Abstracts


SOCIETY OF
CARDIOVASCULAR
ANESTHESIOLOGISTS
Knowledge • Care • Investigation
#SCA2026
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ANNUAL MEETING
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ABSTRACTS
Thursday, April 23 - Sunday, April 26
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SCIENTIFIC PROGRAM ABSTRACTS

Best of Meeting Oral Abstracts and Early Career Investigator Awards: Session 1

Friday, April 24, 2026 | 3:00 PM - 5:00 PM

Best of Meeting Oral Abstracts and Early Career Investigator Awards: Session 1

SCA-BOM1-ECI-OP1

Association of Postoperative Atrial Fibrillation with Cause-Specific In-Hospital Mortality After Cardiac Surgery

Presenting Author: Grace S. Lee, MD

Introduction

Postoperative atrial fibrillation (poAF) is the most common complication after cardiac surgery (1), but its pathophysiology remains debated. Traditional mechanistic explanations focus on cardiac factors such as ischemia–reperfusion injury, postoperative volume shifts, or altered atrial conduction. Emerging evidence suggests that poAF may instead reflect a heightened systemic inflammatory response (2). As dysregulated inflammation drives postoperative sepsis and multiple organ failure (MSOF), we questioned whether poAF is linked to these specific patterns of in-hospital mortality. We conducted this retrospective cohort study to determine whether poAF is associated with sepsis- and MSOF-related cause of death (COD) during index hospitalization. We hypothesized that patients who develop poAF after cardiac surgery would have higher likelihood of sepsis- and MSOF-related death.

Methods

We examined records of 15,256 adults (>20 years) who underwent isolated coronary artery bypass grafting (CABG), CABG + mitral valve repair or replacement (MVR), or CABG + aortic valve repair or replacement (AVR) using cardiopulmonary bypass between 2002–2016 at two tertiary centers (Figure 1). PoAF was defined via established methodology (3). In-hospital deaths were identified via MERLIN platform (4) which supports rapid extraction of EHR data. Discharge summaries were manually reviewed for COD using predefined criteria. CODs were categorized as cardiac, stroke-related, sepsis/MSOF, or other (Table 1). Analyses were restricted to patients who died during index hospitalization. Group differences were evaluated using chi-square or Fisher's exact tests, and unadjusted odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for each category. Time from surgery to death was compared using box-and-whisker plots.

Results

Among 92 in-hospital deaths, 45 (49%) occurred in patients with poAF. The distribution of COD differed between groups (Figure 2). Patients with poAF had a greater incidence of sepsis/MSOF deaths, whereas cardiac deaths were more common among those without poAF. Odds-ratio

analysis (Figure 3) confirmed higher odds of sepsis/MSOF mortality among patients with poAF (OR 4.19, 95% CI 1.05–16.7; p = 0.045). Conversely, odds of cardiac death were lower in patients with poAF (OR 0.34, 95% CI 0.15–0.77; p = 0.030). Patients with poAF also had a longer median time from surgery to death. When stratified by COD, cardiac deaths occurred earliest, while sepsis/MSOF deaths occurred later (Figure 4).

Conclusions

PoAF was associated with higher odds of sepsis/MSOF death and lower likelihood of cardiac death. The timing of events is notable. Although many cardiac deaths occurred during the postoperative window in which poAF appears (days 2–5), cardiac deterioration leading to these deaths began earlier, with ICU care prolonging survival into the poAF window. In contrast, sepsis/MSOF deaths occurred later, consistent with a delayed inflammatory trajectory. These findings suggest that poAF may identify patients particularly susceptible to postoperative inflammatory instability rather than direct cardiac complication. Further studies should investigate these mechanisms and explore targeted preventive or surveillance approaches to improve outcomes.

Image Uploads:

Table 1. Baseline Characteristics of Patients With and Without Postoperative Atrial Fibrillation. *		
Characteristic	**No POAF (n=47)**	**POAF (n=45)**
Demographic characteristics		
Age, mean ± SD	71.0 ± 11.8	76.3 ± 10.2
BMI, mean ± SD	26.7 ± 4.7	27.4 ± 6.7
Sex		
Female	24 (51.1%)	21 (46.7%)
Male	23 (48.9%)	24 (53.3%)
Race		
Caucasian	43 (91.5%)	37 (82.2%)
Asian	2 (4.3%)	4 (8.9%)
Black	1 (2.1%)	3 (6.7%)
Hispanic	1 (2.1%)	1 (2.2%)
Other	0 (0.0%)	0 (0.0%)
Procedure		
CABG	25 (53.2%)	21 (46.7%)
CABG + AVR	18 (38.3%)	16 (35.6%)
CABG + MVR	4 (8.5%)	8 (17.8%)
Emergency surgery		
Emergent	12 (25.5%)	9 (20.0%)
Not emergent	35 (74.5%)	36 (80.0%)
Lung disease		
None	37 (78.7%)	39 (86.7%)
Mild	6 (12.8%)	5 (11.1%)
Moderate	3 (6.4%)	0 (0.0%)
Severe	1 (2.1%)	1 (2.2%)
Diabetes type		
No Diabetes	25 (53.2%)	20 (44.4%)
Non-insulin-dependent diabetes mellitus	14 (29.8%)	16 (35.6%)
Insulin-dependent diabetes mellitus	7 (14.9%)	10 (22.2%)
Smoking Status		
Never	21 (44.7%)	16 (35.6%)
Past	13 (27.7%)	18 (40.0%)
Current smoker	7 (14.9%)	5 (11.1%)
Unknown	6 (12.8%)	6 (13.3%)
Other preexisting conditions		
Hypertension	38 (80.9%)	39 (86.7%)
Hyperlipidemia	37 (78.7%)	39 (86.7%)
Peripheral vascular disease	15 (31.9%)	12 (26.7%)
Unknown	1 (2.1%)	0 (0.0%)

*Note: POAF = Postoperative Atrial Fibrillation, BMI = Body Mass Index, SD = Standard Deviation, CABG = Coronary Artery Bypass Grafting, AVR = Aortic Valve Replacement, MVR = Mitral Valve Replacement, n = Sample size (number of patients)

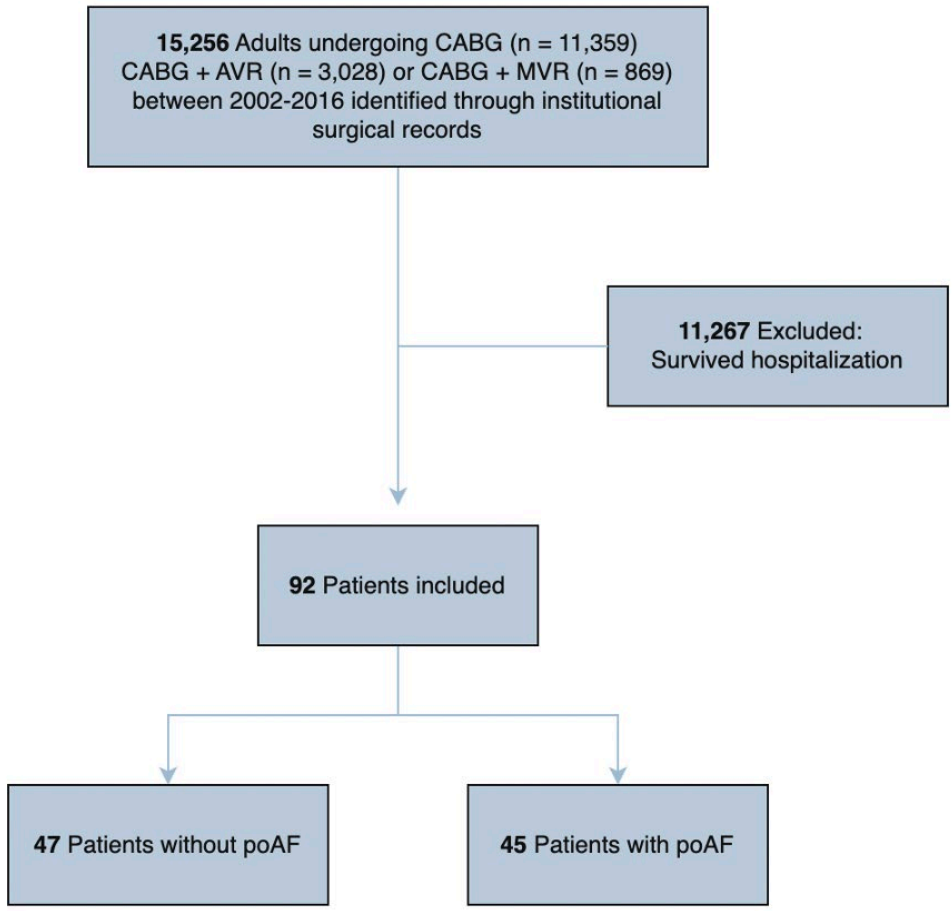


Figure 1. Strengthening the Reporting of Observational studies in Epidemiology (STROBE) flow diagram of patient inclusion for the in-hospital mortality cohort.

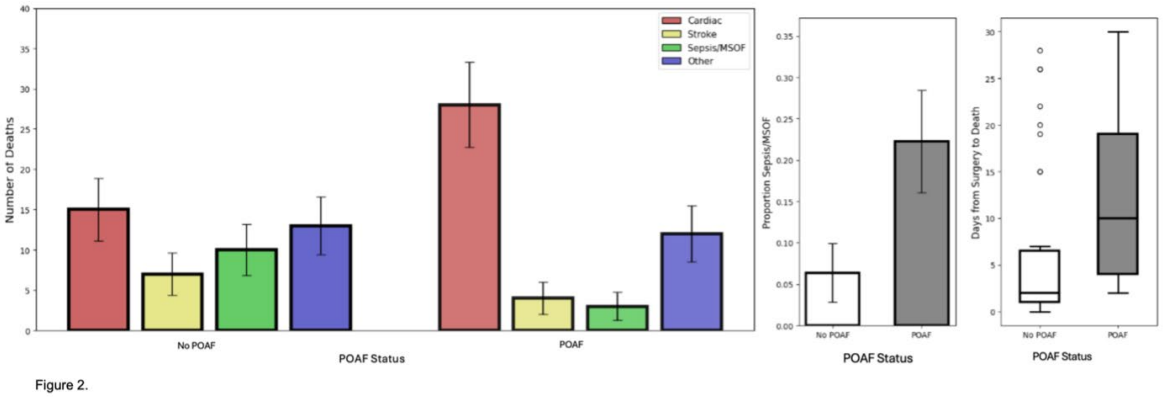


Figure 2.

Left: Distribution of in-hospital deaths by cause, stratified by postoperative atrial fibrillation presence.
Middle: Proportion of in-hospital deaths attributed to sepsis and multiple organ failure by postoperative atrial fibrillation status.
Right: Postoperative interval from surgery to in-hospital death among patients with and without postoperative atrial fibrillation.

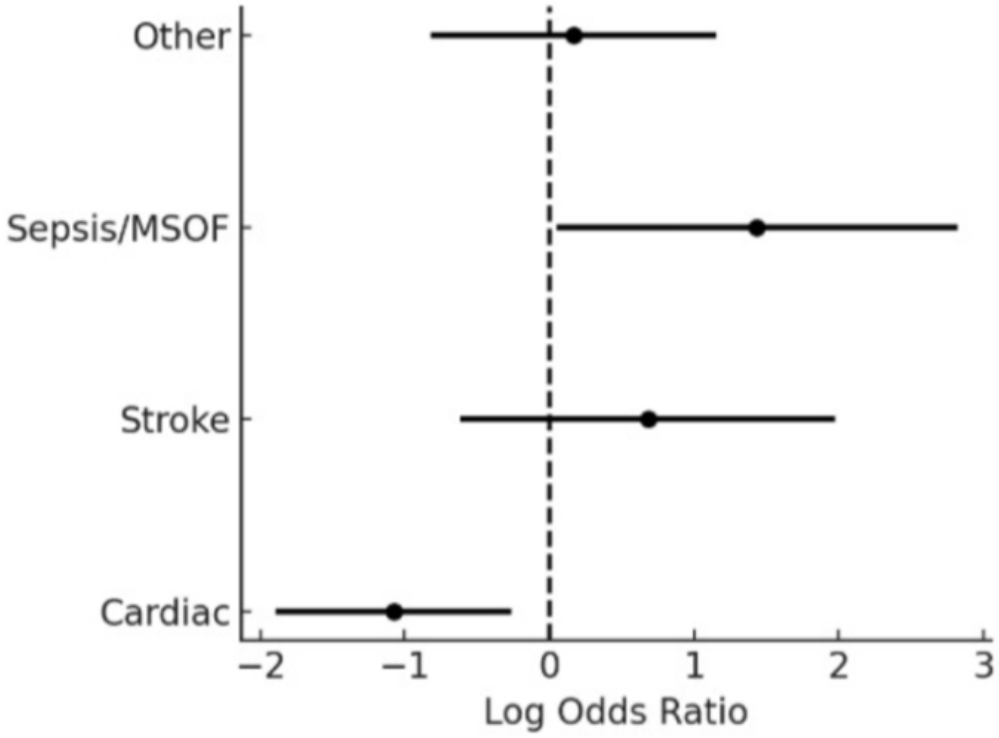


Figure 3. Adjusted log odds ratios for in-hospital mortality in relation to postoperative atrial fibrillation.

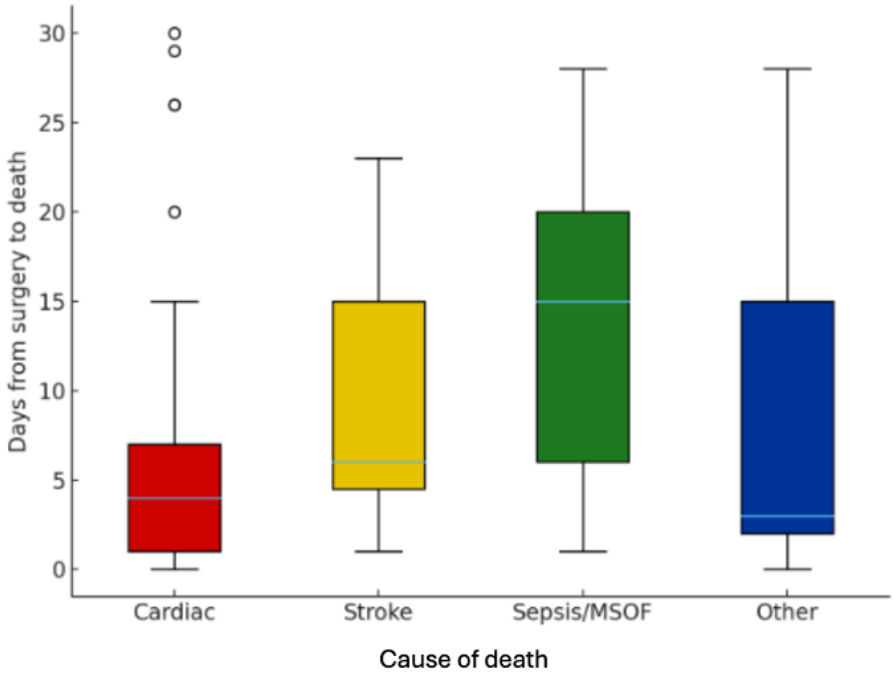


Figure 4. Distribution of time from surgery to in-hospital death by cause of death category.

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SCA-BOM1-ECI-OP2

Effect of an Operating Room Extubation Protocol on Length of Stay After Elective Cardiac Surgery: A Quasi-Experimental Analysis

Presenting Author: Chelsea J. Messinger, MD, PhD

Background: Early postoperative extubation (within 6 hours of leaving the OR) is associated with improved morbidity and mortality after cardiac surgery.[1] Operating-room extubation (ORE) represents the earliest feasible approach, yet existing evidence is inconsistent due to selection bias, center-level heterogeneity, and uncontrolled intraoperative factors.[2-5] In late 2024, we implemented a standardized ORE consideration protocol (Figure 1) for all elective cases, generating a quasi-experimental setting to estimate the protocol's safety and causal effect on post-operative intensive care unit (ICU) and hospital length of stay.

Methods: We used a difference-in-differences design incorporating 5,981 elective cardiac surgical cases requiring cardiopulmonary bypass at two academic centers (November 2022–July 2025). Massachusetts General Hospital (MGH; n=3,455) adopted the protocol; Brigham and Women's Hospital (BWH; n=2,526) served as a contemporaneous control. Transplants, mechanical circulatory support devices, and reoperations for chest washout or other complications were excluded. Outcomes included ICU length of stay, hospital length of stay, 30-day mortality, and reintubation. Effects were estimated via multivariable regression with year-month–clustered standard errors to account for temporal autocorrelation. Identification assumptions were evaluated using pre-policy parallel-trends tests and falsification checks. Sensitivity analyses included minimally adjusted and fully adjusted specifications.

Results: ORE utilization at MGH rose from 4.7% to 18.3% post-policy, whereas BWH increased from 0.6% to 6.8% (Figure 2). Patient demographics and comorbidity profiles were similar across periods; mean age was 64 years, and 71% were male. Within MGH in the post-protocol period, ORE patients were younger and had lower prevalences of major comorbidities and complex operative features (e.g., redo sternotomy, circulatory arrest), though overall case mix at the hospital level was similar pre/post-protocol implementation. The protocol was associated with a nonsignificant reduction in ICU length of stay (-3.5 hours; 95% CI -17.0 to 9.9) and hospital length of stay (-14.0 hours, 95% CI -32.4 to 4.5) (Figure 3). Effect estimates were directionally consistent across model specifications, with no meaningful attenuation after covariate adjustment. There was no effect on 30-day mortality (risk difference 0.3%; 95% CI −1.2% to 1.7%). Among MGH ORE patients,

reintubation rates were low pre- and post-protocol implementation (3.2% vs 2.2%, respectively). Parallel-trends assumptions were satisfied for all endpoints, and falsification checks for the primary outcome supported design validity.

Conclusions: Protocolized ORE implementation substantially increased ORE use without evidence of increased mortality or reintubation, supporting safety and operational feasibility in well-selected elective cases. Although the study was underpowered to detect small differences in length of stay, effect estimates favored modest reductions. This natural experiment provides rigorous, causally interpretable evidence on the safety profile of ORE protocols and underscores the need for randomized trials to determine comparative effectiveness of ORE versus early ICU extubation.

Image Uploads:

Operating Room Extubation Protocol

PATIENT SELECTION CRITERIA

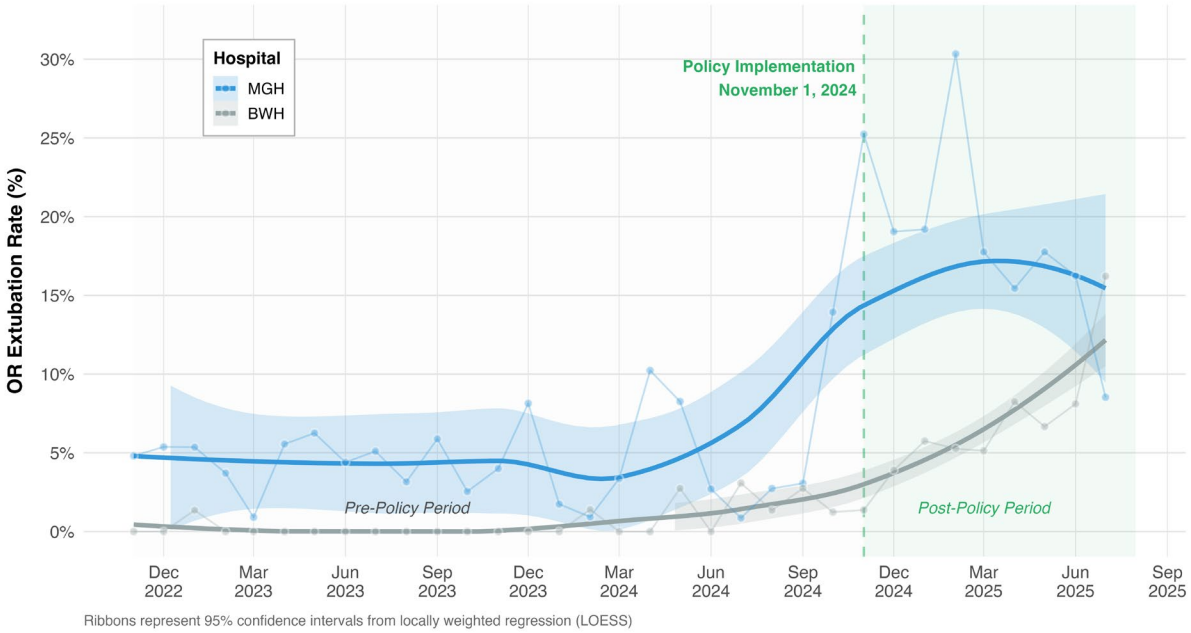
Category	Details
Eligible Procedures	CABG; AVR; MVR; MVr; Aortic Surgery (Non-Urgent); Other (Congenital, Mass Excisions, etc.)
Patient Exclusions (Relative)	Age >75; EF <50%; CKD ≥ Stage 3a; Redo; BMI >45; COPD; IABP; Neuromuscular Disease; Compromised Pre-op O_2; Difficult Airway; Significant Frailty
Procedure Exclusions (Relative)	Bleeding Concern; Vulnerable Graft; High-Level Vasopressors/Inotropes; Persistent EKG Ischemia
Briefing	Plan for OR extubation discussed at pre-operative huddle

PERIOPERATIVE PROTOCOL

Phase	Protocol Elements
Pre-Anesthesia	Minimize Midazolam; Avoid Long-Acting Sedatives (e.g., Gabapentin)
Intra-Operative	Early Narcotic Dosing; BIS Monitoring; Continuous Muscle Relaxation Monitoring; Room/Forced Air Warming Near End of Case; Avoid Deep Cooling; TV ≤ 8 ml/kg (IBW); Intra-op Acetaminophen
Pre-Extubation	Warm to 35°C (Bladder); Muscle Relaxant Reversal; Local Anesthetic at Incision Site; Administer 2 Antiemetics
Post-Extubation/ICU	Per Protocol Management; Early PT Evaluation

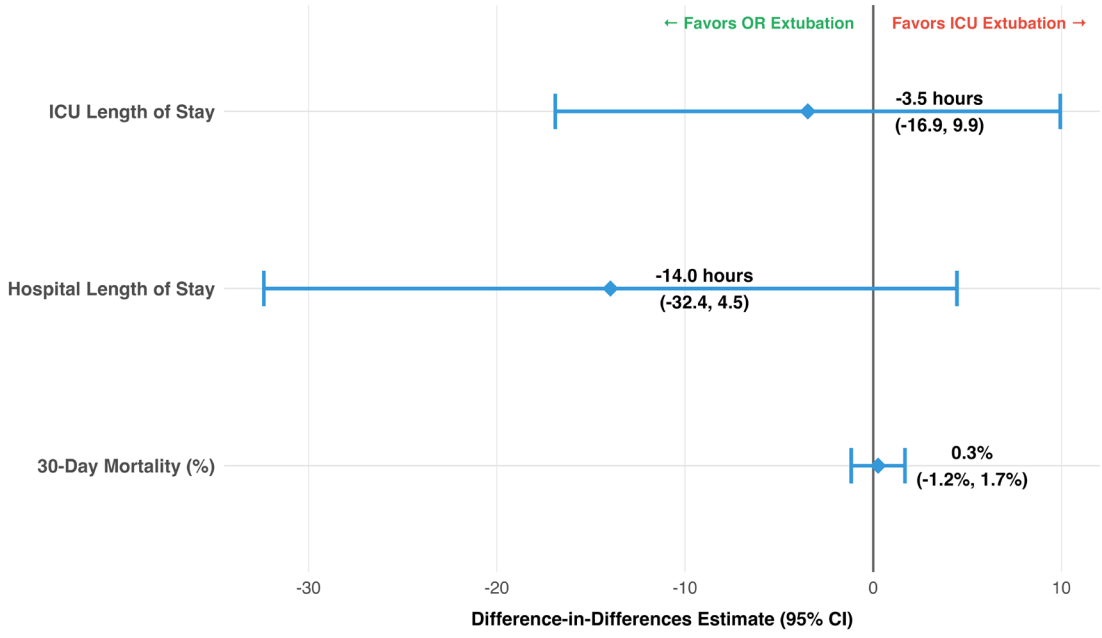
Abbreviations: AVR, aortic valve replacement; BMI, body mass index; BIS, bispectral index; CABG, coronary artery bypass grafting; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; EF, ejection fraction; EKG, electrocardiogram; IABP, intra-aortic balloon pump; IBW, ideal body weight; ICU, intensive care unit; MVR, mitral valve replacement; MVr, mitral valve repair; O_2, oxygenation; PT, physical therapy; TV, tidal volume

Natural Experiment: Operating Room Extubation Adoption



Ribbons represent 95% confidence intervals from locally weighted regression (LOESS)

Difference-in-Differences Analysis: Effect of OR Extubation Protocol



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SCA-BOM1-ECI-OP3

Tricuspid Annular Dynamism And Tenting Geometry As Surrogates Of Right Ventricular–Pulmonary Arterial Coupling In Cardiac Surgery

Presenting Author: Vahid Kiarad, MD, MPH

Background:

Right ventricular–pulmonary arterial (RV–PA) coupling is a physiologic marker of right ventricular adaptation to afterload and an important determinant of perioperative outcomes.[1] 3D assessment of dynamic tricuspid annular geometry is increasingly being studied in the context of valve interventions.[2] Whether intraoperative tricuspid annular dynamism and tenting morphology track with invasively measured RV–PA coupling remains unknown. This study investigated whether intraoperative tricuspid annular dynamism metrics are associated with invasively derived RV–PA coupling and whether these geometric parameters can discriminate between normal and impaired coupling in patients undergoing cardiac surgery.

Methods:

In a prospective observational cohort of adult cardiac surgery patients undergoing simultaneous intraoperative TEE and pulmonary artery catheter monitoring, RV pressure-volume data were obtained and subjected to single-beat end-systolic pressure–volume analysis using the V_0 method.[3] Comprehensive 2D and 3D tricuspid annular datasets were acquired, and dynamic geometric indices-including annulus area, annulus area change, tenting height, tenting volume, perimeter, and axis dimensions were quantified (using the automated tricuspid quantification software of GE Healthcare System) and indexed to body surface area. (Figure 1) Low coupling was defined as Ees/Ea < 0.8. Group comparisons, nonlinear regression, and ROC analyses were used to evaluate associations between 3D dynamic indices and RV PA coupling.

Results:
Fifty-three patients were included; 17 (32%) exhibited low RV–PA coupling. Baseline demographic, comorbidity, perioperative characteristics, and postoperative outcomes did not differ significantly between groups. (Figure 2) Two tricuspid dynamism parameters distinguished low from preserved coupling. Indexed tenting volume was significantly higher in the low-coupling group (1.04 ± 0.60 vs 0.72 ± 0.48 mL, p = 0.044), while annulus area change was significantly reduced (11.9 ± 5.2% vs 15.8 ± 5.9%, p = 0.025). (Figure 3) Nonlinear regression demonstrated strong relationships between coupling and both parameters: a positive nonlinear association for annulus area change (R^2 = 0.96) and an inverse relationship for indexed tenting volume (R^2 = 0.95). (Figure 4) ROC analyses showed moderate discriminatory performance. Annulus area change yielded an AUC of 0.683 with an optimal cutoff < 11%, while indexed tenting volume had an AUC of 0.671 with a threshold >0.712 mL/m^2 for identifying patients with impaired RV PA coupling (Ees/Ea < 0.8) (Figure 5).

Conclusion:

Intraoperative tricuspid annular dynamism, particularly reduced annulus area change and increased indexed tenting volume, is associated with impaired RV-PA coupling during cardiac surgery. These geometric indices demonstrate moderate ability to discriminate abnormal coupling and may provide practical echocardiographic surrogates when invasive pressure-volume analysis is not feasible. Further validation in larger cohorts is warranted to determine their utility for intraoperative physiologic risk stratification.

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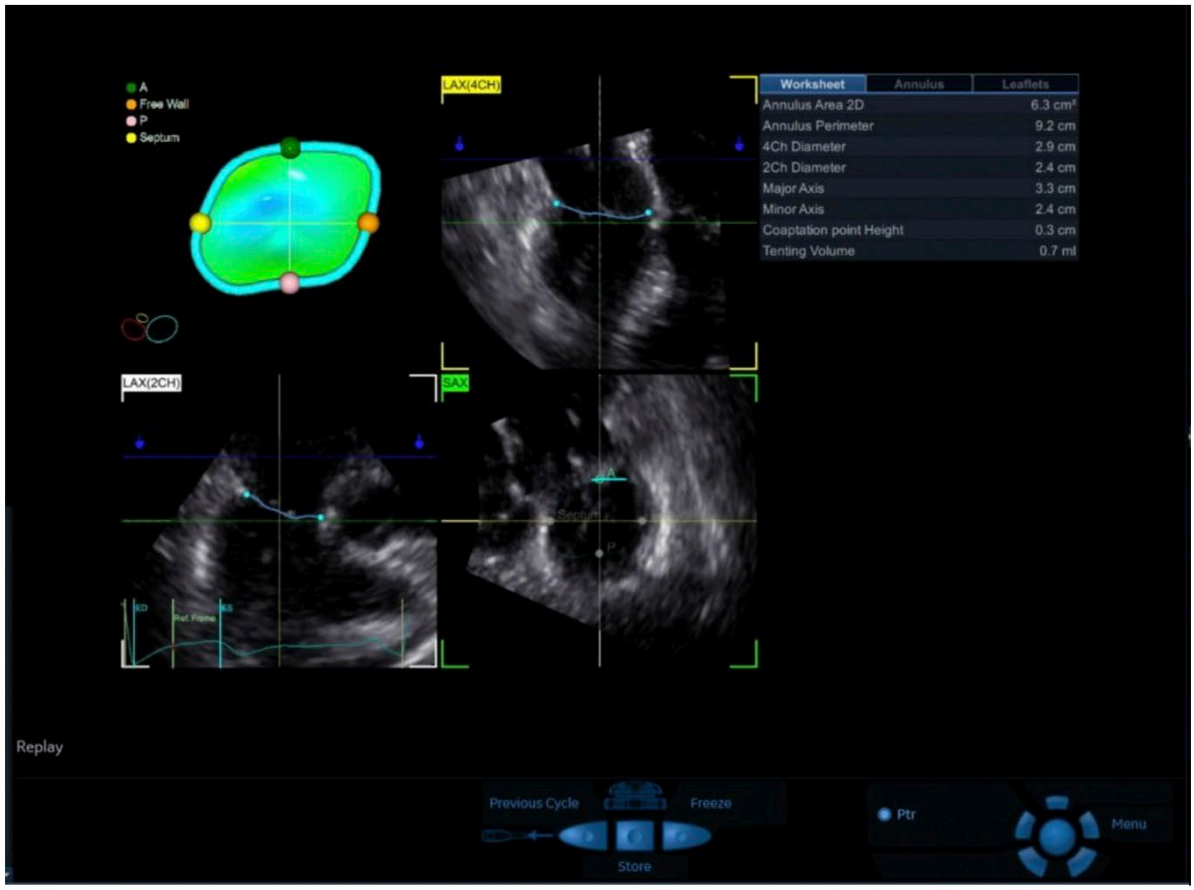


Figure 1: Tricuspid Quantification software in GE Healthcare

Variable	Overall (n=53)	Normal RV–PA Coupling (n=36)	Low RV–PA Coupling n=17)	p-value
Demographics				
Age, mean (SD)	68.2 (10.8)	69.3 (11.0)	65.9 (10.1)	0.28
Male, n (%)	39 (73.6%)	25 (69.4%)	14 (82.4%)	0.32
Race, n (%)				0.54
White	30 (56.6%)	22 (61.1%)	8 (47.1%)	
Black	8 (15.1%)	4 (11.1%)	4 (23.5%)	
Asian	4 (7.5%)	2 (5.5%)	2 (11.8%)	
Other	7 (13.2%)	5 (13.8%)	2 (11.8%)	
Baseline characteristics				
Diabetes mellitus, n (%)	14 (26.4%)	8 (22.2%)	6 (35.2%)	0.34
Hypertension, n (%)	35 (66%)	24 (66.6%)	11 (64.7%)	0.65
Chronic lung disease, n (%)	6 (11.3%)	4 (11.1%)	2 (11.7%)	0.97
Preop creatinine, mean (SD)	1.17 (0.84)	1.23 (0.98)	1.00 (0.23)	0.42
Previous CABG, n (%)	1 (1.8%)	1 (2.7%)	0 (0.0%)	0.48
Previous valve surgery, n (%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	—
Perioperative characteristics				
TR severity, n (%)				0.82
None	33 (62.2%)	23 (63.8%)	10 (58.8%)	
Mild	10 (8.8%)	7 (19.4%)	3 (17.6%)	
Moderate	4 (8.2%)	2 (6.1%)	2 (12.5%)	
Severe	2 (3.7%)	1 (2.7%)	1 (5.8%)	
Surgery type, n (%)				0.98
CABG	24 (45.2%)	16 (44.4%)	8 (47.1%)	
CABG + Valve	8 (15.1%)	5 (13.8%)	3 (21.4%)	
Single valve	14 (26.4%)	10 (27.7%)	4 (23.5%)	
Multiple valves	3 (5.6%)	2 (5.5%)	1 (5.8%)	
Perfusion time (min), mean (SD)	95.3 (44.1)	92.7 (38.7)	101.8 (56.5)	0.55
Outcomes				
STS Major morbidity & mortality, n (%)	6 (11.3%)	6 (16.6%)	0 (0.0%)	0.07
30-day mortality, n (%)	2 (3.8%)	2 (5.5%)	0 (0.0%)	0.32
Postop stroke, n (%)	2 (3.8%)	2 (5.5%)	0 (0.0%)	0.32
Renal failure, n (%)	3 (5.7%)	3 (8.3%)	0 (0.0%)	0.21
Prolonged ventilation, n (%)	2 (3.8%)	2 (5.5%)	0 (0.0%)	0.32
Deep sternal infection, n (%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	—
Reoperation for ischemia, n (%)	1 (1.8%)	1 (2.7%)	0 (0.0%)	0.48
Prolonged pressor/inotrope, n (%)	25 (47.2%)	14 (38.9%)	11 (64.7%)	0.08
ICU LOS (hours), mean (SD)	45.4 (30.0)	48.9 (32.4)	37.1 (22.5)	0.26
Hospital LOS (days), mean (SD)	6.8 (8.5)	7.8 (10.0)	4.8 (2.6)	0.28
Ventilation time (hours), mean (SD)	17.4 (51.6)	21.2 (61.1)	8.2 (6.9)	0.47

Figure 2: Baseline and Demographic Characteristics

Variable	Normal RV–PA coupling (mean ± SD)	Low RV–PA coupling (mean ± SD)	p-value
Tricuspid dynamism (BSA-indexed)			
Sphericity index	76.4 ± 10.6	79.7 ± 6.5	0.243
BSA-indexed coaptation point height (cm)	0.302 ± 0.325	0.247 ± 0.106	0.514
BSA-indexed maximum tenting height (cm)	0.293 ± 0.092	0.318 ± 0.116	0.409
BSA-indexed tenting volume (mL)	0.720 ± 0.481	1.044 ± 0.598	**0.044**
BSA-indexed annulus area 3D (cm^2)	5.464 ± 1.997	5.785 ± 1.236	0.546
BSA-indexed annulus area 2D (cm^2)	5.255 ± 1.864	5.639 ± 1.178	0.441
Annulus area change (%)	15.78 ± 5.89	11.94 ± 5.16	**0.025**
BSA-indexed annulus perimeter (cm)	6.081 ± 1.161	6.177 ± 0.714	0.757
BSA-indexed 4CH diameter (cm)	1.838 ± 0.295	1.875 ± 0.228	0.646
BSA-indexed 4CH diastolic diameter (cm)	1.913 ± 0.287	1.909 ± 0.219	0.958
BSA-indexed 2CH diameter (cm)	1.699 ± 0.508	1.708 ± 0.267	0.947
BSA-indexed major diastolic axis (cm)	2.174 ± 0.407	2.171 ± 0.240	0.981
BSA-indexed major axis (cm)	2.119 ± 0.411	2.122 ± 0.233	0.977
BSA-indexed minor axis (cm)	1.619 ± 0.382	1.687 ± 0.246	0.506
BSA-indexed excursion (cm)	0.480 ± 0.210	0.439 ± 0.199	0.502

Figure 3: Dynamism Parameters

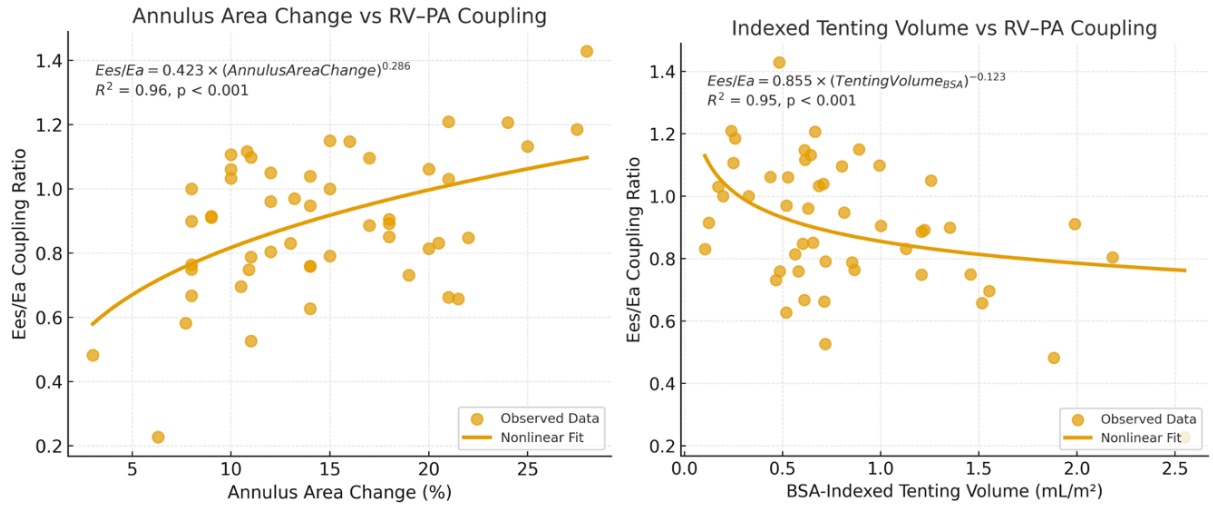


Figure 4: Non-linear associations between RV PA coupling ratio and a)Annulus area change and b) Tenting volume indexed to body surface area

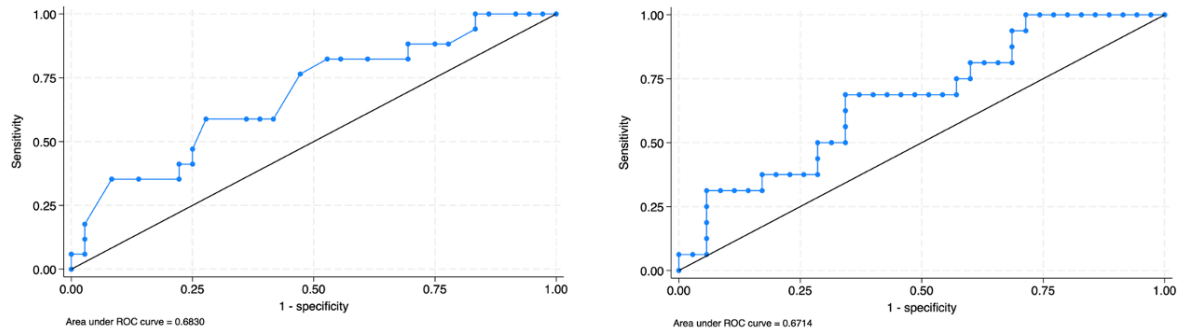


Figure 5 : ROC Curve Analysis used to arrive at optimal cutoffs (Youden index) for dynamism parameters to predict low RV PA Coupling (<0.8)

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SCA-BOM1-BOM-OP4

Oxytocin neuromodulation reduces ventricular arrhythmias post-MI

Presenting Author: Jessica Diaz-Rivera, MD

Background:

Maladaptive autonomic remodeling that occurs following chronic myocardial infarction (MI) increases spinal neural excitability, propagates sympathetic overdrive, and predisposes to ventricular tachyarrhythmia (VT). Our prior work suggests that neuromodulation via spinal cord stimulation (SCS) and pharmacologic therapy can reduce autonomic remodeling and cardiac sympathoexcitation, thus providing a novel therapy for VTs (1-3). Intranasal (IN) OXT is a non-invasive, existing pharmacotherapy with demonstrated safety, and verified central nervous system effects, including spinal cord mechanisms (4-6). Preliminary studies show that exogenous OXT may reduce acute ischemia-reperfusion (IR)-induced sympathoexcitation and cardiac arrhythmias but its long-term impact on spinal neural networks and myocardial excitability is yet to be established. We aimed to test the hypothesis that long-term reactive OXT therapy vs. SCS reduces adverse cardiospinal neural network remodeling and VTs after MI in a porcine model.

Methods:

Twenty-four Yorkshire pigs were randomized to healthy control (n=4), chronic MI (n=5), chronic MI + IR (n=5), chronic MI + IR + long-term SCS (n=5), or chronic MI + IR + long-term IN-OXT (n=5). MI was induced by left circumflex microsphere injection with 5-week maturation. SCS (T1–T4, 50 Hz, 0.4 ms) or daily IN-OXT (20U) began 24 h post-MI and continued to terminal study. Acute LAD IR (1-h occlusion, 2-h reperfusion) was used to evaluate sympathoexcitation and VT risk. High-density mapping quantified activation-recovery interval (ARI) and VT burden. T2–T3 spinal segments underwent in-situ RNA hybridization for OXT receptor (OXTR), neuronal (Rbfox3), microglial (CXCR3), astrocytic (GFAP), excitatory (TLX3), inhibitory (Pax2), and apoptotic (CC3) markers. Cell populations and colocalization were quantified in QuPath. One-way ANOVA with Tukey testing compared groups.

Results:

Chronic MI + acute IR increased sympathoexcitation (decrease in delta ARI) and ventricular arrhythmias (VT+VF), which were mitigated with long-term SCS and IN OXT (Fig 1). With respect to spinal cord neural remodeling, chronic MI and IR led to: increased microglia expression and

increased apoptosis in neurons and astrocytes (Figs 2A,B,C). While long-term SCS and IN OXT, during MI + IR, decreased reactive gliosis, as well as apoptosis. MI + IR alone also led to decreased OXTR expression in neurons overall, (RBFOX3+OXTR) but an increase in excitatory neuronal expression (RBFOX3+ OXTR+ TLX3), (Fig2 D,E). Importantly, SCS and IN OXT therapies mitigated these changes by increasing OXTR expression in neurons and increasing OXTR+ inhibitory+ neuronal expression (Fig 2F)

Conclusion:

In this study, we demonstrate that chronic MI+IR induced myocardial injury leads to adverse neural remodeling in the thoracic spinal cord, and a decrease in OXTR expression. Chronic reactive thoracic SCS neuromodulation, as well as, non-invasive reactive chronic IN OXT therapy, mitigated these molecular changes and reduced myocardial sympathoexcitation and ventricular arrhythmias. Continued work on OXT expression and influence in the spinal cord could lead to potential treatments for ischemia-induced autonomic dysregulation in chronic cardiac disease.

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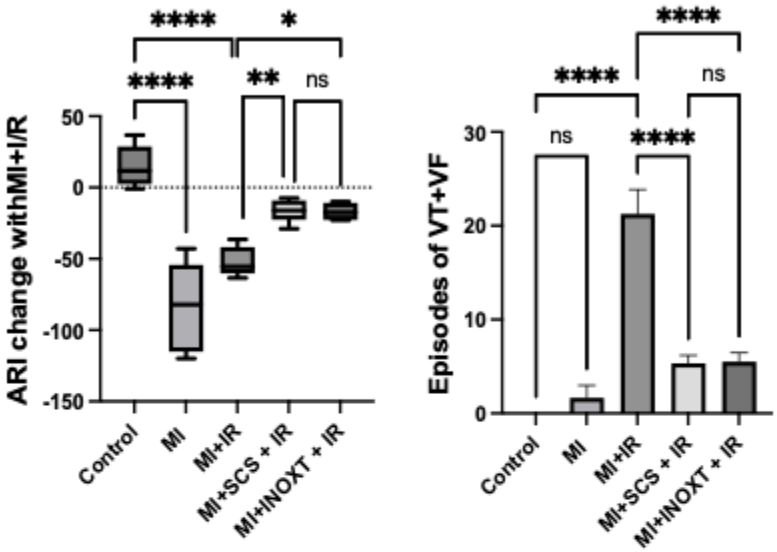


Figure 1. Cardiac Electrophysiology: A. Chronic MI and acute I/R increased sympathoexcitation as demonstrated by a reduction in ARI duration and **B.** increased episodes of ventricular tachycardia (VT) and ventricular fibrillation (VF). Reactive long-term SCS and IN OXT treatment reduced sympathoexcitation and decreased ventricular arrhythmias. Data presented at mean and SE, *p<0.05, **p<0.01, ****p<0.0001, one-way ANOVA & post-hoc Tukey)

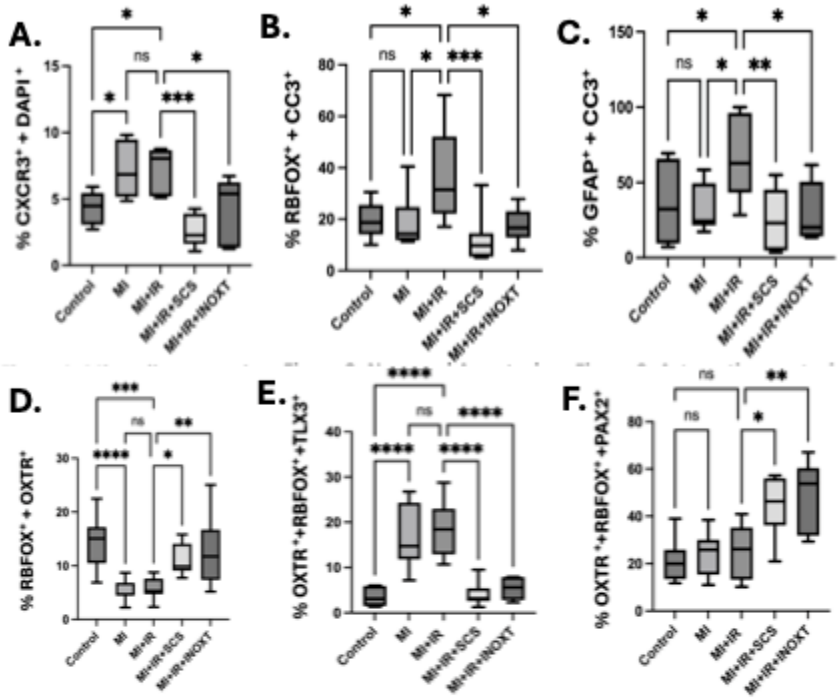


Figure 2. Neural Remodeling: In-situ RNA hybridization on Thoracic (T2-T3) spinal cord sections demonstrated that Chronic MI + acute IR: A. increased microglia expression (with no change in neuron or astrocyte expression), **B.** increased apoptosis in neurons and **C**. increased apoptosis in astrocytes. All of which were decreased with long-term SCS and IN OXT therapy. **D.** Chronic MI + IR alone also led to decreased OXTR expression in neurons overall and E. an increase in excitatory cells. **E,F.** SCS and IN OXT therapies mitigated these changes by increasing OXTR expression in neurons back to baseline and increasing OXTR+ inhibitory neuron expression. Data expressed as mean and 5–95% CI, *p<0.05, **p<0.01, ****p<0.0001, one-way ANOVA & post-hoc Tukey)

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SCA-BOM1-BOM-OP5

Oxytocin neuromodulation reduces ventricular arrhythmias post-MI

Presenting Author: Una Choi, MD

Background: Tranexamic acid (TXA) and ε-aminocaproic acid (EACA) are anti-fibrinolytic medications commonly used to control or prevent bleeding during cardiac surgery with cardiopulmonary bypass. Despite their similarities, few studies to date have directly compared these anti-fibrinolytics for outcomes other than blood loss or transfusion risk.[1,2] We aimed to explore differences between these medications among a wider range of clinical outcomes.
Methods: We utilized a large administrative claims and electronic health records database to conduct a retrospective cohort study of patients undergoing cardiovascular surgery with cardiopulmonary bypass who received either (1) TXA without EACA, or (2) EACA without TXA on the day of surgery. We queried the network up to November 2025 for eligible patients at least 18 years of age. We propensity score matched patients based on characteristics, type of cardiovascular surgery, hemoglobin on the day of surgery, and comorbidities including multiple forms of heart disease, aortic aneurysm, chronic kidney disease, lipid disorders, hypertension, neoplasms,

nicotine dependence, pulmonary disease, overweight and obesity, type 2 diabetes, and use of anticoagulation medications. After matching, we compared cohorts for 14-day and 30-day risk of mortality, acute kidney injury (AKI), thrombotic complications such as pulmonary embolism, stroke, acute myocardial infarction (MI), and seizure. Inclusion and exclusion criteria, matching comorbidities, and outcomes were identified using administrative codes such as from the International Classification of Diseases 10th edition (ICD-10).

Results: In total, we identified 48,738 patients who received TXA without EACA and 85,712 patients who received EACA without TXA. After matching, there were 46,503 patients in both cohorts. For 14-day outcomes, we found a significant reduction among the TXA cohort in risk for AKI (16.68% vs. 18.09%, Relative Risk (RR) 0.92 [95% Confidence Interval 0.90-0.95]), thrombotic complications (5.08% vs. 6.03%, RR 0.84 [0.80-0.89]), stroke (4.08% vs. 4.58%, RR 0.89 [0.84-0.94]), and acute MI (14.95% vs. 16.69%, RR 0.90 [0.87-0.92]) compared to the EACA cohort.

For 30-day outcomes, we found the TXA cohort to similarly have a reduction in risk for AKI (18.93% vs. 20.20%, RR 0.94 [0.91-0.96]), thrombotic complications (6.51% vs. 7.38%, RR ti0.88 [0.84-0.92]), stroke (4.74% vs. 5.33%, RR 0.89 [0.84-0.94]), and acute MI [17.35% vs. 18.75%, RR 0.92 [0.90-0.95]). The risk for seizures was significant at the 30-day mark, showing an increase in risk among the TXA cohort (2.03% vs. 1.82%, RR 1.12 [1.02-1.22]).

Conclusion: Among patients undergoing cardiac surgery with cardiopulmonary bypass, we found TXA had a lower risk for multiple post-operative outcomes than EACA, highlighting the potential to improve post-operative outcomes in high-risk surgeries. Similar to prior randomized controlled trials, our study suggests TXA may carry an associated risk of post-operative seizures.

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Table 1. 14-day and 30-day outcomes for patients undergoing cardiac surgery with cardiopulmonary bypass and given exclusively TXA or EACA.

	14-day outcomes					
	TXA	EACA	RR	95% CI		P-value
Patients before match	48738	85712	--	--	--	--
Patients after match	46503	46503	--	--	--	--
Post-match age at surgery (years)	63.0±12.5	62.9±12.5	--	--	--	--
Mortality	2.25%	2.35%	0.96	0.88	1.04	0.31
Acute kidney injury	16.68%	18.09%	0.92	0.90	0.95	<0.0001
Thrombotic complication	5.08%	6.03%	0.84	0.80	0.89	<0.0001
Stroke	4.08%	4.58%	0.89	0.84	0.94	0.0001
Acute MI	14.95%	16.69%	0.90	0.87	0.92	<0.0001
Seizure	1.72%	1.58%	1.09	0.98	1.20	0.11
	30-day outcomes					
	TXA	EACA	RR	95% CI		P-value
Patients before match	48738	85712	--	--	--	--
Patients after match	46503	46503	--	--	--	--
Post-match age at surgery (years)	63.0±12.5	62.9±12.5	--	--	--	--
Mortality	3.01%	3.19%	0.94	0.88	1.02	0.12
Acute kidney injury	18.93%	20.20%	0.94	0.91	0.96	<0.0001
Thrombotic complication	6.51%	7.38%	0.88	0.84	0.92	<0.0001
Stroke	4.74%	5.33%	0.89	0.84	0.94	<0.0001
Acute MI	17.35%	18.75%	0.92	0.90	0.95	<0.0001
Seizure	2.03%	1.82%	1.12	1.02	1.22	0.018

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SCA-BOM1-BOM-OP6

Association of Preoperative Lacunar Infarction with Delirium and Stroke after Acute Type A Aortic Dissection Repair

Presenting Author: Yu-Hua K. Cheng, MD

Background

So far, little is known about preoperative lacunar infarct (PLI) and its association with neurologic deficits in patients with acute type A dissection (ATAAD). We seek to study the impact of PLI on delirium and stroke after ATAAD repair.

Methods

Among 1304 adults undergoing aortic surgery from 01/2016–10/2024, 795 had preoperative non-contrast brain CT. After excluding left carotid or innominate involvement and other conditions, final analysis included 241 patients with ATAAD, who were divided into PLI and no PLI groups (Fig 1). PLIs were categorized into 5 groups based on their predominant location: 1) thalamus, 2) basal ganglia and adjacent areas (internal or external capsule), 3) deep white matter (centrum semiovale and corona radiata), 4) brainstem and cerebellum, and 5) subcortical regions (frontal, occipital or insular).

Primary outcomes were postoperative stroke and delirium. Binary logistic or modified Poisson regression were used to assess the association of PLI with delirium and stroke.

Results

Mean age was 54.8±12.7 years (26.1% ≥65 years; 69 women, 28.6%). Hypertension was seen in 169 patients (70.1%), smoking in 129 (53.5%) and prior cerebrovascular accident (CVA) in 14 (5.8%). Operations included 45 ascending and/or root repairs (18.7%), 53 hemiarch repairs (22.0%) and 143 total arch repairs (TAR) (59.3%). Mean cardiopulmonary bypass (CPB) time was 222±84 min. Hypothermic circulatory arrest (HCA) was used in 191 (79.2%). Nadir temperature was 26.9±2.2°C (Table 1).

Preoperative lacunar infarct was detected in 142 patients (58.9%), who tended to have more CVA (9.2% vs 1%; *P*=0.004) than no PLI group. Locations of LI were thalamus in 4 (2.8%), basal ganglia and adjacent areas in 82 (57.7%), deep white matter in 7 (4.9%), brainstem and cerebellum in 17 (12%) and subcortical lobar regions in 32 (22.5%) (Table 2).

Postoperative delirium occurred in 29.5% (71/241) for the whole series, which was 31.7% (45/142) in PLI group and 26.3% (26/99) in no PLI group (*P*=0.392) (Table 3). The incidence varied significantly with the locations of PLI (*P*=0.049), which was highest in deep white matter (57.1%), followed by brainstem (52.9%), and lowest in basal ganglia (25.6%).

Stroke occurred in 5% (12/241), which was 7% (10/172) in PLI group vs 2% (2/99) in no PLI group (*P*=0.093).

Predictors of postoperative delirium were lacunar infarct in deep white matter (odds ratio [OR] 4.86; 95% confidence interval [CI] 1.14–25.11; *P*=0.038), TAR (OR 3.68, 95% CI 1.47–10.84; *P*=0.009) and CPB time (min) (OR 1.005; 95% CI 1.002–1.009; *P*=0.006). Lacunar infarcts in subcortical lobar

regions (frontal/occipital/insular) were predictive of stroke (OR 16.83; 95% CI 1.87–154.43; *P*=0.007). Prior CVA was not associated with delirium or stroke.

Conclusion

Preoperative lacunar infract was detected in 59% of patients with ATAAD. Such patients showed a higher incidence of postoperative stroke and delirium, albeit non-significant. Lacunar infarcts involving the brainstem were associated with higher risk of delirium, while lacunar infarcts in subcortical lobar regions (frontal/occipital/insular) were linked to increased risk of stroke. Reviewing preoperative brain CT scan for lacunar infarcts may identify predictive clues to stroke and delirium after ATAAD repair.

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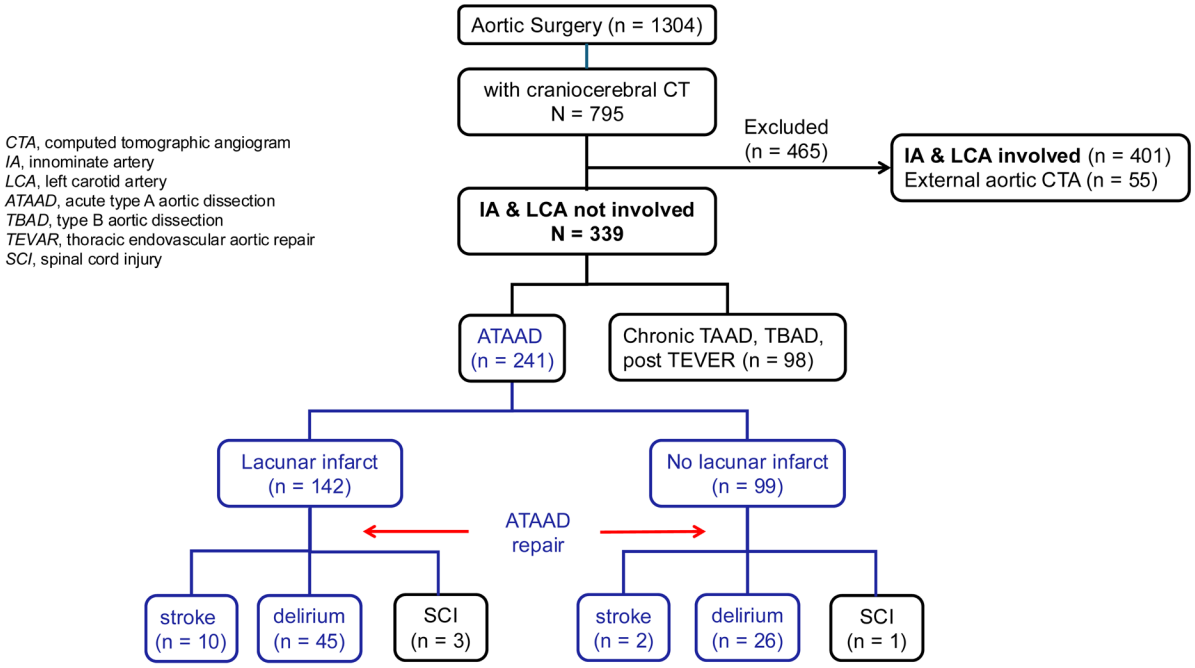


Table 1. Baseline Characteristics and Operative Data by Preoperative Lacunar Infarct

Variable	Entire cohort (n = 241, %)	PLI (n = 142, 58.9%)	No PLI (n = 99, 41.1%)	*P* value
Baseline characteristics				
Age, year	54.8 ± 12.7	59.2 ± 11.3	48.4 ± 11.9	<0.001
≥ 65 years	63 (26.1)	54 (38.0)	9 (9.1)	<0.001
Male	172 (71.4)	100 (70.4)	72 (72.7)	0.773
Body mass index, kg/m^2	23.9 ± 4.3	23.8 ± 4.1	24.0 ± 4.5	0.404
Hypertension	169 (70.1)	109 (76.8)	60 (60.6)	0.010
Ever smoker	129 (53.5)	76 (53.5)	53 (53.5)	0.891
Alcohol use	73 (30.3)	43 (30.3)	30 (30.3)	0.878
Prior cerebrovascular accident	14 (5.8)	13 (9.2)	1 (1.0)	0.004
stroke	11 (4.6)	10 (7.0)	1 (1.0)	0.030
intracerebral hemorrhage	3 (1.2)	3 (2.1)	0 (0)	0.271
Diabetes mellitus	9 (3.7)	7 (4.9)	2 (2.0)	0.315
Previous cardiac surgery	33 (13.7)	21 (14.8)	12 (12.1)	0.704
Coronary artery disease	13 (5.4)	10 (7.0)	3 (3.0)	0.857
Chronic kidney injury	6 (2.5)	5 (3.5)	1 (1.0)	0.405
LVEF, %	50.6 ± 4.3	50.6 ± 3.4	50.6 ± 5.4	0.977
LVEDD, mm	52.1 ± 8.7	49.6 ± 6.6	55.7 ± 10.1	<0.001
Beta-blocker use	171 (71.0)	103 (72.5)	68 (68.7)	0.565
Preoperative cardiac tamponade	46 (19.1)	32 (22.5)	14 (14.1)	0.426
Cardiogenic shock	2 (0.8)	2 (1.4)	0 (0)	0.475
Syncope	3 (1.2)	2 (1.4)	1 (1.0)	0.475
Operative data				
CPB time, min	221.5 ± 83.9	215.9 ± 83.3	229.4 ± 84.6	0.724
Flow rate, L/min/m^2	2.71 ± 0.14	2.73± 0.15	2.69 ± 0.12	0.010
Cross-clamp time, min	135.6 ± 47.5	131.5 ± 43.6	141.2 ± 52.0	0.983
HCA time, min	22 (16–30)	21 (16–30)	22 (16–30)	0.977
HCA temperature, °C	26.9 ± 2.2	27.2 ± 2.2	26.4 ± 2.2	0.018
Cerebral protection				0.106
unilateral ACP	108 (44.8)	66 (46.5)	42 (42.4)	
bilateral ACP	14 (5.8)	12 (8.5)	2 (2.0)	
retrograde cerebral perfusion	69 (28.6)	36 (25.4)	33 (33.3)	
Second CPB run	8 (3.3)	6 (4.2)	2 (2.0)	0.476
Aortic procedure				0.309
ascending and/or root repair	45 (18.7)	24 (16.9)	21 (21.2)	
hemiarch repair	53 (22.0)	28 (19.7)	25 (25.3)	
total arch repair	143 (59.3)	90 (63.4)	53 (53.5)	

Values are expressed as mean ± standard deviation, median (interquartile range) or n (%)
PLI, preoperative lacunar infarct; *LVEF*, left ventricular ejection fraction; *LVEDD,* left ventricular end-diastolic diameter; *CPB*, cardiopulmonary bypass; *HCA*, hypothermic circulatory arrest*; ACP*, antegrade cerebral perfusion

Table 2. Association of lacunar infarct location and prior CVA with postoperative stroke and delirium

Predominant location of lacunar infarct	Number of patients (n = 191, %)	Prior CVA, n (%)	Delirium, n (%)	Stroke, n (%)
Thalamus	4 (2.8)	1 (25.0)	2 (50.0)	1 (25.0)
Basal ganglia and adjacent areas (internal and external capsules)	82 (57.7)	5 (6.1)	21 (25.6)	4 (4.9)
Deep white matter	7 (4.9)	0 (0)	4 (57.1)	1 (14.3)
Brainstem and cerebellum	17 (12.0)	6 (35.3)	9 (52.9)	1 (5.9)
Subcortical lobar regions (frontal/occipital/insular)	32 (22.5)	1 (3.1)	9 (28.1)	3 (9.4)
Total	142 (100)	13 (9.2)	45 (31.7)	10 (7.0)
P value	Not applicable	0.012	0.049	0.680

Values are expressed as n (%)

CVA, cerebrovascular accidents (including stroke and intracerebral hemorrhage)

Table 3. Operative Outcomes by Preoperative Lacunar Infarct

Variable	Entire cohort (n = 241, %)	PLI (n = 142, 58.9%)	No PLI (n = 99, 41.1%)	*P* value
Neurological Deficit				
Delirium	71 (29.5)	45 (31.7)	26 (26.3)	0.392
Stroke	12 (5.0)	10 (7.0)	2 (2.0)	0.093
Spinal cord injury	4 (1.7)	3 (2.1)	1 (1.0)	0.677
Mortality and Morbidity				
Operative mortality	11 (4.6)	8 (5.6)	3 (3.0)	0.055
in-hospital death	2 (0.8)	2 (1.4)	0 (0)	0.168
30-day mortality	9 (3.7)	6 (4.9)	3 (3.0)	0.166
Re-exploration for bleeding	10 (4.1)	5 (3.5)	5 (5.1)	0.745
Acute kidney injury	14 (5.8)	9 (6.3)	5 (5.1)	0.784
Dialysis	19 (7.9)	10 (7.0)	9 (9.1)	0.630
Delayed chest closure	14 (5.8)	7 (4.9)	7 (7.1)	0.579
Gastrointestinal dysfunction	9 (3.7)	6 (4.2)	3 (3.0)	0.740
Time to extubation, hour	18.5 (11.2–41.6)	19.8 (12.7–58.9)	17.8 (10.0–31.2)	0.206
IABP or ECMO	4 (1.7)	0 (0)	4 (4.0)	0.027
Length of ICU stay, hour	84.5 (60.7–136.1)	88.4 (60.8–150.2)	80.8 (59.3–128.4)	0.140
Length of stay, day	15.8 ± 6.3	15.9 ± 6.5	15.6 ± 6.0	0.713

Values are expressed as mean ± standard deviation, median (interquartile range, IQR) or n (%)
PLI, preoperative lacunar infarct; *IABP*, intra-aortic balloon pump; *ECMO*, extracorporeal membrane oxygenation; *ICU*, intensive care unit

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SCA-BOM1-BOM-OP7

Bioadhesive Ultrasound (BAUS) for Continuous Monitoring of Impella™ LVAD Patients in the ICU

Presenting Author: Kitae Chang, MD

Background:

Cardiogenic shock (CS) remains a leading cause of morbidity and mortality, and the most critically ill patients often require temporary MCS, including the percutaneous Impella™ LVAD.1 Current monitoring relies on intermittent echocardiography and radiographic imaging to assess cardiac size and function, as well as Impella™ positioning and performance. However, given the dynamic nature of cardiac function and the known tendency of Impella™ devices to migrate with patient movement, continuous rather than intermittent imaging may enhance device safety and more effectively guide clinical management.2 In collaboration with MIT engineers, we have developed a wearable, Bioadhesive Ultrasound (BAUS) platform, capable of providing continuous cardiac ultrasound imaging (Figure 1). In this trial, we evaluate the safety and feasibility of the BAUS to monitor cardiac function and Impella™ positioning in patients with CS supported by Impella™ LVAD therapy.

Methods:

In this ongoing prospective, single arm, open-label, proof-of-principle study initiated on September 29, 2025, data on safety and feasibility of the BAUS device have been collected for three adult ICU patients with an Impella™ device (IRB 2024P001095). All patients enrolled were male and greater than 18 years of age (Table 1). Only one of the patients had an Impella™ for postoperative mechanical support in addition to ECMO support. On all patients, the probe was placed to visualize the parasternal long view along with the full length of the Impella™ (Figure 2). The device recorded video clips that were of five to 10 seconds in duration every 30 minutes for as long as the device was attached.

Results:

The total duration the BAUS device was attached to patients ranged from three days to eight days (Table 2). None of the patients experienced any complications including, but not limited to skin reactions or pain. For each patient, there was a total of one episode where the device detached by

itself. In patient 1, the device had to be repositioned twice to allow for the ICU team to perform procedures. In patients 2 and 3, the device had to be taken off and replaced a cumulative total of three times so the ICU team could perform formal bedside TTE exams. The device was terminally removed in the patients due to patient request or decannulation of the Impella™ device. An improved battery life and a desire to have a completely wire-free design were points of feedback.

Conclusion:
Our preliminary results on a limited cohort of patients suggest that the BAUS device is safe and easy to apply onto patients. There were no safety incidents and the device could be adhered to patients for at least 3 days and up to 8 days to produce near continuous monitoring with rare unintended detachments. Even following patients' positional changes, the probe was able maintain a parasternal long view and visualize the Impella™ cannula. An obvious limitation of these early findings is the small sample size thus far. The trial is ongoing, and there are improvements to the design of the BAUS that will help address some of the challenges that have been encountered. In summary, BAUS shows early promise to be a useful technology to provide monitoring for highly vulnerable CS patients supported by Impella™ LVADs.

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	Patient 1	**Patient 2**	**Patient 3**
Age (years)	58	25	71
Gender	Male	Male	Male
Self-Reported Race	White	Unknown	White
Height (cm)	167.6	182	179.1
Weight (kg)	81.7	63.7	76.8
BMI (kg/m^2)	29.08	19.23	23.94
Sternotomy Incision	Yes	No	No
Etiology of cardiogenic shock	Acute myocardial infarction	Biventricular heart failure	Non-ischemic cardiomyopathy
Presence of ECMO	Yes	No	No

Table 1. Baseline characteristics

	Patient 1	Patient 2	Patient 3
Duration Device Attached to Patient (days)	3	8	3
Frequency of Device Repositioning (events)	2	2	1
Frequency of Device Detachment (events)	1	1	1
Reason for discontinuation	Provider preference	Patient preference	Decannulation of Impella
Skin Changes (yes/no)	No	No	No

Table 2. Safety and Feasibility Data

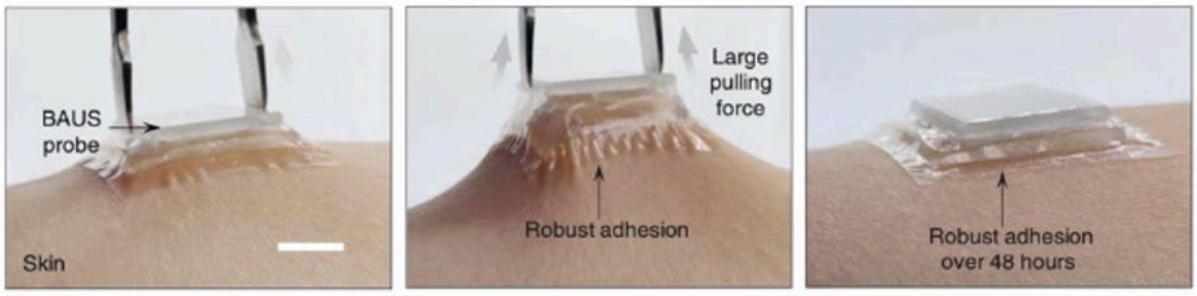


Figure 1. BAUS Device. This is an earlier prototype depiction of the BAUS device[3] (Taken from Wang et al., Science, 2022)

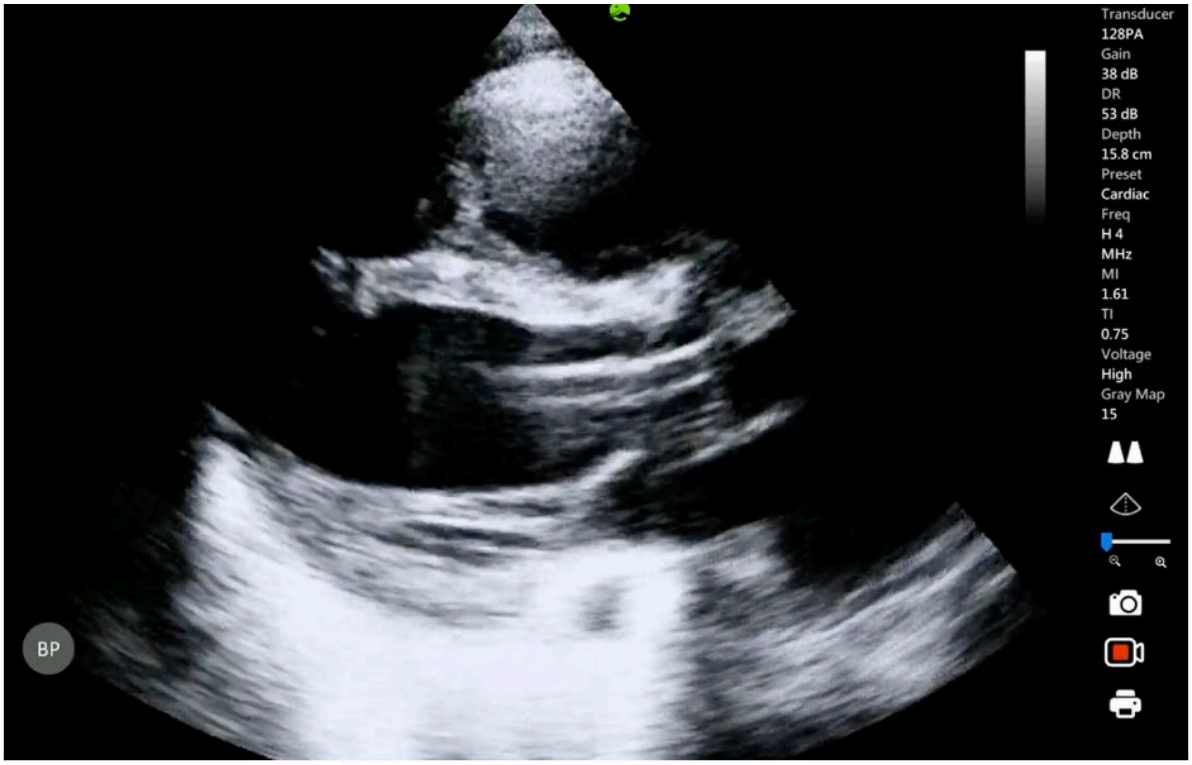


Figure 2. Parasternal long view with Impella™. This is the view obtained by the BAUS on Patient 2.

References

Reference 1

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SCA-BOM1-BOM-OP8

Association of Intraoperative Milrinone Administration and Diastolic Dysfunction In Cardiac Surgical Patients

Presenting Author: Chung Hon Shawn Cheuk, MD

Introduction: Milrinone, a phosphodiesterase 3 inhibitor, is used in cardiac surgery to augment left and right ventricular function through inotropy, pulmonary vasodilation and lusitropy. While milrinone has been shown in animal models to improve lusitropy, its direct effects on diastolic dysfunction in humans are not well-characterized. The primary aim of this study is to evaluate the association of milrinone adminstration with diastolic function in cardiac surgical patients. We have previously linked the development of post operative atrial fibrillation (POAF) with the presence of diastolic dysfunction.[1] Our secondary aim is to evaluate the use of milrinone on POAF.

Methods: Cardiac surgical patients of the PALACS (Posterior Left Pericardiotomy for the Prevention of Atrial Fibrillation after Cardiac Surgery) echo sub-study trial were included.[2] All patients underwent a complete prospective intraoperative TEE protocol. Diastolic dysfunction was defined as lateral e' < 10 cm/s, and grading of diastolic function was performed using E/e' cut offs of grade 1 E/e'≤8, grade 2 E/e' 9 to 12, grade 3 E/e'≥13, along with criteria based on mitral inflow and pulmonary wave flow velocities at two time points, before sternotomy and after chest closure.[1] Hemodynamics were acquired at corresponding TEE points. Intraoperative medication administration was obtained from available electronic anesthesia records. POAF was defined as the occurrence of in-hospital atrial fibrillation during continuous cardiac rhythm monitoring or standard 12-lead ECG.

Results: A total of 382 patients (62 y/o, 75% male, 45% CABG) were included. 76 patients (20%) were given milrinone. Patients given milrinone were more likely to be receiving beta blockers and have reduced LV ejection fraction (Table 1). Overall, 62% of patients had intraoperative diastolic dysfunction. The proportion of patients with improved diastolic dysfunction was greater in cardiac surgery patients with milrinone use vs without milrinone (60% vs 45.7%, p=0.055). On preoperative TEE, median E/e' ratio was higher in patients with milrinone use (7.7 [IQR, 5.9-10.0]; vs 6.9 [IQR, 5.1-8.6]; p=0.006), while lateral e' was higher in patients without milrinone use (9.3 [IQR 7.9-11.1]; vs 8.7 [IQR 6.7-10.1]; p=0.007) (Table 2). On multivariable analysis, milrinone use and LV ejection fraction were independently associated with improvement in diastolic dysfunction (OR 0.45 [95% CI, 0.24-0.83], p=0.01; OR 0.97 [95% CI, 0.94-0.99], p=0.01, respectively) while epinephrine use was independently associated with stable or worsening diastolic dysfunction (OR 1.72 [95% CI, 1.06-2.82], p=0.03) (Figure 1). The incidence of POAF was similar in both groups (20.2% vs 23.5%, p=0.56). On multivariable analysis, milrinone use was not associated with decreased incidence of POAF (OR 0.79 [95% CI, 0.41-1.45], p=0.45) (Figure 2).

Conclusion: Milrinone administration in patients undergoing cardiac surgery is independently associated with improvement in intraoperative diastolic dysfunction. Further large-scale prospective studies are needed to evaluate the lusitropic effects of milrinone and its impact on clinical outcomes.

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Figure 1: Multivariable model of perioperative variables and changes in diastolic dysfunction

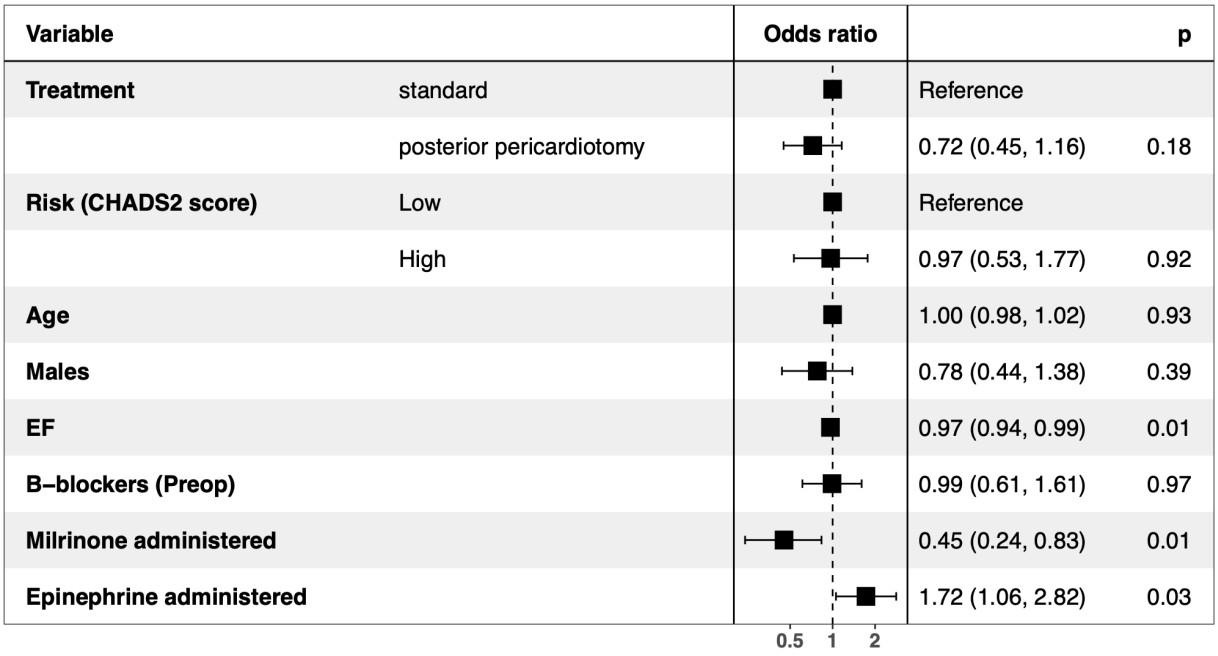


Figure 2: Multivariable model of perioperative variables and POAF

Variable		Odds ratio	p
Treatment	standard	Reference	
	posterior pericardiotomy	0.44 (0.26, 0.73)	0.002
Risk (CHADS2 score)	Low	Reference	
	High	1.32 (0.72, 2.42)	0.364
Age		1.07 (1.04, 1.10)	<0.001
Males		0.58 (0.30, 1.06)	0.085
EF		0.98 (0.96, 1.01)	0.257
B–blockers (Preop)		1.19 (0.72, 1.96)	0.505
Milrinone administered		0.79 (0.41, 1.45)	0.451
Epinephrine administered		0.98 (0.58, 1.65)	0.942

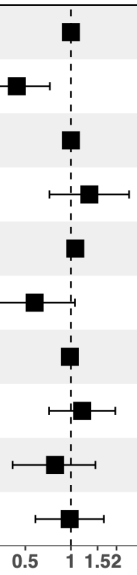


Table 1: Descriptive statistics of included patients

	Overall	No Milrinone	Milrinone	p
N	382	306	76	
Age, yr (median [IQR])	62 [53, 70]	61 [53, 69]	66 [56.8, 71]	0.10
Female sex	95 (24.9)	75 (24.5)	20 (26.3)	0.86
Race				0.74
Asian	15 (3.9)	11 (3.6)	4 (5.3)	
Black or African American	23 (6.0)	20 6.5)	3 (3.9)	
Others	45 (11.8)	35 11.4)	10 (13.2)	
White	299 (78.3)	240 (78.4)	59 (77.6)	
Body mass index, kg/m2 (median [IQR])	27.8 [24.7, 30.5]	27.7 [24.4, 30.5]	27.8 [25.6, 30.2]	0.41
Body surface area, m2 (median [IQR])	2.0 [1.9, 2.2]	2.0 [1.9, 2.2]	2.0 [1.9, 2.1]	0.64
Hypertension	265 (69.4)	209 (68.3)	56 (73.7)	0.44
Diabetes mellitus	83 (21.7)	63 (20.6)	20 (26.3)	0.35
Smoking				0.30
Never	209 (54.7)	171 (55.9)	38 50.0)	
Current	22 (5.8)	15 (4.9)	7 (9.2)	
Previous	151 (39.5)	120 (39.2)	31 (40.8)	
New York Heart Association class (III–IV)	30 (7.9)	21 (6.9)	9 (11.8)	0.23
Chronic lung disease	12 (3.1)	10 (3.3)	2 (2.6)	1
Previous myocardial infarction	51 (13.4)	36 (11.8)	15 (19.7)	0.10
Preoperative hematocrit (median [IQR])	39.7 [35.8, 43.2]	39.5 [35.5, 43.1]	40.6 [37.1, 43.6]	0.27
Previous stroke	13 (3.4)	10 (3.3)	3 (3.9)	1
β-blockers	176 (46.1)	150 (49.0)	26 (34.2)	0.029
CHA2DS2-vASc score	2.1 (1.6)	2.0 (1.6)	2.4 (1.6)	0.06
EuroSCore II (median [IQR])	1.3 [0.9, 2.2]	1.3 [1.0, 2.2]	1.6 [0.9, 3.3]	0.15
Left atrial length, cm (median [IQR])	4 [3.6, 4]	4 [3.5, 4]	4 [3.7, 4.0]	0.44
Left ventricular ejection fraction (median [IQR])	60 [55, 65]	60 [55, 65]	57.50 [52.4, 65]	0.011
Type of surgery				
Coronary artery bypass grafting	171 (44.8)	132 (43.1)	39 (51.3)	0.25
Aortic valve procedures	202 (52.9)	154 (50.3)	48 (63.2)	0.06
Aortic procedures	163 (42.7)	133 (43.5)	30 (39.5)	0.62
Posterior pericardiotomy	190 (49.7)	158 (51.6)	32 (42.1)	0.17
Cross-clamp time, min (median [IQR])	79 [61, 100]	78.5 [61, 98.3]	80 [62, 100.5]	0.61
Cardiopulmonary bypass time, min (median [IQR])	102 [82, 123]	102 [82, 122]	104 [84, 128]	0.27
Operative time, min (median [IQR])	300.5 [255, 356]	298 [254, 354]	312 [262.5, 368]	0.23

Table 2: Baseline preoperative TEE data

Variable (Median [IQR]), Preoperative data	Overall	No Milrinone	Milrinone	p
N	382	306	76	
Left atrial indices				
Left atrial volume, ml	46.7 [34.6, 62.7]	44.9 [34.0, 60.0]	49.7 [36.6, 65.2]	0.17
Left atrial area, cm2	15.6 [13.0, 19.7]	15.5 [12.9, 19.5]	17.1 [13.7, 20.7]	0.10
Left atrial length, cm	5.1 [4.2, 5.7]	5.1 [4.2, 5.7]	5.1 [4.4, 5.8]	0.44
Left atrial reservoir average, %	33.9 [27.6, 40.2]	34.4 [28.6, 40.6]	29.7 [24.7, 38.1]	0.001
Left atrial appendage velocity, cm/s	43.2 [31.2, 54.3]	43.3 [32.5, 55.4]	41.4 [29.1, 51.7]	0.28
Mitral inflow indices				
E/e' ratio	7.0 [5.3, 8.9]	6.9 [5.1, 8.6]	7.7 [5.9, 10.0]	0.006
Mitral e/A ratio	1.3 [1.1, 1.7]	1.3 [1.1, 1.7]	1.3 [1.1, 1.7]	0.99
Tissue Doppler interrogation septal e', cm/s	6.9 [5.7, 8.1]	7.1 [5.9, 8.2]	5.9 [4.6, 7.4]	<0.001
Tissue Doppler interrogation lateral e', cm/s	9.2 [7.6, 10.9]	9.3 [7.9, 11.1]	8.7 [6.7, 10.1]	0.007
Mitral e, cm/s	64.1 [53.3, 75.0]	63.9 [53.3, 74.8]	65.9 [54.9, 74.9]	0.29
Mitral A, cm/s	47.0 [36.0, 59.2]	46.7 [35.7, 59.0]	48.5 [39.4, 66.0]	0.16
Mitral A-wave duration, ms	140 [120, 150]	140 [120, 150]	130 [120, 140]	0.029
Mitral valve deceleration time, ms	170 [140, 210]	180 [140, 210]	170 [140, 210]	0.43
Mitral A-pulmonary A-wave duration, ms	140 [120, 150]	140 [120, 150]	130 [120, 140]	0.029
Left ventricular outflow tract velocity time integral, cm	19.2 [15.6, 23.7]	19.4 [15.7, 23.8]	18.90 [15.6, 22.6]	0.5
Pulmonary vein flow				
Pulmonary vein systolic flow, cm/s	39.9 [31.0, 50.6]	40.5 [30.5, 50.9]	38.0 [33.1, 46.8]	0.52
Pulmonary vein diastolic flow, cm/s	34.9 [27.7, 42.1]	34.7 [27.4, 42.0]	35.7 [29.7, 43.6]	0.41
Pulmonary atrial reversal flow duration, ms	16.7 [13.0, 20.7]	16.5 [13.0, 20.7]	17.1 [12.7, 20.2]	0.93
Pulmonary A-wave duration, ms	110 [80, 140]	110 [80, 140]	115 [82.5, 140.8]	0.53
Pulmonary systolic flow/diastolic flow	1.2 [0.9, 1.4]	1.2 [0.9, 1.4]	1.1 [0.9, 1.4]	0.35
Diastolic dysfunction	211 (61.9)	162 (59.3)	49 (72.1)	0.07

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SCA-BOM1-BOM-OP9

Intraoperative driving pressure and post-operative pulmonary complications in lung resection patients without pre-existing chronic lung disease: a multicenter retrospective cohort study

Presenting Author: Philip Maykowski, MD MPH

Introduction:

Post-operative pulmonary complications (PPC) occur in up to 80% of surgeries and range from hypoxemia responsive to minimal oxygen therapy to respiratory failure requiring prolonged mechanical ventilation (1). Driving pressure is a potentially modifiable component of intraoperative lung protective ventilation and a major target for interventions to reduce postoperative pulmonary complications. However, few studies have evaluated its effects in lung resection patients, a group with unique intraoperative mechanics and high frequency of chronic lung disease both of which can affect risk of PPC. This study aimed to quantify the relationship between driving pressure and PPC within 7 days after surgery in lung resection patients without baseline chronic lung disease.

Methods:

We conducted a retrospective cohort study on patients undergoing lung resection (open, video-assisted, or robotic) between January 1, 2016 and December 31, 2023 at three large academic medical centers in Boston and New York, USA. Perioperative data was extracted from electronic medical records (EMR). The primary exposure was driving pressure reported as medians for the case. Patients with EMR diagnoses of COPD or asthma, and patients with median intraoperative SpO_2 of < 95%were removed (Figure 1). The primary outcome was a composite outcome of PPC defined as at least one of the following: (Grade 1) mild hypoxemia w/ SpO_2 of 90-92% on room air or 278mmHg ≤ PaO_2/FiO_2 < 301mmHg; (Grade 2) Bronchospasm, hypoxemia (SpO_2 < 90% when on room air or imputed PaO_2/FiO_2 < 278mmHg on O_2), and atelectasis; (Grade 3) pleural effusion, pneumonia, pneumothorax, and post-operative mechanical ventilation for < 48 hours, and (Grade 4) post-operative mechanical ventilation for ≥ 48 hours. To examine the relationship between driving pressure and PPC, we created a multivariable mixed model logistic regression with medical center and surgeon as random effects.

Results:

We studied 6525 patients in the original cohort and removed 1223 (19%) patients for COPD, 509 (8%) for asthma, and 175 (3%) for intraoperative SpO_2 of < 95% (Figure 1). The final cohort included 4617 patients (mean age 64.6, 41% male), of whom 185 (4%) had heart failure, and 280 (6%) had cerebrovascular disease. Overall, 3380 patients (73%) developed PPCs, (7% grade 1, 54% grade 2, 11% grade 3, 0.4% grade 4). Multivariable analysis identified age, sex, body mass index, heart failure, cerebrovascular disease, surgery duration, intraoperative SpO_2, and intraoperative driving pressure as significant risk factors (Table 1). We found that for every 1 cmH_2O increase in median intraoperative driving pressure, the odds of post-operative pulmonary complications increased 4% (OR 1.04 CI_{95}: 1.02-1.06).

Conclusion:
In patients without pre-existing lung disease, an increase in intraoperative driving pressure is associated with a clinically meaningful increase in the odds of postoperative pulmonary complications (4% per 1 cmH_20). In addition, optimization of cardiovascular and cerebrovascular comorbidities both may reduce PPC burden in these patients.

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Figure 1: Patient selection

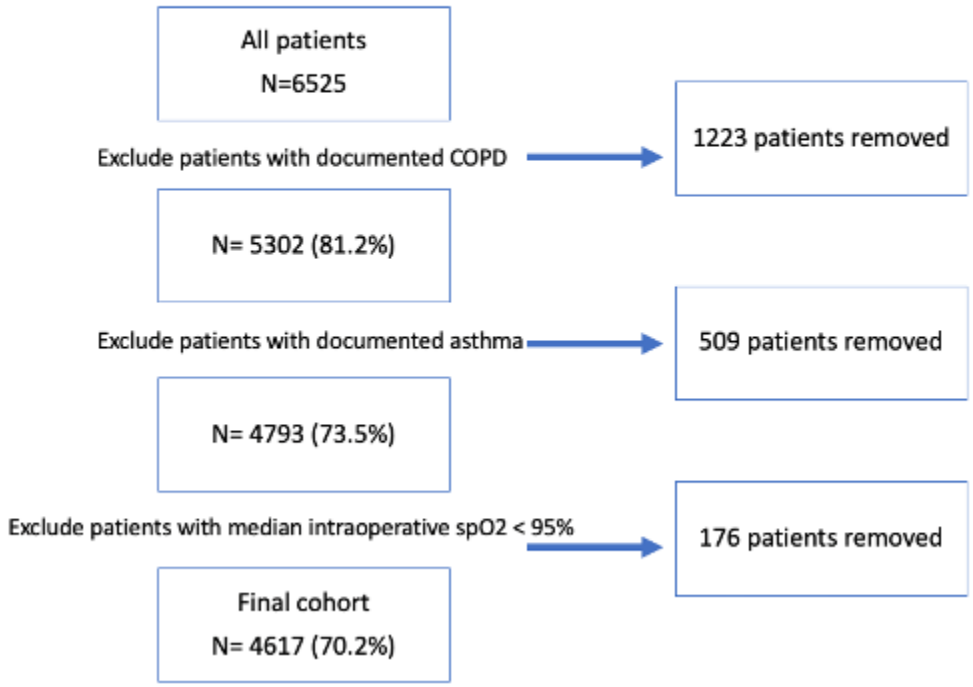


Table 1: Univariable and multivariable models assessing risk of PPCs in those without baseline respiratory disease

Clinical characteristic	Total (n=4617)	Univariable OR	Univariable P-value	Multivariable OR (CI_{95})	Multivariable P-value
Age (years) [mean, ±SD]	64.6 (±12.8)	1.02	<0.001	1.02 (1.01-1.02)	<0.001
Sex – Male (n,%)	1890 (40.9%)	0.97	0.60	0.76 (0.65-0.88)	<0.001
BMI [mean, ±SD]	27.1 (±5.6)	1.11	<0.001	1.08 (1.06-1.10)	<0.001
Smoker (n,%)	1517 (33.5%)	1.23	0.006	1.03 (0.87-1.21)	0.76
Heart failure (n,%)	185 (4.0%)	2.11	<0.001	1.71 (1.11-2.64)	0.015
Cerebrovascular disease (n,%)	280 (6.0%)	2.07	<0.001	1.63 (1.15-2.34)	0.007
GERD (n,%)	1002 (21.7%)	1.56	<0.001	1.26 (1.05-1.52	0.013
Block*	888 (19.2%)	1.95	<0.001	1.50 (1.19-1.88)	<0.001
Intraoperative					
Driving pressure (cm H_2O) [mean, ±SD]	16.0 (±4.3)	1.12	<0.001	1.04 (1.02-1.06)	<0.001
SpO_2 O (%)[mean, ±SD]	98.5 (±1.4)	0.71	<0.001	0.75 (0.71-0.79)	<0.001
FiO_2 (%) [median, IQR]	94 (76-96)	1.01	<0.001	1.01 (1.00-1.02)	<0.001
Duration (Hours) [mean, ±SD]	2.9 (±1.5)	1.34	<0.001	1.32 (1.24-1.42)	<0.001
Fluid balance** [mean, ±SD]	9.9 (±4.6)	0.98	0.07	1.02 (1.00-1.03)	0.09

*Peripheral nerve block, epidural, or spinal included in anesthetic
** mL/KG of ideal body weight/hour

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SCA-BOM1-BOM-OP10

Echocardiographic Correlates of Pressure-Volume-Derived Indices: Advancing Intraoperative Assessment of Right Ventricular Function

Presenting Author: Vahid Kiarad, MD, MPH

Background:

Right ventricular (RV) function is a major determinant of postoperative outcomes in cardiac surgery, yet accurate intraoperative assessment remains challenging. RV-pulmonary arterial (RV-PA) coupling, quantified by the ratio of end-systolic to arterial elastance (Ees/Ea), provides a physiologic index of RV performance and overcomes many limitations of traditional metrics.[1,2] Three-dimensional (3D) transesophageal echocardiography (TEE) offers global RV volumetric assessment and may serve as a practical surrogate for invasive coupling. This study evaluates the relationship between intraoperative 3D echocardiographic RV indices and invasively derived single-beat RV-PA coupling metrics and assessed the ability of echocardiographic parameters to discriminate impaired RV-PA coupling and predict adverse perioperative outcomes.

Methods:

In a prospective observational cohort of adult cardiac surgery patients undergoing simultaneous intraoperative TEE and pulmonary artery catheter monitoring, RV pressure-volume data were obtained and subjected to single-beat end-systolic pressure–volume analysis using the V_0 method.[3] 3D RV volumes and functional indices were quantified from multibeat full-volume acquisitions using vendor-validated RV quantification software. Patients were classified as having normal or low coupling using an Ees/Ea threshold of < 0.8. Correlations between echocardiographic parameters and RV-PA coupling were analyzed using Spearman coefficients, restricted cubic splines, and ROC analysis. Multivariable logistic regression adjusted for perfusion time and STS risk score explored associations of these metrics with prolonged postoperative vasopressor/inotrope use (> 24 hours after surgery).

Results:

Among 78 patients, 28.2% demonstrated low RV-PA coupling (Figure 1, 2). Patients with impaired coupling had significantly larger indexed end-systolic volume and reduced 3D ejection fraction (EF) and 4D-derived fractional area change (FAC). 3D EF correlated most strongly with coupling (ρ = 0.94, $p < 0.001$) and showed excellent discriminative performance (AUC = 0.94; optimal cutoff < 44.2%) to predict low RV-PA coupling. 4D-derived FAC (AUC = 0.86; cutoff < 40.3%) and indexed ESV (AUC = 0.75; cutoff >30.5 mL/m^2) also differentiated impaired coupling, whereas TAPSE and TAPSE/PASP did not (Figure 3, 4). Exploratory adjusted analyses demonstrated significant

associations between lower Ees/Ea, reduced 3D EF, reduced 4D FAC/RVSP, and prolonged postoperative vasopressor/ inotrope use (>24 hours after surgery). (Figure 5, 6)

Conclusion:
Intraoperative 3D echocardiographic volumetric indices- particularly 3D EF and 4D-derived FAC- closely reflect invasively measured RV-PA coupling and effectively identify patients with impaired coupling during cardiac surgery. These findings support 3D TEE-derived global RV systolic indices as practical intraoperative surrogates for physiologic RV-PA interaction assessment and highlight their potential role in identifying patients at risk for adverse perioperative outcomes.

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Variable	Overall (N=78)	Normal RV-PA Coupling Ratio (n=56)	Low RV-PA Coupling Ratio (n=22)	p-value
Demographics				
Age, mean (SD)	66.8 (10.4)	68.3 (10.8)	63.2 (8.6)	0.05
Male sex, n (%)	62 (79.4)	44 (78.5)	18 (81.8)	0.8
Race, n (%)				0.91
White	53 (67.9)	38 (67.9)	15 (68.1)	
Black	9 (11.5)	6 (10.7)	3(13.6)	
Asian	6 (7.7)	5 (8.9)	1 (4.5)	
Other	10 (12.8)	7 (12.5)	3 (13.6)	
Baseline characteristics				
Diabetes mellitus, n (%)	21 (26.9)	15 (26.7)	6 (27.2)	0.96
Hypertension, n (%)	59 (75.6)	42 (56)	17 (77.2)	0.93
Chronic lung disease, n (%)	11 (14.1)	7 (12.5)	5 (18.2)	0.52
Preop creatinine, mean (SD)	1.12 (0.69)	1.17(0.79)	1.01 (0.23)	0.37
Previous CABG, n (%)	1 (1.3)	1 (1.7)	0 (0.0)	0.52
Previous valve surgery, n (%)	0	0	0	-
Perioperative characteristics				
TR severity, n (%)				0.69
None	49 (62.8)	36 (64.3)	18 (59.1)	
Mild	19 (24.4)	12 (21.4)	7 (31.8)	
Moderate	7 (9.0)	6 (10.7)	1 (4.5)	
Severe	3 (3.8)	2 (2.5)	1 (4.5)	
Surgery type, n (%)				0.92
CABG	42 (53.2)	29 (51.8)	13 (59.1)	
CABG + Valve	10 (12.8)	8 (14.3)	2 (9.1)	
Single valve	21 (26.9)	15 (26.8)	6 (27.3)	
Multiple valves	4 (5.1)	3 (3.8)	1 (4.5)	
Perfusion time (min), mean (SD)	94.1 (38.9)	99.2 (42.4)	81.1 (24.2)	0.08
Outcomes				
Major Morbidity and Mortality, n (%)	9 (11.5)	8 (14.2)	1 (4.5)	0.23
30-day mortality, n (%)	2 (2.6)	2 (3.5)	0 (0.0)	0.37
Postop stroke, n (%)	3 (3.8)	3 (5.3)	0 (0.0)	0.27
Renal failure, n (%)	3 (3.8)	2 (5.3)	0 (0.0)	0.27
Prolonged ventilation, n (%)	4 (5.1)	3 (5.3)	1 (4.5)	0.88
Deep sternal infection, n (%)	0	0	0	-
Reoperation (ischemia), n (%)	1 (1.3)	1 (1.8)	0 (0.0)	0.53
Prolonged pressor/inotrope, n (%)	34 (43.6)	18 (32.1)	16 (72.7)	**0.002**
ICU LOS (hours), mean (SD)	45.6 (37.0)	46.0 (30.6)	44.3 (51.0)	0.86
Hospital LOS (days), mean (SD)	6.7 (6.9)	7.4 (7.9)	4.9 (2.9)	0.16
Ventilation time (hours), mean (SD)	14.4 (41.1)	16.3 (47.5)	9.6 (15.0)	0.55

Figure 1: Baseline Characteristics

Variable	Normal RV PA Coupling (mean ± SD) (n=56)	Low RV PA Coupling (mean ± SD) (n=22)	p- value
3D ESV (mL) (End-systolic volume)	57.8 ± 34.3	81.0 ± 32.9	**0.011**
3D EDV (End diastolic volume) (mL)	111.8 ± 49.3	127.3 ± 38.8	0.202
3D ESV indexed to body surface area (mL/m²)	29.7 ± 16.7	38.7 ± 14.6	**0.038**
3D EDV indexed (mL/m²)	57.4 ± 23.9	60.9 ± 16.3	0.535
3D SV (Stroke Volume) (mL)	53.6 ± 17.2	46.2 ± 13.0	0.08
3D EF (Ejection Fraction) (%)	50.0 ± 5.7	37.5 ± 7.3	**<0.001**
TAPSE (Tricuspid Annular Plane Systolic Excursion) (mm)	14.7 ± 4.7	11.9 ± 5.0	0.09
2D FAC (Fractional Area Change) (%)	39.3 ± 8.7	34.3± 8.0	0.055
4D derived FAC (%)	42.3 ± 7.5	31.3 ± 9.1	**<0.001**
S' Velocity	0.23 ± 0.17	0.15 ± 0.03	0.19
EDP (End-diastolic pressure) (mmHg)	14.1 ± 4.4	15.5 ± 4.3	0.219
ESP (End-systolic pressure) (mmHg)	38.0 ± 12.8	41.9 ± 15.7	0.269
PAP (Pulmonary Artery Pressure) mean (mmHg)	27.7 ± 7.8	30.1 ± 9.5	0.269
PAPi (Pulmonary Artery Pulsatility Index)	2.17 ± 1.62	1.63 ± 0.79	0.151
PA Systolic Pressure (mmHg)	40.3 ± 10.1	40.7 ± 12.1	0.909
RV Systolic Pressure (mmHg)	40.9 ± 9.2	40.1 ± 11.5	0.769
Ees (End-systolic elastance)	0.83 ± 0.44	0.63 ± 0.30	0.067
Ea (Effective arterial elastance)	0.80 ± 0.36	1.03 ± 0.61	0.064
Ees/Ea ratio	1.04±0.17	0.67±0.14	**<.001**
TAPSE/PASP	0.36 ± 0.14	0.34 ± 0.14	0.72
2D FAC/RVSP	0.98 ± 0.28	0.93 ± 0.32	0.52
4D derived FAC/RVSP	1.05 ± 0.27	0.87 ± 0.28	**0.018**

Figure 2: Echocardiographic and Hemodynamic Parameters

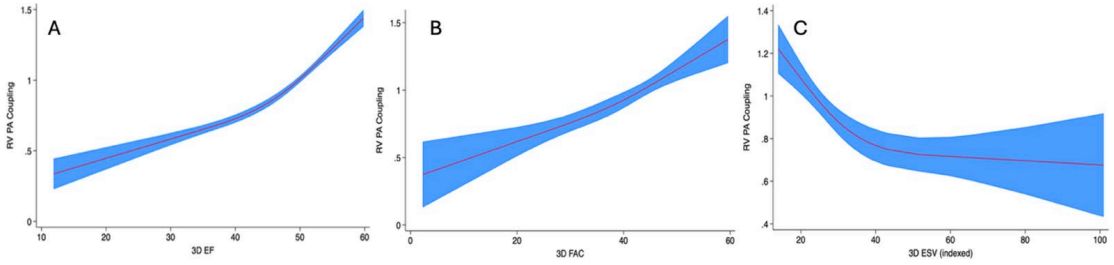


Figure 3: Restricted cubic spline associations

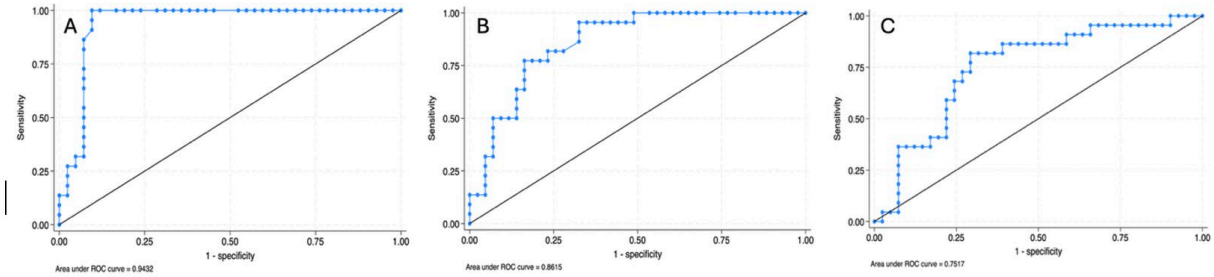


Figure 4: ROC curve analysis for predicting low RV PA coupling

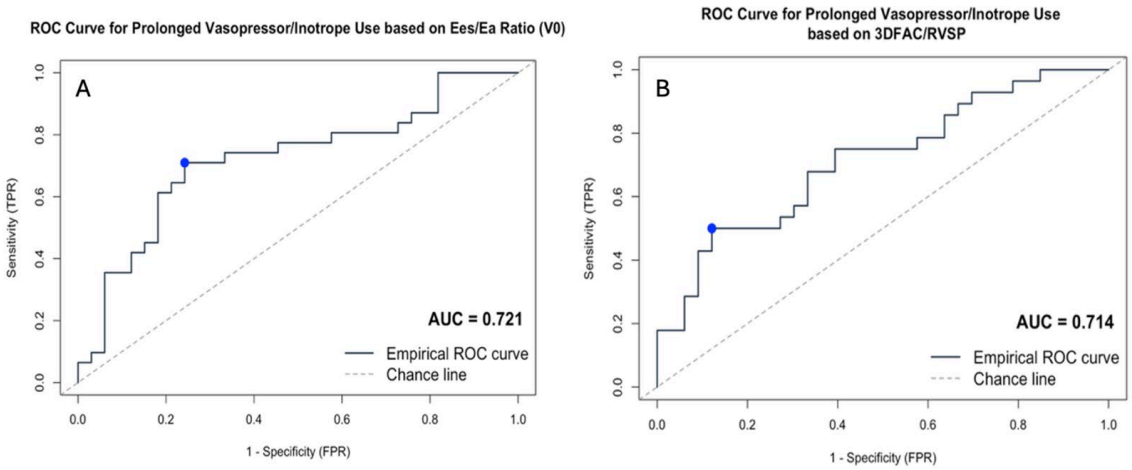


Figure 5: ROC Curve Analysis

Predictive Index	Odds Ratio (95% CI)	P value
Low RV–PA coupling (Vo) (<0.885)	9.61 (2.58 -35.75)	0.001
Low 3DFAC/RVSP ratio (<0.826)	5.19 (1.36 -19.80)	0.016
RVEF	0.88 (0.80 -0.96)	0.004
ESVi	1.05 (1.01-1.12)	0.03
FAC	0.86 (0.78-0.94)	0.001

Figure 6: Associations of indices with prolonged vasopressor/ inotrope use

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SCIENTIFIC PROGRAM ABSTRACTS

Best of Meeting Oral Abstracts and Early Career Investigator Awards: Session 2

Saturday, April 25, 2026 | 3:00 PM - 5:00 PM

Best of Meeting Oral Abstracts and Early Career Investigator Awards: Session 2

SCA-BOM2-ECI-OP1

Deep phenotyping of the right ventricle using optical flow identifies new pattern of motion associated with perioperative mortality after cardiac surgery

Presenting Author: Nicolas T. Quach, MD

Background:

Right ventricular (RV) dysfunction is associated with significant morbidity and mortality in the perioperative period. The RV is a complex structure with mainly two axes of motion: radial and longitudinal. These two axes of motion can be visualized via transesophageal echocardiography (TEE) in the RV inflow-outflow (RVIO) view. In this study, we use dense optical flow, a computer vision strategy that calculates apparent motion at every pixel, to derive highly granular vector fields that quantify myocardial motion and identify their association with all-cause mortality after cardiac surgery.

Methods:

In this retrospective study, 1557 intraoperative pre-cardiopulmonary bypass RVIO videos from 918 patients undergoing cardiac surgery from 2020-2023 were collected along with timestamp-matched invasive pulmonary artery catheter data. Patients' age, sex, surgery type and preoperative transthoracic echocardiogram (TTE) report were also collected. Patients in this cohort were followed up through 2025 for all-cause mortality. Dense optical flow was calculated from these RVIO videos. A fine-tuned vision transformer was used to select for vectors corresponding to the RV free wall, and their motion separated into radial and longitudinal components. Systolic and diastolic frames were identified using time-stamped match ECG data from lead II. Peak systolic and diastolic values for total, component-wise velocities were obtained. Multivariate Cox regression models were used to model all-cause mortality. Hazard ratios and false discovery rate (FDR) corrected p-values for covariates were calculated. Performance of the Cox regression models was assessed using concordance index and models were compared using the likelihood ratio test.

Results:

A multivariable Cox regression fit to age, sex, surgery, preoperative left ventricular ejection fraction (LVEF) and RV total systolic and diastolic velocity showed no significant association between total peak velocity and mortality. When velocity is separated into components and an analogous Cox regression is run, peak radial diastolic velocity is significantly associated with mortality (HR 1.10, p=0.027). When comparing models fit with age, sex, surgery, LVEF, invasive hemodynamic

parameters with and without component-wise velocities, the model containing both velocities and hemodynamic parameters was significantly superior to that containing hemodynamic parameters alone (likelihood ratio test p=0.003). In a sub-analysis of patients with normal preoperative RV function, a model fit to age, sex, surgery, preoperative LVEF and component-separated RV velocities continued to show that peak radial diastolic velocity is significantly associated with mortality (HR 1.12, p=0.021).

Conclusion:

When adjusted for age, sex, surgery, and preoperative LVEF, increased RV peak radial diastolic velocity is independently associated with increased mortality, which remains true even in a sub-analysis of patients with normal preoperative RV systolic function. In all, our study represents the first use of optical flow to more thoroughly phenotype RV motion, and demonstrates its value by identifying a new clinically relevant RV motion phenotype associated with perioperative mortality.

Image Uploads:

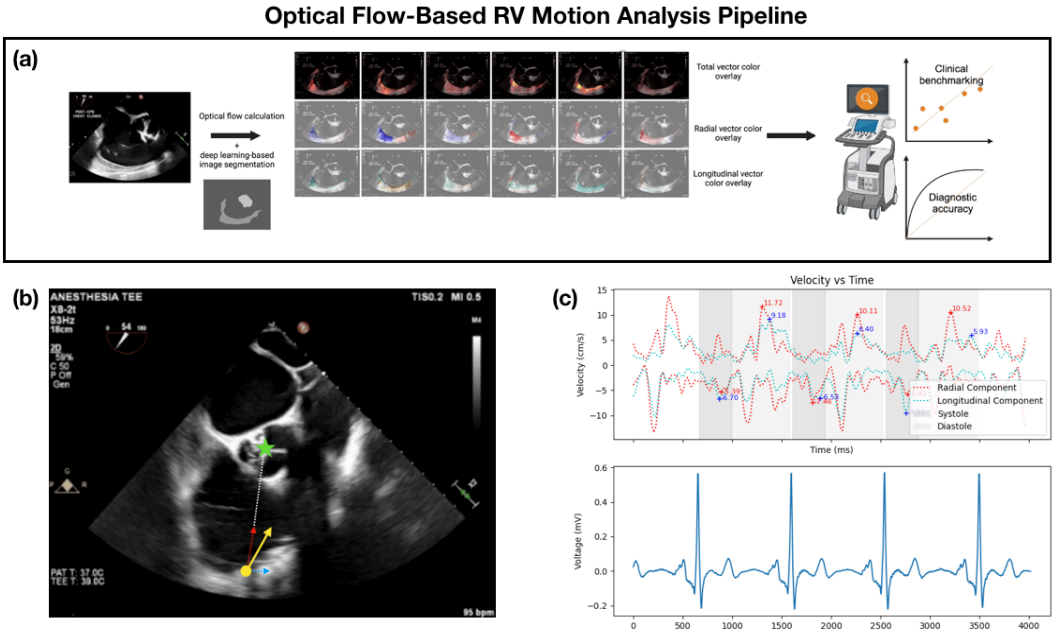


Figure 1. Diagram of motion analysis pipeline and 99th percentile component-wise optical flow based velocity waveforms (a) Diagram of motion analysis pipeline. Optical flow is calculated for all frames. The aortic valve and RV free wall are segmented using a fine-tuned vision transformer model. Segmentation masks are used to isolate optical flow corresponding to the RV free wall. (b) Sample frame from RVIO video demonstrating component-wise analysis of total velocity vector (yellow) into radial component (red) and longitudinal component (blue) using the AV segmentation mask centroid (green star) as a reference point. (c) The 99th percentile radial and longitudinal components or velocity can then be plotted as discreet waveforms. Red and blue crosses denote maximal radial and longitudinal velocity values in systole and diastole, respectively. ECG data from lead II time-stamp matched with the RVIO video is used for cardiac cycle determination. Gray shading represents systolic frames. White shading represents diastolic frames. *cm = centimeters; s = second; mV = millivolt; AV = aortic valve; RVIO = right ventricular inflow-outflow; ECG = electrocardiogram.*

	Total Velocity				Component Velocity			
	HR	95% CI	P-value	FDR corrected P-value	HR	95% CI	P-value	FDR corrected P-value
Demographics								
Age	**1.04**	**[1.02,1.05]**	**0.0001***	**0.0012***	**1.04**	**[1.02,1.06]**	**0.0002***	**0.0019***
Male	0.84	[0.53,1.31]	0.4338	0.6572	0.81	[0.51,1.27]	0.3559	0.5297
Surgery								
CABG	1.22	[0.70,2.15]	0.4833	0.6572	1.31	[0.74,2.31]	0.3541	0.5297
Valve	1.31	[0.73,2.39]	0.3627	0.6572	1.29	[0.71,2.35]	0.3953	0.5297
Aorta	0.99	[0.62,1.61]	0.9940	1.000	1.04	[0.65,1.69]	0.8572	0.9157
Other	0.72	[0.40,1.30]	0.2816	0.6572	0.78	[0.43,1.40]	0.4022	0.5297
Preop LV Function								
LVEF	0.99	[0.97,1.01]	0.2038	0.6572	0.99	[0.97,1.01]	0.2319	0.5297
Optical Flow Velocity								
Total Systolic Velocity	0.99	[0.94,1.06]	0.9810	0.9940	—	—	—	—
Total Diastolic Velocity	1.02	[0.96,1.08]	0.5111	0.6572	—	—	—	—
Radial Systolic Velocity	—	—	—	—	0.99	[0.91,1.08]	0.9157	0.9157
Radial Diastolic Velocity	—	—	—	—	**1.10**	**[1.02,1.17]**	**0.0049***	**0.0267***
Longitudinal Systolic Velocity	—	—	—	—	0.95	[0.86,1.04]	0.2363	0.5297
Longitudinal Diastolic Velocity	—	—	—	—	0.97	[0.90,1.05]	0.4334	0.5297
N =			**918**				**918**	
C-Index =			**0.63**				**0.66**	

	Hemodynamics				Hemodynamics + Velocities			
	HR	95% CI	P-value	FDR corrected P-value	HR	95% CI	P-value	FDR corrected P-value
Demographics								
Age	**1.04**	**[1.02,1.05]**	**0.0002***	**0.0019***	**1.04**	**[1.02,1.06]**	**0.0002***	**0.0027***
Male	0.89	[0.57,1.41]	0.6251	0.6924	0.86	[0.54,1.36]	0.5069	0.7081
Surgery								
CABG	1.35	[0.77,2.34]	0.2922	0.5845	1.48	[0.85,2.59]	0.1688	0.5665
Valve	1.20	[0.68,2.13]	0.5348	0.6924	1.16	[0.64,2.06]	0.6258	0.7152
Aorta	1.10	[0.67,1.83]	0.7038	0.7038	1.16	[0.69,1.93]	0.5754	0.7081
Other	0.68	[0.38,1.23]	0.2004	0.4861	0.74	[0.41,1.34]	0.3258	0.6286
Preop LV Function								
LVEF	0.99	[0.98,1.01]	0.2038	0.6924	0.99	[0.98,1.01]	0.7107	0.7581
Hemodynamics								
CVP	0.93	[0.79,1.11]	0.4391	0.6924	0.95	[0.79,1.13]	0.5633	0.7081
Mean PAP	1.15	[0.96,1.37]	0.1231	0.4861	1.13	[0.94,1.36]	0.1770	0.5665
PA Pulse Pressure	0.99	[0.97,1.01]	0.4675	0.6924	0.99	[0.97,1.02]	0.5114	0.7081
PA Pulsatility Index	1.12	[1.02,1.23]	0.0191	0.1147	1.10	[0.99,1.20]	0.0506	0.2700
RV Step Up	0.89	[0.74,1.06]	0.2025	0.4861	0.90	[0.75,1.09]	0.2850	0.6286
Optical Flow Velocity								
Total Systolic Velocity	–	–	–	–	–	–	–	–
Total Diastolic Velocity	–	–	–	–	–	–	–	–
Radial Systolic Velocity	–	–	–	–	0.99	[0.91,1.08]	0.8493	0.8493
Radial Diastolic Velocity	–	–	–	–	**1.11**	**[1.03,1.18]**	**0.0033***	**0.0261***
Longitudinal Systolic Velocity	–	–	–	–	0.95	[0.86,1.04]	0.2592	0.6286
Longitudinal Diastolic Velocity	–	–	–	–	0.96	[0.89,1.04]	0.3536	0.6286
N =			**918**				**918**	
C-Index =			**0.67**				**0.69**	

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Reference 3

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SCA-BOM2-ECI-OP2

Iatrogenic blood loss due to phlebotomy in the intensive care unit following cardiac surgery: potential target for intervention in patient blood management programs

Presenting Author: Rachel A. Gioscia-Ryan, MD, PhD

Introduction:

Patient blood management – a multidisciplinary approach to managing patient blood status to improve outcomes – is an emerging pillar of perioperative care in cardiac surgery. Pharmacologic interventions reducing perioperative blood loss by 150-200 mL in cardiac surgery patients are associated with improvement in meaningful clinical endpoints including transfusions and mortality; thus, other strategies to minimize blood loss by a similar magnitude could be correspondingly impactful. Frequent laboratory testing is integral to postoperative care in the intensive care unit (ICU) but may also represent an important source of iatrogenic blood loss. Whether the blood volume withdrawn over the course of ICU admission could be minimized by using the smallest commercially available (small-volume, SV) phlebotomy tubes has not been well studied in cardiac surgery patients. Thus, the purpose of this study was to characterize blood loss due to phlebotomy postoperatively to inform development of a prospective intervention involving use of SV tubes to reduce iatrogenic blood loss in cardiac surgery patients.

Methods:

Data were collected on 7382 adult cardiac surgery patients undergoing procedures with use of cardiopulmonary bypass and ICU length of stay (LOS) < 31 days from two academic medical centers between November 2022 and November 2025 (IRB protocol 2025P002206). Patients were stratified into quartiles of estimated blood loss (EBL) due to phlebotomy in the ICU (calculated by enumerating distinct lab draws during ICU stay), with patients with EBL greater than 400 mL (400+) investigated as an additional group. Absolute standardized differences (ASD) were calculated to assess imbalance between quartiles. Descriptive statistics, tables, and figures were generated using R, R Studio, and Microsoft Excel.

Results/Discussion:

Patient characteristics of the entire cohort and each quartile of EBL are described in Table 1. As expected, higher EBL was associated with longer ICU LOS, lower nadir hemoglobin concentration, and higher transfusion and 30-day mortality rates. Patients in the 400+ group comprised 7.7% of the entire cohort and had a median ICU LOS of 10.8 days and 30-day mortality of 7% (Table 2). The absolute volume and proportion of EBL attributable to different types of phlebotomy tubes was estimated (Table 3, Figure 1), with lithium heparin chemistry tubes accounting for the greatest proportion. For tube types with SV alternatives available, the projected blood volume that could be conserved with use of SV tubes was also determined. In the 4th quartile and 400+ group use of SV tubes is projected to conserve 98 and 202 mL, respectively, reflecting an approximately 33% reduction in the total EBL from phlebotomy.

Conclusion:

Our results indicate that use of SV tubes for phlebotomy in the ICU following cardiac surgery is projected to reduce iatrogenic blood loss over the course of ICU admission by as much as 33%, and conserve more than 200 mL of blood in patients with ICU LOS exceeding 10 days. These data provide important proof-of-concept evidence to support a prospective intervention trial incorporating SV phlebotomy tubes into a perioperative patient blood management program for adult cardiac surgery patients.

Image Uploads:

Table 1: Patient characteristics

	Overall N = 7382	Quartile One n = 1864	Quartile Two n = 1846	Quartile Three n = 1835	Quartile Four n = 1837	ASD
Age, years	66 [58, 72]	65 [57, 71]	66 [58, 72]	67 [59, 73]	66 [57, 73]	0.10
Male	5199 (70)	1365 (73)	1350 (73)	1280 (70)	1204 (66)	0.10
Race						0.14
American Indian or Alaska Native	7 (0)	2 (0)	1 (0)	0 (0)	4 (0)	
Asian	246 (3)	57 (3)	65 (3)	69 (4)	55 (3)	
Black or African American	252 (3)	43 (2)	51 (3)	49 (3)	109 (6)	
Identifying with more than one race	19 (0)	5 (0)	1 (0)	5 (0)	8 (0)	
Native Hawaiian or Other Pacific Islander	3 (0)	0 (0)	0 (0)	1 (0)	2 (0)	
Unavailable	577 (8)	124 (7)	140 (8)	147 (8)	166 (9)	
White	6278 (85)	1633 (88)	1588 (86)	1564 (85)	1493 (81)	
Height, inches	68 [65, 70]	68 [66, 71]	68 [65, 70]	68 [65, 70]	68 [64, 70]	0.11
Weight, pounds	185 [159, 213]	189 [163, 215]	187 [161, 213]	183 [159, 214]	180 [152, 208]	0.11
BMI, kg/m^2	28.1 [25.0, 31.8]	28.4 [25.3, 31.9]	28.2 [25.3, 31.6]	28.2 [25.1, 32.0]	27.6 [24.3, 31.8]	0.06
BSA, m^2	2.0 [1.8, 2.1]	2.0 [1.8, 2.1]	2.0 [1.8, 2.1]	2.0 [1.8, 2.1]	1.9 [1.8, 2.1]	0.13
Hypertension	3251 (44)	871 (47)	818 (44)	816 (45)	746 (41)	0.06
Coronary Artery Disease	1714 (23)	378 (20)	478 (26)	472 (26)	386 (21)	0.09
Heart Failure	950 (13)	130 (7)	129 (7)	187 (10)	504 (27)	0.30
Chronic Kidney Disease	579 (8)	87 (5)	115 (6)	162 (9)	215 (12)	0.15
Diabetes	1785 (24)	418 (22)	435 (24)	462 (25)	470 (26)	0.04
ICU Length of Stay, days	2.0 [1.0, 3.8]	0.9 [0.8, 1.1]	1.7 [1.0, 2.0]	2.7 [2.0, 3.1]	5.9 [4.2, 8.1]	1.63
Baseline Hemoglobin, g/dL	13.6 [12.3, 14.7]	14.0 [13.0, 14.9]	13.8 [12.7, 14.8]	13.6 [12.3, 14.7]	12.7 [10.4, 14.1]	0.39
First ICU Hemoglobin, g/dL	10.9 [9.7, 12.0]	11.4 [10.4, 12.4]	11.0 [10.0, 12.1]	10.7 [9.5, 12.0]	10.1 [9.0, 11.4]	0.39
Nadir ICU Hemoglobin, g/dL	8.6 [7.3, 10.0]	10.0 [8.9, 10.9]	9.3 [8.2, 10.4]	8.2 [7.3, 9.4]	7.0 [6.6, 7.8]	1.08
Baseline Iron, mcg/dL	52 [35, 78]	58 [31, 85]	59 [41, 82]	52 [34, 77]	51 [34, 74]	0.13
Baseline TSAT, percent	18 [12, 28]	19 [12, 28]	21 [14, 30]	19 [12, 28]	18 [12, 26]	0.15
Intraoperative Blood Transfusions	2 [1, 3]	1 [1, 2]	1 [1, 2]	2 [1, 2]	2 [1, 4]	0.29
Postoperative ICU Blood Transfusions	2 [1, 3]	1 [1, 2]	1 [1, 2]	1 [1, 2]	2 [1, 4]	0.38
30 Day Mortality	176 (2)	11 (1)	11 (1)	23 (1)	131 (7)	0.19

Data were stratified by quartiles of estimated blood loss from phlebotomy. Quartiles were defined as follows: Quartile One, 1 to 58ml; Quartile Two, 59 to 96ml; Quartile Three, 97 to 175ml; and Quartile Four 176 to 2303ml. Data are presented as median [quartile 1, quartile 3] for continuously scaled data and frequency (percentage) for categorical data. Percentages were rounded to the nearest whole number; therefore, totals may not equal 100%. Baseline hemoglobin was missing for 416 cases: 42 in Quartile One, 74 in Quartile Two, 94 in Quartile Three, and 206 in Quartile Four. First and nadir ICU hemoglobin was missing for 2 cases in Quartile Two and Four. Baseline iron was missing for 6187 cases: 1699 in Quartile One, 1660 in Quartile Two, 1590 in Quartile Three, and 1238 in Quartile Four. Baseline TSAT was missing for 6247 cases: 1708 in Quartile One, 1674 in Quartile Two, 1602 in Quartile Three, and 1263 in Quartile Four. 95 patients contributed more than one procedure to the sample, with a max of three procedures contributed. ASD = absolute standardized difference, BMI = body mass index, BSA = body surface area, ICU = intensive care unit, TSAT = transferrin saturation.

Table 2: Characteristics of patients with 400 mL or greater estimated blood loss

	400+ n = 570
Age, years	64 [54, 72]
Male	389 (68)
Race	
American Indian or Alaska Native	1 (0)
Asian	12 (2)
Black or African American	46 (8)
Identifying with more than one race	8 (1)
Native Hawaiian or Other Pacific Islander	1 (0)
Unavailable	61 (11)
White	441 (77)
Height, inches	68 [64, 70]
Weight, pounds	176 [149, 204]
BMI, kg/m2	27.1 [23.4, 30.9]
BSA, m2	1.9 [1.8, 2.1]
Hypertension	215 (38)
Coronary Artery Disease	95 (17)
Heart Failure	213 (37)
Chronic Kidney Disease	82 (14)
Diabetes	140 (25)
ICU Length of Stay, days	10.8 [7.8, 15.8]
Baseline Hemoglobin, g/dL	11.8 [9.4, 13.8]
First ICU Hemoglobin, g/dL	9.7 [8.7, 11.1]
Nadir ICU Hemoglobin, g/dL	6.7 [6.3, 7.0]
Baseline Iron, mcg/dL	51 [35, 72]
Baseline TSAT, percent	18 [12, 27]
Intraoperative Blood Transfusions	3 [2, 5]
Postoperative ICU Blood Transfusions	4 [2, 7]
30 Day Mortality	90 (16)

Baseline hemoglobin was missing for 92 cases. First and nadir ICU hemoglobin was missing for one case. Baseline iron was missing for 332 cases. Baseline TSAT was missing for 346 cases.

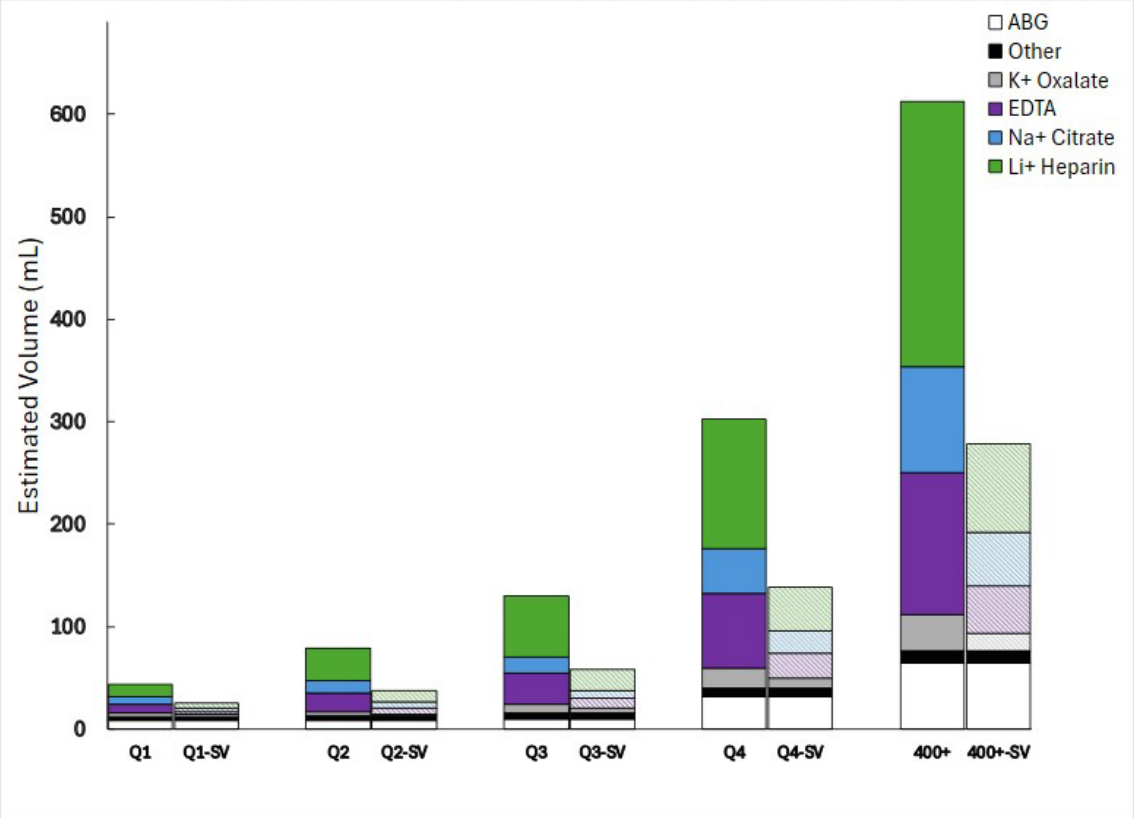
Table 3: Estimated blood loss according to tube type and projected volume conservation with use of small volume tubes

Tube type	*Group*	*Median volume (mL)*	*Proportion (%)*	*Tube size (mL)*	*Smaller volume available (mL)*	*Projected Volume Conserved (mL)*
Sodium Citrate				4	2	
	Quartile 1	8	18			4
	Quartile 2	12	15			6
	Quartile 3	16	12			8
	Quartile 4	44	15			22
	400 mL+	104	17			52
Potassium Oxalate				4	2	
	Quartile 1	4	9			2
	Quartile 2	4	5			2
	Quartile 3	8	6			4
	Quartile 4	20	7			10
	400 mL+	36	6			18
Lithium Heparin				6	2	
	Quartile 1	12	27			4
	Quartile 2	32	41			11
	Quartile 3	60	46			20
	Quartile 4	126	42			42
	400 mL+	258	42			86
EDTA				6	2	
	Quartile 1	8	18			3
	Quartile 2	18	23			6
	Quartile 3	30	23			10
	Quartile 4	72	24			24
	400 mL+	138	23			46
ABG				1	n/a	
	Quartile 1	8	18			
	Quartile 2	9	11			
	Quartile 3	10	8			
	Quartile 4	32	11			
	400 mL+	64	10			
Other				varied	n/a	
	Quartile 1	4	9			
	Quartile 2	4	5			
	Quartile 3	6	5			
	Quartile 4	8	3			
	400 mL+	12	2			

Projected total volume conservation (mL)	
Quartile 1	13
Quartile 2	25
Quartile 3	42
Quartile 4	98
400 mL+	202

The estimated blood volume loss (as group median and proportion of total) attributable to different types of phlebotomy tubes was estimated using the quantity of distinct laboratory tests and common volumes for each type of tube. To project the potential volume that could be conserved by use of small-volume tubes (if available), the median (per group) volume for each type of tube was multiplied by the ratio of the small to common volume. The projected volume savings for each tube type were summed to determine a total projected volume conservation for each group. ABG = arterial blood gas, EDTA = ethylenediaminetetraacetic acid, Other = serum, serum separator, sodium heparin tubes.

Figure 1: Estimated blood loss according to tube type and projected volume conservation with use of small volume tubes



The estimated blood volume loss attributable to different types of phlebotomy tubes across quartile groups and in patients with 400 mL or greater of total estimated blood loss (Q1, Q2, Q3, Q4, 400+) and the projected reduction in blood volume loss with use of small volume (SV) tubes (hashed boxes, Q1-SV, Q2-SV, Q3-SV, Q4-SV, 400+-SV). ABG= arterial blood gas; Other = serum, serum separator, sodium heparin tubes; K+ Oxalate = potassium oxalate; EDTA = ethylenediaminetetraacetic acid; Na+ Citrate = sodium citrate; Li+ Heparin = lithium heparin.

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SCA-BOM2-ECI-OP3

Machine Learning–Derived Predictors of Postcardiotomy Cardiogenic Shock After Cardiac Surgery

Presenting Author: Roopvir Kaur, MD

Background: Post-cardiotomy cardiogenic shock (PCCS) is defined as the inability to separate from cardiopulmonary bypass (CPB) or the development of persistent low cardiac output syndrome despite maximal pharmacological support, often necessitating temporary mechanical circulatory support (MCS).[1] Its incidence can be as high as 6% depending on the definition used, and it carries significant mortality and morbidity, reaching 46% to 67% in contemporary cohorts.[2] The objective of this study was to develop a predictive model for PCCS in patients undergoing cardiac surgery.

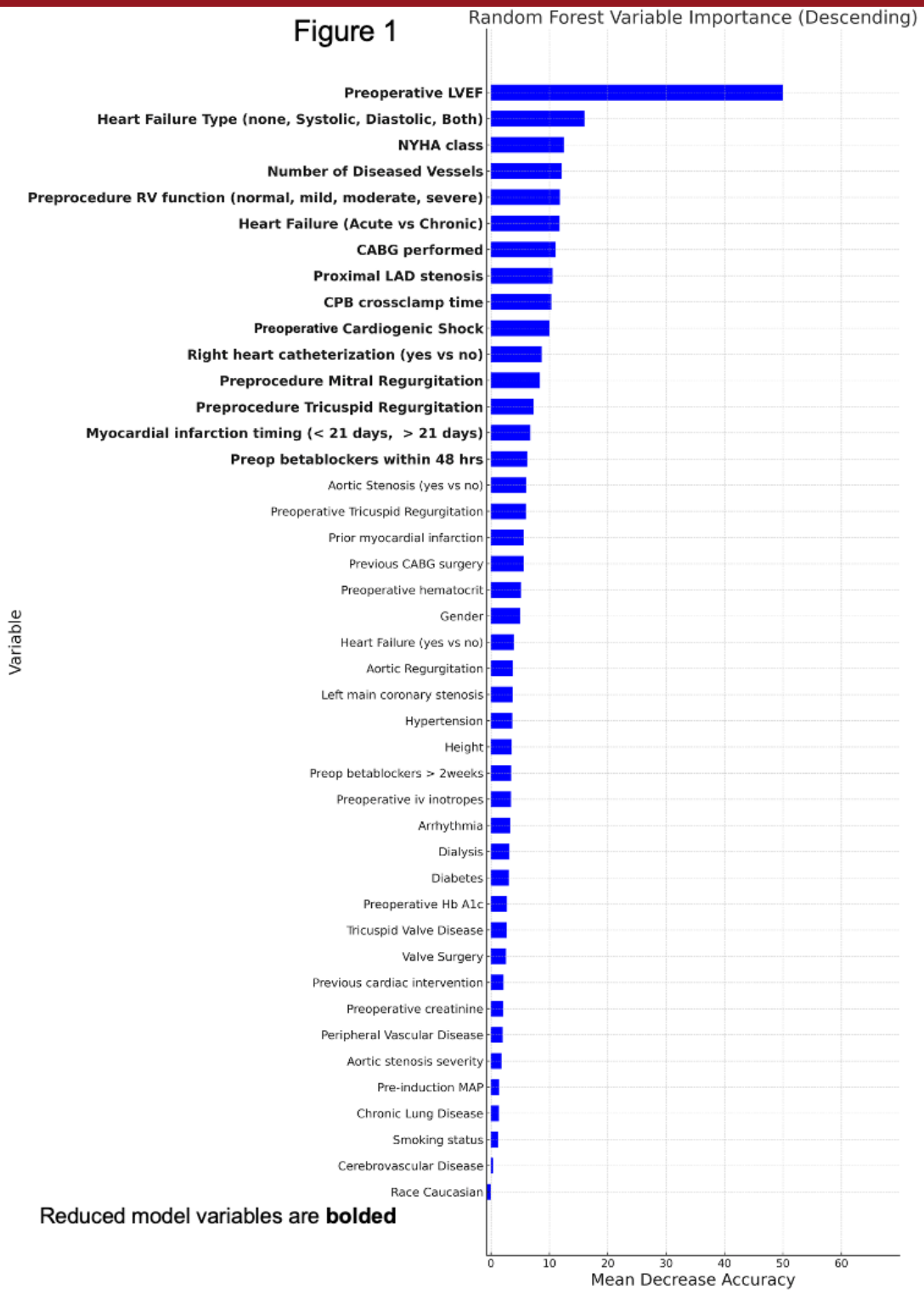
Methods: We performed a retrospective single center cohort study approved by the institutional review board using our Society of Thoracic Surgeons (STS) database between January 2018 and August 2023. Adult patients undergoing coronary artery bypass graft (CABG) and/or valve surgery (aortic, mitral, tricuspid) were included. PCCS was defined as either 1) intraoperative or postoperative use of any type of temporary MCS or 2) intraoperative death[1]. Cases were randomly split 70%/30% into training and testing datasets. Variables considered are demographic, clinical, hemodynamic, procedural, and echocardiographic factors. Supervised learning using a Random Forest (RF) algorithm was conducted to select features among variables that met a univariate threshold of $p< 0.1$. Feature importance was quantified using the mean decrease in accuracy (MDA). The optimal number of top features for inclusion in the final model was determined through RF cross-validation. Model performance was assessed on the testing dataset. A multivariable logistic regression model incorporating all selected features was performed on the full cohort to evaluate independent predictors.

Results: Of 5069 patients included, 476 patients (9.4%) developed PCCS. Selected characteristics are presented in Table 1. We identified 79 relevant variables available preoperatively through separation from CPB. Univariate analysis yielded 43 variables ($p< 0.1$) for feature selection. Based on MDA and RF cross-validation error rates, a reduced 15-variable model was selected, with preoperative left ventricular ejection fraction (LVEF) emerging as the strongest predictor (MDA

70.37) (Figure 1). Both the reduced and full models demonstrated excellent discrimination performance (AUC 0.882 and 0.878, respectively) (Figure 2). Multivariable logistic regression results (Table 2) were consistent, identifying preoperative LVEF, CPB cross-clamp time, preprocedural right ventricular dysfunction of any severity, moderate or severe mitral regurgitation, and recent myocardial infarction as important independent predictors of PCCS(AUC=0.897). Conclusions: Our findings demonstrate that PCCS can be accurately predicted using a focused set of preoperative and intraoperative variables, with excellent model discrimination. This approach may enable earlier identification of high-risk patients and support targeted perioperative management strategies.

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Figure 1



Reduced model variables are **bolded**

Figure 2

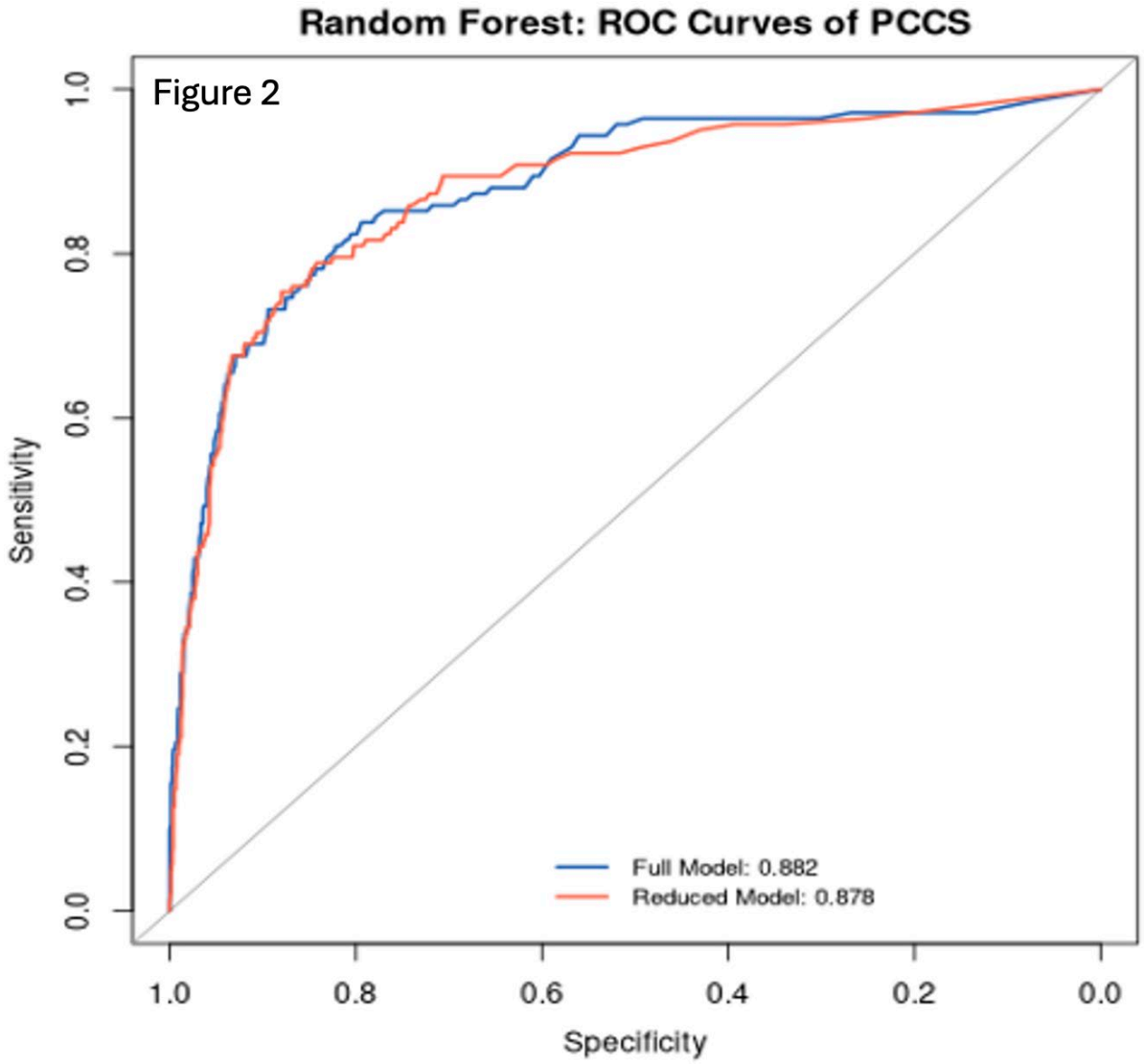

Random Forest: ROC Curves of PCCS
Sensitivity
Specificity
1.0
0.8
0.6
0.4
0.2
0.0
Full Model: 0.882
Reduced Model: 0.878

Table 1. Selected characteristics of the patient population

Variable	No PCCS (N=4603)	PCCS (N=476)	Total (N=5079)
Preoperative LVEF	55.0 [10.0, 87.0]	35.0 [10.0, 65.0]	55.0 [10.0, 87.0]
Heart Failure (Acute vs Chronic)			
Acute	473 (10.3%)	73 (15.3%)	546 (10.8%)
Acute on Chronic	395 (8.6%)	153 (32.1%)	548 (10.8%)
Chronic	2519 (54.7%)	205 (43.1%)	2724 (53.6%)
Heart Failure Type			
Systolic and Diastolic	217 (4.7%)	94 (19.7%)	311 (6.1%)
Diastolic	2478 (53.8%)	110 (23.1%)	2588 (51.0%)
Systolic	658 (14.3%)	222 (46.6%)	880 (17.3%)
Preoperative Cardiogenic Shock	246 (5.3%)	94 (19.7%)	340 (6.7%)
Preprocedure RV Function			
Normal	2739 (59.5%)	194 (40.8%)	2933 (57.7%)
Mild Dysfunction	427 (9.3%)	117 (24.6%)	544 (10.7%)
Moderate Dysfunction	69 (1.5%)	36 (7.6%)	105 (2.1%)
Severe Dysfunction	4 (0.1%)	14 (2.9%)	18 (0.4%)
Prior myocardial infarction	1270 (27.6%)	232 (48.7%)	1502 (29.6%)
Preprocedure Mitral Regurgitation			
None	2097 (45.6%)	147 (30.9%)	2244 (44.2%)
Mild	1107 (24.0%)	120 (25.2%)	1227 (24.2%)
Moderate	533 (11.6%)	125 (26.3%)	658 (13.0%)
Severe	866 (18.8%)	84 (17.6%)	950 (18.7%)
NYHA heart failure class			
Class I	530 (11.5%)	20 (4.2%)	550 (10.8%)
Class II	1777 (38.6%)	137 (28.8%)	1914 (37.7%)
Class III	917 (19.9%)	197 (41.4%)	1114 (21.9%)
Class IV	146 (3.2%)	33 (6.9%)	179 (3.5%)
Number of Diseased Vessels			
None	404 (8.8%)	30 (6.3%)	434 (8.5%)
Three	1829 (39.7%)	274 (57.6%)	2103 (41.4%)
Two	580 (12.6%)	76 (16.0%)	656 (12.9%)
CABG Performed			
No	2071 (45.0%)	124 (26.1%)	2195 (43.2%)
Planned	2521 (54.8%)	351 (73.7%)	2872 (56.5%)
Unplanned	11 (0.2%)	1 (0.2%)	12 (0.2%)
Heart Failure (yes vs no)	3403 (73.9%)	432 (90.8%)	3835 (75.5%)
Preop betablockers > 2weeks	2491 (54.1%)	333 (70.0%)	2824 (55.6%)
Preop betablockers within 48 hrs	3011 (65.4%)	380 (79.8%)	3391 (66.8%)
Pre-induction MAP	92.4 [30.0, 149]	89.0 [30.0, 148]	92.4 [30.0, 149]
Preoperative Hb A1c	5.90 [1.00, 16.4]	6.20 [4.10, 15.2]	5.90 [1.00, 16.4]
CPB crossclamp time	95.0 [0, 398]	98.6 [0, 297]	97.0 [0, 398]
Diabetes	1588 (34.5%)	226 (47.5%)	1814 (35.7%)
Arrhythmia	1166 (25.3%)	176 (37.0%)	1342 (26.4%)
Proximal LAD stenosis	23.7 (35.8)	32.8 (40.7)	24.5 (36.4)
Right heart catheterization (yes vs no)	1661 (36.1%)	228 (47.9%)	1889 (37.2%)
Preoperative Tricuspid Regurgitation			
None	2985 (64.8%)	239 (50.2%)	3224 (63.5%)
Mild	1058 (23.0%)	139 (29.2%)	1197 (23.6%)
Moderate	326 (7.1%)	60 (12.6%)	386 (7.6%)
Severe	234 (5.1%)	38 (8.0%)	272 (5.4%)
Preoperative iv inotropes	67 (1.5%)	23 (4.8%)	90 (1.8%)
Previous CABG surgery	147 (3.2%)	44 (9.2%)	191 (3.8%)
Previous cardiac intervention	1550 (33.7%)	213 (44.7%)	1763 (34.7%)
Dialysis	157 (3.4%)	36 (7.6%)	193 (3.8%)
Peripheral Vascular Disease	464 (10.1%)	79 (16.6%)	543 (10.7%)
Preoperative creatinine	1.31 (1.31)	1.60 (1.66)	1.34 (1.35)
Tricuspid Valve Disease	1801 (39.1%)	234 (49.2%)	2035 (40.1%)

Abbreviations: CABG, coronary artery bypass graft; CPB, cardiopulmonary bypass; Hb, hemoglobin; LAD, left anterior descending; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; NYHA, New York Heart Association; PCCS, post-cardiotomy cardiogenic shock; RV, right ventricle.

Table 2. Results of multivariable Logistic Regression modeling PCCS by 15 predictors

Feature	Term	OR (95% CI)	p-value
Preoperative LVEF	Preoperative LVEF	0.90 (0.88, 0.91)	<0.0001
Heart Failure Type	Systolic and Diastolic vs None	0.72 (0.25, 2.49)	0.56
	Diastolic vs None	0.73 (0.26, 2.5)	0.59
	Systolic vs None	0.65 (0.23, 2.23)	0.45
NYHA heart failure class	I vs. No	0.59 (0.31, 1.05)	0.082
	II vs. No	0.72 (0.47, 1.11)	0.14
	III vs. No	0.94 (0.62, 1.11)	0.77
	IV vs. No	0.72 (0.37, 1.35)	0.31
Number of Diseased Vessels	One vs None	1.10 (0.62, 1.88)	0.74
	Three vs None	1.22 (0.68, 2.16)	0.5
	Two vs None	1.80 (0.99, 3.21)	0.050
Preprocedure RV function	Mild vs. Normal	1.67 (1.22, 2.28)	0.001
	Moderate vs. Normal	2.03 (1.12, 3.62)	0.018
	Severe vs. Normal	25.21 (5.8, 125.08)	<0.0001
Heart Failure **(Acute vs Chronic)**	Acute vs None	2.33 (0.64, 7.2)	0.16
	Acute on chronic vs None	3.08 (0.86, 9.32)	0.062
	Chronic vs None	2.12 (0.59, 6.46)	0.21
CABG Performed	Planned vs. No	1.34 (0.79, 2.28)	0.28
	Unplanned vs No	1.09 (0.04, 9.25)	0.95
Proximal LAD stenosis	Proximal LAD stenosis	1.00 (0.996, 1.003)	0.78
CPB crossclamp time	CPB crossclamp time	1.01 (1.008, 1.014)	<0.001
Preoperative Cardiogenic Shock	Preoperative Cardiogenic Shock	0.99 (0.66, 1.48)	0.95
RHC (yes vs no)	RHC (yes vs no)	1.40 (1.08, 1.82)	0.011
Preprocedure Mitral Regurgitation	Mild vs. None	0.97 (0.71, 1.32)	0.84
	Moderate vs. None	1.65 (1.17, 2.32)	0.004
	Severe vs. None	1.64 (1.09, 2.44)	0.017
Preprocedure Tricuspid Regurgitation	Mild vs. None	1.05 (0.71, 1.52)	0.82
	Moderate vs. None	1.22 (0.74, 1.98)	0.42
	Severe vs. None	1.59 (0.85, 2.86)	0.14
MI timing (< 21 days, >21 days)	MI timing (< 21 days, >21 days)	1.56 (1.07, 2.28)	0.020
Preop betablockers within 48 hrs	Preop betablockers within 48 hrs	1.91 (1.41, 2.62)	<0.0001

Abbreviations: CABG, coronary artery bypass graft; CPB, cardiopulmonary bypass; LAD, left anterior descending; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NYHA, New York Heart Association; PCCS; Post-cardiotomy cardiogenic shock; RHC, right heart catheterization; RV, right ventricle.

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SCA-BOM2-BOM-OP4

Comprehensive Assessment of the Response to Nitric Oxide in Patients with Combined Pre- and Post-capillary Pulmonary Hypertension

Presenting Author: Rafael Vinagre, MD

Background: Although it is typically assumed that the pulmonary vasodilation induced by inhaled nitric oxide (iNO) improves right ventricular (RV) function in the setting of pulmonary hypertension (PH), recent data suggest this may not be universally true (1). Factors such as whether the PH is acute or chronic and the etiology of chronic disease may affect how effectively iNO improves RV to pulmonary arterial (PA) coupling, i.e. the balance between contractility and afterload. Etiology is particularly relevant in patients with chronic PH secondary to left heart disease who exhibit both pre-and post-capillary contributions. The present study was designed to test the hypothesis that iNO does not improve RV:PA coupling in patients with combined pre-and post-capillary PH.

Methods: Data digitally recorded under an IRB-approved protocol from 18 patients undergoing diagnostic right heart catheterization were used for the study. A PA catheter was placed under local anesthesia and RV and PA pressures measured before and during inhalation of 40 ppm NO in 30% oxygen. At each time point cardiac output and stroke volume (SV) were measured by thermodilution. Single beat analysis was applied to the RV pressure waveform in combination with SV to derive end-systolic elastance (Ees) as a measure of contractility and effective PA elastance as a measure of total afterload, and their ratio (Ees/Ea) then used as a coupling metric (2). To provide context for the unitless Ees/Ea value, RV ejection fraction (RVEF) was also derived (3) and pressure volume (PV) loops modeled (4) for calculation of stroke work (SW), pressure volume area (PVA, a correlate of oxygen consumption), and efficiency (SW/PVA). Baseline and iNO data were compared by paired t or Rank Sum tests with a p value $\leq$ 0.5 considered significant.

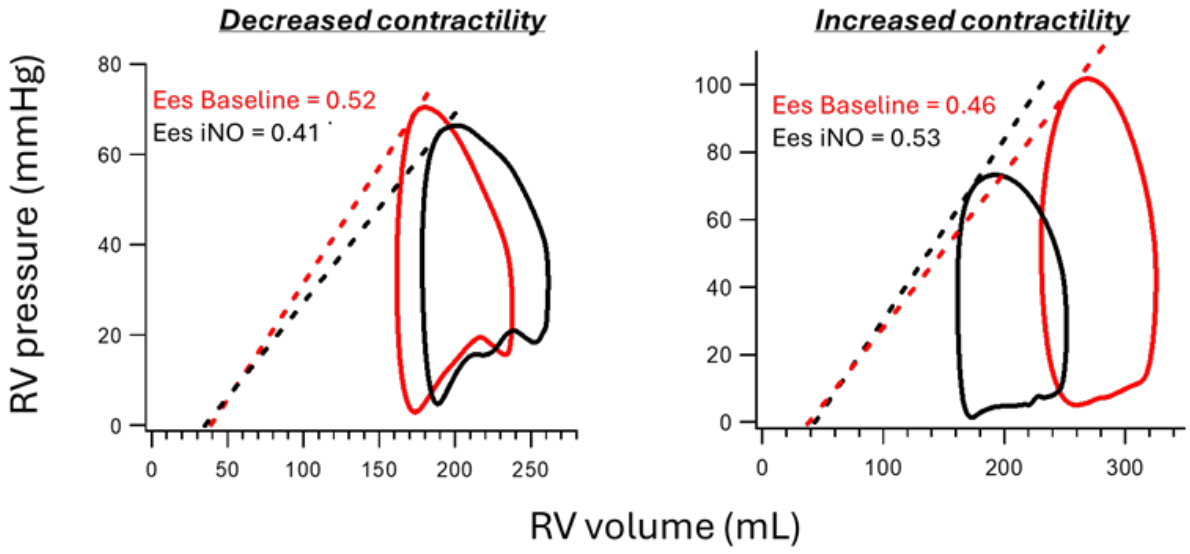
Results: Data are shown in table 1. On average iNO produced only modest decreases in mPAP and PVR that were coupled with improved Ees/Ea and RVEF due to a fall in Ea with no net change in Ees. However, PAWP was unchanged and the effect of iNO on Ees was highly variant with 11/18 patients showing a reduction $\leq$ 10% and 5/18 showing an increase $\geq$ 10%. Intriguingly, within the dataset the change in Ees/Ea was more closely correlated with changes in Ees ($r=0.562$, $p= 0.02$) than reductions in Ea ($r=-0.319$, $p=0.2$). Figure 1 compares PV loops when Ees increased or decreased.

Conclusions: While iNO is commonly used to treat RV dysfunction in patients with PH, efficacy may depend on whether the etiology of PH is pre- or post-capillary. Furthermore, data suggest that iNO may produce a negative inotropic effect in some patients that can offset a reduction in afterload and limit improvement on RV:PA coupling (1). The present study focused on patients with combined pre- and post-capillary PH and found that iNO produced a consistent but modest improvement in Ea and Ees/Ea thus disproving the study hypothesis. However, the effect on Ees was highly variable with the majority of patients exhibiting a decrease that dampened the impact of reduced afterload on improving RV:PA coupling. These preliminary findings underscore the need for individualized assessment of iNO responsiveness and challenge the assumption that afterload reduction alone predicts improvement in RV performance.

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Table 1	Baseline		iNO	
	MEAN	SD	MEAN	SD
mPAP (mmHg)	47	11	43 *	11
PAWP (mmHg)	17	1.5	17	2.6
RVEDP (mmHg)	17	4.0	15	5.2
CO (L/min)	6.3	1.9	6.3	1.8
SV (mL)	78	21	79	23
PVR (WU)	5.7	3.3	4.9*	3.1
Ees (mmHg/mL)	0.65	0.45	0.64	0.57
Ea (mmHg/mL)	0.80	0.37	0.73*	0.38
Ees/Ea	0.75	0.34	0.88*	0.40
RVEF (%)	0.41	0.11	0.44*	0.12
SW (mmHg* mL)	3832	1569	3344*	1137
PVA (mmHg* mL)	9469	4672	7424*	3324
SW/PVA	0.44	0.10	0.50	0.16
			* = $p \leq 0.5$	

Figure 1



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SCA-BOM2-BOM-OP5

Cryoablation vs Thoracic Epidural Analgesia for Postoperative Pain After Lung Transplantation

Presenting Author: Alana Davidson, BS

BACKGROUND

Inadequate analgesia after lung transplantation is challenging and may impair recovery and functional outcomes.[1,2] Thoracic epidural analgesia is the current standard at our institution but is associated with risks, including hypotension, delayed mobilization, and rarely epidural hematoma.[1,3,4] Cryoablation of the intercostal nerves has emerged as a potential alternative opioid-sparing strategy, though studies in lung transplant patients are limited.[5] We aimed to compare opioid utilization among patients receiving thoracic epidural analgesia versus intraoperative cryoablation. We hypothesized that thoracic epidural analgesia is associated with lower total opioid use when compared to cryoablation in the first seven days after lung transplantation.

METHODS

We conducted a retrospective cohort study of adult patients undergoing lung transplantation at Duke University Hospital from January 1, 2020, to April 30, 2025. Data were obtained from electronic medical record review and UNOS. We identified 16 cryoablation patients that met our

inclusion criteria which included first-time BOLTs or SOLTs performed with a clamshell incision that did not also have a cardiac procedure or other organ transplant, did not have a delayed sternal closure, and were not on ECMO upon arrival to the ICU postoperatively. Of the 16 patients identified, 10 received a rescue epidural (Figure 1). We achieved good matching between cryoablation and thoracic epidural patients at a 1:2 ratio using propensity score nearest-neighbor matching (Table 1).

We used descriptive statistics to compare cryoablation versus thoracic epidural patients. Our primary outcome was total opioid consumption converted to oral morphine milligram equivalents (MME). Secondary outcomes included reintubation, time and distance of first ambulation, intraoperative time, and ICU and hospital length of stay. Significant differences between groups were determined by either T-test, Mann-Whitney U-Tests, Chi-Square Tests, or Fisher Exact Tests.

RESULTS

Following matching, cryoablation patients had higher lung Composite Allocation (CAS) scores compared to epidural patients (32.99 ±4.69 vs 27.58 ±5.89; p = 0.003) (Table 1). Although cryoablation patients had high opioid consumption on POD1, (52.50 [38.50, 81.88] vs 26.82 [13.96, 45.62]; p = 0.006) they had lower cumulative opioid consumption over 7 days (p = 0.020) (Figure 2, Table 2). No other outcome measures of interest were found to be significant.

CONCLUSIONS

Intraoperative intercostal nerve cryoablation was associated with lower total opioid consumption over the first postoperative week, despite high opioid use on POD1. These findings suggest that early in the postoperative course there may be an analgesic gap, but overall, there is a potential longer-term opioid-sparing benefit for cryoablation versus thoracic epidural. Secondary clinical outcomes were largely comparable. It should be noted that a large percentage of the cryoablation cohort received a rescue epidural (62.5%). These data suggest that cryoablation may represent an alternative to thoracic epidural analgesia in lung transplantation, although studies are needed to determine who would benefit from cryoablation alone and when supplemental neuraxial analgesia may be required.

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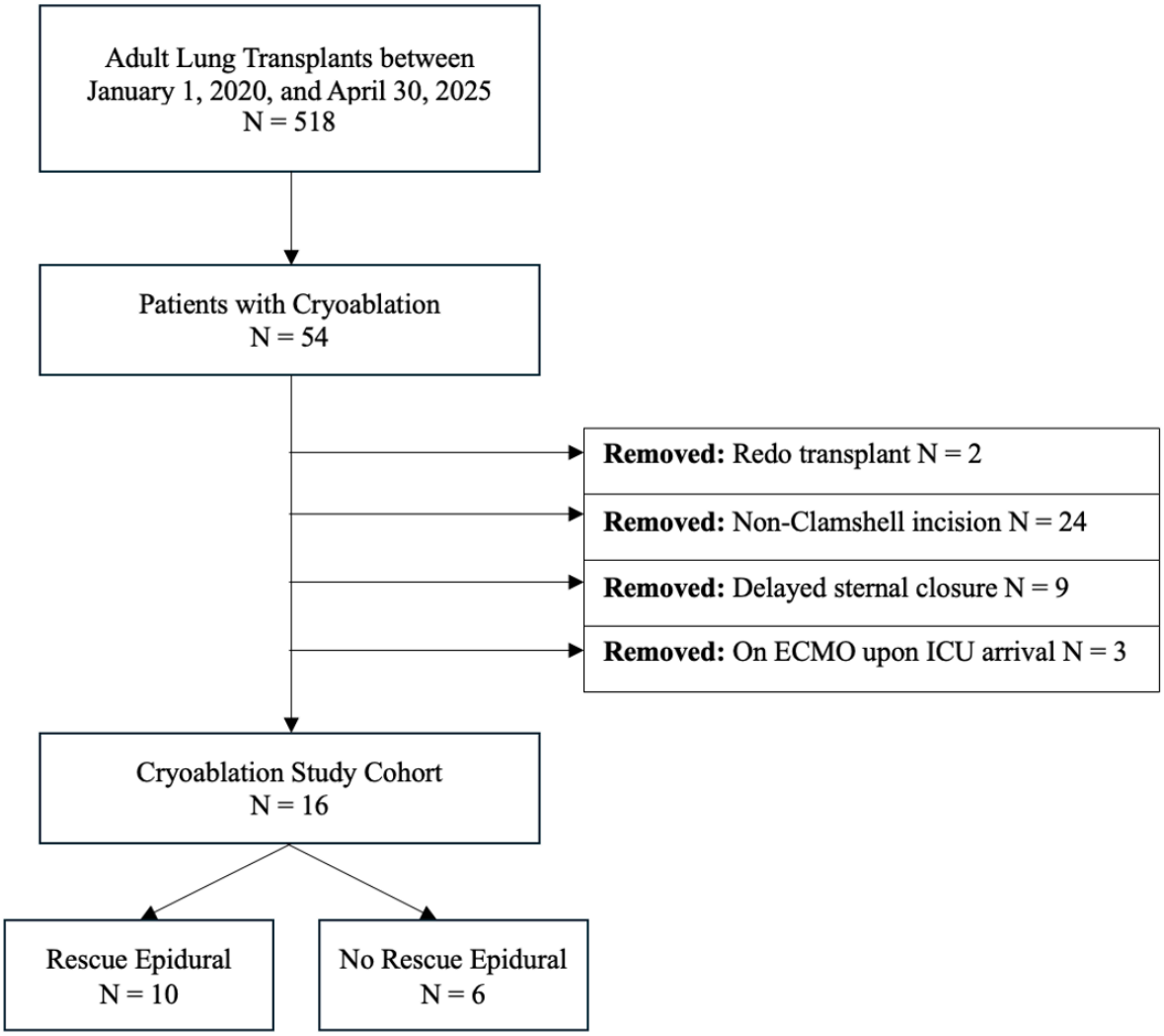


Figure 1. Consort Diagram

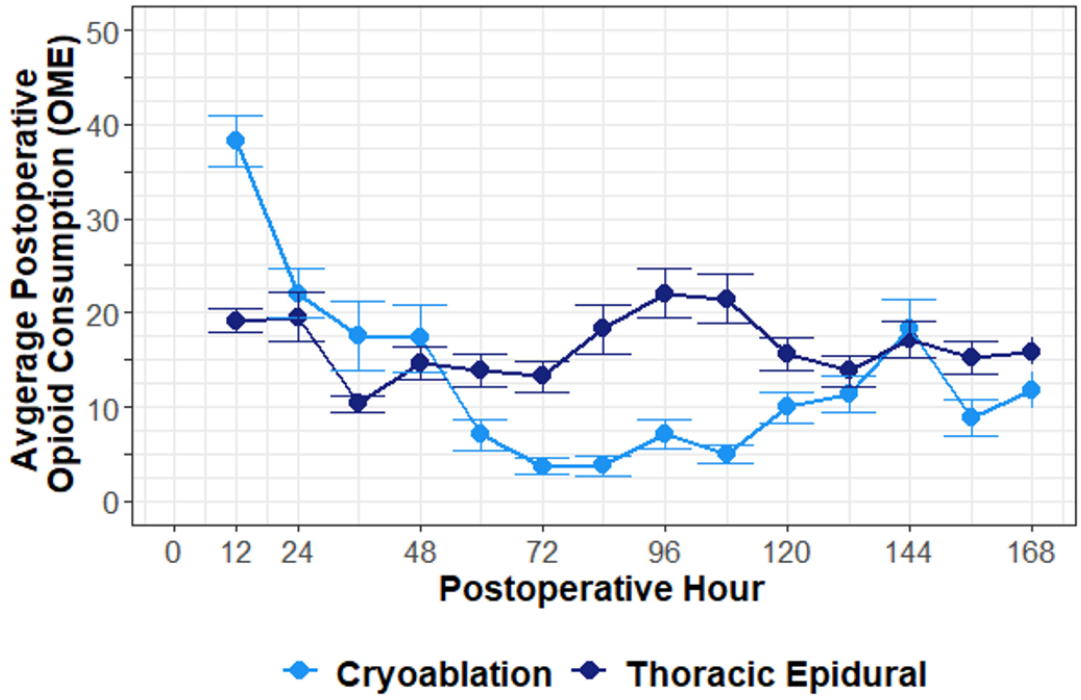


Figure 2. Total postoperative opioid consumption (OME) over the first 7 days by procedure group. Measurements are averages with 95% confidence intervals.

	Overall	Cryoablation	Thoracic Epidural	P-value
N	*48*	*16*	*32*	
Total Postoperative Opioids (OME) (median [IQR])	0.00 [0.00, 24.18]	0.00 [0.00, 0.00]	0.00 [0.00, 203.03]	0.020
POD1	35.00 [15.22, 55.00]	52.50 [38.50, 81.88]	26.82 [13.96, 45.62]	0.006
POD2	6.25 [0.00, 30.00]	2.50 [0.00, 33.12]	7.50 [0.00, 30.00]	0.889
POD3	0.00 [0.00, 20.41]	0.00 [0.00, 19.06]	0.00 [0.00, 22.50]	0.882
POD4	0.00 [0.00, 23.12]	0.00 [0.00, 16.88]	0.00 [0.00, 27.05]	0.560
POD5	0.00 [0.00, 31.88]	6.25 [0.00, 20.62]	0.00 [0.00, 41.25]	0.754
POD6	0.00 [0.00, 55.12]	7.50 [0.00, 56.25]	0.00 [0.00, 38.25]	0.255
POD7	1.64 [0.00, 41.15]	3.75 [0.00, 27.05]	1.64 [0.00, 46.88]	0.693
Biopsy Proven Rejection (30 Days) = Yes (%)	0 (0.0)	0 (0.0)	0 (0.0)	--
Reintubation = Yes (%)	7 (14.6)	1 (6.2)	6 (18.8)	0.398[b]
PGD Grade 2 or 3 (72 Hours) = Yes (%)	10 (21.3)	0 (0.0)	10 (32.3)	0.010[b]
Time to first ambulation (Hours) (median [IQR])	1.24 [0.75, 1.78]	1.20 [0.71, 1.72]	1.25 [0.79, 1.87]	0.736
First ambulation distance (median [IQR])	350.00 [150.00, 400.00]	400.00 [187.50, 400.00]	300.00 [150.00, 400.00]	0.512
ICU Length of Stay (Hours) (median [IQR])	3.85 [2.43, 7.11]	3.04 [2.17, 7.82]	3.97 [2.78, 7.11]	0.347
Postoperative Hospital Length of Stay (Days) (median [IQR])	18.53 [15.81, 27.20]	21.06 [15.64, 35.10]	18.23 [16.06, 21.38]	0.710

Table 2. Postoperative outcomes of interest by procedure group. Significant differences between procedure groups determined by Mann-Whitney U-tests and Fisher Exact Tests[b]

	Overall	Cryoablation	Thoracic Epidural	P-value
N	*48*	*16*	*32*	
Transplant Year = 2024 (%)	48 (100.0)	16 (100.0)	32 (100.0)	--
BOLT (%)	45 (93.8)	15 (93.8)	30 (93.8)	>0.999 [b]
Age (years) (median [IQR])	65.00 [58.75, 69.00]	64.00 [60.75, 69.25]	66.50 [58.75, 69.00]	0.818
Sex = Male (%)	30 (62.5)	9 (56.2)	21 (65.6)	0.752[c]
Race (%)				0.840[b]
Caucasian/White	40 (83.3)	14 (87.5)	26 (81.2)	
Black or African American	5 (10.4)	1 (6.2)	4 (12.5)	
Other	3 (6.2)	1 (6.2)	2 (6.2)	
BMI (kg/m2) (mean (SD))	25.15 (3.77)	24.39 (4.62)	25.53 (3.29)	0.329[a]
Transplant Indication (%)				>0.999 [b]
Obstructive Lung Disease	7 (14.6)	2 (12.5)	5 (15.6)	
Restrictive Lung Disease	41 (85.4)	14 (87.5)	27 (84.4)	

Table 1. Variables used in propensity score matching after matching was performed. Matching was done using the nearest neighbor technique with a 1:2 ratio. Significant differences between procedure groups determined by Mann-Whitney U-tests, Independent Samples T-Tests[a], Fisher Exact Tests[b] and Chi-Square Tests[c]

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SCA-BOM2-BOM-OP6

Cystatin C Thresholds for Early Prediction of Acute Kidney Injury after Cardiac Surgery

Presenting Author: Sreekanth R. Cheruku, M.D., M.P.H.

significant morbidity and mortality. While serum creatinine is most commonly used to diagnose postoperative AKI, this biomarker has a delayed peak which can occur up to 48 hours after kidney injury, is inaccurate in non-steady state conditions, and affected by variations in muscle mass. Cystatin C (cysC) is produced by all nucleated cells, freely filtered in the glomerulus, and has been found to be a robust predictive biomarker for postoperative in-hospital AKI after cardiac surgery[1-3]. We used data from a cohort of prospectively enrolled cardiac surgery patients to determine the optimal cutpoints for cysC which discriminate between patients who develop and do not develop post-operative in-hospital AKI.

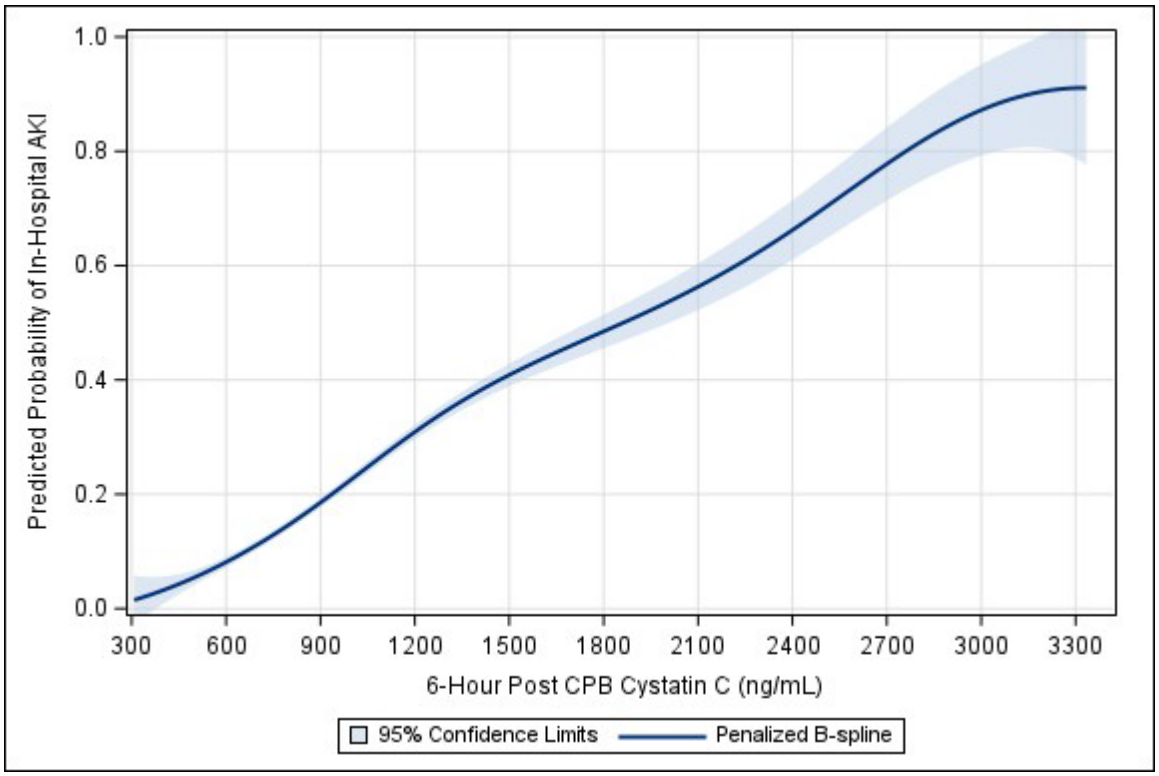
Methods: The study included 610 patients who underwent non-emergent cardiac surgery with cardiopulmonary bypass (CPB). Clinical variables were extracted from a database of prospectively collected data and cysC was assayed from blood samples collected 6 hours post-CPB. In-hospital AKI was defined using the Kidney Disease: Improving Global Outcomes (KDIGO) serum creatinine criteria: a rise in postoperative serum creatinine compared to pre-operative serum creatinine that was ≥0.3 mg/dL within the first 48 hours after surgery or ≥1.5 times the preoperative serum creatinine at any time through postoperative day seven or earlier if hospital stay was less than 7 days. Logistic regression was used to estimate the odds of postoperative in-hospital AKI from the cysC measurements with and without adjustment for clinical covariates. Receiver operating characteristic (ROC) analysis was implemented to identify the optimal cutpoint for the 6-hour post CPB measurement of cysC in discriminating postoperative in-hospital AKI.

Results: The incidence of postoperative in-hospital AKI was 18.03%. A two-fold increase in cysC measured 6-hour post CPB was associated with 3.561 greater predicted odds of developing in-hospital AKI (OR 3.561; 95% CI: 2.098–6.044; AUC=0.7593; $p< 0.0001$). Using the optimal cutpoint from the ROC, the logistic regression revealed, while adjusting for clinical covariates, that those who were above the cutpoint of 955 ng/mL were 3.55 times more likely to develop in-hospital AKI following cardiac surgery (OR 3.55; 95% CI: 2.17–5.80; AUC=0.7555; $p< 0.0001$; Figure 1).

Conclusion: cysC is an early an accurate predictor of postoperative in-hospital AKI after cardiac surgery. The cutpoints derived in this study should be validated in other cardiac surgery cohorts. Developing accurate cutpoints for cysC may allow early identification of cardiac surgery

patients at risk for post-operative AKI and enable treatments to mitigate this risk.

Images Uploads:



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SCA-BOM2-BOM-OP7

Disrupted FGF-21 Signaling is Associated with Impaired Cardiac Function in Both Metabolic and Non-metabolic Steatotic Liver Disease

Presenting Author: Juan D. Valencia, MD, MPH

Background

FGF-21 is a key hepatokine that plays a central role in regulating systemic metabolism, particularly during stress and fasting. Its effects on the heart are not well defined, with prior studies reporting both protective actions in heart failure with preserved ejection fraction (HFpEF) and associations with concentric hypertrophy. This study aimed to evaluate how FGF-21 influences cardiac health in mouse models of non-metabolic liver disease versus diet-induced obesity.

Methods

Eight-week-old male and female C57BL/6J mice were assigned to control, high-fat diet (HFD), or methionine–choline–deficient (MCD) diet groups. Diets were administered for 24 weeks beginning upon arrival. Transthoracic echocardiography (Vevo 2100) was performed under isoflurane anesthesia 3 days after arrival (baseline) and again after completion of the diet period. Systolic function was assessed using B-mode and M-mode imaging from the parasternal short-axis view to measure left ventricular (LV) dimensions and calculate fractional shortening (FS) and ejection fraction (EF). Diastolic function was evaluated from transmitral inflow velocities (E and A waves) to derive the E/A ratio. Parameters were compared using paired t-tests in GraphPad Prism. After sacrifice, plasma, liver, and heart samples were collected for histological analysis (H&E and Picrosirius Red) and for ELISA quantification of plasma FGF-21 and cardiac FGFR-1.

Results

Both diets produced steatotic liver disease with marked hepatic fat accumulation and fibrosis (Fig. 1A). Cardiac histology revealed increased fibrosis in HFD and MCD groups relative to controls (Fig. 1A). HFD resulted in a clear reduction in systolic function, while MCD diet produced a comparable degree of systolic impairment (Fig. 2A–B). Diastolic dysfunction was more prominent in MCD mice, which showed signs of elevated filling pressures (Fig. 2C–D). L-waves were observed in 30% of HFD mice, indicating abnormal filling despite non-significant changes in E/e' (Fig. 2H). Plasma FGF-21 levels were low in controls and significantly elevated in MCD and HFD groups, consistent with diet-induced FGF-21 upregulation and potential FGF-21 resistance (Fig. 1B). Correspondingly, cardiac FGFR-1 expression was reduced in both dietary groups (Fig. 1C).

Conclusion

Both diets impaired cardiac function and produced elevated FGF-21 with reduced FGFR-1, indicating disrupted FGF-21 signaling. These findings suggest a shared liver–heart axis contributing to cardiac injury across different forms of steatotic liver disease.

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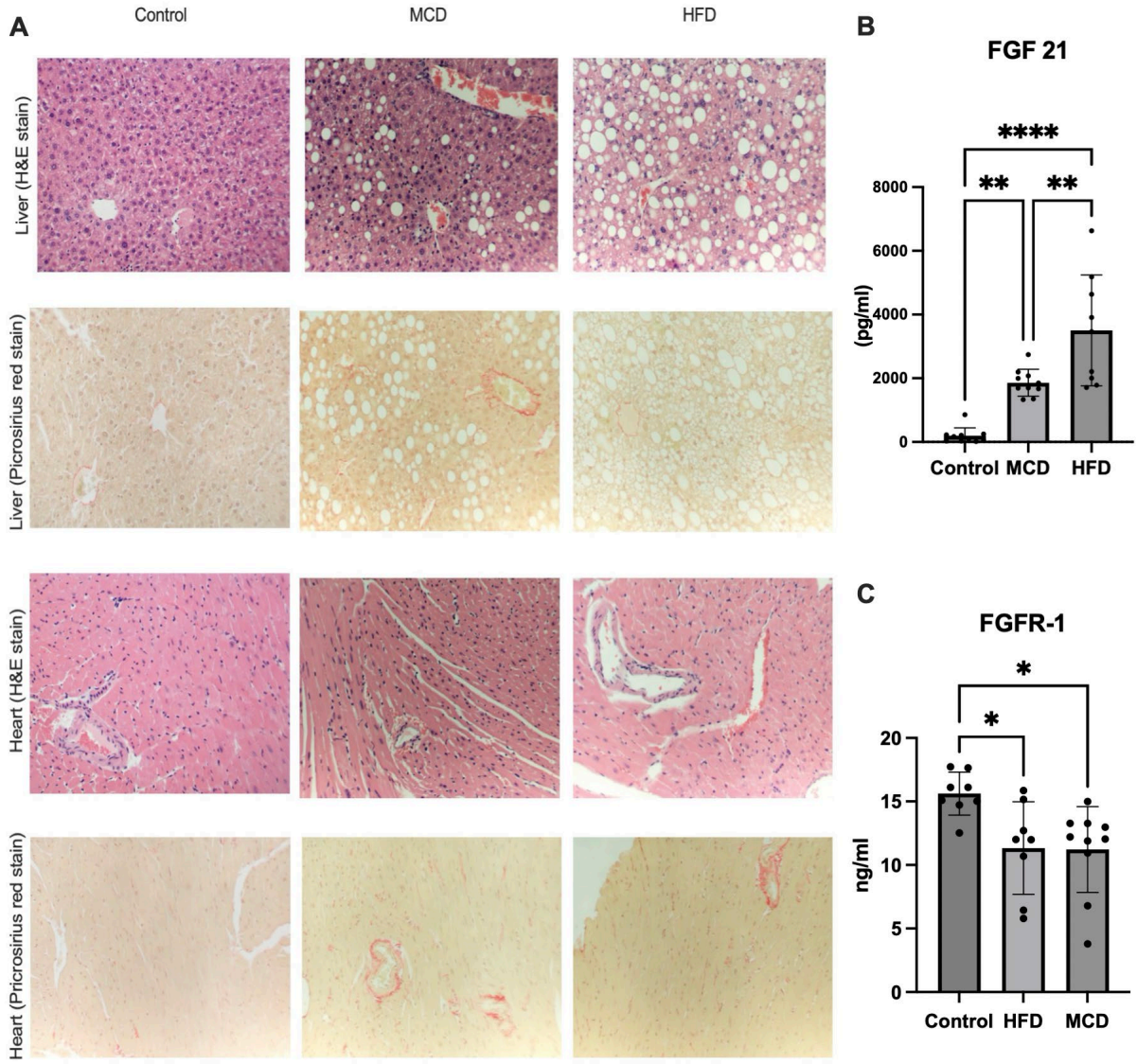


Figure 1: Histological Assessment of Liver and Heart Tissues and Corresponding FGF-21/FGFR-1 levels. A. Representative H&E and Picrosirius Red staining of liver and heart samples from control, MCD, and HFD mice. **B.** Serum FGF-21 concentrations measured by ELISA. **C.** Cardiac FGFR-1 levels measured by ELISA. FGF-21: Fibroblast growth factor 21. FGFR-1: Fibroblast growth factor receptor 1. * = $p<0.05$, ** = $p<0.01$ **** = $p<0.0001$

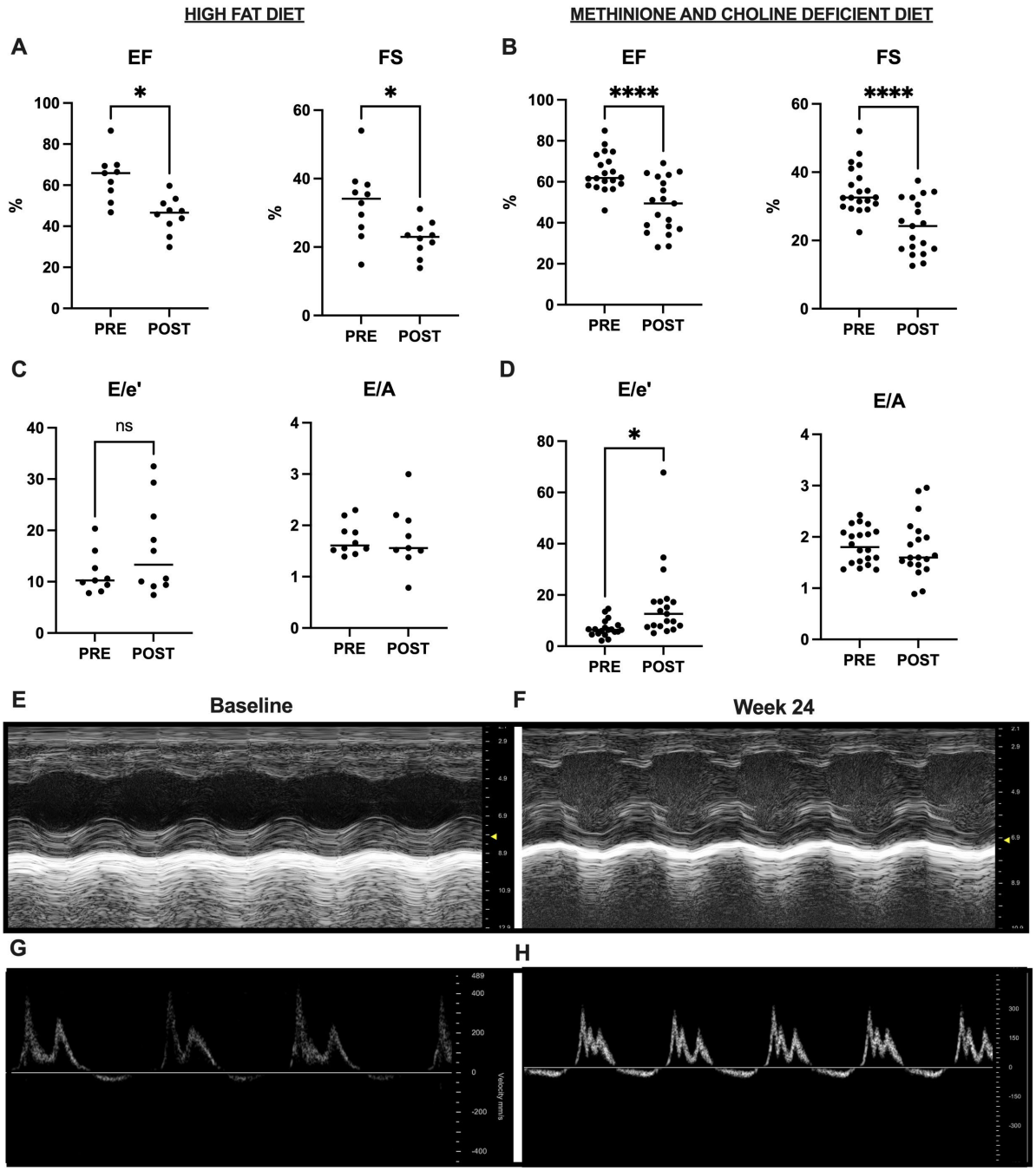


Figure 2: Echocardiographic evaluation of Systolic and Diastolic Function Before and After HFD or MCD. **A-B.** Ejection Fraction and Fractional Shortening at baseline vs 24 weeks of both HFD and MCD diets. **C-D.** E/e' and E/A ratio at baseline vs 24 weeks of both HFD and MCD diets. **E-F.** representative SAX image at baseline and post HDF respectively. **G-H.** Representative mitral valve pulse wave doppler at baseline and post HFD diet. EF = ejection fraction; FS = fractional shortening. E/e'. E = mitral valve E velocity based on pulse wave doppler; e' = lateral mitral annulus velocity based on tissue doppler. * = $p<0.05$, **** = $p<0.0001$

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SCA-BOM2-BOM-OP8

Intraoperative Effective Height Predicts Mid-Term Autograft Insufficiency After the Ross Procedure

Presenting Author: Christopher R. Glover, MD

Background

The Ross procedure is the only option for aortic valve replacement that preserves normal life-expectancy.[1] In patients under the age of forty, there is a twenty-year reduction in life expectancy after replacement with a bioprosthetic tissue valve, while mechanical valve replacement adds the additional comorbidity of lifelong anticoagulation.[1,2] While valve-sparing techniques, such as the Valve Sparing Aortic Root Replacement (VSARR) also preserve a normal life expectancy, not all surgical candidates are eligible based on valve pathology. As a result, Ross procedures are increasing globally.[3] However, the Ross procedure is technically challenging, with the possibility for postoperative complications, even in the absence of significant intraoperative findings on transesophageal echocardiography (TEE). One such outcome that can require surgical reintervention is clinically significant autograft insufficiency (AGI).[4] The severity of aortic valve insufficiency after tissue valve repair is inversely proportional to the aortic valve effective height, but this relationship has not been established following the Ross procedure.[5] In this single center, retrospective cohort study, we hypothesized that a greater autograft effective height measured on intraoperative TEE is correlated with a lower severity of postoperative AGI at mid-term (>1 month) outpatient follow up after the Ross procedure.

Methods

Patients over eighteen years of age with aortic valve insufficiency and/or stenosis who underwent the Ross procedure at Massachusetts General Hospital between 1/1/2024 and 9/30/2024 were identified. Data from the Society of Thoracic Surgeons Adult Cardiac Registry was acquired for each patient who met eligibility. Autograft effective height was measured by an echocardiographer blinded to post-operative AGI from archived intraoperative transesophageal echocardiographic

images. Presence and degree of postoperative AGI was obtained from the most recent transthoracic echocardiography report completed at least one month postoperatively or most proximate to aortic valve reintervention.

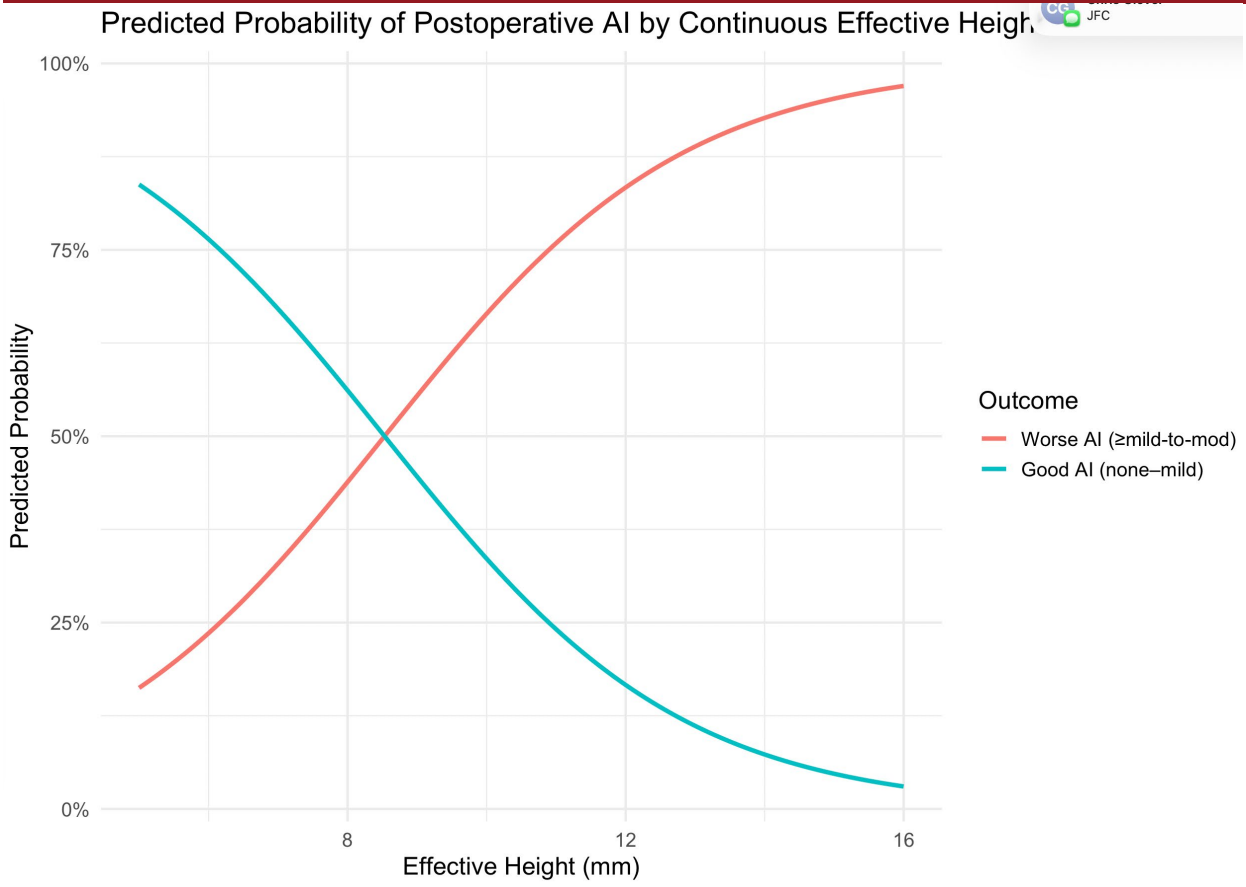
Results

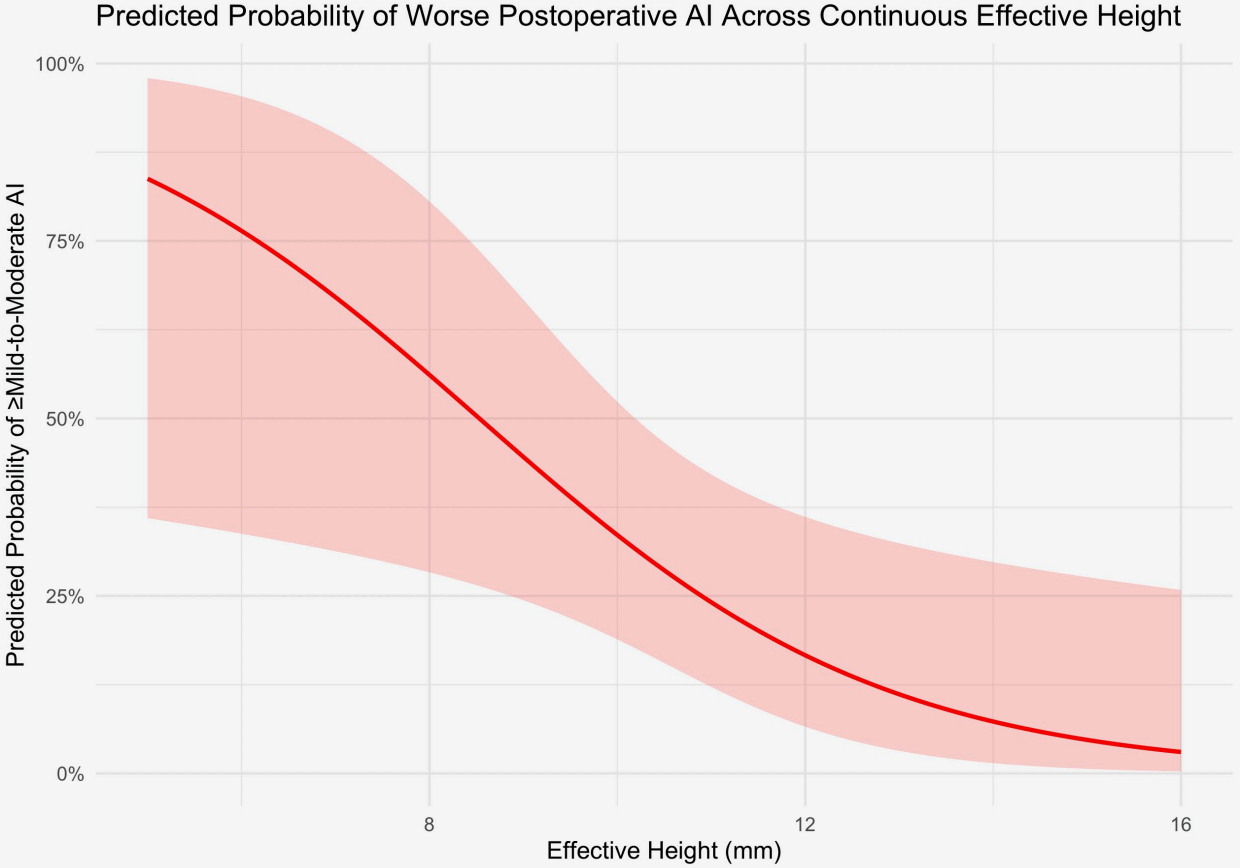
A multivariable ordinal logistic regression adjusting for age, height, and weight was performed. Greater autograft effective height was independently associated with lower postoperative AGI grade (n = 42; adjusted OR per 1-mm increase: 0.72; 95% CI, 0.55-0.95; p = 0.02). When postoperative AGI was dichotomized into “none-mild” vs “≥mild-to-moderate,” effective height ≥10.5 mm was independently associated with 80% lower odds of ≥mild-to-moderate postoperative AGI (adjusted OR, 0.19; 95% CI, 0.04-0.76). None of the candidate covariates (age, height, and weight) were significantly associated with AGI, and none confounded the relationship between effective height and AGI severity.

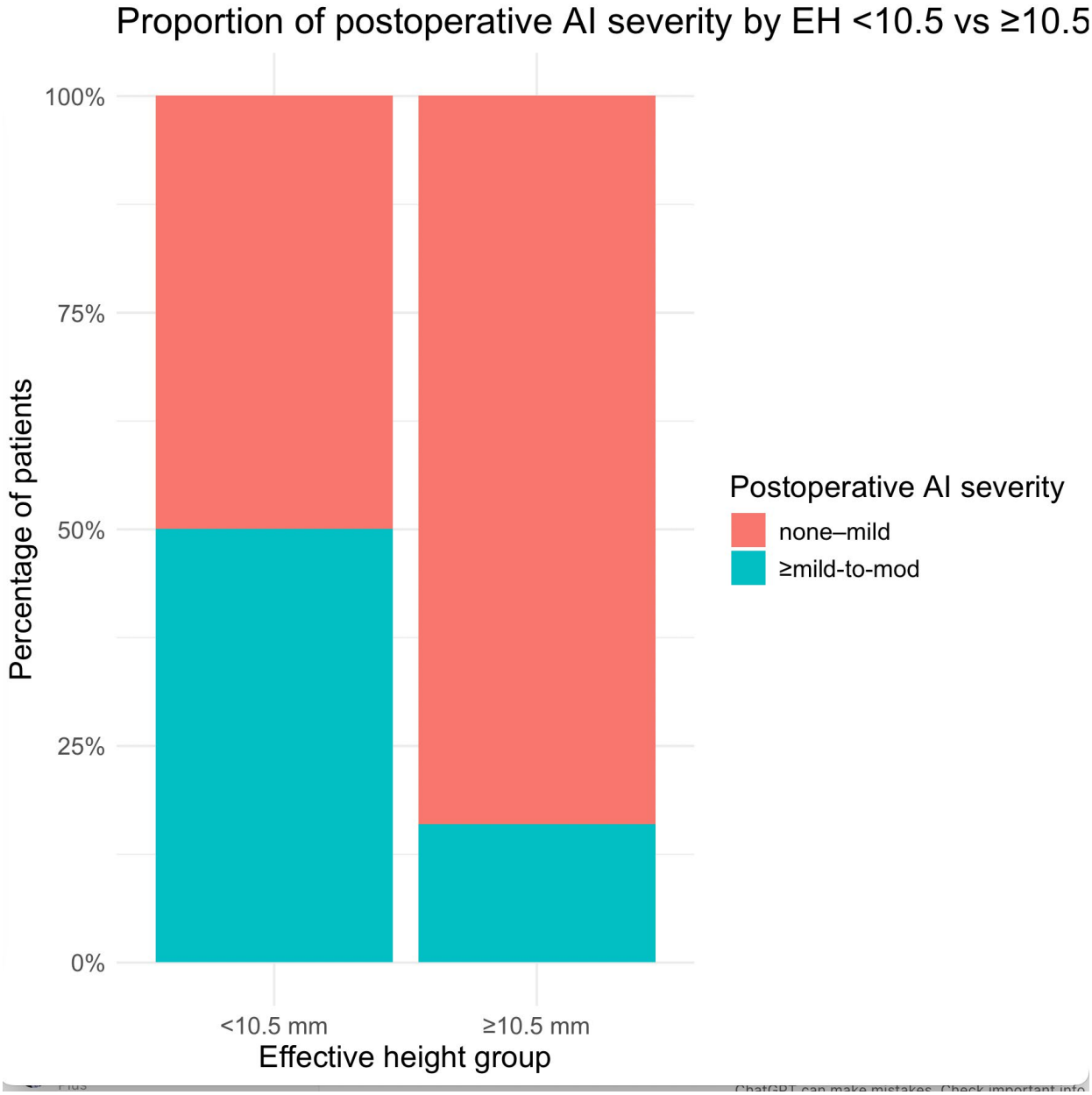
Conclusion

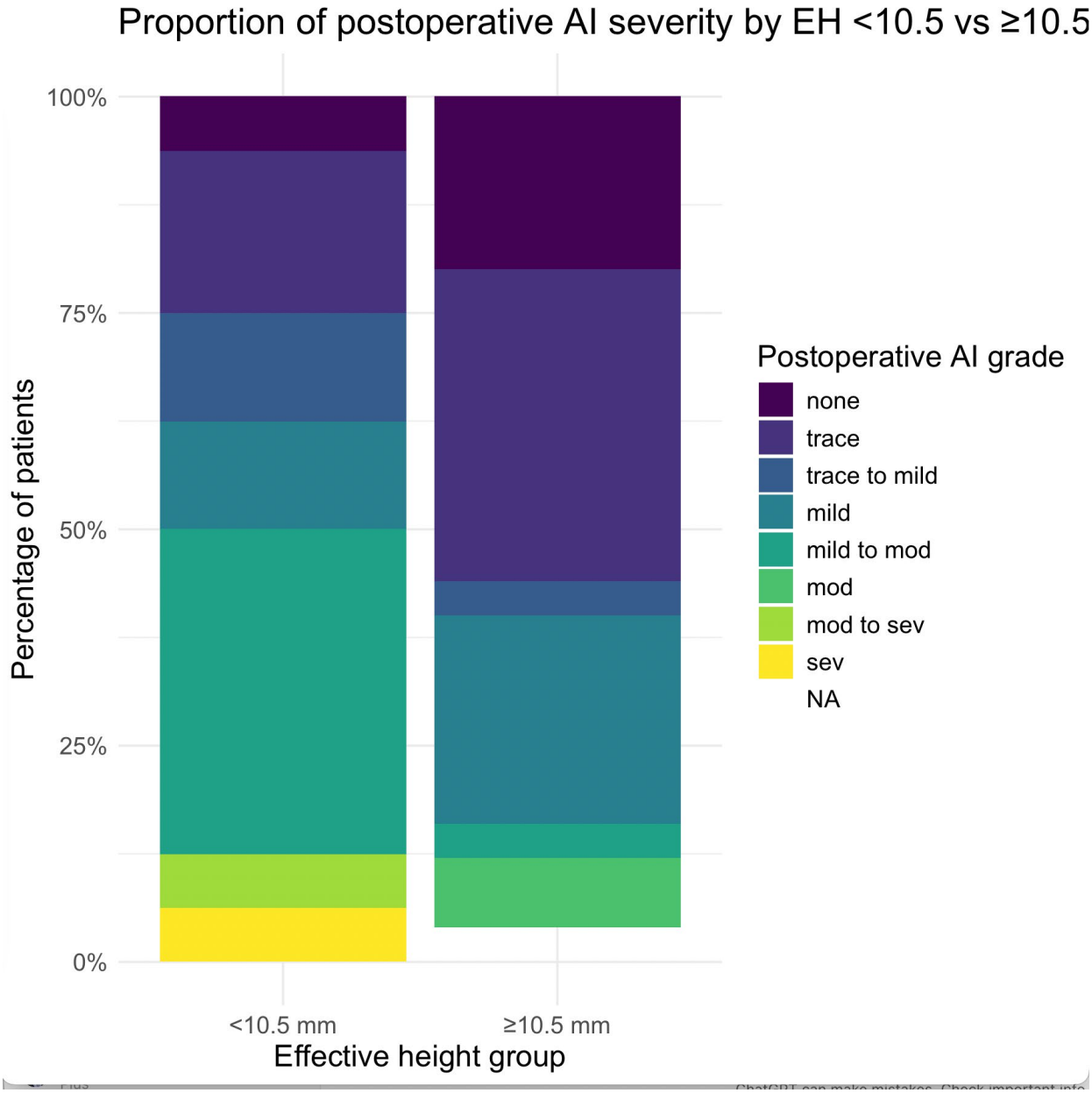
In this single center, retrospective cohort study, greater autograft effective height is associated with a lower degree of postoperative AGI. This relationship could provide a means for intraoperative echocardiographers to more accurately predict the development of clinically significant postoperative AGI, leading to fewer valve interventions. Larger studies are needed to further establish this relationship and elucidate longer-term outcomes.

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Predicted Probability of Postoperative AI by Continuous Effective Heigh
100%
75%
50%
25%
0%
Predicted Probability
8
12
16
Effective Height (mm)
Outcome
Worse AI (≥mild-to-mod)
Good AI (none–mild)


Predicted Probability of Worse Postoperative AI Across Continuous Effective Height
Predicted Probability of ≥Mild-to-Moderate AI
100%
75%
50%
25%
0%
8
12
16
Effective Height (mm)


Proportion of postoperative AI severity by EH <10.5 vs ≥10.5
100%
75%
50%
25%
0%
Percentage of patients
<10.5 mm
≥10.5 mm
Effective height group
Postoperative AI severity
none–mild
≥mild-to-mod



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SCA-BOM2-BOM-OP9

Perfusion-Related Factors Associated with Acute Kidney Injury in Non Emergent Adult Cardiac Surgery, A Bootstrap Reduced Prediction Model Incorporating Pre and Intra Operative Variables

Presenting Author: Ronald L. Gurbal, MD

Introduction: Acute kidney injury (AKI) occurs in 15-50% of adult cardiac surgery patients but varies widely because of institutional practices and populations. Identifying intraoperative factors that increase the risk of AKI is the first step in targeted management for prevention. We hypothesized that intraop factors, specifically prolonged time with high PaO2 on cardiopulmonary bypass (CPB), magnitude/duration of MAP<65mmHg, and mean vasoactive inotropic score (VIS)1, were linked to KDIGO AKI; we aimed to add these to our risk prediction model already incorporating preop factors.

Methods: IRB-approved, retrospective single academic center study of non-emergent adult CABG, aortic, mitral, and/or tricuspid valve surgical patients without pre-existing renal failure. The primary outcome was any stage AKI based on KDIGO (Kidney Disease Improving Global Outcomes), defined by baseline and daily creatinine up to 7 days postop. The secondary outcome was AKI stage 2 or 3. Patients were randomly split into training (70%) and testing (30%) datasets. Univariate logistic regression was performed on the training dataset, and variables meeting $p < 0.1$ (Table 1) were incorporated in the initial model. Backward selection, eliminating the least significant variable one at a time until the model reached the minimum AIC (Akaike information criterion), was conducted on the original training to determine the original model. This procedure was repeated on 100 bootstrapped datasets to form a reduced model that includes variables with a selection frequency of more than 80%. The PaO2 >280mmHg area under the curve/time on CPB (PaO2>280 AUC/time)

and preoperative creatinine were included regardless. ROC AUC was computed for both models for primary and secondary outcomes, respectively.

Results: Of 6,112 patients meeting the I/E criteria, 41.3% developed any stage postoperative AKI (2,116 stage 1, 263 stage 2, and 151 stage 3). Increasing age, BMI, preop creatinine, and medical history including arrhythmias, diabetes, hypertension, peripheral vascular disease, and COPD were associated with increased AKI risk (table 1). Intraop variables such as valve surgery, longer CPB time, higher MAP< 65 AUC/time and higher VIS mean also showed increased risk. The PaO2>280 AUC/time was not predictive in any models. Model performance was similar between the original model with 15 predictors and the reduced model with 10 predictors from the bootstrapping approach for both any AKI and stage 2/3, with a higher AUC (=0.797) for AKI stage 2/3 (Fig. 1).

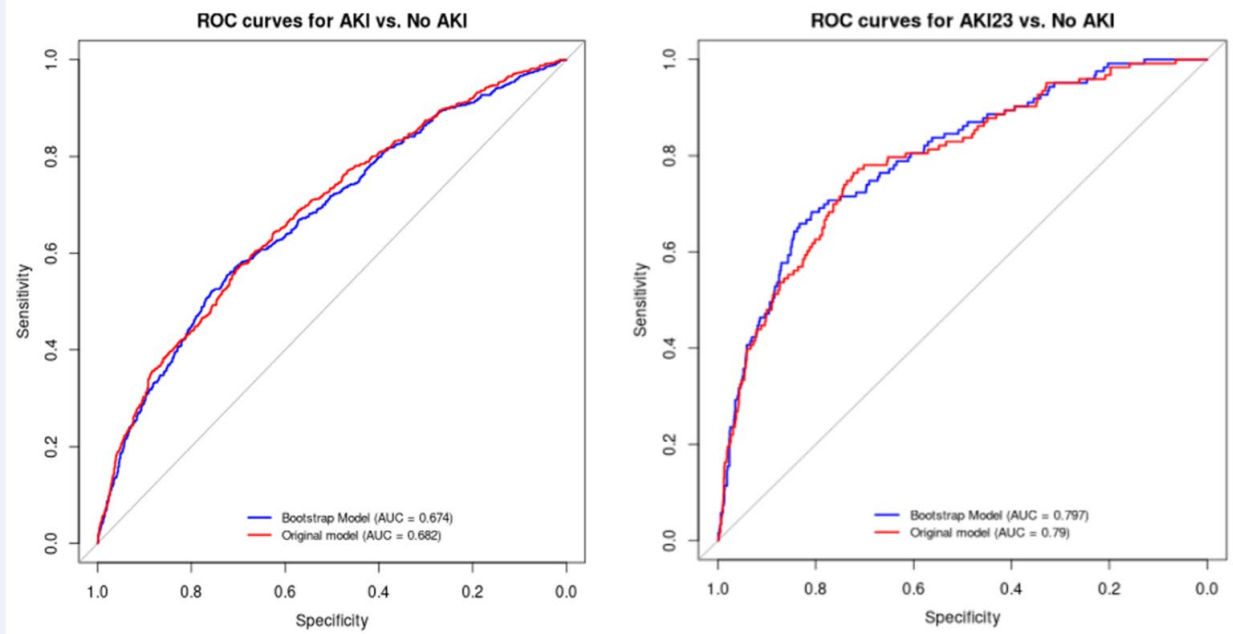
Conclusion: Our risk prediction model is well powered to identify patients at risk of developing clinically meaningful (stage 2/3) AKI. Intraoperative hemodynamic variables representing blood pressure and its management were associated with AKI risk. However, more modifiable factors such as PaO2>280 AUC/time on CPB were not associated with AKI risk within the range used on CPB at our institution. Future work includes incorporating DO2i into these models.

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Variable	KDIGO Stage 0	1	2	3	No AKI vs. any stage	No AKI vs.stage 2/3
	n=3582	n=2116	n=263	n=151	OR (95% CI)	OR (95% CI)
Age	63.31 (12)	65.43 (11.6)	63.94 (11.9)	65.14 (11.4)	**1.01 (1.00, 1.01)***	
BMI	28.25 (5.8)	29.62 (6)	30.96 (6.7)	29.74 (5.6)	**1.03 (1.02, 1.04)***	**1.06 (1.04, 1.08)***
Hypertension (%)	2248 (63)	1556 (74)	188 (71)	94 (62)	**1.19 (1.05, 1.36)***	1.22 (0.94, 1.58)
Diabetes (%)	1175 (33)	1045 (49)	154 (59)	97 (64)	**1.63 (1.43, 1.85)***	**3.41 (2.62, 4.44)***
HBA1C	6.11 (1.2)	6.42 (1.4)	6.39 (1.6)	6.09 (1.1)	1.03 (0.98, 1.08)	1.07 (0.95,1.19)
Insulin dependent (%)	716 (20)	606 (29)	67 (26)	32 (21)		**0.58 (0.41, 0.83)***
Arrhythmia history (%)	1237 (34)	960 (45)	160 (61)	106 (70)	**1.58 (1.40, 1.77)***	**2.68 (2.11, 3.41)***
PVD (%)	298 (8)	244 (11)	38 (14)	14 (9)	**1.22 (1.01, 1.46)***	**1.58 (1.1, 2.24)**
COPD (%)	237 (7)	209 (10)	33 (13)	9 (6)	**1.26 (1.04, 1.64)**	
Prior MI (%)	2166 (60)	1505 (71)	169 (64)	81 (54)	1.15 (0.96, 1.36)	
Preop Creatinine	1.04 (0.3)	1.16 (0.4)	1.02 (0.3)	1.28 (0.6)	**1.94 (1.64, 2.29)***	1.25 (0.9, 1.72)*
Angina (%)	348 (9.7)	198 (9.4)	18 (6.8)	8 (5.3)		
Hypercholesterolemia (%)	2118 (59)	1362 (64)	148 (56)	87 (58)		
Beta blocker preop (%)	1956 (55)	1278 (60)	159 (60)	81 (54)		
Inotropes preop (%)	57 (2)	41 (2)	5 (2)	5 (3)		
ARB/Ace Inhibitor preop (%)	152 (4)	113 (5)	19 (7)	5 (3.3)		
Hgb preop	11.03 (1.5)	10.79 (1.6)	10.88 (1.5)	10.75 (1.8)		
LVEF (%) 55+ (ref)	1441 (40.2)	722 (34.1)	106 (40.3)	50 (33.1)		
45-54	665 (18.6)	423 (20.0)	49 (18.6)	32 (21.2)		
35-44	309 (8.6)	232 (11.0)	27 (10.3)	16 (10.6)		
25-34	181 (5.1)	140 (6.6)	20 (7.6)	11 (7.3)		
15-24	101 (2.8)	117 (5.5)	11 (4.2)	10 (6.6)		
Unknown EF	885 (24.7)	482 (22.8)	50 (19.0)	32 (21.2)		
Tobacco (%)	395 (11)	258 (12)	38 (14)	12 (8)		
Procedures (%) CABG	2306 (64)	1614 (76)	179 (68)	87 (58)	**1.43 (1.20, 1.71)**	1.45 (0.99, 2.11)
Aortic Valve	604 (17)	443 (21)	67 (25)	49 (32)		
Mitral Valve	1453 (41)	676 (32)	123 (47)	84 (56)		1.34 (0.90, 2.00)
Tricuspid Valve	297 (8)	204 (9)	38 (14)	27 (18)		0.84 (0.58, 1.19)
IABP preop	22 (1)	22 (1)	3 (1)	4 (3)		
Redo Cardiac Surgery	320 (8.9)	268 (12.7)	41 (15.6)	28 (18.5)		
Intraoperative Variables						
MAP<65 AUC/time	2.40 (2)	2.63 (2.4)	2.57 (2.5)	2.54 (2.4)	**1.10 (1.04, 1.17)**	**1.16 (1.04, 1.3)**
Vasoactive Inotrope Score, intraop mean	7.2 (7.6)	9.1 (12.3)	10.8 (9.2)	11.9 (8.6)	**1.02 (1.01, 1.03)***	**1.02 (1.01, 1.04)***
CPB Time (minutes)	167.4 (68.2)	176.8 (76.1)	218.4 (98.4)	251.2 (99.9)	**1.43 (1.34, 1.52)***	**2.04 (1.82, 2.28)***
Cross clamp time (mins)	74.2 (63.0)	79.7 (65.9)	88.1 (78.5)	103.3 (96.6)		
P_aO_2>280AUC/time on CPB	24.4 (31.1)	24.57 (31.9)	21.94 (32.0)	18.44 (33.6)	1.00 (1.00, 1.00)*	1.00 (1.00, 1.00)*
$P_aO_2$min on CPB	181.8 (72.3)	181.0 (71.8)	172.9 (72.8)	174.9 (65.9)		
Tmin on CPB (Fahrenheit)	86.8 (8.5)	86.3 (9.6)	84.9 (9.9)	84.2 (9.3)		
Tmax on CPB (Fahrenheit)	98.3 (0.7)	98.3 (0.7)	98.4 (0.6)	98.5 (0.8)		
Hgbmin on CPB	10.8 (1.4)	10.6 (1.4)	10.6 (1.3)	10.3 (1.4)		
RBC transfusion intraop+24h, any (%)	141 (4)	87 (4)	15 (6)	11 (7)		

*Variable Included in Bootstrap Reduced Model; **Bold**: p<0.05 in GLM of full dataset; PVD: Peripheral Vascular Disease; VIS: Vasoactive Inotropic Score; CABG: Coronary Artery Bypass Grafting; CPB: Cardiopulmonary Bypass; IABP: Intraaortic Balloon Pump; ARB: Angiotensin Receptor Blocker; SD: Standard Deviation

Formulas: VIS = dopamine dose (mcg/kg/min) + dobutamine dose (mg/kg/min) + 100x epinephrine dose (mg/kg/min) + 100x norepinephrine dose (mg/kg/min) + 10,000x vasopressin dose (U/kg/min) + 10x milrinone dose (mg/kg/min); P_aO_2>280AUC/time on CPB = AUC of P_aO_2>280 mmHg/CPB time; MAP<65 AUC/time = AUC of MAP<65 mmHg/CPB time, z score transformed



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SCA-BOM2-BOM-OP10

Randomized Controlled Trial of Phenylephrine vs Vasopressin in Cardiac Surgery

Presenting Author: Jordan M. Stefko, MD

Background

During the perioperative period of cardiac surgery, intravenous phenylephrine or vasopressin are often used. While both vasopressin and phenylephrine cause vasoconstriction, their site of action at the kidney is markedly different. Vasopressin binds to V1 receptors at the glomerular efferent arterioles, causing glomerular efferent arteriolar vasoconstriction and thus increases glomerular filtration. In contrast, alpha-1 adrenergic agents mediate vasoconstriction of the afferent renal arteriole, therefore decreasing glomerular perfusion pressure and filtration. This study was

conceived to further explore previous investigational outcomes by comparing vasopressin to phenylephrine; a strict alpha-1 agent.

Methods

This is a randomized, open label study to investigate vasopressin versus phenylephrine in cardiac surgery. All patients >18 years of age presenting for coronary artery bypass graft (CABG), valve surgery, or combined CABG and valve surgery where included. Patients with left ventricular ejection fraction < 35%, mean pulmonary artery pressure > 35 mmHg, > mild right ventricular dysfunction, planned radial arterial graft, or circulatory arrest were excluded. Patients were randomized day of surgery to receive phenylephrine or vasopressin as first line vasopressor. Administration of study drug commenced for MAP < 65 following the protocol in Figure 1 and dosing in Table 1. We hypothesized that vasopressin treated patients will have reduced incidence of acute kidney injury (AKI) compared to patients treated with phenylephrine. A 15% absolute reduction in AKI was used for power analysis. With alpha set at 0.05, a sample size of 130 patients per treatment group (N=260) would provide 80% power to detect a significant difference between the 2 treatments. Primary outcome is AKI as defined by Kidney Disease Improving Global Outcomes criteria. Secondary outcomes are 30-day mortality, stroke, myocardial infarction, re-operation, sternal infection, atrial fibrillation, intensive care unit length of stay, and total vasopressor hours.

Results

267 patients completed the study protocol and were randomized to either phenylephrine (134) or vasopressin (133). The incidence of AKI in the study cohort was 17.2%. No significant difference was observed in AKI by treatment group (p=0.986). A statistically significant decrease in the incidence of new onset atrial fibrillation (19.5% vs 31.2%; p=0.027) and postoperative hospital length of stay (5.16 vs 6.14 days; p=0.040) was observed in the vasopressin group. No difference was observed in 30-day mortality, stroke, re-operation, or sternal infection.

Conclusion

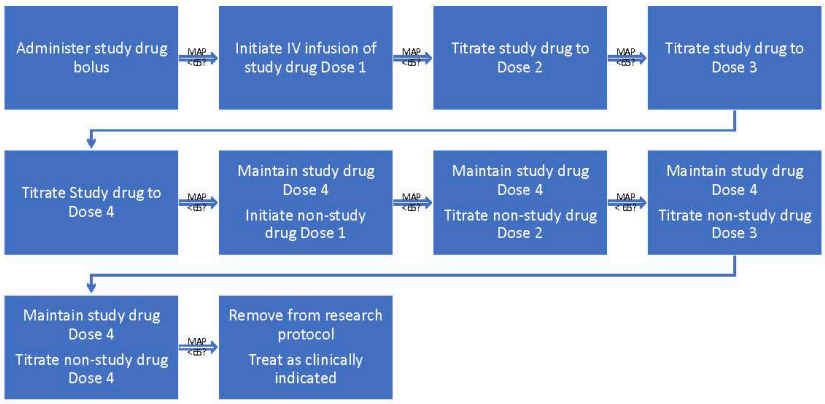
Our hypothesis that vasopressin treated patients will have reduced AKI outcome compared to patients treated with phenylephrine was not observed. Our observed incidence of AKI (17.2%) was much lower than historical data (24-42%), resulting in an underpowered study to detect our primary outcome. While not primarily powered to detect our secondary outcomes, we did observe a significant decrease in postoperative atrial fibrillation in the vasopressin group. This is a notable finding as postoperative atrial fibrillation is associated with increased risk of stroke, death, hospital length of stay, and 30-day readmission.

Image Uploads:

Table 1

	Phenylephrine	Vasopressin
Bolus Dose	100mcg/ml	0.4 U/ml
Dose 1	0.25 mcg/kg/min	0.01 U/min
Dose 2	0.5 mcg/kg/min	0.02 U/min
Dose 3	0.75 mcg/kg/min	0.03 U/min
Dose 4	1.0 mcg/kg/min	0.04 U/min

Figure 1


Administer study drug bolus
MAP <65?
Initiate IV infusion of study drug Dose 1
MAP <65?
Titrate study drug to Dose 2
MAP <65?
Titrate study drug to Dose 3
Titrate Study drug to Dose 4
MAP <65?
Maintain study drug Dose 4
Initiate non-study drug Dose 1
MAP <65?
Maintain study drug Dose 4
Titrate non-study drug Dose 2
MAP <65?
Maintain study drug Dose 4
Titrate non-study drug Dose 3
Maintain study drug Dose 4
Titrate non-study drug Dose 4
MAP <65?
Remove from research protocol
Treat as clinically indicated

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SCA-BOM2-BOM-OP11

Three-Way Comparison of Noninvasive Continuous Blood Pressure Cuff, Radial, and Femoral Arterial Catheters in Cardiac Surgery: A Prospective Observational Study

Presenting Author: Tahir El Meskine, MD

Background: The accuracy of continuous noninvasive blood pressure monitoring remains a key safety consideration in cardiac anesthesia. Prior evaluations of the ClearSight (CS) system have compared it exclusively with radial arterial cannulation (RC), yet its ability to approximate central arterial pressure has not been established. While the radial artery is frequently used because of its accessibility and lower complication rate, the femoral arterial cannula (FC) is considered a more reliable surrogate for central pressure, particularly during shock states and following cardiopulmonary bypass (CPB), when substantial radial–femoral pressure gradients (RFPG) may develop [1-3]. Accordingly, we sought to evaluate the performance of CS against simultaneous RC and FC measurements during cardiac surgery requiring CPB and to determine whether CS was non-inferior to RC in its ability to reproduce FC-derived central pressures.

Methods: As a predefined secondary objective within a prospective single-center study (NCT04092855), adult patients undergoing elective cardiac surgery received CS and RC monitoring before induction, while FC was placed after induction. Systolic (SBP), diastolic (DBP), and mean

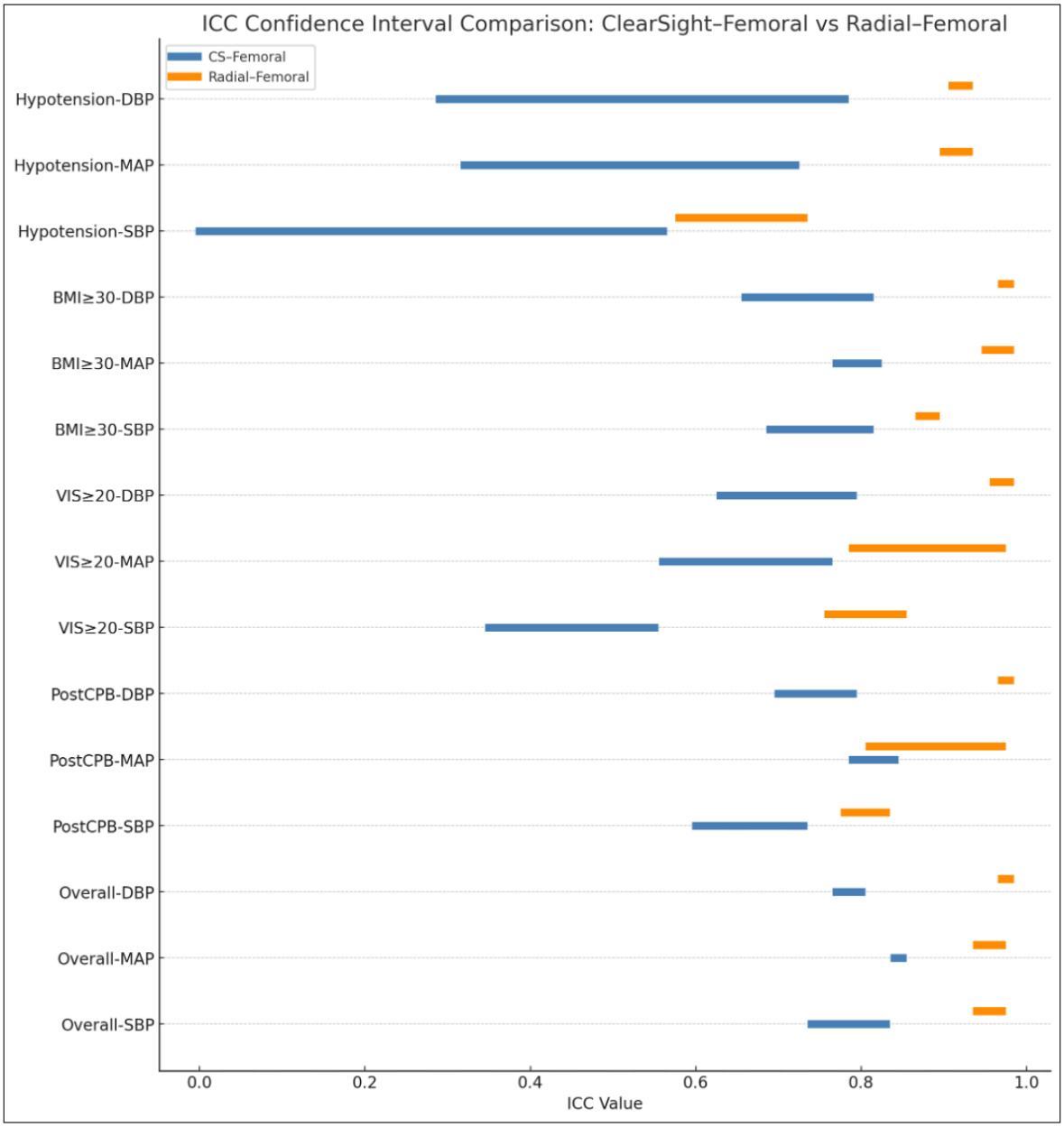
(MAP) pressures from all three systems were recorded continuously and averaged over 60-second intervals. Agreement was evaluated using Bland–Altman analysis, Association for the Advancement of Medical Instrumentation (AAMI) criteria (bias < 5 mmHg and SD < 8 mmHg), and intraclass correlation coefficients (ICC). Subgroup analyses were performed for post-CPB, hypotension (femoral SBP < 90 mmHg), Vasoactive-Inotropic Score (VIS) ≥ 20, and obesity (BMI ≥ 30 kg/m^2). Non-inferiority of CS relative to RC for estimating FC-derived pressure was defined by overlap of ICC confidence intervals between the CS–FC and RC–FC comparisons.

Results: Twenty-nine patients contributed 7,256 triplets of simultaneous measurements. CS met AAMI criteria for MAP and DBP across all subgroups, while SBP accuracy was below AAMI standards for all measurements. ICCs demonstrated moderate-to-good correlation for MAP [0.57-0.84] and DBP [0.63-0.77] and poor-to-moderate correlation for SBP [0.32-0.74] (Figure 1). Reduced performance was most particularly observed during hypotension and high VIS. When referenced to FC, non-overlapping ICC confidence intervals indicated that CS did not match the ability of RC to reproduce FC-derived pressures.

Conclusion: CS provides clinically acceptable performance for MAP and DBP under diverse intraoperative conditions, while SBP accuracy never met the predefined thresholds. The performance of the CS across all pressure components (SBP, DBP and MAP) was most negatively affected by hypotension and high vasoactive support. CS was found to be inferior to RC in assessing central arterial pressures. Further evaluation is warranted to better define its role in perioperative hemodynamic monitoring and if it can be used an alternative to invasive access in certain situations.

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Images

Figure 1
Intraclass correlation coefficients (ICC) interval comparison between the ClearSight-Femoral and Radial-Femoral



Abbreviations: CPB, cardiopulmonary bypass; SBP, systolic blood pressure; MAP, mean arterial pressure; DBP, diastolic blood pressure; VIS, Vasopressor-Inotrope Score; BMI, body mass index

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SCIENTIFIC PROGRAM ABSTRACTS

Poster Session 1

Friday, April 24, 2026 | 12:00 PM - 1:00 PM

Scientific Posters: Session 1

SP 1 - Kiosk 1 - Poster 1
Building a Multimodal Large Language Model for Physician–Patient Communication
Presenting Author: *Drake Ao*

BACKGROUND: With the widespread application of large language models (LLMs) in natural language processing tasks, their potential in medical settings is increasingly drawing attention[1]. For instance, ChatGPT and similar models are used to answer patient questions, offer health advice, and assist with treatment[2].And GPT is an advanced large language model that enables developers to build virtual simulation scenarios from text prompts, removing the need for branching-path simulations with prewritten dialogue[3].Physician–patient communication is central to healthcare services, yet it faces challenges such as information asymmetry, inefficiency, and patient comprehension barriers[4], Medical students lack repeatable, quantifiable communication training scenarios, making it particularly difficult to self-assess empathy skills such as “tone and intonation”[5] .This study aims to develop a large language model tailored for doctor patient communication. By integrating a multimodal virtual patient system (MVP), it establishes a closed-loop process of “dialogue-evaluation-feedback” to enhance the quality of doctor-patient interactions and support communication training for medical students.

METHODS: Integrated 100,000 anonymous Chinese doctor-patient dialogue transcripts with 800 hours of annotated audio featuring acoustic characteristics such as emotion, speech rate, and fundamental frequency to establish a multimodal corpus aligned across text, speech, and emotion. Employing a strategy combining domain-adaptive pre training with instruction-based fine-tuning, we developed the MedTalk-LLM large language model for medical communication. This model integrates multimodal information such as speech intonation to enhance both professional expertise and empathetic capabilities.

RESULTS: Preliminary experiments demonstrate that the MVP speech module can decompose audio events and emotions in real time: within a one-minute conversation, the system detects an average of 12 intonation events (pauses, stress, sudden changes in speech rate) and classifies emotions (anxiety/joy/neutral, etc.). The decomposition results show high consistency with manual annotations, providing reliable quantitative evidence for subsequent RATING scoring.

CONCLUSIONS: Compared to existing pure text-based large language models for doctor-patient communication, MVP employs dual-channel modeling of “semantics-tone” to upgrade traditional LLMs' single-text output into a three dimensional representation encompassing meaning, emotion,

and events. This shifts the training objective from merely "getting it right" to achieving "accurate and empathetic communication," significantly enhancing feedback granularity and credibility. However, the system currently exhibits insufficient robustness against overlapping speech, dialects, and background noise. Future plans include expanding multilingual and multilingual corpora, optimizing noise suppression modules, and conducting multicenter clinical trials to propel large-scale medical communication models from the "text era" into the true "multimodal era."

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SP 1 - Kiosk 1 - Poster 2

Changes in Dynamic Arterial Elastance After Deep Hypothermic Circulatory Arrest are Associated with Post-Operative Acid-Base Status

Presenting author: *Jace Bradshaw, MD*

Background: Deep hypothermic circulatory arrest (DHCA) during cardiac surgery is associated with vasoplegic syndrome, leading to longer hospital stays and higher mortality. Dynamic arterial elastance (Ea_{dyn}) is the ratio of pulse pressure variation to stroke volume variation PPV/SVV, capturing how changes in flow translate into pressure. A higher Ea_{dyn} indicates that a given increase in stroke volume produces a larger rise in arterial pressure (stiff/vasoconstricted system). Conversely, a lower Ea_{dyn} indicates that blood pressure is less responsive to flow changes (compliant/vasodilated system). Intraoperative Ea_{dyn} determination may lead to earlier detection of vasoplegic syndrome, which would allow for earlier intervention. We hypothesize that Ea_{dyn} decreases after cardiopulmonary bypass (CPB) and DHCA and is associated with acidosis.

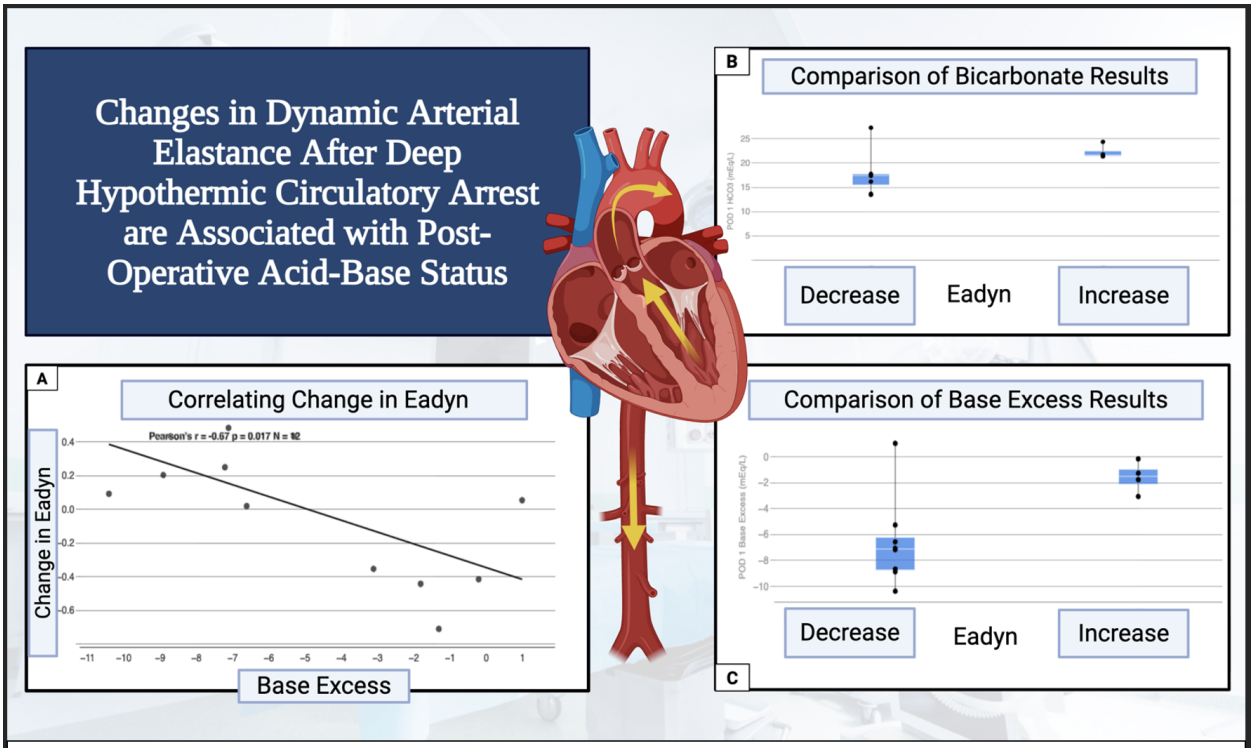
Methods: Adult canines (male and female, 18-25 kg) underwent DHCA for 90 min with antegrade cerebral perfusion. Femoral arterial waveforms were acquired in LabChart pre- and post-bypass. Waveforms were filtered and beats were detected using a trough-anchored model. Stroke volume (SV) per beat used the Liljestrand–Zander surrogate: SV = k (PP/(P_{sys} + PP_{dys}) with proportionality (k) determined from transesophageal echocardiogram-derived SV. For each respiratory cycle, PPV and SVV were computed, and Ea_{dyn} was derived. Primary analysis compared Ea_{dyn} from all respiratory cycles pre- vs post-bypass compared per-canine medians across pre- and post-bypass for all datasets (Mann–Whitney). Secondary analysis compared Ea_{dyn} to laboratory data (base excess, bicarbonate, pH, lactate, electrolytes) and neurologic outcomes determined by two validated assessments (Pittsburgh and Finnish Battery of Neurologic Tests).

Results: Arterial pressure data from a total of N=4486 pre-bypass respiratory cycles and N=5374 post-bypass respiratory cycles were extracted from 13 canine surgeries. While the pooled pre-to-post change in median Ea_{dyn} showed a decrease from 0.73 to 0.63 (< 0.001), the post-bypass change in Ea_{dyn} was not uniform among animals. While 9 animals showed a decrease in Ea_{dyn} (vasodilation), 4 animals showed an *increase* (vasoconstriction and preload dependence). This divergence was strongly associated with metabolic status. The "Ea_{dyn} Increase" group had a significantly better acid-base profile, with a higher median base excess (-1.55 vs -7.15, p=0.0485) and higher bicarbonate (21.6 vs 17.4, p=0.0485) than the "Ea_{dyn} Decrease" group (Fig 1). Across the entire cohort, DEadyn was significantly correlated with base excess (Pearson's r = -0.67, p=0.02), indicating that a *greater drop* in Ea_{dyn} was associated with *worse* acidosis (Fig 1). No significant associations were found with neurological outcomes.

Conclusions: In a canine model, Ea_{dyn} is useful for determining arterial load coupling to SV responsiveness and possible vasoplegia. While many animals exhibit a drop consistent with

vasoplegia, some develop an increase. Higher post-DHCA Ea_{dyn} is strongly associated with a more compensated acid-base status. Ea_{dyn} may serve as a critical, real-time hemodynamic parameter for phenotyping postoperative vasoreactivity and identifying underlying metabolic acidosis, but the causality of this relationship is unknown.

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(A) Each point represents one animal; line depicts simple linear regression (Pearson's r = –0.57, p = 0.017; Spearman ρ = –0.60, p = 0.041). Animals demonstrating a greater decrease in Eadyn exhibited more negative base excess values. (B) Bicarbonate concentrations stratified by whether each animal showed a median decrease (N=9) vs increase (N=4) in Eadyn after DHCA (C) Base excess values similarly stratified by direction of Eadyn change, demonstrating that decreases in Eadyn were associated with more pronounced metabolic acidosis.

References

Reference 1
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SP 1 - Kiosk 1 - Poster 3

Delayed Post-Resuscitation Hyperthermia is Associated with High Mortality in a Rat Model of Ventricular Fibrillation Cardiac Arrest

Presenting Author: *Mark Sourial, DO*

Introduction: Out-of-hospital cardiac arrest (OHCA) affects over 450,000 individuals annually in the United States. Despite successful resuscitation, severe cerebral and myocardial dysfunction often result from ischemia-reperfusion injury. Literature suggests that the rate of reperfusion impacts organ injury, and that ischemic post-conditioning (IPOC), controlled pauses during cardiopulmonary resuscitation (CPR), may mitigate this damage. This study utilized a clinically relevant rat model of ventricular fibrillation (VF) cardiac arrest to characterize post-resuscitation physiological trends, investigating the relationship between core body temperature, arrest duration, and survival outcomes.

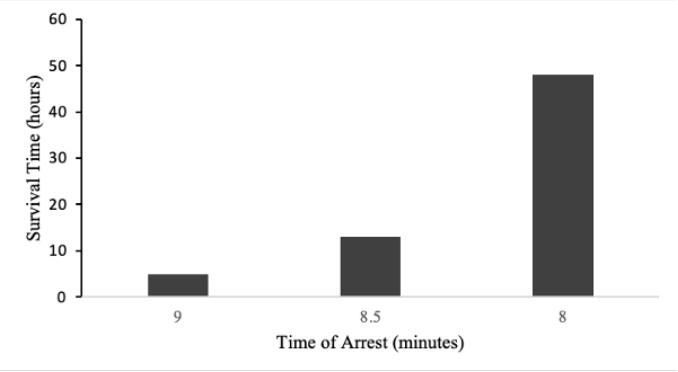
Methods: All experimental procedures were IACUC-approved. Adult rats underwent femoral catheterization for hemodynamic monitoring and telemetry implantation for continuous electroencephalogram (EEG) and core temperature recording. Baseline behavioral testing was performed. Cardiac arrest was induced via esophageal electrical stimulation to trigger sustained VF. Animals were subjected to a timed arrest period followed by mechanical CPR. The protocol optimized "no-flow" duration to achieve consistent Return of Spontaneous Circulation (ROSC). Post-resuscitation monitoring included continuous telemetry and survival analysis. Data are mean ± standard error of the mean (SEM). Statistical significance was determined using Student's t-test and log-rank tests (alpha=0.05).

Results: We established a consistent VF model with reliable ROSC by adjusting the no-flow interval. In the initial cohort (n=5), animals demonstrated initial neurological recovery, including normal movement and grooming. However, survival data indicated high delayed mortality; only one animal (20%) achieved long-term survival. Telemetry revealed a severe, delayed hyperthermic response. While baseline core temperatures averaged 36.2°C, post-resuscitation monitoring showed a progressive rise, spiking >39.0°C (peak ~39.1°C at 6 hours) in non-survivors. This hyperthermia strongly correlated with neurological deterioration and mortality, contrasting with the anticipated immediate hypoxic injury.

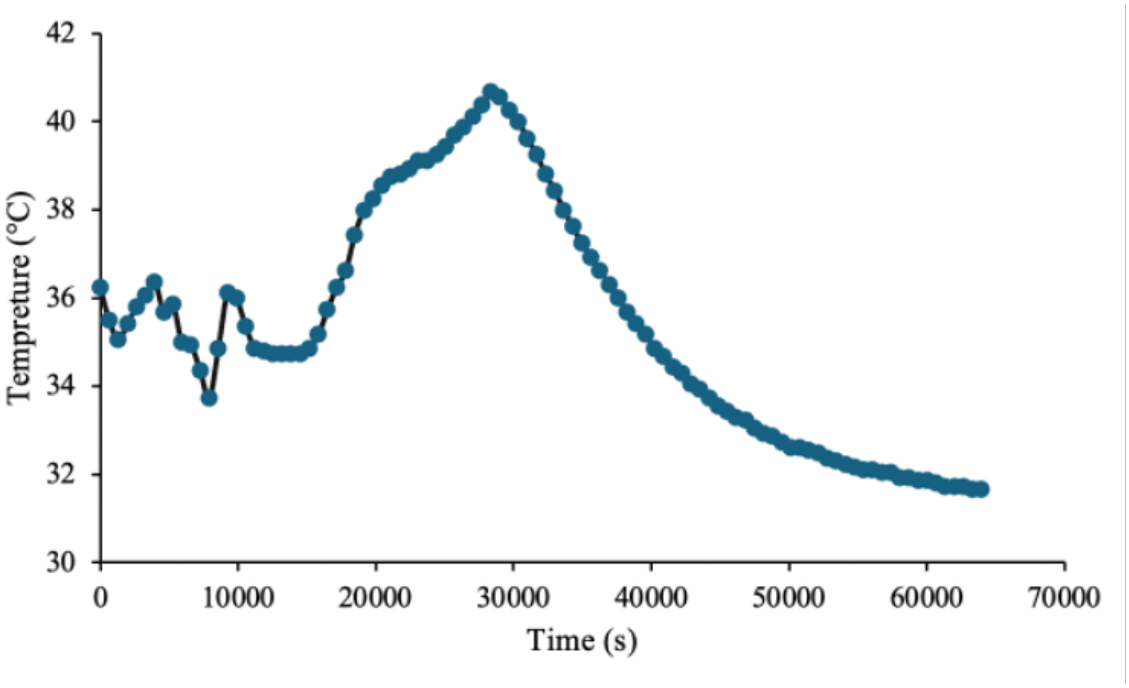
Conclusion: This study characterizes a lethal, delayed hyperthermic response in a rat model of VF cardiac arrest. While immediate ROSC was achievable, post-resuscitation hyperthermia appears to be a primary driver of delayed mortality and neurological injury. These findings suggest that therapeutic temperature management or pharmacological blockade of this spike may be essential

for long-term survival in rodent models. Future work will focus on blunting this response to accurately evaluate the efficacy of IPOC protocols.

Image uploads::







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SP 1 - Kiosk 2- Poster 1

Association of Dexmedetomidine Use with Complete Heart Block after Transcatheter Aortic Valve Replacement: A Retrospective Cohort Study

Presenting Author: *Eric Fried MD*

Introduction: Transcatheter aortic valve replacement (TAVR) is a percutaneous procedure to replace a dysfunctional aortic valve. Complete heart block is a common complication after TAVR and requires pacemaker (PPM) placement.1 Dexmedetomidine (DEX), a sedative-analgesic agent that has been shown to reduce post-operative delirium, especially in high-

risk patients, is often used in TAVR procedures. Known side effects of DEX are hypotension,bradycardia, and cardiac conduction abnormalities.2,3,4 We hypothesized that intraoperative DEX use would be associated with higher rates of PPM placement after TAVR.

Methods: Anesthesia and TAVR data for all cases between January 1,2019 and July 1,2025 were obtained from our departmental perioperative data warehouse and the cardiology TAVR registry. Only TAVR procedures performed under MAC in patients without an existing PPM were included in the analysis. The use of DEX, normalized by patient weight, was categorized into four levels: not used, low dose (< 1mcg/kg), medium dose (1-2mcg/kg), and high dose (>2mcg/kg). Mixed effect logistic regression was built used to assess the risk-adjusted odds ratio for pacemaker implantation at each dexmedetomidine level after accounting for patient demographics, comorbidities, and procedural characteristics, including valve type, size, and deployment position.

Results: 2,022 cases were included. There were no statistically significant differences in PPM rates at any dose of DEX: low dose (odds ratio (OR) 0.98 [0.64, 1.50], p=0.92), medium dose (OR0.78 [0.50, 1.22], p=0.28), and high dose (OR 1.05 [0.61, 1.82], p=0.85). (Figure 1) There was an increased rate of PPM placement in male patients (OR1.89 [1.36, 2.64], p<0.001), and those patients on dialysis (OR2.32 [1.11, 4.86], p=0.025), with a high risk preop EKG (OR 2.64 [1.75, 3.99], p<0.001), or who received a self-expanding valve (OR 2.65 [1.77, 3.96], p< 0.001). Larger size self-expanding valves (OR 1.25 [1.12, 1.39], p< 0.001), and post-balloon dilation were also associated with PPM placement (OR 1.51 [1.05, 2.18], p=0.028). There was a significant reduction in pacemaker rates when balloon-expandable valves were deployed in a more aortic position (OR0.96 [0.94, 0.98], p< 0.001).

Conclusions: Dexmedetomidine, when used in TAVR procedures, is not associated with an increased risk of post-op pacemaker implantation.

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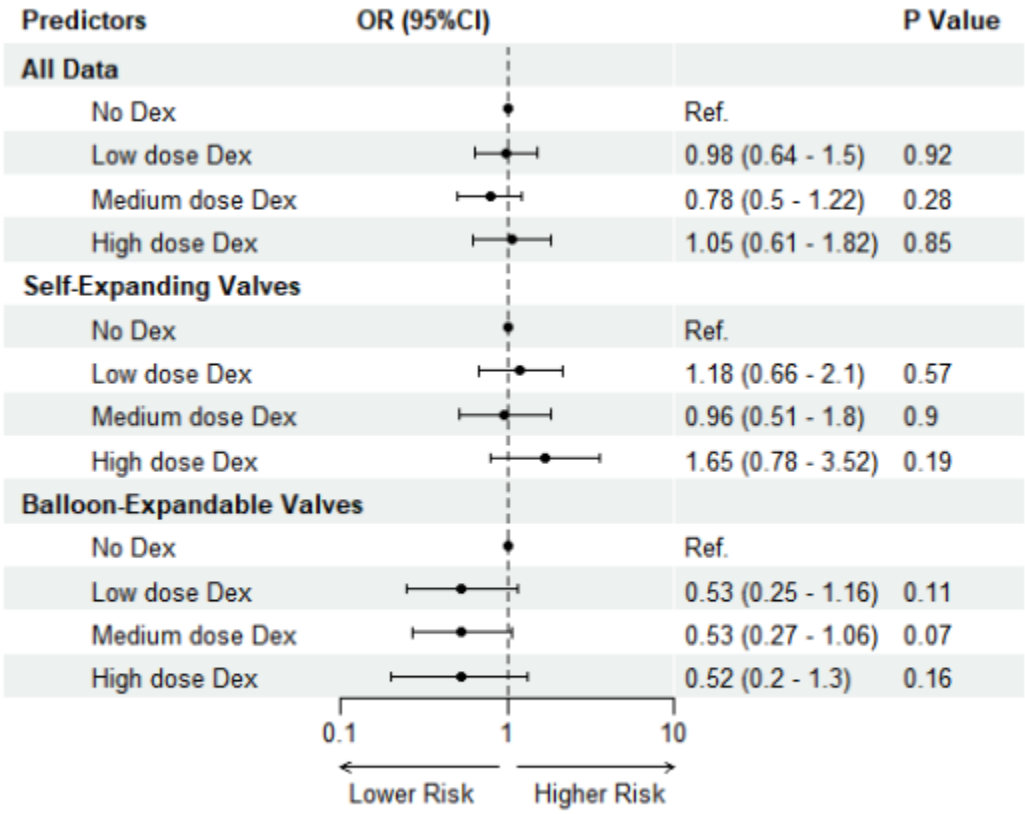


Figure 1. Odds Ratio of Pacemaker Implantation by DEX Dose Levels (No DEX is a reference group).

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SP 1 - Kiosk 2- Poster 2

Gabapentin for post operative analgesia in cardiac surgery: a systematic review and meta-analysis of randomized controlled trials

Presenting Author*: Fani Nhuch, MD*

Introduction: Opioids remain the cornerstone for managing moderate-to-severe postoperative pain after cardiac surgery, but their use is frequently associated with adverse effects such as nausea, vomiting, and respiratory depression. Gabapentin has emerged as a component of multimodal analgesia aimed at reducing opioid requirements and related complications. However, previous meta-analyses in cardiac surgery have been limited by small sample sizes and significant heterogeneity.This systematic review and meta-analysis aim to update the evidence on the efficacy and safety of gabapentin for postoperative analgesia in adults undergoing cardiac surgery.

Methods: We conducted a systematic search of PubMed, Embase, and the Cochrane up to October 01, 2025, to identify randomized controlled trials (RCTs) comparing perioperative gabapentin with placebo in adult patients undergoing any cardiac surgical procedure via sternotomy. The primary outcome was acute postoperative pain at 24 hours. Secondary outcomes included pain at other time points, opioid consumption, chronic pain, and adverse events (nausea, vomiting, sedation, delirium). Continuous and dichotomous outcomes were pooled using mean differences and risk ratios, respectively, with 95% confidence intervals and random-effects models. Risk of bias was assessed using the Cochrane RoB-2 tool.

Results: A total of six RCTs, totaling 753 patients, were included in the final analysis. (Figure 1). The mean age of patients ranged from 50 to 69.9 years, and 81.7% were male. (Table 1) For the primary outcome, gabapentin did not show a statistically significant difference compared to placebo in rest pain (MD -0.34; 95% CI -0.71 to 0.04; p = 0.079) or in cough-elicited pain at 24 hours (MD -0.69; 95% CI -1.38 to 0.00; p = 0.051). (Figure 2,3) Gabapentin did, however, demonstrate a statistically significant reduction in cough-elicited pain at 6 hours (MD -1.24; 95% CI -2.45 to -0.03; p = 0.044). (Figure 4) No statistically significant differences were found between the groups for other time-points or for adverse events, including somnolence, nausea, vomiting, and occurrence of atrial fibrillation.

Conclusion: Despite a statistically significant cough-elicited pain reduction at 6 hours, the arguably small clinical impact and lack of overall pain reduction at all other time-points indicates that gabapentin does not significantly reduce acute postoperative pain following cardiac surgery when compared to placebo. These findings suggest that the routine use of gabapentin as a primary component of multimodal analgesia for acute pain after cardiac surgery may not be warranted.

Keywords: Cardiac Surgery; Gabapentin; Meta-analysis; Pain Management; Postoperative Pain.

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Figure 1:

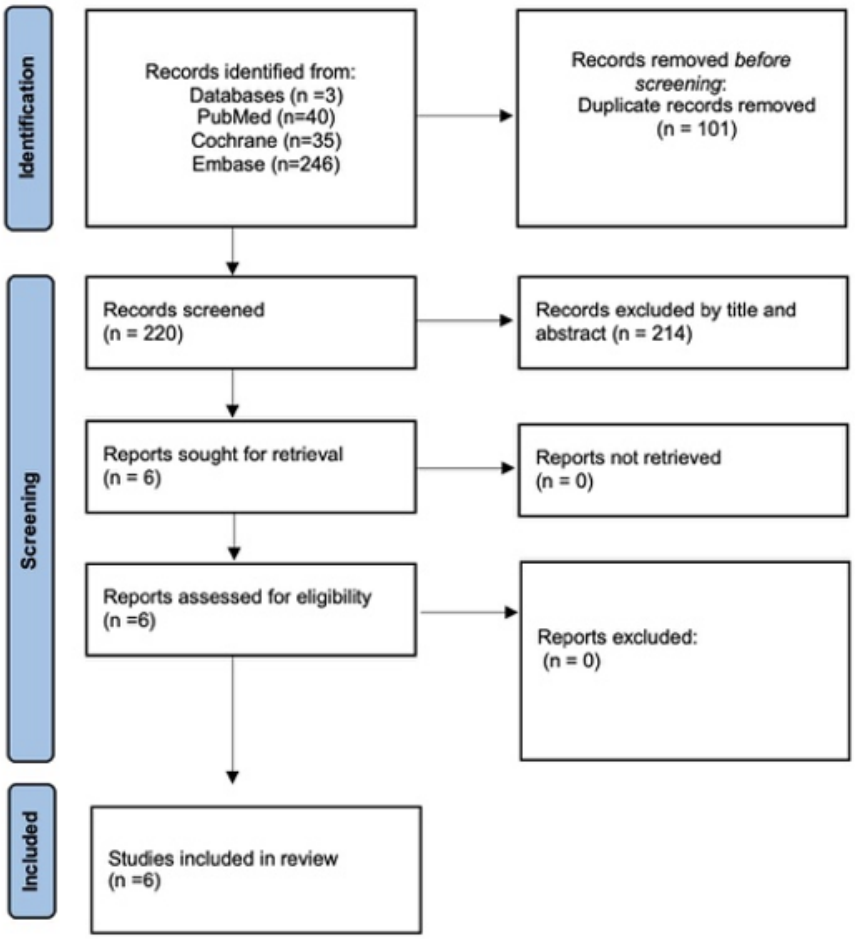


Table 1 Baseline characteristics

Author, year	Intervention	n	Age (years)	Male (%)	Diabetes (%)	Post operative pain assessment
Baos et al., 2025	Gabapentin (600mg pre-operatively, 300mg twice daily for POD one and two)	252	68.9 ± 2.31	78.4	18.1	NRS
	Placebo	249	69.9 ± 2.49	76.3	18.1	NRS
Hanouraet al., 2018	Gabapentin (600mg pre-operatively)	19	61.7 ± 7.6	89.5	63.2	CPOT, NRS
	Placebo	19	61 ± 7.1	98.5	63.2	CPOT, NRS
Soltanzadeh et al., 2011	Gabapentin (800 mg pre-operatively, 400mg two hours after extubation)	30	58.2 ± 8.3	100	10	NRS
	Placebo	30	55.2 ± 8.1	100	13	NRS
Ucak et al., 2011	Gabapentin (1200 mg pre-operatively,1200mg POD one and two)	20	57.4± 11.4	55	6	VAS
	Placebo	20	62.8 ± 10.9	65	5	VAS
Menda et al., 2010	Gabapentin (600mg pre-operatively)	30	52±6	100	23	VRS
	Placebo	30	50±8	100	17	VRS
Rapchuk et al., 2010	Gabapentin (1200 mg pre-operatively, 600mg twice daily for POD one and two)	27	61.8 ± 8.7	81.5	11	VAS
	Placebo	27	58.6 ± 11.1	96.3	3	VAS

Age: mean, standard deviation; POD: Postoperative day; NRS: numeric rate scale; CPOT: critical care pain observational tool; VAS: visual analog score; VRS: verbal rating scale

Figure 2: Pain 24 hours at with movement:

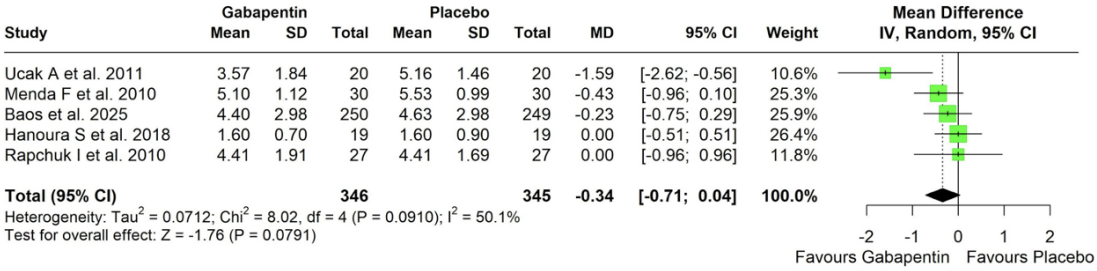


Figure 3: Pain 24 hours at rest:

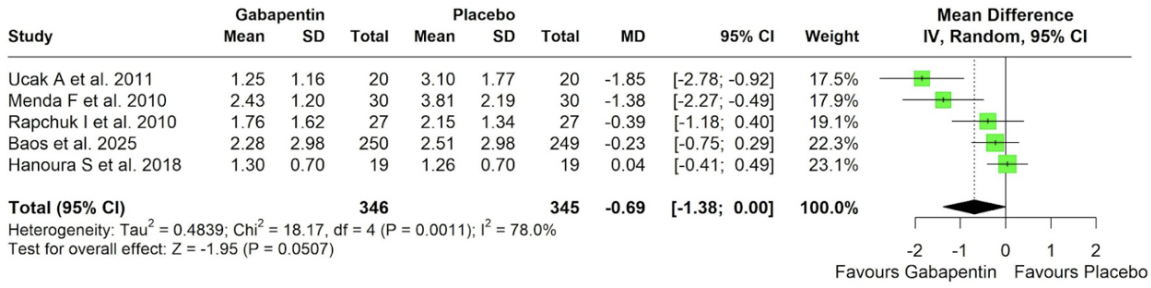
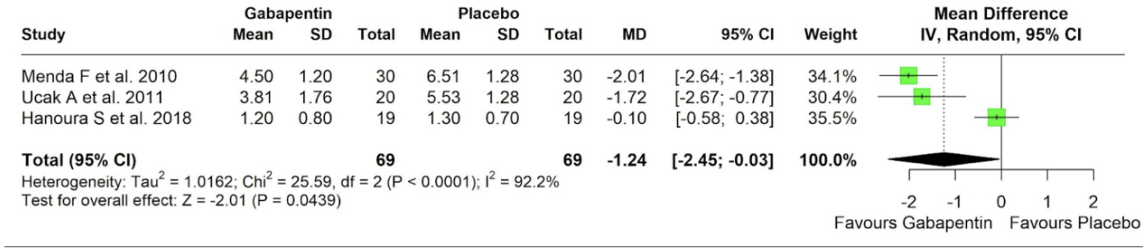


Figure 4: Pain 6 hours with movement:



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SP 1 - Kiosk 2- Poster 3

FIBRINOGEN LEVELS AS A PREDICTOR OF BLEEDING AFTER ADULT CARDIAC SURGERIES – A CLINICAL PROSPECTIVE OBSERVATIONAL STUDY

Presenting Author: *Kanagarajan Natarajan, MD,PDCC*

Introduction: Bleeding after cardiac surgeries is a major complication and an important determinant of morbidity and mortality. Serum fibrinogen levels seem to be an important hemostatic factor, but the relationship between the perioperative fibrinogen levels and the risk of bleeding post operatively have not been elucidated. We aimed at finding the incidence of

hypofibrinogenemia in adult patients undergoing cardiac surgeries and the correlation between preoperative and postoperative fibrinogen levels and post operative chest drain.

Materials And Methods: After institute ethical committee clearance and informed consent 167 adult patients undergoing on pump cardiac surgeries were included in the study. Patients with liver dysfunction, preoperative platelet count less 1 lakh/dl, INR > 1.5 were excluded from the study. Anti platelets and anticoagulants were stopped prior to the surgery according to standard guidelines. Baseline demographic data, preoperative serum creatinine, CPB duration, preoperative hematocrit, nadir hematocrit and temperature, preoperative baseline fibrinogen levels and platelet count, post CPB fibrinogen levels and platelet count prior to protamine administration, and post operative chest drain in first 48 hours were recorded. All patients underwent standardized anesthetic and surgical techniques as per institute protocol.

Statistical Analysis: Quantitative data are expressed as mean ± SD. Pearson's correlation test was used for analysis of continuous variables. Correlations between the measured laboratory indices and clinical and demographic data were performed using the t test. A p value < 0.05 is considered significant.

Results: 0.031 Of the 167 patients 122 were males (73.05%) and 45 were females (26.95%). Mean age was 56.59 ± 13.4. About 15 patients (9.0%) had preoperative baseline fibrinogen less than 150mg% and 40 patients (24%) had low post CPB fibrinogen levels. There is a weak negative correlation between preoperative fibrinogen levels (r = -0.165, p -0.033) , and a weak negative correlation between preoperative fibrinogen levels and post operative product usage(r = -0.197, p – 0.011) both of which are statistically significant. The chest drain in first 48 hrs was 262.9 ± 225.1ml (range – 20ml to 2070 ml). Multivariate regression analysis showed preoperative fibrinogen [coefficient – (-0.719), p – 0.05] as a independent predictor of post operative chest drain. (R= 0.176, R2 = 0.031) with a weak correlation.

Conclusion: We conclude that preoperative fibrinogen levels may be a predictor of post operative bleeding in cardiac surgeries. Fibrinogen levels may be an useful screening tool to identify individuals at added risk for bleeding. Weaker association warrants larger sample size to elucidate the role of fibrinogen in a detailed fashion.

References

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SP 1 - Kiosk 2- Poster 4

Comparison of Six Myocardial Infarction Criteria for Predicting Coronary Bypass Surgery Mortality

Presenting Author: *Wanwoo Seo, MD*

Introduction*:* Various definitions of perioperative myocardial infarction after exist, yet their relative prognostic utility remains unclear.

Objective: To compare the prognostic value of six different perioperative myocardial infarction definitions in predicting operative and 1-year all-cause mortality after coronary artery bypass grafting, and determine the criteria with the greatest incremental predictive value.

Design: Observational cohort study.

Setting: At a tertiary center in Korea between January 2006 and December 2019.

Participants: 3,211 patients undergoing elective coronary artery bypass grafting.

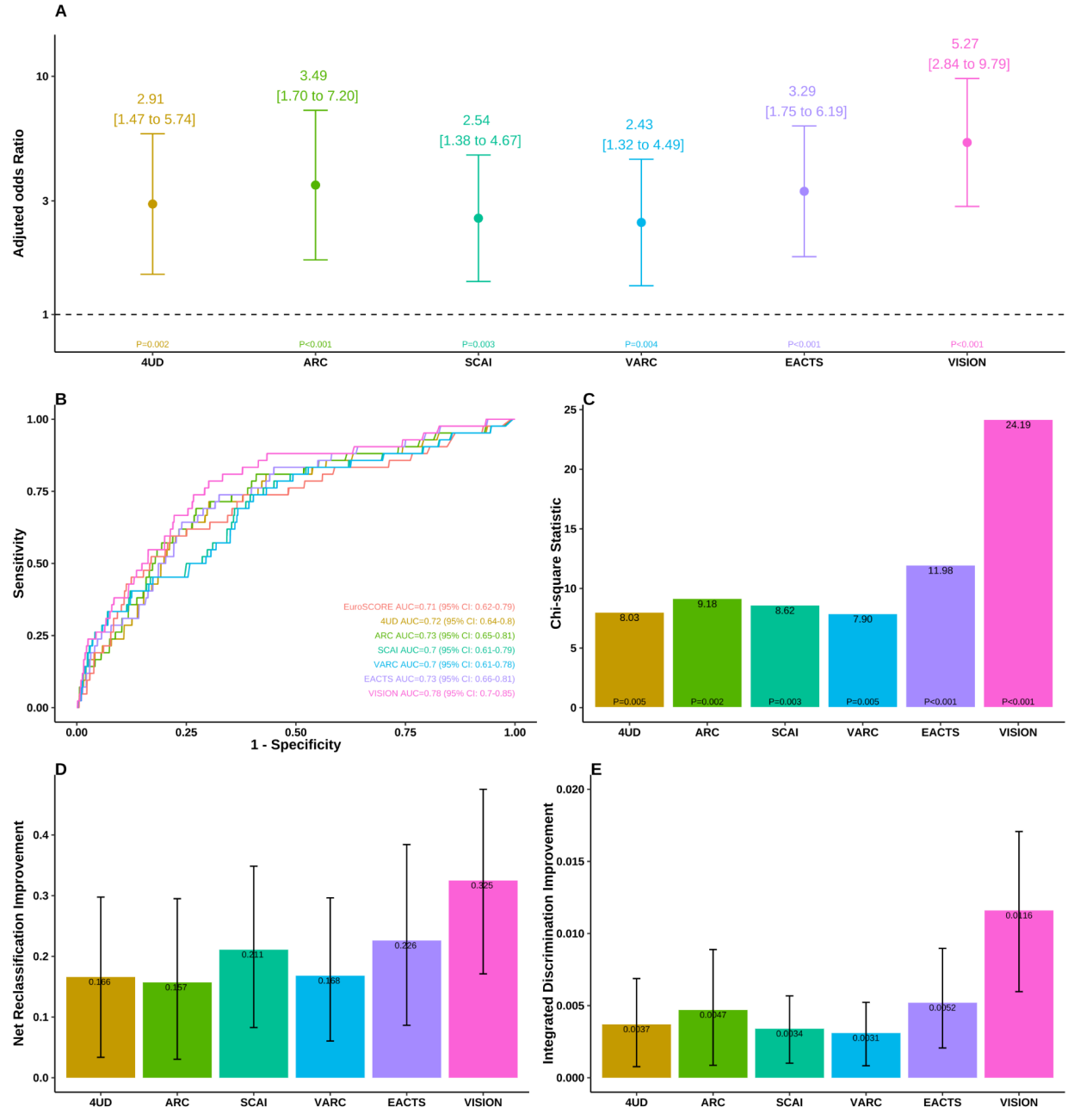
Exposures: Perioperative myocardial infarction was classified using the following six definitions: (i) the Fourth Universal Definition of Myocardial Infarction (4UD), (ii) Academic Research Consortium (ARC), (iii) Society for Cardiovascular Angiography and Interventions (SCAI), (iv) Valve Academic Research Consortium (VARC), (v) European Association of Cardio-Thoracic Surgery (EACTS), and (vi) the myocardial injury criteria proposed by the Vascular Events in Surgery Patients Cohort Evaluation (VISION) Cardiac Surgery study.

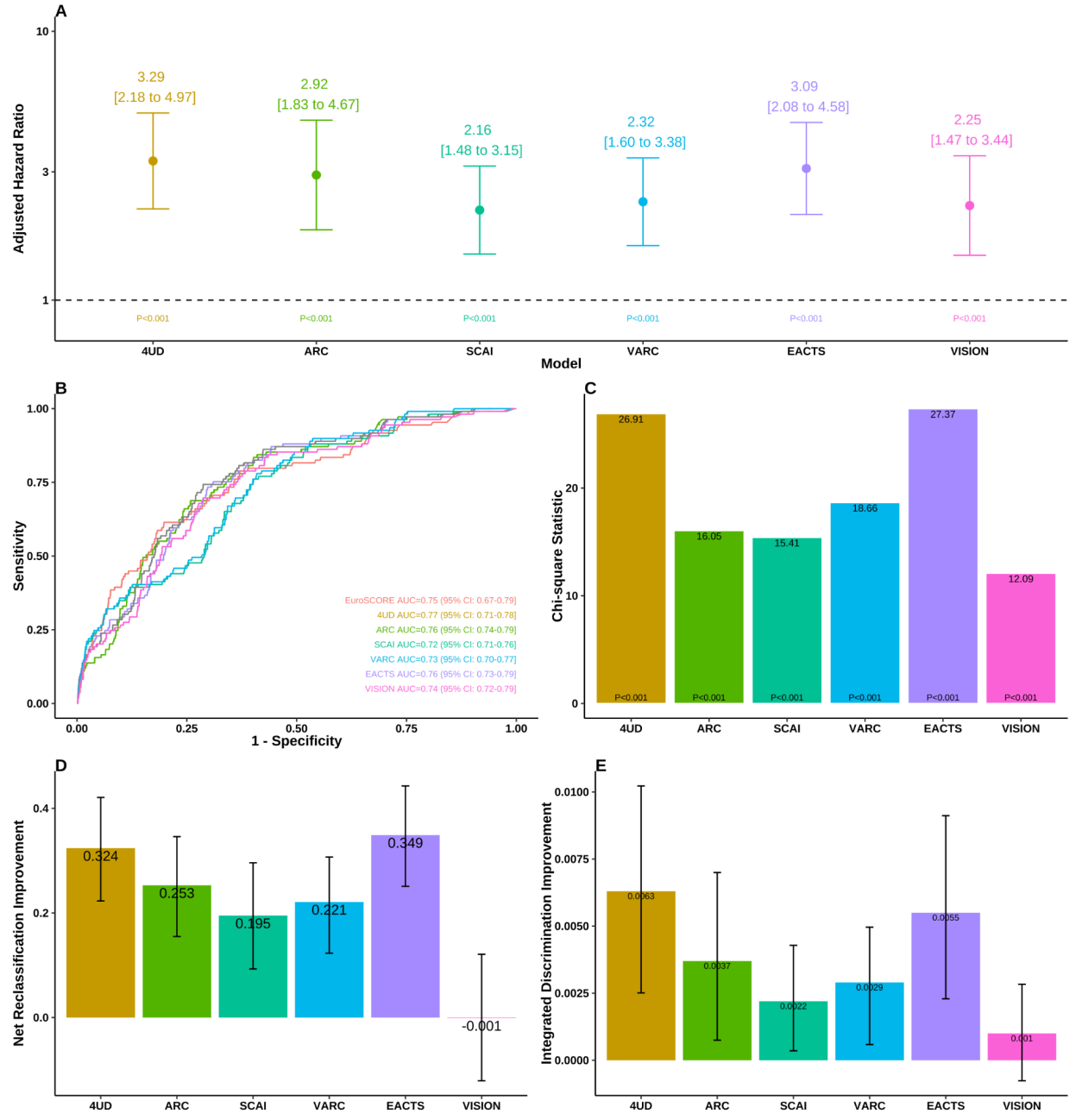
Main Outcomes and Measures: The primary endpoints were operative mortality (within 30 days or prior to discharge) and 1-year all-cause mortality. Predictive performance was assessed using area under the receiver operating characteristic curve (AUC), likelihood ratio tests (LRT), net reclassification improvement (NRI), and integrated discrimination improvement (IDI).

Results: Operative and 1-year mortality was 1.3% and 3.4%, respectively. For operative mortality, the VISION criteria showed the strongest association (odds ratio [OR] 5.27; 95% confidence interval [CI] 2.84–9.79) and had the highest incremental values (AUC 0.78; LRT χ^2 = 24.19; NRI 0.325; IDI 0.0116). For 1-year mortality, the 4UD and EACTS definitions had the strongest associations (4UD: HR 3.29, 95% CI 2.18–4.97; EACTS: HR 3.09, 95% CI 2.08–4.58) and superior incremental predictive values (4UD: AUC 0.77, LRT χ^2 = 26.91; NRI 0.324; IDI 0.0063; EACTS: AUC 0.76, LRT χ^2 = 27.37; NRI 0.349; IDI 0.0055).

Conclusions and Relevance: Among various definitions for perioperative myocardial infarction following coronary artery bypass grafting, the VISION myocardial injury criteria showed the greatest incremental values in predicting operative mortality, whereas for 1-year all-cause mortality, the 4UD and EACTS definitions are the most effective predictors.

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4UD AUC=0.72 (95% CI: 0.64-0.8)
ARC AUC=0.73 (95% CI: 0.65-0.81)
SCAI AUC=0.7 (95% CI: 0.61-0.79)
VARC AUC=0.7 (95% CI: 0.61-0.78)
EACTS AUC=0.73 (95% CI: 0.66-0.81)
VISION AUC=0.78 (95% CI: 0.7-0.85)
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SP 1 - Kiosk 2- Poster 5

Cardiopulmonary Bypass Flows Relative to Baseline and Post Operative Kidney Injury
Presenting Author: *Belle Benanzea-Fontem, MD*

Introduction: Cardiopulmonary bypass (CPB) has been associated with acute kidney injury (AKI) with varying incidence of 1-40%1. Unfortunately, mild degrees of AKI after cardiac surgery can contribute to increased mortality and morbidity2. The mechanism of injury is thought to be closely related to reduced renal blood flow and oxygen availability3. Some have shown that increased CPB flows can be renally protective in patients undergoing cardiac surgery4,5. Other studies have demonstrated that differences in baseline blood pressure and CPB mean arterial pressure may in fact increase the risk of AKI6. Despite these trends there have been no studies comparing the risk of AKI in patients when there are differences in pre-operative baseline cardiac index and the index while on CPB.

Methods: We conducted a single-center retrospective cohort study of patients who underwent cardiac surgery requiring CPB between 2021 and 2025. Data were extracted from electronic medical records. Patients were separated into two groups: those with CPB index 20% below baseline index and all other remaining patients. Baseline index was extracted from the intraoperative record as the median index between the time of placement of a pulmonary artery catheter and the initiation of CPB. CPB index was determined to be the median index between the initiation and separation of CPB as indicated on the intraoperative record. The primary outcome of AKI incidence was assessed using the AKIN criteria using generalized linear models. Secondary outcomes included reintubation, lactate following separation from CPB, blood product

transfusion, 30 day and 6-month mortality, and length of ICU stay. Univariate and multivariate analyses were performed on both primary and secondary outcomes using known covariates associated with AKI.

Results: Patient demographics of our study can be found in Table 1. The risk of AKI, first lactate following separation from CPB, reintubation, ICU LOS, and RBC transfusion were significantly higher in the group of patients with CPB index less than 20% of baseline in the univariate model (Table 2). These outcomes remained significant in the multivariate model except for lactate and RBC transfusion. There was no significant difference in 30 day or 6 month mortality.

Discussion: Our study suggests that large deviations between a patient's baseline cardiac index and the cardiac index chosen during CPB may increase patient morbidity by raising the risk of post-operative AKI in addition to other post-operative performance metrics. Further studies with more patients, from multiple centers, and using randomization are needed to elevate these correlations to true causations.

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	Pump flow 20% Below Baseline (n=117)	Remainder of Patients (n=734)	P-Value
Average Age	56 ±1.3	64 ±0.5	**<0.001**
Sex (Males)	83 (70%)	523 (71.2%)	
Mean Body Mass Index	27.4± 0.47	27.12±0.18	0.53
Mean Pre-operative Creatinine	1.01±0.04	0.96±0.01	0.21
Mean Pre-Operative Hemoglobin	12.17±0.23	12.96±0.07	**<0.001**
Mean Bypass Time (Minutes)	156.79 ±5.43	139.47 ±1.98	**<0.001**
Mean Lowest Temp in Pump (∘C)	26.6 ±0.66	24.06 ±0.26	**<0.001**
Mean Ejection Fraction	54.18±1.49	55.61±0.5	0.31

Table 1. Baseline characteristics of patients within both groups

	Relative Risk (95% Confidence interval)	Mean ±SD in Pump flow 20% Below Baseline	Mean ±SD in Remaining Patients	P-value Univariate	P-value Multivariate
AKI	1.46 (1.15, 1,85)			**0.003**	**0.021**
Reintubation	5.01 (2.88, 8.73)			**<0.001**	**<0.001**
30 day Mortality	0			0.42	0.56
6 month Mortality	2.09 (0.21, 19.93)			0.51	0.94
First Lactate Post Pump		29.07 ± 1.67	24.14 ±0.52	**0.005**	0.88
Length of Stay (Hours)		412.13 ±57.47	188.17 ±10.46	**<0.001**	**<0.001**
Transfusions					
Red Blood Cells (ml)		385.03±51.82	249.93±16.5	**0.004**	0.31
Plasma (ml)		133.8±24.53	102.95±9.46	0.23	0.52
Platelets (ml)		224.1±31.41	203.35±12.52	0.54	0.92
Cryofibrinogen (ml)		35.43±6.57	32.44±2.53	0.66	0.99

Table 2. Primary and secondary outcomes with associated p-values.

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SP 1 - Kiosk 2- Poster 6
One Valve, Four Cusps: Incidental Detection of a Rare Aortic Anatomy
Presenting Author: *Austin Le, MD*

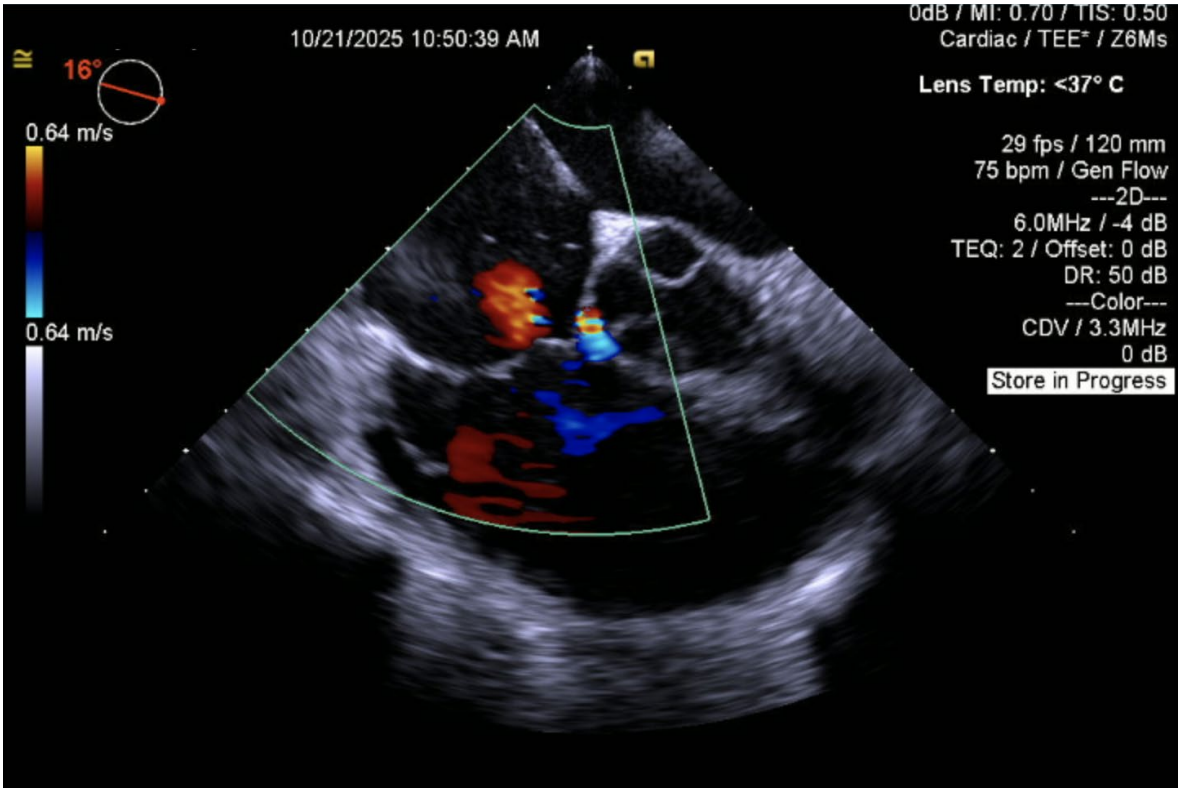
Background / Introduction: Quadricuspid aortic valve is an uncommon congenital malformation with a prevalence estimated at less than 0.05%. Its presentation ranges from asymptomatic to progressive aortic regurgitation, and it is increasingly recognized through advanced echocardiographic imaging. For cardiovascular anesthesiologists, transesophageal echocardiography plays a key role in detecting structural anomalies that influence both perioperative decision-making and long-term care.

Case Report A 27-year-old female with no significant past medical history presented with dyspnea on exertion and palpitations. Physical examination revealed a systolic murmur at the right second intercostal space. Transesophageal echocardiography showed normal left ventricular systolic function and a normal ejection fraction. A restrictive ventricular septal defect with left-to-right shunting and mild tricuspid regurgitation were noted. Detailed assessment of the aortic valve revealed four similarly sized cusps consistent with a Hurwitz and Roberts type A quadricuspid aortic valve. Leaflet motion was preserved, and only trivial aortic regurgitation was detected. No associated dilation of the aortic root was observed. Her symptoms were attributed to underlying congenital structural abnormalities, and she was referred for longitudinal cardiology follow-up.

Discussion: The Hurwitz and Roberts type A quadricuspid configuration, characterized by four equal cusps, is one of the more balanced morphologies and is typically associated with variable degrees of aortic regurgitation. Although this patient exhibited only trivial regurgitation, quadricuspid valves remain susceptible to progressive insufficiency, cusp fibrosis, and aortopathy over time. The presence of additional congenital lesions, including ventricular septal defect, has been reported in association with this anomaly. Comprehensive transesophageal echocardiography was essential in identifying the valve morphology, quantifying regurgitation, and evaluating for concomitant structural abnormalities. Early diagnosis is crucial as management depends on serial imaging and symptoms related to evolving valvular dysfunction or associated lesions. Conclusion This case emphasizes the importance of meticulous echocardiographic evaluation in recognizing rare congenital aortic valve variants. Identification of a type A quadricuspid aortic valve, even with trivial regurgitation, enables appropriate surveillance and informs long-term clinical management. Echocardiographic expertise within the perioperative environment allows anesthesiologists to contribute meaningfully to the detection and multidisciplinary care of structural heart disease.

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References

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SP 1 - Kiosk 2- Poster 7

Incidental VSD During Cardiac Surgery, to fix or not to fix?
Presenting Author: *Jasmine Tuteja, MD*

Background: Ventricular septal defects (VSD) are the most common congenital cardiac anomaly in children, and also one of the most common heart defects in adults. They can either be congenital or acquired, with the acquired form usually occurring after ischemic events or inflammatory conditions. The decision of whether or not to repair a VSD in adulthood is made based on various factors, such as presence of hemodynamic compromise or signs of left ventricular dysfunction. Another congenital anomaly found in many adults is a patent foramen ovale (PFO). Although there exists literature on how to proceed with incidentally found intraoperative PFOs, no literature exists on how to proceed with incidentally found VSDs. We look at a case in which a patient presented for a coronary artery bypass grafting (CABG) and was found to have an incidental membranous VSD intraoperatively on transesophageal echocardiography (TEE).

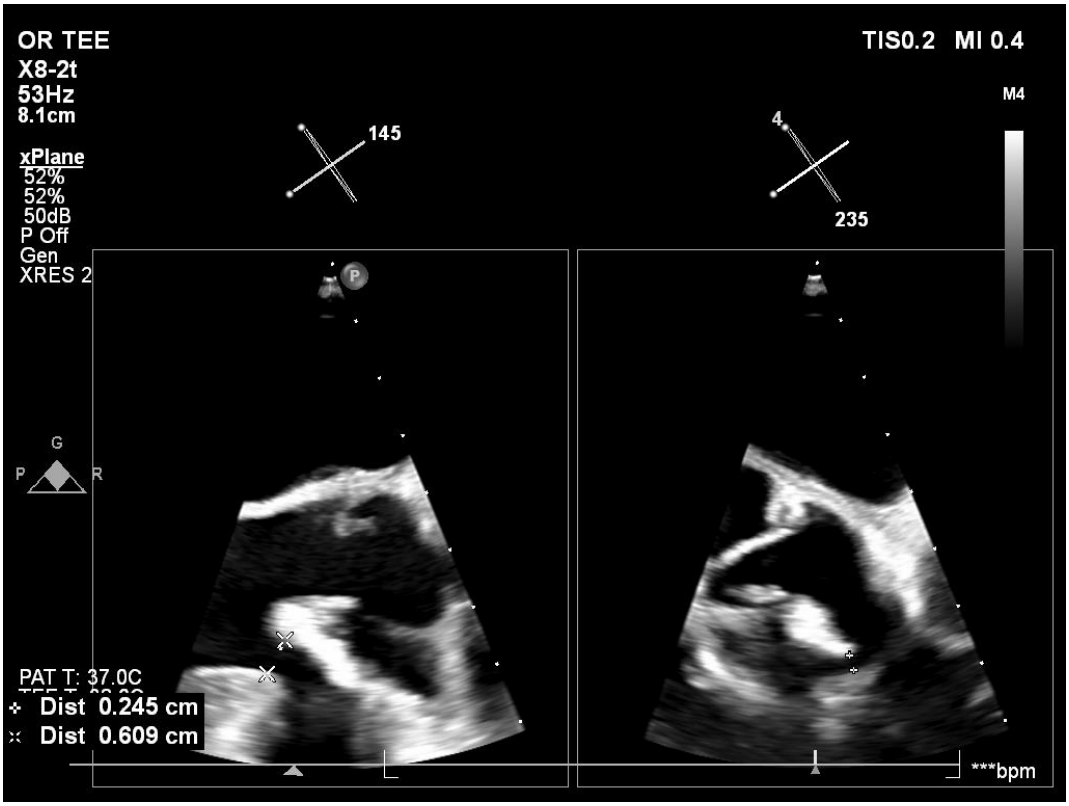
Case Report: A 66-year-old male with history of hypertension and hyperlipidemia presented for CABG due to multivessel coronary artery disease. Intraoperative TEE unexpectedly revealed a 2.5mm x 6.1mm membranous VSD with left-to-right shunting, dilated RV, and normal RV function. RVSP was 23 mmHg with an RA pressure of 5 mmHg, resulting in a peak VSD gradient of 59 mmHg. The incidental VSD posed a significant intraoperative management dilemma. The absence of a preoperative PA catheter and unexpected finding necessitated real-time decision-making. Without preoperative PA catheter data, real-time hemodynamic assessment by the surgical and anesthesia teams was essential. TEE findings, combined with direct intraoperative hemodynamic measurements showing RV pressures of 20/5 mmHg and PA pressures of 19/7/12 mmHg, were crucial for assessing the VSD's hemodynamic significance. Ultimately, due to the patient's hemodynamic stability and the dimensions of the defect being amendable to percutaneous repair, the decision was made to not repair the VSD during the current CABG operation.

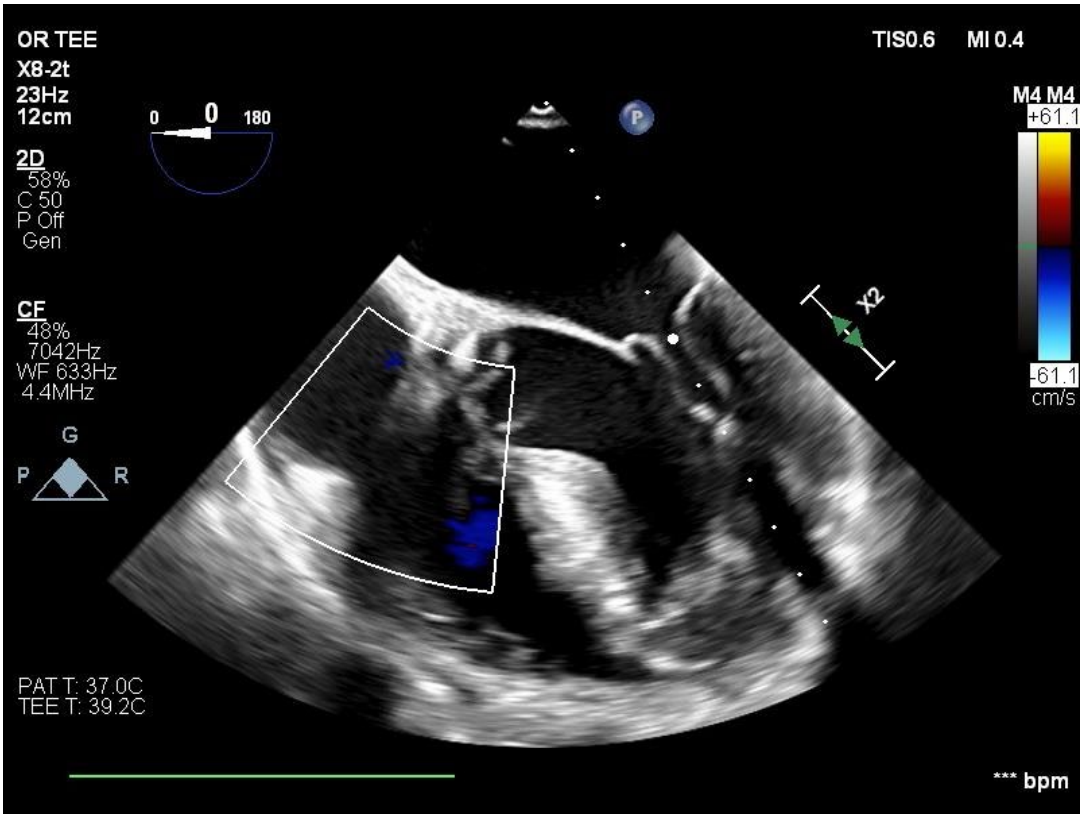
Discussion: This case highlights the critical role of intraoperative TEE in uncovering unexpected findings and guiding surgical decisions. Intraoperative TEE can not only identify surgical complications such as aortic dissection, tamponade or valvular trauma, but can also reveal incidental findings that may necessitate intervention depending on severity. In this particular case, the decision to defer VSD repair was based on the patient's hemodynamic stability and the procedural risks involved with the repair. Aortic regurgitation, left ventricular dilation, and pulmonary hypertension are usually indications for closure, of which this patient had none. Procedural risks of surgical VSD repair can include valvular incompetence, arrhythmias, and progression of pulmonary hypertension.

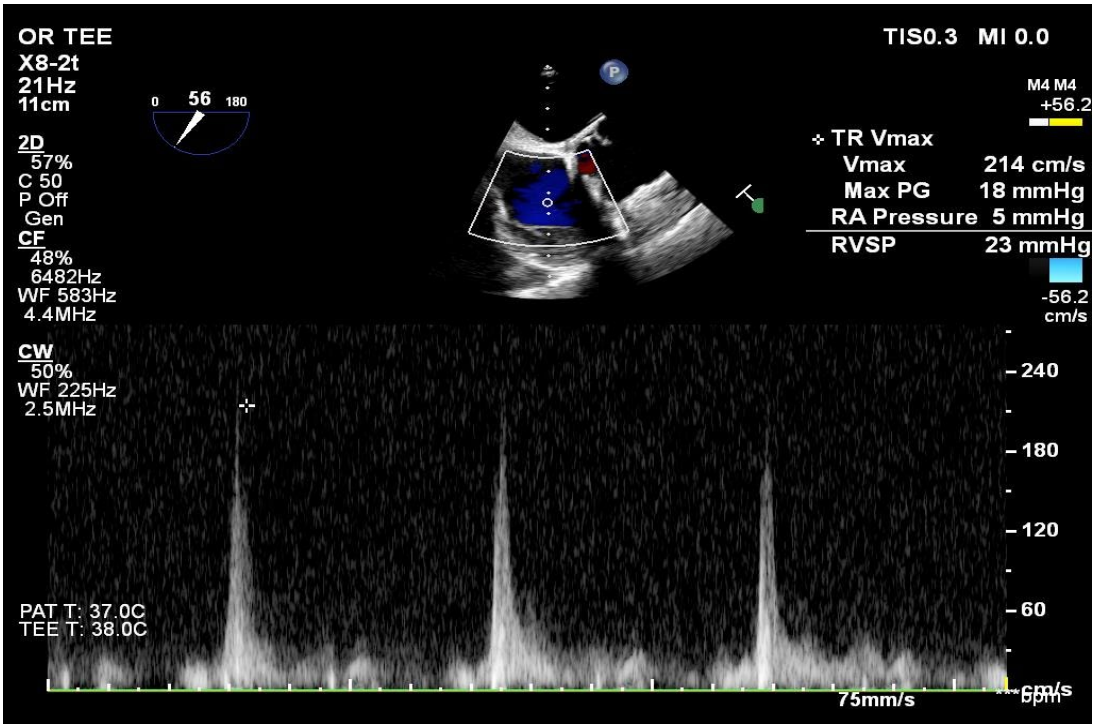
Conclusion: This case emphasizes the challenges of unexpected findings during surgery and the

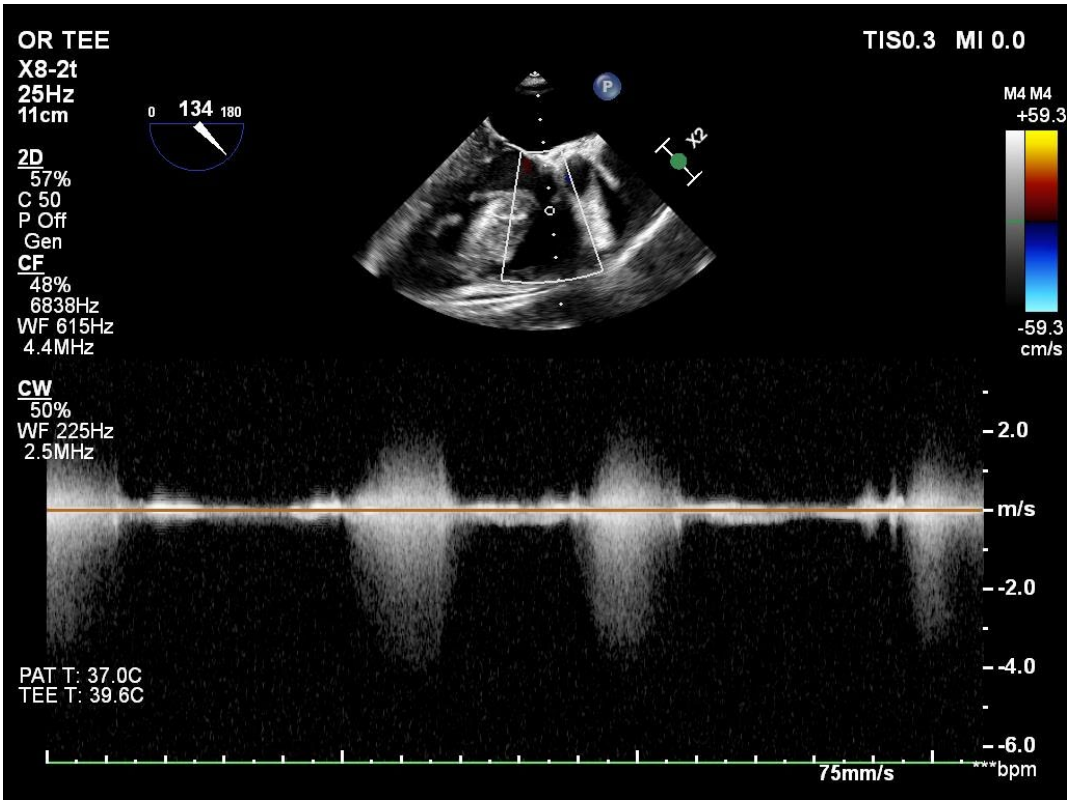
need for further research on managing incidentally discovered VSDs. Non-invasive options remain for future management.

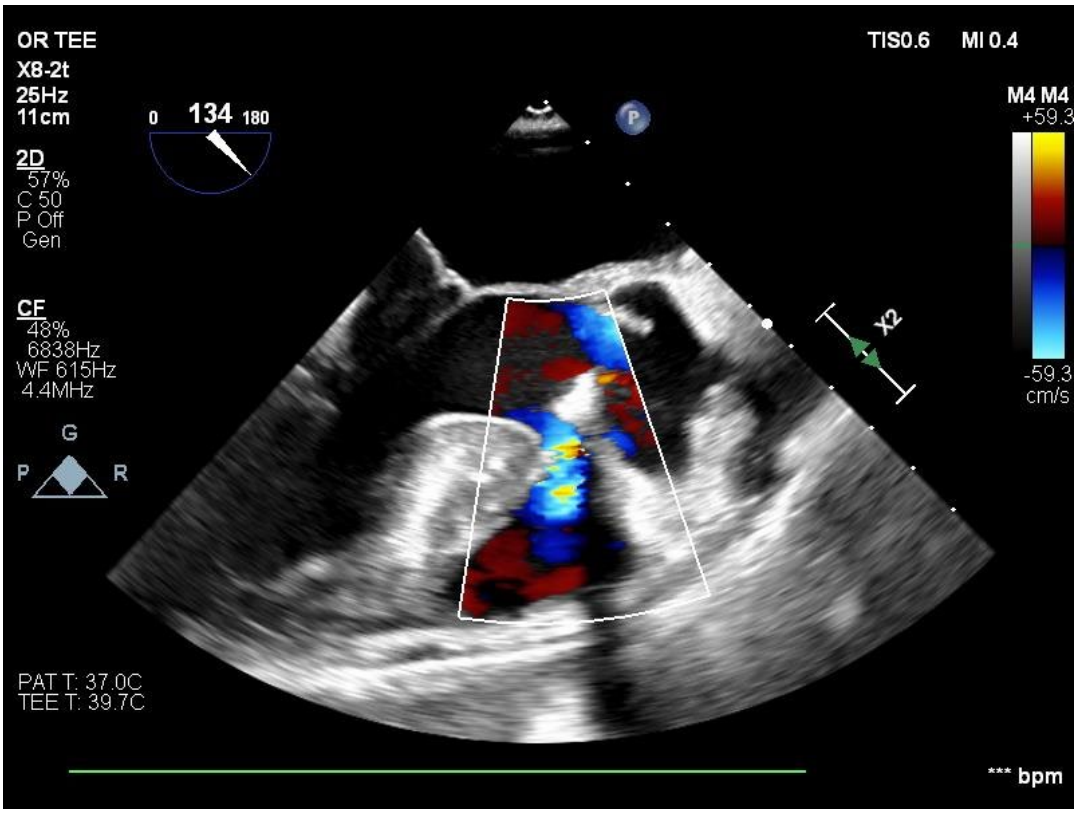
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SP 1 - Kiosk 3- Poster 1
Impact of Pectointercostal Fascial Plane Blocks on Outcomes Following Midline Sternotomy Cardiac Surgery
Presenting author: *Nienke Schouten, PhD*

Introduction: Midline sternotomy cardiac surgery is associated with significant postoperative pain, poor postoperative mobilisation, respiratory complications and prolonged intensive care unit (ICU) stay1. The most recent enhanced recovery after cardiac surgery recommendations advocates for the use of chest wall regional anaesthesia as part of an opioid sparing multimodal approach2.. Emerging evidence supports the use of bilateral pectointercostal fascial plane blocks (PIFBs) in midline sternotomy cardiac surgery to reduce postoperative pain, encourage early mobilisation and improve respiratory outcomes3. This quality improvement project (QIP) evaluates the role of PIFBs in reducing post-operative pain, total post-operative opioid consumption and time-to-extubation, as well as ICU and hospital stay in patients undergoing routine midline sternotomy cardiac surgery.

Methods:All ICU patients undergoing elective and urgent midline sternotomy cardiac surgery were identified by the ICU audit team. 60 (or is it 80) patients who did not receive a PIFB were initially assessed as the control group. This was followed by a period of training and then implementation of bilateral PIFBs. Patients receiving bilateral PIFBs were tracked using QR codes positioned in cardiac theatres. Data were extracted from electronic patient records and physical ICU charts. Respiratory complications were followed up until the point of discharge. Data were analysed using R.

Results: 184 patients (median age 67, 129 men) who underwent routine midline sternotomy

cardiac surgery were included. 104 of these patients received bilateral PIFBs following surgery. Time to extubation was significantly reduced in the PIFB group, median (IQR) 227.5 (170-305) minutes, compared to the control group, median (IQR) 280 (199.5 - 416.5) minutes (p = 0.0035, 95% CI for difference 18.0 - 87.0) (figure 1). Median pain scores on postoperative day one were significantly reduced in the PIFB group with a median (IQR) score of 1 (0.33-1.63) compared to 1.67 (0.83 – 2.92) (p = 0.0002, 95% CI 0.33 - 1.00). Postoperative NIV use was similar between group (X2 (1, N = 184) = 0.0005, p =0.9). Reintubation rates were 1.25% in the control group and 2.88% in the PIFB group (X2 (1, N = 184) = 0.57, p =0.45). Readmission rates were unchanged by PIFBs (X2 (1, N = 184) = 0.26, p =0.61). No difference between groups was found in time to sit out of bed (p = 0.18, 95% CI -5.00 – 1.00), hospital stay (p = 0.14, 95% CI -1.00 – 0.00), ICU stay (p = 0.44, 95% CI -1.00 – 3.00). No adverse events were reported in patients receiving PIFBs. No complications related to PIFBs were found.

Discussion: Regional anaesthetic blocks are sparingly used in routine midline sternotomy cardiac surgery, but have shown promising results in recent studies. In this QIP, PIFBs significantly reduced both time-to-extubation and postoperative pain following routine surgery. They have the added benefit of very low complication rate, easy to learn and quick to administer. The added benefit makes it appealing as an added element to the patients surgical journey and as part of routine use in any enhanced recovery after cardiac surgery programme.

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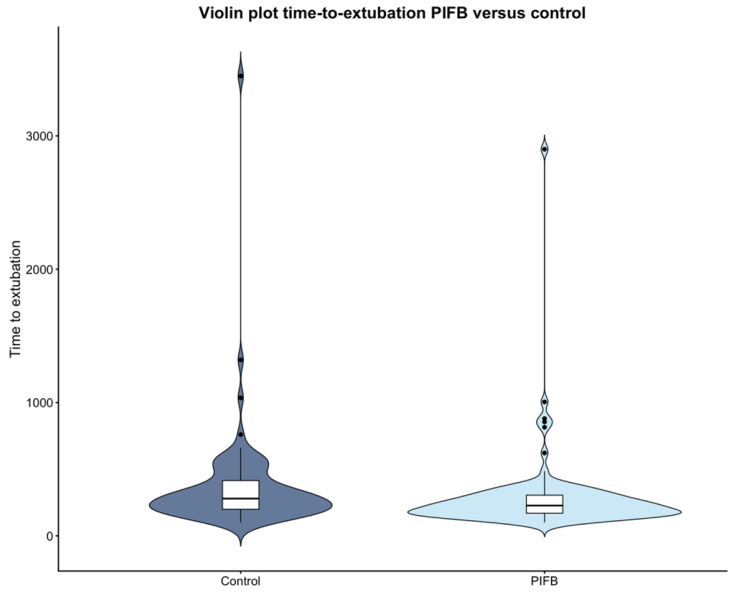


Figure 1 – violin plot visualising time-to-extubation (minutes) in PIFB versus control group (outliers included)

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SP 1 - Kiosk 3- Poster 2

Comparison of pulmonary artery catheter-derived variables and mean arterial blood pressure on the association with acute kidney injury after off-pump coronary artery bypass grafting

Presenting Author: *Jaeyeon Chung,MD*

Background: The intraoperative use of pulmonary artery catheter (PAC) in cardiac surgery remains controversial, and current guidelines recommend selective use in high-risk patients. However, recent evidence suggests that PAC use in off-pump coronary artery bypass grafting (OPCAB) might reduce mortality. Given growing interest in intraoperative hypotension as a modifiable risk factor, we compared the PAC-derived variables, such as cardiac index (CI) and mixed venous oxygen saturation (SvO2), with mean blood pressure (MBP), on the association with acute kidney injury (AKI) after OPCAB.

Methods: This single-center retrospective observational study included adult patients who underwent OPCAB between January 2011 and May 2025. The primary outcome was development of AKI within seven days after surgery, which was defined by the serum creatinine criteria of Kidney Disease: Improving Global Outcomes. Area under the curve (AUC) and duration below CI (2.0, 1.8, 1.6, 1.4, and 1.2 L/min/m2), SvO2 (70, 65, 60, 55, and 50%), and MBP (70, 65, 60, 55, and 50 mmHg) thresholds during the intraoperative period were calculated. Postoperative AKI associations were compared using multivariable logistic regression analysis. Additionally, to determine the clinically significant thresholds for each hemodynamic variable, we evaluated the association of the interval-based exposure, defined as the duration spent within each CI, SvO_2, and MBP interval, using multivariable logistic regression analysis. The risk of cumulative all-cause mortality was compared using Kaplan-Meier curves.

Results: Among the 1,517 patients included in the study, 283 (18.7%) patients developed AKI. AUC for CI thresholds at or below 1.8 L/min/m2 and duration thresholds at or below 1.6 L/min/m2 were significantly associated with postoperative AKI (Table 1). SvO_2 showed significance only below 65%. Conversely, MBP was not associated with AKI at any cutoffs. In the interval-based exposure analysis, only the duration spent with a CI < 1.4 L/min/m^2 was consistently associated with postoperative AKI, whereas MBP and SvO_2 showed no threshold-dependent pattern (Figure 1). Kaplan–Meier curves stratified by the duration spent with a CI < 1.4 L/min/m^2 (no exposure, < 1 hr, ≥1 hr) showed significant inter-group differences (All pairwise log-rank P < 0.05, Figure 2), with shorter duration of low cardiac index associated with more favorable long-term survival.

Conclusions: A decrease in cardiac index during OPCAB is associated with increased postoperative acute kidney injury, where as MBP and SvO2 showed no significant

associations. The association between CI and postoperative AKI seems particularly prominent when CI below 1.4 L/min/m2.

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SP 1 - Kiosk 3- Poster 3

Effect of Local Anesthetic Chest Tube Injection on Pleuritic Pain After Cardiac Surgery.

Presenting Author: *Rosalie Roy*

Background: Pleuritic pain after cardiac surgery is common and often severe, limiting respiration and early mobilization. Chest tube–directed local anesthetic (LA) injection is widely believed to be effective in practice, yet high-quality evidence in cardiac surgery is scarce 1. We evaluated the analgesic efficacy of bupivacaine injected through pleural drains in patients experiencing postoperative pleuritic pain.

Methods: In this prospective double-blinded randomized controlled trial, adult cardiac surgery patients reporting pleuritic pain (Numerical Pain Score [NPS] ≥3) received either blinded bupivacaine 0.5% (10 mL) or blinded placebo (NS 0,9%; 10 ml) through their pleural drain. Pain at rest and during mobilization was assessed at baseline and every 15 minutes post-injection. The primary outcome was adequate pain relief (≥25% NPS reduction) during mobilisation at 30 minutes after injection. Secondary outcomes included absolute ΔNPS, time to maximal analgesia, and duration of effect. Patients without ≥25% reduction in NPS at 30 minutes were withdrawn from the blinded phase and—per protocol and for ethical reasons—received open-label rescue bupivacaine. The sample size was determined to detect a 25% absolute difference in NPS between

groups with 80% power and a two-sided alpha of 0.05 based on the results of a previous local observational study. Group comparisons used t-tests or Mann–Whitney tests for continuous data and chi-square or Fisher exact tests for categorical data.

Results: Fifty patients completed the protocol (25 per group). Adequate relief occurred more frequently with bupivacaine than placebo (69% vs 44%, p=0.0304). Bupivacaine produced greater reductions in NPS both at rest (Δ2.68 vs Δ1.72, p=0.0534) and during mobilization (Δ3.57 vs Δ1.70, p< 0.001). Time to maximal effect was significantly shorter with bupivacaine (23.7 vs 43.8 minutes, p< 0.001). Among non-responders who received open-label rescue bupivacaine, analgesic response was highest (adequate relief 90%).

Conclusion: Chest tube bupivacaine injection provides rapid and clinically meaningful relief of pleuritic pain after cardiac surgery. The pain relief associated with placebo injection and augmented response to open-label rescue suggests that patient expectancy and contextual factors may potentiate analgesic efficacy, an important consideration for multimodal postoperative pain management 2. These findings support the integration of chest-tube bupivacaine injection into multimodal postoperative analgesia strategies in cardiac surgery.

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Effect of Local Anesthetic Chest Tube Injection on Pleuritic Pain After Cardiac Surgery.

Injection pain outcomes

Variable	BUPI (25 patients)	NS (25 patients)	p
Injections (nb)	39	34	
Adequate pain relief * (nb)	27	15	0.0304
Adequate pain relief * (%)	69	44	0.0304
Time for maximal pain reduction (min)	23,7	43,8	0.0005
Total time of relief (min)	31,7	32,1	0.9409
Decrease NPS at rest	2,68	1,72	0.0534
Decrease NPS – repos (%)	45,4	34,0	0.1942
Decrease NPS - mobilisation	3,57	1,70	0.0009
Decrease NPS – effort (%)	46,9	29,3	0.0156
Adequate pain relief RESCUE BUPI (%)	90	89	0.9274
Decrease NPS – rest / RESCUE BUPI	3,60	2,78	0.2497
Decrease NPS – rest (%) /RESCUE BUPI	53,0	50,7	0.8447
Decrease NPS – mobilisation / RESCUE BUPI	3,55	4,00	0.6087
Decrease NPS – effort (%) / RESCUE BUPI	54,7	48,4	0.5288

*NPS decrease ≥ 25%

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SP 1 - Kiosk 3- Poster 4
Association of Post-Cardiopulmonary Bypass Oxygen Delivery Index with Risk of Cardiac Surgery-Associated Acute Kidney Injury
Presenting Author: *Erik Roberts, MD, PhD*

Background: Acute kidney injury (AKI) after cardiac surgery remains a frequent complication. Low oxygen delivery index (DO2i) during cardiopulmonary bypass (i.e., < 270 to 300 mL/min/m2) has been associated with increased AKI risk. For the non-bypass period, studies have focused on components of oxygen delivery (i.e., cardiac index, hemoglobin, and blood pressure) rather than the integrated DO2i. We hypothesized that optimization of the post-bypass DO2i above a threshold of 300 mL/min/m2 would correlate with reduced AKI risk.

Methods: We conducted a multicenter retrospective observational cohort study of 6749 adult cardiac surgeries involving cardiopulmonary bypass and cardiac output measurement across 7 hospitals from 2014 to 2022 using integrated data from the Multicenter Perioperative Outcome Group (MPOG) and the Society of Thoracic Surgeons (STS). The primary outcome was AKI following cardiac surgery, defined as a postoperative serum creatinine increase from baseline of ≥ 0.3 mg/dL within 48 hours or ≥ 1.5x within 7 days. The primary predictor was median post-bypass DO2i , estimated at the minute-level using the pulse pressure-based Liljestrand-Zander method for calculating cardiac output (calibrated to intermittent cardiac output measurements) with hemoglobin imputed from intermittent blood gases. The probability of AKI was predicted from logistic regression models using a natural cubic spline transformation of the median post-bypass DO2i. Chronic kidney disease (CKD) stage was examined as an additional predictor.

Results: Of the 6749 cases, 1757 (26%) developed a postoperative AKI. We identified an AKI risk inflection point at a median post-bypass DO2i of ~300 mL/min/m2. Above this threshold, probability of AKI plateaued at ~22%. Below this threshold, AKI risk increased linearly by ~2% per 10 mL/min/m2 decrease in DO2i . Patients with stage 3 CKD had >10% higher AKI risk across the DO2i range. While the DO2i inflection point was similar to those with normal renal function, only CKD3 patients demonstrated further risk reduction at DO2i levels >300 mL/min/m2.

Conclusion: Achieving a post-cardiopulmonary bypass DO2i above a threshold of ~300 mL/min/m2 is associated with a decreased risk of AKI. Patients with pre-existing CKD may

benefit from a higher oxygen delivery target. Future prospective studies should examine post-bypass and intensive care unit DO2i optimization explicitly.

Image uploads:

Figure 1: Probability of AKI vs. Median Post-Bypass DO2i

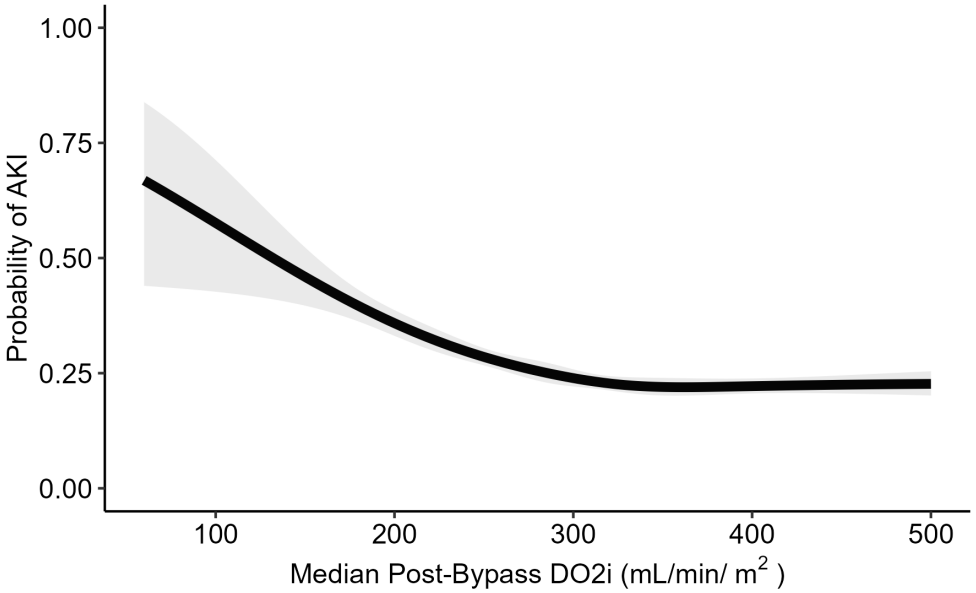
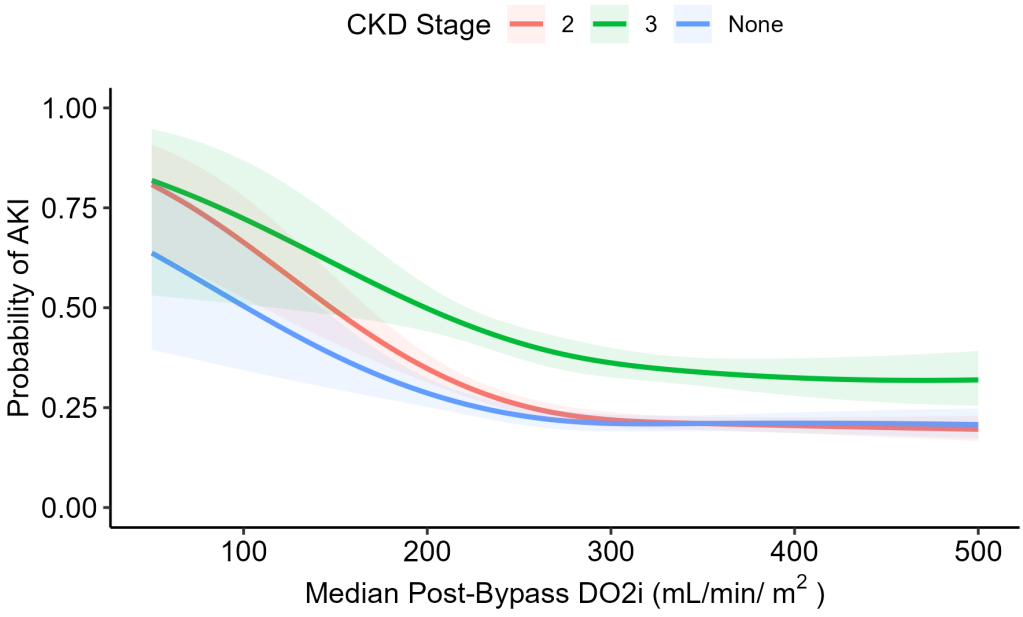


Figure 2: Probability of AKI vs. Median Post-Bypass DO2i (by CKD Stage)



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SP 1 - Kiosk 3- Poster 5
Seeing What Transesophageal Echocardiogram Couldn't: Using Epicardial Echocardiogram for Right Ventricular Foreign Body Removal
Presenting Author: *Katie An, MD*

Introduction: Transesophageal echocardiogram (TEE) has largely replaced the epicardial echocardiogram for intraoperative evaluation during adult cardiac surgeries. Epicardial echocardiograms do however remain useful with selective advantages over TEE. We describe a unique use of the epicardial echocardiogram to remove a foreign body from the right ventricle.

Case Report: A 48-year-old male with a history of hypertension and severe crush injury underneath a motor vehicle approximately 20 years ago initially presented to an outside hospital for nausea, emesis, and episodic chest pain radiating down his left arm. The patient did not know the specific details of his year-long hospital course after the accident other than undergoing multiple orthopedic and abdominal surgeries, having an IVC filter, and being in a prolonged coma. Imaging studies revealed several linear radiodensities in the right ventricular apical wall suspicious for migrated IVC filter fragments (Figure 1). Given the high-risk location that precluded a minimally invasive approach, he underwent open retrieval via right ventriculotomy. The patient was intubated, and TEE was performed to locate the emboli as they did not pierce the ventricular wall and were not visible outside the epicardium. The TEE however did not clearly visualize the emboli because of their antero-apical location (Figure 2). After discussing with the surgeon, an epicardial ultrasound was performed. The filter fragments were able to be located by this approach (Figure 3). The patient was then placed on cardiopulmonary bypass, and a targeted right ventriculotomy was completed. The foreign bodies were successfully retrieved without complications (Figure 4). The patient was extubated and subsequently discharged on POD#3.

Discussion: Intraoperative echocardiograms have been integral in clinical decision-making during cardiac surgery over the last several decades. Though TEE has become the standard, epicardial ultrasound can be an essential adjunct in certain scenarios such as ours. There have been few case studies on the use of TEE or TTE to identify foreign bodies in the heart [1,2], but none using intra-operative epicardial echocardiograms to our knowledge. An epicardial image can provide higher image resolution, particularly for anterior structures, without the chest wall or esophagus in the way [3]. Its use, however, must be coordinated with the surgeon as it interferes with the surgical field.

Conclusion: This case presents a useful application of the epicardial echocardiogram in identifying and retrieving a foreign body in the right ventricle. Though TEE is the predominant modality for intra-operative cardiac evaluation, epicardial echocardiogram remains indispensable when TEE windows are poor particularly for apical or anterior structures.

Image uploads:



Figure 1. CT chest showed hyper-dense linear structures suspicious for IVC filter strut emboli.

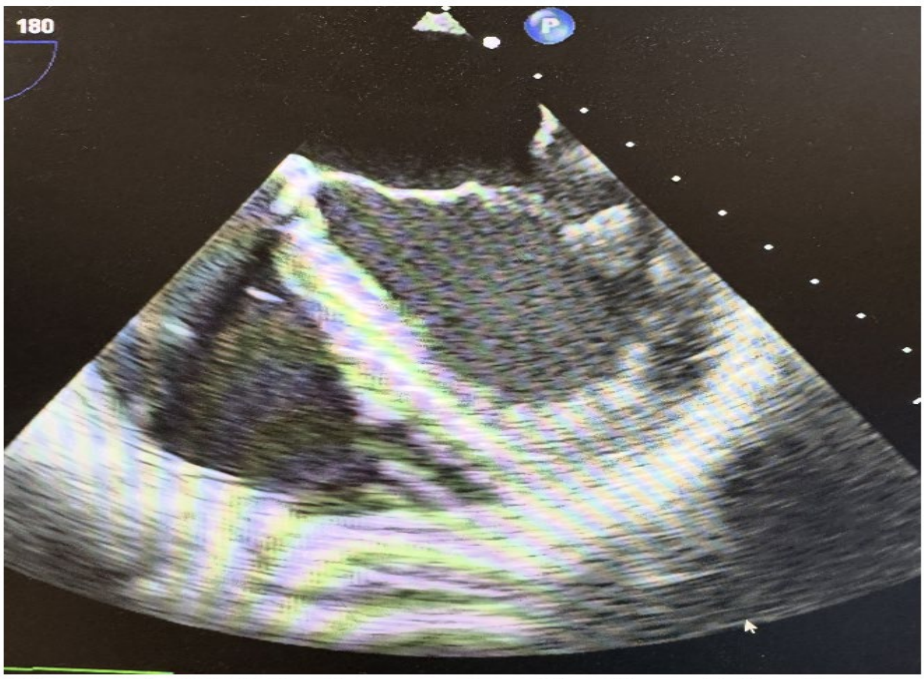


Figure 2. TEE had poor visualization of the foreign bodies in the right ventricle. Mid-esophageal four-chamber view is shown here and the foreign body cannot be seen.



Figure 3. The filter fragments were clearly visualized on the epicardial ultrasound for a targeted ventriculotomy.

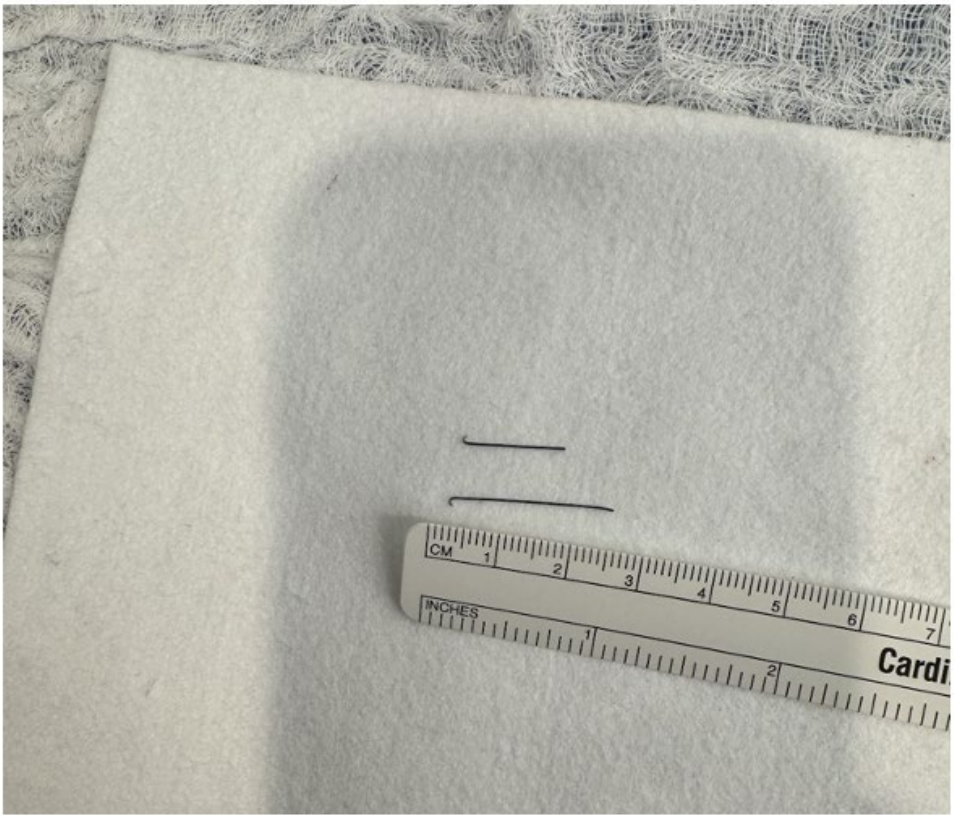


Figure 4. The retrieved IVC filter fragments.

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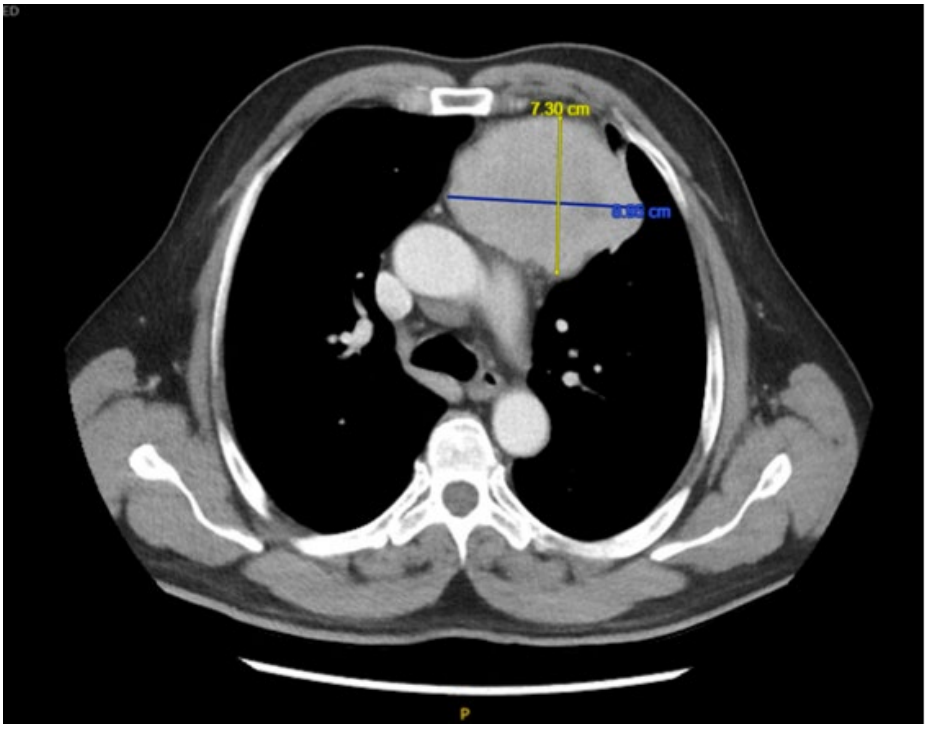
SP 1 - Kiosk 3- Poster 6
Anesthetic considerations for a large symptomatic mediastinal mass
Presenting Author: *Jessalyn Yam*

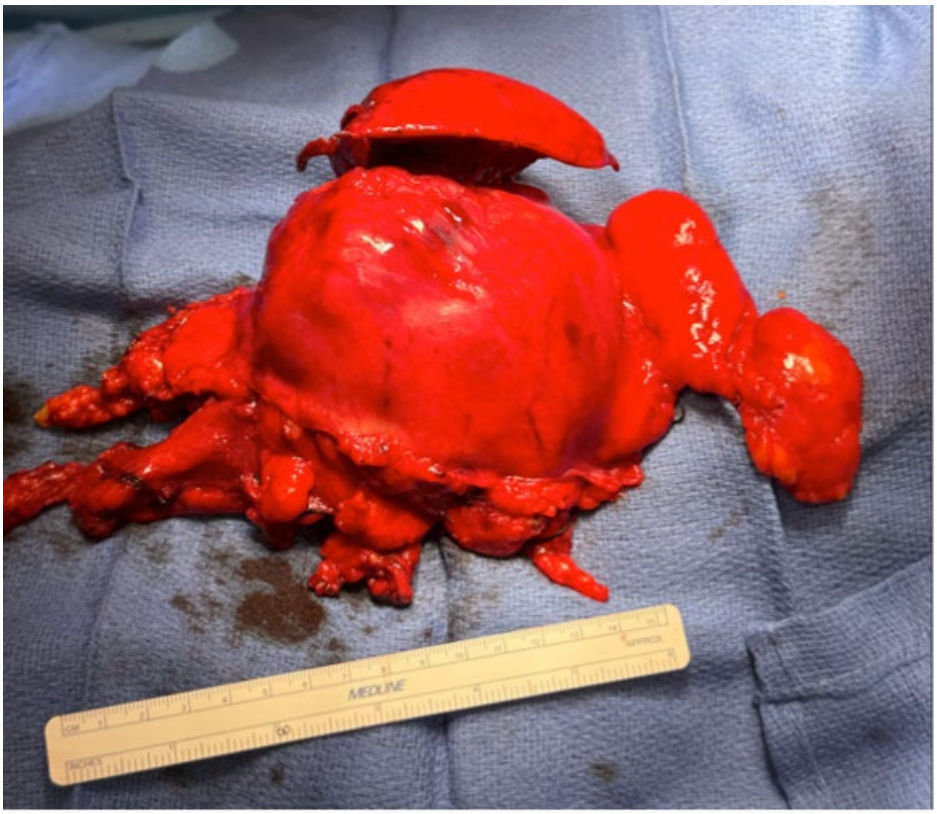
Introduction: Anterior mediastinal masses pose considerable challenges during the induction of anesthesia due to the risk of profound cardiopulmonary compromise. There are currently no standard guidelines available due to the heterogeneity of these masses. Earlier recommendations—largely derived from small case series and reports from the 1970s to 1990s—advocated for conservative strategies such as maintaining spontaneous ventilation and avoiding neuromuscular blockade (1). However, recent literature challenges these long-held principles by exploring ventilatory mechanics during induction and muscle relaxation.

Case Report: A 58-year-old male with no prior medical history presented with two months of left facial pain and several days of new-onset facial numbness, intermittent left oral commissure drooping, and a 10-lb unintentional weight loss secondary to thymic carcinoma. He denied dyspnea, orthopnea, or chest pain and was able to lie supine without symptoms. CT chest revealed a heterogeneous 9 × 7.3 × 7.5 cm anterior mediastinal mass in the left pre-vascular space, causing mild mass effect on the superior pulmonary trunk without airway or great vessel invasion. The patient was scheduled for median sternotomy with radical thymectomy and left upper-lobe wedge resection. After confirming positional tolerance, anesthesia was induced with rapid-sequence induction, followed by positive-pressure ventilation and neuromuscular blockade. A reinforced endotracheal tube and advanced airway equipment were available but ultimately not required. Ventilation was maintained with an I:E ratio of 1:2 and low peak airway pressures, without any hemodynamic deterioration after initiation of positive pressure ventilation. No airway compromise or cardiovascular instability occurred. The patient was extubated uneventfully in the operating room and discharged home on postoperative day 3.

Discussion: Thymic carcinomas are a common cause of anterior mediastinal masses and can grow to substantial size, though surgical outcomes of complete resection are often favorable. Despite its large size, this patient exhibited no postural or respiratory symptoms, indicating low peri-induction risk. For even low-risk mediastinal masses, the recommended airway strategy emphasizes maintaining spontaneous ventilation, with the option of awake fiberoptic intubation to assess for dynamic airway collapse (2). Controlled induction was deemed appropriate, with backup plans including a reinforced endotracheal tube, video laryngoscopy, fiberoptic bronchoscopy, and access to rigid bronchoscopy. Although some reports recommend standby extracorporeal circulation, its utility is limited in true emergencies, as cannulation and initiation often require several minutes during which significant hypoxia or neurologic injury may occur (3). In this setting, safe induction was achieved without relying on awake techniques or preservation of spontaneous ventilation, thereby avoiding redundant patient discomfort. This case highlights the importance of comprehensive risk stratification and individualized anesthetic planning in patients with large mediastinal masses to ensure perioperative safety while minimizing patient distress when appropriate.

Image uploads:


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MEDLINE

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SP 1 - Kiosk 3- Poster 7
Exploring FEIBA Use in Major Cardiothoracic Surgery: Insights from Yale New Haven Hospital
Presenting Author: *Rita Asuquo, MD*

Background: Factor Eight Inhibitor Bypass Activity (FEIBA) is an activated prothrombin complex concentrate used to manage refractory bleeding in Hemophilia A & B. Early 2000s studies suggested benefits in cardiovascular surgery, including reduced return to the operating room, lower transfusion requirements, and decreased chest tube output, prompting off-label use in cardiac surgery. Yale New Haven Hospital (YNHH) formalized a FEIBA protocol in 2020. However, there has been little change in research about bleeding outcomes. Now, FEIBA dosing stems from a small subset of literature that extrapolated from hemophilia dosing, and our administration is commonly guided by subjective clinical judgment without objective triggers. Importantly, this pro-coagulant comes with heavy risks. Thus, we conducted a descriptive analysis to characterize FEIBA utilization patterns and associated postoperative outcomes.

Methods: We retrospectively reviewed all adult patients who underwent major cardiacthoracic or vascular surgery at YNHH during 2023-2024 and received FEIBA intraoperatively. We evaluated demographic variables, surgery type, surgeon and anesthesiologist seniority, bypass duration, transfusion requirements, postoperative chest tube output, length of stay, and 30-day morbidity and mortality. Outcomes were summarized using descriptive statistics.

Results: 118 patients met inclusion criteria. On average, patients receiving FEIBA were older

with elevated BMIs; incidence of prior co-morbidities included chronic kidney disease (8.5%) and with a small number of patients having a pre-existing coagulopathy prior to surgery (6.8%).
Seniority of surgeons did not impact rate of returning to OR or post-operative hemoglobin, however patients of senior surgeons had longer hospital stays and greater chest tube output; minimal difference between outcomes of senior and junior anesthesiologists. Transfusion-adjusted hemoglobin changes were largely negative, consistent with continued post-op bleeding despite resuscitation and FEIBA administration. Aortic operations and cardiac transplants represented the largest proportion of cases receiving FEIBA. Average hospital stay was 18.2 days (median: 13, IQR: 15). Average chest tube output was 2.3L (median: 1.56L, IQR: 1.37).

49 patients (41.5%) experienced at least one major morbidity or mortality event within 30 days; a total of 115 morbidity/mortality events occurred. The most frequent complication was return to the operating room for re-exploration. Other complications included death, renal failure requiring CRRT/CVVH, and thrombosis.

Conclusions: At YNHH from 2023-2024, patients who receive FEIBA had substantial postoperative morbidity, most commonly returning to the OR. Analysis of coagulation studies post-operative, seems to suggest continued bleeding is occurring even after FEIBA administration. Our center consists of older, higher BMI patients receiving an aortic surgery, this could have large implications at YNHH. Given the frequency of FEIBA use and the significant complication burden observed, dosing practices and indications warrant further evaluation. This highlights the need for protocol refinement and support the feasibility of a future high-powered, prospective study.

Image uploads:

	Senior Surgeon	Junior Surgeon
Number of Cases	83.2%	16.8%
Average Bypass Time	248 minutes (~4.1 hours)	230 minutes (~3.8 hours)
Average Length of Stay	19.1 days	13.5 days
Average Chest Tube Output	~4911 mL	~1703 mL
Pre-Operative Hemoglobin (Hgb)	Similar (no major difference)	
Post-Operative Hemoglobin (Hgb)	Similar (both dropped by ~3-4 g/dL)	
Return to OR Rate	31.31%	31.58%
Intraoperative Transfusion (RBC, Plasma, Platelets, Cryoprecipitate)	Higher on average	Lower on average

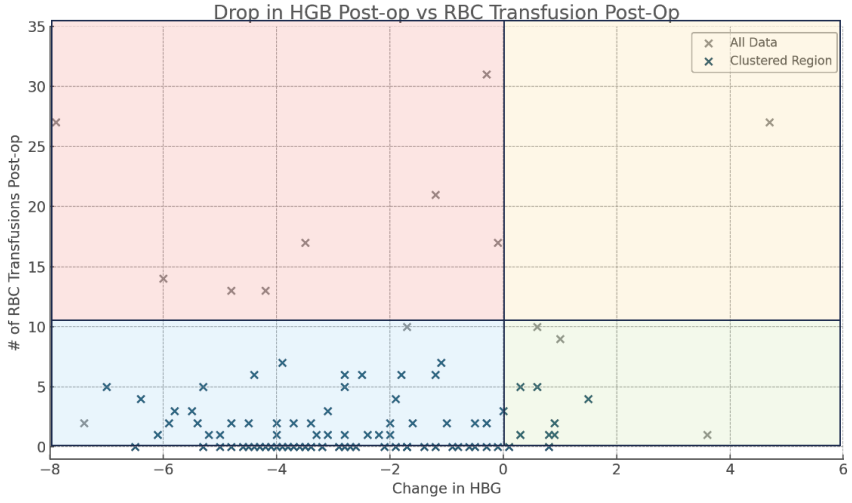
	Yes	No	% of cases with FIEBA Administration
Jr Anesthesiologist	8	9	8/17 = **47%**
Sr Anesthesiologist	64	35	64/100 = **64%**
Jr Surgeon	13	5	13/18 = 72%
Sr Surgeon	59	39	59/99 = 60%

	Yes	No	% of cases with FIEBA Administration
CABG	0	1	0%
Cardiac: Aorta	28	21	28/49 = 57%
Cardiac: Aorta + Valve	9	12	9/21 = 43%
Cardiac: Aorta + CABG	9	3	9/12 = 75%
Cardiac: Transplant	11	2	11/13 = **85%**
Cardiac: 1-Valve	7	2	7/9 = 78%
Cardiac: 2+ Valve	5	2	5/7 = 71%
Cardiac: Other	1	0	100%
Vascular: Thoracic Aorta	3	1	¾ = **75%**

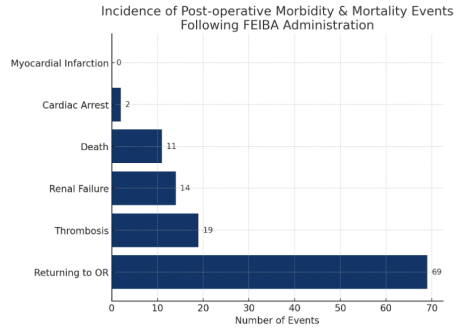
	Yes	No	% of cases with FIEBA Administration
0 – 1 hr	0	0	8/17 = 47%
1 – 3 hr	18	11	18/29 = 62%
3 – 5 hr	31	19	31/50 = 62%
5 – 8 hr	17	15	17/31 = 53%
8 Hr +	5	1	5/6 = 80%

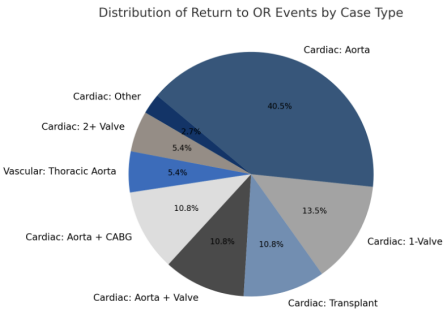
$$\frac{\textit{Post Operative HBG} - \textit{Preoperative HBG}}{\textit{\# of Intraoperative pRBCs}}$$

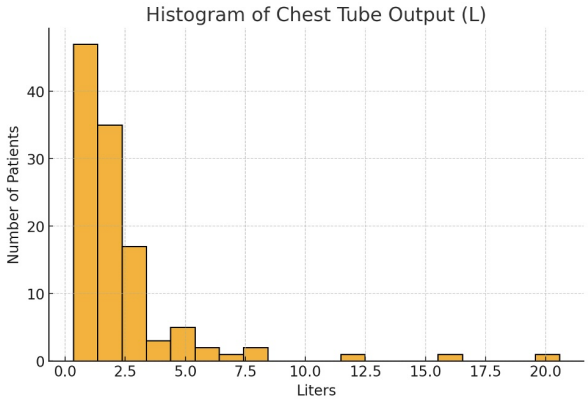
Hemoglobin Change/Unit Transfused by Case Type	
Cardiac: Aorta	-1.4
Cardiac: Aorta + Valve	-2.8
Cardiac: Transplant	-0.7
Cardiac: Aorta + CABG	-4.0
Cardiac: 1-Valve	-2.0
Cardiac: 2+ Valve	-1.6
Vascular: Thoracic Aorta	-0.5
Cardiac: Other	-0.7
Cardiac: CABG	0



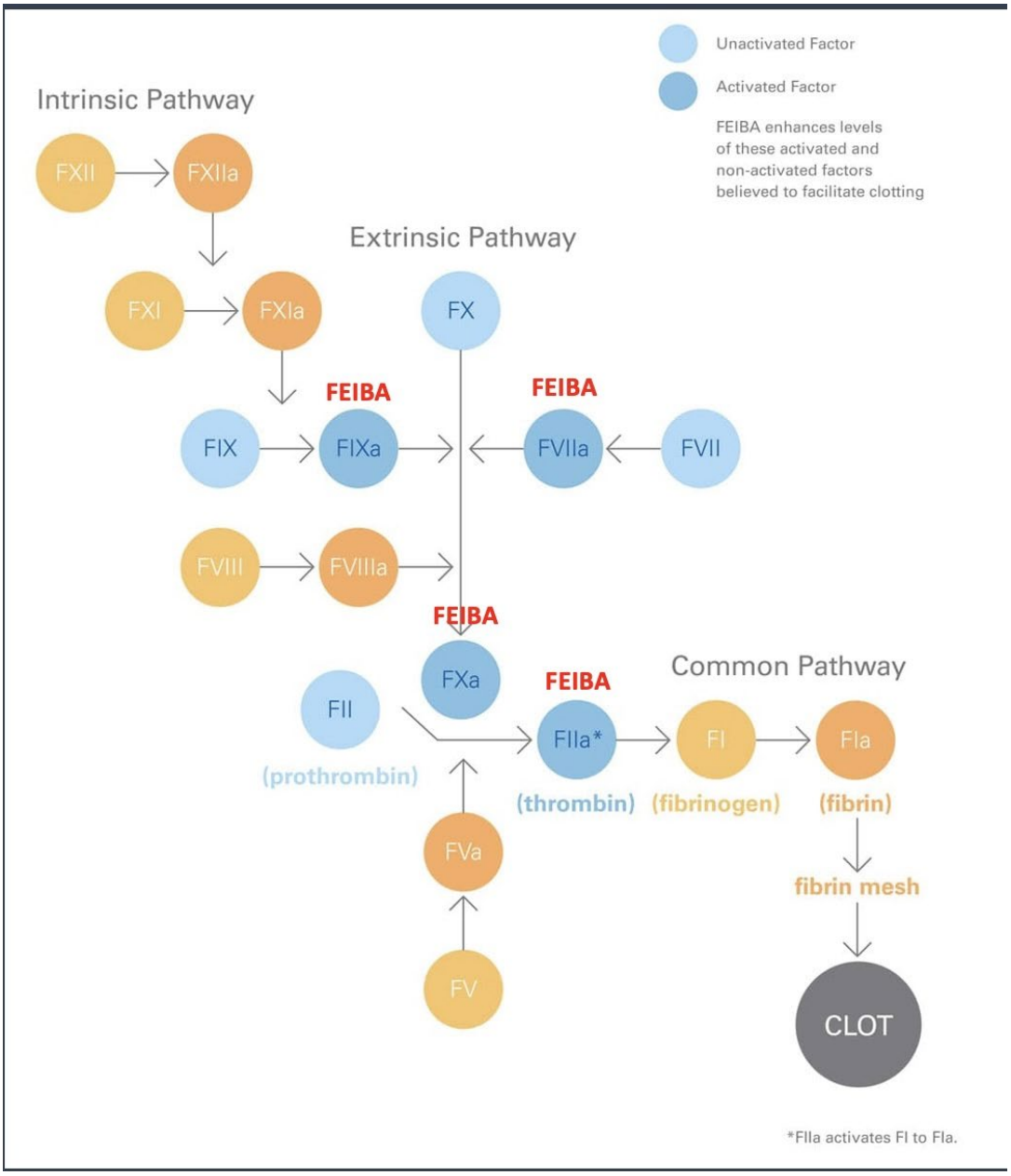
patients







	Chest Tube > Than Median	Chest Tube < Than Median
CABG	0	1
Cardiac: Aorta	28	22
Cardiac: Aorta + Valve	8	13
Cardiac: Aorta + CABG	8	4
Cardiac: Transplant	5	13
Cardiac: 1-Valve	4	4
Cardiac: 2+ Valve	4	3
Cardiac: Other	1	0
Vascular: Thoracic Aorta	3	0



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SP 1 - Kiosk 4- Poster 1
Diagnosing and Treating Preoperative Anemia in Lung Transplant Recipients
Presenting Author: *Madeline McGeagh, MD*

Background: Preoperative anemia in lung transplant (LT) recipients is associated with poor postoperative outcomes5. There is little evidence to support diagnosing, typing and treating anemic patients prior to LT. We hypothesize it is feasible for anemic patients to have their anemia typed and treated prior to LT. We propose such an intervention will result in improvement in hemoglobin and potentially reduce transfusion.

Methods: This is a single center retrospective cohort analysis of LT patients from January 2017-March 2025. Donor and recipient data, procedural characteristics, anemia diagnosis, and related timepoints were collected. As of September 7th, 2023, a preoperative evaluation process began that included screening and diagnosing anemic LT candidates and referral for possible treatment of anemia.

Cohort identification: Patients were grouped by whether they were anemic or not within 3 months of listing (< 12g/dL in women, < 13g/dL in men)1; anemic patients were further stratified by whether they were treated or not and compared (Figure 1).

Outcomes: Primary outcomes of interest were improvement in hemoglobin between listing and transplant and intraoperative transfusion requirements. Secondary outcomes measured included Dyke classification of bleeding3, primary graft dysfunction5, AKI5, and achievement of textbook outcome4. Significant differences between groups were determined by either T-test, Mann-Whitney U-Tests, Chi-Square Tests, or Fisher Exact Tests.

Results: The final cohort consisted of 704 patients with 275 (39.1%) diagnosed with anemia. Preoperative anemia was associated with younger age, female sex, African American race, smaller BMI, diagnosis of cystic fibrosis or immunodeficiency and history of prior chest surgery (Table 1). Of the 275 anemic patients, the most common diagnosis was iron deficiency anemia (N= 69; 25.1%, Table 2). Largely, the anemia was not treated (N=246; Anemia-U), with 186 patients not referred and not treated, and 60 referred but not able to be treated. There were 29 (10.5%) anemia patients that received treatment prior to their transplant (Anemia-T). These patients were younger (51 [30-65] vs 60 [42-67], p = 0.302) and were more likely to have recent transplant (p< 0.001). Treated anemic patients have higher rates of improved hemoglobin (median increase 0.2 g/dL) from listing to transplant (55.2% vs 30.1%, p = 0.014) (Table 2). We did not observe an improvement in bleeding or transfusion rates in the treated group compared to the untreated group. All secondary outcomes were found to be not significantly different or trending between groups (Table 3).

Conclusion: Identifying and treating anemia with improvement in hemoglobin prior to lung transplant is feasible. In our cohort, although 89 anemia LT patients were referred, 60 did not receive an intervention and only 29 received therapy. Lack of improvement in markers of bleeding and outcome could be related to too short a time from treatment to transplant (34 days), an increased number of redo chest patients, or anemia refractory to treatment. Future studies will identify whether earlier referral and treatment are beneficial in this high-risk population.

Image uploads:

Figure 1. Consort Diagram showing stratification of cohort. Total number of untreated anemic patients is 246 (*Anemia-U*). Of these, 186 patients were not referred to anemia clinic and not treated, while 60 were referred but not treated. 29 patients were referred and received treatment prior to transplant (*Anemia-T).*

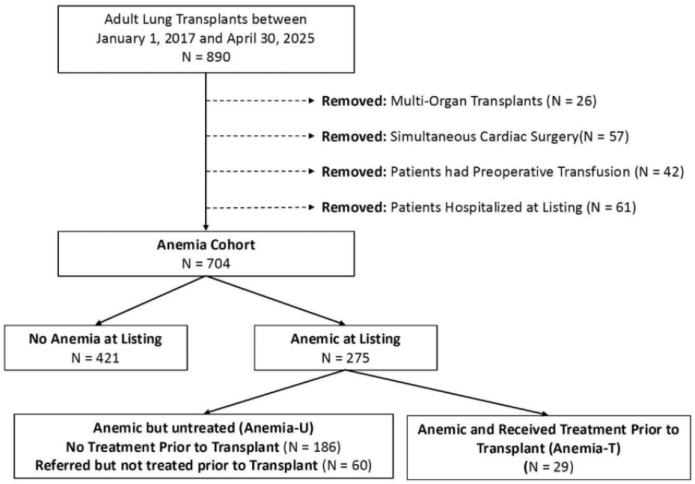


Table 1. Demographic and preoperative clinical characteristics of cohort stratified by anemia status at the time of listing and by anemia treatment status. Significant differences determined by Mann-Whitney U-tests, Independent Samples T-Tests[a], Fisher Exact Tests[b] and Chi-Square Tests[c]. *Abbreviations: IQR = interquartile range, BMI = body mass index, SD = standard deviation, CKD = chronic kidney disease.*

	Overall	Not Anemic	Anemic	P-value	Untreated (Anemia-U)	Treated (Anemia-T)
N	*704*	*421*	*275*		*246*	*29*
Transplant Year (%)				0.570[c]		
2017	75 (10.7)	40 (9.5)	35 (12.7)		31 (12.6)	4 (13.8)
2018	80 (11.4)	43 (10.2)	37 (13.5)		36 (14.6)	1 (3.4)
2019	96 (13.6)	57 (13.5)	39 (14.2)		37 (15.0)	2 (6.9)
2020	118 (16.8)	78 (18.5)	40 (14.5)		38 (15.4)	2 (6.9)
2021	90 (12.8)	53 (12.6)	37 (13.5)		34 (13.8)	3 (10.3)
2022	86 (12.2)	52 (12.4)	34 (12.4)		32 (13.0)	2 (6.9)
2023	52 (7.4)	33 (7.8)	19 (6.9)		17 (6.9)	2 (6.9)
2024	82 (11.6)	47 (11.2)	27 (9.8)		16 (6.5)	11 (37.9)
2025	25 (3.6)	18 (4.3)	7 (2.5)		5 (2.0)	2 (6.9)
Bilateral lung transplant (%)	635 (90.2)	377 (89.5)	251 (91.3)	0.536[c]	224 (91.1)	27 (93.1)
Age (years) (median [IQR])	64.00 [55.00, 68.00]	65.00 [59.00, 69.00]	60.00 [44.50, 67.00]	<0.001	61.50 [48.00, 67.00]	51.00 [30.00, 65.00]
Sex (%)				<0.001[c]		
Female	271 (38.5)	112 (26.6)	157 (57.1)		139 (56.5)	18 (62.1)
Male	433 (61.5)	309 (73.4)	118 (42.9)		107 (43.5)	11 (37.9)
Race (%)				0.020[c]		
Caucasian/White	597 (85.5)	368 (88.2)	221 (81.0)		202 (82.1)	19 (70.4)
Black or African American	80 (11.5)	37 (8.9)	43 (15.8)		35 (14.2)	8 (29.6)
Other	21 (3.0)	12 (2.9)	9 (3.3)		9 (3.7)	0 (0.0)
BMI (kg/m2) (mean (SD))	24.83 (3.66)	25.47 (3.27)	23.82 (4.03)	<0.001[a]	23.96 (3.92)	22.64 (4.72)
Indication for Transplant (%)				<0.001[b]		
A: Obstructive Lung Disease	142 (20.2)	84 (20.0)	56 (20.4)		51 (20.7)	5 (17.2)
B: Pulmonary Vascular Disease	10 (1.4)	4 (1.0)	6 (2.2)		5 (2.0)	1 (3.4)
C: Cystic Fibrosis or Immunodeficiency	35 (5.0)	6 (1.4)	29 (10.5)		23 (9.3)	6 (20.7)
D: Restrictive Lung Disease	517 (73.4)	327 (77.7)	184 (66.9)		167 (67.9)	17 (58.6)
Transplant Number (%)				0.002[b]		
1	664 (94.9)	405 (96.7)	251 (91.9)		227 (93.0)	24 (82.8)
2	30 (4.3)	14 (3.3)	16 (5.9)		13 (5.3)	3 (10.3)
3	6 (0.9)	0 (0.0)	6 (2.2)		4 (1.6)	2 (6.9)
Lung Allocation Score (mean (SD))	45.21 (11.04)	44.44 (10.36)	46.35 (11.90)	0.045[a]	46.34 (11.75)	46.49 (14.55)
Lung Composite Allocation Score (mean (SD))	27.93 (5.97)	27.75 (6.32)	28.43 (5.57)	0.524[a]	27.45 (6.04)	30.73 (3.43)
Prior Chest Surgery (%)	226 (32.1)	116 (27.6)	109 (39.6)	0.001[c]	94 (38.2)	15 (51.7)
Last Preoperative Creatinine (mg/dL) (median [IQR])	0.90 [0.70, 1.00]	0.90 [0.80, 1.00]	0.80 [0.70, 1.00]	0.006	0.80 [0.70, 1.00]	0.80 [0.70, 0.80]
CKD Diagnosis (%)	51 (7.2)	24 (5.7)	23 (8.4)	0.225[c]	21 (8.5)	2 (6.9)

Table 2: Preoperative anemia diagnosis and characteristics of cohort stratified by anemia status at the time of listing and by anemia treatment status. Significant differences determined by Mann-Whitney U-tests, Independent Samples T-Tests[a], Fisher Exact Tests[b] and Chi-Square Tests[c]. *Abbreviations: IQR = interquartile range.*

	Overall	Not Anemic	Anemic	P-value	Untreated (Anemia-U)	Treated (Anemia-T)	P-value
N	*704*	*421*	*275*		*246*	*29*	
Anemia Diagnosis Type							
Chronic Disease (%)	12 (4.4)	--	12 (4.4)	--	9 (3.7)	3 (10.3)	0.121[b]
Blood Loss Related (%)	1 (0.4)	--	1 (0.4)	--	1 (0.4)	0 (0.0)	>0.999[b]
Iron Deficiency (%)	69 (25.1)	--	69 (25.1)	--	43 (17.5)	26 (89.7)	<0.001[b]
Other (%)	4 (1.5)	--	4 (1.5)	--	3 (1.2)	1 (3.4)	0.361[b]
Hemoglobin at Listing (g/dL) (median [IQR])	12.80 [11.80, 13.90]	13.70 [12.90, 14.40]	11.50 [10.60, 12.20]	<0.001	11.60 [10.70, 12.30]	11.20 [10.40, 11.50]	0.024
Preoperative Hemoglobin at Transplant (g/dL) (median [IQR])	12.70 [11.70, 13.70]	13.40 [12.70, 14.20]	11.40 [10.50, 12.20]	<0.001	11.50 [10.60, 12.20]	11.10 [9.90, 12.30]	0.448
Days Between Treatment and Surgery (median [IQR])	--	--	--	--	--	34.00 [24.00, 77.50]	--

Table 3. Primary and secondary outcomes compared between Anemia-U and Anemia-T groups. Intraoperative and postoperative day 1 blood product utilization and Dyke bleeding score, as well as secondary outcomes of interest stratified by anemia status at time of listing and by anemia treatment status. Significant differences determined by Mann-Whitney U-tests, Fisher Exact Tests[b] and Chi-Square Tests[c]. *Abbreviations: IQR = interquartile range, POD1 = postoperative day 1, PGD = primary graft dysfunction, RRT = renal replacement therapy*

	Untreated (Anemia-U)	Treated (Anemia-T)	P-value
N	*246*	*29*	
Change in Hemoglobin from Listing (g/dL) (median [IQR])	-0.05 (0.89)	0.20 (1.31)	0.179
Hemoglobin Improved from listing (%)	73 (29.7)	16 (55.2)	0.010[c]
Intraoperative + POD1 Blood Products (Units) (median [IQR])			
Red Blood Cells	3.00 [1.00, 5.00]	4.00 [2.00, 8.00]	0.042
Cryoprecipitate	0.00 [0.00, 2.00]	1.00 [0.00, 2.00]	0.220
Fresh Frozen Plasma	0.00 [0.00, 2.00]	1.00 [0.00, 4.00]	0.022
Platelets	0.00 [0.00, 1.00]	1.00 [0.00, 4.00]	0.019
Delayed Sternal Closure (%)	41 (16.7)	12 (41.4)	0.003[c]
Chest Tube Output (mL) (12 Hours) (median [IQR])	607.50 [446.25, 900.00]	760.00 [560.00, 1692.00]	0.016
Received Factor VII (%)	5 (2.0)	1 (3.4)	0.491[b]
Received PCC/Factor IX (%)	8 (3.3)	3 (10.3)	0.098[b]
Re-Exploration for Bleeding (%)	52 (21.1)	11 (37.9)	0.072[c]
Dyke Severe/Massive Bleed (%)	102 (41.5)	19 (65.5)	0.023[c]
Dyke Bleeding Classification (%)			0.038[b]
None/Insignificant	12 (4.9)	2 (6.9)	
Mild	33 (13.4)	2 (6.9)	
Moderate	99 (40.2)	6 (20.7)	
Severe	80 (32.5)	12 (41.4)	
Massive	22 (8.9)	7 (24.1)	
Postoperative Hospital Length of Stay (Days) (median [IQR])	23.59 [16.73, 40.31]	21.99 [17.76, 46.78]	0.904
AKI (%)	103 (41.9)	16 (55.2)	0.242[c]
KDIGO Stage (%)			0.101[b]
0	143 (58.1)	13 (44.8)	
1	56 (22.8)	6 (20.7)	
2	30 (12.2)	4 (13.8)	
3	17 (6.9)	6 (20.7)	
Achieved Textbook Outcome (%)	55 (22.4)	5 (17.2)	0.640[b]
Intraoperative Complication (%)	15 (6.1)	1 (3.4)	>0.999[b]
PGD 3 at 48 or 72 Hours (%)	62 (27.4)	9 (31.0)	0.851[c]
Extubated >48 Hours Postop (%)	112 (45.5)	18 (62.1)	0.136[c]
Re-Intubated (%)	56 (22.8)	6 (20.7)	0.986[c]
Dialysis/RRT within 30 Days (%)	29 (11.8)	5 (17.2)	0.377[b]
Hospital Readmission within 30 Days (%)	60 (24.9)	6 (20.7)	0.788[c]
Re-intervention (%)	84 (34.1)	14 (48.3)	0.194[c]
Surgical (%)	78 (31.7)	14 (48.3)	0.114[c]
Bronchoscopy (%)	4 (1.6)	0 (0.0)	>0.999[b]
Radiological (%)	8 (3.3)	0 (0.0)	>0.999[b]
Mortality (90 Days) (%)	11 (4.5)	3 (10.3)	0.173[b]
ECMO within first 72 Hours (%)	39 (16.3)	9 (31.0)	0.090[c]
Tracheostomy within 7 Days (%)	58 (23.6)	13 (44.8)	0.025[c]
Biopsy Proven Rejection within 30 Days (%)	22 (9.3)	0 (0.0)	0.143[b]

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SP 1 - Kiosk 4- Poster 2

On-Table Extubation Following Hypothermic Circulatory Arrest: A Case Series Demonstrating Feasibility and Safety in High-Risk Cardiac Surgery Patients

Presenting Author: *Nicholas Owen, DO*

Background:Early extubation, or fast-track extubation (FTE), defined by the Society of Thoracic Surg eons as extubation within 6 hours of ICU arrival, is an established benchmark associated with reduced costs and similar outcomes after cardiac surgery. Recent data suggest that on-table extubation (OTE), or extubation in the operating room, may further improve outcomes, including lower mortality and shorter ICU stay. Our institution, an early adopter of OTE, achieves it in ~70% of cardiac surgeries. Building on this experience, we applied OTE to patients requiring hypothermic

circulatory arrest (HCA), a group traditionally excluded due to perceived risk.

Methods: A retrospective review was conducted of all cardiac surgeries performed at our institution between March 2021 and January 2025. Patients undergoing HCA were identified, and those extubated intraoperatively were analyzed. Selection for OTE was made case-by-case after multidisciplinary discussion of patient and surgical factors including BMI, airway, neurological status, coagulation correction, hemodynamic stability, and oxygenation on minimal ventilatory settings. Demographics, comorbidities, operative details, and postoperative outcomes were collected. All patients received intraoperative methadone, superficial parasternal blocks, and met established extubation criteria.

Results: Of 85 cardiac surgeries involving HCA, 12 patients (14%) underwent successful OTE. Mean age was 64 ±14 years; 8 were male, and mean BMI was 29.7 (range 19–36). Five (42%) were emergent cases for acute Type A dissection. No patient required postoperative reintubation or re-exploration for bleeding. Initial ICU oxygen therapy was nasal cannula (9), high-flow nasal cannula (2), or BiPAP (1). There were no cases of pneumonia, pulmonary embolism, pleural effusion requiring drainage, or pneumothorax. Median ICU stay was 21.5 hours (IQR 17.3–30.6), and median hospital stay was 6.6 days (IQR 5.0–9.7). No mortalities occurred. Mean HCA duration was 31.7 ± 21.7 minutes; 10 patients (83%) received antegrade cerebral perfusion. Mean vasoactive-inotropic score at extubation was 3.9 (range 0–32). All received methadone (mean 11.9 ±4.6 mg; 0.17 ±0.05 mg/kg).

Conclusion: This case series demonstrates that on-table extubation can be safely achieved in selected patients undergoing cardiac surgery with hypothermic circulatory arrest, including emergent cases. Despite the traditionally high-risk nature of this population, no mortalities or reintubations occurred, and ICU and hospital stays were favorable. These results suggest fast-track recovery principles can extend beyond elective, low-risk cases when guided by a structured, multidisciplinary approach. OTE may allow earlier neurologic assessment and faster recovery, with potential benefits in morbidity and resource use. Further prospective studies are warranted to define its broader applicability among high-risk cardiac surgery patients.

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SP 1 - Kiosk 4- Poster 3

Preoperative Diastolic Dysfunction and Postoperative Mortality in Patients Undergoing Lower Extremity Bypass Grafting

Presenting Author: *Pratyush Devarasetty, MD*

Introduction:The relationship between left ventricular diastolic dysfunction and clinical outcomes has been increasingly studied.1However, studies differ on the effect diastolic dysfunction has on outcomes in the non-cardiac surgery patient population.2-3 This study aims to evaluate the association of diastolic dysfunction and mortality in patients undergoing lower extremity bypass grafting surgeries using the new 2025 American Society of Echocardiography (ASE) diastolic function guidelines.4

Methods: This retrospective study identified 518 adult patients who underwent lower extremity bypass grafting surgery at a single center between January 2018 and December 2022. Diastolic dysfunction grading was determined using the 2025 ASE diastolic function guidelines (Figure 1). Only patients who had a transthoracic echocardiography (TTE) study within 1 year prior to surgery and sufficient TTE data to determine diastolic dysfunction grading were included. Postoperative mortality was assessed at 30 days and 1 year after the index surgery. The association between diastolic dysfunction and mortality was evaluated using survival analysis and reported as a hazard ratio.

Results:Of the 224 patients with sufficient echocardiographic data for diastolic dysfunction grading, 144 had a TTE within 1 year prior to surgery (Figure 2). Table 1a and 1b show the demographics, baseline characteristics, and outcomes for the study cohort. Patients were dichotomized into normal left atrial pressure (LAP) (normal or grade I) or elevated LAP (grade II or III) groups based on their diastolic dysfunction grading. Compared to the normal LAP group, the elevated LAP group had significantly more atrial fibrillation (31.6% vs. 15.1%, p=0.05), congestive heart failure (47.4% vs. 19.8%, p=0.002), and chronic kidney disease (47.4% vs 23.6%, p=0.01). The elevated LAP group also had significantly more pulmonary hypertension (RVSP 38.5 vs 25.0 mmHg, p< 0.001) and worse RV-PA coupling (TAPSE/RVSP 0.49 vs 0.89 mm/mmHg p< 0.001) than the patients in the normal LAP group. The 30 day (5.3% vs. 1.9%, p=0.609) and 1 year mortality (18.4% vs. 8.5%, p=0.171) were higher in the elevated LAP group than the normal LAP group. Patients with an elevated LAP showed a trend towards increased mortality compared to those with a normal LAP, with a hazard ratio of 2.26 (95% CI 0.84–6.08, p=0.105) (Table 2). Figure 3 shows the survival curve of the study cohort dichotomized into normal and elevated LAP groups.

Conclusion: Preoperative TTE to evaluate diastolic dysfunction prior to lower extremity bypass grafting surgery may help identify vascular surgery patients at higher mortality risk. The present findings invite further investigation into integrating diastolic assessment into routine preoperative evaluation to enhance optimization.

Image uploads:

Figure 1. 2025 ASE diastolic function guidelines

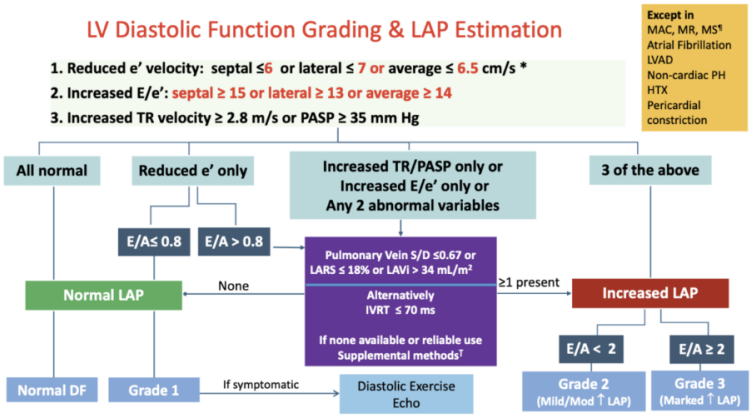


Figure 3 Algorithm for estimation of mean LAP for patients in sinus rhythm and who do not have severe primary MR, any degree of mitral stenosis (MS), or moderate or severe MAC. The algorithm should also not be applied to patients in atrial fibrillation, heart transplant (HTX) recipients, noncardiac PH, pericardial constriction or LV assist device (LVAD). *For annular e′ velocity, age-adjusted lower limits of normal values shown in Table 6 can be applied in place of the values shown in this figure. ¶The algorithm should also not be applied to patients with mitral valve repair, mitral valve replacement, or mitral-transcatheter edge-to-edge repair. *DF*, Diastolic function; †, PR end-diastolic velocity ≥ 2m/s, PA diastolic pressure ≥ 16 mm Hg, mitral inflow L-wave velocity ≥ 50 cm/s, Ar-A duration > 30 ms, and/or a decrease in mitral E/A ratio of ≥50% with Valsalva maneuver.

Figure 2. Consort diagram for study cohort.

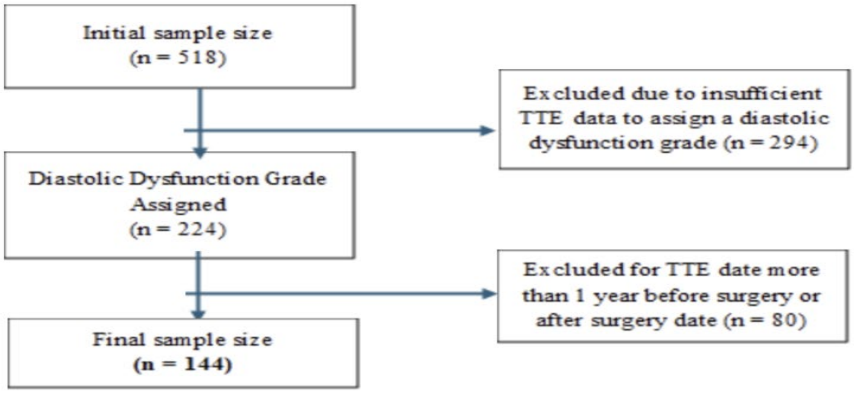


Figure 3. Survival curve of the study cohort based on normal left atrial pressure (red line) and elevated left atrial pressure (blue line).

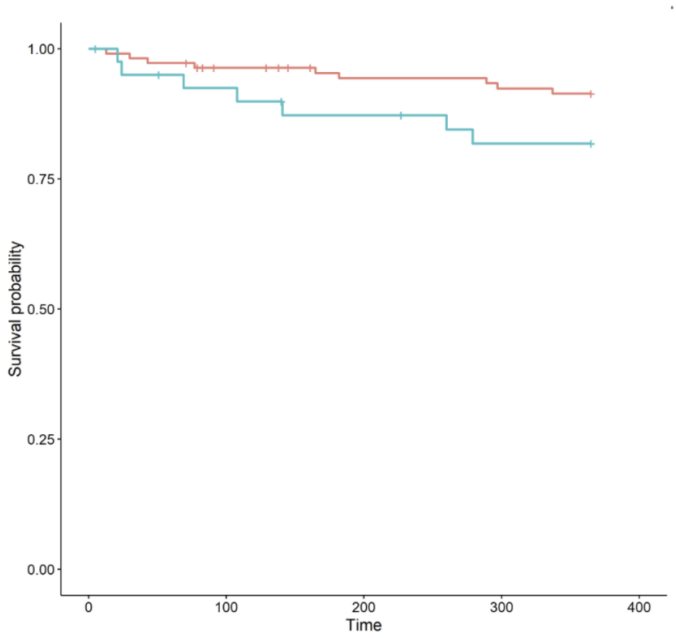


Table 1a. Demographics, baseline characteristics of the study cohort dichotomized into normal and elevated left atrial pressure groups.

	Normal LAP (n = 106)	Elevated LAP (n = 38)	Total (n = 144)	P value
Demographics				
Age	65.4 ± 10.8	69.2 ± 12.0	66.4 ± 11.3	0.070
Sex (Male)	71 (67.0%)	24 (63.2%)	36 (72.0%)	0.820
Race				0.162
Asian	0 (0.0%)	1 (2.6%)	1 (0.7%)	
American Indian	2 (1.9%)	1 (2.6%)	3 (2.1%)	
Black	45 (42.5%)	23 (60.5%)	68 (47.2%)	
Hispanic	1 (0.9%)	0 (0.0%)	1 (0.7%)	
White	56 (52.8%)	13 (34.2%)	69 (47.9%)	
Not reported	2 (1.9%)	0 (0.0%)	2 (1.4%)	
Lost to follow-up within 1 year	8 (7.5%)	4 (10.5%)	12 (8.3%)	0.820
Comorbidities				
Hypertension	97 (91.5%)	34 (89.5%)	131 (91.0%)	0.963
Hyperlipidemia	66 (62.3%)	20 (52.6%)	86 (59.7%)	0.398
Diabetes mellitus	51 (48.1%)	16 (42.1%)	67 (46.5%)	0.655
Chronic kidney disease	25 (23.6%)	18 (47.4%)	43 (29.9%)	0.011
End stage renal disease	3 (2.8%)	5 (13.2%)	8 (5.6%)	0.049
Coronary artery disease	48 (45.3%)	21 (55.3%)	69 (47.9%)	0.386
COPD	24 (22.6%)	9 (23.7%)	33 (22.9%)	>0.999
Congestive heart failure	21 (19.8%)	18 (47.4%)	39 (27.1%)	0.002
Atrial fibrillation	16 (15.1%)	12 (31.6%)	28 (19.4%)	0.050
Cerebrovascular accident	30 (28.3%)	9 (23.7%)	39 (27.1%)	0.736
Immunosuppression	8 (7.5%)	5 (13.2%)	13 (9.0%)	0.480
Obesity	36 (34.0%)	7 (18.4%)	43 (29.9%)	0.112
Morbid obesity	5 (4.7%)	1 (2.6%)	6 (4.2%)	0.937
Smoking status				0.677
Active	47 (44.3%)	15 (39.5%)	62 (43.1%)	
Former	50 (47.2%)	18 (47.4%)	68 (47.2%)	
Never	9 (8.5%)	5 (13.2%)	14 (9.7%)	

Values are mean (SD) or n (%). Abbreviations: LAP, left atrial pressure; COPD, chronic obstructive pulmonary disease; TAPSE, tricuspid annular plane systolic excursion; RVSP, right ventricular systolic pressure; LV, left ventricle; RV, right ventricle.

Table 1b. The Echocardiography parameters and outcomes of the study cohort dichotomized into normal and elevated left atrial pressure groups.

Echocardiography Parameters	Normal LAP (n = 106)	Elevated LAP (n = 38)	Total (n = 144)	P value
TAPSE (mm)	21.0 [17.0, 24.5]	23.0 [18.0, 25.0]	21.0 [17.0, 25.0]	0.327
RVSP (mmHg)	25.00 [12.50, 30.00]	39.50 [34.45, 51.25]	28.00 [15.00, 35.75]	<0.001
TAPSE/RVSP (mm/mmHg)	0.89 [0.66, 1.41]	0.49 [0.40, 0.65]	0.79 [0.49, 1.20]	<0.001
Tricuspid Regurgitation				0.012
None	16 (15.1%)	0 (0.0%)	16 (11.1%)	
Trace/Mild	86 (81.1%)	33 (86.8%)	119 (82.6%)	
Moderate	4 (3.8%)	4 (10.5%)	8 (5.6%)	
Severe	0 (0.0%)	1 (2.6%)	1 (0.7%)	
Mitral Regurgitation				0.009
None	24 (22.9%)	0 (0.0%)	24 (20.0%)	
Trace/Mild	78 (74.3%)	14 (93.3%)	92 (76.7%)	
Moderate	3 (2.9%)	0 (0.0%)	3 (2.5%)	
Severe	0 (0.0%)	1 (6.7%)	1 (0.8%)	
LV Ejection Fraction				0.062
<15%	0 (0.0%)	1 (2.6%)	1 (0.7%)	
15-25%	2 (1.9%)	3 (7.9%)	5 (3.5%)	
>25%-35%	3 (2.8%)	3 (7.9%)	6 (4.2%)	
>35%-45%	8 (7.5%)	0 (0.0%)	8 (5.6%)	
>45%-55%	11 (10.4%)	3 (7.9%)	14 (9.7%)	
>55%	82 (77.4%)	28 (73.7%)	110 (76.4%)	
RV Function				0.248
Normal	99 (94.3%)	35 (92.1%)	134 (93.7%)	
Mild Dysfunction	6 (5.7%)	2 (5.3%)	8 (5.6%)	
Moderate Dysfunction	0 (0.0%)	1 (2.6%)	1 (0.7%)	
Severe Dysfunction	0 (0.0%)	0 (0.0%)	0 (0.0%)	
RV Size				0.693
Normal	100 (95.2%)	35 (92.1%)	135 (94.4%)	
Mild Dilation	4 (3.8%)	2 (5.3%)	6 (4.2%)	
Moderate Dilation	1 (1.0%)	1 (2.6%)	2 (1.4%)	
Severe Dilation	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Outcomes				
30 Day Readmission	30 (28.3%)	6 (15.8%)	36 (25.0%)	0.190
30 Day Mortality	2 (1.9%)	2 (5.3%)	4 (2.8%)	0.609
1 Year Mortality	9 (8.5%)	7 (18.4%)	16 (11.1%)	0.171

Table 2. Hazard ratio models for one-year mortality based on normal and elevated left atrial pressure (LAP) to assess association with mortality.

	Hazard Ratio	P value
Normal LAP (reference)	1.0 (reference)	N/A
Elevated LAP	2.26 (0.84, 6.08)	0.105

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SP 1 - Kiosk 4- Poster 4

Impact of Postoperative Vasopressor Use on Acute Kidney Injury Following Orthotopic Heart Transplantation

Presenting Author: *Kevin Maar*

INTRODUCTION: Hemodynamic instability following orthotopic heart transplantation (OHT) frequently requires vasoactive and inotropic management. This study examined the relationship between postoperative vasoactive therapy and acute kidney injury (AKI) in OHT patients.

METHODS: Vasoactive therapy agents, maximum dose, therapy duration, and clinical data of 581 OHT recipients was retrospectively analyzed (January 2012–December 2024) after institutional review board approval. The primary outcome was AKI staged using Kidney Disease: Improving Global Outcomes (KDIGO) categories. A binomial logistic regression model adjusted for preoperative albumin, preoperative creatinine, transfusion requirements, IMPACTscore, cardiopulmonary bypass time, diabetes mellitus status, and preoperative mechanical circulatory

support was used. The secondary outcome was postoperative dialysis requirement.

RESULTS: Three hundred nine patients (53.2%) had AKI. 120 (20.8%) had stage 1, 29 (5%) had stage 2, 160 (27.8%) had stage 3 KDIGO. 140 (24.1%) required postoperative dialysis. In the multivariable logistic regression, norepinephrine (days; AOR: 1.142 (1.081 - 1.21) p< .001) and epinephrine (days; AOR: 1.150 (1.029 - 1.29) p=0.014) therapy duration were independently associated with an increased risk for AKI. Maximum vasopressin dose (AOR: 4.102 (1.182 - 14.244) p=0.026) and maximum terlipressin dose (AOR: 3.101 (1.131 - 8.499) p=0.028) were also independently associated with an increased risk of AKI. An increased risk of postoperative dialysis could be observed in higher norepinephrine and epinephrine therapy duration (days; AOR: 1.135 (1.088 - 1.184) p< .001 and AOR: 1.122 (1.036 - 1.214) p=0.005 respectively).

CONCLUSION: Postoperative vasopressor pharmacotherapy may have an impact on AKI after OHT. The intricate pharmacological and pathophysiological mechanisms warrant further investigation into this topic.

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SP 1 - Kiosk 4- Poster 5
Utility of the Superior Vena Cava Collapsibility Index (SVC-CI) to Predict Fluid Responsiveness in Patients with Coronary Artery Disease Undergoing Surgical Revascularization
Presenting Author: *Parker Burrows, MD*

Background/Introduction: Fluid responsiveness is a growing term aimed at investigating the transient state regarding fluid resuscitation in surgical and critical care patients. Given the risk of morbidity and mortality for under and over resuscitation, extensive research has investigated which variables may help guide this clinical decision. Dynamic variables, such as echocardiography combined with inferior vena cava collapsibility, have gained popularity compared to historic static assessment variables, such as central venous pressure or pulmonary artery occlusion pressure. Recently, the superior vena cava collapsibility index (SVC-CI) utilizing transesophageal echocardiography (TEE) has explored assessing fluid responsiveness in surgeries such as craniotomies, elective general surgeries, and open vascular surgeries1-3 . An opportunity is present to extend this exploration into cardiac surgery.

Methods: Forty-three patients to date have undergone isolated coronary artery bypass grafting (CABG) via cardiopulmonary bypass (CPB) at a single tertiary care center. Participants were divided into two cohorts based on preoperative left ventricular ejection fraction (LVEF ≥40% and < 40%). At two separate surgical timepoints, after anesthetic induction but before sternotomy (pre-CBP) and after separation from cardiopulmonary bypass and chest closure (post-CBP), participants were given an augmented fluid bolus via a change in bed position from 15° reverse Trendelenburg to 15°

Trendelenburg. A TEE generated cardiac output (CO) and SVC-CI were obtained at each designated timepoint. Participants were considered fluid responders based on an increase in CO by 15% between position changes. A receiver operator characteristic (ROC) curve was generated, and the area underneath (AUROC) was calculated. The Youden Index was utilized to determine an optimal cutoff value for the SVC-CI to predict fluid responders.

Results/Discussion: Based on our preliminary data, of the 43 participants that underwent isolated CABG utilizing CPB who met our inclusion and exclusion criteria, echocardiographic data was available for analysis in 28 participants at the pre-CPB timepoint and 26 participants at the post-CPB timepoint. Twelve (43%) participants at the pre-CPB timepoint and 10 (38%) participants during the post-CPB timepoint were fluid responders. The AUROC at the pre-CBP timepoint was 0.542 with an optimal cutoff value of 36.1% with sensitivity of 0.5 and specificity of 0.75. The AUROC at the post-CBP timepoint was 0.381 with an optimal cutoff value of 34.1% with sensitivity of 0.3 and specificity of 0.81. All but 2 participants had preoperative LVEF ≥40%, so subgroup analysis was not completed with the preliminary data.

Conclusion: To date, based on our preliminary data, the SVC-CI does not appear to be a useful marker of fluid responsiveness in patients undergoing CABG with CPB. This dynamic marker performed better at the pre-CPB timepoint. Other right heart echocardiographic metrics, such as tricuspid annulus plane systolic excursion (TAPSE), have been shown to lose utility after pericardiotomy. Based on our power analysis, we plan to enroll a total of 120 participants, with an equal number in each LVEF cohort.

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SP 1 - Kiosk 4- Poster 6

Assessing Perioperative Ultrasound Training Needs Among Anesthesia Clinicians: A Multimodal Departmental Evaluation

Presenting Author: *Adil Manji, MD*

Background: Point-of-care ultrasound (POCUS) has become an essential tool in anesthesiology for perioperative decision-making, revealing critical pathology that can alter management.1 Fundamental POCUS skills are now included in anesthesiology training requirements and board certification exams.2 However, there is little standardization of POCUS training across programs, and many anesthesiologists lack formal ultrasound training.3 We conducted a departmental needs assessment to guide development of a structured perioperative ultrasound curriculum.

Methods: REDCap surveys were administered in the Beth Israel Deaconess Medical Center Department of Anesthesia, targeting faculty, trainees (residents/fellows), CRNAs and advanced providers. The survey covered ultrasound certification status, clinical use patterns, perceived barriers, educational needs, and self-assessed confidence. Responses were collected anonymously and analyzed descriptively, including subgroup comparisons by professional role. Faculty subspecialty data (e.g., ICU or cardiac training) were compared to institutional demographics to evaluate sample representativeness.

Results: Out of approximately 75 eligible clinicians, 40 completed the survey (response rate ~53%). The survey population included a representative mix of anesthesia faculty and trainees, with a slightly higher proportion of ICU and cardiac-trained clinicians among respondents compared to departmental averages. Formal POCUS training, defined as either certification (e.g., NBE, POCUS Academy) or certificate programs (e.g., ASA POCUS Certificate), was reported by approximately 20% of respondents, a relatively high figure likely reflecting the presence of cardiac and ICU-trained faculty. Among those, 12% held full certification and 8% reported certificate completion. Over 90% expressed interest in further POCUS training. Notably, around 75% of respondents supported the development of a formal perioperative POCUS service, highlighting perceived clinical value. Only about half of faculty felt comfortable supervising ultrasound exams, and both faculty and trainees acknowledged significant gaps in their ultrasound proficiency. Analysis revealed that confidence and experience were limited across all roles (attending, trainee, CRNA, NP), suggesting that educational gaps are not limited to a single subgroup. Most clinicians agreed that POCUS would be valuable in acute perioperative scenarios (for example hypotension or respiratory distress), but barriers hindered routine use, such as time and workflow constraints, limited equipment or image management resources, and documentation challenges.

Conclusion: This needs assessment demonstrates a strong demand for structured POCUS education and highlights clear deficiencies in skills, infrastructure, and support systems across provider roles. We recommend implementing a structured perioperative ultrasound curriculum featuring longitudinal training, faculty upskilling, improved equipment availability, and workflow integration. These steps will support broader adoption and competency in POCUS across anesthesiology practice. Future efforts should include surveying a wider population to validate these findings and inform scalable implementation strategies.

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SP 1 - Kiosk 4- Poster 7
A Knot Too Far: Transesophageal Echocardiography Identifies Unintended Aortic Valve Constraint
Presenting Author: *Vincent Franco, MD*

Introduction:Transesophageal echocardiography (TEE) is integral to modern cardiac surgery, providing real-time anatomic and functional assessment that directly influences intraoperative decision-making. While TEE is routinely used to confirm preoperative diagnoses and evaluate surgical repairs, its greatest value emerges when unexpected pathology appears after cardiopulmonary bypass (CPB). This case illustrates how comprehensive, systematic TEE evaluation identified a new, unanticipated valvular lesion following mitral valve repair, prompting immediate corrective intervention and preventing postoperative morbidity.

Case Report: A 70-year-old woman with severe mitral valve regurgitation due to ruptured P2 chordae underwent elective mitral valve repair. Following induction of anesthesia, TEE confirmed severe posterior leaflet prolapse without aortic valve pathology. After cannulation and initiation of CPB, the surgical team performed left atrial appendage amputation, triangular P2 resection, cleft closure, Gore-Tex neochord placement, and implantation of a 28-mm Physio ring. The patient was re-warmed, de-aired, and separated from CPB. Post-repair TEE demonstrated excellent mitral leaflet coaptation without residual regurgitation.
However, a new, moderate-to-severe aortic insufficiency (AI) was immediately identified on TEE—an unexpected finding given the absence of preoperative AI. Color doppler revealed an eccentric regurgitant jet originating from the noncoronary cusp. Given the timing and appearance, iatrogenic injury or annular impingement from a mitral annuloplasty suture was suspected. The surgical team reinitiated CPB and cross-clamped the aorta. Inspection via aortotomy confirmed a mitral annuloplasty stitch inadvertently placed at the non-coronary cusp, restricting leaflet mobility. The stitch was removed, restoring normal cusp motion. The aortotomy and atriotomy were re-closed, de-airing was completed, and the patient was weaned from CPB without difficulty. Final TEE demonstrated no residual aortic or mitral regurgitation. Post operatively, the patient was transferred to the ICU in stable condition and extubated on POD 1.

Discussion: This case highlights the indispensable role of comprehensive intraoperative TEE, not only in validating the mitral valve repair, but also in diagnosing unexpected complications. Systemic comprehensive TEE should be performed, including multiplane interrogation of valve anatomy, doppler evaluation of regurgitant severity, and systematic evaluation of iatrogenic causes such as stitch impingement, subvalvular entrapment, annuloplasty ring distortion, and

leaflet perforation. Early identification allowed intraoperative correction, avoiding postoperative hemodynamic instability, reoperation, and increased morbidity.

Conclusion: Comprehensive intraoperative TEE is essential for high-quality cardiac surgical care. In this case, meticulous post-repair TEE identified a new iatrogenic aortic insufficiency that was not clinically apparent. Prompt recognition and communication enabled immediate surgical correction and an optimal clinical outcome. This case reinforces that TEE must be systematic and vigilant to ensure safe and successful cardiac valve surgery.

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SP 1 - Kiosk 5- Poster 1

Intraoperative Coronary Sinus Injury: Case Series Review of Risk Factors and Transesophageal Echocardiogram Assessment

Presenting Author: *Quinton Riter, DO*

Introduction: Coronary sinus injury (CSI) is a rare but potentially life-threatening complication of retrograde cardioplegia (direct and indirect cannulation), with an incidence of 0.06–0.6%.(3) It is often caused by mechanical stress from high cardioplegia pressures, challenging or mispositioned cannula placement, balloon overinflation, or excessive cardiac manipulation. These mechanisms can tear, dissect, or perforate the compliant, low-pressure coronary venous sinus.(3) As cardiac anesthesiologists, prompt recognition and assessment of CSI is critical for guiding management. We present a case series of four intraoperative CSIs, highlighting key transesophageal echocardiography (TEE) findings and management considerations.

Case Series: All cases were performed under general anesthesia with TEE, arterial monitoring, and

central venous access. Retrograde cardioplegia cannulas (direct or indirect) were placed prior to initiation of cardiopulmonary bypass by 4 different surgeons. (For details, see table 1)

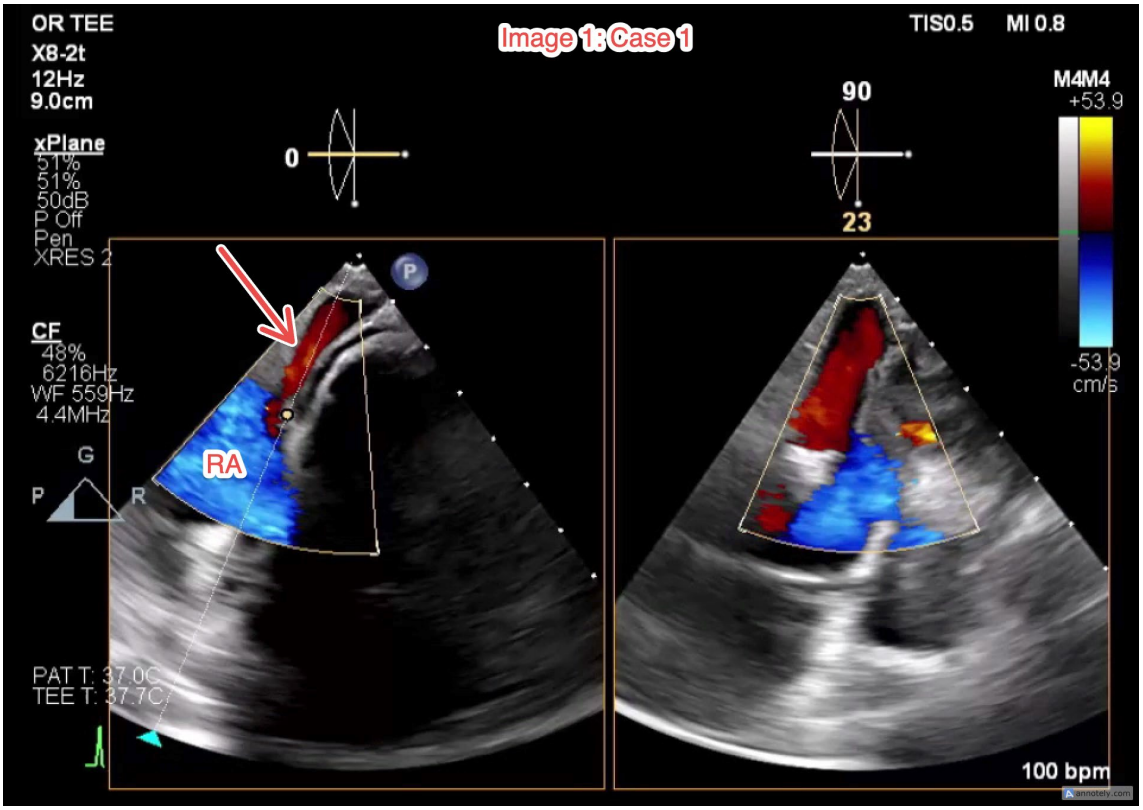
Discussion: CSI is an uncommon complication of retrograde cardioplegia cannulation. Risk factors include delivery pressures > 40 mmHg, difficult cannula placement, cannula malposition, operator inexperience, and excessive cardiac manipulation.(1,3) Although diagnosis may be challenging, early recognition is essential to guide timely management and prevent further morbidity. TEE aids in the detection of CSI and in assessing its hemodynamic significance by evaluating mitral inflow and pulmonary vein flow, identifying structural abnormalities, and monitoring interval changes—particularly after correction of coagulopathy. In parallel, continuous vigilance for increased surgical bleeding, acute tamponade, conduction disturbances, and hemodynamic instability is warranted, as these may indicate ongoing coronary sinus disruption, atrial ventricle groove instability, expanding hematoma formation, or development of false lumens. The coronary sinus is visualized with the TEE mid esophageal (ME) 4 chamber, ME 2 chamber, and modified bicaval views which can also be used to guide cannula placement. The ME 4 chamber, ME commissural, ME 2 chamber, and ME aortic long axis assess for encroachment on left atrium (LA), LA collapse, and pseudo-mitral stenosis physiology which may be the first indications of CSI.(2) Evaluation of lesion progression, injury location, and coronary sinus patency guides management decisions. Mitral inflow obstruction, hemodynamic instability, or an expanding hematoma may warrant surgical repair. Coronary sinus injuries can be managed with direct suturing, hemostatic packing agents, or venous/pericardial patch techniques, though repair is often challenging given the coronary sinus' posterior location.(1) Stable abnormalities from CSIs may be managed conservatively with serial monitoring. Treatment centers on correcting coagulopathy to promote thrombosis within the low-pressure system and on careful preload optimization. Adequate preload helps maintain LA pressure and prevents hematoma-related LA collapse and functional mitral stenosis, whereas excessive preload may worsen coronary sinus dilation or propagate LA dissection.(1)

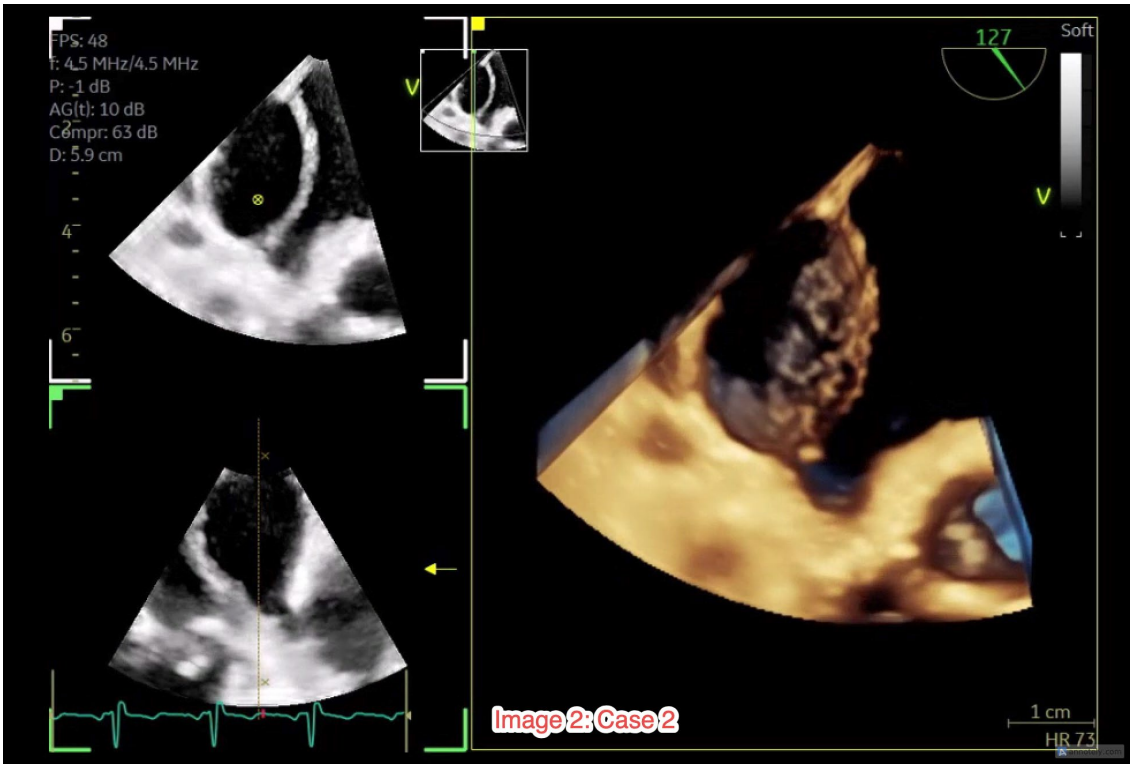
Conclusion: TEE allows for prompt recognition and tailored management of CSI resulting in improved patient care.

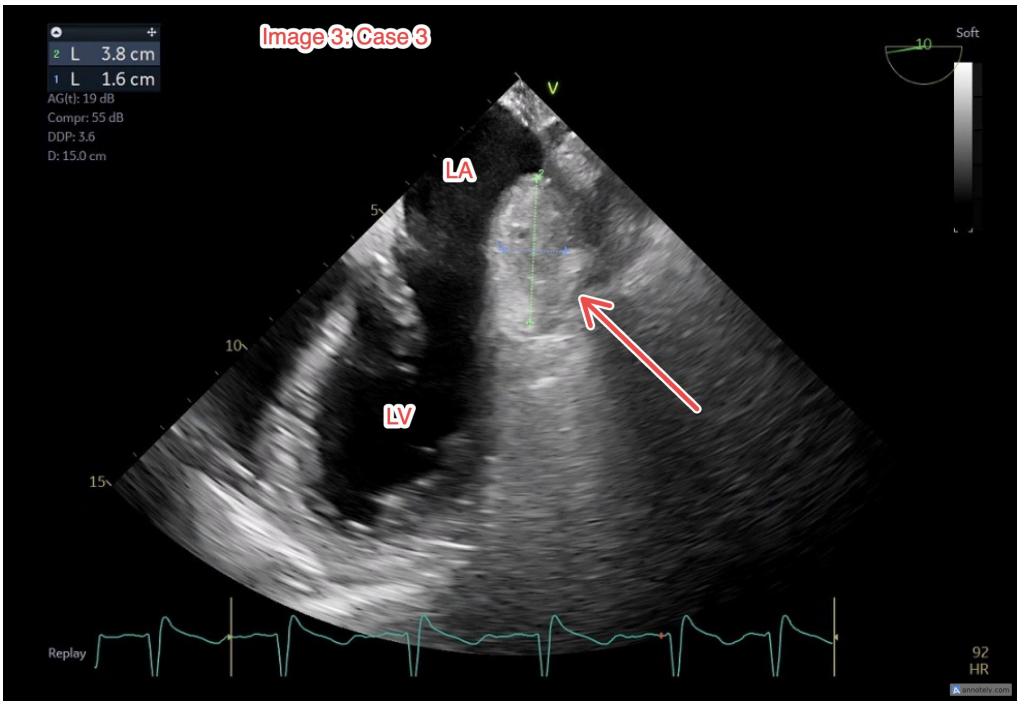
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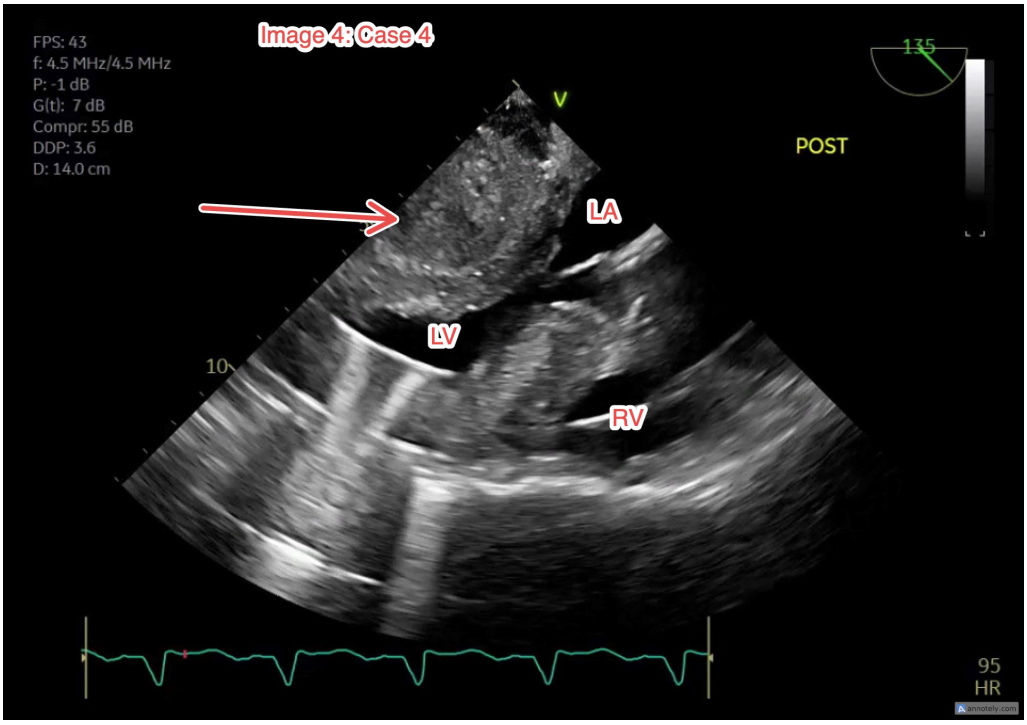
Table 1: Summary of Cases.	Column1	Column2	Column3	Column4	Column5	Column6	Column7	Column8	Column9
	Age	Comorbidities	Surgery	CPB time	Method of Cardioplegia	Main TEE finding	Further Evaluation of CSI w/w.o TEE	Follow Up Echocardiography Results	Special Considerations
Case 1	72yoF	Hodgkin lymphoma s/p radiation c/b radiation induced heart disease with moderate AI/AS, moderate-severe MR with mild stenosis, and moderate TR	AVR, MVR, TVr	123 minutes	Antergrade and direct retrograde	Hematoma noted tracking along inferior and lateral walls of left atrium without expansion after protamine	(1) No mitral inflow obstruction (2) The visual component of the hematoma appeared decreased in size after protamine & chest closure with no wall motion abnormalities on TEE	Hospital course complicated by problems associated with patient's radiation history including dysphagia (EGD, corpak) and AFib eventually requiring PPM. Follow up POD8 echo showing resolution of hematoma.	Surgery noted as complex due to radiation heart disease with calcification of aortomitral curtain
Case 2	71yoF	MI s/p PCI to LAD (1/2019) and distal ramus, LCx (1/2021), HTN, HLD, Postop AF (Self converted)	CABG (LIMA-LAD, SVG-PDA, SVG-Ramus)	89 minutes	Antergrade and indirect retrograde	Bi-directional flow between the RA and false lumen located in posterior LA following CPB, most likely due to coronary sinus injury induced LA dissection	(1) No mitral inflow obstruction (2) No hemodynamic instability following cessation of CPB (3) False lumen initially originating from posterior wall of the LA, which expanded anteriorly despite protamine and chest closure	Discharged POD5. POD12 TTE no signs of residual LA dissection secondary to coronary sinus injury	[1] Surgical team noted resistance with placing the retrograde cannula. A total of 3 attempts were made for final placement [2] Manipulation of the heart to access the posterior coronaries for grafting may facilitate retrograde cannula migration/misplacement
Case 3	70yoF	Thoracic aortic aneurysm, Afib and MV prolapse	David procedure, b/l PVI & LAAC	153 minutes	Antergrade and indirect retrograde	Large hypoechoic cavity in the posterolateral left atrial wall, which reduced in size after discontinuation from CPB. A low velocity flow was noted in the cavity. The hypoechoic cavity measured 3.5 cm X 1.1 cm	(1) No mitral inflow obstruction (2) No longer flow through the false lumen which eventually clotted off after protamine administration	Discharged POD7. POD4 TTE no signs of false cavity with flow	
Case 4	75yoM	HFrEF (LVEF 20%), CAD with MVD, pAF/AFL s/p failed ablation (Eliquis), HTN, HLD, Hypothyroidism, and CKD	HM3 LVAD placement, CABG, MAZE, and LAAL	131 minutes	Antegrade and direct retrograde	Following cessation of CPB, LA & LV noted to be compressed by a coronary sinus hematoma but with adequate HM3 flow. No mitral inflow obstruction.	Lateral wall hematoma decompressed and closed with interrupted pledgeted sutures. No mitral inflow obstruction.	Postoperative course c/b PDA laceration which was repaired. POD14 TTE noting residual lateral wall hematoma indenting into the LV cavity without hemodynamic effects.	(1) Manipulation of the heart to access the posterior coronaries for grafting may facilitate retrograde cannula migration/misplacement (2) Hematoma noted on postoperative TTE may be confounded by PDA laceration but nonetheless, no hemodynamic consequences from the hematoma.

Abbreviations: s/p- status post, c/b- complicated by, MVD- mitral valve disease, AF-atrial fibrillation, AFL- atrial flutter, PVI- pulmonary vein isolation, LAAC- left atrial appendage clip, CPB- cardiopulmonary bypass, HM3- Heartmate 3, POD- post operative day




FPS: 48
T: 4.5 MHz/4.5 MHz
P: -1 dB
AG(t): 10 dB
Compr: 63 dB
D: 5.9 cm
127
Soft
Image 2: Case 2
1 cm
HR 73


Image 3: Case 3
L 3.8 cm
L 1.6 cm
AG(t): 19 dB
Compr: 55 dB
DDP: 3.6
D: 15.0 cm
Soft
LA
LV
Replay
92
HR



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SP 1 - Kiosk 5- Poster 2

Parallel Extracorporeal Membrane Oxygenation Support During Lung Transplantation

Presenting Author: *Camila Magalhaes Funatsu, MD*

Introduction: Extracorporeal membrane oxygenation (ECMO) has evolved from an experimental therapy in the 1970s into a cornerstone of support for refractory respiratory and circulatory failure. Advances in circuit technology and physiology have expanded its use, particularly in lung transplantation, where ECMO serves as both a bridge to transplant and a means of perioperative

support. Standard veno-venous (VV) or veno-arterial (VA) ECMO may be insufficient in patients with severe right ventricular dysfunction or intolerance of single-lung ventilation. Hybrid and parallel ECMO circuits have emerged as innovative strategies to optimize flow and oxygen delivery when conventional configurations are inadequate.

Case Report: A 65-year-old man with progressive pulmonary hypertension, suspected pulmonary veno-occlusive disease, and chronic hypoxemic respiratory failure was transferred for lung transplant evaluation. Despite prior trials of pulmonary vasodilators, his respiratory status worsened with escalating oxygen needs and right ventricular (RV) failure.
On admission, he required high-flow oxygen, inhaled nitric oxide, and multiple vasopressors. Echocardiography showed preserved LV function (EF 55–60%) with severe RV dilation and dysfunction, moderate tricuspid regurgitation, and pulmonary artery systolic pressure of 77 mmHg. Due to worsening hypoxemia, VV ECMO was initiated via a left subclavian bicaval dual-lumen cannula. As RV failure advanced, he was transitioned to veno-venoarterial (V-VA) ECMO with the addition of a femoral arterial cannula.

The patient remained ECMO-dependent with renal replacement therapy for volume control. Right and left heart catheterization confirmed severe pulmonary hypertension and mild coronary artery disease. He was listed for bilateral lung transplantation.

Intraoperatively, suprasystemic pulmonary pressures prevented tolerance of single-lung ventilation. A parallel ECMO strategy was employed: central VA ECMO (aortic and right atrial cannulation) was added to the peripheral circuit, achieving flows of 3.0–3.5 L/min, controlled reperfusion, and adequate oxygenation. After implantation, the central circuit was decannulated, while peripheral ECMO was maintained. Postoperatively, femoral arterial decannulation with tracheostomy occurred on day 6, and VV ECMO decannulation on day 9.

Discussion: This case demonstrates the utility of parallel ECMO in lung transplantation complicated by pulmonary hypertension and RV failure. Conventional VV or VA ECMO may be insufficient in these settings. By combining peripheral and central support, parallel circuits can stabilize hemodynamics, optimize flows, and ensure controlled graft reperfusion.
Conclusion Parallel ECMO strategies provide an important tool in complex lung transplantation cases where standard support is inadequate. Their use should be tailored to patient physiology and guided by a multidisciplinary team, recognizing the associated risks and technical challenges.

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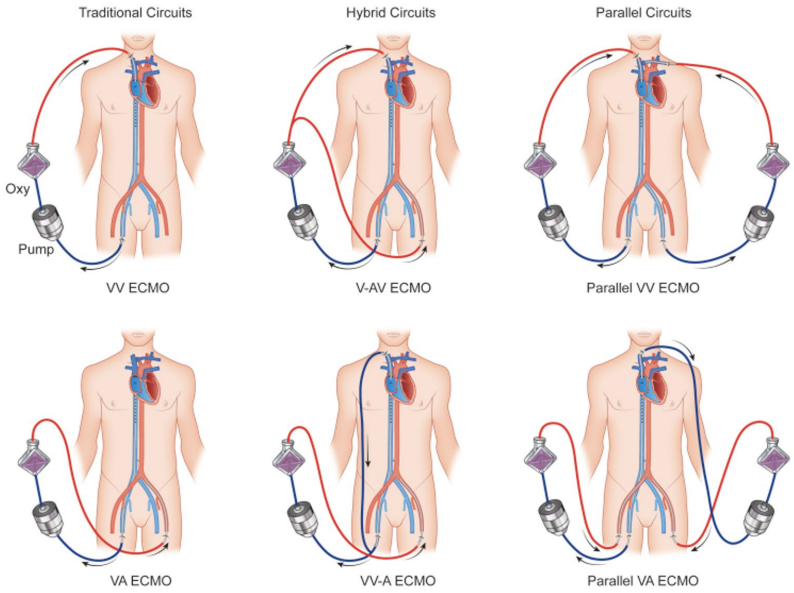


Fig 1. - Various extracorporeal membrane oxygenation configurations including standard, hybrid and parallel circuits (Hybrid and parallel extracorporeal membrane oxygenation circuits. Shah, Aakash et al. JTCVS Techniques, Volume 8, 77 – 85)

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SP 1 - Kiosk 5- Poster 3
Mass Exodus: A 3-Year Case Review of Thrombus And Vegetation Extraction with an On-Circuit Mechanical Aspiration System: Indications, Anesthetic Management, Procedural Success, and Outcomes
Presenting Author: *Jeremy Tang, MD*

Introduction: Intracardiac masses present major anesthetic challenges due to embolic potential, hemodynamic instability, and associated sepsis or coagulopathy. Vegetations >10 mm are associated with significantly increased embolization and mortality, emphasizing the importance of timely intervention and meticulous perioperative planning. An on-circuit mechanical aspiration system (AngioVac, Angiodynamics, Latham, NY) offers a minimally invasive alternative to open surgery for their extraction; however, anesthesia-specific guidance remains limited in the literature. This case series describes anesthetic strategies, physiologic perturbations, and perioperative outcomes during on-circuit aspiration of right- and left-sided intracardiac masses.

Case Presentation: Over a 3-year period, 14 patients underwent on-circuit aspiration. Across the cohort, 15 masses were categorized as intracardiac thrombi, or infectious masses Mass locations included the tricuspid valve (40.0%), right atrium (26.7%), mitral valve (20.0%), left atrium (6.1%), and right ventricle (6.1%). Thrombus size was documented in 10 patients, with a median dimension of 2.85 cm (range 1.8–6.1 cm).

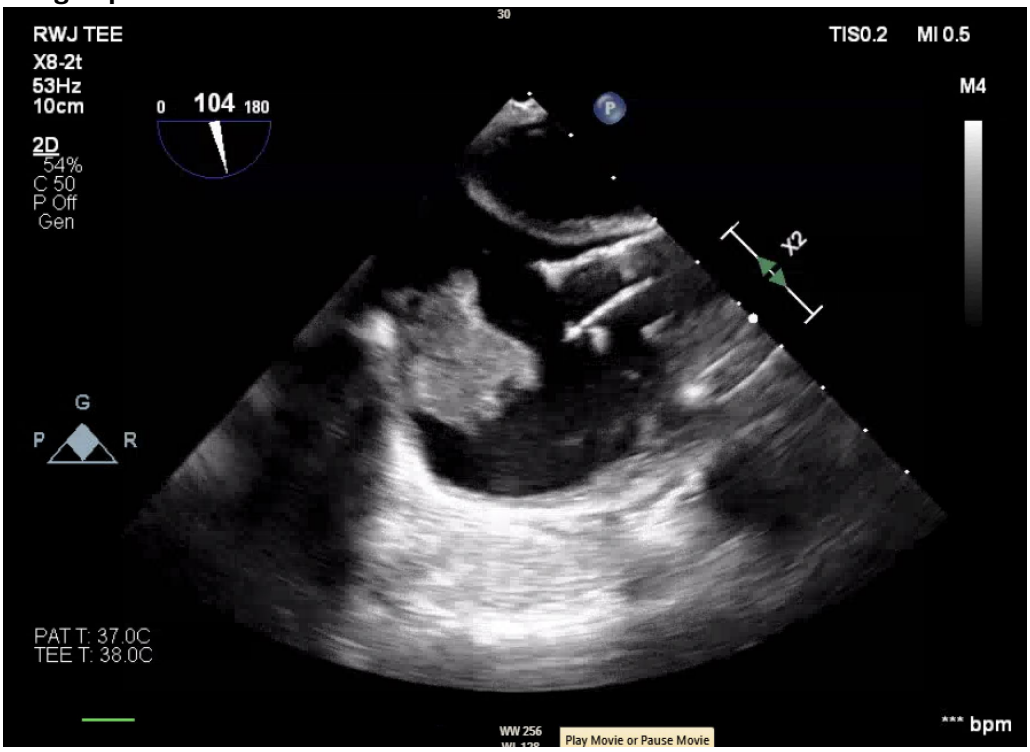
Anesthetic induction posed substantial risk: one patient (7.1%) experienced cardiac arrest at induction, consistent with preload dependence and potential RV inflow obstruction by the mass. Fifty percent of patients required vasoactive infusions, particularly during cannulation and initiation of high-flow suction, which often reduced venous return. Transesophageal echocardiography (TEE) was essential for characterizing masses, guiding the cannula, and evaluating for residual clot.

Perioperative complications occurred in 28.6% of patients, including thrombus embolization, cardiac arrest, and postoperative neck swelling, with no case requiring conversion to open surgery. Post-procedure echocardiography demonstrated complete mass removal in 10 patients, partial removal in 2, and no intervention in 2. Four patients (28.6%) received perioperative transfusions, and 3 (21.4%) required continuation of vasoactive infusions postoperatively. Eleven patients (78.5%) were extubated in the operating room, while the remaining patients were extubated at a mean of 16.5 hours postoperatively. Procedural success—defined as documented removal or absence of conversion with survival beyond 30 days and symptom resolution—was 85.7%. Two patients (14.3%) experienced 30-day mortality. Median ICU and hospital lengths of stay were 5 and 19 days, respectively, with overall survival of 71.4% at study completion.

Discussion: This case series highlights the anesthesia-specific complexity of on-circuit aspiration of intracardiac masses. Key considerations include: (1) risk-stratified induction in patients with preload depend

hemodynamic shifts requiring early vasopressor readiness; and (3) pathology-specific management, as vegetation/endocarditis cases often involve sepsis and RV dysfunction, whereas thrombotic cases reflect coagulopathy or inadequate anticoagulation. These findings underscore the importance of proactive hemodynamic planning and tailored anesthetic strategies in patients undergoing intracardiac on-circuit aspiration procedures.

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SP 1 - Kiosk 5- Poster 4

When Heparin Becomes a Hazard: Perioperative Management of Alpha-Gal Syndrome in Cardiac Surgery

Presenting Author: *Iqbal Khan, MD*

Background: Alpha-gal syndrome is an allergy that has garnered increasing attention due to its delayed hypersensitivity response to galactose-α-1,3-galactose, a carbohydrate found in non-primate mammalian tissues. Frequently induced by the bite of the Lone Star tick, the condition is particularly relevant in perioperative care due to the presence of mammalian-derived products in multiple anesthetic agents and medications, including heparin. Given the delayed onset and unnoticed sources of exposure, anesthetic management of sensitized patients presents a significant safety challenge, particularly for those undergoing cardiac surgery.

Case Report: A 69-year-old male with coronary artery disease, paroxysmal atrial fibrillation, and type 2 diabetes was scheduled for elective CABG with possible maze and left atrial appendage clipping. Earlier that spring, after multiple Lone Star tick bites, he developed delayed vomiting, diarrhea, and hives after eating beef on two occasions. Allergy testing confirmed alpha-gal syndrome with IgE α-gal >100 kU/L. Preoperative evaluation showed a Mallampati III airway, atrial fibrillation, EF 40% with global hypokinesis, and 100% proximal LAD occlusion with collateral flow. Because cardiac surgery requires high-dose heparin and many perioperative agents contain mammalian-derived components, a multidisciplinary team created a modified plan. The Vanderbilt desensitization protocol (1300 units over 4.5 hours) was extended to a 24-hour ICU regimen with continuous monitoring. The patient received scheduled diphenhydramine and prednisone. Vasoactive infusions were mixed only in preservative-free saline, and pre-mixed medications were avoided. Induction was performed via inhalation with norepinephrine support. Lipid-based IV agents such as propofol were avoided. Two central lines and a pulmonary artery catheter were placed, and two units of autologous blood were collected before bypass. Protamine remained available for reversal. The operation was uneventful. The patient tolerated systemic heparin without anaphylaxis, separated from bypass with minimal inotropes, and required only autologous transfusion. Recovery was stable, and he remains well nearly one year later.

Discussion: This case highlights the critical need to recognize and accommodate alpha-gal allergy in the surgical setting. The hidden presence of mammalian-derived ingredients in medications and equipment necessitates proactive identification and substitution. While some studies suggest that alpha-gal–sensitized patients may tolerate intravenous heparin following pre-treatment, risks remain, particularly in those with elevated IgE titers. Establishing institution-specific perioperative protocols and conducting detailed allergy histories are vital steps in improving patient safety.

Conclusion: Alpha-gal syndrome presents a unique and growing challenge in anesthetic and surgical practice. This case demonstrates how a collaborative, preemptive strategy—including desensitization, excipient auditing, and alternative medication preparation—can enable safe surgical outcomes in high-risk patients. Increased awareness and protocol development will be essential as incidence of this condition continues to rise.

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SP 1 - Kiosk 5- Poster 5
Managing Catastrophic Pulmonary Artery Injury During Re-do Left Pneumonectomy Without Cardiopulmonary Bypass
Presenting Author: *Shriya Sharma, MD*

Introduction: Pulmonary artery injury is the most feared intraoperative complication during anatomic pulmonary resection, with reported incidence of 0.5–2.9% in minimally invasive lobectomies. Risk factors include prior thoracic surgery, induction chemotherapy, and dense hilar adhesions. Emergent conversion to thoracotomy is often required to control hemorrhage, preserve lung parenchyma, and maintain patient survival, posing significant anesthetic and cardiovascular challenges.

Case Presentation A 73-year-old male with prior myocardial infarction, early heart failure, prior left upper lobe wedge resection, mediastinal lymphadenectomy, and 50 pack-year smoking history underwent planned robotic left upper lobectomy for recurrent invasive adenocarcinoma after neoadjuvant chemotherapy. Preoperatively, an erector spinae plane block and arterial line were placed; anesthesia was induced with a 39 Fr double-lumen endotracheal tube.

Extensive adhesions were encountered robotically, requiring 6 hours of lysis and lymphadenectomy. Dense hilar adhesions obscured the left pulmonary artery and superior vein, causing minor bleeding and necessitating conversion to open thoracotomy.

During open dissection, proximal control of the left pulmonary artery and vein was extremely challenging, with a near-tear of the pulmonary artery occurring while attempting to divide an aberrant hilar vessel. The cardiac surgery team was immediately available and repaired the

vascular injury with running prolene sutures. Throughout the event, the patient remained hemodynamically stable with minimal vasopressor support, despite 2 L estimated blood loss and ongoing transfusion (6 units pRBC, 2 units platelets).

After successful vascular repair, the left pneumonectomy was completed with meticulous bronchial stapling under bronchoscopy guidance. Hemostasis was confirmed, and a 24 Fr chest tube was placed. The patient was extubated on postoperative day 1 with stable renal function after fluid resuscitation for rhabdomyolysis from prolonged lateral decubitus positioning. Postoperatively, he experienced brief episodes of asymptomatic atrial flutter managed with metoprolol and anticoagulation.

Discussion: Pulmonary artery injuries are recognized as the leading cause of urgent conversion to thoracotomy during minimally invasive pulmonary resections. Blunt and sharp dissection, prior chemotherapy, and large tumors increase risk. In this case, extensive adhesions and prior surgery created an exceptionally high-risk scenario. Emergent conversion to open thoracotomy allowed precise vascular control, avoiding cardiopulmonary bypass, which is associated with transitory immunologic and hemodynamic perturbations. The off-pump approach minimized additional cardiac stress while permitting definitive management of life-threatening hemorrhage. Multidisciplinary coordination between anesthesiology, thoracic surgery, and cardiac surgery was critical, highlighting the anesthesiologist's role in maintaining hemodynamic stability and facilitating complex vascular repair.

Conclusion: This case demonstrates that even catastrophic pulmonary artery injury can be managed safely without cardiopulmonary bypass, emphasizing the critical role of anesthesiologists in complex thoracic procedures.

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SP 1 - Kiosk 5 - Poster 6

Hybrid Repair of Degenerative Mitral Regurgitation: A Case Report

Presenting Author: *Thalia Casanova, BS*

Introduction: Degenerative mitral regurgitation (MR) is the leading indication for mitral valve repair (MVr) and most often results from P2 dysfunction. Large, myxomatous P2 segments with long anterior leaflets present a repair challenge because neochord-only or full-resection

techniques may increase the risk of systolic anterior motion (SAM), restrict mobility, or require a smaller annuloplasty ring. We present two cases where a hybrid repair strategy was selected to optimize valve geometry and minimize complications.

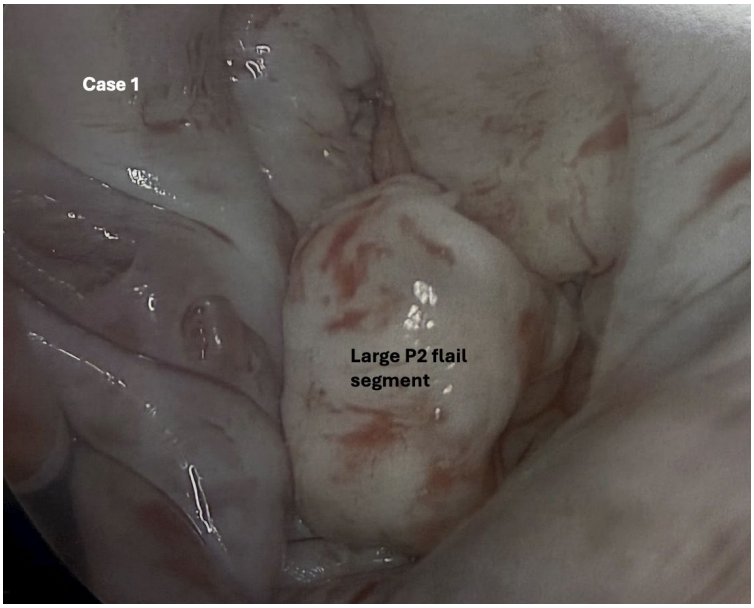
Case Report: Case 1: A 68-year-old male with acute diastolic heart failure presented with respiratory failure secondary to severe MR. Echocardiography demonstrated a flail P2 scallop, severe MR, and preserved systolic function (EF 70%). Hemodynamics showed pulmonary hypertension (PA 74/23 mmHg), wedge pressure 23 mmHg, and a cardiac index of 1.5 L/min/m2. Coronary angiography revealed mild non-obstructive disease.
Case 2: A 50-year-old male with ventricular fibrillation, atrial fibrillation, and multiple prior critical conditions was found to have severe MR. Echocardiography showed a flail P2 with ruptured chordae, EF 60–65%, and severe left atrial enlargement. Hemodynamics revealed right atrial pressure 1 mmHg, PA pressure 23/6 mmHg, wedge pressure 9 mmHg, and a cardiac index of 2.0 L/min/m2. Coronary angiography showed no coronary disease. NT-proBNP was mildly Elevated.

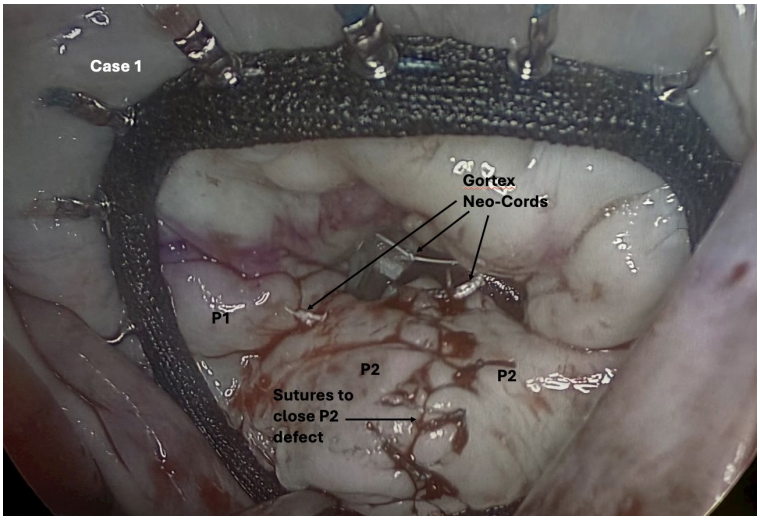
Both patients underwent MVr via median sternotomy with bicaval cannulation and Del Nido cardioplegia. Intraoperative inspection confirmed enlarged, myxomatous P2 scallops. A hybrid repair was performed consisting of limited P2 resection, reconstruction, six Gore-Tex neochords, and large annuloplasty rings (38-mm for Case 1 and 42-mm for Case 2). Case 2 also required atrial fibrillation ablation. Cardiopulmonary bypass and cross-clamp times were 101/78 minutes for Case 1 and 116/81 minutes for Case 2. Postoperative TEE in both cases showed trace MR (3 mm Hg and 2 mm Hg), excellent coaptation, and low gradients without SAM or obstruction. Each patient had an uneventful recovery.

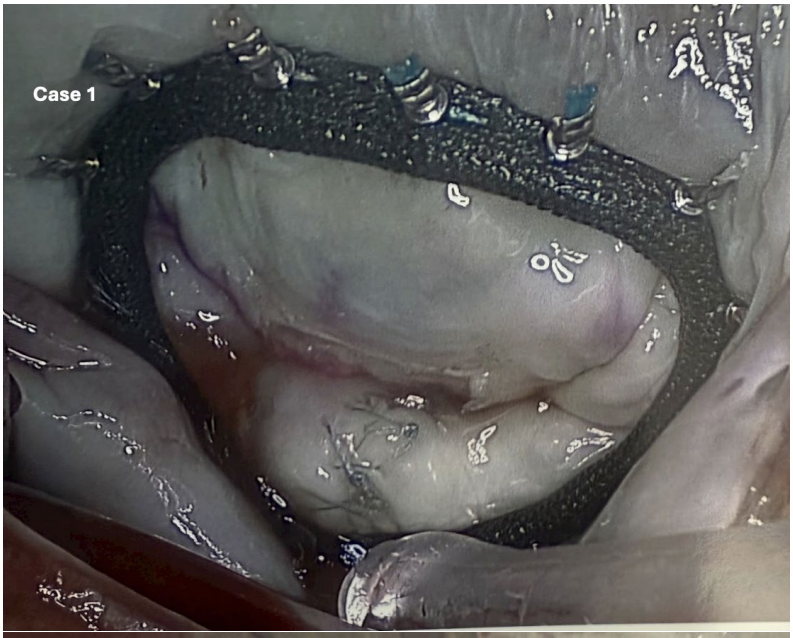
Discussion; These cases highlight the importance of adapting MVr to anatomy rather than one technique. Both patients had large P2 scallops with elongated anterior leaflets, increasing SAM risk. A neochord-only strategy would shift the coaptation line anteriorly, whereas very short chords leave excess bulk and raise gradients. Full quadrangular resection with sliding plasty would shorten the posterior leaflet, reduce mobility, and likely require a smaller ring. The hybrid approach of limited P2 resection
with neochordal reconstruction addressed these challenges by removing tissue while preserving mobility, allowing the coaptation zone to remain posterior and enabling use of a larger ring.

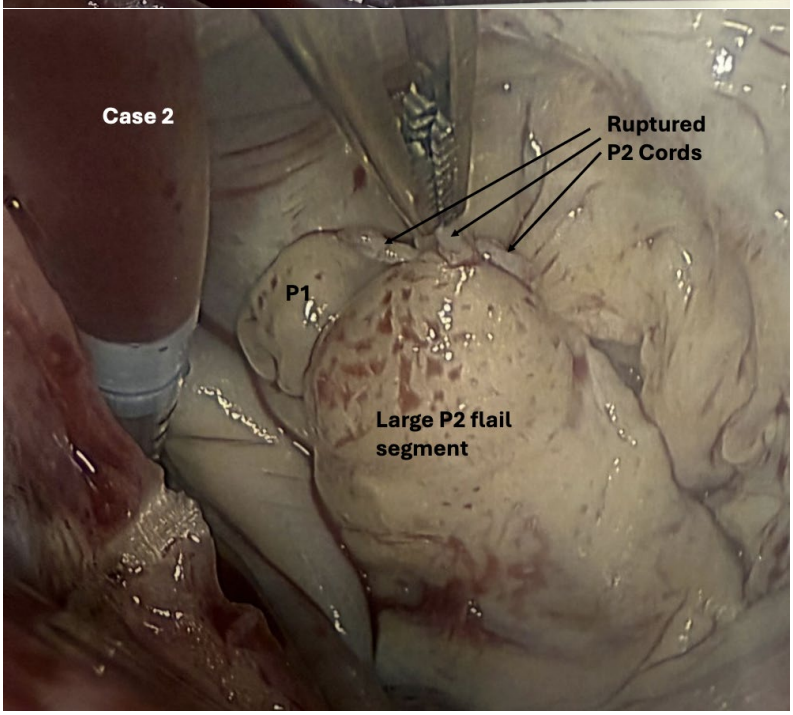
Conclusion: Hybrid P2 repair combining limited resection with neochordal reconstruction is an effective strategy for large, myxomatous P2 lesions. This approach preserves mobility, minimizes SAM risk, and supports use of a larger annuloplasty ring. These cases illustrate a reproducible technique for anatomically complex degenerative MR where neither resection-only nor neochord-only methods are optimal.

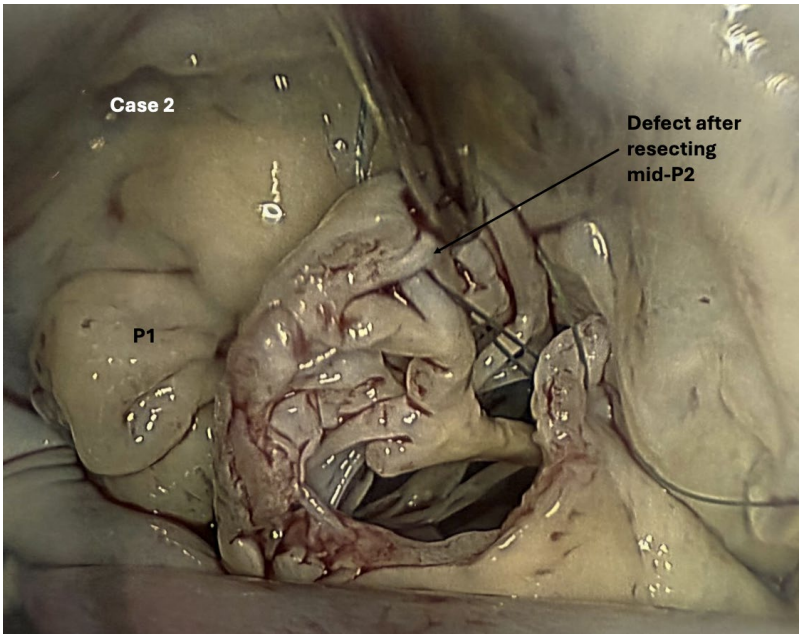
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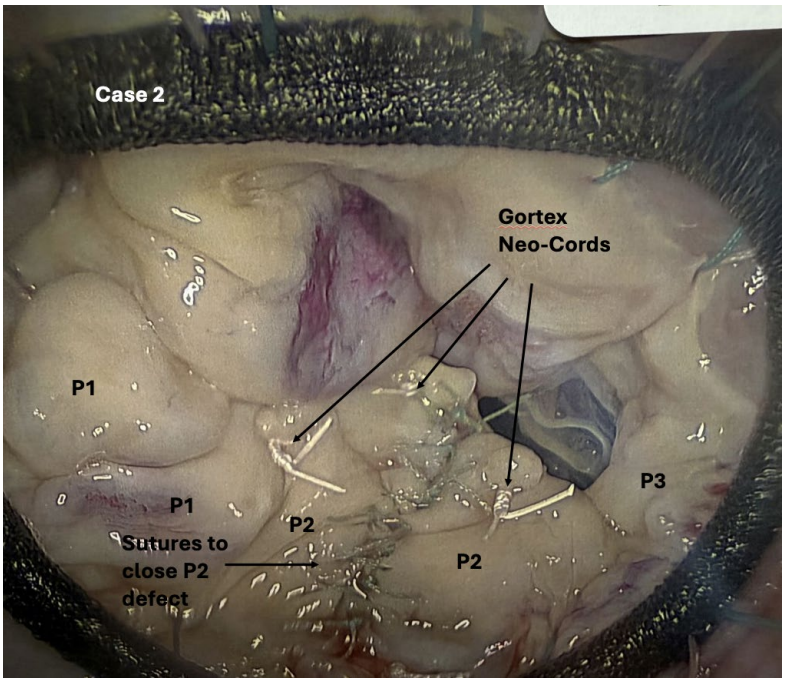

Case 1
Large P2 flail segment


Case 1
Gortex
Neo-Cords
P1
P2
P2
Sutures to close P2 defect


Case 1


Case 2
Ruptured
P2 Cords
P1
Large P2 flail
segment





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SP 1 - Kiosk 5 - Poster 7
The SVC Squeeze: A Case of Acute Intraoperative Superior Vena Cava Syndrome During Cardiac Surgery
Presenting Author: *Yannan Huang, MD*

Introduction: Superior vena cava (SVC) syndrome is a rare complication of cardiac surgery caused by vessel compression or occlusion. Typical presentation is subacute, occurring in the weeks after initial surgical intervention. Here, we present a unique case of acute intraoperative SVC syndrome secondary to stenosis from overlying SVC cannulation site sutures.

Case Report: A 76 year old male underwent bioprosthetic mitral valve replacement, MAZE procedure, left atrial appendage clip, and posterior pericardial window. Preoperative TEE demonstrated normal biventricular function and myxomatous degeneration of the mitral valve with bileaflet prolapse causing moderate to severe mitral regurgitation. Bicaval cannulation was performed for optimal visualization of the mitral valve. Overall OR course was uneventful until sternotomy closure when the patient developed profound hypotension refractory to increasing vasopressor and inotropic support as well as increased CVP from 18 to 53 mmHg. The patient was also noted to have new onset facial edema with a plethoric cyanotic appearance. Intraoperative TEE ME bicaval view showed turbulent flow pattern (fig 1) with concern for acute SVC syndrome secondary to stenosis at the prior SVC cannulation site. Release of overlying sutures under TEE guidance led to immediate improvement of CVP and return of laminar flow (fig 2). However, subsequent significant bleeding from the SVC cannulation site and left atrial suture line necessitated emergent return to cardiopulmonary bypass and bovine pericardial patch repair. The case was further complicated by vasoplegia and coagulopathy requiring significant vasoactive support and extensive blood product transfusion. Ultimately, adequate hemostasis was achieved and the patient was transferred to the cardiothoracic intensive unit for recovery. Post-operative course was complicated by complete heart block requiring permanent pacemaker placement. The patient was discharged home on POD 8 without further issues.

Discussion: In SVC syndrome, inhibition of venous return from vessel obstruction or compression can lead to life-threatening hemodynamic instability. Common causes include mediastinal malignancies and vessel thrombus or stenosis from semi-permanent intravascular catheters/devices. Onset post cardiac surgery is typically gradual and insidious. Symptoms include dyspnea, facial swelling, neck vein distention, and headache which can progress to respiratory failure, obstructive shock, and coma from cerebral edema. While acute intraoperative development is rare, this patient's objective findings of new onset facial plethora, acutely rising CVP, and refractory hypotension were consistent with obstructive shock secondary to decreased venous return. TEE demonstrating turbulent flow within the SVC confirmed our suspicions. Discussion with the surgical team revealed the cause to be oversuturing at the SVC cannulation site due to initial tissue friability with resolution following prompt surgical intervention.

Conclusion: Acute SVC syndrome during cardiac surgery is rare and clinically challenging to

diagnose, especially given the lack of subjective symptoms in an anesthetized patient. This case highlights how timely recognition and prompt treatment can greatly improve patient outcomes.

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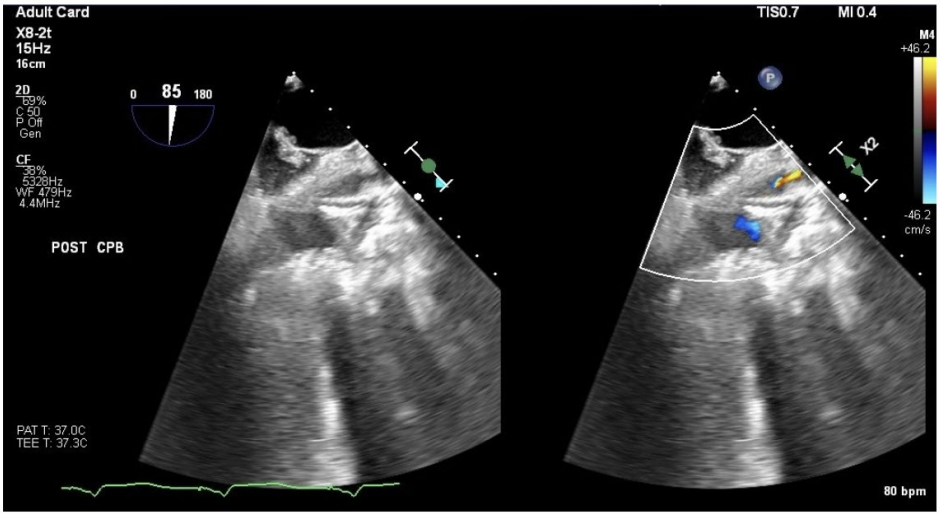


Figure 1. TEE midesophageal bicaval view with color flow mapping showing turbulent flow in the SVC

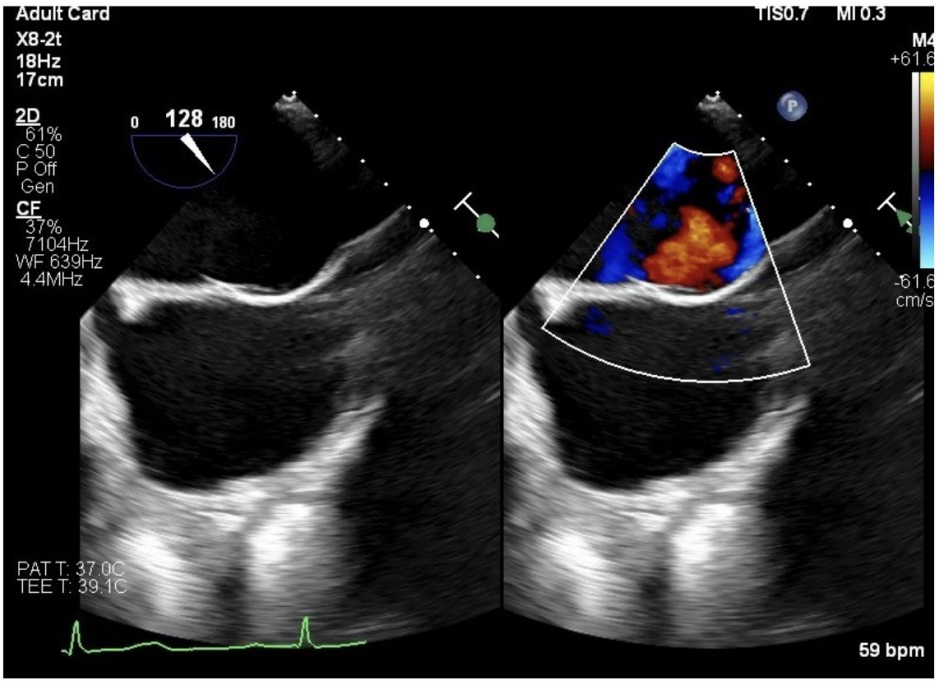


Figure 2. TEE midesophageal bicaval view with color flow mapping showing return of laminar flow in the SVC after suture release

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intraoperative acute superior vena cava syndrome." Annals of Cardiac Anaesthesia 16.2 (2013): 133-136.

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SP 1 - Kiosk 6 - Poster 1
Towards Earlier Detection of Hyperfibrinolysis by ROTEM
Presenting Author: *Andrew Milewski, MD, PhD*

Introduction: Hyperfibrinolysis (HF)—the excessive and premature breakdown of blood clots—increases the numbers of blood products needed to treat bleeding patients and increases mortality1-3. Early antifibrinolytic (AF) therapy reduces these risks, but only when given early4,5. Although viscoelastic testing (VET) permits rapid, targeted identification of specific coagulopathies, HF—when it occurs—typically becomes apparent >30min after starting the test. In this time, patients may lose large volumes of blood despite receiving cryoprecipitate, platelets, and plasma. In this preliminary study, we investigate the time needed for VET to identify HF and explore EXTEM-HEPTEM intersections (EHIs) as a potential early predictor of HF.

Methods: The dataset comprised rotational thromboelastometry (ROTEM) tests performed from 3/2024-8/2025 on four ROTEM sigma devices at two independent academic medical centers. HF was defined as a decrement of ≥15% in the EXTEM curve from its maximum value within 60min of the clotting time (CT) measurement plus a decaying or flat FIBTEM. EXTEM CT is shorter than HEPTEM CT. The EXTEM curve thus rises and falls earlier than the HEPTEM curve when HF occurs, and the two curves should therefore intersect. Three timepoints in the EHI process were examined. 1. Approaching intersection: time that EXTEM and HEPTEM came within 1mm of each (-1mm timepoint). 2. Intersection: time that EXTEM and HEPTEM had equal amplitudes (0mm). 3. After intersection: time that HEPTEM rose to ≥1mm above EXTEM (1mm). Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated for the entire dataset and in a subset of curves obtained for patients undergoing open repairs of thoracoabdominal aortic aneurysms (ORTAAAs). Time savings were calculated as the difference between the 1mm, 0mm, and -1mm times and the times at which the standard definition for HF was met.

Results: HF occurred in 2.5% (56/2,250) of ROTEM tests and in 15% (17/113) of the ORTAAA tests. The standard HF criteria were met over a broad range of times (8.0-58.4min, median 36.4min, IQR [23.5-47.6]). EHIs were sensitive (100%) and specific (80% for -1mm, 94% for 0mm, and 97% for 1mm) for predicting HF. The PPV improved as the curves approached, crossed, and continued to separate (11% for -1mm, 30% for 0mm, and 43% for 1mm) and the NPV was 100% for each case.

The sensitivity, specificity, and NPV were similar for the ORTAAA tests, but the PPV was higher: 49% (-1mm), 81% (0mm), and 94% (1mm). The -1mm timepoint
was reached at a median of 25.8min (IQR [16.1,37.0]), 0mm at 33.1min (21.1,41.5), and 1mm at 35.5min (22.1,44.7). The average time savings were: 9.3min (95% CI [6.5,12.1]) at -1mm timepoint, 4.9min ([2.9,6.8]) at 0mm, and 3.0 min ([1.6,4.3]) at 1mm.

Conclusions: EHIs are highly sensitive for predicting HF. The PPV of EHIs are low in populations with low HF prevalence but higher in populations, like ORTAAs, with higher HF prevalence. Also, greater prediction accuracy was achieved at the expense of time savings. EHIs can yield clinically meaningful (>10 min) time savings. These preliminary findings suggest that early curve features can predict HF. Additional studies are needed to identify and test other early indicators for HF.

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SP 1 - Kiosk 6 - Poster 2
Dabigatran is not inferior to Heparin in Inhibiting Clot Propagation and Strength.
Presenting Author: *Michael Eaton, MD*

Background: Bivalirudin is a commonly recommended alternative to heparin for anticoagulation for

cardiopulmonary bypass (CPB) in patients with heparin-induced thrombocytopenia (HIT). However, it has been shown that while bivalirudin delays clot initiation, it does not inhibit clot propagation or clot strength (1) and thus may put patients at risk for thrombosis during CPB. Dabigatran is another direct thrombin inhibitor that has shown promise as an anticoagulant in animal models of CPB. (2,3) It is unknown how dabigatran affects clot propagation and strength,

Methods: 10 consenting volunteers provided fresh whole blood specimens that were aliquoted and spiked with Heparin (5 U/mL), Bivalirudin (15 µg/mL), or Dabigatran (2.4 and 5 ng/mL), or Dabigatran (2400 ng/mL). Tissue-factor stimulated thromboelastograms (rapidTEG,) were run in duplicate on the TEG 5000 analyzer. Markers of clot propagation (angle and maximum rate of thrombus generation MRTG) and clot strength (Maximum Amplitude, MA) were compared between the different anticoagulants.

Results and Conclusions: In a non-inferiority analysis, using a 10% difference threshold, Dabigatran 2.4 mcg/ml and 5 mcg/ml were non-inferior to heparin 5 units/ml) in decreasing propagation. Dabigatran 5mcg/ml was non-inferior to heparin 5 u/ml in decreasing clot strength. Using paired t-tests, dabigatran 2.4 mcg/ml and 5mcg/ml were superior to heparin 5u/ml and bivalirudin in inhibiting clot propagation, and dabigatran 5 mcg/ml was superior to heparin 5 u/ml in inhibiting clot strength. Dabigatran may be a superior substitute for heparin anticoagulation for CPB in patients with contraindications to heparin such as HIT.

References

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SP 1 - Kiosk 6 - Poster 3

Fibrinogen Concentrate (Fibryga®) Dosing Guided by Quantra® QPlus Point of Care Viscoelastic Testing. A Pilot Study of a formula using the FCS

Presenting Author: *Rudy Berthezene, MD*

Introduction: Fibrinogen (Fib) is a key component of hemostatic function. This precursor to insoluble fibrin is hemodiluted and degraded by cardiopulmonary bypass (CPB), which could lead to excessive post operative bleeding.1 Viscoelastic testing (VET), SEER Sonorheometry (HemoSonics Quantra® QPlus, Durham, NC) (Q+) is a point of care (POC) real time coagulation analyzer returning the Fib contribution to clot strength (FCS). With estimated total body Fib level, one can solve for a target dose of Fib. We conducted a pilot study utilizing mathematical dosing using FCS to guide Fib administered. Fib dose (g) = [Target Fib conc (g/L) x circulating blood volume (L)] – Calculated Fib (g) Calculated Fib (g) = (FCS (hPa) x 0,91) x circulating blood

volume (L)

Methods: Twenty-three patients were informed and signed an institutional approved consent for use of fibrinogen concentrate (Fibryga®, Octapharma, Parsippany, NJ, USA). Q+ FCS (hPa) has a 91.7% sensitivity for Claus Fib.2 Anesthetic, surgical and perfusion techniques were performed following standard of care procedures. Fifteen minutes after protamine administration, a Q+ test was run to calculate the Fib dose needed to be administered. One hour after administration a second Q+ was run to evaluate closeness to target. Data was analyzed for mean, median and SD when appropriate. CS and FCS were compared between times by one way ANOVA. Approximation to target Fib (FCS) level was broken into categories of 0-10%, 0-25% and >25% variance. It was arbitrarily accepted that less than ± 25% variance from target FCS was successful for this pilot. Bleeding (up to 24 h) was assessed.

Results: In 78% of patients the formula allowed approximate dose targeting (± 25%) and 52% of patients exhibited a 0-10% variance. 80% of the patients outside the acceptable range received transfusions of blood products. These patients also tended to bleed more at 24 hrs post-surgery (1190mL (IQR 1155) vs 785mL (IQR 575), p=0,47).

Conclusions: This pilot study showed that Fib dosing using the FCS is reasonable. In 78% of patients the formula allowed dose targeting (± 25%) and 52% of patients had a <10% variance from target. Most patients with variance showed underdosing. Considerably more Fib would have to be infused to reach a 2-3g/L target but was limited by the hospital pharmacy for cost constraints. Persistent bleeding occurred in 3 patients (re-explored for surgical origin). The use of an estimated circulating volume using pre-CPB weight could cause inaccuracy. As well, active bleeding, receiving blood products, fluids, or vasoactive drugs makes the formula less accurate in predicting Fib dose. POC-VET with Q+ using FCS as an estimate of Fib is now a quick and exciting way to tailor dosing to each patient. Future work will expand upon this pilot to find the best use of FCS in cardiac patients to individually guide Fib dosing.

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Table 1 : Population demographics

	(n=23)
Patients characteristics	
Sex Ratio (M/F)	10,5
Age (yrs)	70 [60 ; 74]
Weight (kg)	74 [70 ; 84]
Taille (m)	1,74 [1,71 ; 1,78]
Circulating blood volume (L)	5,0 [4,8 ; 5,2]
Protocol datas	
Fibrinogen infused (g)	1,56 [1 ; 2]
Targeted FCS (kPa)	1,8 [1,5 ; 2,0]
Total postoperative bleeding (mL)	875 [475 ; 1320]
Surgical procedures	
CABG	5
Valve replacement	13
Bentall	3
Tirone David	2
(Reoperation)	(2)

Results in median (Q1 ; Q3)

Table 2: Values before and after Fib administration

	Before	*After*	*p*
Fib lab ***(g/L)***	2,0 [1,8 ; 2,4]	2,5 [2,2 ; 2,7]	*0,01*
QPlus FCS ***(kPa)***	1,5 [1,2 ; 1,7]	1,7 [1,1 ; 2,0]	*0,02*
QPlus CS ***(kPa)***	12,8 [10 ; 14,6]	13,4 [11,9 ; 16,7]	*0,02*
Bleeding flow ***(mL/h)***	240 [150 ; 480]	210 [88 ; 360]	*ns*

Results in median (Q1 ; Q3)

References

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SP 1 - Kiosk 6 - Poster 4
Perioperative coagulation management in elective cardiac surgery
Presenting Author: *Andy Ngo, MD*

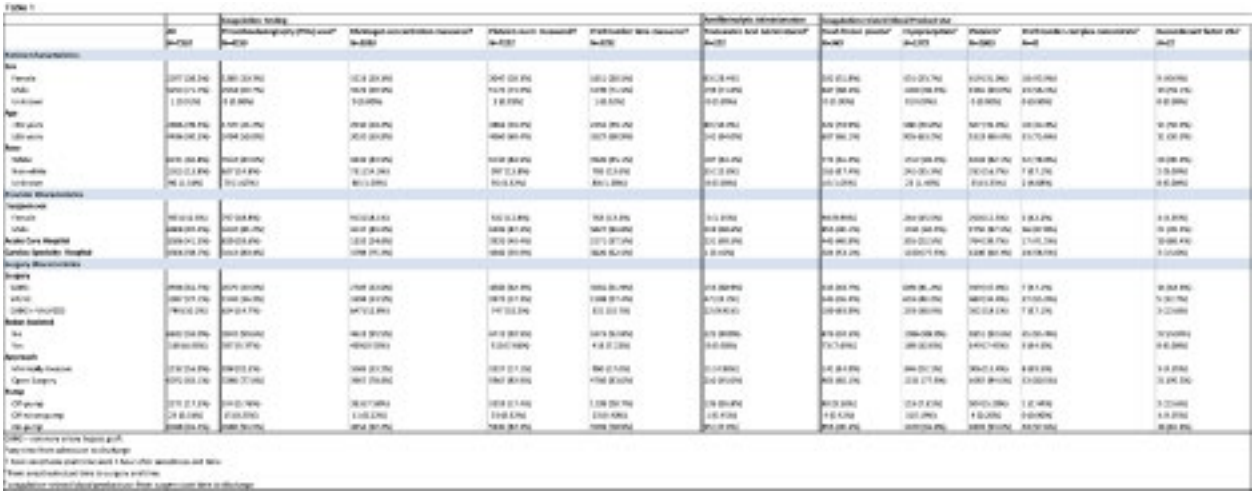
Background: Optimal perioperative coagulation management can improve clinical outcomes in cardiac surgery, where transfusion rates approach 40%(1). This study evaluated perioperative bleeding-risk assessment and management practices in patients undergoing elective cardiac surgery.

Methods: Using data from electronic health records and the STS Adult Cardiac Surgery Database, we conducted a retrospective cohort study to examine patients who underwent elective isolated coronary artery bypass graft (CABG), valve repair/replacement, or CABG+valve surgery from July 2020 to December 2024 in a U.S. integrated healthcare delivery system. We assessed the proportion of surgeries where thromboelastography (TEG), conventional coagulation tests, antifibrinolytic medications, coagulation-related blood products, and cell salvage were used.

Results: Table 1 shows the patient, provider, and surgery characteristics for the 7332 cardiac surgeries included in this study, and the usage of coagulation testing, antifibrinolytics, and coagulation-related blood products. Cell salvage was used in 5241 (71%) surgeries. Overall, 4239 (57.8%) surgeries had TEG performed at least once. Out of the 7332 cardiac surgeries, TEG was

performed in 308 (4.2%) pre-operatively, 3981 (54.3%) intra-operatively, and 631 (8.6%) post-operatively. Compared to the proportion of overall surgeries with each of these characteristics, TEG usage was higher in surgeries performed at cardiac specialty hospitals, on-pump, via a minimally invasive approach, by a female surgeon, and when it was a valve or CABG+valve surgery. TEG usage did not appear to vary by patient demographics. Intraoperative use of conventional coagulation tests showed 7222 (98.5%) surgeries had a platelet count, 5791 (79.0%) a prothrombin time, and 5050 (68.9%) a fibrinogen concentration measured. No obvious differences in usage of platelet counts and prothrombin time were observed by patient, provider, or surgery characteristics, but fibrinogen concentration measurement followed similar patterns to TEG usage. Tranexamic acid was administered in 222 (3.0%) surgeries; epsilon aminocaproic acid was administered in 1 (0.01%) surgery. Coagulation-related blood product usage showed that fresh frozen plasma was transfused in 949 (12.9%) surgeries, cryoprecipitate in 1575 (21.5%), and platelets in 2000 (27.3%). Cardiac specialty hospitals administered cryoprecipitate proportionately more frequently and fresh frozen plasma proportionately less frequently than acute care hospitals, while the latter accounted for almost all the tranexamic acid and recombinant factor VIIa used. Differences were also observed by surgery type, and on- vs off-pump status.
Conclusion: Use of TEG, coagulation testing, and related products varied substantially between cardiac specialty and acute-care hospitals, by surgery type, and off- vs on-pump status. This suggests that there are likely opportunities for standardization to optimize care quality and resource stewardship. Further research will be needed to determine where differences are driven by differences in patient complexity and types of procedure performed, as well as what impact variation in practice has on patient outcomes.

Image uploads:



References

Reference 1 Wyler von Ballmoos MC, Kaneko T, Iribarne A, et al. The Society of Thoracic Surgeons Adult Cardiac Surgery Database: 2023 Update on Procedure Data and Research. Ann

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SP 1 - Kiosk 6 - Poster 5
Institutional Product and Factor Usage as Function of Procedure Type Submission Type: Scientific Program

Presenting Author: *Samuel Belgique, MD*

Background: Significant perioperative bleeding in cardiac surgery is driven by CPB-associated coagulopathy, including dilutional effects, platelet dysfunction, and fibrinogen depletion [1]. Contemporary patient blood management guidelines emphasize multimodal, algorithm-based strategies using restrictive transfusion thresholds and viscoelastic-guided factor replacement, reserving concentrates for refractory bleeding [2]. We sought to describe baseline product and hemostatic factor utilization across procedure types at our center as groundwork for future interventions.

Methods: We conducted a single-center retrospective review of adult cardiac surgeries, categorizing cases as isolated non-hypothermic circulatory arrest (HCA) procedures (CABG/valve/ascending aorta without HCA), multiple non-HCA procedures, or aortic repair with HCA. Patients were grouped by whether they received either prothrombin complex concentrate (PCC) or recombinant factor 7a (rF7a) intraoperatively. Variables evaluated included demographics, preoperative antipla time, and nadir temperature. Continuous variables were described by mean ± standard deviation and compared using t-tests. Categorical variables were described as a percentage of each of the two groups and compared using χ^2 tests (see Table 1). Intraoperative administration of RBCs, FFP, platelets, cryoprecipitate, desmopressin, PCC, and rF7a were reported by procedure type (see Table 2).

Results: Among 152 patients, 25 (16%) received PCC or rF7a. Hemostatic agent use was more common in female patients (56% vs 33%; p=0.03), aortic repair with HCA (76% vs 11%; p< 0.001), emergent surgery (56% vs 6%; p< 0.001), and redo sternotomy (24% vs 9%; p=0.026). Patients receiving PCC or rF7a had longer CPB times (131.5±62.9 vs 84.0±33.2 min), lower nadir temperatures (27.9±4.7°C vs 33.3±2.8°C), higher preoperative INR (1.3±0.3 vs 1.1±0.2), and lower hemoglobin (11.6±2.8 vs 13.0±2.2). Antiplatelet use was less common among those requiring agents (12% vs 36%; p=0.018). PCC utilization increased stepwise by procedure complexity: isolated non-HCA (2.1%), multiple non-HCA (11.1%), and aortic repair with HCA (51.5%), with similar trends observed for all blood product types.

Conclusion: Blood product and hemostatic factor utilization increased with procedural complexity, peaking in aortic repair with HCA. Variation in transfusion patterns and factor use in our cohort likely reflects established bleeding risk factors identified in prior studies, including procedural complexity and CPB duration, surgical urgency and redo sternotomy, female sex, low preoperative hemoglobin, and elevated INR [3,4,5,6]. The analysis of 'any factor use' as a surrogate indicator of massive coagulopathy is a simple and previously unexplored method that identified risk factors of

bleeding after cardiac surgery consistent with earlier studies. In addition, these findings identify procedure types and clinical contexts most likely to benefit from future standardized interventions and provide a foundation for developing evidence-informed pathways to reduce transfusion exposure and improve hemostatic management in high-risk cardiac surgery populations.

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Table 1. Baseline patient demographic and clinical Information

	Managed with prothrombin complex concentrate or recombinant factor 7a?		
	No (n = 127)	Yes (n = 25)	P value
Age (years)	61.3 ± 12.6	59.1 ± 15.3	0.441
Female sex	42 (33%)	14 (56%)	0.030
Weight (kg)	90.6 ± 22.7	94.5 ± 22.5	0.451
Preoperative medications			
Anticoagulants	25 (20%)	2 (8%)	0.162
Antiplatelets	46 (36%)	3 (12%)	0.018
Preoperative labs			
Hemoglobin	13.0 ± 2.2	11.6 ± 2.8	0.012
Platelet count	214 ± 68.0	240.8 ± 91.8	0.124
INR	1.1 ± 0.2	1.3 ± 0.3	<0.001
Fibrinogen	485.1 ± 218.6	315 ± 149.9	0.346
Primary surgery			
Isolated non-HCA surgery*	89 (70%)	3 (12%)	<0.001
Multiple non-HCA surgeries*	24 (19%)	3 (12%)	0.409
Aortic repair with HCA	14 (11%)	19 (76%)	<0.001
Redo sternotomy	11 (9%)	6 (24%)	0.026
Emergent surgery	8 (6%)	14 (56%)	<0.001
Cardiopulmonary bypass time (min)	84 ± 33.2	131.5 ± 62.9	<0.001
Nadir core temperature (°C)	33.3 ± 2.8	27.9 ± 4.7	<0.001

HCA=hypothermic circulatory arrest; mean ± standard deviation and 2-tailed t-test performed for continuous variables; Chi square tests performed for categorical variables measured as a percentage of patients receiving or not receiving PCC or rF7a.
*non-HCA surgeries include coronary artery bypass grafting, valve surgery, and ascending aortic repair without HCA.

Table 2. Blood product and hemostatic agent administration, by procedure type

	Isolated non-HCA surgery (n = 92)	Multiple non-HCA surgeries (n = 27)	Aortic repair with HCA (n = 33)
Blood product			
Red blood cells	0.6 ± 1.3	1.3 ± 1.7	1.8 ± 2.4
Fresh frozen plasma	0.3 ± 0.8	0.6 ± 1.2	2.2 ± 2.6
Platelets	0.2 ± 0.4	0.4 ± 0.6	1.2 ± 0.6
Cryoprecipitate	0.1 ± 0.4	0.5 ± 0.9	2.0 ± 1.2
Hemostatic agents			
Desmopressin	9 (9.8%)	6 (22.2%)	18 (54.5%)
Prothrombin complex concentrate	2 (2.1%)	3 (11.1%)	17 (51.5%)
Recombinant factor 7a	1 (1.0%)	0 (0%)	8 (24.2%)

HCA=hypothermic circulatory arrest. Mean ± standard deviation provided for the number of blood products given; number of patients (percentage of the total number of patients having that procedure) having received each hemostatic agent given.

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SP 1 - Kiosk 6 - Poster 6

Perioperative blood product transfusion is not associated with higher rates of intraluminal thrombus (ILT) following non-emergency aortic arch replacement surgery Presenting Author: *Ben Chisnall, MBBS*

Background: Intraluminal thrombus (ILT) is a recognised complication of aortic arch replacement surgery using a frozen elephant trunk (FET) prosthesis, with a postoperative incidence of around 10% (1, 2). ILT formation has been linked to increased postoperative mortality and morbidity (2). Risk factors for ILT include aneurysmal aortic disease, central graft position, prothrombotic medical conditions, and conservative management of postoperative bleeding (1, 3). Perioperative blood product transfusion is usually required for these cases, however, as patients who have undergone aortic arch replacement tend to have some degree of coagulopathy after separation from cardiopulmonary bypass (CPB). We hypothesised that there may be an association between intra- and post-operative blood product transfusion and the development of ILT in the postoperative period.

Methods: This was a retrospective study looking at all patients who had undergone non-emergency aortic arch replacement surgery with insertion of a FET graft at our institution between January 2017 and June 2025. Intraluminal thrombus was defined as the presence of thrombus within the aortic graft at any point post-operatively, identified on computed tomography (CT) imaging. Transfusion data was taken from the theatre and ICU patient records, for the duration of admission to theatres and ICU. Statistical analysis was performed using Mann-Whitney U testing of samples (due to unequal group sizes) on IBM SPSS software.

Results: Out of 164 patients undergoing aortic arch replacement, 14 (8.5%) developed ILT. All patients received blood product transfusion either intra-operatively or post-operatively on ICU. Results are summarised in Tabl 1.
There was no statistically significant difference in volume of blood products received between the two groups.

Conclusion: Our study shows that the volume of perioperative blood product transfusion is not associated with intraluminal thrombus formation following aortic arch replacement surgery. These findings have not been shown before and provide reassurance that correction of intra- or post-operative coagulopathy does not correlate with ILT formation. This study was limited by a small sample size in the ILT group, which is to be expected given its relatively low incidence. **Image uploads:**

Blood component	ILT (n = 14) Mean (range)	No ILT (n = 150) Mean (range)	P value
Red cells (units)	4.57 (14 – 0)	3.96(26 – 0)	0.419
Platelets (units)	1.85 (5 – 0)	2.04 (19 – 0)	0.708
FFP (units)	3.5 (14 – 0)	3.80 (32 – 0)	0.320
Cryoprecipitate (units)	0.92 (4 – 0)	1.45 (8 – 0)	0.520
Fibrinogen concentrate (units)	1.14 (8 – 0)	0.38 (8 – 0)	0.457
All blood products (units)	12 (34 – 2)	11.6 (93 – 1)	0.581

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SP 1 - Kiosk 7 - Poster 1

Obesity and Outcomes in Patients Undergoing Veno-Venous Extracorporeal MembraneOxygenation: A Minnesota Consortium Study

Presenting Author: *Juan Morales Behaine, MD*

Background:Obesity is increasingly prevalent in critical care,affecting up to one-third of U.S. ICU patients.Although historically viewed as a relative contraindication to veno-venous extracorporeal membrane oxygenation(VV-ECMO) due to technical challenges and comorbidities,recent studies have challenged this perspective by demonstrating the feasibility and safety of VV-ECMO in obese patients.Evidence remains conflicting,with some studies reporting similar or improved outcomes in obese compared with non-obese patients,while others describe higher mortality,particularly in COVID-19–related ARDS.This study evaluates the relationship between obesity,in-hospital mortality,and ELSO-defined complications in adults supported with VV-ECMO for COVID-19 and non-COVID-19 ARDS using the Minnesota Consortium database between January 2017 and January 2025.

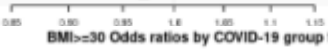
Methods:This multi-center observational cohort study includes adult VV-ECMO patients enrolled in the Minnesota Consortium database between 01/01/2017 and 01/01/2025.The dataset contains all patients who required VV-ECMO in four large academic centers in Minnesota during this period.The primary outcome was in-hospital mortality,with secondary outcomes consisting of ELSO-defined complications.Multivariable logistic regression assessed the association between both continuous and categorical BMI and clinical outcomes.An interaction test evaluated differences in the effect of BMI on outcomes between COVID and non-COVID groups.Model estimates,including odds ratios,95% confidence intervals,and p-values are reported.A complete case analysis was performed due to few missing observations.

Results:A total of 420 patients were included.There was insufficient data to conclude that the association between obesity and outcomes differed by COVID-19 status for all outcomes($p > 0.05$ for interaction test).Nonetheless,when evaluated as a continuous variable,BMI was associated with increased in-hospital mortality in non–COVID-19 patients(OR 1.06 per kg/m2, 95%CI 1.02–1.10, $p=0.003$),but no such association was observed in COVID-19 patients.Higher BMI also showed increased rates of renal complications in both COVID-19 (OR 1.07,95%CI 1.01–1.14,$p =0.018$), and non–COVID-19 patients (OR 1.04, 95%CI 1.00–1.07,$p=0.04$) as well as higher rates of infectious complications in non–COVID-19 patients(OR 1.05, 95%CI 1.02–1.09,$p=0.003$)(Figure 1).When analyzed categorically,a BMI≥30 was associated with higher in-hospital mortality(OR 2.17,95%CI 1.24–3.82,$p=0.007$) and higher rates of infectious complications (OR 2.49,95%CI 1.45–4.35,$p=0.001$) in non–COVID-19 patients(Figure 2).Categorization by obesity grades non-obese and obese I, II, and III showed that classes II and III were associated with higher mortality in non–COVID-19 patients compared to non-obese, whereas class I was not(Figure 3).An increased odds of infectious complications was also observed in non–COVID-19 patients (Figure 4).Overall,our findings add to the existing evidence and contrast with previous studies reporting no difference or improved mortality outcomes in obese ECMO
Patients.1-3

Conclusion:In this cohort,higher BMI was associated with increased mortality and infectious complications in non–COVID-19 VV-ECMO patients and increased renal complications in COVID-19 patients.

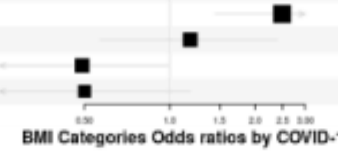
Continuous BMI Odds ratios by COVID-19 group for outcomes

Outcome	COVID-19 group	Odds Ratio	95% C.I.	p-value	Interaction p-value
Death	Non-COVID	1.06	(1.02, 1.10)	0.003	0.07
	COVID	1.00	(0.94, 1.05)	0.9	
Mechanical	Non-COVID	1.02	(0.99, 1.06)	0.18	0.77
	COVID	1.01	(0.95, 1.08)	0.71	
Hemorrhagic	Non-COVID	1.04	(1.00, 1.07)	0.06	0.09
	COVID	0.98	(0.92, 1.03)	0.42	
Pulmonary	Non-COVID	0.98	(0.94, 1.03)	0.5	0.11
	COVID	1.05	(0.99, 1.11)	0.15	
Cardiovascular	Non-COVID	1.02	(0.99, 1.06)	0.22	0.83
	COVID	1.03	(0.97, 1.10)	0.33	
Renal	Non-COVID	1.04	(1.00, 1.07)	0.04	0.3
	COVID	1.07	(1.01, 1.14)	0.018	
Neurologic	Non-COVID	0.98	(0.94, 1.03)	0.49	0.13
	COVID	1.06	(0.98, 1.14)	0.17	
Infectious	Non-COVID	1.05	(1.02, 1.09)	0.003	0.25
	COVID	1.01	(0.95, 1.07)	0.72	
Limb	Non-COVID	0.95	(0.91, 1.00)	0.06	0.64
	COVID	0.97	(0.91, 1.05)	0.47	



BMI>=30 Odds ratios by COVID-19 group for outcomes

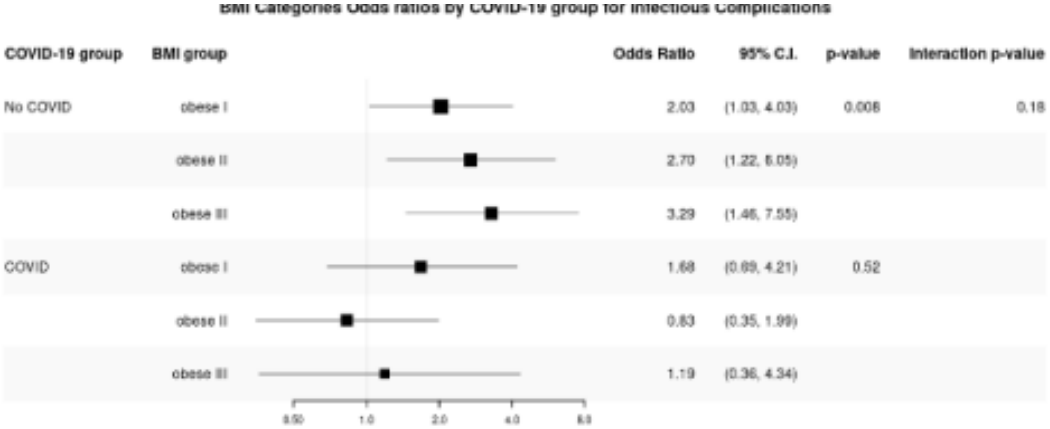
Outcome	COVID-19 group	Odds Ratio	95% C.I.	p-value	Interaction p-value
Death	Non-COVID	2.17	(1.24, 3.82)	0.007	0.06
	COVID	0.93	(0.47, 1.83)		
Mechanical	Non-COVID	1.24	(0.73, 2.12)	0.43	0.6
	COVID	1.59	(0.75, 3.36)		
Hemorrhagic	Non-COVID	1.75	(0.94, 3.36)	0.08	0.043
	COVID	0.67	(0.34, 1.33)		
Pulmonary	Non-COVID	1.02	(0.51, 2.03)	0.96	0.52
	COVID	1.41	(0.69, 3.02)		
Cardiovascular	Non-COVID	0.86	(0.47, 1.60)	0.64	0.34
	COVID	1.41	(0.65, 3.26)		
Renal	Non-COVID	1.50	(0.88, 2.57)	0.14	0.85
	COVID	0.99	(0.43, 2.26)		
Neurologic	Non-COVID	0.86	(0.43, 1.70)	0.66	0.3
	COVID	1.64	(0.62, 4.88)		
Infectious	Non-COVID	2.49	(1.45, 4.35)	0.001	0.1
	COVID	1.18	(0.57, 2.40)		
Limb	Non-COVID	0.49	(0.23, 0.99)	0.05	0.96
	COVID	0.50	(0.21, 1.18)		



BMI Categories Odds ratios by COVID-19 group for Death Complications

COVID-19 group	BMI group	Odds Ratio	95% C.I.	p-value	Interaction p-value
No COVID	obese I	1.47	(0.74, 2.98)	0.018	0.15
	obese II	2.46	(1.08, 6.01)		
	obese III	3.99	(1.58, 11.59)		
COVID	obese I	0.75	(0.33, 1.69)	0.6	
	obese II	1.31	(0.56, 3.09)		
	obese III	0.74	(0.24, 2.3)		





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SP 1 - Kiosk 7 - Poster 2
Post-operative oxygen delivery and delirium after cardiac surgery with cardiopulmonary bypass: a multisite retrospective cohort study of 1,690 patients
Presenting Author: *Elijah Christensen, MD, PhD*

Introduction: Delirium is a common, distressing, and dangerous complication after cardiac surgery with cardiopulmonary bypass (CPB) with few modifiable risk factors. This study aims to determine the association between post-operative oxygen delivery (DO2i) and the risk of delirium and assess the critical threshold for DO2i associated with delirium risk after surgery.

Methods: This study is a multi-site retrospective cohort study with 1,690 patients undergoing elective cardiac surgery with CPB who had multiple cardiac output measurements with a

pulmonary artery catheter and arterial blood gas analyses.

Results: The cohort of 1,690 patients analyzed included 185 patients diagnosed with delirium within 7 days of surgery (11%). Patients with delirium had a lower post-operative DO2i (mean difference of 26 ml/min/m2, 95% CI 14 to 36, p< 0.0001) compared to the 1,505 patients without delirium. In a multivariable analysis controlling for age, sex, race, heart failure, diabetes, obstructive sleep apnea, body mass index, CPB time, circulatory arrest time, aortic cross clamp time, carotid stenosis, and cognitive impairment the post-operative mean DO2i was not independently associated with delirium. An exploratory analysis by sex identified an independent association between delirium and an increased mean DO2i in the first 8 hours of ICU arrival and decreased mean DO2i between 8 and 16 hours and 24 and 32 hours in male patients. Post-operative DO2i was inversely correlated with CPB time with a decrease of 0.27 ml/min/m2 per minute of CPB exposure ($p < 0.0001$). The critical threshold of DO2i after cardiac surgery is lower with increased age and female sex. Conclusions: Post-operative DO2i over 7 days is not independently associated with the risk of delirium but significantly correlated with CPB time. Older patients may have a lower DO2i critical threshold for risk of delirium and the mean DO2i is independently associated with delirium in male patients only during the first 32 hours after ICU admission.

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SP 1 - Kiosk 7 - Poster 3

Tapia Syndrome after Coronary Artery Bypass Grafting

Presenting Author: *Daniel Schneider. MD*

Introduction: Tapia syndrome is a rare complication characterized by concurrent injury to the hypoglossal and recurrent laryngeal branches of the vagus nerves, typically presenting after airway manipulation or prolonged intubation in the perioperative setting. Diagnosis can be challenging due to a wide range of presentations, including dysphonia, tongue deviation, and dysphagia. Risk factors that
have been associated with developing Tapia syndrome include difficult airway manipulation, prolonged intubation, extreme neck flexion or extension during surgery, and high cuff pressures. Treatment is largely supportive, with most patients requiring rehabilitation to regain proper function.

Case Presentation: We report the case of a 62 year-old male with a past medical history significant for HTN, CAD and type 2 diabetes mellitus. He underwent an uneventful 4 vessel CABG, was admitted to the cardiovascular ICU in stable condition. On postoperative day four ENT was consulted due to the patient complaining of hoarseness and trouble moving food on the left side of his mouth. Transnasal flexible laryngoscopy was performed which demonstrated full mobility of the right true vocal fold with hypomobility of the left true vocal fold without any visible lesions. The patient was discharged and followed up with ENT whose physical exam showed left tongue weakness, furrowing, and subtle deviation to the left. Flexible laryngoscopy was repeated with findings of full immobility of the left true vocal fold and pooling of secretions in the pyriform sinus. The patient was counseled that there were options such as hyaluronic acid dermal fillers for the left vocal fold, referral to speech and language therapy, and conservative management. The patient opted for conservative management after being advised that the immobility was likely to resolve spontaneously.

Discussion: Tapia syndrome is a rare complication characterized by injury to the hypoglossal and recurrent laryngeal branches of the vagus nerves, typically occurring after airway manipulation such as intubation, laryngoscopy, or prolonged surgical positioning. It has significant overlap with routine post-extubation symptoms. Diagnosis is largely clinical and relies on findings of unilateral tongue deviation, dysphonia, hoarseness, dysphagia, and impaired gag reflex. These complications often lead to prolonged swallowing dysfunction, aspiration risk, voice abnormalities, and extended rehabilitation needs. Management is generally supportive, involving corticosteroids, speech therapy, swallowing therapy, and ENT follow-up.

Conclusion: Anesthesia providers must be aware of the hypoglossal and vagus nerves during intubation and ensure that they are implementing careful positioning, avoiding excessive force, maintaining endotracheal tube cuff pressures, and conducting a complete assessment of laryngeal function after extubation. In unconventional head positions, anesthesia teams should reassess alignment throughout the case to prevent nerve compression.

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SP 1 - Kiosk 7 - Poster 4
Detection of wire malposition by transesophageal echocardiography prevented major injury during fluoroscopic guided transcatheter aortic valve replacement Submission Type:
Presenting Author: *Kailash Panchapakesan, MD*

Introduction: This echocardiographic finding illustrates a situation during a valve-in-valve TAVR in which fluoroscopic guidance proved insufficient, and the use of transesophageal echocardiography prevented potential catastrophic injury.

Brief Summary of the Clinical Case: A 73-year-old male presented with a bioprosthetic valve failure of an existing transcatheter aortic valve replacement (TAVR), and was scheduled for a valve in valve TAVR . His medical history included severe aortic valve stenosis status post TAVR in 2021, atrial fibrillation, hypertension, sick sinus syndrome status post dual-chamber pacemaker, HFrEF, diabetes, and obesity. Surveillance transthoracic echocardiography demonstrated valve degeneration of the prior TAVR valve with leaflet thickening, restricted leaflet motion and elevated aortic valve gradients consistent with severe bioprosthetic valve failure. He also had reduced ejection fraction (45%).

At Rush, these procedures are typically performed under monitored anesthesia care with fluoroscopic guidance. In the usual fashion, the aortic valve was crossed with a glide wire using an Amplatz Left catheter (AL-1) under fluoroscopic guidance. Once the aortic valve was crossed, a stiff wire was positioned into the left ventricular apex. In the meantime, the patient began to have large volume emesis and was promptly intubated without complication. Given the change in clinical status, TEE was introduced. There was difficulty advancing the TAVR valve across the existing valve. At this point, the location and position of the wire used to deliver the TAVR valve was in question. TEE guidance was used. It demonstrated that the wire had passed through a paravalvular leak rather than through the bioprosthetic valve. This was also confirmed using

fluoroscopy.

Discussion: Paravalvular leak (PVL) after transcatheter aortic valve implantation (TAVI) is common and reported to occur more frequently than after surgical aortic valve replacement Pre-procedural risk factors include annulus shape, LVOT calcification, bicuspid aortic valve. The mid-esophageal AV long axis view was able to demonstrate the wire passing para valvular. In patients higher pre-procedural risk for para valvular leak, both initial TAVR and valve in valve procedures might benefit from TEE as a default imaging modality.

Impact of Echocardiographic findings: This malposition was identified before advancing further into the procedure and valve deployment, preventing possible catastrophic vascular or myocardial injury. The wire passing para valvular on first pass during a valve in valve procedure may be rare, but it highlights the value of TEE as an imaging modality for higher risk and more complex cases.
Teaching Points of the Case
Although valve-in-valve TAVR procedures often proceed uneventfully, this case underscores two critical points -
The importance of preparedness to secure the airway in the event of aspiration, and the value of having TEE readily available.
TEE offers unique anatomic visualization that fluoroscopy alone cannot provide, particularly when there is uncertainty regarding catheter or wire positioning.

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SP 1 - Kiosk 7 - Poster 5
Brain Death Determination on ECMO: Decision-Making When Physiology is Hijacked Presenting Author: *Theodore Lang, MD*

Introduction: Brain death is a medicolegal determination with requirements that can make its application in clinical practice challenging.1 The physiologic derangements present in critical illness further complicate the issue. To our knowledge, no standardized step-by-step protocol for determining brain death in patients on venoarterial-ECMO (VA-ECMO) is described in the literature.2 Inability to induce hypercarbia, frequent oxygen desaturations, and presence of hemodynamic instability also makes apnea testing more difficult in this patient population.3-4 We present a case demonstrating successful brain death determination for a patient on VA-ECMO.

Methods: All clinical information was collected by chart review.

Results: A 33-year-old female without significant past medical history presented to an outside hospital with acute myocardial infarction due to thromboembolism from a suspected patent foramen ovale. During revascularization, the patient developed profound cardiogenic shock requiring a percutaneous Impella device and peripheral VA-ECMO. At transfer to our hospital, the patient had decerebrate posturing, minimally responsive pupils, and dusky-appearing lower limbs without distal pulses. Despite conversion to central VA-ECMO with pelvic vessel reconstruction, serial head imaging was supportive of irreversible anoxic-ischemic brain injury. On exam, she was comatose with a core temperature >36oC and a mean arterial pressure >60 mmHg. There were absent brainstem reflexes and laboratory evaluation was unremarkable. There were >5 half-lives since the last administration of CNS-depressing medications.

For apnea testing, the patient was preoxygenated with FiO2 100% for 15 minutes on the ventilator followed by attachment of an end tidal carbon dioxide monitoring device. Second, the VA-ECMO settings were changed to a sweep flow rate of 1 L/min with FdO2 100%. The patient was disconnected from the ventilator and a baseline arterial blood gas (ABG) was collected. Serial ABG measurements were taken every 10 minutes until arterial carbon dioxide levels (PaCO2) were 20 mmHg higher than the baseline value. Thereafter, the patient was observed for respiratory effort for two minutes. Total time for apnea testing was 34 minutes and the patient remained hemodynamically stable with adequate oxygenation. A positive apnea test supporting brain death was confirmed by the absence of spontaneous respirations and a PaCO2 >60 mmHg and >20 mmHg above the patient's pre-apnea levels.

Conclusion: The case highlights a successful VA-ECMO apnea testing protocol for brain death determination without need for further ancillary studies (Fig 1.). Our protocol ensures a preoxygenation period of 15 minutes with FiO2 100% on mechanical ventilation followed by FdO2 100% on VA-ECMO.
Such requirements may obviate the need for supplemental oxygen therapy and mitigate concern for frequent desaturations. Also, induction of hypercarbia using a sweep flow rate of 1 L/min with an end target PaCO2 >60 mm Hg may shorten the required observation period and improve the quality of testing since these conditions likely approach the highest respiratory threshold or apnea point.5 The case offers clinicians additional considerations to optimize brain death determination for patients on VA-ECMO.

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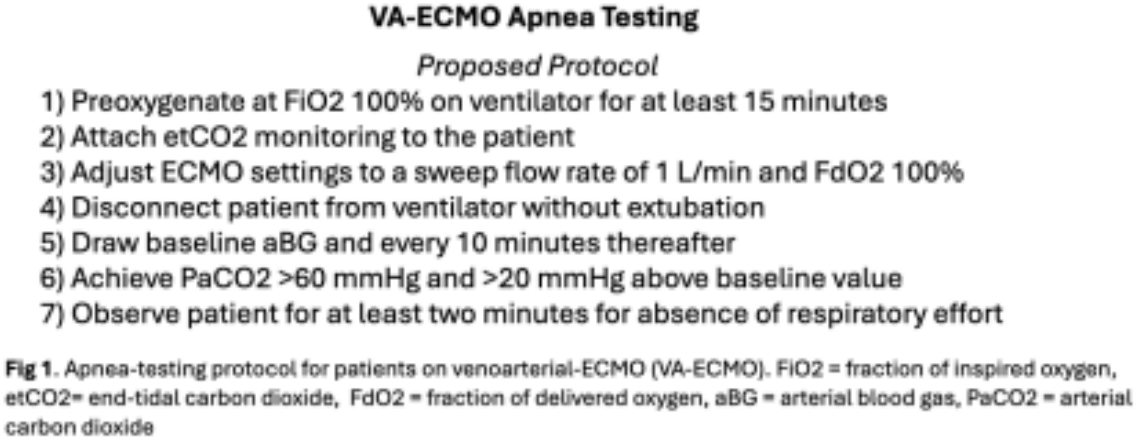


Fig 1. Apnea-testing protocol for patients on venoarterial-ECMO (VA-ECMO). FiO2 = fraction of inspired oxygen, etCO2= end-tidal carbon dioxide, FdO2 = fraction of delivered oxygen, aBG = arterial blood gas, PaCO2 = arterial carbon dioxide

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SP 1 - Kiosk 7 - Poster 6

Two Independent Leadless Pacemakers, One Operation: Perioperative Management of Dual Independent Systems

Presenting Author: *Behnaz Hatami. MD*

Introduction: Leadless pacemakers have transformed cardiac rhythm management by eliminating transvenous leads and generator pockets, thereby lowering infection and mechanical complication risks. However, the coexistence of two leadless pacemakers from different manufacturers is rare, and perioperative management strategies are not yet standardized.

Case Presentation: A 33-year-old woman with a medical history of sick sinus syndrome status post pacemaker, systemic lupus erythematosus, Ehlers-Danlos syndrome, autoimmune thyroiditis, seizure disorder, familial dysautonomia, and mast cell activation syndrome presented for robotic hysterectomy. She received a Biotronik dual-chamber pacemaker in 2021, which was removed in 2023 after MSSA endocarditis with a right-atrial lead vegetation. To minimize future infection risk, a Medtronic Micra AV leadless pacemaker was implanted in the right ventricle, followed by a St. Jude Aveir leadless pacemaker in the right atrium at a different facility. Preoperative interrogation revealed the Micra RV device set to VDD 70 bpm and the St. Jude Aveir RA device set to AAIR 60 bpm, with an underlying normal sinus rhythm of approximately 72 bpm. Although the infraumbilical surgical field did not mandate device reprogramming, the Micra's absence of magnet mode and the dual-device configuration prompted reprogramming to asynchronous MRI mode (VOO 90 bpm). The Aveir was left unchanged at AAIR 60. General anesthesia was maintained with total intravenous propofol, and intraoperative hemodynamics remained stable throughout an uneventful procedure. Postoperative interrogation showed normal device function, and settings were restored to baseline.

Discussion: This case highlights the evolving complexity of care in patients with leadless pacemakers. As emphasized by Ramirez et al. and the ASA Practice Advisory on cardiac implantable electronic devices (CIEDs), the anesthesiologist must verify device type, pacing dependency, and magnet response during preoperative assessment. Surgical electromagnetic interference (EMI) can cause oversensing and inappropriate inhibition of pacing output. Evidence suggests that leadless devices may be less susceptible to EMI, but the potential for malfunction warrants vigilance. The ASA and AHA recommend strategies such as minimizing monopolar electrocautery, using short bursts, and ensuring the current path does not cross the CIED system.
Due to differences in manufacturer programming, these systems may be unable to coordinate atrial and ventricular activity therefore, hemodynamic instability from AV synchrony loss should be anticipated. Close monitoring and multidisciplinary coordination remain central to safety. To our knowledge, this is one of the first reported cases describing anesthetic management of concurrent independent atrial and ventricular leadless pacemakers.

Conclusion: With the expanding use of leadless pacemakers, a comprehensive understanding of manufacturer-specific device modes, combined with coordinated multidisciplinary planning, is critical to optimizing patient safety.

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SP 1 - Kiosk 8 - Poster 1

Right Ventricle to Pulmonary Artery Coupling and Major Morbidity and Mortality After Vascular Surgery

Presenting Author: *Louisa Walker, MD*

Introduction: The right ventricle to pulmonary artery (RV-PA) coupling ratio has been utilized to assess right ventricular function in relation to pulmonary artery afterload in cardiac surgical patients and those undergoing transcatheter cardiac procedures. Studies evaluating the prognostic value of this RV-PA coupling have largely involved invasive monitoring such as right heart catheterization to obtain PA systolic pressure (PASP) or use of high-fidelity conductance catheters. Several of these studies have found an association between patients with low RV-PA coupling ratios and increased risk for major operative morbidity and mortality. However, it is unknown whether a low RV-PA coupling ratio is associated with worse outcomes in patients undergoing non-cardiac surgery.

Methods: This retrospective cohort study was conducted via chart review of patients who had lower extremity bypass grafting surgery at a single institution between January 2018 and December 2022. Only patients who had a transthoracic echocardiogram (TTE) that reported both a tricuspid annular plane systolic excursion (TAPSE) and right ventricular systolic pressure (RVSP) measurement done within one year prior to index surgery were included in the analysis. The RV-PA coupling ratio was calculated by dividing the TAPSE (mm) by the RVSP (mmHg). The primary

outcome was mortality within one year of surgery. Secondary outcomes included 30-day readmission, 30-day mortality, and postoperative myocardial infarction within one year of surgery. The Youden index was used to determine the optimal cut-off point for classifying patients into a low versus high RV-PA coupling ratio group based on one-year mortality.

Results: A total of 527 patients underwent lower extremity bypass grafting in the study period, and 121 of those had a TTE that included both TAPSE and RVSP measurements within one year prior to surgery. Of the 121 patients, 16 (13.2%) died within one year of surgery. There were 106 patients in the high RV-PA coupling ratio group (TAPSE/RVSP ≥0.38) and 15 in the low RV-PA coupling ratio group (TAPSE/RVSP < 0.38) (Table 1). One-year postoperative mortality was 8.5% (n=9) in the high RV-PA coupling ratio group and 46.7% (n=15) in the low RV-PA coupling ratio group (Figure 1). The rate of mortality at 30 days after surgery was 13.3% in the low RV-PA coupling ratio group compared to 1.9% in the high RV-PA coupling ratio group. The low RV-PA coupling ratio group also had a 13.3% incidence of postoperative myocardial infarction compared to 1.9% in the high RV-PA coupling ratio group.

Conclusion: In patients undergoing lower extremity bypass surgery with preoperative TTE-measured TAPSE and RVSP, those with a RV-PA coupling ratio below 0.38 mm/mmHg had higher rates of mortality within one year of surgery. Though this study was limited by a single center and small sample size with a relatively low one-year mortality rate, the results demonstrate a tendency towards increased mortality and postoperative complications in patients with RV-PA coupling ratios below the cutoff.

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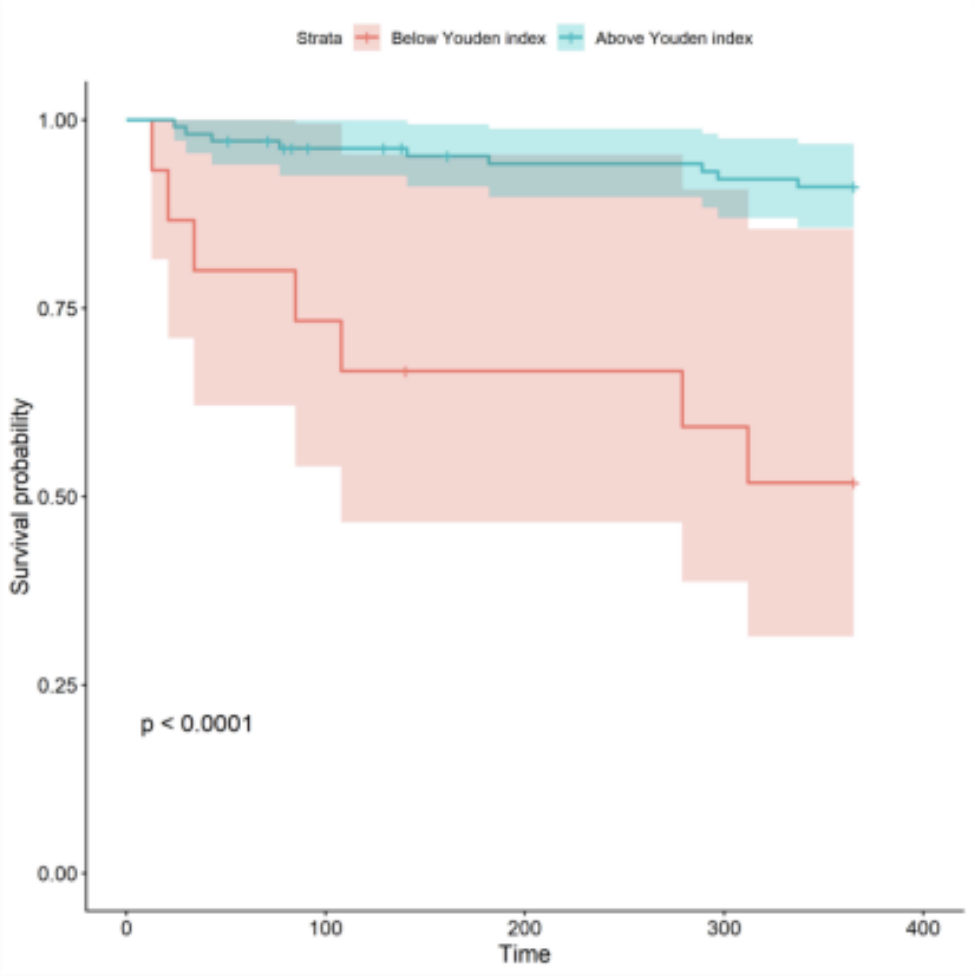


Figure 1. Survival curves for those patients above versus below the Youden index cutoff of an RV-PA coupling ratio of 0.38, as determined by TTE-measured TAPSE divided by RVSP. Shading indicates the 95% confidence intervals.

Table 1. Patient characteristics and outcomes stratified by TAPSE/RVSP cutoff of ≥0.38 mm/mmHg

TAPSE/RVSP	Total (n=121)	≥0.38 (n=106)	<0.38 (n=15)	p-value
Demographics				
Age	66.5 ± 11.2	66.0 ± 11.2	69.8 ± 10.9	0.223
Sex				0.117
Female	42 (34.7%)	40 (37.7%)	2 (13.3%)	
Male	79 (65.3%)	66 (62.3%)	13 (86.7%)	
Lost to follow-up within 1-year	9 (7.4%)	8 (7.5%)	1 (6.7%)	>0.999
Preoperative Comorbidities				
Hypertension	111 (91.7%)	98 (92.5%)	13 (86.7%)	0.794
Diabetes mellitus	61 (50.4%)	52 (49.1%)	9 (60.0%)	0.605
Chronic kidney disease	37 (30.6%)	28 (26.4%)	9 (60.0%)	**0.019**
End stage renal disease	6 (5.0%)	5 (4.7%)	1 (6.7%)	>0.999
Coronary artery disease	61 (50.4%)	52 (49.1%)	9 (60.0%)	0.605
Congestive heart failure	36 (29.8%)	25 (23.6%)	11 (73.3%)	**<0.001**
Atrial fibrillation	24 (19.8%)	16 (15.1%)	8 (53.3%)	**0.002**
Obesity	32 (26.4%)	29 (27.4%)	3 (20.0%)	0.770
TTE Parameters				
TAPSE (mm)	21.0 [17.0, 25.0]	21.6 [18.0, 25.0]	13.0 [11.5, 15.4]	**<0.001**
RVSP (mmHg)	28.20 [15.00, 40.00]	27.00 [14.25, 34.75]	52.00 [44.50, 62.00]	**<0.001**
TAPSE/RVSP ratio	0.75 [0.49, 1.19]	0.84 [0.58, 1.32]	0.26 [0.24, 0.28]	**<0.001**
TR Severity				0.283
None	8 (6.6%)	8 (7.5%)	0 (0.0%)	
Mild	101 (83.5%)	88 (83.0%)	13 (86.7%)	
Moderate	10 (8.3%)	9 (8.5%)	1 (6.7%)	
Severe	2 (1.7%)	1 (0.9%)	1 (6.7%)	
LV Ejection Fraction				**<0.001**
>55%	88 (72.7%)	83 (78.3%)	5 (33.3%)	
>45%-55%	11 (9.1%)	10 (9.4%)	1 (6.7%)	
>35%-45%	9 (7.4%)	7 (6.6%)	2 (13.3%)	
>25%-35%	6 (5.0%)	4 (3.8%)	2 (13.3%)	
>15-25%	5 (4.1%)	2 (1.9%)	3 (20.0%)	
<15%	2 (1.7%)	0 (0.0%)	2 (13.3%)	
RV Systolic Function				**<0.001**
None	105 (86.8%)	99 (93.4%)	6 (40.0%)	
Mild dysfunction	12 (9.9%)	6 (5.7%)	6 (40.0%)	
Moderate dysfunction	2 (1.7%)	0 (0.0%)	2 (13.3%)	
Severe dysfunction	2 (1.7%)	1 (0.9%)	1 (6.7%)	
Outcomes				
30-day mortality	4 (3.3%)	2 (1.9%)	2 (13.3%)	0.121
1-year mortality	16 (13.2%)	9 (8.5%)	7 (46.7%)	**<0.001**
Myocardial infarction	4 (3.3%)	2 (1.9%)	2 (13.3%)	0.121

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SP 1 - Kiosk 8 - Poster 2

Echocardiographic Assessment of the Pulmonic Autograft in Patients Undergoing the Ross Procedure

Presenting Authors: *Simone Redaelli, MD*

Background: The Ross procedure is a highly specialized cardiac operation in which the diseased aortic valve is replaced with the patient’s native pulmonary valve (autograft), followed by reconstruction of the right ventricular outflow tract with a cadaveric pulmonary valve (homograft). Emerging data suggests favorable long-term outcomes of the Ross procedure1, yet criteria for determining whether a native pulmonary valve is suitable for use as an autograft remain poorly defined. Specifically, the prognostic significance of preoperative pulmonary valve echocardiographic findings and their relationship to durability of the Ross procedure failure have not been fully elucidated. The aim of this study was to evaluate native pulmonary regurgitation prior to the Ross procedure and to assess the echocardiographic features of regurgitation after autograft.

Methods: This retrospective study included patients deemed eligible by pre-procedural surgical and echocardiographic evaluations who underwent the Ross procedure at a tertiary hospital from January 2022 to October 2023. Patient demographics and surgical characteristics were collected from electronic health records after IRB approval. The severity and origin of regurgitation of both the native pulmonary valve and the neo-aortic autograft were assessed using echocardiographic reports and echocardiography Image uploads: review. For transesophageal echocardiography, we reviewed the middle esophageal (ME) right ventricle inflow-outflow, ME ascending aorta short-axis, ME aortic valve long axis, upper esophageal aortic arch short axis and transgastric right ventricle basal views. For transthoracic echocardiography (TTE), we reviewed parasternal long- and short-axis and the apical five-chamber view.

Results: We included 29 consecutive patients. 10 (34.5%) were female, mean (±SD) age 44 ± 8. Patient characteristics are presented in Table 1. 24 (82.8%) patients underwent surgery for a bicuspid aortic valve, and mixed aortic regurgitation and stenosis was the most common preoperative finding (48.3%). 19 (65.5%) patients had trace or trace-to-mild native pulmonary regurgitation, and none had more than mild central pulmonary regurgitation. 19 (65.5%) had trace or trace-to-mild autograft regurgitation immediately after the procedure. At the first TTE follow-up (median [IQR] 4 [4-5] days post procedure), 17 (58.6%) patients had trace or trace-to-mild autograft regurgitation, 4 (13.7%) had mild autograft regurgitation, and one had moderate-to-severe regurgitation. At a second TTE follow-up (median [IQR] 181 [84-202] days post procedure), 8 patients showed worsening autograft regurgitation, although in most cases (75%) the severity did not exceed mild. Notably, 3 patients improved from trace to none. Trends in autograft regurgitation are shown in Figure 1 and Table 2.

Conclusion: In this single-center cohort, native pulmonary regurgitation was uniformly minimal prior to the Ross procedure, and early postoperative autograft regurgitation was mild or less and generally stable over short-term follow-up. Larger studies with longer follow-up are needed to clarify the prognostic value of preoperative pulmonary valve findings and to refine criteria for optimal autograft selection.

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Table 1. Patient characteristics and operative variables

Demographics	
Age, years	44 ± 8
Female, n (%)	10 (34.5%)
Height (median, IQR), cm	170 (163-178)
Weight (median, IQR), kg	88 (80-103)
BMI (median, IQR), kg/m^2	30.6 (27.4 -33.7)
Comorbidities	
Hypertension, n (%)	14 (48.3%)
Diabetes mellitus, n (%)	1 (3.4%)
Coronary artery disease, n (%)	3 (10.3%)
Intraoperative	
CPB time (median, IQR), min	223 (215-237)
Aortic cross-clamp time (median, IQR), min	171 (148-182)
Postoperative	
Hospital LOS (median, IQR), days	6 (5-8)
ICU LOS (median, IQR), days	2 (2-2)

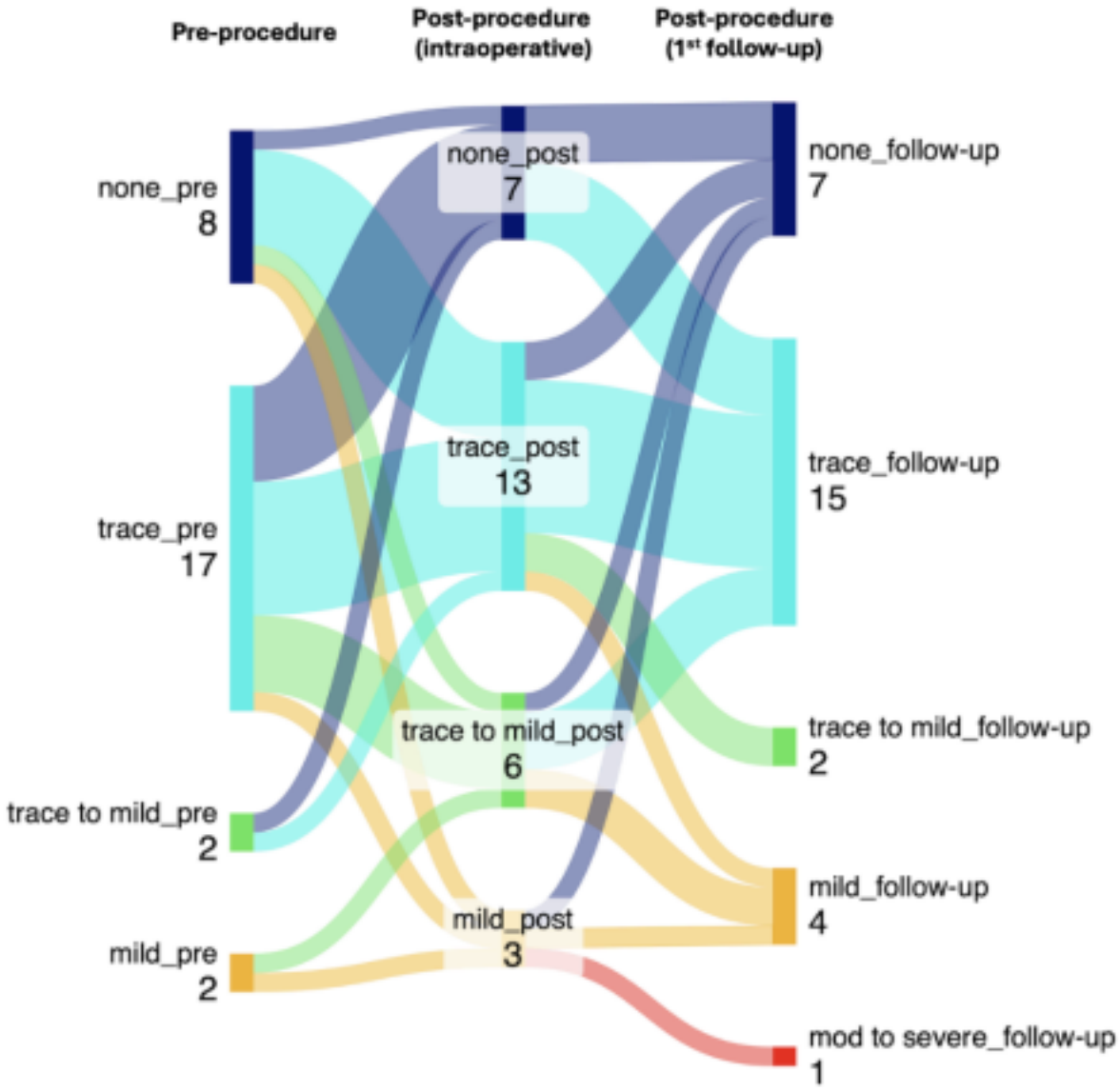
Abbreviations: BMI, Body mass index; CPB, Cardiopulmonary bypass; LOS, Length of stay; ICU, Intensive care unit

Table 2. Severity and origin of valve regurgitation

Patient	Pulmonary insufficiency preoperative		Autograft insufficiency intraoperative		Autograft insufficiency postoperative (1st follow-up)		Autograft insufficiency postoperative (2nd follow-up)	
1	mild	central	mild	both	none		trace	central
2	none		mild	commissural	mild	commissural	mild	commissural
3	trace	both	mild	commissural	moderate-to-severe	commissural	severe	both
4	none		none		none		trace	central
5	trace		none		trace	central	trace	central
6	trace	central	none		trace	central	trace	
7	trace	central	none		none		trace	
8	trace	central	none		none		trace	central
9	trace	central	none		trace	central	none	
10	trace-to-mild	central	none		trace	central	trace	central
11	none		trace		trace	central	trace	central
12	trace	both	trace		trace-to-mild	commissural	none	
13	none		trace	both	mild	both	mild	both
14	none		trace	both	trace		trace	
15	trace-to-mild	both	trace	both	none		none	
16	none		trace	central	none		none	
17	none		trace	central	trace	commissural	trace	central
18	trace	central	trace	central	trace	central	none	
19	trace	central	trace	central	trace	central	trace	
20	trace	central	trace	central	trace-to-mild	central	trace-to-mild	central
21	trace	central	trace	central	trace	central	trace	central
22	trace		trace	commissural	trace		trace	
23	trace	both	trace	commissural	trace		trace	
24	trace	central	trace-to-mild	both	trace	commissural	mild	both
25	trace	central	trace-to-mild	both	mild	both	mild-to-moderate	both
26	trace	central	trace-to-mild	both	none		mild	
27	none		trace-to-mild	central	trace	central	trace	central
28	trace	central	trace-to-mild	central	trace	central	trace	central
29	mild	central	trace-to-mild	commissural	mild	commissural	mild	commissural

Assessment of valve regurgitation was performed before the procedure (intraoperative), immediately after the procedure (intraoperative), at first follow-up (median [IQR] 4 [4-5] days post-procedure), and at second follow-up (median [IQR] 181 [84-202] days post procedure).

Figure 1. Temporal changes of native pulmonic valve (pre-procedure) and autograft (post-procedure) regurgitation



From left to right, the graph displays changes in valve-regurgitation severity across different time points. The severity is color-coded: blue = no regurgitation, light blue = trace, green = trace to mild, yellow = mild, and red = moderate to severe. Numbers indicate the number of patients for each group. Severity of valve regurgitation was assessed by transesophageal echocardiography before the procedure (intraoperative) and immediately after the procedure (intraoperative). The first follow-up assessment (post-procedure) was obtained by transthoracic echocardiography at median [IQR] 4 [4-5] days post-procedure. *Abbreviations*: mod to severe: moderate to severe

References
Reference 1 Ryan WH, Squiers JJ, Harrington KB, Goodenow T, Rawitscher C, Schaffer JM, DiMaio JM, Brinkman WT. Long-term outcomes of the Ross procedure in adults. Ann Cardiothorac Surg. 2021 Jul;10(4):499-508. doi: 10.21037/acs-2021-rp-fs-28. PMID: 34422562; PMCID: PMC8339616.

SP 1 - Kiosk 8 - Poster 3
Validation of Echocardiographically Measured Left Ventricular Mass Ratio for Heart Transplant Size Matching in Obese Donors and Recipients
Presenting Author: *Christian O'Donnell, MD*

Background: The predicted heart mass (PHM) ratio is commonly used for donor-to-recipient size matching in heart transplantation but may be less predictive in obesity due to the dependence on body weight for left ventricular mass (LVM) estimation. We recently demonstrated that using transthoracic echocardiographically (TTE) measured donor LVM for size-matching, using core lab interpretation of donor TTEs, is more predictive of one-year transplant outcomes than the PHM ratio. However, there is a need for validation in obese donors and recipients using the local TTE upon which donor heart acceptance decisions are based.

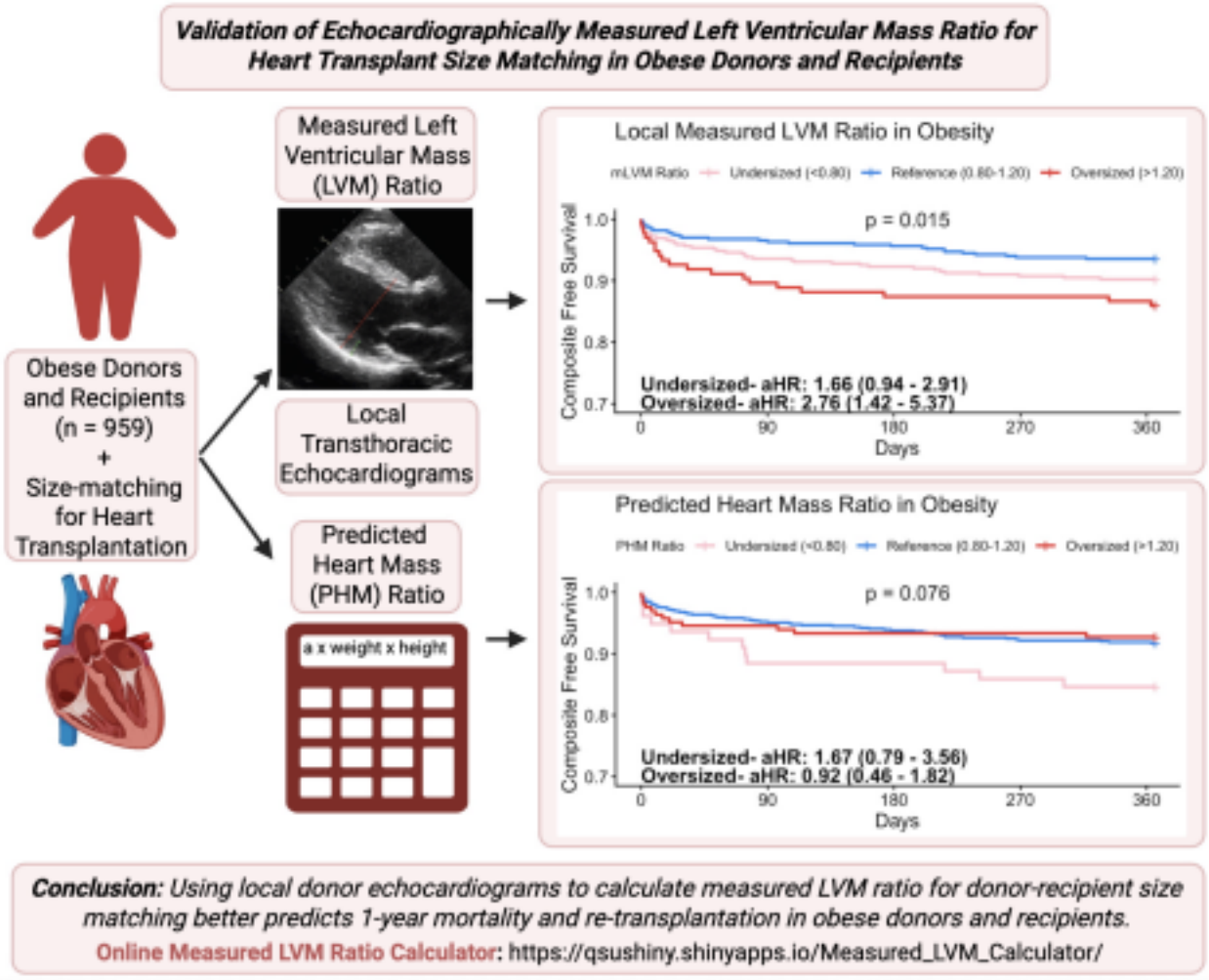
Methods: The Donor Heart Study was a prospective, multicenter, observational cohort study that collected echocardiograms from brain dead donors. Local TTE measurements of the donor performed at the time of transplant offer were collected for obese donors and recipients (body mass index [BMI] ≥30kg/m2) and used to calculate the locally measured LVM ratio (donor measured LVM/recipient predicted LVM) using an online calculator that we developed (https://qsushiny.shinyapps.io/Measured_LVM_Calculator/). The association of the measured LVM ratio with the primary composite outcome of 1-year mortality or re-transplantation was analyzed by adjusted Cox proportional hazard models and Kaplan-Meier survival curve analyses. Secondary analyses compared the association of PHM ratio and the combination of measured LVM and PHM ratio with the primary outcome. Undersized matches were defined as a donor/recipient size ratio< 0.8, ideal sized 0.8-1.2, and oversized >1.2.

Results: In 959 obese heart transplants, from mostly male donors (68.4%) and recipients (75.4%) with a median BMI of 31 kg/m2 there was a 19.0% (IQR: 10.0 – 33.0) median absolute difference between the predicted and measured LVM ratio. Undersized and oversized transplants by the measured LVM ratio had early separation from ideally size-matched transplants by Kaplan-Meier analysis (log-rank p-value = 0.015), where the primary outcome occurred in a U-shaped pattern (undersized: 9.8%, ideal size-matched: 6.4%, oversized: 14.1%; p = 0.017). After multivariable adjustment, the measured LVM ratio predicted oversized matches had a greater than 2-fold increased risk (adjusted hazard ratio [aHR]: 2.76 [95% CI: 1.42 – 5.37], p= 0.003) for the 1-year composite outcome, and there was a strong trend for increased risk with undersized matches (aHR: 1.66 [95% CI: 0.94 – 2.76], p = 0.078). In contrast, the PHM ratio did not

significantly differentiate the primary outcome (log-rank p-value = 0.082) and was not predictive after adjustment. Comparative receiver operator characteristic curves demonstrated that combining the measured LVM ratio with PHM ratio (AUC: 0.66) had better discrimination than using the PHM ratio alone (AUC: 0.56) (Delong p-value = 0.050).

Conclusion: Using local TTE measurements of donor LVM for size-matching in obese donors and recipients better predicts poor 1-year heart transplant outcomes than the currently used PHM ratio. Combining the novel measured LVM ratio method with the traditional PHM ratio may provide a more precise method for donor-recipient matching in the high-risk obese heart transplant population.

Image uploads:



Reference

Reference 1 O'Donnell, C., Tapaskar, N., Sanchez, P.A., Wayda, B., Santana, E.J., Pulgrossi,

R.C., Steffner, K., Zhang, S., Weng, Y., Sun, L.Y., Malinoski, D., Zaroff, J., Haddad, F., Khush, K.K. The Association of Echocardiographically Measured Donor Left Ventricular Mass and 1-Year Outcomes After Heart Transplantation. JACC: Heart Failure. 2025. PMID: 39570237.

SP 1 - Kiosk 8 - Poster 4

It is worse than it looks: Echocardiographic guidance of infective endocarditis valve replacement using a full commando procedure

Presenting Author: *Rodney Chen, MD*

Introduction: Infective endocarditis is a relatively rare diagnosis that carries an almost 30% mortality rate despite advanced diagnosis and management (1,2). Often, because of previously undetected fistulae, abscesses, and vegetations, perioperative echo becomes essential to guide surgical intervention. Our patient is a 64-year-old male with uncontrolled type 2 diabetes, hyperlipidemia, and severe aortic stenosis with previous bioprosthetic surgical AVR done in 2023, who presented with malaise, leukocytosis, and hyperglycemia with glucose > 400 and hemoglobin A1c > 12%, and was subsequently found to have an aortic root abscess and mitral valve endocarditis. Pre-operative transesophageal echo demonstrated an aortic root abscess and a large mobile mitral valve vegetation with suspected leaflet perforation prompting surgical mitral valve replacement. However, intraoperative transesophageal echo revealed a compromised aorto-mitral curtain, with an aortic root to left atrial fistula (previously thought to be the mitral valve perforation) and an aortic root to right atrial fistula.

Summary of the Clinical Case: Based on perioperative transesophageal echo findings, the surgical team proceeded with aortic root replacement, mitral valve replacement, and reconstruction of the intervalvular fibrous body with a bovine pericardial patch (commando procedure). The patient required a second cardiopulmonary bypass runs to deal with bleeding around the aortic root as the tissues had been 'obliterated' and achieving hemostasis was difficult. He was supported on VA ECMO for three days postoperatively to support hemodynamics and keep the aortic root depressurized.

Discussion: Mitral valvular vegetations is clearly visualized on mid-esophageal 4-chamber view with further characterization of the lesion in mid-esophageal long axis view and with 3D echo views
Thickening along the tricuspid valve in mid-esophageal 4-chamber view suggests vegetations along the tricuspid valve in addition to the mitral and aortic valves
Color flow doppler demonstrates a type 1 mitral regurgitant jet across the anterior mitral leaflet secondary to infectious erosion of the native valve in the long axis view. This is further demonstrated in a true view 3D tissue model of the mitral valve
3D echo imaging of the mitral valve demonstrates a clear regurgitant jet as well as a large valvular

vegetation. Fistula tracts are characterized extending into the left and right atria with a 3D aortic root image

A mid-esophageal long axis view demonstrates a clear aortic root-to-left atrium fistula

Significant valvular thickening of the aortic valve can be visualized on mid-esophageal long axis view

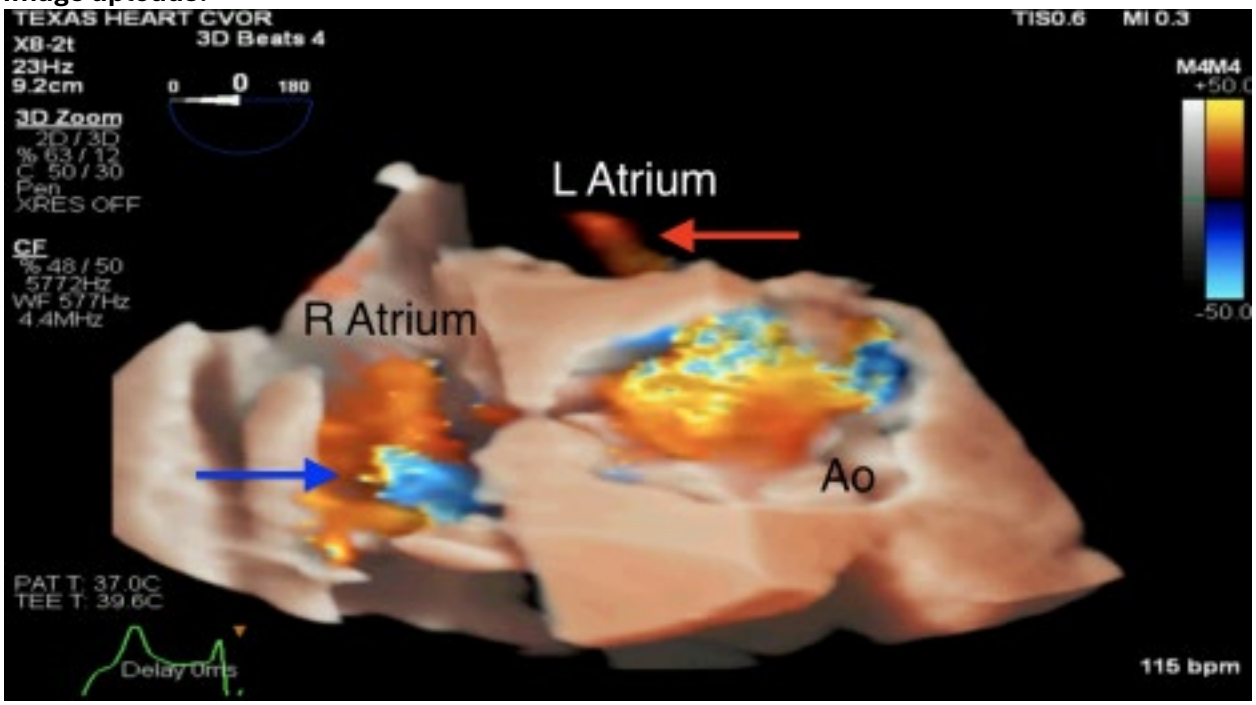
Post-procedural long axis view demonstrates a well-seeded bioprosthetic mitral valve. 3D post-procedural mitral valve demonstrates significant perivalvular leak, which was repaired

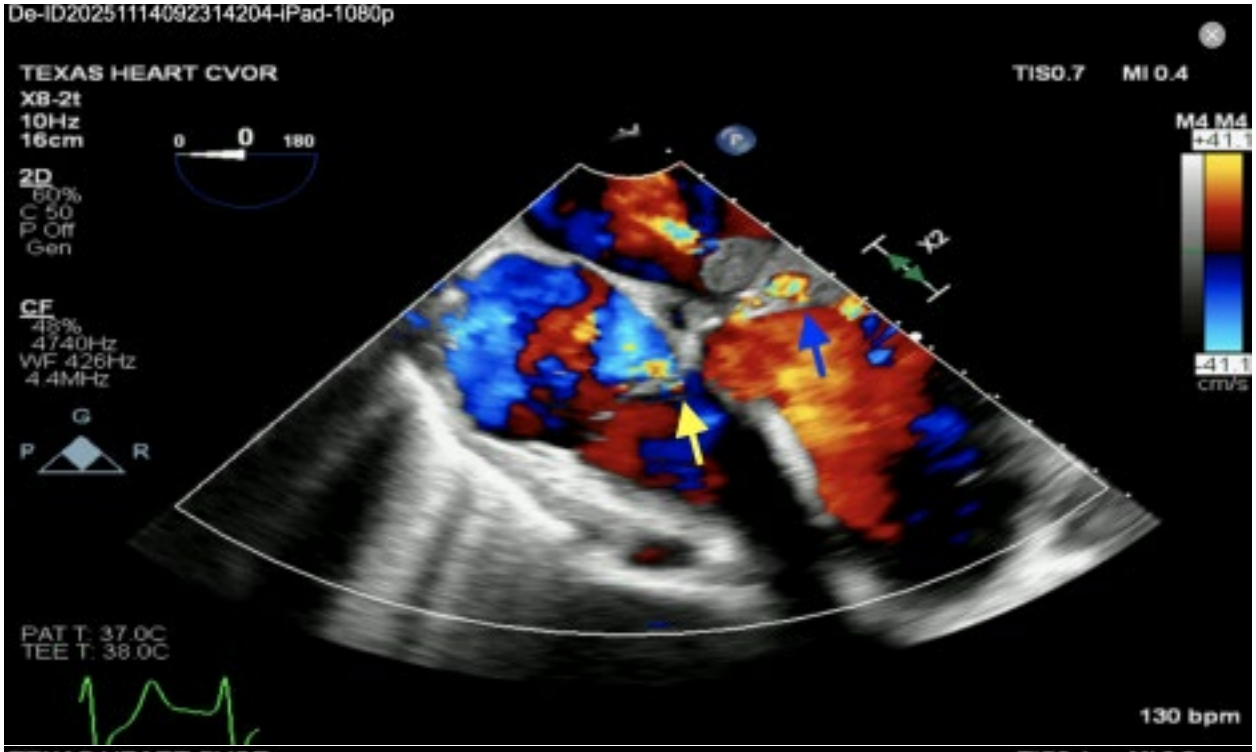
Impact of Echocardiographic findings

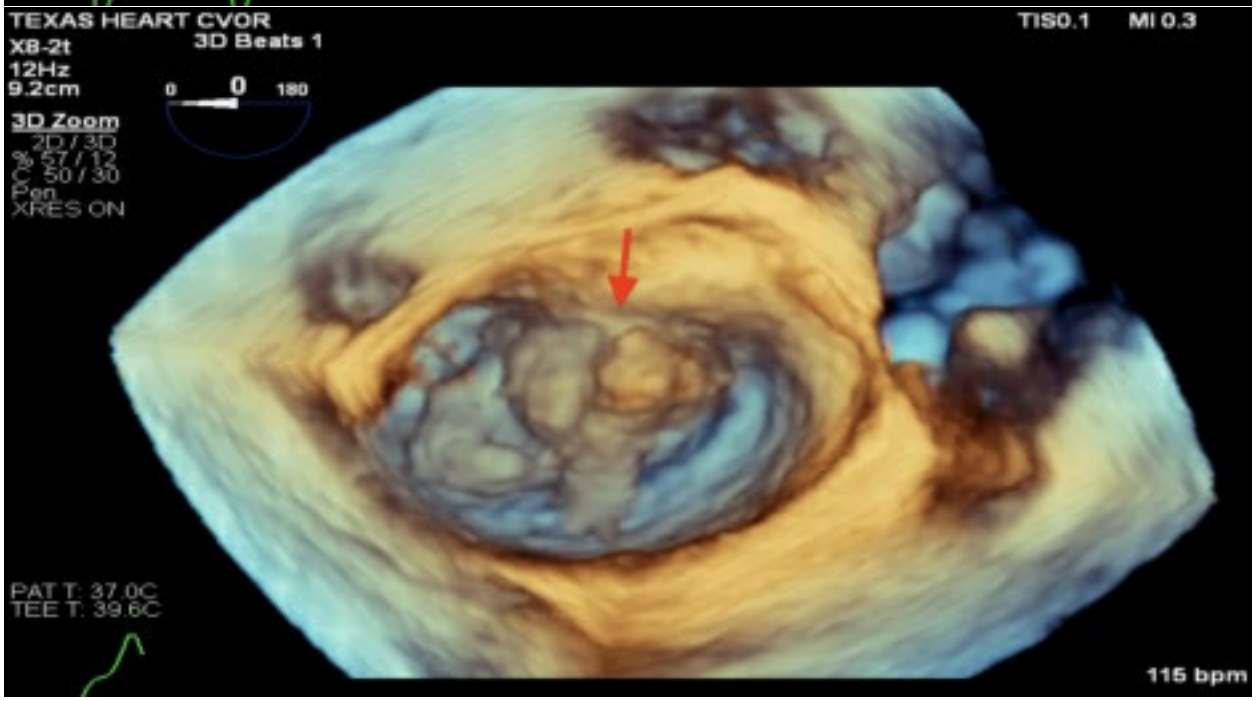
Echocardiographic guidance helped identify new fistulae previously not appreciated on pre-operative imaging as well as identifying tricuspid leaflet involvement.

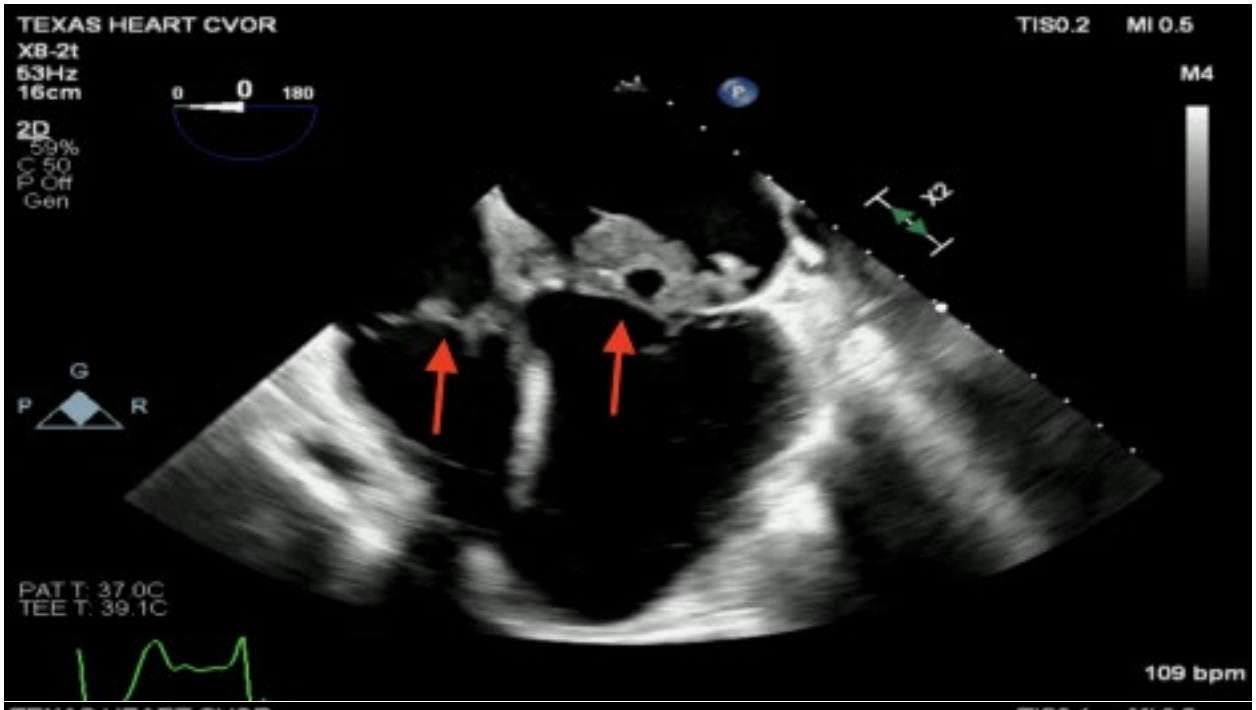
Teaching Points of the Case: A comprehensive transesophageal echo exam is critical to diagnosis and perioperative management of infective endocarditis. An apparent and overlying lesion can readily mask additional echo findings that might significantly alter surgical approach to valve repair.

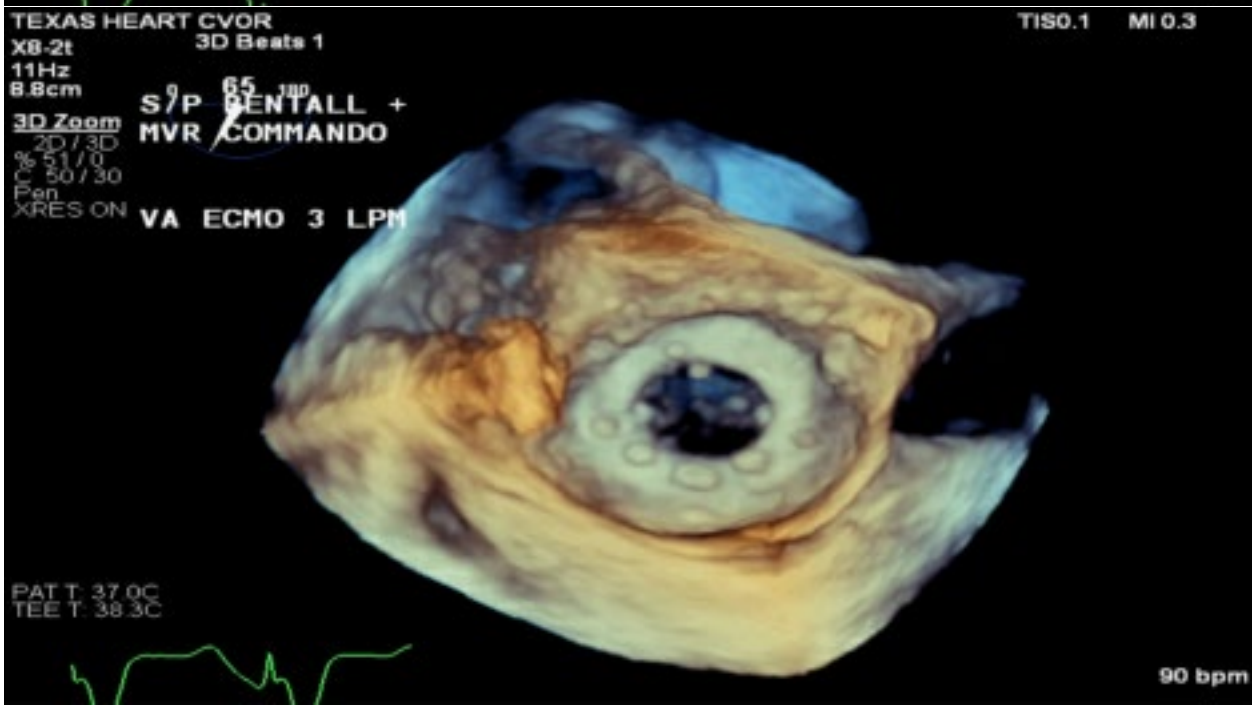
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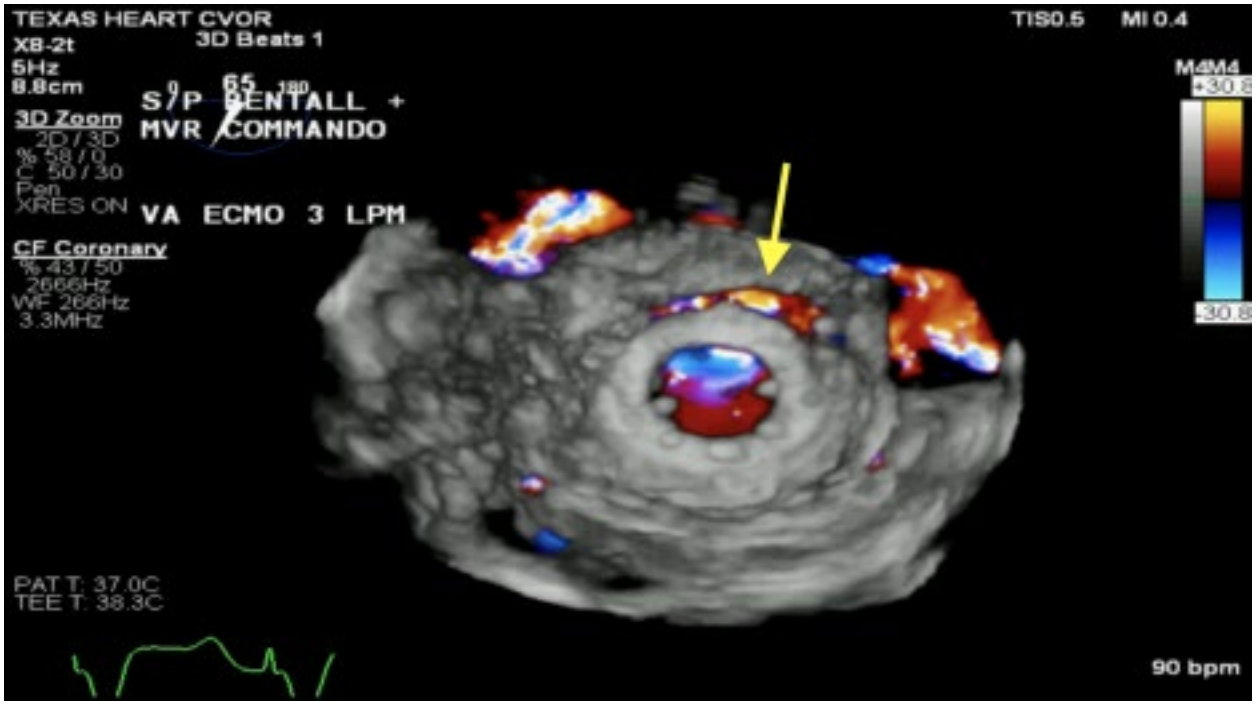



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109 bpm


TEXAS HEART CVOR
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PAT T: 37.0C
TEE T: 38.3C
90 bpm



References
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Reference 2 Chambers, H. F., et al. (2020). "Native-Valve Infective Endocarditis." N Engl J Med 383(6): 567-576.

SP 1 - Kiosk 8 - Poster 5
Pulmonary Arterial End-Diastolic Forward Flow in Carcinoid Heart Disease
Presenting Author: *Andrew Stone, MD*

Introduction Paragraph: Pulmonary arterial end-diastolic forward flow (EDFF) occurs when the diastolic pressure in the right ventricle (RV) exceeds the diastolic pressure in the pulmonary artery (PA) during atrial contraction. EDFF has been associated with diastolic dysfunction of the right ventricle, especially after repair of tetralogy of Fallot (TOF). Carcinoid syndrome (CS) metastasized to the liver can cause carcinoid heart disease (CaHD) through serotonin (5-HT) production that induces endocardial fibrosis (1). This fibrosis primarily affects the tricuspid and pulmonic valves, resulting in tricuspid (TR) and pulmonic regurgitation and/or stenosis (PR/PS) due to thickened and restricted leaflets. EDFF may be found in the intraoperative transesophageal echocardiographic exam of patients with CaHD as seen in the following cases.

Brief Summary of the Clinical Case: A 62-year-old female with a history of CaHD presented for tricuspid valve replacement (TVR) and pulmonic valve replacement (PVR). Preoperative testing demonstrated a moderately dilated RV with normal function, severe TR, PS, severe PR, elevated right sided filling pressures, normal pulmonary artery pressures and normal left-sided filling pressure. Preinduction invasive central monitors were placed while on octreotide and vasopressor

infusions. TEE showed torrential TR and severe PR due to restricted and thickened leaflets that looked “fixed in the open position”. The spectral Doppler envelope of the PA revealed EDFF during atrial contraction. The TVR and PVR were performed without issue and the patient was transferred to the ICU intubated on support with phenylephrine, vasopressin, and dobutamine.

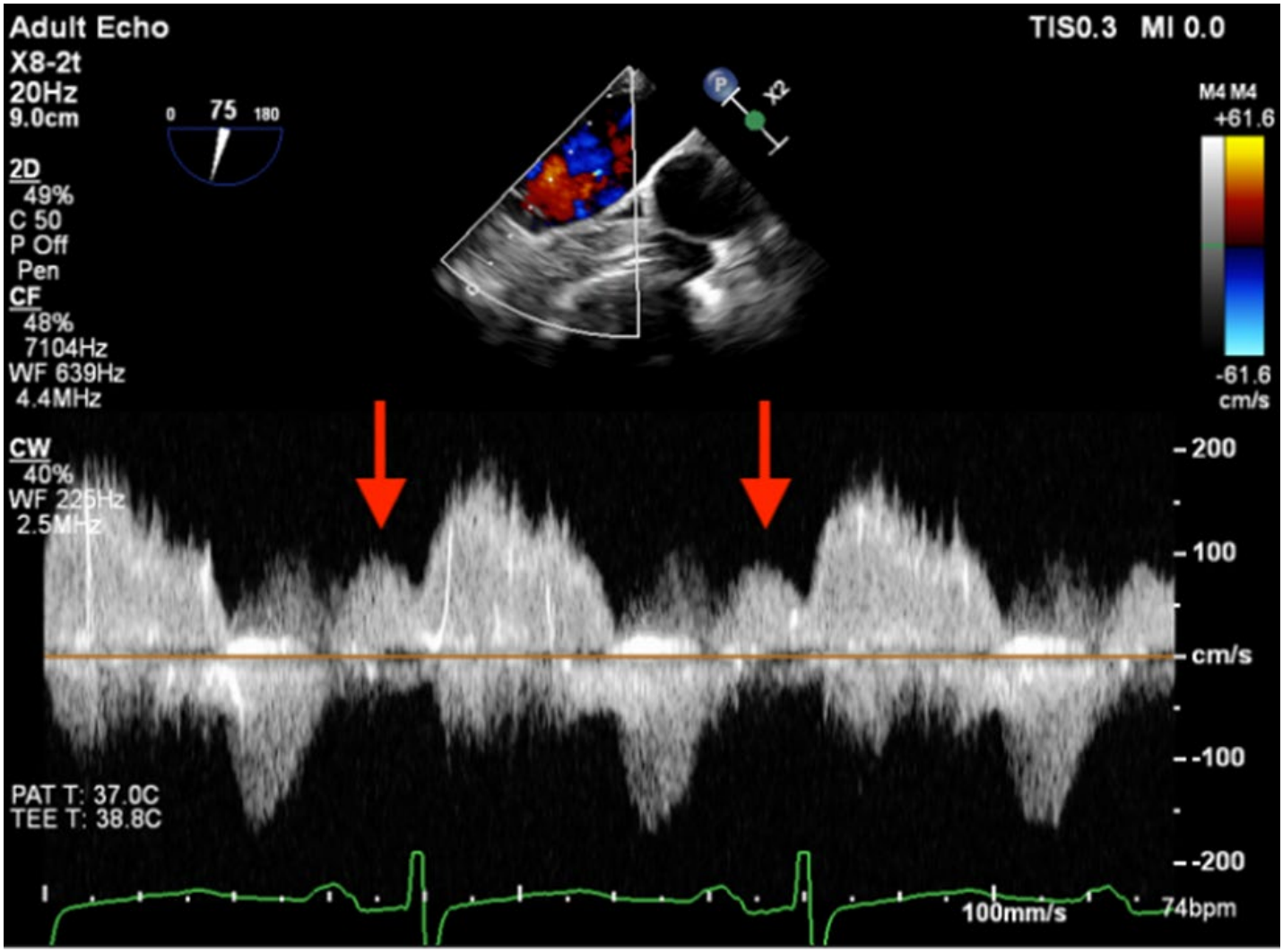
Discussion: Videos and Image uploads: 1 through 4 are from the current, presented case. They used RV inflow-outflow view and aortic arch in short axis, with color flow doppler (CFD) and CWD to evaluate TVR and PVR. The aortic arch view used CFD to evaluate PR and EDFF through the PV, and CWD to image the EDFF and the PR index ratio.

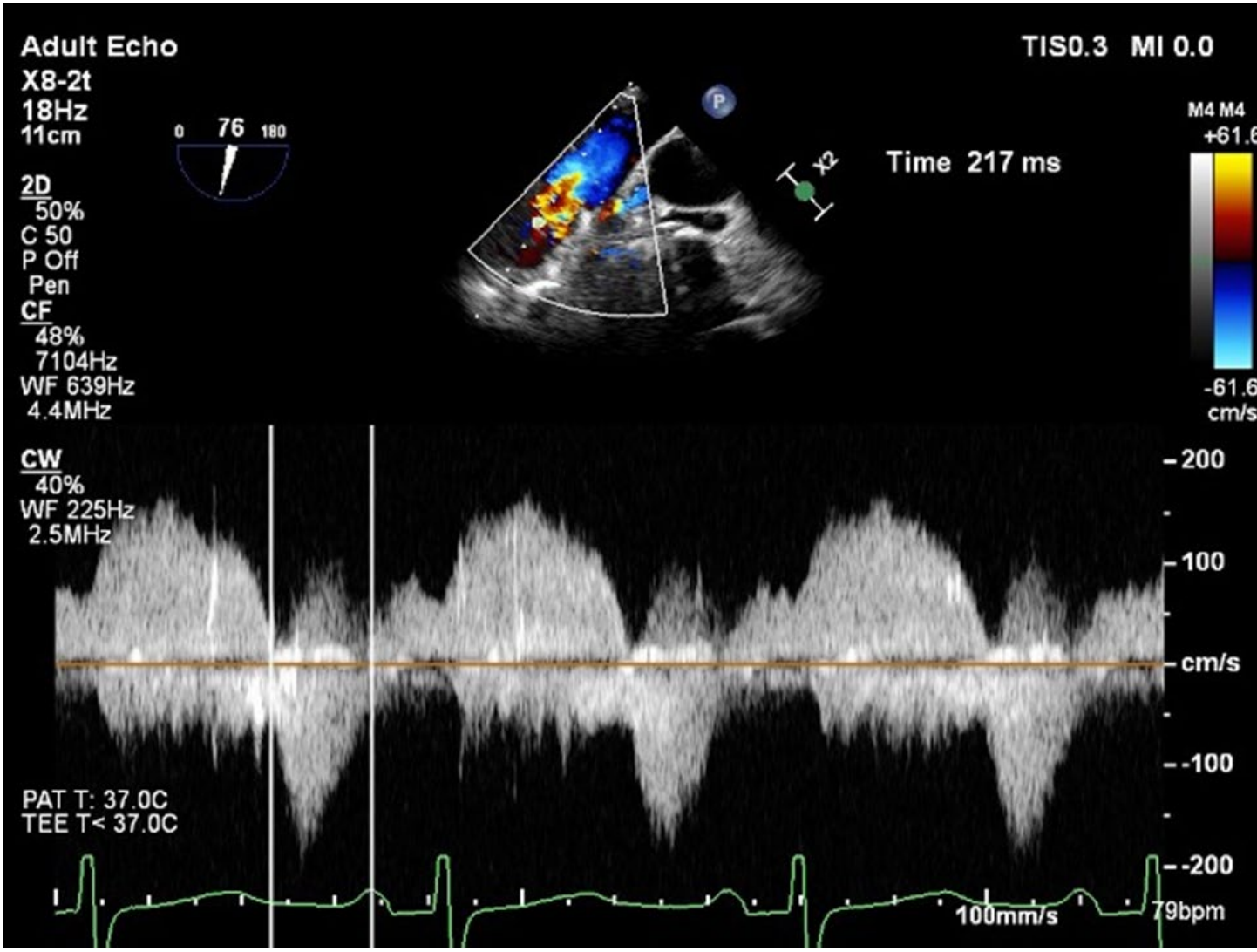
Image uploads: 5 and 6 are a prior case with CaHD and severe TR and severe PR, using the same views.

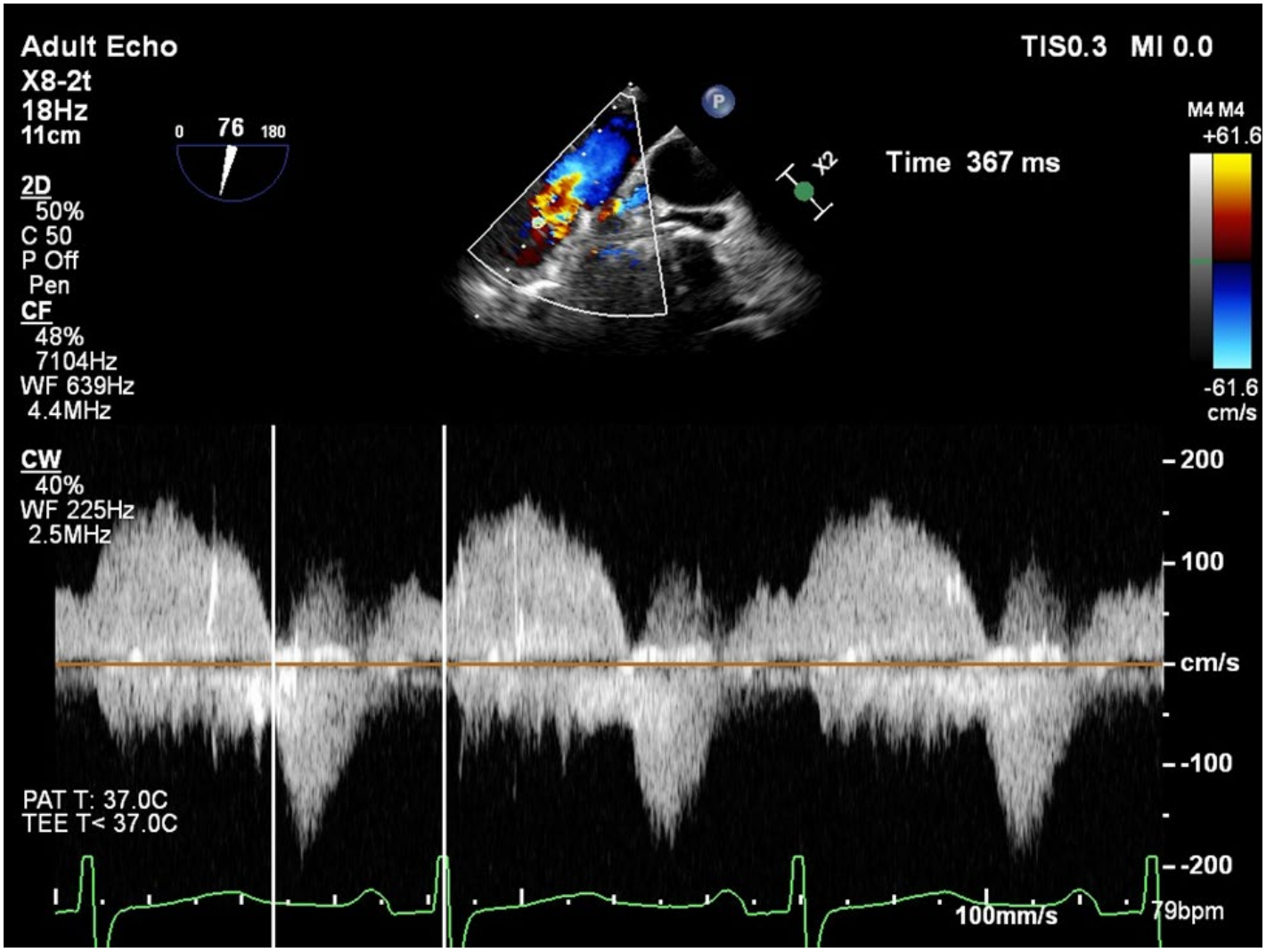
Impact of Echocardiographic findings: These findings can indicate the severity of the regurgitation and possibility of RV dysfunction, which can affect induction considerations, inotropic requirements and ability to separate from cardiopulmonary bypass. RV function has important implications in perioperative morbidity and mortality.

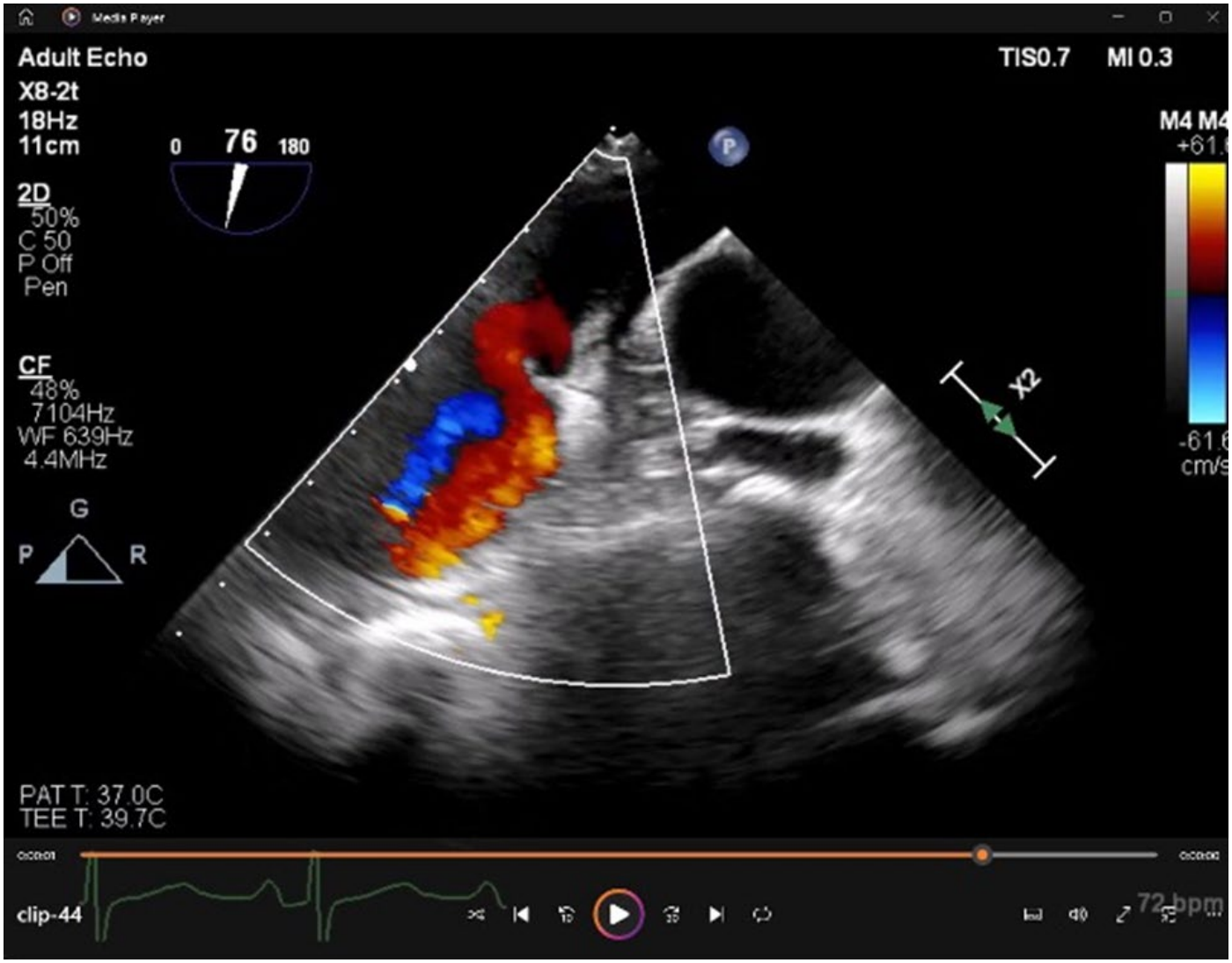
Teaching Points of the Case: Here we show CaHD with severe TR and PR with EDFF from a restricted PV in two separate cases. Notably, failure to recognize EDFF can cause underestimation of PR index. In the previous case (image 6) the PR index was incorrectly calculated because CWD was used to
determine diastole, instead of the EKG. Clinicians must use the EKG to determine the PR index in these cases as you cannot assume forward flow on CWD indicates the beginning of systole. We attribute the EDFF to the restricted and retracted PV leaflets, as can be seen in image 4 and 5, where the PV leaflets remain open at end-diastole. This leaves a channel through which the atrial kick pushes blood into the RV and through the PV. In cases of severe PR, there can be very rapid equalization of pressures, which can easily be missed on CFD. Therefore, it is critical to interrogate with CWD and correctly calculate the PR index, otherwise the severity of PR can be underestimated.

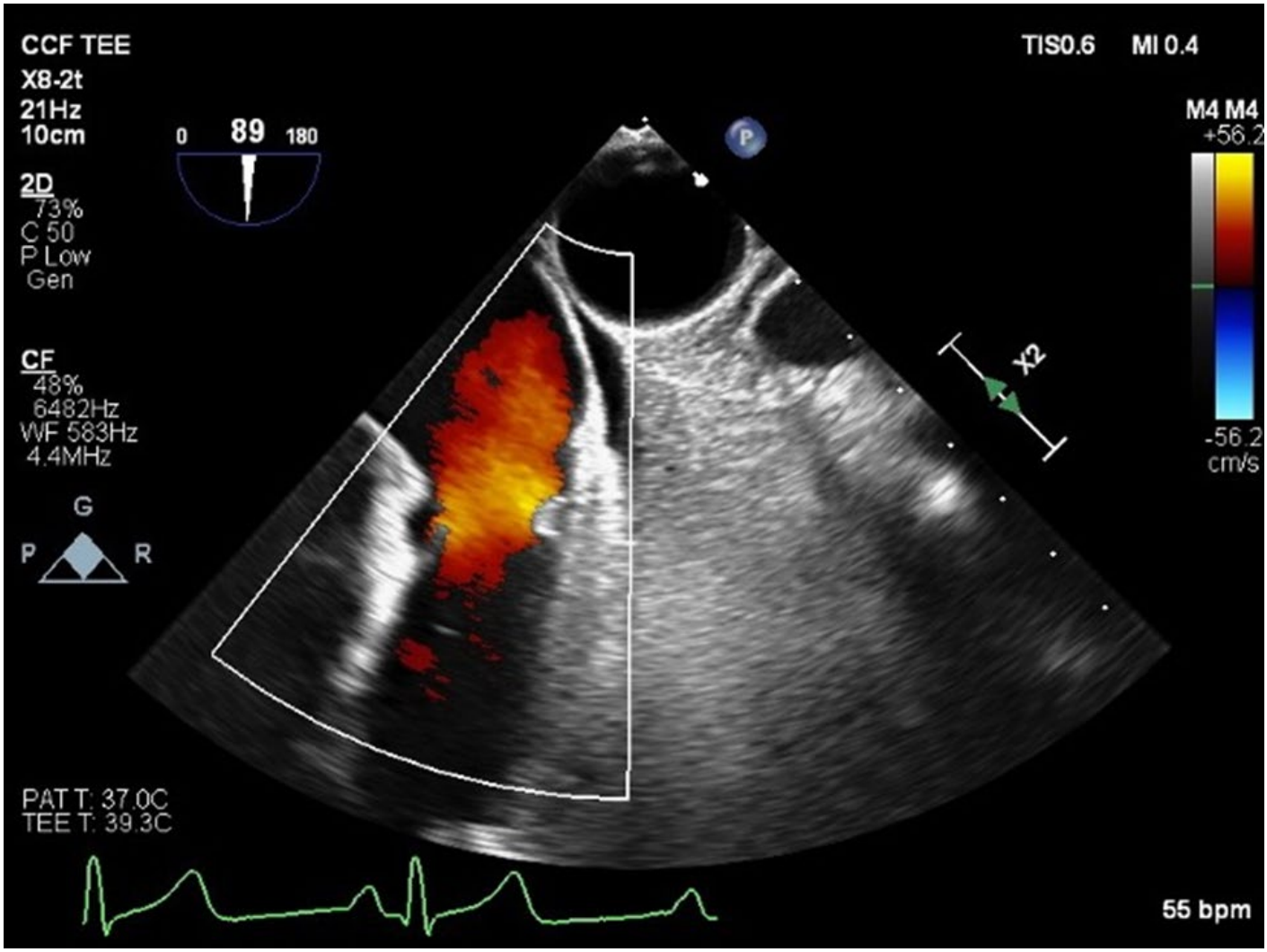
Image uploads:

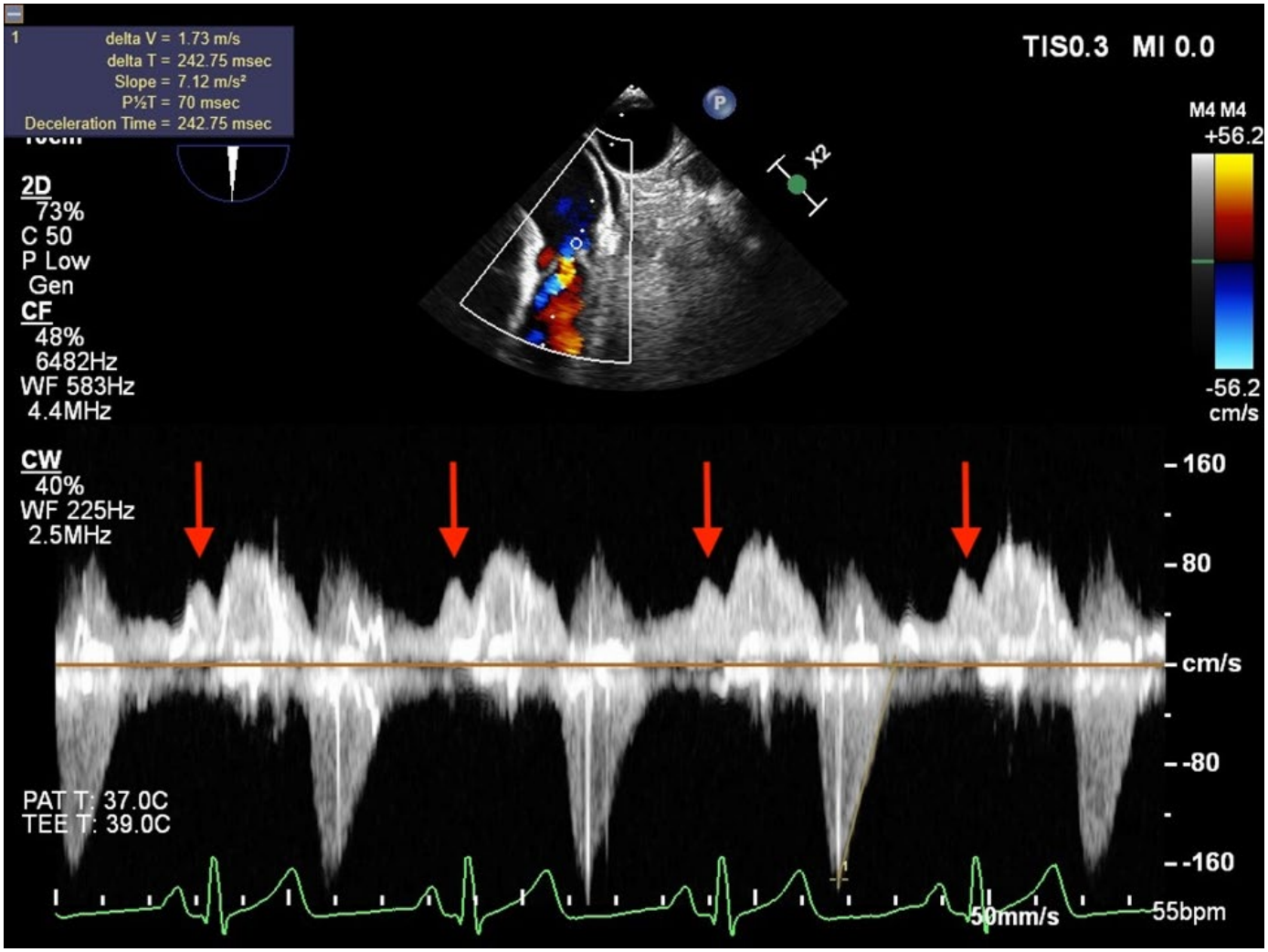

Adult Echo
X8-2t
20Hz
9.0cm
0
75
180
2D
49%
C 50
P Off
Pen
CF
48%
7104Hz
WF 639Hz
4.4MHz
CW
40%
WF 225Hz
2.5MHz
TIS0.3 MI 0.0
M4 M4
+61.6
-61.6
cm/s
200
100
cm/s
-100
-200
PAT T: 37.0C
TEE T: 38.8C
100mm/s
74bpm


Adult Echo
X8-2t
18Hz
11cm
0 76 180
2D
50%
C 50
P Off
Pen
CF
48%
7104Hz
WF 639Hz
4.4MHz
CW
40%
WF 225Hz
2.5MHz
PAT T: 37.0C
TEE T< 37.0C
TIS0.3 MI 0.0
Time 217 ms
M4 M4
+61.6
-61.6
cm/s
200
100
cm/s
-100
-200
100mm/s
79bpm


Adult Echo
X8-2t
18Hz
11cm
0 76 180
2D
50%
C 50
P Off
Pen
CF
48%
7104Hz
WF 639Hz
4.4MHz
CW
40%
WF 225Hz
2.5MHz
PAT T: 37.0C
TEE T< 37.0C
TIS0.3 MI 0.0
Time 367 ms
M4 M4
+61.6
-61.6
cm/s
200
100
cm/s
-100
-200
100mm/s
79bpm


Media Player
Adult Echo
X8-2t
18Hz
11cm
TIS0.7
MI 0.3
0
76
180
2D
50%
C 50
P Off
Pen
CF
48%
7104Hz
WF 639Hz
4.4MHz
G
P
R
X2
M4 M4
+61.
-61.
cm/s
PAT T: 37.0C
TEE T: 39.7C
clip-44
72 bpm


CCF TEE
X8-2t
21Hz
10cm
0
89
180
2D
73%
C 50
P Low
Gen
CF
48%
6482Hz
WF 583Hz
4.4MHz
G
P
R
TIS0.6
MI 0.4
M4 M4
+56.2
-56.2
cm/s
x2
PAT T: 37.0C
TEE T: 39.3C
55 bpm



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SP 1 - Kiosk 9 - Poster 1
Remimazolam for Procedural Sedation in the ASA 3 and 4 Population Undergoing Transesophageal Echocardiography and/or Cardioversion: A Retrospective Study
Presenting Author: *Conner Clay, MD*

Introduction: Remimazolam is a novel, ultra-short-acting benzodiazepine that was created for intravenous sedation. It works on the GABA type A receptor and is rapidly metabolized by tissue esterase to an inactive metabolite. It is often used for sedation in patients with poor cardiac function, with the advantages over other anesthetics of presumed hemodynamic stability, predictable onset, quick recovery, and minimal respiratory depression. Existing literature has been published on its use for sedation and induction of general anesthesia for endoscopic GI procedures and cardiac surgery. These studies have emphasized the need to study specific patient subgroups to optimize the drug's use across diverse clinical scenarios.

Methods: A retrospective cohort evaluation of ASA 3 or 4 patients who have undergone procedural sedation with remimazolam compared to propofol for either TEE, cardioversion, or TEE with cardioversion from 2021-2024 across all Mayo centers. Primary outcomes measured were percent drop mean arterial pressure (MAP) post-induction, vasopressor use during procedure, and desaturation events (SpO2 < 90%) during the procedure. Our secondary outcomes compared unplanned intubation events and length of stay in PACU.

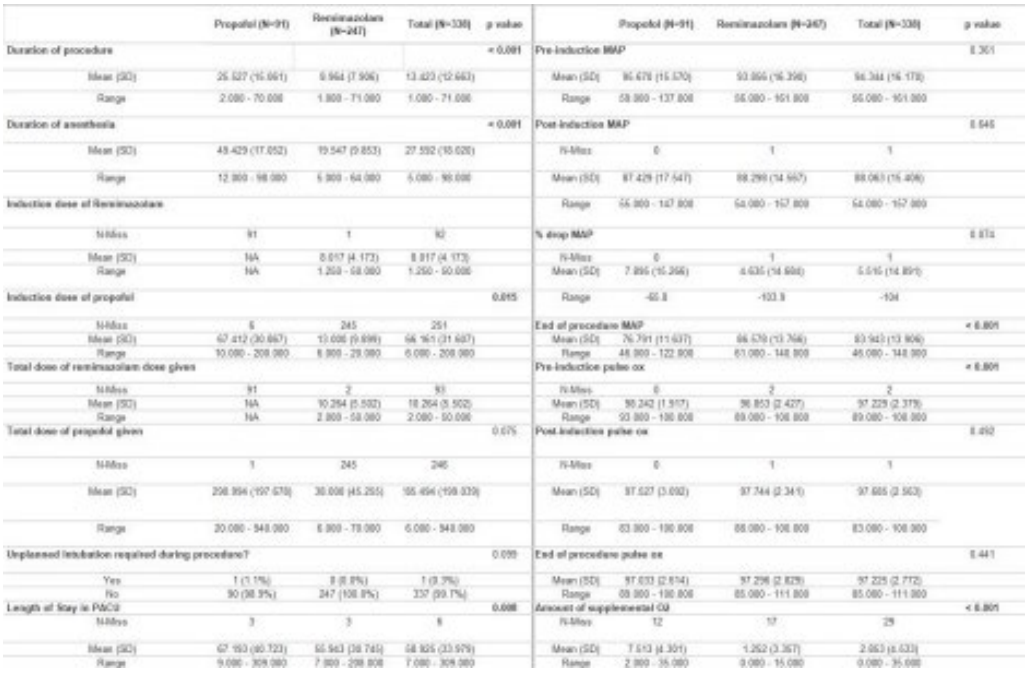
Results/Discussion: We identified 91 patients who received only propofol during their procedure and 247 patients who received only remimazolam. There was no significant difference in pre-induction MAP (p=0.36), post-induction MAP (p=0.65) or percent MAP drop (p=0.07). However, end of procedure MAP significantly differed, with a mean of 86.6 mmHg in the remimazolam group vs. 76.8 mmHg in the propo groups. Desaturation events occurred in 8 patients in the propofol group and none in the remimazolam group. While the limited number of desaturation events restricted statistical evaluation, the distribution of events suggests a potential respiratory safety advantage with the use of remimazolam. 1 unplanned intubation event was noted among propofol patients, compared to none among remimazolam patients (p=0.099). Length of PACU stay in minutes (67.1 vs. 55.9) was significantly shorter for the remimazolam group (p=0.008). The duration of TEE was the longest (29.6 min), followed by TEE with cardioversion (12.8 min), followed by cardioversion (6.8 min).

Conclusion: Remimazolam is noninferior to propofol for use in procedural sedation for TEE and/or Cardioversion for ASA 3 or 4 patients. Remimazolam resulted in increased end-procedure MAP compared to propofol. Furthermore, PACU stay is decreased among patients sedated with remimazolam. These findings support remimazolam as effective, time-efficient sedation with manageable hemodynamic effects in cardiac procedures.



Image uploads:

	Propofol (N=91)	Remimazolam (N=247)	Total (N=338)	p value
Gender				**0.049**
Female	40 (44.0%)	80 (32.4%)	120 (35.5%)	
Male	51 (56.0%)	167 (67.6%)	218 (64.5%)	
Age				0.815
Mean (SD)	70.495 (13.509)	70.814 (10.144)	70.728 (11.130)	
Range	34.000 - 94.000	33.000 - 93.000	33.000 - 94.000	
ASA				0.687
III	60 (65.9%)	157 (63.6%)	217 (64.2%)	
IV	31 (34.1%)	90 (36.4%)	121 (35.8%)	
Heart Failure				0.232
N-Miss	50	107	157	
HFpEF	15 (36.6%)	66 (47.1%)	81 (44.8%)	
HFrEF	26 (63.4%)	74 (52.9%)	100 (55.2%)	
Cardiomyopathy				**0.003**
N-Miss	3	24	27	
Yes ischemic	6 (6.8%)	36 (16.1%)	42 (13.5%)	
Yes non-ischemic	25 (28.4%)	88 (39.5%)	113 (36.3%)	
No	57 (64.8%)	99 (44.4%)	156 (50.2%)	
Ejection Fraction in last echo prior to procedure				**0.025**
N-Miss	2	12	14	
Mean (SD)	53.000 (14.023)	48.838 (15.084)	49.981 (14.895)	
Range	15.000 - 75.000	10.000 - 76.000	10.000 - 76.000	
Status of procedure				0.507
elective	89 (97.8%)	244 (98.8%)	333 (98.5%)	
urgent/emergent	2 (2.2%)	3 (1.2%)	5 (1.5%)	
What procedure did the patient undergo?				**< 0.001**
TEE	40 (44.0%)	2 (0.8%)	42 (12.4%)	
Cardioversion	16 (17.6%)	66 (26.7%)	82 (24.3%)	
TEE and cardioversion	35 (38.5%)	179 (72.5%)	214 (63.3%)	
Inpatient prior to procedure?				**< 0.001**
Yes	30 (33.0%)	25 (10.1%)	55 (16.3%)	
No	61 (67.0%)	222 (89.9%)	283 (83.7%)	



	Propofol (N=91)	Remimazolam (N=247)	Total (N=338)	p value
Duration of procedure				< 0.001
Mean (SD)	25.527 (15.661)	9.964 (7.906)	13.423 (12.663)	
Range	2.000 - 70.000	1.000 - 71.000	1.000 - 71.000	
Duration of anesthesia				< 0.001
Mean (SD)	48.429 (17.052)	19.547 (9.853)	27.592 (18.020)	
Range	12.000 - 98.000	5.000 - 64.000	5.000 - 98.000	
Induction dose of Remimazolam				
N-Miss	91	1	92	
Mean (SD)	NA	8.817 (4.173)	8.817 (4.173)	
Range	NA	1.250 - 50.000	1.250 - 50.000	
Induction dose of propofol				0.015
N-Miss	5	245	251	
Mean (SD)	67.412 (30.667)	10.000 (9.999)	66.161 (31.607)	
Range	10.000 - 200.000	5.000 - 20.000	5.000 - 200.000	
Total dose of remimazolam dose given				
N-Miss	91	2	93	
Mean (SD)	NA	10.264 (5.502)	10.264 (5.502)	
Range	NA	2.000 - 50.000	2.000 - 50.000	
Total dose of propofol given				0.075
N-Miss	1	245	246	
Mean (SD)	208.994 (197.679)	30.000 (45.255)	55.494 (109.939)	
Range	20.000 - 940.000	5.000 - 70.000	5.000 - 940.000	
Unplanned Intubation required during procedure?				0.055
Yes	1 (1.1%)	8 (3.2%)	1 (3.3%)	
No	90 (98.9%)	247 (100.8%)	337 (99.7%)	
Length of Stay in PACU				0.008
N-Miss	3	3	6	
Mean (SD)	67.193 (60.723)	55.943 (39.745)	58.925 (33.979)	
Range	9.000 - 309.000	7.000 - 208.000	7.000 - 309.000	

	Propofol (N=91)	Remimazolam (N=247)	Total (N=338)	p value
Pre-Induction MAP				0.301
Mean (SD)	96.670 (15.370)	93.866 (16.398)	94.344 (16.178)	
Range	58.000 - 137.000	56.000 - 161.000	56.000 - 161.000	
Post-Induction MAP				0.645
N-Miss	0	1	1	
Mean (SD)	87.429 (17.547)	88.299 (14.667)	88.063 (15.406)	
Range	55.000 - 147.000	54.000 - 157.000	54.000 - 157.000	
% drop MAP				0.874
N-Miss	0	1	1	
Mean (SD)	7.896 (15.268)	4.635 (14.684)	5.515 (14.891)	
Range	-60.0	-103.0	-104	
End of procedure MAP				< 0.001
Mean (SD)	76.791 (11.637)	66.570 (13.766)	69.943 (13.906)	
Range	48.000 - 122.000	51.000 - 148.000	46.000 - 148.000	
Pre-Induction pulse ox				< 0.001
N-Miss	0	2	2	
Mean (SD)	98.242 (1.517)	96.853 (2.427)	97.229 (2.375)	
Range	93.000 - 100.000	89.000 - 100.000	89.000 - 100.000	
Post-Induction pulse ox				0.492
N-Miss	0	1	1	
Mean (SD)	97.527 (3.092)	97.744 (2.341)	97.685 (2.563)	
Range	83.000 - 100.000	88.000 - 100.000	83.000 - 100.000	
End of procedure pulse ox				0.441
Mean (SD)	97.033 (2.614)	97.296 (2.829)	97.225 (2.772)	
Range	88.000 - 100.000	85.000 - 111.000	85.000 - 111.000	
Amount of supplemental O2				< 0.001
N-Miss	12	17	29	
Mean (SD)	7.513 (4.301)	1.252 (3.367)	2.653 (4.633)	
Range	2.000 - 35.000	0.000 - 15.000	0.000 - 35.000	

	TEE (N=42)	Cardioversion (N=82)	TEE and cardioversion (N=214)	Total (N=338)	p value
Duration of procedure					**< 0.001**
Mean (SD)	29.595 (12.543)	6.805 (8.974)	12.785 (11.133)	13.423 (12.663)	
Range	11.000 - 69.000	1.000 - 71.000	1.000 - 70.000	1.000 - 71.000	
Duration of anesthesia					**< 0.001**
Mean (SD)	55.619 (13.874)	23.354 (8.906)	23.715 (16.421)	27.592 (18.020)	
Range	33.000 - 98.000	5.000 - 48.000	6.000 - 90.000	5.000 - 98.000	
Length of Stay in PACU					**0.001**
N-Miss	3	1	2	6	
Mean (SD)	77.744 (52.153)	55.827 (23.123)	56.646 (32.315)	58.925 (33.979)	
Range	9.000 - 309.000	7.000 - 126.000	9.000 - 208.000	7.000 - 309.000	

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SP 1 - Kiosk 9 - Poster 2

Multicenter Study: Long-Term and Short-Term Outcomes of Perioperative Red Blood Cell Transfusion during Lung Transplant

Presenting Author: *Yue Qiu, MD*

Background: Lung transplant is associated with a significant risk of transfusion and transfusion may be associated with poor outcomes. While previous single-center studies have demonstrated associations between perioperative packed red blood cell (PRBC) transfusion and adverse short-term outcomes, including primary graft dysfunction (PGD)(1,2) and mortality (3), the impact on long-term graft function and overall survival remains inadequately characterized. Existing evidence shows conflicting results, with some studies suggesting increased mortality with high-volume transfusions (5) while others found no significant differences in overall mortality(6). This study evaluated whether perioperative RBC transfusion independently predicts short-term and long-term outcomes after lung transplantation.

Methods: A multicenter retrospective study was conducted across 8 major lung transplantation centers in good standing with the United Network for Organ Sharing, where more than 40 lung transplants are performed annually. Adult patients who underwent lung transplantation between 2015-2018 were included. Preoperative patient characteristics, intraoperative variables including

blood product transfusion, and postoperative survival and organ outcomes were collected. Exposure was any RBC transfusion intraoperatively or within 72 hours postoperatively. The primary outcome was 1-year mortality, and the secondary outcomes included 1-year graft failure and short-term clinical outcomes. Inverse probability of weighting (IPW)-adjusted logistic regression estimated the relationship between perioperative RBC transfusion and clinical outcomes. Covariate balance was confirmed using standardized mean differences and visualized with a Love plot. (Figure 1)

Results: The analysis cohort included 695 lung transplant recipients (mean age 58 years; 59% male; 75% White), and 69% participants received perioperative RBC transfusion (baseline characteristics by RBC exposure - Table 1). One-year mortality was significantly increased in patients receiving RBC transfusions as compared to those who did not; mortality 14.6% RBC group vs. 6.1% non-RBC, p = 0.002 (Table 2). This association remained significant after IPW and multivariable adjustment (aOR=3.52, 95% CI: 1.12–11.08, p =0.032, Table 2). Additionally, RBC transfusion was significantly associated with primary graft dysfunction within 72 hrs (aOR=2.30, 95%CI: 1.28-4.11, p=0.005).

Conclusion: This multicenter study provides robust evidence that perioperative RBC transfusion is independently associated with significantly increased one-year mortality and PGD within 72 hours following lung transplantation. These findings underscore the importance of implementing blood conservation strategies and restrictive transfusion protocols in lung transplant recipients.

Image uploads:

Table 1: Baseline Characteristics of the Cohort by RBC Exposure

Level	Unweighted No RBC (n = 214)	Unweighted Any RBC (n = 481)	Unweighted p	Weighted No RBC (wN = 612.6)	Weighted Any RBC (wN = 690.2)	Weighted p
	61.26 (9.67)	56.42 (12.43)	<0.001	59.31	57.78	0.166
Female	63 (29.4%)	224 (46.6%)	<0.001	242.2 (39.5%)	288.1 (41.7%)	
Male	151 (70.6%)	257 (53.4%)	<0.001	370.4 (60.5%)	402.1 (58.3%)	
Other	164 (76.6%)	360 (74.8%)	0.879	472.6 (77.2%)	521.8 (75.6%)	
Caucasian	10 (4.7%)	24 (5.0%)	0.879	26.8 (4.4%)	32.5 (4.7%)	0.918
Hispanic	40 (18.7%)	97 (20.2%)	0.879	113.2 (18.5%)	135.8 (19.7%)	
Other	26.37 (3.83)	25.17 (4.55)	0.001	26.12	25.41	
	38.95 (13.00)	46.07 (18.63)	<0.001	41.12	44.04	0.045
	17 (7.9%)	26 (5.4%)	0.003	58.2 (9.5%)	37.1 (5.4%)	<0.001
Iowa	8 (3.7%)	48 (10.0%)	0.003	22.0 (3.6%)	73.6 (10.7%)	<0.001
Louisville	37 (17.3%)	65 (13.5%)		98.7 (16.1%)	99.7 (14.4%)	
Ohio State	32 (15.0%)	75 (15.6%)		92.4 (15.1%)	109.1 (15.8%)	
Colorado	22 (10.3%)	30 (6.2%)		68.2 (11.1%)	45.9 (6.6%)	
Kentucky	62 (29.0%)	112 (23.3%)		181.8 (29.7%)	146.8 (21.3%)	
Florida	35 (16.4%)	124 (25.8%)		90.3 (14.7%)	176.9 (25.6%)	
Minnesota	1 (0.5%)	1 (0.2%)		1.0 (0.2%)	1.0 (0.1%)	
Pittsburgh	61.26 (9.67)	56.42 (12.43)		59.31	57.78	

BMI= Body Mass Index, LAS score = Lung allocation score, SD = standard deviation, RBC= Red Blood Cell

Table 2: Associations between RBC transfusion and post-operative outcomes (n=695)

Outcome	Unadjusted Associations			IPW-Adjusted Associations	
	No RBC (n =214, 31%)	Any RBC (n = 481, 69%)	Chi-Squared p-value	OR (95% CI)	p-value
1-year Mortality	13 (6.1%)	70 (14.6%)	0.002	3.52 (1.12–11.08)	0.032
1-year Graft Failure	8 (3.8%)	44 (9.2%)	0.019	3.70 (0.86–15.86)	0.078
Endobronchial Biopsy Evidence of Rejection	23 (10.9%)	52 (11.0%)	0.99	1.59 (0.74–3.42)	0.23
PGDwithin 72 hrs	79 (36.9%)	258 (54.0%)	<0.001	2.30 (1.28–4.11)	0.005
ECMO during hospitalization	7 (3.3%)	84 (17.6%)	<0.001	4.71 (0.42–52.31)	0.206

RBC = red blood cell; IPW = inverse probability weighting; OR = odds ratio; CI = confidence interval; PGD = primary graft dysfunction; ECMO = extracorporeal membrane oxygenation.

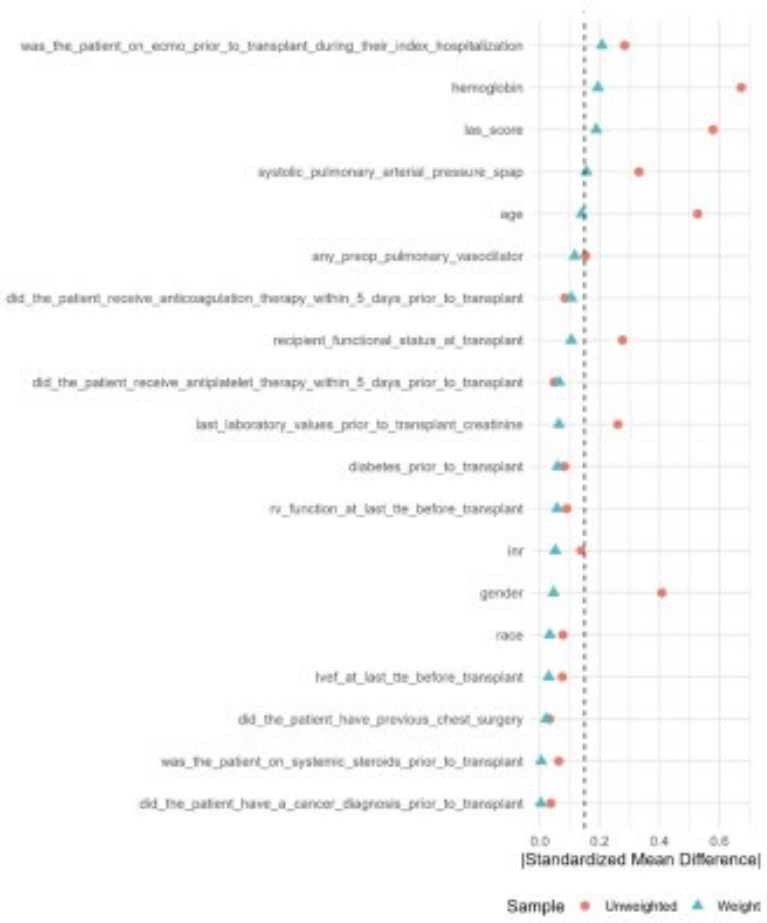


Figure 1: Figure 1. Love plot of weighted and unweighted standardized mean differences (SMDs) for all covariates included in the propensity score model for any RBC transfusion. The vertical dashed line marks an SMD of 0.15, the threshold used to define acceptable covariate balance in this analysis.

SMD = standardized mean difference; RBC = red blood cell; IPW = inverse probability weighting; BMI = body mass index; MPAP = mean pulmonary arterial pressure; LVEF = left ventricular ejection fraction; INR = international normalized ratio; CAD = coronary artery disease.

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SP 1 - Kiosk 9 - Poster 3

Patterns in the use of postoperative intravenous iron after cardiac surgery from 2005-2025

Presenting Author: *Una Choi, MD*

Background: Postoperative anemia affects up to 90% of patients undergoing cardiac surgery and is associated with worse clinical outcomes. This anemia is predominantly iron-restricted in nature. Oral iron may be employed but has limited efficacy in the postoperative setting and low levels of treatment adherence. Alternatively, intravenous (IV) iron provides rapid and complete iron repletion; however, its use following cardiac surgery remains poorly quantified. In this analysis, we quantify temporal trends in intravenous (IV) iron utilization and corresponding iron deficiency laboratory values from 2005-2025.

Methods: A large claims and electronic health record database (TriNetX Research Network) was queried with three periods of interest: (1) 05/01/2005-05/01/2015; (2) 05/01/2015-05/01/2020; and (3) 05/01/2020-05/01/2025. We examined adult (aged ≥18 years) cardiac surgery patients with moderate-to-severe postoperative anemia (i.e., hemoglobin (hgb) ≤11 g/dL) within 1-5 days after surgery. We extracted all postoperative IV iron infusions administered in the first 30 days after surgery, including iron sucrose, iron-dextran, sodium ferric gluconate complex, ferumoxytol, and ferric carboxymaltose. To assess changes in iron deficiency laboratory collection and treatment of patients with laboratory-defined postoperative iron deficiency anemia (IDA), we conducted a sub-analysis of patients with postoperative hgb ≤11 g/dL and a transferrin saturation ≤20% or ferritin ≤30 ng/mL. Chi-square tests were performed to compare IV iron infusion rates with significance defined as $p < 0.05$.

Results: A total of 271,507 patients were included. There were significant increases in IV iron utilization over time ($p< 0.001$), including rates of 1.65% from 2005-2015, 3.92% from 2015-2020, and 4.85% from 2020-2025 (Table 1). Next, we assessed temporal changes in assessment of postoperative iron labs (ferritin or transferrin saturation) in those with moderate-to-severe postoperative anemia. Similarly, there were significant increases over time ($p< 0.001$), including 1.47% in 2005-2015, 2.94% in 2015-2020, and 4.15% in 2020-2025. Among those with iron labs obtained after surgery, 55.28% met laboratory criteria for IDA. The overall treatment rate with postoperative IV iron was 27.01%. Although treatment of postoperative IV iron increased modestly over time (Table 2), these changes were not statistically significant.

Conclusion: In this large database study, we demonstrate increases in postoperative IV iron use among cardiac surgical patients from 2005-2025. However, utilization remained low, with approximately 4.85% of patients with moderate-to-severe postoperative anemia receiving IV iron. Laboratory testing for iron deficiency remained similarly uncommon, employed in less than 5% of patients. When postoperative IDA was discovered, only 1 in 4 patients received IV iron therapy, with no significant changes over time. The persistently low rates of iron testing and IV iron utilization represent a modifiable target for quality improvement, particularly if future trials demonstrate improvements in clinical outcomes such as mortality, readmission, or recovery with postoperative IV iron therapy in this high-risk population.

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Table 1 – 30-day postoperative intravenous iron infusions among cardiac surgical patients with postoperative hemoglobin ≤11 g\dL.

Years	# years	n (hgb≤11)	Medication	n	%	Mean ± SD (days)	Median	Min	Max
2005-2015	10	40926	iron sucrose	364	0.89%	11 ± 8	10	1	30
			iron sucrose and iron-dextran	233	0.57%	4 ± 6	1	1	29
			sodium ferric gluconate complex	49	0.12%	7 ± 7	4	1	27
			ferumoxytol	22	0.05%	11 ± 7	10	1	23
			iron-dextran complex	9	0.02%	9 ± 8	6	1	21
			ferric carboxymaltose	0	0.00%	--	--	--	--
			Any IV iron	*677*	1.65%	--	--	--	--
2015-2020	5	95598	iron sucrose	1856	1.94%	8 ± 8	5	1	30
			iron sucrose and iron-dextran	1100	1.15%	4 ± 6	1	1	30
			sodium ferric gluconate complex	677	0.71%	7 ± 8	3	1	30
			iron-dextran complex	49	0.05%	8 ± 8	4	1	29
			ferumoxytol	33	0.03%	15 ± 8	17	1	29
			ferric carboxymaltose	32	0.03%	13 ± 10	11	1	30
			Any IV iron	*3747*	*3.92%*	--	--	--	--
2020-2025	5	134983	iron sucrose	2643	1.96%	9 ± 8	5	1	30
			sodium ferric gluconate complex	2185	1.62%	7 ± 8	3	1	30
			iron sucrose and iron-dextran	1318	0.98%	7 ± 8	3	1	30
			iron-dextran complex	198	0.15%	9 ± 8	5	1	30
			ferumoxytol	110	0.08%	13 ± 9	12	1	30
			ferric carboxymaltose	86	0.06%	15 ± 9	16	1	29
			Any IV iron	*6540*	4.85%	--	--	--	--

Table 2 – 30-day postoperative intravenous iron infusions among cardiac surgical patients with postoperative hemoglobin ≤11 g/dL and iron deficiency (i.e., ferritin ≤30 ng/ml or TSAT ≤20%)

Years	# years	n	Medication	n	%	Mean ± SD (days)	Median	Min	Max
2005-2015	10	232	iron sucrose	44	0.11%	3 ± 7	1	1	29
			sodium ferric gluconate complex	6	0.01%	1 ± 1	1	1	2
			ferumoxytol	1	0.00%	7	7	7	7
			iron sucrose and iron-dextran	1	0.00%	1	1	1	1
			ferric carboxymaltose	0	0.00%	--	--	--	--
			Any IV iron	*52*	22.41%	--	--	--	--
2015-2020	5	1445	iron sucrose	221	0.23%	4 ± 5	1	1	30
			sodium ferric gluconate complex	105	0.11%	3 ± 5	1	1	27
			iron sucrose and iron-dextran	27	0.03%	4 ± 6	1	1	20
			ferric carboxymaltose	7	0.01%	3 ± 4	1	1	13
			iron-dextran complex	7	0.01%	7 ± 9	1	1	20
			ferumoxytol	3	0.00%	4 ± 3	3	2	7
			Any IV iron	*370*	*25.61%*	--	--	--	--
2020-2025	5	3310	sodium ferric gluconate complex	367	0.27%	4 ± 5	1	1	30
			iron sucrose	360	0.27%	4 ± 6	1	1	26
			iron sucrose and iron-dextran	151	0.11%	4 ± 6	1	1	28
			iron-dextran complex	27	0.02%	5 ± 7	2	1	24
			iron sucrose and sodium ferric gluconate complex	7	0.01%	4 ± 5	2	1	15
			ferumoxytol	7	0.01%	9 ± 10	4	1	26
			ferric carboxymaltose	6	0.00%	19 ± 7	2	6	28
			Any IV iron	*925*	27.95%	--	--	--	--

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SP 1 - Kiosk 9 - Poster 4

How is Angiotensin II used perioperatively in cardiac surgery? A tertiary medical center's experience.

Presenting Author: *Aubrey Greer, MD*

Background: Pharmacologic hemodynamic support with vasoactive medications is critical in the perioperative care of cardiac surgery patients. While various drugs and regimens are available, there are significant knowledge gaps in perioperative practice patterns with no clear data supporting one strategy over another. Angiotensin II (AT2) is a relatively new vasopressor with a

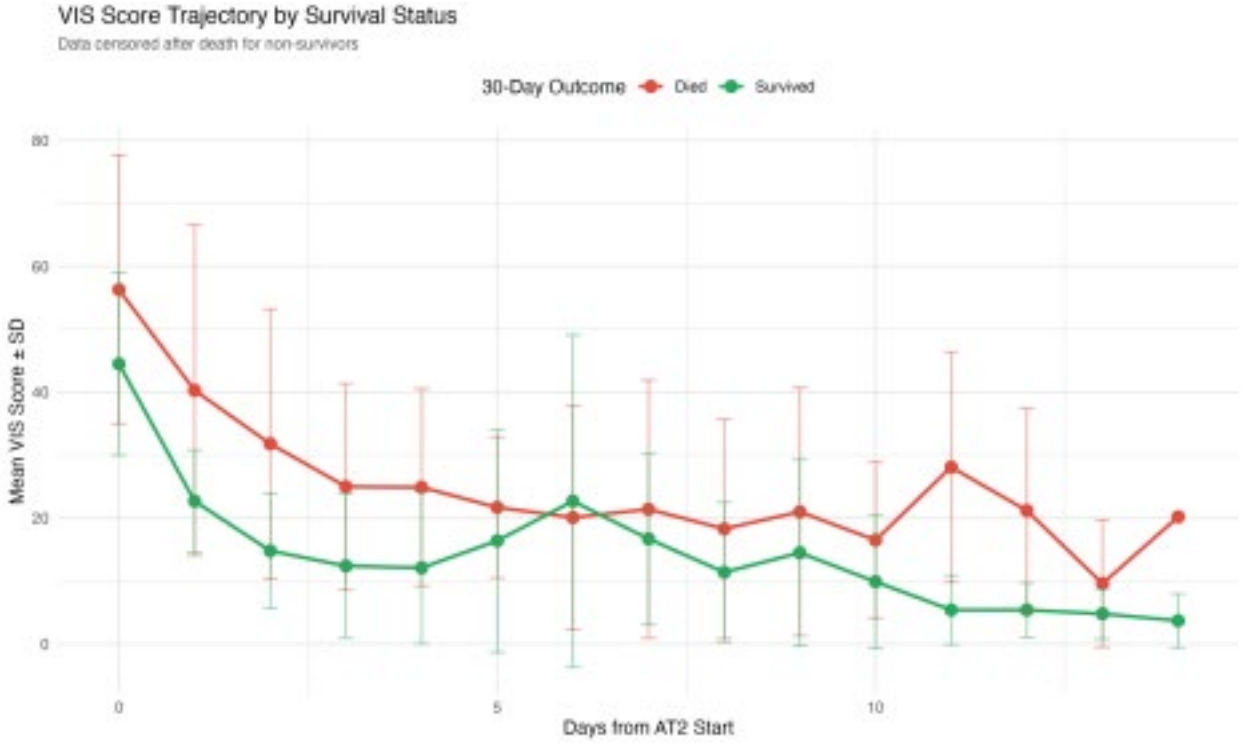
unique mechanism of action indicated for distributive shock. This study aims to describe practice patterns of perioperative AT2 use for cardiac surgery patients at a tertiary academic center and is the preliminary analysis for a multi-institutional project.

Methods: The institutional research patient data registry (RPDR) and EHR were queried to identify cardiac surgery patients who underwent a procedure requiring cardiopulmonary bypass (CPB) and received angiotensin II during the same admission. The extracted data included patient demographics, procedure information, biventricular function, presence and type of mechanical circulatory support, vasoactive-inotropic scores (VIS), methylene blue/hydroxocobalamin administration, and outcomes. Analysis was performed using RStudio.

Results: Between 2024-2025, 38 patients underwent 41 cardiac surgeries with CPB and received AT2. The cohort was predominantly male (86.8%) with mean age 63 years. Procedures included complex multi-component (39%), heart transplant (22%), and isolated procedures (24%), valve + CABG (7%), and combined valve procedures (7%). Mechanical support was common (63% ECMO), with 95% receiving concurrent methylene blue. Three AT2 timing phenotypes emerged: intraoperative-only, postoperative-only, and persistent (intra-op and post-op). Among the 38 patients who received AT2, significant VIS reduction was observed across all timing cohorts. The intraop-only group (n=5) showed a 36% VIS reduction (p=0.04), while the postop AT2 group (n=33) demonstrated 33% reduction within 24 hours (p< 0.001). Overall 30-day mortality was 68%, with marked differences by timing: intraop-only 20%, postop-only 73%, and persistent 50%. Among the patients receiving postoperative AT2 (n=33), the non-survivors (n=24, 73% of postop cohort) had a trend towards higher baseline VIS (56.3 vs 44.5, p=0.08), blunted response (28% vs 49% reduction), and persistent high VIS (>20) after Day 3, suggesting multi-organ failure and refractory vasoplegia.

Conclusions: In this preliminary cohort, AT2 produced significant VIS reductions (33%, p< 0.001), confirming pharmacologic efficacy. Thirty-day mortality varied by timing phenotype (intraop-only 20%, postop-only 73%, persistent 50%), though small subgroup sizes limit interpretation. Non-survivors trended toward higher baseline VIS and attenuated response, raising the possibility that treatment timing and baseline hemodynamic severity may help identify patients with refractory vasoplegia. Larger multi-institutional studies are needed to clarify these associations.

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References

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SP 1 - Kiosk 9 - Poster 5

Intraoperative Hemodynamics in Thoracic Surgery: Do Higher MAP Targets Improve or Worsen Postoperative Outcomes?

Presenting Author: *Kwame Wiredu, MD, PhD*

Introduction: Optimal intraoperative mean arterial pressure (MAP) targets for thoracic surgery are undefined, despite the unique hemodynamic perturbations imposed by one-lung ventilation, non-supine positioning, and minimally invasive approaches often involving CO2 insufflation and the accompanying alterations in intrathoracic pressures. Current American Heart Association/American Stroke Association guidance for noncardiac surgery recommends maintaining MAP ≥70 mmHg to reduce perioperative cerebrovascular risk, 1 a threshold commonly extrapolated to thoracic procedures. However, higher MAP targets have not consistently improved outcomes in other surgical populations,2,3 raising concern that aggressive blood pressure augmentation during thoracic surgery may be unnecessary or harmful. This study tested the hypothesis that higher intraoperative MAP is associated with worse postoperative outcomes in thoracic surgery.

Methods: This secondary analysis used high-fidelity arterial blood pressure waveforms and perioperative clinical data from the VitalDB open database,4 comprising 6,388 noncardiac surgical patients at Seoul National University Hospital (2016–2017). Thoracic cases were restricted to open, robotic, or videoscopic procedures. For each patient, arterial pulse waves between surgical start and end were processed using a three-step algorithm: second-order recursive low-pass Butterworth filtering, slope-sum transformation, and adaptive-threshold pulse detection, followed by computation of pulse-wise MAP and case-level time-weighted MAP (Figure 1A, B). Primary outcomes were hospital length of stay, ICU length of stay, and in-hospital mortality. Multivariable logistic, negative binomial, and Cox proportional hazards models assessed associations between twMAP and outcomes, adjusting for age, sex, BMI, ASA status, intraoperative vasopressor use, and other clinical covariates, with analyses stratified by surgical approach.

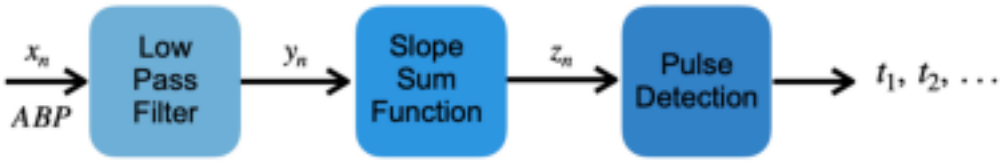
Results: Among 640 thoracic surgery patients, 97 underwent open, 12 robotic, and 531 videoscopic procedures. Open cases more often involved ASA III or higher, emergency surgery, and greater intraoperative blood loss, whereas age, BMI, and preoperative pulmonary function were similar across approaches. In-hospital mortality was 5% (n=30). Unadjusted analyses demonstrated significant differences in ICU stay, hospital stay, and mortality by approach. Intraoperative twMAP clustered around 85–91 mmHg, reflecting a high-pressure strategy. After adjustment, higher twMAP was independently associated with longer hospital length of stay, but not ICU length of stay, and Cox modeling showed no mortality benefit ($p = 0.581$).

Conclusion: Thoracic surgery patients in this large physiologic cohort were managed at relatively high intraoperative MAPs, and higher twMAP was linked to prolonged hospitalization without

improvement in ICU stay or survival. These findings challenge the assumption that higher MAP targets are intrinsically protective in thoracic surgery and suggest that aggressive blood pressure augmentation may be detrimental. Prospective trials are needed to define thoracic-specific MAP targets and to evaluate whether more conservative pressure strategies can enhance recovery and optimize resource utilization.

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A


x_n
ABP
Low Pass Filter
y_n
Slope Sum Function
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Pulse Detection
$t_1, t_2, \ldots$

B

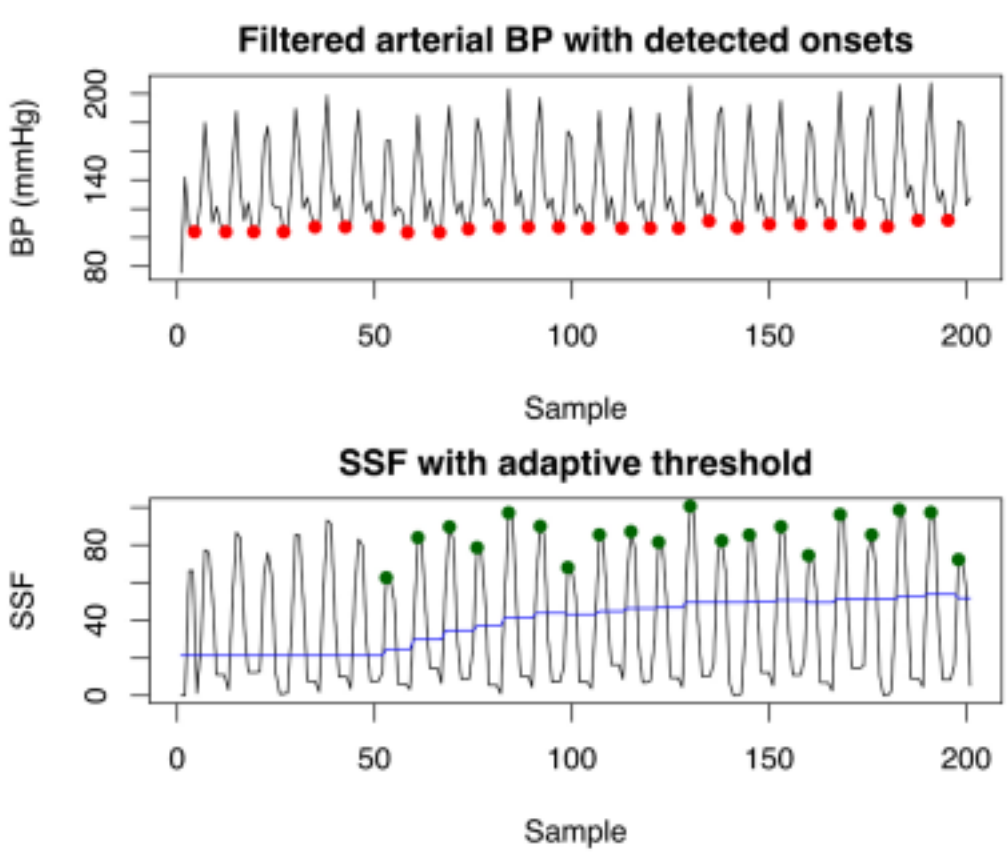

Filtered arterial BP with detected onsets
BP (mmHg)
80
140
200
0
50
100
150
200
Sample
SSF with adaptive threshold
SSF
0
40
80
0
50
100
150
200
Sample

Figure 1

	Open (N=97)	Robotic (N=12)	Videoscopic (N=531)	Overall (N=640)	P-value
Age (years)					
Mean (SD)	58 (± 15)	62 (± 15)	59 (± 14)	59 (± 14)	0.537
Sex					
Female	27 (28 %)	3 (25 %)	248 (47 %)	278 (43 %)	0.004
Male	70 (72 %)	9 (75 %)	283 (53 %)	362 (57 %)	
BMI (Kg/m2)					
Mean (SD)	22 (± 3.1)	24 (± 2.4)	23 (± 3.1)	23 (± 3.1)	0.027
ASA Physical Status					
1	10 (10 %)	1 (8 %)	95 (18 %)	106 (17 %)	0.000
2	60 (62 %)	10 (83 %)	385 (73 %)	455 (71 %)	
3	25 (26 %)	1 (8 %)	50 (9 %)	76 (12 %)	
4	2 (2 %)	0 (0 %)	1 (0 %)	3 (0 %)	
Emergency Operation					
No	83 (86 %)	12 (100 %)	498 (94 %)	593 (93 %)	0.028
Yes	14 (14 %)	0 (0 %)	33 (6 %)	47 (7 %)	
Preoperative PFT					
Borderline obstructive	0 (0 %)	0 (0 %)	2 (0 %)	2 (0 %)	0.173
Mild obstructive	5 (5 %)	2 (17 %)	27 (5 %)	34 (5 %)	
Mild restrictive	6 (6 %)	0 (0 %)	14 (3 %)	20 (3 %)	
Mixed or pure obstructive	5 (5 %)	0 (0 %)	10 (2 %)	15 (2 %)	
Moderate obstructive	3 (3 %)	0 (0 %)	10 (2 %)	13 (2 %)	
Moderate restrictive	2 (2 %)	0 (0 %)	3 (1 %)	5 (1 %)	
Normal	73 (75 %)	10 (83 %)	464 (87 %)	547 (85 %)	
Severe restrictive	3 (3 %)	0 (0 %)	1 (0 %)	4 (1 %)	
Preoperative Hypertension					
No	72 (74 %)	5 (42 %)	367 (69 %)	444 (69 %)	0.143
Yes	25 (26 %)	7 (58 %)	164 (31 %)	196 (31 %)	
Preoperative Diabetes					
No	87 (90 %)	11 (92 %)	472 (89 %)	570 (89 %)	0.987
Yes	10 (10 %)	1 (8 %)	59 (11 %)	70 (11 %)	
Preoperative Hemoglobin					
Mean (SD)	12 (± 2.1)	12 (± 2.6)	13 (± 1.6)	13 (± 1.8)	0.000
Missing	9 (9.3%)	2 (16.7%)	50 (9.4%)	61 (9.5%)	
Duration of Surgery (Hours)					
Mean (SD)	2.5 (± 1.9)	4.3 (± 3.2)	1.8 (± 1.1)	2.0 (± 1.3)	0.000
Estimated Blood Loss (mL)					
Mean (SD)	310 (± 280)	290 (± 210)	120 (± 140)	160 (± 190)	0.000
Missing	35 (36.1%)	4 (33.3%)	252 (47.5%)	291 (45.5%)	
Total Crystalloid (mL)					
Mean (SD)	1000 (± 990)	1300 (± 660)	630 (± 400)	700 (± 560)	0.000
Missing	6 (6.2%)	0 (0%)	33 (6.2%)	39 (6.1%)	
PRBCs Transfused (units)					
Mean (SD)	0.21 (± 0.69)	0 (± 0)	0.0075 (± 0.11)	0.038 (± 0.29)	0.000
Length of Hospitalization (Days)					
Mean (SD)	13 (± 20)	10 (± 9.9)	5.6 (± 5.4)	6.8 (± 9.8)	0.000
Length of ICU Stay (Days)					
Mean (SD)	1.2 (± 2.6)	0.42 (± 0.51)	0.56 (± 1.6)	0.65 (± 1.8)	0.013
In-hospital Mortality					
No	87 (90 %)	12 (100 %)	511 (96 %)	610 (95 %)	0.037
Yes	10 (10 %)	0 (0 %)	20 (4 %)	30 (5 %)	

Regression Analysis Results				
Variable	estimate	std.error	statistic	p.value
(Intercept)	1.39	0.50	2.76	0.01
Time Weighted MAP	0.00	0.00	2.30	0.02
Age	0.01	0.00	3.18	0.00
Sex (male)	0.24	0.05	4.68	0.00
BMI	−0.03	0.01	−4.10	0.00
ASA II	0.08	0.07	1.10	0.27
ASA III	0.39	0.10	4.03	0.00
ASA IV	1.40	0.33	4.26	0.00
Emergency Case (yes)	0.50	0.09	5.67	0.00
Vasopressor Use	0.07	0.05	1.32	0.19
Preoperative PFT: Mild obstructive	0.29	0.39	0.73	0.47
Preoperative PFT: Mild restrictive	0.67	0.41	1.63	0.10
Preoperative PFT: Mixed/pure obstructive	0.72	0.41	1.76	0.08
Preoperative PFT: Moderate obstructive	0.44	0.41	1.07	0.28
Preoperative PFT: Moderate restrictive	0.28	0.45	0.62	0.53
Preoperative PFT: Normal	0.44	0.38	1.15	0.25
Preoperative PFT: Severe restrictive	1.79	0.48	3.76	0.00
Preoperative HTN: Yes	−0.02	0.05	−0.39	0.70
Preoperative DM: Yes	−0.02	0.07	−0.24	0.81
Preoperative Hemoglobin	−0.06	0.02	−4.08	0.00
Duration of Operation	0.00	0.00	9.07	0.00
Approach: Robotic	−0.01	0.19	−0.07	0.94
Approach: Videoscopic	−0.04	0.08	−0.48	0.63
Operation type: Minor resection	−0.16	0.05	−2.96	0.00

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SP 1 - Kiosk 9 - Poster 6

Pulmonary hypertension in non-cardiac surgery: a large single-center retrospective cohort study

Presenting Author: *Maximilian Staebler, MD*

Background: Pulmonary hypertension (pHTN) is an important disease entity that influences perioperative management and outcomes1. As the prevalence of pHTN is increasing among surgical populations 2, anesthesia providers must recognize and manage these high-risk patients to prevent perioperative complications. However, existing single-center studies have assessed only small cohorts, limiting our understanding. Here, we characterize the risk profile and adverse outcomes in a large cohort of pHTN patients undergoing non-cardiac surgery.

Methods: An echocardiography database at Massachusetts General Hospital was queried to identify patients with echocardiographic evidence of pHTN, defined as a tricuspid regurgitation (TR) jet velocity > 2.8 m/s. Of 28,742 patients, 15,385 had echocardiographic evidence of pHTN. We identified 3,730 patients who underwent non-cardiac surgery within 1 year without interval normalization of the TR velocity and compared them to a control group of 2,639 patients without echocardiographic evidence of pHTN. Shapiro-Wilks testing was performed to assess normality. All variables (non-normally distributed) were analyzed using the Mann Whitney U test or the chi-square test.

Results: Demographic and surgical data are shown (Tables 1 and 2). Patients in the pHTN group had higher rates of heart failure (29.7% vs 26.5%, p< 0.001) and pre-existing pHTN diagnosis (7.72% vs

3.34%, p < 0.001) but lower rates of coronary artery disease (16.4% vs 18.4%, p = 0.042). pHTN patients were more commonly assigned ASA 4 classification (27.3% vs 18.6%, p < 0.001), and less commonly assigned ASA 3 (63.5% vs 67.9%, p < 0.001) compared to controls. Postoperatively, pHTN patients more frequently required at least one new vasopressor (21.1% vs 18.1%, p = 0.0045) or inotrope (2.90% vs 2.02%, p = 0.044) initiation. pHTN patients were more likely to be treated with vasopressin or norepinephrine (vasopressin: 4.99% vs 3.46%, p = 0.0056, norepinephrine: 11.0% vs 7.56%, p < 0.001) and required a longer duration of post-operative vasopressors and inotropes (see Table 3). Mortality was higher in pHTN patients at 30 days (8.47% vs 5.61%, p < 0.001) and 90 days (14.5% vs 10.2%, p < 0.001), as
was post-operative length of stay. There was no difference in post-operative ICU admission (4.42% vs 3.76%, p = 0.23). pHTN patients were less likely to be discharged home (35.0% vs 46.6%, p < 0.001) and more likely to be discharged to nursing facilities (28.6% vs 20.7%, p < 0.001) or long-term care facilities (6.19% vs 4.56%, p = 0.0067).

Conclusion: Pulmonary hypertension carries an independent risk of perioperative mortality and increased healthcare utilization following non-cardiac surgery. Additionally, only 7.72% of patients with echocardiographic evidence of pHTN carried a formal diagnosis, suggesting underdiagnosis despite these increased risks. Our findings highlight the need for improved recognition and perioperative management strategies in this vulnerable patient population.

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Table 1. Patient Demographics

	pHTN Group (%, N= 3730)	Control Group (%, N=2369)	p-value
Sex (M)	1933 (51.8)	1271 (53.7)	p = 0.17
Median Age at Surgery	72.3 [IQR 62.3-80.4]	72.0 [IQR 63.1-79.4]	p = 0.22
Median BMI	27.0 [IQR 23.4-31.6]	26.6 [IQR 23.2-30.8]	p = 0.02
ASA Classification			p < 0.01
1	1 (0)	2 (0)	p = 0.56
2	269 (7.2)	281 (11.9)	p < 0.001
3	2369 (63.5)	1608 (67.9)	p < 0.001
4	1019 (27.3)	441 (18.6)	p < 0.001
5	71 (1.9)	35 (14.8)	p = 0.25
6	0 (0)	1 (0)	p = 0.39
Rate of Medical Comorbidities			
Chronic Obstructive Pulmonary Disease (COPD)	360 (9.65)	202 (8.53)	p = 0.16
Coronary Artery Disease	610 (16.4)	436 (18.4)	p = 0.04
Atrial Fibrillation (AF)	1071 (28.7)	673 (28.4)	p = 0.79
Heart Failure (CHF)	1107 (29.7)	627 (26.5)	p < 0.001
Myocardial Infarction	258 (6.92)	150 (6.33)	p = 0.40
Cerebrovascular Accident	340 (9.12)	237 (10.0)	p = 0.27
Pre-existing Pulmonary Hypertension Diagnoses	288 (7.72)	80 (3.34)	p < 0.001

Table 2. Surgery Type and Acuity

Surgery Type	pHTN Group (%, N=3730)	Control Group (%, N=2369)	p-value
Orthopedic Surgery	658 (17.6)	395 (16.7)	p = 0.35
General Surgery	421 (11.3)	331 (14.0)	p = 0.002
Emergent and Urgent Surgery	417 (11.2)	211 (8.91)	p = 0.005
Vascular Surgery	370 (9.90)	189 (7.98)	p = 0.01
Urologic Surgery	361 (9.70)	257 (10.8)	p = 0.15
Neurosurgery	302 (8.10)	217 (9.16)	p = 0.16
Thoracic Surgery	269 (7.21)	181 (7.64)	p = 0.57
Renal Transplant Surgery	188 (5.04)	115 (4.85)	p = 0.79
Interventional Radiology	180 (4.83)	90 (3.80)	p = 0.06
Radiology	157 (4.21)	84 (3.55)	p = 0.22
Surgical Oncology	106 (2.84)	96 (4.05)	p = 0.01
Gynecologic Surgery	101 (2.71)	85 (3.59)	p = 0.06
Plastic Surgery	70 (1.88)	46 (1.94)	p = 0.93
Burn Surgery	57 (1.53)	30 (1.35)	p = 0.47
Oral and Maxillofacial Surgery	37 (0.99)	20 (0.84)	p = 0.65
Endoscopy	19 (0.51)	12 (0.50)	p = 1
Other	17 (0.46)	10 (0.42)	p = 1

Table 3. Post-operative Outcomes

	pHTN Group (%, N=3730)	Control Group (%, N=2369)	P-value
Rate of at least one vasopressor post-operatively	786 (21.1)	428 (18.1)	p = 0.005
Rate of new Norepinephrine administration	410 (11.0)	179 (7.56)	p < 0.001
Rate of new Phenylephrine administration	428 (11.5)	267 (11.3)	p = 0.84
Rate of new Vasopressin administration	186 (4.99)	82(3.46)	p = 0.006
Rate of at least one inotrope post-operatively	108 (2.90)	48 (2.02)	p = 0.04
Rate of new Epinephrine administration	77 (2.06)	37 (1.56)	p = 0.19
Rate of new Dobutamine administration	16 (0.43)	10 (0.42)	p = 1.0
Rate of new Milrinone administration	23 (0.62)	14 (0.59)	p = 1.0
Rate of new Dopamine administration	9 (0.24)	1 (0.04)	p = 0.10
Median Duration of Post-operative Vasopressors (days)	2.99 [IQR 0.84-9.1]	1.94 [IQR 0.65 - 6.15]	p < 0.001
Median Duration of Post-operative Inotropes (days)	3.06 [IQR 1.02 - 6.85]	3.97 [IQR 1.31 - 11.89]	p = 0.14
New ICU Admission Post-operatively	165 (4.42)	89 (3.76)	p = 0.23
Post-operative Mortality at 30 days	316 (8.47)	133 (5.61)	p <0.001
Post-operative Mortality at 90 days	539 (14.5)	241 (10.2)	p < 0.001
Median Time To Death (days)	364 [IQR 58 - 1005]	483 [IQR 95 - 1137]	p < 0.001
Median Post-operative Length of Stay (LOS)	5.48 [IQR 1.72-11.6]	3.70 [IQR 1.53 - 8.52]	p < 0.001
Discharge Disposition			
Home	1305 (35.0)	1104 (46.6)	p < 0.001
Hospice	49 (1.31)	22 (0.93)	p = 0.17
Home with services	786 (21.1)	512 (21.6)	p = 0.61
Nursing Facility	1065 (28.6)	490 (20.7)	p < 0.001
Long Term Care	231 (6.19)	108 (4.56)	p = 0.007
Expired	275 (7.37)	113 (4.77)	p < 0.001
Other	19 (0.50)	20 (0.83)	p = 0.10

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SP 1 - Kiosk 10 - Poster 1

ROBIN Technique for Refractory Dynamic LVOT Obstruction Following Valve-in-Ring TMVR

Presenting Author: *Darshan Rola, DO*

Introduction: Dynamic left ventricular outflow tract (LVOT) obstruction is an uncommon but challenging complication following transcatheter mitral valve replacement (TMVR), particularly in patients with prior annuloplasty rings, elongated anterior mitral leaflets, or multiple reinterventions. Standard approaches—such as LAMPOON or septal reduction—may not fully relieve obstruction. The retrograde oriented basal incision of the anterior leaflet (ROBIN) technique is an emerging option for refractory cases.

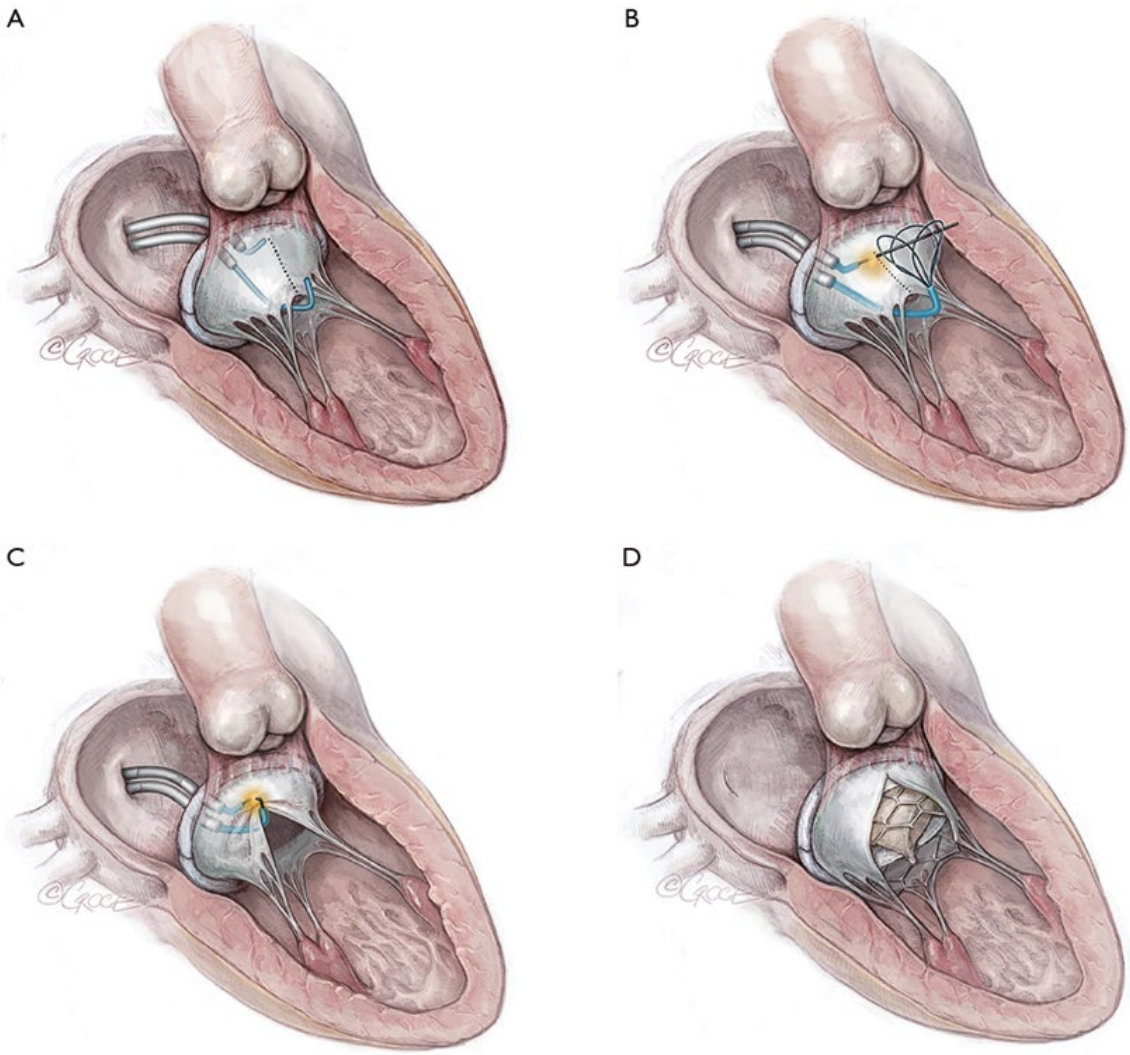
Case Report: An 83-year-old man with hypertension, prior mitral valve repair, and progressive valvular disease underwent TMVR with a LAMPOON procedure. His post-procedure course was complicated by paravalvular leak, dynamic LVOT obstruction, alcohol septal ablation, permanent pacemaker implantation for complete heart block, and atrial septal defect closure. He remained symptomatic with NYHA class III limitation despite these interventions. Repeat imaging showed persistent systolic anterior motion of an elongated anterior mitral leaflet with provocable LVOT obstruction. Stress echocardiography revealed a post-exercise aortic gradient of 33 mmHg. Invasive hemodynamics demonstrated no fixed obstruction at baseline, but post-extrasystolic LV–aortic gradients approached 100 mmHg, confirming severe dynamic LVOT obstruction.

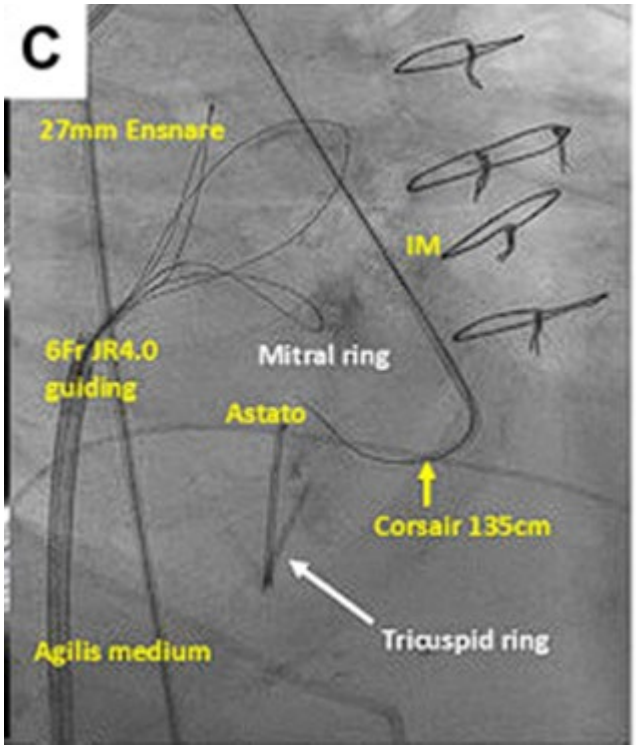
Given his refractory symptoms, anterior leaflet modification using the ROBIN technique was performed. Retrograde arterial access was used to position a guiding catheter against the ventricular aspect of the basal anterior leaflet. Under fluoroscopy and transesophageal echocardiography (TEE), a wire within a microcatheter was advanced and electrified in short pulses to perforate the leaflet. Several attempts were required due to leaflet thickness and restricted mobility from the prior ring. After successful traversal, the wire was externalized into the left atrium, establishing a controlled leaflet tract. Sequential balloon dilation was then performed to achieve complete basal leaflet splitting while preserving the integrity of the valve-in-ring prosthesis. TEE confirmed that the divided leaflet cleared the LVOT without interacting with the implanted device.

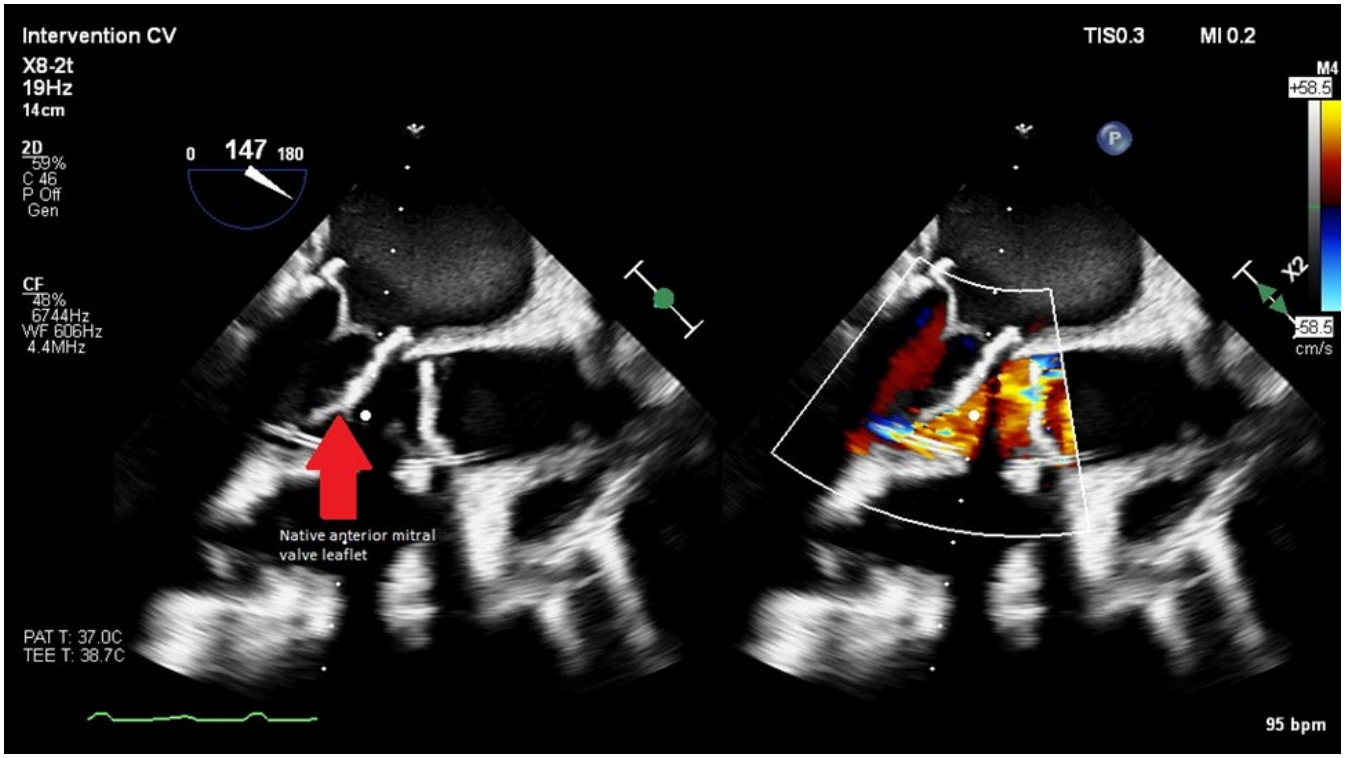
Discussion: The procedure immediately abolished post-extrasystolic LVOT gradients without introducing new transmitral or aortic valve dysfunction. Post-procedure TEE demonstrated stable valve positioning, resolution of paravalvular leak, improved LVOT flow, a mean gradient of 5.6 mmHg, and preserved biventricular function. In this anatomically complex patient—whose prior repair limited the effectiveness of LAMPOON and septal reduction—the ROBIN technique provided effective relief of dynamic obstruction.

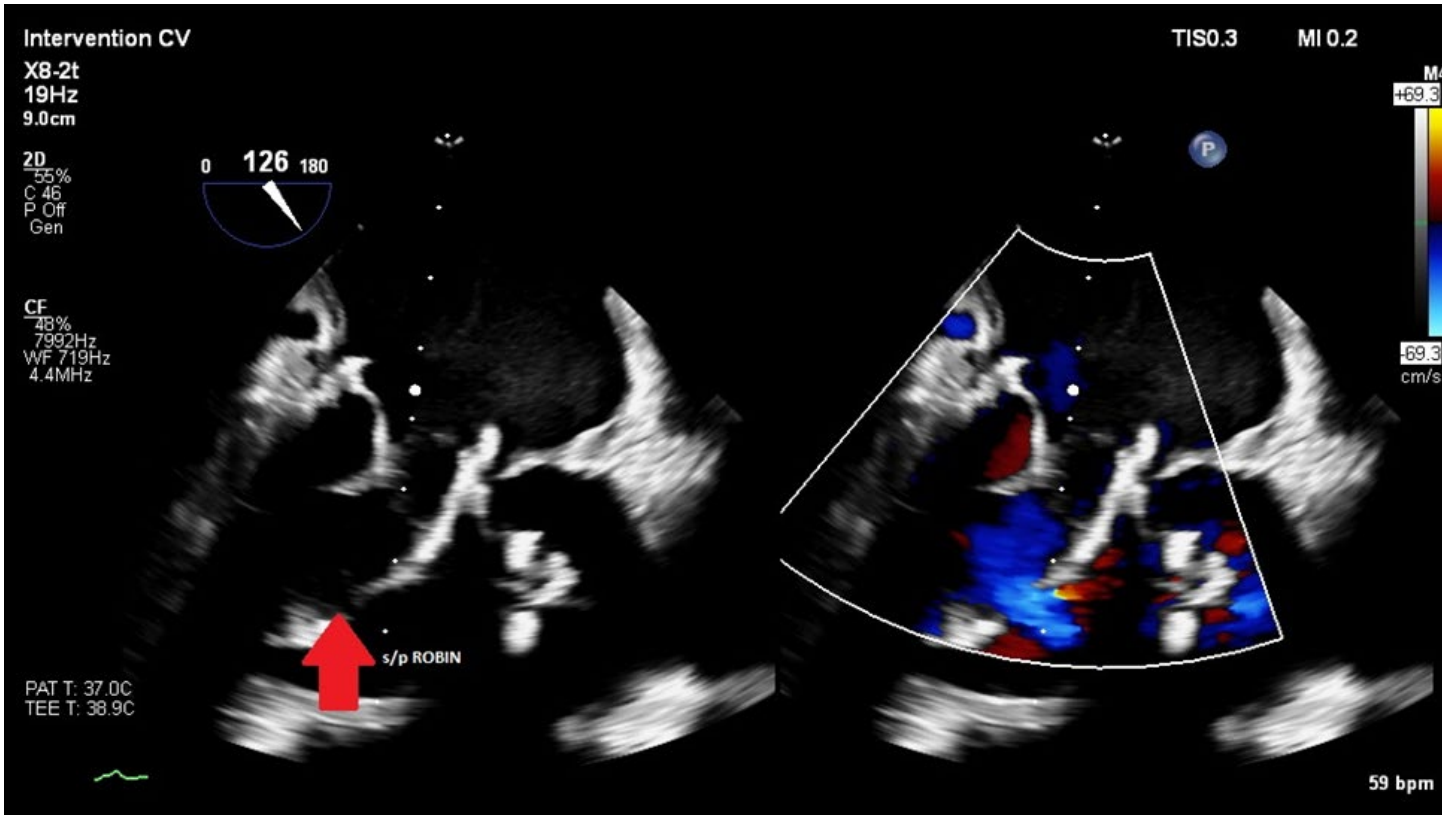
Conclusion: This case highlights the utility of the ROBIN leaflet-modification technique for severe dynamic LVOT obstruction following complex TMVR. ROBIN produced immediate hemodynamic improvement without compromising prosthetic valve function and may serve as a valuable adjunct when obstruction persists despite LAMPOON or septal reduction.

Image uploads:




C
27mm Ensnare
IM
6Fr JR4.0
guiding
Mitral ring
Astato
Corsair 135cm
Agilis medium
Tricuspid ring


Intervention CV
X8-2t
19Hz
14cm
2D
59%
C 46
P Off
Gen
0
147
180
CF
48%
6744Hz
WF 606Hz
4.4MHz
TIS0.3
MI 0.2
M4
+58.5
-58.5
cm/s
X2
Native anterior mitral
valve leaflet
PAT T: 37.0C
TEE T: 38.7C
95 bpm



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SP 1 - Kiosk 10 - Poster 2

Bulges, Blocks, and Beats: The Consequences of Atrial Septal Aneurysm Repair, a case series

Presenting Author: *Saad Ali, MD*

Background/Intro: Atrial septal aneurysm (ASA) is rare, with an incidence of 1–2%(1). Repair is even

less common and reserved for symptomatic patients or those at thromboembolic risk, but intervention on the interatrial septum carries risk for perioperative atrial arrhythmias. Mechanical stretch, localized conduction heterogeneity, and inflammatory remodeling during device-based or surgical manipulation may increase atrial irritability (2). This case series summarizes perioperative arrhythmia patterns in patients undergoing ASA correction and highlights related anesthetic considerations.

Methods: We reviewed echocardiography reports from 2019–2025 and identified ~2700 studies with ASA-related keywords. Only four patients underwent ASA repair. Clinical data, TEE findings, prior arrhythmia history, anesthetic course, and postoperative rhythm outcomes were reviewed. Two patients were symptomatic; two underwent repair for primary thromboembolic prevention. Two had associated septal defects with shunting; two underwent transcatheter closure. All cases used intraoperative echocardiography when an anesthesiologist was involved and were performed under general anesthesia.

Postoperatively, two patients were extubated immediately; two remained intubated. Two developed postoperative arrhythmias (patients #1 and #2). Patient #3 was pacemaker-dependent in complete AV block preoperatively and remained unchanged. Patient #4, with a history of atrial fibrillation post-ablation, remained in sinus rhythm. Average length of stay was seven days (range 2–19). There were no 30-day mortalities.

Discussion: Across these cases, interatrial septal pathology was associated with perioperative atrial arrhythmias, including new-onset arrhythmias in patients without prior rhythm disease. Manipulation of abnormal septal tissue appears to unmask atrial irritability through stretch, inflammation, or local conduction disruption. This risk is not limited to septal-directed procedures; catheter-based interventions that cross or compress the septum may also provoke arrhythmias even when the septum is not the procedural target.
These findings are relevant to structural heart procedures such as TAVR. In patients with known ASAs, intraoperative TEE may be useful to assess for septal instability or worsening pathology regardless of procedural focus. For individuals at risk of postoperative atrial fibrillation due to septal manipulation, prophylactic left atrial appendage ligation may be considered to reduce long-term anticoagulation needs.

Conclusion: This case series demonstrates a consistent pattern of perioperative atrial arrhythmias following interventions involving or affecting the interatrial septum. The findings highlight the importance of vigilant rhythm monitoring, hemodynamic optimization, and routine intraoperative TEE to guide intervention and to detect septal injury as it occurs. Larger studies are needed to clarify arrhythmia risk, refine perioperative management, and determine the role of prophylactic LAA ligation in patients with septal pathology.

Image uploads:

Patient	Echocardiographic Findings	Indications for Repair	Procedure	Post Operative Course
#1: 40y Female	ASA with PFO	Transient neurological symptoms	General Anesthesia; transcatheter PFO closure and ASA repair	Paroxysmal Atrial Flutter; subsequently resolved without treatment. Discharged on POD #3
#2: 73y Female	ASA with spontaneous echo contrast and atrioventricular outflow tract flow acceleration	Recurrent syncopal episodes ("Drop" attacks)	General anesthesia; open repair of ASA and LAA ligation with cardiopulmonary bypass	Rapid Atrial Fibrillation in immediate post operative period, successfully treated with amiodarone. Discharged on POD #4
#3:74y Male	ASA with spontaneous echo contrast and severe AI	Shortness of breath related to AI; patient was asymptomatic from ASA	General anesthesia; aortic valve replacement, repair of ASA, and LAA ligation with cardiopulmonary bypass	Remained in complete AV block. Patient already pacemaker dependent preoperatively. Discharged on POD #19
#4: 88y Male	ASA with small ASD	Shortness of breath; small ASD had become larger after TAVR, possibly iatrogenically	General Anesthesia; transcatheter repair of ASD and ASA	Remained in NSR. Prior Afib ablation. Discharged on POD #2

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SP 1 - Kiosk 10 - Poster 3

Correlation between Intraoperative Invasive Hemodynamic and Color Flow Doppler Assessment After Percutaneous Edge-to-Edge Mitral Valve Repair

Presenting Author: *Usman Ahmed, MD*

Background: Percutaneous edge-to-edge mitral valve repair (pMVr) is widely used for severe primary and secondary mitral regurgitation (MR).1 Intraoperative assessment typically incorporates invasive left atrial (LA) pressure measurements and echocardiographic color flow Doppler (CFD) indices2; however, the degree to which these modalities correlate remains unclear. CFD grading is semi-quantitative and velocity-based, whereas LA pressure assessment assumes a pressure–volume relationship potentially influenced by ventricular compliance and loading conditions. This study evaluated the relationship between intraoperative changes in LA pressures and CFD-derived MR severity following pMVr.

Methods: A retrospective analysis was conducted using a transcatheter valve therapy registry at a tertiary center. Adults undergoing pMVr from 2015–2020 were included. Demographics, comorbidities, echocardiographic parameters, and intraoperative hemodynamic measurements were collected. MR severity was assessed using a 4-grade CFD scale. Peak and mean LA pressures were measured continuously during the procedure and indexed to left ventricular pressures. Statistical analysis included nonparametric testing, Spearman correlation, and multivariable linear regression adjusting for clinical and procedural covariates.

Results: Among 140 patients, median age was 80 years and 61% were male. Most patients (84%) had severe MR preoperatively. Peak LA pressure decreased from 47.5 mmHg to 28 mmHg ($p< 0.001$), and mean LA pressure decreased from 24 mmHg to 17 mmHg ($p< 0.001$). MR grade improved from 4 to 2 ($p< 0.001$).(Figure 1-3) Although both modalities demonstrated significant reductions, the magnitude of MR grade reduction exceeded the reduction in LA pressures. No correlation was found between change in MR grade and change in peak LA pressure ($R^2=0.16$; $p=0.96$) or mean LA pressure ($R^2=0.23$; $p=0.43$) in adjusted analyses. (Figure 4-6)

Conclusion: Intraoperative reductions in MR severity measured by CFD do not correlate with reductions in LA pressures following pMVr. These findings likely reflect fundamental mechanistic differences between velocity-based Doppler assessment and pressure-derived hemodynamic measurement, along with variability in ventricular compliance, clip-related flow changes, and operator-dependent imaging factors. Despite the absence of correlation, combined hemodynamic and echocardiographic assessment remains essential for intraoperative evaluation of procedural success. Further research should determine which echocardiographic parameters best predict clinical outcomes after pMVr.

Image uploads:

Characteristics	Central Tendency/ Frequency (n=140)
Age	80 (72 – 88)
Female	57 (38.8%)
BMI	24.69 (22.4 – 29.4)
Smoking	10 (7.1%)
Hypertension	112 (80%)
Diabetes Mellitus	44 (31.4%)
Renal Failure	7 (5%)
Prior MI	39 (27.9%)
NYHA Classification	
NYHA II	18 (12.9)
NYHA III	79 (56.4)
NYHA IV	43 (30.7)
Creatinine	1.4 (1.0 – 1.8)
Pacemaker	28 (20)
Prior Cardiac Surgeries	8 (5.7)
Prior CABG	37 (26.4)
Prior TMVR	7 (5)
Prior Stroke	15 (10.7)
Cardiomyopathy	50 (34)
Ischemic	24 (17.1)
Non-ischemic	25 (17.9)
Atrial Fibrillation	89 (63.6)
Ischemic Regurgitation	
Paroxysmal	26 (18.6%)
Persistent	34 (24.3)
Six minute walk distance	527 (260 – 817)
KCCQ 12 score	39.6 (22.9 – 59.7)

Figure 1: Descriptive baseline demographics of 140 patients undergoing percutaneous edge-to-edge clip mitral valve leaflet repair.

Characteristics	Central Tendency/ Frequency (n=140)
LV Ejection Fraction	45 (30 - 60)
Fluoroscopy time	26.9 (19 – 38.7)
Fluoroscopy Dose	767 (403 – 1263)
Pre-operative LA peak pressure	47.5 (40 – 58)
LA Dimension	4.75 (4.20 – 5.30)
LA Volume Index	54.50 (47.0 – 67.3)
Pre-operative MR grade	
II	1 (0.7)
III	22 (15.7)
IV	117 (83.6)
Number of Clips used	
1	75 (51.7%)
2	59 (40.1%)
3	9 (6.1%)
4	2 (1.4%)
Total Fluid Received During Case	741 +- 615
Total Fluid Input	947 +- 647
Total Fluid Output	147 +- 270
Post-operative LA peak Pressure	28 (22 – 34)
Post-operative MR grade	
I	4 (2.9)
I	53 (37.9)
II	69 (49.3)
III	14 (10)
Post-operative Creatinine	1.4 (1.0 – 1.8)
Discharge Location	
Deceased	4 (2.9)
Home	107 (76.4)
Hospice	2 (1.4)
Extended Care/ Rehab	24 (17.1)
Other	3 (2.1)

Figure 2: Descriptive intraoperative characteristics of 140 patients who underwent percutaneous edge-to-edge clip mitral valve leaflet repair.

	Pre-MitraClip Mean (SD)	*Post-MitraClip Mean (SD)*	*Effect Estimate (95% CI)*	*p-value*	*Correlation to difference in MR grade*	
					Unadjusted [a] *R^2 (p value)*	***Adjusted*** [b] *aR^2 (p value)*
Peak LA Pressure (mmHg)	47 (38 – 57)	28 (20 – 33)	1.76 (1.50, 2.01)	<0.001	0.11 (*p=0.24*)	0.16 (0.96)
Mean LA pressure (mmHg)	24 (18 – 28)	17 (14 – 22)	1.68 (1.27, 2.09)	<0.001	0.04 (p=0.80)	0.23 (0.43)
MR quantification	4 (3 – 4)	2 (1 – 2)	3.09 (2.70, 3.45)	<0.001	--	

Figure 3: Comparison between hemodynamic and echocardiographic measures pre-Mitra clip placement and post Mitra-clip placement in 140 patients who underwent percutaneous edge-to-edge clip mitral valve leaflet repair. Effect estimates were calculated to quantify the difference in the two measures on a matching scale.

[a] Unadjusted spearman correlation was used to test correlation between difference in LA pressures and MR grading.

[b] Adjusted correlations were reported using linear regression analysis. Model was adjusted for age, gender, body mass index, smoking status, hypertension, diabetes, chronic renal failure, prior myocardial infarction, heart failure classification (NYSE), ejection fraction, number of clips used, left atrial dimension, left atrial volume index.

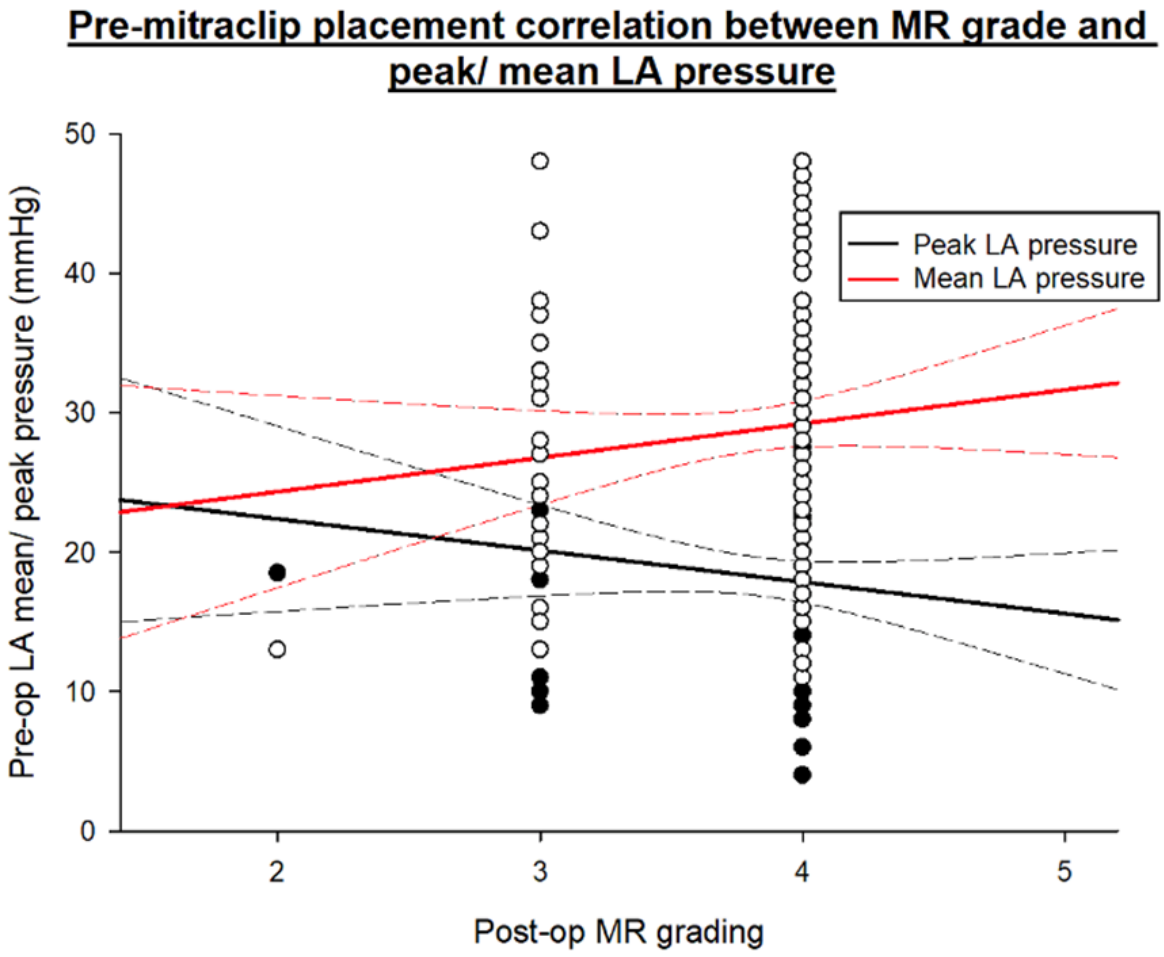


Figure 4: Correlation between MR grade and LA pressures before placing Mitra clips intraoperatively

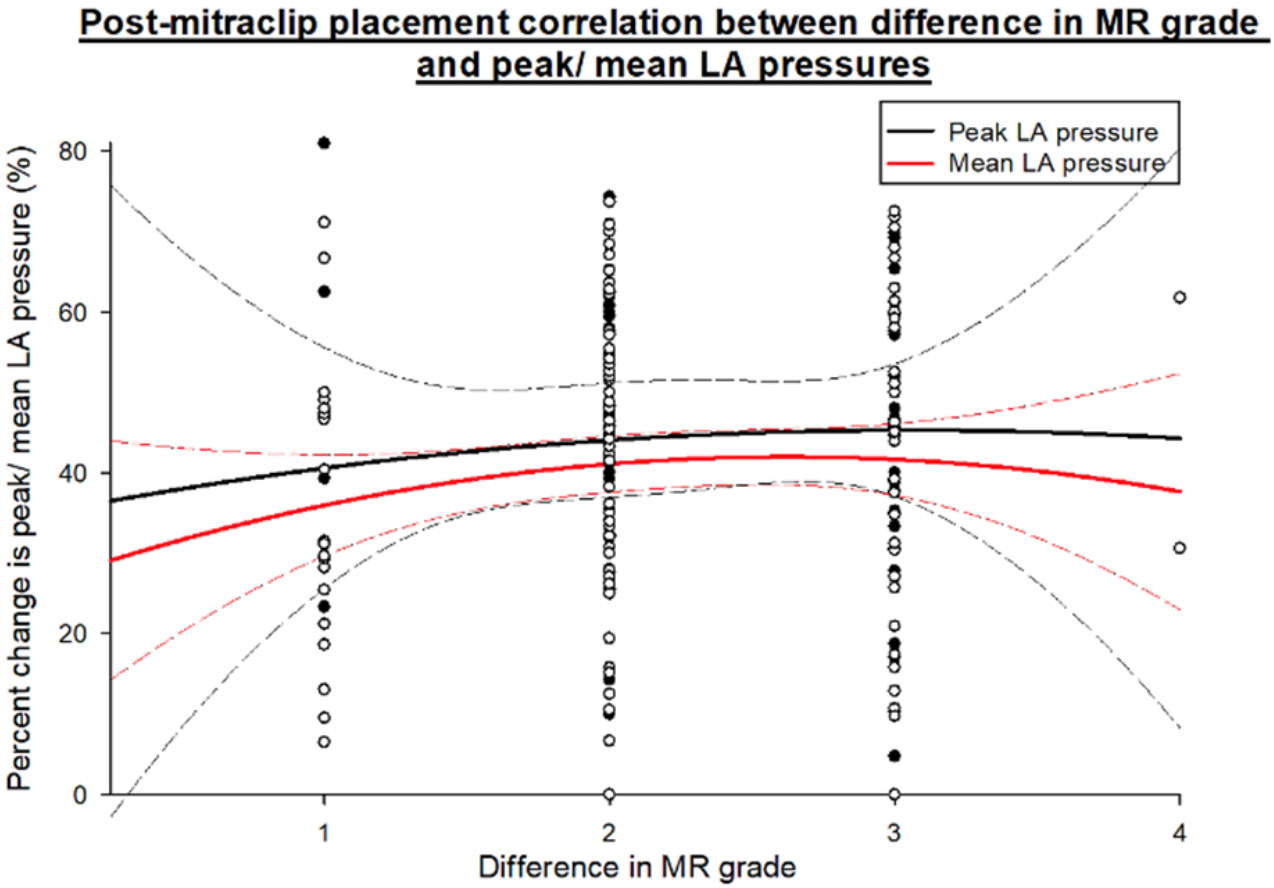


Figure 5: Correlation between MR grade and LA pressures after placing Mitra clips intraoperatively

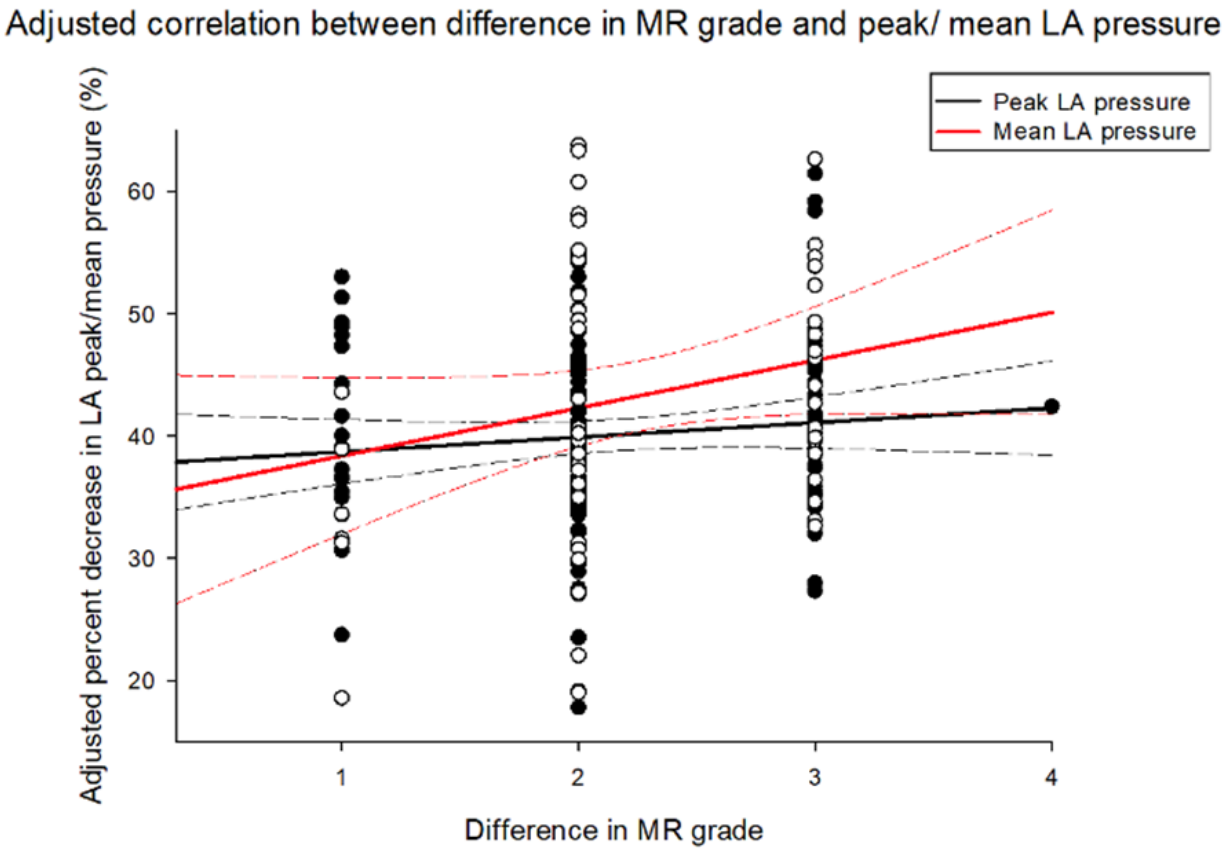


Figure 6: Adjusted correlation between difference MR grade and percentage difference in LA pressures after placing Mitra clips.
- Difference was calculated by subtracting pre-Mitra clip value from post-Mitra clip values
- Adjusted for age, BMI, gender, smoking status, diabetes mellitus type 2, hypertension, prior renal failure, prior myocardial infarction, NYSE heart failure classification, ejection fraction, number of clips used, left atrial dimension, left atrial volume index

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SP 1 - Kiosk 10 - Poster 4

Coronary Vasospasm Prevention and Hemodynamic Stability During Pulse Field Ablations: A Single-Center Experience

Presenting Author: *Jessica Chan, DO*

Introduction: Pulsed field ablation (PFA) has emerged as a non-thermal, myocardial-selective modality for atrial arrhythmia treatment, offering reduced collateral tissue injury compared with radiofrequency and cryoablation. While PFA is well established for pulmonary vein isolation, limited data describe anesthetic and hemodynamic considerations during cavotricuspid isthmus (CTI) ablation for typical atrial flutter. Prior angiographic studies demonstrate that PFA delivered along the CTI can provoke significant right coronary artery (RCA) vasospasm, often requiring large doses of nitroglycerin for mitigation. As PFA adoption expands, understanding real-world anesthetic patterns, vasopressor use, and periprocedural safety is essential. This study presents a retrospective review of CTI ablations performed using the FARAPULSE™ Pulsed Field Ablation System (Boston Scientific, Marlborough, MA), with specific focus on nitroglycerin and phenylephrine utilization, hemodynamic stability, and procedural outcomes.

Methods: A retrospective single-center review was conducted of consecutive patients who underwent CTI ablation using the FARAPULSE™ system over a 6-month period at Loma Linda University Health. All procedures were performed under general anesthesia. Data extracted included total nitroglycerin dosing (bolus and infusion), phenylephrine bolus requirements, intraoperative blood pressure trends, arrhythmia termination, and complications. A structured narrative literature synthesis was also performed to contextualize institutional findings within the existing evidence base for PFA safety and vasospasm physiology.

Results: Across the 6-month review period, nitroglycerin was routinely administered prophylactically to mitigate anticipated RCA vasospasm during CTI PFA. Phenylephrine bolus requirements increased correspondingly, consistent with expected vasodilatory effects of nitroglycerin. Despite this, hemodynamic parameters remained stable with standard anesthetic management. Procedural success rates were high, and there were no episodes of pericardial tamponade, coronary injury, or hemodynamic collapse. One patient experienced a watershed cerebral infarct
temporally associated with the procedure—the sole major complication observed. No esophageal, phrenic nerve, or pulmonary venous complications were identified.

Conclusion: In this retrospective review, CTI ablation using the FARAPULSE™ PFA system was generally safe and well tolerated under general anesthesia. Anticipatory management of coronary vasospasm with nitroglycerin was effective, though it increased vasopressor utilization. Overall hemodynamic stability was preserved, and the complication rate was low, with one watershed stroke representing the only major adverse event. As PFA expands beyond pulmonary vein isolation

to CTI and other atrial targets, ongoing collection of multicenter real-world data will be essential to refine anesthetic strategies, vasospasm-prevention protocols, and long-term safety assessment.

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SP 1 - Kiosk 10 - Poster 5

Innovative, High-Value Perioperative Imaging by Cardiac Anesthesiology: A Quality-Driven, Cost-Efficient Model for Structural Heart and Electrophysiology Care Presenting Author: *Jacob Schaff, MD, FASE*

Introduction: Successful delivery of advanced electrophysiology and structural heart interventions relies on timely, expert, and highly reliable imaging. At our institution, anesthesiologist-led perioperative echocardiography has become critical to the safety, efficiency, and procedural success of left atrial appendage occlusion (LAAO), TAVR, cardiac ablation workflows, structural heart procedures, and selective non-cardiac surgical cases. By integrating advanced imaging directly into anesthesia services, we created a high-reliability, patient-centered model that strengthens every phase of care. A comprehensive anesthesia-driven imaging program was developed to provide real-time TEE and TTE across multiple procedural domains. In

electrophysiology, anesthesiologist-performed TEE is essential for clearing the left atrial appendage (LAA) for thrombus prior to cardiac ablation, allowing same-day assessment and immediate progression to ablation for urgent or add-on cases. This eliminates delays inherent to scheduled cardiology-driven TEE slots and ensures continuous workflow for EP teams. During Watchman implantation and LAAO procedures, anesthesia-led TEE optimizes device sizing, positioning, and peri-device leak assessment while providing immediate detection of complications such as effusion, device thrombosis, or hemodynamic instability. The model extends seamlessly to structural heart care. For TAVR, anesthesia-performed TTE provides rapid valve assessment, gradient evaluation, and confirmation of procedural success, enabling streamlined workflow without requiring cardiology FTEs or echo technicians. In non-cardiac surgery, anesthesia-led TEE/TTE offers real-time diagnostic capability for unexplained hypotension, valvular disease, or cardiac dysfunction, expanding clinical safety and decision-making at the point of care. Operational benefits are substantial. For LAA clearance, integrating TEE into the anesthetic reduced evaluation time from 35–85 minutes to 13–20 minutes, improving cath lab turnover by 22–65 minutes per case and saving $2,750–$8,125 per procedure. Across ~80 annual cases, this yields $220,000–$650,000 in efficiency savings. A streamlined urgent EP pathway reduced hospital length of stay by 104 days annually through same-day rule-out and treatment. Eliminating the need for 2.0 cardiology FTEs and associated technicians generated $2.6M in direct savings and $1.8M in recovered cardiology outpatient revenue, totaling $4.8M in measurable value.

Our experience demonstrates that anesthesiology-led perioperative imaging is not supplemental—it is foundational to the success of EP and structural heart programs. This integrated model enhances safety, accelerates care, improves throughput, reduces costs, and elevates the patient experience—offering a scalable blueprint for community hospitals advancing cardiovascular services.

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SP 1 - Kiosk 10 - Poster 6

Urgent Transcatheter Edge-to-Edge Mitral Valve Repair Versus Surgery in Cardiogenic Shock: A Single-Center Comparative Analysis

Presenting Author: *Usman Ahmed, MD*

Background: Cardiogenic shock complicated by severe mitral regurgitation is associated with high mortality, and surgical mitral valve intervention has traditionally been the standard therapeutic option.1,2 However, operative risk is frequently prohibitive in this population, limiting timely surgical management. Transcatheter edge-to-edge repair offers a less invasive strategy, but direct comparative data between urgent transcatheter repair and surgery in the setting of cardiogenic shock remain limited.3 This study evaluated clinical outcomes associated with both approaches in a high-risk cohort.

Methods: A retrospective analysis was conducted of patients presenting with cardiogenic shock and severe mitral regurgitation who required urgent intervention. Patients underwent either surgical mitral valve repair/replacement or transcatheter edge-to-edge repair. Primary outcomes included 30-day and 1-year all-cause mortality and heart-failure readmission. Secondary endpoints included peri-procedural complications, transfusion requirements, ventilation duration, intensive care utilization, and postoperative mitral regurgitation severity. Shapiro Wilk test was used to assess normality of distribution of variables. Chi-square tests, t-tests and log rank test (for survival analysis) were used to compare baseline characteristics and outcomes between the 2 cohorts. A two-tailed p-value < 0.05 was considered significant.

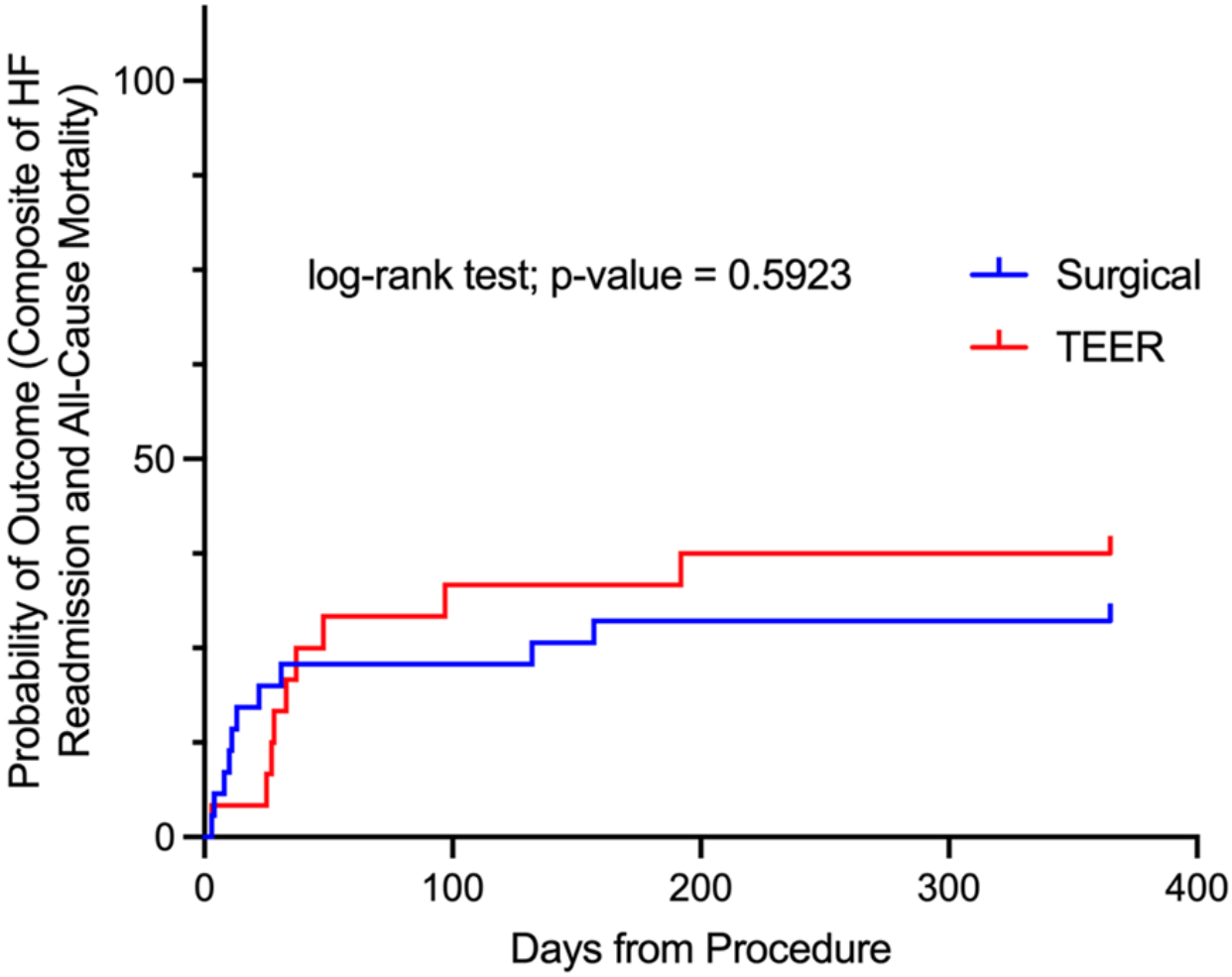
Results: Fifty-eight patients were included (35 surgical, 23 transcatheter). Patients undergoing transcatheter repair were older and more frequently presented with advanced heart-failure symptoms. Both interventions resulted in significant reduction in mitral regurgitation. Compared with surgery, transcatheter repair was associated with fewer transfusions (2.3 ± 0.7 vs. 7.0 ± 6.1 units), lower rates of prolonged ventilation (30.4% vs. 65.7%), and shorter intensive care unit stays (107 ± 103 vs. 191 ± 160 hours). There were no significant differences in the incidence of 1-year mortality (26.1% vs. 14.3%) or 1-year heart-failure readmission. (Figure 1,2). Survival analysis revealed no differences in the primary outcomes between the two groups. (Figure 3)

Conclusion: In patients with cardiogenic shock and severe mitral regurgitation, urgent transcatheter edge-to-edge repair achieved comparable survival and readmission rates to surgical intervention despite higher baseline clinical risk. Reduced peri-operative morbidity, including lower transfusion requirements, shorter ventilation duration, and decreased intensive care length of stay, supports its role as a viable therapeutic alternative for appropriately selected high-risk patients.

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Variable	Surgical (35)	TEER (23)	p-value
Sex (Female)	15 (42.9%)	8 (34.8%)	0.539
Age, y (mean ± SD)	64 ± 12	73 ± 13	0.009
Race			
White	17 (48.6%)	20 (87.0%)	0.036
Black	2 (5.7%)	0 (0%)	0.036
Asian	1 (2.9%)	1 (4.3%)	0.036
Hispanic	2 (5.7%)	0 (0%)	0.036
Other	13 (37.1%)	2 (8.7%)	0.036
BMI (mean ± SD)	27.2 ± 4.9	24.7 ± 4.6	0.057
Hypertension	21 (60.0%)	19 (82.6%)	0.069
Diabetes	13 (37.1%)	10 (43.5%)	0.629
Smoking history	21 (60.0%)	7 (30.4%)	0.028
Chronic lung disease	5 (14.3%)	5 (21.7%)	0.462
Home oxygen use	1 (2.8%)	1 (4.3%)	0.761
Infective endocarditis	8 (22.9%)	0 (0%)	0.014
Prior myocardial infarction (MI)	15 (42.8%)	10 (43.4%)	0.963
Prior PCI	7 (20%)	6 (26%)	0.587
Atrial fibrillation/flutter	12 (34.3%)	10 (43.5%)	0.963
Peripheral vascular disease (PVD)	5 (14.3%)	2 (8.7%)	0.523
Prior stroke	5 (14.3%)	1 (4.3%)	0.224
Prior transient ischemic attack (TIA)	5 (14.3%)	1 (4.3%)	0.224
Pacemaker	3 (8.6%)	4 (17.4%)	0.313
Preoperative creatinine (mg/dL, mean ± SD)	1.8 ± 2.02	1.65 ± 0.18	0.721
Diseased vessels			
0	21 (60.0 %)	12 (52.2 %)	
1	2 (5.7 %)	1 (4.3 %)	
2	4 (11.4 %)	5 (21.7 %)	
3	8 (22.9 %)	5 (21.7 %)	0.763
Acute coronary syndrome (ACS)	12 (34.2%)	6 (26.1%)	0.509
NYHA Class I	1 (2.9%)	0 (0.0%)	0.29
NYHA Class II	2 (5.7%)	0 (0.0%)	0.157
NYHA Class III	10 (28.6%)	3 (13.0%)	0.155
NYHA Class IV	9 (25.7%)	20 (87.0%)	<0.001
Not documented	12 (34.3%)	0 (0.0%)	<0.001
Prior cardiac arrest	3 (8.6%)	2 (8.7%)	0.986
Prior HF admission	28 (80.0%)	9 (39.1%)	0.002
MR – Mild	2 (5.7%)	0 (0.0%)	0.443
MR – Moderate	6 (17.1%)	3 (13.0%)	0.443
MR – Severe	27 (77.1%)	20 (87.0%)	0.443
Aortic stenosis (AS)	2 (5.7%)	3 (13.0%)	0.331
Aortic insufficiency (AI)	7 (20.0%)	1 (4.3%)	0.091
LVEF (mean ± SD)	50.7 ± 16.7	45.0 ± 17.1	0.214
STS risk score (operative mortality; mean ± SD)	18.698 ± 20.2909	20 ± 14	0.807

	Surgical	TEER	p-value
Intra-aortic balloon pump (IABP) use	18 (51.4%)	13 (56.5%)	0.704
Postoperative echo: Trivial/Trace MR	13 (37.1 %)	1 (4.3 %)	
Mild MR	20 (57.1 %)	15 (65.2 %)	
Moderate MR	1 (2.9 %)	6 (26.1 %)	
Severe MR	1 (2.9 %)	1 (4.3 %)	0.006
Permanent stroke	1 (2.9%)	0 (0%)	0.414
Post-operative acute kidney injury	6 (17.1%)	9 (39.1%)	0.061
Operative mortality	1 (2.9%)	0 (0%)	0.414
Total RBC units (mean ± SD)	**7.0 ± 6.1**	**2.3 ± 0.7**	**0.036**
Major bleed	2 (5.7%)	1 (4.3%)	0.818
Permanent stroke	1 (2.9%)	0 (0%)	0.414
Prolonged ventilation	**23 (65.7%)**	**7 (30.4%)**	**0.009**
ICU hours (mean ± SD)	**191 ± 160**	**107 ± 103**	**0.05**
ICU readmission	1 (2.9%)	1 (4.3%)	0.761
Hospital LOS, days (mean ± SD)	19.4 ± 12.5	35.8 ± 68.1	0.186
30-day all-cause mortality	3 (8.6%)	4 (17.4%)	0.313
1-year mortality	5 (14.3%)	6 (26.1%)	0.262
1-year HF readmission	5 (14.3%)	3 (13.0%)	0.893
Composite outcome (1-year mortality+ HF readmission)	11 (31.4%)	9 (39.1%)	0.546



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SCIENTIFIC PROGRAM ABSTRACTS

Poster Session 2

Saturday, April 25, 2026 | 12:15 PM - 1:15 PM

Scientific Posters: Session 2

SP 2 - Kiosk 1 - Poster 1

Minimally Invasive Mitral Valve Surgery Is Associated with a Lower Incidence of Postoperative Atrial Fibrillation - Insights from the Bi-institutional, Retrospective PROMISE Study

Presenting Author: *Melina Heine*

Background: Postoperative atrial fibrillation (POAF) is the most common complication after cardiac surgery and is associated with worse outcomes. Minimally invasive cardiac surgery (MICS) has evolved rapidly with the aim of reducing surgical trauma. Although MICS involves higher procedural complexity, patients undergoing MICS may benefit from reduced postoperative complications and shorter recovery periods2,3. However, the impact of minimally invasive surgical approaches on the POAF incidence remains understudied.

Methods: A bi-institutional, retrospective cohort study was conducted, recruiting 1,280 adult patients who underwent mitral valve surgery with cardiopulmonary bypass (CPB) at Mass General Brigham between January 2016 and April 2025 (IRB #2025P001733). Patients younger than 18 years of age, patients with a history of atrial fibrillation or cardiac assist devices, and patients who underwent concomitant aortic valve, aortic, or CABG surgery were excluded from the analysis. After this exclusion, the surgical approaches encompassed endoscopic (n=46), robotic (n=106), thoracotomy (n=449) and sternotomy (n=679) patients. Data were extracted from RPDR and our institutional data warehouse/EHR. Propensity score matching (1:1, nearest neighbor, caliper=0.2) was performed, comparing MICS (endoscopic, robotic and thoracotomy) versus sternotomy, adjusting for age, sex, BMI, hypertension, diabetes, pulmonary hypertension, CHF, COPD, CKD, LVEF (categorial, intraoperative TEE before CPB), CPB time, procedure details (single mitral valve, multi-valve, mitral valve and additional procedure), Charlson Comorbidity Index (weighted) and preoperative beta-blocker therapy. After matching, a logistic regression was performed on the matched cohort to compare the odds of in-hospital POAF between MICS and sternotomy. Additional pairwise PSM was performed for each approach versus sternotomy, correcting for pairwise comparisons. The in-hospital outcome POAF was defined according to current consensus.

Results: In the matched cohort (328 MICS versus 328 sternotomy patients), sternotomy was associated with significantly higher odds of POAF compared to MICS (OR 1.94, 95% CI 1.39-2.73, p< 0.001). Pairwise, matched comparisons (33 endoscopic; 96 robotic and 293 thoracotomy cases matched to sternotomy) demonstrated significantly lower POAF incidence for robotic versus sternotomy (OR 4.2, 95% CI 2.07-9.04, p< 0.001) and for thoracotomy versus sternotomy approaches (OR 2.01, 95% CI 1.4-2.89, p< 0.001). No significant difference in POAF incidence was observed between the endoscopic and sternotomy groups (OR 1.29, 95% CI

0.48-3.51).

Conclusions: MICS, particularly through a robotic and thoracotomy approach, is associated with a lower incidence of POAF after mitral valve surgery compared with conventional sternotomy. While the study did not identify a significant association for the endoscopic approach, the finding must be interpreted in light of the modest sample size for the endoscopic cases. These findings suggest that the surgical approach may be a modifiable factor in reducing POAF risk after mitral valve surgery. Further studies are needed to confirm these observations, elucidate the underlying mechanisms, and to evaluate the impact of POAF after MICS.

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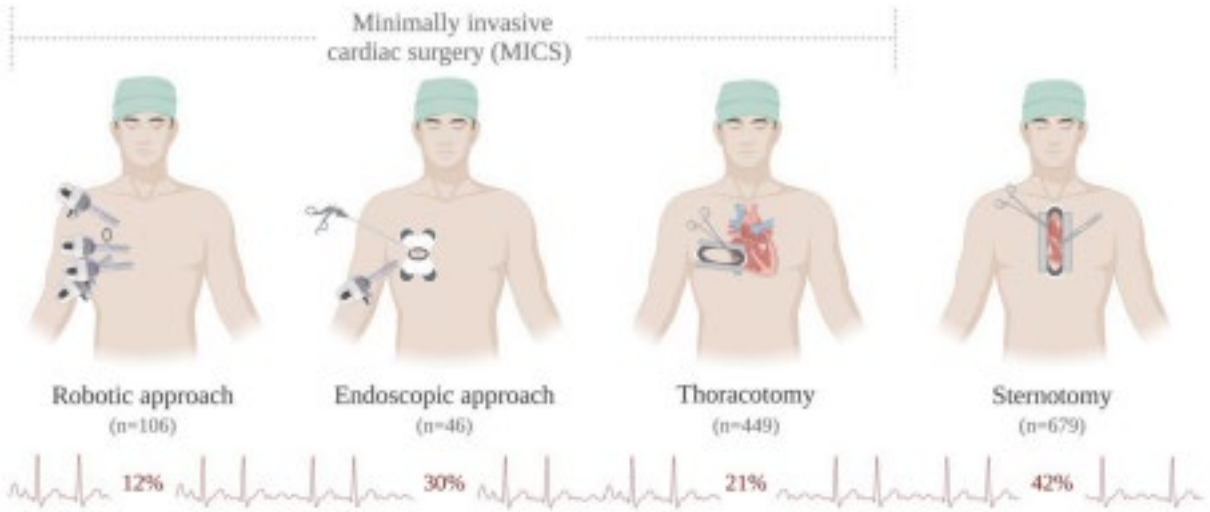


Figure 1: Overview of surgical approaches to the mitral valve and corresponding incidence of postoperative atrial fibrillation

Table 1: Patient demographics, clinical characteristics, and perioperative variables (unmatched cohort)

	Total (n=1280)	Endoscopic (n=46)	Robotic (n=106)	Sternotomy (n=679)	Thoracotomy (n=449)	p-value*
Demographics						
Age (median ± IQR), years	63 [54, 70]	65 [57.5, 71]	61 [55.3, 67]	64 [55, 71]	61 [52, 69]	0.002
Sex, No. (%)						<0.001
Male	721 (56.5)	19 (44.2)	74 (69.8)	355 (52.3)	273 (60.8)	
BMI (median ± IQR)	25.52 [23.06, 28.41]	24.58 [22.9, 28.9]	25.7 [23.6, 27.7]	25.85 [22.95, 28.97]	25.09 [23.03, 27.62]	0.016
Baseline clinical characteristics						
Arterial hypertension, No. (%)	436 (34.1)	15 (32.6)	47 (44.3)	225 (33.1)	149 (33.2)	0.142
Diabetes, No. (%)	55 (4.3)	14 (30.4)	1 (0.9)	29 (4.3)	11 (2.4)	<0.001
Stroke, No. (%)	33 (2.6)	2 (4.3)	1 (0.9)	25 (3.7)	5 (1.1)	0.032
CHF, No. (%)	53 (4.1)	1 (2.2)	4 (3.8)	36 (5.3)	12 (2.7)	0.156
CAD, No. (%)	116 (9.1)	5 (10.9)	10 (9.4)	56 (8.2)	45 (10)	0.741
CKD, No. (%)	42 (3.3)	3 (6.5)	1 (0.9)	26 (3.8)	12 (2.7)	0.211
COPD, No. (%)	24 (1.9)	2 (4.3)	2 (1.9)	17 (2.5)	3 (0.7)	0.088
CCI (weighted)	0 [0, 1]	1 [0, 3]	0 [0, 1]	0 [0, 2]	0 [0, 1]	<0.001
Betablockers (home meds)	262 (20.5)	15 (32.6)	16 (15.1)	162 (23.9)	69 (15.4)	<0.001
LVEF within 6 months prior (%)	65 [60, 68]	65 [53, 65]	65 [61.25, 68.5]	65 [60, 68]	65 [60, 69]	0.228
Intraoperative factors						
Mitral valve procedure performed, No. (%)						<0.001
Repair	1101 (86.0)	39 (84.8)	102 (96.2)	529 (77.9)	431 (96.0)	
Replacement	179 (14)	7 (15.2)	4 (3.8)	150 (22.1)	18 (4.0)	
CPB (median ± IQR), min	134 [106, 163.5]	139 [123.5, 157]	164 [146.25, 190.75]	111 [90, 142]	151 [130, 178.25]	<0.001
Aortic cross clamp time (median ± IQR), min	93 [75, 115]	82 [73.5, 89.5]	111 [101, 131.75]	84 [66, 107]	101 [85.5, 120.5]	<0.001
Operation time (median ± IQR), hours	244 [218, 280]	233 [201.25, 263]	264 [237.5, 295.5]	235 [205.5, 275]	251 [229, 283]	<0.001
Postoperative characteristics (before hospital discharge)						
POAF in the hospital, No. (%)	**406 (31.7)**	**14 (30.4)**	**13 (12.3)**	**284 (41.8)**	**95 (21.2)**	**<0.001**
Length of ICU stay (LOS, median ± IQR), days	1.24 [0.97, 2.5]	1.87 [1.06, 2.63]	1.09 [0.88, 2.14]	1.74 [1, 2.96]	1.13 [0.96, 2.04]	<0.001
Length of hospital stay (median ± IQR), days	5.11 [4.15, 7.03]	5.04 [4.75, 6.49]	4.89 [3.85, 5.88]	5.95 [4.88, 7.84]	4.94 [3.99, 6.04]	<0.001

*Kruskal-Wallis tests for non-normally distributed continuous variables and chi-square or Fisher's exact tests for categorical variables
IQR – interquartile range, **BMI** – body mass index, **BSA** – body surface area, **CHF** – congestive heart failure, **CAD** – coronary artery disease, **CKD** – chronic kidney disease, **COPD** – chronic obstructive pulmonary disease, **CCI** – Charlson Comorbidity Index, **LVEF** – left ventricular ejection fraction, **LAVI** – left atrial volume index, **CPB** – cardiopulmonary bypass, **LOS** – length of stay, **POAF** – postoperative atrial fibrillation

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SP 2 - Kiosk 1 - Poster 2

Renal Recovery After Cardiac Surgery-Associated Acute Kidney Injury in Adults: A Systematic Review

Presenting Author: *Kabungo Mulumba, MD, MBA*

Background: Cardiac surgery–associated acute kidney injury (CSA-AKI) significantly increases long-term mortality and progression to chronic kidney disease (CKD). Although the incidence and risk factors of CSA-AKI are well described, the epidemiology of renal recovery remains poorly defined, and recovery criteria vary substantially across studies. We conducted a systematic review to characterize recovery rates, definitions, and predictors of renal recovery after CSA-AKI across cardiac surgical populations.

Methods: A protocol was registered on PROSPERO. MEDLINE and Cochrane CENTRAL were searched for studies published from 2000 onward evaluating adults who developed AKI after cardiac surgery requiring cardiopulmonary bypass (CPB). Eligible study designs included prospective and retrospective cohorts, case-control studies, case series, randomized trials, and systematic reviews. Screening and full-text review were performed in duplicate, with conflicts resolved by a third reviewer. Data extraction and risk of bias assessment were performed using the Newcastle–Ottawa Scale (NOS). Recovery definitions, timing of assessment, recovery rates, and predictors were synthesized descriptively due to heterogeneity.

Results: The search yielded 5,286 publications; 30 studies met criteria after screening (Figure 1). Studies represented diverse surgical populations, including coronary artery bypass graft (CABG)/valvular/aortic procedures, Left ventricular assist device (LVAD) implantation, bridge-to-transplant LVAD (BTT-LVAD), and heart transplantation. Recovery definitions were highly heterogeneous across studies, encompassing dialysis discontinuation, return of serum creatinine to baseline, eGFR-based thresholds, or improvement in CKD stage (Figure 3). Half of studies were rated low risk of bias, 40% moderate, and 10% high (Figure 2). Recovery rates varied by surgical

category (Figure 4): 1) CABG/valvular/aortic: 55–65%. 2) LVAD: 33–67% 3) BTT-LVAD: 40–60% recovered prior to transplant, with additional improvement post-transplant. 4) Heart transplant: approximately 66% recovery among patients with transient renal replacement therapy (RRT) and less than 30% among those with persistent dialysis dependence.

Follow-up ranged from 48 hours to greater than 1 year, limiting comparability. Consistent predictors of renal recovery included younger age, higher baseline eGFR, shorter CPB duration, shorter LVAD support, and transient rather than persistent RRT need. Predictors of non-recovery included older age, pre-existing CKD, higher transfusion burden, cardiorenal anemia, elevated right-sided pressures, and prolonged CPB or ischemic time.

Conclusion: Renal recovery after CSA-AKI is highly variable and is defined inconsistently across studies, precluding quantitative pooling and limiting prognostic accuracy. Across surgical categories, approximately 30-70% of patients experience renal recovery. Younger age, preserved baseline kidney function, shorter CPB and LVAD durations, and transient RRT requirement consistently favor recovery. Standardizing recovery definitions and extending follow-up are essential for future studies aimed at improving prognostication and guiding postoperative and long-term management for patients with CSA-AKI.

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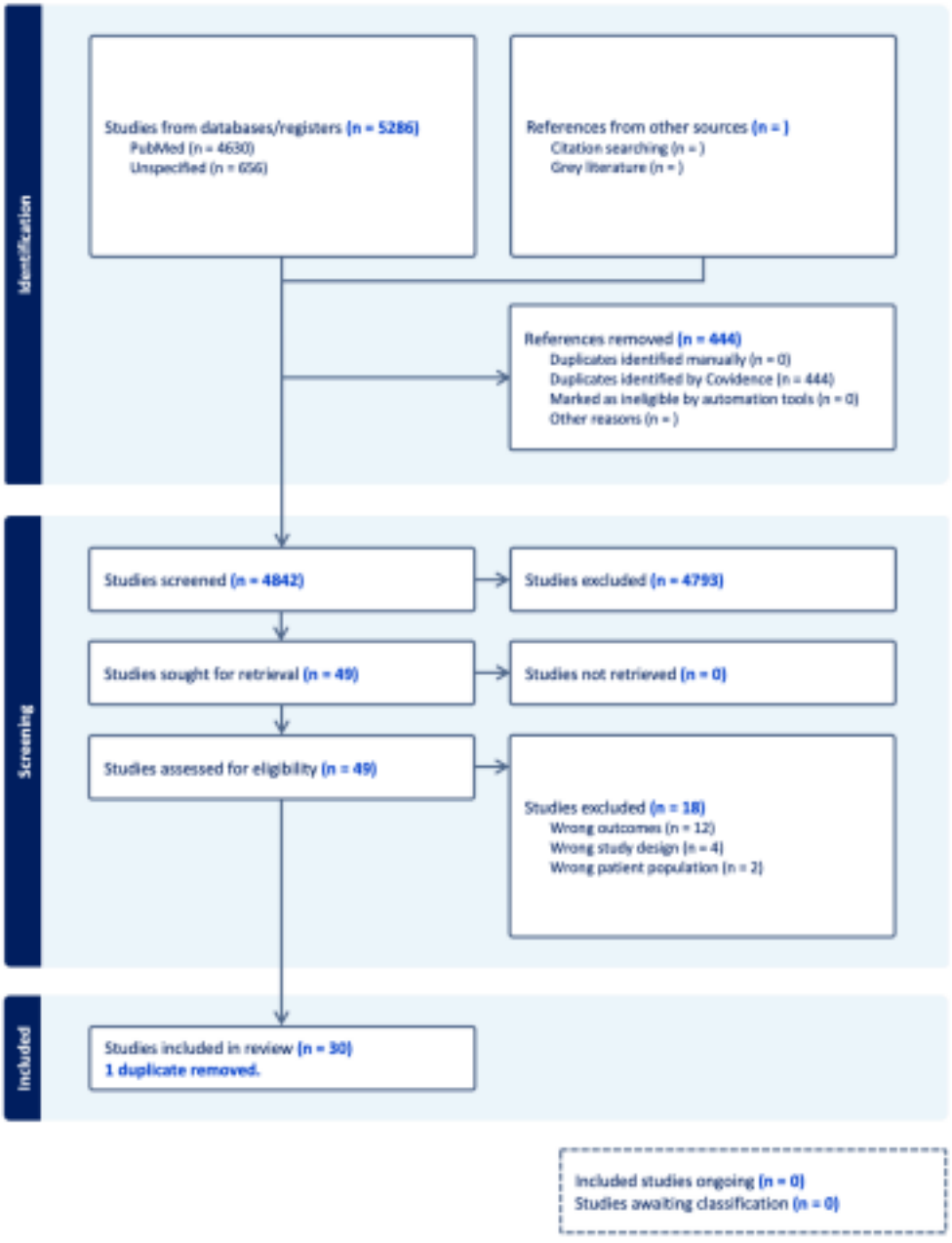


Figure 1: Identification of studies via databases and registers. 5286 articles identified on PubMed and Cochrane libraries. 30 studies identified for systemic review.



Fig 2. Risk of bias for multiple outcomes according Newcastle-Ottawa Scale (NOS) tool: summary of review authors' judgment about each domain, presented as 1) judgement across each domain per study and as 2)percentages across included studies.

Recovery Definition	Number of Studies
RRT/Dialysis Discontinuation	13
Complete reversal of AKI within 48 h of AKI onset.	1
Post surgery serum creatinine returning to or below baseline within 1–30 days	1
Return of serum creatinine to <1.5x the baseline at the time of survival discharge	1
Improvement in eGFR that resulted in a CKD stage change to one of lesser severity.	1
eGFR trend over time	4
Serum creatinine returns to <50% above baseline within 48 h of AKI onset (ADQI consensus).	1
Decrease of AKI stage based on KDIGO criteria	2
Decrease in postoperative creatinine values.	1
SCr concentrations <= 44mmol/L above baseline values	1
Reduction in SCr to <1.25 times baseline SCr	1
Decrease in peak creatinine no higher than baseline values after CABG and no new dialysis by postoperative day 7 (or at discharge if this occurred before postoperative	1
SCr being lower than baseline SCr + 29.74 µmol/L at discharge or death.	1
1)ATN : SCr at discharge returned to within baseline SCr + 0.5 mg/dL; 2) ADQI: returned to within 50% above baseline SCr; 3) Pannu et al. returned to within 25% above baseline SCr; 4) KDIGO [6]: estimated glomerular filtration rate (eGFR) at discharge ≥60 mL/min/ 1.73 m2 (Chronic Kidney Disease Epidemiology Collaboration equation (CKD-EPI formula)) and 5) Bucaloiu et al.: eGFR at discharge ≥90% of baseline eGFR	1

Fig 3. Definitions of renal recovery employed across various studies

Category	Count	Incidence of AKI	RRT Requirement	Renal Recovery	Predictors of non-recovery	Predictors of Renal Recovery	Key findings
CABG, Valvular and Aortic Surgeries	18	25 - 30%	1- 10%	55 -65% recovered	older age, baseline CKD, prolonged CPB and ischemic time, large transfusion burden, low mean perfusion pressure reflecting renal hypoperfusion and venous congestion	younger age, shorter CPB duration, shorter RRT duration	renal recovery improved survival . Even recovered patients faced higher late mortality and CKD risk compared to those who never developed AKI. Persistent AKI (Cr not returning to baseline) was strongest predictor of 5-year mortality in one study
Heart Transplants	4	10 - 20%	12 - 15% peri-transplant	66% of patients requiring transient pre-transplant RRT recovered. <30% recovery in patients with persistent RRT dependence. In on study 49% of persistent RRT patients developed dialysis dependence ESRD at 10 years.	ongoing AKI or need for RRT predicted high mortality. Presence of cardio-renal anemia increased one-year mortality	transient renal dysfunction from heart failure and not CKD	recovery common in patients with transient renal dysfunction but rare in persistent or structure kidney disease. Transient improvement in eGFR post transplant wanes over time due to nephrotoxic immunosuppresive drugs
LVAD Implantations	5	20 - 30%	14 - 20%	33 - 67% based on indepdance from RRT or improvement in CKD stage	pre-existing CKD, advanced age, elevated right atrial pressures, prolonged CPB and abnormal renal imaging	younger age, higher baseline eGFR and hemodynamic unloading	Early functional improvement in kidney function 2-3 months post LVAD. Benefit diminishes over time, particulalary beyond one year. Renal recovery correlates with survival
BTT LVAD Implantations (LVAD + HT)	3	45 - 60%	10 - 20%	40- 60% had renal recovery close to baseline after LVAD and additional 20-30% had further improvement after transplantation	lower baseline eGFR, prolonged LVAD duration, older age, extended CDPB and ischemic time during transplantation.	shorter LVAD support, higher pre-transplant eGFR, younger age	>25% improvement in eFGR sustained over 3 months associated with 40-55% reduction in mortality. >50% of patients with reduced renal function who undergo BTT-LVAD show improvement in renal function at transplant

Fig 4. Summary of findings by type of Surgery. LVAD (Left Ventricular Assist Device), BTT (Bridge To Transplant), AKI (Acute kidney Injury), CKD(Chronic Kidney Disease) . Cr,(Creatinine) RRT (Renal Replacement therapy), CABG (Coronary Bypass Graft Surgery),

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SP 2 - Kiosk 1 - Poster 3

Beta-adrenergic agonists to differentiate low-flow states in the left atrial appendage (LAA) from thrombus on transesophageal imaging: Use cases for ephedrine and isoproterenol during Watchman implant

Presenting Author: *Jacob Schaff, MD*

Background: Left atrial appendage (LAA) occlusion is increasingly used to reduce thromboembolic risk in patients with atrial fibrillation. Accurate pre-procedural TEE assessment is critical to identify true thrombus, which is a contraindication to device placement, versus low-flow states such as spontaneous echo contrast (SEC) or "sludge," which may mimic thrombus. Traditional imaging relies on static echogenicity and Doppler velocities, but differentiation can remain uncertain.

Mechanistic Rationale: LAA emptying is highly sensitive to atrial contractility, preload, and heart rate. Low velocities (< 20 cm/s) promote blood stasis and SEC formation, potentially mimicking thrombus. Beta-adrenergic agonists enhance atrial contractility and heart rate, increasing LAA flow. Isoproterenol, a non-selective beta-1/beta-2 agonist, directly increases atrial contractility and LAA emptying velocity. Ephedrine, an indirect sympathomimetic, improves systemic blood pressure and preload, producing a modest increase in LAA flow. Observing the response of echogenic material to pharmacologic augmentation allows differentiation of reversible low-flow SEC from persistent thrombus.

Case-Based Illustration: We report a series of patients undergoing pre-procedural TEE for LAA occlusion planning with indeterminate LAA echogenicity. Ephedrine (5–10 mg IV) was used to optimize systemic hemodynamics, followed by titrated isoproterenol infusion (1–10 µg/min) to augment LAA contractility and emptying velocities, continuously monitored via Doppler.

· In cases of low-flow SEC, echogenic material resolved with increased LAA emptying velocity (from 14 ± 3 cm/s to 28 ± 5 cm/s), confirming absence of thrombus and allowing safe device planning.

· In cases of true thrombus, echogenic material persisted despite maximal tolerated pharmacologic augmentation, confirming the contraindication for LAA occlusion.
No sustained arrhythmic complications were observed, highlighting the safety of carefully titrated beta-agonist use in this context.

Discussion: Dynamic pharmacologic assessment of LAA function during pre-procedural TEE adds a functional dimension to traditional imaging, improving diagnostic confidence in differentiating low-flow states from thrombus. Isoproterenol provides a more predictable and robust increase in LAA velocities compared with ephedrine. This approach has the potential to reduce unnecessary procedure cancellations or delays and improve peri-procedural safety.

Conclusion: Beta-adrenergic agonists represent a practical and physiologically guided adjunct in pre-procedural TEE evaluation for LAA occlusion. Augmentation of LAA flow can help in distinguishing low-flow SEC from thrombus, facilitating safe and timely device placement.

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SP 2 - Kiosk 1 - Poster 4

Automated Echocardiographic Assessment of Impella Micro-Axial Flow Pump Depth Using Machine Learning-Based Approach (Impella™ SmartView): A Real-World Registry Analysis

Presenting Author: *Suzanne Bennett, MD, FCCM*

Background: Appropriate positioning of Impella micro-axial flow pumps (mAFP) across the aortic valve is essential to optimize ventricular unloading and minimize device-related complications. Transthoracic echocardiography (TTE), particularly the parasternal long-axis (PLAX) view, is the standard modality for assessing mAFP depth; however, measurement is challenged by variable image quality and substantial inter- and intra-observer variability. Machine-learning (ML)–based echocardiographic analysis may improve reproducibility and reduce clinical burden, but requires validation against expert human interpretation within real-world datasets.

Methods: We analyzed PLAX TTE loops from the prospective, multicenter Long-Term Outcome and Quality Indicator Impella (LOQI) Registry, including patients supported with Impella CP or 5.5 devices. From 76 patients, 200 echocardiographic loops were sampled, including repeated loops to assess intra-observer variability. Three expert clinicians independently performed manual mAFP depth measurements. An ML-based algorithm (Impella SmartView, UltraSight Ltd.) automatically identified the aortic valve plane and pump inlet to compute insertion depth. Algorithm performance was evaluated using mean absolute error (MAE) against expert consensus, with a prespecified clinical acceptability threshold of < 0.5 cm, and contextualized relative to inter- and intra-observer variability.

Results: The ML-based approach achieved an overall MAE of 0.45 cm (95% CI 0.40–0.50) compared with expert consensus, meeting the prespecified performance goal. Agreement for loop acceptability classification was moderate (κ = 0.51), with high sensitivity for acceptable Image

Uploads. Inter-observer MAE ranged from 0.46 to 0.71 cm, while intra-observer MAE ranged from 0.28 to 0.50 cm. Algorithm performance was comparable to intra-observer variability for two of three observers and demonstrated less variability than inter-observer agreement.

Conclusions: In this multicenter, real-world feasibility study, automated ML-based assessment of mAFP depth from standard TTE loops demonstrated accuracy comparable to expert human interpretation and within the bounds of inherent observer variability. Integration of ML-based mAFP depth assessment may improve consistency of device management and reduce clinical workload in patients supported with mechanical circulatory support.

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SP 2 - Kiosk 1 - Poster 5
Microplastics in Cardiopulmonary Bypass Circuits

Presenting Author: *Zeyu Song, MS, CCP*

Background: Microplastics (< 5 mm) have been identified in human blood and multiple organs, suggesting ongoing environmental and iatrogenic exposure. Cardiopulmonary bypass (CPB) circuits contain polymer-based components and may introduce microplastics directly into the circulation, yet this potential exposure pathway remains largely uncharacterized.

Objective: To quantify microplastics present within CPB circuits and associated perfusate and to determine whether exposure to volatile anesthetics influences microplastic release.

Methods: An in vitro CPB model was circulated with Plasmalyte A under five conditions: Plasmalyte A sampled directly from the manufacturer's bag, Plasmalyte A after a 0.2 µm pre-bypass filter only, CPB with a pre-bypass filter and no volatile anesthetic, CPB with 5% isoflurane, and CPB with 8% sevoflurane. Circuits were standardized for flow, temperature, duration, and tubing configuration. Post-oxygenator samples were analyzed using two-stage vacuum filtration, stereomicroscopy, and pyrolysis–gas chromatography/mass spectrometry to detect and quantify polymers commonly present in CPB components, including polymethyl methacrylate (PMMA), polypropylene (PP), polystyrene (PS), polyvinyl chloride (PVC), polyethylene (PE), and nylon 6 (PA6).

Results: Microplastics were detected under all conditions, including Plasmalyte A sampled directly from the manufacturer's bag. Concentrations were 5.73 ± 3.97 µg/L after filtration alone, 6.84 ± 2.18 µg/L with isoflurane, and 7.24 ± 5.33 µg/L with sevoflurane, with no significant difference between CPB groups (ANOVA $p = 0.748$). The 0.2 µm pre-bypass filter reduced microplastic mass by approximately 23.6% (0.887 → 0.678 µg/L) but did not eliminate particles. Multiple polymer types were identified across all CPB conditions.

Conclusion: CPB circuits represent a potential source of intravascular microplastic exposure during cardiac surgery. Although filtration reduced particulate burden, microplastics remained detectable, and volatile anesthetic exposure did not significantly increase release. These findings support further investigation into ultrafine filtration strategies and circuit material selection to mitigate microplastic exposure during CPB.

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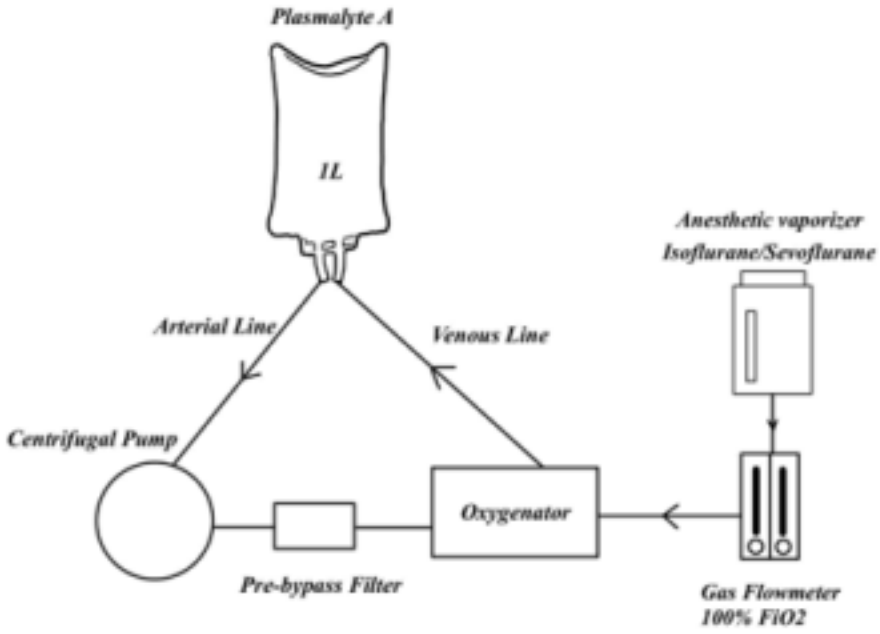


Figure 1. Schematic of the CPB circuit used for microplastic analysis, including pre-bypass filtration and optional volatile anesthetic exposure.

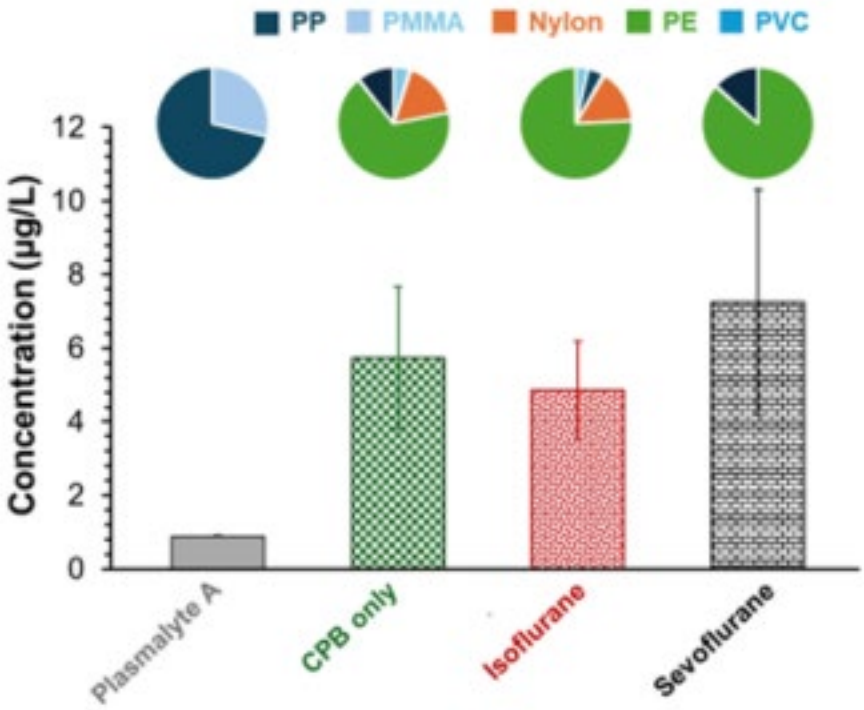


Figure 2. Concentrations and chemical compositions of microplastics under four conditions: (1) Plasmalyte A, (2) CPB only, (3) Isoflurane, and (4) Sevoflurane. The fraction of PS is much smaller than the other polymer types and is therefore not shown in the pie chart.

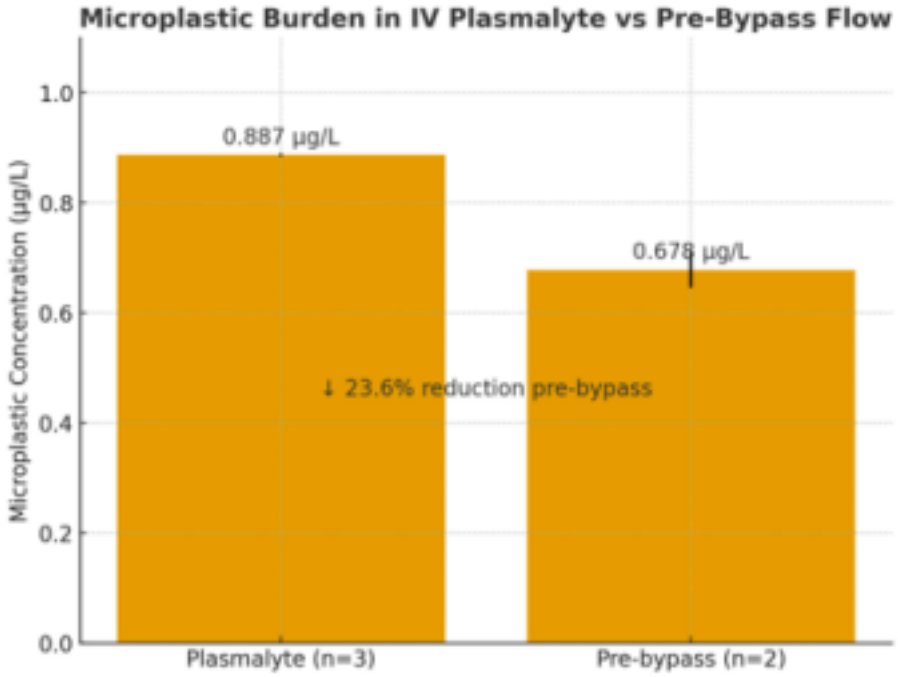


Figure 3. Mean microplastic mass was higher in Plasmalyte A sampled directly from the bag (0.887 µg/L) compared with fluid measured after going through the pre-bypass filter (0.678 µg/L). Values represent the combined average of three Plasmalyte samples and two filtered samples, demonstrating an approximate 23.6% reduction in microplastic concentration before entering the bypass circuit. Error bars indicate standard deviation.

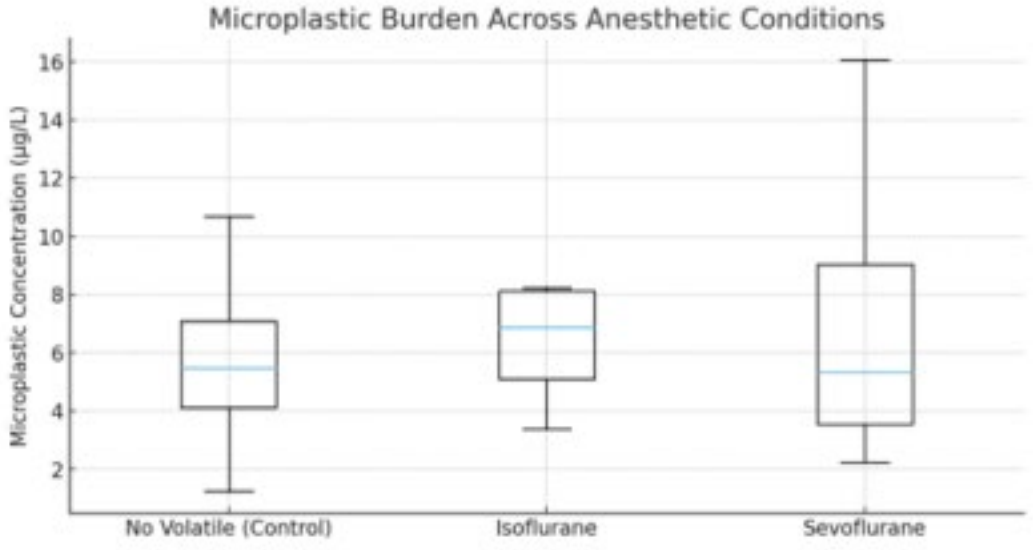


Figure 4. Microplastic concentration distributions for perfusate exposed to no volatile anesthetic (control), isoflurane, and sevoflurane. Each condition represents n=4 runs. Median, interquartile range, and outlier values are shown.

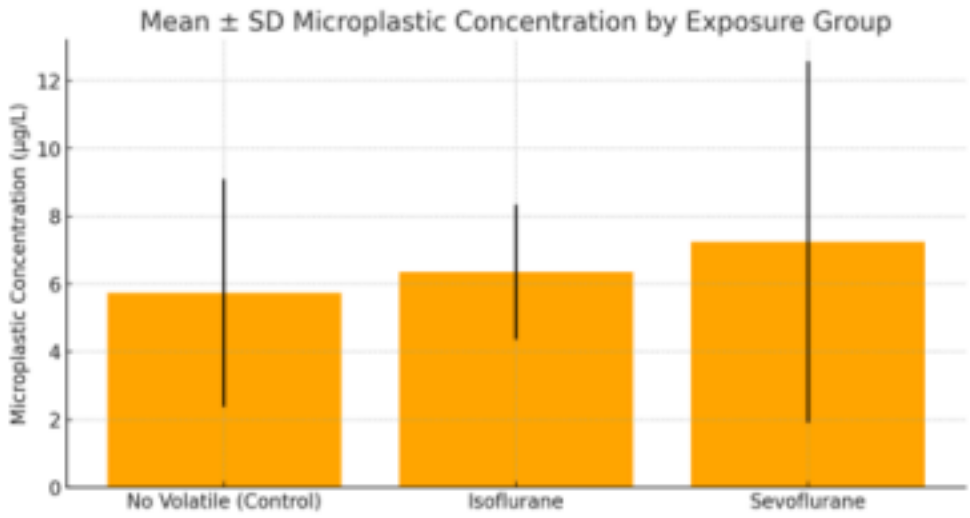


Figure 5. Mean ± SD microplastic concentration in perfusate under three exposure conditions: control (no volatile), isoflurane, and sevoflurane (n=4 per group). Sevoflurane demonstrated the highest mean value, although ANOVA showed no statistically significant difference between groups.

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SP 2 - Kiosk 1 - Poster 6

The Impact of Early Extubation and Core Temperature on the Patient Undergoing Open Heart Surgery

Presenting Author: *Steven Insler, DO, FAOCA*

Background: Extubation time and postoperative core body temperature may influence recovery and complications following cardiothoracic surgery (CTS). Prolonged intubation (>6 hours) and hypothermia (core temperature < 36°C) have been associated with increased complications, higher healthcare costs, and extended hospital stays.

Methods: From an initial cohort of 8,112 patients undergoing CTS between January 2023 and April 2024, 4,890 met inclusion criteria. Patients were stratified by extubation time (< 6 vs. ≥6 hours) and postoperative temperature (< 36°C vs. ≥36°C). Data collected included demographics, ventilation duration, infection status, procedure type, and length of stay. Non-parametric tests (Wilcoxon rank-sum, Chi-square/Fisher's exact) were used due to non-normal distributions, with significance set at $\alpha = 0.05$.

Results: Most variables differed significantly between groups, except postoperative atrial fibrillation (AFib). Patients extubated ≥6 hours with temperatures < 36°C upon CVICU arrival had significantly longer hospital stays and higher incidence of acute kidney injury (AKI) (Tables 1 and 2). Complex procedures (e.g., AVR + MV replacement, MV repair + CAB) were consistently associated with increased AKI risk and prolonged hospitalization. AFib rates remained stable across groups but trended lower in patients extubated within 6 hours and with temperatures ≥36°C.

Conclusions: Extubation time, postoperative temperature, and procedure complexity are key determinants of outcomes following CTS, particularly AKI and length of stay. AFib incidence appears largely unaffected, though a trend toward reduced risk was observed in patients extubated early and normothermic postoperatively.

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Table 1. Summary for Patients who Extubate < 6 hours, Temp ≥ 36 C

Characteristic	AVR N = 336[1]	AVR + CAB N = 103[1]	AVR + MV Replace N = 22[1]	CAB N = 430[1]	MV Repair N = 292[1]	MV Repair + CAB N = 30[1]	MV Replace N = 54[1]	MV Replace + CAB N = 7[1]	Other N = 581[1]	p-value[2]
Length of Stay										<0.001
Mean (SD)	7.9 (4.9)	10.5 (8.4)	13.1 (5.9)	9.5 (5.5)	6.4 (2.8)	9.5 (5.6)	11.9 (8.3)	15.6 (16.8)	9.0 (5.3)	
(Min-Max)	(4.0-44.0)	(5.0-61.0)	(6.0-28.0)	(4.0-79.0)	(4.0-26.0)	(5.0-29.0)	(4.0-47.0)	(6.0-50.0)	(4.0-38.0)	
Median (Q1, Q3)	6.0 (5.0, 8.0)	8.0 (6.0, 11.0)	12.0 (9.0, 16.0)	8.0 (6.0, 12.0)	6.0 (5.0, 7.0)	7.5 (6.0, 10.0)	9.0 (7.0, 15.0)	7.0 (6.0, 26.0)	7.0 (6.0, 10.0)	
Unknown	0	0	0	0	0	0	0	0	1	
Postoperative AFib within 48 hours	67 (19.9%)	19 (18.4%)	3 (13.6%)	86 (20.0%)	51 (17.5%)	9 (30.0%)	10 (18.5%)	2 (28.6%)	99 (17.0%)	>0.99
Postoperative Acute Kidney Injury	25 (7.4%)	14 (13.6%)	4 (18.2%)	47 (10.9%)	11 (3.8%)	5 (16.7%)	7 (13.0%)	1 (14.3%)	60 (10.3%)	0.004

[1]n (%)

[2]Kruskal-Wallis rank sum test; Fisher's Exact Test; p-values adjusted using the Holm–Bonferroni procedure

Table 2. Summary for Patients who Extubate ≥ 6 hours, Temp < 36 C

Characteristic	AVR N = 89[1]	AVR + CAB N = 66[1]	AVR + MV Replace N = 34[1]	CAB N = 126[1]	MV Repair N = 31[1]	MV Repair + CAB N = 41[1]	MV Replace N = 33[1]	MV Replace + CAB N = 25[1]	Other N = 533[1]	p-value[2]
Length of Stay										<0.001
Mean (SD)	11.5 (8.8)	17.4 (13.1)	21.1 (14.4)	17.0 (10.3)	11.1 (5.5)	18.3 (11.6)	22.1 (15.8)	20.4 (16.2)	20.7 (17.4)	
(Min-Max)	(4.0-53.0)	(6.0-72.0)	(5.0-68.0)	(5.0-74.0)	(5.0-27.0)	(6.0-48.0)	(6.0-65.0)	(7.0-70.0)	(5.0-135.0)	
Median (Q1, Q3)	8.0 (7.0, 12.0)	13.5 (8.0, 24.0)	16.5 (13.0, 25.0)	15.0 (10.0, 20.0)	9.0 (7.0, 13.0)	14.0 (9.0, 25.0)	16.0 (9.0, 28.0)	14.0 (10.0, 24.0)	15.0 (9.0, 25.0)	
Postoperative AFib within 48 hours	21 (23.6%)	16 (24.2%)	6 (17.6%)	24 (19.0%)	10 (32.3%)	10 (24.4%)	9 (27.3%)	7 (28.0%)	115 (21.6%)	>0.99
Postoperative Acute Kidney Injury	12 (13.5%)	17 (25.8%)	5 (14.7%)	42 (33.3%)	8 (25.8%)	14 (34.1%)	11 (33.3%)	14 (56.0%)	194 (36.4%)	<0.001

[1]n (%)

[2]Kruskal-Wallis rank sum test; Pearson's Chi-squared test; p-values adjusted using the Holm–Bonferroni procedure

SP 2 - Kiosk 2 - Poster 1

Association Between Intraprocedural Hypotension and Postoperative Acute Kidney Injury After Transcatheter Tricuspid Valve Repair - A Retrospective Cohort Study.

Presenting Author: *Arun Muthukumar, MD*

Introduction: Transcatheter tricuspid valve repair (TTVR) is increasingly performed in high-risk

patients, typically under general anesthesia. [1] Although these patients often have multiple comorbidities that elevate AKI risk, the association between intraprocedural hypotension and postoperative AKI remains unclear. [2] Therefore, we conducted a study to determine whether total hypotension duration during TTVR is independently associated with postoperative AKI.

Methods: After Institutional Review Board approval (Henry Ford Hospital), we conducted a retrospective cohort study of 140 patients from 168 who underwent TTVR under general anesthesia between January 2024 and July 2025. Continuous variables were summarized as mean ± SD or median (IQR), and categorical variables as counts (percentages). Groups with and without AKI were compared using Student's t-test or Wilcoxon rank-sum test for continuous variables; χ^2 or Fisher's exact tests for categorical variables.

The exposure was total intraprocedural hypotension duration, defined as cumulative minutes with mean arterial pressure (MAP) < 65 mmHg. The primary outcome was postoperative AKI, defined as a binary variable according to KDIGO creatinine criteria (presence vs absence of AKI as 0 vs 1, during the index hospitalization). A multivariable logistic regression assessed association between hypotension duration and AKI, adjusting for baseline creatinine, age, body mass index (BMI), and left ventricular ejection fraction (LVEF). A secondary analysis categorized hypotension duration into 0–5, 6–20, and >20 minutes, with 0–5 minutes as reference, adjusting for baseline creatinine. Two-sided $p < 0.05$ was considered statistically significant and analyses were performed using a LOGIT model (SAS v9).

Results: A total of 140 patients had complete data for baseline creatinine, total hypotension duration, and AKI status. The mean age was 77.4 ± 9.9 years, mean BMI was 27.6 ± 5.6 kg/m^2, and mean LVEF was 53.5 ± 11.7%. AKI occurred in 42 (25.0%) patients.
The median total duration of intraprocedural hypotension (MAP < 65 mmHg) was 10 minutes (IQR 1–25), with a mean of 17.1 ± 21.2 minutes. Patients who developed AKI had clinically longer hypotension compared with those without AKI (19.4 ± 25.3 vs 16.4 ± 19.7 minutes; p = 0.48). Baseline creatinine was higher in patients with AKI (2.19 ± 1.70 vs 1.39 ± 1.34 mg/dL; p = 0.0075).

In the primary multivariable model adjusting for baseline creatinine, age, BMI, and LVEF, total hypotension duration was not significantly associated with AKI (adjusted OR 1.01 per additional minute; 95% CI 0.99–1.03; p = 0.47). In contrast, several variables had independent association with post-operative AKI: baseline creatinine (adjusted OR 1.52 per 1 mg/dL; 95% CI: 1.18–1.94; p = 0.0013), older age (OR 1.06; 95% CI: 1.01–1.11; p = 0.018), higher BMI (OR 1.08; 95% CI: 1.00–1.16; p = 0.040), and lower LVEF (OR 0.96; 95% CI: 0.93–0.99; p = 0.022). Neither 6–20 minutes nor >20 minutes of hypotension during the procedure was associated with increased AKI risk compared to 0–5 minutes cohort (p>0.50).

Conclusion: No significant independent association was identified between total intraprocedural hypotension duration and AKI in patients who underwent TTVR under general anesthesia.

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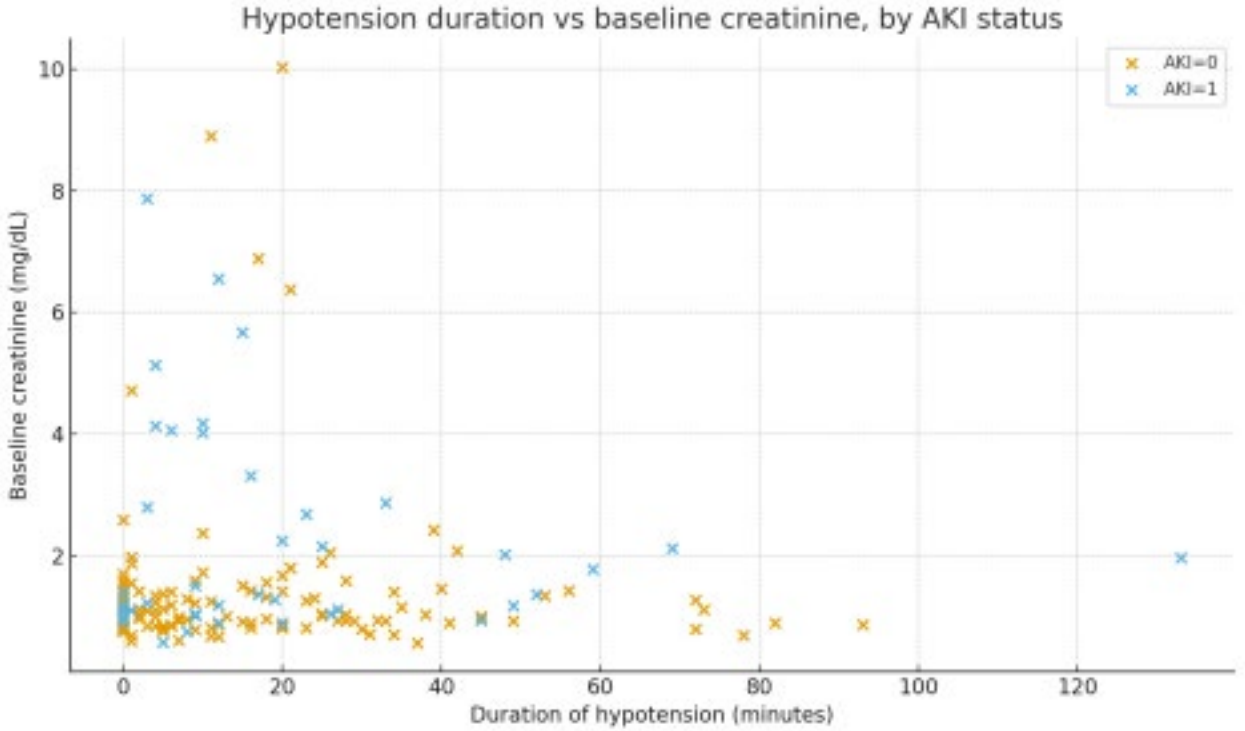


Table 1: Demonstrates the relation between independent variables and post-operative Acute Kidney Injury.

Predictor Variable	*OR*	*95% CI for OR*	*p-value*
Total hypotension duration (per min)	1.01	0.99 – 1.03	0.47
Baseline creatinine (per 1 mg/dL)	1.52	1.18 – 1.94	0.0013
Age (per year)	1.06	1.01 – 1.11	0.018
BMI (per kg/m²)	1.08	1.00 – 1.16	0.040
LVEF (per 1% increase)	0.96	0.93 – 0.99	0.022

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SP 2 - Kiosk 2 - Poster 2
Incidentally Diagnosed Double Inferior Vena Cava during a Right Atrial Thrombectomy
Presenting Author: *Sidney Akabogu, MD*

Introduction Paragraph: A dual inferior vena cava (IVC) system is a rare congenital anomaly occurring in approximately 1-3% of the population*.This condition results from the persistence of both left and right supracardinal veins during embryonic development*. In a typical dual IVC system, two distinct IVCs arise from each iliac vein, with the left IVC crossing to the right side at the level of the left renal vein. While usually asymptomatic, understanding this variation in anatomy is crucial for accurate diagnoses, surgical planning, and prevention of potential issues in these patients. We present a case of a patient with an IVC filter who was undergoing a right atrial thrombectomy and was found to have a dual IVC system.

Brief Summary of the Clinical Case: A 27-year-old female with a past medical history of antiphospholipid syndrome, IVC filter placement and anticoagulation therapy due to a history of deep venous thromboses (DVT) and pulmonary emboli (PE) presented with increasing dyspnea on exertion. She had recently had a miscarriage and a subsequent dilation and curettage (D&C) and had been suffering from daily vaginal spotting since. Of note, the patient reported that she had discontinued her anticoagulation since her D&C.
Initial investigations included a transthoracic echocardiogram (TTE) that showed normal right ventricular (RV) function, no tricuspid regurgitation, but did exhibit a moderately sized thrombus in the right atrium with evidence of microcalcifications. After a multidisciplinary team discussion, it was felt that even though the patient was hemodynamically stable, it was prudent to proceed with a transcatheter removal of the thrombus under general anesthesia using an alphavac system.
After an uneventful induction of general anesthesia, placement of left radial arterial line, and a right internal jugular vein double lumen central line a transesophageal echocardiogram (TEE) was performed. The TEE confirmed normal RV function and normal valvular anatomy.
However, the right atrial (RA) mass was attached to a dual caval system septum at the cavoatrial junction. On further investigation, one cava was standard and the other extended directly into the hepatic vein. The findings were reported to the interventional cardiologists, and the rest of the procedure was uneventful with a successful aspiration of the RA mass.

Discussion: Increased risk of DVTs: slower venous return can increase the risk of clot formation

Challenges with thrombosis management may warrant special considerations for treatment IVC filter placement in a patient with a dual caval system
Preventing misdiagnosis which could prevent unnecessary interventions or delayed proper treatment
Surgical Planning
Potential anatomical complications with a dual caval system

Impact of Echocardiographic findings: The precise location of the origin of the dual caval system, and the knowledge of the congenital anomaly is essential for surgical planning during retroperitoneal vascular procedures Hemodynamically unstable patients that require ECMO, may require additional investigation on where to cannulate

Teaching Points of the Case: While most cases with a dual caval system are asymptomatic, awareness of this anatomical variation is crucial for a cardiovascular anesthesiologist to be aware of to ensure proper diagnosis and management of potential issues.

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SP 2 - Kiosk 2 - Poster 3
Continuous Spinal Blocks in Patients with Major Cardiac Comorbidities: A Case Series
Presenting Author: *Matthew Fitzsimons, MD, MBA*

Introduction: As medical science and clinical care have improved, an increasing number of patients with major cardiac comorbidities are undergoing non-cardiac surgeries. These lesions include coronary artery disease in the setting of severely reduced ejection fraction, aortic stenosis (AS), and severe pulmonary hypertension. This population presents several challenges to anesthetic management; their lesions typically make them sensitive to changes in preload, afterload, and heart rate which can be precipitated by the induction of anesthesia. While preoperative optimization can alleviate some of these challenges, urgent or time-sensitive surgeries may preclude the ability to properly optimize a given patient. One such example are arthroplasties after trauma.

Where lower-extremity orthopedic procedures are traditionally amenable to regional anesthetic techniques, such as intrathecal blocks; such techniques can precipitate changes in hemodynamic

parameters that are poorly tolerated in patients with these cardiac comorbidities, in particular those with fixed cardiac output lesion, such as AS. A continuous spinal technique alleviates this side effect by allowing slow titration of the medication to the desired anesthetic level. A literature review demonstrates that continuous spinal blocks have been utilized to great effect in the field of obstetrics and orthopedics. However, there are no examples of this technique being utilized in patients with fixed cardiac output lesions.

This case series highlights five anesthetics where a continuous spinal block was administered to patients with fixed cardiac output lesions undergoing non-cardiac surgical procedures.

Case Series:
Case 1:
An 82-year-old female with severe pulmonary hypertension undergoing a hip hemiarthroplasty after trauma.
Case 2:
An 81-year-old female with an nSTEMI undergoing an intermedullary nail fixation after trauma. Case 3:
A 72-year-old female with severe pulmonary hypertension and HOCM undergoing perineal proctectomy.
Case 4:
A 79-year-old male with severe aortic stenosis undergoing a left total knee replacement with revision to a spacer for a prosthetic joint infection.
Case 5:
A 88-year-old male with severe coronary artery disease and severe aortic stenosis undergoing a hip hemiarthroplasty after trauma.

Results: All patients successfully had their procedures performed under continuous spinal technique with an intrathecal catheter without conversion to general anesthesia. The highest dose of infusion across all patients was 0.08 mcg/kg/min norepinephrine equivalents. The highest total of vasoactive agents used was 0.8mg norepinephrine equivalents. 3 of 5 patients were sedated with dexmedetomidine; 1 of 5 was sedated with propofol. 1 of 5 was not given additional sedation. The patient receiving propofol required the highest quantity of vasoactive agents. Conclusion: Traditionally, patients with fixed cardiac output are typically relegated to general anesthesia. Severe aortic stenosis and/or severely reduced ejection fraction precludes the patient from generating an increase in cardiac output to overcome the reduction in afterload associated with single shot spinal. Here we demonstrate that a continuous spinal technique allows for neuraxial approach when desired without hemodynamic collapse.

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SP 2 - Kiosk 2 - Poster 4
Methadone: Are Two Dose Better Than One in Cardiac Surgical Patients?
Presenting Author: *Michael Wisniewski, MD*

Background: Methadone is a Mu opioid agonist, N-methyl-d-aspartate (NMDA) receptor antagonist, and Serotonin-Norepinephrine Re-uptake Inhibitor, offering long-acting multimodal pain control via administration of a single drug. Its rapid onset of action and long elimination half-life make it advantageous for cardiac

surgical procedures, where there is a need for rapid analgesia intraoperatively and long-acting analgesia postoperatively. While methadone has been shown in surgical populations to improve pain scores and reduce postoperative opioid consumption, the optimal dosing and timing of methadone is still unclear. This is relevant in cardiac surgical patients; a recent study demonstrated that methadone plasma concentrations decrease 48% during cardiopulmonary bypass (CPB). We hypothesize that patients receiving two doses of methadone (one before and one after CPB) will have improved pain scores and decreased opioid consumption as compared to patients receiving only a single dose before CPB.

Methods: Patients greater than 18 years of age undergoing elective cardiac surgery requiring sternotomy and CPB who received methadone intraoperatively were identified retrospectively from our Enhanced Recovery After Cardiac Surgery database and electronic health record between May 2023 and July 2025. Patients were divided into two separate cohorts based on methadone administered intraoperatively: those receiving a single dose of methadone (10 mg before CPB) and those receiving two doses of methadone (10 mg before CPB and 10 mg after CPB). Primary outcomes included average pain scores and morphine milligram equivalents (MME) consumed on post operative days (POD) 0, 1, 2 and 3. Secondary outcomes included duration of mechanical ventilation, intensive care unit (ICU) length of stay, total length of stay, and the need for antiemetics. Patient

demographics, clinical characteristics, intraoperative medication administration and outcomes were compared between the two groups by Chi-square and student -t tests.

Results: A total of 276 patients were identified in the study period. There was no significant difference with respect to clinical and procedural characteristics between the two groups (Table 1). There was a significant difference in intraoperative use of nerve blocks, ketamine and dexmedetomidine between groups (Table 2). Patients receiving two doses of methadone received less MMEs across POD 0, 1, 2 and 3; however, this was not statistically significant (Table 3). There was no significant difference between pain scores (POD 0, 1, 2 and 3), ICU and hospital length of stay, and antiemetic use (Table 3). Patients receiving two methadone doses had a statistically significant decrease in duration of mechanical ventilation (p =0.034)

Conclusion: Two doses of 10 mg of methadone did not improve pain outcomes compared to a single 10 mg dose. Planned additional statistical analysis adjusted to nerve blocks and other factors might reveal a difference as there is a trend towards improved opioid consumption in the two doses group. The results also demonstrate that two doses of methadone significantly decreased the duration of mechanical ventilation, with no increase in ICU length of stay or antiemetic use, suggesting benefit in postoperative pain control.

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Table 1. Demographic and Clinical Characteristics by Methadone Dose Count

	All *n=276*	*One Dose* *n=189*	*Two Doses* *n=87*	*p-value*
Age, Mean (SD)	64.8 (11.1)	64.2 (11.8)	66.0 (9.3)	0.47
Sex	n=276	n=189	n=87	0.32
Female	79 (29%)	58 (31%)	21 (24%)	
Male	197 (71%)	131 (69%)	66 (76%)	
Body mass index, Mean (SD)	31.5 (10.5)	31.4 (12.1)	31.8 (5.7)	0.09
ASA status				0.75
ASA 3	20 (7%)	15 (8%)	5 (6%)	
ASA 4	251 (91%)	171 (90%)	80 (92%)	
Diabetes mellitus	29 (10%)	20 (10%)	9 (10%)	>0.99
Tobacco use	11 (7%)	10 (10%)	1 (2%)	0.18
Alcohol abuse	9 (6%)	5 (5%)	4 (8%)	0.57
Opioid use disorder	2 (1%)	2 (2%)	0 (0%)	
Serum creatinine, Mean (SD)	1.1 (0.6)	1.1 (0.7)	1.0 (0.3)	0.10
Procedure details				0.20
Coronary artery bypass graft	93 (34%)	55 (29%)	38 (44%)	
Pulmonary thromboendarterectomy	7 (3%)	6 (3%)	1 (1%)	
Repair / replacement aortic valve	81 (29%)	62 (33%)	19 (22%)	
Repair / replacement mitral valve	16 (6%)	10 (5%)	6 (7%)	
Repair / replacement tricuspid valve	6 (2%)	6 (3%)	0 (0%)	
Replacement aortic root	27 (10%)	18 (10%)	9 (10%)	
Resection / repair ascending aortic aneurysm	35 (13%)	24 (13%)	11 (13%)	
Total procedure time, Mean (SD)	270.3 (98.2)	272.9 (101.9)	264.7 (89.8)	0.67

Table 2. Preoperative and intraoperative analgesic medications

	All n=276	One Dose n=189	Two Doses n=87	p-value
Midazolam	238 (86%)	161 (85%)	77 (89%)	0.58
Fentanyl	213 (77%)	151 (80%)	62 (71%)	0.12
Ketamine	233 (84%)	152 (80%)	81 (93%)	0.006
Dexmedetomedine	116 (42%)	71 (38%)	45 (52%)	0.037
Lidocaine	252 (91%)	177 (94%)	75 (86%)	0.06
Hydromorphone	12 (4%)	12 (6%)	0 (0%)	0.021
Acetaminophen	160 (58%)	106 (56%)	54 (62%)	0.37
Nerve block	111 (40%)	95 (50%)	16 (18%)	<0.001

Table 3. Primary and Secondary Outcomes by Methadone Dose Count

	All n=276	One Dose n=189	Two Doses n=87	p-value
Pain scores POD 0, Mean(SD)	6.4 (3.7)	6.4 (3.7)	6.4 (3.8)	0.83
Pain Scores POD1, Mean(SD)	7.0 (2.9)	6.8 (3.0)	7.3 (2.6)	0.18
Pain Scores POD2, Mean (SD)	5.9 (3.0)	5.7 (3.1)	6.2 (2.6)	0.36
Pain scores POD 3, Mean (SD)	5.1 (3.2)	4.9 (3.4)	5.5 (2.8)	0.27
MME POD0, Mean(SD)	29.5 (66.8)	31.4 (73.1)	25.4 (50.8)	0.89
MME POD1, Mean (SD)	30.1 (80.2)	32.1 (93.1)	25.8 (40.2)	0.44
MME POD2, Mean (SD)	21.9 (108.8)	24.8 (130.2)	15.6 (26.7)	0.59
MME POD 3, Mean (SD)	15.3 (56.6)	16.1 (65.3)	13.7 (30.3)	0.61
Ventilation hours, Mean (SD)	1.4 (4.3)	1.7 (4.7)	0.9 (3.2)	0.034
ICU stay, Mean (SD)	49.8 (100.2)	54.3 (113.1)	40.1 (63.0)	0.67
Ondansetron antiemetic use	95 (34%)	64 (34%)	31 (36%)	0.79

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SP 2 - Kiosk 2 - Poster 5
TEG 6 HN R time Discrepancies in Heparinized Patients
Presenting Author:*Patrick Ward, DO*

Introduction: Thromboestograms have been a mainstay in coagulation monitoring in cardiac surgery patients since they became widely available. The TEG 5000 (Haemonetics) offered the use of heparinase that could measure the patient's coagulation status regardless of the effect of heparin. This practice allowed for anesthesiologist to mobilize resources sooner in patients that would need more blood products or hold off on unnecessary transfusions. The TEG 6 cartridge based system was later implemented that allowed for less variability between samples and allowed for multiple tests to be run at once. However, it did not allow for heparinase to be added to the cartridge. In April of 2024 the FDA approved the Global Hemostasis-HN assay cartridge (Haemonetics) for viscoelastic testing on fully heparinized patients for the TEG 6 cartridge-based system. The TEG 6 HN cartridge allows for reversal of five units of heparin per one milliliter of blood. When implementing our protocol for utilization of the TEG 6 Global Hemostatis-HN cartridge, we saw elevated R-times while fully heparinized on cardio-pulmonary bypass without any increase MACFF and MACRTH. In reviewing the literature and discussion with Haemonetics, we could not determine a cause for the prolonged R-time. We wanted to look at the trends of different cases at our institution that had a TEG 6 HN cartridge run while fully heparinized and

determine factors that could be effecting the results

Methods: A chart review was done of all patients that underwent cardio-pulmonary bypass at the University of Missouri between August 2024 and March 2025. We looked at weight of patient, total bypass time, bladder temperature when samples drawn, initial heparin bolus, amount and timing of heparin redosing, and R-time while fully heparinized and after reversal with protamine. We then determine the difference between the R-times. We excluded any case that did not have a post protamine TEG 6 HN run, missing data regarding heparin redoses, or data regarding transfusion of blood products.

Results: We had 79 cases performed in that time frame, 22 cases were included into the study. These cases were majority coronary artery bypass grafting cases with rare aortic valve replacements. The average difference in R times between heparinized and post protamine was 6.38 seconds. Bypass times showed no difference in R time difference. Heparin redosing was limited however was not done within 10 mins of TEG sample being drawn. Initial heparin dose also did not show to be a factor. However the MACRTH and the MACFF stayed consistent between the two tests.

Conclusion: While no one factor that we found influenced the prolonged R-time, it is a significant finding. The average difference being 6.22 seconds, with the most being as high as 11.2 seconds. We saw a difference in R times in every patient. All the cases returned to a normal value post protamine without intervention. This data shows that while the TEG 6 HN is a great tool for determining if the patient is in need of fibrinogen or platelets prior to reversal of heparin, its utilization for clotting factors remains to be determined. A larger multi centered study would help elucidate variables between institutions and patient populations.

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Patient	Date	Weight	Baseline R	35C Venous R	Post Protamine R & Time	Difference of R	Start of Bypass
Patiet 1	8/16/2024	61.6	5.5	13.8	9.4	4.4	8:44
Patient 3	8/19/2024	114.7	3.3	17	5.8	11.2	10:00
Patient 4	8/21/2024	115.65	5.7	17	5.6	11.4	10:10
Patient 5	8/23/2024	84.85	4.7	10.5	6.1	4.4	9:33
Patient 6	8/28/2024	88.2		10.7	6.5	4.2	10:26
Patient 8	9/6/2024	86.8	4.6	11.3	5.7	5.6	9:08
Patient 9	9/11/2024	92.1	3.2	9.2	5.7	3.5	9:23
Patient 10	9/12/2024	69.8	4.1	9.7	8.5	1.2	12:52
Patient 59	2/5/2025	76.7		11.9	6.7	5.2	9:26
Patient 60	2/10/2025	87		10.2	4.6	5.6	10:27
Patient 66	2/26/2025	97.1		9.9	5.7	4.2	10:12
Patient 67	3/6/2025	61.1		12	6.7	5.3	14:25
Patient 68	3/7/2025	87.9		12.8	6.8	6	9:17
Patient 69	3/10/2025	88.7		9.2	5.5	3.7	9:29
Patient 70	3/12/2025	88.4		12.4	5.5	6.9	21:56
Patient 71	3/12/2025	162.8		12.9	6.5	6.4	19:41
Patient 72	3/14/2025	81.6		11.3	7.7	3.6	9:05
Patient 73	3/14/2025	76.3	7.9	15.8	5.5	10.3	17:26
Patient 74	3/17/2025	78.3		17	8.1	8.9	8:51
Patient 75	3/24/2025	120.1	4.7	15	5.7	9.3	10:09
Patient 76	3/26/2025	67.5	5.7	15	5.5	9.5	10:36
Patient 77	3/26/2025	71.8	5.3	10	6.3	3.7	
Patient 78	3/26/2025	112.2	n/a	11.6	5.7	5.9	9:59
Patient 79	3/28/2025	91.6	5.6	14.6	5.5	9.1	8:50
					Average R Time Difference	6.229166667	

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SP 2 - Kiosk 3 - Poster 1

Opioid-free anaesthesia for lung cancer surgery: a propensity scored analysis

Presenting Author: *Tony Vassalos, MD MBCHB*

Background: Opioid-sparing patient surgical pathways are commonplace in enhanced recovery programmes, demonstrating the perceived benefits of avoiding opioid-related perioperative complications. Evidence of opioid related hyperalgesia, immunomodulation and increased cancer reccurrence rates further promotes this concept. In this single-centre, prospective, propensity scored study in a tertiary care hospital in Scotland, UK we investigated the impact of opioid-free anaesthesia on post-operative clinical outcomes in patients undergoing elective lung cancer resection.

Methods: A prospective cohort of elective, adult patients undergoing lung cancer resection (December 2021 – November 2023) using opioid-free anaesthesia (intravenous lidocaine (1.5mg/kg) and propofol 1% (1-2mg/kg) induction; adjuvant analgesics of intravenous magnesium sulphate (50mg/kg), clonidine (1mcg/kg) and dexamethasone (6.6mg)) were compared to a retrospective cohort (January 2020 - November 2021) of opioid-based anaesthesia (intravenous propofol 1% (1-2mg/kg) and target-controlled remifentanil induction with morphine as required). Anaesthesia was maintained with sevoflurane (SevofluraneTM, AbbVie Ltd, Maidenhead, UK) in every patient with depth of anaesthesia monitored using the bispectral index (BIS; range 40-60; Medtronic, Minneapolis, MN, USA). Every patient received intravenous paracetamol and ketorolac unless contraindicated. No oral premedication was administered in either group before surgery and all patients were anaesthetised by the same anaesthetist. All patients received regional analgesia and the same post-operative pain management in recovery and beyond, including opioids as required. All analyses were performed using Minitab (version 22; Minitab LLC, State College, PA, USA) at a 5% significance level. To adjust for between group differences, propensity scores were computed using logistic regression with age, gender, surgical access, thoracoSCORE, lung transfer factor and smoking status as covariates. The scores were included as covariates in the regression models to compare clinical outcomes (cardio-respiratory complications, delirium, pain scores at 24 and 48 hours, hospital stay and 30-day mortality) between the groups.

Results: Opioid-free (n=53; 66.98±10.66yrs; 1M:1.2F; ThoracoSCORE 1.87±2.19; 74% lobectomy; 26% sublobar) compared to opioid-based patients (n=76; 66.79±11.99yrs; 1M:1.5F; ThoracoSCORE 1.37±1.45; 67% lobectomy; 33% sublobar) underwent surgical resection utilising open (13% vs 22%), robotic (8% vs 14%) and video assisted (79% vs 49%) access respectively. Complete clinical outcome data were not available for seven patients in the opioid-based (n=69) and four patients in the opioid-free (n=49) cohort. No significant differences were found in propensity scored adjusted clinical outcomes between opioid-free and opioid-based cohorts.

Conclusion: Opioid-free thoracic anaesthesia is equivalent to current, multi-modal, opioid-based anaesthesia with similar post-operative acute pain scores, cardio-respiratory complications and hospital stay. Reliable peri-operative regional analgesia is integral to good post-operative outcomes after lung cancer resection.

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Clinical Outcomes	Opioid-based Group (n=69)	Opioid-free Group (n=49)	P Value
Pain scores (0-4)			
24 hours	2 (1-3)	2 (2-3)	0.919
48 hours	2 (1.5-2)	2 (1.25-3)	0.612
Patients with no complications	46 (66)	36 (73)	0.126
Hospital stay (days)	5 (4-9)	6 (4-8)	0.465
Respiratory complications (air leak, lower respiratory tract infection, atelectasis)	14 (20)	11 (22)	0.520
Cardiovascular complications (atrial fibrillation, MACE)	9 (13)	4 (8)	0.384
Delirium	2 (2)	0	
30 day mortality	1 (1)	0	

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SP 2 - Kiosk 3 - Poster 2

Evaluating AI Chatbot Responses to Patient Questions Regarding Anesthesia for Cardiopulmonary Bypass: A Pilot Study

Presenting Author: *Dhanush Bathala, DO*

Background: With the increasing use of AI Chatbots, patients will have an unparalleled level of access to health information to their concerns. These questions will extend to their care and expectations for surgery and perioperative care. Currently, few studies have examined AI Chatbot responses to questions regarding anesthesia- with fewer examining patient questions to anesthesia during cardiac surgery. The aim of this study was to pilot an understanding of the current state of information provided to patients by AI Chatbots to questions regarding anesthesia for cardiopulmonary bypass (CPB) surgery using tools already established for qualitative assessment.

Methods: Using the “People Also Asked” feature on google, four questions were extracted based on keyword searches regarding anesthesia and CPB. These questions were inputted into 3 AI Chatbots: ChatGPT, Gemini, and Perplexity. Chatbots were chosen based on whether they had a free version publicly available. Responses from each chatbot were collected, and blinded in an excel sheet for rating.
Chatbot responses were rated with 2 standardized health education qualitative assessment tools: Flesch Kincaid Score (for readability), and PEMAT-P score (for understability and actionability). Scoring for the PEMAT-P was completed for each response by two independent reviewers, and an inter-reviewer score was also calculated to assess for reviewer similarity. Mean scores were calculated for each chatbot, and one-way ANOVA testing was completed across the three chatbots to assess for any differences.

Results: Across the three AI chatbots assessed, ChatGPT demonstrated the highest average readability by Flesch Kincaid scoring at 40.6. Gemini followed next at 37.3, and finally Perplexity at 31.4. These findings indicated overall low readability by all AI Chatbots per Flesch Kincaid scoring- with a goal readability score for medical education material at around 60-80. When evaluated using the PEMAT-P scoring rubric for understandability, Perplexity had the highest average score at 82.1, followed by Gemini at 79.0, and finally ChatGPT at 77.6. The highest average PEMAT-P actionability score was also seen in Perplexity at 60.6, followed by Gemini at 49.4, and ChatGPT at 45.0. One-way ANOVA testing showed no statistical significant differences in readability, understandability, or actionability across all three chatbots.

Conclusion: In this initial evaluation of AI generated responses to questions regarding

anesthesia during CPB, all three chatbots demonstrated moderate levels of understandability with low levels of actionability and readability. While these findings are encouraging, there is room to improve chatbot responses. This initial study highlights the need for further evaluation of more patient questions, adding layers of evaluation such as accuracy and verification of source material provided from AI Chatbots. These enhancements can help address limitations present in our pilot study, and provide a stronger understanding of the current state of AI responses to cardiac anesthesiology topics. Furthermore, this study highlights the need to investigate or develop patient education tools to be tailored to assess AI Chatbots across not only for cardiac anesthesiology but all medical specialties.

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SP 2 - Kiosk 3 - Poster 3

A Tale of Two Protamine Reactions

Presenting Author: *Brady Dangelser, MD*

Introduction: Protamine is the only available reversal agent of unfractionated heparin and is essential in the management of cardiopulmonary bypass. Through electrostatic interaction protamine neutralizes heparin; however, not without potential side-effects or adverse reactions. We discuss two separate cases of major protamine reactions - IgE mediated anaphylaxis and compliment mediated pulmonary vasoconstriction and their echocardiographic findings on intra-operative TEE.

Case presentation:
Case #1: A 65-year-old male with a history of prior ascending aorta and total arch replacement who underwent an open thoracoabdominal aortic aneurysm repair for his enlarging 7.1 cm descending thoracic aorta aneurysm. The patient remained hemodynamically stable throughout the operation and tolerated distal unclamping of the aorta. Approximately six minutes following administration of protamine, the patient became acutely hypotensive requiring 0.2 mcg/kg/min of norepinephrine to maintain appropriate perfusion pressure. On TEE, biventricular size and function remained at baseline. Peak inspiratory pressures on the ventilator rose ten points. Given the proximity of hemodynamic collapse to protamine administration with prior protamine exposure and increased peak airway pressures, the patient was treated for anaphylaxis with an epinephrine infusion; a tryptase level was drawn and consistent with an anaphylactic reaction.

Case #2: A 67-year-old male with hypertrophic cardiomyopathy status post septal myomectomy complicated by ventricular tachycardia status post CRT presented in acute on chronic decompensated heart failure and underwent orthotopic heart transplant (OHT). On the day of OHT, baseline pulmonary arterial (PA) pressures were consistently in the low 30s over low 20s. The patient was hemodynamically stable upon separation from cardiopulmonary bypass until protamine administration. Following protamine test dose, the PA pressures became supra-systemic with new onset severe right ventricular dysfunction noted in the surgical field and on TEE. The patient was loaded with endotracheal milrinone with gradual improvement in right ventricular function and return to baseline PA pressures. Discussion: Major protamine reactions such anaphylaxis and acute pulmonary hypertension both have an incidence of less than 1%, however, these often occur at tenuous times following separation from CPB1,2. Case one demonstrated severe vasoplegia with increased peak airway pressures consistent with an IgE mediated anaphylactic reaction – treated with low dose epinephrine and volume resuscitation. TEE demonstrated normal right ventricular function with a hyperdynamic left ventricle. Meanwhile, the second case demonstrated a precipitous climb in PA pressures with florid right ventricular failure on TEE secondary to protamine-mediated acute pulmonary hypertension. In both cases, TEE allowed for rapid narrowing of the differential diagnosis and successful management of severe protamine reactions. Conclusion: While rare, severe protamine reactions are a life-threatening event. TEE is essential to the early recognition and treatment of these severe reactions.

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SP 2 - Kiosk 4 - Poster 1

The Downstream Effect of Implementing a Preoperative Screening Process for Iron-Deficiency in Cardiac Surgery Patients

Presenting Author: *Grace Bronner*

Background: Preoperative anemia is one of the strongest predictors of perioperative transfusion and is associated with worse outcomes. preoperative anemia management, there remains a gap in implementation. Loyola University Medical Center (LUMC) was not routinely screening for preoperative iron deficiency. Thus, the prevalence of iron deficiency and IDA in cardiac surgery

patients at LUMC was unknown. This study intended to examine the screening rate and results after implementing a formal screening process for iron deficiency in patients undergoing cardiac surgery.

Methods: This retrospective, observational pilot study included adult patients undergoing elective cardiac surgery with cardiopulmonary bypass at LUMC from 3/3/2025-6/30/2025. Excluded patients were those undergoing emergency surgery, transplant surgery, or receiving mechanical circulatory support devices. The primary outcome was the rate of preoperative screening for iron deficiency. Secondary outcomes included the prevalence of anemia and IDA, number of patients preoperatively treated with iron, rate of red blood cell (RBC) transfusion, number of RBC units transfused, and length of stay (LOS). Data analyses were conducted via Stata v.19 (Stata Corp, College Station, TX).

Results: Of patients reviewed (N=85), 75% (64/85) were preoperatively screened for iron deficiency, and 52% (33/64) of screened patients were iron deficient. All patients (N=85) were assessed for preoperative anemia, with 32% having anemia (hemoglobin < 13.0 g/dL). Iron deficiency status was not known for all anemic patients. Of the patients screened for iron deficiency, 11% (7/64) had IDA. Preoperative treatment with iron was prescribed for 5% of patients.
Anemic patients displayed an increased need for and greater amount of RBC transfusion than non-anemic patients. 81% of anemic patients received perioperative RBC transfusion compared to 26% of non-anemic patients ($p< 0.001$). Additionally, 86% of known IDA patients required RBC transfusion. Patients who received at least one RBC unit displayed an estimated 5 day increase in their hospital LOS compared to those who received no RBC units (β: 5.1, 95% CI [2.36, 7.76], $p< 0.001$).
Non-anemic iron deficiency (NAID) trended towards reduced odds of RBC transfusion (odds ratio 0.54, 95% CI [0.20, 1.46], $p=0.221$). The use of acute normovolemic hemodilution (ANH) was associated with markedly reduced odds of RBC transfusion (OR 0.16, $p< 0.001$). Notably, the majority of patients who received ANH were patients with NAID.

Conclusion: The prevalence of preoperative anemia at LUMC is consistent with published literature. Patients with preoperative anemia were more likely to require RBC transfusion, and patients with IDA specifically demonstrated an increased need for transfusion. Both the number of RBC units and need for RBC transfusion displayed a statistically significant association with hospital LOS. The study findings provide data for resource allocation to establish preoperative anemia management for cardiac surgery patients at LUMC. Further investigation is needed on the use of ANH and the treatment of patients with NAID.

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SP 2 - Kiosk 4 - Poster 2

Distinct Patterns of Injury and Death Result from Oxygenator Failures During Cardiopulmonary Bypass

Presenting Author: *Jace Bradshaw, MD*

Background: Cardiopulmonary bypass (CPB) oxygenators facilitate essential intraoperative gas exchange, enabling complex surgical interventions. Although rare, CPB oxygenator failures can cause catastrophic morbidity and mortality. Published analyses of CPB-specific oxygenator failures are limited, and existing extracorporeal membrane oxygenation (ECMO) reports may not extrapolate to cardiac surgery. Our study aimed to systematically examine CPB oxygenator failure reports that resulted in patient harm within the MAUDE database between April 2015 and April 2025 using descriptive analyses and natural language processing.

Methods: We retrospectively analyzed FDA Manufacturer and User Facility Device Experience (MAUDE) reports (April 2015–April 2025) describing CPB oxygenator failures with injury or death. ECMO events, malfunctions without harm, and duplicates were excluded. Manufacturer, device problem, and patient problem distributions were compared between injury and death cohorts using ANOVA, chi-square, and Fisher's exact tests. Narrative text underwent artificial intelligence natural language processing (NLP) (TF–IDF, n-gram analysis, non-negative matrix factorization) to identify recurring clinical themes.

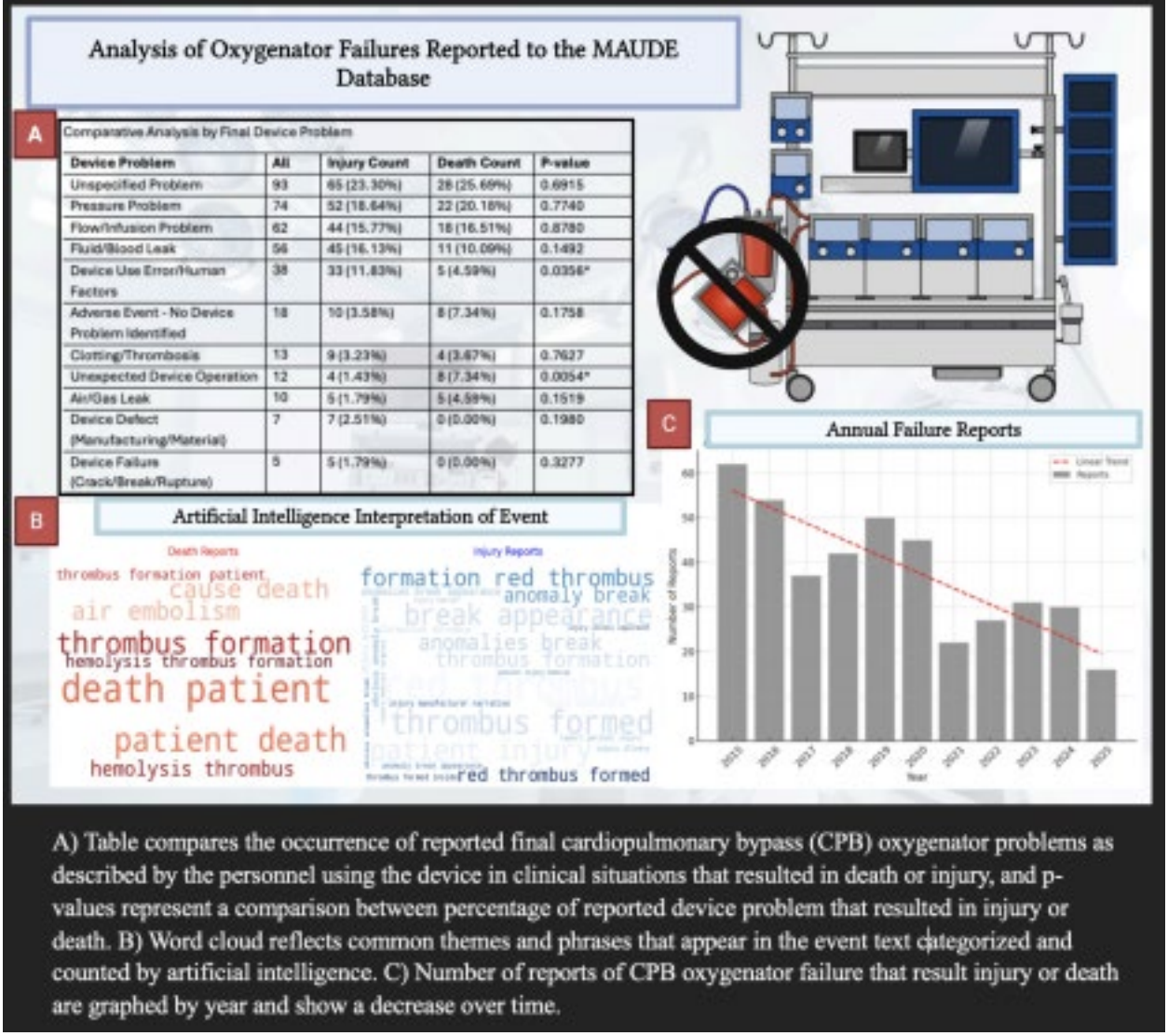
Results: Of 1,022 screened reports, 388 unique events met inclusion criteria: 279 injuries

(71.9%) and 109 deaths (28.1%). Three manufacturers accounted for 88% of events. Manufacture A was used in 169 (60.6%) of the injury reports but only 25 (11.1%) of death reports. Manufacturer B devices were used in 31 (11.1%) of injury reports but 47 (43.1%) of death reports. The incidence of failure for each manufacturer could not be calculated. Device problem categories differed by outcome: “Unexpected Device Operation” was significantly more common in deaths (7.3% vs. 1.4%, p=0.0054), whereas “Human Factors” was more common in injuries (11.8% vs. 4.6%, p=0.036) (Figure 1). Patient problem analysis showed that blood loss and hemorrhage predominated in the injury cohort, while thromboembolic events (9.2% vs. 3.6%, p=0.038) were more commonly reported in the death cohort. NLP analysis of free-text narratives revealed three dominant themes: (1) intraoperative performance and monitoring failures (e.g., abnormal blood gas results, flow rate discrepancies); (2) hemolysis and thromboembolic complications (including air embolism as a recurrent fatal mechanism); and (3) quality management (Figure 1). These themes highlighted device-specific vulnerabilities and gaps in intraoperative detection of failure modes. When assessed by year, overall, the number of reported oxygenator failures that result in death or injury is decreasing. (Figure 1)

Conclusions: Oxygenator failures during CPB leading to patient harm demonstrate distinct associations with specific device problems and patterns of patient injuries. Fatal events are more often linked to sudden operational failure and thromboembolic complications, underscoring the importance of vigilant monitoring, preparedness for oxygenator exchange, and device design improvements. NLP enhances the detection of clinically relevant patterns and may guide targeted safety interventions.

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Figure 1: Results of MAUDE Database Analysis of Reported Oxygenator Failures



Device Problem	All	Injury Count	Death Count	P-value
Unspecified Problem	93	65 (23.30%)	28 (25.69%)	0.6915
Pressure Problem	74	52 (18.64%)	22 (20.18%)	0.7740
Flow/Infusion Problem	62	44 (15.77%)	18 (16.51%)	0.8780
Fluid/Blood Leak	56	45 (16.13%)	11 (10.09%)	0.1492
Device Use Error/Human Factors	38	33 (11.83%)	5 (4.59%)	0.0356*
Adverse Event - No Device Problem Identified	18	10 (3.58%)	8 (7.34%)	0.1758
Clotting/Thrombosis	13	9 (3.23%)	4 (3.67%)	0.7627
Unexpected Device Operation	12	4 (1.43%)	8 (7.34%)	0.0054*
Air/Gas Leak	10	5 (1.79%)	5 (4.59%)	0.1519
Device Defect (Manufacturing/Material)	7	7 (2.51%)	0 (0.00%)	0.1980
Device Failure (Crack/Break/Rupture)	5	5 (1.79%)	0 (0.00%)	0.3277

A) Table compares the occurrence of reported final cardiopulmonary bypass (CPB) oxygenator problems as described by the personnel using the device in clinical situations that resulted in death or injury, and p-values represent a comparison between percentage of reported device problem that resulted in injury or death. B) Word cloud reflects common themes and phrases that appear in the event text categorized and counted by artificial intelligence. C) Number of reports of CPB oxygenator failure that result injury or death are graphed by year and show a decrease over time.

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SP 2 - Kiosk 4 - Poster 3

Determination of ED 90 of intrathecal morphine for postoperative analgesia in adult open heart surgery using sequential allocation biased coin method

Presenting Author: *Dhawan MBBS, MD, DM*

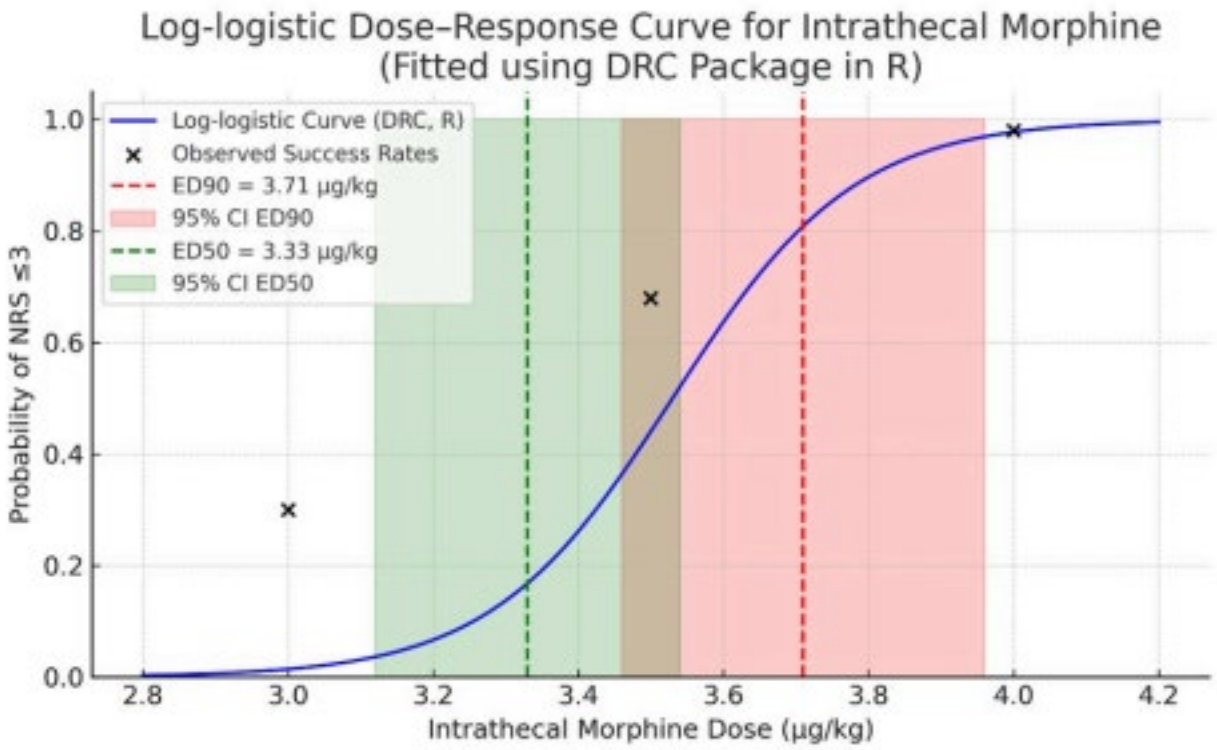
Introduction: Effective postoperative pain control remains a cornerstone of enhanced recovery after cardiac surgery. Although intrathecal morphine (ITM) is a time tested gold standard analgesic strategy, its optimal dose that balances efficacy and safety for 24 hours post operative analgesia in cardiac surgical population remains undefined. This study aimed to determine the ED_{90} of intrathecal morphine for postoperative analgesia in adult open heart surgery using a biased-coin up-down sequential allocation design. ED_{90} can then be used for comparison with various other methods of fast track cardiac surgery.

Methods: In this prospective, double-blind, single-centre trial, all eligible patients undergoing elective open-heart surgery via median sternotomy were enrolled. The initial dose was set at 4.0 µg/kg, the lowest dose of intrathecal morphine reported for adult open-heart surgery. Subsequent dose modifications were determined using a biased-coin up-and-down sequential allocation method, based on the analgesic response of the preceding patient. A computer generated list of random numbers, prepared by the pharmacology co-investigator using Microsoft Excel 2016 (Microsoft Corporation, Redmond, WA, USA), guided the allocation process. A 90:10 probability rule was applied to favour retention of the current dose versus upward or downward adjustment based on the analgesic outcome observed in the preceding patient. Dose increments were maintained at 0.5 µg/kg to align with clinically appropriate titration intervals. The dosing range was restricted between 2 µg/kg and 6 µg/kg to minimise the risk of under- or overdosing. The primary endpoint was analgesic success, defined as NRS ≤3 at rest and during deep breathing/coughing at 24 hours after administration of spinal anaesthesia, with no more than two rescue doses of 1 µg/kg fentanyl. Secondary outcomes included time to first rescue analgesia, total opioid consumption at 6,12, 24 and 48hours, RAAS at extubatation, 6, 12, 24 and 48 hours, mechanical ventilation duration, adverse events, and patient satisfaction.

Results: The ED_{90} for intrathecal morphine was 3.71 µg/kg [95% CI: 3.46–3.96], with 100% success at 4.0 µg/kg. Time to first rescue analgesia was significantly prolonged ($p< 0.001$) and total rescue opioid requirement was significantly reduced at 6, 12, 24 and 48hours for 4mic/kg. A statistically significant difference was observed for RAAS at all assessed time points. Patients in the 4.0 µg/kg group demonstrated a shorter and less variable duration of mechanical ventilation compared to the 3.0 µg/kg and 3.5 µg/kg groups. No cases of respiratory depression, pruritus, or spinal hematoma were observed. Postoperative nausea vomiting was observed in 12.5% (3/24) of patients receiving 4.0 µg/kg. Patient satisfaction at 48 hours was highest in the 4.0 µg/kg group ($p = 0.0017$).

Conclusion: This is the first study to define the ED_{90} of intrathecal morphine to be 3.7mic/kg, offering a robust, evidence-based foundation for clinical adoption. These findings support the integration of weight-based intrathecal morphine dosing into opioid-sparing, enhanced recovery cardiac anaesthesia protocols.

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SP 2 - Kiosk 5 - Poster 1

Blood Before Balloon? Anesthetic Optimization for Anemia-Driven ST-Elevation Myocardial Infarction

Presenting Author: *Bruce Kardach, DO*

Background: A Type 1 ST-elevation Myocardial Infarction (STEMI) results from acute coronary blockage, whereas a Type 2 MI stems from supply-demand mismatch. While classic MI symptoms present with sudden, left-sided crushing chest pain, symptoms in women more commonly present with epigastric pain, nausea, dyspnea, and/or fatigue, either in addition to or independent of chest pain. Although standard pre-operative optimization with heparin, DAPT, and a statin is key to minimizing morbidity and mortality, this protocol can precipitate catastrophic iatrogenic hemorrhage. This complication worsens the underlying supply-demand mismatch, creating a critical management crossroads for the anesthesiologist.

Case Description: A 76-year-old female with a complex history including CAD (inferior STEMI s/p PCI to RCA + RPDA and staged POBA to LAD), HFrEF, ESRD-associated anemia (baseline Hgb 11 g/dL), and a 2-day history of nausea and epigastric pain presented post-dialysis with new onset chest pain and an inferior STEMI for an urgent left heart catheterization (LHC). Her initial optimization with heparin and DAPT precipitated severe hemoptysis, hemorrhagic shock (nadir Hgb 5.7 g/dL), and DKA. Following MTP with 3 units pRBC, 1 unit FFP, vasopressor support, and elective intubation prior to an emergent EGD showing no active bleed, the STEMI pattern on her EKG resolved, unmasking a likely Type 2 MI.

During a subsequent, ultimately negative, second-look EGD with a pre-procedural Hgb of 9.6 g/dL, her course was complicated by two cardiac arrests (asystole, then PEA) precipitated by hypoxia and bradycardia, with ROSC after one round of CPR for each event. The LHC was consequently delayed for one week for further medical optimization. The eventual LHC revealed patent vessels with TIMI 3 flow and significant collaterals perfusing a known chronic total occlusion of the ostial RCA. No culprit lesion was identified, and DAPT was discontinued.
Discussion

This case highlights two crucial anesthetic considerations. First, resuscitation for her iatrogenic hemorrhage served as a diagnostic modality in that resolution of ST-changes post-transfusion indicates a Type 2 MI, allowing an emergent LHC to be potentially re-categorized as urgent or semi-urgent. Second, achieving a numerical hemoglobin goal does not necessarily equate to clinical stability. While guidelines on anemia in ACS discuss a potential benefit for a liberal

transfusion strategy, this patient's initial improvement was likely a multifactorial result of increased oxygen-carrying capacity and preload. The initial resuscitation, while life-saving, was insufficient to restore full stability, as evidenced by the subsequent cardiac arrests.

Conclusion: In managing a Type 2 STEMI unmasked by MTP for iatrogenic hemorrhage, correcting the hemoglobin is simply a first step. Anesthesiologists must recognize that profound physiological fragility can persist despite resolved ST-changes and normalized lab values. This mandates heightened vigilance and a low threshold to consider delaying non-emergent procedures for maximal medical optimization. Crucially, the anesthetic plan must anticipate significant hemodynamic collapse, which is not wholly unexpected in a seemingly optimized patient.

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SP 2 - Kiosk 5 - Poster 2

Case Report: Rapidly Progressive Cardiac Involvement in Granulomatosis with Polyangiitis

Presenting Author: *Alexander Zhang, MD*

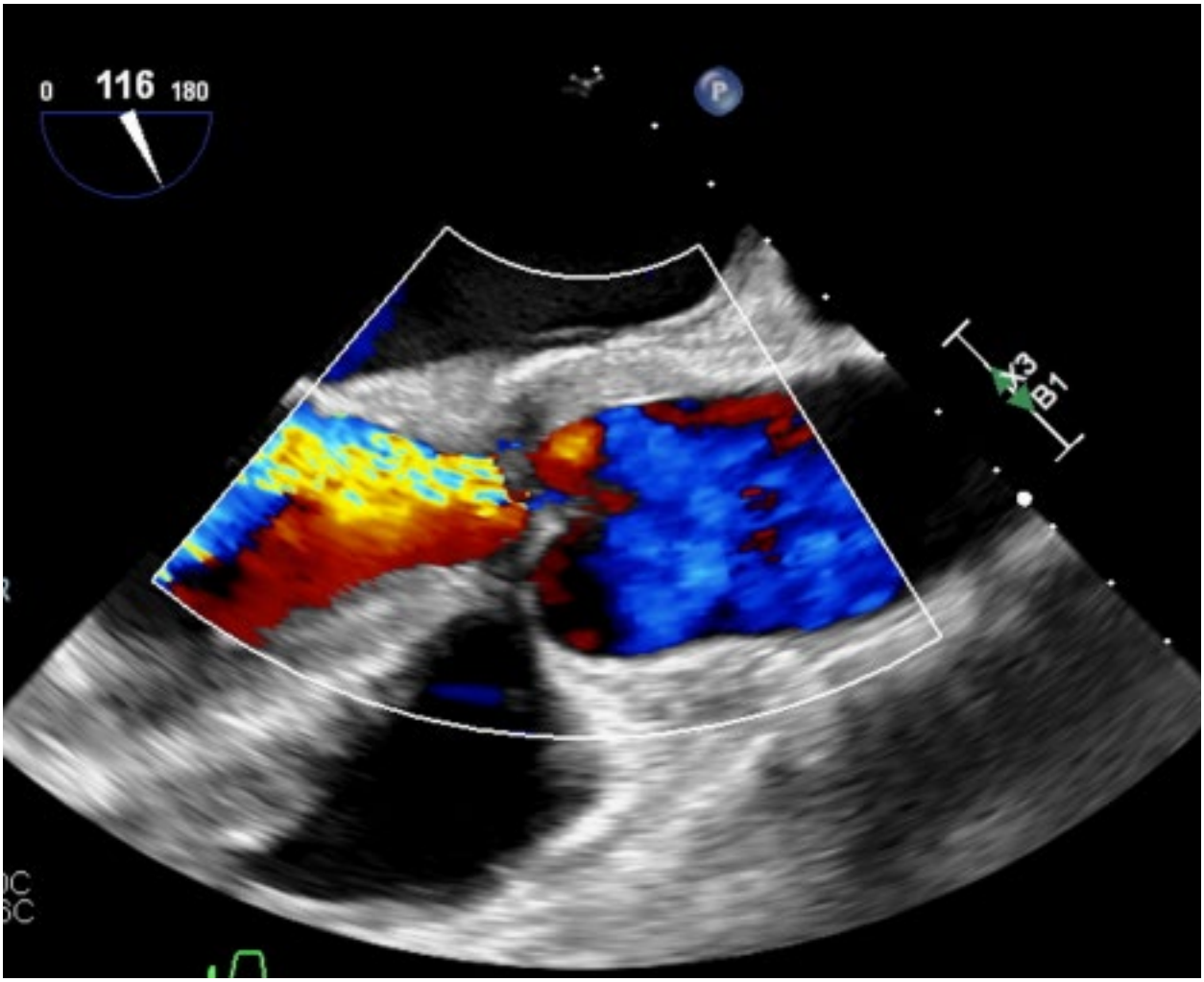
Introduction: Granulomatosis with polyangiitis (GPA) is an ANCA-associated vasculitis (AAV) primarily affecting the lungs, kidneys, and respiratory tracts. Cardiac involvement is uncommon (3–12%) and underrecognized. We present the case of a 49-year-old male with GPA who developed rapidly progressive cardiac disease. This case highlights the need for early cardiac evaluation and timely immunosuppressive escalation in patients with GPA who present with cardiac manifestations.

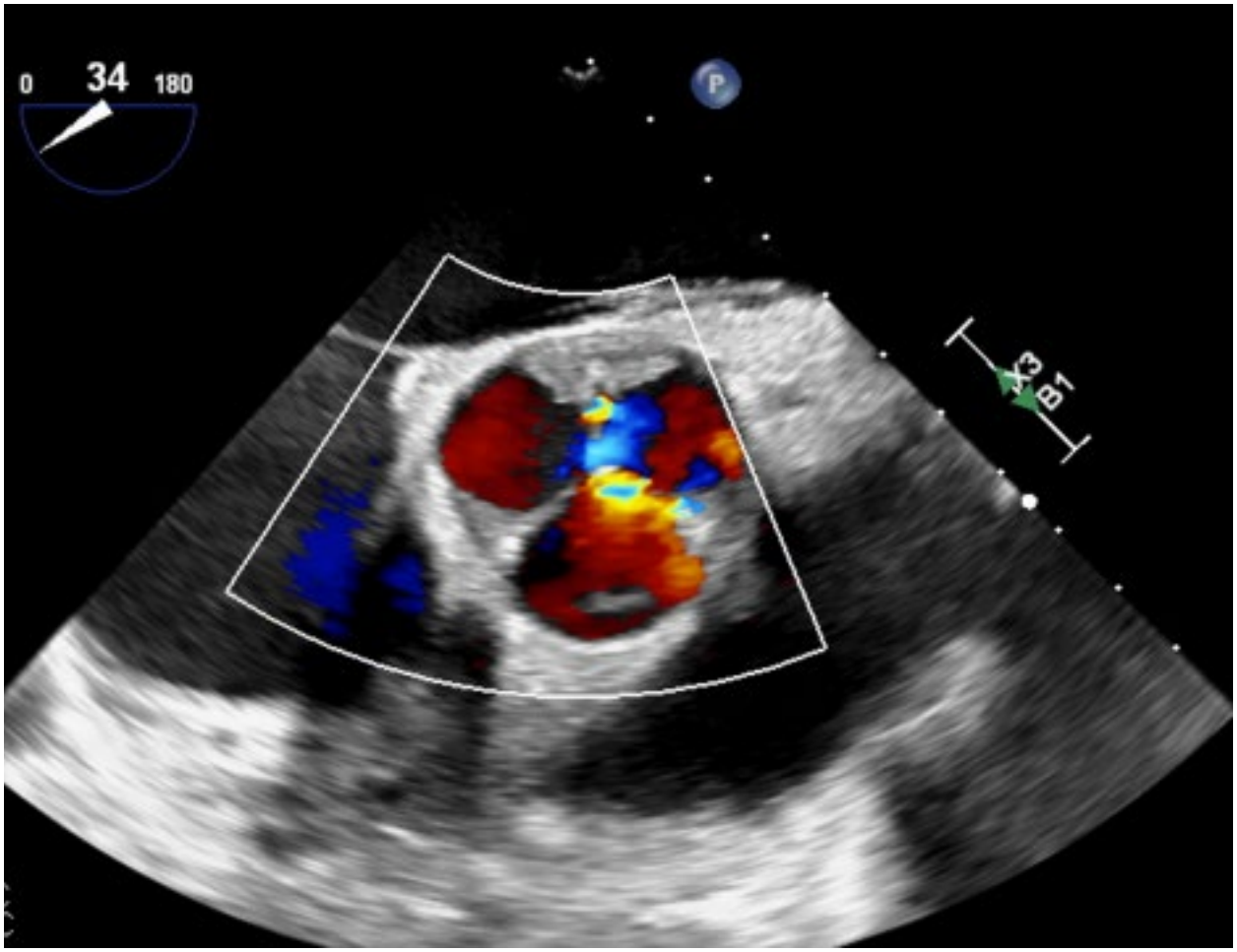
Case Report: A 49-year-old male with GPA initially presented with a large pericardial effusion and impending tamponade requiring pericardial window and colchicine. TTE revealed new mild aortic regurgitation (AR). Eight months later, he developed complete heart block requiring a Micra permanent pacemaker. At this time, his AR was severe with newly reduced EF (45%) and dilated cardiomyopathy. Cardiac MRI revealed inflammatory changes near the aortic and mitral subvalvular regions. He underwent a Bio-Bentall procedure one month later. Intraoperative TEE showed thickened aortic cusps and severe AR. Postoperatively, TEE confirmed intact valve function. Histopathology of the native aortic valve demonstrated mixed acute and chronic inflammation consistent with possible GPA pending exclusion of infection.

Discussion AAV is an autoimmune disease against neutrophilic MPO and PR3 leading to deposition of granulomas. The autoimmune phenomena result in partial and total occlusion of blood vessels, tissue necrosis, and structural deformities. The most common systemic effects are otolaryngeal, pulmonary, and renal.1 The estimated prevalence of GPA is 2.3 - 140 per 1 million persons. 2 3 The cardiac manifestations of GPA occur due to deposition of granulomas in coronary vessels, conduction pathways, myocardial tissue, and valves leading to pericarditis, cardiomyopathy, myocarditis, coronary artery disease, valvular disease, and severe conduction disorders.2 4 Approximately 46-61% of GPA patients have cardiac involvement on advanced cardiac imaging but are often asymptomatic or have subclinical findings. 5 6 7 8 According to the European Alliance of Associations for Rheumatology (EULAR) 2022 guidelines regarding AAV, tissue biopsy is recommended to establish the diagnosis of vasculitis, and high-quality antigen specific assay PR3 and MPO-ANCA are recommended if AAV is suspected.
Immunomodulators are the gold standard of treatment for ANCA vasculitis.9 Current recommendations do not call for routine cardiac screening for asymptomatic AAV.

Conclusion: Although rare, GPA can present with significant cardiac manifestations. A multidisciplinary approach involving cardiology, cardiac surgery, rheumatology, and anesthesia is essential for optimal management, including immunosuppression and procedural planning. Earlier collaboration in this case may have prompted suspicion of GPA-related pericarditis and earlier diagnostic testing. Given the high prevalence of subclinical disease and heterogeneous presentation, new or evolving cardiac symptoms warrant may support selective screening or protocolized evaluation in GPA patients who present with cardiac involvement.

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SP 2 - Kiosk 5 - Poster 3

Real-Time Angiographic Documentation of Intraoperative Coronary Vasospasm During Aortic Surgery: A Three-Case Series and Literature Review

Presenting Author: *Yao Ding, MD*

Introduction: Coronary artery spasm (CAS) during aortic surgery is rare but can precipitate catastrophic events like myocardial ischemia and cardiac arrest. Intraoperative hemodynamic instability is usually attributed to surgical factors or bleeding, so CAS is easily overlooked without real-time imaging. We describe three cases of intraoperative CAS during complex aortic procedures where emergent angiography definitively diagnosed the spasm and guided life-saving therapy.

Case Report Case 1: A 69-year-old man with a penetrating aortic arch ulcer and moderate Lcx stenosis underwent branched thoracic endovascular repair. Stent deployment caused mean arterial pressure to fall despite escalating catecholamines, with new ST-depression and bradycardia progressing to pulseless electrical activity. After resuscitation, complete heart block persisted. Angiography revealed right coronary ostial spasm without fixed stenosis. Intravenous nicardipine and nitroglycerin resolved the spasm and restored hemodynamics. The patient was discharged without recurrent ischemia.

Case 2: A 66-year-old man with a bicuspid aortic valve and ascending aortic aneurysm underwent ascending/arch replacement. Post-CPB, cardiac manipulation triggered hypotension and paroxysmal supraventricular tachycardia. Recurrent inferior ST elevation coincided with right ventricular dilatation and severe tricuspid regurgitation on TEE. Angiography revealed critical right coronary ostial spasm without obstruction. Intracoronary nitroglycerin, corticosteroids, and nicardipine resolved the spasm and normalized ECG/TEE findings. He was extubated safely and discharged in sinus rhythm.

Case 3: A 59-year-old man with coronary disease underwent off-pump aortic debranching and stenting. During chest closure, abrupt hypotension with marked ST elevation occurred, unresponsive to vasopressors. Angiography showed no significant stenosis but diffuse slow flow consistent with CAS, and troponin I increased. On background nitroglycerin, intravenous nicorandil was administered, normalizing blood pressure and ECG. The timing argued against Kounis syndrome. He recovered well and was discharged in good condition.

Discussion All three patients had cardiovascular comorbidities but no history of vasospastic angina. Each episode manifested as sudden, refractory hypotension with arrhythmias. Real-time angiography was critical, documenting transient spasm or slow flow in segments distinct from pre-existing lesions. Stability was only restored with specific vasodilators (nitrates/calcium channel blockers). These findings suggest that complex aortic surgery—characterized by sympathetic surges, rapid hemodynamic shifts, and direct manipulation near the aortic root—can unmask latent coronary hyperreactivity. Timely recognition and intervention for CAS are crucial for survival. Conclu

Intraoperative CAS represents a rare, high-risk, and elusive complication of aortic surgery. Our observations emphasize that early consideration, recognition, and intervention are vital for patient survival when standard resuscitation fails. These findings support the hypothesis that perioperative strategies to modulate coronary vasoreactivity warrant prospective investigation in high-risk patients.

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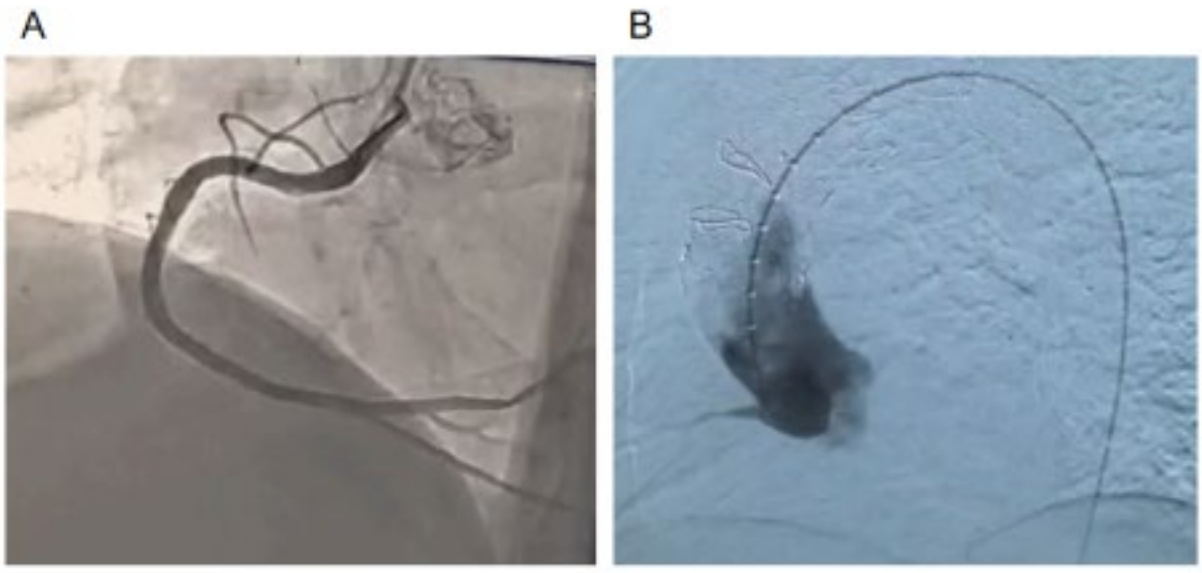


Figure 1. Contrast between preoperative anatomy and intraoperative spasm (Case 1)
(A) Preoperative coronary angiography.
(B) Intraoperative emergency angiography during hemodynamic collapse, demonstrating severe spasm at the ostium of the RCA.

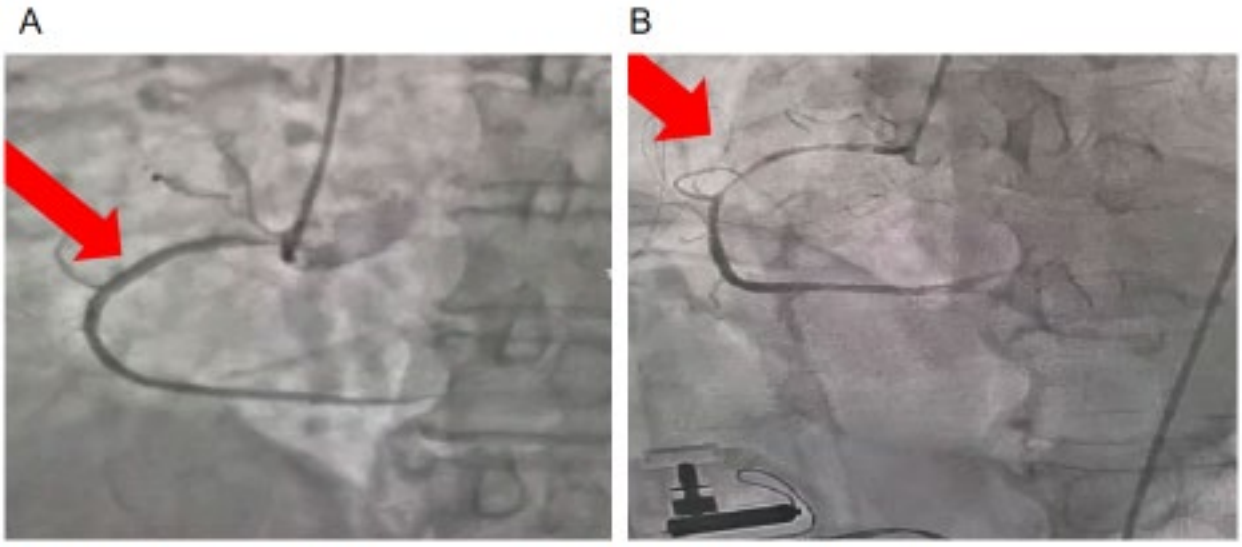


Figure 2. Contrast between preoperative anatomy and intraoperative spasm (Case 2)
(A) Preoperative coronary angiography.
(B) Intraoperative emergency angiography.



Figure 3. Hemodynamic instability and angiographic findings (Case 3)
(A) Intraoperative monitor snapshot showing marked ST-segment elevation coinciding with abrupt hypotension during chest closure.
(B) Subsequent emergency coronary angiography showing diffuse slow flow

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SP 2 - Kiosk 5 - Poster 4
Takes Two to Tango: Coordinating Anesthesia for Combined Lung–Liver Transplantation
Presenting Author: *Dimpie Shah, MD*

Introduction: Combined lung–liver transplantation is rare and physiologically complex, requiring

tight coordination between cardiothoracic anesthesia, thoracic surgery, and transplant teams. Patients with end-stage interstitial lung disease (ILD) and cirrhosis have limited cardiopulmonary reserve, altered hemodynamics, and major transfusion needs. We present a case of idiopathic pulmonary fibrosis (IPF) related to World Trade Center exposure undergoing successful bilateral lung and liver transplantation.

Case Description: A 69-year-old man with ILD/IPF on home oxygen, cryptogenic cirrhosis with portal hypertension, and prior PCI underwent combined lung–liver transplantation. Anticipating significant hemodynamic shifts, two Cordis introducers were placed in the right internal jugular vein: one for pulmonary artery (PA) catheter monitoring, and the second kept open for possible venovenous bypass (VVB) cannulation during the liver phase. A right antecubital rapid infusion catheter (RIC) provided high-flow Belmont access. Lung transplantation was performed first, followed by orthotopic liver transplantation without the need for extracorporeal support. The patient tolerated the procedure well, was extubated postoperatively, and was eventually discharged to rehabilitation with excellent graft function.

Discussion: Dual-organ transplantation requires advance planning for rapid changes in preload, pulmonary vascular resistance, and oxygenation. VVB is frequently employed during liver transplantation to decompress the splanchnic venous system and reduce hemodynamic instability during inferior vena cava manipulation. Standard VVB needs two large bore cannulae, one femoral for drainage and the second for internal jugular return. One RIJ Cordis was therefore placed specifically to preserve a ready pathway for quick conversion to VVB if hemodynamics deteriorated during the anhepatic phase.

A second RIJ Cordis was required because the cardiothoracic surgeon requested a PA catheter to continuously track RV performance, pulmonary pressures, and cardiac output. Together, the dual Cordises and peripheral RIC created a system that enabled real-time cardiopulmonary monitoring, immediate readiness for VVB, and unimpeded Belmont transfusion capability, avoiding line competition and the need for emergent access under instability.

Mechanical support strategies for lung–liver transplantation also vary. Cardiopulmonary bypass (CPB) provides full support but increases inflammation, bleeding, and coagulopathy—undesirable in cirrhosis. ECMO offers gas-exchange support and reduced inflammatory activation but still requires anticoagulation. Proceeding without extracorporeal support, as in this case, avoids these complications but demands meticulous monitoring, stable access for rapid transfusion, and the ability to escalate immediately to VVB if needed.

This case underscores the importance of deliberate vascular access planning, integration of cardiopulmonary monitoring, and coordinated multidisciplinary management to safely navigate the physiologic complexities of combined lung–liver transplantation.

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SP 2 - Kiosk 5 - Poster 5

TEE-Guided Diagnosis and Management of LV Aneurysm with Contained Rupture in the Operating Room

Presenting Author: *Camila Magalhaes Funatsu, MD*

Introduction: Left ventricular (LV) aneurysms and pseudoaneurysms are rare complications of MI associated with significant morbidity. True aneurysms may lead to HF, arrhythmias, and thrombus formation but have low rupture risk. Pseudoaneurysms carry a high risk of rupture and sudden death. We present the perioperative management of a patient with delayed anteroapical MI complicated by severe LV dysfunction, hemopericardium, and a large apical aneurysm with contained rupture.

Case Report: A 46 y/o M w/ HTN collapsed at home after several weeks of chest and back pain. He arrived as a STEMI alert with mild troponin elevation and TTE showing EF 20–25%, apical akinesis, an aneurysmal apex, and a small pericardial effusion. Cardiac cath revealed a very late anteroapical infarction, CTO of the distal LAD, a large apical aneurysm, and elevated right-sided pressures. IABP was placed and he was transferred to the CVICU. There was concern for impending free-wall rupture and prompting emergent surgical repair.

In the OR, GA was induced and invasive monitoring included a radial arterial line, RIJ introducer w/ PA catheter, and TEE. Pre-bypass TEE showed a large apical aneurysm with contained rupture and pseudoaneurysm features: narrow neck, bidirectional flow, myocardial discontinuity, moderate

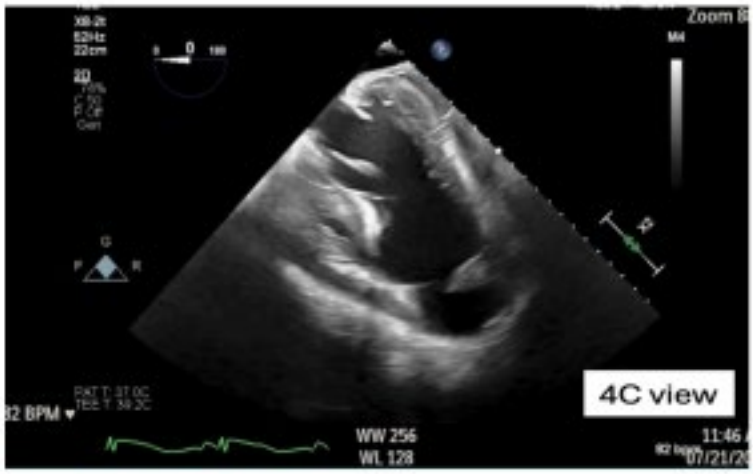
hemopericardium, and EF ~25%. Peripheral cannulation and sternotomy revealed extensive clot within the pseudoaneurysm. The aneurysmal sac was resected and the LV reconstructed. Post-repair TEE confirmed complete exclusion of the pseudoaneurysm, no residual shunt, and improved EF (45–50%). Postoperatively, he remained stable on IABP and low-dose vasopressors, which were weaned over 48 hours. Chest tubes and IABP were removed on POD 3, and he was discharged home on POD 15.

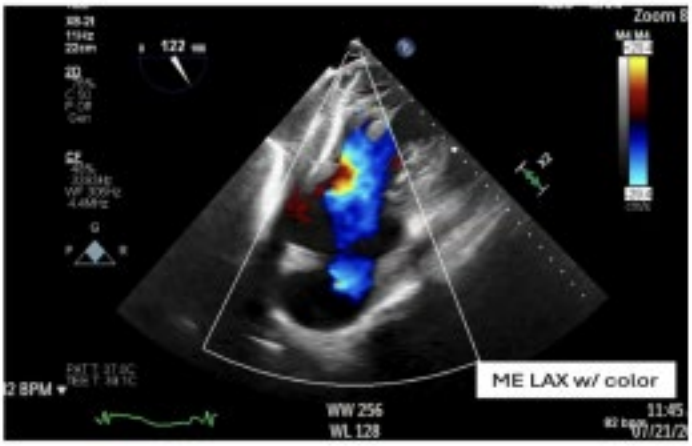
Discussion True LV aneurysms form when all three myocardial layers thin and bulge after MI, usually within 2 weeks. They are broad-based, dyskinetic, contained by scar, usually anterior/apical, and rarely rupture. Pseudoaneurysms arise from contained free-wall rupture, where blood exits through a myocardial defect but is held by pericardium or scar. Their walls lack myocardium, more commonly involve the posterior or lateral wall, and carry a high risk of rupture requiring urgent repair. TEE features helpful in differentiating the two include: True aneurysm: – Wide – Dyskinetic but continuous myocardium – Thinned but intact wall Pseudoaneurysm: – Narrow neck (ratio < 0.5) – Saccular shape – Myocardial discontinuity – Flow into sac, often bidirectional
In this case, TEE confirmed an aneurysm with contained rupture and pseudoaneurysm characteristics, guiding anesthetic decision-making and the urgency of surgical repair.

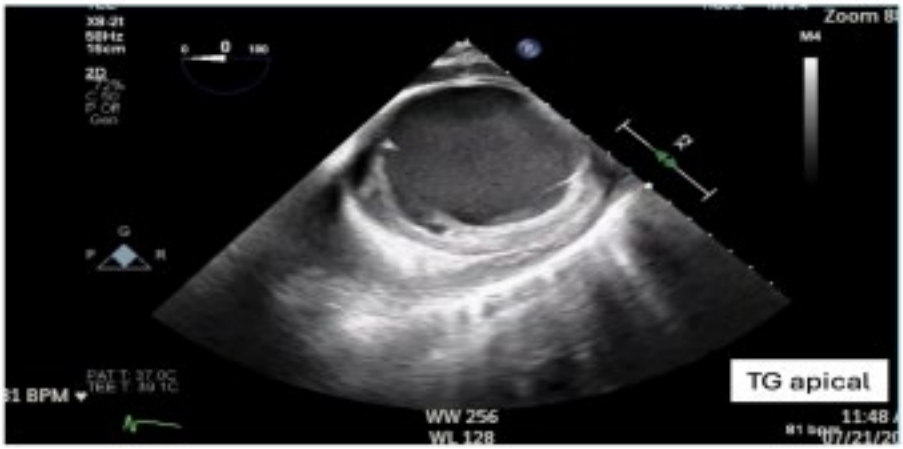
Conclusion: Distinguishing true aneurysm from pseudoaneurysm, particularly via TEE, directly affects perioperative planning, hemodynamic goals, and timing of intervention. Maintaining stable hemodynamics before bypass reduces wall stress and rupture risk. This case highlights the essential role of intraoperative TEE and anesthetic strategy in the management of catastrophic post-MI complications.

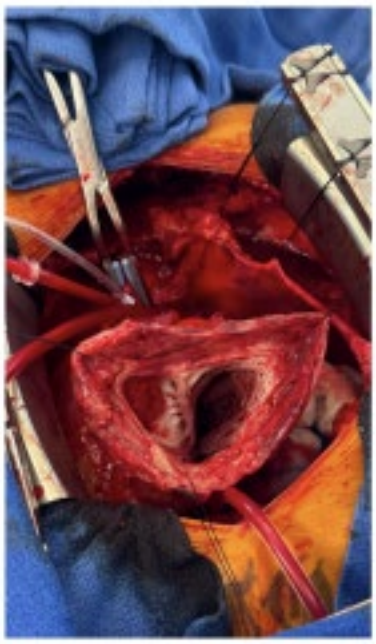
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Pre repair TEE








Post Repair TEE





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SP 2 - Kiosk 5 - Poster 6

Anesthetic Management of a Patient with a Left Ventricular Assist Device Undergoing a Minimally-Invasive Ivor Lewis Esophagectomy

Presenting Author: *Alexander White, MD, FRCPC, FANZCA*

Introduction: Patients with durable left ventricular assist devices (LVADs) increasingly present for non-cardiac surgery (1), though thoracic surgery in this population remains rare, with limited literature to guide management. We present a case of an LVAD patient undergoing robot-assisted minimally invasive Ivor Lewis esophagectomy (MIE).

Case Report: A 74-year-old male with a HeartMate III (Abbott Laboratories, Chicago IL) LVAD as destination therapy for ischemic cardiomyopathy presented with gastrointestinal (GI) bleeding and was diagnosed with distal esophageal adenocarcinoma. He underwent MIE with curative intent.

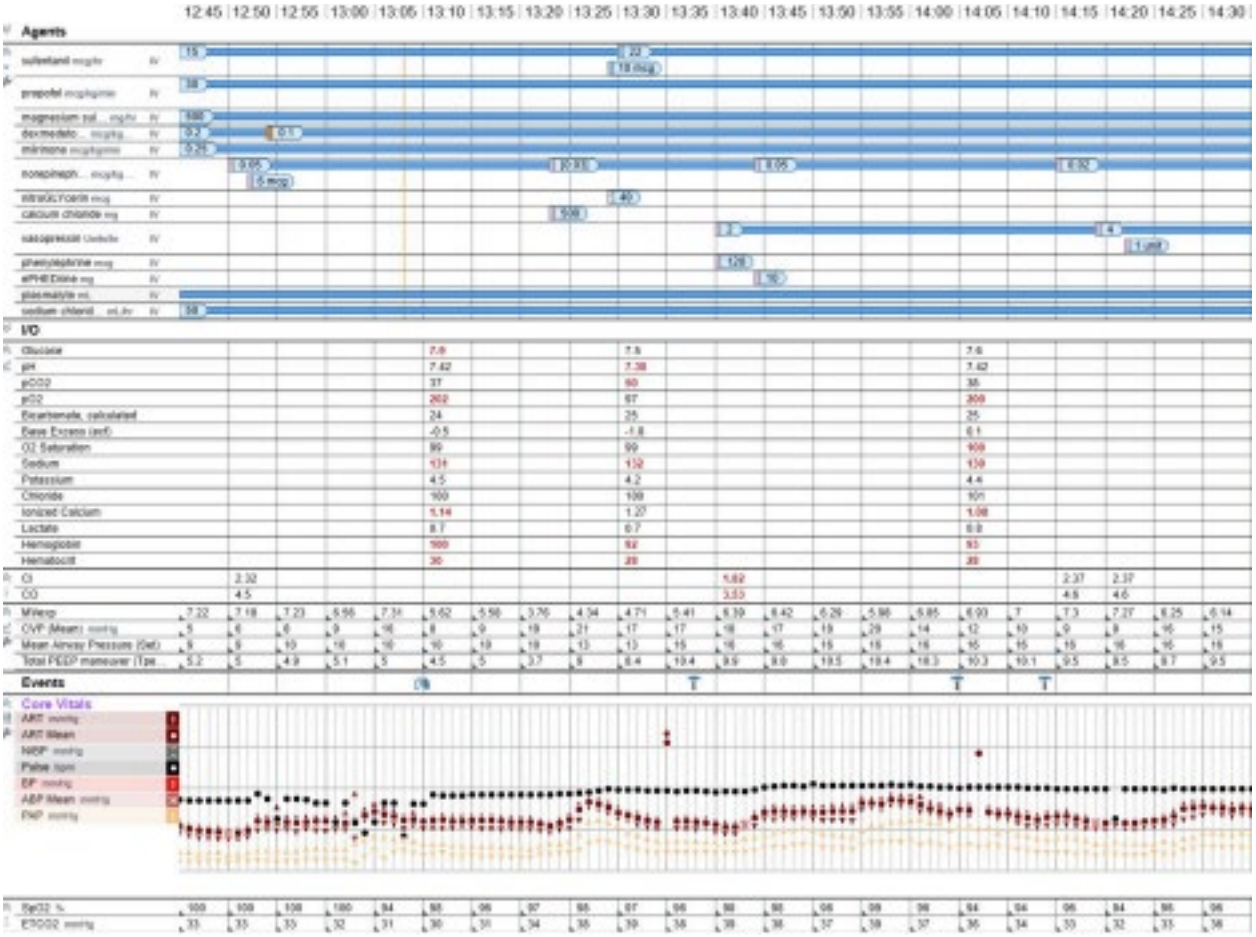
Warfarin was held due to the GI bleed. We avoided thoracic epidural analgesia given the avoidance of a full thoracotomy and the anticipated need for postoperative anticoagulation. Transesophageal echocardiography (TEE), typically useful for LVAD monitoring, was not possible due to the operation.

Intraoperatively, LVAD flow, power, and pulsatility index (PI) were monitored with a perfusionist available to adjust settings and a cardiac surgeon on standby. A left radial arterial line was placed before induction. Pulse oximetry may be unreliable at times due to lack of pulsatile flow, so oxygenation was also assessed using cerebral oximetry. Anesthesia was induced and maintained with propofol and sufentanil, and the patient was intubated with a 41 Fr double-lumen tube. A pulmonary artery (PA) catheter was placed. A milrinone infusion was started in anticipation of increased pulmonary vascular resistance from abdominal insufflation and one-lung ventilation (OLV) with capnothorax. We targeted a mean arterial pressure (MAP) of 60–80 mmHg and an LVAD flow of 4–4.5 L/min.

After OLV and capnothorax, PA pressures, $EtCO_2$, and $PaCO_2$ increased, with decreases in minute ventilation, LVAD flow, MAP, and cardiac output (CO) (Figure 1). In addition to milrinone, we increased afterload with norepinephrine and vasopressin to support RV perfusion, administered ephedrine for RV inotropy, and increased minute ventilation. $PaCO_2$, LVAD flow, PA pressures, and MAP improved without LVAD speed adjustment. Throughout the case, fluctuations in LVAD flow and MAP were managed by integrating CVP, PA pressures, PI, and CO to determine whether fluid therapy, inotropy, or afterload adjustment were necessary. Intravenous hydromorphone and dexmedetomidine, plus intercostal nerve blocks provided postoperative analgesia. The patient was extubated in the operating room free of vasopressor support, and was discharged home 2 weeks later with no anastomotic leak.

Discussion: MIE presents challenges to the LVAD patient, as abdominal insufflation with reverse Trendelenburg positioning, followed by OLV, chest insufflation, and lateral decubitus positioning with mediastinal manipulation, can compromise RV function and LVAD filling. Our case demonstrates the feasibility of LVAD management in MIE, even without the use of TEE. There was close communication with the surgeons to ensure positioning changes and insufflation were performed gradually, avoiding sudden reductions in preload. Invasive pressure monitoring integrated with LVAD parameters provided hemodynamic guidance. To our knowledge, this is the first case report describing an esophagectomy in a patient with an LVAD.

Image Uploads



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SP 2 - Kiosk 6 - Poster 1

Hyperfibrinolysis is common in open thoracoabdominal aortic aneurysm repairs and increases intraoperative transfusions, a pilot study

Presenting Author: *Andrew Milewski, MD, PhD*

Background: Numerous conditions—including ischemia-reperfusion activation, shock-induced fibrinolytic upregulation, and dilution and consumption of key fibrinolytic inhibitors—may precipitate hyperfibrinolysis (HF) in open repairs of thoracoabdominal aortic aneurysms (ORTAAAs).1 Owing to concerns around maintaining perfusion to the spinal cord and a lack of data supporting their use, antifibrinolytics are not given prophylactically in these surgeries but are instead given only after HF has been identified. Any coagulopathy in ORTAAAs, surgeries that entail large volumes of recirculated blood and extensive vascular suture lines exposed to deliberately augmented arterial pressures, can cause loss of large blood volumes. Owing to the long times (>30 minutes) typically needed for detection, HF is a particularly insidious coagulopathy that may trigger a vicious cycle of bleeding, dilution, consumptive coagulopathy, and high volume transfusion. Nevertheless, the prevalence of intraoperative HF has not been characterized in ORTAAAs, nor has the association between HF and intraoperative blood product use been explored.2 In this preliminary study, we investigate the local prevalence of HF and its effect on intraoperative blood product use in a pilot sample of ORTAAAs.

Methods: Intraoperative ROTEM data were matched to ORTAAAs performed at Weill Cornell (WC) from 4/19/2024–8/19/2025. HF was defined as ≥15% decrement of the EXTEM maximum clot firmness within 60 minutes of the clotting time measurement accompanied by a decaying or flat FIBTEM curve. Mann-Whitney U tests and Student's t-tests were employed to examine differences in intraoperative volumes of cell-salvage blood and numbers of red blood cells (RBCs), plasma, platelets, and cryoprecipitate (cryo) between cases in which ROTEM did or did not detect HF.

Results: Of the 42 patients who underwent ORTAAA, intraoperative ROTEM was performed in 41 cases. HF was detected in 33% (95% CI [20%,50%]) of cases (N = 14). The composition by gender, age, type of aneurysm, presence of dissection, and proportion of cases employing left atrial to femoral artery (LA/FA) partial bypass were similar between the HF and non-HF groups, as were the procedure times and aortic cross-clamp times, regardless of whether LA/FA bypass was employed. No statistically significant differences were found between the HF and non-HF groups for pre- or post-operatively measured Hgb, hematocrit, platelets, fibrinogen, INR, or aPTT. More cryo was used when HF occurred compared to cases in which HF was not detected (3.1±0.9 vs 2.0±0.6, p = 0.033). Trends were also seen towards greater numbers of RBCs (5.6±2.2 vs 3.5±1.1, p = 0.100), platelets (1.5±0.6 vs 1.1±0.4, p = 0.114), and plasma (2.6±1.1 vs 1.8±0.7, p = 0.144) in the HF group, but statistical significance was not reached in this underpowered pilot sample.

Conclusions: HF is common in ORTAAA and a study in a larger retrospective cohort is planned to better characterize the trends toward greater numbers of intraoperative blood products seen in this pilot study. Future studies are needed to identify the timing of HF onset in ORTAAAs and to examine whether targeted antifibrinolytic therapy can reduce intraoperative transfusion needs in this population.

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SP 2 - Kiosk 6 - Poster 2

Targeted therapy with factor concentrates for coagulation management in cardiac surgical patients: the ATN-108, FIBRES, and FARES-II randomized controlled trials

Presenting Author: *Jerrold Levy, MD, FCCM, FAHA*

Background: Cardiac surgery presents multiple challenges for bleeding management. Anticoagulation is required to prevent circuit thrombosis for cardiopulmonary bypass (CPB). Unfractionated heparin is the mainstay anticoagulant, but heparin resistance (HR), defined as >500 IU/kg without achieving a target activated clotting time of 480s occurs in ~10% of patients, often attributed to reduced antithrombin (AT) levels. AT is a critical cofactor for heparin effectiveness, and AT concentrate is a mainstay of therapy for managing HR, but is currently used off-label (not yet approved in US or Canada for acquired AT deficiency or HR).

Post CPB, coagulopathic bleeding is a serious complication with multiple causes. In North America, standard treatment for coagulation factor replacement has included allogeneic blood products (ABPs), such as cryoprecipitate (cryo) and frozen plasma (FP). However, these are associated with risks, including transmissible infections, adverse reactions, and availability. The pathogen-inactivated factor concentrates fibrinogen concentrate (FC) and four-factor prothrombin complex concentrate (4F-PCC), and AT concentrate instead of FP for treating HR, present favorable alternatives.

We describe post-hoc analyses from two major randomized trials of factor concentrates for coagulation management in patients undergoing cardiac surgery with CPB, aimed at characterizing changes in conventional coagulation tests, and the design of a new trial examining the efficacy of AT concentrate in patients with HR.

Methods: FIBRES (NCT03037424) compared FC (Octafibrin/FIBRYGA) vs cryo at 11 Canadian hospitals. FARES-II (LEX-211; NCT05523297) compared 4F-PCC (Octaplex/BALFAXAR) vs FP at 12 hospitals in Canada/US. ATN-108 (NCT06096116) is comparing AT concentrate (Atenativ) vs placebo at ~35 centers in Europe, US, Canada, and Central Asia. Study designs are summarized in Table 1 and Fig. 1.

Results: In total, 1,155 patients were treated in FIBRES (n=735) and FARES-II (n=420). In FIBRES, for the primary endpoint, mean ABP transfusions 24-h post-CPB were 16.3 (95% CI 14.9, 17.8) U in the FC group vs 17.0 (15.6, 18.6) U in the cryo group (ratio 0.96; 1-sided 97.5% CI −∞, 1.09; $p< 0.001$ for noninferiority). This coincided with a significantly higher increase in plasma fibrinogen with FC vs cryo (Fig. 2a). In FARES-II, the primary endpoint of effective hemostasis was achieved in 77.9% (n=166) of 4F-PCC vs 60.4% (n=125) of FP patients (diff. 17.6%; 95% CI 8.7, 26.4; $p< 0.0001$ for superiority). This coincided with a significantly higher proportion of patients on 4F-PCC vs FP achieving successful INR reduction (Fig. 2b). Rates of thromboembolic events and death were comparable between groups.

For ATN-108, recruitment began in Q4 2024. Target enrollment is ~120 patients, and completion is anticipated in Q1 2028.

Conclusion: FIBRES and FARES-II demonstrated efficacy and comparable safety of FC vs cryo and 4F-PCC vs FP in cardiac surgery patients requiring coagulation factor replacement. Algorithms for managing heparin resistance and coagulopathy, using highly purified factor concentrates guided by point-of-care testing, have the potential to reduce allogeneic transfusions and improve outcomes in surgical patients.

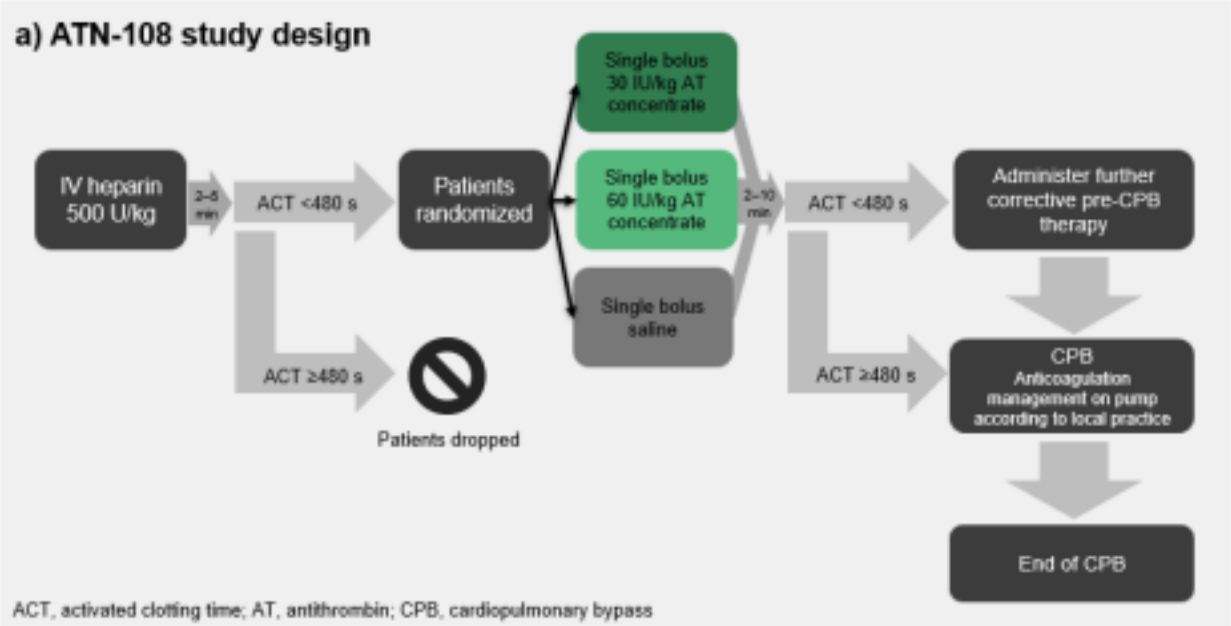
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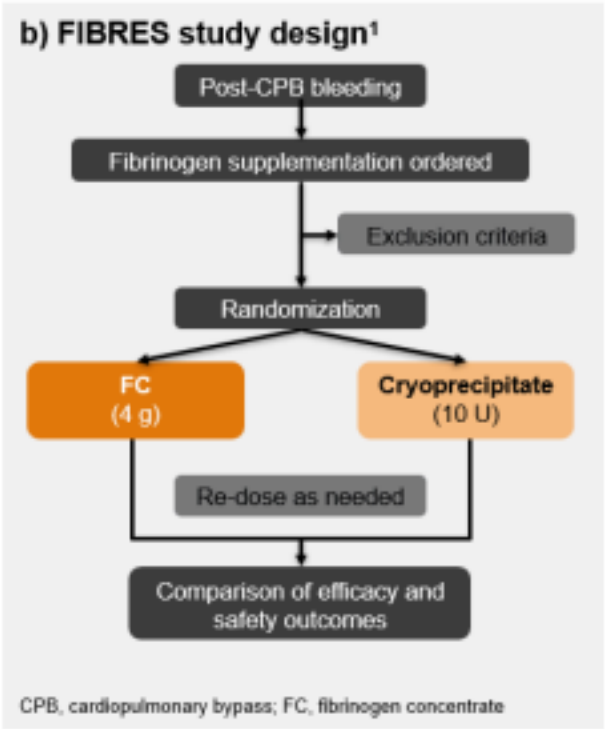
Table 1. Study overview

	ATN-108	FIBRES	FARES-II
Indication	Restoring/maintaining heparin responsiveness in patients undergoing CPB	Bleeding patients with acquired fibrinogen deficiency	Bleeding patients requiring coagulation factor replacement
Study design	Prospective, multicenter, randomized, placebo-controlled, double-blind, Phase 3 study	Prospective, multicenter, randomized, active-control, single-blinded, Phase 3 study	Prospective, multicenter, randomized, active-control, partially blinded, Phase 3 study
Status	Ongoing	Completed	Completed
Patients	Adult patients with planned cardiac surgery with CPB	Adult patients undergoing cardiac surgery with CPB	Adult patients undergoing cardiac surgery with CPB
	Heparin-resistant: pre-CPB activated clotting time <480 s between 2 and 5 min following IV administration of 500 U/kg UFH	Fibrinogen supplementation ordered for post-CPB hemorrhage with known or suspected acquired hypofibrinogenemia	Coagulation factor replacement ordered for management of bleeding or anticipated bleeding and known or suspected coagulation factor deficiency
Study treatment (IV infusion)	***Antithrombin concentrate:*** Bolus injection of 30 or 60 IU/kg ***Placebo (saline):*** Bolus injection of 0.6 or 1.2 mL/kg	***Fibrinogen concentrate:*** 4 g each time fibrinogen supplementation was ordered ***Cryoprecipitate:*** 10 U each time fibrinogen supplementation was ordered	***4F-PCC:*** 1,500 IU for BW ≤60 kg; 2,000 IU for BW >60 kg ***FP:*** 3 U for BW ≤60 kg; 4 U for BW >60 kg
		Re-dosed as needed during the first 24 h after termination of CPB, after which only cryoprecipitate was used for fibrinogen replacement	A second dose could be administered within 24 h after the first dose if the patient continued to have at least moderate bleeding and suspected coagulation deficiency
Primary endpoint	Efficacy: Comparison of the percentage of patients in each group in whom no further therapy containing antithrombin (i.e., FP or other antithrombin concentrates) is needed for restoring pre-CPB heparin responsiveness after administration of antithrombin concentrate or placebo, and for maintaining it during CPB	Efficacy: Comparison of the total number of units of ABPs (RBCs, pooled or apheresis platelets, and plasma) administered during the first 24 h after termination of CPB	Efficacy: Comparison of hemostatic treatment response to 4F-PCC versus FP, defined as 'effective' if no additional hemostatic interventions (such as administration of any systemic hemostatic agents or surgical re-opening for bleeding) were required from 60 min to 24 h after initiation of the first dose of IMP
Safety	AEs, including TEEs, and deaths	AEs, including TEEs, and deaths	AEs, including TEEs, and deaths

4F-PCC, four-factor prothrombin complex concentrate; ABPs, allogeneic blood products; AEs, adverse events; BW, body weight; CPB, cardiopulmonary bypass; FP, frozen plasma; IMP, investigational medicinal product; IU, international units; IV, intravenous; RBCs, red blood cells; TEEs, thromboembolic events; U, units; UFH, ultrafractionated heparin

Figure 1.


a) ATN-108 study design
IV heparin 500 U/kg
3–5 min
ACT <480 s
Patients randomized
Single bolus 30 IU/kg AT concentrate
Single bolus 60 IU/kg AT concentrate
Single bolus saline
2–10 min
ACT <480 s
Administer further corrective pre-CPB therapy
ACT ≥480 s
Patients dropped
ACT ≥480 s
CPB
Anticoagulation management on pump according to local practice
End of CPB
ACT, activated clotting time; AT, antithrombin; CPB, cardiopulmonary bypass


b) FIBRES study design[1]
Post-CPB bleeding
Fibrinogen supplementation ordered
Exclusion criteria
Randomization
FC (4 g)
Cryoprecipitate (10 U)
Re-dose as needed
Comparison of efficacy and safety outcomes
CPB, cardiopulmonary bypass; FC, fibrinogen concentrate

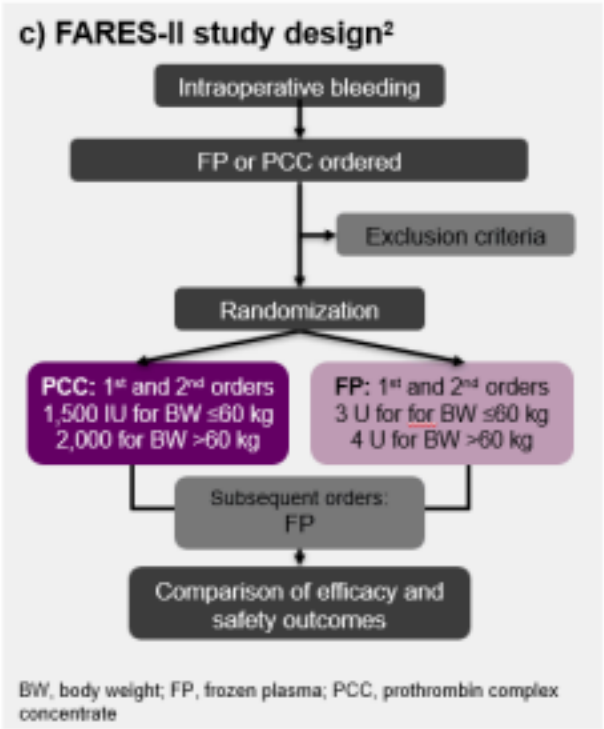
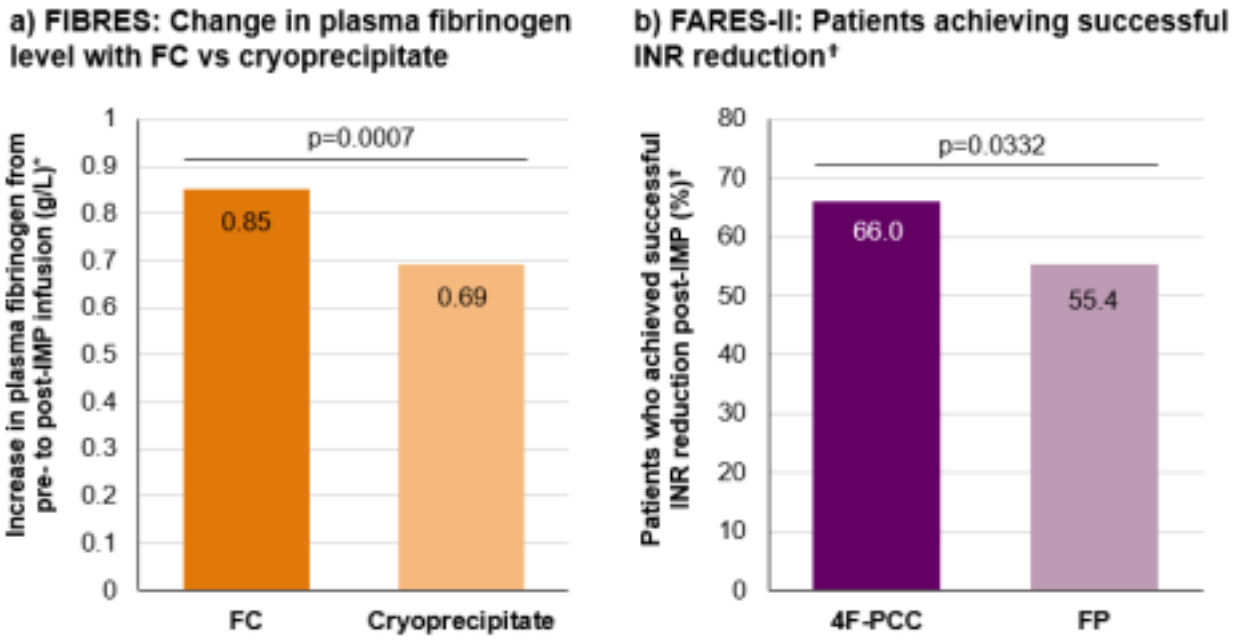

c) FARES-II study design[2]
Intraoperative bleeding
FP or PCC ordered
Exclusion criteria
Randomization
PCC: 1st and 2nd orders 1,500 IU for BW ≤60 kg 2,000 for BW >60 kg
FP: 1st and 2nd orders 3 U for for BW ≤60 kg 4 U for BW >60 kg
Subsequent orders: FP
Comparison of efficacy and safety outcomes
BW, body weight; FP, frozen plasma; PCC, prothrombin complex concentrate

Figure 2.



* Only measurements taken within 75 minutes before and after start of the infusion are included in the analysis

† INR reduction was considered successful if the magnitude of the reduction was >1.0 or the post-treatment level dropped below 1.5.

4F-PCC, four-factor prothrombin complex concentrate; FC, fibrinogen concentrate; FP, frozen plasma; IMP, investigational medicinal product; INR, international normalized ratio.

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SP 2 - Kiosk 6 - Poster 3

Fibrinogen concentrate restores plasma fibrinogen levels and increases clot strength in patients with acquired fibrinogen deficiency: An integrated analysis from Phase 2 and Phase 3 studies

Presenting Author: *Andres Brainsky, MD*

Background: Fibrinogen supplementation is recommended to restore hemostasis in patients with acquired fibrinogen deficiency (AFD) who are bleeding in various therapeutic settings.1–4 Fibrinogen concentrate (FC) has been shown to effectively elevate plasma fibrinogen levels in patients with AFD, including those undergoing cardiac surgery.5–7 To further examine the efficacy and safety of FC for restoring plasma fibrinogen levels and clot strength, we analyzed a pooled dataset from two previously published randomized controlled trials (RCTs) that investigated the

efficacy and safety of FC for the treatment of AFD in patients undergoing cardiovascular surgery with cardiopulmonary bypass who had intraoperative bleeding. Both trials were placebo-controlled and included a Phase 2 single-center study in patients undergoing aortic replacement surgery (NCT00701142) and a Phase 3 multicenter study in patients undergoing complex cardiovascular surgery (NCT01475669).8–9

Methods: All patients who received FC or placebo in the original RCTs were pooled in this integrated analysis population and used to evaluate outcomes between participants who received FC and those who received placebo. The primary endpoint was the change from pre- to post-infusion plasma fibrinogen levels. In addition, changes in clot strength were assessed using viscoelastic testing (ROTEM®; FIBTEM maximum clot firmness [MCF]). The difference between the treatment groups for efficacy endpoints was assessed using least squares (LS) mean difference. Safety analysis included all treatment-emergent adverse events (TEAEs). Results In total, 213 subjects were included in the pooled population; 107 received FC and 106 received placebo. The majority of subjects in both groups were male (FC: 73.8%; placebo: 71.7%;). Age and BMI were similar between the FC and placebo groups (mean age 62.7 vs 63.3 years and mean BMI 26.40 vs 26.18 kg/m2, respectively). Pre-infusion plasma fibrinogen levels were comparable between the FC and placebo groups (mean 1.79 vs 1.71 g/L), as was pre-infusion clot strength (mean MCF 9.9 vs 9.3 mm;

Table 1). The plasma fibrinogen level change from pre- to post-infusion was significantly greater in the FC group than in the placebo group (LS mean change 1.02 vs 0.18 g/L; $p< 0.0001$; Table 1) and post-infusion plasma fibrinogen levels were higher in the FC group compared with the placebo group (mean 2.83 vs 1.90 g/L; Table 1). The change in MCF was significantly greater in the FC group compared with the placebo group (LS mean change 8.4 vs 1.3 mm; $p< 0.0001$; Table 1). No clinically relevant differences were observed between the FC and placebo groups in the proportion of patients experiencing TEAEs, severe TEAEs or treatment-related TEAEs, while the proportion of subjects with TEAEs leading to death was lower in the FC group (1.9% vs 8.5% in the placebo group; Table 2).

Conclusion: In this integrated analysis, FC was effective in restoring plasma fibrinogen levels to the normal physiological range (2–4 g/L)10 and increasing clot strength in bleeding patients with AFD undergoing complex cardiac surgery, with a safety profile comparable to placebo. This pooled analysis provides further evidence of the efficacy and safety of FC in patients with AFD undergoing complex cardiac surgery.

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Table 1: Efficacy endpoints

	Pooled clinical trial data (Phase 2 and Phase 3 RCTs) (N=213)	
	Fibrinogen concentrate (n=107)	Placebo (n=106)
Plasma fibrinogen levels (g/L)*		
Pre-infusion	n=106	n=106
Mean (SD)	1.79 (0.61)	1.71 (0.47)
Post-infusion†	n=106	n=104
Mean (SD)	2.83 (0.63)	1.90 (0.48)
Change from pre- to post-infusion†	n=106	n=104
LS mean (SE)	1.02 (0.06)	0.18 (0.05)
LS mean difference (SE); p-value	0.84 (0.08); p<0.0001	
FIBTEM MCF (mm)		
Pre-infusion	n=104	n=101
Mean (SD)	9.9 (4.2)	9.3 (3.5)
Post-infusion†	n=103	n=101
Mean (SD)	18.9 (4.4)	10.5 (4.1)
Change from pre- to post-infusion†	n=103	n=101
LS mean (SE)	8.4 (0.4)	1.3 (0.4)
LS mean difference (SE); p-value	7.1 (0.6); p<0.0001	

*As assessed by Clauss assay.
†Post-infusion was at end of surgery: in the Phase 2 study, this was at last suture; and in the Phase 3 study at skin closure.
FIBTEM, fibrinogen thromboelastometry; LS, least squares; MCF, maximum clot firmness; RCTs, randomized controlled trials; SD, standard deviation; SE, standard error.

Table 2: Adverse events

	Pooled (Phase 2 and Phase 3 RCTs) (N=213)	
	Fibrinogen concentrate (n=107)	Placebo (n=106)
Any TEAE, n (%)	99 (92.5)	94 (88.7)
Any severe TEAE, n (%)	45 (42.1)	45 (42.5)
Treatment-related TEAE*, n (%)	10 (9.3)	10 (9.4)
Any TEAE leading to death, n (%)	2 (1.9)	9 (8.5)

*Assessed by the investigator as at least possibly related to the treatment.
RCTs, randomized controlled trials; TEAE, treatment-emergent adverse event.

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SP 2 - Kiosk 6 - Poster 4

Viscoelastic tests did not reduce transfusion during lung transplantation: The QuanTrans Multicentric Randomized Trial

Presenting Author: *Julien Fessler, MD*

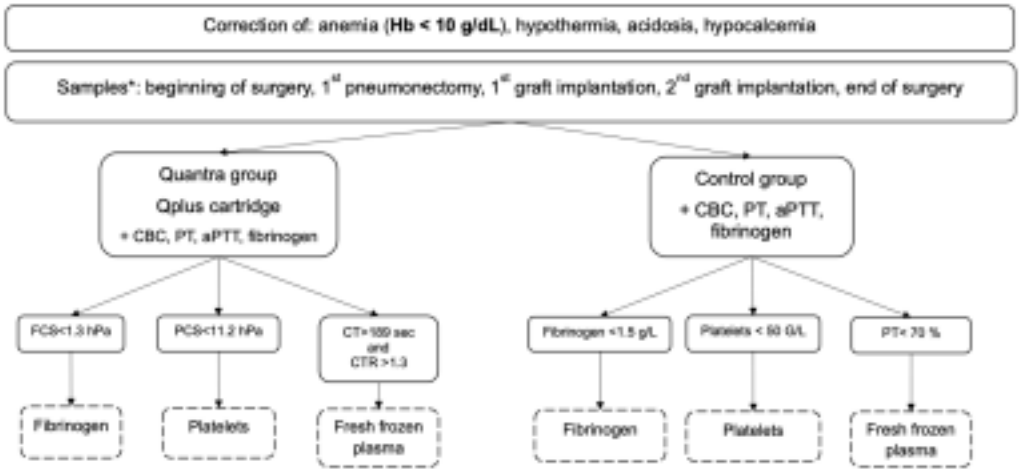
Background: Transfusion is frequent in lung transplantation despite improvements in surgical techniques. It is a deleterious risk factor for primary graft dysfunction, chronic rejection, and mortality. The main objective of this study is to determine if a transfusion algorithm based on a whole blood viscoelastic test with Quantra® test reduces transfusion during lung transplantation compared to the protocol based on a standard coagulation test.

Methods: We conducted a multicentric randomized clinical trial including patients undergoing double-lung transplantation between May 2023 and October 2024 at four hospitals in France. We excluded patients under cardiopulmonary bypass, preoperative ECMO, and multiorgan transplantation. Patients were randomized to be managed either based on the Quantra QPlus Cartridge or based on standard coagulation tests (PT, aPTT, or fibrinogen). The blood samples were collected at five surgical time points: upon arrival at the operating room, after the first pulmonary artery clamping, after the first graft implantation, after the second graft implantation, and after surgery. Based on a historical cohort, the primary outcome was a 30% reduction in the number of blood products (red blood cell packs and fresh frozen plasma), with a target of 7.95 (SD: 6.33). Quantitative variables are compared using the T-test or Mann-Whitney/Wilcoxon test. Qualitative variables are compared using the Chi2 test or Fisher test. The protocol was registered in Clinical Trials (NCT05798286) and approved by the “Comité de Protection des Personnes Sud-Est V”.

Results: Of the 128 patients included in the study, 55 were randomized to the Quantra group and 56 to the Standard group. In the Quantra group, 2 patients underwent a redo transplantation, whereas only 1 patient in the standard group did. There was no difference in the primary outcome, the number of intraoperative blood products transfused, 3.89+/- 6.96 in the Quantra group and 2.67 +/- 3.6 in the Standard group, p = 0.923, nor in the number of red blood cell packs of fresh frozen plasma. A post hoc analysis revealed that 640 subjects would have been necessary to observe a statistically significant difference.

Conclusion: Patient blood management based on viscoelastic test with Quantra did not reduce the transfusion during double lung transplantation in this multicentric randomized control trial. The results of our study reflect the global improvement and changes in clinical practice in transfusion during lung transplantation and question the systematic use of viscoelastic tests.

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	Quantra group (N=55)	Standard group (N= 56)	P value
Age	55.44 +/- 13.04	56.71 +/- 8.20	0.53
Women	21 (38.2%)	20 (35.7%)	0.78
Weight	63.49 +/- 12.77	70.04 +/- 13.18	0.009
Height	169.67 +/- 8.85	169.48 +/- 8.74	0.91
BMI	22.03 +/- 3.98	24.37 +/- 4.17	0.005
Suppurative	5 (9.1%)	1 (1.8%)	0.17
Obstructive	31 (56.4%)	29 (51.8%)	
Restrictive	13 (23.6%)	20 (35.7%)	
PHT	2 (3.6%)	0 (0.0%)	
Others	4 (7.3%)	6 (10.7%)	
RBC packs	2.38 +/- 2.63	2.18 +/- 2.78	0.47
FFP packs	1.65 +/- 4.23	1.57 +/- 2.93	0.45
Platelets units	0.22 +/- 0.74	0.09 +/- 0.44	0.12
Redo	3 (5.5%)	1 (1.8%)	0.29
PMH thoracic surgery	8 (14.5%)	12 (21.4%)	0.34
Aspirin	9 (16.4%)	10 (17.8%)	0.83

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SP 2 - Kiosk 6 - Poster 5

Early Experience with Point-of-Care Coagulation Monitoring Using a Miniaturized Laser Speckle Analyzer in Impella Patients

Presenting Author: *Aubrey Greer, MD*

Background: Patients supported with Impella devices require careful anticoagulation management to balance thrombotic and hemorrhagic risks. Conventional coagulation testing involves delays between sample collection and result availability, limiting timely clinical decision-making. The iCoagLab is a laser speckle-based coagulation analyzer that provides comprehensive coagulation profiling from ultra-low blood volumes (< 60 µL) in under 15 minutes. We previously demonstrated strong correlations between iCoagLab metrics and conventional laboratory tests in cardiac surgery ICU patients using a benchtop device. Here, we evaluate a miniaturized point-of-care iCoagLab console in patients with Impella mechanical circulatory support.

Methods: We conducted a prospective pilot study in patients with Impella devices at our institution. Non-citrated whole blood samples were obtained and tested at the bedside using the point-of-care iCoagLab console. Three lyophilized reagent assays were used: activated kaolin (intrinsic pathway), prothrombin time (extrinsic pathway), and fibrinogen. Time-matched samples were sent to the core laboratory for conventional coagulation testing (aPTT, PT, fibrinogen, platelet count). iCoagLab metrics were correlated with core laboratory results using Pearson's correlation coefficient.

Results: Twelve paired measurements were obtained from 7 patients. iCoagLab maximum amplitude (MA), a measure of clot strength, showed significant correlation with core laboratory platelet count (r=0.645, p=0.024) and moderate correlation with core fibrinogen (r=0.594, p=0.070). iCoagLab fibrinogen correlated with core fibrinogen (r=0.538, p=0.135). iCoagLab ACT showed a negative correlation with core aPTT (r=-0.525, p=0.097). This may reflect the influence of fibrinogen substrate on whole-blood clot detection that is not captured by plasma-based aPTT testing and may also be influenced by the anticoagulation agent used.

Conclusions: The miniaturized iCoagLab point-of-care console demonstrates promising correlation with conventional laboratory testing in Impella-supported patients, particularly for clot strength parameters. The significant MA-platelet correlation supports the device's utility for rapid bedside assessment. The divergent ACT-aPTT relationship may reflect known differences between whole-
blood viscoelastic and plasma-based assays in mechanical circulatory support patients. Larger studies are needed to validate these findings and enrollment in this study is ongoing.

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SP 2 - Kiosk 6 - Poster 6

Thromboembolic Outcomes Following Fibrinogen Concentrate Versus Cryoprecipitate Use in Major Cardiovascular Surgery: A Real-World Analysis

Presenting Author: *Melissa Cushing, MD*

Background: Perioperative coagulopathy is common in major cardiovascular (CV) surgery and often involves low fibrinogen levels requiring replacement to maintain hemostasis.1-3 Replacement options include plasma, cryoprecipitate (cryo), and fibrinogen concentrate (FC). A recent randomized trial in CV surgery found FC noninferior to cryo for hemostatic efficacy and suggested fewer thromboembolic events (TEEs), though interpretation was limited by heterogeneity and sample size.4 We evaluated real-world postoperative TEE incidence among patients receiving FC or cryo during major CV surgery.

Methods: We conducted a retrospective cohort study using electronic health record data from the TriNetX Dataworks US network. Adults (≥18 years) who underwent major CV surgery between January 1, 2016 and December 31, 2024, and received either FC or cryo at the index surgery (patient's first qualifying surgical procedure) were included. Patients receiving both products during the index surgery, those with hereditary clotting factor deficiencies or primary thrombophilias, and those undergoing another surgery within 30 days with the alternate therapy were excluded. Procedures were identified using ICD-10-PCS and CPT codes and limited to open surgical approaches, including coronary artery bypass grafting (CABG), valve procedures, aortic surgery, septal defect repair, heart transplant, and complex congenital operations. Propensity score matching (1:2, optimal fixed ratio) was used to balance baseline characteristics, including age, sex, race, ethnicity, Charlson Comorbidity Index, history of thromboembolic events within 3 months prior, prophylactic anticoagulation, baseline hemoglobin, and 3-month history of cancer and metastatic cancer. The primary outcome was the incidence of 30-day postoperative TEEs. Logistic regression estimated TEE risk difference, and Kaplan–Meier (KM) analysis with log-rank testing compared time to TEE.

Results: A total of 19,930 patients were identified (4,091 received FC; 15,839 received cryo). Among index surgeries, the most common procedure types were CABG (27.3%), aortic valve (20.6%), aortic surgery (12.9%), and mitral valve (12.1%) (Figure 1). After matching, 10,734 patients remained (3,578 FC; 7,156 cryo). In the matched cohort, overall median age was 66 years (IQR 57–73), and 30.6% were female.

The FC group had lower 30-day rates of overall TEEs compared with the cryo group (8.0% vs. 9.5%), including stroke, transient ischemic attack, arterial thromboembolism, venous thromboembolism (VTE), and pulmonary embolism (PE). VTE/PE rates were also lower (2.1% vs 3.1%). Adjusted odds of all TEEs were lower with FC (OR: 0.79; 95% CI: 0.68–0.92; p = 0.002), as were odds of VTE/PE (OR: 0.63; 95% CI: 0.48–0.83; p = 0.001). KM analysis demonstrated a lower 30-day cumulative incidence and delayed onset of overall TEEs (Figure 2) and VTE/PE (Figure 3) in the FC group compared with cryo (log-rank p = 0.005 and p = 0.001, respectively).

Conclusion: In this large, real-world matched cohort of patients undergoing major CV surgery, FC was associated with a lower incidence of 30-day TEEs compared with cryo. These findings build on existing trial data and suggest that FC may offer a favorable thrombotic profile in the perioperative setting.

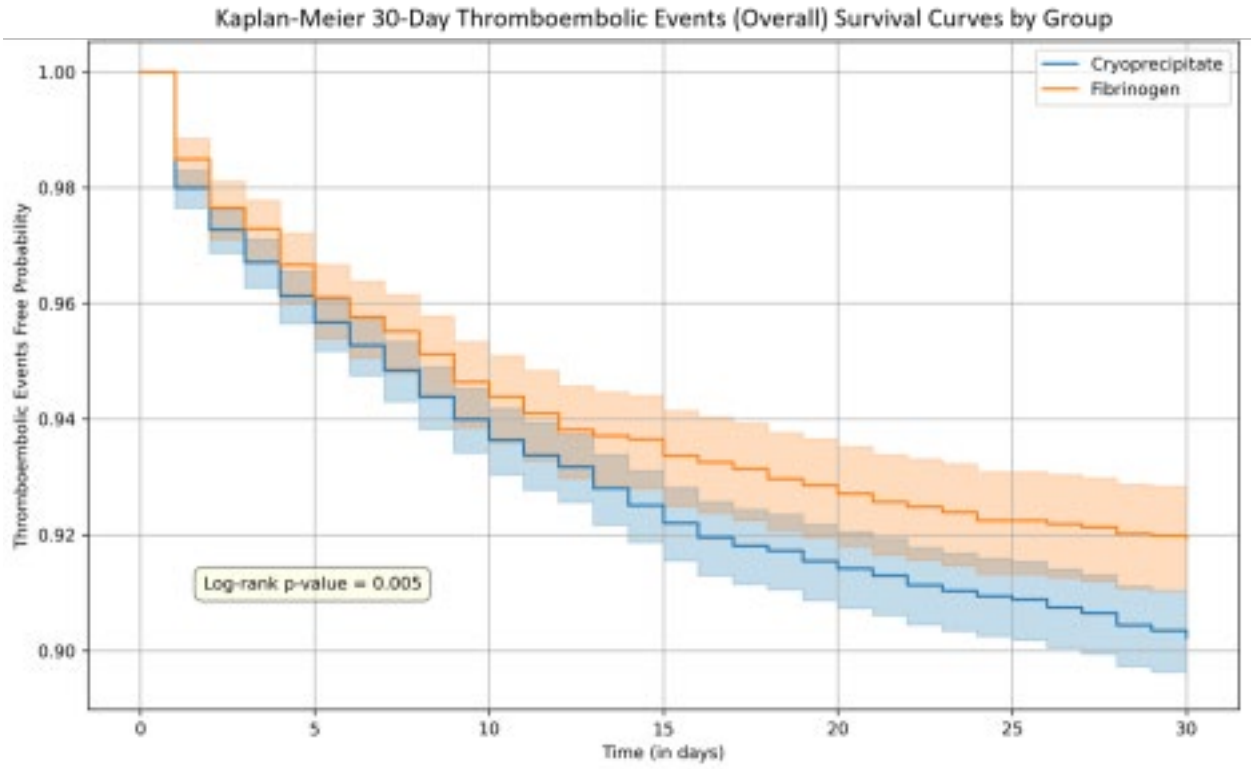
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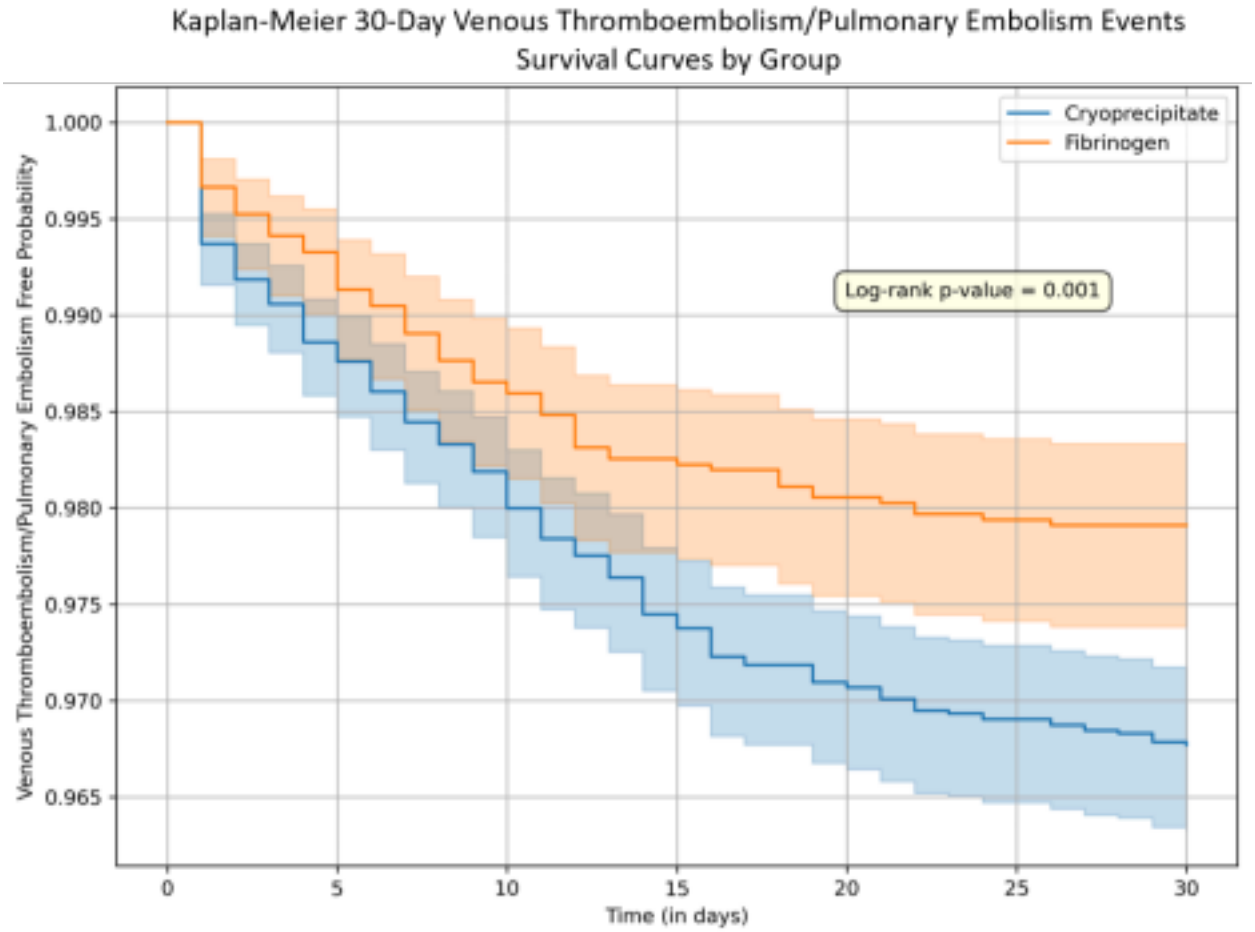
Surgery Type*	All Surgeries (N=32,191)		Surgeries with FC (n = 6,351)		Surgeries with Cryo (n = 25,818)		Relative Frequency of FC vs Cryo Use***
	n	%	n	%	n	%	
Coronary artery bypass graft (CABG) surgery	6772	27.3%	1919	28.5%	6953	26.9%	1.06
Aortic valve procedure	6630	20.6%	1196	18.9%	5441	21.1%	0.89
Surgery on aorta	4135	12.9%	587	9.2%	3548	13.7%	0.67
Mitral valve procedure	3886	12.1%	917	14.4%	2979	11.5%	1.25
Tricuspid/pulmonary valve procedure	1475	4.6%	286	4.5%	1187	4.6%	0.98
ASD (atrial septal defect)/VSD (ventricular septal defect) repair	1089	3.1%	189	2.7%	840	3.3%	0.82
Heart transplant	744	2.3%	160	2.5%	576	2.2%	1.13
Complex congenital	30	0.1%	5	0.1%	26	0.1%	0.81
Other**	5481	17.0%	1260	19.9%	4221	16.5%	1.14

* Please note that the N listed here represents the number of surgeries, counting each patient only once per surgery group. For example, if a patient had two CABG procedure codes and one mitral valve procedure code at index, they would be counted once in the CABG surgery group and once in the mitral valve procedure group.

** "Other" includes structural or adjunct cardiac procedures not categorized as CABG, valve, aortic, transplant, or congenital. Examples include LAA resection, ventricular restorative, pericardial or pulmonary artery repair, and minor procedures.

*** Indicates how frequently FC was used relative to Cryo for each surgery type; values <1 indicate greater FC use, and values >1 indicate greater Cryo use.





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SP 2 - Kiosk 7 - Poster 1

Long Term Outcomes After Intraoperative or Postoperative ECMO Initiation for Post-cardiotomy Cardiogenic Shock Following Aortic Valve Surgery: A National Retrospective Propensity-Matched Study

Presenting Author: *Yousef Rafati, MD*

Background: Post-cardiotomy cardiogenic shock is a life-threatening condition characterized by reduced cardiac output after failure to wean from cardiopulmonary bypass or postoperative cardiac dysfunction.1 Veno-Arterial Extracorporeal Membrane Oxygenation (VA-ECMO) is increasingly utilized as a bridge to recovery in this setting, with use ranging from 0.4% to 3.7%.2 While prior studies have focused on in-hospital outcomes, data on long-term survival and functional recovery of patients' placed on VA-ECMO support after valve replacement is limited.3 Small patient numbers and heterogeneous perioperative characteristics have hindered rigorous analyses. Thus, we aimed to characterize five-year survival and functional outcomes of intraoperative and post-operative VA-ECMO initiation for post-cardiotomy cardiogenic shock following aortic valve surgery.

Methods: We conducted a retrospective cohort study using the TriNetX Health Research Network, comprising data from 66 U.S. healthcare organizations and over 135 million individuals. Patients undergoing aortic valve replacement who received intraoperative or early postoperative VA-ECMO were identified using the ICD-10 codes. Demographics and perioperative data from November 2015 onward were collected. Baseline covariates were balanced using 1:1 propensity score matching. Outcomes, including five-year survival and functional outcomes, were assessed after the index window. Kaplan-Meier curve was used to evaluate five-year post-operative mortality. Hazard ratios and confidence intervals were calculated to compare the cohorts and evaluate outcomes after matching.

Results: A total of 56,732 patients underwent aortic valve replacement, of whom 553 received intraoperative or postoperative VA-ECMO support. After propensity score matching for demographics and comorbidities, 549 patients were included in each cohort. Baseline characteristics before and after matching are shown in Table 1. At five years, individuals requiring VA-ECMO cannulation had a significant risk for mortality (hazard ratio [HR]: 5.79; 95% confidence interval [CI]: 4.58-7.32; $P < 0.001$) with a median survival of 45 days and 30.5% survivability at the end of the time window (Figure 1), sepsis and shock (HR: 4.17; 95% CI: 2.63-6.62; $P < 0.001$), gastrointestinal hemorrhage (HR: 2.48; 95% CI: 1.60-3.84; $P < 0.001$), pneumonia (HR: 1.72; 95% CI: 1.24-2.39; $P < 0.001$), encephalopathy (HR: 2.53; 95% CI: 1.79-3.57; $P < 0.001$), cerebral infarction (HR: 1.98; 95% CI: 1.38-2.81; $P < 0.001$), depression (HR: 1.91; 95%

CI: 1.25-2.92; P < 0.05), anxiety (HR: 1.65; 95% CI: 1.17-2.33; P < 0.05), ventilator dependence (HR: 4.54; 95% CI: 2.86-7.21; P < 0.001), and dialysis dependence (HR: 5.20; 95% CI: 3.77-7.18; P < 0.001).

Conclusion: Patients' requiring intraoperative or postoperative VA-ECMO after aortic valve surgery experience increased morbidity and reduced functional outcomes. Although overall ECMO survival remains low, long-term outcomes are favorable with five-year survival notable among survivors. These results underscore the impact of post-cardiotomy cardiogenic shock requiring ECMO on functional outcomes and highlight the need for future research to identify factors that improve long-term survival and quality of life in this population.

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Table 1. Baseline characteristics of patients who underwent aortic valve replacement with and without the need for VA-ECMO before and after propensity score matching (PSM)

Factor	Before PSM			After PSM		
	ECMO Cohort (n = 523)	Control Cohort (n = 56,179)	*P* Value	ECMO Cohort (n = 549)	Control Cohort (n = 549)	*P* Value
Age, years						
18-39	7.78%	5.16%	<0.01	7.65%	6.56%	0.48
39-60	28.39%	25.98%	0.20	28.60%	29.87%	0.64
61-80	60.04%	63.40%	0.10	59.93%	59.02%	0.76
81-102	3.44%	5.03%	0.08	3.46%	4.19%	0.53
Sex						
Male	62.93%	68.77%	<0.01	62.66%	63.21%	0.85
Female	36.53%	30.72%	<0.01	36.79%	36.43%	0.90
Race						
White	66.00%	77.28%	<0.01	65.76%	59.02%	<0.05
Hispanic	7.60%	4.83%	<0.01	7.65%	9.11%	0.38
Black	7.05%	6.60%	0.67	7.10%	7.10%	1.00
Asian	1.81%	4.52%	<0.01	1.82%	1.82%	1.00
Cardiac Comorbidities						
Coronary Artery Disease	60.58%	57.54%	0.15	60.29%	58.65%	0.58
Essential hypertension	58.41%	60.90%	0.23	58.29%	58.47%	0.95
Hyperlipidemia	54.79%	56.86%	0.32	54.65%	52.82%	0.54
Type 2 diabetes mellitus	33.64%	25.70%	<0.01	33.52%	35.88%	0.41
Overweight and obesity	31.83%	25.16%	<0.01	31.69%	32.79%	0.70
Systolic heart failure	30.38%	14.77%	<0.01	29.87%	30.97%	0.70
Diastolic heart failure	22.97%	15.92%	<0.01	22.95%	21.86%	0.66
Biventricular heart failure	15.73%	1.04%	<0.01	15.12%	11.66%	0.09
Right heart failure	13.20%	0.95%	<0.01	12.57%	10.57%	0.30
Atrial fibrillation and flutter	45.39%	27.13%	<0.01	45.17%	44.99%	0.95
Acute myocardial infarction	23.51%	12.13%	<0.01	23.32%	22.40%	0.72
Cardiac arrest	13.74%	1.63%	<0.01	13.12%	14.39%	0.54
Peripheral vascular disease	8.68%	7.42%	0.26	8.56%	7.10%	0.37
Pulmonary Comorbidities						
Chronic obstructive pulmonary disease	14.47%	10.03%	<0.01	14.57%	13.48%	0.60
Pulmonary embolism	6.51%	2.52%	<0.01	6.38%	6.19%	0.90
Pulmonary hypertension	23.87%	12.49%	<0.01	23.50%	26.78%	0.21
History of nicotine dependence	29.84%	25.01%	<0.01	29.69%	27.69%	0.46

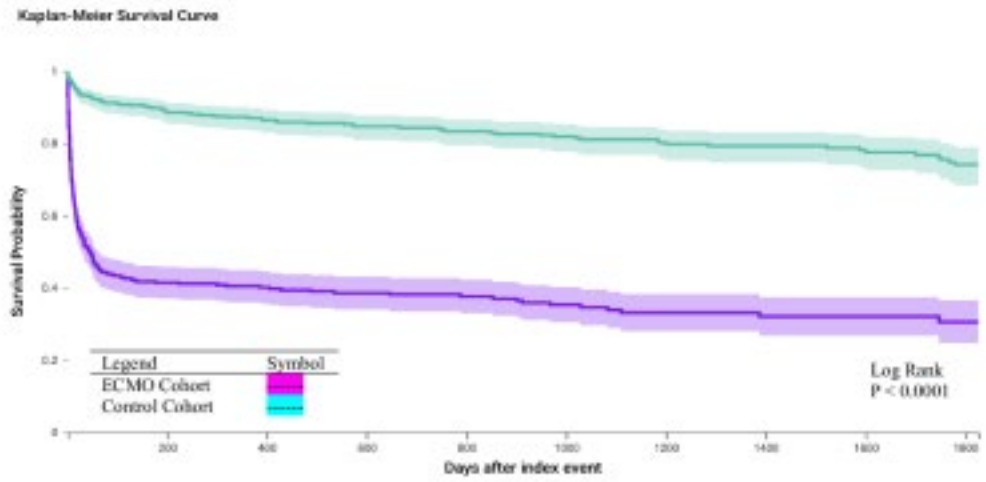


Figure 1. Kaplan-Meier Survival Curve between the ECMO and Control Cohort

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SP 2 - Kiosk 7 - Poster 2
Perioperative Hemodynamic Predictors of Right Ventricular Failure After Impella 5.5 Implantation

Presenting Author: *Nicolas Kumar, MD*

BACKGROUND: Right ventricular failure (RVF) is a major driver of morbidity, renal dysfunction, and mortality in patients requiring Impella 5.5 support for isolated left ventricular failure.1 Accurate early risk stratification is critical for determining the need for biventricular mechanical support and optimizing perioperative management.2 Although several hemodynamic indices of RV function are validated in durable left ventricular assist device (LVAD) populations, their predictive utility in Impella 5.5 patients and across perioperative timepoints remains unclear.3

METHODS: We performed a retrospective cohort study of 198 patients who were supported with Impella 5.5 for cardiogenic shock at two tertiary centers. Four hemodynamic predictors—pulmonary artery pulsatility index (PAPi), right atrial–to–pulmonary capillary wedge pressure ratio (RA:PCWP), right ventricular stroke work index (RVSWI), and mean pulmonary–to–mean arterial pressure ratio (mPAP:MAP)—were assessed at three timepoints: pre-Impella, immediately post-activation upon arrival to the intensive care unit, and 24 hours after support initiation. Logistic regression models (unadjusted and adjusted for age, vasoactive-inotropic score, Impella flow, and duration of support) evaluated associations with RVF. Using the INTERMACS' definition, RVF was defined as a composite of right ventricular mechanical circulatory support (MCS), inhaled pulmonary vasodilator therapy for 48 hours or greater, or inotrope use for ≥14 days. Receiver operating characteristic (ROC) analysis determined optimal discriminative thresholds.

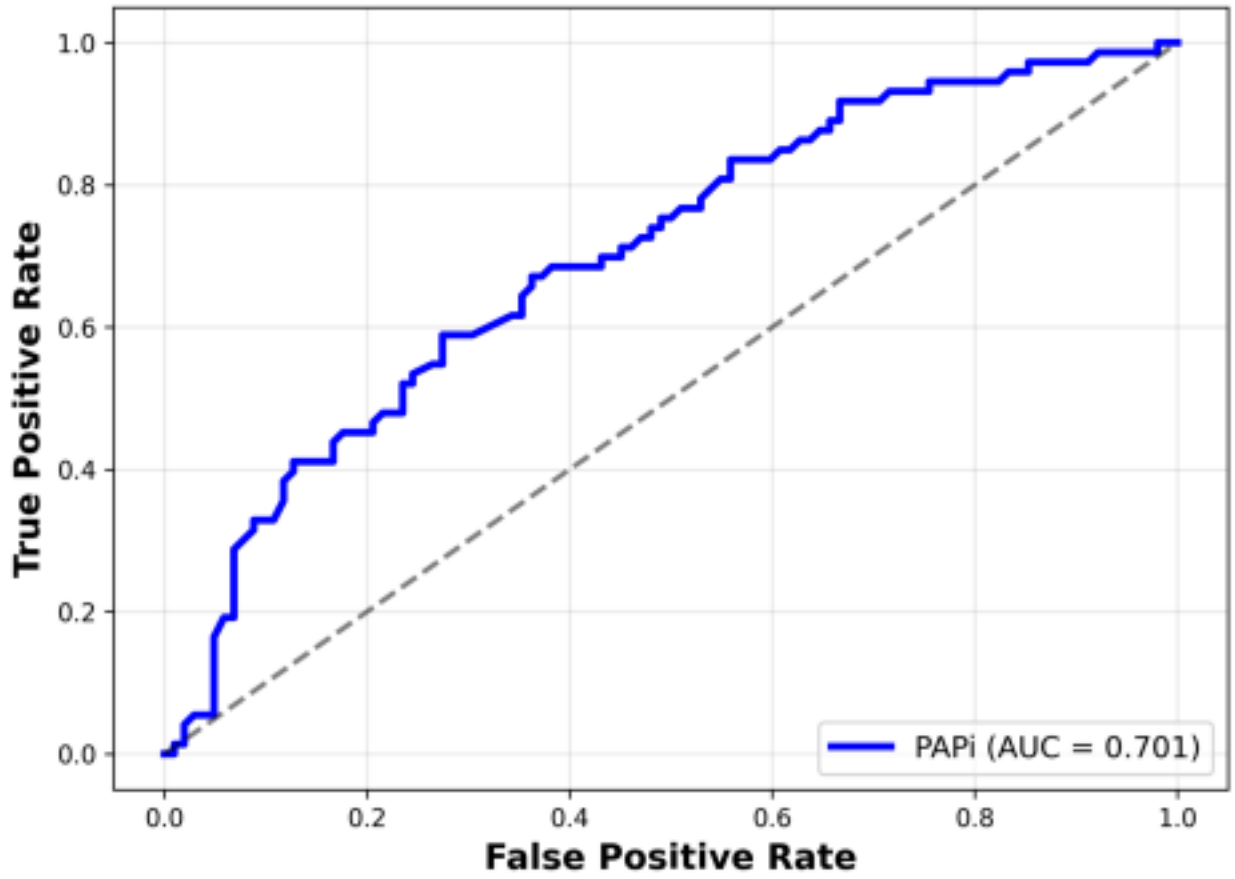
RESULTS: RVF occurred in 77 patients (38.9%) (Table 1). Pre-Impella, PAPi strongly predicted RVF

(adjusted OR 0.67; 95% CI: 0.53, 0.85, p< 0.001), and RA:PCWP predicted both 30-day mortality (OR 12.0; 95% CI: 1.83, 78.9, p=0.010) and renal failure requiring dialysis (OR 18.1; 95% CI: 3.13, 104.6, p=0.001). Immediately post-Impella 5.5 activation, PAPi remained predictive of RVF (OR 0.62; 95% CI: 0.46, 0.82, p< 0.001) and predicted need for dialysis (OR 0.55; 95% CI: 0.38, 0.79, p=0.001). At 24 hours, none of the four hemodynamic parameters remained predictive of RVF. Neither RVSWI nor mPAP:MAP were predictive at any time point.
PAPi demonstrated good discriminative ability (AUC 0.701), with an optimal threshold of 1.87 (sensitivity 58.9%, specificity 72.5%) (Figure 1).

CONCLUSIONS: PAPi was the most consistent and clinically applicable predictor of RVF, retaining significance from baseline through early Impella 5.5 support before diminishing by postoperative day 1. These findings parallel proposed PAPi thresholds in durable LVAD populations and highlight the value of calculating a pre-Impella PAPi to identify patients who may potentially benefit from early RV mechanical support. Trending PAPi immediately after activation may guide fluid, inotrope, and Impella power (P)-level titration to mitigate perioperative RVF risk.

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Variable	No RVF (n=121)	RVF (n=77)	P-value
Age, years	61 (54-68)	63 (54-70)	0.815
Male sex, n (%)	95 (78.5)	57 (74.0)	0.493
BMI, kg/m²	26.5 (23.4-29.5)	27.2 (24.4-30.8)	0.154
Vasoactive-Inotropic Score	2.5 (0.0-7.0)	7.3 (0.0-16.8)	<0.001
Impella 5.5 Flow, L/min	4.0 (3.5-4.4)	4.1 (3.6-4.6)	0.262
Duration of Impella 5.5 Support, days	9.0 (6.0-16.0)	15.0 (8.0-27.0)	<0.001
Outcomes			
30-day Mortality, n (%)	8 (6.6)	21 (27.3)	<0.001
Renal Failure Requiring Dialysis, n (%)	11 (9.1)	29 (37.7)	<0.001



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SP 2 - Kiosk 7 - Poster 3
Transition to ECMO: The Hidden Burden of Early Inflammation and Organ Injury
Presenting Author: *Dandan Zhu, PhD*

Backgroud: Extracorporeal membrane oxygenation (ECMO) is initiated when sepsis-related cardiopulmonary failure progresses beyond conventional support yet early predictors of such deterioration remain unclear. In addition, although ECMO is thought to amplify systemic inflammation, in-vivo evidence is limited.

Method: Samples from 106 hospitalized patients with confirmed COVID-19 were categorized by sampling time, with T1 defined as the first serum collection obtained after hospital admission. A panel of infectious, inflammatory, coagulation, complement markers as well well serum surrogates of neurological and cardiovascular injury were assessed. Clinical status, morbidity, and mortality were extracted from electronic medical records.

Results: Among the 106 enrolled patients, 70 patients (AdmMin) were admitted without invasive ventilation (Min Group). Subsequently, 36 required intubation and 10 patient eventually required ECMO (3 at admission, 7 at later times. MV group: intubated patients without ECMO. Patient on ECMO were younger as compared to Min or MV group. Expectedly SOFA and APACHE score were higher in both ECMO or MV versus Min. Surprisingly, these scores were not different between ECMO and MV. Inflammatory profiling revealed that C-reactive protein and procalcitonin of MV and ECMO group were all elevated as compared to Min Group.WBC levels were similar between Min and MV, but both groups differed significantly from ECMO. However, most MV–ECMO comparisons were not statistically significant, indicating that ECMO progression occurred from a baseline of already heightened systemic inflammation rather than from an acute inflammatory escalation.Beyond classical inflammatory profiles, neurological biomarkers showed the strongest discriminatory signal. GCS scores differed across all three groups ($p< 0.001$) with highest in vs lowest in patients. ECMO patients also exhibited significantly elevated KLK6 and NCAM-1 levels compared with Min and MV, suggesting the presence of early neuro-injury or blood–brain-barrier vulnerability in those who progressed to ECMO. Clinically, patients requiring invasive ventilation or ECMO group experienced substantially worse outcomes than Min group, including increased stroke and deep-vein thrombosis at 6 months ($p< 0.01$, $p< 0.05$), higher short- and long-term mortality ($p< 0.001$), and prolonged ICU and hospital length of stay ($p< 0.001$). No statistically significant differences were observed among the three groups with respect to other infectious markers, non-specific inflammatory markers, coagulation markers, complement components, complement activation markers or cardiovascular injury markers.

Conclusions: Patients who ultimately required ECMO demonstrated a distinct early-admission phenotype characterized by severe physiological derangement, systemic inflammation, and early

neurological injury signals, even before ECMO initiation. Although inflammatory markers were elevated in both MV and ECMO groups, MV–ECMO differences were largely nonsignificant, indicating that ECMO progression emerged from a pre-existing state of profound multisystem stress rather than an acute inflammatory surge.

Keywords: COVID-19, ECMO, Immunological response, Neurological injury, Clinical Implications

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SP 2 - Kiosk 7 - Poster 4

Lactate clearance in cardiogenic shock – a tool to get the right therapy for the right patient?

Presenting Author: *Florian Falter, MD, FRCA, FFICM, PhD*

Introduction: A large number of patients admitted in cardiogenic shock after out of hospital cardiac arrest (OOHCA) do not survive to intensive care (ICU) or hospital discharge (1). About one third of patients who die have a neurological cause of death. Instituting extracorporeal mechanical circulatory support (MCS) is increasingly becoming a therapeutic option on patients with cardiogenic shock (2) ,but should be avoided in patients with poor neurological prognosis. Currently there are no reliable tools to assess patients' neurological condition at the time of arrival in the catheter lab. The association between elevated lactate levels and increased mortality has been shown repeatedly. However, no reliable timepoint or lactate plasma level at which mortality

increases have been defined (3). Complete lactate clearance has been shown to be a strong predictor of survival (4). We tested the hypothesis if lactate clearance is able to guide the timing of MCS initiation in patients in cardiogenic shock after OOHCA.

Methods: After IRB approval, this retrospective observational study included 77 patients admitted to cardiac catheterisation laboratories at Royal Papworth Hospital for primary percutaneous coronary intervention after OOHCA and with Society of Cardiovascular Angiography and Interventions (SCAI) class D or E cardiogenic shock. They had lactate measurements taken on admission, on arrival in ICU, and then at 6 hours, 12 hours and 18 hours after ICU admission to calculate clearance (defined as % reduction from baseline). None of the patients received MCS at any point. Patients were divided into those who survived to discharge and those who died during ICU admission.

Statistical analysis was performed using Medcalc with Kruskal-Wallis analysis.

Results: Out of 77 patients included, 45 (58.4%) died during ICU admission and

32 (41.6%) survived to ICU discharge; 29 (64.4%) died of a cardiac cause, 16

(35.6%) died of a neurological cause. Patients who arrested before arrival of

emergency services had the highest incidence of death by neurological causes

(Table 1).

Lactate values were significantly higher in non-survivors at all timepoints (Table 2), but there was overlap in lactate levels between groups, not allowing to define a cut-off level for mortality.

There was no statistically significant difference in lactate clearance between groups at ICU admission, 6 hours and 12 hours. However, this turned significant at 18 hours (Table 2).

Conclusion: This study shows that patients with SCAI class D or E CS who did not survive ICU admission had similar lactate clearance for the first 12 hours, which subsequently dropped off between 12 and 18 hours when compared to survivors. There appears to be an inflection point between 12 - 18 hours after ICU admission, where lactate clearance has the potential serve as a biomarker to demarcate survivors from non-survivors.

This may provide useful guidance when considering establishing patients on MCS. The 12-hour window provides some time to ensure that advanced mechanical therapy is appropriate.

Image Uploads

	Arrest location				
	No arrest	Before EMS arrival	After EMS arrival	Medical ward or ED	Catheter lab
Cardiac cause of death	14	6	2	1	6
Neuro cause of death	2	11	2	0	0

Table 1

	Dead (n= 45)	Alive (n= 32)	P value
Age	68 (61.00 - 72.00)	62 (54.00 - 67.28)	0.041
Lactate cath lab	4.5 (3.30 - 5.05)	2.55 (1.48 - 3.98)	0.016
Lactate ICU admission	3.8 (2.29 - 4.50)	1.6 (1.24 - 2.78)	0.0024
Lactate ICU 6h	3.6 (1.89 - 4.55)	1.6 (1.00 - 2.22)	0.0037
Lactate ICU 12h	2.6 (0.90 - 1.88)	1.3 (1.48 - 4.12)	0.0032
Lactate ICU 18h	2.5 (1.39 - 3.30)	1.2 (0.90 - 1.50)	0.0009
Lactate clearance ICU admission (%)	23.8 (9.66 - 36.61)	3.4 (-4.80 - 26.30)	0.077
Lactate clearance ICU 6h (%)	15.0 (-0.51 – 25.38)	20.6 (-11.65 - 28.04)	0.91
Lactate clearance ICU 12h (%)	30.6 (14.81 – 40.1)	33.3 (20.85 – 50.75)	0.54
Lactate clearance ICU 18h (%)	2.5 (1.39 – 3.3)	45.6 (31.82 - 52.6)	0.0001

Table 2

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SP 2 - Kiosk 8 - Poster 1

Ventricular Septal Defect Associated with a Perimembranous Ventricular Septal Aneurysm

Presenting Author: *Andrew Whang, MD*

Introduction Paragraph: A perimembranous ventricular septal aneurysm (pVSA) is a rare condition associated with other congenital abnormalities such as a ventricular septal defect (VSD), and can be mistaken for a sinus of Valsalva aneurysm (SOVA). Its formation may be related to a spontaneous closure of a congenital VSD by approximation of the septal leaflet of the tricuspid valve across the defect.1 A pVSA can cause arrythmias, obstruction of the right ventricular outflow tract, tricuspid insufficiency, aortic valve (AV) prolapse, and bacterial endocarditis.2 We present a case of an incidentally found VSD associated with a pVSA that was initially mistaken for a ruptured SOVA.

Brief Summary of the Clinical Case: A 75-year-old female with a history of hypertension presented with a complaint of palpitations, shortness of breath, and near syncope. Initial transthoracic echocardiography (TTE) findings were concerning for a severely calcified AV, severe aortic stenosis, and a tricuspid valve with an echogenic mass possibly attached to the septal leaflet and septum, later thought to be a SOVA. She was transferred to our institution for a higher level of care. Intraoperative transesophageal echo (TEE) revealed a VSA with a pVSD, without evidence of a SOVA. The patient subsequently underwent an AV replacement and VSD closure with a patch.

Discussion: The aneurysm was identified in the mid-esophageal four chamber (ME 4C) view, long axis (ME LAX) view, and RV inflow-outflow view. The aneurysm was visualized below the AV (Fig 1, 2, and 3).

The VSD with left to right shunt was interrogated using color and continuous wave Doppler. The peak velocity was 6.24 m/s (peak gradient 156 mmHg) and the calculated Qp/Qs was 2.4:1 (Fig 4, Vid 1 and 2).
Diastolic flow reversal in the descending aorta may be seen with a SOVA fistula. However, it was absent in this case as it is usually not associated with a VSD (Fig 5).

The presence of a pVSA was confirmed by the surgical team.

Postoperatively, the VSD was repaired. No residual flow across the defect was seen using color Doppler.

Impact of Echocardiographic findings

It can be difficult to differentiate SOVA and pVSA because they occur anatomically adjacent to each other at the level of aortic annulus. Echocardiography can help with differentiating the two and with surgical planning. In this case, initial TTE identified the aneurysm as a SOVA, which was later identified as a pVSA by intraoperative TEE.

Teaching Points of the Case

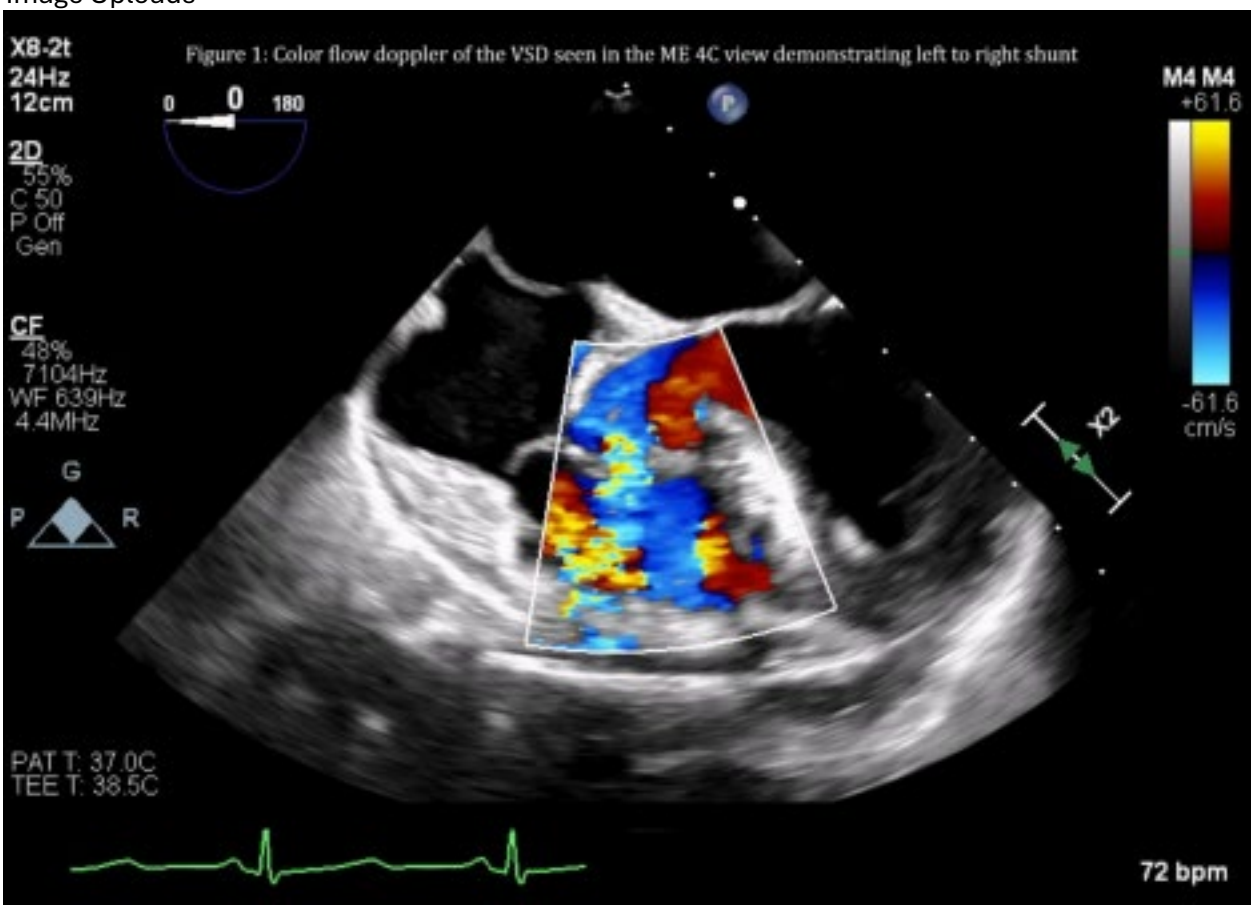
A pVSA is a rare congenital abnormality although it is possible to find it in older patients.4 They are typically found below the level of the aortic annulus.

Careful attention should be paid to exclude SOVA, which are located above the aortic annulus. SOVA with rupture typically have a more aggressive course depending on the fistula size and repair is recommended.

Depending on the size of the ruptured SOVA, spectral Doppler can show diastolic reversal of flow in the descending aorta in the absence of severe aortic regurgitation, as well as continuous high-velocity flow across the aneurysm with accentuation of the waveform in diastole. A VSD will typically have high velocity turbulent flow during systole and low or absent velocity in diastole. This can help differentiate a SOVA from a VSD.3

Given limited views with TTE, it can be difficult to differentiate whether the aneurysm is located above or below the annulus.5

Image Uploads



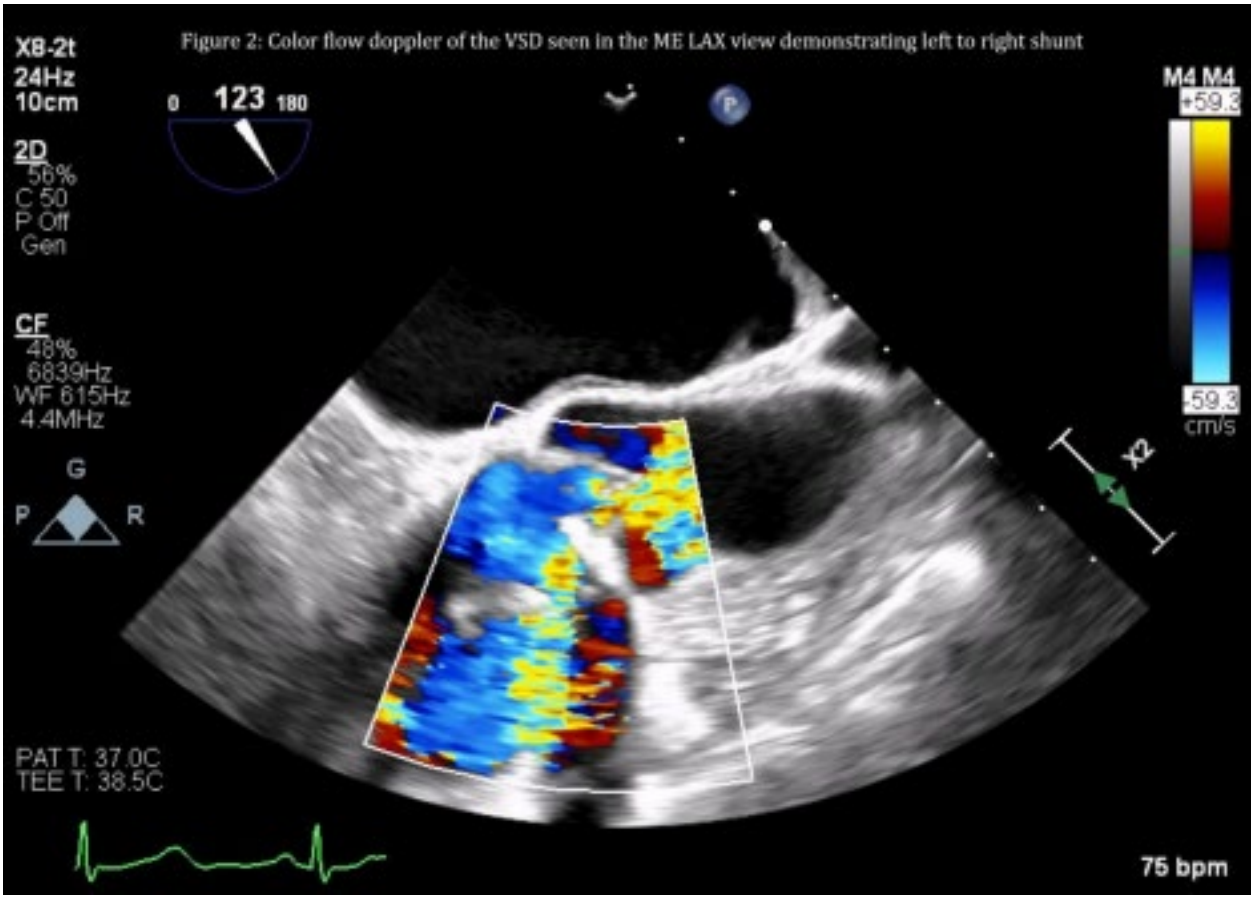


Figure 2: Color flow doppler of the VSD seen in the ME LAX view demonstrating left to right shunt

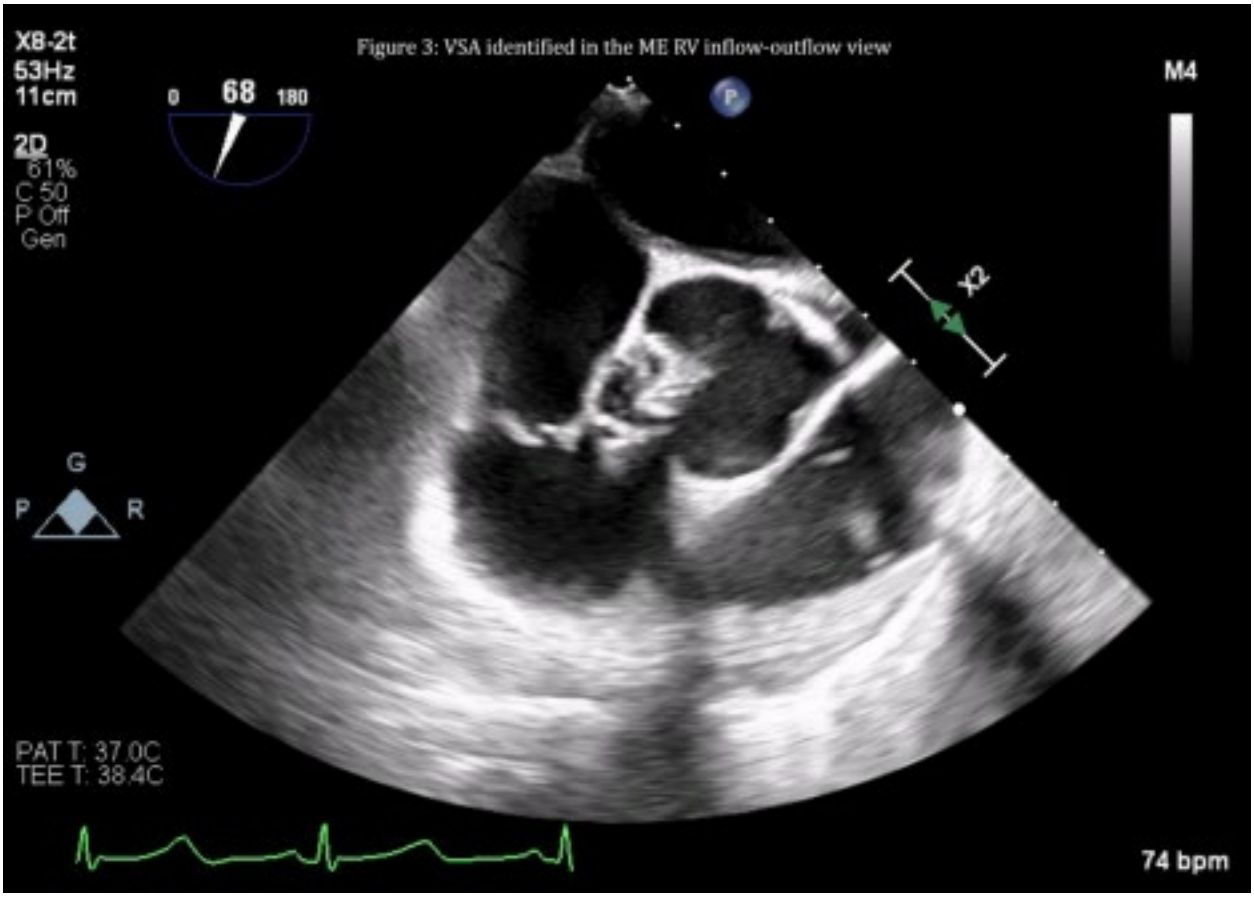
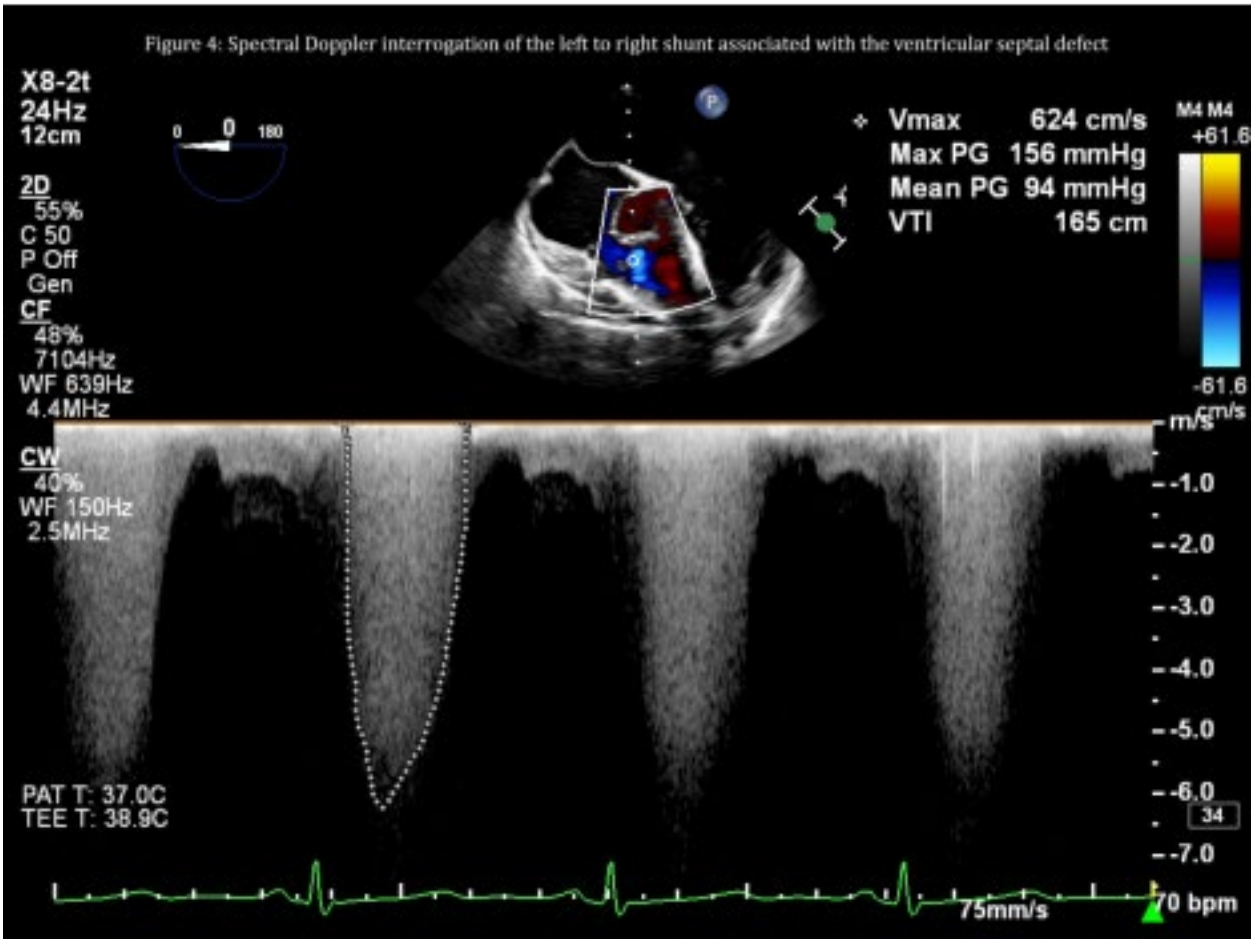


Figure 3: VSA identified in the ME RV inflow-outflow view

Figure 4: Spectral Doppler interrogation of the left to right shunt associated with the ventricular septal defect



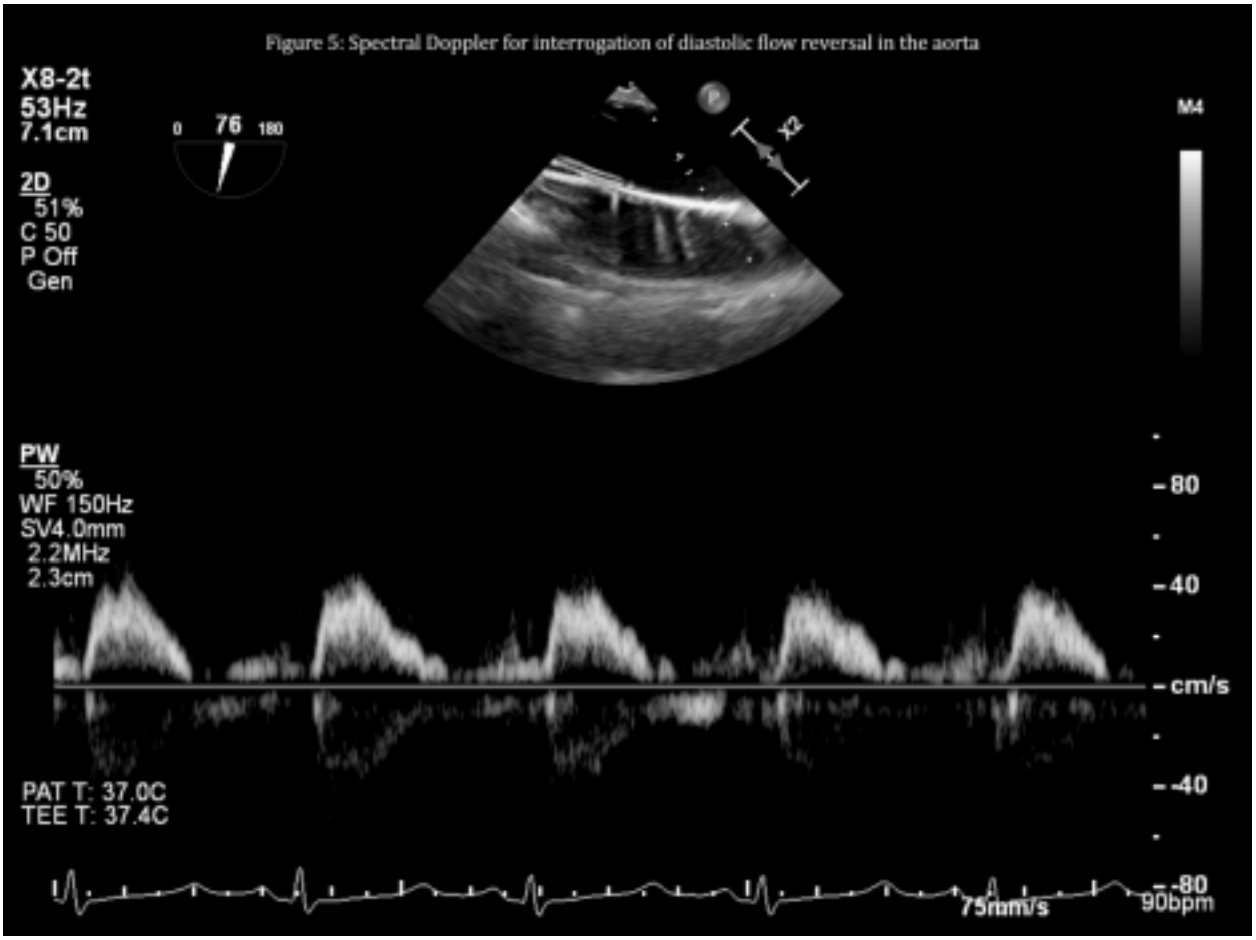


Figure 5: Spectral Doppler for interrogation of diastolic flow reversal in the aorta

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SP 2 - Kiosk 8 - Poster 2

Dynamic Evolution of Periannular Complications in Infective Endocarditis: A Case Highlighting the Role of Intraoperative TEE

Presenting Author: *Ricardo Sant Anna, MD*

Introduction Paragraph: Infective endocarditis is a dynamic condition in which TEE findings may evolve rapidly. This case illustrates how surgical planning must adapt to newly identified periannular complications and highlights the essential role of detailed intraoperative TEE in guiding management.

Brief Summary of the Clinical Case: A 53 yo woman with methicillin-sensitive S aureus AV infective endocarditis. TTE revealed severe aortic insufficiency with large aortic valve vegetations. Moderate MR was also noted, with destruction of the central fibrous trigones and additionally, a complex periaortic abscess was confirmed on TEE. She was set to undergo AV and root replacement plus MV repair. Intraoperative TEE found an unrecognized aortic root pseudoaneurysm, an aorta–left atrial fistula, and significant erosion of the anterior mitral leaflet. The surgical plan was revised, and the patient underwent aortic root replacement with a 24-mm Artivion aortic homograft which was sutured on the base of the anterior mitral valve leaflet (complex mitral valve repair), and reconstruction of the central fibrous trigones.

Discussion: Aorto-cavitary fistulas, abscesses, and pseudoaneurysms are key periannular complications of infective endocarditis, each with distinct TEE features. In this case, a preoperative TEE demonstrated an abscess as echolucent or echodense regions near the valve annulus without color Doppler flow. Intraoperative TEE revealed a newly formed aorto-cavitary fistula, identified by a continuous, high-velocity, turbulent Doppler jet from the aorta into the receiving chamber, typically arising when an abscess ruptures under systemic pressure.
Fistulas most frequently form between the aortic root and right-sided chambers, but extensive periannular destruction can produce fewer common connections. In this patient, infection extended beyond the aortic annulus, causing a complex abscess, erosion of the central fibrous trigones, an aortic root pseudoaneurysm, and a rare aorta–left atrial fistula. Posterior extension toward the mitral–aortic intervalvular fibrosa explains the left-sided fistula. Early recognition of fistulas is critical, as it directly influences surgical strategy.
3D TEE provides superior anatomical detail and spatial orientation compared with 2D imaging,

enabling accurate evaluation of vegetations, abscesses, pseudoaneurysms, and fistulas. Despite lower temporal and lateral resolution than 2D TEE, 3D imaging improves detection of periannular complications and enhances communication between imaging specialists and surgeons, supporting optimized surgical outcomes in complex or unexpected cases.

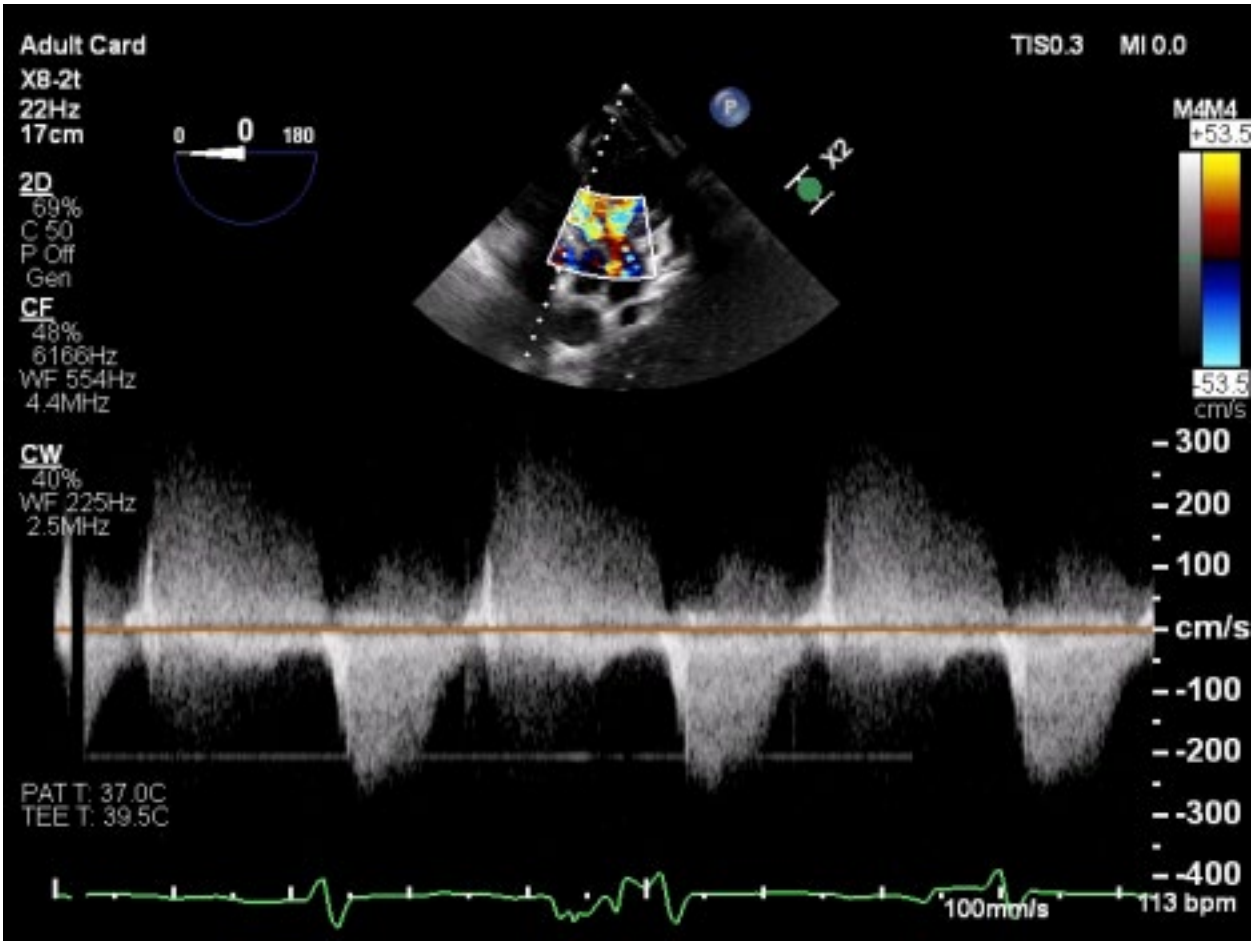
Impact of Echocardiographic findings: Patients with infective endocarditis may present with a wide range of complications. Given the dynamic nature of the disease, intraoperative findings can significantly influence surgical decision-making. In this case, we observed how an area previously identified as a perivalvular abscess had evolved into more than one type of fistulous communication. In all patients with endocarditis, it is essential to remain vigilant for newly developing complications.

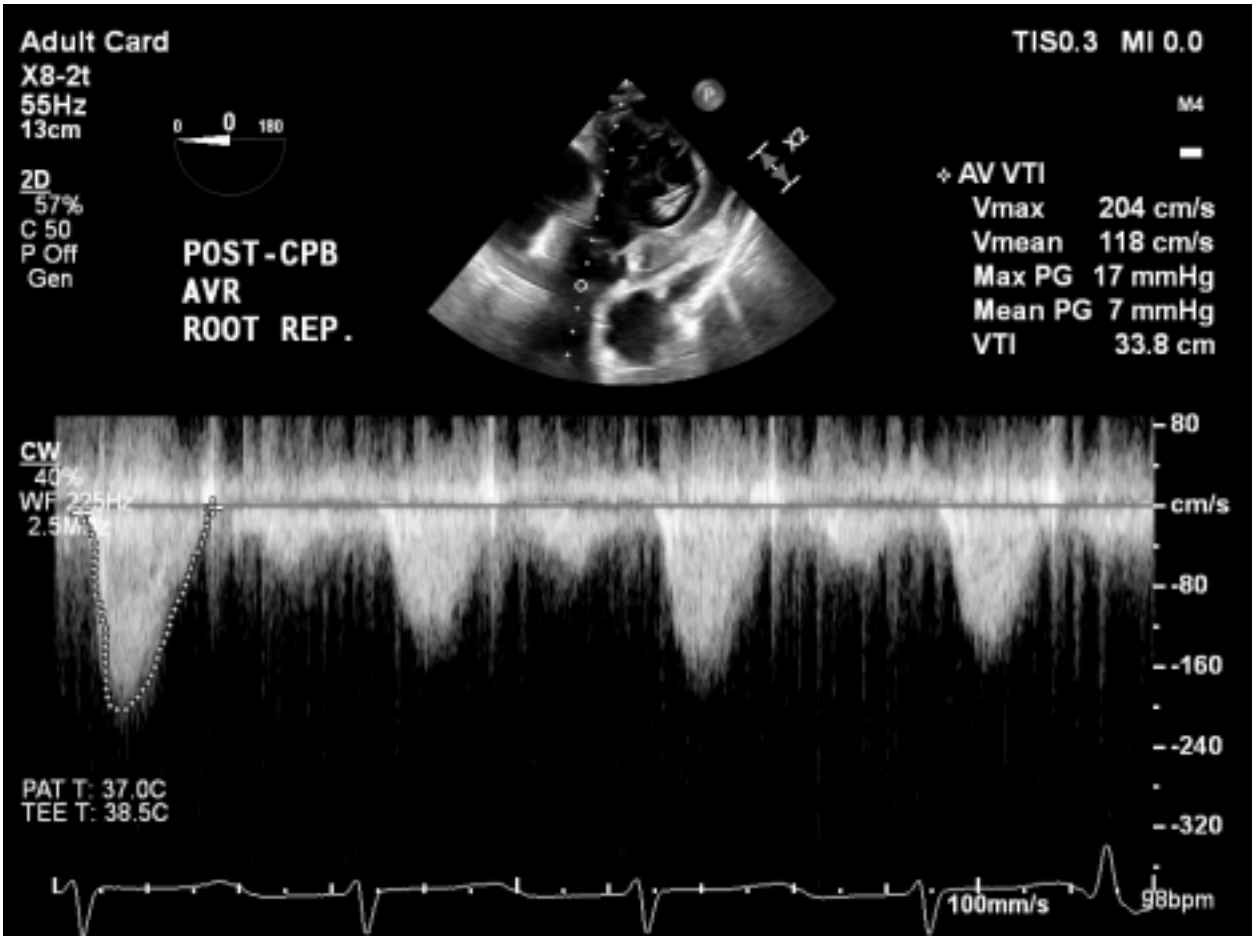
Teaching Points of the Case
Systematic evaluation of all potential complications associated with infective endocarditis.

Identification of the most common anatomical locations for intracardiac fistulas. Role of 3D

echocardiography in the assessment of periannular complications.

Image Uploads


Adult Card
X8-2t
22Hz
17cm
0
0
180
2D
69%
C 50
P Off
Gen
CF
48%
6166Hz
WF 554Hz
4.4MHz
CW
40%
WF 225Hz
2.5MHz
TIS0.3
MI 0.0
M4M4
+53.5
-53.5
cm/s
300
200
100
cm/s
-100
-200
-300
-400
PAT T: 37.0C
TEE T: 39.5C
100mm/s
113 bpm



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SP 2 - Kiosk 8 - Poster 3

Transesophageal Echocardiographic Detection of Thoracic Stent Graft Narrowing After Aortic Arch Replacement

Presenting Author: *Natalie Koons, DO*

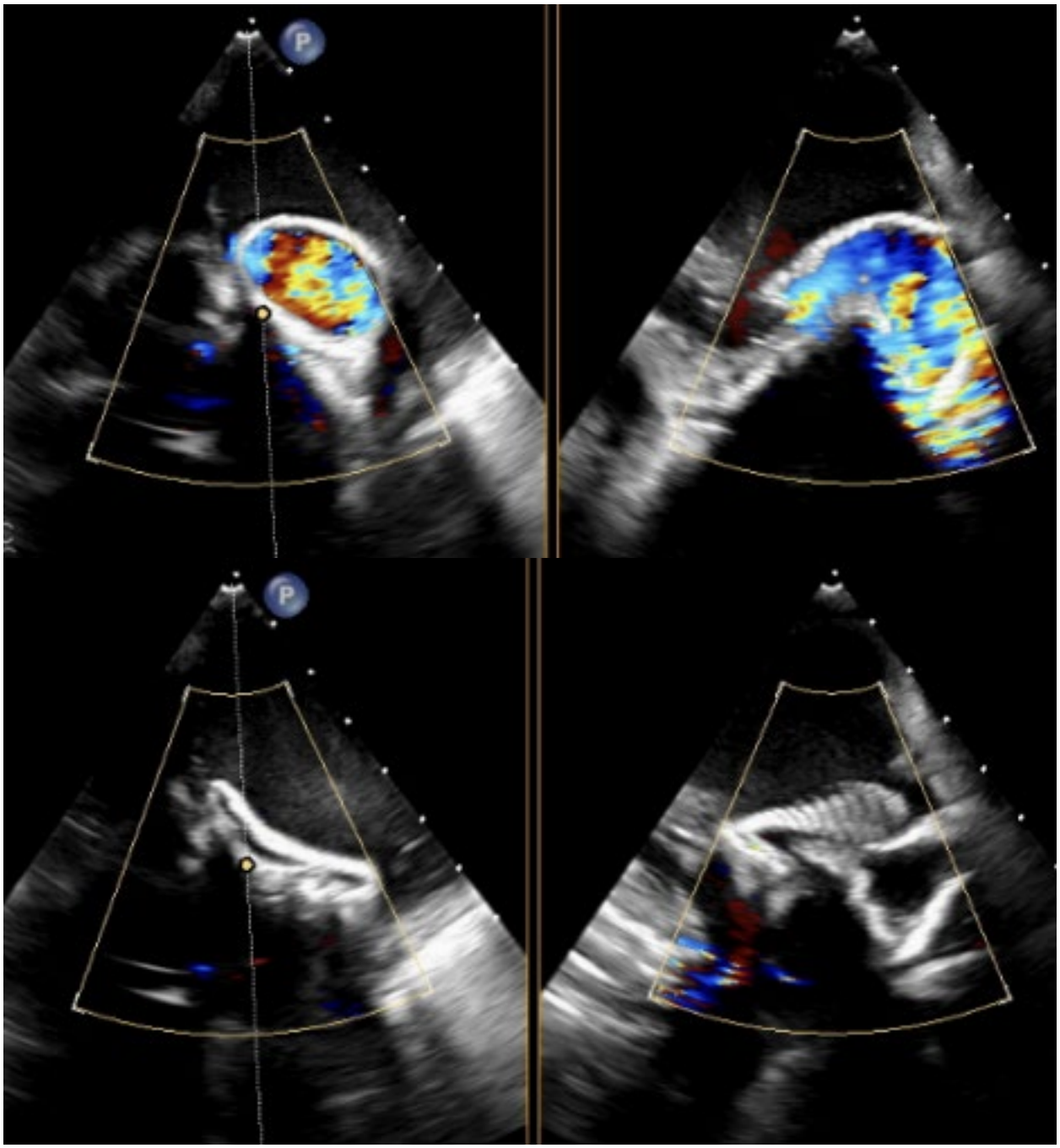
Introduction: Thoracic endovascular aortic repair (TEVAR) has become the standard surgical intervention for blunt aortic trauma due to its immediacy and minimally invasive technique.1,2 However, these procedures are not without potential complications, including endoleak, cerebrovascular ischemia, and infections, especially in patients with concomitant trauma.3 We present the complex management of an infected TEVAR with a type Ia endoleak with transesophageal echocardiogram (TEE) evidence of graft kinking after aortic arch replacement.

Case Presentation: A 31-year-old male with a history of substance use disorder, and suicide attempt with multiple traumatic injuries, intracranial hemorrhage, and traumatic grade 3 descending thoracic aortic injury status-post TEVAR, presented for a total aortic arch replacement for an expanding aneurysmal sac caused by a type Ia endoleak. General anesthesia was induced with standard American Society of Anesthesiologists (ASA) monitoring, a central venous catheter and TEE probe were placed, as in standard procedures for sternotomy with cardiopulmonary bypass (CPB). Cerebral oximetry and neuroprotective modalities were used to complete a zone 2 arch replacement with circulatory arrest. After successfully weaning from CPB, there was disruption of the proximal anastomosis requiring reinitiation of CPB. A graft-to-graft anastomosis to the arch was performed and the patient was, again, weaned from CPB. At this time, there was a significant difference in the femoral and brachial arterial pressures that appeared to be caused by the positioning of the aortic graft. TEE showed flow acceleration noted at the level of the elephant trunk in the proximal descending thoracic aorta with a peak gradient of 17 mmHg (Image 1). TEE confirmed significant narrowing at the thoracic stent graft (Image 2) that was relieved with anterior positioning of the aorta and worsened with chest closure. The chest was left open, packed with laps to elevate the aorta, and the patient was transferred to the cardiovascular intensive care unit (CVICU). Upon presentation to the CVICU, right brachial (102/67 (79)) and left femoral pressures (70/61 (65)) continued to be unequal. Due to the obstruction along the aortic graft, the patient was taken back to the operating room for a proximal extension prosthesis with additional TEVAR stent and balloon expansion resulting in resolution of flow acceleration in the proximal descending thoracic aorta. The chest was closed and the patient was stably transferred back to the CVICU. After an 8-week hospitalization, the patient was discharged to a skilled nursing facility for further rehabilitation.

Discussion: Graft kinking following aortic procedures is an unfavorable complication that has been well documented.4,5 Complex anatomy, thrombus formation, and graft diameter discrepancy can all be causes of graft kinking. Intraoperative TEE identification of blood flow gradients within a replaced aortic graft is important to ensure the absence of turbulent flow and graft kinking that could ultimately cause hemodynamic compromise.

Conclusion: Our case illustrates the extensive management of a patient who underwent an aortic arch replacement for an infected TEVAR with a type Ia endoleak with subsequent graft kinking.

Image Uploads



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SP 2 - Kiosk 8 - Poster 4

HemodynamicEffects of Bolus vs Infusion Anesthesia During Transesophageal Echocardiography in Patients with Severe Left Ventricular Systolic Dysfunction

Presenting Author: *Renee Cress*

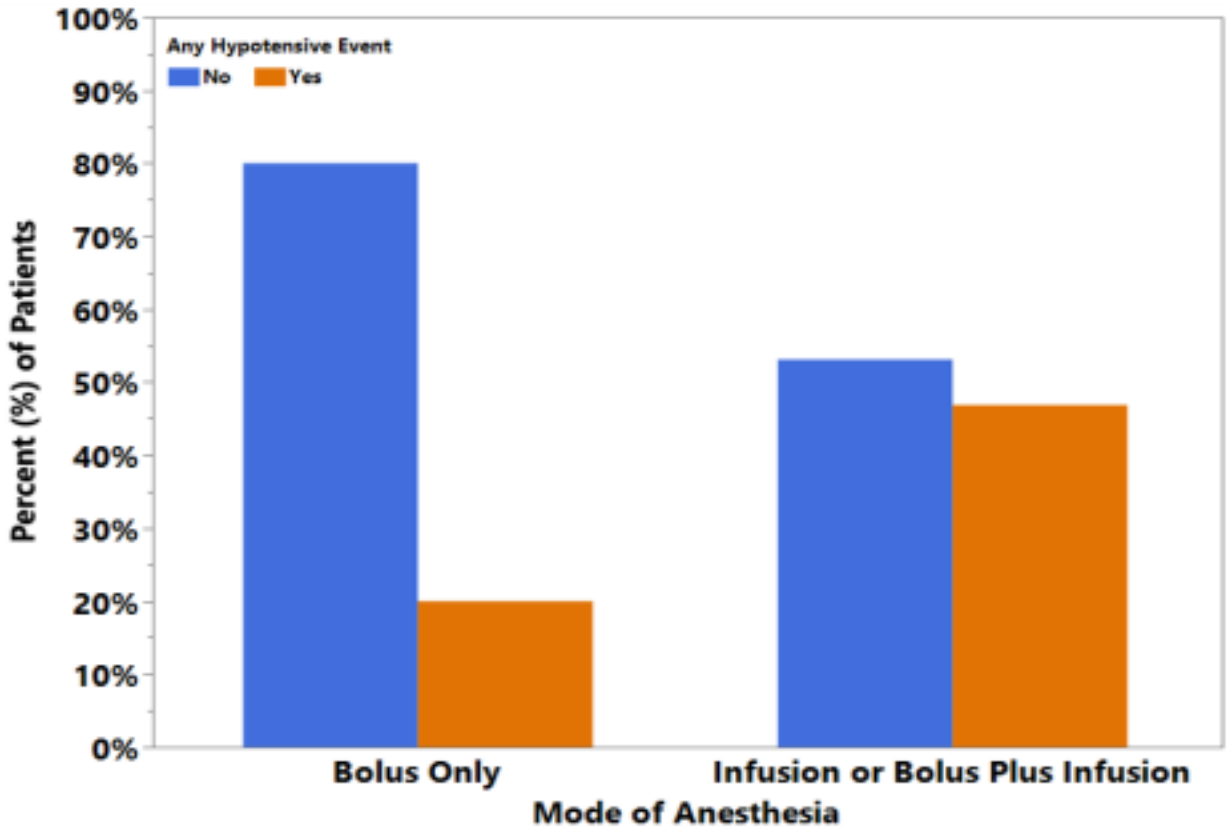
Background/Introduction: Transesophageal echocardiography (TEE) is a semi-invasive diagnostic tool frequently performed under moderate sedation to evaluate cardiac structure and function in high-risk patients (1). Although generally safe, use of anesthetics during TEE poses an increased risk in patients with severely reduced left ventricular ejection fraction (LVEF < 30%), including arrhythmias, respiratory compromise, and hemodynamic instability (1, 2, 3). This study assesses trends in hemodynamics associated with different anesthetic management strategies during TEE in patients with LVEF < 30%.

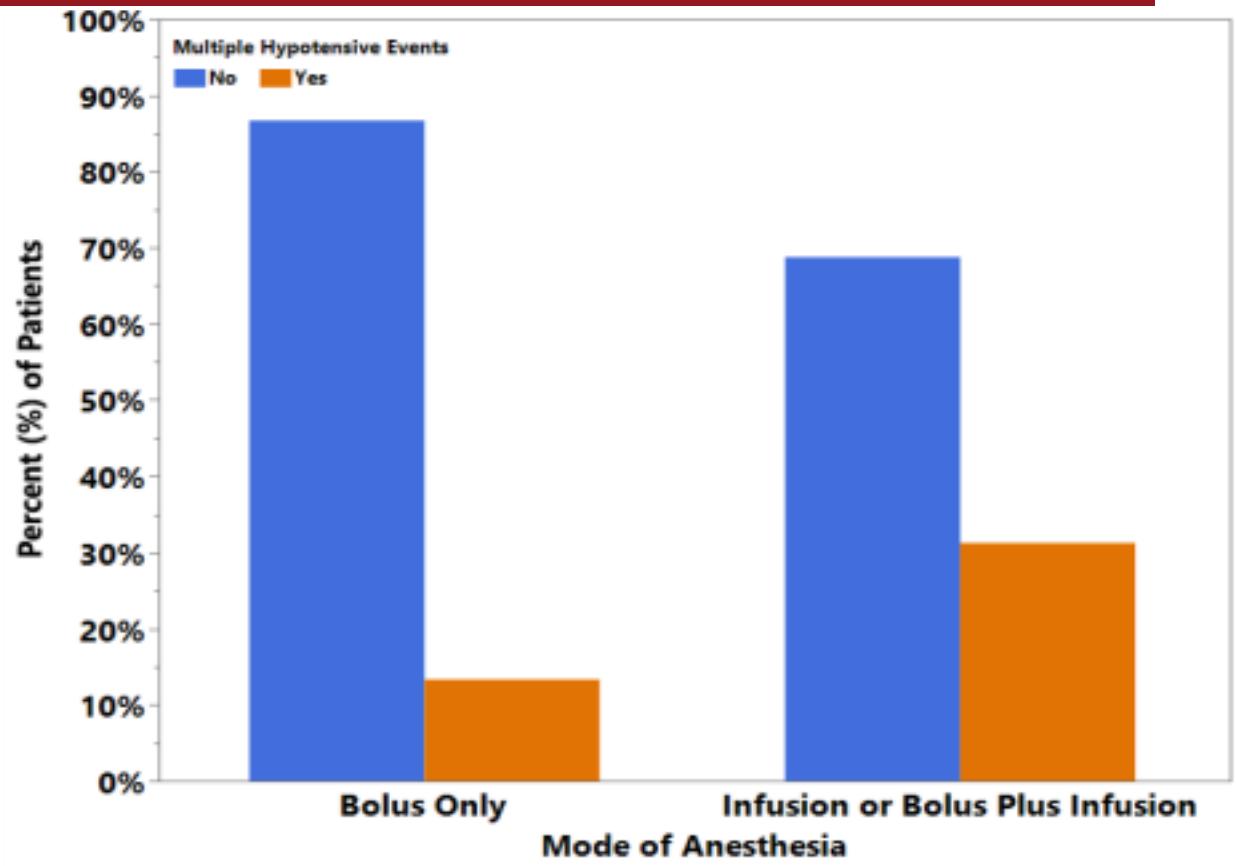
Methods: We conducted a single-institution retrospective observational study of adult patients (≥18 years) with LVEF < 30% who underwent anesthetic management for TEE between January 1, 2022, and December 31, 2024. Of 111 eligible encounters, 32 were excluded due to procedures performed without an anesthetist (eg, bedside studies), concurrent ablation procedures, or missing records, leaving 79 encounters for analysis. The variables for this analysis included anesthetic technique (medications used, mode of administration, total anesthetic dose), intraoperative vitals, and postoperative complications. Patients were categorized into three groups: boluses only, infusions only, and boluses + infusions, with latter two groups combined for analysis. Hemodynamic instability was defined as systolic blood pressure < 90 mmHg or mean arterial pressure < 50 mmHg. Descriptive statistics and comparative analyses (Fisher's exact test, Z-test, and chi-square test) were performed.

Results/Discussion: Among the 79 encounters, 15 received boluses only, 14 received infusions only, and 50 received both. Hypotension occurred most often in the bolus + infusion group (48%), followed by the infusion-only group (43%) and the bolus-only group (20%; P < 0.142). In two-group analysis, patients managed with boluses alone had fewer hypotensive episodes (20% vs. 47%; P = 0.058) and fewer multiple hypotensive episodes (13% vs. 33%; P = 0.128), compared with those receiving any type of infusions. While not statistically significant—likely due to limited sample size—these trends may be clinically relevant in practice. Of note, patients receiving infusions had a higher median total propofol dose than those receiving boluses only (247 mg vs. 80 mg; P = 0.0132), which likely contributed to the increased incidence of hypotension observed in the infusion groups.

Conclusion: Among patients with LVEF < 30% undergoing TEE, bolus-only anesthetic management was associated with non-significantly fewer hypotensive events and lower total propofol requirements compared with infusion-based techniques. Expanding the dataset to earlier years may improve the power and further clarify the observation of these associations.

Image Uploads





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SP 2 - Kiosk 8 - Poster 5
A Distance in Dispute: Concordance of C-Sept Distance Across TEE Planes
Presenting Author: *Ahmed Yousef, MD*

Background: Systolic anterior motion (SAM) of the mitral valve remains a major contributor to dynamic left ventricular outflow tract obstruction, making reliable preemptive assessment critical. Seminal studies in echocardiography have established the coaptation-septal distance (C-sept) as a key predictor of SAM—noting that distances less than 2.5 cm at the onset of systole increase risk. Maslow et al. originally described the measurement in the TEE 5-chamber view in 1999.1 In 2014, Varghese et al. extended this analysis to the Long-axis view.2 However, it remains unknown if C-sept values vary across these imaging planes, reflecting differences in leaflet orientation and septal interrogation. To account for this potential discrepancy, we explore the role of measurement of C-sept in 3D imaging—particularly multiplane reconstruction (MPR) aligned through the center of A2–P2—and its agreement and potential interchangeability with conventional measurements of C-sept in 5-chamber and Long-axis.

Methods: Twenty adult cardiac surgical patients undergoing intraoperative TEE were included. For each patient, C-sept was measured at the onset of systole using three modalities: mid-esophageal 5-chamber, Long-axis, and 3D MPR aligned orthogonally through the center of A2–P2. All measurements were performed by a single observer. Agreement between modalities was evaluated using linear regression and Bland–Altman analysis.

Results: C-sept values obtained from 5-chamber, Long-axis, and 3D MPR views were similar in each patient with no systematic over- or under-estimation. Correlation analysis demonstrated strong linear relationships between all modality pairs with the highest concordance observed between Long-axis and 3D MPR. Agreement between 5-chamber and 3D MPR was also high, though associated with slightly wider dispersion. Bland–Altman analysis showed small mean biases for each comparison (ranging from -1.6 to 1.25 mm) and 95% limits of agreement that remained narrow and clinically acceptable (Figure 1). No patient demonstrated outlier measurements that suggested modality-specific error.

Discussion: Our finding of strong agreement among 5-chamber, Long-axis, and 3D MPR measurements of C-sept suggests that 3D MPR aligned through A2–P2 may provide a reliable alternative to conventional 2D C-sept assessment. Bland-Altman analysis confirms minimal mean bias and acceptably narrow limits of agreement for each modality pair, supporting interchangeability of measurements. Accurate estimation of septal distance is critical for SAM risk stratification during mitral valve repair. Published models incorporating C-sept have shown strong discriminative performance for SAM.2 Further novel methods for assessment of SAM risk have been published including one validating anterior leaflet tip-septal distance as a predictor.3 Importantly, 3D-derived measurements provide a consistent anatomic reference helping to reduce imager variability. In the case of C-sept, 3D MPR enforces alignment through the midline coaptation point, reducing possible plane-dependent discordance seen in 2D views and accounting for asymmetric leaflet geometry or challenging imaging windows. Further studies should explore 3D-derived C-sept distances in larger cohorts and in patients with SAM physiology.

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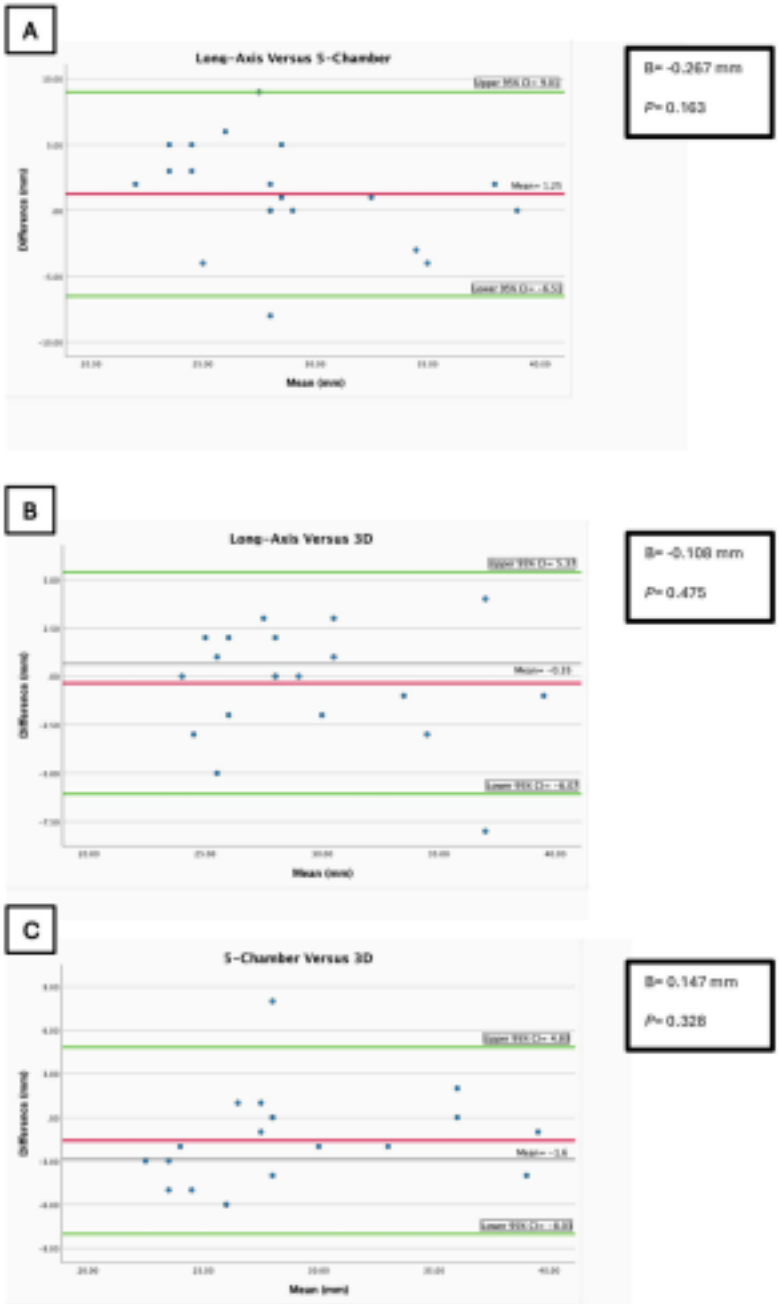


Figure 1. Bland–Altman Analysis of C-Sept Distance Agreement
Bland–Altman plots comparing C-sept distance between (A) Long-axis vs 5-chamber, (B) Long-axis vs 3D MPR, and (C) 5-chamber vs 3D MPR. Mean bias and 95% limits of agreement indicate minimal systematic error and acceptable variability between measurement techniques. Linear regression unstandardized coefficients (B) and correlation t-tests demonstrate strong concordance between modalities.

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SP 2 - Kiosk 8 - Poster 6

Minimization of Systolic Anterior Motion in Robotic Mitral Valve Repair

Presenting Author: *John Yin, MD*

Background: Systolic anterior motion (SAM) is a complication of mitral valve repair (MVR) with an incidence rate of 4-13%1,2. Various transesophageal echocardiography (TEE) measurements have been identified as risk factors in the prediction of SAM3. Most of these parameters were studied on patients undergoing a conventional MVR approach and have not been examined in the context of robotic MVR. We hypothesize that patients who develop SAM after robotic MVR have cardiac structural characteristics that can be predicted by TEE.

Methods: Data was collected on 197 patients who underwent isolated robotic MVR at our institution from September 2021 to August 2025. TEE acquired structural measurements of previously published predictors of SAM were discussed with the surgical team prior to the start of the case. These risk factors included position of the coaptation point (AL/PL ratio), anterior and posterior leaflet heights (AML, PML), aorto-mitral angle, coaptation to basal septum distance (C-sept) and basal septum and ventricular sizes. For simplicity, low risk consisted of 0-3 risk factors and high risk consisted of 4-7 risk factors. Surgical outcomes were compared between low and high-risk groups. Post-hoc TEE measurements were also compared between patients who developed SAM and patients who did not. Univariable association between various TEE measurements and SAM were evaluated using Firth logistic regression.

Results: The incidence of SAM in 197 patients who underwent robotic MVR was 3.5% (n = 7/197), as shown in Table 1. Of those patients who developed SAM, 2 had valve replacements and 5 were managed with volume administration, esmolol, and afterload augmentation for treatment. Univariate firth logistic regression found associations with the development of SAM: aorto-mitral angle (OR 0.95, CI 0.9 - 0.99), C-sept distance (OR 0.78, CI 0.64-0.93) and left ventricular end systolic diameter (LVESD) (0.72, CI 0.57-0.87). Furthermore, patients with higher documented SAM risk factors were also associated with the likelihood of developing SAM (OR 2.35, 1.27-4.83) (Figure 1). Predictably, there were group differences in risk factors in the high (N = 89) and low (N = 108) SAM risk patients that surgeons were aware of intraoperatively (Table 2). However, there were no significant differences in surgical complexity as measured by both Fujita Complexity Score4 and Mitral Valve Complexity score5. While not statistically significant, the high SAM risk group trended towards higher mitral valve repair time with a larger annuloplasty band compared to commissural diameter. The high SAM risk group was also observed to contain 100% of patients with SAM.

Conclusion: Larger C-sept, aorto-mitral angle and LVESD is associated with a reduction in SAM risk in robotic MVR. A major limitation for predictive validity in this study is the low SAM outcome. However, careful discussion of patients at high risk of SAM may allow robotic MVR to be completed with an overall lower SAM incidence than reported in literature, especially using a simple risk stratification strategy. Surgical adjustments preventing SAM such as placement of larger annuloplasty bands or having more resection length may be informed by risk stratification.

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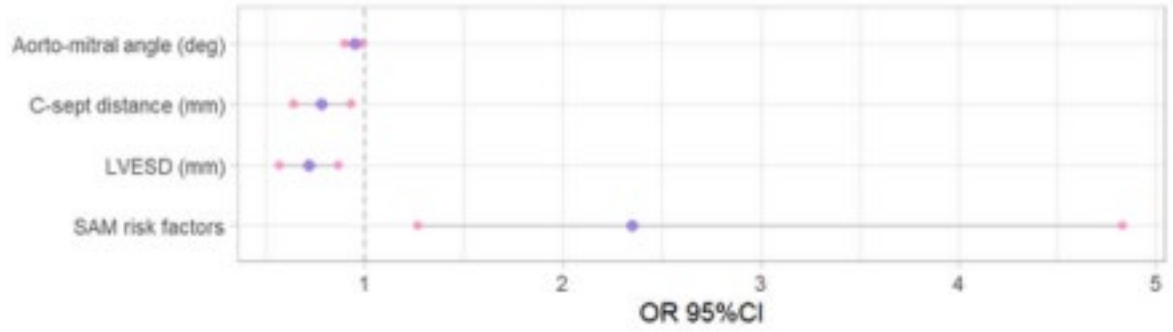


Figure 1. Univariable Associations with SAM. Odds ratios (ORs) and 95% confidence intervals (CIs) from univariable Firth logistic regression model.

Table 1. Clinical Characteristics of Patients with SAM vs No SAM.

SAM Outcome		
Characteristics	**No,** N = 190	**Yes,** N = 7
Age, Median (IQR)	62 (54 to 69)	61 (59 to 69)
Sex (Male), n (%)	127 (67)	2 (29)
Pre-op LVEF%, Median (IQR)	62 (58 to 63)	60 (58 to 63)
AL/PL ratio, Median (IQR)	1.5 (1.2 to 1.8)	1.5 (1.1 to 1.6)
Aorto-mitral angle (deg), Median (IQR)	120 (113 to 127)	108 (101 to 115)
C-sept distance (mm), Median (IQR)	27 (25 to 31)	24 (19 to 26)
Basal septal diameter (mm), Median (IQR)	14 (12 to 16)	13 (10 to 16)
LVESD (mm), Median (IQR)	31 (28 to 34)	24 (22 to 27)
LVEDD (mm), Median (IQR)	51 (46 to 57)	50 (43 to 52)
AML (mm), Median (IQR)	28 (25 to 32)	26 (25to 29)
PML (mm), Median (IQR)	20 (16 to 23)	19 (17 to 24)
Bileaflet disease (Yes), n (%)	41 (22)	2 (29)
Instrument Active Time (min), Median (IQR)	99 (81 to 112)	117 (95 to 121)

Table 2. Comparison of Pre-Stratified SAM Risk Groups.

	Pre-op SAM Risk Groups		
Characteristics	**Low, N = 108**	**High, N = 89**	**P-value[1]**
Age, Median (IQR)	62 (53 to 71)	63 (56 to 69)	0.83
Sex (Male), n (%)	70 (65)	59 (66)	0.83
Pre-op LVEF%, Median (IQR)	60 (58 to 63)	63 (58 to 63)	0.81
AL/PL ratio, Median (IQR)	1.6 (1.3 to 1.8)	1.3 (1.1 to 1.6)	<0.001*
Aorto-mitral angle (deg), Median (IQR)	122 (117 to 132)	115 (108 to 120)	<0.001*
C-sept distance (mm), Median (IQR)	29 (26 to 32)	26 (23 to 29)	<0.001*
Basal septal diameter (mm), Median (IQR)	13 (10 to 14)	15 (13 to 17)	<0.001*
LVESD (mm), Median (IQR)	31 (28 to 36)	29 (27 to 34)	<0.005*
LVEDD (mm), Median (IQR)	52 (48 to 57)	48 (45 to 56)	<0.041*
AML (mm), Median (IQR)	28 (24 to 34)	28 (25 to 32)	0.529
PML (mm), Median (IQR)	18 (15 to 21)	21 (18 to 25)	<0.001*
Bileaflet disease (Yes), n (%)	26 (24)	17 (19)	0.40
Commissure-commissure (CC) diameter (mm), Median (IQR)	43 (39 to 48)	42 (39 to 47)	0.43
Annuloplasty size (mm), Median (IQR)	32 (28 to 34)	32 (30 to 34)	0.54
Ratio of CC/Implant size, Median (IQR)	1.36 (1.25 to 1.47)	1.31 (1.21 to 1.44)	0.10*
Fujita Complexity Score, Median (IQR)	1 (1 to 2)	1 (1 to 2)	0.76
Mitral Valve Complexity Score, Median (IQR)	1 (1 to 4)	1 (1 to 3)	0.76
Technique Score, Median (IQR)	2 (2 to 3)	3 (2 to 3)	0.62
Time of valve repair (min), Median (IQR)	52 (39 to 71)	61 (46 to 68)	0.07*
SAM post MVR, n (%)	0	7 (100%)	0.003*

1Mann-Whitney test; Pearson's Chi-squared; Fisher's Exact test

Risks included: systolic anterior motion (SAM) risk factors described as aorto-mitral angle < 120 degrees, anterior height/posterior height ratio < 1.3, anterior mitral length > 20mm, posterior mitral length > 15mm, coaptation-septum distance < 25mm, basal septum > 15mm, left ventricular end diastolic diameter < 45mm.

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SP 2 - Kiosk 9 - Poster 1

Transformer Based Patient Response Modeling Across All Perioperative Phases for Cardiac Surgery Associated Acute Kidney Injury Management

Presenting Author: *Yao Ji, PhD*

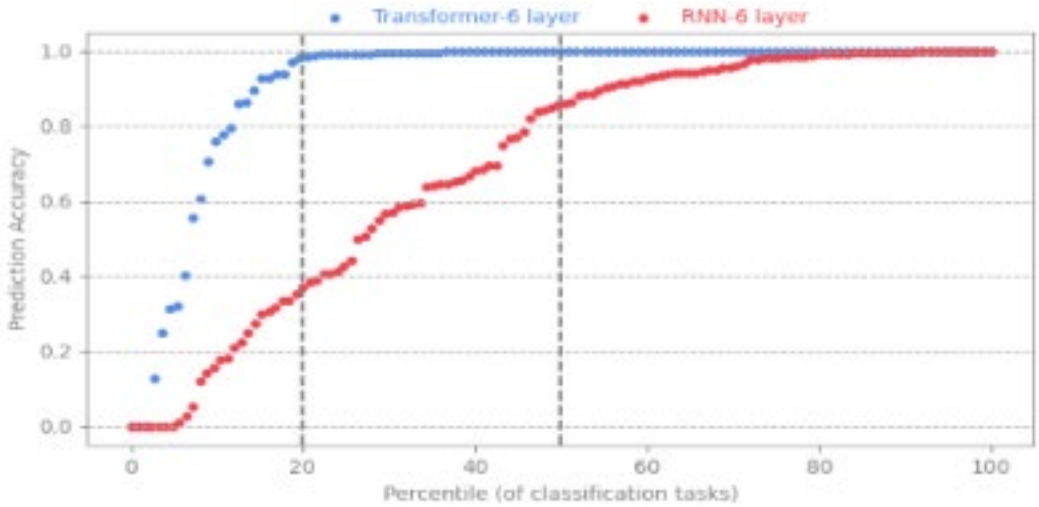
Background: Cardiac surgery associated with acute kidney injury (AKI) significantly affects surgical outcomes resulting in higher all-cause mortality, dialysis, and persistent renal dysfunction rates. AKI management is complex and inherently sequential: each intervention alters immediate physiology and the downstream recovery trajectory. Reinforcement learning (RL) optimizes sequential decisions under uncertainty. We aim to develop a transformer-based patient response model for AKI management. Methods: De-identified adult cardiac surgery cases from the Society of Thoracic Surgeons (STS) Adult Cardiac Surgery Database1, including a total of 108,258 patients from 2014 to 2024 were analyzed on secure high-performance computing (HPC) servers maintained by the University of Louisville. For each patient, we construct a four-stage trajectory consisting of preoperative, intraoperative, postoperative, and early post-discharge follow-up stages. At each decision step t=0,1,2,3, the state $s_t \in S$ captures the patient's current clinical status (e.g. demographics, vital signs). The action $a_t \in A$ represents possible clinical interventions (e.g., procedure type, medications). The system then transits to a new state s_{t+1} according to the transition kernel $P(s_{t+1}|s_t,a_t,s_{t-1},a_{t-1},\ldots,s_0,a_0)$. We trained several layer of decoder-only transformer models and recurrent neural network (RNN) baselines to predict the patients' next state across multiple clinical variables giving all the previous state action pair $(s_t,a_t,s_{t-1},a_{t-1},\ldots,s_0,a_0)$, thereby learning the patient state transition kernel P for downstream RL-based policy optimization. For 6-layer transformer, the training time is 150 hours on one Nvidia® A30 GPU. The inference time is within seconds.

Results: Transformers consistently outperformed RNNs on attribute prediction across all perioperative phases. Target attributes totaled 126: 18 intraoperative, 89 postoperative, and 19 post-discharge. For evaluation, its accuracy is defined as the proportion of patients with correctly predicted states divided by the total test patients. We observe 1) for 80% of all126 attributes prediction tasks, the 6-layer transformer reaches 97% accuracy versus only 36% for the 6-layer

RNN. 2) For the 6-layer transformer (or 6-layer RNN), the average accuracy when using the preoperative stage to predict intraoperative attributes is 94% (RNN 72%), when using the preoperative and intraoperative stage information to predict postoperative attributes is 90% (RNN 66%), and when using all three previous stages to predict post-discharge attributes is 96% (RNN 83%). These results show that the transformer achieves near-perfect accuracy on a large fraction of tasks and substantially outperforms the RNN model, underscoring the advantage of transformer architectures for RL-oriented clinical modeling.

Conclusion: A transformer-based model encoding preoperative, intraoperative, postoperative, and post-discharge information achieved reliable next-state prediction supporting realistic simulation of alternative intervention sequences. These findings represent an important step toward developing accurate clinical decision support systems to reduce AKI incidence following cardiac surgery.

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SP 2 - Kiosk 9 - Poster 2
Metallomics and Metabolomics in Pulmonary Arterial Hypertension
Presenting Author: *Emma Huang*

Background: Pulmonary arterial hypertension (PAH) is a disease of the pulmonary vasculature with increasing prevalence worldwide and no effective therapy that improves mortality. The etiology of PAH is complex, involving genetic, epigenetic, and environmental factors that cause inflammation, oxidative stress, and metabolic dysregulation. Environmental metal exposure has been linked to increased cardiovascular disease incidence. We hypothesized that metal exposure and associated metabolic dysregulations may induce or exacerbate PAH pathogenesis.

Methods: Plasma samples from 498 idiopathic PAH (IPAH) patients, 100 scleroderma-associated PAH (APAH) patients from the PAH Biobank, and 88 control subjects underwent measurement of 27 metal concentrations using inductively coupled plasma mass spectrometry (ICP-MS). Metals measured included Be, Na, Mg, Al, K, Ca, V, Cr, Mn, Fe, Ni, Cu, Zn, As, Se, Mo, Ag, Cd, Sb, Ba, Ti, Pb, Th, U, Co, Pt, and W. Additionally, serum samples from healthy controls (n=51), scleroderma associated PAH (APAH, n=52), and IPAH (n=51) were analyzed for polar metabolites using parallel two-dimensional liquid chromatography-mass spectrometry.

Results: Principal component analysis of plasma metallomics clearly separated IPAH from SPAH and controls, indicating significant metallomics pattern differences among groups. Several metals including cobalt and platinum were significantly different in PAH patients compared to controls. In the PAH group, cobalt significantly correlated with tungsten (Pearson correlation ρ=0.75, p=0.001). Statistical univariate metabolomics analysis identified 205 metabolites with significant abundance alterations in APAH versus controls, demonstrating substantial metabolic dysregulation associated with PAH.

Conclusion: Distinct metallomics and metabolomics profiles differentiate PAH subtypes from controls, with military-relevant metal exposures potentially contributing to disease pathogenesis. These findings suggest that environmental metal exposure may represent an underappreciated risk factor for PAH development and progression, warranting further investigation into preventive and therapeutic strategies targeting metal-induced oxidative stress and metabolic dysfunction.

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SP 2 - Kiosk 9 - Poster 3

Feasibility, acceptability, and outcome selection for assessment of a hypobaric oxygenator compared to standard oxygenator: A case control study

Presenting Author: *Nathan Clendenen, MD, MS*

Background: Cardiac surgery with cardiopulmonary bypass (CPB) is effective for reducing mortality with severe cardiovascular disease and improving quality of life but it is associated with a high-risk of perioperative cerebral vascular accident, delirium, and acute kidney injury (1). Hypobaric oxygenation is a possibly beneficial intervention for reducing the exposure to GME generated during cardiac surgery with CPB (2). The purpose of this study was to determine the feasibility and acceptability of the use of a hypobaric oxygenator in a future clinical trial. Secondary objectives included determining the severity of GME after CPB determined by transesophageal echocardiography (TEE). We also compared the diagnostic performance of trainee echocardiographers' ability to quantify gaseous microemboli (GME) to an expert. Our hypothesis is that increased exposure to GME is associated with a lower venous cerebral saturation and reduced urine output and that a future randomized trial will need to account for TEE assessment variability due to the echocardiographers experience level.

Methods: We reviewed data from patients undergoing cardiac surgery with CPB with a standard or hypobaric oxygenator (Spectrum VT200 or VT160). The study was approved by the University of Colorado IRB (# 17-2455). Cases were matched to controls with a 1 to 4 ratio based on surgeon, procedure, and Euroscore II value. We included patients undergoing complex elective cardiac surgery with CPB. We compared groups using appropriate parametric or nonparametric tests as appropriate. For variables that were significant we performed regression to compare the groups while controlling for age, sex, CPB time, and EuroSCOREII. Fellows (3) were compared to a gold standard echocardiographer (TS). Each reviewed post-CPB TEE Image Uploads and were blinded to group and scored the presence of air on an ordinal scale (1 = none, 5 = extreme). Trainee results were averaged and compared to the gold standard score.

Results: Implementing the use of a hypobaric oxygenator was feasible, acceptable, and did not disrupt usual care for patients undergoing cardiac surgery with CPB. Our matching scheme resulted in some imbalance between the groups (Table 1). We did not observe a significant difference in the severity of GME detected by TEE or a difference in cerebral oxygen saturation or urine output score between groups. In a crude analysis we observed a significant difference in the max VO2/DO2, nadir DO2, time below a mean arterial pressure 55 mm Hg, and DO2i time dose response. Controlling for age, sex, CPB duration, and Euroscore2 value we noted an independent association between max VO2/DO2 (estimate for hypobaric filter 0.11, p = 0.01) and nadir DO2 (estimate for hypobaric filter -79.7, p =0.007). Trainee GME scores were correlated with the gold standard (0.80 p = 0.02) but significantly overestimated the severity of GME (+0.16, p = 0.02). The

ROC AUC for the average trainee result was 0.81 (range for individual trainees 0.68 – 0.88).

Conclusions: Implementation of a hypobaric oxygenator for elective cardiac surgery is feasible, acceptable, and independently associated with a high max VO2/DO2 and lower nadir DO2 compared to a standard oxygenator. Assessment of GME by TEE varies with experience level.

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	All (N = 51)	Control (n = 40)	Hypobaric Oxygenator (n =11)	P value
Age	65 ± 13	64 ± 13	70 ± 11	0.03
Sex (Male)	41/51 (80%)	35/40 (88%)	6/11 (55%)	0.02
BMI	27± 4	27 ± 4	26 ± 5	0.62
EuroSCOREII	3.28% ± 4.90%	3.46% ± 5.48%	2.60% ± 1.40%	0.26
Urgent procedure	8/51(16%)	7/40 (18%)	1/11 (9%)	0.47
CABG	11/51 (22%)	9/40 (23%)	2/11 (18%)	-
MVR	19/51 (37%)	15/40 (38%)	4/11 (36%)	-
Combined	21/51 (41%)	16/40 (40%)	5/11 (46%)	-
Repeat sternotomy	3/51 (6%)	3/40 (8%)	0/11 (0%)	0.22
CPB time (min)	138 ± 46	135 ± 47	150 ± 43	0.37
Aortic cross clamp	97 ± 35	100 ± 37	87 ± 28	0.26

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SP 2 - Kiosk 9 - Poster 4
The Effect of Perioperative Transfusion and Anemia on Adverse Outcomes in a Cardiac Surgical Population
Presenting Author: *Reema Shah, DO*

Introduction: Anemia is common in cardiac surgery and is linked to increased morbidity, mortality, and prolonged hospitalization. Intraoperative blood loss and hemodilution during cardiopulmonary bypass often worsen anemia and increase perioperative RBC and non-RBC transfusions. While transfusions are often beneficial, they carry the risk of immunomodulation, lung and renal injury, volume overload, and infection. Understanding whether postoperative complications are driven by anemia, transfusion exposure, or underlying illness severity is essential for improving blood management. This study evaluated the association between a predefined set of clinical and perioperative risk factors on postoperative outcomes including infection, prolonged ventilation, renal complications, rebleeding, readmission, length of stay (LOS), and mortality. It then assessed whether perioperative RBC transfusion and preoperative anemia independently predicted these adverse events

Methods: We performed a retrospective review of our hospital's Adult Cardiac Surgery Society of Thoracic Surgeons Database from January 2018 to March 2024. Although our practice follows STS 2021 transfusion guidelines, final transfusion decisions remained at the discretion of the operative team. Univariate analyses were performed to look for associations of our predefined risk factors with adverse outcomes. After discovery of univariate predictors, multivariable analyses were performed using logistic regression for binary outcomes (infection, prolonged ventilation, renal complications, rebleeding, readmission, mortality), and linear regression for length of stay. Models included transfusion exposure, anemia severity, demographic/physiologic variables, renal function, and surgical acuity. A secondary model incorporating bypass time and perfusion variables was performed in a subset with complete data. Statistical significance was set at $p < 0.05$.

Results: 5,482 patients were included in the initial analysis. Perioperative RBC transfusion was significantly associated with multiple adverse outcomes including infection, prolonged ventilation, and renal complications (Table 1). After adjusting for bypass time and the use of retrograde autologous prime (RAP), perioperative RBC transfusion remained significantly associated with the aforementioned adverse outcomes (Table 3). Tables 2 and 4 show the multivariate analyses of preoperative hemoglobin with adverse outcomes. Mortality was not significantly associated with perioperative RBC transfusion or preoperative anemia.

Conclusion: Perioperative RBC transfusion and low preoperative hemoglobin are significant independent risk factors for multiple adverse outcomes. These include prolonged ventilation,

renal complications, length of stay, and readmission to the hospital. These results emphasize the significance of optimizing preoperative hemoglobin and reducing transfusion risk before cardiac surgery. Preoperative anemia is a modifiable risk factor whose diagnosis and treatment with IV iron (if iron deficient) may serve to decrease the risk of postoperative complications. Image Uploads

Table 1. Perioperative RBC Transfusion as a Predictor of Adverse Outcomes

Outcome	Odds Ratio (OR)	95% CI	*p*-value
Infection	3.421	1.695 – 6.901	<0.001*
Prolonged Ventilation	3.930	2.551 – 6.055	<0.001*
Renal Complications	1.710	1.043 – 2.806	0.033*
Length of Stay	1.048	-0.648 – 2.744	0.226
Readmission	1.170	0.848 – 1.615	0.338
Mortality	1.307	0.210 – 8.145	0.774

Table 2. Preoperative Hemoglobin as a Predictor of Adverse Outcomes

Outcome	Odds Ratio (OR)	95% CI	*p*-value
Infection	0.930	0.864 - 1.00	0.051
Prolonged Ventilation	0.902	0.859 – 0.947	<0.001*
Rebleeding	0.964	0.885 – 1.050	0.395
Renal Complications	0.879	0.824 – 0.938	<0.001*
Length of Stay	-0.466	-0.724 - -0.208	<0.001*
Readmission	0.920	0.876 – 0.966	<0.001*
Mortality	0.912	0.755 – 1.101	0.338

Table 3. Perioperative RBC Transfusion as a Predictor of Adverse Outcomes (Adjusted for CPB time and RAP)

Outcome	Odds Ratio (OR)	95% CI	*p*-value
Infection	5.339	1.626 – 17.534	0.006*
Prolonged Ventilation	4.341	2.353 – 8.011	<0.001*
Renal Complications	2.098	1.036 – 4.249	0.039*
Length of Stay	1.755	-0.398 – 3.908?	0.110
Readmission	1.103	0.734 – 1.658	0.638
Mortality	1.528	0.185 – 12.616	0.694

Table 4. Preoperative Hemoglobin as a Predictor of Adverse Outcomes (Adjusted for CPB time and RAP)

Outcome	Odds Ratio (OR)	95% CI	*p*-value
Infection	0.921	0.843 – 1.007	0.069
Prolonged Ventilation	0.884	0.833 – 0.938	<0.001*
Rebleeding	0.981	0.885 – 1.088	0.717
Renal Complications	0.902	0.833 – 0.977	0.012*
Length of Stay	-0.589	-0.902 - -0.276	<0.001*
Readmission	0.930	0.877 – 0.987	0.017*
Mortality	0.872	0.719 – 1.058	0.165

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SP 2 - Kiosk 10- Poster 1

Expedited Hydroxocobalamin Administration for Vasoplegia Syndrome: Process Improvement Without Clear Clinical Effect

Presenting Author: *William Wright, MD*

Introduction: Vasoplegia syndrome (VS) is a severe complication of cardiac surgery characterized by profound vasodilation that is refractory to vasopressors. Hydroxocobalamin (HCB) is a nitric oxide modulator used in the multimodal treatment of VS for its vasopressor-sparing effects. Although the optimal timing of HCB in the treatment of VS is not known, limited evidence suggests that earlier administration may improve responsiveness. At our institution, HCB was historically available only by delivery from central pharmacy, creating delays in treatment. As part of a quality improvement initiative aimed at expediting care, HCB was stocked in an automated dispensing cabinet (ADC) adjacent to the cardiac operating rooms. We hypothesized that our intervention would shorten treatment delays and be associated with enhanced responsiveness to HCB, as reflected by greater increases in mean arterial pressure (MAP) and/or reductions in vasopressor requirements.

Methods: This observational study included all patients who underwent cardiothoracic surgery involving cardiopulmonary bypass (CPB) and received intraoperative HCB at a single academic medical center from November 2022 through September 2025. Data were extracted from review of medical records. Patients were analyzed in pre- vs. post-intervention groups according to whether they received HCB delivered from central pharmacy vs. retrieved from the ADC. “Decision to treat” was defined as the time of electronic order (pre-intervention) or ADC withdrawal (post-intervention). The primary process outcome was length of time from decision to treat to medication administration. Responsiveness to HCB was assessed by comparing MAP and norepinephrine equivalent (NEE) trends between the groups in the two hours following HCB administration. NEE was calculated using previously published weighting of vasoactive infusions. Continuous variables between the groups were compared using Mann-Whitney U tests, while categorical variables were compared using Fisher exact tests. MAP and NEE trends were examined using linear mixed models with a random intercept to account for repeated observations.

Results: Twenty-four patients were included (15 pre- vs. 9 post-intervention). The groups were similar in demographics, comorbidities, cardiac function, need for mechanical circulatory support, preoperative medications, and laboratory values. Total surgical and CPB durations were

also comparable between the groups. Time from decision to treat to HCB administration was significantly shorter in the post-intervention group (16.5 vs. 40.0 minutes, p = .003). MAP increased more rapidly in the pre-intervention group (modeled slope of 0.10 mmHg/min, p < .001) compared to the post-intervention group (0.01 mmHg/min, p = .763) following HCB administration (p = .043), whereas the magnitude of NEE decrease over time did not differ between groups (p = .465).

Conclusion: Although stocking HCB in a nearby ADC significantly shortened time to administration, our intervention did not translate into improved hemodynamic responsiveness. Interpretation of our findings is limited by the retrospective design and small, heterogenous cohort. Larger prospective studies are needed to better characterize how timing influences responsiveness to HCB in VS.

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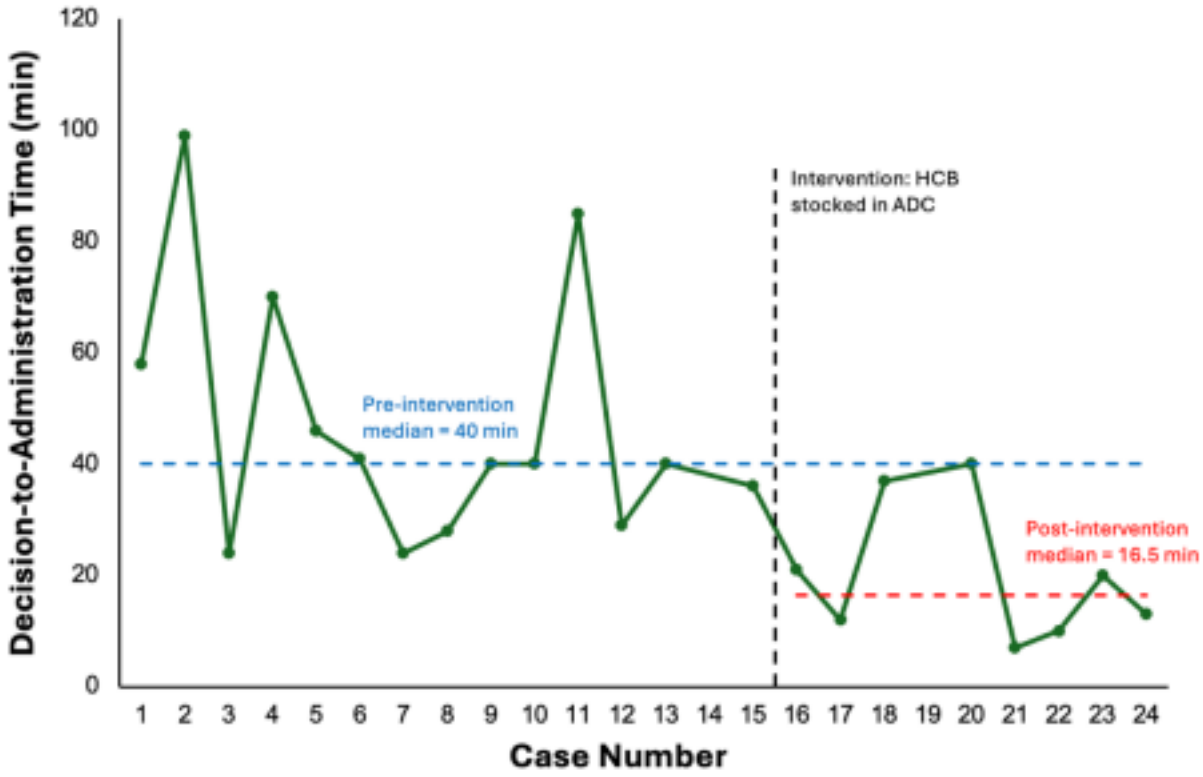


Figure 1. Run chart of time from decision to treat to HCB administration for 24 cardiac surgery cases. The dashed black vertical line indicates the point at which HCB was made available in an ADC. The dashed blue and red horizontal lines indicate the median pre- and post-intervention times to administration. Time from decision to treat to HCB administration was significantly shorter in the post-intervention period (16.5 vs. 40 minutes, $p = .003$). Abbreviations: ADC, automated dispensing cabinet; HCB, hydroxocobalamin.

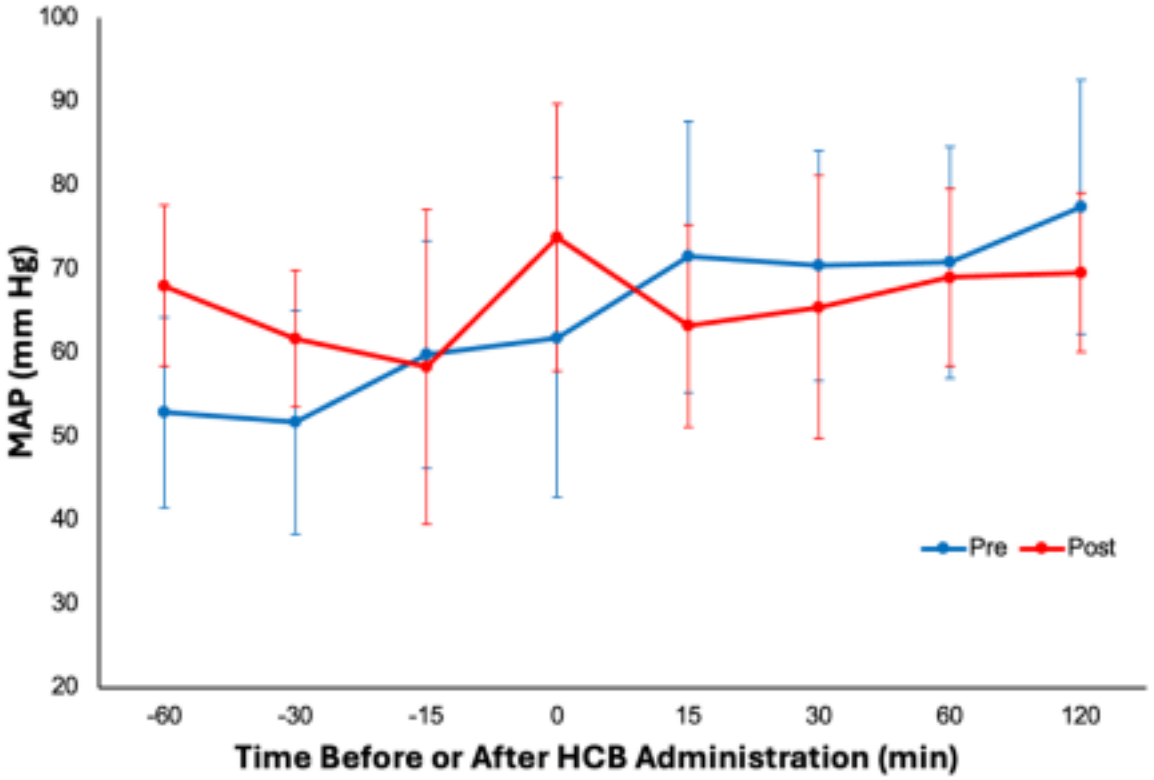


Figure 2. MAP trends during the hour before and two hours after HCB administration. In the total cohort, MAP increased at a modeled slope of 0.064 mmHg/min during the two hours after HCB administration (p = .005). There was a significant difference in MAP change over time between groups following HCB administration (p = .043), such that those in the pre-intervention group showed a more rapid increase (0.10 mmHg/min, p <.001) compared to those in the post-intervention group (0.01 mmHg/min, p = .763). Error bars represent one standard deviation around the mean. Abbreviations: HCB, hydroxocobalamin; MAP, mean arterial pressure.

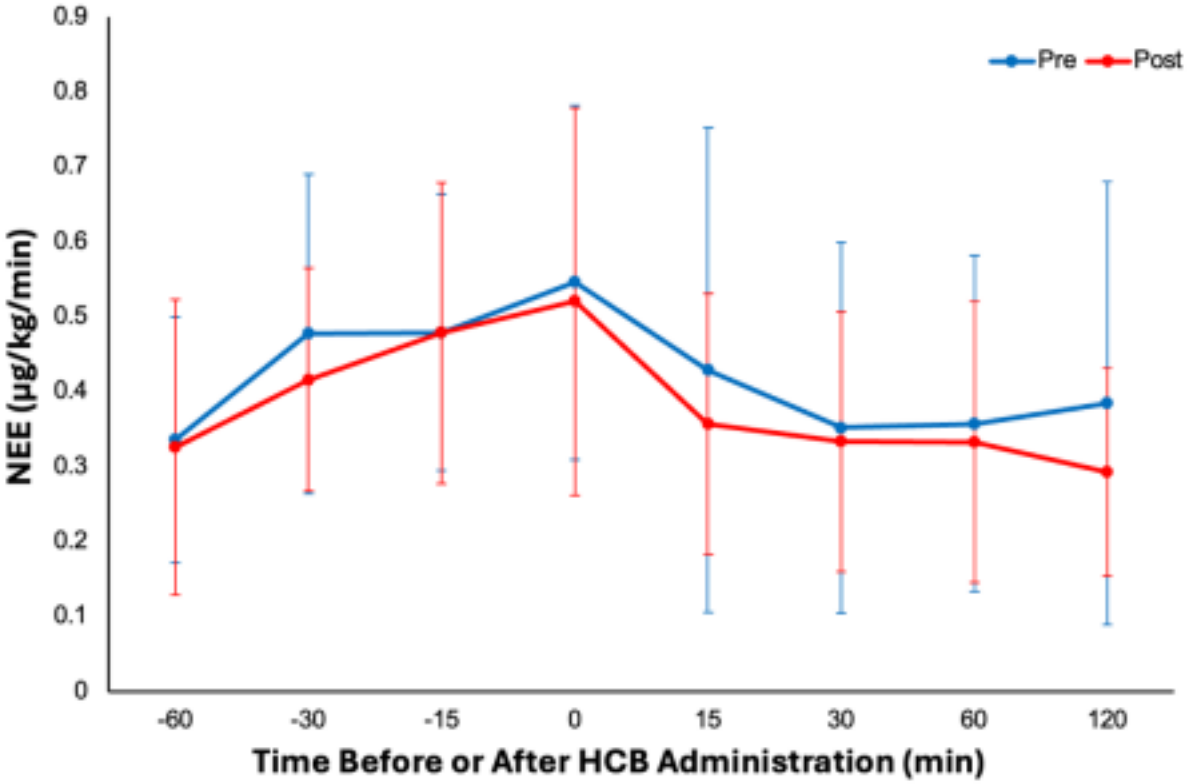


Figure 3. NEE trends during the hour before and two hours after HCB administration. In the total cohort, NEE decreased by a modeled slope of 0.001 µg/kg/min per minute during the two hours after HCB administration (p = .007). There was no significant difference in NEE change over time between groups following HCB administration (p = .465). Error bars represent one standard deviation around the mean. Abbreviations: HCB, hydroxocobalamin; NEE, norepinephrine equivalents.

Table 1. Baseline characteristics

	Pre-Intervention	Post-Intervention	*p* value
	n = 15	*n* = 9	
Age (years)	51 (40.5-59.5)	61 (41-76)	0.270
Male, n (%)	13 (87)	8 (89)	1
BMI (kg/m^2)	26.1 (23.1-26.9)	26.8 (24.5-28.7)	0.371
Hypertension, n (%)	6 (40)	5 (56)	0.675
CAD, n (%)	6 (40)	6 (67)	0.400
HFrEF, n (%)	10 (67)	4 (44)	0.403
IE, n (%)	5 (33)	1 (11)	0.351
DM, n (%)	2 (13)	3 (33)	0.326
CKD, n (%)	5 (33)	1 (11)	0.351
Thyroid disease, n (%)	1 (7)	2 (22)	0.533
LVEF (%)	30 (17.5-42.5)	50 (25-55)	0.219
MCSD, n (%)	5 (33)	5 (56)	0.403
Prior sternotomy, n (%)	2 (13)	4 (44)	0.150
ACE-I/ARB use, n (%)	2 (13)	1 (11)	1
Beta blocker use, n (%)	6 (40)	3 (33)	1
Amiodarone use, n (%)	4 (27)	4 (44)	0.412
Creatinine (mg/dL)	1.1 (0.9-1.3)	1.9 (1.0-2.1)	0.188
eGFR (mL/min/1.73 m^2)	64 (52-72)	36 (29-54)	0.089
Hemoglobin (g/dL)	9.2 (8.2-10.0)	8.9 (7.6-9.6)	0.591
Hemoglobin A1c (%)	5.7 (5.4-6.0)	5.9 (5.9-6.1)	0.140

Abbreviations: ACE-I, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; CAD, coronary artery disease; CKD, chronic kidney disease; DM, diabetes mellitus; eGFR, estimated glomerular filtration; IE, infective endocarditis; LVEF, left ventricular ejection fraction; MCSD, mechanical circulatory support device.
Continuous variables are presented as median (interquartile range).

Table 2. Case specifics and clinical outcomes

	Pre-Intervention $n = 15$	Post-Intervention $n = 9$	p value
Surgical procedure*			
CABG	3	1	1
Valve procedure	9	3	0.400
Aorta repair	3	1	1
Device placement	8	4	1
Heart transplantation	1	0	1
Lung transplantation	0	2	0.130
Redo sternotomy, n (%)	2 (13)	3 (33)	0.326
Surgery duration (minutes)	291 (230-487)	414 (351-566)	0.238
CPB duration (minutes)	110 (104-172)	190.0 (100-231)	0.387
Timing of HCB relative to CPB initiation (minutes)	108 (85.5-144)	140 (68-246)	0.520
Total blood products (mL)	1291 (577.5-5188)	5155 (4214-6968)	0.035
Total intravenous fluids (mL)	3198.0 (1777-6922)	9468 (6333-10934)	0.021
Concomitant use of adjunct therapies			
Methylene blue, n (%)	6 (40)	2 (22)	0.657
Angiotensin II, n (%)	9 (60)	3 (33)	0.400
Stress dose steroids, n (%)	9 (60)	3 (33)	0.400
Antihistamines, n (%)	4 (27)	0 (0)	0.259
30-Day mortality, n (%)	5 (33)	2 (22)	0.669
In-hospital mortality, n (%)	6 (40)	3 (33)	1
AKI, n (%)#	11 (79)	6 (75)	1
Need for RRT, n (%)#	5 (36)	6 (75)	0.183
30-Day stroke, n (%)	5 (33)	0 (0)	0.118

Abbreviations: AKI, acute kidney injury; CABG, coronary artery bypass grafting; CPB, cardiopulmonary bypass; HCB, hydroxocobalamin; RRT, renal replacement therapy.
Continuous variables are presented as median (interquartile range).
*Patients may have undergone more than one type of procedure.
Analyses for AKI and need for RRT exclude patients who were already on RRT prior to surgery (pre-intervention $n = 14$; post-intervention $n = 8$).

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SP 2 - Kiosk 10- Poster 2

Workplace Harassment, Incivility, and Disrespect: A Cross-Sectional Survey of the Society of Cardiovascular Anesthesiologists Membership

Presenting Author: *Christopher Van Akin, MD*

Background: Workplace harassment, incivility, and disrespect (HID) is an important challenge in anesthesiology associated with patient safety and workforce retention. We sought to assess the prevalence and types of HID in the operating room (OR) and intensive care unit (ICU) and evaluate psychological safety and intention to leave current career position from the perspective of cardiothoracic and vascular anesthesiologists and intensivists.

Methods: A 38-item survey was anonymously administered via REDCap to members of the Society of Cardiovascular Anesthesiologists (SCA) through two email communications in November 2025. All members of the SCA in 2025 with an active email address were eligible to participate. General descriptive statistics (percentages of respondents) were used to summa

Results: 134 surveys were analyzed. The survey response rate was 4 % and completion rate was 66%. Most respondents were male, identified as White, and worked within the United States. Regional geographic participation in the United States was lowest in the Southwest and greatest in the Northeast, and participation outside of the United States was from Canada, Europe, Australia, and New Zealand. Most respondents were adult cardiac anesthesiologists, with a significant percentage also identified as thoracic, transplantation, vascular, and critical care specialists. Most respondents were academic physicians, but private practice and hospital employed physicians were significantly represented. There was balanced representation of respondents with at least, 5, 10, or 20 years of clinical experience, and a smaller percentage of respondents had less than 1 year of workplace experience (Table 1).

55% of respondents indicated that they experienced HID within the last year. Undermining, microaggressions, verbal workplace violence, and bullying/harassment were the most common types of incivility reported, followed by identity-based discrimination and physical violence (Table 2). Most respondents indicated that, within the last year, supervisors or co-workers were condescending, paid little attention to their opinions, or doubted their judgement in matters under

their responsibility (Table 3). Undermining, microaggressions, and identity discrimination were the types of HID most likely to be experienced monthly (Figure 1).

Our data suggest that survey respondent's experience of HID within the last year may relate to inadequate psychological safety in the primary work team, and it may also be associated with to intention to leave their current job (Table 4).

Conclusion: Workplace HID was reported by most respondents in this study. This is the first study to examine HID from the perspective of cardiovascular, thoracic anesthesiologists and intensivists. The response rate may limit generalizability of these results, but these data are consistent with prior reports in anesthesiology, cardiology, cardiac surgery, and vascular surgery that indicate that HID is an important concern in the workplace. Future studies should examine the relationship between identity and workplace discrimination, and examine barriers to psychological safety in the workplace specifically for cardiothoracic and vascular anesthesiologists and intensivists.

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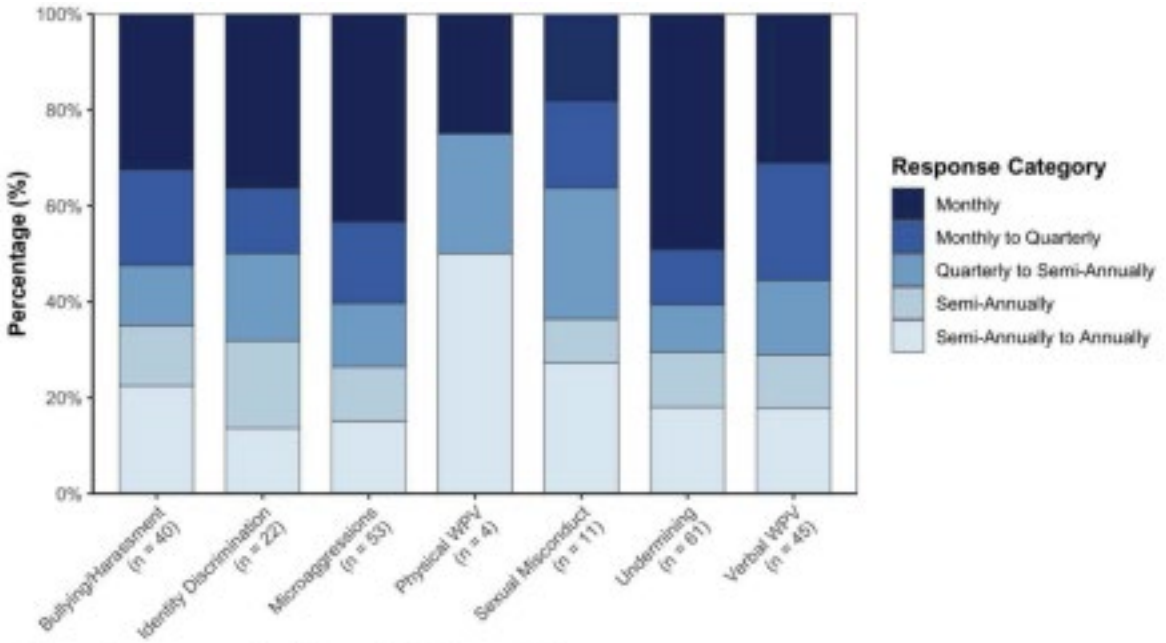


Figure 01. Frequency Distribution of Workplace Incivility.

Table 1. Baseline demographic and clinical characteristics	*n (%)*
Survey ***(n=134)***	
Complete	88 (65.7)
Not complete	46 (32.3)
Gender ***(n=128)***	
Male	74 (57.8)
Female	48 (37.5)
Prefer not to answer	6 (4.7)
Race/Ethnicity ***(n=138)****	
White	81 (58.7)
Black/African American	8 (5.8)
Asian	20 (14.5)
American Indian/Alaska Native	3 (2.2)
Hispanic/Latino	9 (6.5)
None of these	6 (4.3)
Prefer not to answer	11 (8.0)
Primary region of work ***(n=131)***	
United States	120 (91.6)
Outside of the United States	11 (8.4)
Subregion of the United States ***(n=115)***	
Northeast	50 (43.5)
Southeast	20 (17.4)
Midwest	17 (14.8)
Southwest	7 (6.1)
West	21 (18.2)
Subregion outside the United States ***(n=9)***	
Canada	2 (22.2)
Europe	3 (33.3)
Australia/New Zealand	4 (44.4)
Clinical subspecialty ***(n=129)****	
Adult Cardiac	127 (98.5)
Pediatric Cardiac	6 (4.6)
Thoracic	39 (30.2)
Transplantation	25 (19.4)
Vascular	26 (20.2)
Critical Care	28 (21.7)
Other	2 (1.5)
Practice or employment model ***(n=131)***	
Private practice management	5 (3.8)
Private practice group (physician-owned)	16 (12.2)
Academic	87 (66.4)
Hospital employed	21 (16.0)
Self-employed	1 (0.8)
Other	1 (0.8)
Years in primary clinical specialty ***(n=131)***	
< 1 year	7 (5.4)
1–4 years	22 (16.8)
5–9 years	33 (25.2)
10–20 years	32 (24.4)
> 20 years	37 (28.2)

n=number of respondents with available data for each item.
**Participants could select more than 1 response.*

Table 2. Workplace incivility in the primary work team/unit in the last year

Workplace incivility (a) (n=134)	*Yes n (%)*
Any workplace incivility (≥1 type)	74 (55.3)
Subtypes of workplace incivility (a) (n=134)	*Yes n (%)*
Physical workplace violence	4 (3.0)
Verbal workplace violence	45 (33.6)
Identity-based discrimination	22 (16.4)
Sexual misconduct	11 (8.2)
Bullying/harassment	40 (29.9)
Microaggressions	53 (39.6)
Undermining	61 (45.5)

(a) Refers to the stem: "How often have you experienced or witnessed any of the following in your primary work team/unit in the past year?" "≥1 episode in the past year" includes responses of Monthly, Monthly to Quarterly, Quarterly to Semi-Annually, Semi-Annually, or Semi-Annually to Annually on the original frequency scale. Responses of Annually or Less, "Have experienced this, but more than 1 year ago", and blank/no response were not counted as episodes in the past year.
n=number of respondents with available data.

Table 3. Experiences with supervisors or co-workers (n=118)

Experience during the last year (a)	*Yes (b) n (%)*	*No (b) n (%)*
...put you down or acted in a condescending way	88 (74.6)	30 (25.4)
...paid little attention to your statements or opinions	96 (81.4)	22 (18.6)
...made demeaning or derogatory remarks about you	60 (50.8)	58 (49.2)
...addressed you in unprofessional terms (publicly or privately)	63 (53.4)	55 (46.6)
...ignored or excluded you from professional camaraderie	59 (50.0)	59 (50.0)
...doubted your professional judgment in matters under your responsibility	81 (68.6)	37 (31.4)
...made unwanted attempts to discuss your personal matters	37 (31.4)	81 (68.6)

(a) Refers to the stem: "Were you ever in a situation in which any of your supervisors or co-workers...".
(b) No = Never or Have experienced this, but more than one year ago. Yes = Once or twice, Sometimes, Often, or Many times.
n=number of respondents with available data for each item.

Table 4. Perceptions of primary work unit/team within the last year

Primary work unit/team climate (a) (n=101)	*Yes n (%)*	*No n (%)*
Easy to speak up about what is on my mind	62 (67.4)	30 (32.6)
Mistakes in the unit/team are often held against staff	42 (45.7)	50 (54.3)
People in the unit/team are comfortable discussing problems and disagreements	57 (62.6)	34 (37.4)
People in the unit/team are eager to share information about what does and does not work	64 (70.3)	27 (29.7)
Keeping information to oneself is the best way to get ahead in the unit/team	33 (35.9)	59 (64.1)
Intention to leave current job (b) (n=100)	*Yes n (%)*	*No n (%)*
I would like to find a better job	44 (44.0)	56 (56.0)
I often think about leaving this job	43 (43.0)	57 (57.0)
I have plans to leave this job within 1 year	23 (23.0)	77 (77.0)

(a) Refers to the stem: "How descriptive is each of the following of your current primary clinical work team/unit within the last year?" For these items, Yes = slightly accurate or moderately accurate; No = highly inaccurate, moderately inaccurate, slightly inaccurate, or neither accurate nor inaccurate.
(b) Refers to the stem: "With respect to my intention to leave this organization…" For these items, Yes = agree slightly or agree strongly; No = disagree, disagree slightly, or neutral.
n=number of respondents with available data for each item.

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SP 2 - Kiosk 10- Poster 3
Patient Perspectives on Extubation Timing after Cardiac Surgery
Presenting Author: *Lauren Gibson, MD*

Background**:** Early extubation after cardiac surgery expedites recovery and reduces costs,1 such that extubation within 6 hours has been adopted as a quality metric by the Society of Thoracic Surgeons (STS).2 Extubating patients in the operating room (OR) may further shorten hospital stays, reduce costs, and increase the likelihood of discharge home,3 although some studies have reported higher rates of adverse outcomes compared with extubation in the intensive care unit (ICU).4 As rates of OR extubation increase, further research is needed to clarify the comparative efficacy of OR vs ICU extubation. Patient-reported outcomes are an important component of this assessment, yet few studies have examined patient perceptions of the extubation process.5 The objective of this study was to quantify recall of endotracheal intubation and postoperative pain among patients extubated in the ICU versus the OR and to describe qualitative patient experiences.

Methods: Elective cardiac surgical patients at Massachusetts General Hospital were surveyed at the time of ICU discharge over a 2-month period (IRB#2025P002647). After verbal consent, patients were asked about their immediate postoperative experience using a standardized questionnaire (Table 1). Surgery type and date, extubation location, and first recorded postoperative pain score were abstracted from the electronic medical record. Binary outcomes are summarized as risks with risk ratios (RR) and 95% confidence intervals (CI); initial pain scores are reported as medians with

interquartile ranges (IQR). The association between patient-reported comfort and pain score was evaluated using Spearman's rank correlation. Qualitative responses are presented descriptively.

Results: Sixty patients were surveyed, including 46 (77%) extubated in the ICU and 14 (23%) extubated in the OR (Table 2). Compared with ICU-extubated patients, those extubated in the OR were less likely to recall having an endotracheal tube (7.1% vs. 54%; RR 0.13, 95% CI 0.02-0.89) and more likely to recall feeling comfortable on awakening (79% vs. 57%; RR=1.29, 95% 0.90-1.85)(Figure 1). Initial pain scores were similar (median 6.5 vs 7, respectively). There was a weak, nonsignificant inverse correlation between patient-reported comfort on awakening and initial pain scores (Spearman ρ = -0.17, p=0.2). Among those who recalled the endotracheal tube, 40% mentioned it spontaneously as one of their earliest postoperative memories. None reported it as a positive experience; reports ranged from "uncomfortable but fine" or "challenging" to "the worst part" or "horrific" (Table 3). When asked about opportunities for improving their experience, 18% of patients cited inadequate pain control and 8% cited
discomfort related to the endotracheal tube. Conclusion: Patients extubated in the OR after elective cardiac surgery were less likely to recall endotracheal intubation, an experience generally described negatively, and more likely to report being comfortable on awakening. These findings suggest that OR extubation may enhance patient experience during the immediate postoperative period. Future prospective studies should include patient-reported outcomes alongside traditional safety measures to more fully characterize the impact of OR extubation.

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1. What is the first thing that you remember on awakening from surgery?
2. Were you comfortable when you first woke up?
3. Do you remember having a breathing tube?
4. How could we improve your experience in the early post-operative period?

Table 1. Patient survey questions.

	All patients (n=60)	ICU extubated (n=46)	OR extubated (n=14)
Post-operative day, median (range)	2 (1-4)	2 (1-4)	1.5 (1-3)
Surgery type, n (%)			
CABG	19 (32%)	15 (33%)	4 (29%)
Mitral valve surgery	13 (22%)	9 (20%)	4 (29%)
Aortic surgery	12 (20%)	10 (22%)	2 (14%)
Aortic valve replacement	7 (12%)	5 (11%)	2 (14%)
Congenital	5 (8%)	4 (9%)	1 (7%)
Other	4 (7%)	3 (7%)	1 (7%)

Table 2. Surgical characteristics including post-operative day of survey and surgery type.

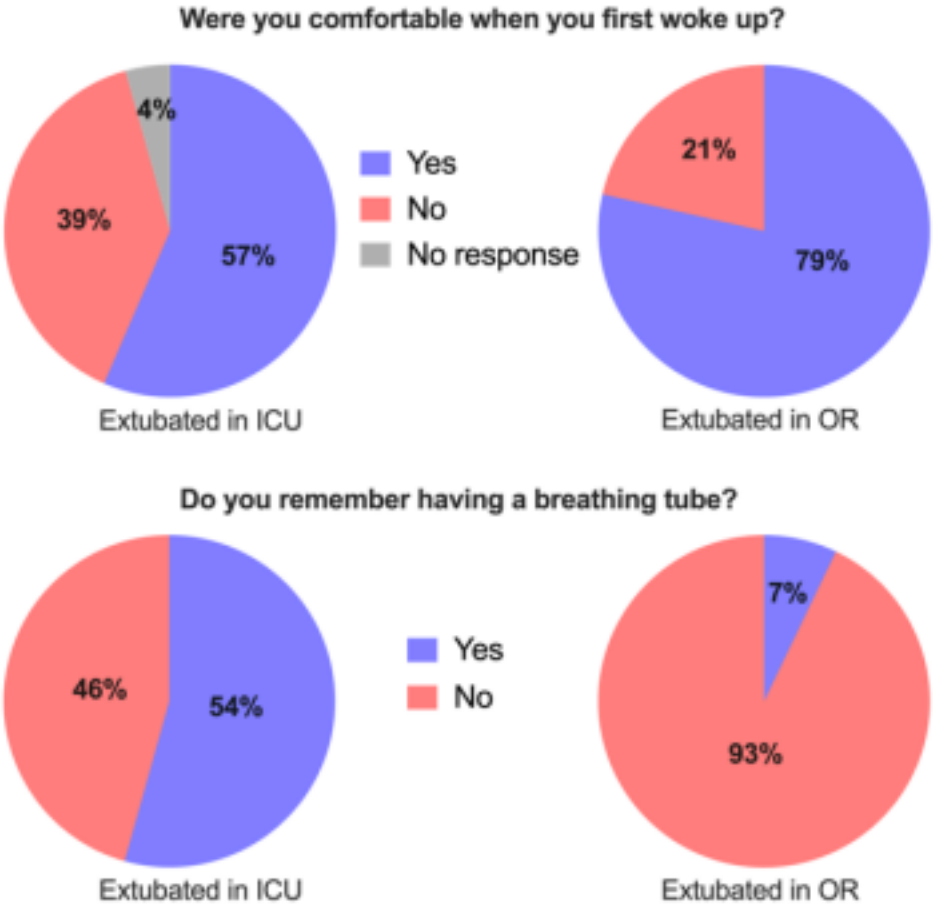


Figure 1. Proportion of ICU and OR extubated patients reporting comfort upon wakening (top) and recalling intubation (bottom).

Strongly negative comments
"[I remember] gagging on tube, screaming to get it out"*
"I woke up with the breathing tube. I couldn't breathe and thought I was going to die [...] I was scared"*
"The breathing tube was horrific. I was worried about it and that part bothered me the most"*
"The breathing tube was the worst part"
"I was most anxious about the breathing tube"
Slightly negative comments
"[I remember] wanting them to pull out the breathing tube" *
"I still had the breathing tube in and was uncomfortable" *
"[I was] uncomfortable from the breathing tube, my throat hurt"
"The breathing tube didn't bother me much"
"I wanted the breathing tube out"
"My hands were tied. I tried to push [the breathing tube] out with my tongue"
"Other than the breathing tube, I was comfortable"
"[The breathing tube] came out overnight. It was challenging but fine"
"[Having the breathing tube] was challenging but fine"
"I really didn't like it, but it was doable and I knew about it ahead of time"
"It was uncomfortable but fine"
"It was uncomfortable. I couldn't talk or swallow and my hands were tied. I didn't like it"
Neutral comments
"I expected [the endotracheal tube] and knew not to panic"*
"[I remember] chewing on the breathing tube"*
"[I remember] people telling me they were taking the breathing tube out"*
"I couldn't talk [...] not a big deal"
"It didn't really bother me"
"I had to cough it out"

Table 3. Patient-reported comments regarding endotracheal tube. Asterisk (*) indicates that the comment was made in response to the first survey question ("What is the first thing that you remember on awakening from surgery?"), prior to specific questioning about the endotracheal tube.

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SP 2 - Kiosk 10 - Poster 4

A Prospective, Randomized Controlled Trial Comparing Efficacy of Rectus Sheath Block Versus Control in Mitigating Postoperative Opioid Requirements Following Chest Tube Placement

Presenting Author: *John Choi, DO*

Introduction: Cardiac surgical pain is multifactorial, arising from sternotomy, rib retraction, vein or artery harvesting, and chest tube placement. Chest tubes, in particular, can cause substantial postoperative pain that may impair recovery and respiratory mechanics. Inadequate analgesia is associated with higher opioid use, prolonged hospitalization, and increased risk of complications such as postoperative pneumonia. The rectus sheath block (RSB), which targets the anterior cutaneous branches of the intercostal nerves, may provide improved analgesia and reduce opioid requirements.

Objective: This study evaluates whether RSB offers superior analgesia compared with conventional pain management in patients requiring chest tubes after cardiothoracic surgery. The primary endpoint is total opioid consumption (IV morphine milligram equivalents [MME]) on postoperative days (POD) 0–5. Secondary endpoints include ICU length of stay (LOS), total postoperative LOS, incidence of postoperative nausea or vomiting (PONV), and need for rescue block.

Methods: This prospective, randomized controlled trial was conducted at a tertiary academic center with Institutional Review Board approval. Eligible patients were 18–85 years old undergoing cardiothoracic surgery with chest tube placement. Exclusion criteria included ASA class V, urgent or emergent operations, chronic opioid use, and contraindications to local anesthetic. Participants were randomized to RSB or control (standard care). Care teams were blinded to group assignment. Postoperative opioid administration was extracted from electronic medical records and converted to IV MME. Secondary outcomes included ICU LOS, total LOS, PONV, and rescue block frequency. Univariate analyses used Mann-Whitney U tests for continuous variables and Chi-squared or Fisher’s exact tests for categorical variables.

Results: Eighty-four patients have been enrolled (44 RSB, 40 control). Median age was 65 years in both groups. Sixteen patients (19%) were female; the RSB group had a higher proportion of females (27.3%) than the control group (10%) (p=0.08). BMI, hypertension, hyperlipidemia, diabetes, alcohol or drug use, and chronic opioid use did not differ between groups. Preliminary analysis demonstrated no significant differences in opioid consumption on POD0–5, ICU or total LOS, PONV, or need for rescue block.

Conclusion: This trial examines whether RSB reduces postoperative opioid use and enhances recovery after chest tube placement. While RSB may theoretically limit opioid exposure, PONV, and LOS, early results show no significant differences between RSB and control groups across primary or secondary endpoints. Additional analysis will be performed once full enrollment is reached. Future work will assess final quantitative outcomes, long-term effects of chest tube–related pain, and whether RSB provides incremental benefit when used with erector spinae or pecto-intercostal fascial plane blocks.

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SP 2 - Kiosk 10 - Poster 5

Postoperative Body Surface Area-Guided Ventilation and Respiratory Acidosis

Presenting Author: *Daniel Aziz, MD*

Background: Patients receiving mechanical ventilation after cardiac surgery often receive prescriptive tidal volumes, while respiratory rate and minute ventilation are less standardized. This may result in inadequate respiration and increased acidemia, which is linked to poor cardiovascular performance and outcomes.1 We hypothesized that mechanical ventilation settings tailored to body surface area (BSA) may increase minute ventilation, reduce respiratory acidosis and decrease acidemia.

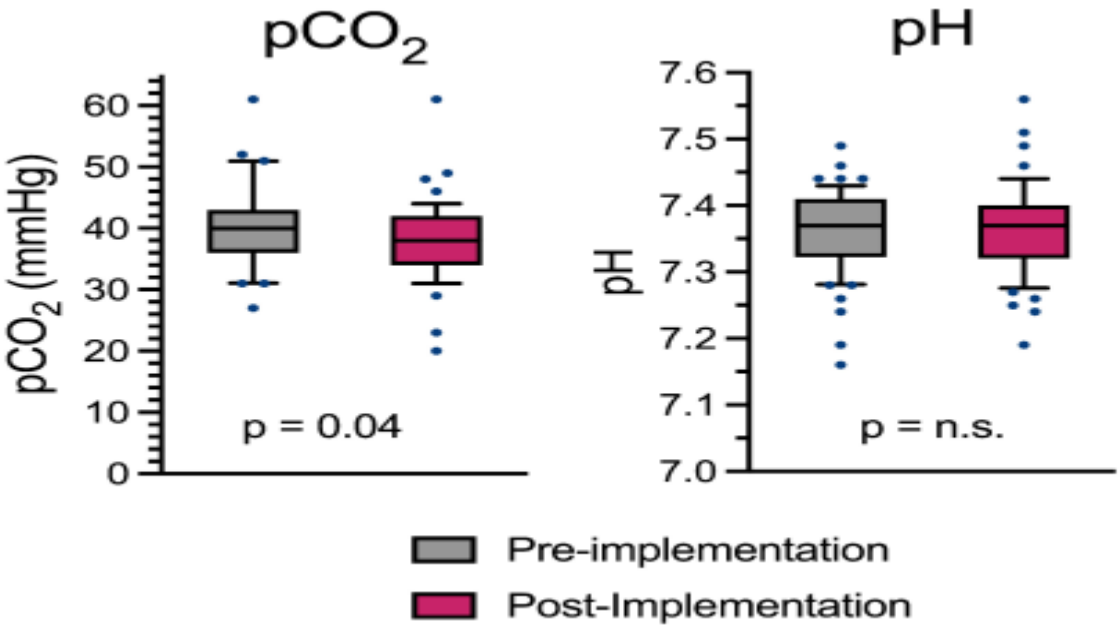
Methods: We analyzed consecutive adult patients who received open-heart surgery at Vanderbilt University Medical Center pre- and post-implementation of a BSA-guided minute ventilation quality improvement (QI) project. The ventilation project prescribed a 20-second inspiratory breath upon intensive care unit (ICU) arrival, a tidal volume of 7 ml/kg ideal body weight, and a minute ventilation of 4.0 L/m2 for women or 4.5 L/m2 for men. Prior to the project, patients were ventilated with a tidal volume range (6-8 mL/kg ideal body weight) and a respiratory rate at the discretion of

the adm arrival (T1). Ventilation parameters were adjusted based on the results of the ABG, and a second ABG was measured 60-120 minutes later (T2).

Results: One hundred eighteen consecutive patients (63 pre and 55 post-implementation) were studied. Median (interquartile range) age was 64 (54, 70) years, and 42 (35%) patients were women. BSA was 2.06 (1.82, 2.24) m2 in the pre-implementation group and 2.07 (1.87, 2.27) m2 in the post-implementation group. Surgery type (41% coronary bypass, 22% valve, 8% aorta, 13% combined, 8% transplant, and 8% other) and cardiopulmonary bypass duration (116 [91, 156] minutes) were similar between groups. Initial (T1) minute ventilation was 0.6 L/min higher and respiratory rate was 2 breaths/min higher in the post- compared to the pre-implementation group (9.1 vs 8.6 L/min, p=0.02 and 20 vs 18 breaths/min, p< 0.001). Tidal volume did not differ (487 vs. 487 mL, p=0.48). T1 ABG values pH (7.36), pCO2 (40 mmHg), pO2, (145 mmHg), and
base excess (-1.9) did not differ. At T2 (median 88.5 min after T1), respiratory rate and minute ventilation remained increased in the post-implementation group. T2 pCO2 was 2.0 mmHg lower in the post-implementation group (38.1 vs 40.1 mmHg, p=0.04; figure), BE was 1.04 lower (-3.4 vs -2.4, p=0.04), and HCO3 was 2.9 mEq/L lower (21.7 vs 24.6 mEq/L, p=0.05). pH remained similar between groups (7.37 vs 7.37, p=0.99), as did pO2 (136 vs 137 mmHg, p=0.50) and lactate (2.8 vs 2.6 mmol/L, p=0.39). Median postoperative ventilation time was 9 hours post-implementation vs 12 hours pre-implementation, and 13% of both groups were reintubated.

Conclusion: A BSA-targeted ventilation protocol increased minute ventilation and respiratory rate following cardiac surgery compared to usual care. The ventilation protocol was associated with reduced pCO2 an hour after surgery and a normal pH, despite increased metabolic acidosis in the post-implementation group. These findings suggest that BSA-targeted post cardiac surgery ventilation may reduce respiratory acidosis but have limited effects on acidemia.

Image Uploads



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SCIENTIFIC PROGRAM ABSTRACTS

Poster Session 3

Sunday, April 26, 2026 | 9:00 AM - 10:00 AM

Scientific Posters: Session 3

SP 3 - Kiosk 1 - Poster 1

Temporal Trends in the Epidemiology of Surgical Site Infections After Cardiac Surgery in a Tertiary Academic Center

Presenting Author: *Alan Iwahashi, MD*

Background: Surgical site infections (SSIs) are a potentially preventable cause of serious morbidity in cardiac surgery patients, occurring at a rate of approximately 1-3% in the literature2. Infection prevention interventions are standard practice in the perioperative care of these patients. In our large tertiary academic center, we conducted a retrospective descriptive analysis of SSI data to identify risk factors and long-term trends after cardiac surgery, which may present areas for future investigation.

Methods: 9,196 cardiac surgeries performed at DUH between January 1, 2018, and June 30, 2025 were identified and categorized as: Coronary Artery Bypass Graft (CABG), Valve, CABG + Valve, Other Cardiac, and Other Multi Cardiac. The “Other Cardiac” group includes heart transplants, heart-lung transplants, aortic/ventricular septal defect surgeries, atrioventricular repairs, and aortic aneurysms. “Other Multi Cardiac” includes any combined cardiac surgery other than CABG + Valve. We excluded patients who required reoperation following their index procedure and patients requiring mechanical circulatory support devices. Our final cohort consisted of 7,397 patients, of which 119 (1.6%) had a superficial or deep SSI detected (Figure 1). Using descriptive statistics, we summarized demographic and perioperative clinical characteristics by SSI status. Continuous variables were summarized by either mean (SD) or median [IQR] depending on the normality of their distribution, and categorical variables were summarized as frequency (%). Significant differences between SSI status were determined by T-test, Mann-Whitney U-Tests, Chi-Square Tests, or Fisher Exact Tests (Table 1). Additionally, we visualized the rate of SSI over time for the entire cohort and by surgical group (Figure 2).

Results / Discussion: In our cohort, of 119 total SSIs, 72 (61%) were invasive. SSI patients are predominantly female (Male SSI = 53.8% vs Male No SSI = 64.3%, $p = 0.023$), have a higher percentage of Black/African American patients (24.4% vs 16.4%, $p = 0.014$), and are more common in CABG procedures (55.5% vs 31.6%, $p < 0.001$). From 2021 to 2023, CABG-related SSIs approached that of non-CABG groups. SSI patients also have higher rates of obesity (54.6% vs 36.9%, $p < 0.001$), hypoalbuminemia (35.3% vs 24.1%, $p = 0.006$), diabetes (47.1% vs 27.3%, $p < 0.001$), and CKD (33.6% vs 24.9%, $p = 0.038$). Finally, SSI patients spend more time under anesthesia (7.55 Hours [6.38, 9.13] vs 7.23 Hours [6.02, 8.65], $p = 0.005$) and have longer postoperative hospital length of stays (6.86 days [4.99, 9.87] vs 5.98 days [4.86, 8.75], $p = 0.020$) (Table 2).

Conclusion: Several patient and procedural factors were associated with increased risk of SSIs, particularly in isolated CABG surgery, female, Black/African American patients, and those with obesity, diabetes, hypoalbuminemia, and CKD. Future investigations can evaluate the effect of various preventative interventions on SSI incidence using time-interrupted analyses.

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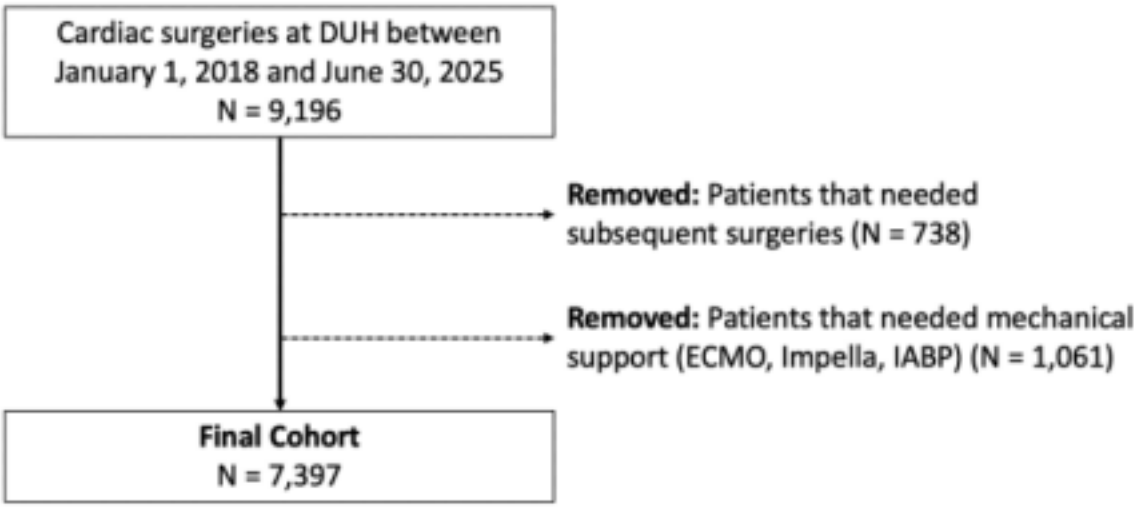


Figure 1: Consort Diagram

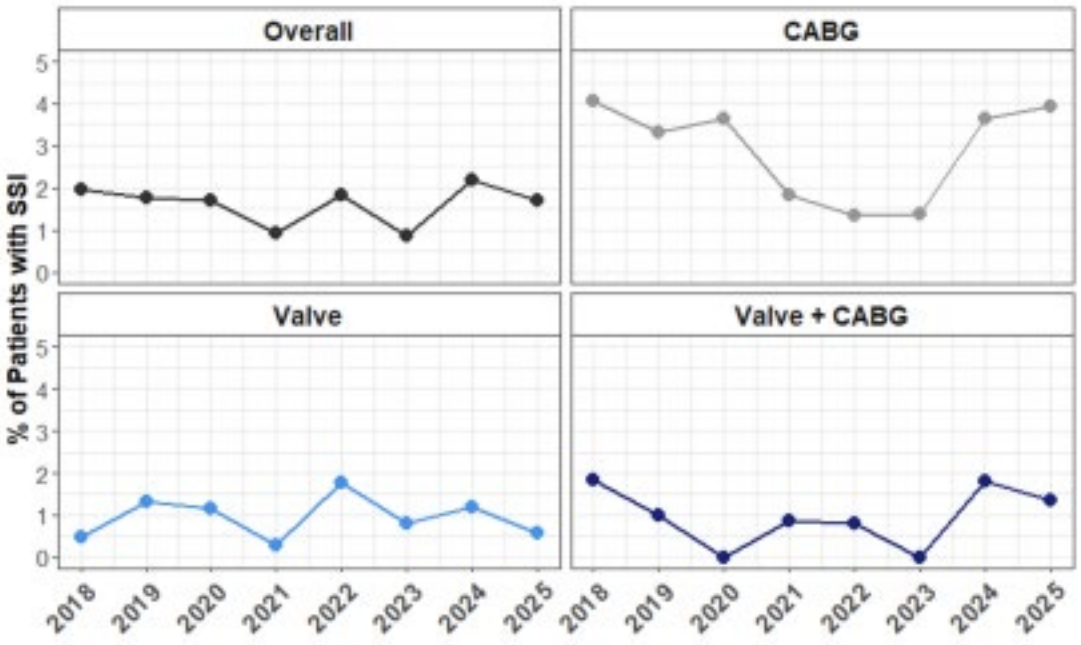


Figure 2: Rate of SSI over time for the cohort and by surgical group

	Overall	No SSI	SSI Detected	P-value
N (%)	*7397(100)*	*7278 (98.4)*	*119 (1.6)*	
Age (Years) (median [IQR])	63.00 [49.00, 71.00]	63.00 [49.00, 71.00]	59.00 [48.00, 68.00]	0.059
Adult (%)	6400 (86.5)	6295 (86.5)	105 (88.2)	0.677[c]
Sex = Male (%)	4745 (64.1)	4681 (64.3)	64 (53.8)	0.023[c]
Race (%)				0.014[b]
Caucasian/White	5477 (74.0)	5397 (74.2)	80 (67.2)	
Black/African American	1239 (16.8)	1210 (16.6)	29 (24.4)	
Other	453 (6.1)	443 (6.1)	10 (8.4)	
Not Reported/Declined	228 (3.1)	228 (3.1)	0 (0.0)	
Ethnicity (%)				0.721[b]
Hispanic	330 (4.5)	326 (4.5)	4 (3.4)	
Not Hispanic/Latino	6682 (90.3)	6571 (90.3)	111 (93.3)	
Not Reported/Declined	385 (5.2)	381 (5.2)	4 (3.4)	
Surgery Type (%)				<0.001[b]
CABG	2368 (32.0)	2302 (31.6)	66 (55.5)	
Valve	2757 (37.3)	2730 (37.5)	27 (22.7)	
Valve + CABG	817 (11.0)	809 (11.1)	8 (6.7)	
Other Cardiac	864 (11.7)	850 (11.7)	14 (11.8)	
Other Multi-Cardiac	591 (8.0)	587 (8.1)	4 (3.4)	
ASA Score (%)				0.057[b]
1	1 (0.0)	1 (0.0)	0 (0.0)	
2	104 (1.4)	103 (1.4)	1 (0.8)	
3	2190 (29.6)	2168 (29.8)	22 (18.5)	
4	5075 (68.6)	4979 (68.4)	96 (80.7)	
5	26 (0.4)	26 (0.4)	0 (0.0)	
Emergent = Yes (%)	122 (1.7)	120 (1.7)	2 (1.8)	0.717[b]
BMI (mean (SD))	28.08 (51.71)	28.05 (52.12)	29.97 (9.64)	0.689[a]
Obesity = Yes (%)	2752 (37.2)	2687 (36.9)	65 (54.6)	<0.001[c]
Last Preoperative Albumin (90 days) (mean (SD))	3.63 (0.52)	3.63 (0.52)	3.42 (0.49)	<0.001[a]
Hypoalbuminemia (%)	1794 (24.3)	1752 (24.1)	42 (35.3)	0.006[c]
Last Preoperative Hemoglobin (A1C) (90 Days) (mean (SD))	5.80 [5.40, 6.40]	5.80 [5.40, 6.30]	6.10 [5.60, 8.10]	<0.001
Diabetes = Yes (%)	2040 (27.6)	1984 (27.3)	56 (47.1)	<0.001[c]
Last Preoperative Creatinine (90 Days) (median [IQR])	1.00 [0.80, 1.20]	1.00 [0.80, 1.20]	1.00 [0.80, 1.30]	0.672
CKD = Yes (%)	1852 (25.0)	1812 (24.9)	40 (33.6)	0.038[c]
COPD = Yes (%)	416 (5.6)	404 (5.6)	12 (10.1)	0.054[c]
Smoking Status (%)				0.063[b]
Current Smoker	702 (9.5)	681 (9.4)	21 (17.6)	
Never Smoked	2176 (29.4)	2148 (29.5)	28 (23.5)	
Passive Smoke	15 (0.2)	15 (0.2)	0 (0.0)	
Quit Smoking	2132 (28.8)	2099 (28.8)	33 (27.7)	
Smoking Status Unknown	2372 (32.1)	2335 (32.1)	37 (31.1)	
Preoperative Antibiotics Administered (%)				>0.999[b]
Ordered, not administered	2 (0.0)	2 (0.0)	0 (0.0)	
Administered	7391 (99.9)	7272 (99.9)	119 (100.0)	
No orders or administrations	4 (0.1)	4 (0.1)	0 (0.0)	
Preoperative Intranasal Antibiotic Decolonization (%)				0.210[b]
Completed	3301 (44.6)	3255 (44.7)	46 (38.7)	
Not Applicable per checklist/Iodine allergy	48 (0.6)	46 (0.6)	2 (1.7)	
Not Done/No Documentation	4027 (54.4)	3956 (54.4)	71 (59.7)	
Unknown	21 (0.3)	21 (0.3)	0 (0.0)	
Preoperative Chlorhexidine Bath Procedure Followed = Yes (%)	5190 (70.2)	5114 (70.3)	76 (63.9)	0.158[c]

Table 1. Demographic and preoperative clinical characteristics for the entire cohort and by SSI status. Significant differences between SSI status determined by Mann-Whitney U-tests, Independent Samples T-Tests[a], Fisher Exact Tests[b] and Chi-Square Tests[c]

	Overall	No SSI	SSI Detected	P-value
N (%)	*7397(100)*	*7278 (98.4)*	*119 (1.6)*	
Anesthesia Duration (Hours) (median [IQR])	7.23 [6.02, 8.67]	7.23 [6.02, 8.65]	7.55 [6.38, 9.13]	0.005
Procedure Duration (Hours) (median [IQR])	5.10 [3.99, 6.44]	5.10 [3.99, 6.43]	5.59 [4.31, 6.82]	0.011
CPB Time (Hours) (median [IQR])	3.50 [2.23, 5.38]	3.50 [2.23, 5.38]	3.17 [2.00, 4.98]	0.260
Cross Clamp Time (Minutes) (median [IQR])	97.00 [69.00, 139.00]	97.00 [69.00, 139.00]	86.00 [66.00, 133.00]	0.227
Minimum Temperature After CPB (median [IQR])	97.70 [97.30, 98.10]	97.70 [97.30, 98.10]	97.70 [97.08, 98.10]	0.670
Intraoperative Hypothermia (%)				0.534[b]
Yes	346 (4.7)	341 (4.7)	5 (4.2)	
No	4380 (59.2)	4315 (59.3)	65 (54.6)	
Unknown	2671 (36.1)	2622 (36.0)	49 (41.2)	
Intraoperative Blood Products (median [IQR])				
Total Units	1.00 [0.00, 3.00]	1.00 [0.00, 3.00]	1.00 [0.00, 2.00]	0.208
RBC Units	0.00 [0.00, 1.00]	0.00 [0.00, 1.00]	0.00 [0.00, 1.00]	0.552
Cryoprecipitate Units	0.00 [0.00, 1.00]	0.00 [0.00, 1.00]	0.00 [0.00, 0.00]	0.002
Fresh Frozen Plasma (FFP) Units	0.00 [0.00, 0.00]	0.00 [0.00, 0.00]	0.00 [0.00, 0.00]	0.166
Platelets Units	0.00 [0.00, 1.00]	0.00 [0.00, 1.00]	0.00 [0.00, 1.00]	0.110
Max POD1 Glucose (median [IQR])	160.00 [143.00, 185.00]	160.00 [143.00, 185.00]	166.00 [146.00, 197.50]	0.063
Postoperative MV (First Extubation) (Hours) (median [IQR])	4.92 [3.15, 9.08]	4.92 [3.15, 9.07]	5.08 [3.45, 10.99]	0.295
First Postoperative ICU LOS (Hours) (median [IQR])	28.25 [21.52, 67.77]	28.25 [21.52, 67.72]	28.42 [21.10, 73.63]	0.878
Total Postoperative ICU LOS (Hours) (median [IQR])	31.93 [21.78, 70.18]	31.90 [21.80, 69.98]	36.60 [21.48, 79.28]	0.804
Postoperative Hospital LOS (Days) (median [IQR])	5.99 [4.86, 8.78]	5.98 [4.86, 8.75]	6.86 [4.99, 9.87]	0.020
Mortality (30 Days) (%)	74 (1.0)	74 (1.0)	0 (0.0)	0.634[b]
Discharge Disposition (%)				0.763[b]
Expired	71 (1.0)	70 (1.0)	1 (0.8)	
Home Health Service/Hospice	836 (11.3)	819 (11.3)	17 (14.3)	
Home or Self Care	6009 (81.2)	5916 (81.3)	93 (78.2)	
Skilled Nursing Facility	381 (5.2)	375 (5.2)	6 (5.0)	
Other	100 (1.4)	98 (1.3)	2 (1.7)	

Table 2. Intra and Postoperative Clinical Characteristics for the entire cohort and by SSI status. Significant differences between SSI status determined by Mann-Whitney U-tests, and Fisher Exact Tests[b]

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adults undergoing cardiac surgery. Cochrane Database of Systematic Reviews 2024, Issue 12. Accessed 30 November 2025.

SP 3 - Kiosk 1 - Poster 2

Optimizing Gas Exchange During MAC Sedation for TAVR: A Quality Improvement Study

Presenting Author: *Andrew Torck, MD*

Background/Introduction: During MAC sedation for transcatheter aortic valve replacements (TAVRs), high-risk patients (e.g. obstructive sleep apnea, pulmonary hypertension, or RV dysfunction) often experience hypoventilation, hypercapnia, and unanticipated airway interventions. These events increase procedural risk and can delay case progression, therefore, minimizing complications in these patients is critical. Despite available technology, trans-nasal humidified rapid-insufflation and
ventilatory exchange (THRIVE) remains underutilized. Literature supports using THRIVE in spontaneously breathing patients to reduce desaturation and promote gas exchange1. Rapid insufflation provides improved oxygen delivery and slows $EtCO_2$ rise in sedated patients2,3. A protocolized approach to MAC for TAVRs may reduce practice variability, airway rescue rates, and procedural risk for these high-risk patients.

Methods: Setting
A single-center QI project evaluating a standardized THRIVE protocol during MAC for TAVRs. Outcomes were compared between a control group (simple face mask) and an intervention group (THRIVE).

Patient Selection

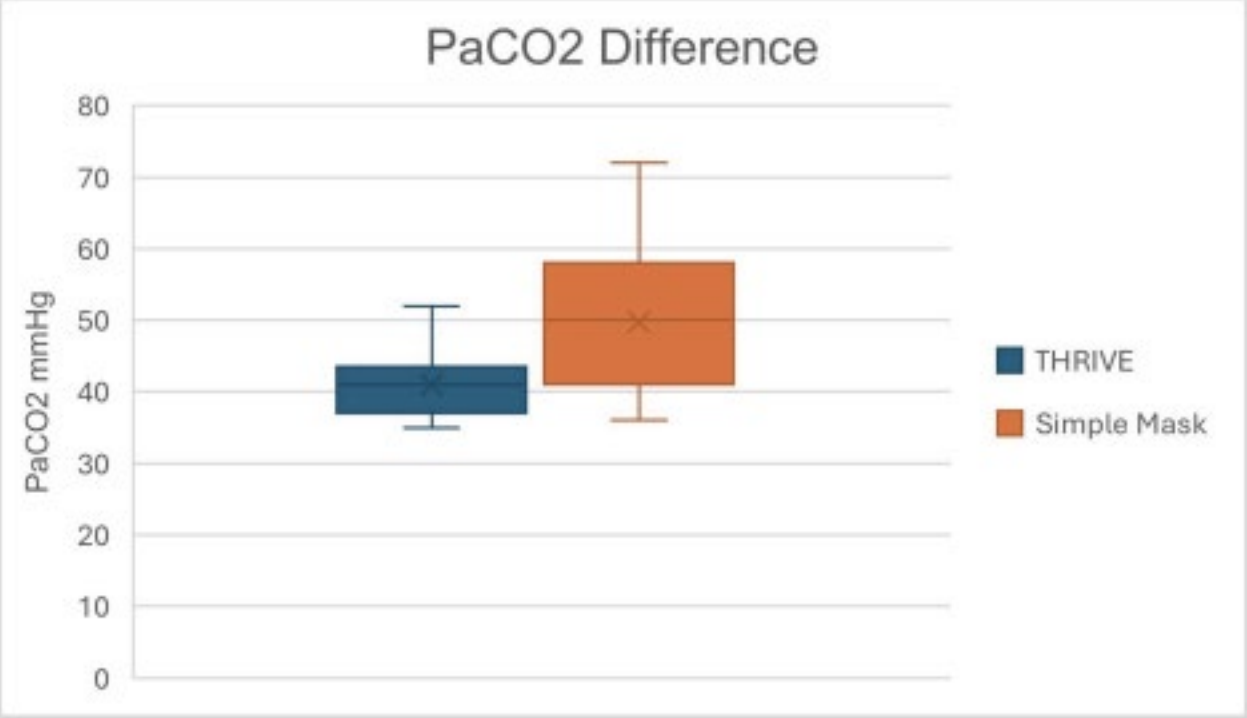
Adults undergoing elective TAVR with MAC were included. Patients were grouped by surgical date to streamline implementation. Control (n = 13): standard simple face mask. Intervention (n = 11): THRIVE via Optiflow system.

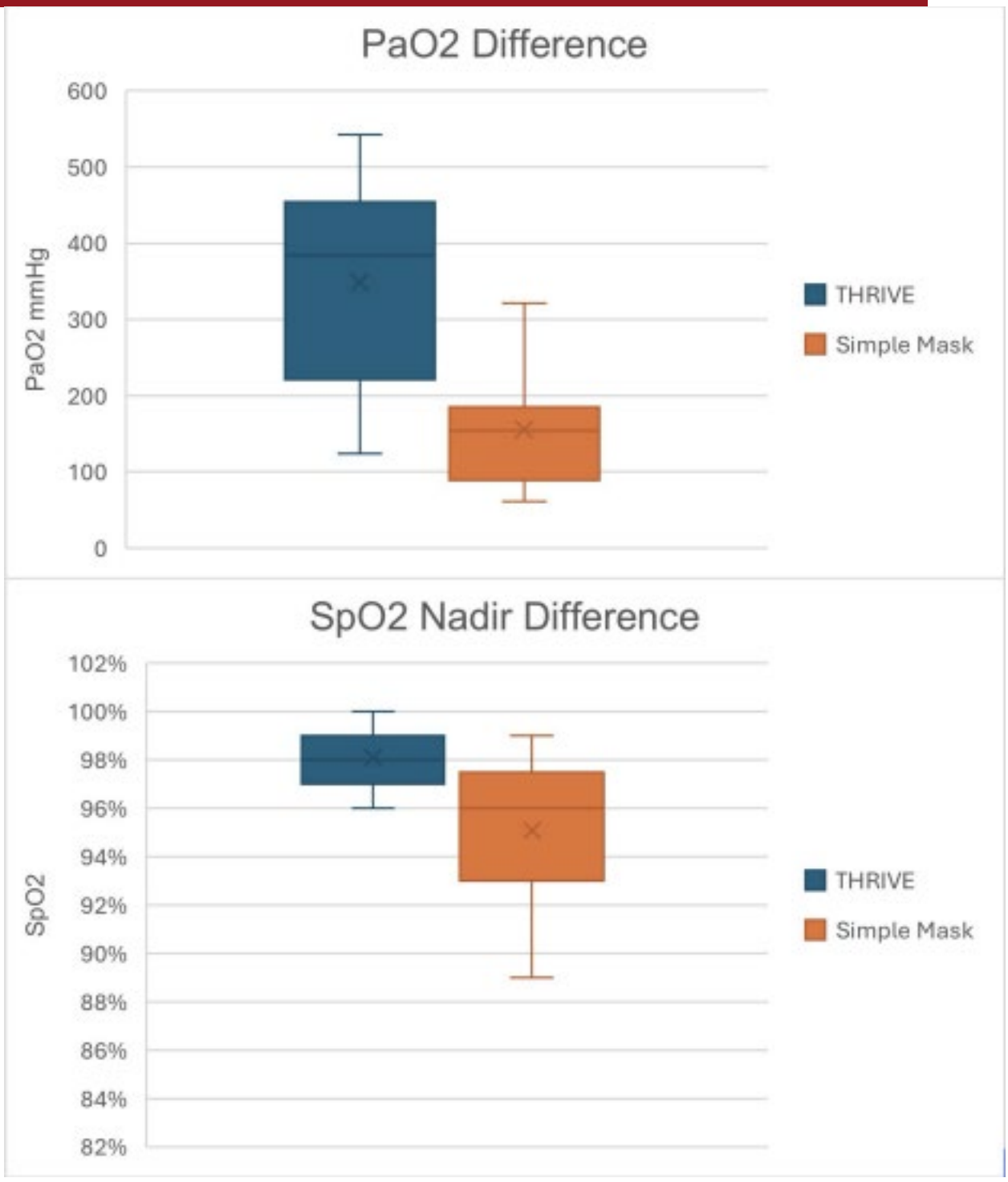
Results: The THRIVE group consistently demonstrated superior respiratory metrics compared to control with less SpO2 decline (Median: 98% vs. 96%; p = 0.004), lower PaCO2 (Median: 41 mmHg vs. 50 mmHg; p = 0.0256), and PaO2 over twice as high as control (Median: 384 mmHg vs. 154 mmHg; p = 0.002). There was no significant difference in arterial pH (p = 0.2364). The frequency of intraoperative airway interventions (jaw thrust, adjuvant airway insertion, escalation to mask ventilation, or conversion to general) was lower with THRIVE (Median: 0 vs. 2 interventions per case; p = 0.0003).

Conclusion: This project shows that a standardized THRIVE protocol is associated with improved gas exchange and fewer airway interventions compared to control. These fragile patients have minimal reserve leaving little margin for error and risk rapid decompensation. As such, every opportunity to optimize peri-procedural gas exchange should be pursued. Prior literature supports the physiological benefits of high-flow nasal oxygenation in a variety of clinical contexts1–3. THRIVE is known to prolong safe apnea time, improve oxygenation, and provide low-level CO2 wash out, making it a valuable tool for maintaining spontaneous ventilation4,5. However, its application in cardiac anesthesia remains limited. These findings provide early evidence that THRIVE can

enhance intraoperative safety in high-risk TAVR patients by improving gas exchange and minimizing airway interventions. As a result, THRIVE may signal a shift in the anesthetic management of cardiac patients with limited reserve. This warrants future prospective randomized studies to further quantify the benefits of THRIVE during TAVR and other cardiac procedures. These studies may ultimately guide the development of practice standards that reduce perioperative risk in this vulnerable patient population.

Image Uploads





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SP 3 - Kiosk 1 - Poster 3

Streamlining Society of Thoracic Surgeons (STS) data collection: Leveraging Epic-electronic medical record integration for efficient and reliable cardiac surgery data capture.

Presenting Author: *Ala Jamal, MD*

Background: The Society of Thoracic Surgeons (STS) Adult Cardiac Surgery Database is a cornerstone for quality improvement and research initiatives in cardiac surgery. At UC Davis Health, we share the vision by collecting the required STS data for every index-cardiac surgery case on cardiopulmonary bypass (CPB). However, the necessary data are dispersed throughout the Epic-electronic medical record (EMR), making the process time-consuming and prone to omissions, especially for variables that become available only after specific postoperative time points (e.g., pain scores on post-operative day 3 and at discharge).

Methods/Results: A custom SmartPhrase was developed within Epic Hyperspace® May 2025-version, in collaboration with the Informatics team, by identifying and aggregating the unique Epic codes for each required data field of interest. The tool extracts preoperative vitals, intraoperative medications and events, postoperative laboratory data, pain scores, and other required elements from patients' chart into a single standardized progress note. The SmartPhrase was validated on 20 patient-records with previously collected STS data, demonstrating 100% concordance with manual chart review. Following successful validation, the tool was launched on October 1, 2025. Since implementation,

data collection time per case decreased from 20–30 minutes to less than 3 minutes. To ensure ongoing data integrity, a weekly quality assurance process was established in which two patient records are randomly selected, and SmartPhrase-generated data are compared to manual chart review.

Discussion: Initial validation confirmed complete agreement between SmartPhrase and manual data collection. The significant time-reduction in data collection has been sustained, and ongoing weekly audits continue to demonstrate accuracy. This workflow system not only streamlines data collection but also establishes a solid framework for continuous quality monitoring. The approach is adaptable to other complex data collection needs within the EMR.

Conclusion: Integrating Epic data codes into a single SmartPhrase assured reduction in time-requirement for STS data collection while maintaining accuracy. This approach streamlines a previously fragmented process, supports continuous quality assurance, and offers a scalable model for other institutional data collection systems.

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Change in STS Data Collection Efficiency Before and After SmartPhrase Integration

Metric	Pre-Implementation (Manual Review)	Post-Implementation (SmartPhrase)
Time per case	20–30 minutes	<3 minutes
Workflow sessions per case	2–3	1
Chart locations needed	>10	1
Risk of omissions	Moderate	Near-zero
QA discrepancies	Not routinely measured	0 (weekly audits to date)

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SP 3 - Kiosk 1 - Poster 4

Association of Induction with Intravenous Methadone with Analgesic Outcomes in Adult Robotic-Assisted Mitral Valve Repair Surgery: A Retrospective Observational Cohort Study

Presenting Author: *Kaber Karimi, MD*

Introduction: Methadone is a long-acting μ-opioid receptor agonist and N-methyl-D-aspartate (NMDA) receptor antagonist with increasing research suggesting sustained postoperative analgesia after cardiac surgery due to its prolonged duration of action (4-6 hours). Despite increasing interest in opioid-sparing techniques, data comparing Methadone to standard opioid strategies upon induction remain limited, with even less data regarding robot-assisted cardiac surgeries. This study evaluated whether Methadone administration on induction is associated with reduced postoperative pain scores in adult patients undergoing robot-assisted surgery.

Methods: We conducted a single-center, retrospective observational cohort study of adult patients (≥18 years) undergoing elective robotic-assisted mitral valve repairs between May 2025 to October 2025. Patients were grouped based on receipt of Intravenous methadone at induction (exposed) versus fentanyl-only induction (control). Exclusion criteria included pts >70 years of age, chronic methadone therapy, emergency surgery, conversion to median sternotomy, not extubated in OR, and missing postoperative analgesia data.

Data was obtained from the EPIC electronic medical record including demographics, comorbidities, procedure type, anesthesia medications, and postoperative outcomes. The primary outcome was post-operative pain scores (Numeric Rating Scale 0–10) during the first 24 hours following ICU arrival. Secondary outcomes included total opioid consumption (converted to Oral Morphine Equivalent). Unadjusted comparisons were performed using appropriate parametric or non-parametric tests comparing mean scores of post-op pain scores and opioid consumption

Results: A total of 41 patients met inclusion criteria, including 15 receiving induction methadone and 26 receiving fentanyl-only induction. Upon analysis of primary outcomes, induction methadone showed reduction in 24-hour pain scores (mean 2.97 +/- 2.11 in exposed vs 5.09 +/- 2.11 in control) and overall lower opioid requirements (mean 12.56 in exposed +/- 12.58 vs 15.42 +/- 19.5 in unexposed).

Conclusions: In this retrospective cohort of cardiac surgical patients, intravenous methadone administered at induction showed reduced postoperative pain scores and lower opioid. These findings support the potential role of methadone as an opioid-sparing analgesic strategy in cardiac anesthesia and justify further prospective evaluation, though further investigation is needed to determine statistically significant differences in data outcome.

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SP 3 - Kiosk 1 - Poster 5

Improving Perioperative Temperature Regulation in Cardiac Anesthesia: A QI Initiative for TAVR Cases

Presenting Author: *Joshua Ogogo,MD*

Background: Body temperature (36–37.5°C) is regulated by the hypothalamus through hormonal, metabolic, and behavioral mechanisms, with behavior—driven by skin temperature—being most effective. Perioperative hypothermia (core < 36°C) commonly occurs due to anesthesia, heat redistribution, and a cool environment, primarily via radiation, and increases the risk of cardiac complications, infection, coagulopathy, delayed recovery, and longer hospital stays. Although Transcatheter Aortic Valve Replacement (TAVR) is a minimally invasive alternative to open surgery, it still carries a significant risk of hypothermia despite limited exposure and extensive draping.

Methods: This quality improvement project aimed to reduce intraoperative and postoperative hypothermia in patients undergoing TAVR. A multidisciplinary team implemented an enhanced active rewarming strategy to maintain normothermia from the operating room through transfer to the step-down unit.

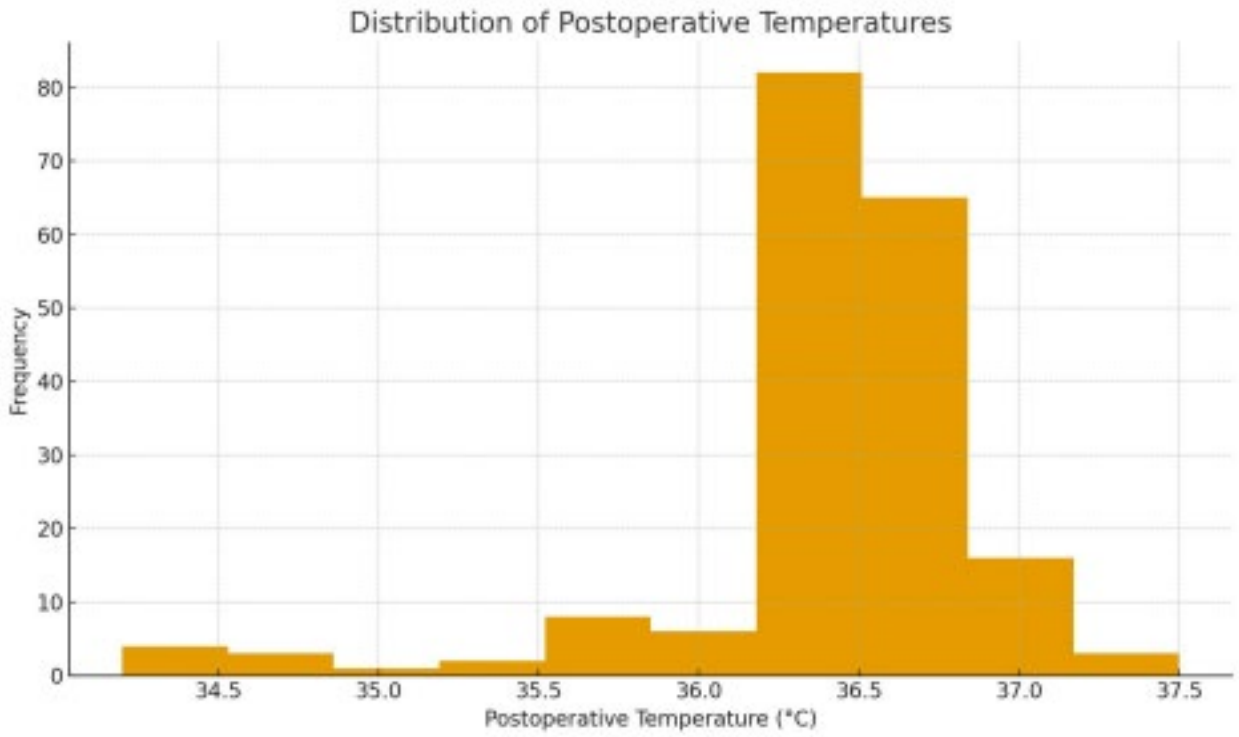
The intervention used a standardized dual forced-air warming protocol with both underbody and upper-body Bair Hugger devices for all eligible patients. Operating room temperature was maintained at 70°F. Intraoperative temperature was measured with skin thermometry during sedation and nasopharyngeal probes under general anesthesia at predefined intervals. Postoperative temperature on step-down unit arrival was measured orally.

Conducted prospectively from January to November 2025, the project included all consecutive TAVR patients at our institution (N = 190). Collected data included demographics, intraoperative and postoperative temperature values, warming interventions, and hypothermia incidence on arrival to the step-down unit. Process adherence and outcomes were compared with pre-intervention baseline data to evaluate the strategy's effectiveness.

Results: A total of 190 TAVR patients had postoperative oral temperatures measured in the step-down unit. After implementing the enhanced intraoperative warming strategy, postoperative temperatures shifted noticeably toward normothermia. The mean temperature was 36.41°C (IQR 36.1–36.7°C), and only 9.5% of patients arrived with a temperature below 36.0°C. Compared with earlier patterns of frequent mild hypothermia, these findings indicate a substantial reduction in low postoperative temperatures with the combined under-body and upper-body forced-air warming and controlled OR ambient temperature.

Conclusion: Perioperative hypothermia remains a significant risk in TAVR procedures despite their minimally invasive nature. Anesthesia impairs thermoregulation, and heat loss occurs rapidly, making temperature maintenance crucial to prevent complications and support recovery. Implementing a standardized rewarming strategy—dual forced-air warming with controlled OR temperature—improved postoperative thermal outcomes, shifting temperatures toward normothermia and reducing hypothermia on step-down unit arrival. These results highlight the effectiveness of protocolized warming and the importance of structured temperature management in TAVR perioperative care.

Image Uploads



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SP 3 - Kiosk 2 - Poster 1

Postpartum Readmissions and Morbidity Trajectories Among Pregnant Patients with Aortic Disease: A Retrospective Cohort Study Using the Premier Healthcare Database

Presenting Author: *Afif Kraitem, MD*

Background: The pregnant or postpartum state increases the risk of dissection in patients with aortic disease by fivefold.1,2 This is assumed to be due to heightened vulnerability associated with connective-tissue remodeling and fluid changes during and after pregnancy.3 Postpartum morbidity among obstetric patients with aortic disease remains poorly characterized. We aim to describe postpartum readmission patterns among patients with aortic disease and examine differences across aortic disease subtypes.

Methods: Using the Premier Healthcare Database (2016–2020), we identified pregnant patients with aortic disease using ICD-10 codes and categorized them into six subgroups: congenital non-genetic aortic disease, Marfan syndrome, Ehlers–Danlos syndrome (EDS), other congenital malformation syndromes, aortic aneurysm, and aortic ectasia. Primary outcome was all-cause readmission up to 1 year postpartum. We examined the indications for readmission, defined as cardiovascular (arrhythmia requiring intervention, heart-failure exacerbation, or aortic complications) or obstetric (hypertensive disorders, postpartum hemorrhage, or infection). Descriptive statistical analyses were performed.

Results: Among 10,745 delivery hospitalizations with aortic disease, the one-year readmission rate was 510 per 10,000 for patients with aortic disease. Among aortic disease readmissions, 21% were due to cardiovascular conditions typically for arrhythmia-related hospitalizations or heart-failure exacerbations. Obstetric readmissions occurred at around 73%, comparable to the general obstetric population and were predominantly related to hypertensive disorders. No postpartum readmissions for aortic dissection occurred in any aortic disease subgroup. Aortic surgery was required within 1 year postpartum in 0.6% of the aortic disease cohort.

Conclusions: Postpartum hospitalization among pregnant patients with aortic disease (5.1%) was nearly twofold higher than in the general obstetric population (2.8% or 287 per 10,000), driven primarily
by arrhythmia-related and heart-failure–related readmissions. Although no postpartum aortic

dissections occurred in this cohort, morbidity patterns varied by aortic phenotype, indicating that postpartum care needs should be created specifically for various aortic disease subtypes, particularly in the Marfan syndrome cohort which had a 35% readmission rate due to cardiovascular complications. Cardiac anesthesiologists, during their experience caring for patients with aortic disease, can aid the PHT by highlighting which patients are at highest risk of readmission for cardiac arrhythmias or heart failure based on the occurrence of these issues predelivery. A key study limitation is the inability to determine whether patients were readmitted to hospitals outside of their delivery hospital system. Though PHT patients tend to represent to their home delivery center, we may be underreporting complications in this population.

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Aortic Disease Subtype	Congenital (non-genetic)	Marfan syndrome	Ehlers-Danlos syndrome	Other congenital syndromes	Aneurysm	Ectasia	Unspecified Marfan	Overall aortic cohort
Total N	226	76	404	26	121	161	164	1,338
1-Year Readmission (% of readmissions)	17 (7.6%)	3 (3.9%)	10 (2.5%)	1 (3.8%)	12 (9.9%)	9 (5.6%)	11 (6.7%)	58 (5.1%)
Cardiovascular Readmissions* (% of readmissions)	3 (17.7%)	2 (66.7%)	2 (20%)	0 (0%)	4 (33.3%)	3 (33.3%)	3 (27.3%)	12 (20.7%)
Obstetric Readmissions** (% of readmissions)	14 (82.4%)	1 (33.2%)	8 (80%)	0 (0%)	8 (66.7%)	6 (66.6%)	7 (63.6%)	42 (72.4%)
Aortic Surgery within 1 year (% of subgroup)	1 (0.4%)	1 (1.3%)	0 (0%)	0 (0%)	5 (4.1%)	2 (1.2%)	2 (1.2%)	7 (0.6%)

* Cardiovascular readmissions include arrhythmia requiring intervention, heart-failure exacerbation, or aortic complications.
** Obstetric readmissions include hypertensive disorders, postpartum hemorrhage, and infection.

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SP 3 - Kiosk 2 - Poster 2
Postoperative anemia is associated with inferior postoperative outcomes after cardiac

surgery

Presenting Author: *Ryan Nicholson, MD, MPH*

Background: While the risks of preoperative anemia are well documented, the impact of postoperative anemia is less known. Postoperative anemia impacts up to 90% of patients after cardiac surgery. Research is required to characterize the relationship between moderate-severe and severe postoperative anemia and short- and long-term outcomes.

Methods: A claims and electronic health record database (TriNetX Research Network) was queried from 01/2005-10/2024. All cohorts contained cardiac surgery patients aged ≥18 years without preoperative anemia, i.e. with preoperative hemoglobin (hgb) ≥13 g/dL. We compared patients with moderate-severe anemia, i.e. hgb ≤11 g/dL, or severe anemia (hgb ≤8 g/dL) within 1-5 days postoperatively with those without. To assess any time-related changes, we examined 3 periods of interest: 14-, 30- and 365-day postoperative outcomes.

We propensity score matched for age, demographics, valvular surgery, use of cardiopulmonary bypass, neoplasms, chronic kidney disease, circulatory system diseases, respiratory system diseases, obesity/overweight, stroke, liver disease, perioperative blood product transfusions, nicotine dependence, and alcohol related disorders.

Outcomes of interest were mortality, acute kidney injury (AKI), stroke, deep venous thrombosis (DVT), pulmonary embolism (PE), acute myocardial infarction (MI), and red blood cell (RBC) transfusion.

Results: 129,178 patients had postoperative moderate-severe anemia while 11,872 patients did not. Post-match, 11,570 patients were in each cohort. In our 14-day, 30-day, and 365-day comparisons, postoperative anemia was associated with increased postoperative AKI, DVT, acute MI, and RBC transfusion risk (Figure 1). Mortality risk was increased at 14- and 30-day postoperatively but not at 365-days postoperatively. There was no difference in PE risk at any of the time points tested.

35,344 patients had postoperative severe anemia while 136,480 did not. Post-match, 33,649 patients were in each cohort. Postoperative severe anemia was associated with increased risk of all outcomes at all time points (Tables 1-3).
Conclusion: In this large, propensity-score matched study, postoperative moderate-severe anemia was associated with more inferior outcomes at 14-days postoperatively than 365-days postoperatively. In contrast, severe anemia was associated with inferior short- and long-term outcomes, suggesting a lower postoperative hemoglobin is particularly detrimental.

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Figure 1 – Impact of moderate-severe postoperative anemia (hemoglobin ≤11 g/dL) on 14-, 30-, and 365-day postoperative outcomes

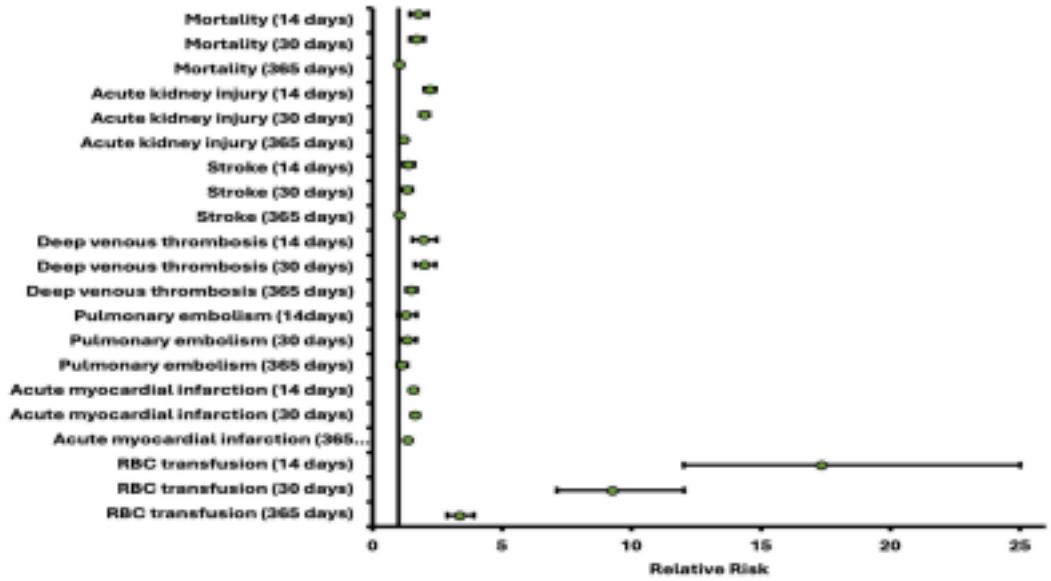


Table 1 – Impact of postoperative severe (hemoglobin≤8 g/dL) anemia among patients with preoperative hemoglobin ≥13.1 g/dL on 14-day postoperative outcomes

	Anemia (hgb≤8)	No anemia (hgb>8)	RR [95% CI]	P-value
Patients before match	35344	136480	--	--
Patients after match	33649	33649	--	--
Post-match age at surgery (years)	63 ± 12	63 ± 12	--	--
Mortality	3.56%	1.45%	2.46 [2.21-2.73]	<0.0001
Acute kidney injury	11.85%	6.47%	1.83 [1.74-1.92]	<0.0001
Stroke	3.40%	2.12%	1.61 [1.47-1.76]	<0.0001
DVT	2.62%	1.29%	2.03 [1.81-2.28]	<0.0001
Pulmonary embolism	1.17%	0.84%	1.40 [1.20-1.63]	<0.0001
Acute myocardial infarction	9.63%	7.82%	1.23 [1.17-1.29]	<0.0001
RBC transfusion	7.18%	1.39%	5.15 [4.67-5.68]	<0.0001

Table 2 – Impact of postoperative severe (hemoglobin≤8 g/dL) anemia among patients with preoperative hemoglobin ≥13.1 g/dL on 30-day postoperative outcomes

	Anemia (hgb≤8)	No anemia (hgb>8)	RR [95% CI]	P-value
Patients before match	35344	136480	--	--
Patients after match	33649	33649	--	--
Post-match age at surgery (years)	63 ± 12	63 ± 12	--	--
Mortality	4.80%	1.96%	2.45 [2.24-2.67]	<0.0001
Acute kidney injury	14.63%	7.83%	1.87 [1.79-1.95]	<0.0001
Stroke	4.42%	2.79%	1.59 [1.47-1.72]	<0.0001
DVT	3.79%	1.88%	2.01 [1.83-2.21]	<0.0001
Pulmonary embolism	1.74%	1.28%	1.35 [1.20-1.53]	<0.0001
Acute myocardial infarction	12.41%	10.06%	1.23 [1.18-1.29]	<0.0001
RBC transfusion	7.66%	1.67%	4.58 [4.19-5.02]	<0.0001

Table 3 – Impact of postoperative severe (hemoglobin≤8 g/dL) anemia among patients with preoperative hemoglobin ≥13.1 g/dL on 365-day postoperative outcomes

	Anemia (hgb≤8)	No anemia (hgb>8)	RR [95% CI]	P-value
Patients before match	35344	136480	--	--
Patients after match	33649	33649	--	--
Post-match age at surgery (years)	63 ± 12	63 ± 12	--	--
Mortality	8.35%	4.11%	2.03 [1.91-2.16]	<0.0001
Acute kidney injury	20.72%	11.78%	1.76 [1.70-1.82]	<0.0001
Stroke	7.35%	5.24%	1.40 [1.32-1.49]	<0.0001
DVT	5.87%	3.20%	1.84 [1.71-1.97]	<0.0001
Pulmonary embolism	2.99%	2.18%	1.37 [1.25-1.51]	<0.0001
Acute myocardial infarction	17.59%	14.50%	1.21 [1.17-1.26]	<0.0001
RBC transfusion	9.01%	2.43%	3.71 [3.44-4.01]	<0.0001

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SP 3 - Kiosk 2 - Poster 3

Comparison of Brachial and Central Pulse Pressures in Patients Undergoing Open Heart Surgery with Cardiopulmonary Bypass: Implications for the Acute Kidney Injury-predictive Pulse Pressure Threshold

Presenting Author: *Robert Thomas, MD*

Introduction: Patients undergoing cardiac surgery with cardiopulmonary bypass often exhibit reduced aortic compliance and microvascular dysfunction that elevate pulse pressure (PP). Elevated brachial PP (bPP), especially above 50 mmHg, has been linked to increased postoperative acute kidney injury (AKI). Central pulse pressure (cPP) may better reflect aortic load because peripheral amplification raises brachial systolic values above central aortic pressure. Because bPP and cPP may differ in prognostic significance, direct comparison with noninvasive technology is needed.

Methods: We performed a secondary analysis of prospectively collected hemodynamic data in adults undergoing elective coronary artery bypass grafting (CABG) or combined valve/CABG surgery. The subjects were classified into three pre-existing hypertension categories: No HTN (bSBP 128 to 105 mmHg), Moderate HTN (bSBP 159 to 141 mmHg), and Severe HTN (bSBP 191 to 161 mmHg). Each patient underwent pre-induction and post-induction of anesthesia assessments using the Sphygmocor Xcel system, with triplicate measurements averaged. Variables included bPP, cPP, and their difference. Group differences and induction-related changes were analyzed using standard statistical methods.

Results: The raw data is summarized in Table 1. Forty-eight subjects were included. Pre-induction bPP and cPP rose progressively with hypertension severity. After induction, both values declined substantially across groups, narrowing inter-group differences. PP≥50 mmHg was far more common in the Moderate and Severe hypertension than in the No hypertension group of patients. Across all groups and states, bPP remained higher than cPP, with differences of 6–18 mmHg. Induction produced significant decreases in both pressures, greatest in the Severe group. Pre-induction bPP and cPP differed significantly among hypertension groups; post-induction differences were attenuated, with only Severe versus No hypertension remaining distinct for bPP. Age correlated positively with bPP and cPP pre-induction but not post-induction.

Conclusion: bPP exceeded cPP at both time points, indicating they are not interchangeable

for perioperative risk assessment. Because cPP may more accurately reflect central arterial load, it may offer stronger predictive value for outcomes such as AKI. Noninvasive waveform technology enables feasible perioperative evaluation of central pressures. Further study should refine hypertension phenotypes using central pressures and confirm the prognostic utility of cPP in cardiac surgery.

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Group-State	bPP	cPP	Difference
No HTN Pre	52	42	10
Mod HTN Pre	69	55	14
Sev HTN Pre	89	71	18
No HTN Post	41	35	6
Mod HTN Post	46	37	8
Sev HTN Post	52	43	9

SP 3 - Kiosk 2 - Poster 4

From Vegetation to Fistulation: Aortic Valve Endocarditis Complicated by Aorto-Atrial Fistula

Presenting Author: *Yannan Huang, MD*

Introduction: Aorto-atrial fistulas (AAF) are a rare and complex condition that occur when there is blood flow between the aorta and either the right or left atrium. These malformations can be congenital or acquired, most commonly from infective endocarditis in which inflammation and bacterial destruction undermine the integrity of the aortic and atrial walls. We present a case of AAF in a patient with native aortic valve endocarditis who underwent bioprosthetic aortic valve replacement for severe aortic regurgitation (AR) and aortic root repair with pericardial patch.

Case Presentation: A 79 year old male with a history of hypertension and chronic low back pain presented with two weeks of worsening bilateral lower extremity edema. Labs were significant for anemia of chronic disease, hyponatremia, and elevated BNP. Left heart catheterization was unremarkable. Preoperative TEE revealed preserved ejection fraction, a sinus of valsalva aneurysm, and severe AR with a 3mm x 5mm echogenic mobile mass on the aortic valve consistent with endocarditis secondary to Granulicatella adiacens. Lumbar MRI ordered for history of low back pain was positive for spine discitis and osteomyelitis. Intraoperative TEE showed severe AR and associated holodiastolic aortic regurgitant flow reversal in the descending aorta. A mobile vegetation was again seen on the R coronary cusp (fig 1). New aorto-right atrial fistula was noted (fig 2) and suspected likely due to right atrial fistulation of previously seen sinus of valsalva aneurysm

secondary to infectious extension and erosion. Postprocedure TEE showed a well-seated bioprosthetic valve without AR and successful fistula closure. Postoperatively, the patient developed symptomatic slow atrial fibrillation requiring Mirca leadless pacemaker placement. The patient was discharged home on POD 10 to complete a six week course of IV ceftriaxone.

Discussion: In this case we describe an aorto–right atrial fistula secondary to infective endocarditis, ultimately requiring aortic root repair with a pericardial patch. Although uncommon, fistula formation represents a severe form of periannular extension, in which infection spreads beyond the valve annulus and erodes into adjacent cardiac structures. For endocarditis-related AAF, the incidence ranges from 1.6% to 5.8% of patients with native and prosthetic valves, respectively (1). The management of AAF requires definitive surgical intervention to remove infected tissue, restore valvular function, and close the fistulous tract. While both TTE and TEE can be used to diagnose AAF, TEE offers higher resolution of the valve and periannular complications due to higher signal-to-noise ratio and proximity to the heart and aorta (2).

Conclusion: While this patient's presentation was subtle, his progressive lower extremity edema and elevated BNP were consistent with left-to-right shunt physiology and developing heart failure. The causative organism, Granulicatella adiacens, is a rare but aggressive pathogen associated with increased rates of heart failure and perivalvular complications (3). This case highlights the importance of early recognition and treatment of aorto-atrial fistulas in patients with infective endocarditis, particularly when significant valvular dysfunction is present.

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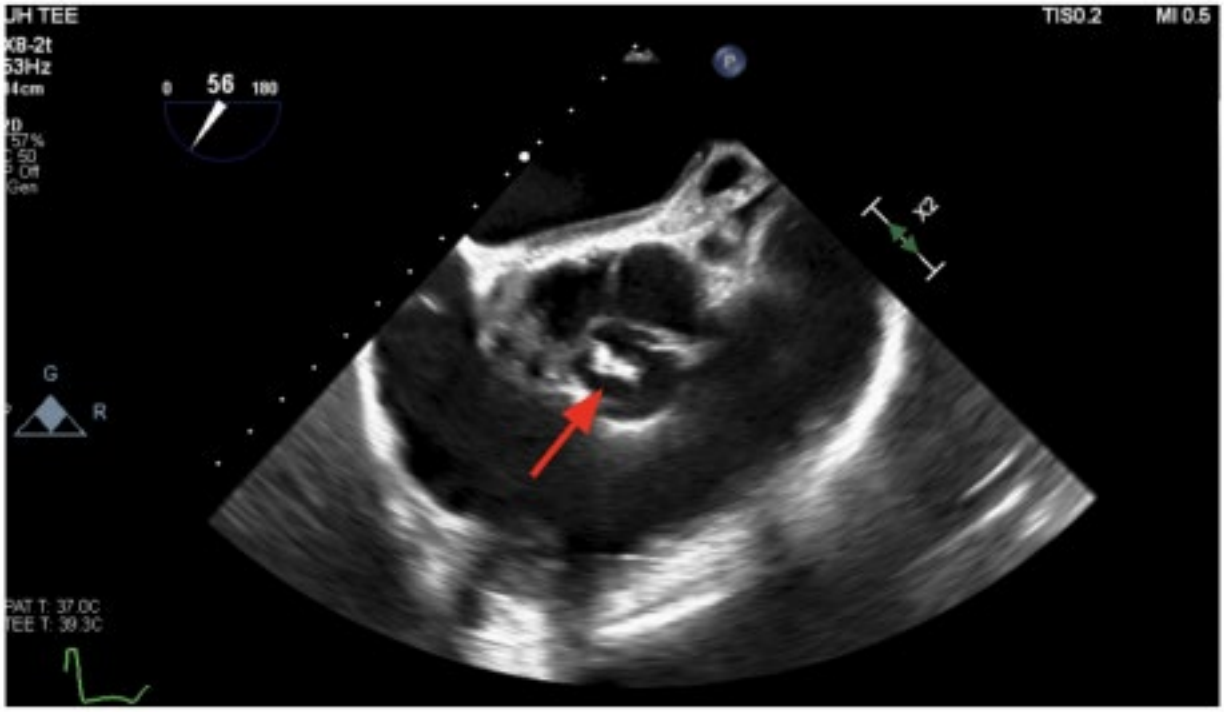


Figure 1. TEE midesophageal aortic valve short axis view with a mobile vegetation visualized on the R coronary cusp of the aortic valve

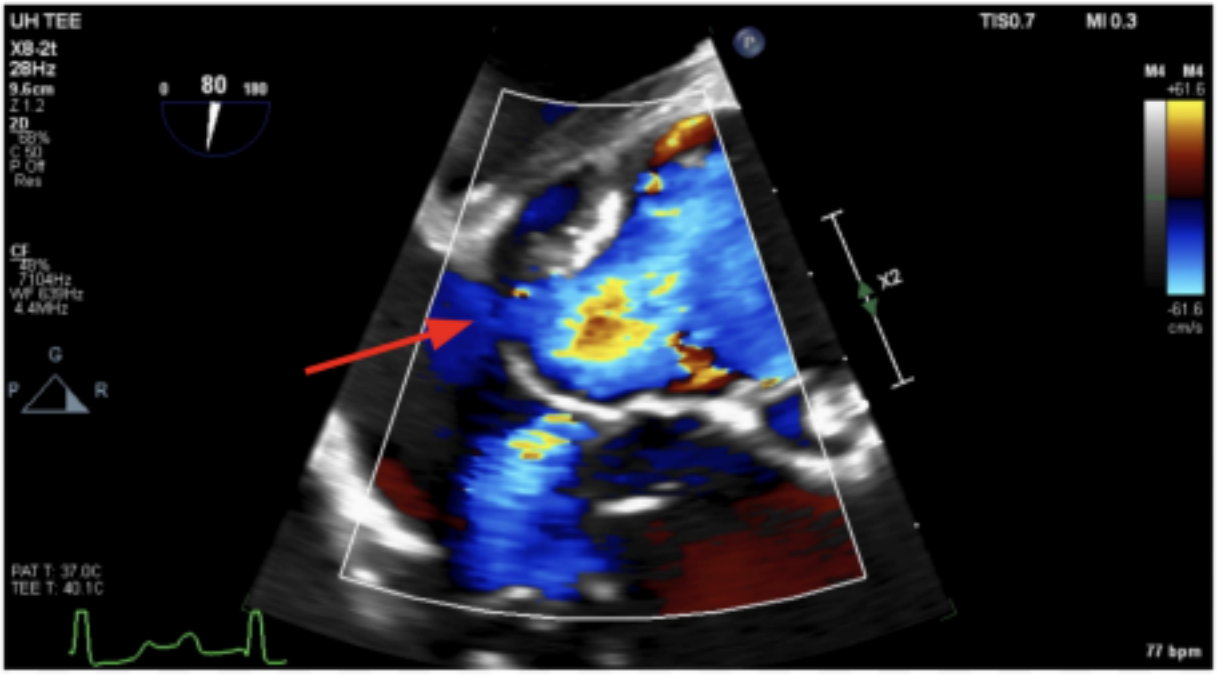


Figure 2. TEE midesophageal aortic valve short axis view with color flow doppler showing fistulous connection between right atrium and aortic root

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SP 3 - Kiosk 2 - Poster 5

Monitored Anesthesia Care Does Not Increase Complications in Adults with Congenital Heart Disease Undergoing Cardiac Catheterization

Presenting Author: *Amy Baker*

Introduction: Adults with Congenital Heart Disease (ACHD) is a rapidly growing population due to advances in surgical reconstruction and medical management of congenital heart disease over the last few decades. These patients often require additional interventions later in life to address residual disease and are generally considered high risk. While monitored anesthesia care (MAC) has been shown to be safe in the general population, no studies have reported outcomes of MAC in the ACHD population. Our aim was to identify whether MAC is associated with equal or higher rates of complications compared to general anesthesia (GA) in ACHD patients undergoing cardiac catheterization. We performed a single-center, retrospective review of ACHD patients undergoing cardiac catheterization at a nascent congenital heart disease program between July 2018 and May 2025. A total of 193 patients undergoing 228 catheterization procedures were identified through the PedCath database. Cases were divided into two groups based on anesthesia type, MAC (n = 28) or GA (n = 200). Extracted information included demographics, periprocedural data, ACHD diagnosis, anesthesia record, and relevant anesthesia risk factors. CHD category was defined using anatomic classification of lesions into minor, moderate, and complex by the American Heart Association. Data analysis was performed using StataBE version 19.5. Fisher's exact test was used for categorical variables and the Wilcoxon-Mann-Whitney for continuous variables to compare outcomes between MAC and GA groups.

Table 1 shows patients who received MAC had similar racial and ethnic backgrounds and anesthesia duration compared to GA patients. However, MAC patients were significantly older ($p < 0.001$) with higher BMI ($p = 0.049$) and were more likely to have asthma ($p = 0.049$) and obstructive sleep apnea ($p < 0.01$) compared to GA patients.

As shown in Table 2, MAC had a 100% procedural success rate (defined as completion of intended procedure) with no minor complications and one major complication (peri-procedural cardiac arrest requiring CPR, with return of spontaneous circulation within 1 minute).

Figure 1 demonstrates that the group of patients who received MAC during their catheterization procedures had roughly equivalent distributions of CHD severity compared to those who received GA. As shown in Figure 2, the type of catheterization procedures differed significantly between anesthesia groups (Fisher's exact test, p = 0.002). MAC was predominantly used for diagnostic procedures (57.1%) and pulmonary hypertension studies (25.0%), while GA was more commonly used for interventional procedures including angioplasty (13.8%), valve replacement (8.5%), and structural closures (10.1%).

Figure 3 shows the distribution of anesthesiologists who were assigned to the cases in the MAC and GA groups. The 28 MAC patients were assigned to 8 distinct anesthesiologists, indicating the low complication rate was not due to individual provider skill but rather reflects MAC safety across multiple physicians. MAC can be safe and appropriate for ACHD patients undergoing cardiac catheterization. Our results suggest that either MAC or GA can be chosen for these patients using shared decision making rather than utilizing risk factors for poor outcomes alone.

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Table 1. Patient Demographics by Anesthesia Type. *p = 0.034, **p= 0.049, ***p= 0.014

	Monitored Anesthesia Care	General Anesthesia
n	28	189
Age in years (median, [IQR])	39 (35-50)	29 (21-39)
Sex		
n, Female (%)	16 (57)	111 (56)
n, Male (%)	12 (43)	78 (41.3)
Race		
n, White (%)	27 (96.43)	164 (97.62)
n, Black (%)	1 (3.57)	16 (8.47)
n, Asian (%)	0	1 (0.53)
n, Decline (%)	0	8 (4.23)
Ethnicity		
%Hispanic	21	26
% Non-Hispanic	61	60
% Decline	18	14
BMI (median, [IQR])	31.57 [23.15-34.8]*	25.8 [22.25-30.8]*
Smoking = Yes (%)	7.7	12
Asthma = Yes (%)	18**	7
OSA = Yes (%)	25 ***	10
Anesthesia Time, hours (mean, (sd))	3.46 (1.06)	3.84(1.45)

Table 2. Primary Outcomes by Anesthesia Type.

	Monitored Anesthesia Care	General Anesthesia
Total n	28	189
Procedure Success, n (%)	28 (100)	185 (97.8)
Minor Complications, n (%)	0	17 (9)
Major Complications, n (%)	1 (3.57)	2 (1.06)
Mortality, n (%)	0	1 (0.53)
Hemodynamic Support, n (%)	0	4 (2.12)
Respiratory Support, n (%)	0	3

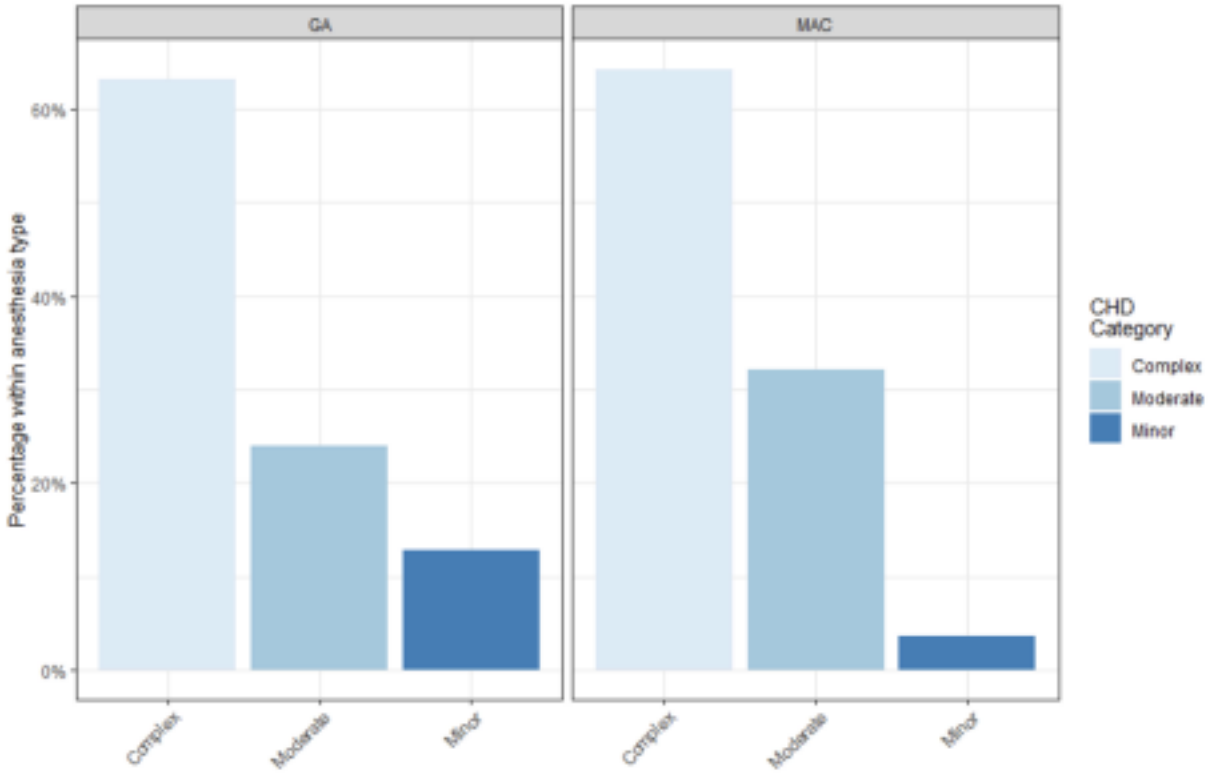


Figure 1. Distribution of Congenital Heart Disease Severity by Anesthesia Modality. Categories made to reflect structural anatomy severity scores as described by the American Heart Association.

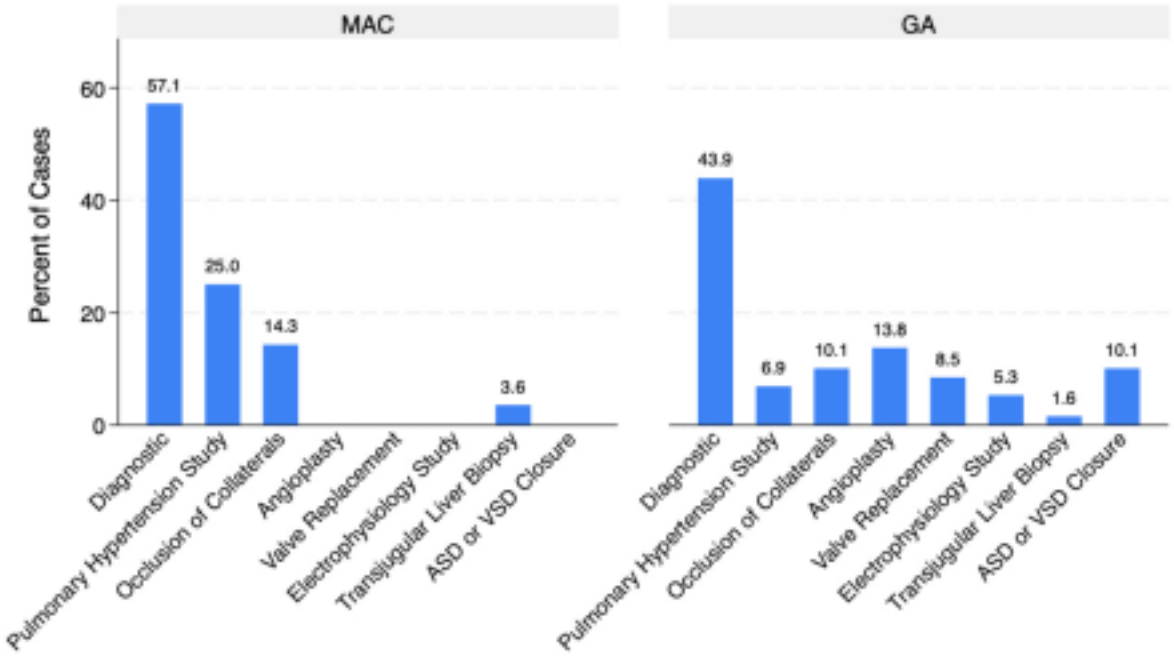


Figure 2. Distribution of Catheterization Procedures by Anesthesia Modality. Percent of total cases per anesthesia modality for each type of case is reported on columns.

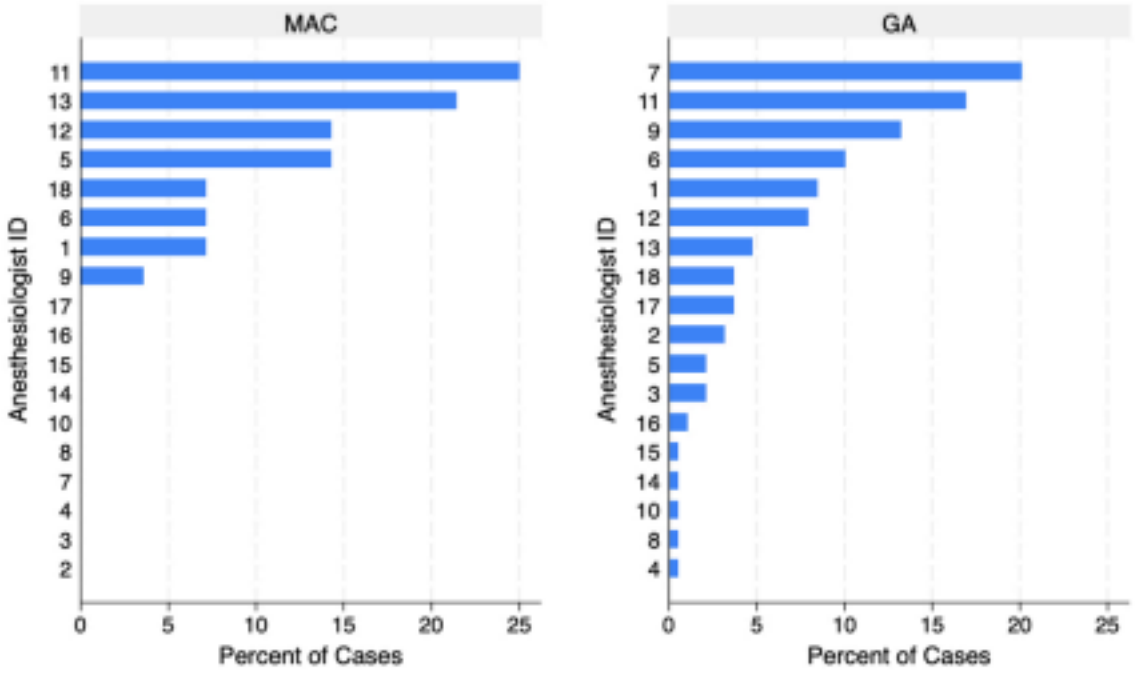


Figure 3. Distribution of Individual Anesthesiologists Performing Cases by Anesthesia Modality. Anesthesiologists were assigned a unique numerical identifier to maintain confidentiality.

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SP 3 - Kiosk 2 - Poster 6

When Inspiration Becomes Desperation: Salvage ECMO for Explosive Negative Pressure Pulmonary Edema

Presenting Author: *Vincent Franco, MD*

Introduction: Negative pressure pulmonary edema (NPPE) most commonly results from forceful inspiratory efforts against upper-airway obstruction, producing markedly negative intrathoracic pressure, abrupt increases in pulmonary capillary hydrostatic pressure, and rapid alveolar flooding. Although uncommon, NPPE may progress to life-threatening hypoxemia requiring advanced cardiopulmonary support.

Case Report: A 37-year-old morbidly obese woman with antiphospholipid syndrome (on enoxaparin and clopidogrel), May–Thurner syndrome, asthma, and opioid use disorder on methadone presented for elective lower-extremity debridement. Induction led to difficult mask

ventilation and transient desaturation, improving with neuromuscular relaxation. A 7.0 ETT was placed; severe wheezing and elevated airway pressures resolved with albuterol. The intraoperative course remained stable.

At emergence, after administration of 2 mg/kg sugammadex, the patient developed rapidly increasing serous and then frothy secretions within the ETT. At this point the patient exhibited adequate oxygenation and respiratory efforts and was following commands therefore the decision was ultimately made to extubate. Immediately post-extubation respiratory failure occurred with profuse pink frothy secretions and precipitous desaturation to 50%. Reintubation was complicated by massive secretions obscuring visualization; an ETT was finally placed after aggressive suctioning. Ventilation remained ineffective with peak pressures nearing 50 cmH_2O and severely limited tidal volumes. Approximately 1.5L of frothy, pink tinged fluid was suctioned. Persistent refractory hypoxemia prompted emergent cardiothoracic surgical consultation.

The patient became bradycardic and pulseless; ROSC was achieved after brief CPR and one round of epinephrine. She was emergently cannulated for VA-ECMO with immediate improvement in oxygenation. TEE demonstrated a hyperdynamic LV, normal RV, and no evidence of pulmonary embolism. Bronchoscopy confirmed diffuse frothy pulmonary edema throughout both bronchial trees. As cardiac function was preserved, she was transitioned to VV-ECMO for isolated respiratory failure, decannulated on day 3, extubated on day 5, and discharged home three weeks later.

Discussion: This case demonstrates fulminant NPPE immediately after reversal with sugammadex. Rapid restoration of diaphragmatic strength is a proposed mechanism increasingly recognized in sugammadex-associated NPPE. Comorbid asthma and difficult mask ventilation increased susceptibility to upper-airway obstruction and forceful inspiratory efforts. The abrupt clinical transition, massive airway fluid burden, and refractory hypoxemia are characteristic of severe NPPE and may mimic aspiration, bronchospasm, and acute PE. Early recognition and rapid escalation to ECMO were critical.

Conclusion: NPPE can progress from subtle airway obstruction to catastrophic hypoxemia within minutes. High-risk patients receiving rapid neuromuscular reversal warrant heightened vigilance during emergence. When conventional ventilation fails, early engagement of ECMO teams can be lifesaving.

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SP 3 - Kiosk 2 - Poster 7
It's Not Just a Transplant When Scleroderma Meets the OR: BLT with Off-pump CABG on VA-ECMO

Presenting Author: *Behnaz Hatami, MD*

Introduction: Scleroderma creates anesthetic challenges via pulmonary hypertension with RV dysfunction, esophageal dysmotility with aspiration risk, and Raynaud's phenomenon complicating perfusion assessment. We present management of a patient with these comorbidities undergoing bilateral lung transplant (BLT) with off-pump CABG on VA-ECMO.

Case Description: A 61-year-old woman with scleroderma, pulmonary hypertension on maximal

vasodilators, severe RV dilation/dysfunction, pericardial effusion, Raynaud's, and esophageal dysmotility presented for BLT and CABG. She was cachectic and functionally limited (< 4 METs).

Femoral access was obtained pre-induction for rapid VA-ECMO cannulation. Induction was tailored to preserve RV perfusion.

Despite fasting, bronchoscopy after single-lumen intubation revealed retained food with spillage into the lungs. After suctioning, a PA catheter and central line were placed, VA-ECMO was initiated via ascending aortic and femoral venous cannulation, and the ETT was exchanged to a left double-lumen. BLT via clamshell incision with donor lung downsizing and off-pump CABG (SVG to LAD) were completed on VA-ECMO. She was weaned from ECMO and transferred intubated to the ICU.

A T5-6 epidural catheter was placed for analgesia. Early imaging showed primary graft dysfunction that progressed to grade 3 at 72h. Her course was complicated by mucopurulent secretions requiring serial bronchoscopies, delirium, and atrial fibrillation. She was extubated but later needed re-intubation for acidosis and hypoxia; gastric contents were again seen at the cords. Achalasia-like aperistalsis and probable right vocal-cord paresis caused persistent aspirations. She ultimately underwent tracheostomy with a trach collar by day, nocturnal ventilatory support, and placement of a GJ feeding tube for long-term enteral feeding.

Volume overload, acute kidney injury, and concern for rejection versus pulmonary edema required titrated diuresis, a temporary tacrolimus holiday, and pulse-dose steroids. Over subsequent weeks, she was eventually decannulated and discharged to rehabilitation on room air with enteral nutrition.

Discussion: Patients with pulmonary hypertension are at high risk for RV failure during induction, positive-pressure ventilation, and particularly during one-lung ventilation; therefore, preemptive planning for ECMO should be considered. In scleroderma, esophageal dysmotility renders fasting unreliable, increasing aspiration risk and often necessitating prolonged airway protection, tracheostomy, and enteral feeding.

Raynaud's complicates peripheral perfusion monitoring as radial arterial line placement is contraindicated, and digital perfusion is unreliable.

Postoperatively, overlapping etiologies of respiratory failure require a multimodal assessment integrating imaging, bronchoscopy, hemodynamic monitoring, and immunologic evaluation. This case underscores the importance of disease-specific and multidisciplinary management in scleroderma patients undergoing complex BLT plus CABG with extracorporeal support.

Conclusion: This case highlights how scleroderma reshapes anesthetic strategy in BLT with concomitant cardiac surgery. Anticipatory ECMO planning, aspiration-focused airway management, and multidisciplinary care are essential.

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SP 3 - Kiosk 3 - Poster 1
Nitroglycerin for Prevention of Coronary Spasm During Arrhythmia Ablation: A Case Series
Presenting Author: *Stephanie Lam, MD*

Introduction: Pulsed field ablation (PFA) is increasingly used to treat atrial fibrillation (AF) and atrial flutter (AFL). The cavotricuspid isthmus (CTI) and portions of the mitral annulus lie near coronary vessels 1–4 and coronary vasospasm during ablation is a concern; VF and cardiac arrest 5 have been reported. Very aggressive (3 mg bolus) prophylactic nitroglycerin (NTG) has been used in multiple studies, though anesthetic considerations are not discussed.

Case 1
A 69-year-old male with AF/AFL, MVP, and OSA underwent PFA of the CTI. Pre-operative TTE showed possible HCM with preserved function. Under GA with arterial line monitoring, 1.6 mg IV NTG was given in 100–400 mcg increments over 13 minutes. Vasopressor support included norepinephrine 0.03 mcg/kg/min, phenylephrine 50 mcg/min, vasopressin 8 U. BP decreased from 120s/70s to 90s/50s (MAP > 65). No EKG changes occurred.

Case 2
A 52-year-old female underwent RHC and repeat PVI and CTI ablation for recurrent AFL. Her history included HTN, HFpEF, PAPVR (Qp:Qs 1.1), prior convergent MAZE with LAA ligation, and epicardial/endocardial ablations. TTE showed LVEF 60–65% and mild RA/RV enlargement, and mildly elevated left-sided filling pressures. Under GA, 1 mg IV NTG was given before ablation, followed by phenylephrine (500 mcg bolus, 40 mcg/min infusion). NIBP at 3-min intervals showed SBP increased from 100s to 130s mm Hg (MAP > 65). No EKG changes occurred.

Case 3

A 78-year-old female with HTN, HLD, CAD, CHF, cardiac amyloidosis with severe LVH (LVEF 45-50%), PH, and AF/AFL underwent PVI and CTI PFA. History included Prinzmetal angina with prior bradycardic, stroke-like episodes. She had mildly decreased RV function with RV hypertrophy and a dilated main PA consistent with the PH. Under GA, intracoronary NTG 7.5 mcg (2.5 mcg increments over 7 min) was given immediately before ablation. NIBP at 3-min intervals showed BP decreased from 90s/60s to 80s/40s (MAP decreased 65 to the low 50s) and responded to phenylephrine 100 mcg. No EKG changes occurred.

Discussion: Prophylactic NTG during PFA is increasingly used, but no standardized protocol exists. Published regimens vary from 1-3 mg IV bolus to infusions or repeat boluses, often exceeding doses typically used by anesthesiologists.2,3 For intracoronary dosing, one protocol recommends 1 mg.3 Similar variability occurred in our cases, with differences in dose, route, and monitoring (only one patient had an arterial line). Notably, Case 3 received only 7.5 mcg intracoronary. Despite the variability, hypotension was manageable with routine vasopressors. Attention should be paid to dose selection, as excessive vasodilation can abruptly reduce preload and systemic pressure, impairing coronary perfusion in patients with limited coronary reserve. Those with RV dysfunction or PH may be particularly vulnerable because of RV preload dependence and reliance on adequate right coronary perfusion.

Conclusion: Given unclear incidence of coronary vasospasm and absence of a defined protocol, practice remains heterogeneous. These findings highlight the need for close coordination with the interventional team and anesthesia-focused guidance on monitoring, anticipatory hemodynamic management, and risk stratification when NTG is used during PFA.

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Table 1.

Category	Variable	Case 1	Case 2	Case 3
Baseline Characteristics	Age / Sex	69 / M	52 / F	78 / F
	Key Cardiac History	AF/AFL, MVP, suspected HCM	Recurrent AFL, HFpEF, PAPVR, prior convergent MAZE, multiple ablations	CAD, CHF, cardiac amyloidosis, Prinzmetal angina, AF/AFL
	LV	LVEF 60-65%; mild asymmetric LVH; grade II diastolic dysfunction	LVEF 60–65%; normal size and wall thickness	LVEF 45–50%, severe LVH; infiltrative pattern
	RV	Normal RV size/function; RVSP 38 mm Hg	Mildly enlarged; normal systolic function	Low-normal systolic function; RVH
	Pulmonary Pressures	Mildly elevated	PASP 35 mm Hg; PCWP 17	PASP 41 mm Hg
	Coronary Evaluation	No significant CAD	No CAD work-up	Known vasospastic angina, negative stress test
	Other Comorbidities	OSA	HTN	HTN, HLD
	Prior Procedures	None	MAZE, LAA ligation, multiple ablations	None
Procedural & NTG Details	Procedure	CTI PFA	Repeat PVI + CTI PFA	PWI + CTI PFA
	Monitoring	GA with arterial line	GA with NIBP only	GA with NIBP only
	NTG Protocol	1.6 mg IV in 100–200 mcg increments over 13 min	1 mg IV bolus	7.5 mcg intracoronary (2.5 mcg increments over 7 min)
	Glycopyrrolate	0.2 mg	0.3 mg	0.3 mg
	Vasopressor Support	NE infusion, phenylephrine infusion, vasopressin bolus	Phenylephrine bolus + infusion	Phenylephrine bolus
	Hemodynamic Response	SBP 120s → 90s (MAP > 65)	SBP 110s → 100s; (MAP >65)	SBP 90s → 80s (MAP 65 → low 50s)
	ECG Changes	None	None	None
	Procedural Outcome	Completed; no spasm	Completed; no spasm	Completed; no spasm

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SP 3 - Kiosk 3 - Poster 2

Volume Removal Using Cardiopulmonary Bypass as an Independent Predictor of Acute Kidney Injury in Cardiac Surgery

Presenting Author: *Paul Pantapalangkoor, MD*

Background: Multiple methods of volume removal may be utilized during cardiopulmonary bypass (CPB) to mitigate hemodilution, with the aim of reducing red blood cell transfusion.1–3 Acute kidney injury (AKI) is a frequent complication in cardiac surgery and increases mortality.4 Volume removal may cause intravascular contraction and reduced renal perfusion. The influence of various volume removal strategies on the incidence of postoperative AKI remains an underexplored and modifiable variable. We investigated the relationship between total fluid removal during CPB and the incidence of acute kidney injury (AKI) in a large cohort of patients undergoing cardiac surgery.

Methods: This was a retrospective, single-center study of 1,922 adult patients undergoing cardiothoracic surgery requiring CPB. Patient demographic data and intraoperative variables were extracted from the electronic medical record. We performed univariate analysis of

retrograde autologous prime (RAP), ultrafiltration (UF), modified ultrafiltration (MUF), and zero-balance ultrafiltation (ZBUF) volumes and the incidence of postoperative AKI (defined by AKIN criteria). We additionally analyzed the relationship between AKI and total intraoperative fluid removal both as an absolute volume (mL) and indexed to body weight (mL/kg). Other known independent predictors of AKI were also included. Data was analyzed using a linear model reporting mean and standard deviation.

Results: Total volume removed using individual ultrafiltration techniques did not show a significant correlation with AKI (Table 1). However, total fluid removed during CPB (sum of RAP, MUF, UF, and ZBUF) was highly correlated with incidence of postoperative AKI. Patients with postoperative AKI had a higher average volume of fluid removed (2996.6 mL) compared to the non-AKI group (2504 mL) ($p < 0.001$). The correlation remained significant when total volume removal was indexed to body weight (AKI group: 39.6 mL/kg vs. non-AKI group: 33.1 mL/kg; $p < 0.001$) (Table1). The presence of DM, CHF, COPD, CKD were also associated with an increased incidence of postoperative AKI in univariate analysis.
Conclusion: In this retrospective study, volume removal via any one of the single hemoconcentration methods was not correlated with postoperative AKI in univariate analysis. However, total volume removal during CPB, which included RAP and all means of ultrafiltration, was significantly associated with postoperative AKI even when indexed to body weight. In univariate analysis, this finding likely suggests high-volume fluid removal utilizing the CPB circuit is a result of increased surgical complexity and longer CPB duration, and not necessarily the cause of AKI. The next step is to perform a multivariate analysis of the data set controlling for associated variables to determine if CPB-mediated fluid removal is independent risk-factor for postoperative AKI. The multivariate analysis will be completed shortly and provide a more nuanced understanding of perioperative fluid manipulation and renal protection. Multivariate analysis results will be included in the final submission.

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Postoperative AKI (AKIN Criteria) Univariate Analysis				
		AKI Absent (n = 1244)	AKI Present (n = 674)	P-Value
Age	Mean (SD)	59.6 (15.0)	61.7 (13.5)	0.003
BMI	Mean (SD)	26.7 (5.0)	27.3 (5.4)	0.014
Sex	M	849 (68.2%)	461 (68.4%)	1
	F	393 (31.6%)	213 (31.6%)	
DM	Present	110 (16.3%)	564 (83.7%)	< 0.001
	Absent	1135 (91.2%)	109 (8.8%)	
Congestive Heart Failure CHF	Present	140 (11.3%)	139 (20.6%)	< 0.001
	Absent	1104 (88.7%)	535 (79.4%)	
Chronic Obstructive Pulmonary Disease (COPD)	Present	22 (1.8%)	25 (3.7%)	0.009
	Absent	1222 (98.2%)	648 (96.3%)	
Chronic Kidney Disease (CKD)	Present	22 (1.8%)	60 (8.9%)	< 0.001
	Absent	1222 (98.2%)	614 (91.1%)	
Preoperative GFR	Mean (SD)	75.2 (13.1)	67.4 (22.9)	0.027
Total Volume Removed (mL)	Mean (SD)	2504 (1937.8)	2996 (2438.7)	**< 0.001**
Total Volume Removed per Kilogram (mL/kg)	Mean (SD)	33.1 (26.5)	39.6 (35.1)	**< 0.001**
RAP Volume (mL)	Mean (SD)	546.2 (190.1)	539.8 (202.8)	0.49
Modified Ultrafiltration (MUF)	Mean (SD)	937.2 (455.7)	956.5 (518.7)	0.73
Ultrafiltration (UF)	Mean (SD)	968.0 (827.8)	918.2 (823.6)	0.26
Zero-Balance Ultrafiltration (ZBUF)	Mean (SD)	898.6 (738.7)	914.0 (640.6)	0.86

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SP 3 - Kiosk 3 - Poster 3
End-of-life Trajectories in Patients with Temporary Mechanical Circulatory Support
Presenting Author: *Susana Arango, MD*

Background: Use of temporary mechanical circulatory support (tMCS), including extracorporeal membrane oxygenation (ECMO) and percutaneous ventricular assist devices (pVAD) (e.g., Impellas), has expanded across a wide range of clinical settings. Patients supported with tMCS exhibit highly variable clinical courses preceding death, ranging from rapid multi-organ failure to those deemed to be on a “bridge to nowhere”, and these end-of-life (EOL) trajectories in patients on tMCS are poorly understood. Our aim was to characterize these clinical trajectories by focusing on etiology and circumstances of death, and EOL care support.

Methods: We conducted a retrospective cohort study at two U.S. academic medical centers, including adult patients who received tMCS (veno-venous ECMO, veno-arterial ECMO, and pVAD) from January to September 2025. Cases were captured by querying operative and procedural documentation for terms associated with placement of ventricular assist devices, and extracorporeal membrane oxygenation. Demographic and clinical data were extracted for all patients.

Results: Among 255 patients receiving tMCS, 50 (20%) died during the index admission. In the patients who died, median age at death was 69 years (IQR 57, 77), and 16 (32%) were female. Indications for tMCS included coronary syndromes (34%), postcardiotomy shock (22%), extracorporeal cardiopulmonary resuscitation (ECPR, 18%), acute decompensated heart failure (16%), respiratory failure (8%), and distributive shock (2%). Initial tMCS included ECMO-only in 23 patients (46%), pVAD-only in 20 (40%), and both in 7 (14%). Median duration of tMCS was 7 days (IQR 1, 15), with 11 (22%) patients on support for < 24 hours. Withdrawal of life-sustaining tMCS occurred in 31 patients (62%). Of these, the reason for withdrawal was multiorgan failure (44%), perceived poor neurological prognosis (8%), nonsurvivable or non-operable hemorrhage (6%), “bridge to nowhere” (4%), or family request (2%). Liberation from tMCS due to anticipated recovery occurred in 16 patients (32%), these patients died a median of 4.5 days after decannulation (IQR 3, 21). Three patients (6%) died while on tMCS. Palliative care consultations were provided to 11 patients (22%) at a median of 7 days proximate to initiation of tMCS (IQR 2-14). In patients who received palliative care, the median time from cannulation to withdrawal was 28 days (IQR 11, 36) versus 10 days (IQR 2, 17) in those who did not. Spiritual support services were received by 33 (66%) patients or their families.

Conclusions: In our cohort, patients on tMCS most commonly died following withdrawal of

support, while death after liberation or during ongoing support was less frequent. Although multi-organ failure was the primary reason for withdrawal, family request and patients on a "bridge to nowhere" occurred, highlighting areas of ongoing interest. Palliative care consultations were inconsistent and occurred late in the course of tMCS, reflecting the clinical uncertainty and variability in patient trajectories. Improved characterization of these trajectories may guide timely and patient-centered approaches to withdrawal of support.

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SP 3 - Kiosk 3 - Poster 4

Sugammadex-Induced Anaphylaxis Exacerbating Systolic Anterior Motion of the Mitral Valve After Coronary Artery Bypass Surgery

Presenting Author: *Giovanna Patafio, DO*

Introduction: Anaphylaxis in the perioperative setting can produce circulatory collapse. The associated vasodilation, tachycardia, and hypercontractility can exacerbate systolic anterior motion (SAM) of the mitral valve, leading to dynamic left ventricular outflow tract obstruction (LVOT). Even in patients without structural predisposition, anaphylaxis has been reported to trigger significant LVOT obstruction. Sugammadex, widely used for reversal of aminosteroid neuromuscular blockade, is a recognized cause of perioperative anaphylaxis, with reported incidences of 0.02–0.04 percent and rapid onset characterized by hypotension, tachycardia, and flushing. This case illustrates how sugammadex-induced anaphylaxis acutely worsened pre-existing SAM after open-heart surgery, resulting in severe hemodynamic instability.

Case Report: A 74-year-old male with coronary artery disease, cardiomyopathy, hypertension,

type 2 diabetes, and sleep apnea underwent elective coronary artery bypass grafting with cardiopulmonary bypass. Preincision transesophageal echocardiography (TEE) demonstrated systolic anterior motion (SAM) without significant left ventricular outflow tract (LVOT) obstruction. The procedure and separation from bypass were uncomplicated, and the patient remained stable without vasopressor support, supporting a plan for on-table extubation. After chest closure, the TEE probe was removed and sugammadex was administered. Quickly, the patient developed severe hypotension, tachycardia, and urticaria. Anaphylaxis was suspected after other reversible causes were excluded. Epinephrine was administered without improvement. Repeat TEE revealed severe mitral regurgitation with worsening SAM and dynamic LVOT obstruction. These TEE findings indicated distributive shock exacerbating the patient's underlying physiology. Epinephrine was discontinued due to ionotropy, causing worsening hypercontractility. Phenylephrine, intravenous fluids, methylprednisolone, and diphenhydramine were administered, resulting in hemodynamic stabilization and return to baseline TEE findings. The patient remained intubated and was transported to the intensive care unit with sedation. He was successfully extubated the following day, and the remainder of his recovery was uncomplicated.

Discussion: Reports of anaphylaxis-associated LVOT obstruction highlight that vasodilation, tachycardia, reduced preload, and inotropic stimulation can exacerbate SAM. Hemodynamics in this patient improved with the restoration of systemic vascular resistance and cessation of ionotropes. This case emphasizes the essential role of intraoperative TEE in distinguishing dynamic obstruction from distributive shock, particularly in cardiac surgery patients with known SAM.

Conclusion: Sugammadex-induced anaphylaxis can precipitate or worsen dynamic LVOT obstruction in patients with pre-existing SAM, leading to rapid and severe hemodynamic compromise. Early recognition of both the allergic response and the mechanical component of shock, aided by intraoperative TEE, is critical for effective treatment.

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SP 3 - Kiosk 3 - Poster 5
Predicting postoperative atrial fibrillation after cardiac surgery using machine learning
Presenting Author: *Dong Ju Lee, MD*

Background: Postoperative atrial fibrillation (POAF) is a frequent complication after cardiac surgery that is associated with increased morbidity. However, existing risk scores have limited accuracy, highlighting the need for more effective prediction models. In this study, we developed and validated a machine-learning (ML) model predicting POAF following cardiac surgery using various perioperative variables.

Methods: A total of 6,859 adult patients who underwent cardiac surgery at Seoul National University Hospital (SNUH) or Seoul National University Bundang Hospital (SNUBH) between October 2004 and October 2021 were included. Top 20 perioperative variables were input to develop a prediction model based on extreme gradient boosting. The final model was internally validated using 516 patients from the SNUH test set and externally validated in 1,701 patients from the SNUBH cohort. The areas under the receiver operating characteristic curve (AUROC) and precision recall curve (AUPRC), and 95% confidence intervals (CIs) were evaluated to assess model performance.

Results: The incidence of new-onset POAF after cardiac surgery was 37.2%. The AUROC and AUPRC were 0.891 (95% CI, 0.859–0.920) and 0.861 (95% CI, 0.819–0.898), respectively, for internal validation and 0.726 (95% CI, 0.700–0.751) and 0.646 (95% CI, 0.607–0.684) for external validation.

Conclusions: A ML model developed with perioperative data demonstrated strong performance in predicting new-onset POAF after cardiac surgery. This approach may enable risk-tailored monitoring and prophylaxis while providing the basis for improved perioperative decision-making.

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SP 3 - Kiosk 3 - Poster 6

Perioperative Benzodiazepine Exposure and Acute Postoperative Respiratory and Infectious Complications in Cardiac Surgery: A Retrospective Study Using the INSPIRE Dataset

Presenting Author: *Ali Moinuddin, BSc*

Background: Cardiac surgery patients are highly susceptible to postoperative respiratory complications, sepsis, & pneumonia. Benzodiazepines (BZD) depress ventilatory drive, impair airway protection, & modulate immune function, fueling interest in BZD-sparing strategies. The INSPIRE dataset provides granular perioperative data from ~130,000 operations at Seoul National University Hospital (2011-2020). This study evaluated whether perioperative BZD use, by timing & dose, was independently associated with postoperative respiratory failure & major infections after cardiac surgery & aimed to characterize phase-specific risks.

Methods: In this retrospective cohort analysis using INSPIRE, adults 18-90 years old undergoing cardiac surgery were identified from procedural metadata. Perioperative BZD exposure (midazolam, diazepam, lorazepam, other) was determined from time-stamped administrations & categorized into three mutually exclusive groups: no BZD, preoperative/intraoperative BZD (± ICU dosing), & ICU-only BZD within the first 24 postoperative hours. The primary outcome was a composite of acute postoperative respiratory failure or major infection during the index hospitalization. Multivariable logistic regression adjusted for demographics, baseline functional status, operative & cardiopulmonary bypass durations.

Results: The cohort comprised 1,858 cardiac operations among 1,790 patients (mean age 63.7 years; 63.5% male), primarily ASA class 3 under general anesthesia. Overall, 767 operations (41.3%) received any perioperative BZD (141 preoperative/intraoperative, 607 ICU-only), whereas 1,091 operations had no BZD exposure. There were 76 primary composite events. In fully adjusted models, any BZD exposure was associated with higher odds of the composite outcome (OR 3.58, 95% confidence interval [2.02-6.33]), respiratory failure alone (OR 5.45 [2.30-12.92]), & major infection alone (OR 2.79 [1.37-5.68]). Relative to no BZD use, preoperative/intraoperative BZD showed increased odds of respiratory failure (OR 12.79 [4.73-34.63]) & major infection (OR 4.67 [1.90-11.49]), while ICU-only BZD was also associated with increased risk, with smaller effect sizes; respiratory failure (OR 4.02 [1.61-10.04]), major infection (OR 2.77 [1.33-5.78]). BZD-exposed patients had longer ICU stays (median 3.70-4.89 vs 1.84 days) & hospital stays (21-27 vs 15 days).
Discussion: Perioperative BZD exposure was common & associated with markedly higher odds of acute respiratory failure, major infection, & prolonged ICU + hospital LOS after cardiac surgery. These findings remain susceptible to confounding by illness severity & model overfitting given low event counts, but the phase-specific pattern suggests particular risk with pre- & intraoperative doses. Contemporary randomized evidence like the B-Free trial suggests BZD restriction may not substantially reduce delirium; in this subcohort no delirium was documented. BZD administration before & during cardiac surgery was associated with increased odds of postoperative acute respiratory failure & infection, supporting cautious, selective BZD use & motivating further dose-response & propensity-based analyses to reduce preventable complications without compromising essential sedation.

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SP 3 - Kiosk 3 - Poster 7
Temporary Impella 5.5 Assistance Facilitating Complex Soft-Tissue Resection and

Reconstruction for Massive Tumoral Calcinosis

Presenting Author: *Michael Guertin, MD*

Introduction: An estimated 56.2 million people live with heart failure (HF) globally, a number that continues to rise1. Advanced HF patients face substantial perioperative risk during non-cardiac surgery2. When surgery is required before pursuing therapies such as transplantation or durable LVAD support, the care team faces a difficult dilemma. This case highlights the use of temporary mechanical circulatory support (MCS) to enable resection of an extensive tumoral calcinosis in a patient undergoing dual-organ transplant evaluation.

Case Report: A 52-year-old male in combined heart and kidney transplant evaluation presented with a right hip tumoral calcinosis measuring 13x17.7x20.2 cm. Aspirations provided only transient relief, and he reported progressive pain and limited ambulation. Given its size and functional impact, resection was required to advance transplant candidacy. His history included nonischemic cardiomyopathy (EF 25%), 2+ mitral regurgitation, ventricular tachycardia with ICD, ESRD from polycystic kidney disease on IHD, hypertension, and prior stroke.

After multidisciplinary discussion, the team proceeded with resection and flap reconstruction supported by Impella 5.5. Preoperative catheterization showed mildly elevated filling pressures and a cardiac index of 2.2 L/min/m^2. A PA catheter was left in place. After brachial arterial line placement and induction with etomidate and rocuronium, he was intubated and a TEE probe inserted for monitoring and device guidance. Baseline PA pressures were 49/29 (mean 40) mmHg with CVP 28 mmHg; low-dose epinephrine and vasopressin were started.

After heparinization (11,000 units; ACT 245 seconds), the Impella was inserted via the right axillary artery, positioned 5.3 cm below the aortic valve with improved PA pressures of 34/23 (mean 28) mmHg and CVP 21 mmHg prior to right hip incision. Repositioning to left lateral decubitus triggered arrhythmias, corrected with TEE-guided adjustment. Intraoperatively, several episodes of decoupling between Impella flow and native LV contraction were managed with inotropes and P8 suppor

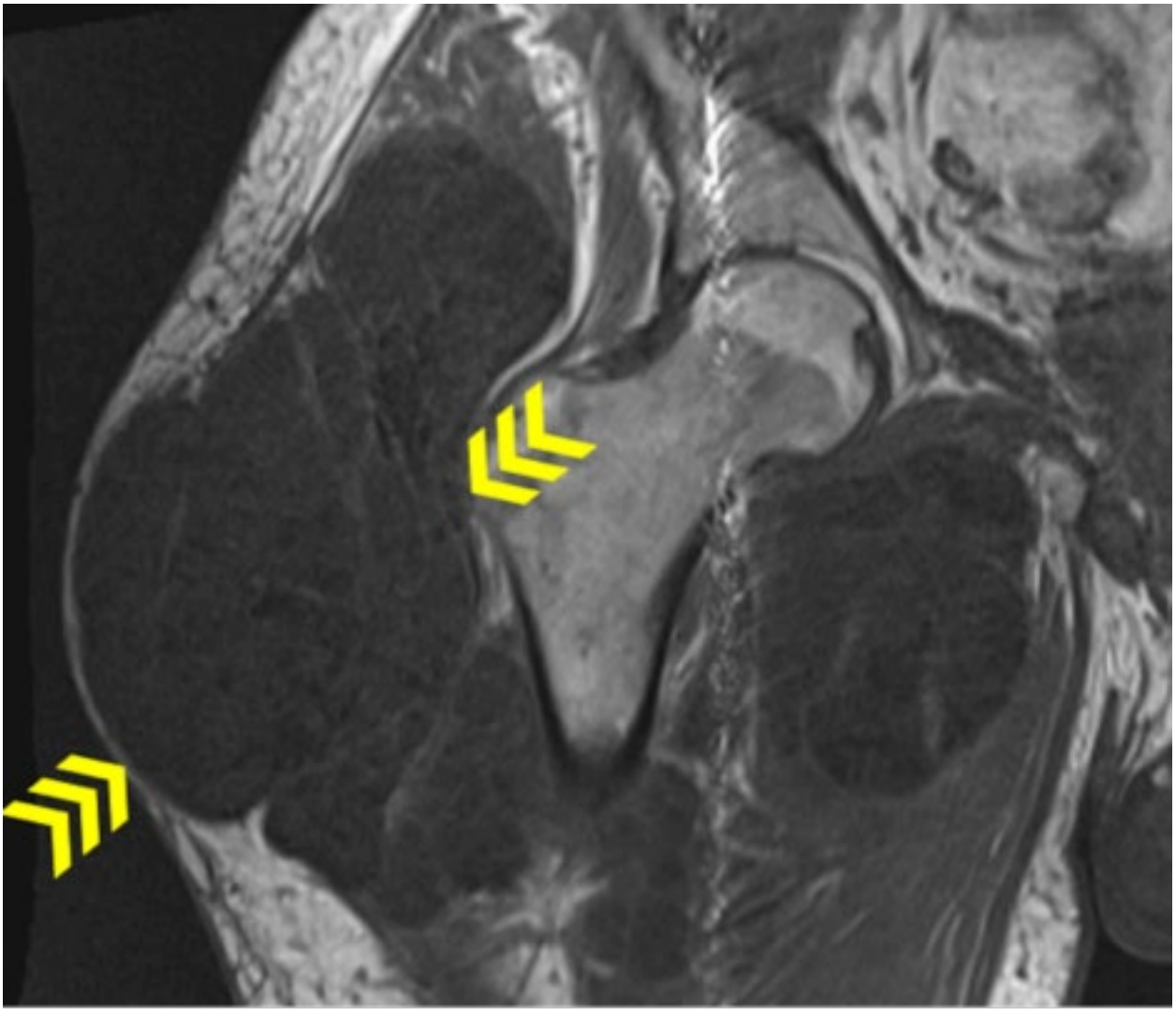
Postoperatively, he was transferred to the SICU and successfully extubated within four hours. He was weaned off inotropes, and the Impella was removed on POD 4. The patient was discharged home on POD 7. He is now listed as status 5 for heart transplantation and status 7 for kidney transplantation.

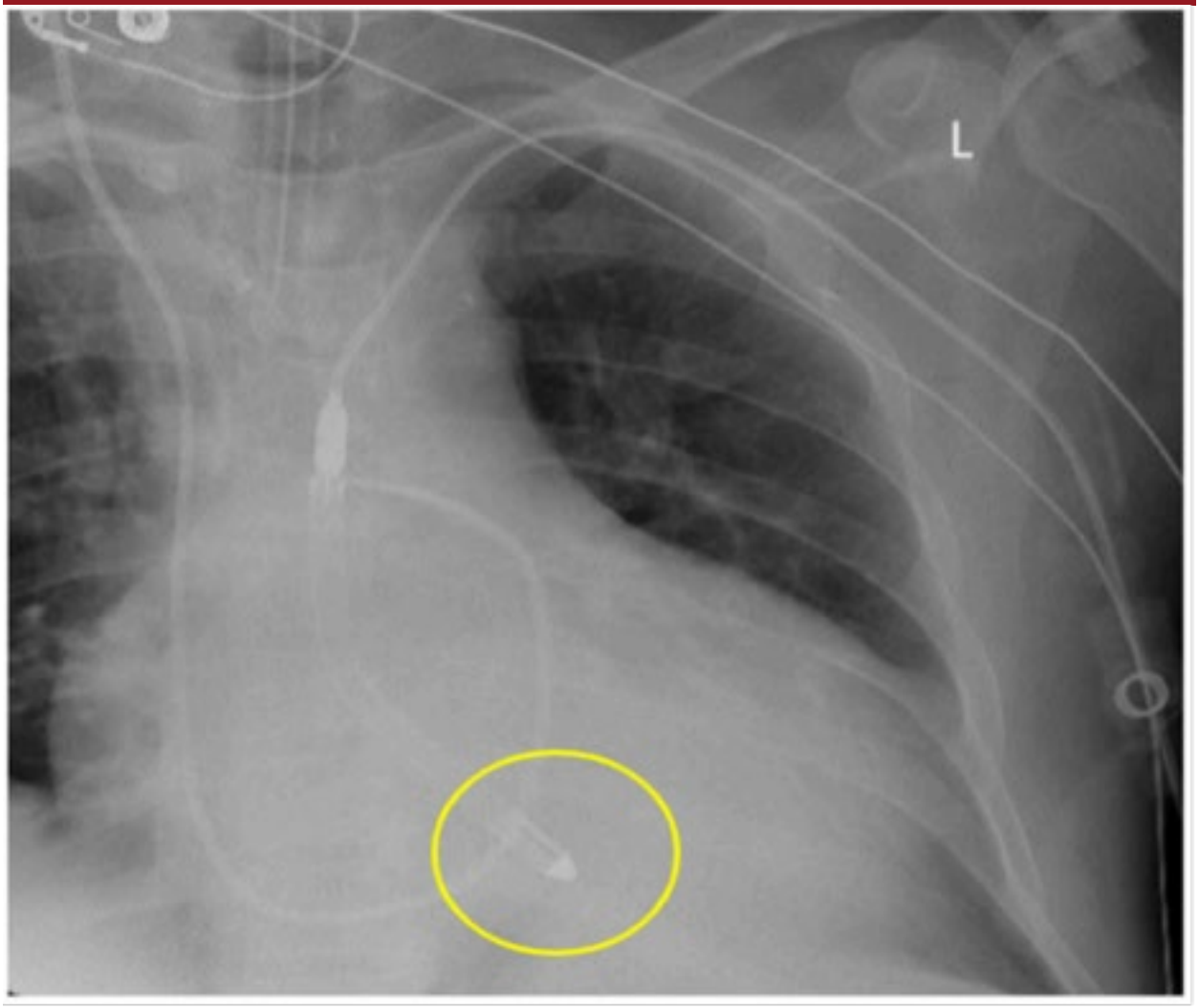
Discussion: As non-cardiac surgical needs rise among advanced HF patients, temporary MCS can support high-risk individuals through essential procedures. Prior work, including its use for bariatric surgery in pre-transplant candidates3, shows its ability to expand surgical eligibility. In this case, the Impella 5.5 provided stability and preserved perfusion during periods of device-native decoupling, enabling safe completion of a complex resection. Continuous TEE

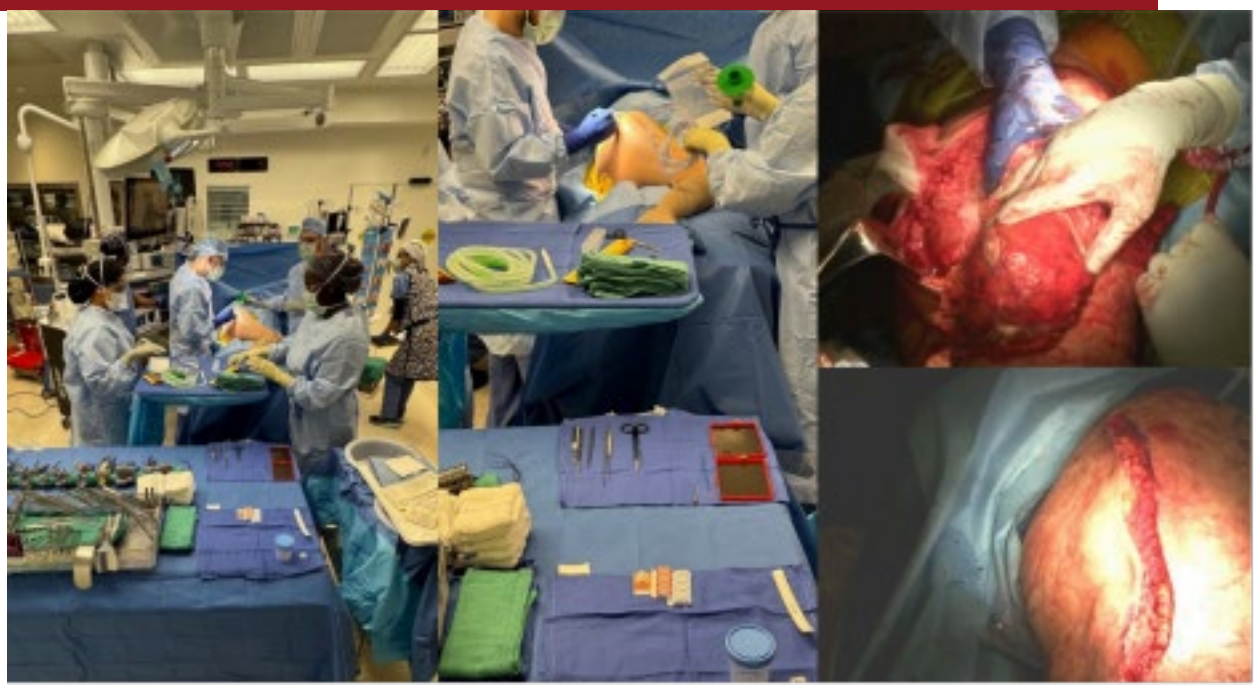
and invasive monitoring were essential for positioning and support optimization.

Conclusion: Temporary Impella 5.5 support enabled safe resection of a massive tumoral calcinosis in a patient with severely limited cardiac reserve, facilitating continued transplant evaluation. Temporary MCS should be considered for selected advanced HF patients requiring high-risk non-cardiac surgery.

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SP 3 - Kiosk 4 - Poster 1

Anesthetic Management of Complex Caval Reconstruction in a Patient with Extensive Collateral Circulation due to Concurrent SVC and IVC Occlusions

Presenting Author: *Michael Guertin, MD*

Introduction: Systemic lupus erythematosus (SLE) increases the risk of venous thromboembolism and renal failure. Patients with SLE and end-stage renal disease (ESRD) often require long-term hemodialysis, predisposing them to venous stenosis and thrombosis. Occlusions of the SVC and IVC promote extensive venous collateral pathways1. During cardiac surgery, these collaterals

create major challenges regarding vascular access and hemorrhage control. This case highlights the need for rigorous anesthetic planning and multidisciplinary coordination to optimize outcomes in this high-risk population.

Case Report: 26-year-old female with SLE, ESRD, and chronic SVC occlusion s/p stenting, presented after hemodynamic instability during dialysis. Workup revealed extensive mediastinal collaterals and calcified SVC/IVC thromboses extending into the right atrium. The patient was scheduled for sternotomy, caval embolectomy and reconstruction.

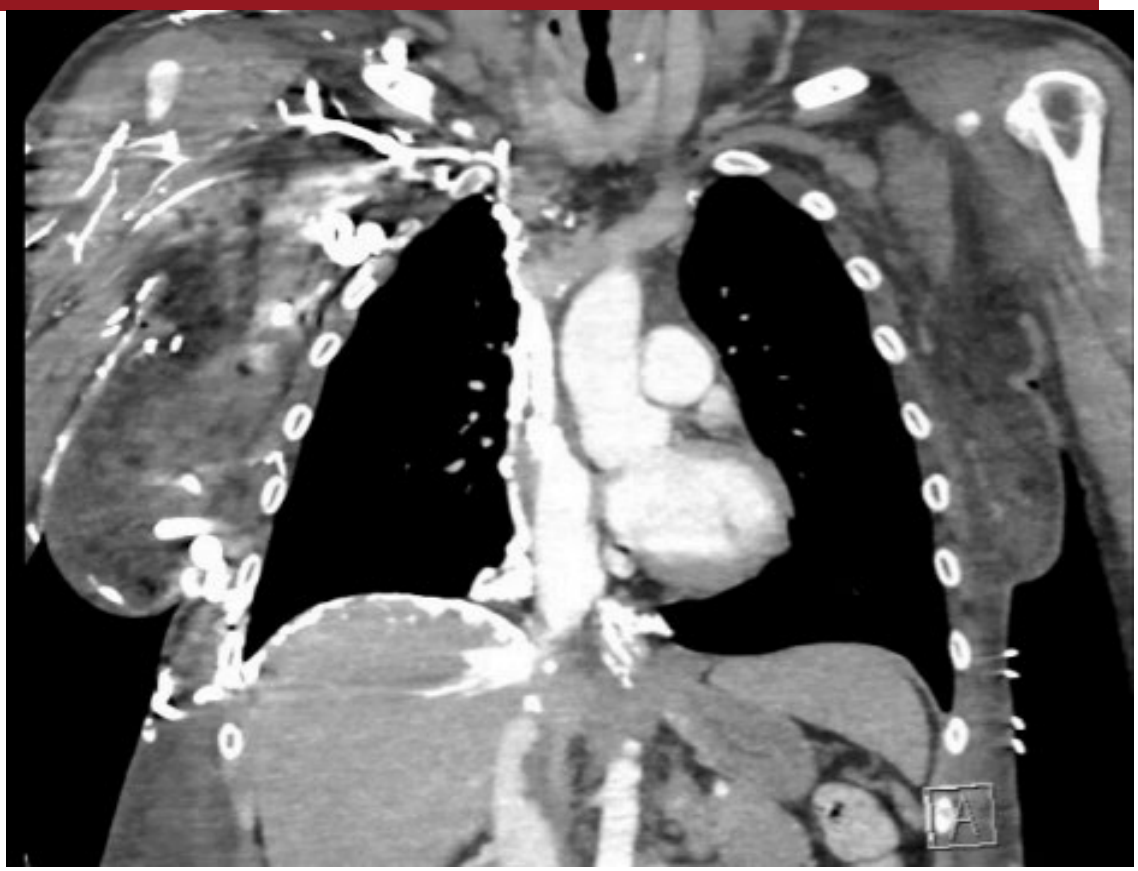
Anesthetic planning prioritized large-bore access and massive transfusion readiness. After a stable induction, a radial arterial line was placed. A Left IJ 9 Fr MAC introducer was placed after visualization of a stenotic right IJ. Additional access included two PIVs and a femoral dialysis catheter. Baseline CVP was remarkably elevated at 65-70 mmHg. With a starting Hgb of 7.1 g/dL and in anticipation of significant bleeding, RBC units were administered prior to incision. On sternotomy, significant hemorrhage was noted from collateral vessels and the sternum itself. Aggressive transfusions and vasopressors were required until initiation of cardiopulmonary bypass (CPB) via ascending aorta and right atrium cannulas. An extensive, calcified mass extending from the level of confluence of the innominate veins to the right atrium was resected. IVC thrombectomy was unsuccessful and deferred for later percutaneous intervention. Weaning from CPB revealed hemodynamic improvements (CVP 33-35 mmHg). Post-bypass coagulopathy was managed in routine fashion with blood products and desmopressin. The patient was extubated on POD 2and later discharged to acute rehab on POD 10.

Discussion: Perioperative management of chronic caval obstruction is uniquely challenging, particularly with combined SVC and IVC occlusions. Chronic SVC syndrome alone increases venous pressures2. Dual obstruction is rarely described and creates extreme venous hypertension with dependence on delicate mediastinal collaterals, effectively forming a high-flow vascular malformation. This anatomy presents a rare etiology for uncontrolled hemorrhage at incision and sternotomy. Unlike common sternotomy
complications, hemorrhage in these cases can stem from indistinct, diffuse collaterals that lack a discrete focal point to repair. Consequently, surgical control is exceptionally difficult, and initiating CPB often offers limited benefit. Because venous obstruction lies proximal to routine cannulation sites, traditional bypass configurations may not adequately decompress of the upstream collateral network.

Conclusion: This case of chronic caval obstruction requiring complex surgical revision underscores the critical necessity for meticulous, individualized anesthetic planning and preparedness to aggressively manage ongoing, uncontrolled hemorrhage for a prolonged period.

Image Uploads





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SP 3 - Kiosk 4 - Poster 2
RV Dysfunction in Open, Minimally Invasive, and Robotic Assisted Mitral Valve Repair
Presenting Author: *Melissa Kimlinger, MD*

Background: Robotic and minimally invasive approaches for mitral valve repair or replacement (MVR) are increasingly performed to enhance perioperative recovery. However, evidence is limited regarding whether these approaches reduce right ventricular (RV) dysfunction. We evaluated patients who underwent open, minimally invasive, or robotic MVR with transthoracic

echocardiographic follow-up within the first year post-operatively to assess differences and trends in RV function.

Methods: Patients who underwent MVR at a quaternary care hospital from January 2021 through June 2024 and had at least 1 postoperative transthoracic echocardiogram within 365 days were included. Patients undergoing concomitant major cardiac procedures such as aortic surgery, additional valvular procedures, coronary artery bypass grafting, or others, were excluded. Continuous variables are reported as median (interquartile range) and categorical variables as percentages. Logistic regression was used to assess factors associated with postoperative RV dysfunction.

Results: A total of 278 patients were included: 128 open, 93 minimally invasive, and 57 robotic. Median age was 64 years (IQR 55-71), 60% were male, and median BMI was 25.7 kg/m2 (IQR 23.1-28.5). Age, sex, and BMI were similar across approaches. Aortic Cross clamp and cardiopulmonary bypass times were longer in minimally invasive and robotic procedures than open (aortic cross clamp time: minimally invasive 123 [105-145] minutes, robotic 123 [100-150] minutes, open 111 [94-141] minutes, $p< 0.01$; cardiopulmonary bypass time: minimally invasive 207 [179-229] minutes, robotic 207 [176-241] minutes, open 162 [131-189], $p< 0.01$) minutes.

Postoperative RV dysfunction occurred in 16.1% of open MVR's, 13.3% of minimally invasive, and 10.0% of robotic MVRs. Left ventricular dysfunction was observed in 18.5% of open, 9.5% of minimally invasive, and 6.0% of robotic patients. In multivariable analysis, robotic approach was associated with decreased odds of RV dysfunction compared to open approach (OR 0.19, $p=0.02$). Minimally invasive approach was not associated with significantly lower rates of RV dysfunction (OR 0.39, $p=0.08$). Preoperative left ventricular ejection fraction ($p< 0.01$) and cardiopulmonary bypass time ($p< 0.01$) independently predicted postoperative RV dysfunction.

Conclusions: Our findings indicate that patients undergoing robotic MVR may have lower incidence of RV dysfunction as compared to Open MVRs. The results need to be interpreted cautiously as patients selected for robotic procedures often have fewer comorbidities. Prior studies have shown similar results – minimally invasive and robotic approaches have longer procedural times, yet similar rates of mortality, stroke, major morbidity, and RV function. Further larger multi-institutional studies are needed to assess the generalizability of our findings.

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SP 3 - Kiosk 4 - Poster 3

Comparing Clinical Quality and Readability of Large Language Models (ChatGPT versus MediSearch) in Patient Education for Transcatheter Aortic Valve Replacement

Presenting Author: *William Jones, DO*

Background: Untreated aortic stenosis (AS) will eventually lead to heart failure [1]. This condition particularly affects aging populations, with more than 3% of adults over 75 experiencing the disease [2]. The introduction of transcatheter aortic valve replacement (TAVR) has fundamentally changed how we manage severe symptomatic AS. As TAVR expands to broader patient populations, inadequate patient understanding of risks, benefits, and procedural steps remains a barrier to shared decision-making. LLMs have the potential to enhance patient comprehension, yet their reliability in explaining advanced cardiovascular interventions has not been defined. This study systematically evaluated two LLMs, ChatGPT and MediSearch, to characterize differences in reliability, quality, and readability of health information. The findings may inform development of safer, more effective AI-driven patient engagement systems in cardiovascular care.

Methods: One hundred patient-focused TAVR questions were obtained from Google's "People Also Ask" feature. The free versions of ChatGPT and MediSearch were queried. Outputs were independently evaluated across two domains: modified DISCERN scores for reliability and quality, and Flesch-Kincaid and Coleman-Liau scales for readability. We summarized DISCERN and readability indices (Flesch–Kincaid, Coleman–Liau) and assessed normality for each paired comparison using the Shapiro–Wilk test. For ChatGPT vs MediSearch, we performed paired t-tests when both measures were normally distributed and Wilcoxon signed-rank tests otherwise. Effect sizes were estimated for the paired comparisons. Statistical significance was set at $p < 0.05$. Analyses were conducted in R (version 4.3.1) using tidyverse, ggpubr, and rstatix.

Results: MediSearch achieved significantly higher DISCERN scores ($p < .001$), demonstrating stronger reliability and alignment with evidence-based recommendations. MediSearch averaged a DISCERN score of 4.52 (95% CI: 4.42 ± 0.104), whereas ChatGPT averaged a score of 2.66 (95% CI: 2.66 ± 0.139). MediSearch also generated significantly lower readability scores ($p < .001$), indicating language better suited for patients with higher literacy levels. MediSearch averaged a Flesch-Kincaid score of 9.954 (95% CI: 9.954 ± 0.483) and a Coleman-Liau index averaged 14.915 (95% CI: 14.915 ± 0.511). In comparison, ChatGPT averaged a Flesch-Kincaid score of 15.097 (95% CI: 15.097 ± 0.392) and a Coleman-Liau index averaged 16.442 (95% CI: 16.422 ± 0.316). (Table 1)

Conclusion: MediSearch delivered greater clinical quality and more readable content than ChatGPT, indicating that specialized LLMs may better support patient understanding of TAVR. By providing information that is both reliable and accessible, MediSearch shows potential to improve shared decision-making and reduce knowledge gaps in structural heart care. These findings highlight the value of continued evaluation of AI-generated educational materials and the importance of designing tools that balance accuracy with clarity. This early investigation

contributes foundational evidence to guide future development of safe, effective digital communication in cardiovascular medicine.

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ChatGPT	DISCERN	Mean	2.66	MediSearch	DISCERN	Mean	4.52
		Median	3.00			Median	5.00
		SD	0.70			SD	0.52
		95% CI	2.66 ± 0.139			95% CI	4.52 ± 0.104
	FK	Mean	15.10		FK	Mean	9.95
		Median	15.30			Median	10.05
		SD	1.97			SD	2.43
		95% CI	15.097 ± 0.392			95% CI	9.954 ± 0.483
	CL	Mean	16.44		CL	Mean	14.91
		Median	16.28			Median	15.01
		SD	1.59			SD	2.58
		95% CI	16.442 ± 0.316			95% CI	14.915 ± 0.511

Table 1: Summary Statistics Displaying Each Measurement Modality by NLP Models

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SP 3 - Kiosk 4 - Poster 4

Longitudinal Analysis of Diversity in ACTA Fellowship Programs from 2012 to 2024

Presenting Author: *Dhanush Bathala, DO*

Background: With increasing interest in the field of anesthesiology, and a diverse patient population there is a need to ensure that the next generation of anesthesiologists represent the communities they serve. Although some studies have examined the current representation of anesthesiology residents, few studies have aimed to examine whether progress in representation is being achieved in adult cardiothoracic anesthesiology. The aim of this study is to examine trends in race/ethnicity and gender representation amongst adult cardiothoracic anesthesiology (ACTA) fellows between 2012-2024.

Methods: Self-reported race/ethnicity and gender data for ACTA fellows were collected from the Accredited Council for Graduate Medical Education (ACGME) Data Resource books for academic years 2011-2012 till 2023-2024, the first and recent years reported respectively.

Gender categories reported included male, female, non-binary, and not reported. Due to limited sample size in the non-binary and not reported groups, only fellows identifying as male or female were used for analysis. Race/ethnicity categories reported included White, Asian, Black, Hispanic, American Indian/Alaskan Native, Native Hawaiian/Pacific Islander, Multiple Ethnicity, Other, and Unknown. To ensure a sufficient sample size for analysis, those who identified as Black, Hispanic, American Indian/Alaskan Native, and Native Hawaiian/Pacific Islander were grouped together as underrepresented in medicine (URM) in line with the AAMC's definition. Additionally those who identified as multiple ethnicities, other, and unknown were grouped as Other.

Chi-square analysis was performed to assess significance in demographic differences across years, and linear regression was performed to evaluate trends in percent representation of groups over time. Analysis was performed using SPSS,with $p < 0.05$ considered significant.

Results: Between 2012to 2024, a total of 2,397 ACTA fellows were analyzed. During this time, fellows identifying as female ranged from 25 to 33%, and those identifying as male ranged from 67 to 75%. Over this period, chi square analysis showed a significant change in the gender distribution ($p < 0.01$). Linear regression analysis indicated a significant upward trend in the percentage of female fellows ($B=0.71$, $p < 0.01$), indicating a gradual increase in female representation.
From 2012-2024, fellows who identified as URM comprised between 8 to 14% of fellows, with the lowest rates of representation between 2012-2014. White fellows comprised the largest portion of fellows, commonly between 54 to 62% annually. Over this period, chi-square analysis showed a significant change in the race/ethnicity distribution over time ($p < 0.05$). Linear regression showed a significant increase in URM fellows ($B=0.42$, $p < 0.05$) and Asian fellows ($B=0.33$, $p < 0.05$) over this period.

Conclusion: Our findings reveal that the race/ethnicity and gender of ACTA fellows have slowly evolved since 2012. During this period, there was a significant increase in the percentage of female fellows and the percentage of fellows identifying as URM and Asian. While positive, these findings still highlight the need to continue promoting pathways to sustain growth in representation in Adult Cardiothoracic anesthesiology.

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SP 3 - Kiosk 4 - Poster 5

Maternal morbidity among pregnant patients with aortic diseases: A Retrospective Cohort Study Using the Premier Healthcare Database

Presenting Author: *Daniel Aziz, MD*

Background: Cardiothoracic anesthesiologists often participate in peripartum planning for patients with aortic disease.1,2 Despite substantial heterogeneity across aortic pathologies, maternal outcomes are frequently generalized under a single “aortopathy" umbrella.3 We aim to characterize outcomes of pregnant patients with aortic disease by phenotype and evaluate whether morbidity varies by mode of delivery. We hypothesize that although morbidity may be slightly higher than in the general obstetric population, morbidity events are rare, differ by disease subtype, and not associated with mode of delivery.

Methods: We conducted a retrospective cohort study of delivery hospitalizations in the Premier Healthcare Database (2016–2020). Patients with aortic disease were identified using ICD-10 codes and classified into congenital aortic disease, heritable Marfan syndrome, Ehlers–Danlos syndrome (EDS), other congenital malformation syndromes, aortic aneurysm, and aortic ectasia. Mode of delivery was categorized as vaginal, intrapartum cesarean, or planned cesarean. Primary outcomes included aortic dissection, cardiovascular morbidity, and Severe Maternal Morbidity events relevant to aortic disease. Secondary outcomes were ICU admission, postpartum hemorrhage, hypertensive disorders of pregnancy, thromboembolic events, and readmission within one year.

Results: Among 4.8 million delivery hospitalizations, 9,299 (0.19%) involved aortic disease. Patients with aortic disease were older (mean 30.4 vs 28.1 years) and had higher rates of chronic hypertension (9.1% vs 3.6%) and connective-tissue disorders (7.7% vs 0.1%). Mode of delivery in the aortic disease cohort (53.3% vaginal, 10.8% intrapartum cesarean, 34.9% planned cesarean) differed slightly from the general obstetric cohort (66% vaginal, 11% intrapartum cesarean, 23% planned cesarean). Cardiovascular complications occurred in 60 per 10,000 delivery hospitalizations among patients with aortic disease, compared with 10 per 10,000 in the general obstetric population— reflecting overall low event rates. Morbidity varied by aortic disease subtype, with the highest rates among patients with heritable syndromes (Marfan 3.7%, EDS 3.4%) and aneurysmal disease (9.9%). No codes for aortic dissection were present in any aortic disease subgroup during the delivery hospitalization.

Conclusion: Pregnant patients with aortic disease experienced low absolute rates of major cardiovascular complications at delivery hospitalization. Cesarean delivery was more common in the aortic cohort than in the general obstetric population consistent with expert recommendations for cesarean delivery in patients with extreme dilation. Subtype-specific differences in morbidity reinforce the importance of individualized peripartum planning based on disease phenotype, as emphasized in the recent ESC guidelines. A key limitation is that administrative data lack aortic size measurements, preventing validation of guideline-based risk stratification by aortic diameter.

Image Uploads

	Congenital	Heritable Marfan	Heritable EDS	Other Congenital	Aneurysm	Ectasia	Unspecified Marfan (Q87.4)	Overall
Total N	205	76	101	28	121	161	261	1130
Cesarean Delivery	38.7%	39.5%	26.5%	14.6%	58.9%	45.3%	37.6%	44.9%
Vaginal Delivery	48.8%	35.5%	57.7%	42.3%	24.8%	32.9%	42.7%	46.7%
Intrapartum CD	16.6%	9.2%	11.1%	11.5%	9.2%	10.6%	8.7%	10.8%
Cardiovascular Morbidity Composite	3.9%	3.9%	0.9%	0.0%	8.8%	1.9%	1.3%	3.8%
Obstetric Hemorrhage	4.4%	10.5%	2.7%	3.8%	5.8%	4.3%	4.9%	4.8%
ICU Admission	13.2%	11.8%	1.7%	0.0%	14.0%	6.8%	4.9%	8.8%

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SP 3 - Kiosk 4 - Poster 6

Retrospective analysis of arterial line failure in a population of cardiac surgery patients

Presenting Author: *Christina Le, MD*

Background: Arterial catheter failure is described as inaccurate waveform or loss of blood return with withdrawal of the arterial line.1 This phenomenon is disconcerting in cardiac surgery due to the occurrence of sudden hemodynamic shifts, frequent blood sampling, and nonpulsatile flow during bypass. Most studies on arterial line failure take place in the intensive care unit (ICU) setting, one of which found the incidence to be 26%.2 Older studies in the cardiac surgery population found the arterial line failure rate to be 4.9%.3 Thus, the data on arterial line failure rate and associated risk factors is inconsistent and outdated. The goal of our study is to characterize

the incidence of radial arterial line failure (RALF) in cardiac surgery and identify its associated risk factors.

Methods: This is a single-center, IRB-approved retrospective study of coronary artery bypass graft (CABG) surgeries from January 2023 to December 2024. The study population included patients undergoing CABG with cannulation of the radial artery for arterial line access. We defined RALF as placement of a second arterial line intraoperatively or during postoperative day 0 or 1 in the ICU. Cases with complete data for arterial line laterality during preoperative left heart catheterization (LHC) and CABG were included. Univariate (UVM) and multivariate (MVM) regression models were performed to determine factors associated with RALF.

Results: There were 341 CABG cases during the study period, 277 of which met study criteria. (Figure 1) The incidence of RALF was 20.2%. Characteristics significant in the UVM included intraoperative inotrope use (P = 0.03) and duration of time during the procedure when mean arterial pressure (MAP) was < 50 mm Hg (P = 0.01). In the MVM, both variables remained significant (P = 0.04 and P = 0.001, respectively). Additionally, cannulation of the right radial artery for CABG (P = 0.01) and same arterial line laterality as preoperative LHC (P = 0.02) were associated with higher odds of RALF. (Table 1)

Conclusion: Our analysis revealed the incidence of RALF to be comparable to previously reported rates during hospitalization.1 Interestingly, intraoperative inotrope use, duration of time with MAP < 50 mm Hg, CABG radial arterial line placement on the right, and radial artery cannulation on the same side as preoperative LHC were strong risk factors. This may be explained by both poor perfusion of the catheter from decreased cardiac output and prior insult to the vessel during LHC, leading to poor waveform on arterial pressure monitoring. Identification of cardiac surgery patients at increased risk may be crucial to preoperative planning to anticipate and prevent arterial line failure.

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Figure 1. Study inclusion and exclusion criteria. *CABG, coronary artery bypass graft. LHC, left heart catheterization.*

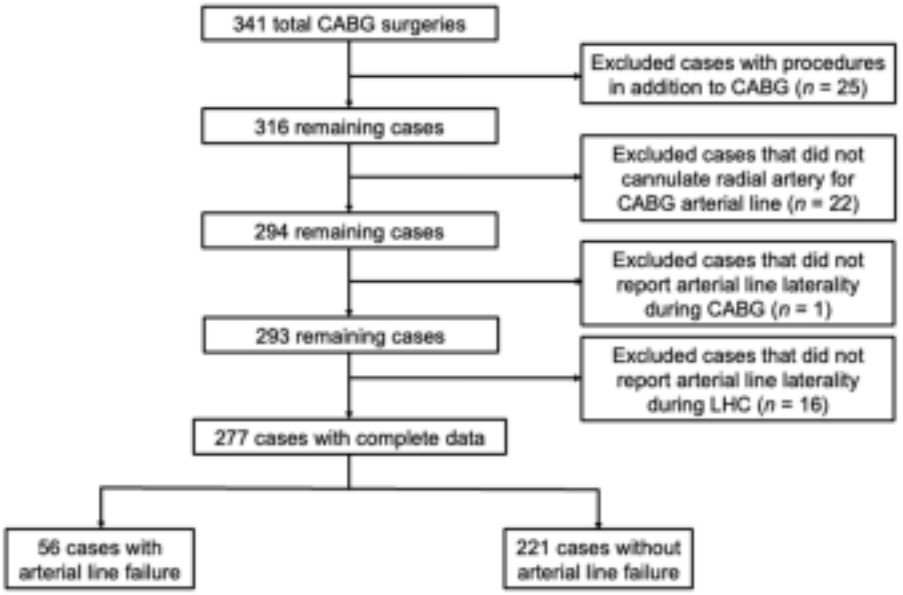


Table 1. Univariate and multivariate regression analysis for identification of variables associated with radial arterial line failure.

Variable	Arterial line failure cases (n = 56)	Cases without arterial line failure (n = 221)	Univariate *P*-value	Multivariate *P*-value
Age	64.6 ± 11.2	65.1 ± 10.3	0.73	0.78
Male (vs Female)	42 (75%)	186 (84%)	0.11	0.10
Body mass index (kg/m²)	28.1 ± 5.1	27.8 ± 5.4	0.64	0.69
Preoperative hemoglobin (g/dL)	13.4 ± 1.6	13.2 ± 2.0	0.36	0.20
Preoperative creatinine (mg/dL)	1.25 ± 1.68	1.42 ± 1.90	0.53	0.74
Preoperative ejection fraction (%)	52.0 ± 15.8	55.8 ± 13.6	0.12	0.42
LHC radial arterial line laterality on right (vs left)	54 (96%)	219 (99%)	-	-
CABG radial arterial line laterality on right (vs left)	41 (73%)	143 (65%)	0.23	**0.01***
Same laterality for LHC and CABG radial arterial lines	31 (55%)	128 (58%)	0.73	**0.02***
Duration of time between LHC and CABG (days)	44.2 ± 77.1	38.7 ± 51.2	-	-
Anesthesia case duration (min)	502.9 ± 97.2	490.0 ± 100.9	0.36	0.20
Median preinduction systolic blood pressure (mm Hg)	151.1 ± 28.7	158.3 ± 32.9	0.14	0.48
Median preinduction diastolic blood pressure (mm Hg)	79.8 ± 21.2	81.7 ± 34.3	0.69	0.53
Cardiopulmonary bypass time (min)	118.1 ± 41.4	155.2 ± 40.4	0.64	0.48
Intraoperative vasopressor use	55 (98%)	204 (92%)	0.11	0.12
Intraoperative inotrope use	35 (63%)	103 (47%)	**0.03***	**0.04***
Duration of time with MAP <50 mm Hg	17.2 ± 26.9	10.5 ± 13.6	**0.01***	**0.001***
Intraoperative pRBC transfused (mL)	228.5 ± 375.1	206.3 ± 381.1	0.70	0.70
Intraoperative plasma transfused (mL)	58.8 ± 152.6	51.1 ± 155.5	0.74	0.72
Intraoperative platelets transfused (mL)	173.6 ± 287.4	162.5 ± 267.4	0.79	0.75
Intraoperative cryoprecipitate transfused (mL)	25.2 ± 52.4	17.3 ± 47.5	0.28	0.46
Maximum intraoperative glucose (mg/dL)	254.1 ± 42.7	249.3 ± 46.2	0.48	0.79
Hospital length of stay (days)	10.8 ± 6.5	10.2 ± 7.9	-	-

Continuous data reported as mean ± standard deviation. Categorical values expressed as *n* (percentage). * denotes statistical significance, with *P* < 0.05. *LHC, left heart catheterization. CABG, coronary artery bypass graft. MAP, mean arterial pressure. pRBC, packed red blood cells.*

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SP 3 - Kiosk 4 - Poster 7

Management of Massive Hemorrhage and Acute Tamponade During Leadless Permanent Pacemaker Implantation Attempt

Presenting Author: *Joseph Israeli, MD*

Introduction: Leadless permanent pacemaker implantations carry risks. The rate of cardiac perforation is low (0.8%), but it is double that of traditional pacemaker implantation (0.4%) (1), and its sudden presentation demands constant readiness.

Case Report: An 84-year-old woman with a history of breast cancer presented for leadless pacemaker implantation under MAC for syncope due to sinus pauses. Fulminant hypotension occurred shortly before device deployment. Cardiac perforation was confirmed when injected contrast opacified the pericardial space. She was promptly induced with etomidate, intubated, and resuscitative transfusion was begun. TEE revealed a large pericardial effusion and thrombus. Emergent pericardiocentesis was performed. Hemodynamics improved with transfusion, but the effusion persisted. Protamine (for prior heparin), PCC, and factor seven were administered, but at this point 1-2 liters (L) of blood was drained without any sign of slowing. Cardiothoracic surgery was called emergently. After median sternotomy the pericardium was opened gradually. Hemodynamics improved as blood was released, and clot was removed. Three perforations were repaired; the primary one was dime sized and on the inferomedial margin of the RV, while two smaller ones were on the anterior RV. Following hemostasis, RV and RA epicardial leads were sewn to the heart and then tunneled. CPB was not used, and the chest was then closed. TEE showed normal heart function without residual effusion. She received about 4L in blood products total (PRBC, FFP, cryoprecipitate, and platelets), and 6L of blood total were drained to the salvage system. She arrived to the ICU intubated without pressors. She was extubated the next day, and the remainder of her course was uncomplicated.

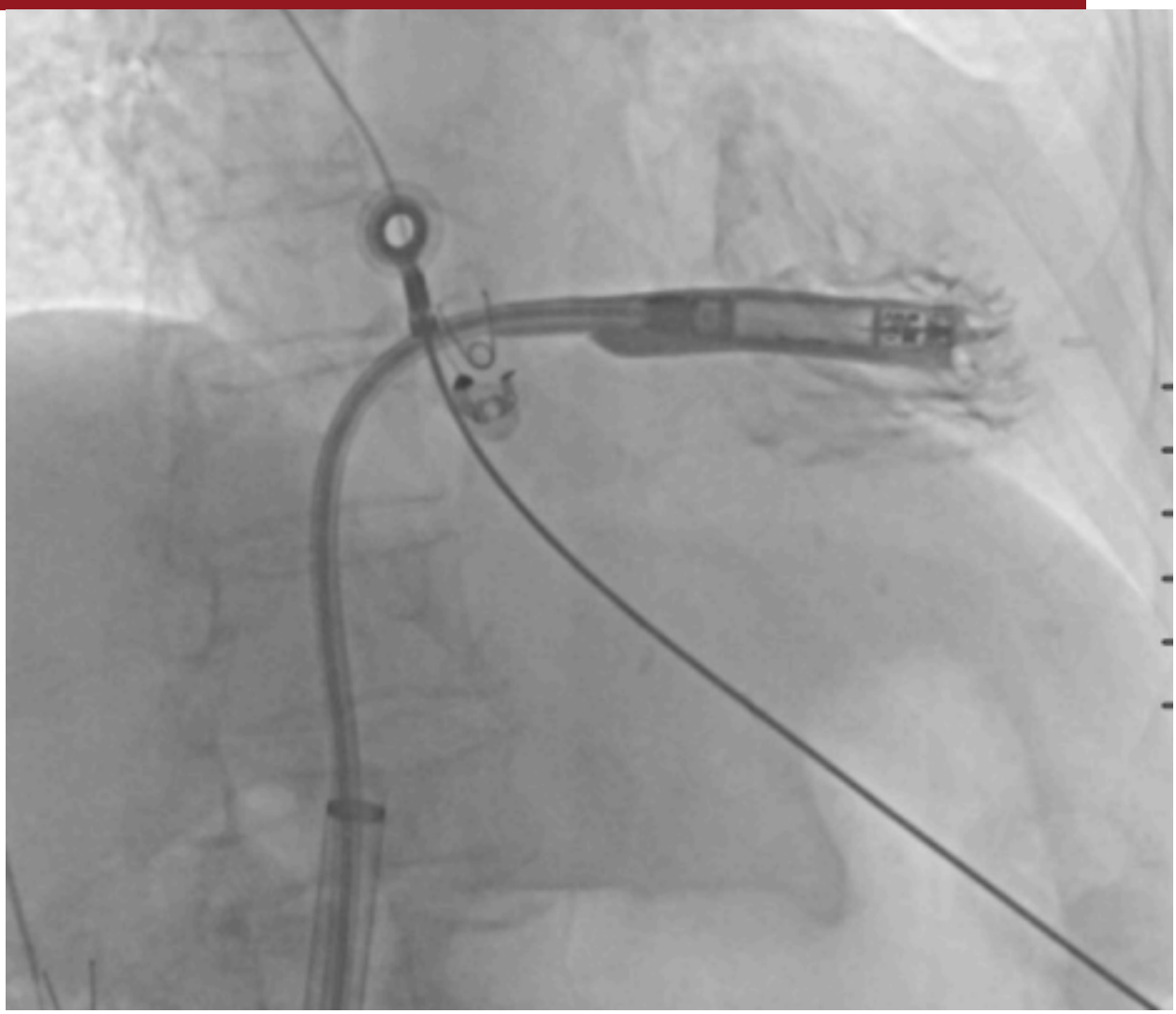
Discussion: In pericardial hemorrhage, the patient is afflicted by a dual shock state: both hypovolemic from the hemorrhage and obstructive from the tamponade. Expeditious hemorrhage control (via balanced blood product resuscitation and hemostasis) and relief of the obstruction (via pericardiocentesis or surgical drainage) are crucial. When the pericardium has thrombus, surgery is preferred to pericardiocentesis (2). Positive pressure ventilation can decrease cardiac output, so

airway management and hemodynamics must be vigilantly monitored. We avoided CPB to prevent heparinization and exposure to bypass. The mnemonic “fast, full, and tight” (describing heart rate, preload, and afterload respectively) may help guide temporization but risks oversimplification; one must dynamically evaluate preload, contractility, and afterload to optimize cardiac output. The most specific echocardiographic finding in tamponade is diastolic RV collapse, but RA inversion and notable respiratory variation in ventricular inflow are also classic (3). Slow relief of the tamponade is key to lowering the risk of pericardial decompression syndrome (PDS). This manifests with hemodynamic decline within hours of decompression despite initial improvement, as the rapid preload increase overwhelms the heart (4).

Conclusion: Early identification is key to treating cardiac tamponade. One must temporize appropriately and ensure definitive treatment. TEE is excellent for guiding treatment and confirming resolution.

Image Uploads

Pros and Cons of Leadless vs. Conventional Pacemaker				
Pros:	Lower infection risk (lead and pocket infections notably)	No lead related complications	Less device-related reintervention	Simplified procedure for patients with limited venous access
Cons:	Suboptimal degree of AV synchrony	Higher risk of cardiac perforation during placement	Inability to pace the conduction system	Lack of experience with long term device extraction



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SP 3 - Kiosk 5 - Poster 1
Transcatheter Aortic Valve Mass Aspiration in a High-Risk Patient
Presenting Author: *Joseph Israeli, MD*

Introduction: Life-threatening cardiac conditions affect people of all ages and physical health. Emerging techniques are being applied for new indications to treat patients at too a high risk for conventional surgery.

Case Report: Patient was a 93-year-old woman with a history of multiple recent admissions in the last two months for unstable angina and NSTEMI (despite imaging confirming nonocclusive CAD), acute on chronic CHF, severe mitral regurgitation, moderate aortic stenosis, persistent atrial fibrillation on anticoagulation, found to have a symptomatic aortic valve mass. Echocardiography demonstrated a pedunculate mass measuring 13mm x 10mm on the base of the left coronary cusp adjacent to the commissure. It prolapsed into the left main coronary artery during systole. After multidisciplinary meetings, a percutaneous approach to mass aspiration was selected due to her advanced age and comorbidities. Preparations were made for emergent TAVR and/or PCI if needed. Induction of general anesthesia was uneventful and included a pre-induction left brachial arterial line and TEE placement. The interventionalists placed a cerebral embolic protection (CEP) device and accessed the femoral vessels. Through the right femoral arterial access, a 7 French guide catheter helped position a snare around the mass's base. Using the left femoral arterial access, an 18 French aspiration catheter was placed above the mass under TEE and fluoroscopic guidance for mass aspiration. AV bypass was initiated during the excision and aspiration and left to run for 12 minutes after excision to aspirate any embolic debris. Post procedural TEE confirmed that the residual mass was less than 0.2 mm. Coronary angiogram and TEE were performed and unchanged from baseline. The patient was extubated successfully at the end of the procedure and discharged home the next day.

Discussion: Traditional treatment of aortic valve papillary fibroelastoma is surgical resection, but this can be daunting in high-risk patients (1). We present a case of the use of percutaneous aspiration to treat this lesion. While this technique has been documented for right sided cardiac masses and venous thrombi, its use for our indication is off-label and not described in the literature (2).
Anticipating potential challenges and initiating multidisciplinary preparations with cardiology, surgery, and perfusion teams was crucial. Considerations included: device limitations (the large and rigid cannula is designed for larger venous circulation), potential aortic valve damage and conversion to emergent TAVR, prophylaxis of cerebral/systemic embolization (we used a CEP device and inverted AV bypass to filter debris), hemodynamic instability needing mechanical support, possible incomplete aspiration and need for further surgery, peripheral vascular injury,

and the risk of coronary artery injury necessitating emergent PCI (3).

Conclusion: Emerging techniques display great promise in treating left sided cardiac lesions in patients with prohibitive surgical risk. We describe a successful percutaneous catheter aspiration technique for an aortic valve mass. TEE guidance is key to surgical precision and prompt detection of complications. Further studies are needed for this to become more commonplace in the future.

Image Uploads

Aortic Valve Mass Differential .

More Benign Findings	Imaging Artifact	Lambl's Excrescences (LE)	Valvular Strands (VS)	Myxoma
Features:	Reverberation, side-lobe artifacts, dropout, and misidentification of normal anatomy/embryological variants can cause this. Essential to have an experienced echocardiographer evaluate in multiple planes to avoid inappropriate/unnecessary interventions.	Strand-like densities on valve edges. Usually benign, but can be embolic. Thin and mobile. Most often on left-sided valves.	Similar to LE but can be longer or thicker. Differentiate based on echocardiography. Often used interchangeably with LE in the literature, but there may be subtle histological differences.	Rarely found on aortic valve, but included on differential because it is a heart mass. Most commonly found in left atrium, attached by a stalk to the fossa ovalis. Can also occur in RA, LV, and rarely RV.
Can't Miss Findings	**Infective Endocarditis (IE)/Aortic Root Abscess (ARA)**	**Primary or Metastatic Tumor**	**Thrombus**	**Papillary Fibroelastoma**
Features:	IE masses are mobile and irregular masses that pose a high embolic risk. ARA is a complication of IE that appears as a mass or cavity near valve annulus. ARA can cause persistent bacteremia, heart block (if extension into septum and conducting system), new murmurs.	Primary malignant tumors (like sarcoma) can rarely present as masses on valve. Associated with rapid growth, invasion, and possibly systemic symptoms. Metastatic tumors (lung, esophageal, etc.) can mimic other masses and attach to valve.	Most common in prosthetic valves but can also affect native valves, especially if hypercoagulable. Typically less mobile than other masses and they are associated with valvular issues.	Most common primary cardiac tumor of aortic valve. Small, mobile, homogeneous. Mass with stalk, usually on aortic side of valve. Can cause embolic events from friability/mobility. Can prolapse into coronary arteries.

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SP 3 - Kiosk 5 - Poster 2

Esophageal Balloon-Guided PEEP Optimization Enables Safe Alveolar Recruitment in Super Morbid Obesity With Severe ARDS

Presenting Author: *Shriya Sharma, MD*

Introduction: Super morbid obesity (BMI >50) profoundly alters respiratory and cardiovascular mechanics. Elevated pleural pressures from massive thoracoabdominal load reduce functional lung volume and distort airway pressure interpretation, often masking severe under-recruitment. In ARDS, standard PEEP tables may be ineffective or harmful. Esophageal balloon monitoring allows estimation of transpulmonary pressure (PL = Paw – Pes), enabling individualized PEEP titration that balances alveolar recruitment with hemodynamic safety. We present a case in which PL-guided PEEP facilitated oxygenation and cardiopulmonary stability in a patient with BMI 74 and severe ARDS.

Case: A 60-year-old male (BMI 74) with OSA, COPD/asthma, atrial fibrillation/flutter, and tachyarrhythmia-induced cardiomyopathy (EF 45%) presented with hypoxemic respiratory failure unresponsive to noninvasive ventilation (PaO_2 53 mmHg, FiO_2 60%). After intubation, and ventilation with PEEP 16–18 cmH_2O and plateau pressures 36–38 cmH_2O failed to improve oxygenation (PaO_2/FiO_2 < 80). Concern for severe pleural pressure elevation prompted esophageal balloon placement.

Initial measurements showed: end-expiratory Pes 24 cmH_2O → PL,exp = –8 cmH_2O, indicating marked collapse despite high PEEP; end-inspiratory Pes 28 cmH_2O → PL,insp = 8 cmH_2O, within acceptable limits. PEEP was titrated to 28 cmH_2O to achieve PL,exp ≈ 0 cmH_2O. Oxygenation, compliance, and driving pressure improved without hemodynamic instability or RV dysfunction.

Ventilatory Response to PL-Guided PEEP

Parameter Pre-titration Post-titration

PaO_2 (mmHg) 53 80

FiO_2 (%) 100 80

Plateau (cmH_2O) 38 36

Driving Pressure 22 16
Compliance (mL/cmH_2O) 18 25

Hemodynamics remained stable with no increase in vasoactive support, and bedside echocardiography showed no RV dilation. The patient avoided proning and ECMO, was weaned off sedation, and was successfully extubated on ICU day 19.

Discussion: In super morbid obesity, elevated pleural pressures make airway pressures unreliable predictors of lung stress. This patient exhibited severe under-recruitment masked by high plateau pressures that predominantly reflected chest wall load. PL-guided PEEP allowed safe application of high PEEP while maintaining physiologic transpulmonary pressures, improving oxygenation and compliance without compromising RV function.

Conclusion: Esophageal balloon-guided PEEP provided effective and hemodynamically safe alveolar recruitment in a patient with BMI 74 and severe ARDS, avoiding escalation to proning or ECMO. This case illustrates that transpulmonary pressure–guided ventilation allows individualized, physiology-driven management in extreme obesity, addressing the limitations of standard PEEP strategies and improving ventilatory safety and effectiveness.

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SP 3 - Kiosk 5 - Poster 3

Cardiac Arrest After Induction in a Patient with Pulmonary Embolism and Clot-in-Transit: The Role of ECMO in Perioperative Rescue

Presenting Author: *Camila Magalhaes Funatsu, MD*

Introduction Pulmonary embolism (PE) with right heart clot-in-transit (CIT) represents a critical perioperative challenge with high mortality. A combination of RV dysfunction, pHTN, and hemodynamic instability places these patients at extreme risk during induction of anesthesia. ECMO has emerged as a lifesaving adjunct when rapid hemodynamic collapse occurs. We present a case of cardiac arrest immediately following induction for planned thrombectomy, successfully managed with emergent institution of VA ECMO and surgical pulmonary embolectomy.

Case Report: A 55 y/o obese woman with HTN presented with acute dyspnea and presyncope, found to have left popliteal/femoral vein thrombosis, a large saddle PE with severe RV

dilation/dysfunction, and a large mobile RA thrombus. She was transferred from the Virgin Islands for higher-level care and scheduled for thrombectomy. Preoperatively, she was hemodynamically stable on room air without vasopressor support. Anesthesia was induced, intubation was uneventful, but shortly thereafter, the patient developed pulseless VTach/VFib. ACLS was initiated, but profound cardiovascular collapse ensued. The cardiothoracic surgery team proceeded with emergent sternotomy and central VA ECMO cannulation was rapidly obtained. ECMO was initiated, restoring perfusion and hemodynamic stability. Full heparinization and conversion to CPB were achieved. The RA was opened, revealing extensive thrombus within the RA and RV trabeculations, which were evacuated. Inspection of the pulmonary arteries revealed large thrombi at the main bifurcation extending into segmental branches bilaterally. The emboli were carefully extracted under low-flow CPB, ensuring clearance of all branches. After complete embolectomy, the patient was rewarmed, weaned from CPB, and transitioned back to VA ECMO via central aortic and right femoral venous cannulation. The chest was partially closed due to coagulopathy.

The patient was transferred to CVICU. Postoperative course was complicated by a left hemothorax requiring washout and subsequent conversion to tunneled central cannulation. Gradual RV recovery was observed on serial TEE. She was decannulated on POD 10, weaned from mechanical ventilation, and discharged to long-term rehabilitation.

Discussion: This case highlights the hemodynamic vulnerability of patients with acute PE and clot-in-transit during induction. Minimal reductions in preload, afterload, or contractility can trigger cardiovascular collapse. ECMO provides an immediate means of stabilizing perfusion and oxygenation while enabling definitive surgical intervention. Early coordination between anesthesia, cardiac surgery, and perfusion teams allowed rapid initiation of ECMO and restoration of circulation. Elective pre-induction femoral cannulation for standby ECMO may have mitigated peri-induction arrest risk.

Results: Anesthetic management of patients with PE and clot-in-transit requires planning and readiness for rapid deterioration. Emergent ECMO initiation can be lifesaving in cases of catastrophic collapse. This case illustrates the importance of multidisciplinary coordination and the vital role of ECMO as both a bridge to embolectomy and recovery of RV function.

Image Uploads

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SP 3 - Kiosk 5 - Poster 4

From Genetic Discovery to Transplant: A Clinical Course of Titin-Related Dilated Cardiomyopathy

Presenting Author: *Michael Guertin, MD*

Introduction: Titin truncating variant (TTNtv) cardiomyopathy is the most common genetic cause of dilated cardiomyopathy (DCM), accounting for up to 25% of end-stage and familial cases. The disease progressive ventricular dilation, systolic dysfunction, and electrical instability arising from disruption of titin's role in sarcomere structure1-2. Management of TTNtv cardiomyopathy involves guideline-directed medical therapy (GDMT) as first-line treatment. However, long-term outcomes remain suboptimal; an observational study found that only 11% of TTNtv-positive patients achieve full ejection fraction recovery at 5 years, compared to 30% in non-TTNtv DCM, likely due to intrinsic myofibrillar dysfunction and irreversible myocardial remodeling3. For those with progression to end stage TTNtv DCM, left ventricular assist device (LVAD) or orthotopic heart transplantation (OHT) is indicated. Median survival post-transplant now exceeds 12 years, with 1-, 3-, and 5-year survival rates of 90.3%, 84.7%, and 79.6%, respectively, compared to a median survival of less than 2 years for stage D heart failure without advanced therapies4.

This case highlights the progression of TTNtv-DCM, characterized by multiple heart failure exacerbations, arrhythmias, and limited GDMT tolerance, culminating in heart transplantation for advanced, refractory disease.

Case Report: A 59-year-old asymptomatic female with no significant past medical was incidentally

found to have a left ventricular ejection fraction (LVEF) of 16%, along with mild mitral regurgitation and mild pulmonary hypertension.

For the next two years, she experienced multiple hospitalizations for tachycardia and heart failure exacerbations. Evaluation by the advanced heart failure team revealed a pathogenic heterozygous TTN mutation.

Attempts to up titrate GDMT were limited by hypotension. Despite appropriate therapy, in September 2024, she was admitted to the medical ICU due to acute-on-chronic heart failure exacerbation complicated by cardiogenic shock. After attempts to optimize of milrinone, an Abiomed Impella 5.5® was placed due to increased ectopy and worsening hemodynamics. Despite adequate Impella positioning on echocardiography, amiodarone therapy, and weaning milrinone, the patient continued to have frequent ventricular ectopy. On POD 7, a suitable donor heart became available, and she successfully underwent OHT. She was extubated on POD 1 following OHT and discharged to acute rehabilitation two weeks later.
Discussion: This case highlights the rapid progression and management complexity of TTNtv-DCM. TTNtv disease is marked by impaired contractile reserve, increased fibrosis, and heightened arrhythmogenicity, contributing to poor GDMT tolerance and recurrent decompensation5. These findings explain the limited efficacy of medical therapy in advanced stages and the frequent need for mechanical circulatory support as a bridge to advanced therapies.

Conclusion: OHT remains the definitive therapy for advanced TTNtv-DCM. This case underscores the value of early genetic identification, close monitoring, and coordinated multidisciplinary care, as well as the need for ongoing research into targeted, genotype-driven treatments.

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SP 3 - Kiosk 5 - Poster 5

Case Report: Left Ventricular Outflow Tract Obstruction Secondary to Retained Subvalvular Tissue Following Mitral Valve Replacement for Mitral Regurgitation

Presenting Author: *Abid Mohideen, MD*

Introduction: Preservation of the mitral subvalvular apparatus, including chordae tendineae and leaflets, is standard in mitral valve replacement (MVR) to limit distortion of left ventricular structure and hemodynamics.1,2 Rarely, retained subvalvular tissue may obstruct the left ventricular outflow tract (LVOT). We describe a case of prominent retained subvalvular apparatus in the LVOT identified via transesophageal echocardiography (TEE) following MVR for severe mitral regurgitation (MR) with preservation of hemodynamics causing dilemma for further operative management.

Case Report: A 74-year-old woman with a history of patent ductus arteriosus repair via left thoracotomy, mitral valve prolapse, MR, atrial fibrillation (AF), hypertension, and gastroesophageal reflux disease was repeatedly admitted over 1 month due to AF with rapid ventricular response, pulmonary edema, aspiration, orthopnea, and worsening dyspnea on exertion, requiring BiPAP and diuretics. During her latest hospitalization, an electrocardiogram illustrated diffuse ST-depression and T-wave inversions with troponin < 30. Cardiac catheterization revealed 70% stenosis of her left anterior descending coronary artery and 4+ MR. After cardiac surgical evaluation, she agreed to elective tissue MVR, coronary artery bypass grafting, Maze procedure, and left atrial appendage clipping. Preoperative TEE showed a 37 mm mitral valve annulus without stenosis, thickened leaflets with restriction of the posterior leaflet (P1-P3), 7 mm vena contracta, systolic dominance of pulmonary vein flow, and severe eccentric, posteriorly directed MR. She underwent MVR with standard retention of chordal and leaflet tissue to enhance postoperative left ventricular function. Postoperative TEE of the mitral area visualized successful implantation of a stented bioprosthesis with trace MR, normal leaflet motion, peak velocity of 10 m/s, mean gradient of 5 mmHg, normal mitral annulus, normal motion, and velocity time interval of 28 cm. However, prominent, mobile retained tissue of the subvalvular apparatus was also identified in the LVOT. Though the patient remained hemodynamically stable and the LVOT gradient was stable, concern for obstruction prompted return to cardiopulmonary bypass. The obstructing portion of subvalvular tissue was resected via aortotomy, clearing the TEE findings in the LVOT. She tolerated both procedures well

and
remained hemodynamically stable. Her postoperative course was complicated by volume overload and a pleural effusion that was successfully drained with a pigtail catheter.

Discussion: When feasible, preservation of the subvalvular apparatus in MVR is meant to maintain left ventricular physiologic function. We report a rare example of excess retained chordal and leaflet tissue causing prominent appearance in the LVOT. This case also reiterates the value of perioperative TEE. Decision making in the absence of flow turbulence and increased gradients was difficult but returning to cardiopulmonary bypass was deemed necessary to resect the excess tissue.

Conclusion: This case showcases the nuance in ensuring preservation of the subvalvular apparatus to optimize postoperative ventricular function. Utilizing operative TEE is crucial to dictate diagnosis and corrective measures.

Image Uploads

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SP 3 - Kiosk 5 - Poster 6

Emergent surgical retrieval of an Amplatzer Amulet migrated into submitral apparatus causing severe mitral regurgitation via minithoracotomy: A case report

Presenting Author: *Kevaughn Henry, MD*

Introduction: Left atrial appendage occluder (LAAO) offers a non-pharmacological method to reduce the risk of cardioembolic events in atrial fibrillation. Both the Amulet and Watchmen exhibit similarly high procedural success. The Watchmen is a self-expanding frame enveloped in a permeable fabric placed at the ostium of the LAA, while the Amulet employs a lobe-and-disk design to improve LAA sealing.1 The Amulet IDE trial showed that Amulet was superior in achieving complete LAA occlusion, though has higher risks of pericardial effusion and device embolization.3 We describe a case of Amplatzer Amulet LAAO migration into the LV in the submitral apparatus immediately identified with TEE and was surgically retrieved via minithoracotomy.

Case Report: A 65 year-old male with past medical history of hypertension, hyperlipidemia,

cerebrovascular accident in 2003 with residual unsteady gait, obstructive sleep apnea, HFpEF with EF 65%, coronary artery disease, prophylactic ICD implant 2015, persistent atrial fibrillation previously on Xarelto and underwent 22 mm Amplatzer Amulet placement for left atrial appendage occlusion. On post operative day 2, the patient started having shortness of breath and presented to us with worsening symptoms for a week. The CTA chest showed migration of the LAAO device into the LV and TTE showed migration into the submitral apparatus in close proximity to the posterior mitral leaflet causing an acute severe mitral regurgitation (MR). Intraoperative TEE guided the emergent mini thoracotomy approach with one-lung isolation and device removal was successfully done along with internal LAA closure, mitral valve repair using Cosgrove annuloplasty band. The patient had successful operation with 60 minutes of cross-clamp time, 84 minutes of cardiopulmonary bypass time with no intraoperative complications and was transferred to the ICU.

Discussion: A trial fibrillation is a common cardiac arrhythmia found in elderly and significantly increases the risk of cardioembolic stroke. Given the risk factors with anticoagulation, LAAO device placement has emerged as an alternative method in patients with nonvalvular atrial fibrillation. However, the risks include pericardial effusion/tamponade, thrombosis, and dislodgement.2 Some studies
showed certain characteristics associated with device embolization such as, “lower device volume (24 vs. 41 procedures; p< 0.0001), Watchman 2.5 vs. Watchman FLX devices (0.08% vs. 0.04%; p=0.0048), larger LAA ostia (median 23 vs. 21 mm; p=0.004), and a smaller difference between device and LAA ostia size (median difference 4 vs. 5 mm; p=0.04).2” Conclusion There’s a lack of consensus on the management of migrated LAAO devices, keeping into account patient’s hemodynamics and the device location. Based on a few case studies, if the device is migrated into the left atrium or aorta and the patient is asymptomatic, a percutaneous retrieval can be done.3 However, in cases where the device occluded the LVOT, sudden cardiac death occurred. In our case, LAAO was found in the LV and in the submitral apparatus which was identified in a timely fashion using TEE which helped guide the surgical approach for device retrieval via minithoracotomy with one-lung ventilation.

Image Uploads

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SP 3 - Kiosk 5 - Poster 7
Trial and Error: Airway Management during Robotic Tracheobronchoplasty
Presenting Author: *Holly Huber, MD*

Introduction: Expiratory central airway collapse (ECAC) is characterized by exaggerated tracheobronchial narrowing. Severe cases are increasingly treated with surgical management such as tracheobronchoplasty, which fixes mesh to the cartilaginous airway edges and posterior membrane to prevent expiratory collapse. This technique involves isolated left lung ventilation in the left lateral position and periods of apnea while the surgeon works on the left main bronchus. The “Stop, Drop, and Roll” (DRS) maneuver, which retracts the endotracheal tube (ETT) into the trachea to expose the posterior membrane, presents unique anesthetic challenges2. We present one such case.

Case: A 68-year-old female presented with chronic cough and exertional dyspnea. On awake bronchoscopy, the patient had 80% collapse of the distal trachea with near 100% collapse of both mainstem bronchi. She improved following a trial with stents to her distal trachea and was consented for definitive treatment with robotic tracheobronchoplasty.
Intra-operatively, we used a specialized wire-reinforced 40-cm endotracheal tube (Fuji Wire Reinforced Endotracheal Tube [WRETT]) to allow easier sealing of the left mainstem. During the left bronchoplasty portion of the procedure, the DRS maneuver was used to allow fixation of mesh. During the DRS maneuver, the ETT was retracted, sutures placed and ETT readvanced. The surgical team communicated that the ETT was inadvertently in the right bronchus. The ETT was retracted, there were no identifiable airway structures. Unable to mask or intubate in the lateral position, an LMA was placed and ETT exchanged via a wire exchange catheter kit. The lowest measured SpO2 was 33% during the incident.

For subsequent DRS maneuvers, the fiberoptic scope was placed within the ETT prior to retraction. This allowed for more precise retraction given direct visualization of the lighted end of the fiberoptic scope on the surgical field as well as replacement into the left mainstem bronchus. The remainder of the operation was uneventful, and the patient had a reassuring neurological examination following extubation.

Discussion: Our patient had near 100% collapse of the left and right mainstem bronchi on forced exhalation. Her intra-operative course was complicated by accidental extubation with severe hypoxia following the DRS maneuver.

There have been case reports of using a standard ETT in combination with an EZ blocker without

the DRS method, but there is a risk of suturing the blocker into the airway1. Other airway techniques for this procedure mostly include utilizing the DRS method with some variation of specialized ETT which has risks of accidental extubation given frequency of airway manipulation – especially under the drapes and in a lateral position.

Conclusion: Fiberoptic manipulation of this patient population's airways can be challenging due to positioning, distorted airway anatomy from their disease process, and worsening EDAC with intrathoracic insufflation. We believe using a fiberoptic scope like a lighted stylet to guide retraction and re-advancement of the ETT during the left bronchoplasty portion of the operation provides an additional layer of safety to prevent intra-operative complications of frequent airway manipulation.

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SP 3 - Kiosk 6 - Poster 1

When the Numbers Don't Add Up: Profound Cerebral NIRS Drop During Emergent Aortic Repair Without Neurologic Injury

Presenting Author: *Raymond Powers, MD*

Introduction: Acute aortic syndromes carry high perioperative risk, particularly when complicated by tamponade, hemodynamic collapse, or concern for cerebral malperfusion. Cerebral near-infrared spectroscopy (NIRS) is often used to monitor cerebral perfusion during cardiopulmonary bypass (CPB). We present a case of emergent ascending aortic dissection repair with profound drop in cerebral NIRS during anterograde cerebral perfusion (ACP), further complicated by rapid extension of type A aortic dissection to involve axillary and carotid arteries. Ultimately, no appreciable neurologic injury was found postoperatively.

Case Presentation: A 73-year-old woman with hypertension, Hyperlipidemia, severe mitral regurgitation and ascending aortic dilation was admitted with acute chest pain and new-onset atrial fibrillation. Imaging revealed an intramural hematoma with evolution into a Type A aortic dissection complicated by cardiac tamponade. The patient was taken emergently to the operating room for

ascending aortic repair.

Given tamponade physiology, general anesthesia was carefully induced with sevoflurane to preserve spontaneous ventilation and venous return. After sternotomy and initiation of CPB, repair of the ascending aorta commenced. The patient was cooled to a bladder temperature of 28 °C, a nasopharyngeal temperature of 23 °C. Ice was placed on the head. Shortly after circulatory arrest and ACP initiation, cerebral NIRS readings fell precipitously, with a nadir in the 30's from a baseline in the 80's. Pump flow was maximized, and adjustments were made to hematocrit, $PaCO_2$, and line pressure. Notably, backpressure from the right axillary inflow cannula was within normal limits, cannula positioning was confirmed, and head positioning rechecked. NIRS values remained critically low for nearly 40 minutes, however. With reinitiation of systemic circulation, cerebral NIRS quickly climbed back to baseline readings. Time in minutes was as follows: total bypass 175, cross-clamp 87, ACP 35, circulatory arrest without ACP 4.5.

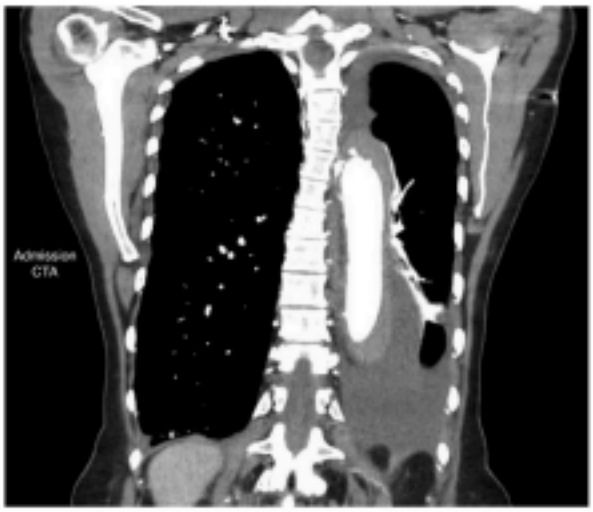
Post CPB, TEE demonstrated possible dissections of the carotid arteries, and extensive circumferential dissection of the descending aorta which was not present pre-CPB. These results were later confirmed with CTA.

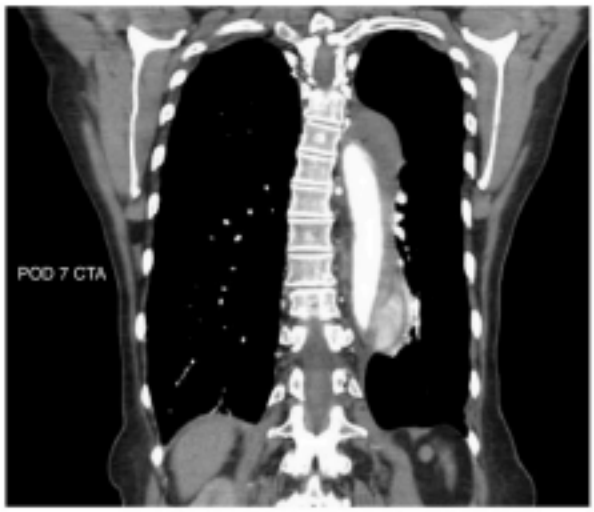
Discussion:

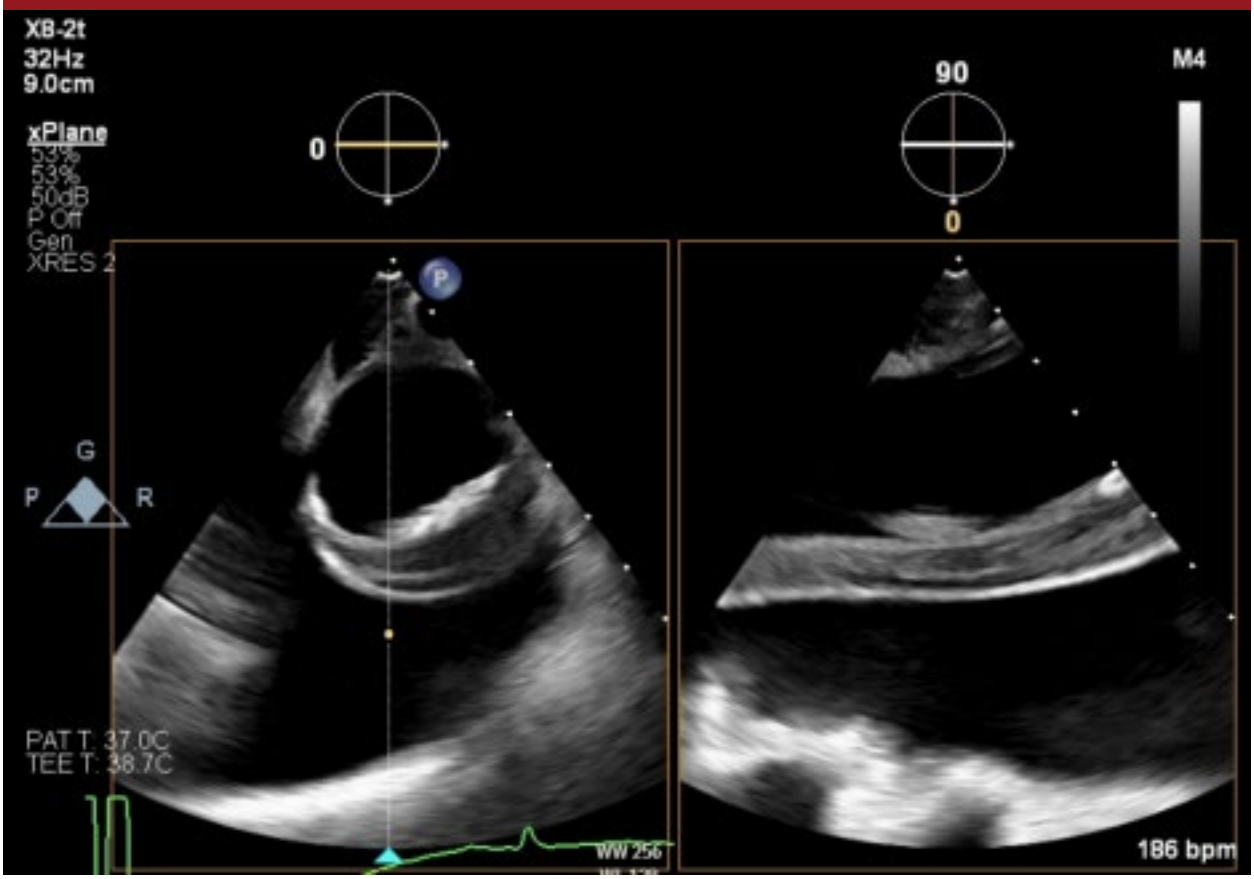
Profound cerebral desaturation during aortic dissection repair typically raises concern for cannula malposition, embolic phenomena, or flow competition from the dissection flap. This case illustrates that critically low NIRS measurements do not uniformly translate to neurologic injury, possibly due to device limitations or successful hypothermic neuroprotection.

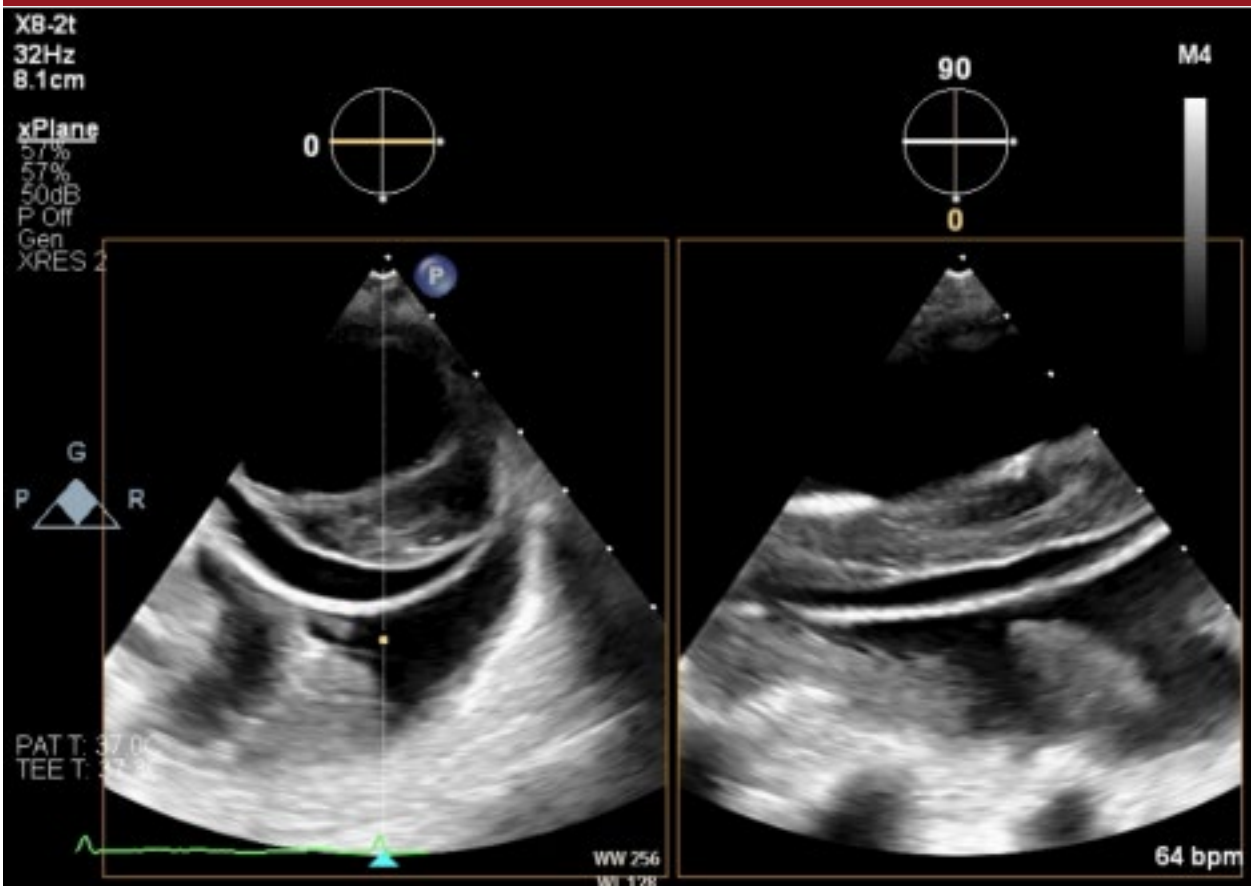
Despite tamponade physiology, massive transfusion, and significant fluctuations in cerebral oximetry, the patient recovered without neurologic deficit and had an ICU stay of only five days. Key contributors likely included rapid diagnosis, careful hemodynamic management, maximization of CPB flow, constant reassessment of perfusion parameters, and neuroprotective hypothermia. This case brings into question whether cerebral NIRS values can correctly predict strokes during hypothermia and demonstrates a superb outcome despite marked and sudden extension of a type A dissection with non-reassuring cerebral NIRS values.

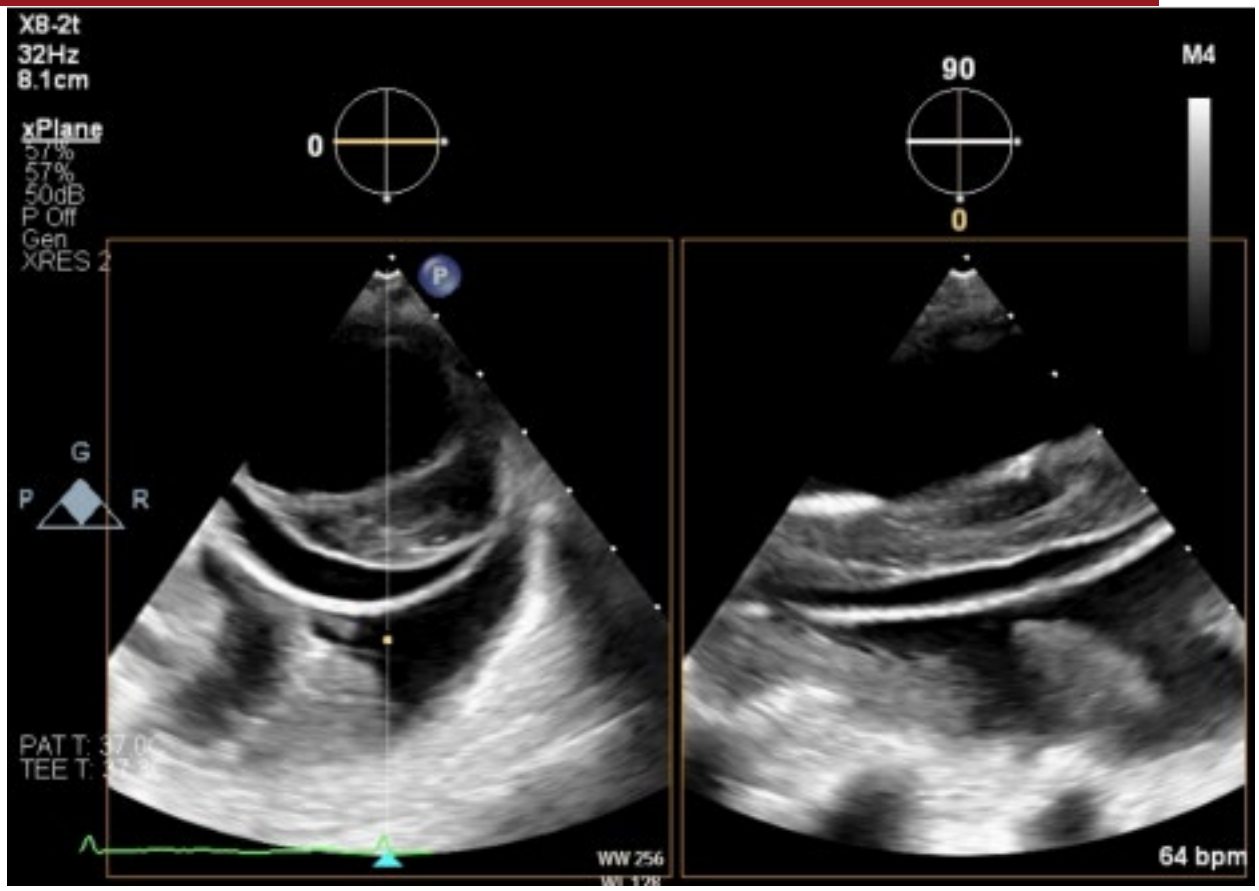
Image Uploads


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WW 256
64 bpm



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SP 3 - Kiosk 6 - Poster 2
Congenital Pulmonary Valve Stenosis in an Adult Patient: Multimodality Imaging
Presenting Author: *Jose Gallegos, MD*

Introduction Paragraph: The current case showcases an uncommon presentation of the pulmonic valve (PV) stenosis and associated pulmonary artery (PA) aneurysm in an adult patient. A transesophageal echocardiography (TEE) was supplemented by epicardial echocardiography (EE) on the surgical field. This technique allowed evaluation of the PV morphology and the mechanisms of obstruction. We aim to highlight the potential benefits of epicardial ultrasound especially when evaluating complex anatomical structures of the anterior heart.

Brief Summary of the Clinical Case: This is a 63-year-old male presenting with known left pulmonary artery aneurysm. Transthoracic echocardiography (TTE) showed mild PV insufficiency and stenosis (concordant with right heart catheterization), however not providing a clear mechanism. Intraoperative 2D and color flow Doppler TEE imaging suggested a lack of the obstruction at the level of the PV annulus but rather echogenic structure and flow acceleration distal to the PV suggestive of a potential supravalvular stenosis (video 1). Similarly, pulsed wave/continuous wave Doppler showed no obstruction at the level of the PV annulus (2mmHg), increased gradient (9-11mmHg) within the PV and further increase to 28mmHg just past the PV, before the aneurysmal main and left PA (figure 1). The lack of the spatial resolution of the 3D TEE was not conducive to discerning the mechanism. The EE, then, revealed significantly thickened tips of the long PV leaflets producing a cone-shaped PV anatomy responsible for the stenosis (video 2). Given that the discerned stenosis was directly related to the PV, a pulmonary homograft was used to replace the PV, which was distally anastomosed to the avalvular aortic homograft.

Discussion

- Epidemiology:
- Isolated PV stenosis is rare in adults accounting for only 8% of adult congenital heart cases
- Usually associated with other congenital heart abnormalities1,2
- Morphology:
- PV stenosis most commonly presents with a dome-shaped valvular apparatus with normal leaflet motion and a small central opening at the thickened tips1,2
- Aneurysmal dilatation of the PA past the point of stenosis
- Evaluating the PV with TEE:
- Decreased spatial resolution when evaluating very anterior structures
- Views:
- Mid-esophageal (ME) RV inflow-outflow view allows for visualization of the RV outflow tract (RVOT) and PV
- Withdrawal of probe into Upper esophagus allows for long axis presentation of PV and PA
- Co-axial alignment for spectral Doppler Measurements
- Best view for 3D imaging
- Color flow Doppler
- Further pinpoint areas of stenosis via flow acceleration

- Spectral Doppler
- Pulsed wave Doppler - Can help assess level of stenosis

Continuous wave Doppler - gradient across entire valve apparatus

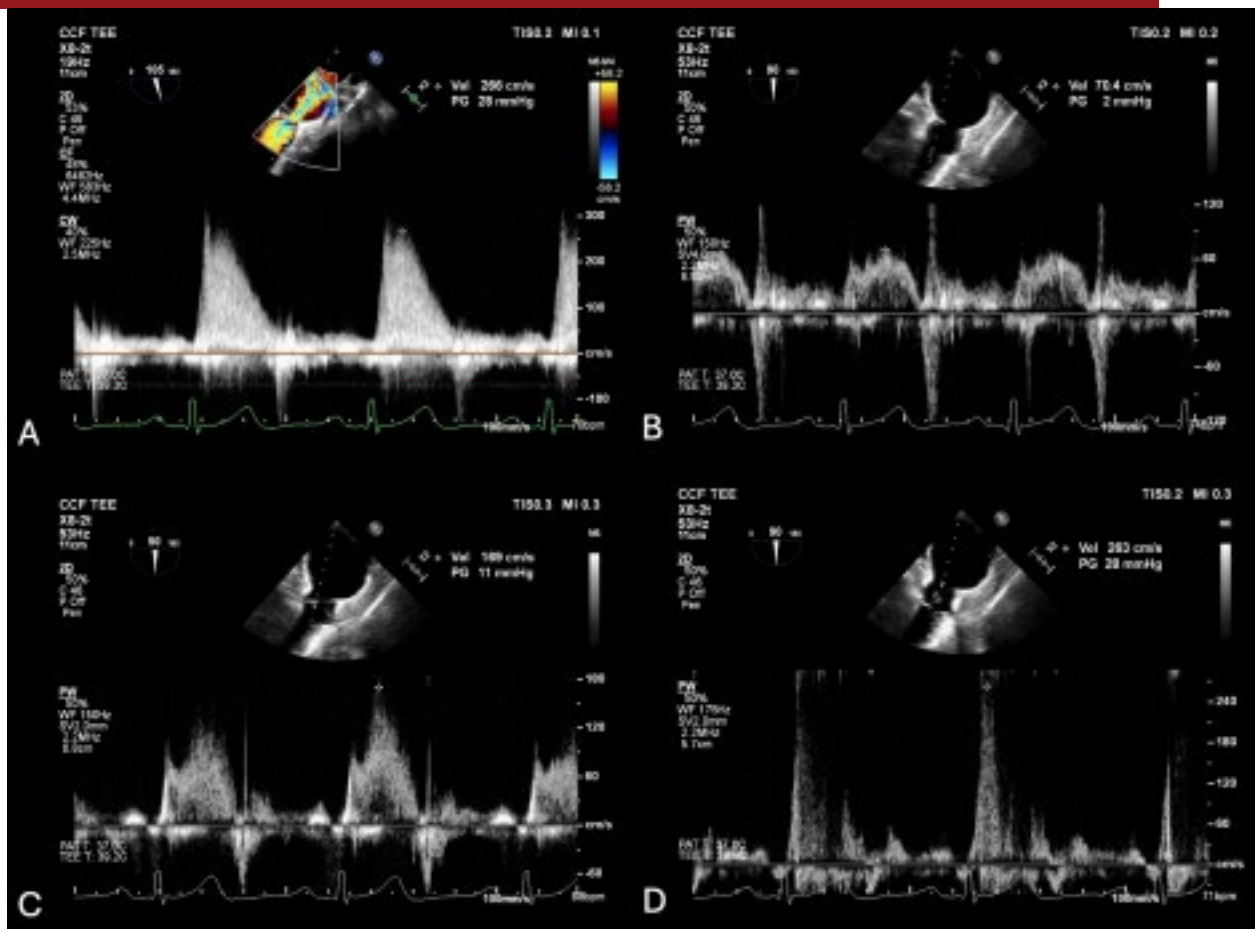
Impact of Echocardiographic findings

The application of Epicardial Echocardiography (EE) provided spatial resolution superiority which in turn allowed for clarification of the PV stenosis mechanism. This influenced surgical decision making and planning by electing for a pulmonary homograft.

Teaching Points of the Case

- Intraoperative EE is an underused tool
- Previous studies and case reports have reported improved acquisition of Image Uploads that have led to changes in intraoperative decision-making3,4
- Pulmonary thromboembolism detection
- Potential a better window for 3D plane slicing
- Decreased injury risk to patient compared to TEE and can be quickly employed

Image Uploads



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SP 3 - Kiosk 6 - Poster 3

Methods of Assessing Discordance between aPTT versus Anti-factor Xa Level among Patients Supported on Extracorporeal Membrane Oxygenation

Presenting Author: *Connie Nguyen, MD*

Introduction: Unfractionated heparin is the anticoagulant of choice to reduce the risk of thrombosis associated with extracorporeal membrane oxygenation (ECMO). Heparin therapy is commonly monitored using activated partial thromboplastin time (aPTT) and anti-factor Xa activity assays. However, discordance between these assays is common, though the definition of discordance is not universally defined. Because patients on ECMO treated with intravenous heparin are at risk for both bleeding and thrombosis, establishing optimal anticoagulation patterns is critical.
We hypothesize that discordance between aPTT and anti-factor Xa activity is common at our high-volume ECMO center, and that assessment of discordance may not be well-defined by simple disagreement between protocolized therapeutic ranges of each assay.

Methods: Retrospective cohort study of adult ECMO patients treated with IV heparin at Columbia University Irving Medical Center (CUIMC). Data was extracted from Extracorporeal Life Support Organization registry,perfusion logs, and medical records. Our institution defines systemic anticoagulation targets as aPTT of 51–83 seconds and anti-Xa level of 0.3–0.7 IU/mL. Comparison of aPTT:anti-Xa discordance was done via 2 approaches:

· 1. Range based, using CUIMC systemic anticoagulation targets:

· a. aPTT < anti-Xa if aPTT was < 51 and anti-Xa 0.3–0.7 or aPTT 51–83s and anti-Xa > 0.7 IU/mL

· b. aPTT = anti-Xa if aPTT was 51–83s and anti-Xa 0.3–0.7 IU/mL

· c. aPTT > anti-Xa if aPTT was > 83s anti-Xa 0.3–0.7 or aPTT 51–83s and anti-Xa < 0.3 IU/mL

· 2. Using a second-degree polynomial regression model in which repeated measures per patient were accounted for using a random intercept per patient and identifying discordant values as those outside of the 95% prediction interval:

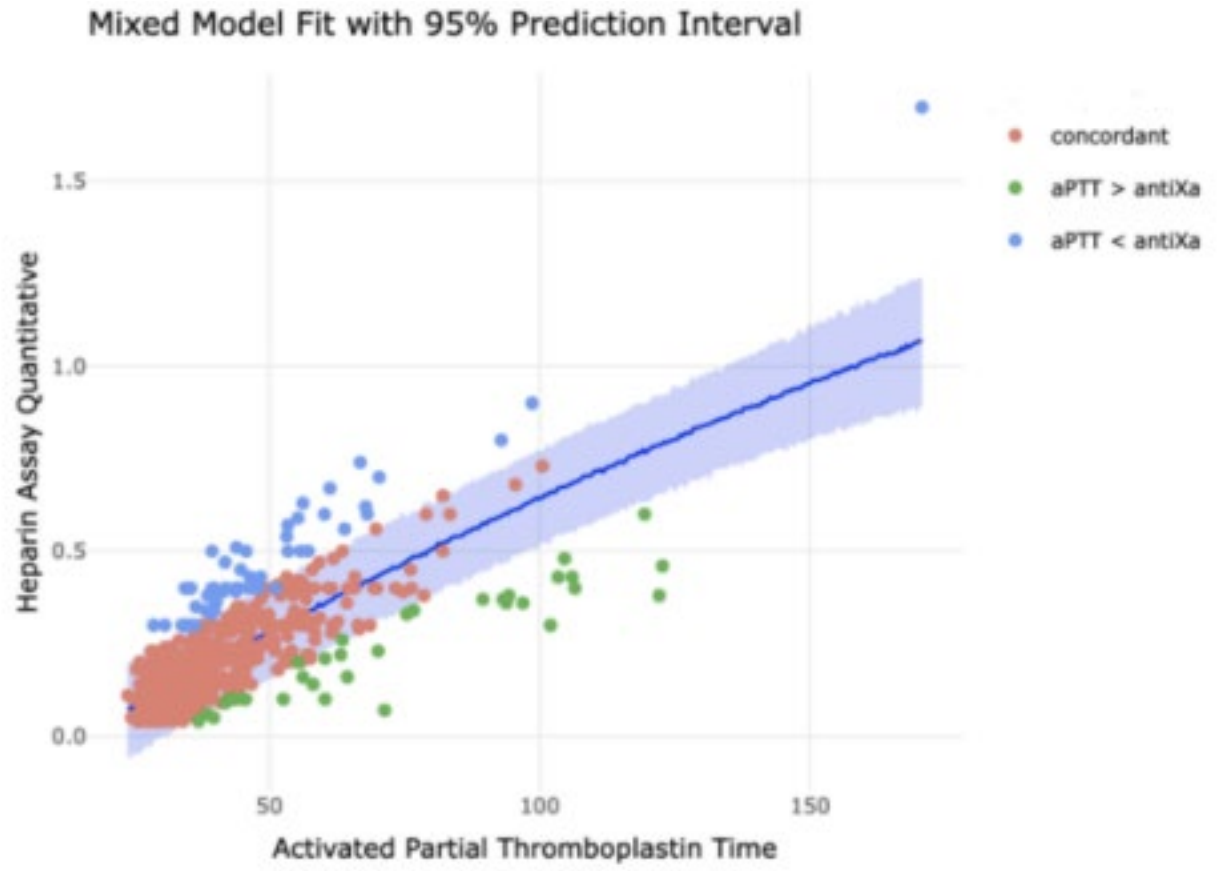
· a. aPTT < anti-Xa (2.5% of data outside of prediction interval)

· b. aPTT > anti-Xa (2.5% of data outside of prediction interval)

The counts of patients in each discordant group were compared with Pearson chi-squared tests.
Results 113 patients with 571 time-matched aPTT:anti-Xa pairs were identified. One patient had 1 assay in which aPTT was below CUIMC goal range and anti-Xa was above goal range and, separately, 1 assay with the opposite groupings. This patient and 2 lab pairs were dropped due to possible lab errors. Counts of patients with discordance using a range-based approach were significantly different than those using the modeled approach (image 1) (48 versus 18 aPTT < anti-Xa and 15 versus 17 aPTT > anti-Xa, simulated p = 2e-04; image 2).

Discussion: Discordance was common in this single-center study. Counts of patients with discordance based on pre-defined ranges for systemic anticoagulation were significantly different than those identified based on predicted values using measured labs. Our modeled approach, in which sensitivity and specificity were balanced with a 95% prediction interval, may identify truly unusual lab pairs which are less likely to be recognized based on discrete ranges, in which lab pairs on the margin of the range may be mis-identified as “discordant”. In the absence of a universal definition of discordance, it remains unclear which approach is superior and their clinical implications. Future studies will aim to determine whether rates of detecting complications, such as bleeding or thrombosis, differ significantly when using either range-based or modeled approaches.

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		Modeled		
	n = 112	aPTT < anti-Xa	aPTT > anti-Xa	Concordant
Range-based	aPTT < anti-Xa	14	4	30
	aPTT > anti-Xa	0	11	4
	Concordant	4	2	43

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SP 3 - Kiosk 6 - Poster 4
A Perplexing Jet and the Search for its Origin
Presenting Author: *Olivia Sonderman, MD, MPH*

Introduction: Our case highlights the role of the basic perioperative transesophageal echocardiography (TEE) in refining the preoperative diagnosis for cardiac surgery patients. It secondarily provides the opportunity to review an uncommon pathology and its echocardiographic findings.

Brief Summary of the Clinical Case: A 47 year-old male with past medical history of Klippel-Feil Syndrome (KFS) and secundum atrial septal defect (ASD) status post repair at 6 years of age was found to have a high velocity, continuous flow jet concerning for Gerbode defect on surveillance transthoracic echocardiography. He was scheduled for open surgical repair.

Post-induction TEE focused on interrogation of a turbulent jet with continuous left to right flow prominent through systole and diastole. Close inspection revealed aneurysmal tissue at the level of the aortic root. In the midesophageal aortic valve short axis view, the left-to-right flow appeared to have continuity with the Sinus of Valsalva.

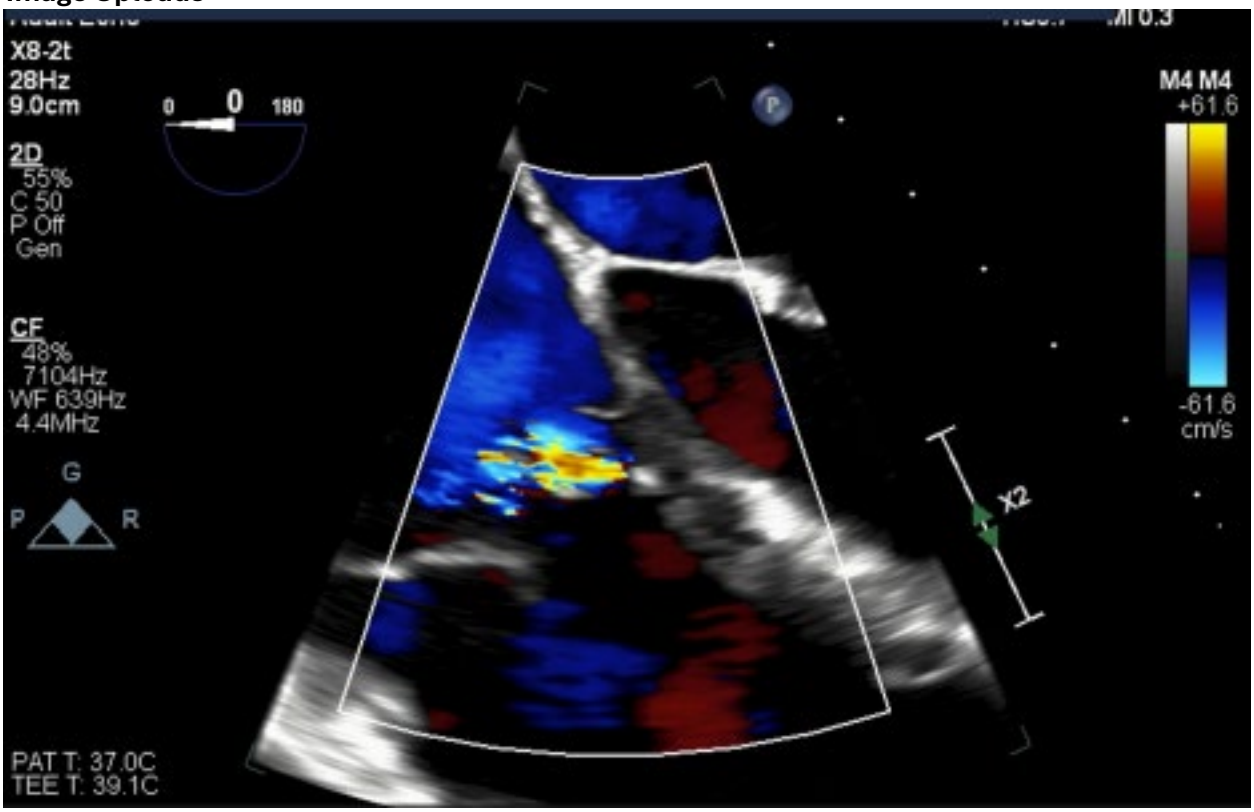
Cardiopulmonary bypass (CPB) was initiated. Prior to placing the aortic cross clamp, the surgeon snared the venous cannulas to isolate the right atrium. In doing so, significant venous return was lost to the CPB circuit suggesting a shunt above the level of the aortic valve. The right atrium was opened and revealed the prior ASD repair. Aneurysmal tissue in the right atrium was unfurled revealing a small opening through which the right-non coronary commissures of the aortic valve were visualized. The finding was consistent with a defect in the noncoronary sinus of valsalva into the right atrium, likely a Sinus of Valsalva aneurysm (SOVA). The defect was repaired with bovine pericardium.

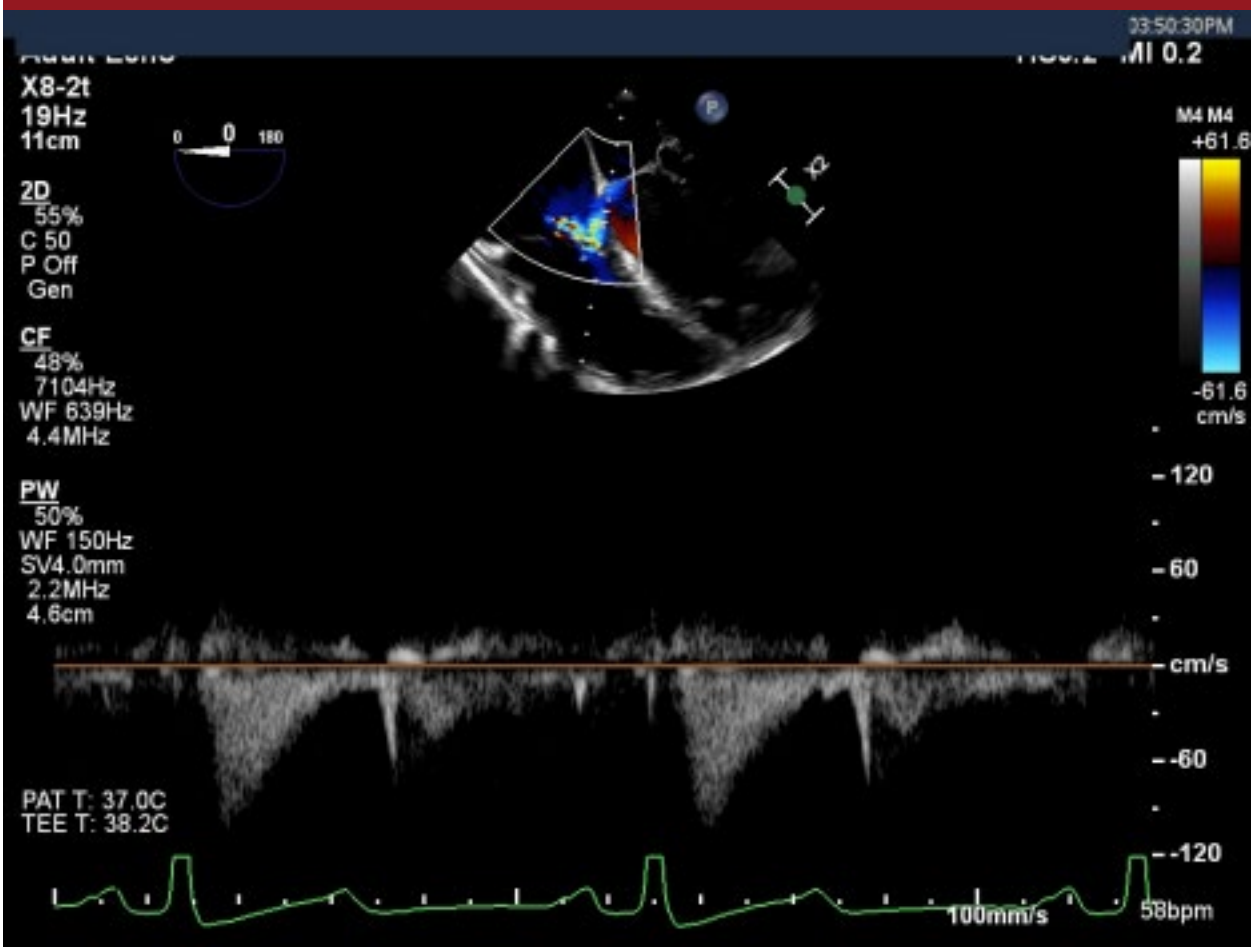
The patient was uneventfully weaned from CPB. Post-CPB evaluation revealed resolution of the left-to-right shunt with normal biventricular function. The patient was discharged to home four days later.

Discussion

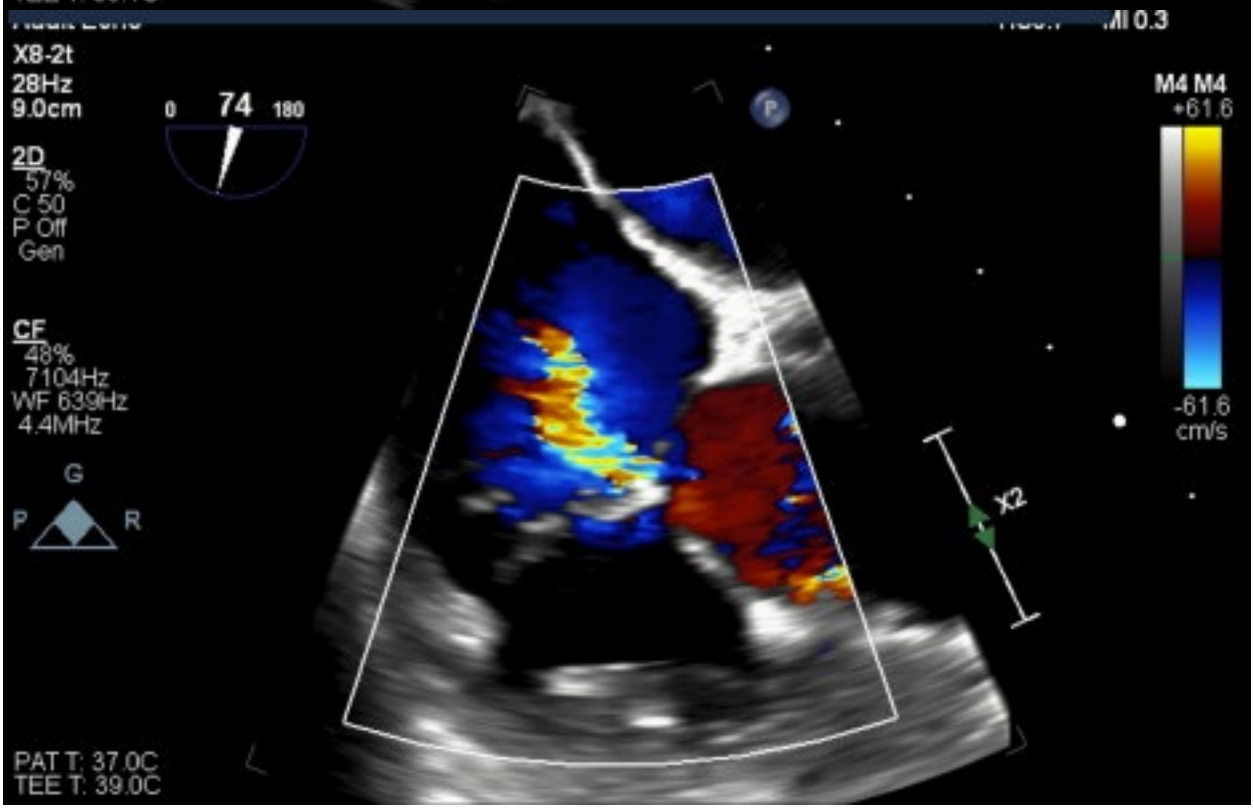
- TEE techniques: carefully define anatomy to differentiate a Gerbode defect from a sinus of valsalva aneurysm rupture
- Midesophageal long axis
- Defines relationship of defect/jet to the RV
- Short axis view of the aortic valve revealing left-to-right flow concerning for SOVA ● Defines relationship of the defect to the atria, aortic valve cusps and coronaries ● RV-inflow-outflow view
- Helpful in localizing and defining ventricular septal defects
- Look for “Windsock Deformity”
- Doppler profiles are helpful
- SOVAR Doppler profile is accentuated in diastole
- Gerbode Defect (or VSD) Doppler is predominant in systole
- Impact of Echocardiographic findings
- Impact of the echocardiographic finding on clinical decision making
- Changes surgical approach (enter the heart from the aorta versus the right atrium) ● Impacts optimal hemodynamic management
- Teaching Points of the Case
- Teaching points:
- Have a high index of suspicion when TEE Image Uploads contradict the preoperative diagnosis ● Review of sinus of Valsalva aneurysm
- Sinus of Valsalva aneurysm (SOVA), a congenital or acquired cardiac defect that is present in roughly 0.09% of the general population
- SOVAs expand over time and may lead to heart failure, cardiac ischemia, tamponade, thromboembolic events, malignant arrhythmias and hemodynamic instability ● In the case of a ruptured SOVA, echocardiographic evaluation with color Doppler will reveal continuous, turbulent flow in systole and diastole because the aorta is a high-pressure system

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SP 3 - Kiosk 6 - Poster 5

Identifying Dysphagia After Aortic Surgery: Predictors and Accuracy of Bedside Swallow Screening

Presenting Author: *Arvin Saleh, MD*

Introduction: Dysphagia is an underrecognized complication following aortic surgery, with reported incidence ranging from 5% to 60%. Clinical complications from dysphagia include aspiration pneumonia, prolonged mechanical ventilation and ICU or hospital stay, and an increased risk of tracheostomy or enteral feeding tube placement. The gold standard diagnostic tests for dysphagia are resource-intensive and thus diagnosis relies on bedside swallow tests. Thus, we seek to 1) identify risk factors associated with postoperative dysphagia in adult post-aortic surgery patients and (2) evaluate the concordance between bedside and formal swallow assessments.

We performed a retrospective cohort study of 125 adults who underwent aortic surgery at Yale New Haven Hospital between 2019 and 2021. Postoperative dysphagia was defined as (1) failure of bedside or formal swallow evaluation, (2) persistent symptoms requiring diet modification, or (3) placement of a nasogastric or gastrostomy feeding tube. Candidate predictors included demographics, preoperative comorbidities, intraoperative variables, and postoperative ventilation duration. Univariable analyses identified variables associated with dysphagia ($p < 0.10$), which were then evaluated in multivariable logistic regression. Because of the limited number of dysphagia events and potential collinearity, bootstrap resampling with 200 replicates was used to generate bias-corrected confidence intervals. Concordance between bedside swallow evaluation and formal instrumental assessment (videofluoroscopy or FEES) was cross-tabulated to determine agreement, sensitivity, and specificity. Statistical significance was defined as $p < 0.05$.

Postoperative dysphagia occurred in 14 patients (11.2%). In multivariable analysis, independent risk factors for dysphagia were cerebrovascular disease (adjusted Odds Ratio (OR) 0.14, 95% CI 0.02–0.71, p=0.017) and congestive heart failure (adjusted OR 0.32, 95% CI 0.09–0.76, p=0.018). This suggests that impaired neurologic reserve, reduced cardiopulmonary function, and baseline frailty predispose these patients to dysphagia. Another independent risk factor for dysphagia is longer ventilation durations (adjusted OR 1.19 per 12 hours of mechanical ventilation, 95% CI

1.13–1.80, p < 0.001), consistent with proposed mechanisms linking extended intubation to impaired laryngeal sensation and swallowing dysfunction. Among the 14 patients who failed the formal swallow evaluation, 13 also failed the bedside test, yielding a sensitivity of 93%. Of the 111 patients who passed the formal evaluation, 108 also passed the bedside test, yielding a specificity of 97%. A strength of this study was the consistency of the recovery setting (two specific ICUs within the hospital) and the nursing staff, which enhanced internal validity. However, these same factors, together with the small sample size, limit the generalizability of these findings to other settings.

Postoperative dysphagia occurred in one in nine patients after aortic surgery and was strongly associated with prolonged mechanical ventilation, cerebrovascular disease, and congestive heart failure. Structured bedside swallow evaluations may serve as a reliable and efficient tool for early identification of swallowing dysfunction in this population.

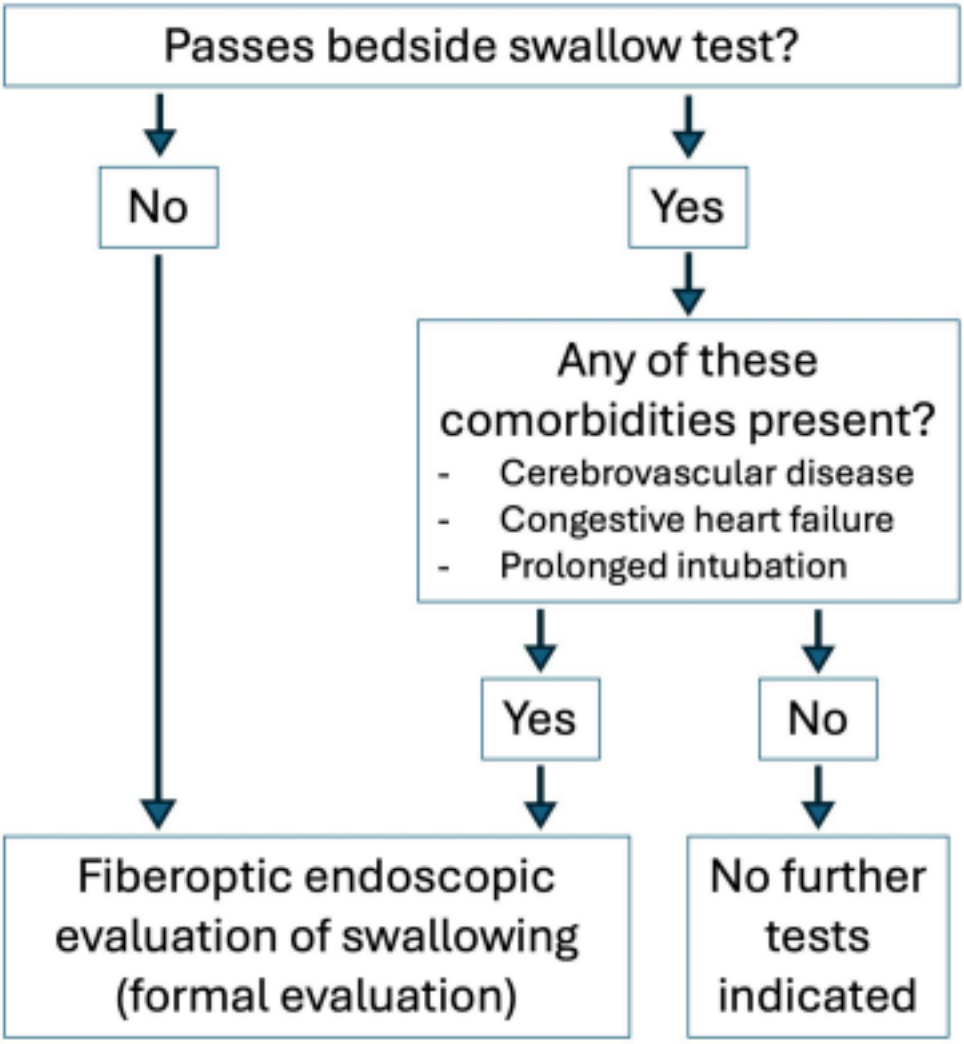
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Variable	Dysphagia Median or n/N (%)	No Dysphagia Median or n/N (%)	p-value
Demographics			
Age (years)	72.0 (65.0–76.0)	68.0 (59.0–74.0)	0.142
Male sex	9/14 (64.3%)	68/111 (61.3%)	0.819
Body Mass Index (BMI)	27.0 (24.0–30.0)	28.0 (25.0–31.0)	0.398
Preoperative Comorbidities			
Hypertension	12/14 (85.7%)	91/111 (82.0%)	1.000
Diabetes mellitus	6/14 (42.9%)	36/111 (32.4%)	0.411
Cerebrovascular disease	4/14 (28.6%)	6/111 (5.4%)	**0.012**
Congestive heart failure	4/14 (28.6%)	12/111 (10.8%)	0.089
Chronic lung disease	3/14 (21.4%)	18/111 (16.2%)	0.698
Gastroesophageal reflux disease (GERD)	4/14 (28.6%)	16/111 (14.4%)	0.250
Intraoperative and Airway Factors			
Cardiopulmonary bypass (CPB) time (min)	152.0 (124.0–181.0)	142.0 (118.0–168.0)	0.312
Cross-clamp time (min)	89.0 (72.0–112.0)	80.0 (65.0–97.0)	0.225
Difficult intubation	2/14 (14.3%)	13/111 (11.7%)	0.675
Postoperative Course			
Duration of mechanical ventilation (hours)	146.0 (102.0–290.0)	18.0 (12.0–26.0)	**<0.001**

Variable	Adjusted OR	95% CI	p-value
Demographic Factors			
Age (per 10 years)	0.70	0.40–1.05	0.093
Male sex	1.00	0.53–1.51	0.998
Body mass index (per 5 units)	1.00	0.86–1.22	0.962
Preoperative Comorbidities			
Cerebrovascular disease	0.14	0.02–0.71	**0.017**
Congestive heart failure	0.32	0.09–0.76	**0.018**
Chronic lung disease	1.00	0.57–1.77	0.998
Gastroesophageal reflux disease (GERD)	0.68	0.32–1.28	0.259
Intraoperative and Airway Factors			
Cardiopulmonary bypass (CPB) time (per 30 min)	1.00	0.86–1.15	0.997
Cross-clamp time (per 20 min)	0.90	0.70–1.12	0.351
Difficult intubation	1.00	0.45–2.01	0.993
Postoperative Variable			
Duration of mechanical ventilation (per 12 hours)	1.19	1.13–3.80	**<0.001**

	Formal Fail (n)	Formal Pass (n)	Total
Bedside Fail	13	3	16
Bedside Pass	1	108	109
Total	14	111	125

Pathway for Identifying High-Risk Dysphagia Patients



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SP 3 - Kiosk 6 - Poster 6
Migration of Left Atrial Appendage Occlusive Device into Left Ventricle
Presenting Author: *Jasmine Tuteja, MD*

Background: Left atrial appendage occlusion (LAAO) is a common method of preventing thromboembolic strokes in patients with atrial fibrillation who are unable to tolerate long-term anticoagulation. While complications are rare, LAAO devices can dislodge and potentially lead to acute heart failure, stroke, or even death. Device type and patient anatomy impact the risk of LAAO device dislodgement.

Case presentation: An 80-year-old man with history of atrial fibrillation presented for LAAO device placement. The patient had a history of cardioversion and ablation, along with history of upper GI bleeds and inability to tolerate long-term anticoagulation. He underwent an uncomplicated implantation of an Amulet device. However, he experienced multiple episodes of asymptomatic non-sustained ventricular tachycardia on post-implant day 1. Transthoracic echocardiogram showed migration of the implant into the apex of the left ventricle. The patient was taken urgently for percutaneous device retrieval, which was unsuccessful. He developed a vasopressor requirement overnight and was taken to the operating room on post-implant day 2. Intraoperatively, the LAAO device was found to be lodged in the LVOT causing a fixed obstruction. He underwent sternotomy, device removal, LAA ligation, mitral valve repair, tricuspid valve repair, and closure of atrial septal defect. The patient was successfully transitioned off cardiopulmonary bypass, requiring both vasopressor and inotropic support. He was extubated the following day and was weaned off hemodynamic support shortly after.

Discussion: LAAO device migration is a rare but serious complication that requires prompt recognition and intervention. Studies have found the incidence of this complication to be 0.2-0.7%. There are various factors that contribute to dislodgement of LAAO devices, such as device under-sizing, patient anatomy, unstable anchoring, and device type. Though Amulet devices are often

used because they fit a wide range of LAAs, studies have found the Amulet is significantly more likely to embolize as compared to the Watchman.

Embolization to the left ventricle, as with our patient, can cause ventricular arrhythmias, papillary muscle rupture, or LVOT obstruction. LVOT obstruction is particularly life-threatening as it can lead to acute heart failure and hemodynamic collapse. Dislodgment of the device typically occurs in the immediate-to-early period after implantation and is an emergent situation. Embolization to the left ventricle is also more likely to need surgical intervention for retrieval, as the device can become trapped by the mitral sub-valvular apparatus.

Percutaneous retrieval is the first-line option, assuming patient hemodynamics and device location allow. In this method, a transseptal approach is often used and tools such as snares can be used to grasp the device and remove it. However, in the case of percutaneous retrieval failure, surgical intervention is required.

Conclusion: This case highlights the importance of early recognition and timely management of LAAO device migration, and the anesthetic considerations when taking care of these patients.

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SP 3 - Kiosk 7 - Poster 1

Right Ventricular Remodeling After Lung Transplantation and Its Association with One-Year Mortality

Presenting Author: *Grace Lee, MD*

Background:Right ventricular (RV) dysfunction is a common feature of end-stage lung disease, and its remodeling after lung transplantation may influence postoperative recovery and outcomes (1). This study aimed to evaluate changes in RV function before and after lung transplantation and determine whether RV remodeling is associated with one-year mortality.

Methods:We conducted a retrospective review of adult lung transplant recipients from 2017 to 2021 who underwent transthoracic echocardiography (TTE) within six months before and after transplantation. RV systolic function was quantified by fractional area change (RVFAC) and categorized qualitatively as normal, mild, or moderate/severe dysfunction. RV remodeling was defined as the difference between post- and pre-transplant RVFAC. Baseline demographics, comorbidities, and echocardiographic parameters were compared between survivors and non-survivors at one year. Univariable logistic regression and Cox proportional hazards models were used to assess associations between RV parameters and one-year mortality.

Results:Among 95 patients (mean age 61.7 ± 11.1 years; 40% female), 10 (10.5%) died within one year. Non-survivors were younger (55 ± 17.6 vs. 62.5 ± 10.4 years, p = 0.05) and had lower pre-transplant creatinine (0.65 ± 0.25 vs. 1.35 ± 1.01 mg/dL, p = 0.04) and higher rates of pre-existing left ventricular dysfunction (10% vs. 0%, p = 0.98). Pre-transplant RVFAC was lower in non-survivors (29.7 ± 6.4 vs. 35.4 ± 12.8, p = 0.18), while post-transplant RVFAC showed modest improvement in both groups (36.5 ± 10 vs. 42.1 ± 10.3, p = 0.10). The magnitude of RVFAC change (post-pre) did not differ significantly between survivors and non-survivors (6.8 ± 13.7 vs. 6.9 ± 15.2, p = 1.0). On univariable analysis, pre-transplant RVFAC (OR 0.92, 95% CI 0.91–1.02, p = 0.18), post-transplant RVFAC (OR 0.94, 95% CI 0.88–1.01, p = 0.10), RVFAC difference (OR 1.00, 95% CI 0.96–1.04, p = 0.99), and RV dysfunction severity were not significantly associated with one-year mortality. Similarly, Cox regression analyses accounting for time to event showed no significant association between these RV parameters and mortality.

Conclusion:Although modest improvement in RV function was observed after lung transplantation, neither baseline nor post-transplant RV function, nor the degree of RV remodeling, predicted one-year mortality. These findings suggest that RV remodeling alone may not be a primary determinant of early post-transplant outcomes and underscore the multifactorial nature of recovery following lung transplantation.

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SP 3 - Kiosk 7 - Poster 2

Echocardiographic Measurement of Pulmonary Artery and Systemic Blood Flow: an In-vivo Comparison

Presenting Author: *Edward Jaffe, MD*

Introduction: Echocardiographic Cardiac Output (CO) measurement by continuity is both accurate and well validated1. Stroke volume is calculated by velocity time integral (VTI) of single beat forward blood flow multiplied by the cross-sectional area (CSA) of the structure across which the

VTI is measured. CO can be measured in the Left Ventricular Outflow Tract (LVOT)2, across the Aortic Valve (AV) and in Right Ventricular Outflow Tract (RVOT) which measures pulmonary blood flow3. These structures were thought to have near circular shape allowing CSA estimation by 2D diameter. However, recent evidence suggests that the RVOT is oval in cross section4, causing measurement error. We purpose that blood flow measured by TEE across the mid-Pulmonary Artery (PA) serves as an accurate alternative measure of pulmonary blood flow. We hypothesize that Mid-PA CO should equate to LVOT blood flow in individuals without shunts or significant valvular disease.

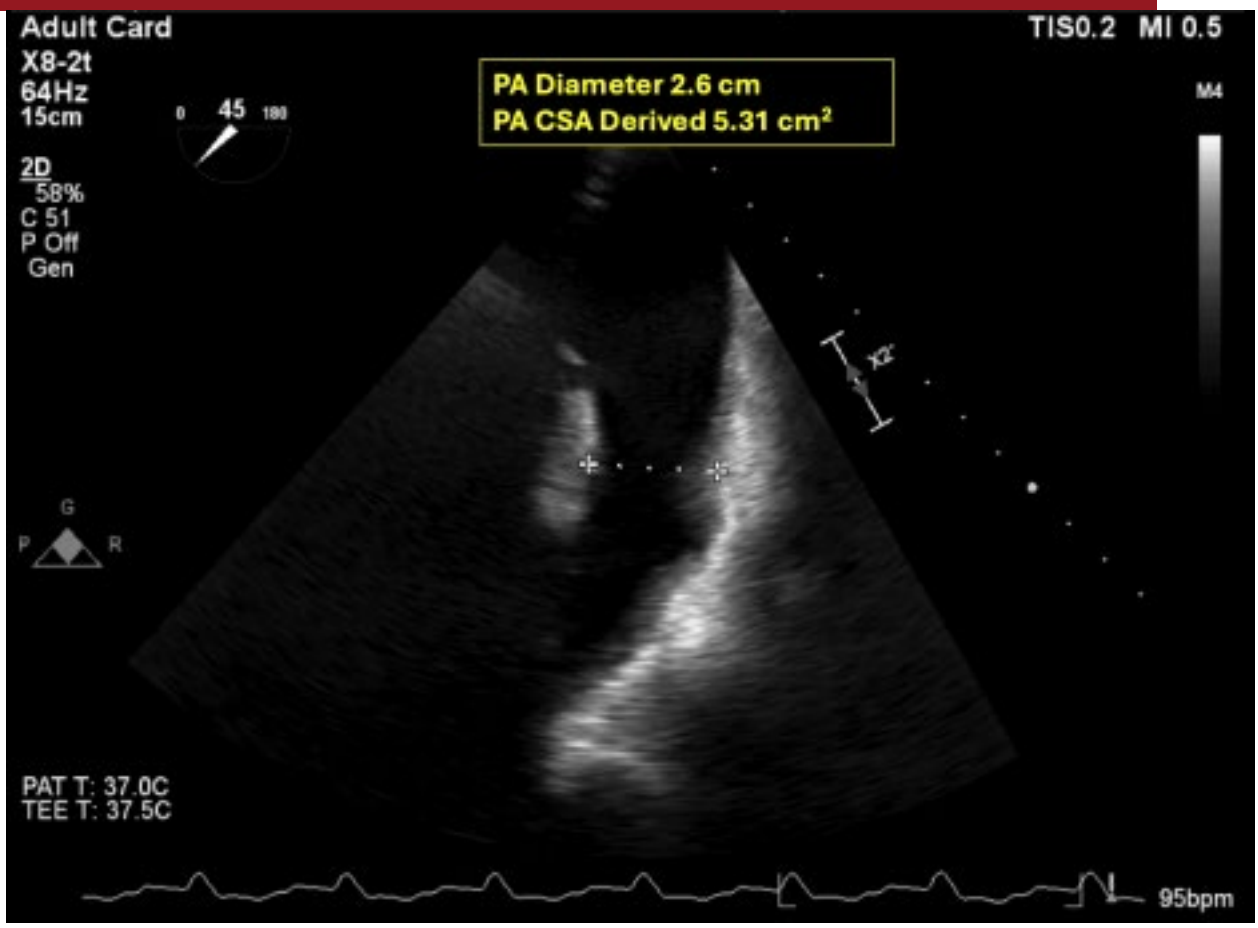
Methods: Intraoperative TEE exams with both LVOT and Mid PA CO assessment were included. In each exam, LVOT and PA CSA was derived from 2D diameters. Exclusion criteria included shunts, greater than moderate valvular regurgitation, or stenosis. 10 exams met criteria. Results were reported as Qp:Qs ratios (Pulmonary CO divided by Systemic CO). Statistics were performed in Microsoft Excel®. Ratios were log10 transformed for statistical analysis and One-Sample T-test was performed.

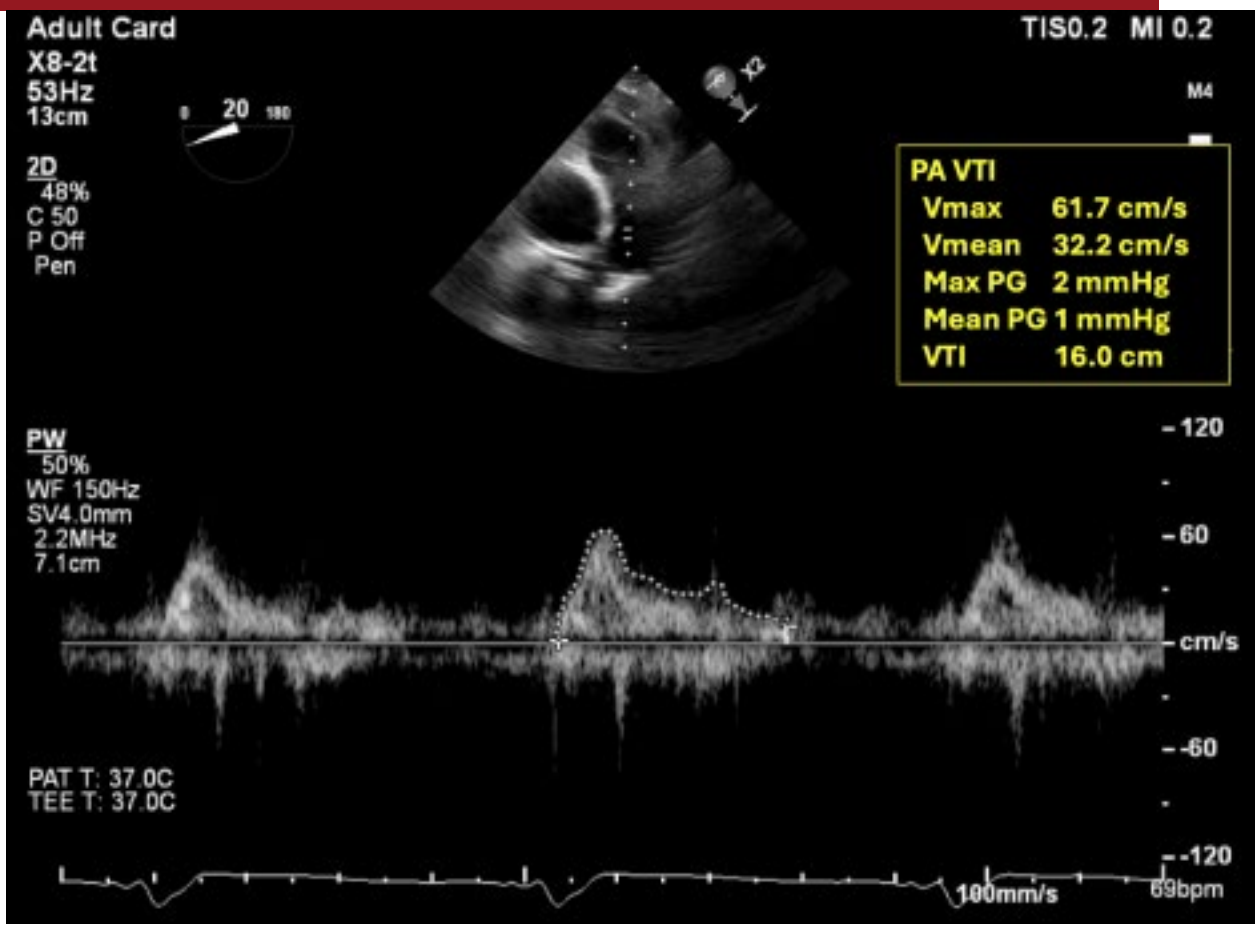
Results: The mean Qp:Qs was 1.1:1 (95% CI 1.0016:1 to 1.2668:1) the Two-Tail P-Value was 0.0475 and the One-Tail P-Value was 0.02376 with a positive T statistic of 2.29.

Discussion: Our null hypothesis was technically rejected. The positive T statistic and statistically significant One-Tail P-Value suggests the true Qp:QS relationship is greater than 1:1 . However, given the lower CI value (1.0016:1) is a clinically indistinguishable from 1:1, we argue the measurements correlate appropriately for clinical purposes. The discrepant Qp:Qs ratio likely stems from CSA calculation. The LVOT is elliptical5, and standard 2D TEE measurement in the mid-esophageal aortic valve long axis view measures the shorter diameter, therefore underestimating its CSA. In further study we will investigate if 3D LVOT CSA measurement will account for this difference.
Limitations include assuming a true Qp:Qs of 1:1 without gold standard (catherization) comparison and potential error from dynamic CO changes between PA and LVOT CO measurement.

Conclusion: Given the close correlation and clinically insignificant difference observed between mid-PA and LVOT measured CO, mid-PA measurement should be considered an acceptable clinical alternative for estimating pulmonary blood flow and systemic CO in patients without shunts or significant valvular disease.

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64Hz
15cm
2D
58%
C 51
P Off
Gen
0
45
180
PA Diameter 2.6 cm
PA CSA Derived 5.31 cm²
TIS0.2 MI 0.5
M4
G
P
R
PAT T: 37.0C
TEE T: 37.5C
95bpm



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SP 3 - Kiosk 7 - Poster 3
Echocardiographic Assessment of Pulmonary Angiosarcoma: Pulmonic Valve Replacement and Pulmonary Thromboendarterectomy

Presenting Author: *Matthew Geiser, MD*

Introduction**:** Pulmonary angiosarcoma is a rare and aggressive cancer with often nonspecific symptoms and a poor prognosis. Tumors can originate in the lungs or pulmonary arteries with treatment options including surgery, radiation, and chemotherapy. When the tumor arises from the main pulmonary artery, pulmonic valve or supravalvular stenosis can occur precipitating right heart dysfunction.
We present a case of pulmonary artery angiosarcoma and the echocardiographic assessment of pulmonary valve obstruction.

Brief Summary of the Clinical Case: A patient presented to an outside hospital with a history of shortness of breath and fatigue. Transthoracic echocardiogram and MRI imaging showed rapidly progressing severe pulmonic valve / supravalvular stenosis (peak gradient 110 mmHg) and severe right ventricular (RV) dysfunction with a concern for angiosarcoma involving the pulmonary artery. Patient was transferred to our institution for consideration of surgical resection.
Patient underwent pulmonic valve replacement, repair of pulmonary artery stenosis, and pulmonary thromboendarterectomy (PTE). Intraoperative transesophageal echocardiography (TEE) showed severe RV with preserved LV function. Flow acceleration was seen in the main pulmonary artery distal to the pulmonic valve, but the valve was difficult to visualize on TEE. Once on CPB, there was noted to be a near occlusive mass involving the pulmonary root and right ventricular outflow track with obliteration of the pulmonic valve. Initial attempts to separate from CPB were unsuccessful due to severe RV dysfunction and hemodynamic instability despite escalation of inotropes, pressors, and initiating iNO. This necessitated VA ECMO deployment. Patient was brought to the ICU with an open chest on VA ECMO with TEE showing severe RV dysfunction. Final pathology was positive for primary pulmonary angiosarcoma.

Discussion: Obtaining an image of the pulmonary valve is more challenging with TEE as it is the most anterior of the heart valves and therefore farthest away from the probe. Additionally, this can make doppler assessment challenging as alignment can vary based on patient anatomy.

Views

- Mid-esophageal right ventricular inflow-outflow
- Mid-esophageal ascending aortic short-axis
- Upper-esophageal aortic arch short-axis
- Transgastric Basal RV view

Grading of pulmonic stenosis is by velocity or peak pressure gradient

- Severe: > 4 m/s or > 64 mmHg
- Moderate: 3-4 m/s or 36-64 mmHg
- Mild: < 3 m/s or < 36 mmHg

Calculations

- PASP = RVSP – PV pressure gradient

Impact of Echocardiographic findings

- Assessing the severity and cause of obstruction
- Identifying the level of obstruction (sub-valvular vs valvular vs supra-valvular) ● Assessing other abnormalities caused by this obstruction (RV dysfunction, tricuspid regurgitation)

Teaching Points of the Case

- Views to assess pulmonic valve and pulmonary artery
- Grading of pulmonic stenosis
- How to differentiate between pulmonic valve stenosis versus supravalvular pulmonic stenosis

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SP 3 - Kiosk 7 - Poster 4

Left Atrial Strain and Predicting Recurrence of Atrial Tachyarrhythmia in Patients undergoing Convergent Maze

Presenting Author: *Arpine Galstyan, MD*

Background: The Convergent Maze is a two-stage hybrid procedure that combines surgical epicardial ablation of left atrial arrhythmogenic sites with catheter-based endocardial ablation in a staged fashion for the treatment of atrial fibrillation (AF). Left atrial strain (LAS) quantification has demonstrated prognostic value in predicting AF recurrence in at-risk individuals undergoing catheter ablation. The objective of the current study is to determine whether two-dimensional speckle tracking LAS predicts six-month freedom from atrial tachyarrhythmias (ATA) and symptoms following the Convergent Maze, and to assess the association between baseline and postoperative LAS phases (LAS reservoir and LAS conduit) and ATA recurrence.

Methods: A total of 45 patients with either persistent AF or atrial flutter (AFL) scheduled to undergo the Convergent Maze were recruited between January 2023 and August 2025. Pre-operative transthoracic echocardiography (TTE) and two-dimensional speckle tracking echocardiography were performed in all patients before and after epicardial ablation during the same hospitalization. The primary outcome was recurrence of ATA or related symptoms within six months of the Convergent Maze following a three-month blanking period. Baseline and post-procedural clinical and echocardiographic variables, including LAS parameters, were compared between patients with and without ATA recurrence using t-tests or Chi-square/Fisher's exact tests as appropriate.

Results/Discussion: Among the 45 patients, eight were either lost to follow-up or had incomplete data. Of the remaining 37, 30 (81%) maintained a stable sinus rhythm (SR) at their six-month follow-up, while seven experienced a recurrence of ATA. In addition, 27 (73%) remained symptom-free, while 10 (27%) did not. Baseline LAS measurements demonstrated mean LAS reservoir and LAS conduit values of 11.2 ± 5.6 and –9.9 ± 4.6, respectively. Post-procedure measurements of LAS reservoir and LAS conduit yielded mean values of 8.7 ± 4.2 and -7.8 ± 4.2, respectively. During the post-procedure assessment, 17 patients (45.9%) were noted to be in AF, 19 (51.4%) were in SR, and one (2.7%) was atrially paced. No significant difference in pre- and post-intervention measurements of LAS reservoir ($p = 0.153$) and LAS conduit ($p = 0.402$) was observed in those who were stable in SR during their six-month follow-up and those who experienced a recurrence in ATA (see Figure 1). Given that approximately 81% of patients remained free of ATA and 73% of patients were free of symptoms at six-months, the Convergent Maze has demonstrated to be an effective treatment option for patients with ATA. However, if LAS is to be used as a predictor of recurrence, assessment at a later point in time is required to better determine its predictive value and to characterize the magnitude of LAS change following endocardial ablation.

Conclusion: This study was the first to investigate the relationship between LAS parameters and ATA recurrence in patients undergoing the Convergent Maze. Further research investigating this relationship is needed, ideally after endocardial ablation.

Image Uploads

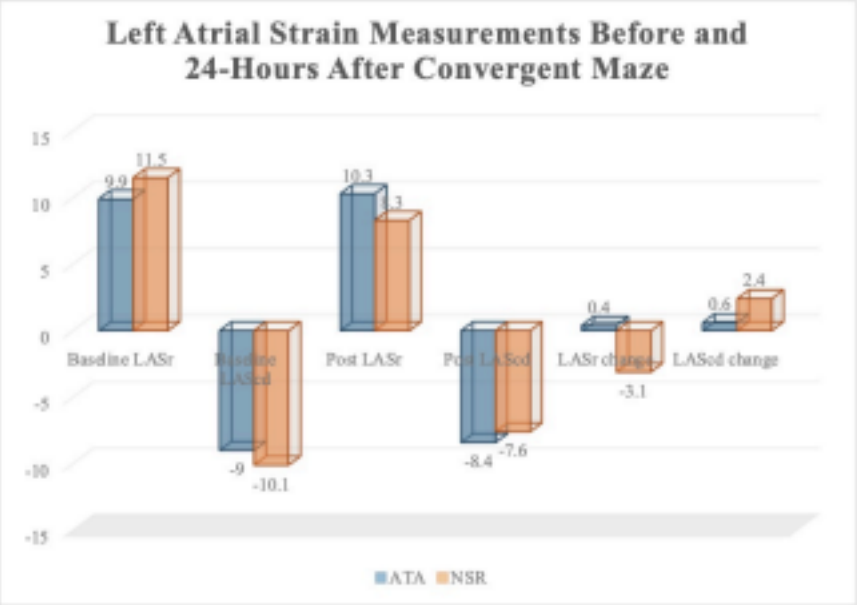


Figure 1. Baseline and post-operative left atrial strain parameters 24-hours after Convergent Maze in patients who remained stable in normal sinus rhythm during their six-month follow-up and those who experienced a recurrence in atrial tachyarrhythmia.
Abbreviations: LASr, Left Atrial Strain reservoir; LAScd, Left Atrial Strain conduit; ATA, Atrial Tachyarrhythmia; NSR, Normal Sinus Rhythm

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SP 3 - Kiosk 7 - Poster 5
3D Tranesophageal echo reconstruction of the inter-atrial septum saves the day
Presenting Author: *Ravie Abozaid, MBBCH*

Introduction**:** 3D TEE has the potential to improve the safety and precision of transeptal puncture (TSP), providing real-time volumetric imaging of the Fossa Ovale (FO), interatrial septum (IAS), and surrounding structures. It is increasingly becoming a primary guiding modality in complex structural heart procedures and related complications 1,2.

Here, we discuss the importance of 3D septal reconstruction to guide TSP during mitral transcatheter edge-to-edge repair (M-TEER) and during management of a large iatrogenic atrial-septal defect (IASD).

Brief Summary of the Clinical Case: 81-year-old female with a history of severe AS (s/p TAVR), permanent pacemaker, nocturnal hypoxia, AF s/p LAAO and severe eccentric mitral regurgitation (MR) presenting for M-TEER. Intraoperative TEE showed significant calcification of the valve and chordae, a broad-based jet and severe MR located in the A2-P2 segment.

TSP was performed, the MitraClip guide sheath was inserted and abrupt movement of the apparatus towards the PFO was noted. Real-time 3D imaging was used to immediately diagnose and define the large IASD, which represented tearing across the intra-atrial septum. 3D MPR was used to measure the IASD defect precisely (Fig 1).

Difficulty with re-crossing the IASD was solved with 3D imaging and guidance across. The MitraClip was deployed in an A2-P2 configuration uneventfully after adequate bileaflet insertion and evaluation of residual MR and mitral hemodynamics.

Evaluation of the residual ASD revealed large-volume left-to-right shunting and high oxygen saturation in the right-sided atrium. Given these findings and her severe tricuspid regurgitation (TR), the decision was made to close the IASD.

Under 3D TEE guidance, an attempt to close the defect with a 48 mm Gore Occluder failed. Then, a 36 mm Amplatzer ASD occluder was successfully deployed in a stable position with no residual shunting.

Discussion

3D TEE with MPR helps identify an IASD, define the defect shape, evaluate the adequacy of the tissue rim, and assess the relation of the defect to surrounding structures – all crucial for determining candidacy for percutaneous closure. 3D, 2D color Doppler can be superimposed to assess shunt flow and identify fenestrations that may be missed 3.

In this case, a very large IASD (1.7 x 2.8 cm) was identified (Fig 2), with a significant shunt and concern for worsening RV function and TR; repair was prudent. Thus, demonstrating why real-time 3D TEE MPR is an indispensable skill for visualizing the FO, interatrial septum, catheters during transeptal puncture, diagnosing and treating IASD and other complications. We advocate routine use to guide transeptal puncture and IASD assessment, so you can use it when complex cases

require it.

Impact of Echocardiographic findings

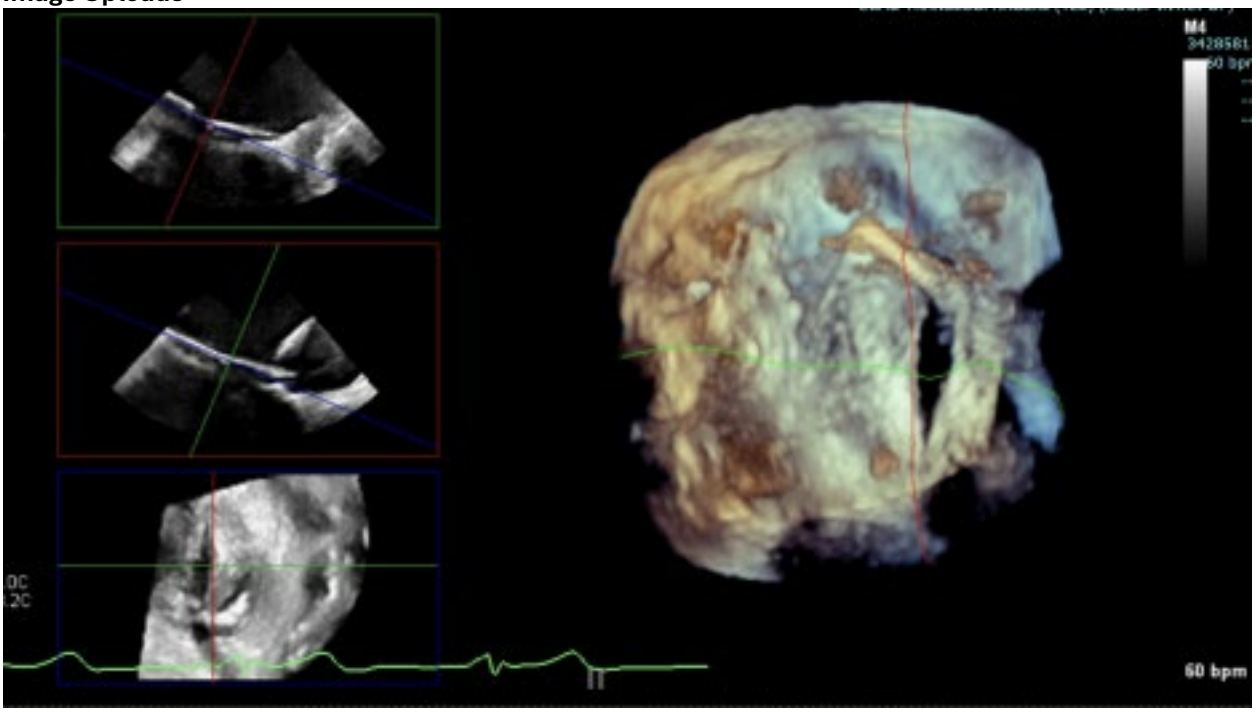
3D TEE guidance is indispensable for navigating complex steps, such as TSP, optimal device positioning, immediate detection of complications and guiding rescue actions.

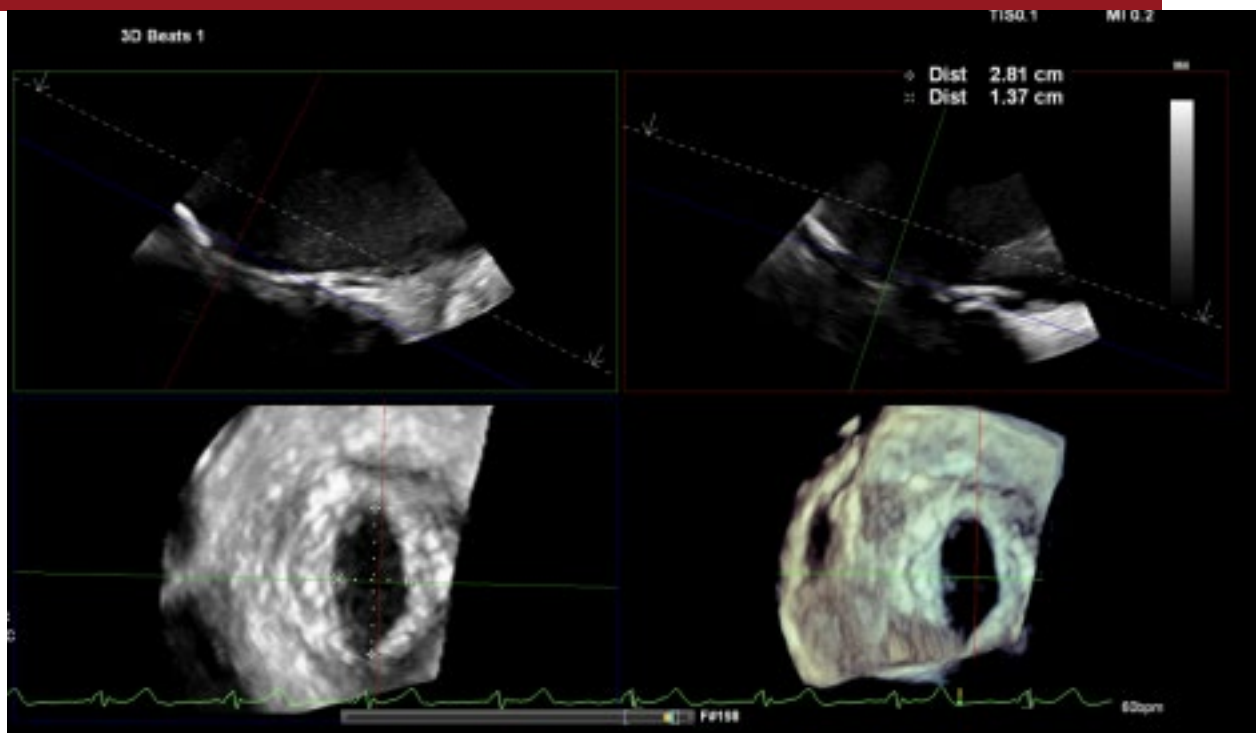
Teaching Points of the Case

3D TEE Practice is key to guiding the proceduralist through difficult transseptal crossing and identifying and treating complications in the heat of battle.

Understanding the anatomy and orientation of the left and right atrial septa and orienting them for the proceduralist relative to known structures can be achieved through routine practice of 3D TEE of the IAS. Ensuring it can be applied when complex cases require it.

Image Uploads





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SP 3 - Kiosk 7 - Poster 6
TEE use for Endovascular Ascending Aortic Repair
Presenting Author: *Nicholas Flores-Conner, MD*

Introduction: Type A aortic dissection is a common emergency procedure in the CT ORs. However, the presence of aortic dissection is rare in the general population and has a high mortality rate if not intervened on. We present a case of type A aortic dissection on a patient who underwent endovascular repair given high surgical risk. TEE was utilized to evaluate the location of the tear, the deployment of the endovascular graft, coronary artery proximity to the graft, and AV insufficiency severity. Ascending aortic endovascular treatment has been attempted on rare occasion with a systematic review published in 2019 reporting a total of 60 cases on the literature. However, this technique is being investigated further and will likely be more present in our ORs in the future.

Brief Summary of the Clinical Case: 76 y.o. F patient with PMH significant for ESRD on PD s/p right nephrectomy 2/2 RCC, HTN, COPD with recent hospitalization and intubation leading to chronic respiratory failure, severe frailty, neurocognitive decline, cirrhosis with ascites, bilateral pleural effusions, hemopericardium, and chronic type B aortic dissection who presented with new diagnosis of type A aortic dissection. Given her complex medical status, chronicity of disease, and frailty, ascending TEVAR was offered as management strategy with no attempt for open sternotomy. The false lumen extended to the aortic root and compromised the integrity of the left and non-coronary cusps creating severe AI. We performed ascending aortic TEVAR with TEE and flouroscopic guidance to minimize the risk of aortic leaflet entrapment and coronary artery occlusion. At the end of the procedure, the AI decreased to moderate. The TEVAR graft extended to the aortic root but did not cause obstruction to coronary flow or leaflet restriction.

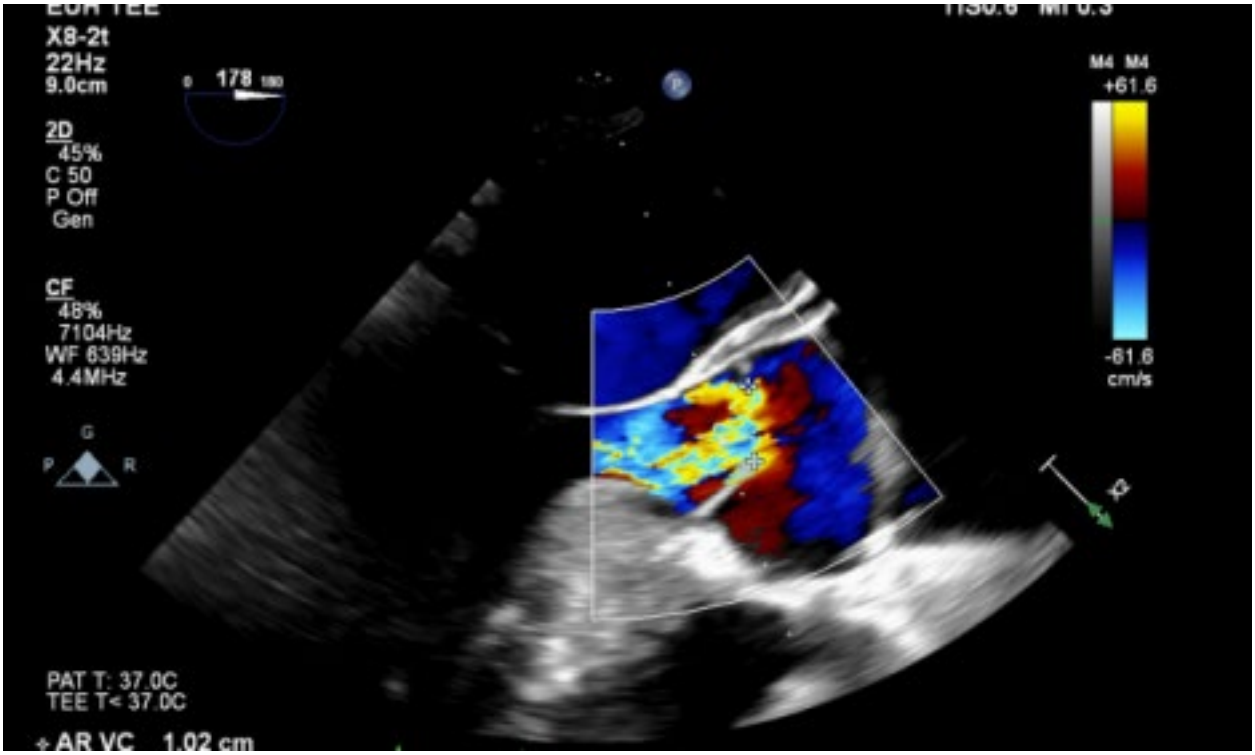
Discussion

- The use of 2D and 3D imaging for quantification of AI and its mechanism can help in determining if the patient will require further interventions like TAVR.
- An anatomical survey allows for location of the coronary ostia, extension of the dissection flap, and origin of the tear to maximize the safety of the graft deployment and minimize the risk of extension, rupture, or inadvertent coronary artery occlusion.
- We utilized 2D imaging of the ME long axis of the AV and long axis of the ascending aorta to identify the extent of the dissection flap and location of the tear.
- X-plane allowed for simultaneous identification of the anatomy of the true lumen and compromise of the non-coronary and left coronary cusps causing severe AI. The same technique was utilized to evaluate for potential leaflet entrapment.
- 3D and 2D imaging was utilized to complete the anatomical survey when the TEVAR graft was being positioned and deployed to ensure occlusion of the tear, and avoid coronary ostia occlusion.

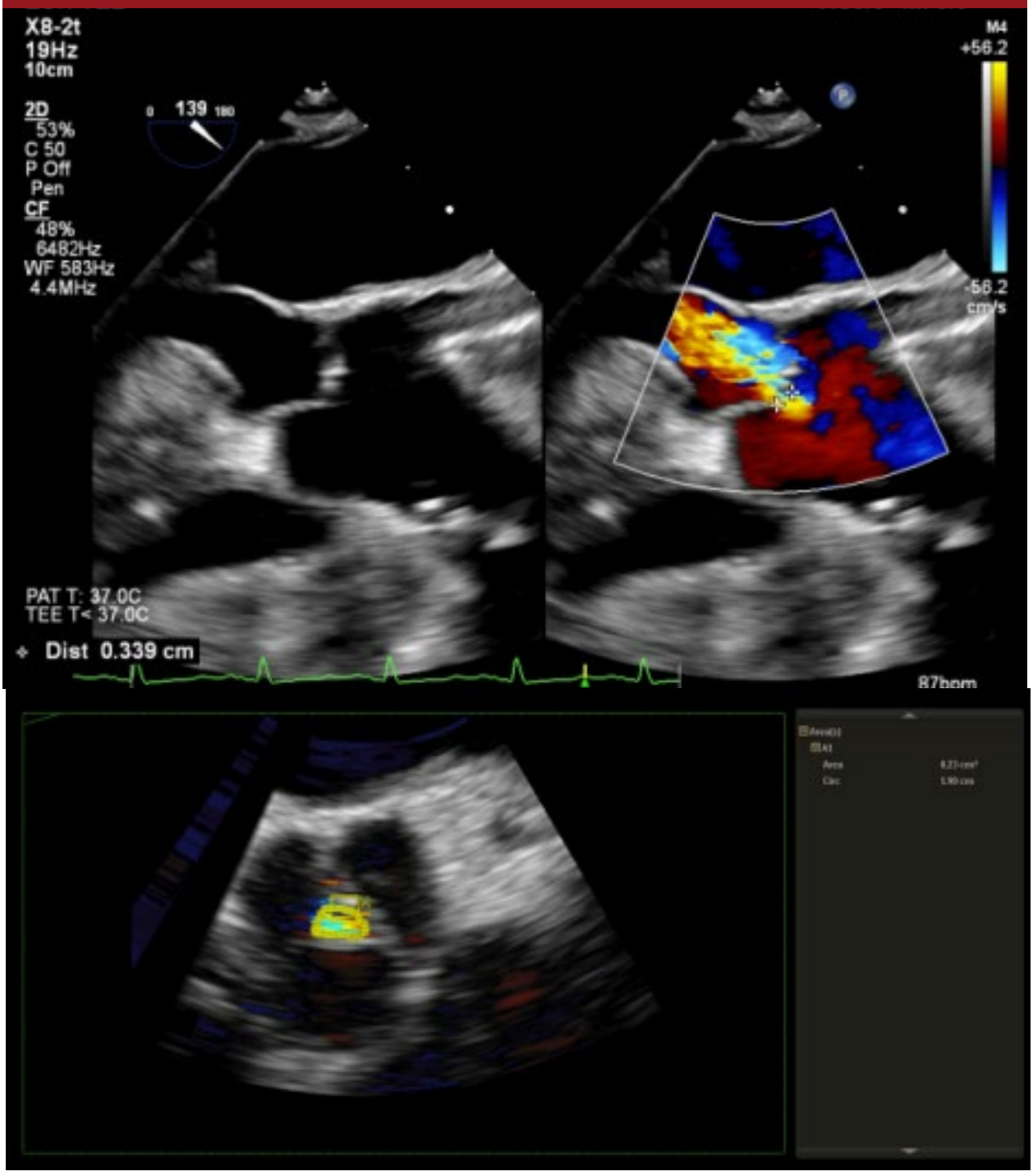
Impact of Echocardiographic findings: Echocardiographic findings were utilized to confirm patency of the coronary ostia, improvement of severity grading after endovascular repair, and leaflet excursion.

Teaching Points of the Case: Endovascular ascending aortic repair is a developing technique that will likely appear in our cardiothoracic operating rooms in the future. We discuss the utility of transesophageal echocardiography to determine appropriate positioning of the ascending graft, patency of the coronary ostia, evaluation of aortic insufficiency, leaflet excursion, and tear obliteration.

Image Uploads




X8-2t
53Hz
10cm
2D
54%
C 50
P Off
Gen
120
M4
PAT T: 37.0C
TEE T< 37.0C
Dist 2.92 cm
93bpm



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SP 3 - Kiosk 8 - Poster 1

Distinct Neutrophil Marker Signatures Associate with Specific Long COVID Symptoms – Implications for Cardiac Anesthesiologists

Presenting Author: *Emma Huang*

Background/Introduction: Long COVID encompasses persistent health problems following acute SARS-CoV-2 infection, including cardiovascular symptoms such as heart palpitations and gastrointestinal manifestations. Neutrophils play a significant role in determining acute COVID-19 severity, and emerging evidence links neutrophil extracellular traps and persistent neutrophil activation to long COVID pathophysiology. However, whether specific neutrophil markers associate with individual long COVID symptoms has not been analyzed. This study aimed to determine whether distinct low-density neutrophil (LDN) and normal-density neutrophil (NDN) markers correlate with specific long COVID symptoms, which has implications for anesthesiologists caring for these patients perioperatively.

Methods: Blood samples were collected from 65 patients with long COVID. Peripheral blood mononuclear cells were isolated and analyzed using mass cytometry (CyTOF) with custom antibody panels targeting LDN and NDN markers. Data were analyzed using Cytobank software and the CyTOF workflow package. Patients were stratified by presence or absence of each symptom, and neutrophil marker expression was compared between groups using Wilcoxon rank-sum testing. Statistical significance was set at p less than 0.05.

Results: Symptom-specific analysis revealed significant associations between individual long COVID symptoms and distinct neutrophil markers. Heart palpitations, the most common cardiovascular symptom, showed the highest number of associations across both LDN and NDN panels, including ndnCD66b mean fluorescence intensity, ndnCD11b mean fluorescence intensity, ldnCD66b mean fluorescence intensity, ldnCD10, and multiple CD66b quartile markers. Rhinorrhea significantly associated with ldnCD63 mean fluorescence intensity markers and ldnCD35 mean fluorescence intensity. From a marker-driven perspective, ldnCD44 mean fluorescence intensity quartile 1 associated significantly with diarrhea and dyspnea. In the NDN panel, ndnCD44 mean fluorescence intensity and ndnCD11b mean fluorescence intensity were most frequently associated with long COVID symptoms.

Conclusion: Specific long COVID symptoms demonstrate significant associations with distinct neutrophil marker signatures, suggesting heterogeneous immune phenotypes underlie different manifestations of post-acute sequelae. Understanding symptom-level immune associations provides a framework for characterizing long COVID heterogeneity and may inform targeted therapeutic approaches and perioperative risk stratification for individual symptom management in surgical patients presenting for cardiac or noncardiac surgeries.

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SP 3 - Kiosk 8 - Poster 2

Parasternal Nerve Block with Rectus Sheath Block As An Adjunct For Postoperative Pain Control Following Median Sternotomy in Cardiac Surgery

Presenting Author: *Stephen Lockwood, MD*

Background: Acute post-operative pain after median sternotomy is common in patients following cardiac surgery and is a strong predictor for the development of chronic post-operative pain [1]. A multimodal management technique during the perioperative period is advocated for in these patients, with regional anesthetic techniques representing a safe, rapid intervention that has the potential to improve patient outcomes in this population. In particular, the parasternal block in conjunction with the rectus sheath block stand as low risk, expeditious procedures that can be implemented by the anesthesiology team. We hypothesize that these blocks performed at the conclusion of surgery during which patients undergo median sternotomy will reduce opioid requirements and overall pain scores in the immediate post operative period

Methods: Following IRB approval a retrospective chart review was carried out from January 2020 through January 2025. Inclusion criteria were adult patients (age >18) undergoing non-emergency

cardiac surgery requiring primary sternotomy who had received ultrasound guided parasternal nerve blocks and rectus sheath blocks in the OR following completion of surgery with either 0.2% ropivacaine or 0.25% bupivicane. Exclusion criteria included patients with open chest at the end of their index surgery, requirement for mechanical circulatory support, heart or lung transplantation, aortic surgery, redo sternotomy, emergency surgery, and conversion from thoracotomy to sternotomy or vice-versa. Primary endpoints were numeric rating scale (NRS) pain scores and oral morphine equivalent (OME) opioid consumption in the first, second, and third 24h periods after end of surgery.

Results: From 2020-2025 a total of 50 patients were identified that met the above criteria ("Block Group"). These were compared with a total of 4201 patients in the same time period ("No Block Group") who met inclusion/exclusion criteria, but did not receive nerve blocks. Statistical analysis was conducted using multivariable linear regression adjusting for gender, age, weight, procedure time, and LOS.
In the first 24 hours following surgery there were higher median NRS pain scores reported for the block groups (6 vs 5.5). This trend continued into the 24-48 hour period (5 vs 4.5) and 48-72 hour period (4.25 vs 3).
In the block group oral morphine equivalent (OME) administration was lower intraoperatively (180 vs 195), but higher in the first 24 hours postoperatively (141 vs 90), the 24-48 hour period (69.5 vs 46), and the 48-72 hour period (40.6 vs 22.5). These differences in OME requirements were statistically significant

Conclusion: Based on our analysis, the incorporation of parasternal nerve blocks with rectus sheath blocks into the multimodal pain regimen of patients undergoing cardiac surgery with median sternotomy does not appear to improve NRS pain scores or reduce opioid requirements in the first 72 hours following surgery. Limitations of this study include its single center, retrospective structure and small sample size. Heterogeneity in block technique and interoperator variability were not accounted for. Similarly the potential for selection bias in those patients who did receive blocks represents a potential confounder.

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	No Block (N=4201)	PSB (N=50)	Overall (N=4251)
Gender			
Female	1315 (31.3%)	15 (30.0%)	1330 (31.3%)
Male	2886 (68.7%)	35 (70.0%)	2921 (68.7%)
Age	66.0 [57.0, 73.0]	64.5 [57.0, 70.0]	66.0 [57.0, 73.0]
Weight	87.9 [75.0, 102]	94.4 [79.9, 104]	88.0 [75.1, 102]
Proc Time (hours)	7.63 [6.38, 9.22]	6.69 [5.82, 7.56]	7.63 [6.37, 9.19]
LOS (days)	8.92 [6.65, 13.6]	7.67 [5.88, 10.0]	8.91 [6.63, 13.5]

Table 1: Patient Demographics

	NoBlock (N=4201)	PSB (N=50)	Overall (N=4251)
Median Pain Score			
0 to 24	5.50 [3.50, 7.00]	6.00 [5.00, 7.88]	5.50 [3.50, 7.00]
24 to 48	4.50 [2.50, 6.00]	5.00 [4.00, 6.50]	4.75 [2.50, 6.00]
48 to 72	3.00 [0, 5.00]	4.25 [2.00, 6.00]	3.00 [0, 5.00]
OME			
Intraop	195 [150, 300]	180 [150, 279]	195 [150, 300]
0 to 24	90.0 [52.5, 131]	141 [96.0, 165]	90.0 [52.5, 133]
24 to 48	46.0 [22.5, 82.5]	69.5 [38.8, 100]	47.5 [22.5, 82.5]
48 to 72	22.5 [5.00, 52.5]	40.6 [15.0, 83.8]	22.5 [5.00, 52.5]

Outcomes	Beta (95% CI)	P value
Median Pain score		
0 to 24	0.81 (0.08, 1.54)	**0.029**
24 to 48	0.66 (-0.06, 1.37)	0.072
48 to 72	0.67 (-0.05, 1.38)	0.068
Opioid consumption (OME)		
0 to 24	27.7 (13.3, 42.1)	**<0.001**
24 to 48	18.1 (5.57, 30.6)	**0.005**
48 to 72	14.6 (4.27, 24.9)	**0.006**

Table 3: Analysis

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SP 3 - Kiosk 8 - Poster 3

Qualitative Right Ventricular Assessment For Patients With Pulmonary Hypertension: The Good……and the Ugly

Presenting Author: *Andrew Maslow, MD*

Introduction: Recent guidelines deemphasize ‘eyeballing’ or visual estimation of right ventricle (RV) size and function, while favoring quantitative analyses (1,2). Despite this, qualitative assessment (visual assessment; ‘eyeballing’) of RV function and size is the most frequently reported and relied upon parameter (3). While studies have compared qualitative measures of the RV to reference measures, analyses between qualitative assessment and patient outcome (4) or hemodynamic correlations are few if at all. The present study explores the relationship between qualitative RV assessment during transthoracic echocardiography (TTE) and hemodynamic performance and outcome in patients presenting with pulmonary hypertension (PHTN).

Design & Setting: Retrospective cohort at a tertiary-care university hospital coronary care unit (CCU).

Participants: 159 adults with PHTN admitted to a CCU who underwent RHC and TTE.

Measurements: Patients were admitted for cardiac (67%), pulmonary (30%) or other (3%) diagnoses. Demographics were not associated with mortality. Data included RHC hemodynamics, TTE qualitative assessments of RV size and function, and LVEF by Simpson’s equation. RV size and function were described as normal, mild, moderate, and severe. For additional analysis RV function and size were combined to form three groups: 1) n=68-normal/normal, mild/normal and normal/mild; 3) n=30- severe/severe, moderate/severe and severe/moderate; and group 2) n=61-all others.

Statistics: Multiple types of regression analyses and model checks to determine how RV size and RV systolic function relate to hemodynamic measures and clinical endpoints at 12 months to include renal function, readmission, and mortality

Results: Neither RV function nor size was associated with outcome. Normal RV function pulmonary artery pressures, resistance, compliance and pulmonary to systemic hemodynamic ratios were significantly lower compared to either moderate or severe RV dysfunction (Table 1). The normal sized RV recorded significantly higher SBP, and lower pulmonary pressures,

pulmonary vascular resistance and pulmonary to systemic hemodynamic ratios compared to mildly, moderately, and severely dilated groups (Table 2). Differences were also reported between mild and severely dilated RV. For both RV function size, there were no differences between mild and moderate and between moderate and severe groups. Combining RV function with size reported pulmonary and pulmonary to systemic hemodynamic differences between groups 1 and 2, 1 and 3, and 2 and 3 (Table 3). There was a borderline significant difference in mortality between groups 1 and 3. For the analyses of RV function, size, and combined groups, there were no significant differences in HR, MAP, CO, SV, PAPi, or SPP. There was no interaction between LVEF and RV size in either of the three analyse

Conclusions: For patients with PHTN, qualitative assessment of RV size or function differentiated hemodynamics between normal size or function from other groups. Neither assessment, alone, was associated with adverse outcome, while combining assessments of RV size with RV function has added value in defining hemodynamic differences and outcome.

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Table 1: Right ventricular function/dysfunction

	Normal n=62	Mild n=37	Moderate n=40	Severe n=20	p value
Heart Rate (bts/min)	85.4+/-16.8	82.7+/-14.4	85.4+/-15.1	96.4+/-26.2	0.161
MAP (mmHg)	94.1+/-15.8	91.2+/-15.0	87.4+/-15.3	90.0+/-18.8	0.204
SBP (mmHg)	131.0+/-20.2	123.9+/-22.0	120.9+/-20.6	120.1+/-27.3	0.013
Cardiac Output (L/min)	5.40+/-1.66	5.04+/-1.33	5.22+/-2.70	4.86+/-1.63	0.323
Stroke Volume (ml/beat)	64.8+/-22.9	62.9+/-20.1	61.9+/-27.9	59.4+/-49.7	0.106
RAP (mmHg)	8.2+/-4.9	9.7+/-5.6	9.9+/-2.3	11.9+/-5.9	0.059
PASP (mmHg)	46.2+/-14.4	53.6+/-18.4	59.4+/-17.2**	66.7+/-20.7**	<0.0001
mPAP (mmHg)	31.0+/-9.5	35.4+/-11.6	39.0+/-11.0@	44.9+/-12.0**	<0.0001
PADP (mmHg)	20.3+/-7.6	23.4+/-8.4	25.7+/-8.6@	31.6+/-8.6**#	<0.0001
PVR (WU)	3.1+/-2.1	3.5+/-2.4	4.4+/-3.1@	6.1+/-4.3**#	<0.0001
PAC	2.9+/-1.6	2.4+/-1.2	2.1+/-1.4	1.9+/-1.8@	0.001
PAPi	4.5+/-4.2	3.7+/-2.5	4.8+/-3.7	4.9+/-7.7	0.647
RAP/PASP	0.18+/-0.12	0.19+/-0.10	0.18+/-0.10	0.19+/-0.09	0.801
PVR/SVR	0.19+/-0.11	0.21+/-0.11	0.27+/-0.15@	0.36+/-0.19**#	<0.0001
mPAP/MAP	0.34+/-0.12	0.39+/-0.13	0.46+/-0.16**	0.52+/-0.16**	<0.0001
PASP/SBP	0.36+/-0.13	0.44+/-0.16	0.50+/-0.17**	0.57+/-0.19**	<0.0001
SPP (mmHg)	85.9+/-16.5	81.5+/-14.0	77.5+/-15.3	78.1+/-19.8	0.056
Mortality (n(%))	12 (19.4%)	4 (10.8%)	10 (25%)	8 (40.0)	0.076
Readmission (n(%))	55 (88.7%)	32 (86.5%)	37 (92.5%)	20 (100%)	0.509
% Decrease GFR	21.9+/-27.8	22.6+/-29.2	24.3+/-25.6	22.4+/-26.3	0.936
% Increase SCr	35.3+/-43.6	39.2+/-52.1	40.2+/-54.0	43.8+/-71.4	0.958

vs normal O = $p < 0.05$; @ = $p < 0.02$; ** = $p < 0.001$;
vs mild # $p < 0.05$

Table 2: Right ventricular size

	Normal n=47	Mild n=31	Moderate n=47	Severe n=34	p value
Heart Rate (bts/min)	86.8+/-16.4	86.0+/-13.2	80.8+/-16.5	92.8+/-22.1	0.110
MAP (mmHg)	96.1+/-16.6	87.5+/-14.7	89.5+/-14.0	90.1+/-17.8	0.099
SBP (mmHg)	134.9+/-22.9	119.3+/-18.1^{O}	122.6+/-21.7^{O}	121.8+/-21.1^{O}	0.006
Cardiac Output (L/min)	5.5+/-1.7	4.9+/-1.4	5.1+/-1.6	5.2+/-2.8	0.455
Stroke Volume (ml/beat)	64.0+/-22.1	59.1+/-21.6	68.0+/-35.7	58.2+/-28.7	0.201
RAP (mmHg)	8.0+/-5.4	9.9+/-4.8	9.3+/-5.0	11.1+/-6.0	0.058
PASP (mmHg)	43.8+/-14.9	51.1+/-17.9	58.0+/-17.3**	64.3+/-17.1$^{**\#}$	< 0.0001
mPAP (mmHg)	29.8+/-10.2	34.9+/-12.0	37.3+/-10.0**	43.0+/-11.1$^{**\#}$	<0.0001
PADP (mmHg)	19.6+/-8.1	23.8+/-8.7^{O}	24.0+/-7.3$^{@}$	29.3+/-9.2$^{**\#}$	<0.0001
PVR (WU)	2.6+/-1.9	3.5+/-1.9^{O}	4.1+/-2.1**	5.9+/-4.4**	<0.0001
PAC	3.1+/-1.7	2.6+/-1.4	2.3+/-1.5^{O}	1.9+/-1.2**	0.0004
PAPi	4.3+/-4.6	3.5+/-2.5	4.7+/-3.1	5.2+/-6.5	0.273
RAP/PASP	0.18+/-0.11	0.21+/-0.14	0.17+/-0.10	0.18+/-0.09	0.519
PVR/SVR	0.15+/-0.08	0.21+/-0.10$^{@}$	0.26+/-0.13**	0.35+/-0.18$^{**\#}$	<0.0001
mPAP/MAP	0.32+/-0.11	0.41+/-0.16$^{@}$	0.43+/-0.13**	0.49+/-0.15**	<0.0001
PASP/SBP	0.33+/-0.12	0.44+/-0.17$^{@}$	0.49+/-0.16**	0.54+/-0.17$^{**\#}$	<0.0001
SPP (mmHg)	88.1+/-16.4	77.6+/-14.4	80.2+/-15.0	79.0+/-17.7	0.027
Mortality (n(%))	8 (17.0%)	6 (19.4%)	11 (23.4%)	9 (26.5%)	0.742
Readmission (n(%))	45 (95.7%)	26 (83.9%)	40 (85.1%)	33 (97.1%)	0.268
% Decrease GFR	20.6+/-30.8	26.6+/-29.2	21.6+/-20.3	23.6+/-29.2	0.595
% Increase SCr	35.4+/-46.9	44.4+/-49.5	32.4+/-39.3	46.0+/-72.8	0.7564

vs normal O = $p < 0.05$; @ = $p < 0.02$; ** = $p < 0.001$;
vs mild # $p < 0.05$

Table 3: Combination of right ventricular size and function

	Group1 n=68	Group 2 n=61	Group 3 n=30	p value
Heart Rate (bts/min)	85.5+/-15.4	84.3+/-15.9	91.2+/-24.3	0.536
MAP (mmHg)	93.9+/-16.1	88.8+/-13.8	90.0+/-19.1	0.136
SBP (mmHg)	130.6+/-21.9	121.0+/-19.0^{O}	122.8+/-25.7	0.018
Cardiac Output (L/min)	5.3+/-1.7	5.1+/-1.3	5.3+/-3.1	0.529
Stroke Volume (ml/beat)	63.0+/-22.7	62.4+/-20.9	63.8+/-46.7	0.277
RAP (mmHg)	8.3+/-5.4	9.6+/-4.6	11.5+/-6.3^{O}	0.027
PASP (mmHg)	44.9+/-14.7	57.0+/-16.4**	67.7+/-18.8$^{**\#}$	<0.0001
mPAP (mmHg)	30.4+/-9.9	37.4+/-10.5**	44.7+/-11.2$^{**\#}$	<0.0001
PADP (mmHg)	22.2+/-7.7	24.4+/-8.0**	30.7+/-8.9$^{**\#}$	<0.0001
PVR (WU)	2.9+/-2.0	3.9+/-2.1$^{@}$	6.4+/-4.4$^{**\#}$	<0.0001
PAC	3.0+/-1.6	2.2+/-1.1$^{@}$	2.00+/-1.8**	<0.0001
PAPi	4.2+/-4.1	4.3+/-2.9	5.3+/-6.8	0.582
RAP/PASP	0.19+/-0.13	0.18+/-0.10	0.18+/-0.09	0.897
PVR/SVR	0.16+/-0.08	0.24+/-0.11**	0.37+/-0.19$^{**\#}$	<0.0001
mPAP/MAP	0.33+/-0.11	0.43+/-0.14**	0.52+/-0.16$^{**\#}$	<0.0001
PASP/SBP	0.35+/-0.12	0.48+/-0.15**	0.57+/-0.19**	<0.0001
SPP (mmHg)	85.6+/-16.1	79.2+/-14.3	78.4+/-19.3	0.037
Mortality (n(%))	12 (17.6%)	11 (18.0%)	11 (36.7%)^{O}	0.097
Readmission (n(%))	61 (89.7%)	53 (86.9%)	30 (100%)	0.251
% Decrease GFR	20.9+/-31.0	25.4+/-20.5	21.4+/-30.3	0.761
% Increase SCr	36.2+/-49.2	38.1+/-38.9	44.7+/-76.9	0.614

vs Group 1 O = $p < 0.05$; @ = $p < 0.02$; ** = $p < 0.001$;
vs Group 2 # $p < 0.05$

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SP 3 - Kiosk 9 - Poster 1

Development and Validation of a Predictive Model for Intraoperative Hypothermia in Patients Undergoing Video-Assisted Thoracoscopic Surgery: A Retrospective Study

Presenting Author: *Kevin Chen, MD*

Objective: To construct and validate a nomogram for predicting intraoperative hypothermia in patients undergoing video-assisted thoracoscopic surgery (VATS).

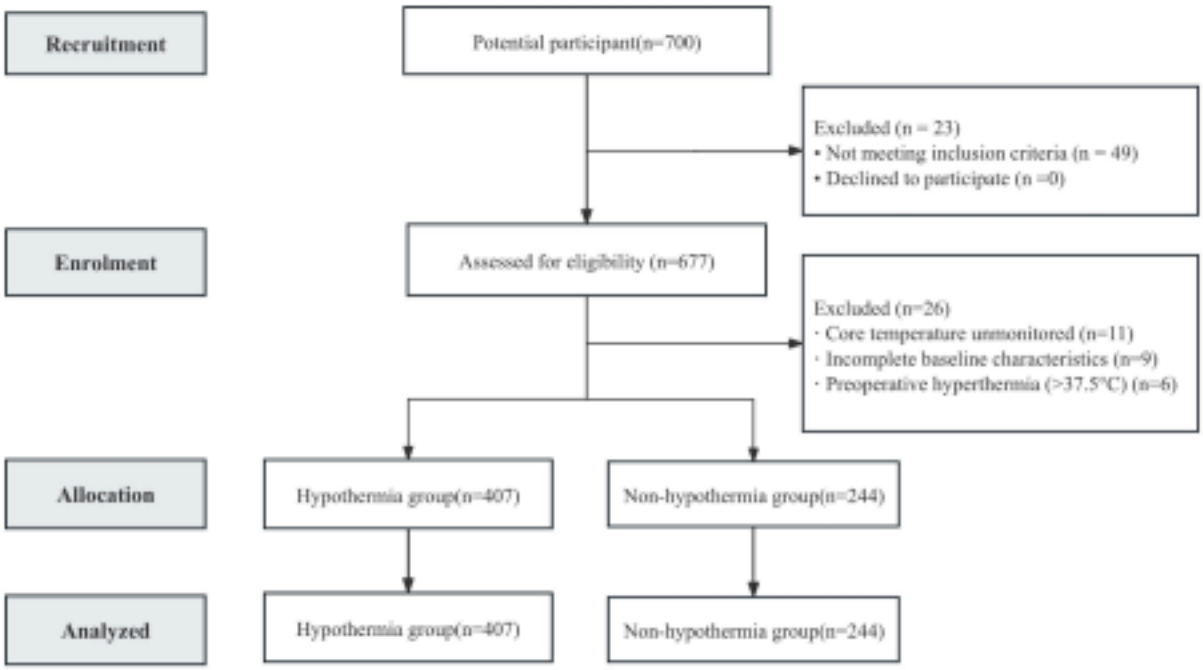
Materials and Methods: This retrospective study included 651 patients who underwent VATS at a tertiary hospital in Beijing (January 2023–August 2025). Thirty-three perioperative variables were assessed. Predictors were selected using least absolute shrinkage and selection operator (LASSO) regression, and multivariable logistic regression was performed to build the model. Restricted cubic spline (RCS) analysis was applied to explore potential nonlinear associations between continuous predictors and intraoperative hypothermia. A nomogram was developed and internally validated by 1000 bootstrap resamples. Model performance was evaluated in terms of discrimination, calibration, and clinical utility using receiver operating characteristic (ROC) analysis, calibration plots, the Hosmer–Lemeshow test, and decision curve analysis (DCA).

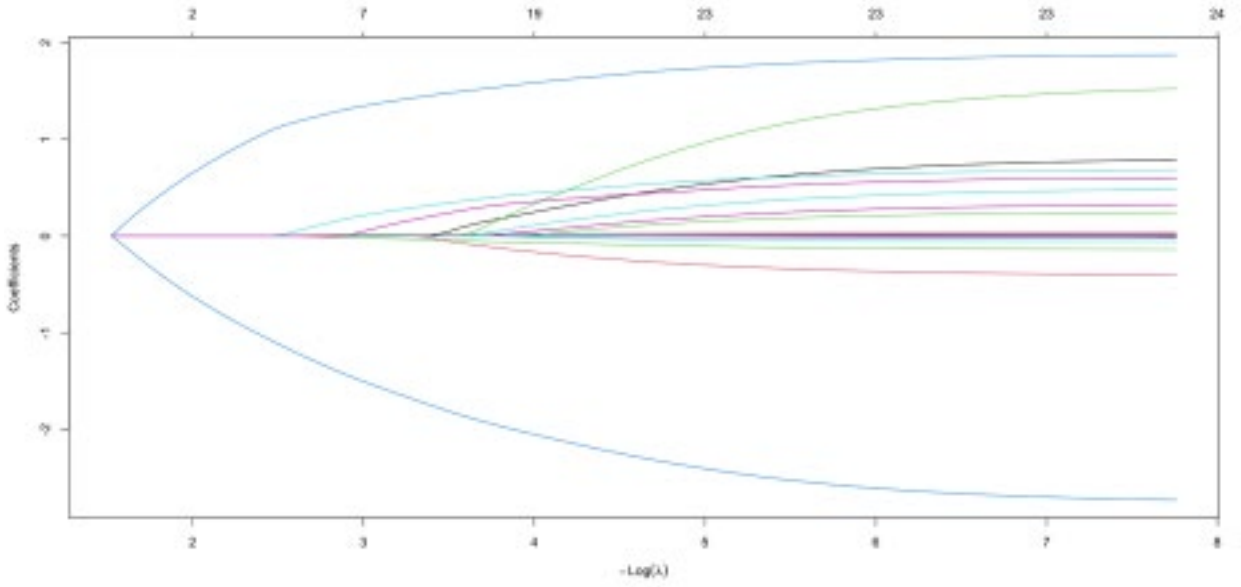
Results: Intraoperative hypothermia occurred in 407 patients (62.52%). Independent risk factors included older age, longer anesthesia duration, intraoperative hypotension, bradycardia, and elevated high-density lipoprotein (HDL), whereas higher body mass index (BMI) and forced intraoperative warming were protective. The nomogram showed good discrimination with an area under the ROC curve of 0.889 (95% CI, 0.865–0.914), sensitivity of 0.717, and specificity of 0.658. Calibration demonstrated close agreement between predicted and observed outcomes (mean absolute error = 0.008), and the Hosmer–Lemeshow test indicated adequate fit (χ^2 = 5.951, P = 0.653). DCA confirmed favorable clinical utility across a wide range of threshold probabilities.

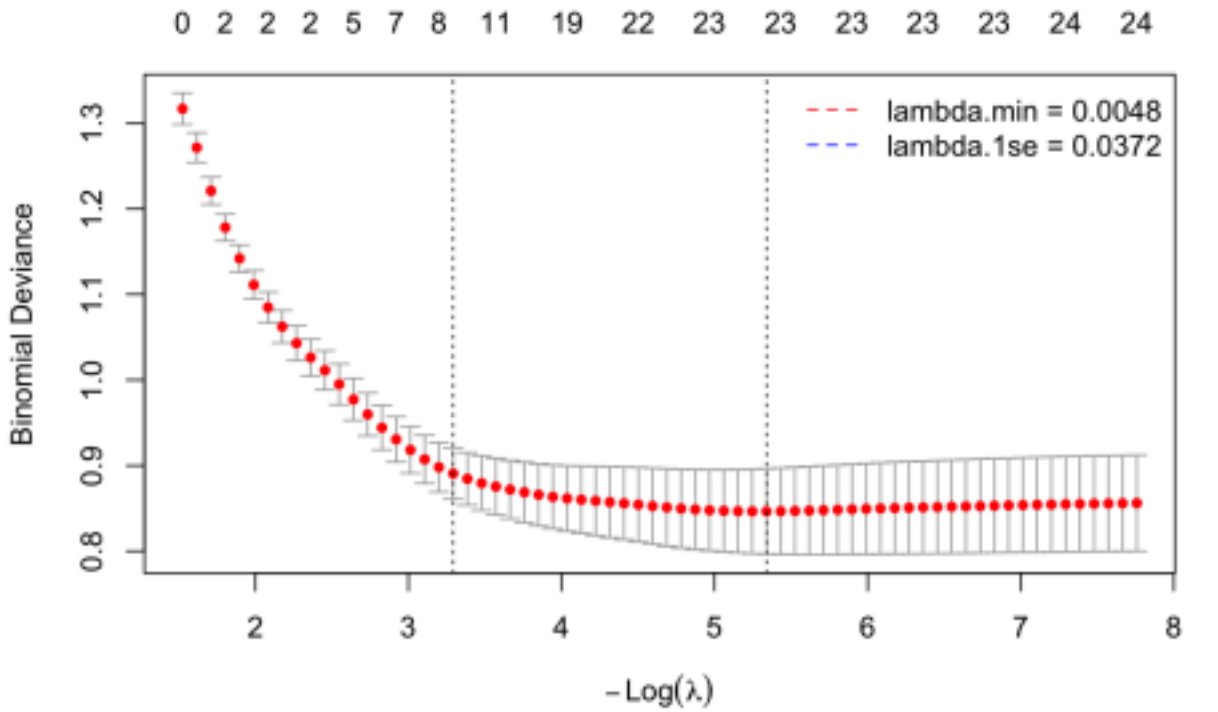
Conclusions: The proposed nomogram demonstrated strong predictive accuracy and clinical value for intraoperative hypothermia in VATS patients. Its application may facilitate early identification of high-risk individuals and support targeted perioperative warming strategies.

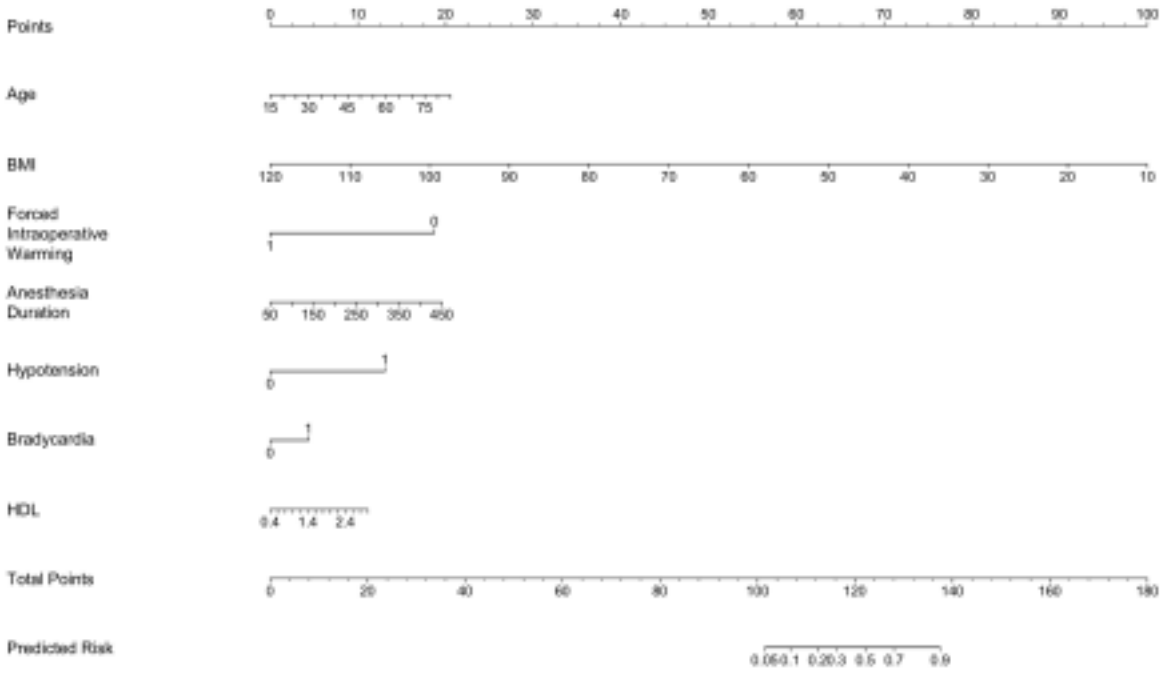
Keywords: video-assisted thoracoscopic surgery, intraoperative hypothermia, prediction model, nomogram

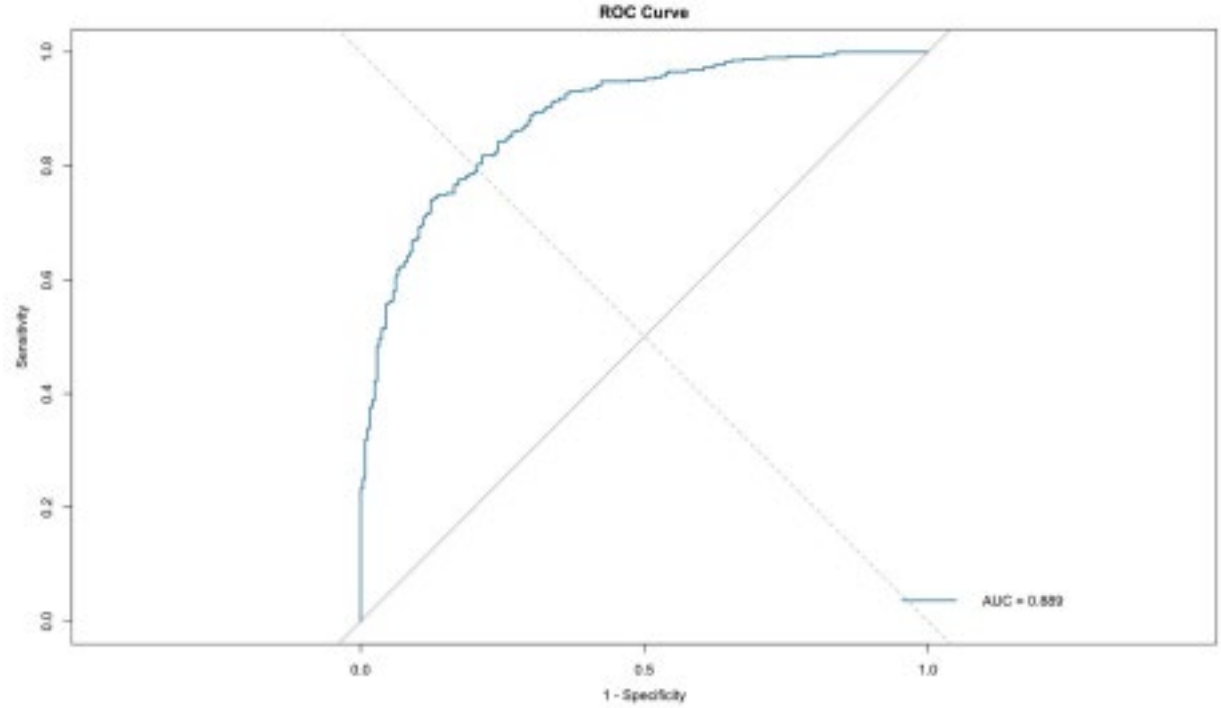
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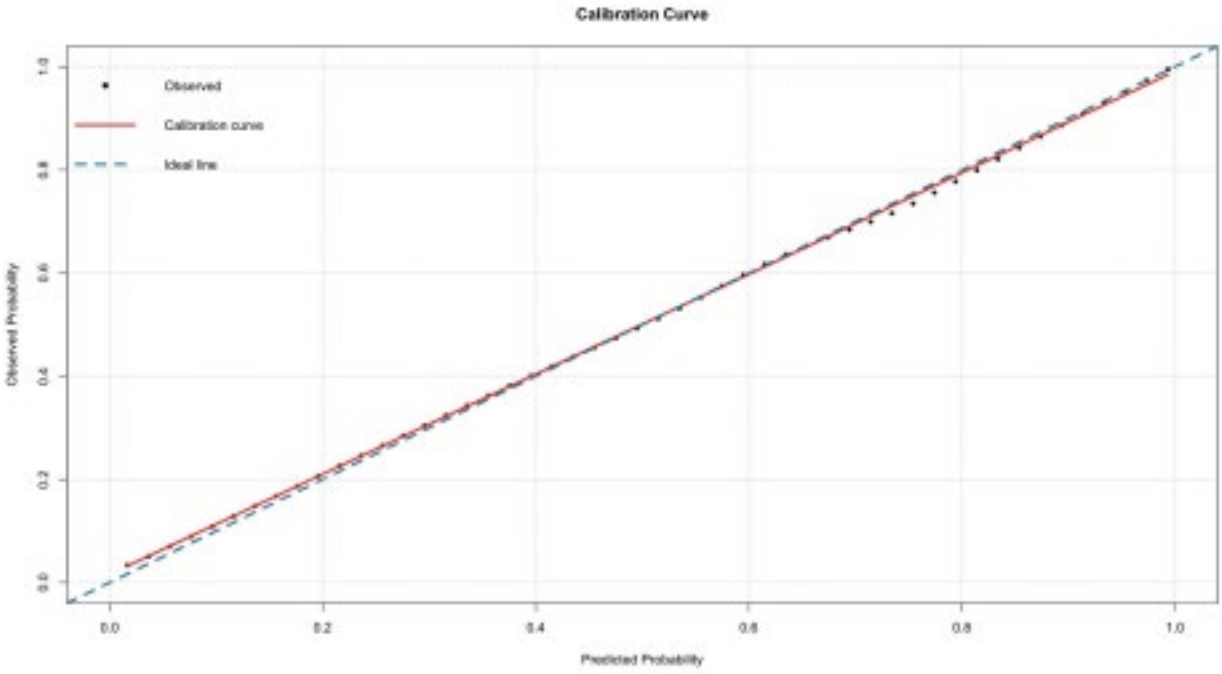





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Binomial Deviance
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lambda.1se = 0.0372
2 3 4 5 6 7 8
−Log(λ)


Points
0 10 20 30 40 50 60 70 80 90 100
Age
15 30 45 60 75
BMI
120 110 100 90 80 70 60 50 40 30 20 10
Forced Intraoperative Warming
0
1
Anesthesia Duration
50 150 250 350 450
Hypotension
1
0
Bradycardia
1
0
HDL
0.4 1.4 2.4
Total Points
0 20 40 60 80 100 120 140 160 180
Predicted Risk
0.05 0.1 0.2 0.3 0.5 0.7 0.9





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SP 3 - Kiosk 9 - Poster 2
Breath Volatile Organic Compound Analysis for Non-Invasive Heart Failure Diagnosis
Presenting Author: *Justin Huang, DO*

Background/Introduction: Heart failure affects approximately 64 million people worldwide and imposes substantial healthcare burden. Current diagnostic methods rely on costly and invasive testing, including echocardiography, cardiac catheterization, and natriuretic peptide measurement. Volatile organic compounds in exhaled breath represent endogenous metabolic byproducts that reflect disease-specific metabolic alterations. Breath analysis offers a non-invasive, rapid, and cost-effective alternative for heart failure diagnosis with potential perioperative applications for anesthesiologists managing cardiac surgical patients. This study aimed to develop and validate a breath-based diagnostic method for congestive heart failure using chemoselective capture and mass spectrometry analysis of exhaled carbonyl compounds.

Methods: Breath samples were collected from 176 patients with congestive heart failure and 32 non-heart failure controls using two collection methods: direct exhalation onto microfabricated chips and 1-liter Tedlar bags. Micropillar chips were pre-coated with 2-(aminooxy)-N,N,N-trimethylethanammonium iodide to chemoselectively capture carbonyl compounds via oximation chemistry. Bagged samples were drawn across chips under applied vacuum. Captured carbonyl-aminooxy adducts were eluted with ethanol and analyzed by ultra-high performance liquid chromatography-mass spectrometry to profile aldehydes, ketones, and hydrocarbons. Group differences were assessed using analysis of variance with statistical significance defined as p less than or equal to 0.05. Receiver operating characteristic curves evaluated diagnostic performance.

Results: Acetone demonstrated significant elevation in heart failure patients compared with controls using both collection methods. Direct breath collection showed acetone elevation (p equals 0.032), while Tedlar bag collection confirmed this finding (p equals 0.0137). Receiver

operating characteristic analysis demonstrated diagnostic accuracy comparable to established biomarkers. Additional volatile organic compounds including aldehydes and hydrocarbons showed differential expression patterns between groups, suggesting potential multi-marker diagnostic panels.
Conclusion: Breath volatile organic compound analysis represents a promising non-invasive screening method for heart failure diagnosis, with acetone identified as a statistically significant biomarker differentiating heart failure from non-heart failure patients. This approach offers rapid, cost-effective point-of-care testing with particular relevance for perioperative risk stratification in patients undergoing cardiac and noncardiac surgery. Validation in larger multicenter cohorts is warranted to establish clinical utility and define optimal diagnostic thresholds for perioperative applications.

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SP 3 - Kiosk 9 - Poster 3

Comparison Between Single-Shot and Bilateral Catheter Erector Spinae Plane Block for Postoperative Pain Control in Cardiac Surgery: A Retrospective Cohort Study

Presenting Author: *Eric Lineburger, MD, MSc,PhD, FASE*

Background Postoperative pain after median sternotomy contributes to respiratory impairment, delayed mobilization, and increased complications. The erector spinae plane (ESP) block has emerged as an opioid-sparing strategy in cardiac anesthesia. This study compared a single bilateral injection technique with intermittent boluses through bilateral catheters.

Methods: This retrospective cohort included adults undergoing median sternotomy between February 2023 and September 2024. IRB approval for retrospective data analysis was obtained on

February 21, 2025 (approval number 7.401.708). In the single-injection group, patients received bilateral ESP injections of 20 mL of 0.375 percent ropivacaine at the beginning of surgery. In the ESP catheter group, the same initial dose was followed by 20 mL per side every eight hours for forty-eight hours. All patients received standardized multimodal analgesia and patient-controlled intravenous analgesia with morphine and ketamine. Primary outcomes were pain on coughing and pain at rest at twenty-four hours and forty-eight hours, measured using a numeric verbal pain score from zero to ten. Secondary outcomes were sedation level, patient satisfaction, use of additional rescue analgesia, and the need for postoperative non-invasive ventilation. Statistical analyses used generalized estimating equations.

Results: The results are summarized in Table 1. A total of two hundred and thirty-five patients were included. Pain on coughing was significantly lower in the catheter group at twenty-four hours (2.16 ± 0.14 versus 1.13 ± 0.13; mean difference −1.03; 95%CI: -1.37 to -0.70; P < 0.001) and forty-eight hours (1.81 ± 0.13 versus 0.99 ± 0.10; mean difference −0.82; 95%CI: -1.14 to -0.51; P < 0.001). Pain at rest was also reduced at twenty-four hours (0.81 ± 0.10 versus 0.24 ± 0.06; mean difference −0.57; 95%CI: -0.80 to -0.33; P < 0.001) and forty-eight hours (0.40 ± 0.08 versus 0.11 ± 0.04; mean difference −0.29; 95%CI: -0.92 to -0.49; P=0.002). Sedation levels were similar at twenty-four hours (mean difference 0.11; 95%CI: -0.33 to 0.11; P=0.314) but were lower in the catheter group at forty-eight hours (mean difference -0.24; 95%CI: -0.46 to -0.02; P=0.030). Satisfaction with pain control and need for rescue analgesia did not differ significantly. The catheter group had a substantially lower requirement for non-invasive
ventilation in the first twenty-four hours after surgery (3.1 percent versus 15.1 percent; P = 0.022).

Conclusion: ESP intermittent boluses through bilateral catheters provided superior analgesia compared with a single ESP bilateral injection technique, resulting in lower pain on coughing and pain at rest, reduced sedation at forty-eight hours, and a lower need for early postoperative non-invasive ventilation. Satisfaction and rescue analgesia were similar between the two strategies. Prospective trials are required to confirm these findings and assess broader clinical implications.

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Table 1. Comparison Between Single-Shot and Bilateral Catheter Erector Spinae Plane Block for Postoperative Pain Control in Cardiac Surgery

Variable	Bolus (n)	Bolus (Mean±SD)	Catheter (n)	Catheter (Mean±SD)	p-value
Patients	150		85		
Measurements	225		137		
Age (years)		62.9 ± 11.3		62.9 ± 9.6	0.938
Pain on coughing 24h	130	2.16 ± 0.14	74	1.13 ± 0.10	<0.001
Pain on coughing 48h	92	1.81 ± 0.13	62	0.99 ± 0.10	<0.001
Analgesia perception 24h (%)	133	95.0 ± 1.8	74	95.0 ± 2.6	0.778
Analgesia perception 48h (%)	92	98.0 ± 1.5	63	98.0 ± 1.6	0.788
Pain at rest 24h (NRS)	133	0.81 ± 0.10	74	0.24 ± 0.06	<0.001
Pain at rest 48h (NRS)	92	0.40 ± 0.08	62	0.11 ± 0.04	0.002
Sedation score 24h (0–5)	133	0.57 ± 0.06	74	0.46 ± 0.09	0.314
Sedation score 48h (0–5)	92	0.59 ± 0.06	63	0.35 ± 0.09	0.030
Opioid rescue consumption 24h (VTBI)	50	84.2 ± 2.0	6	85.8 ± 3.0	0.685
Opioid rescue consumption 48h (VTBI)	32	72.3 ± 3.5	8	67.0 ± 7.4	0.526
Use of NIV 24h (%)	72	15.1 ± 4.2	32	3.1 ± 3.1	0.022
Use of NIV 48h (%)	49	22.5 ± 5.7	30	16.0 ± 6.9	0.463

Legend: NRS = Numerical Rating Scale; VTBI = opioid rescue consumption (volume to be infused from PCA pump); NIV = noninvasive ventilation.

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Postoperative factors: The need for extracorporeal life support (OR 3.04, CI[1.66-5.50], p< 0.001) was associated with increased risk of AKI.
Conclusion: In conclusion, preoperative pulmonary hypertension, preoperative need for life support, and intraoperative packed red cell transfusion are independent risk factors for postoperative AKI following lung transplantation, whereas higher intraoperative fluid administration appears to be protective.

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SCIENTIFIC PROGRAM ABSTRACTS

SUPER ECHO

SUPERECHO
Saturday, April 25, 2026 | 3:00 PM - 5:00 PM

SCA-SuperEcho-01
The Masks We Wear: Eccentric Severe Aortic Insufficiency Concealing a Flailed Posterior Mitral Valve Leaflet Identified by Integrating M-Mode Assessment
Presenting Author*: Aaron Hacker, DO*

Introduction: This case illustrates a rare diagnostic challenge in perioperative echocardiography. A severe, eccentric, posteriorly directed aortic insufficiency (AI) jet produced marked diastolic displacement of the anterior mitral valve leaflet (AMVL), creating a "pseudo-prolapse" that obscured a significant flail P1 scallop and yielded only mild mitral regurgitation (MR). The resulting anterior/atrial positioning of the AMVL throughout the cardiac cycle distorted coaptation geometry, complicated MR quantification, and required careful integration of M-mode and 3D imaging to guide surgical planning.

Brief Summary of the Clinical Case: A 71-year-old man with severe AI presented for elective aortic valve replacement (AVR). Prior transthoracic and transesophageal echocardiographic examinations demonstrated isolated, severe AI with mild mitral regurgitation. Following induction of anesthesia, the intraoperative TEE revealed a previously undiagnosed flail P1 scallop of the posterior mitral valve leaflet (PMVL) but only mild eccentric MR. Since the severity of the MR was mild, describing the mechanism of MR underestimation and potential increased MR severity with a competent AV, following surgical intervention, was critical for surgical decision making.
Quantitative Findings

- MR EROA (PISA): 0.21–0.24 cm^2
- MR regurgitant volume: ≈35 mL
- 3D CFD regurgitant orifice area: ≈0.22 cm^2
- M-mode: inverted E-point, reduced amplitude, diminished E-F slope, diastolic doming, and atrial systolic AMVL positioning.

Color Doppler demonstrated the severe posterior AI jet striking the AMVL, driving it anteriorly/atrially in diastole and maintaining abnormal positioning into systole. This jet-mediated coaptation partially compensated for the flail P1 scallop and masked the true MR severity. Recognizing that AVR alone would normalize AMVL motion and unmask more severe MR, the team proceeded with combined AVR and mitral valve repair.

Discussion: Comprehensive evaluation included 2D and 3D TEE, multiplane leaflet analysis, and integrated quantitative assessment (EROA, regurgitant volume, 3D VCA). M-mode through the mid-AMVL precisely characterized abnormal A-D motion and the markedly diminished E-F slope. The severe eccentric AI jet repeatedly displaced the AMVL anteriorly/atrially, producing paradoxical diastolic motion and maintaining the leaflet in an atrial position into systole. M-mode showed an abnormally reduced E-F excursion (~0.82 cm) and severely blunted slope velocity, confirming restricted ventricular excursion caused by continuous jet impact.
This "pseudo-prolapse" facilitated partial coaptation with the flail PMVL, reducing MR appearance

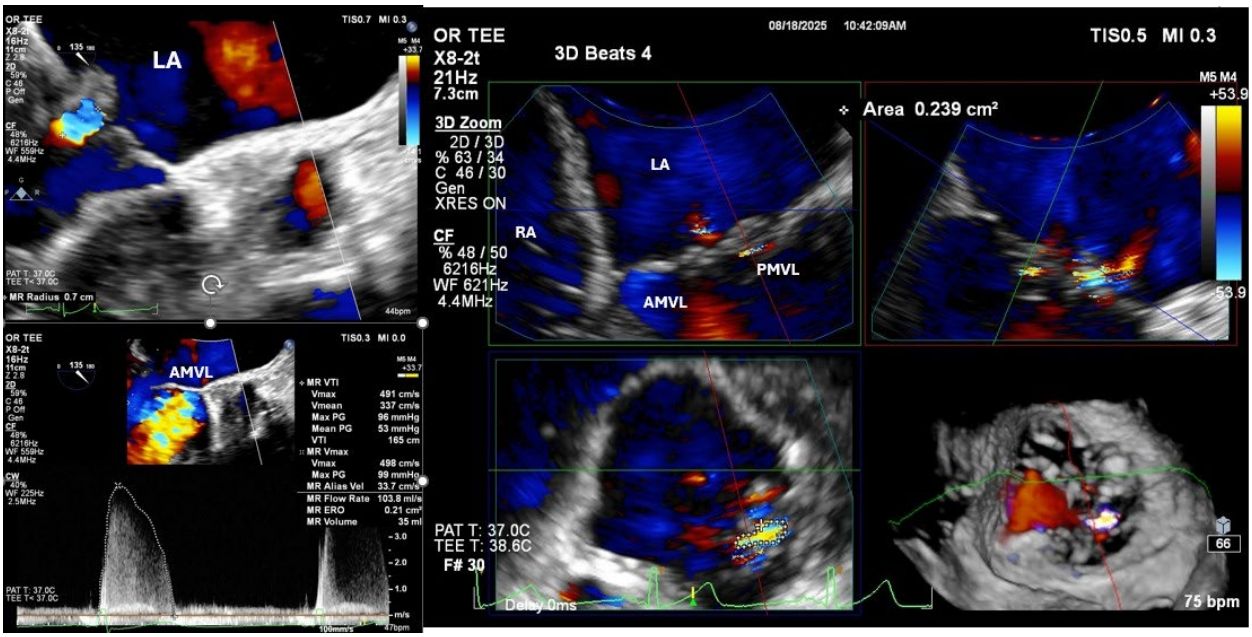
despite a significant anatomic lesion.

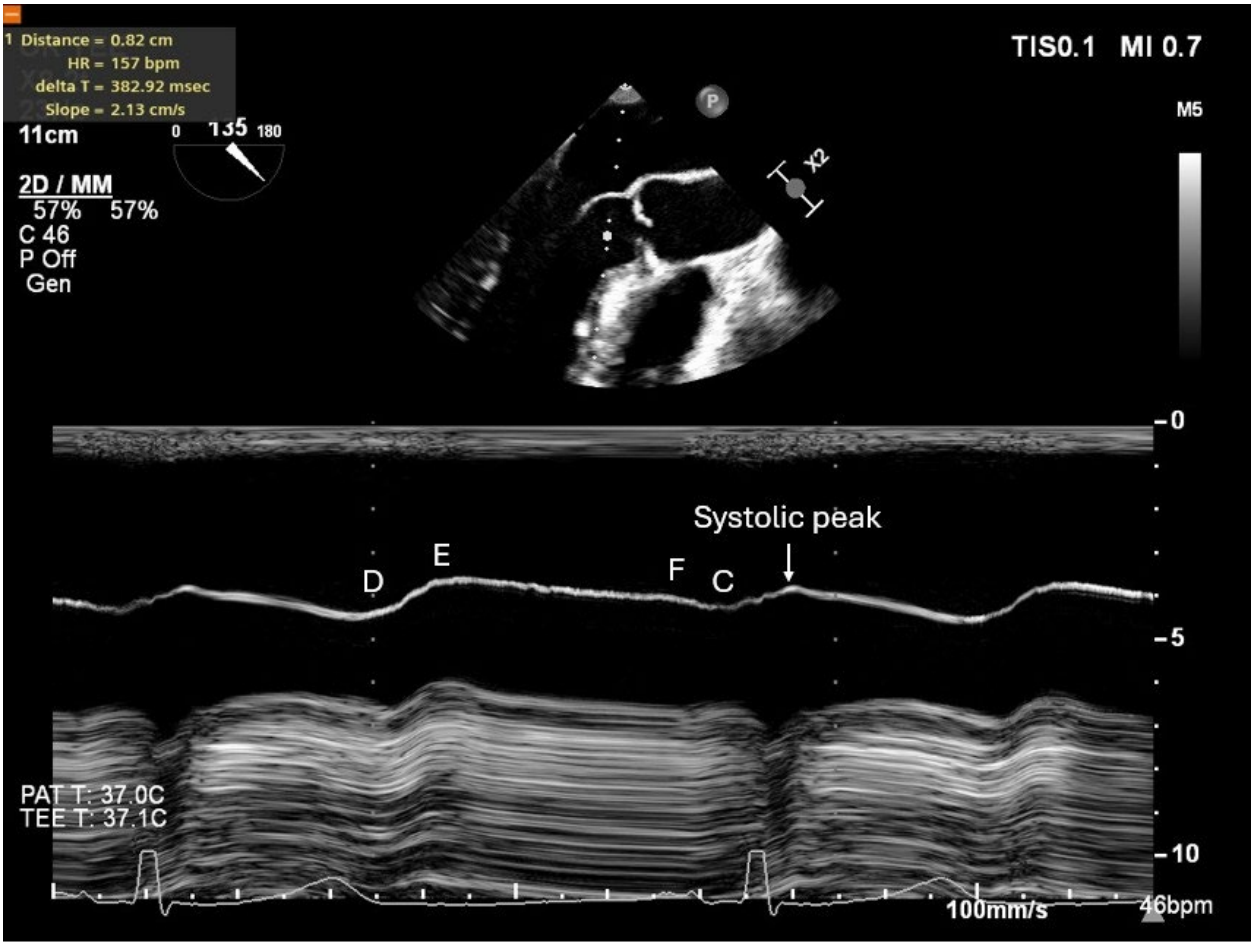
Impact of Echocardiographic findings: Recognizing that the AI-driven AMVL malposition concealed true MR severity was critical. AVR would eliminate the jet, restore normal leaflet kinematics, and likely unmask significant MR requiring repair. The combined intervention was therefore appropriate and preemptive.

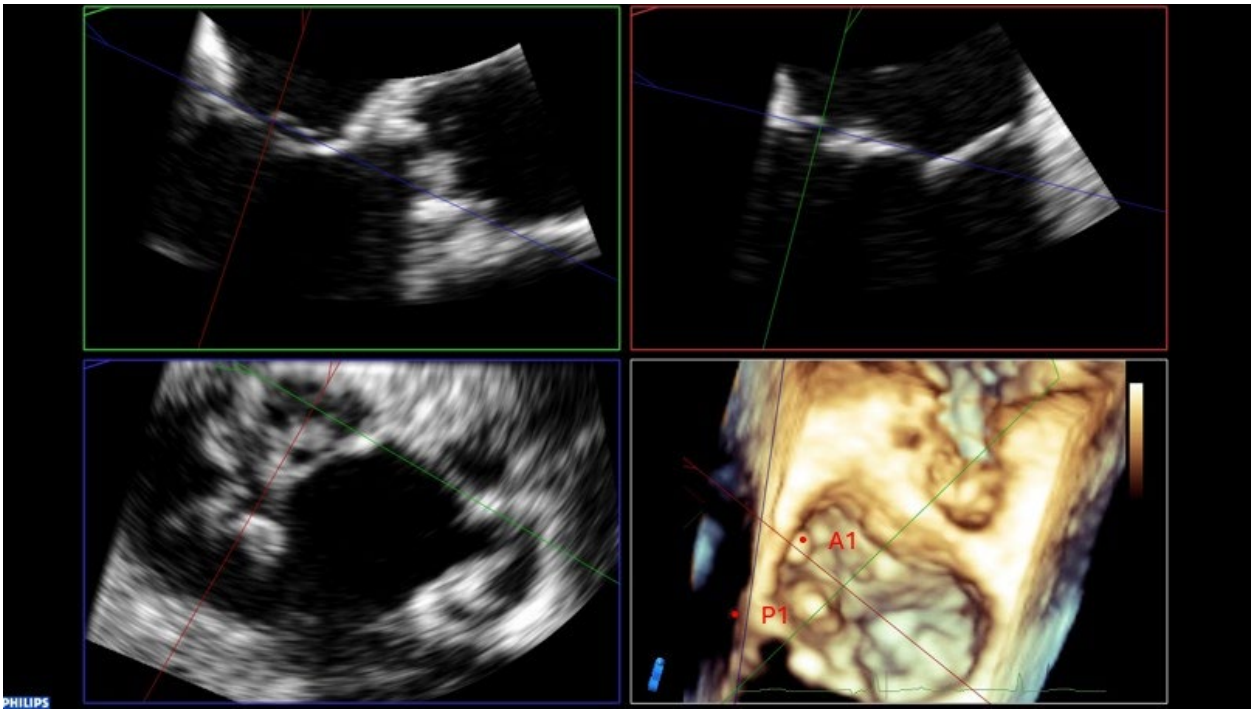
Teaching Points of the Case

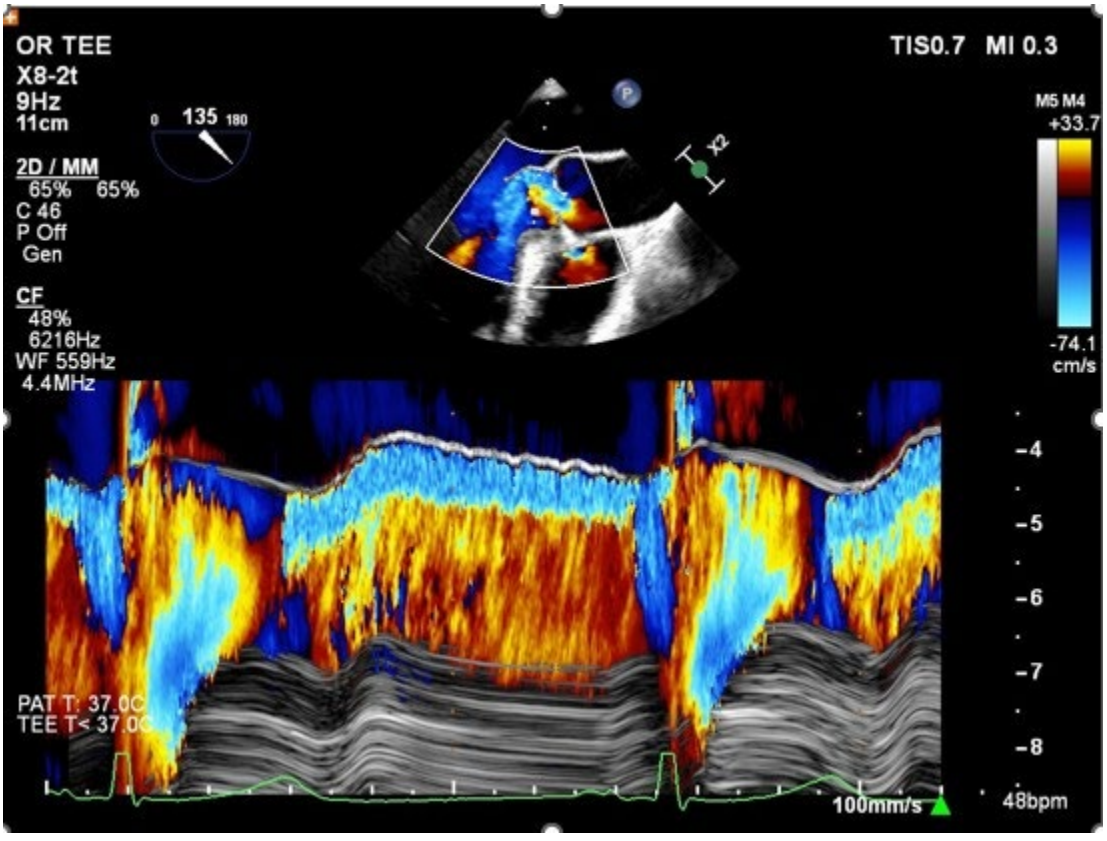
- Diastolic AI jets can generate systolic “pseudo-prolapse,” masking MR severity.
- M-mode remains essential for detecting abnormal AMVL motion patterns.
- 3D TEE is indispensable for distinguishing jet-maintained coaptation from true anatomic competence in mixed valvular disease.

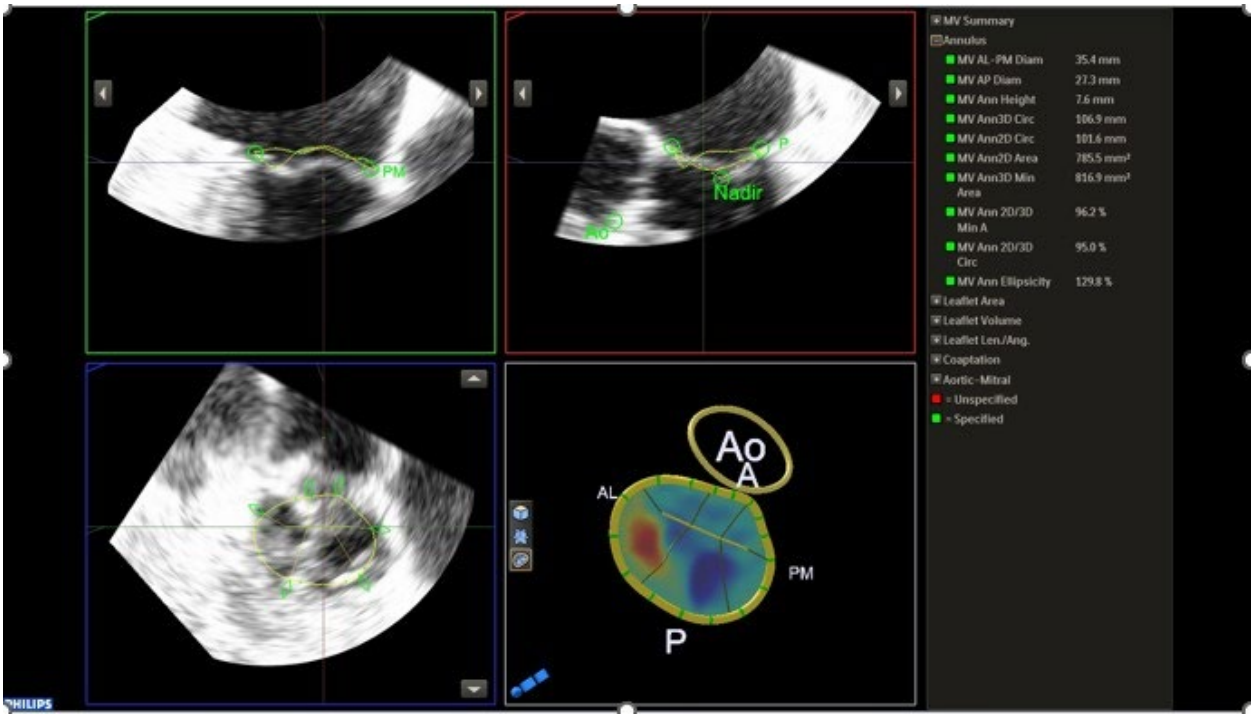
Image uploads




1 Distance = 0.82 cm
HR = 157 bpm
delta T = 382.92 msec
Slope = 2.13 cm/s
11cm
0
135
180
2D / MM
57%
57%
C 46
P Off
Gen
TIS0.1
MI 0.7
M5
P
X2
Systolic peak
D
E
F
C
0
5
10
PAT T: 37.0C
TEE T: 37.1C
100mm/s
46bpm


A1
P1
PHILIPS


OR TEE
X8-2t
9Hz
11cm
2D / MM
65% 65%
C 46
P Off
Gen
CF
48%
6216Hz
WF 559Hz
4.4MHz
TIS0.7 MI 0.3
M5 M4
+33.7
-74.1
cm/s
-4
-5
-6
-7
-8
48bpm
100mm/s
PAT T: 37.0C
TEE T< 37.0C



Video Uploads

Video 1: Legend: Mid esophageal five chamber view: dilated LV, with moderate tenting of the mitral valve, restricted diastolic opening of the mitral valve and atrial positioning. Posterior MV leaflet fail With color flow Doppler (CFD), there is moderate to severe, posteriorly directed, aortic insufficiency jetLegend: Mid esophageal aortic valve long axis view: dilated LV, with moderate tenting of the mitral valve, restricted diastolic opening of the mitral valve. Posterior MV leaflet failWith color flow Doppler (CFD), there is moderate to severe, posteriorly directed, aortic insufficiency jet and trivial to mild anteriorly directed MR ME views with orthogonal view—Mild MR jet

Video 2: 3d view of the mitral valve: Fail of the P1 leaflet. Trueview 3D view of the mitral valve with CFD showing mild, anteriorly directed mitral regurgitation

References

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Hirasawa K et al. J Am Coll Cardiol. 2020; 75:2252–2264.

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SCA-SuperEcho-02
Complex Mitral Valve Decision Making: A Therapeutic Option for Treating Dynamic Left Ventricular Outflow Tract Obstruction After Mitral Valve Repair
Presenting Author: *Survandita Dhawan, MD*

Introduction: Intraoperative echocardiography is a critical component of surgical decision-making during surgical mitral valve (MV) repair. A key element of the echocardiographic examination is evaluating the risk of systolic anterior motion (SAM) post-MV repair. [1,2]. Technological advances in three-dimensional echocardiography (3D-TEE) and the ability to obtain high-resolution multiplanar reconstruction (MPR) further aid in precise measurement of SAM predictors intraoperatively, as well as determine the degree of obstruction of the left ventricular outflow tract (LVOT). Furthermore, two-dimensional echocardiography (2D-TEE) and 3D-TEE, including live MPR, now allow for transcatheter mitral valve therapies [3]. These structural interventions can treat a myriad of conditions, including SAM post-MV repair under physiologically stressful conditions. Here we present a case of a young, and otherwise, healthy patient who underwent MV repair for severe MR and was found to have dynamic LVOT obstruction with exercise after repair, which was addressed with MV edge-to-edge repair (mTEER).

Brief Summary of the Clinical Case: A 38-year-old male with history of myxomatous valve disease initially presented with shortness of breath. He was found to have severe MR and subsequently presented for minimally invasive MV repair. Intraoperative echocardiography showed that , he was high risk for SAM post repair; post-repair, the patient did demonstrate SAM, but the majority of the LVOT was found to be unobstructed, despite SAM being present. The repair was deemed successful, and the patient was subsequently after an uneventful postoperative course. Two years later, he re-presented with worsening, intermittent dyspnea. He underwent a stress echocardiogram which revealed dynamic LV outflow tract obstruction and minimal mitral regurgitation; an extensive workup was unrevealing alternate causes for his dyspnea.

At this point, he was referred to structural cardiology, and he was planned for a detailed TEE examination in the cath lab with plans to proceed with mTEER if deemed feasible. 2D- and 3D-TEE with dobutamine stress demonstrated dynamic LVOT obstruction predominantly due to excessive motion of the anterior MV leaflet with residual mitral regurgitation, as noted postoperatively two years ago. Given these findings, he underwent mTEER with a MitraClip XTW to the A2-P2 segment with immediate resolution of the dynamic LVOTO, which could no longer be reproduced despite high doses (>20mcg/kg/min) of dobutamine infusion,

He was subsequently discharged and remains symptoms-free post MV TEER.

Discussion:

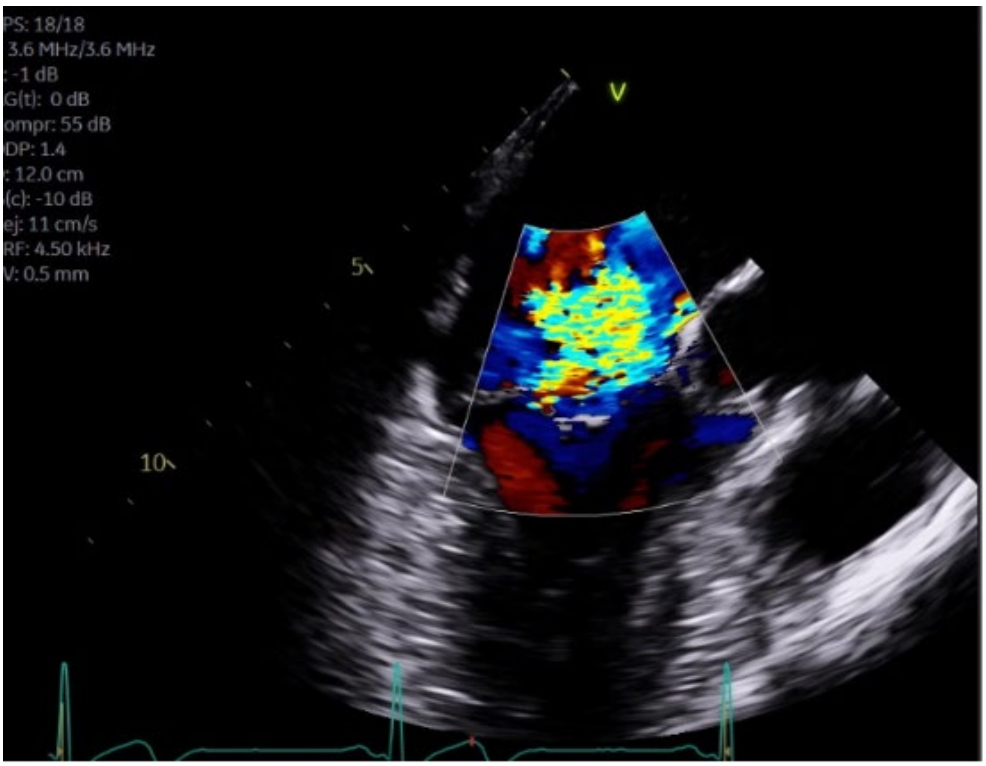
- Three-dimensional imaging with multiplanar reconstruction (3D MPR) provides an accurate measurement of LVOT obstruction in setting of SAM post MV repair.
- TEE is vital for guidance of structural options, such as mTEER.

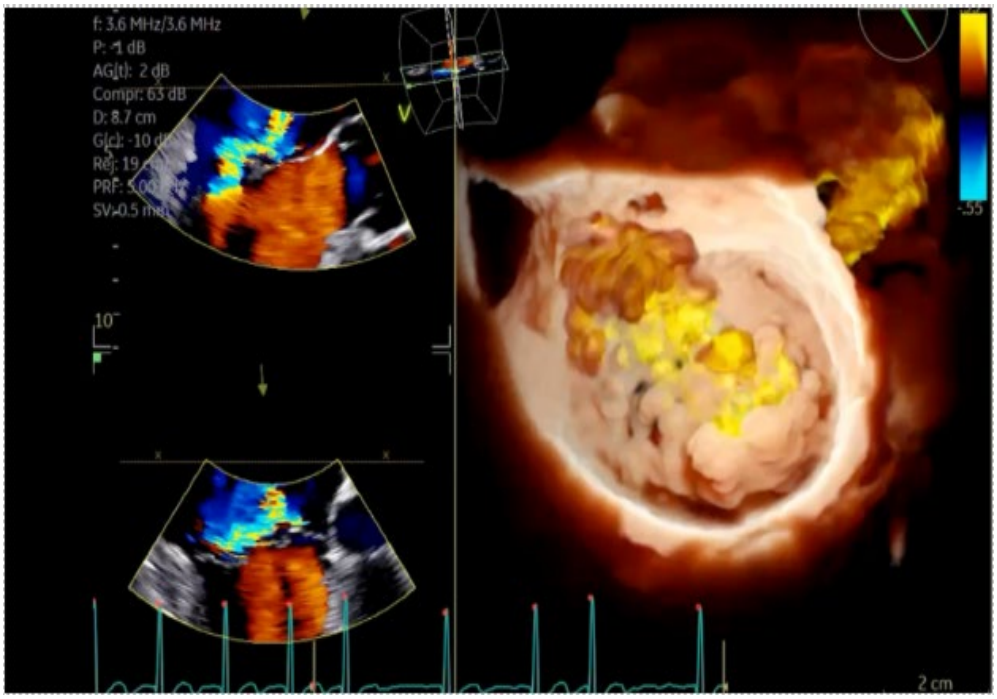
Impact of Echocardiographic findings: MV TEER can be successfully performed in patients with dynamic LVOT obstruction resulting from stress conditions using live three-dimensional echocardiographic guidance.

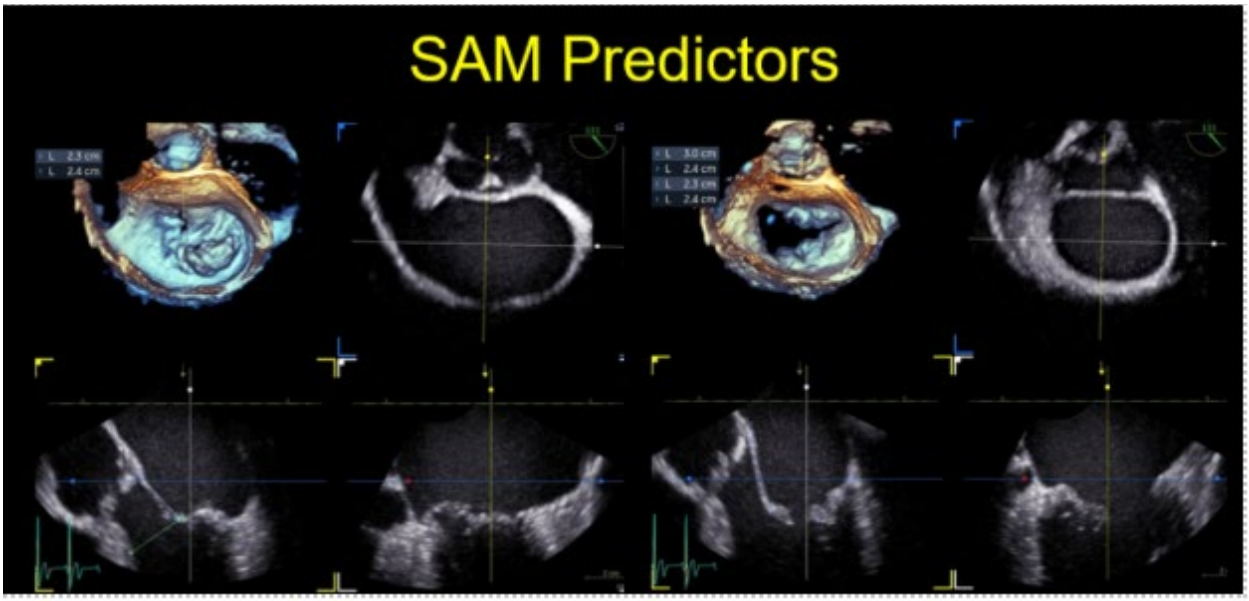
Teaching Points of the Case

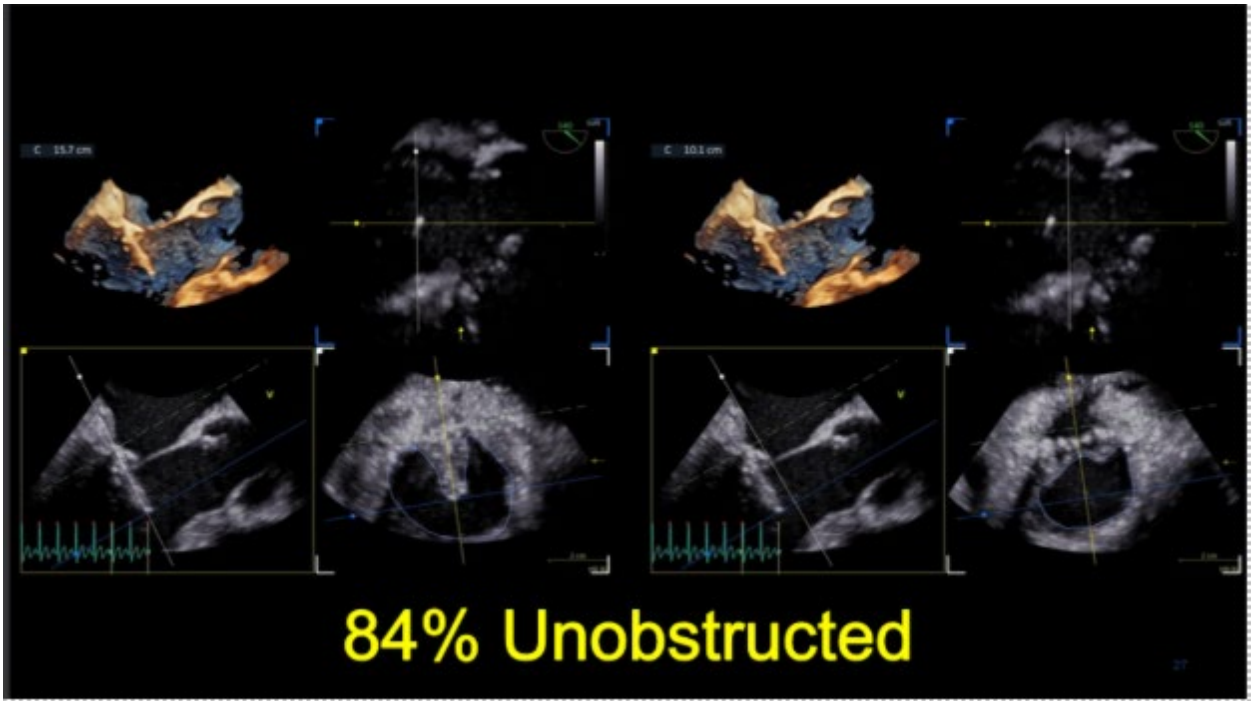
- LVOT obstruction can be deduced from live 3D-TEE and MPR when SAM is present post-repair intraoperatively.
- Structural options, such as MV TEER, is an alternative to classic strategy of MV replacement after failure of MV repair.

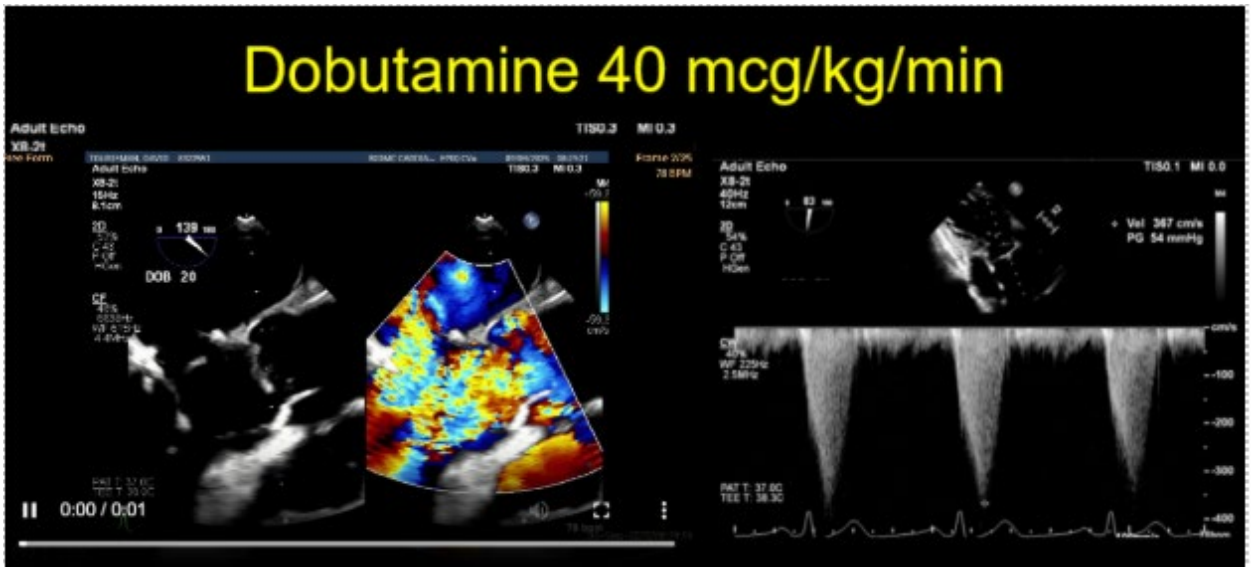
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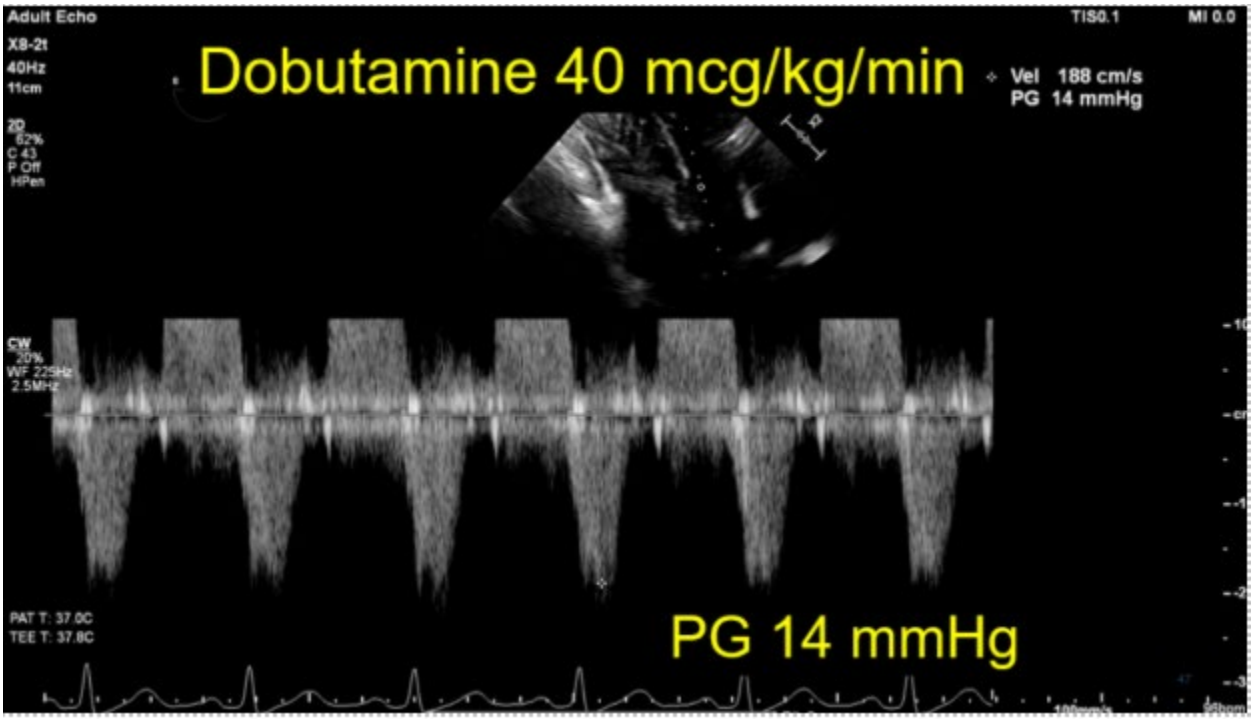



f: 3.6 MHz/3.6 MHz
2 cm


SAM Predictors


84% Unobstructed


Dobutamine 40 mcg/kg/min
Adult Echo
DOB 20
0:00 / 0:01
Adult Echo
Vel 367 cm/s
PG 54 mmHg



Video Uploads:

Video 1: This video encompasses the patient's initial MV repair, including the use of 3D-TEE and MPR to deduce SAM predictors pre-repair of mitral valve. We also used 3D-TEE and MPR to calculate the level of obstruction of the LVOT post-repair in setting of SAM present. Two years later, both 2D-TEE and 3D-TEE was used to determine the exact location causing SAM and leading to dynamic LVOT obstruction under stress physiology. 2D-TEE and 3D-TEE were also utilized to guide catheters and the M-TEER apparatus to the appropriate location (A2-P2 scallop) and deploy the mitraclip. Both 2D-TEE and 3D-TEE were also vital in visualizing lack of SAM after successful mTEER, and therefore lack of dynamic LVOT obstruction under stress physiology.

Video 2: This video encompasses the patient's initial MV repair, including the use of 3D-TEE and MPR to deduce SAM predictors pre-repair of mitral valve. We also used 3D-TEE and MPR to calculate the level of obstruction of the LVOT post-repair in setting of SAM present. Two years later, both 2D-TEE and 3D-TEE was used to determine the exact location causing SAM and leading to dynamic LVOT obstruction under stress physiology. 2D-TEE and 3D-TEE were also utilized to guide catheters and the M-TEER apparatus to the appropriate location (A2-P2 scallop) and deploy the mitraclip. Both 2D-TEE and 3D-TEE were also vital in visualizing lack of SAM after successful mTEER, and therefore lack of dynamic LVOT obstruction under stress physiology.

References

Reference 1

1. Nicoara, A., Skubas, N., Ad, N., Finley, A., Hahn, R. T., Mahmood, F., Mankad, S., Nyman, C. B., Pagani, F., Porter, T. R., Rehfeldt, K., Stone, M., Taylor, B., Vegas, A., Zimmerman, K. G., Zoghbi, W.

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Reference 2
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SCA-SuperEcho-03
Severe Mitral Regurgitation and a Phantom VSD in a patient with ARDS: A Tale of Two Valves
Presenting Author: *Laura Mendez Pino, MD*

Introduction: We present a case of torrential MR caused by a flail posterior mitral leaflet with an associated AV cusp perforation that mimicked a VSD. This was identified in a young adult on VV ECMO support for respiratory failure due to ARDS. This case also offers several educational points: the ECMO configuration had to be changed to LAVA to determine whether the severe MR was contributing to the respiratory failure. His respiratory status dramatically improved with the ECMO reconfiguration, and his clinical picture completely resolved after the MV and AV repair.

Brief Summary of the Clinical Case: A 24-year-old male with a history of ulcerative colitis, vaping, and known MR presented with shortness of breath and rapidly developed ARDS. He was intubated and placed on VV ECMO. Due to worsening hemodynamics, Impella was attempted, but placement failed. He was placed on VV ECMO with a dual-lumen cannula via RIJ approach. He was transferred for lung transplant evaluation.
A TTE ordered after the transfer showed severe MR, while a TEE confirmed a flail P3 segment. Color Doppler demonstrated subaortic flow with right-to-left shunting, concerning for a VSD. The reversed shunt was suspected to be ECMO-related.
To assess whether MR was the main cause of pulmonary edema, VV ECMO was reconfigured to LAVA ECMO. This resulted in rapid improvement in the pulmonary congestion, confirming MR as the cause of the ARDS. Surgery revealed a perforation in the right coronary cusp instead of a VSD, related to the previous Impella attempt. The patient underwent MV repair, AV cusp repair, ASD closure, and ECMO decannulation. He was extubated and discharged home

Discussion

- Multimodal echocardiography was essential in identifying torrential MR caused by flail P3 and excluding papillary muscle rupture or infective endocarditis as underlying causes
- TEE views critical for VSD assessment were systematically used in this case. These include

1. ME SAX at the ventricular level – useful for muscular defects
2. ME LAX view – helpful for mid-anterior muscular or perimembranous VSDs
3. Transgastric inflow and outflow views – best for defect sizing and shunt flow assessment via color Doppler

- Improvement in pulmonary edema after LV unloading with the LAVA ECMO confirmed that torrential MR was the cause
- The perforation of the aortic cusp was probably iatrogenic, associated with the unsuccessful Impella placement attempt
- This case highlights the importance of 3D TEE, color Doppler analysis, and flow timing relative to the cardiac cycle

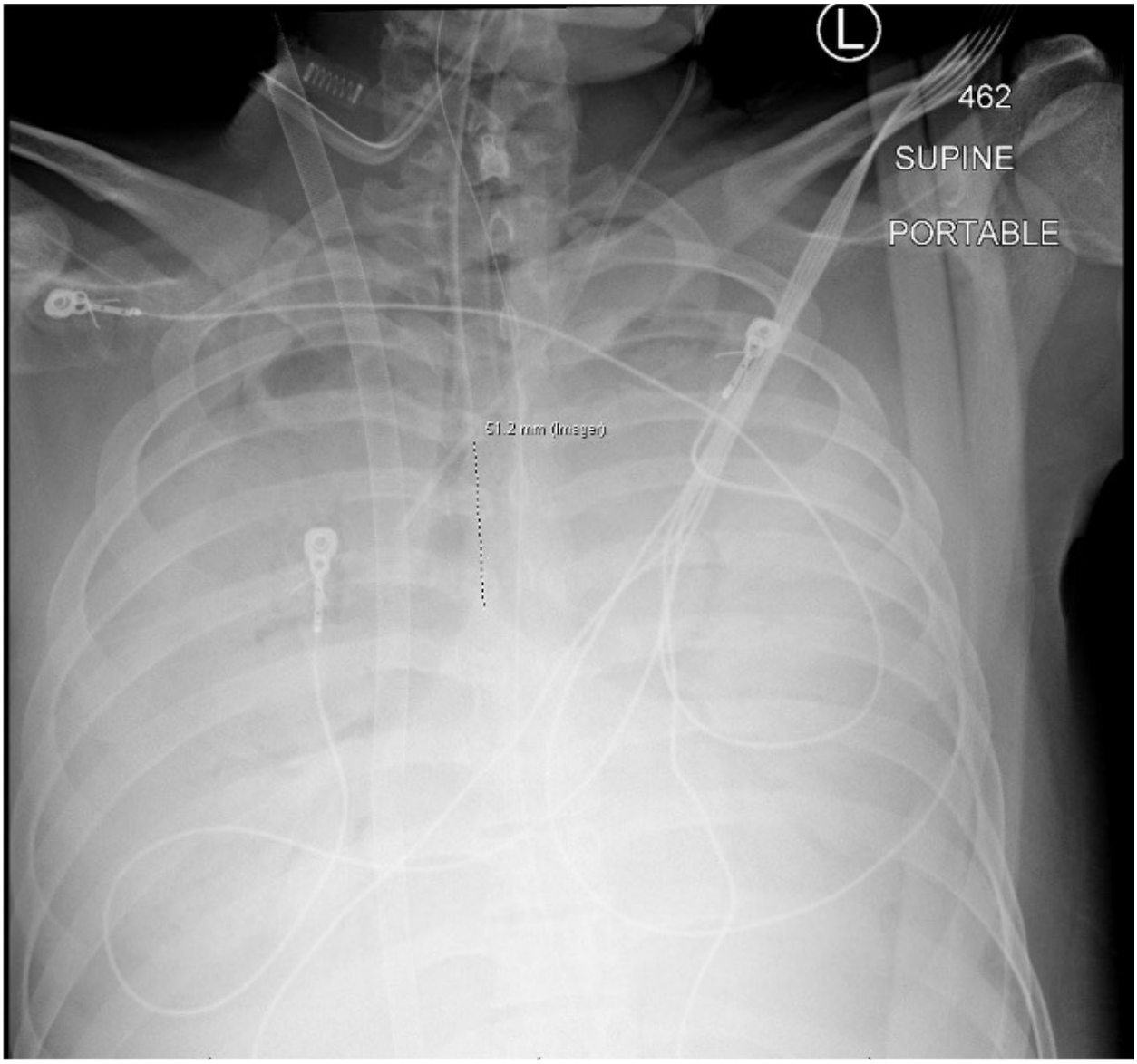
Impact of Echocardiographic findings

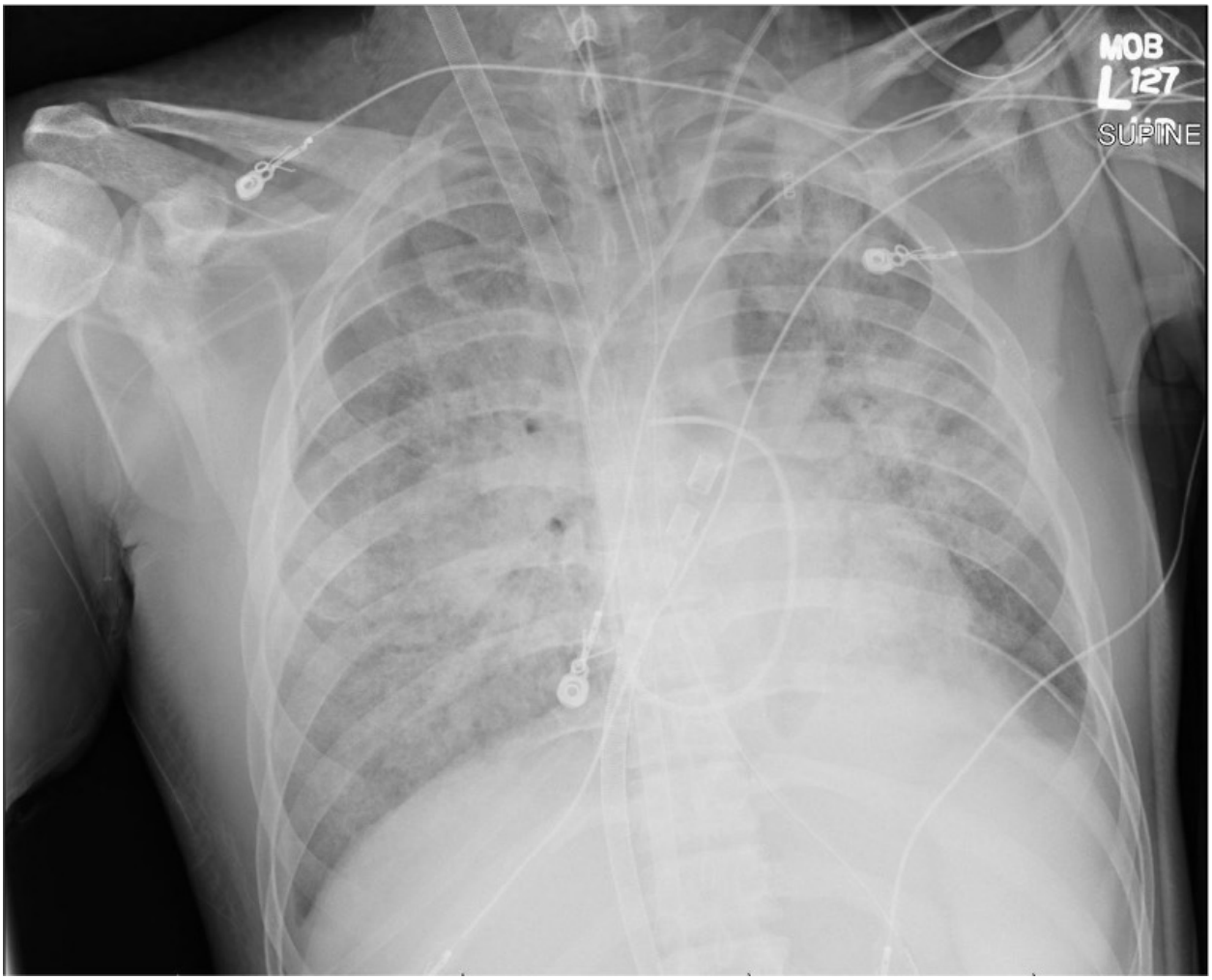
- The finding of severe MR in the setting of an apparent ARDS completely changed the management
- The subaortic color doppler showed flow during diastole, not systole, which is inconsistent with a true VSD and suggests aortic valve perforation as the diagnosis. This highlights the importance of integrating flow timing with anatomical localization

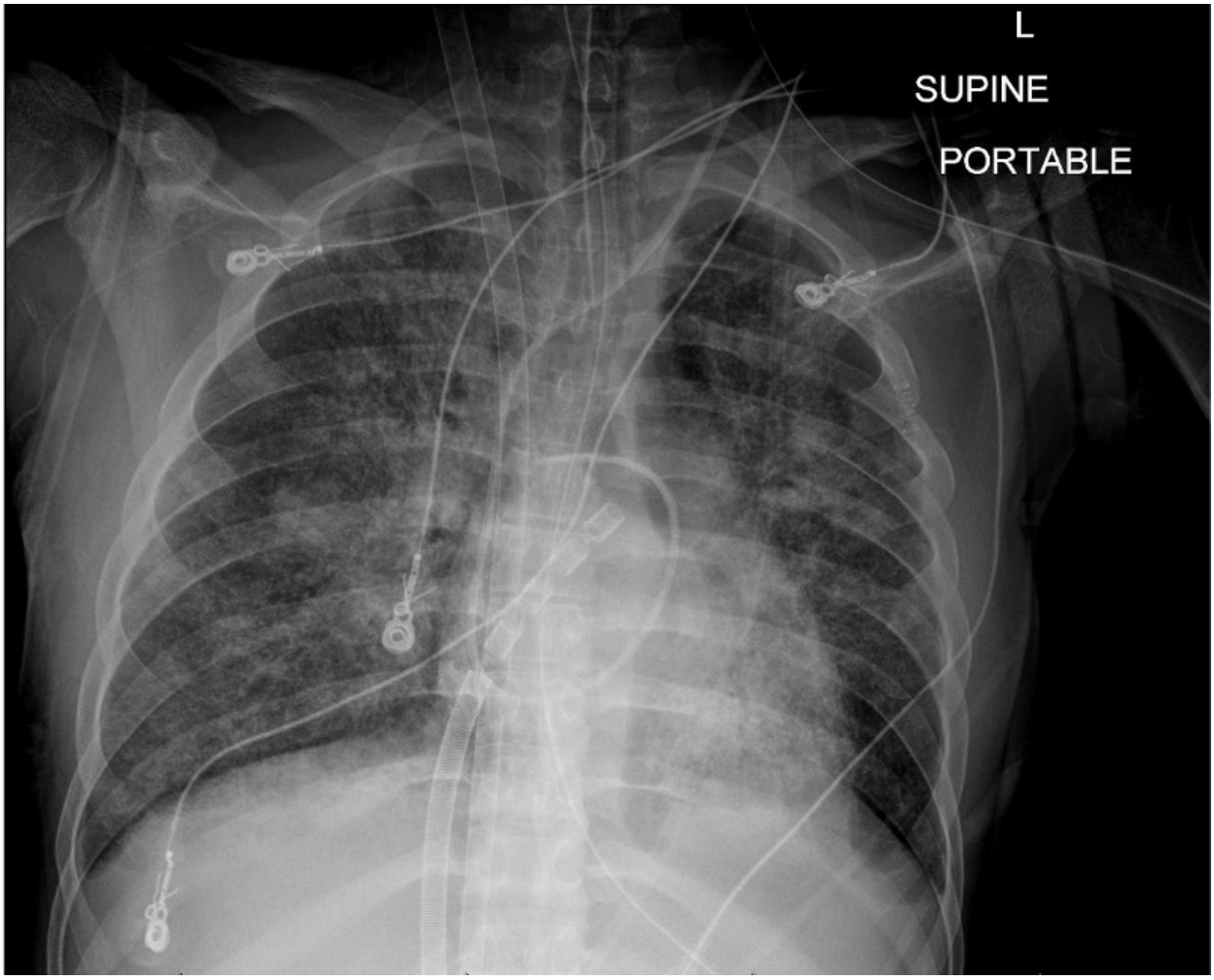
Teaching Points of the Case

Multimodal echo confirmed torrential MR from a flail P3 and ruled out papillary muscle rupture, endocarditis, and true VSD. LAVA ECMO clarified the physiology by improving pulmonary edema with LV unloading. The aortic cusp perforation was likely iatrogenic from the failed Impella attempt, underscoring the need for careful imaging. Overall, advanced TEE and color Doppler were essential to distinguish severe MR from structural defects in this critically ill patient.

Image Uploads


L
462
SUPINE
PORTABLE
51.2 mm (Integer)


MOB
L 127
SUPINE



Video Uploads
Video 1. ME MV flail LAX view, 2. ME MV flail LAX view CFD, 3. ME AV SAX view with color
Video 2. X plane of AV with color, 2. 3D VSD
Video 3. 3D MV, 2. 3D MV
Video 4. Post-repair ME LAX view, 2. Post-repair ME MV LAX view
Video 5. Post-repair ME AV SAX view with color, 2. Post-repair ME AV LAX view

References

Reference 1
Hahn RT, Abraham T, Adams MS, Bruce CJ, Glas KE, Lang RM, Reeves ST, Shanewise JS, Siu SC, Stewart W, Picard MH. Guidelines for performing a comprehensive transesophageal echocardiographic examination: recommendations from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. J Am Soc Echocardiogr. 2013 Sep;26(9):921-64. doi: 10.1016/j.echo.2013.07.009. PMID: 23998692.

SCA-SuperEcho-04
Post Transcutaneous Aortic Valve Replacement paravalvular leak management; The role of advanced 3-Dimensional echocardiography
Presenting Author: *Hesham Ezz, MD, E-AEC*

Introduction**:** Paravalvular leak (PVL) after transcutaneous aortic valve replacement (TAVR) is a feared complication with significant impact on post TAVR outcomes. PVL ≥ mild was associated with increased cardiovascular and all-cause mortality in 2 metanalyses of >45,000 patients[1]. Accordingly, prevention and early detection of TAVR associated PVL is critical. Several modalities can be utilized to localize, define severity and treat PVLs[2] (table 1). This is a report of a patient presenting for vascular plug occlusion of a moderate to severe post TAVR PVL where quantitative 3D echocardiography, grey scale optimization, and advanced software (GlassVueTM) provided a more precise definition of the paravalvular pathology and its severity and proved essential guiding the appropriate intervention

Brief Summary of the Clinical Case: 91-year-old man underwent uncomplicated TAVR procedure with a #26 balloon expandable valve (BE). He presented with progressive exertional dyspnea 4 weeks later. Repeat TTE showed elevated valve gradients and a moderate PVL. TEE was completed showing a potential moderate to severe anterior PVL encompassing ~ 50% of the AV circumference. Decision was made to pursue percutaneous vascular plug under general anesthesia with TEE guidance
Intraoperatively, TEE imaging in 2D color flow showed an anterior eccentric jet (video 1). Further interrogation, utilizing 3D color MPR with “hide greyscale” function and GlassVue™ technology with higher transparency, allowed the visualization of 3 complex PVL jets (figure 1, video 2)
To assess the severity of aortic insufficiency, 3D summative effective regurgitant orifice areas (EROA) is an option, albeit time consuming and subject to image and user limitations. To define the regurgitant severity we resorted to a volumetric method (figure 2). Our assessment of AI was mild to moderate, which may have been underestimated secondary to altered loading conditions (general anesthesia and positive pressure ventilation)
Based on the presence of multiple complex PVL jets, we decided to abort the vascular plug placement, and the patient received a valve-in-valve 26 BE TAVR the following day

Discussion:
2D color assessment
-AV SAX and LAX views (biplane) simultaneous orthogonal imaging is useful

3D assessment
-Multibeat acquisition improves spatial resolution
-'Hide grey scale' and GlassVueTM functions allowed clockface mapping and direction of complex jets relative to the valve structure

-Modality to change surgical course for patient

Volumetric assessment
-2D EROA by PISA limited for eccentric jets
-3D summative tracing EROA/vena contracta via 3D MPR though precise, can be time consuming
-Views: TG RV inflow-outflow, AV LAX, deep TG LAX
-Calculations: LVOT SV - RVOT SV

Impact of Echocardiographic findings: The demonstration of several complex/serpiginous PVL jets helped inform the decision to change the management of this patient from attempted vascular plug occlusion of the presumed 1 large paravalvular leak to valve-in-valve (ViV) TAVR

Teaching Points of the Case
1-Post TAVR PVLs:
-Typically multiple and complex
-Repair options are; balloon dilation, plug, ViV TAVR, and surgical replacement[3]
-2D echocardiography has limited utility, advanced 3D TEE is advantageous in diagnosis and management

2-Volumetric assessment of regurgitant lesions is advantageous compared to doppler in the presence of multiple complex jets

Image Uploads

Table 1. Various modalities for diagnosis and quantification of post TAVR PVL				
Modality	Specific Features	Strengths	Limitations	Clinical Utility
TTE (Transthoracic Echo)	2D/Color Doppler; initial screening; assesses LV size/function, gradients	Widely available; noninvasive; good for anterior leaks and overall cardiac assessment	Acoustic shadowing from prosthesis; suboptimal for posterior leaks; limited spatial resolution	First-line for initial assessment, severity grading, and LV response
TEE (Transesophageal Echo)	2D/3D imaging; multiplanar; color Doppler; direct visualization of sewing ring	Superior localization and characterization; essential for procedural planning	May miss anterior defects; acoustic shadowing; semi-invasive	Gold standard for PVL localization, sizing, and guiding intervention
3D TEE	Multiplanar reconstruction; direct vena contracta measurement	Precise defect sizing and location; improves device selection for closure	Image quality affected by artifacts; less validated for aortic than mitral PVL	Best for procedural planning and device sizing, especially for percutaneous closure
Cardiac CT	High-resolution anatomical imaging; planimetry of regurgitant orifice	Excellent for anatomical detail, defect morphology, and pre-procedural planning	Lacks flow quantification; metal artifacts; may miss small leaks	Adjunct when echo is inconclusive or for complex anatomy; device sizing and procedural guidance
CMR (Cardiac MRI)	Phase-contrast imaging; quantifies regurgitant volume/fraction	Accurate quantification of regurgitant volume; unaffected by acoustic shadowing	Limited availability; prosthetic artifacts; less spatial detail	Useful when echo is inconclusive or discordant; reclassifies severity; guides management
Cardiac Catheterization	Hemodynamic assessment; angiography	Direct measurement of pressures; visualizes regurgitation	Invasive; rarely needed; limited anatomical detail	Reserved for cases with inconclusive noninvasive imaging or for hemodynamic assessment
TAVR: Transcutaneous Aortic Valve Replacement, PVL: Para-Valvular Leak				

Table 1. Modalities for diagnosis of paravalvular leak (PVL). Approaches for PVL treatment include balloon post dilation (BPD), percutaneous vascular plug occlusion, valve-in-valve TAVR and surgical valve replacement.

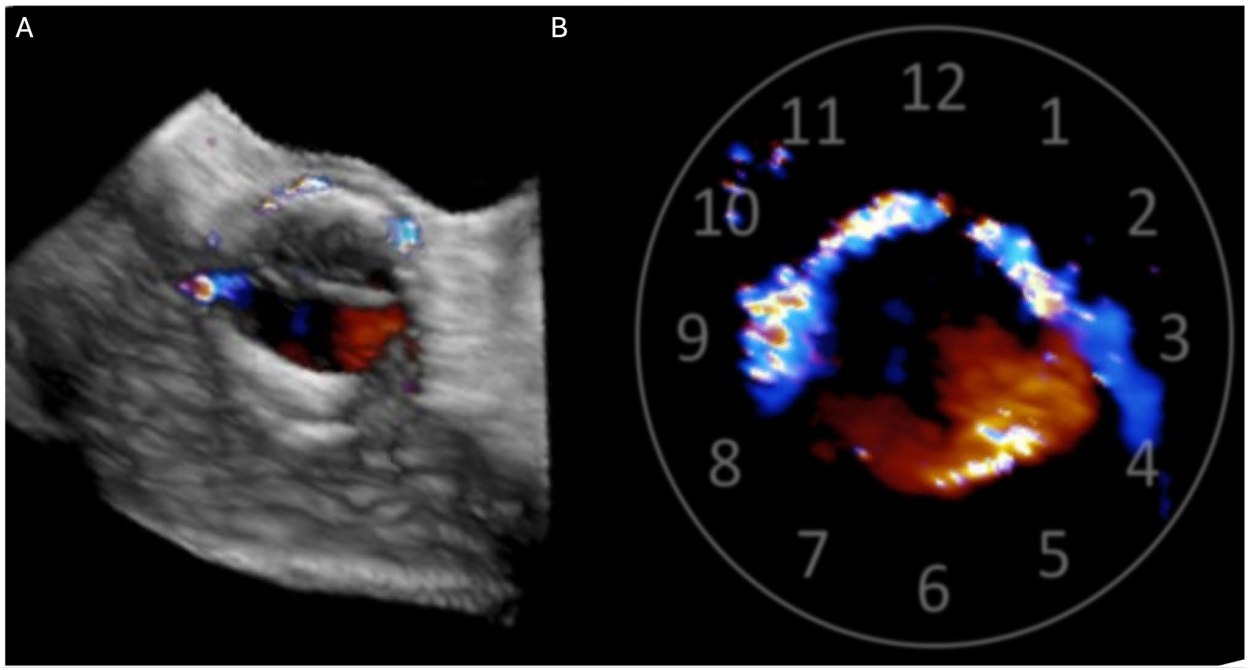


Figure 1. A. 3D multi-beat acquisition enface view of the aortic valve with concurrent color flow Doppler. On visual assessment there are potentially two posterior jets at approximately 11 o'clock and 1 o'clock. There is an anterior jet at approximately 4 o'clock to 6 o'clock, previously identified on 2D imaging. The color flow pattern is different between the anterior and the posterior jets. B. Same 3D multi-beat acquisition enface view of the aortic valve as panel A while using the 3D 'hide greyscale' function. In this image three paravalvular leaks are defined. The two posterior jets both have a narrow, serpiginous course. The anterior jet has a wider course.

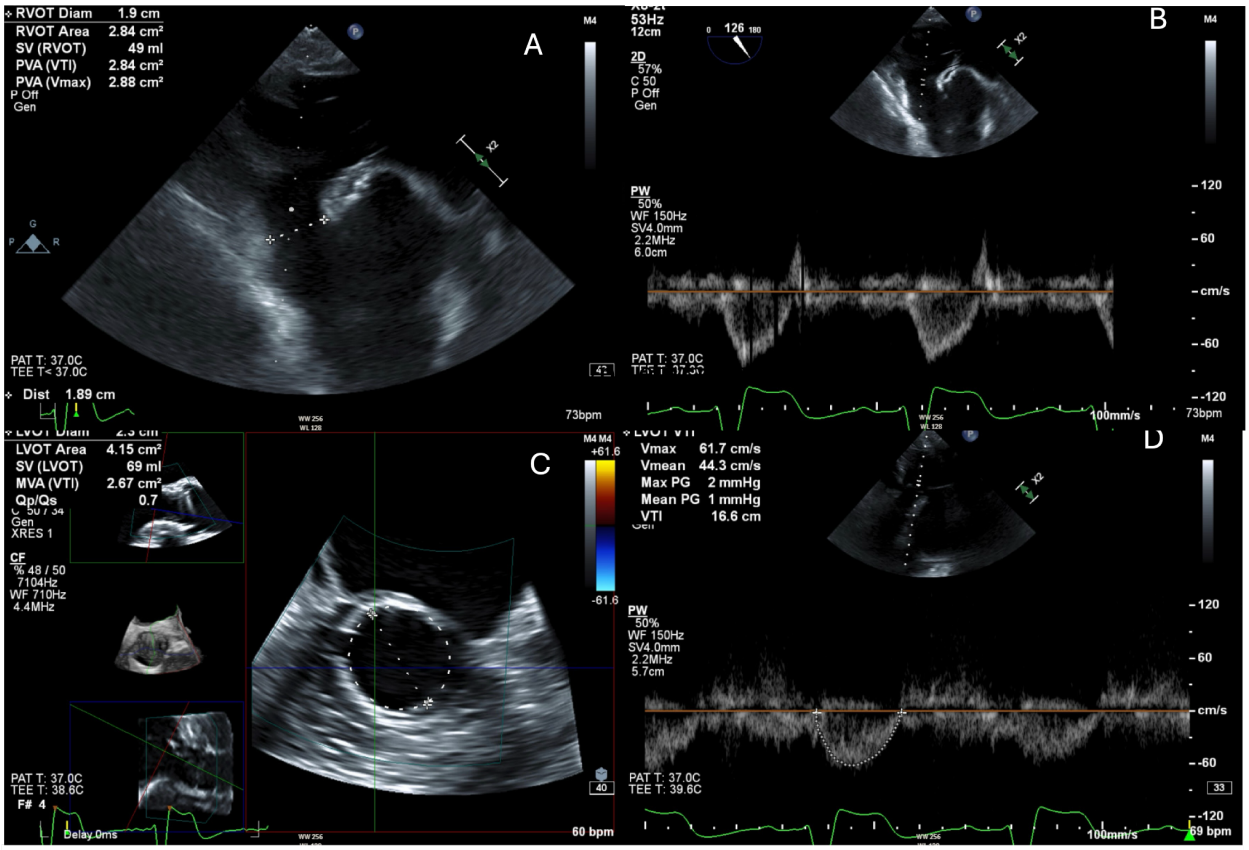


Figure 2. Quantitative echocardiography used to determination both the right ventricular outflow tract (RVOT) stroke volume (SV) and the left ventricular outflow tract (LVOT) stroke volume (SV). A. Transgastric RV inflow-outflow view measuring the diameter of the the RVOT at 1.89 cm. B. Pulse wave Doppler through the RVOT to measure RVOT velocity time integral (VTI). C. 3D assessment of the LVOT utilizing MPR to measure both the LVOT area and diameter. D. Deep transgastric long axis view with pulse wave Doppler at the LVOT to measure LVOT velocity time integral (VTI). The aortic valve regurgitant volume was calculated as the difference between the LVOT SV minus the RVOT SV since the patient had no pulmonic or mitral valve pathologies.
RVOT area was calculated as (***r^2) and RVOT SV was calculated as (RVOT area x RVOT VTI).
LVOT area was directly measured from 3D multiplanar reconstruction (MPR) and LVOT SV was calculated as (LVOT area x LVOT VTI)
Aortic regurgitant volume was calculated as (LVOT SV – RVOT SV). Regurgitant volume was 19 ml
Aortic valve regurgitant fraction was calculated as (AV regurgitant volume/LVOT SV). Regurgitant fraction was 28%
The aortic regurgitation was consistent with mild to moderate severity

Video Uploads

Video 1. 2D Biplane (simultaneous orthogonal imaging) of a mid-esophageal long axis view and mid-esophageal short axis view of the aortic valve with color flow Doppler. There is a an anterior PVL with visually mild severity. There is another potential posterior jet that is difficult to assess.

Video 2. 3D multi-beat acquisition enface GlassvueTM of the aortic valve with concurrent color flow Doppler. 3D optimization utilized to alter the transparency of the view to enhance PVL visualization. There were 2 posterior narrow and high velocity jets originating at the 11 and 1 o'clock position on the enface view with a serpiginous course and emergring at 9 and 3 o'clock on the LVOT side, respectively. A third wider anterior jet was detected, emerging on the LVOT side from 4 to 6

o'clock. The origin of the third jet was difficult to visualize due to the acoustic shadowing from the valve frame.

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(1) van Wely M, Rooijakkers M, Stens N, et al. Paravalvular regurgitation after transcatheter aortic valve replacement: incidence, quantification, and prognostic impact. Eur Heart J Imaging Methods Pract. 2024;2(2):qyae040. doi:10.1093/ehjimp/qyae040

Reference 2
(2) Sá MP, Bloom JP, Osho AA. Paravalvular Leak After Transcatheter Aortic Valve Implantation: Importance of Preprocedural Variables and Intraprocedural Assessment. J Am Heart Assoc. 2024;13(18):e037850. doi:10.1161/JAHA.124.037850

Reference 3
(3) Cruz-Gonzalez I, Rama-Merchan JC, Rodríguez-Collado J, et al. Transcatheter closure of paravalvular leaks: state of the art. Neth Heart J. 2017;25(2):116-124. doi:10.1007/s12471-016-0918-3

SCA-SuperEcho-05
Echocardiography and Management of Quadruple Valve Endocarditis
Presenting Author: *Talha Saif, MD*

Introduction: Infective endocarditis is a well-studied pathology that unfortunately has high rates of in-hospital mortality even in developed countries. Treatment takes into consideration many factors, such as: extent of valve damage, severity of valvular function, patient demographics and etcetera. Culminating these factors with expert clinical opinions dictates whether the patient receives a valve repair, biologic or mechanical valve. In this case report, we present an extraordinary case in which a 33 year old female suffered from quadruple valve endocarditis and was successfully treated allowing her to give birth the following year. We will discuss the echocardiographic findings along with the subsequent surgical management that took place.

Brief Summary of the Clinical Case: A 33 year old female with PMH significant for Shone Complex status post aortic coarctation repair as a child presented to an outside facility with concerns of bright red per rectum, and several months of fever and chills. A CTA performed at the outside hospital showed evidence of consolidation across both lobes and as well as areas of necrosis in both the kidneys and the lungs concerning for septic emboli. She was transferred to Duke University for further management. Her initial TEE demonstrated evidence of vegetations on the pulmonic, tricuspid, and aortic valves with evidence of severe PI, mild TR, and moderate AI as well as a subaortic membrane. The decision was made to replace the pulmonic, tricuspid and aortic valves via sternotomy. However, the TEE in the OR demonstrated a VSD and vegetation on the mitral valve that were not appreciated on prior studies. Given the severity of regurgitant jets on TEE, the initial decision was to perform the following procedures: tricuspid valve debridement, annuloplasty and cleft closure; mitral valve debridement and cleft closure; subaortic membrane

resection with aortic valve repair; pulmonic valve replacement (given severity of PI); and VSD closure. All of the procedures were successful save for the aortic valve repair. Despite the surgeon's best efforts, the aortic valve showed evidence of severe AI on TEE. The aorta was clamped once more, and the decision to do an aortic root replacement was made.

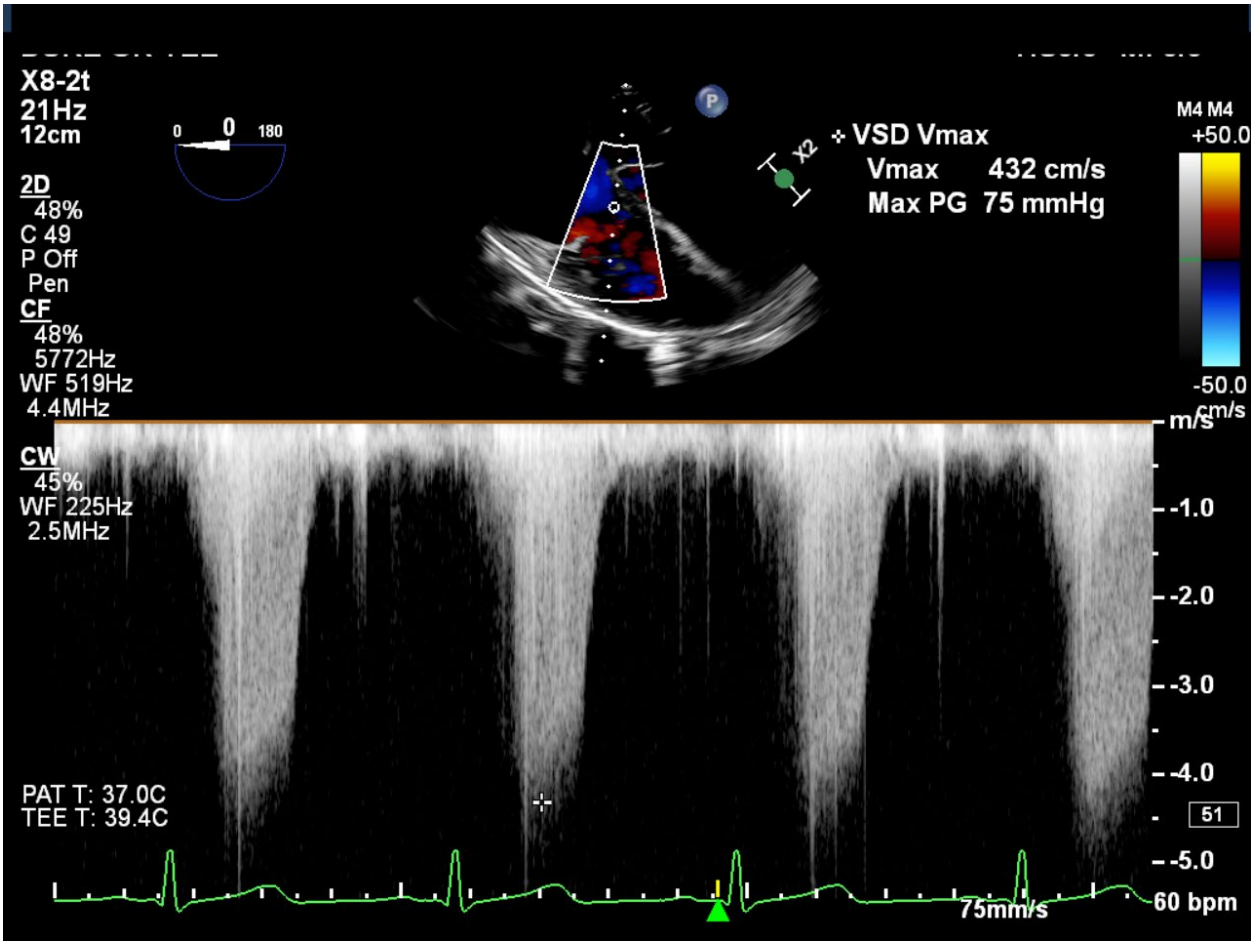
Discussion

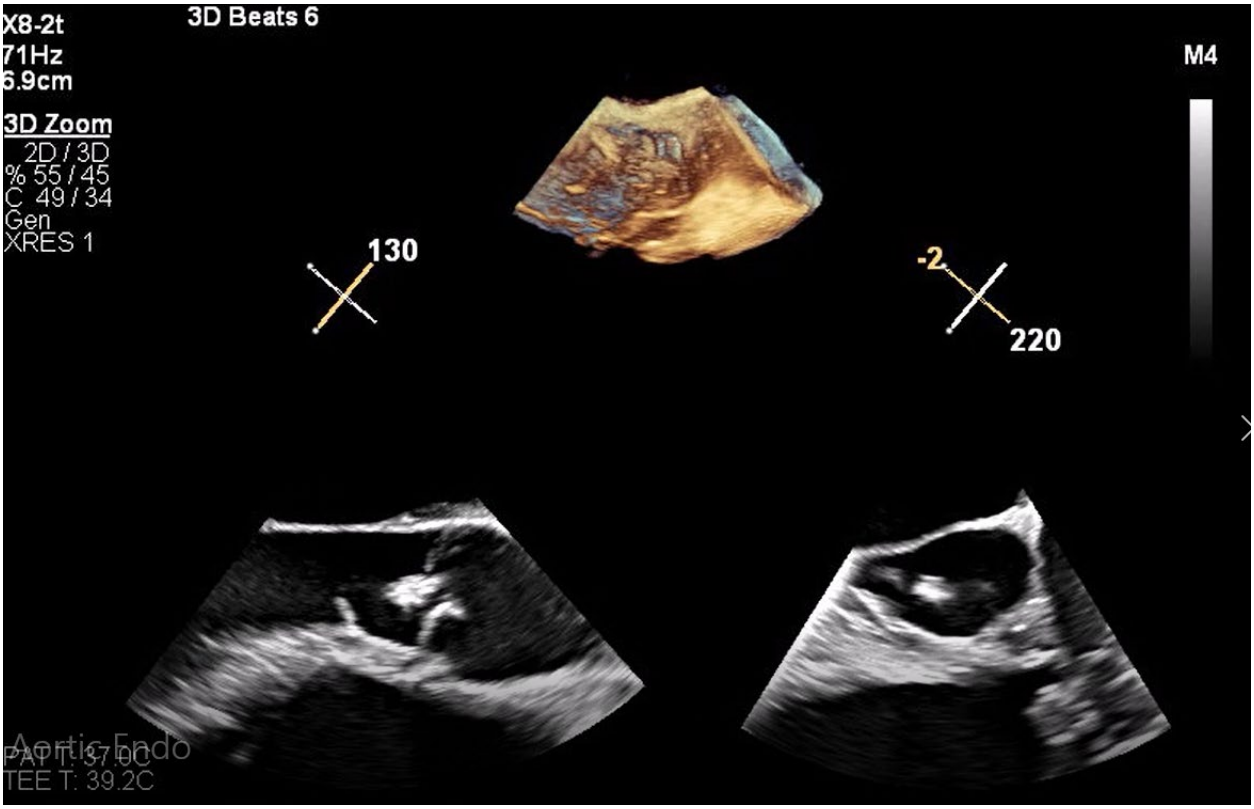
- Pulmonic Valve Views: Upper Esophageal SAX, RVOT Inflow-Outflow
- Tricuspid: ME 4 Chamber, RVOT Inflow-Outflow, Modified Bicaval
- Mitral: ME 4 Chamber, ME AV LAX
- Aortic: ME AV LAX, ME AV SAX, Deep Transgastric
- VSD: ME 4 Chamber

Impact of Echocardiographic findings: The intraoperative echo evaluation demonstrated new findings and subsequently changed the management of each valve. Initially, the plan was to do a PVR, TVR, and AVR. However, with the new findings of the VSD and mitral valve vegetation, a VSD closure and mitral valve debridement and cleft repair was formed. Additionally, the TVR de-escalated to an annuloplasty after further evaluation of the tricuspid valve. The subaortic membrane was resected and aortic valve repair was performed, but the AI did not improve following intervention. Thus, the echo findings prompted us to replace the aortic valve.

Teaching Points of the Case: Intraoperative TEE may showcase new pathologies not previously demonstrated and can severely impact the overall surgical plan. Additionally, it can help gauge the efficacy of the surgical interventions and help the team determine the next course of action.

Image Uploads


X8-2t
21Hz
12cm
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48%
C 49
P Off
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CF
48%
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WF 519Hz
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VSD Vmax
Vmax 432 cm/s
Max PG 75 mmHg
M4 M4
+50.0
-50.0
cm/s
m/s
-1.0
-2.0
-3.0
-4.0
-5.0
PAT T: 37.0C
TEE T: 39.4C
75mm/s
60 bpm



Video Uploads

Video 1: ME 2 Chamber View, Focus on Mitral Valve

Video 2: RVOT Inflow-Outflow View, demonstrating vegetations on pulmonic and tricuspid valve

Video 3: UE Short Axis View, Evidence of PI

Video 4: ME 4 Chamber View, Focus on Tricuspid Valve

SCA-SuperEcho-06
Multimodal Monitoring–Guided Intraoperative Detection of Ascending Aortic Dissection in Minimally Invasive Atrial Septal Defect Closure
Presenting Author: *Gustavo Zunkowski*

Introduction: Aortic injury during cannulation for cardioplegia delivery is rare but highly morbid, and early recognition is essential to prevent catastrophic progression. Transesophageal echocardiography is crucial in minimally invasive cardiac surgery, where direct visualization of the ascending aorta is limited. This case describes an initially subtle aortic wall injury that progressed to ascending aortic dissection during minimally invasive atrial septal defect repair, highlighting the value of continuous multimodal monitoring.

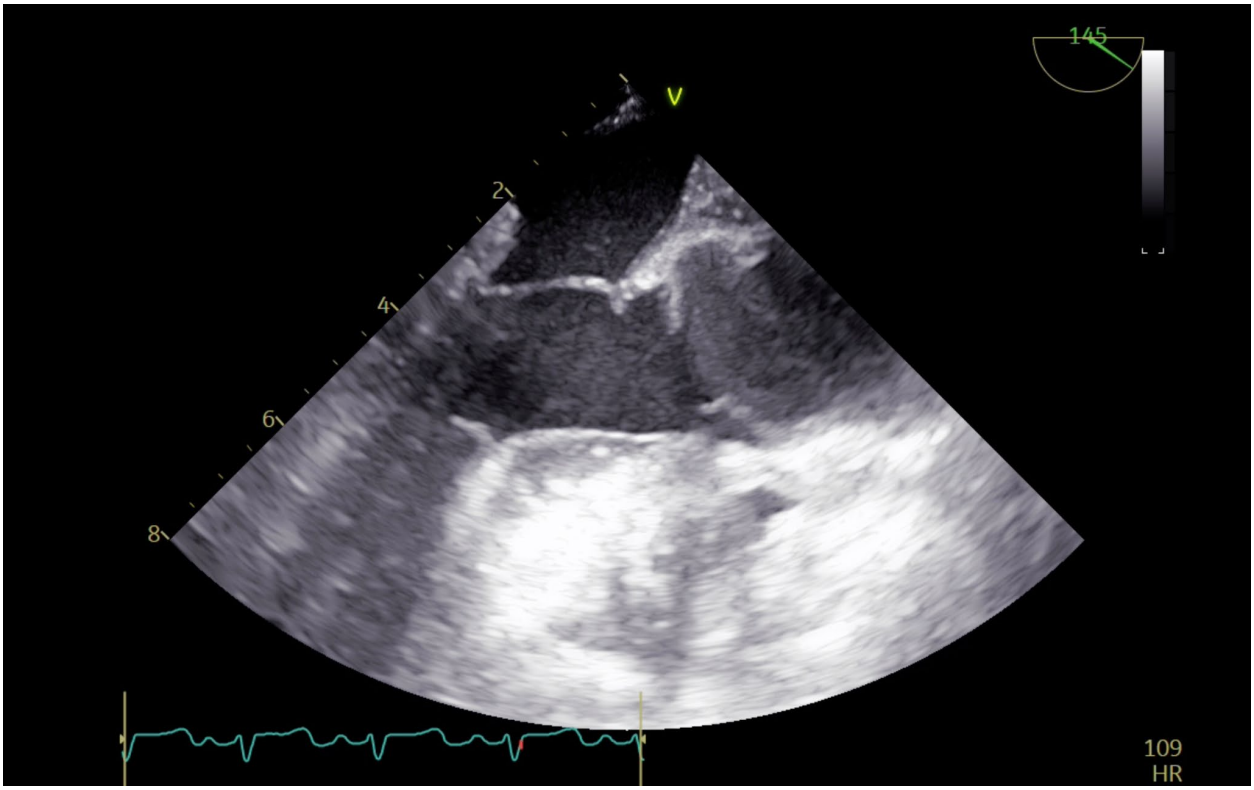
Brief Summary of the Clinical Case: A fifty-eight-year-old woman with arterial hypertension, persistent atrial fibrillation, and an ostium secundum atrial septal defect was scheduled for closure through right mini-thoracotomy. Preoperative transthoracic echocardiography showed a large interatrial communication, severe left atrial enlargement, probable pulmonary hypertension, and preserved ventricular function. After induction, baseline transesophageal imaging confirmed the defect and normal ascending aortic anatomy. During cardiopulmonary bypass, a localized hematoma of the ascending aorta appeared on imaging and was attributed to cardioplegia cannulation. Conversion to sternotomy suggested the lesion was confined to the adventitia. Minutes later, a sudden decline in left-sided cerebral oxygen saturation raised concern for malperfusion. Repeat transesophageal examination, including three-dimensional views, revealed new separation of the aortic wall layers consistent with ascending aortic dissection. Aortotomy confirmed the diagnosis, and the affected segment was replaced with a straight vascular graft. Cerebral saturation normalized after repair, the defect was closed, and the patient was extubated in the operating room and discharged the following day without neurological deficits.

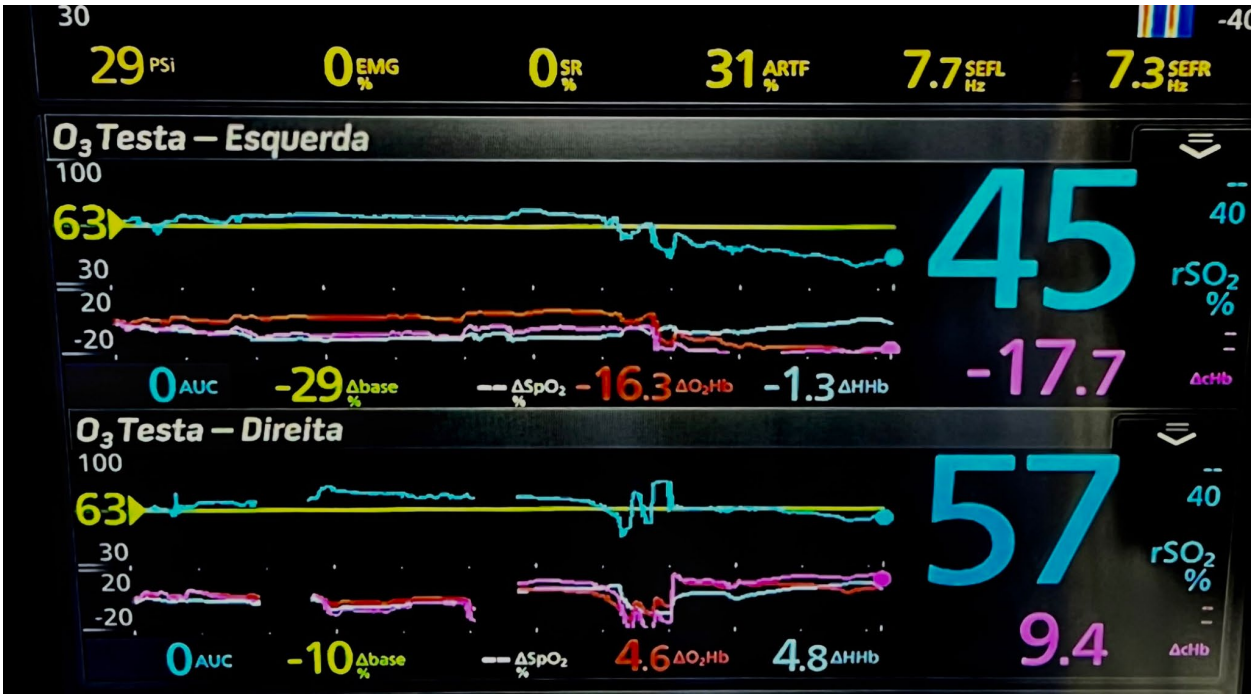
Discussion: This case shows how a seemingly limited aortic wall injury can progress quickly under the hemodynamic stress of cardiopulmonary bypass. The combination of abrupt cerebral desaturation and evolving transesophageal findings indicated early malperfusion and prompted rapid conversion from a minimally invasive approach to sternotomy. Because mini-thoracotomy restricts direct aortic visualization, reliance on imaging becomes essential to detect subtle wall abnormalities. Early integration of physiological and anatomical data enabled timely diagnosis, definitive aortic repair, and prevention of neurological or hemodynamic deterioration.

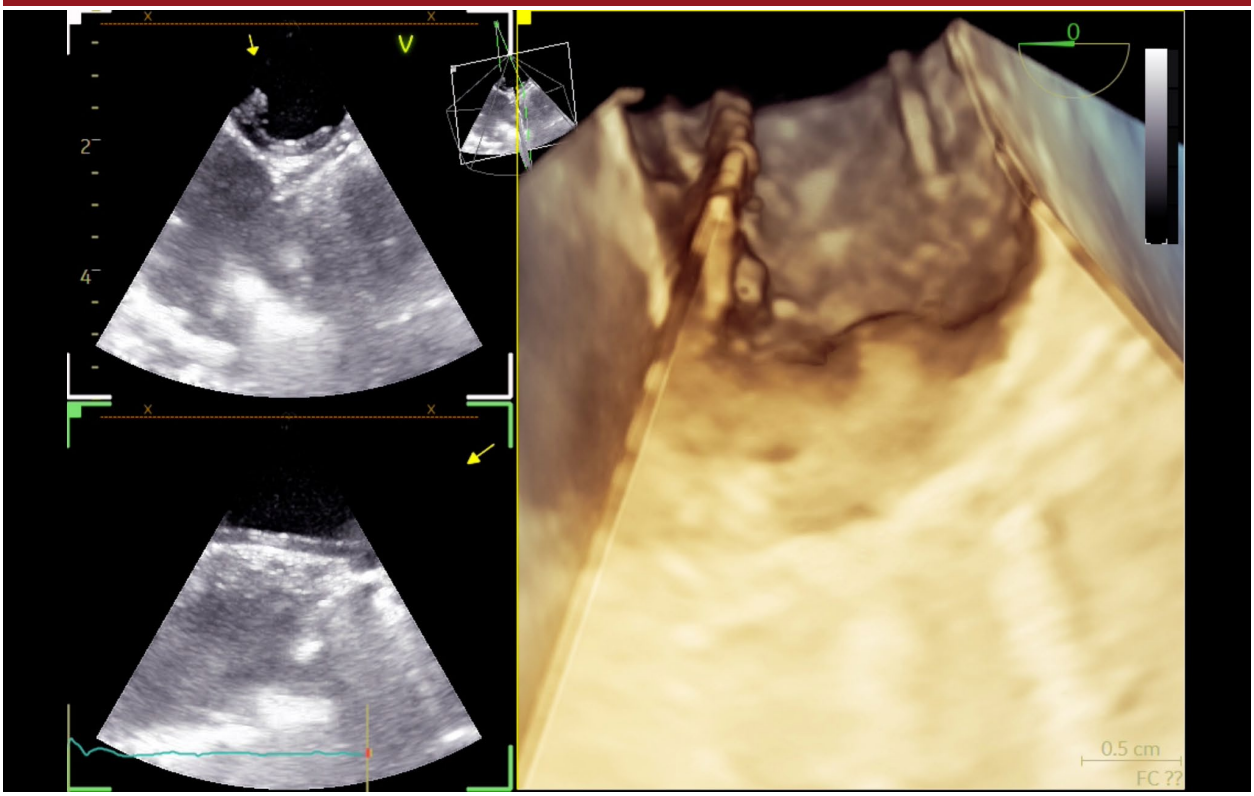
Impact of Echocardiographic findings: Echocardiography altered the intraoperative strategy by identifying the initial aortic injury and later confirming its progression to dissection. These findings prompted immediate conversion to sternotomy, aortotomy, and replacement of the ascending aorta, preventing major complications. Imaging then confirmed restored aortic integrity after repair, allowing safe completion of the procedure and contributing to the favorable outcome.

Teaching Points of the Case: Subtle aortic injuries may progress quickly during minimally invasive cardiac surgery, requiring continuous transesophageal imaging and cerebral oxygenation monitoring. Initial detection of a mural hematoma, followed by sudden cerebral desaturation and new structural abnormalities on repeated imaging, illustrates how combining anatomical and physiological information improves situational awareness and guides timely intervention.

Image Uploads


145
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8
109
HR


30
29 PSi
0 EMG %
0 SR %
31 ARTF %
7.7 SEFL Hz
7.3 SEFR Hz
O3 Testa – Esquerda
100
63
30
20
-20
45
40
rSO2 %
0 AUC
-29 Δbase %
ΔSpO2 %
-16.3 ΔO2Hb
-1.3 ΔHHb
-17.7
ΔcHb
O3 Testa – Direita
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20
-20
57
40
rSO2 %
0 AUC
-10 Δbase %
ΔSpO2 %
4.6 ΔO2Hb
4.8 ΔHHb
9.4
ΔcHb



Video Uploads

Video 1: This video shows a crescentic, echogenic thickening of the aorta wall compatible with an intramural hematoma

Video 2: This video shows a multiplanar reconstruction with 2D and 3D transesophageal echocardiography (TEE) focused on the ascending aorta, and it demonstrates findings consistent with evolving aortic wall dissection.

References

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Biancari F, Jormalainen M, Raivio P, Mustonen C, Honkanen HP, Valo J, Vento A, Juvonen T. Cerebral Oximetry Monitoring in Patients Undergoing Surgery for Stanford Type A Aortic Dissection. J Cardiothorac Vasc Anesth. 2021 Jul;35(7):2019-2025. doi: 10.1053/j.jvca.2020.10.011. Epub 2020 Oct 13. PMID: 33144000.



FELLOW AND RESIDENT ABSTRACTS

“Best in Show” Complex Case Presentations

Friday, April 24, 2026 | 10:30 AM – 11:30 AM

#FR – BIS – OP1

Keeping the Blood Flowing: Perioperative Strategies for Coronary Unroofing in Sickle Cell Disease

Presenting Author: *Jaskaran S. Mangat, DO, CA-1 Resident, AIMMC (Advocate Chicago)*

Introduction

Anomalous origin of the left main coronary artery (MCA) from the right coronary cusp is a rare and potentially lethal congenital anomaly associated with myocardial ischemia and sudden cardiac death. Surgical unroofing is technically challenging and in patients with sickle cell disease (SCD), anesthetic management is further complicated by the risk of Hgb polymerization during hypoxia, hypothermia, acidosis, and low-flow states. We describe perioperative management of severe SCD undergoing LMCA unroofing, highlighting rapid intraoperative complete exchange transfusion, neuroprotection without hypothermia, and coordination of multidisciplinary care.

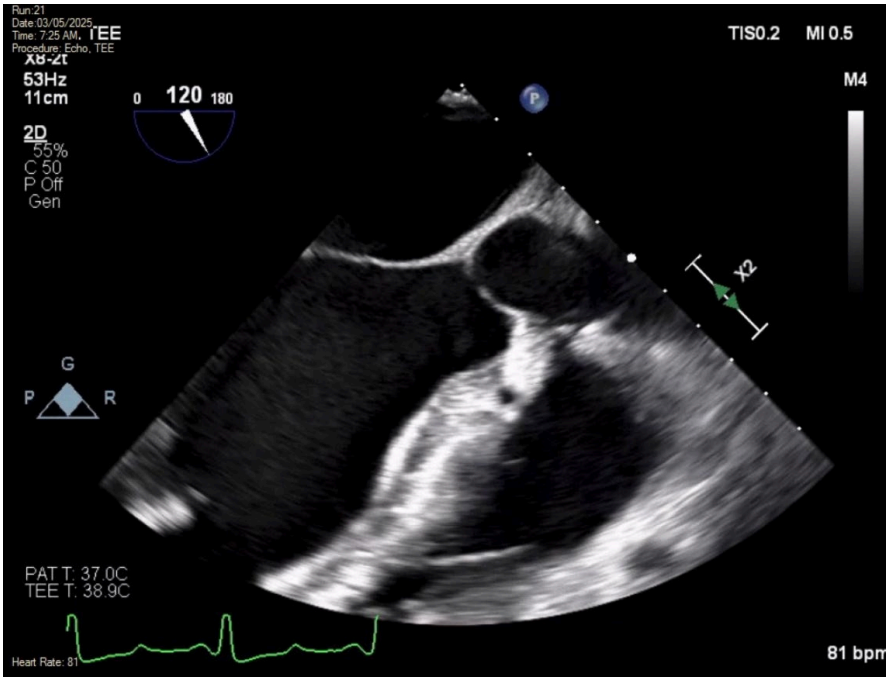
Case Presentation

A 55-year-old African American male (BMI 28.6) with homozygous SCD (Hbs 82.6%), CKD III, hypertension, remote PE with IVC filter, and prior COVID pneumonia with tracheostomy presented with exertional chest pain. Cardiac MRI showed inferior wall motion abnormality. CA and LHC revealed anomalous MCA from the right coronary cusp with an intraseptal/intraconal interarterial course and dynamic compression. To reduce HbS < 30% before cardiopulmonary bypass (CPB), hematology, anesthesia, and surgery jointly planned an abrupt, full-volume exchange transfusion at CPB initiation. Baseline Hgb was 10.9 g/dL; creatinine 1.27 mg/dL. After induction, strict normothermia was maintained to avoid sickling. Upon CB initiation, ~5 L of native blood was drained completely and rapidly, not in the usual slow 1:1 exchange. This total drainage produced an immediate, functionally circulatoryarrest-like state and the heart entered ventricular fibrillation from loss of coronary perfusion. Perfusion team then rapidly infused donor blood through the arterial cannula, restoring full intravascular volume within seconds, followed by successful internal cardioversion to normal sinus rhythm. Importantly, the patient was NOT cooled prior to this arrest-like period; EEG-guided neuroprotection (high-dose propofol and steroids) ensured electrocerebral silence despite normothermia. The RVOT was divided to unroof the intraseptal LMCA, reconstructed with bovine pericardium. Cell salvage was intentionally avoided due to the risk of reinfusing sickled erythrocytes.The patient was transferred to CVICU on milinone 0.25 ug/kg/min and norepinephrine 0.08 ug/kg/min, extubated POD 1, and discharged home POD 8 neurologically intact. TTE post-CPB showed preserved biventricular function and widely patent LMCA. A superficial sternal wound infection one month later resolved with antibiotics.

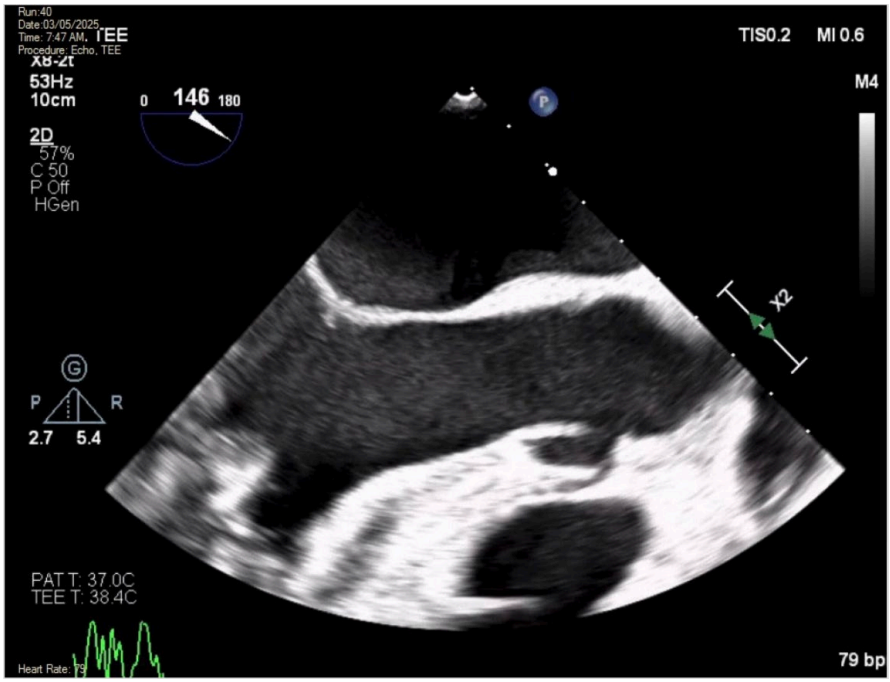
Discussion

Cardiac surgery in SCD is challenging because CB exposes patients to nearly all major sickling factors. Reducing HbS rapidly and maintaining strict normothermia are essential. This case required an unconventional, total-volume exchange strategy, performed instantaneously rather than staged, representing a perfusion approach rarely described in SC patients undergoing cardiac surgery. Neuroprotection was achieved at normothermia using EEG-guided electrocerebral silence instead of traditional hypothermic circulatory arrest—an uncommon strategy in SCD, where cooling may worsen sickling risk. The surgical repair itself-unroofing an intraseptal LMCA via RVOT division-is technically demanding and rarely reported in combination with severe SCD.

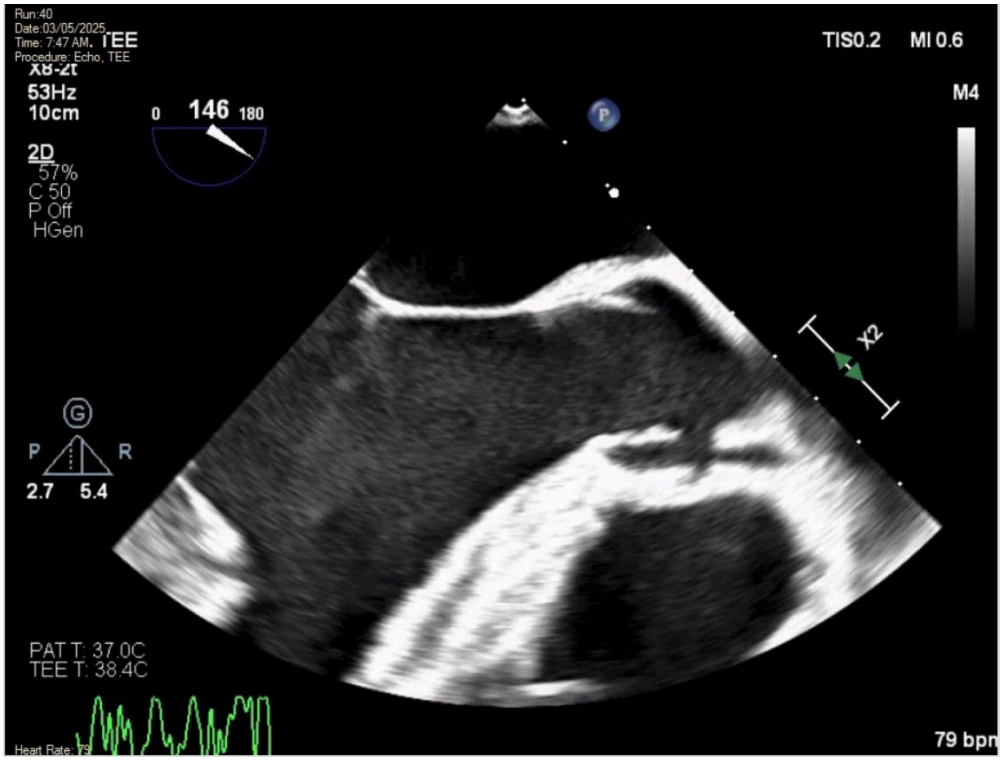
Images



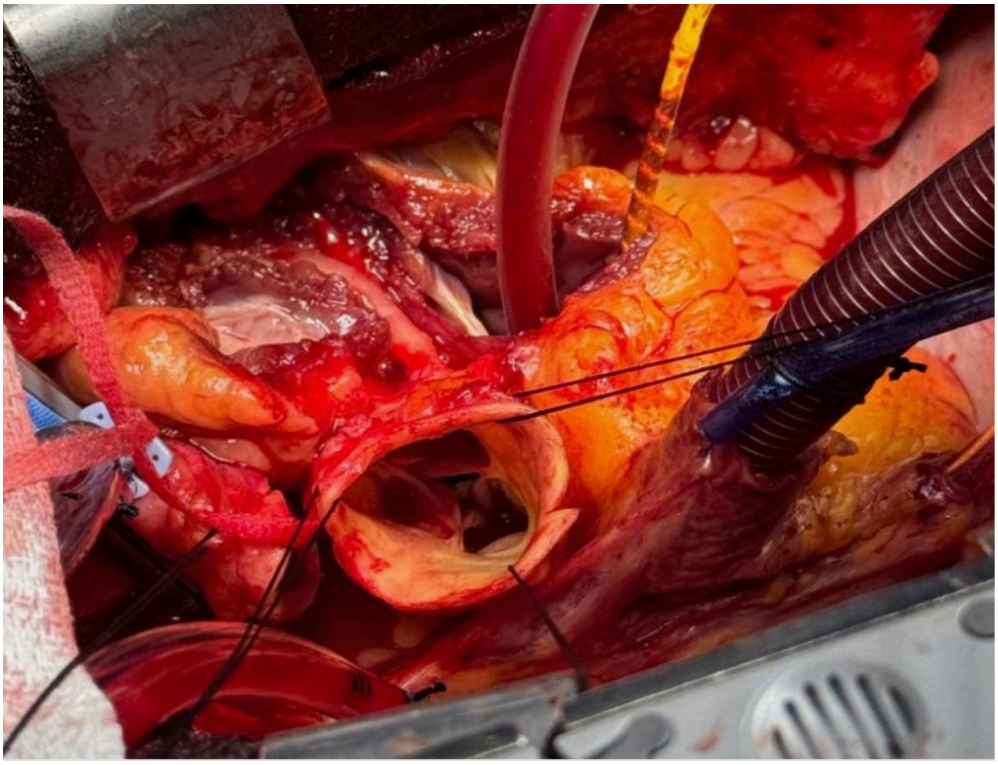
Shows LMCA entering the septum hence the intraconal-intraseptal course



Coronary artery image 1 of 2 showing clear branch



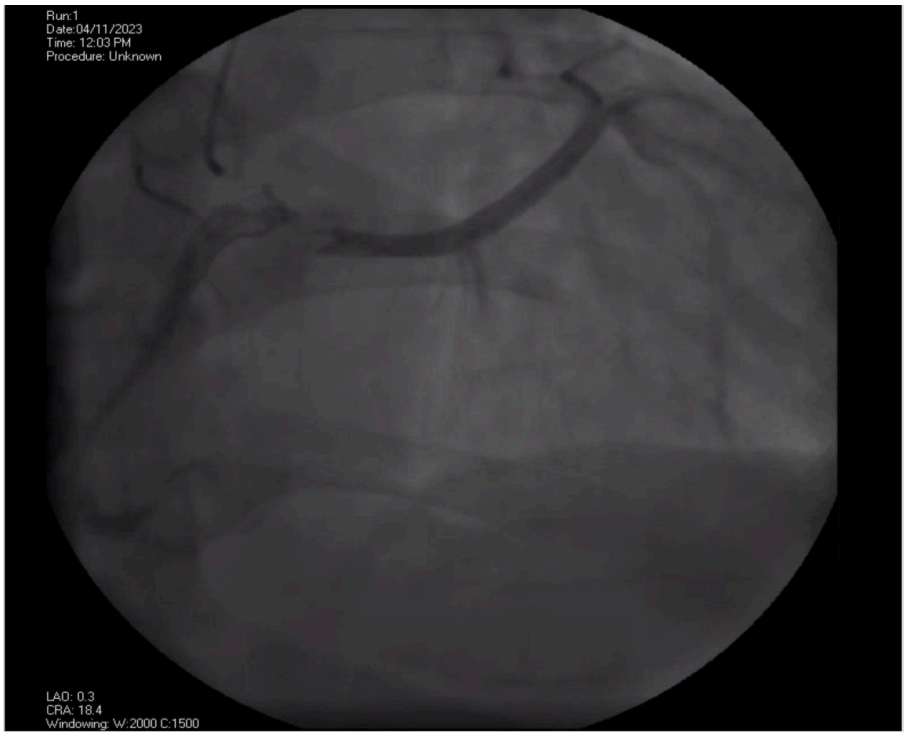
Coronary artery image 2 of 2 showing clear branch (starting from right sinus of valsalva instead of one)



surgical view



Angiography image 1 of 2, showing that both the LMCA (more horizontal course) and RCA (vertical) start from same origin



Angiography image 1 of 2, showing that both the LMCA (more horizontal course) and RCA (vertical) start from same origin

References

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#FR – BIS – OP2

Transcatheter Aortic Valve Replacement Explant after Multiple Iatrogenic Intracardiac Shunts

Presenting Author: *Shane G. Carr, MD, Anesthesiology Resident, Northwestern University*

Introduction

While transcatheter aortic valve replacement (TAVR) has demonstrated considerable clinical success in the treatment of aortic stenosis (AS), particularly among high-risk populations, surgical explantation may be necessary in some cases, most frequently due to endocarditis, structural valve degeneration, or paravalvular leak. One rare complication of TAVR is iatrogenic intracardiac shunt (ICS), occurring in 0.5% of cases. Risk factors for post-TAVR ICS include excessive balloon dilation, oversized valve placement, prior thoracic radiation, and severe annular calcification. When it occurs, it has a morbidity of 50% and a 30-day mortality of 25%.

Case Presentation

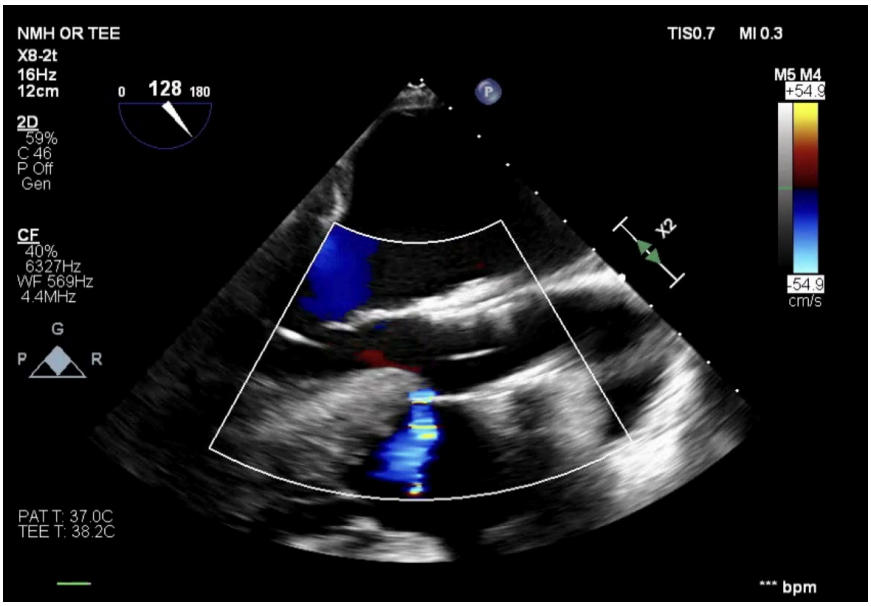
A 78-year-old female with a history of severe AS (mean gradient 71mmHg, area 0.59cm^2), triple-vessel coronary artery disease, and stage IV chronic kidney disease underwent TAVR after declining surgical aortic valve replacement and coronary artery bypass grafting (CABG). The procedure was seemingly uncomplicated with only residual mild perivalvular regurgitation. One week later, she presented with dyspnea and was found to have laboratory findings consistent with hemolysis, including a hemoglobin of 6.7 mg/dL and elevated lactate dehydrogenase. Transthoracic echocardiography revealed a new 0.5 cm perimembranous ventricular septal defect (VSD) with left-to-right-shunt. The decision was made to proceed with TAVR explant, VSD closure, aortic valve replacement (AVR), and three-vessel CABG. Pre-cardiopulmonary bypass (CPB) transesophageal echocardiography (TEE) demonstrated a TAVR valve with a large perivalvular leak, a perimembranous VSD with left-to-right flow, and a previously undetected Gerbode defect. CBP lasted 193 minutes. 45 minutes post-CPB, the patient developed worsening hypoxia, hypercarbia, and lactic acidosis. Over the next two hours, the patient developed RV failure, requiring escalating inotropic and vasopressor support. Inhaled nitric oxide was initiated and diuresis with furosemide was attempted to support the worsening RV function without response. Given the patient's tenuous clinical status, the decision was made to initiate veno-arterial (V-A) extracorporeal membrane oxygenation (ECMO). Postoperatively, the patient's cardiopulmonary function improved, and V-A ECMO decannulation occurred on postoperative day (POD) 2. Urine output remained negligible and continuous veno-venous hemofiltration was initiated. She was extubated on POD 5 and discharged on POD 26 with continued need for intermittent hemodialysis.

Discussion

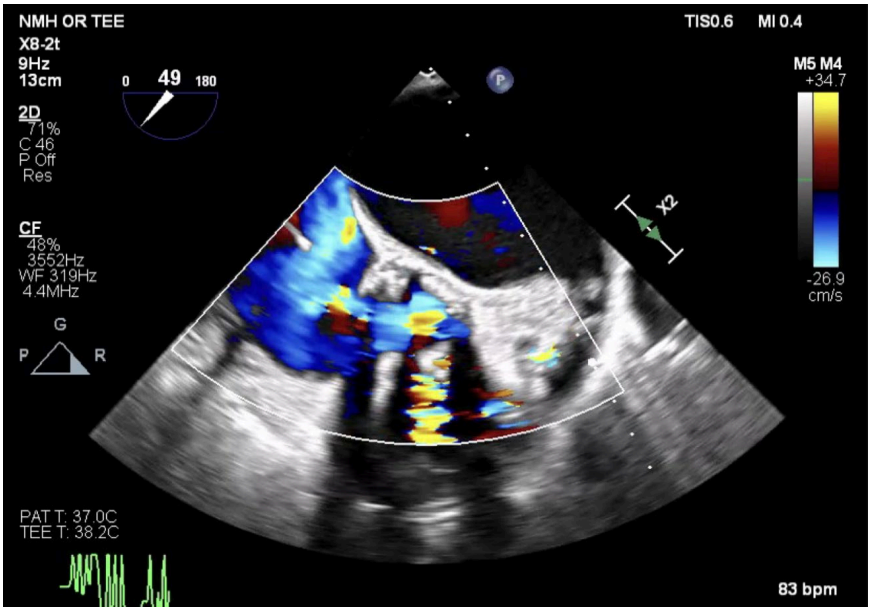
Although rare, ICS after TAVR is associated with substantial morbidity and mortality, attributable to three main factors. First, high-risk patients undergoing TAVR have co-morbidities prohibiting surgery. Second, TAVR explantation due to ICS is frequently an urgent procedure due to acute clinical decompensation. Third, the added complexity of repairing an ICS prolongs CPB time, contributing to worsened postoperative outcomes. In our case, these factors likely compounded, resulting in post-cardiotomy shock and the

development of end-stage renal disease. While TAVR has revolutionized the treatment of AS, it does have unique complications that must be considered.

Images



Perimembranous ventricular septal defect



Gerbode defect

References

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#FR – BIS – OP3

A Rare Presentation of HLHS With Absent Ascending Aorta Managed with Neonatal VAD Support

Presenting Author: Kellen Creech, DO, University of Florida

Introduction

Hypoplastic left heart syndrome with aortic atresia is one of the most severe forms of congenital heart disease, characterized by underdevelopment of the left-sided cardiac structures.[1] Without intervention, this lesion can be fatal in the neonatal period, and even with staged palliation affected infants face significant morbidity. Despite advances, some HLHS patients experience refractory ventricular dysfunction or hemodynamic instability. Mechanical circulatory support, including VADs, can be a bridge to recovery, transplantation, or staged repair in patients with failing single-ventricle physiology. However, the use of durable VADs, such as the Berlin Heart EXCOR, presents challenges including complex anatomy, altered physiology following palliation, and risks of thrombosis, bleeding, and neurologic injury. Reports of VAD support in HLHS with aortic atresia remain limited but support primary VAD insertion compared to rescue VAD insertion.[2] We present the rare case of an infant with an absent ascending aorta with HLHS and LV coronary fistula who underwent implantation of a Berlin VAD for transplant evaluation.

Case Presentation

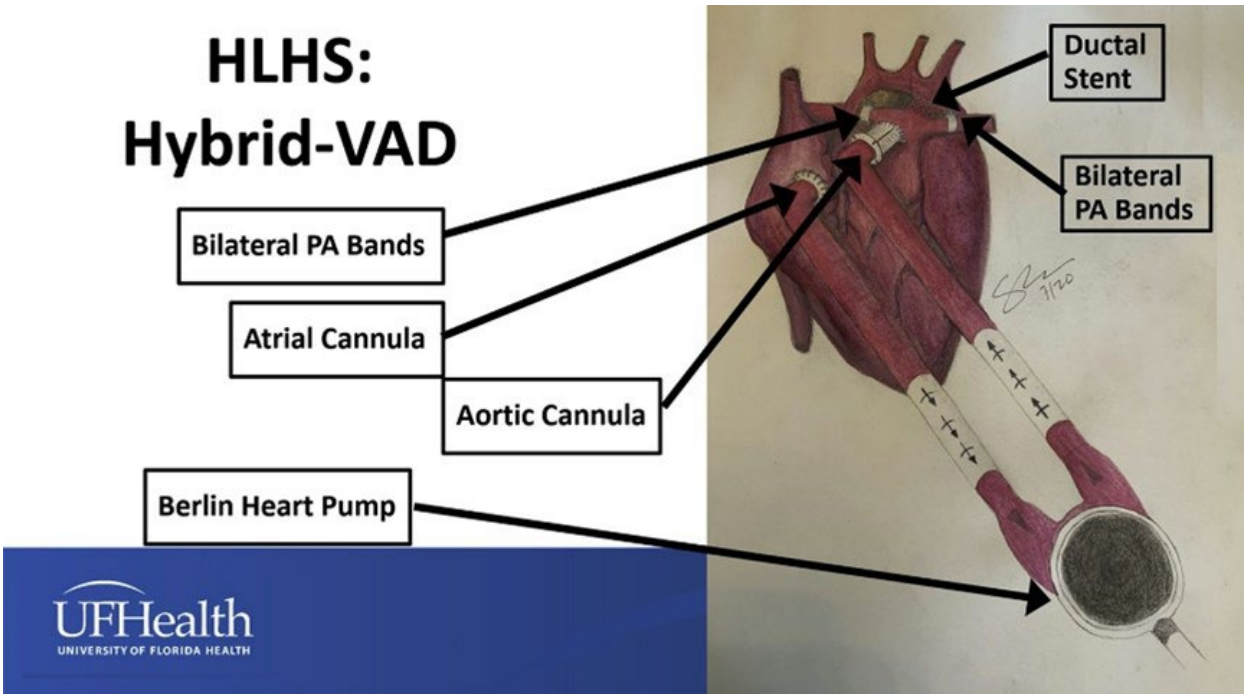
A female with prenatal HLHS was born at 35w1d via emergent C section due to fetal distress. The baby was started on PGE and intubated for poor respiratory effort. The baby also had anasarca and ascites requiring diuretics and paracentesis. Cardiac CT showed HLHS with absence of aorta, LV dependent coronaries with coronary cameral fistula connecting with the LV, PDA and cor triatriatum formation. Baby was transferred to us for transplant evaluation. Baby arrived intubated and on PGE and milrinone infusions. Patient went to the OR for single ventricular assist device, atrial septectomy and bilateral PA bands. Post-bypass, the heart was grossly distended, asystolic and had areas of ischemia throughout. Berlin VAD was changed to PediMag VAD due to hemodynamic instability with lactic acidosis and poor renal function. Despite support with high cardiac indices, she continued to deteriorate over the next 52 days and was not a suitable transplant candidate and ultimately care was withdrawn.

Discussion

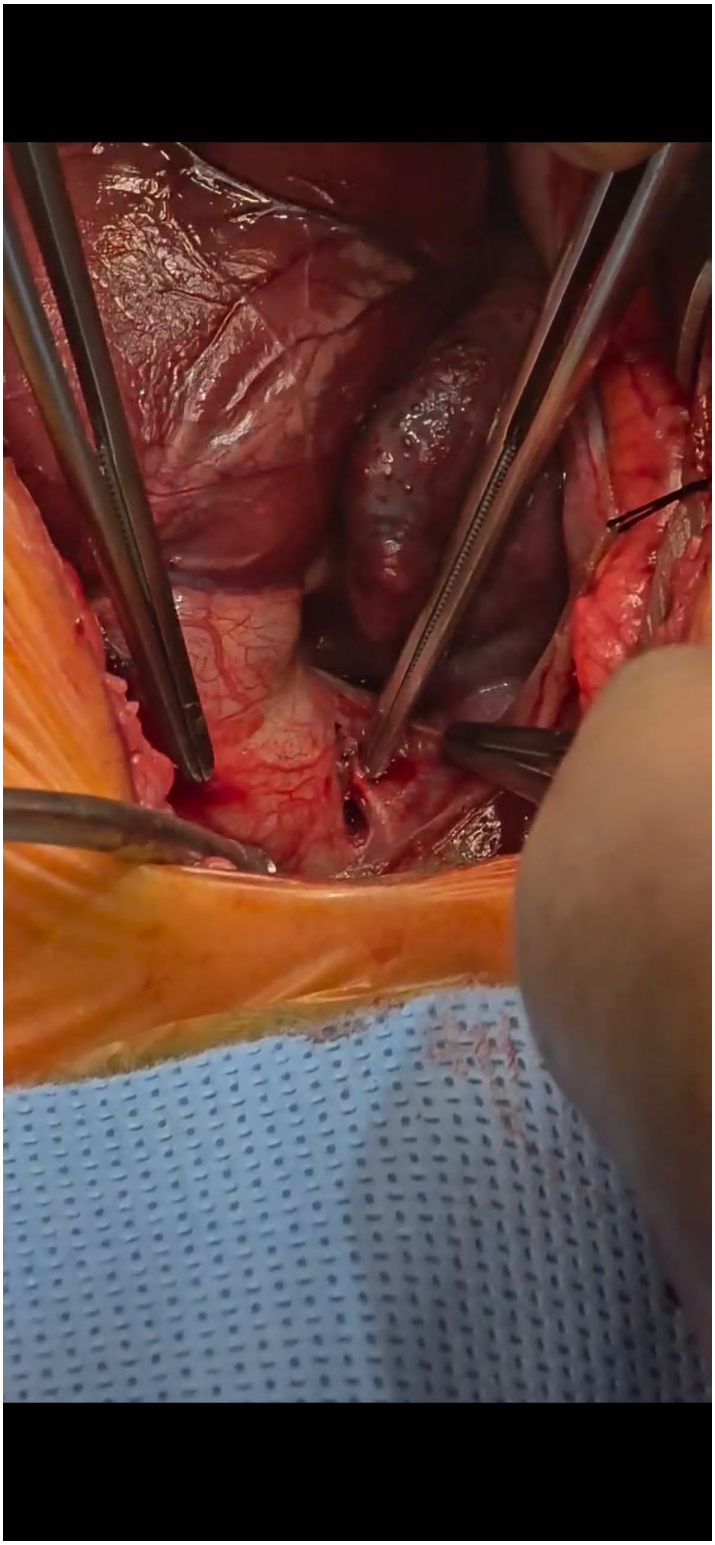
HLHS with absence of the ascending aorta represents one of the rarest and severe variants. In these patients, systemic perfusion is dependent on retrograde flow from the ductus arteriosus into the aortic arch. This physiology presents challenges to surgical palliation and MCS. Use of VADs in single-ventricle patients is managed in our institution in the referenced algorithm with outcomes.[3] The univentricular circulation, ductal-dependent systemic perfusion, and parallel pulmonary–systemic circulations create challenges in achieving adequate blood flow distribution. In this case, the decision for VAD implantation was driven by progressive ventricular dysfunction and inability to maintain end-organ perfusion with medical therapy alone. The absent ascending aorta added a layer of complexity and risk, as systemic output was dependent on ductal flow. Establishing adequate VAD flow in this anatomy requires careful surgical planning to ensure effective perfusion and avoidance of excessive pulmonary blood flow. Our patient's initial stabilization on

VAD support demonstrated that, despite these challenges, MCS can be a viable option in extreme variants of HLHS.

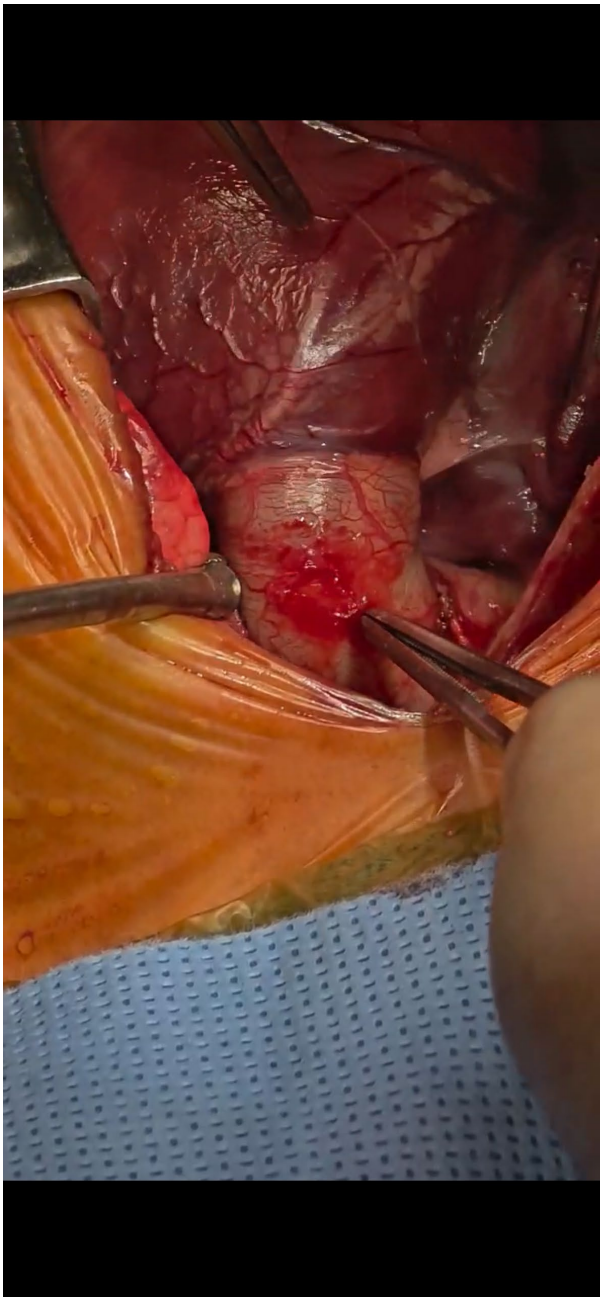
Images



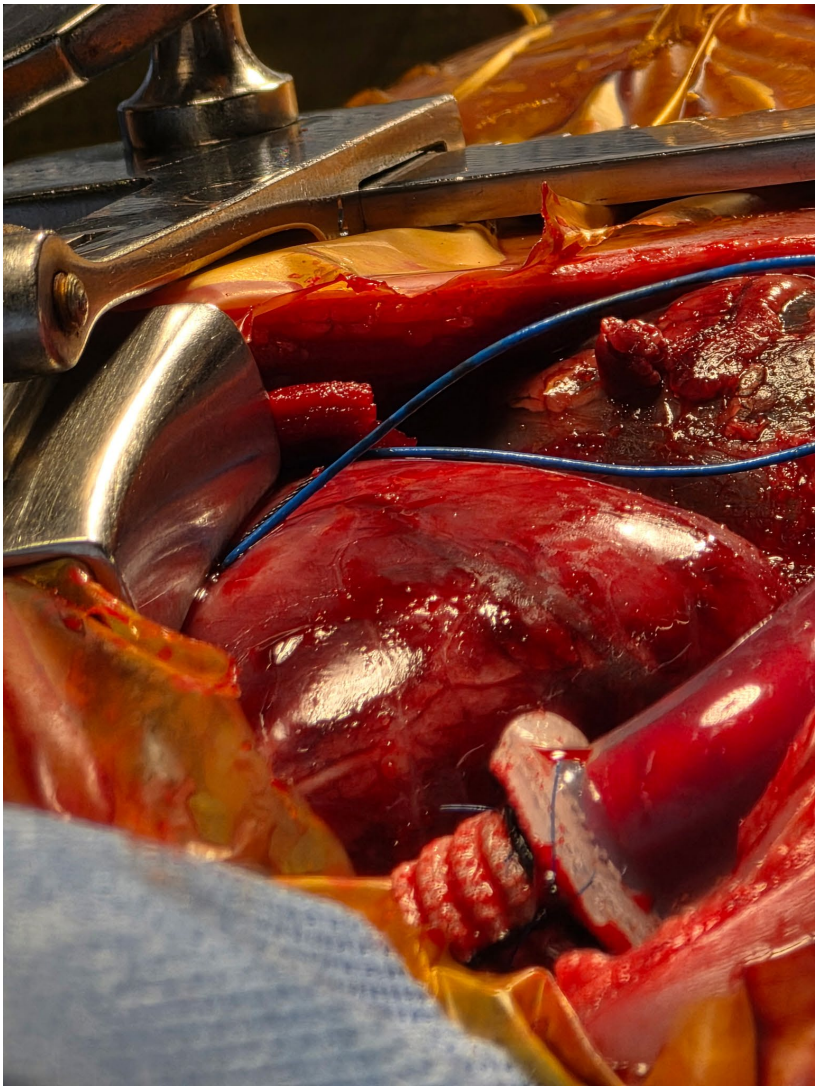
Configuration of Hybrid VAD for HLHS using application of bilateral pulmonary artery bands, stent placement in the patent arterial duct, atrial septectomy if needed, and Berlin Heart VAD insertion. VAD, Ventricular assist device; PA, pulmonary artery.



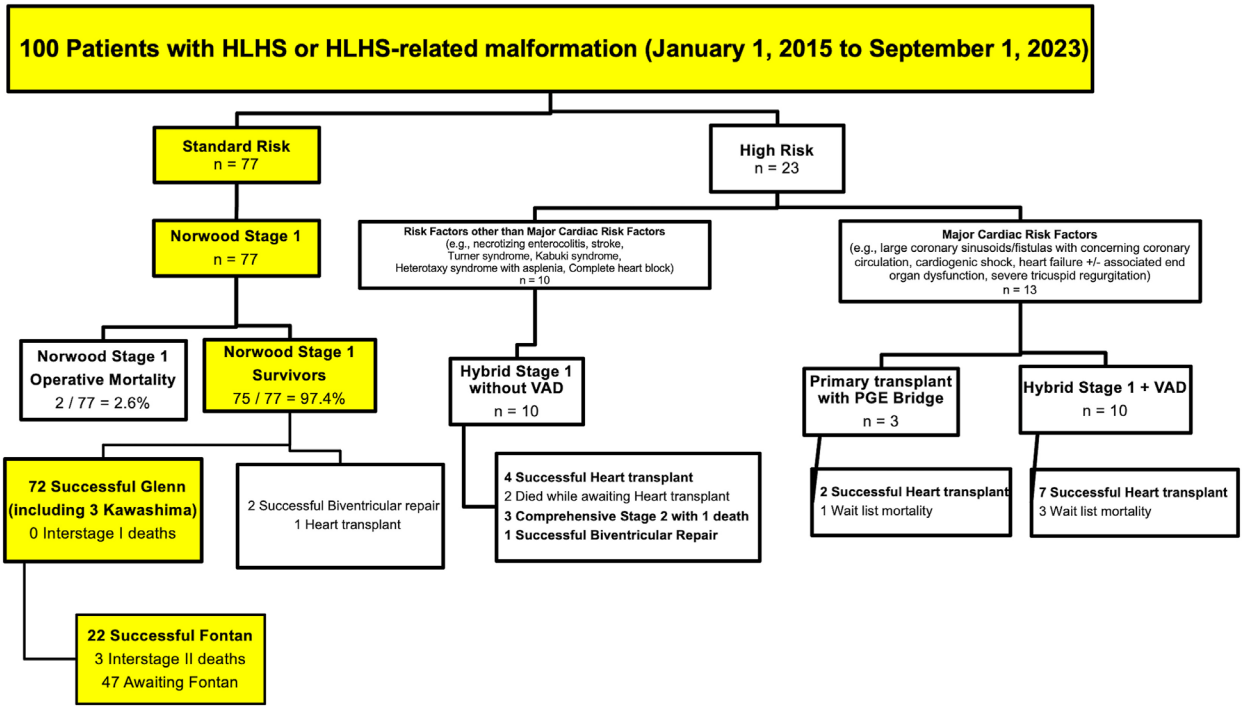
Surgical tool indicating absence of ascending aorta



Surgical tool at the top of the picture indicating coronary cameral fistula



Heart appearing grossly distended with areas of ischemia



HLHS approach algorithm and outcomes from University of Florida Jan 2025

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Mark Steven Bleiweis, Jennifer Co-Vu, Joseph Philip, James C. Fudge, Himesh V. Vyas, Andrew D. Pitkin, Gregory M. Janelle, Kevin J. Sullivan, Curt J. DeGroff, Dipankar Gupta, John-Anthony Coppola, Biagio “Bill” A. Pietra, Frederick Jay Fricker, Susana C. Cruz Beltrán, Giles J. Peek, Jeffrey Phillip Jacobs, Comprehensive Approach to the Management of Patients With Hypoplastic Left Heart Syndrome: Analysis of 100 Consecutive Neonates, The Annals of Thoracic Surgery, Volume 119, Issue 1, 2025, Pages 169-177, ISSN 0003-975, https://doi.org/10.1016/j.athoracsur.2024.05.010. (https://www.sciencedirect.com/science/article/pii/S0003497524003874)

#FR – BIS – OP4

Initiation of Cardiopulmonary Bypass in an Awake Patient with an Ascending Aortic Pseudoaneurysm Causing Obstruction of the Pulmonary Artery and Trachea

Presenting Author: Jonathan V. Petrillo, Fellow, University of California San Diego

Introduction

Awake peripheral cannulation for initiation of cardiopulmonary bypass (CPB) is an established strategy to prevent hemodynamic collapse and airway compromise during induction of anesthesia in patients with major mediastinal compression. We describe the use of this technique in a patient with a large ascending aortic pseudoaneurysm causing life-threatening obstruction of the pulmonary artery and trachea.

Case Presentation

A 73-year-old woman with a history of DeBakey I aortic dissection repair presented with back pain and was found to have proximal graft dehiscence with an 11.2 × 9.6 cm ascending aortic pseudoaneurysm compressing the main pulmonary artery and trachea. She was hemodynamically stable but was noted to have cold agglutinins, prompting initiation of anti-impulse therapy and plasma exchange. The night before the planned repair, she developed worsening hypoxemia.Given the high risk of cardiovascular and airway collapse with induction, the multidisciplinary team elected to perform awake peripheral CPB cannulation. The patient was unable to tolerate the supine position, so cannulation was performed in reverse Trendelenburg using minimal sedation and generous local anesthesia. Femoral arterial and venous access were obtained successfully, with TTE providing real-time confirmation of guidewire and cannula positioning. CPB was initiated prior to anesthetic induction, after which endotracheal intubation was performed without complication. TEE revealed a large ascending aortic pseudoaneurysm with active extravasation compressing the main pulmonary artery. Bronchoscopy showed complete collapse of the left mainstem bronchus and partial collapse of the right. During mediastinal dissection, the pseudoaneurysm ruptured. Reduced venous return necessitated rapid transfusion and a brief period of circulatory arrest to facilitate aortic clamping and restoration of flow. The remainder of the repair proceeded uneventfully. The patient separated from CPB without difficulty and was extubated on postoperative day one.

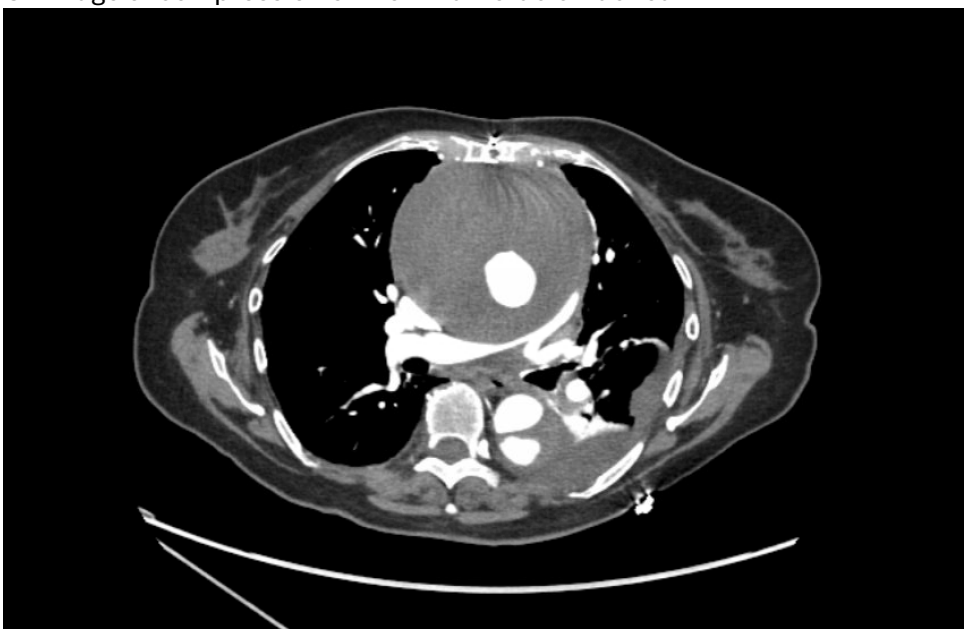
Discussion

This case highlights the effectiveness of awake peripheral CPB cannulation in mitigating the risk of hemodynamic and airway collapse in patients with severe mediastinal compression from complex aortic pathology. TTE provided rapid, reliable confirmation of cannula positioning when patient condition and positioning limited the feasibility of TEE. Early CPB initiation facilitated controlled induction, early cooling, and preparation for circulatory arrest. The favorable outcome underscores the importance of multidisciplinary planning, echocardiographic guidance, and coordinated anesthesia and surgical execution when managing patients with unstable mediastinal and aortic pathology.

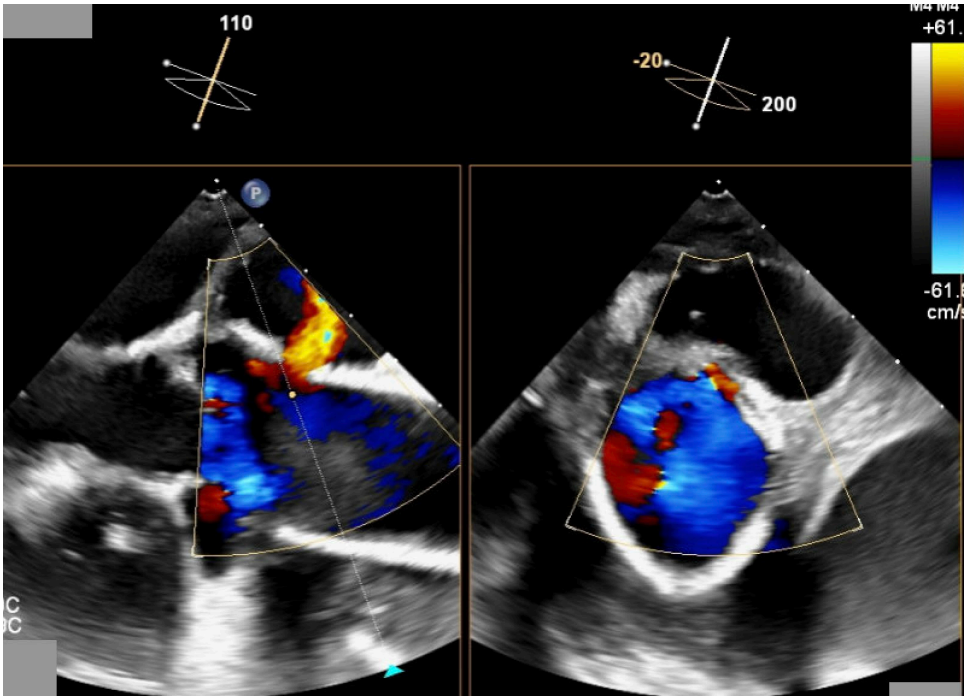
Images



CT image of compression of the intrathoracic trachea



CT image of compression of the pulmonary artery



TEE image of dehiscence of prior aortic repair

References

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#FR – BIS – OP5

Anesthesia for Heart-After-Liver Transplantation and Protection of the Cardiac Graft in a Sensitized Patient

Presenting Author: Shahin Mortazavi, MD, Fellow Adult Cardiothoracic Anesthesiology, University of Washington

Introduction

Since the first combined heart and liver transplantation (CHLT) was performed in 1984, approximately 719 patients have undergone this rare and technically complex procedure. Over time, CHLT has evolved beyond its initial indications of end-stage cardiac and liver failure or familial amyloidosis, with expanding utilization driven by improved surgical techniques and patient selection strategies. An important part of the evaluation of potential heart transplant recipients is the recognition and measurement of the degree of sensitization with calculated panel-reactive antibody (CPRA). Sensitized patients with higher CPRA values (>50%) have an associated increased risk for removal or death on the waitlist, and post transplantation rejection and survival[1]. CHLT may reduce alloimmune-mediated injury to the heart allograft, as the liver from the same donor can absorb donor-specific lymphocytotoxic alloantibodies. In this context, heart-after-liver transplantation (HALT) approach has emerged as a potential strategy to facilitate hepatic absorption of alloantibodies before the heart is implanted in highly sensitized patients requiring a heart transplant[2].

Case Presentation

We report a 38-year-old female with a history of postpartum non-ischemic cardiomyopathy, severe ventricular dysfunction, and CRT-D implantation. Despite optimal medical therapy and two Mitra-clip procedures for severe mitral regurgitation, she developed recurrent ventricular tachycardia (VT). During VT ablation at an outside hospital she experienced VT arrest requiring veno-arterial extracorporeal membrane oxygenation (VA ECMO) and Impella support. She was transferred to the University of Washington Medical Center for a heart transplant evaluation. She was found to have a CPRA of 100%. Given her very high sensitization, she was referred for a HALT. Institutional clinical HALT protocol were followed for desensitization prior to transfer to the operating room with plasmapheresis, administration of eculizumab, methylprednisolone, and rabbit anti-thymocyte globulin at critical stages of the procedure. A combined sternotomy and subcostal incision was made for cardiac dissection and hepatectomy. The explanted liver was utilized for a domino transplantation at our institution. Donor liver implantation proceeded without complication. The orthotopic heart transplant proceeded with recipient cardiectomy and heart implantation in standard fashion with bicaval anastomoses. The patient returned to the OR on post operative day (POD) 2 for bile duct anastomosis. She was extubated on POD 3 with an uneventful ICU course.

Discussion

Combined heart and liver transplantation (CHLT) is most commonly performed as a sequential procedure, with the heart transplanted first due to its limited tolerance for cold ischemia[3]. In contrast, the heart-after-liver transplantation (HALT) approach reverses this order, offering a significant reduction in cardiac allograft rejection risk in highly sensitized patients. However, HALT carries potential drawbacks, including prolonged

cardiac ischemic time and increased reported mortality. As experience with CHLT continues to grow, we present a case highlighting the unique perioperative anesthetic and surgical considerations of performing CHLT in reversed order.

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#FR – BIS – OP6

The Rickety Heparin Bridge: Intraoperative Discovery and Real-Time Embolization of Mechanical Mitral Valve Thrombosis

Presenting Author: Nino Miletic, MD, Mount Sinai Morningside/West

Introduction

Mechanical valve thrombosis (MVT) is a life-threatening complication that most commonly occurs during periods of interrupted or subtherapeutic anticoagulation. Patients with mechanical valves typically require lifelong warfarin with an INR goal of 2.5–3.5. Prior to major surgeries, warfarin is stopped to minimize the risk of catastrophic bleeding. Heparin bridging is controversial but is recommended for high-risk patients. Despite appropriate protocols, the perioperative period remains high risk for MVT formation.

Case Presentation

A 59-year-old woman with a history of atrial fibrillation, rheumatic mitral valve disease status post repair (1993) and mechanical mitral valve replacement (2008), non-ischemic cardiomyopathy, severe aortic stenosis status post transcatheter aortic valve replacement (2025), severe tricuspid regurgitation, and moderate pulmonary hypertension was admitted for optimization before a planned triple re-do sternotomy and tricuspid valve replacement. She was diuresed, started on inodilators, and transitioned from warfarin to an unfractionated heparin infusion (goal PTT 90–100 seconds). Following the induction of anesthesia, pre-incision transesophageal echocardiography (TEE) unexpectedly demonstrated new-onset mechanical mitral stenosis. The lateral leaflet was immobile – a marked change from a TEE performed one month earlier – and highly suspicious for acute thrombotic obstruction (Figure 1). The procedure was aborted and thrombolytic therapy was initiated in the intensive care unit. The patient received low-dose alteplase at 1 mg/hr for 24 hours, and repeat TEE confirmed a thrombus on the ventricular aspect of the lateral leaflet. During real-time imaging, the affected valve leaflet suddenly regained full mobility, and the thrombus was visualized abruptly dislodging and embolizing (Figure 2). The valve then demonstrated normal excursion (Figure 3). The patient emerged from anesthesia neurologically intact and without evidence of systemic embolic injury. She was then transitioned back to therapeutic warfarin, monitored for five days, and discharged home with plans for future intervention.

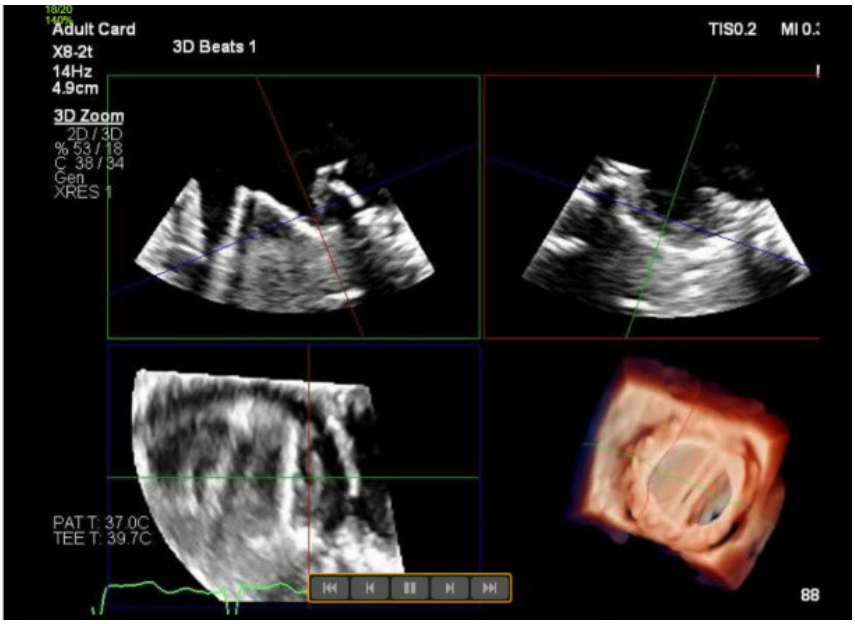
Discussion

MVT occurs in 0.1–5.7% of mechanical valve patients, with bileaflet valves carrying lower risk than older designs [1]. Thrombosis most commonly arises during subtherapeutic anticoagulation or perioperative bridging. Heparin bridging remains controversial and is generally reserved for high-risk patients [2]. Despite achieving a therapeutic PTT (1.5 to 2 times patient's baseline), this patient developed MVT, highlighting the persistent vulnerability of mechanical valves to interruptions in anticoagulation and the need for improved strategies to prevent perioperative thrombosis. To treat MVTs, the PROMETEE trial supports low-dose, slow-infusion tissue plasminogen activator (25 mg over 25 hours), showing high success rates without excess bleeding [3]. However, guidance on post-thrombolysis monitoring is limited [1]. Embolic events may be transient and easily overlooked; in our case, real-time TEE captured embolization that could have been

mistaken for benign restoration of leaflet mobility. Therefore, more evidence is needed to define optimal imaging, neurologic assessment, and duration of surveillance following thrombolytic therapy for MVT.

Images

A



[*NB* Images will be optimized for poster format] Figure 1. Initial findings across mechanical mitral valve on pre-incision TEE exam. (A) 3D TEE mid-esophageal mitral commissural view across the stuck lateral mitral valve leaflet with no apparent thrombus on the atrial aspect of the leaflet.

B

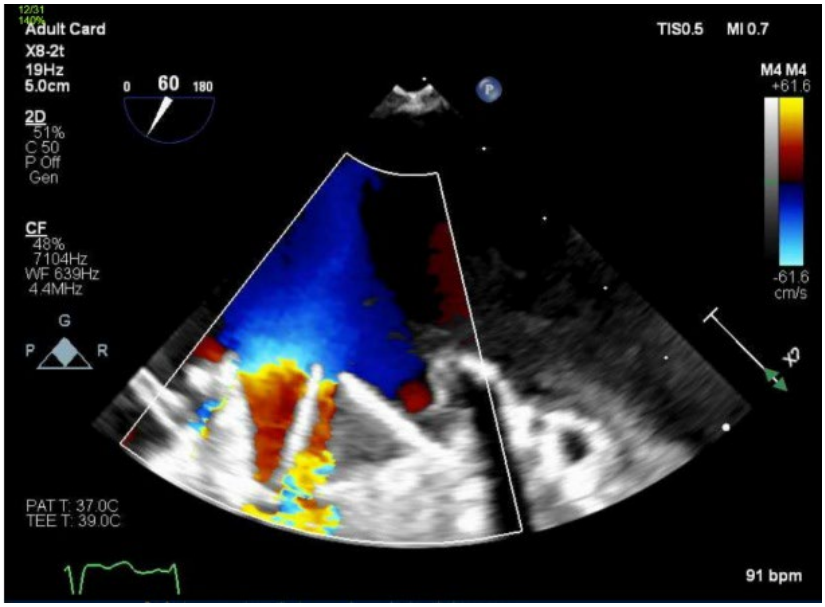


Figure 1 continued. (B) Color doppler during ventricular filling across the mechanical mitral valve.

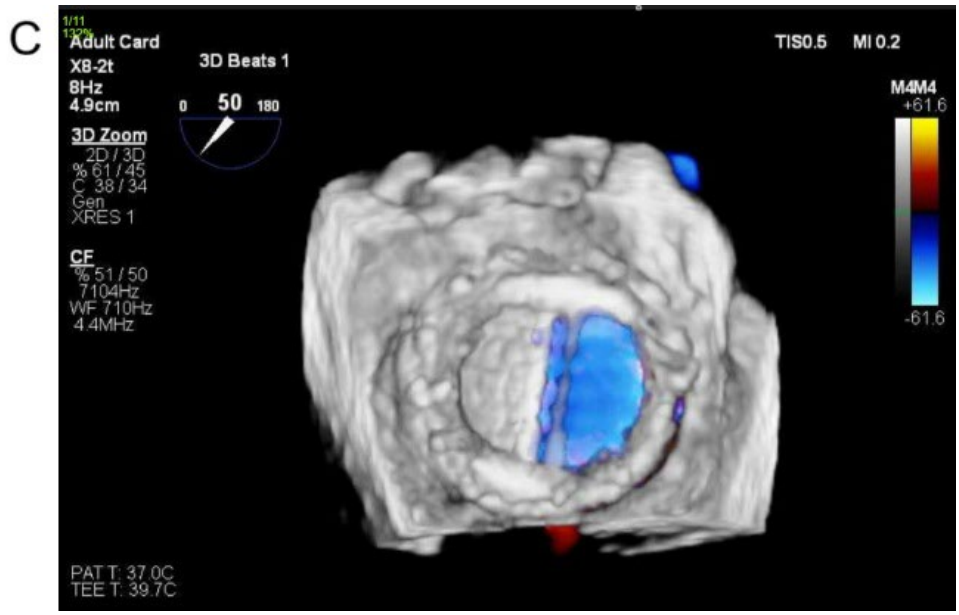


Figure 1 continued. (C) 3D color doppler “en face” view across the mechanical mitral valve with stuck lateral leaflet.

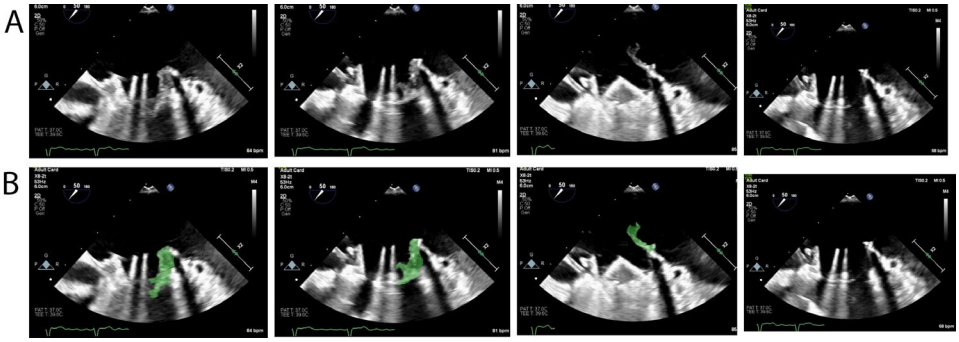


Figure 2. TEE images across the mechanical mitral valve after it suddenly regained mobility. Thrombus became readily visible from the ventricular aspect of the valve. (A) Still captures in series as the thrombus became more apparent before embolizing. (B) Still captures enhanced to show thrombus in green overlay.

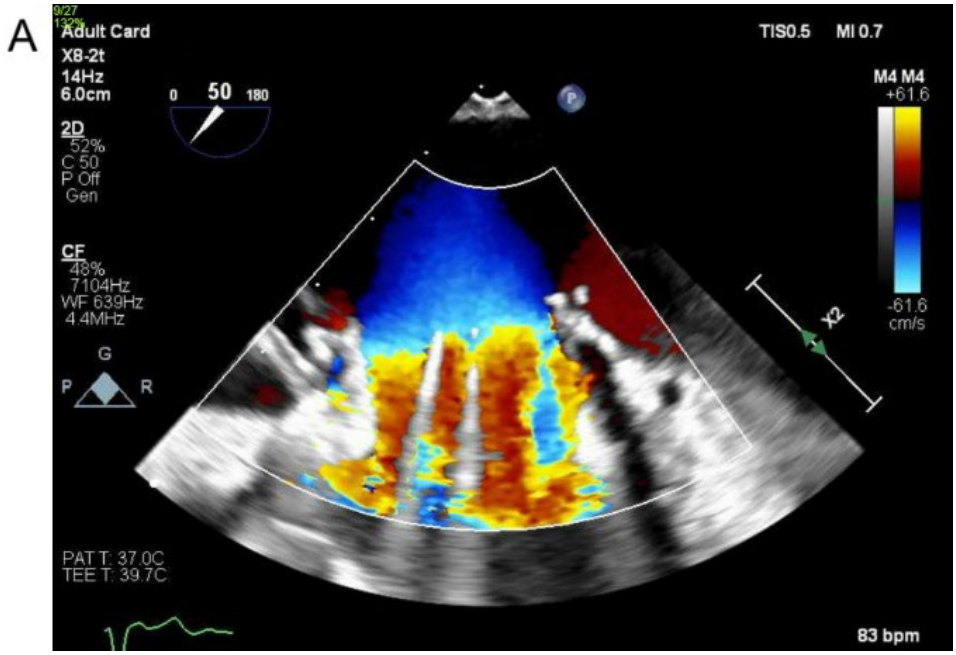


Figure 3. TEE images across the mechanical mitral valve after witness return of bilateral leaflet excursion and thromboembolism. (A) Doppler image showing resolution of mitral stenosis and bilateral leaflet mobility.

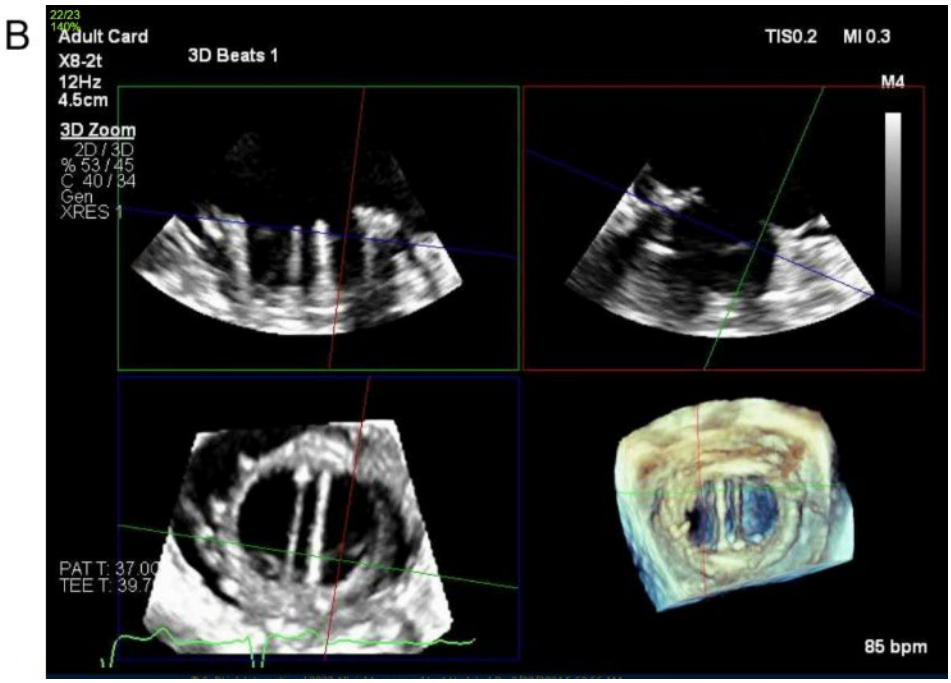


Figure 3 continued. (B) 3D TEE images of the mitral valve showing both leaflets fully opening after thromboembolism witnessed in real-time.


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FELLOW AND RESIDENT ABSTRACTS

Poster Session 1

Friday, April 24, 2026 | 1:30 PM – 2:30 PM

#FR 1 – Kiosk 1 – Poster 1

Quadruple Valve Replacement in the Setting of Carcinoid Heart Disease

Presenting Author: Lydia Duvall, DO, ACTA Fellow, Duke University

Introduction

Left sided valvular disease in the setting of carcinoid heart disease is a rare finding and is likely caused by pulmonary metastasis or a patent foramen ovale[1] Few case reports document left sided valve involvement due to the intrinsic breakdown of serotonin by the pulmonary system,[1] with even fewer reports documenting quadruple valve replacement during a single procedure.

Case Presentation

A 70-year-old male with a known history of carcinoid disease presented to an outside hospital with abdominal pain and increased loose stools. Imaging revealed multiple hepatic lesions, and subsequent pathology confirmed metastatic neuroendocrine tumor. Transthoracic echocardiography demonstrated severe regurgitation of all four valves as well as a patent foramen ovale. The cardiology team initially pursued medical management with diuresis, after which the patient was discharged. Shortly after discharge, the patient developed worsening dyspnea and returned to the hospital with an oxygen saturation of 80% on room air. Given the progression of his symptoms and the severity of his valvular pathology, the multidisciplinary team proceeded with plans for surgical intervention involving quadruple valve replacement.

Discussion

Echocardiography played a central role in the preoperative and intraoperative management of this patient, guiding the decision to proceed with quadruple valve replacement in the setting of carcinoid heart disease. Comprehensive transthoracic and transesophageal imaging demonstrated severe regurgitation of all four cardiac valves. Multiple transesophageal echocardiographic views were essential for evaluating valvular pathology, assessing prosthetic valve position and function, as well as to evaluate for residual intracardiac shunting following patent foramen ovale closure. This case highlights several important teaching points: carcinoid heart disease typically affects right-sided structures because serotonin is metabolized in the lungs and left-sided involvement should prompt investigation for intracardiac shunts or pulmonary metastases. Additionally, this case underscores the challenge of interpreting prosthetic valve function in multivalve replacement surgeries, further emphasizing the important role of high-quality intraoperative transesophageal echocardiography.

Images

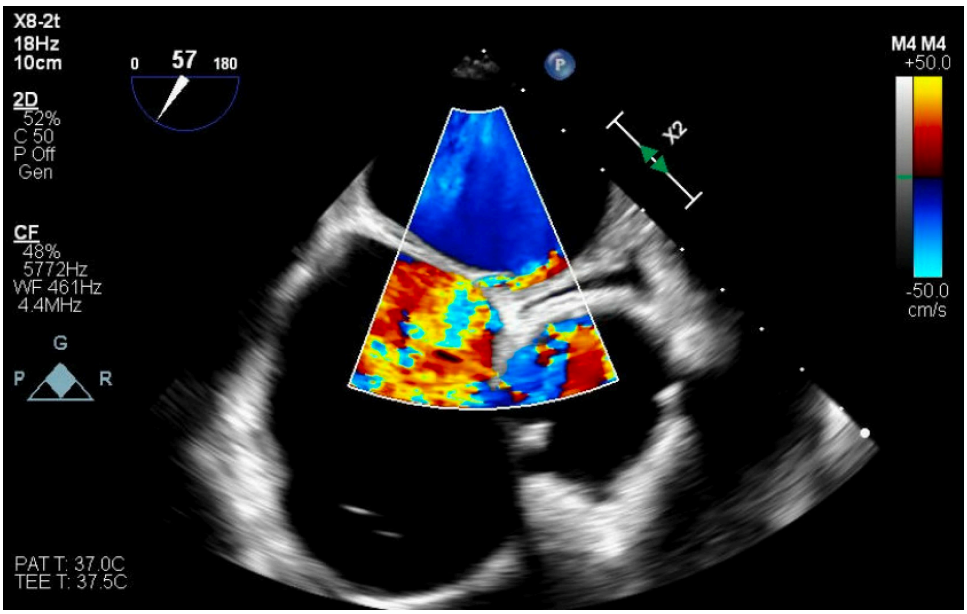


Figure 1: Mid-esophageal bicaval view demonstrating patent foramen ovale

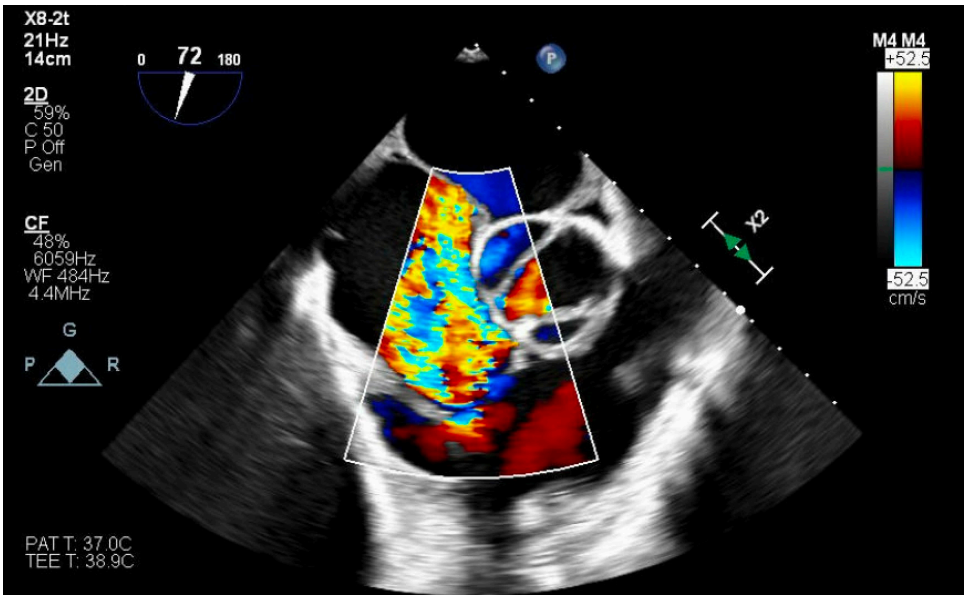


Figure 2: Mid-esophageal right ventricle inflow-outflow view depicting massive tricuspid regurgitation

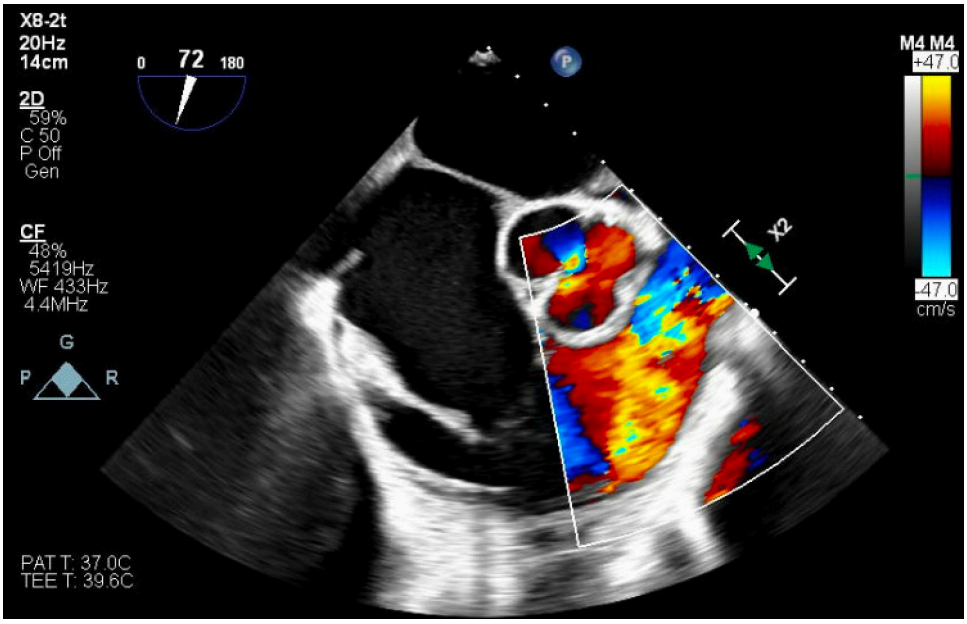


Figure 3: Mid-esophageal right ventricle inflow-outflow view depicting severe pulmonic insufficiency

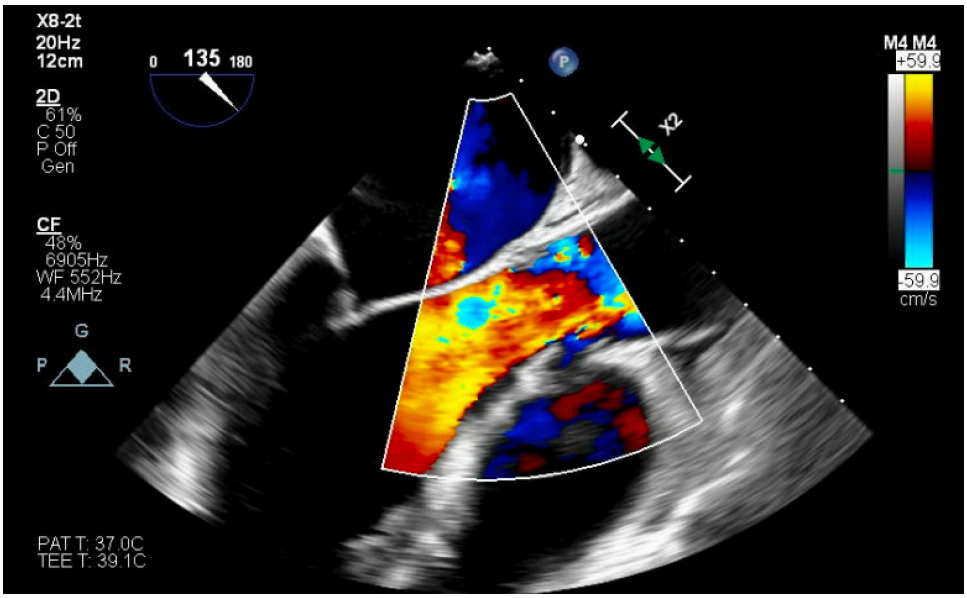


Figure 4: Mid-esophageal aortic valve long axis view demonstrating severe aortic insufficiency

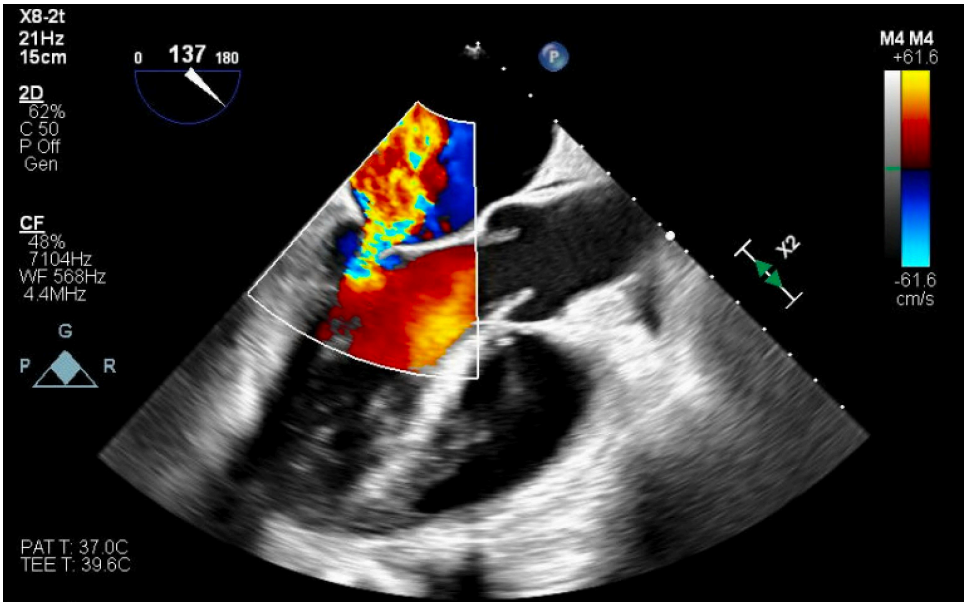


Figure 5: Mid-esophageal aortic long axis view with severe mitral regurgitation

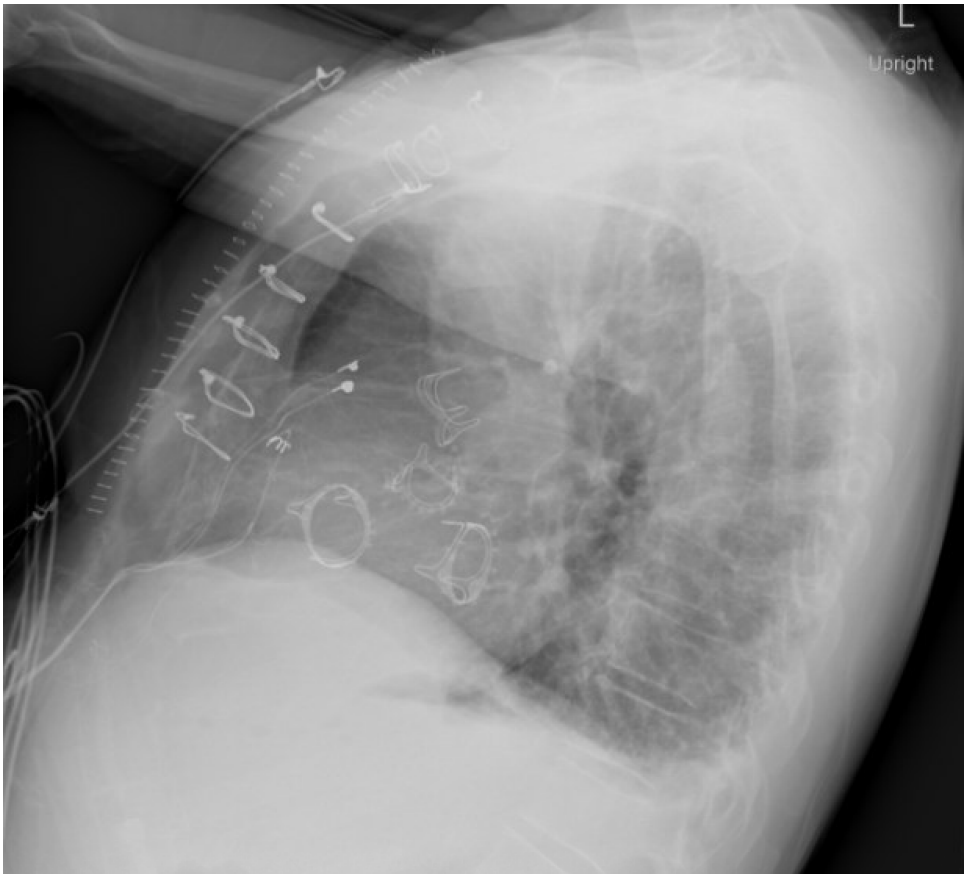


Figure 6: Post operative lateral chest x-ray visualizing four bioprosthetic valves

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#FR 1 – Kiosk 1 – Poster 2

Newly Diagnosed Congenital Heart Disease and Pulmonary Hypertension in Pregnancy

Presenting Author: Lilly Fang, MD, University of Texas Southwestern

Introduction

Pregnancy poses cardiopulmonary changes that have serious implications in patients with congenital heart disease. It is important to consider the patient's individual anatomy and physiology in the context of these changes. This requires a multidisciplinary team discussion to identify hemodynamic goals and create a safe delivery plan.

Case Presentation

A 19-year-old G1 female presented at 21 weeks for prenatal care. She reported no past medical history but was noted to have a prominent holosystolic murmur. This was initially attributed to be a physiologic flow murmur of pregnancy, but a transthoracic echo done at 31 weeks showed a large unrestricted ventricular septal defect (VSD) with an overriding aorta and signs of right ventricular pressure overload. At 34 weeks, she presented with shortness of breath and was admitted for further workup and delivery planning. Right heart catheterization showed severe pulmonary hypertension (PH) with mean PA pressure 61mmHg and left to right shunt with estimated Qp:Qs of 2.4. A multidisciplinary discussion with maternal-fetal medicine, cardiac anesthesiology, cardiology, and pulmonology decided to initiate intravenous epoprostenol to optimize her pulmonary vascular resistance, then deliver via primary cesarean section under neuraxial anesthesia. Femoral sheaths were placed to facilitate prompt initiation of VA-ECMO in case of decompensation. She had an arterial line and epidural placed without complication. She was started on epinephrine, vasopressin, and nitric oxide for inotropy, blood pressure support, and PVR reduction respectively. She then underwent an uncomplicated cesarean section and subsequent post-operative recovery where she was weaned off epoprostenol, started on long-term macitentan and diuretic therapy, and discharged on post-partum day 9.

Discussion

With the advancement of management options for congenital heart disease, life expectancy is increasing, leading to new challenges such as pregnancy. Patients with PH are poorly tolerant of the physiologic changes of pregnancy including increased circulatory volume and are at extremely high risk of cardiopulmonary complications. Additionally, in the presence of a large left to right shunt, decreases in systemic vascular resistance or increases in pulmonary vascular resistance can reverse the shunt and cause hypoxia, pulmonary vasoconstriction, and right heart failure, which is known as Eisenmenger syndrome, and is associated with 30-50% risk of death. In particular, the intra- and immediate post-partum periods carry the highest risk due to rapid hemodynamic changes and fluid shifts. A multidisciplinary team of experts including maternal-fetal medicine, anesthesiology, and cardiology is critical in creating a delivery plan to mitigate these risks. The optimal mode of delivery is controversial. Cesarean section is more controlled but carries more hemodynamic shifts, while prolonged labor and Valsalva maneuvers in vaginal

delivery can precipitate cardiovascular collapse. Regardless of delivery method, epidural anesthesia is preferred but requires slow uptitration to avoid sudden drops in SVR. Advanced therapies such as prostacyclin analogues, sildenafil, and nitric oxide can help optimize PVR to prevent right heart failure and shunt reversal.

Images

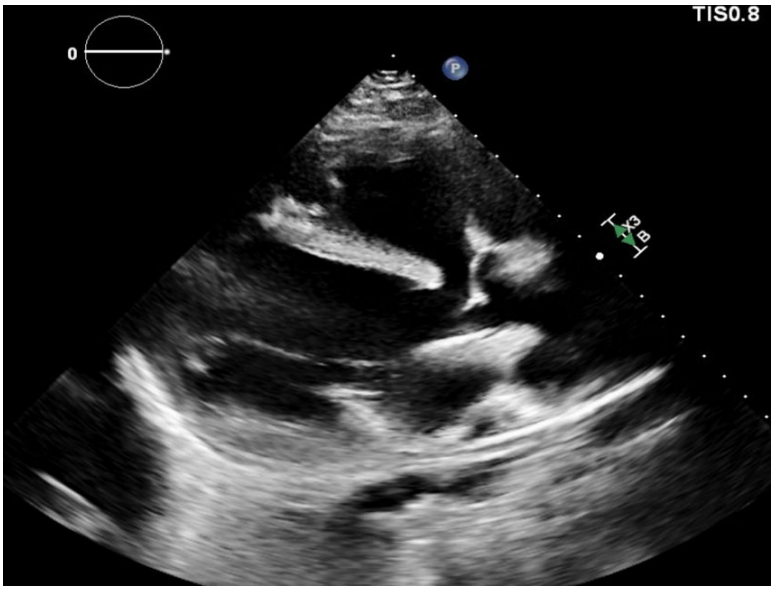


Figure 1. Snapshot of the patient's transthoracic echo with large perimembranous VSD and overriding aorta. The RV is hypertrophic and dilated, suggesting pressure and volume overload.

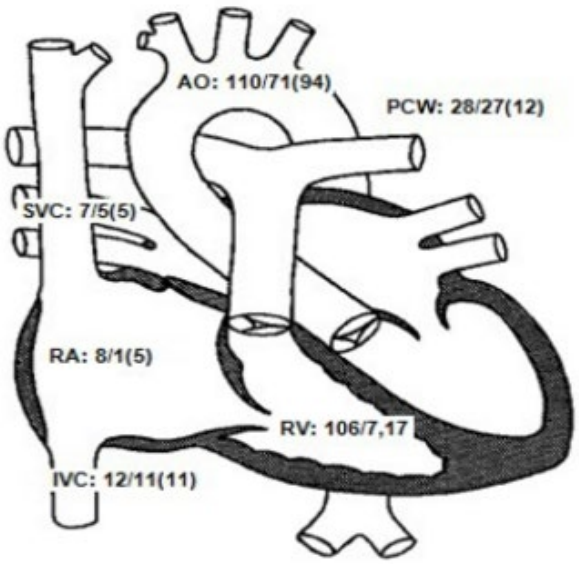


Figure 2. Pressures obtained from the patient's right heart catheterization, which was significant for systemic RV pressures and mean PAP of 61mmHg, indicating severe pulmonary hypertension

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#FR 1 – Kiosk 1 – Poster 3

Cardiac Arrest Due to CO2 Embolism During Laparoscopic Vein Harvesting

Presenting Author: Jake Blum, MD, Johns Hopkins Hospital

Introduction

Carbon Dioxide (CO2) embolism is a relatively common and possibly under-recognized phenomenon during endoscopic saphenous vein harvesting for coronary artery bypass grafting (CABG). CO2 emboli can range from a subclinical incidental finding on transesophageal echocardiography (TEE) to a cause of cardiogenic shock. This abstract describes a case during which CO2 embolism led to hemodynamic collapse which was promptly recognized and treated.

Case Presentation

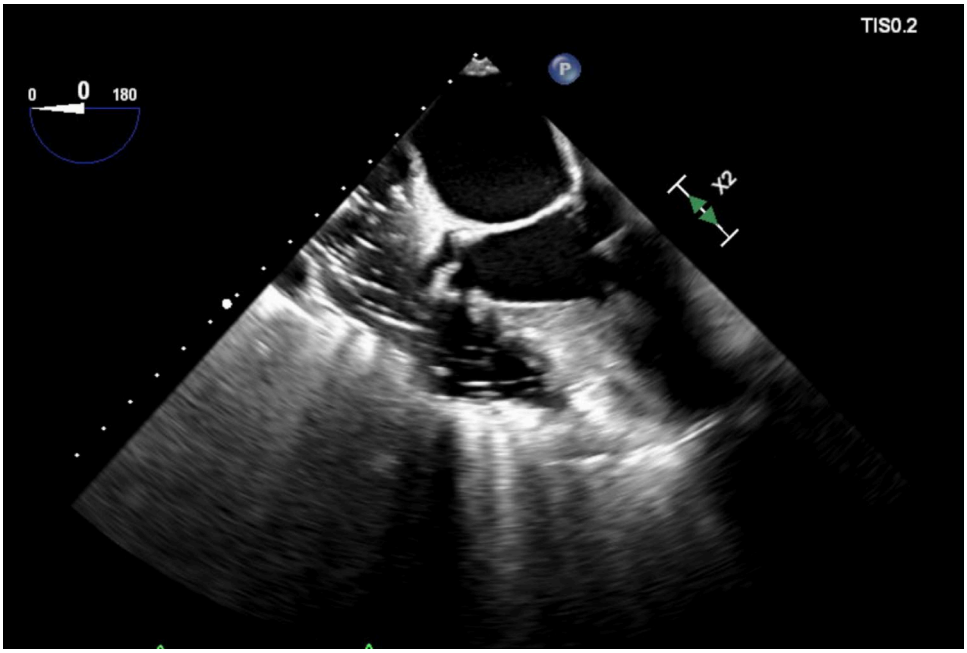
A 66 year-old female with a past medical history of hypertension, chronic pericardial effusion, end-stage renal disease, anemia, and severe aortic stenosis (AS) was referred for aortic valve replacement. Her preoperative workup was notable for 90% mid-RCA stenosis. She was scheduled for mechanical aortic valve replacement and CABG x1 with endoscopic graft harvesting from the right saphenous vein. On the day of surgery, induction of anesthesia, arterial line and central venous line placement took place uneventfully. Preoperative TEE demonstrated normal biventricular function, grade 1 diastolic dysfunction, and severe AS with a dimensionless index of 0.11. No patent foramen ovale was detected via color flow Doppler. The patient tolerated maintenance of general anesthesia, sternotomy, and CO2 insufflation of the right lower extremity (RLE) for saphenous vein harvesting. Shortly following the administration of heparin to prepare for cardiopulmonary bypass (CPB), sudden, profound hypotension was noted as the patient's blood pressure rapidly decreased from 130/60 mmHg to 50/30 mmHg. Cardiac massage was then performed by the attending surgeon. Intraoperative TEE showed a significant amount of gas in the right ventricle. All lines were checked for air entrainment and syringes were inspected to assess the possibility of medication error as potential causes. Around this point in the case, the surgical team notified the room that there had been a likely venous injury within the RLE. A perfusing pressure was regained after roughly one minute of cardiac massage, vasopressor support, and fluid administration. It was felt that CO2 gas embolism from the RLE was the most likely cause of the hemodynamic collapse. After stabilization, the right atrium was noted to be distended in the surgical field. Gas bubbles were appreciated from stay suture sites when the patient was cannulated for CPB. A significant amount of gas was liberated from a dual stage venous cannula placed in the right atrium. The patient was successfully initiated on and weaned from CPB after aortic valve replacement and CABG. The remainder of the perioperative course was unremarkable. The patient was discharged on post operative day eight.

Discussion

The incidence of CO2 embolism during endoscopic saphenous vein harvesting involving CO2 insufflation is more than 4%, though the incidence of "massive" CO2 embolism is overall quite rare at 0.5% (1). The prompt recognition and treatment of such events may be critical to patient safety. Prior case reports

involving hemodynamic distrubance associated with endoscopic vein harvesting (2,3) have reported that TEE has beena valuable diagnostic tool in such instances. Our patient may have been particularly susceptible to hemodynamic collapse from gas embolism given her preload dependent cardiac physiology secondary to severe AS.

Images



References

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#FR 1 – Kiosk 1 – Poster 4

Multidisciplinary Perioperative Coordination for Complex Adrenocortical Adenocarcinoma

Presenting Author: Maianna Dematteis, M.D., Resident, University of Rochester Strong Memorial Hospital

Introduction

Multidisciplinary collaboration is crucial for surgically complex cases. When a disease process spans multiple organ systems, the best outcome requires leveraging the expertise from multiple specialties. We describe a case of a right adrenal tumor with associated inferior vena cava (IVC) invasion and peri-aortic metastasis. The patient's pathophysiology necessitated coordination with multiple sub-specialty surgical services and cardiac anesthesiology to plan for cardiopulmonary bypass (CPB) and to optimize management of a complex case with dynamic intra-operative decision-making.

Case Presentation

A 59-year-old woman with history of hypertension and thyroid cancer presented to the emergency room with post-thyroidectomy fevers. Pan-scan CT imaging showed a right adrenal mass with intracaval thrombus extension and a secondary mass compressing the base of the celiac artery axis (Figures 1 & 2). Bloodwork revealed elevated cortisol levels, concerning for adrenocortical carcinoma. Given the rapid tumor progression on surgical imaging, formal cardiac testing was deferred to avoid surgical delay. Intra-operatively, she underwent general anesthesia with post-induction placement of a left femoral central line, rapid infusion catheter, and an arterial line. On transesophageal echo (TEE), the thrombus was supradiaphragmatic but below the right atrium. Under TEE guidance, the surgical team mobilized the liver and created a pericardial window to gain suprahepatic control of the thrombus. To maintain hemodynamic stability on CPB, infusions of norepinephrine and vasopressin were required. The patient ultimately underwent an en bloc nephrectomy, en bloc diaphragmatic resection, and vena cava reconstruction. Estimated blood loss was seven liters, requiring multiple blood products for resuscitation. Post-operatively, she was transferred to the intensive care unit in stable condition and was ultimately discharged home about one month later.

Discussion

Adrenocortical carcinomas are relatively rare tumors that may present with or without invasion of the IVC. While multiple cases have been reported with variations in surgical approach, the extent to which this tumor spread required a more involved anesthetic and surgical approach. Only a few prior studies detail the use of cardiopulmonary bypass, all of which lacked similar degrees of tumor progression and complexity[1]. Likewise, individual reports of adrenal tumors with IVC involvement describe only low-level, noninvasive thrombi without major circulatory threat[2]. Thus, the significant risk for vascular and visceral compromise required extensive preemptive planning and continuous intra-operative discussion to anticipate hemodynamic instability, coagulopathy, and impact on surgical visualization to guide real-time management. Together, these factors necessitated early coordinated involvement of all involved specialties

and a comprehensive anesthetic strategy.

Images



Figure 1. Adrenal mass invading the IVC



Figure 2. peri-aortic lymph node impeding celiac flow from the aorta

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#FR 1 – Kiosk 1 – Poster 5

A Mass-ive Problem: Anesthetic Considerations for Severe Right Ventricular Outflow Tract Obstruction

Presenting Author: Kevin Welch, MD, Resident, University of North Carolina

Introduction

Large anterior mediastinal masses pose considerable anesthetic challenges due to the risk of compressing vital structures. Many affect the airway, esophagus, cardiac chambers, or vasculature. Isolated compression of the right ventricular outflow tract (RVOT) or main pulmonary artery (PA) are extremely rare, and experience with anesthetic management for patients with this pathology is limited to few case reports. We present a case of a patient requiring debulking of a large mediastinal mass compressing the main PA causing severe right ventricular dysfunction.

Case Presentation

A 47 year-old female presented to the hospital with back pain, dyspnea, weight loss, and a new systolic murmur. Work-up revealed a 9.5cm anterior mediastinal mass found to be compressing the RVOT and PA (Figure 1). She was scheduled for tumor debulking via sternotomy. A preoperative transthoracic echocardiogram (TTE) revealed a severely dilated right ventricle and severe right ventricular dysfunction. Prior to induction, a radial arterial line and internal jugular central venous catheter were placed. Additionally, the surgical team obtained femoral arterial and venous access in the event that emergent VA ECMO initiation was required. Soon after sternotomy, she developed significant hemodynamic instability despite maximal pharmacologic support including norepinephrine, epinephrine, vasopressin, dobutamine and inhaled nitric oxide. Debulking was attempted, however the mass was found to invade the pulmonary artery, requiring the procedure to be aborted. Intraoperative transesophageal echocardiogram (TEE) revealed severe RV dilation and hypokinesis, and a mean gradient across the obstruction of 111mmHg consistent with severe stenosis of the PA (Figure 2). Given the significant pharmacologic support required and evidence of RV failure on TEE, the patient was cannulated for central VA ECMO before transport to the ICU. She underwent palliative radiotherapy and eventual ECMO decannulation.

Discussion

Anesthesiologists caring for patients with mediastinal masses should be prepared for the potential of cardiovascular collapse and fixed RVOT obstruction—a rarely encountered pathology outside of congenital heart defects. Due to high risk of right ventricular failure, preparing for mechanical circulatory support and astute intraoperative monitoring is imperative. Use of intraoperative TEE and PA catheter data are excellent ways to quickly diagnose and manage right ventricular failure. In this case, a PA catheter was unable able to be successfully placed due to severe PA stenosis. Hallmarks of hemodynamic management for these cases should include optimizing right ventricular preload with fluid if needed, augmenting contractility with inotropes, and decreasing RV afterload with pulmonary vasodilators[1,2,3]. However, in this case, the potential benefit of pulmonary vasodilators given the fixed and proximal nature of this obstruction is unclear. Despite

maintaining optimal hemodynamics, this case illustrates the difficulty in anesthetic management of fixed RVOT obstruction, for which there are no standard guidelines.

Images

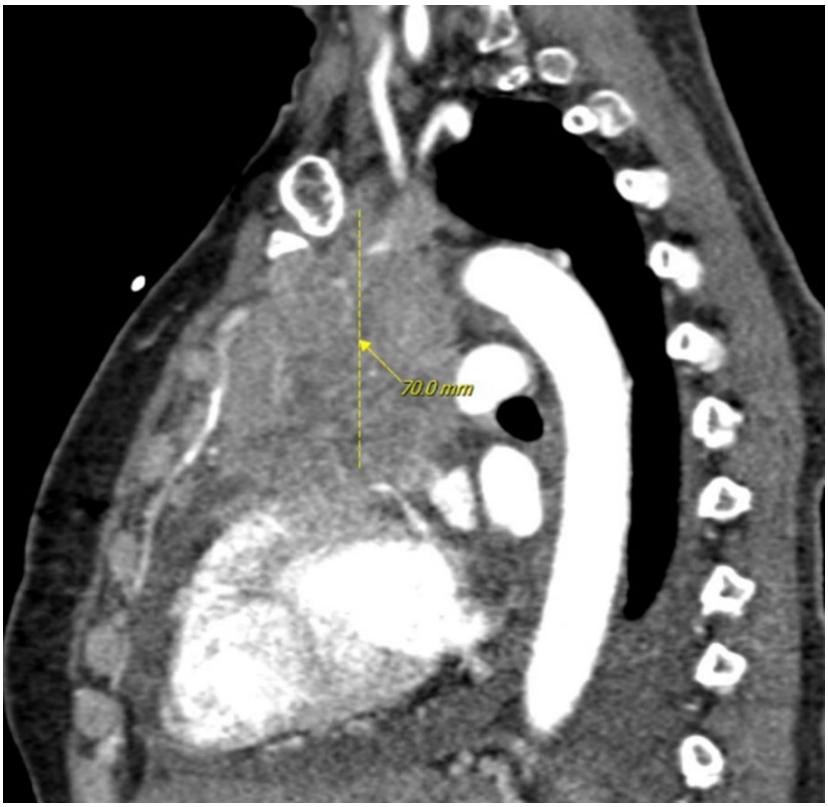


Figure 1A: Sagittal CT visualization of large anterior mediastinal mass with invasion of great vessels

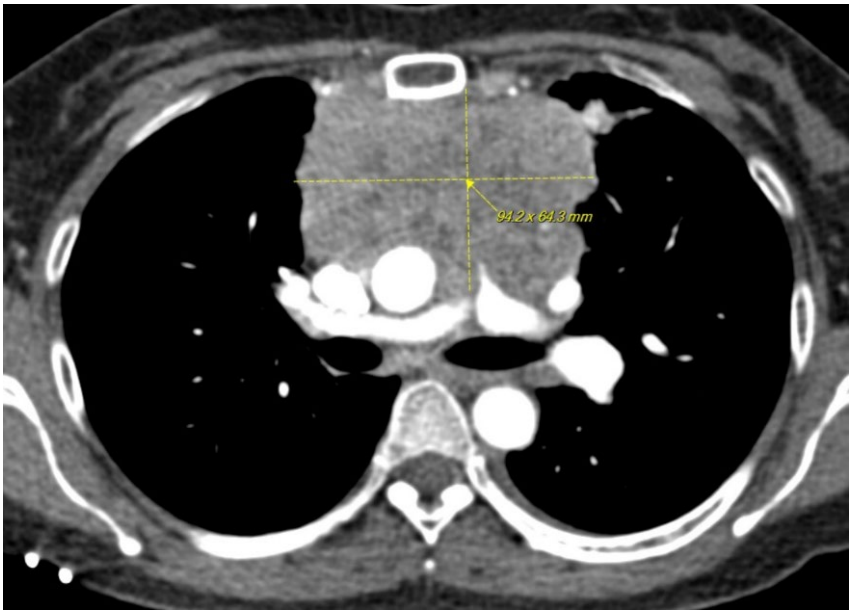


Figure 1B: Transverse CT visualization of large anterior mediastinal mass with invasion of great vessels

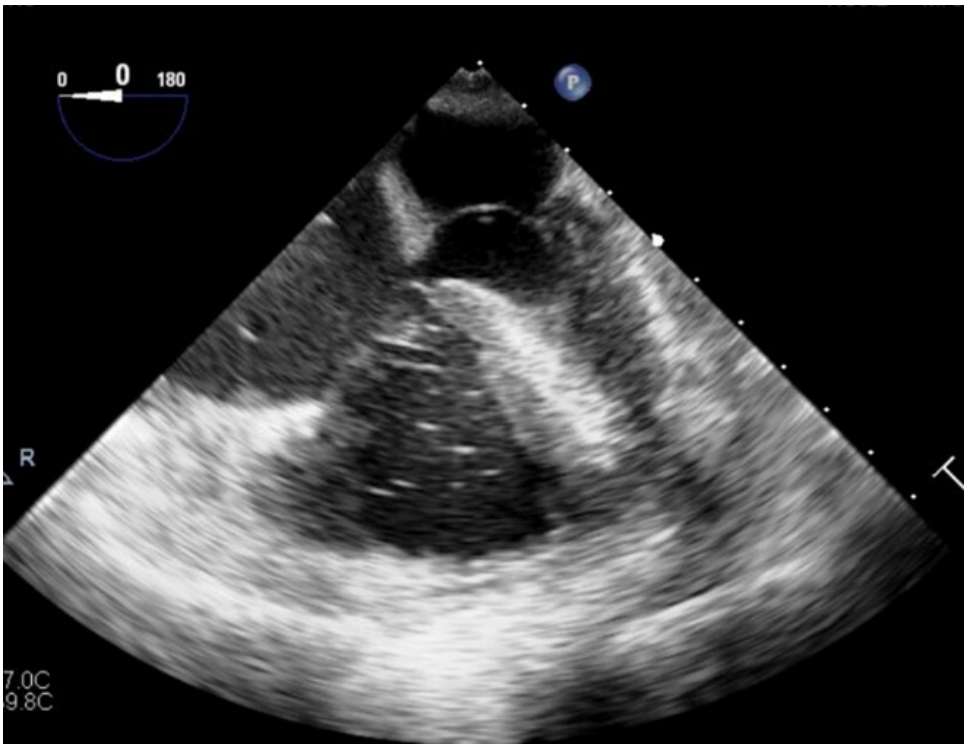


Figure 2A: TEE ME4C showing intraoperative RV failure

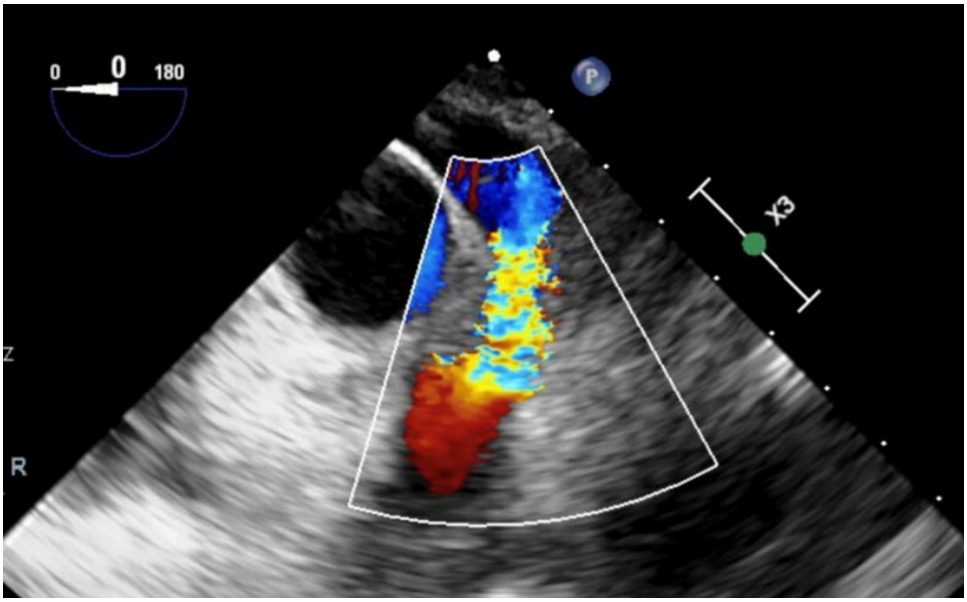


Figure 2B: TEE upper esophageal aorta short axis with color doppler showing intraoperative PA obstruction

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#FR 1 – Kiosk 1 – Poster 6

Non-Cardiac Surgery in a Patient with Carcinoid Heart Disease Complicated by Intraoperative Carcinoid Crisis

Presenting Author: Arjun Johal, Resident, University of California, San Francisco

Introduction

Neuroendocrine tumors may release vasoactive substances that produce carcinoid syndrome in 30–50% of patients, manifesting as flushing, diarrhea, bronchospasm, and in 20–30% —carcinoid heart disease (CHD) secondary to serotonin-mediated fibrotic plaque deposition on right-sided valves, causing severe valvular dysfunction and progressive heart failure. There is risk for carcinoid crisis from perioperative stress and managing this hemodynamic lability in a patient with carcinoid heart disease can be challenging. We present the management of a patient with metastatic carcinoid syndrome and advanced CHD undergoing external fixation of a femur fracture.

Case Presentation

A 74-year-old male with metastatic and long-standing carcinoid syndrome presented with a femoral neck fracture three days after a fall. His history included persistent diarrhea, intermittent flushing, and CHD with torrential tricuspid regurgitation, severe pulmonic regurgitation, moderate mitral regurgitation, severe bi-atrial enlargement, mild pulmonary hypertension, and atrial fibrillation on apixaban. He is on long-acting octreotide every 21 days; his last dose was two weeks prior. Fixation was performed under general anesthesia. In the preoperative area, he received midazolam, diphenhydramine, ondansetron. Induction was achieved slowly with fentanyl and propofol after 100 mcg octreotide bolus and initiation of an octreotide infusion at 100 mcg/hr. Following induction and intermittently intraoperatively, he experienced hypotension that responded poorly to phenylephrine and vasopressin boluses but improved with octreotide boluses. He required high-dose vasopressors throughout the case and his octreotide infusion was titrated up to 200 mcg/hr. Analgesia included long-acting opioids. By the end of surgery his blood pressure normalized and all vasopressors were weaned off. The octreotide infusion was reduced to 100 mcg/hr. He was extubated in the OR, and transferred to the ICU for close hemodynamic monitoring. The octreotide infusion was discontinued 12 hours later, and postoperative pain was controlled with long-acting opioids without further hemodynamic instability. He was discharged home 2 days later.

Discussion

This case illustrates the complexity of anesthesia for a patient with severe carcinoid heart disease undergoing non-cardiac surgery. The main goal is preventing carcinoid crisis by minimizing perioperative stressors and avoiding triggering drugs, including histamine-releasing opioids and beta-agonists. Phenylephrine and vasopressin are preferred vasopressors. Management includes preoperative anxiolysis and octreotide infusion, even though many patients are already on long-acting somatostatin analogs that may lower crisis risk. Regional anesthesia is an excellent strategy; surgical anesthesia using epidural or intrathecal catheters allows controlled sympathectomy, stable hemodynamics, and superior analgesia,

thereby lowering crisis risk in a hemodynamically labile patient with carcinoid heart disease but was avoided here due to recent DOAC use and abnormal coagulation. Intraoperative echo can differentiate hypotension and instability from a carcinoid crisis and from RV dysfunction, which is common in carcinoid heart disease. Dopamine or dobutamine is preferred when RV dysfunction is present.

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#FR 1 – Kiosk 1 – Poster 7

Take a Load Off: LV Unloading in the Patient on Mechanical Circulatory Support

Presenting Author: Yaman Kherallah, MD, Fellow, University of Chicago

Introduction

Venoarterial extracorporeal membrane oxygenation (VA-ECMO) restores systemic perfusion in cardiogenic shock but increases left ventricular (LV) afterload through retrograde aortic flow. This can lead to LV distension, pulmonary edema, arrhythmias, and thrombus formation when the aortic valve remains closed. We present a case of VA-ECMO in a patient with severe aortic stenosis (AS) and prosthetic valve endocarditis who developed persistent LV distension requiring adjunctive transseptal TandemHeart (LivaNova, UK) left-atrial (LA) drainage.

Case Presentation

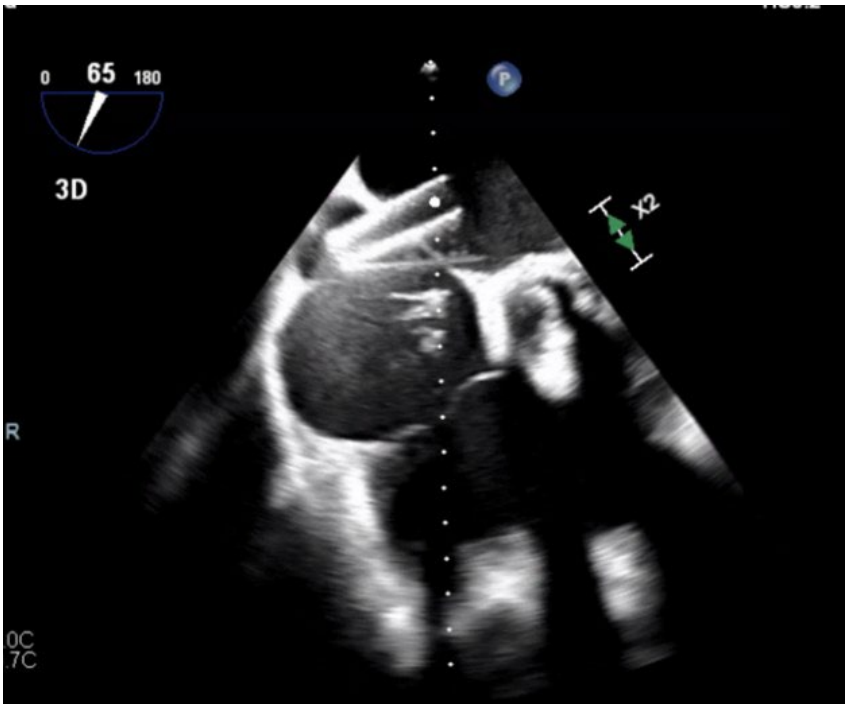
A 23-year-old man with history of VSD and coarctation repairs, aortic valve replacement complicated by prosthetic AS, and complete heart block with CRT-P presented in mixed cardiogenic–septic shock and was transferred from an outside hospital on peripheral VA-ECMO with an atrial septal stent in place for LV decompression. On post-cannulation day 17 he was taken to the operating room for ECMO reconfiguration due to limb ischemia and ongoing LV distension. Intraoperative TEE confirmed severe AS, moderate mitral regurgitation (MR), markedly depressed LV function, and patency of a previously placed atrial-septal stent with left to right shunt. Despite advancement of the venous drainage cannula into the right atrium, LV distension persisted. The surgical team elected to place a TandemHeart, initially planning to place the cannula through the septal stent, but the stent was too small. Instead, they opted to place the cannula adjacent to the stent, under TEE and fluoroscopic guidance. TEE confirmed left-to-right flow, reduced LA/LV dilation, and improved LV function. Despite initial stabilization, refractory sepsis and ischemic colitis led to withdrawal of support on day 31.

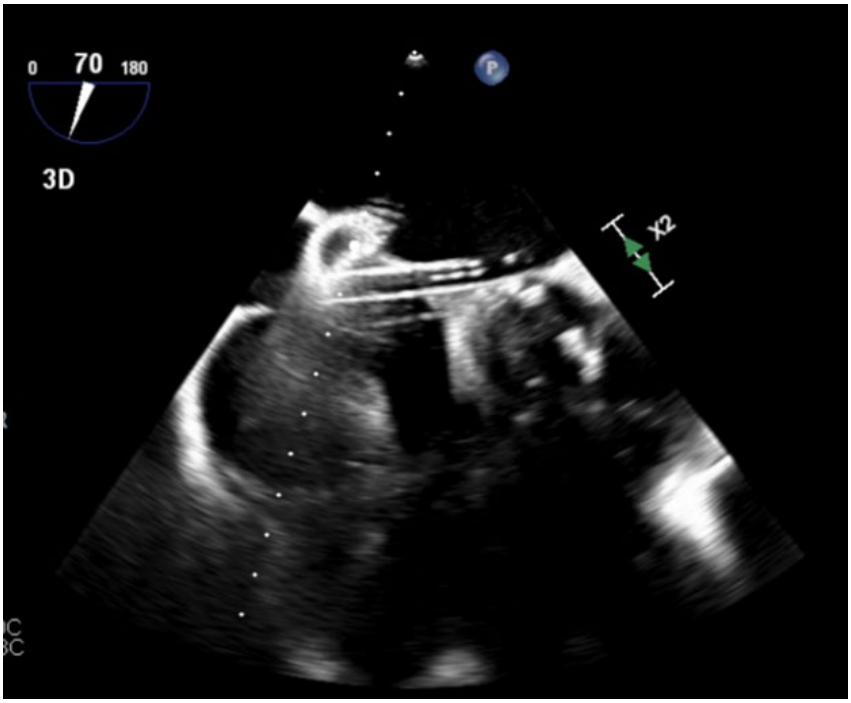
Discussion

In a patient on VA-ECMO, retrograde aortic flow shifts the LV pressure–volume loop rightward, elevating wall stress, exacerbating MR, and promoting pulmonary congestion. When the aortic valve remains closed, aortic root stasis predisposes to thrombus, LV distension, arrhythmias, and pulmonary edema. These factors increase myocardial oxygen demand, limit myocardial recovery, impair the ability to wean mechanical circulatory support devices, and increase mortality[1]. LV unloading during VA-ECMO reduces LV end-diastolic pressure/volume, pulmonary venous hypertension, and LV distension. It has been associated with lower mortality and improved weaning from mechanical support, although trial results are mixed[2,3]. Nonpharmacologic strategies for LV unloading include intra-aortic balloon pump, transaortic microaxial pumps, balloon atrial septostomy, and transseptal LA cannulation. In this case, severe AS and endocarditis precluded transaortic approaches such an Impella (Abiomed, MA), and the pre-existing atrial-septal stent was inadequate. The need to place a TandemHeart cannula adjacent to the stent highlights the critical role of TEE in safe device placement. TEE was essential in guiding transeptal access superior and anterior to the

stent while avoiding the aortic valve (Fig. 1). This demonstrates the feasibility of transseptal left atrial drainage for LV unloading in patients with complex anatomy. Future work should refine patient selection and optimal timing to balance potential benefit against the risk of unnecessary medical intervention.

Images





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#FR 1 – Kiosk 2 – Poster 1

Leadless Pacemaker-Induced Tricuspid Regurgitation Requiring Surgical Management: A Case Report

Presenting Author: Colby Kihara, D.O., Resident physician, University of Colorado

Introduction

Since their introduction, permanent pacemakers have served as a cornerstone in the management of symptomatic bradyarrhythmias. Nevertheless, traditional transvenous systems carry meaningful procedural and post-procedural risks, with reported complication rates of 7.76% to 12.4% within the first 90 days after implantation [1]. Leadless pacemakers were developed to address these limitations by eliminating intravascular leads and device pockets, thereby reducing the risk of lead dislodgement, venous thrombosis, and lead-related endocardial injury. We present a case of severe tricuspid regurgitation (TR) associated with a leadless pacemaker, necessitating surgical device retrieval and tricuspid valve (TV) repair.

Case Presentation

A 74-year-old man with a past medical history notable for atrial fibrillation, hypertension, nonobstructive coronary artery disease, and sick sinus syndrome status post leadless pacemaker placement, later removed due to moderate-to-severe TR, and replaced with an Aveir VR leadless pacemaker and tricuspid annuloplasty with a 28-mm ring, presented with progressive TR. Despite these interventions, a transesophageal echocardiogram (TEE) performed several years after his TV repair and leadless pacemaker placement revealed torrential TR, right ventricular dilation with preserved systolic function, and biatrial enlargement. Cardiothoracic surgical evaluation determined that he was not a candidate for valve-in-valve therapy. Due to ongoing symptomatic TR and concern that the leadless pacemaker was impairing leaflet coaptation, surgical management was recommended. During the current admission, he underwent successful leadless pacemaker retrieval, left atrial appendage ligation, TV replacement, and epicardial cardiac resynchronization therapy implantation. Intraoperative pre-cardiopulmonary bypass TEE showed severe TR with the pacemaker positioned in the basal right-ventricular free wall, abutting the septal leaflet. Post-repair TEE demonstrated no residual TR. His postoperative course was uncomplicated, and he was discharged on day 18.

Discussion

Leadless pacemakers were developed to address key limitations of transvenous systems. Although leadless devices demonstrate a 63% reduction in major complications at 12 months, risks remain, with reported complication rates near 2.7% [2]. While leadless systems were expected to reduce tricuspid valve dysfunction seen with transvenous leads, emerging evidence indicates that TR progression may still occur. Although early data suggested stable or improved valve function in most patients, subsequent studies report worsening TR in up to 43% at one year [3]. This case illustrates an uncommon but clinically significant complication: mechanical interference between a leadless pacemaker and the septal tricuspid leaflet, resulting in torrential TR requiring device extraction and repair of the TV. As leadless pacing technology continues to evolve, further prospective studies are needed to clarify risk factors, optimize device

positioning, and better define patient populations most likely to benefit. Early recognition of device–valve interactions and multidisciplinary management remain essential to improving outcomes.

Images

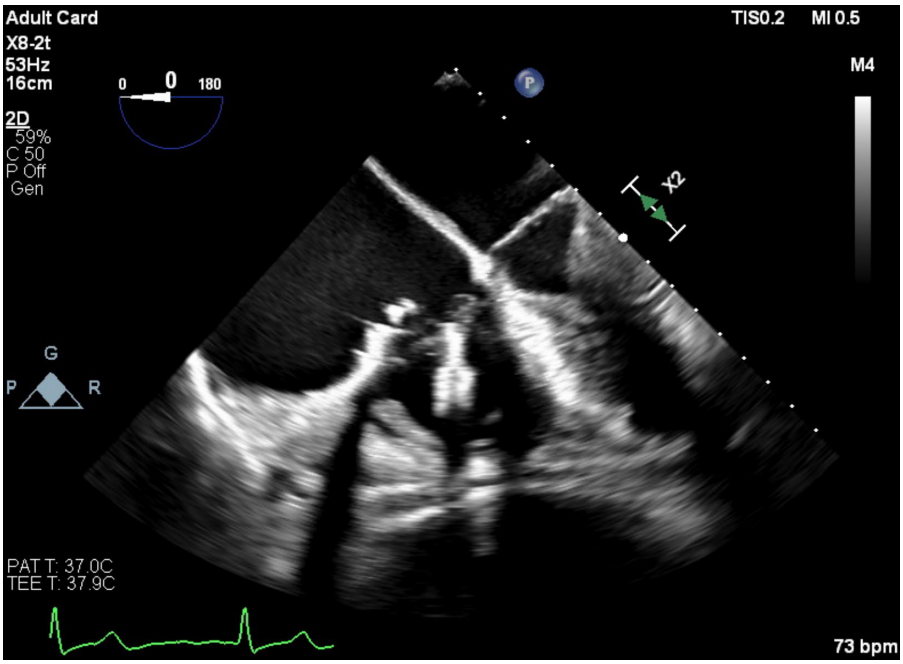


Image showing the leadless pacemaker abutting the tricuspid valve prior to intervention

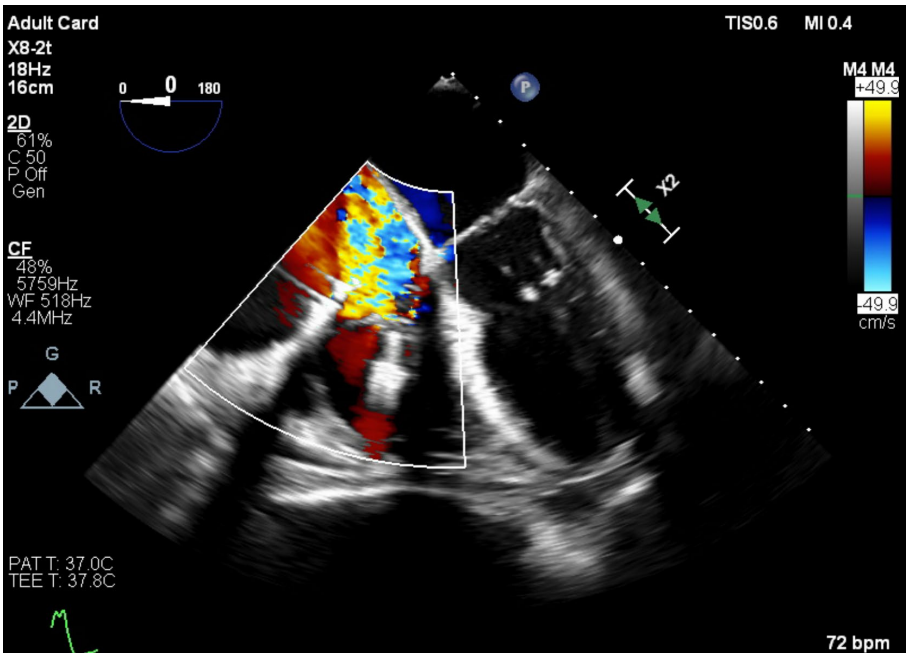


Image demonstrating severe tricuspid regurgitation prior to intervention

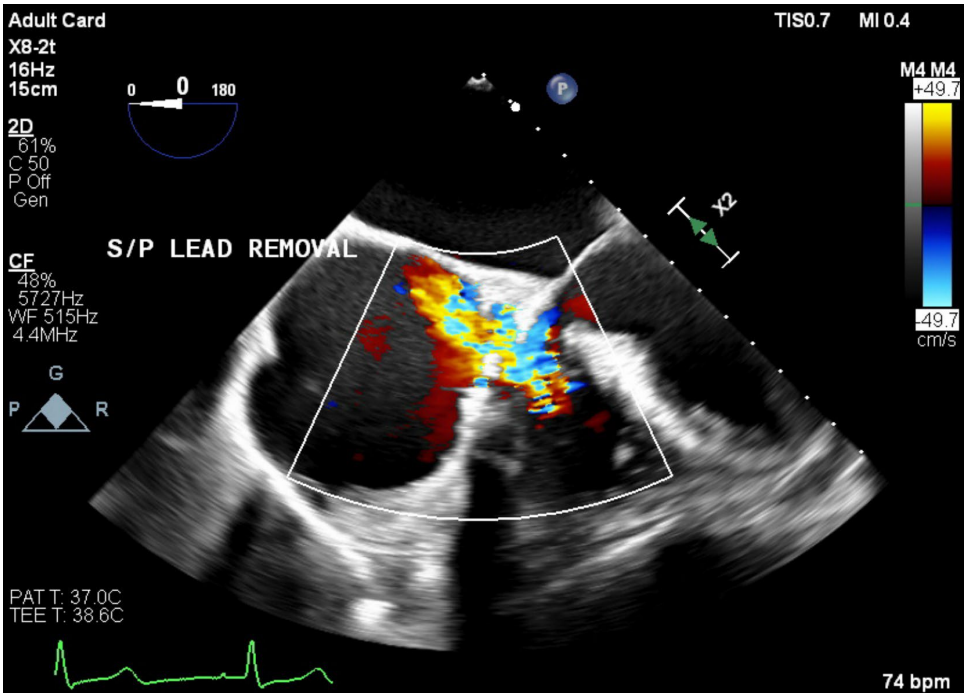


Image demonstrating severe tricuspid regurgitation after lead removal

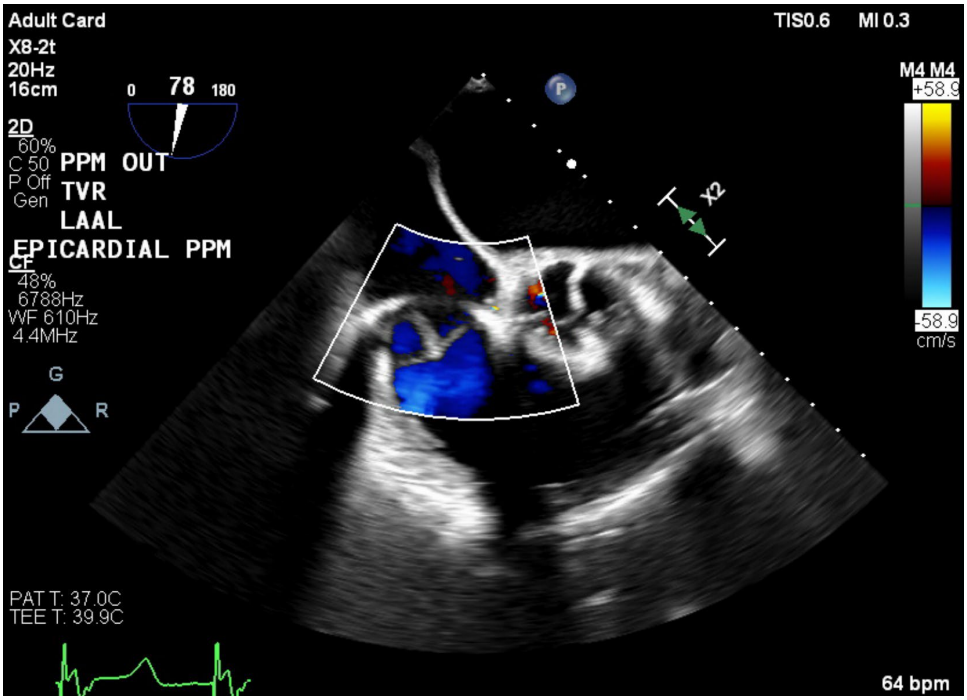


Image after intervention demonstrating no tricuspid regurgitation

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#FR 1 – Kiosk 2 – Poster 2

Transesophageal Echocardiogram-Driven Management of Intraoperative Hemodynamic Instability Due to Systolic Anterior Motion of the Mitral Valve

Presenting Author: Sahrudh Dharanendra, MD, Resident, Emory University School of Medicine Department of Anesthesiology

Introduction

Systolic anterior motion (SAM) of the mitral valve is a dynamic displacement of the anterior leaflet towards the left ventricular outflow tract (LVOT) during systole, resulting in LVOT obstruction. SAM is classically associated with hypertrophic cardiomyopathy or left ventricular hypertrophy. However, SAM can also occur in structurally normal hearts, especially in conditions that involve a hyperdynamic, underfilled left ventricle (Reddy & Ueda, 2014). In the perioperative setting, SAM is an important, but underrecognized cause of intraoperative hypotension. Diagnosis is usually made with echocardiography and requires prompt intervention, typically with volume resuscitation and beta-blockers. We report the case of a 41-year-old male with no significant past medical history who presented with multiple traumatic injuries following a motor vehicle collision. He developed intraoperative hypotension, which was attributed to SAM and responded well to treatment with esmolol, vasopressin, and volume resuscitation.

Case Presentation

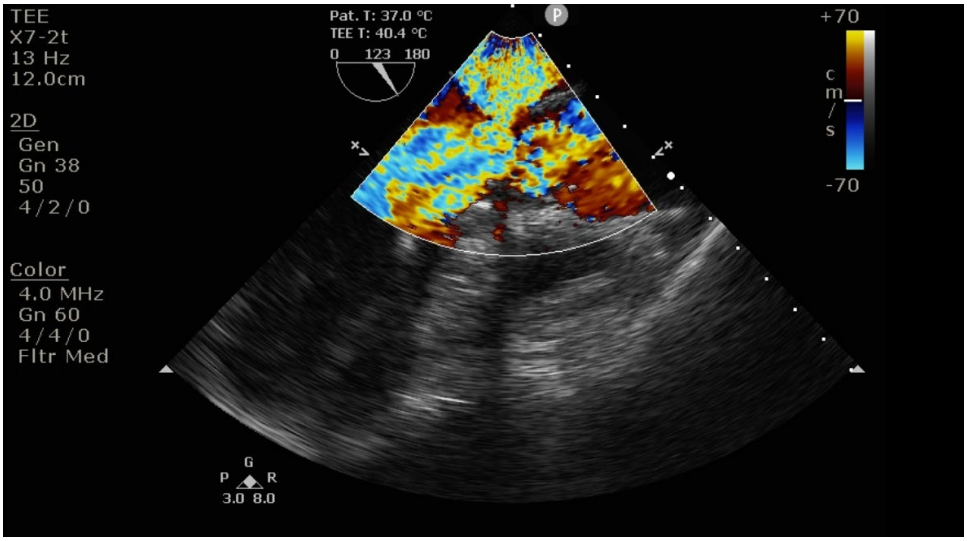
Point-of-care ultrasound team consulted for intraoperative hypotension in a 41-year-old male post motorcycle crash with significant LLE trauma, now 8 hours into emergent wound exploration and revascularization. Prior to consult, patient received significant blood product transfusion (24 units total) and was maintained on a norepinephrine infusion. Patient had episode of ventricular tachycardia with a pulse immediately prior to consult. Initial impression of patient revealed significant hemodynamic instability (arterial line unable to detect pulsatile waveform, NIBP 80s/50s, HR 150s per ECG, and SpO2 mid-high 80s). Transthoracic echocardiogram (TTE) attempted with poor windows. Decision to switch to transesophageal echocardiogram (TEE) which revealed underfilled and hyperdynamic left ventricle. Mitral regurgitation and left ventricular outflow tract (LVOT) flow acceleration demonstrated “Y-sign” consistent with systolic anterior motion of the mitral valve (SAM). 40 mg of esmolol and 2 units of vasopressin administered. HR decreased to 110 bpm with resolution of SAM and dynamic LVOT obstruction, and blood pressure improved to 120s/80s on arterial line. Decision made with surgeons to temporarily close incisions and apply negative pressure wound therapy device in order to transport patient to ICU for further care and resuscitation.

Discussion

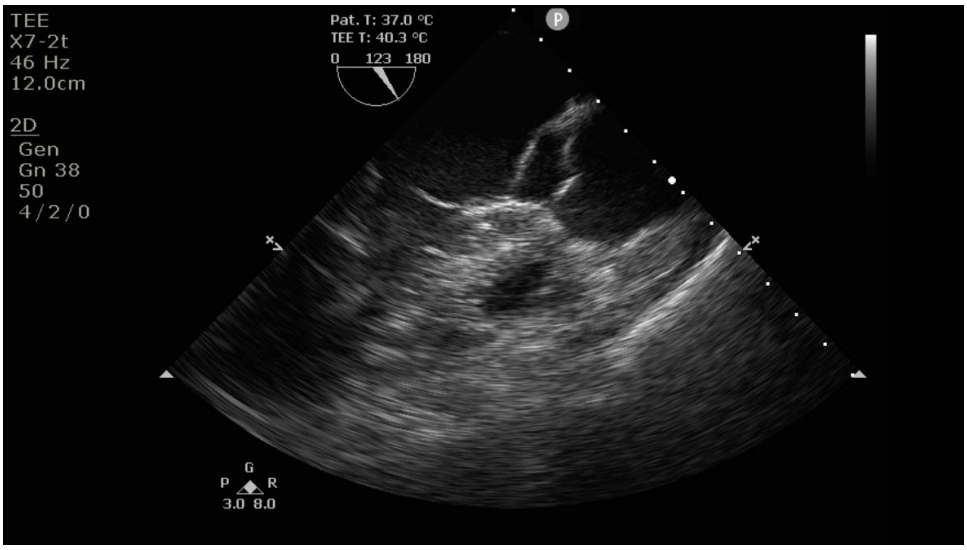
Quickly revealing the source of hemodynamic instability in a perioperative patient is important to reduce mortality and complications, particularly in the setting of emergency surgery. This case showcases the role of intraoperative echocardiography to quickly identify a clinical issue and guide management to improve the patient’s status rapidly. Specifically, the decision to switch from TTE to TEE was crucial in identifying SAM that was contributing to LVOT obstruction causing hemodynamic instability in this patient. In this case,

esmolol was used to slow heart rate, decrease contractility, and improve diastolic filling to resolve LVOT obstruction (Crescenzi et al. 2008), while the use of vasopressin allowed for reduction of norepinephrine and its beta agonism, and resulted in resolution of LVOT obstruction as well (Balik et al. 2020).

Images



TEE image of Y-Sign



Mid-Esophageal LAX view of Anterior Mitral Valve Leaflet causing occlusion

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#FR 1 – Kiosk 2 – Poster 3

The Long Clot: A Tale of Massive Pulmonary Embolism, Mechanical Thrombectomy, and Mechanical Circulatory Support

Presenting Author: David M. Altman, MD, Anesthesiology Resident, Stanford Medicine

Introduction

Pulmonary embolism (PE) is the third leading cause of cardiovascular death, and high-risk PE has traditionally been managed with systemic thrombolysis. Because thrombolysis carries substantial bleeding risk, only about 16% of eligible patients receive it [1]. Mechanical thrombectomy (MT) has recently emerged as an alternative, but it typically requires sedation or general anesthesia, which can be risky in the setting of marked RV strain. In such cases, mechanical circulatory support (MCS) may be required to facilitate MT [2]. We present a case of massive PE in which airway manipulation precipitated cardiovascular collapse, necessitating emergent MCS for support during MT.

Case Presentation

A 48-year-old man with advanced laryngeal squamous cell carcinoma, status post tracheostomy and awaiting total laryngectomy, presented to an outside hospital with acute chest pain and dyspnea. Imaging showed a massive PE with thrombus extending from the right atrium (RA) through the right ventricle (RV) into the main pulmonary artery (PA) (Fig 1), forming a saddle embolus, along with extensive bilateral segmental clot burden. He was transferred to our center for MT. Upon arrival, 5 French micropuncture catheters were placed in the left femoral artery and vein for potential cannulation for venoarterial extracorporeal membrane oxygenation (VA-ECMO). Because his tracheostomy was uncuffed, we elected to exchange it for a cuffed tube to ensure a secure airway, anticipating the need for controlled ventilation during the procedure. We topicalized with 4% lidocaine, and the exchange was initially uneventful. However, immediately after cuff inflation, the patient coughed forcefully and developed acute, persistent hypotension unresponsive to vasopressors and inotropes. Emergent VA-ECMO cannulation was initiated. During cannulation he became pulseless, prompting CPR; ECMO flow was established seven minutes later. Following stabilization, MT proceeded, successfully removing extensive thrombus from the RA, RV, and PAs (Fig 2). He was transferred to the cardiovascular intensive care unit and was found to be neurologically intact after weaning sedation. Several days later, he was transferred, on VA-ECMO, to an outside hospital for his planned laryngectomy, after which he was successfully decannulated and eventually discharged home.

Discussion

This case underscores the importance of anticipating profound hemodynamic instability in massive PE, particularly during airway manipulation, where even small changes in intrathoracic pressure can precipitate RV failure. Here, early placement of micropuncture access substantially reduced the time required to initiate ECMO support. The event also illustrates how routine perioperative decisions can lead to major downstream consequences. Although securing the airway before MT was reasonable, coughing during the exchange may have propagated clot or abruptly increased pulmonary vascular resistance, leading to

hemodynamic collapse despite careful topicalization. Alternatives such as cannulating for ECMO before the exchange or sedating the patient beforehand carried their own risks. Ultimately, this case highlights the need for meticulous planning, multidisciplinary coordination, and immediate readiness to escalate to MCS when caring for patients with massive PE.

Images

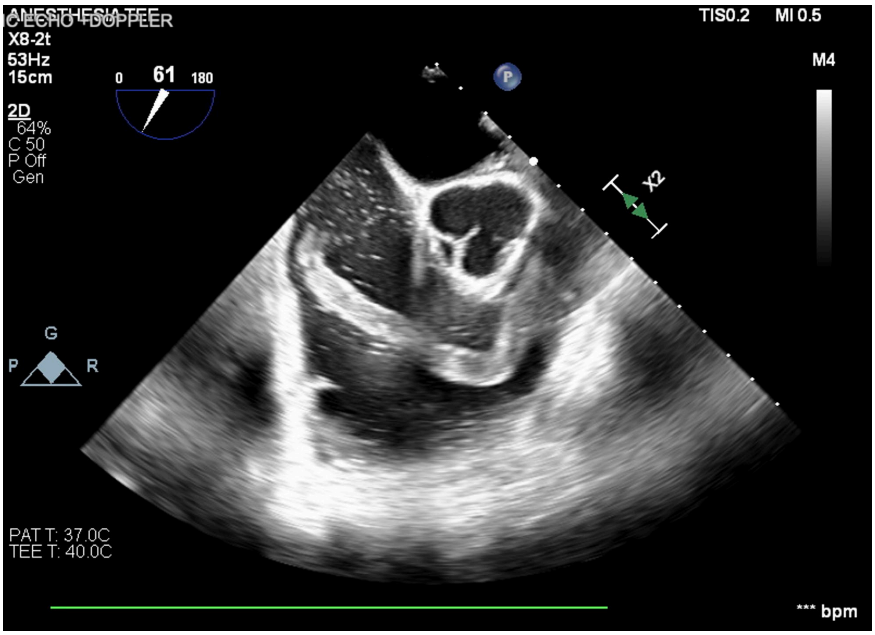


Figure 1: Mid-esophageal RV inflow-outflow view showing clot spanning from RA to PA



Figure 2: Initial long clot removed via suction thrombectomy

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#FR 1 – Kiosk 2 – Poster 4

Fulminant Protamine Reaction After Bentall Procedure: Rapid Hemodynamic Collapse and ECMO Rescue

Presenting Author: Henry G. Liokumovich, MD, Fellow, Ochsner Medical Center

Introduction

Protamine is a polycationic protein mainly utilized as a reversal agent for unfractionated heparin anticoagulation during cardiac and vascular operations. Intravenous protamine administration can cause acute reactions that range from hypotension to anaphylaxis and cardiovascular collapse (1).

Case Presentation

The patient is a 22 year old male with a history of morbid obesity, aortic insufficiency (AI), aortic stenosis (AS) and a proximal ascending aortic aneurysm that underwent a Bentall procedure. Initial intraoperative transesophageal echocardiogram (TEE) showed normal biventricular function with an estimated ejection fraction of 60%, a unicuspid aortic valve, moderate AI, severe AS, and a proximal ascending aortic aneurysm. The surgery proceeded uneventfully and the initial post bypass TEE showed normal biventricular function with a well functioning mechanical aortic valve. A test dose of 10mg protamine was administered, uneventfully, and the rest of the protamine dose was started slowly. After 50mg of protamine was administered, the pulmonary artery (PA) pressures acutely rose to 70s from 30s systolic with a simultaneous drop in systemic pressures to 60s from 110s systolic. The right ventricle free wall also began to dramatically balloon on TEE. A type III protamine reaction was suspected as the most likely differential diagnosis for this presentation. Epinephrine, norepinephrine, vasopressin infusions and inhaled nitric oxide were started. An angiotensin II infusion was subsequently added to augment perfusion pressures. At this point in time, the anterior and lateral walls of the left ventricle also began to appear akinetic despite good flow through the coronary buttons. An intraaortic balloon pump was placed leading to only a small improvement of the cardiac index. The patient was transported to the cardiac surgical intensive care unit (ICU). Shortly after arrival in the ICU, he underwent cardiac arrest requiring internal cardiac massage and emergent VA ECMO cannulation.

Discussion

Protamine administration is associated with a variety of complications that range from systemic

hypotension to catastrophic pulmonary hypertension and right ventricular dysfunction. There are multiple hypothesized mechanisms of action for these complications, including IgE-mediated reactions, complement activation, histamine and thromboxane A2/B2 release (1,2). The reported incidence of intravenous protamine reactions is between 0.08% to 1.2% in the general population and 3% to 26.6% in diabetic patients with a history of using protamine containing insulin (2). Protamine should be administered slowly with close monitoring and closed-loop communication among the operating room team. Quickly identifying a protamine reaction by changes in physical exam, peak airway pressures, invasive blood pressure and PA pressures, if available, is crucial. If a reaction is suspected, then the protamine infusion should be stopped immediately. Acute pulmonary hypertension and hemodynamic collapse should be treated with vasopressor and inotropic medication. Advanced support with mechanical devices, such as ECMO, can be initiated in severe, refractory cases.

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#FR 1 – Kiosk 2 – Poster 5

Iatrogenic Aortic Insufficiency from Coronary Cusp Entrapment During Aortic Arch Stent Deployment

Presenting Author: Parita S. Jani, MD, MPH, Adult Cardiothoracic Anesthesiology Fellow, Washington University in Saint Louis

Introduction

Endovascular repair of aortic arch pathology has expanded with the development of branched and fenestrated devices. These procedures are increasingly offered to patients at high surgical risk. Despite high early technical success, arch stenting carries anesthetic challenges, including cerebral ischemia, coronary obstruction, major vascular injury, and rare valve injury. This case highlights a rare but severe complication—iatrogenic aortic insufficiency (AI) from mechanical trauma to the left coronary cusp during stent deployment.

Case Presentation

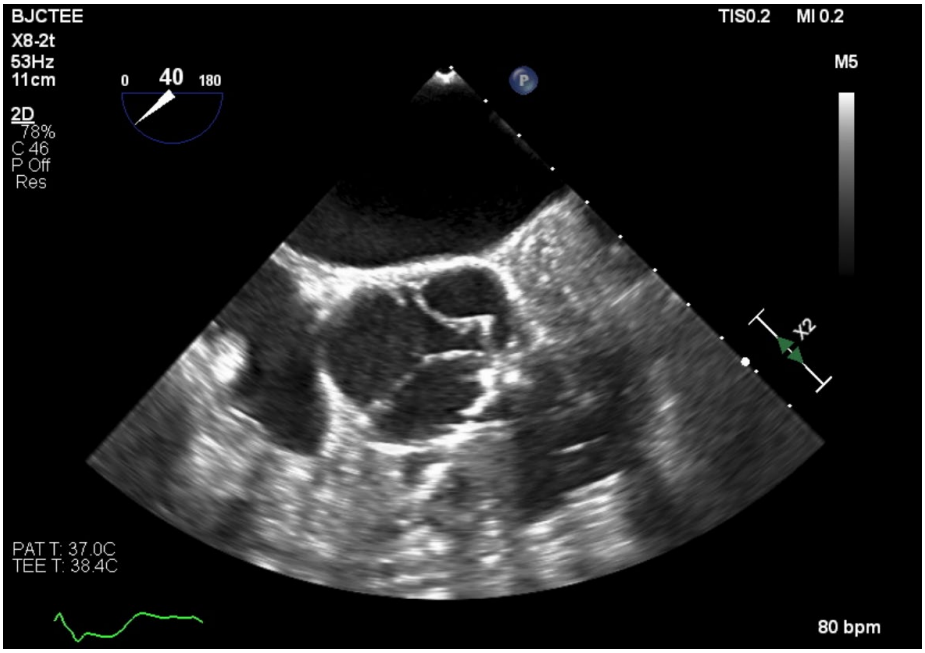
A 79-year-old woman with a penetrating aortic ulcer of the arch and an ascending aortic aneurysm was scheduled for branched endovascular aortic arch repair. Her history included CAD with prior PCI, COPD, hypertension, and T2DM. Prior to this procedure, she underwent cervical revascularization for stroke risk reduction. This case was done under general anesthesia, with a left radial arterial line, a right internal jugular central line, and continuous TEE. Pre-procedure TEE showed normal biventricular function with mild central AI. Given the potential for complications during endovascular repair, perfusion support was on standby for open conversion. Bilateral femoral and right radial arteries were accessed, with wires advanced from the left femoral and right radial arteries to the arch. Sequential arch endograft components were deployed in standard fashion. Early graft migration required repositioning under rapid ventricular pacing (RVP), for which a right femoral venous pacing catheter was positioned in the right ventricle. During deployment of the final graft component at the sinotubular junction, the patient developed abrupt hypotension and bradycardia. TEE demonstrated entrapment of the left coronary cusp (LCC) of the aortic valve by the delivery system. RVP was utilized again to allow disengagement of the device from the cusp; however, the LCC was torn, resulting in torrential central and eccentric AI on TEE. Since all grafts had been deployed and patient remained stable, emergent surgical valve repair was not pursued and she was transferred to the ICU. Her postoperative course was complicated by prolonged intubation due to pulmonary edema and complete heart block requiring temporary pacing. She was transferred out of the ICU after 6 days in normal sinus rhythm with planned elective aortic valve replacement the week after.

Discussion

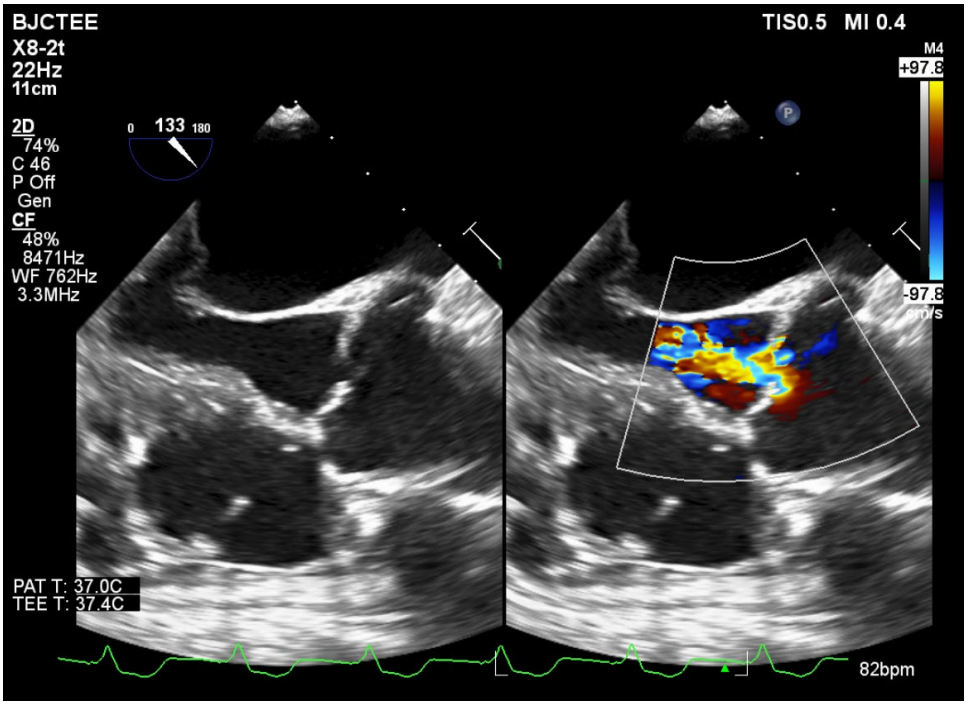
This case highlights a rare but important complication of branched endovascular arch repair: acute severe AI from mechanical interaction between the delivery system and the native aortic valve. Passage of large devices near the aortic root can lead to cusp entrapment or trauma. Sudden hemodynamic instability in these phases should prompt immediate TEE assessment to define the mechanism and determine whether

device disengagement is feasible. In this instance, successful release using RVP was followed by irreversible LCC injury, resulting in severe AI with associated conduction disturbance and pulmonary edema. As endovascular arch technologies proliferate, anesthesiologists will increasingly encounter device-related valve injury. Early recognition and coordinated intraoperative management are essential to avoid hemodynamic instability and guide postoperative care.

Images



Mid-esophageal short-axis view of the aortic valve demonstrating mechanical entrapment of the left coronary cusp (LCC): The delivery system has tethered the left coronary cusp (LCC) in an immobilized, fixed-open position, preventing symmetric leaflet excursion and normal coaptation. This finding explains the patient's immediate post-deployment hemodynamic instability and development of acute severe aortic insufficiency.



Mid-esophageal long-axis view with color Doppler showing torrential aortic regurgitation following leaflet release: After disengagement of the device from the LCC, severe central and eccentric aortic regurgitation persisted due to cusp tearing.

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#FR 1 – Kiosk 2 – Poster 6

TAVR Aortic Insufficiency and Mitral Regurgitation Prompting LAVA-ECMO Complicated by Left Atrial Appendage Perforation

Presenting Author: Danielle C. Isham, MD, Cardiothoracic Anesthesia Fellow, University of Michigan Hospital Department of Anesthesia

Introduction

Valvular dysfunction accounts for 10 to 20% of cardiogenic shock (CS) cases and is challenging to manage, as common mechanical circulatory support (MCS) devices can worsen hemodynamics1. Intraaortic balloon pumps and percutaneous left ventricular (LV) devices are often contraindicated, and conventional venoarterial extracorporeal membrane oxygenation (VA-ECMO) can increase LV filling pressures, promoting pulmonary edema2. Left atrial venoarterial ECMO (LAVA-ECMO) unloads both ventricles through a single transseptal cannula and may serve as a bridge to definitive intervention2. We present a case of acute severe aortic insufficiency (AI) from transcatheter aortic valve replacement (TAVR) leaflet disruption and severe mitral regurgitation (MR) prompting LAVA-ECMO which was complicated by airway bleeding and left atrial appendage (LAA) perforation.

Case Presentation

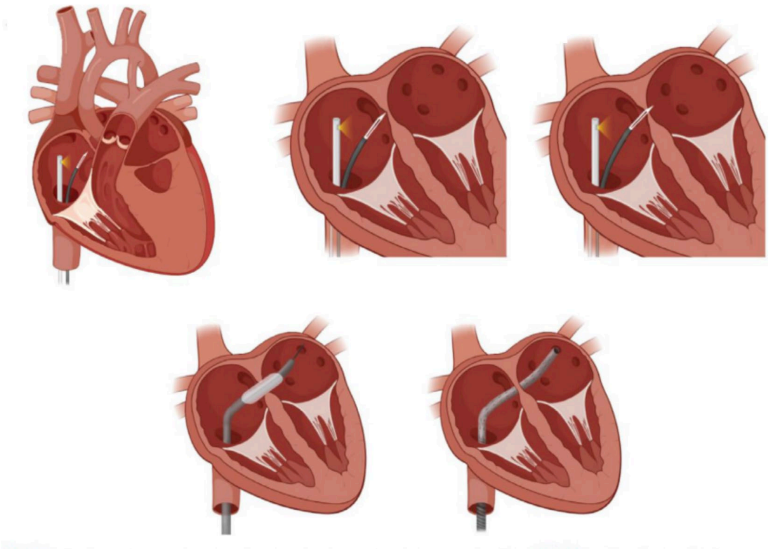
A 73-year-old man with a prior TAVR presented to an outside hospital with syncope from progressive aortic stenosis. While awaiting transfer for surgical replacement, he underwent left heart catheterization which was complicated by cardiac arrest due to acute AI from a torn valve leaflet. He was emergently intubated and placed on VA-ECMO, later converted to LAVA-ECMO because of severe AI, severe MR, and airway bleeding concerning for pulmonary edema. He was then transferred to our institution. On arrival, his ETT and oropharynx were filled with blood of an unclear source, and his airway was managed with careful suctioning. He remained on minimal ventilator support. He was emergently taken to the OR for TAVR explant, aortic valve replacement, aortic root enlargement, and mitral valve repair. After pericardial entry, a large bloody effusion was encountered. TEE demonstrated the LAVA-ECMO cannula passing through the interatrial septum with the tip not visible. He had severely reduced LV function (EF 25-30%) and low-normal RV function. After LAVA cannula withdrawal, brisk bleeding consistent with LAA perforation occurred and the patient was quickly transitioned from ECMO to cardiopulmonary bypass. Emergency blood products were prepared as well as a Belmont rapid infuser. LAA primary repair and clipping, atrial septal defect repair, and the valve procedures were completed. Postoperatively, a pharyngeal wall laceration was found, likely from emergent intubation, which was treated with packing for 24 hours. He was extubated on POD 3. By POD 6, TTE showed recovered LV function with an EF of 65%. His course was further complicated by Klebsiella pneumonia treated with antibiotics. He transferred to step-down on POD 11 and to inpatient rehabilitation on POD 19.

Discussion

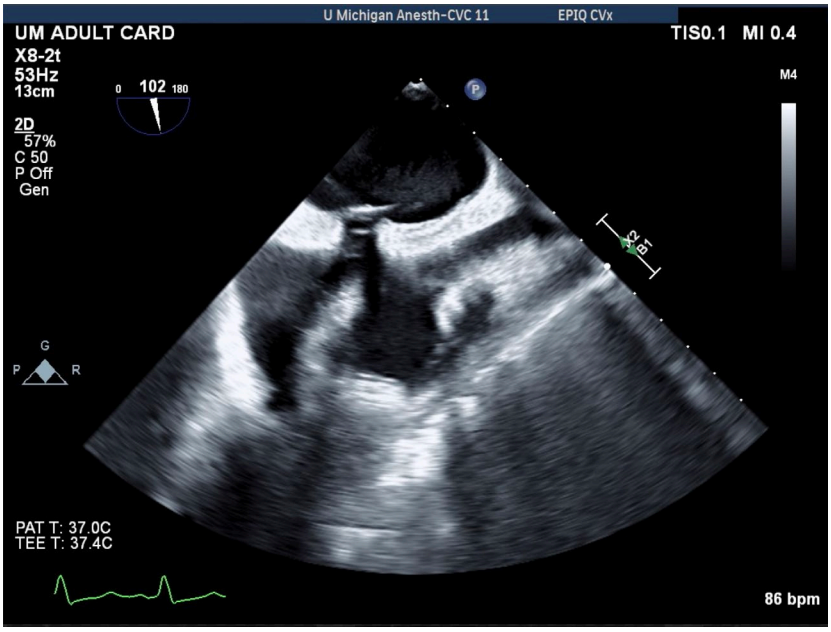
This case demonstrates the use of LAVA-ECMO as salvage support in acute severe AI and MR, conditions in

which other MCS options can be unsafe or ineffective. LAVA-ECMO provides active left atrial drainage and reduces LV filling pressures, limiting pulmonary edema and improving systemic perfusion1. The case also highlights anesthetic challenges including management of a bloody airway of uncertain origin, rapid preparation for major bleeding, and the essential diagnostic value of TEE in identifying LAA perforation. Complex valvular lesions with poor biventricular function required nuanced hemodynamic management and potentially non-traditional MCS.

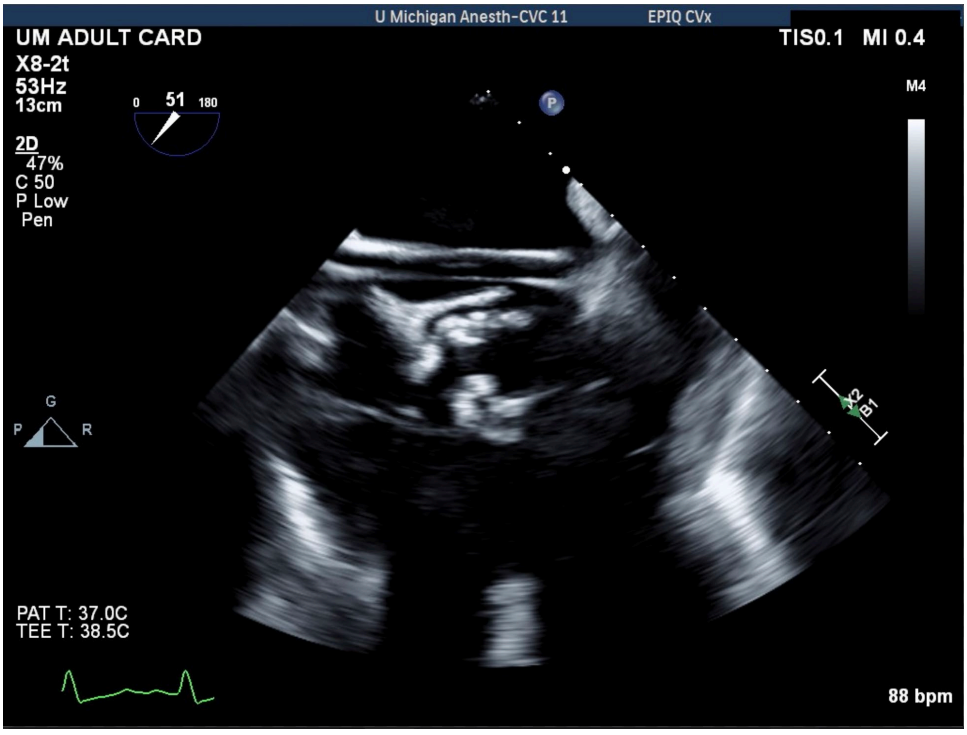
Images



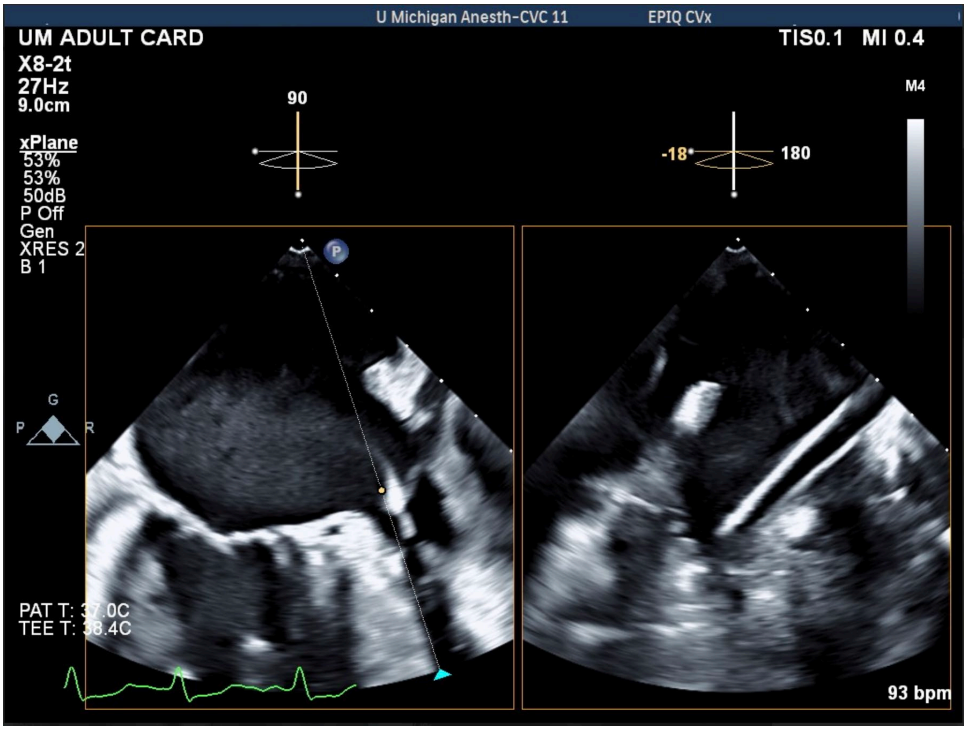
Placement of LAVA-ECMO with intracardiac echocardiography



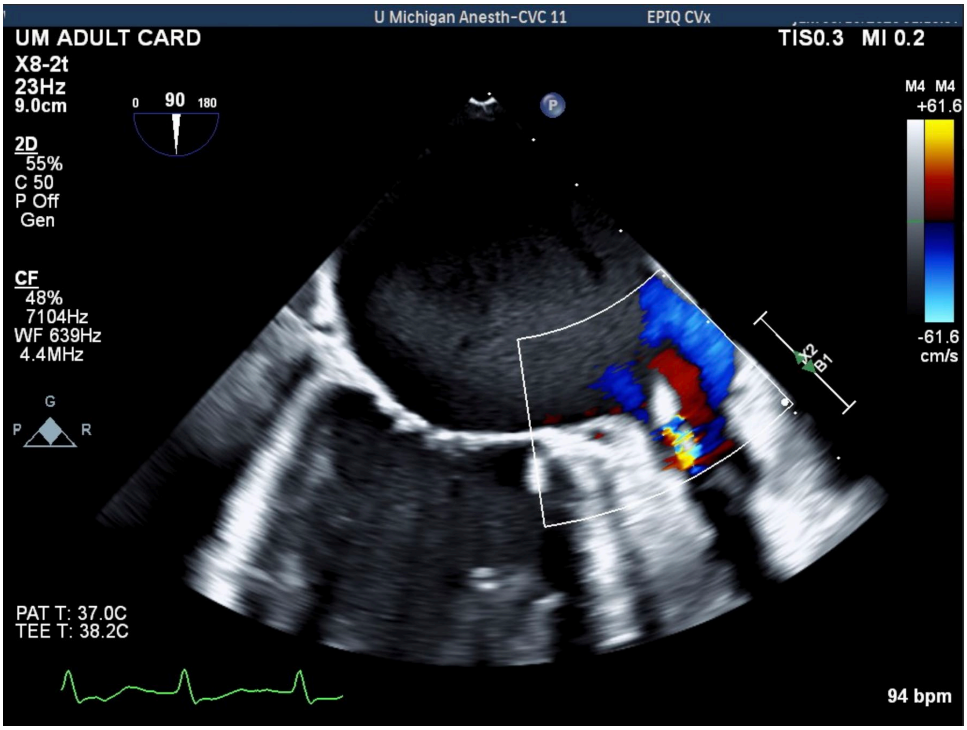
TEE ME bicaval view with cannula crossing interatrial septum



TEE ME RV inflow outflow view of LAVA cannula



TEE ME two-chamber with x-plane of LAVA cannula through LAA



TEE ME two-chamber with color Doppler

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#FR 1 – Kiosk 2 – Poster 7

Cold Agglutinin Disease During Coronary Artery Bypass Surgery

Presenting Author: Benjamin S. Christensen, MD, Fellow, Texas Heart Institute

Introduction

Primary cold agglutinin disease (CAD) accounts for 15% of autoimmune hemolytic anemias with an estimated prevalence of 10 cases per million people[3]. Cold Agglutinins (CA) are auto antibodies (IGM) that become active below physiological temperatures. Clinical significance arises when the thermal amplitude rises to temperatures of 28-31°C or higher[1]. CA form when IgM molecules bind to RBC surface antigens resulting in agglutination with RBCs. Agglutinated RBCs then undergo complement fixation via the classical complement pathway. Cardiopulmonary bypass in patients with CAD carries a large risk of morbidity. Systemic hypothermia can result in blood reaching temperatures that cause agglutination and hemolysis. This can lead to circuit obstruction, impaired pump flow, and worsening hemolysis[1].

Case Presentation

82-year-old female with a past medical history of ischemic cardiomyopathy and an ejection fraction of 25%, hypertension, hyperlipidemia, type 2 diabetes mellitus, who presented for a robotic coronary artery bypass graft. On the day of surgery, blood bank reported that the patient had a positive cold agglutinin screening. Following a multi-disciplinary discussion, the case was postponed pending further workup. Further testing confirmed the diagnosis of cold agglutinin disease. Surgery was then rescheduled for a few weeks later. On the day prior to surgery patient underwent plasmapheresis to remove IgM and was started on Sutimlimab, a monoclonal antibody and complement inhibitor used to reduce hemolysis. For the procedure, the operating room was warmed and the patient kept covered in blankets. A forced air warmer, elevated room temperature, and warmed IV fluids were also utilized. Patient was placed on ECMO support with flows at 3-4 Liters/ min. The surgeon then performed a robotic LIMA to LAD bypass. After the patient was taken to ICU and extubated 5 hours later. Post op course was uncomplicated, and the patient was discharged home 7 days later.

Discussion

In this case CAD was diagnosed prior to the start of the surgical case. Given the nonemergent nature of the case it was rescheduled pending medical optimization. The surgical plan mad it possible to avoid CPB, cardioplegia delivery, and hypothermia. This unique approach substantially reduced the risk of intraoperative cold-induced hemolysis. Unfortunately, such optimal conditions are not always possible. Many cases are diagnosed intraoperatively, often after the patient has already been cooled. If possible, the patient and circuit should be warmed promptly, and the cardioplegia flushed out, then replaced with a warm solution[2]. The decision to proceed or not will depend on various factors including the timing of the discovery, clinical significance, and urgency of the case. In situations where it's deemed necessary to proceed with the procedure, every effort should be made to minimize time spent below the patient's thermal amplitude. The patient should then be warmed and as many portions of the surgery done as possible without hypothermia.

It may also be prudent to consider the administration of a complement inhibitor intraoperatively and post operative plasmapheresis. Following the surgery, care should then be taken to watch out for signs of hemolysis and other thromboembolic complications.

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#FR 1 – Kiosk 3 – Poster 1

HeartMate 3 Left Ventricular Assist Device Placement in a Patient with Large Left Ventricular Pseudoaneurysm

Presenting Author: John N. Filosa, MD, Cedars Sinai Medical Center

Introduction

Left ventricular pseudoaneurysm (LVP) is a rare complication of myocardial infarction (MI) caused by ventricular free wall rupture contained by pericardium.[1,2] If untreated, may lead to rupture, emboli, and compression of nearby structures.[1,2] These patients may develop cardiogenic shock requiring mechanical circulatory support (MCS), such as a durable left ventricular assist device (LVAD).[1,2] Large apical LVPs complicate LVAD placement due to friable tissue consistency at the inflow cannula site, and presence of intracardiac thrombotic material. We present a case of a patient with a large LVP presenting for HeartMate3 (HM3) placement, including the unique surgical approach and TEE considerations.

Case Presentation

A 57-year-old active smoker presented with a month of intermittent chest pain. He was diagnosed with a STEMI and underwent stenting of his left anterior descending artery. One month later, he presented with recurrent chest pain and shortness of breath. On evaluation, TTE showed severely depressed ejection fraction < 20%, and CT showed a large apical LVP of 6 x 6 x 9 cm in size. He was admitted to the intensive care unit (ICU), where a multidisciplinary team discussed treatment options, including transplant, surgical repair, percutaneous closure, and MCS. Based on the patient's psychosocial factors, LVP size, and persistent low cardiac indices without recovery, the team and patient pursued durable LVAD placement and LVP repair. After induction of general anesthesia and line placement, TEE exam was performed. LVP was identified along the apical wall, with spontaneous echo contrast seen in the cavity. RV function was moderately depressed, so inhaled nitric oxide and low dose epinephrine were started. After dissection on peripheral cardiopulmonary bypass (CPB), the LVP was identified and noted to be thin and friable, with surrounding inflammation from MI. The heart was arrested, and LVP was opened and clot removed. The cavity was marsupialized; sutures were passed around the LV, while plicating tissue of LVP to fit around the sewing ring of the HM3. HM3 pump was placed into the LV apex and secured. The HM3 RPM was titrated up, and the patient successfully separated from CPB on low dose dobutamine and epinephrine infusions. The patient was transferred to the ICU in stable condition. The rest of his hospital course was unremarkable, and he was discharged home on POD10 on warfarin and clopidogrel.

Discussion

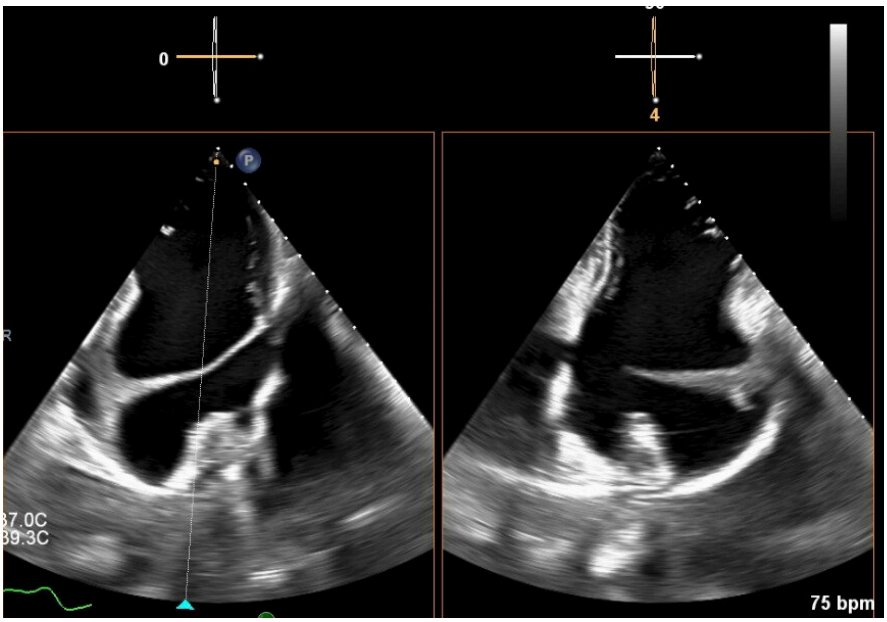
Limited case reports exist describing apical LVP repair during LVAD implantation. Surgical methods include endoventricular patchplasty or partial LV reconstruction with pledgeted sutures around aneurysmal sac.[1,2] In our case, aneurysmatic tissue appeared consistent enough to secure the cannula without additional patch repair. Regardless of approach, given friable apex, TEE is crucial for assessing for proper cannula placement.[3] Baseline TEE is also vital for assessing RV dysfunction, aortic regurgitation, LV thrombus, and

intracardiac shunt.[3] Presence of thrombi in the LVP increases risk of cerebrovascular accidents and pump thrombosis, although extremely rare.[1,2] Overall, acute cardiogenic shock with LVP is complex to medically manage and require the expertise of cardiac anesthesiologists perioperatively.

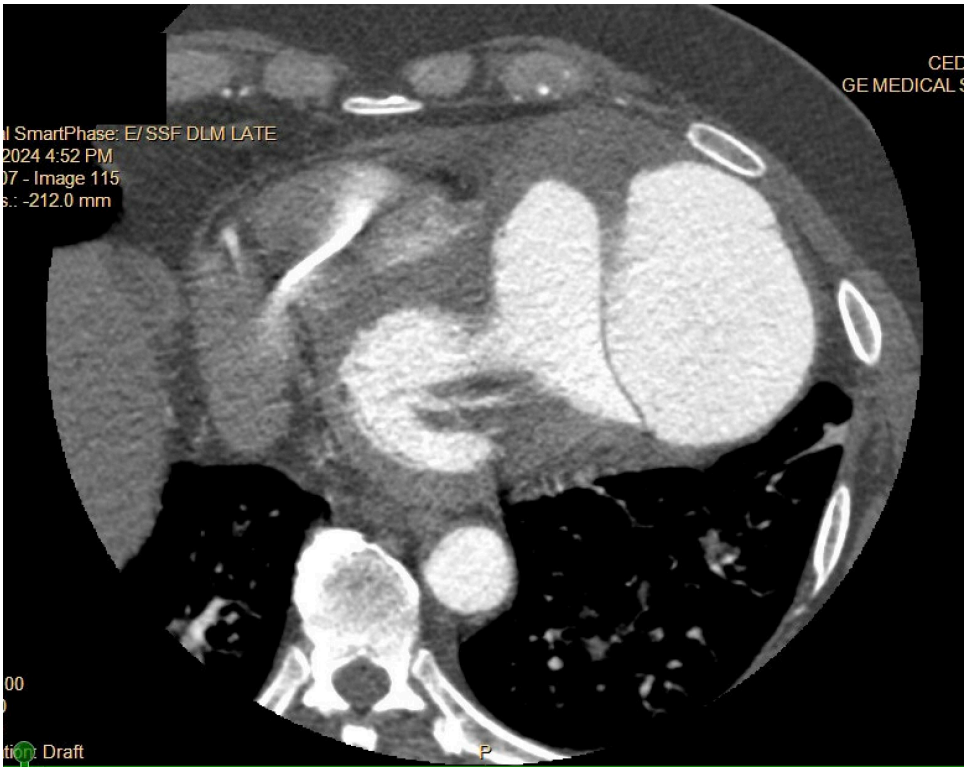
Images



Pseudoaneurysm LAX



LVAD positioning



CT Angio

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#FR 1 – Kiosk 3 – Poster 2

Non-Cardiogenic Pulmonary Edema and Vasoplegia After Cardiopulmonary Bypass

Presenting Author: Max T. Ackerman, MD, Resident, University of Chicago

Introduction

Severe pulmonary edema and vasodilatory shock can arise unexpectedly after cardiopulmonary bypass (CPB). These complications stem from profound inflammatory responses and pose diagnostic and management challenges. We present a case of combined coronary artery bypass grafting (CABG) and aortic valve replacement (AVR) complicated by non-cardiogenic pulmonary edema (NCPE) and severe vasoplegia.

Case Presentation

A 78-year-old male with coronary disease, 30% ejection fraction (EF), severe aortic stenosis (gradient 34 mmHg), and exertional dyspnea underwent three-vessel CABG and AVR. Bypass and cross clamp times were 232 and 190 minutes. Separation from bypass was achieved with dobutamine and phenylephrine infusions. Post-repair transesophageal echo (TEE) showed unchanged EF, a well-functioning 23 mm bioprosthetic valve, trace mitral regurgitation (MR), and normal pulmonary artery pressures (PAP). Over the next hour, the patient required escalating phenylephrine and vasopressin boluses for worsening hypotension. Peak airway pressures gradually rose from 20 to 40 cm H2O with three liters of pink frothy secretions suctioned from the endotracheal tube. TEE excluded MR and aortic outflow tract obstruction, and PAP remained normal. Arterial blood gas showed a P/F ratio < 100, PaCO2 67, and pH 6.9 with lactic acidosis. VV-ECMO was initiated but worsening biventricular function prompted conversion to VA-ECMO. The patient was transferred to the ICU with an open chest, given steroids for presumed inflammation, and initiated on hemodialysis for acidosis. He was decannulated from ECMO postoperative day (POD) 5, extubated POD 7, and discharged POD 19.

Discussion

Vasoplegic syndrome (VS) is a high-output, low-systemic vascular resistance state occurring in 5-44% of adult cardiac surgeries. The pathophysiology involves activated complement and cytokine pathways, upregulated nitric oxide synthase, and vasopressin depletion.[1] Risk factors include prolonged bypass, complex combined procedures, and renin-angiotensin system blockade. Management emphasizes prompt recognition, preload optimization, corticosteroids, and the use of vasoactive agents to restore vascular tone.[1, 2] Clinically, the differential diagnosis includes allergic or protamine reactions. However, the timing of symptoms (40 minutes post-protamine), normal filling pressures, and poor response to epinephrine made these unlikely. Acute diastolic dysfunction due to chest closure was also unlikely given the prolonged requirement for VA-ECMO. Non-cardiogenic pulmonary edema after CPB represents a severe manifestation of endothelial injury. It likely results from increased pulmonary capillary permeability from complement activation, cytokine release, and ischemia-reperfusion injury.[3] Clinically, pulmonary edema and hypoxemia are present despite normal filling pressures as in this case. Management is supportive: lung-protective ventilation, high PEEP, fluid removal as tolerated, steroids, and ECMO for refractory cases. This case

highlights the severity of cardiorespiratory deterioration that may occur with inflammatory vasoplegia after CPB, how inflammatory mechanisms may underlie both NCPE and VS, and the role of ECMO in recovery. Further work is needed to better understand how to best predict and treat this challenging complication.

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#FR 1 – Kiosk 3 – Poster 3

Postoperative Atrial Clot Formation Following HeartMate III Implantation

Presenting Author: Melinda Wei, DO, MBA, Resident Physician, Indiana University School of Medicine Department of Anesthesiology

Introduction

Left ventricular assist devices (LVADs) have been prominent in the treatment of end-stage heart failure. The HeartMate 3 (HM3) LVAD is a magnetically levitated pump which minimizes shear stress, resulting in reduced hemolysis and pump thrombosis compared to previous LVADs[1,2]. Despite pump changes, risks for LVAD thrombosis include presence of a bioprosthetic valve, recent post-op status, subtherapeutic anticoagulation in the post-op period, and low cardiac output states[1,3]. We present a case of left atrial thrombus formation following LVAD implantation, necessitating emergent removal on bypass on POD 3.

Case Presentation

A 73-year-old male with HFrEF, atrial fibrillation, and previous concomitant CABG and MVR was admitted for LVAD evaluation. During his workup, he was maintained on milrinone for inotropic support and a heparin drip given his history of atrial fibrillation. After the workup was completed, he underwent HM3 implantation. Pre-procedure TEE was notable for EF 10% and a severely dilated LA with significant spontaneous echo contrast. The echo contrast cleared with administration of epinephrine suggesting that no clot was present pre-implantation. The VAD was implanted without complication. Anticoagulation was initiated with warfarin on POD 1 and a heparin drip was later added due to subtherapeutic INRs. On POD 3, the patient underwent a thoracentesis for a pleural effusion and soon after became obtunded and hypotensive. At the same time, the VAD had low flow alarms. He was emergently brought to the OR for sternal exploration. TEE showed a left atrial thrombus, and the patient was put on bypass to facilitate successful removal of the clot. Despite the patient needing significant intra-op vasopressors and inotropes, VAD parameters were improved upon weaning from bypass. Postoperatively, the patient was weaned from his vasopressor and inotrope requirement and was discharged on POD 29 without further incident.

Discussion

Cardiac thrombosis in LVAD recipients is multifactorial and aligns with Virchow's triad, which describes three key factors that contribute to thrombosis: endothelial injury, stasis and hypercoagulability. This patient fit all three of these criteria with endothelial injury from recent surgery, increased stasis due to a large left atrium that did not fully decompress with VAD initiation, and hypercoagulability as evidenced by subtherapeutic INRs. The patient's complication illustrates a potential need for a more aggressive anticoagulation strategy in patients that are at elevated intracardiac thrombus risk. For instance, a risk scoring system that considers the presence of preoperative atrial dilation, spontaneous echo contrast, a prosthetic valve, and a history of anticoagulation use may have indicated the need for a more intensive postoperative anticoagulation strategy. Serial imaging such as TEE or CTs in high-risk patients could also guide the anticoagulation strategy and prompt more aggressive treatment if there were early signs of clot

formation. This could potentially prevent an acute decompensation which this patient experienced. In conclusion, the presented case suggests the need for continued research in personalized risk assessment in LVAD patients to minimize postoperative risks, especially those that impact hemostasis.

Images

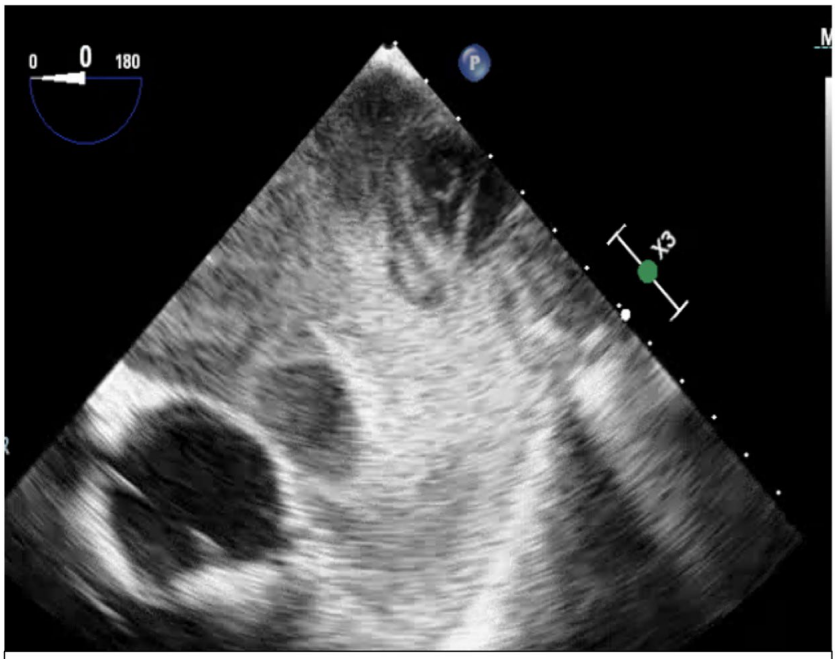


Figure 1. Severely dilated LA with spontaneous echo contrast

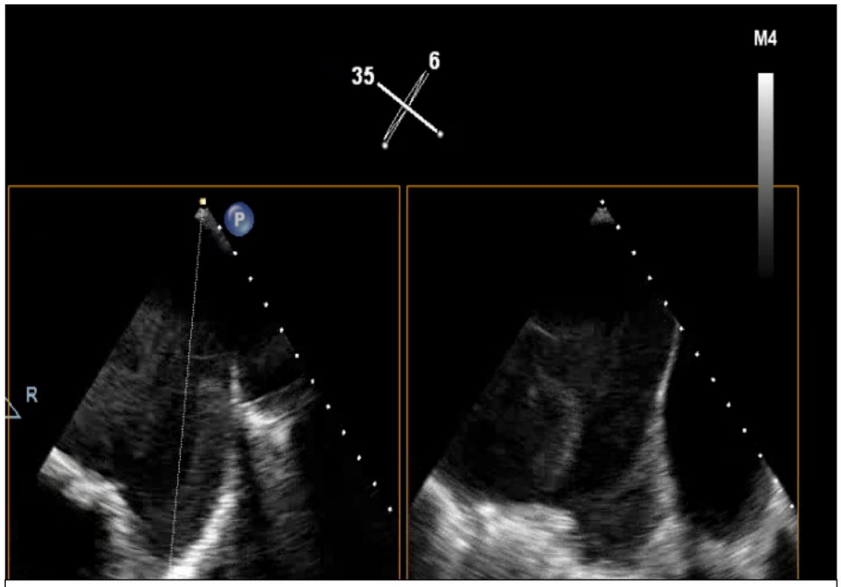


Figure 2. Post epi challenge showing clearance of spontaneous echo contrast and no apparent clot

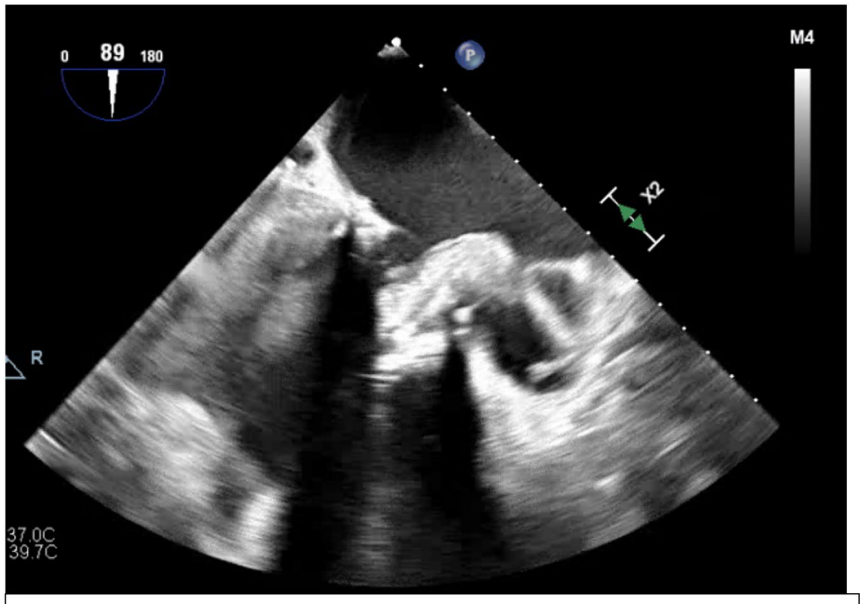


Figure 3. ME 2 chamber with left atrial thrombus

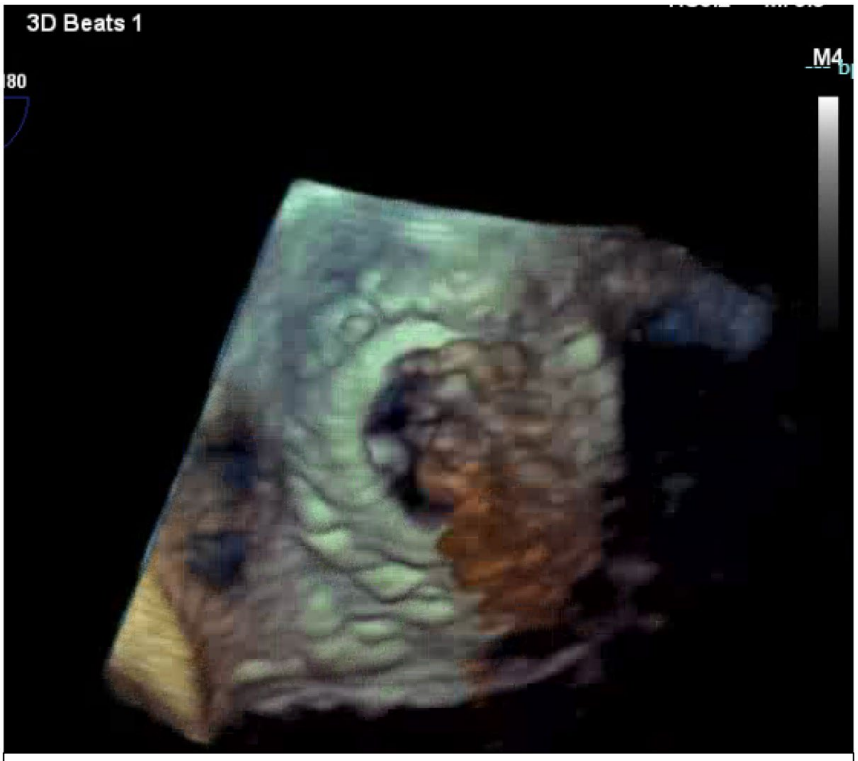


Figure 4. 3D image of clot

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#FR 1 – Kiosk 3 – Poster 4

Autograft Mismatch in the Ross Procedure: Should Obesity Be Quantified More Rigorously in Surgical Decision-Making?

Presenting Author: Felipe Nobre, MD, Clinical Fellow, The University of Western Ontario

Introduction

Prosthesis–patient mismatch (PPM) remains a major determinant of long-term outcomes after aortic valve replacement, particularly when residual gradients hinder left-ventricular reverse remodeling. Although the Ross procedure has regained prominence for its favorable hemodynamics and durability, its applicability in patients with severe or extreme obesity remains poorly characterized. Large body surface areas (BSA) may significantly alter the biomechanical environment of the pulmonary autograft, influencing wall stress, compliance, long-term dilation, and adaptive remodeling. Despite these physiologic considerations, current guidelines do not provide quantitative thresholds for autograft suitability across obesity strata. This case reports the early hemodynamic performance of a Ross procedure in a patient with extreme obesity and highlights the absence of evidence-based, body size–adjusted criteria to guide valve strategy selection.

Case Presentation

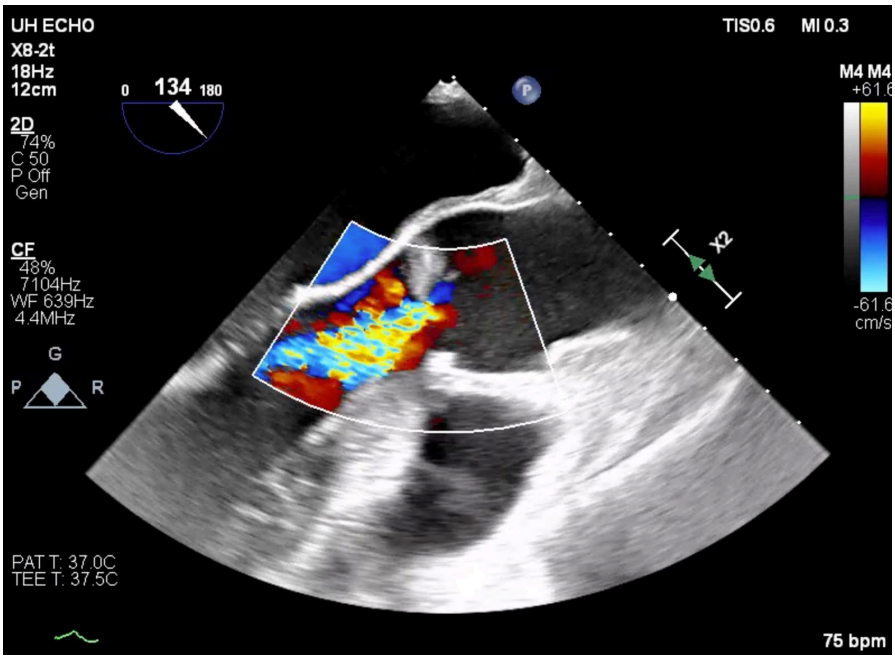
A 36-year-old woman (BMI 52 kg/m^2, BSA 2.91 m^2) with bicuspid aortic valve disease presented with moderate aortic stenosis and insufficiency and NYHA III symptoms. Preoperative echocardiography demonstrated a mean gradient of 31 mmHg, valve area 1.3 cm^2, and preserved ejection fraction. CT imaging showed an aortic basal ring area of 700 mm^2 and a pulmonic ring area of 970 mm^2. Based on annular dimensions, a standard surgical aortic valve replacement would have accepted a 21-mm prosthesis—23 mm at the upper limit—valve sizes associated with low indexed effective orifice area and a high likelihood of moderate-to-severe PPM in a patient approaching 3.0 m^2 of BSA. She underwent a Ross procedure with aortic annuloplasty and sinotubular junction stabilization using a 32-mm graft. Postoperative echocardiography revealed a peak gradient of 21.8 mmHg, mean gradient 12 mmHg, and valve area 2.3 cm^2. The patient was extubated early, experienced an uncomplicated recovery, and was discharged asymptomatic.

Discussion

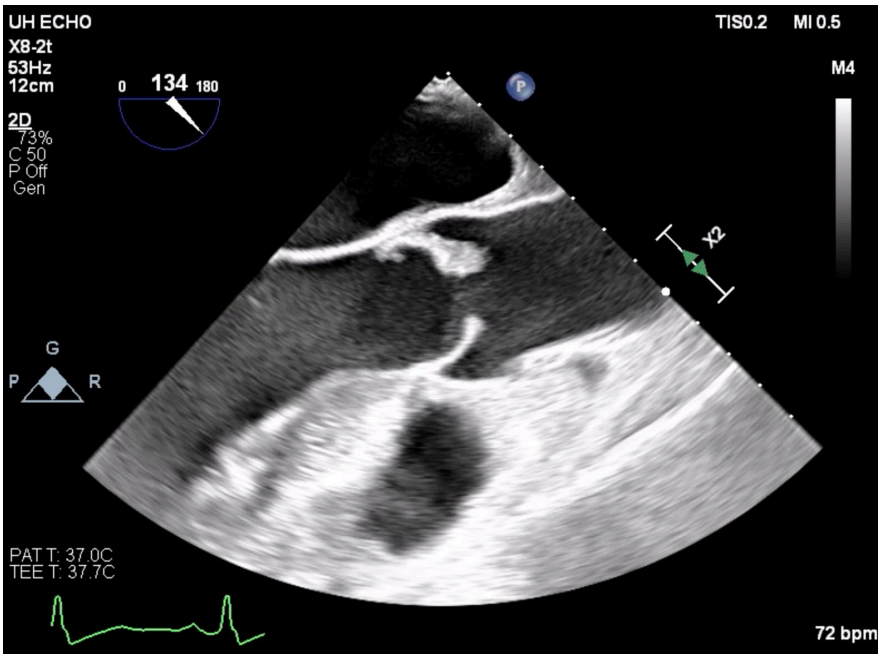
Current recommendations treat obesity as a single category (“BMI >30”), without recognizing the substantial physiologic differences between moderate, severe, and extreme obesity or their implications for autograft behavior. Existing research addressing PPM overwhelmingly focuses on prosthetic valves, leaving the relationship between BSA, autograft diameter, and long-term remodeling largely unexplored. In this case, the Ross procedure provided hemodynamics clearly superior to those expected from a 21–23 mm prosthesis, preventing the predictable PPM burden that would have occurred with standard aortic valve replacement. However, no quantitative framework exists to define the lower limits of autograft size adequacy, to estimate remodeling thresholds in large-BSA patients, or to guide surgical decision-making when obesity approaches extreme ranges. As the prevalence of severe obesity increases and the Ross

procedure gains broader indications, this evidence gap becomes increasingly relevant for individualized patient selection.

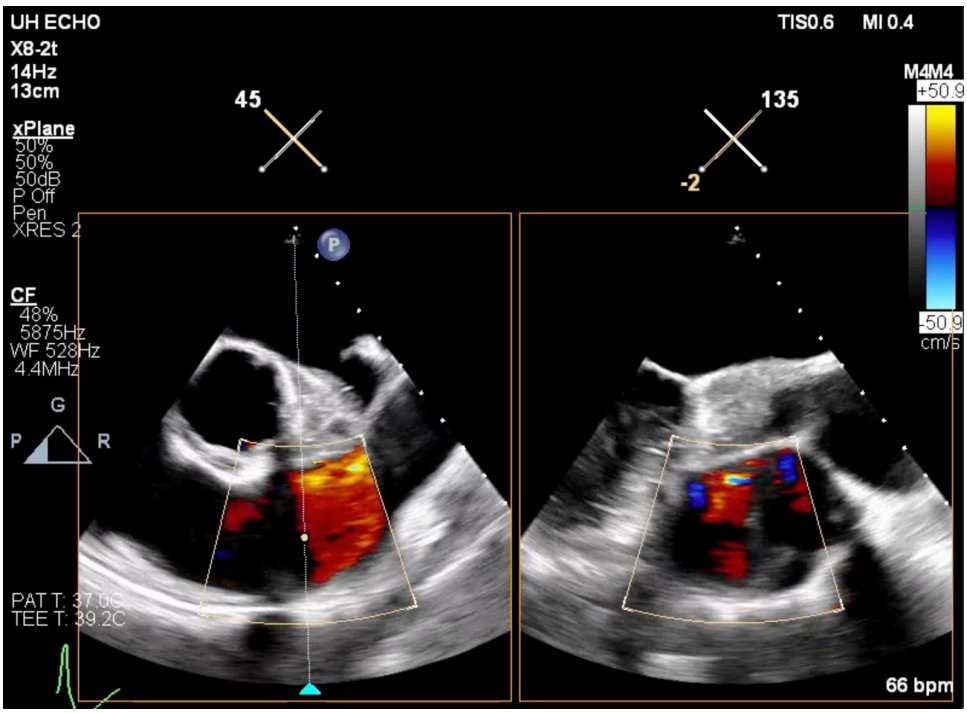
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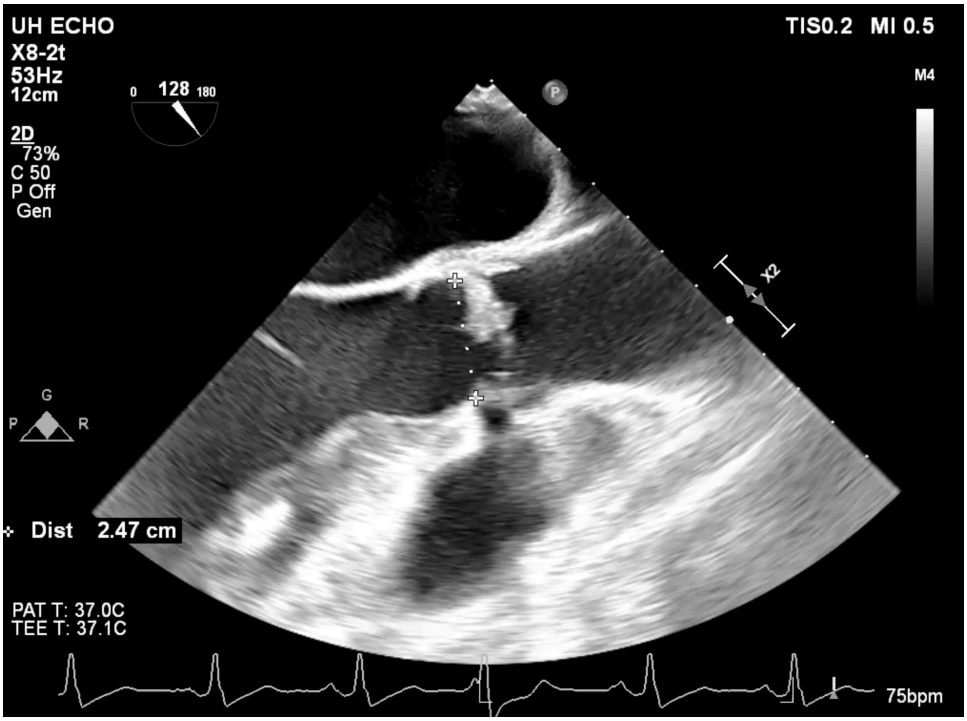
ME LAX Color Doppler focusing on Aortic Valve



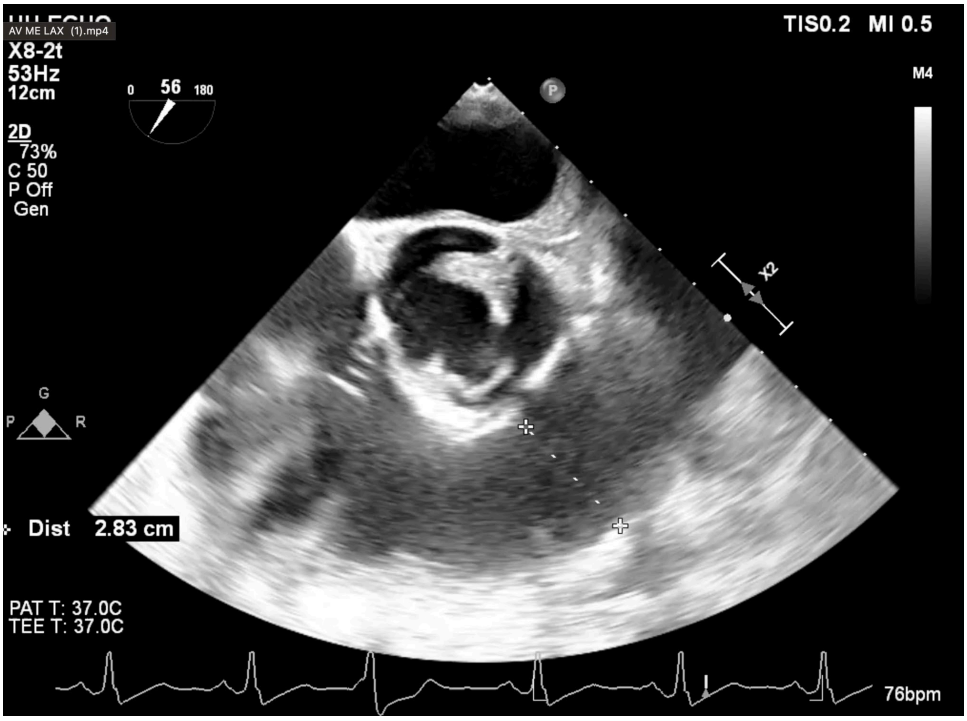
ME LAX focusing on Aortic Valve



ME RV Inflow Outflow focusing on Pulmonic Valve with Multi-plane and color Doppler



Aortic Valve Diameter on ME LAX



Pulmonic Valve diameter

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#FR 1 – Kiosk 3 – Poster 5

Epicardial and Epiaortic Echocardiography in a Post-Esophagectomy Patient Undergoing Valve-Sparing Aortic Root and Ascending Aneurysm Repair

Presenting Author: Mariam Elsaban, MBBS, anesthesiology resident, Mayo Clinic

Introduction

Prior esophagectomy with gastric conduit reconstruction is a relative contraindication to transesophageal echocardiography (TEE) because of the risk of anastomotic injury or perforation (1). In cardiac surgery, this presents a major challenge, as TEE is central to evaluating ventricular function, valvular competence, and confirming the success of surgical intervention while guiding critical intraoperative decisions (2). Alternative hemodynamic tools such as pulmonary artery catheterization and arterial waveform analysis offer useful data but lack the anatomic detail required for real-time surgical decision-making. In patients for whom TEE is unsafe, epicardial and epiaortic echocardiography provide a practical and effective alternative (3).

Case Presentation

A 74-year-old man with distal esophageal adenocarcinoma previously treated with chemoradiation and esophagectomy with gastric conduit and proximal high esophagogastric anastomosis presented for valve-sparing aortic root and ascending aortic aneurysm repair. Transthoracic echocardiography (TTE) showed a 4.9-cm sinus of Valsalva aneurysm and an enlarged ascending aorta with an effaced sinotubular junction. Additional findings included a moderately enlarged right ventricle with borderline systolic dysfunction. CT angiography confirmed a 5.5-cm aortic root aneurysm. Recent endoscopy demonstrated stenosis and ulceration at the thoracic esophagogastric anastomosis; therefore, TEE was deferred because of perforation risk. Multidisciplinary planning led to the decision to rely on epicardial and epiaortic imaging instead. General anesthesia was induced and invasive monitoring placed included a right internal jugular introducer with pulmonary artery catheter, a right radial arterial line, and a left femoral arterial line. Epicardial and epiaortic images were obtained before and after cardiopulmonary bypass to assess valvular function, cusp coaptation, ascending aortic anatomy, and biventricular performance. Following the initial bypass run, epicardial imaging revealed mild to moderate aortic regurgitation with insufficient cusp coaptation. These findings guided the decision to perform a Bentall aortic root replacement using a 29 mm bioprosthetic aortic valved conduit. Final epicardial imaging demonstrated satisfactory biventricular function and competent bioprosthesis. The patient was extubated on postoperative day 1 and discharged on postoperative day 5.

Discussion

Epicardial and epiaortic echocardiography provide valuable anatomic and functional information when TEE is contraindicated, as in patients with prior esophageal reconstruction (1). These modalities allow direct visualization of the aortic root, ascending aorta, and anterior cardiac structures, enabling assessment of cusp coaptation, ventricular performance, and valvular integrity (3). Their limitations include dependence on operator skill, brief interruptions of the operative field, and the need for open surgical exposure. This case demonstrates the utility of epicardial and epiaortic imaging in guiding intraoperative choices between valve-

sparing and valve-replacing strategies and highlights their role in complex aortic surgery when standard TEE cannot be used (2).

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#FR 1 – Kiosk 3 – Poster 6

Mitral TEER With a Twist

Presenting Author: Nicolas Cypro, MD, Cardiothoracic Anesthesia Fellow, Beth Israel Deaconess Medical Center

Introduction

Mitral valve transcatheter edge-to-edge repair (MV-TEER) is a complex intervention requiring nuanced echocardiographic guidance during multiple key procedural steps. It involves interatrial septal puncture and precise positioning of the device delivery system. A suboptimal septal puncture location can make the repair unsuccessful due to inadequate grasp of the anterior or posterior leaflet. Furthermore, is it important to anticipate how the creation of an atrial septal defect (ASD) may impact the patient's physiology. ASD closure after left-sided transcatheter procedures can have both positive and negative impacts, and the decision to do so is made on an individualized basis.

Case Presentation

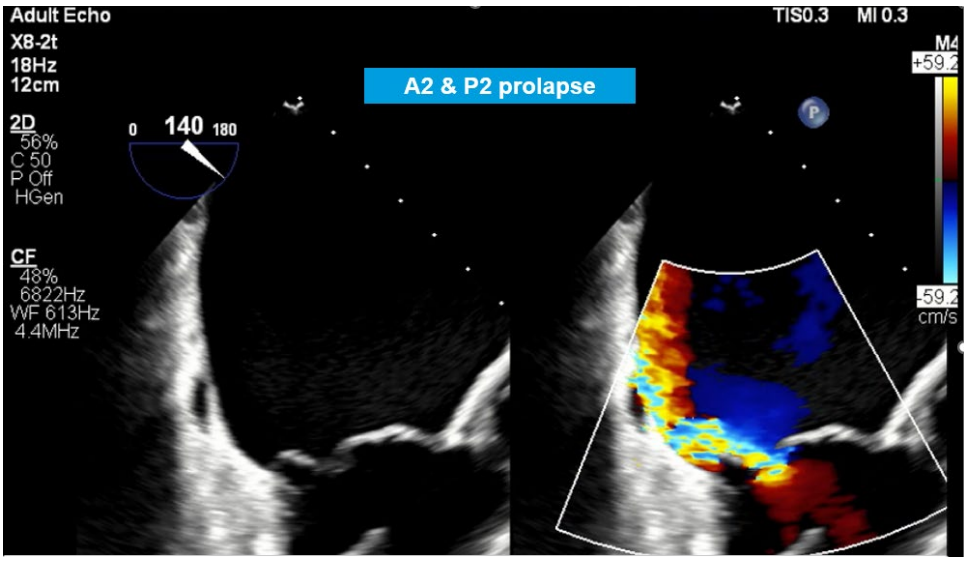
A 73-year-old male with severe symptomatic mitral regurgitation (MR) and otherwise complex medical history demonstrating prohibitive surgical risk presented for MV-TEER. Transesophageal echo (TEE) demonstrated biventricular systolic dysfunction, severe tricuspid regurgitation (TR) and massive biatrial enlargement in addition to severe MR with bilateral leaflet prolapse. A septal puncture was made in the anterior portion of the fossa ovalis measuring 4.3 cm from the mitral valve annulus. The procedural team encountered difficulty grasping the anterior leaflet of the mitral valve due to the sheath coursing from an anterior position and crossing the mitral leaflet coaptation point at an acute angle. Following several attempts, the decision was made to retract the delivery sheath back through the septum and repeat the septal puncture at a more posterior position within the fossa. Immediately when the sheath was withdrawn into the right atrium, we encountered hypoxia and hypotension. Despite ventilation with 100% oxygen, arterial blood gas demonstrated acute hypoxemia and hypercarbia. TEE with color Doppler demonstrated right-to-left shunting across the iatrogenic ASD. The procedural team urgently deployed a septal occlusion device across the defect leading to physiological improvement. We then performed a second septal puncture in a more posterior location which allowed for a more perpendicular approach through the mitral valve annulus. On this attempt both leaflets were successfully grasped and the team deployed two clips with two-grade reduction in MR severity. Upon retraction of the delivery sheath through the septum, bidirectional flow was visualized across the ASD. A second septal occluder was not deployed at this time given reassuring oxygenation, ventilation, and hemodynamics with minimal oxygen support and spontaneous ventilation. The patient's postoperative course was uneventful and he discharged the following day.

Discussion

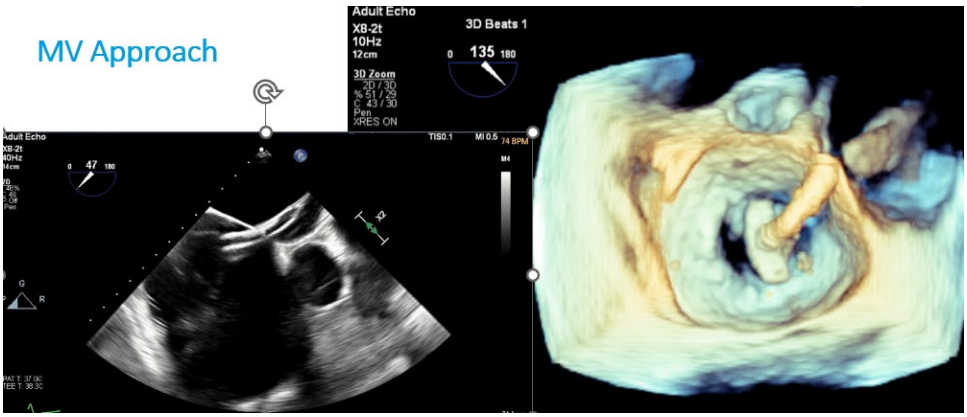
This case highlights the impact that an optimized septal puncture location within the fossa ovalis can have on procedural success. For our patient, an anterior septal puncture did not allow for a perpendicular

approach through the mitral valve, leading to difficulty grasping the anterior mitral leaflet. On the subsequent attempt from a more posterior position, a perpendicular approach through the mitral valve allowed successful leaflet grasp and device deployment. Additionally, it is important to anticipate physiologic disturbances that can result from creation of an iatrogenic ASD and to determine when deploying a closure device may be appropriate.

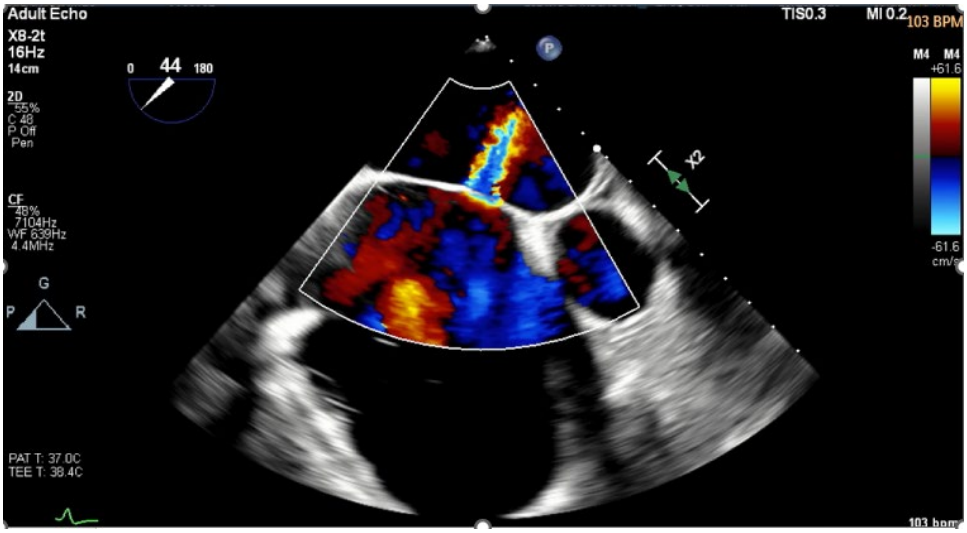
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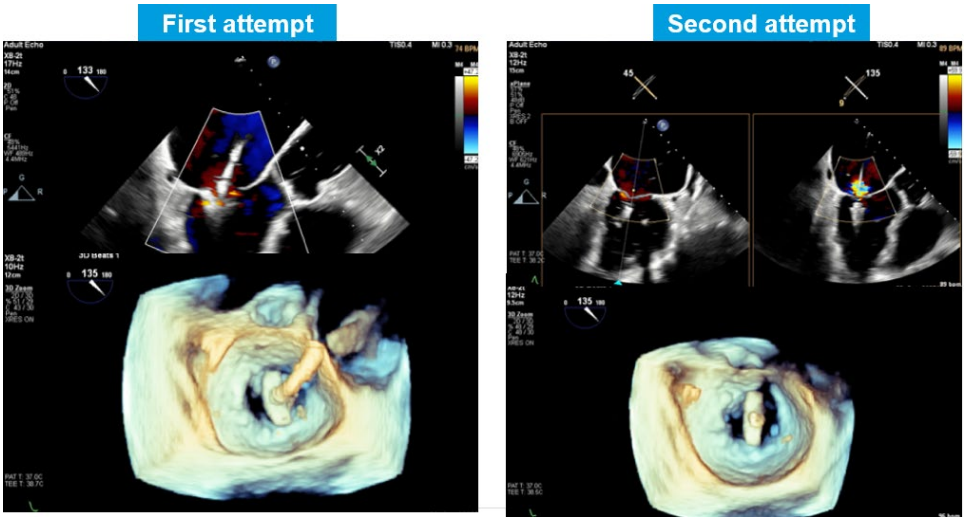
Initial evaluation of the mitral valve demonstrating severe regurgitation with an eccentric posteriorly directed jet and bi-leaflet prolapse



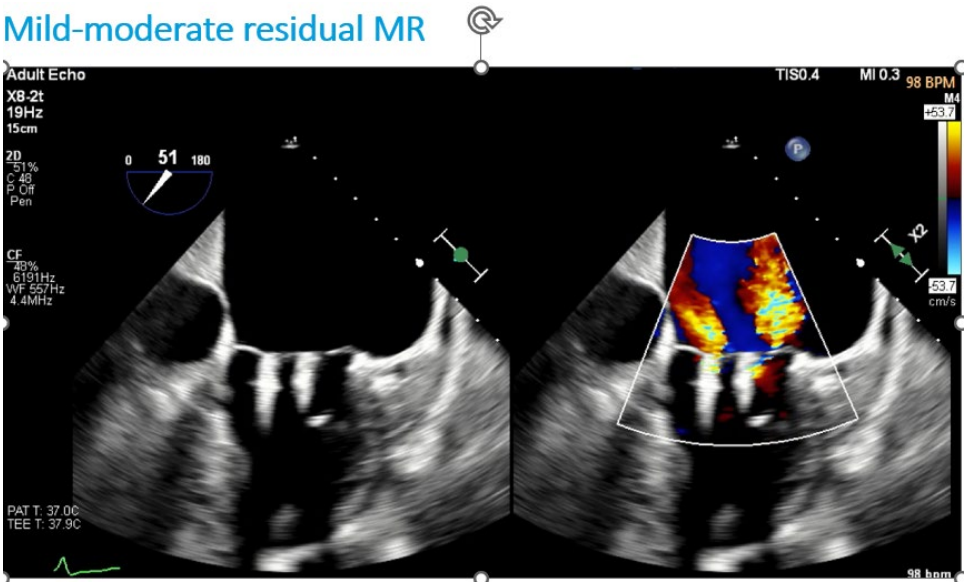
2D and 3D images demonstrating septal puncture location within the interatrial septum and the delivery sheath angle approaching the mitral valve



Right-to-left shunt through the iatrogenic ASD created following withdrawal of the device delivery sheath after unsuccessful first TEER attempt



Comparison of delivery sheath approach angles through the mitral valve during first (unsuccessful) and second (successful) TEER attempts, emphasizing the importance of crossing through the mitral valve annulus in a perpendicular orientation



Result following deployment of two mitral clips with mild-moderate residual MR

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#FR 1 – Kiosk 4 – Poster 1

Cardiac Surgery in a Patient with Lupus Myocarditis, Triple-Positive APS, G6PD Deficiency, and a Rare S–s–/U– Blood Type: A Multidisciplinary Challenge

Presenting Author: Basheer Kayali, M.D., Resident, Lahey Hospital and Medical Center

Introduction

Systemic lupus erythematosus (SLE) and antiphospholipid syndrome (APS) can produce severe cardiac complications, including lupus myocarditis and valvular lesions. In APS, the hypercoagulable state during cardiopulmonary bypass (CPB) confers heightened risks of thrombosis and bleeding [1]. We present a 22-year-old woman with lupus myocarditis and triple-positive APS who required urgent mitral valve replacement (MVR). Her coexisting glucose-6-phosphate dehydrogenase (G6PD) deficiency, an extremely rare S–s–/U– red cell phenotype, and a preoperative hematocrit of 21.5% created additional anesthetic, anticoagulation, and transfusion challenges.

Case Presentation

The patient presented in cardiogenic shock due to lupus myocarditis and acute severe mitral regurgitation. She had diffuse alveolar hemorrhage requiring intubation and a recent right-leg deep vein thrombosis from APS. A percutaneous mitral repair (MitraClip) was attempted as a bridge but worsened the regurgitation, necessitating emergent surgical MVR. Given her tenuous status, the team prepared for rapid venoarterial ECMO cannulation as a contingency during induction and cardiopulmonary bypass. Intraoperative management prioritized hemodynamic stability and blood conservation. Induction was carefully titrated with ongoing inotropes, and vasopressors were adjusted to treat CPB-related vasoplegia (methylene blue was avoided due to G6PD deficiency). Anticoagulation on bypass used high-dose heparin targeting an ACT >2× baseline in the presence of a lupus anticoagulant [1]. Antifibrinolytics were administered to reduce bleeding, and only limited compatible blood was available—advance coordination with the blood bank was essential given the patient's S–s–/U– phenotype [2]. The patient underwent bioprosthetic MVR with tricuspid repair and was weaned from CPB without ECMO support. Postoperatively, she was managed in the ICU with continued steroids, extubation on POD 5, and cautious resumption of heparin under multidisciplinary guidance, ultimately recovering to discharge.

Discussion

This case highlights the importance of meticulous planning and multidisciplinary collaboration for patients with such complex comorbidities. APS confers high risk of intraoperative thrombosis and hemorrhage, warranting careful anticoagulation management and specialized monitoring [1]. Additionally, the patient's erythrocyte phenotype significantly restricted compatible blood availability, posing risks of alloimmunization and future transfusion delay [2]. A team-based approach was crucial: input from cardiology, rheumatology, hematology, and transfusion medicine informed optimal timing of surgery, perioperative immunosuppression, blood product preparation, and anticoagulation strategies. Anesthetic management required readiness for acute deterioration (including ECMO standby) and avoidance of contraindicated

agents (e.g., oxidant drugs in G6PD deficiency). Proactive coordination with the blood bank ensured availability of rare compatible units for this S–s–/U– patient [2]. In summary, thorough preparation and interdisciplinary care were essential for a successful outcome in this challenging case.

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#FR 1 – Kiosk 4 – Poster 2

Severe Biventricular Dysfunction and ECMO-Associated Tricuspid Valve Thrombosis After Redo Replacement: A Case of Coronary Vasospasm, Mechanical Support Challenges, and Multimodal Management

Presenting Author: Hai-yen H. Nguyen, MD, Anesthesia Fellow, Department of Anesthesiology, University of Texas-Houston Anesthesiology

Introduction

Numerous complications can arise after cardiac surgery, and valve surgery adds a unique set of potential issues. We present a case of not only perioperative LV failure following redo tricuspid valve surgery in which coronary vasospasm was suspected, but also thrombosis of the new valve after initiation of ECMO. This case highlights clinical and diagnostic reasoning and a multimodal rescue strategy leading to complete recovery.

Case Presentation

A 37-year-old woman with prior tricuspid valve replacement for drug-associated endocarditis presented with prosthetic valve and pacemaker infection. Preoperative TTE showed normal biventricular function and severe tricuspid stenosis. She underwent redo tricuspid valve replacement with pacemaker extraction. After bypass, TEE revealed new severe LV dysfunction with global hypokinesis and a moderate to severely impaired RV. Coronary ostia appeared patent, but coronary sinus backflow was reduced. The prosthetic valve was explanted, though backflow remained diminished. After implantation of a new valve, targeted nitrate infusion into the coronary sinus produced immediate and reproducible improvement in LV contractility, supporting suspected coronary vasospasm. Verapamil and dobutamine were initiated, but the patient progressed to cardiogenic shock requiring VA-ECMO. Coronary angiography confirmed patent epicardial vessels without flow-limiting lesions. Three days later, during planned ECMO decannulation, intraoperative TEE revealed a decompressed RV with no forward flow across the tricuspid valve during weaning of ECMO. High suspicion of valve thrombosis prompted re-initiation of bypass. The bioprosthetic tricuspid valve was found to be thrombosed, with thrombus in the RV as well. The valve was replaced again and the thrombus evacuated. The patient was weaned from bypass with improved biventricular function and decannulated from ECMO. By postoperative day 7, TTE demonstrated full recovery of both ventricles with stable prosthetic valve performance.

Discussion

Perioperative coronary vasospasm is a rare yet important cause of acute ventricular dysfunction after cardiac surgery, and may mimic epicardial obstruction or myocardial injury. It may be especially difficult to recognize after right-sided procedures1. Global hypokinesis, preserved coronary ostia, reduced coronary sinus backflow, and rapid improvement with nitrates support a dynamic vasomotor process 1,2. Early therapy with nitrates, calcium channel blockers, and optimization of sympathetic and metabolic factors is essential. In refractory cases, VA-ECMO with LV unloading can preserve coronary perfusion and allow

myocardial recovery2. This case also highlights a known consequence of postcardiotomy ECMO: low pulsatility and escalating ECMO flows can reduce cardiac filling, promoting intracardiac stasis and prosthetic valve thrombosis3. These factors likely contributed to both the tricuspid prosthesis clot and the RV thrombus. While supporting the coronary spasm effects, VA-ECMO had its own consequence for the new valve. Maintaining suspicion for vasospasm, initiating early vasodilator therapy, recognizing ECMO-related stasis phenomena, and close communication among anesthesia and surgical teams were crucial in managing unexplained post-bypass LV failure and valve dysfunction.

Images

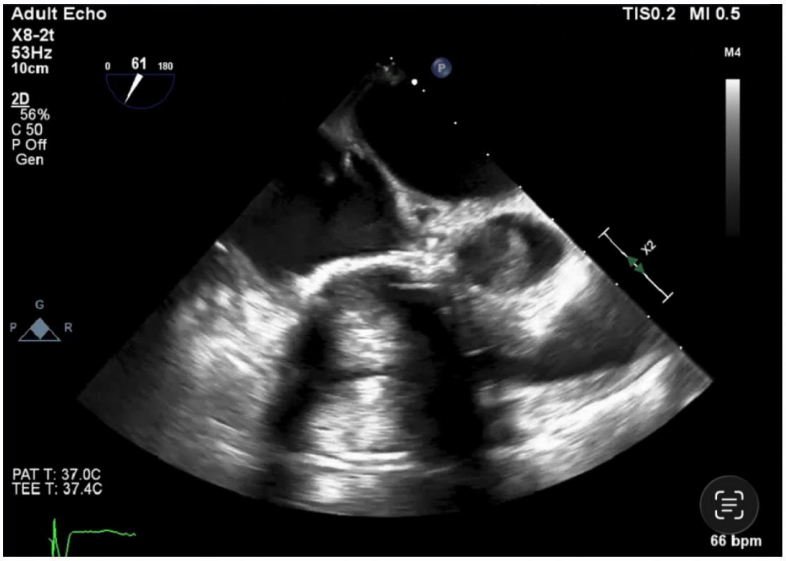


Figure 1: Stenosed tricuspid valve during index case

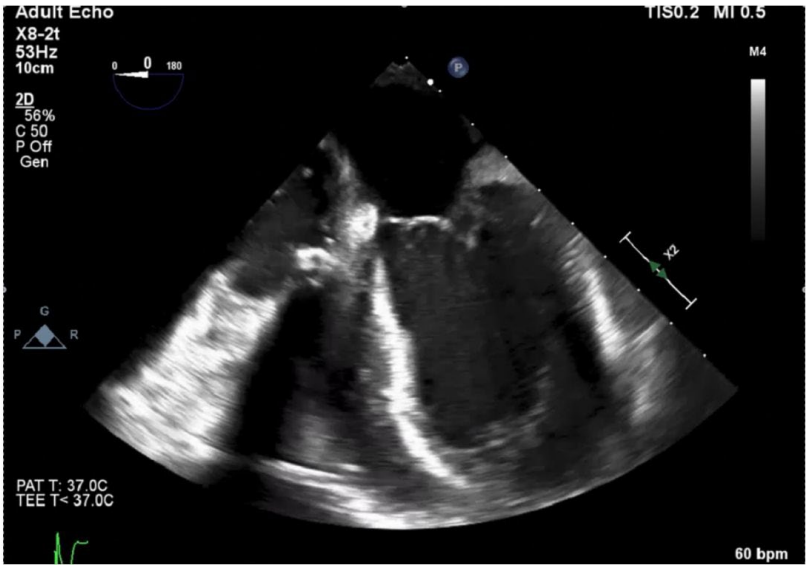


Figure 2: Decompressed right heart during temporary cessation of ECMO flows

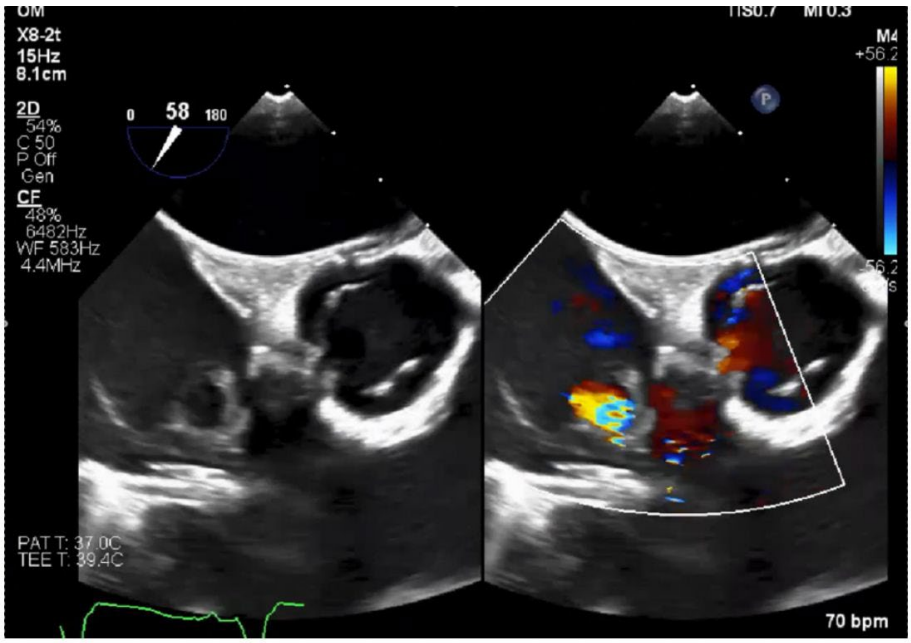


Figure 3: Thrombus visualized on tricuspid valve during OR takeback POD 3 for ECMO decannulation

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#FR 1 – Kiosk 4 – Poster 3

Thermistor port bleeding as an early sign of pulmonary artery catheter entrapment

Presenting Author: Catherine Ha, MD, Resident Physician, NewYork-Presbyterian/Columbia University Medical Center

Introduction

Pulmonary artery catheters (PACs) during cardiac surgery allow for continuous hemodynamic monitoring of measures such as cardiac output and right-sided intracardiac pressures. Broadly, complications associated with PACs include inadvertent trauma, arrhythmias, infection, thrombosis, and misinterpretation of data.[1] Entrapment of the PAC in a surgical suture line is a rare but dangerous complication that is underrecognized intraoperatively. We describe a case of PAC entrapment which presented with bleeding from the thermistor port and confirmed using TEE.

Case Presentation

A 69-year-old male with a history of multi-vessel coronary artery disease and mitral valve prolapse with severe mitral regurgitation presented for elective coronary artery bypass grafting, mitral valve replacement, and patent foramen ovale (PFO) closure. After induction of general anesthesia, the patient underwent uncomplicated placement of a central line with a PAC. A bicaval cannulation strategy was used for cardiopulmonary bypass (CPB). During CPB, transient leaking of blood was noted from the thermistor port of the PAC (Image 1). Soon after separation from CPB, routine mobilization of the PAC demonstrated resistance without changes in the transduced pressure waveforms. On TEE, the PAC appeared to be attached to the right atrial/superior vena caval junction (Image 2). Despite revisions to the superior and inferior vena cava cannulation sutures, the PAC remained tethered. After reinstating CPB, the PAC was found to be sutured to the PFO closure site. The PAC was cut and removed by the surgical team, and the remainder of the surgery proceeded uneventfully. The patient's post-operative course was notable for moderate reduction of his ejection fraction requiring inotropy until post-operative day (POD) 6, after which his ejection fraction recovered to normal. He also experienced variability in his heart rhythm, including atrial fibrillation with intermittent rapid ventricular rate and conversion pauses. He was evaluated by the Electrophysiology team and a dual chamber pacemaker was placed uneventfully on POD 13. He was discharged home on POD 14.

Discussion

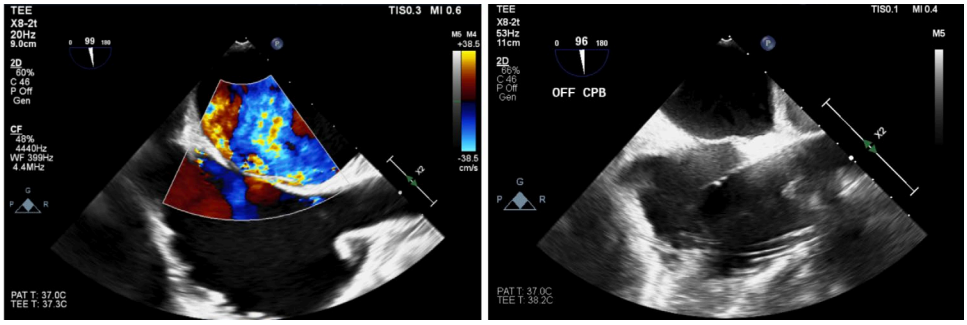
This case highlights that bleeding from the thermistor port is an underrecognized sign of possible PAC entrapment in a surgical suture line.[2,3] Kumar et al. (2025) recently reported the overall incidence of PAC entrapment to be 0.068%, primarily due to inadvertent suturing and in the setting of mitral valve surgery. Of these cases, 77% were discovered post-operatively, the majority of which required re-do sternotomy with associated increased risks of surgical complications and costs. Of the case reports analyzed, three described bleeding from the thermistor port, which were associated with puncturing of the PAC by sutures.[1] The complication described here was discovered with routine mobilization of the PAC prior to chest closure, confirmed with TEE, and corrected during the intraoperative period, though requiring re-initiation of CPB.

Furthermore, bleeding from the thermistor port was noted during the initial run of CPB, supporting its utility as an early indicator of inadvertent suturing of the PAC, with the potential for avoiding morbidity associated with re-initiation of CPB and/or re-do sternotomy.

Images



Blood noted in thermistor port during 1st CPB run.



TEE images in mid-esophageal bicaval view. Left image shows the PFO pre-closure. Right image shows PAC tethered near SVC/RA junction after PFO closure.

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#FR 1 – Kiosk 4 – Poster 4

Leiomyosarcoma of the heart: atypical presentation of persistent nausea and vomiting

Presenting Author: Mackenzie M. Geisman, MD, Resident physician, Los Robles Regional Medical Center

Introduction

Primary cardiac tumor occurrence is rare with a prevalence between 0.001–0.28%, and sarcomas make up a majority of malignant cardiac tumors1. Sarcoma tumors can originate from the heart, pericardium, or the surrounding great vessels. Common symptoms of cardiac tumors include intracardiac obstruction, signs of systemic embolization, stroke, myocardial infarction, or arrhythmias. We present a case of a patient diagnosed with a leiomyosarcoma presenting for progressive nausea and vomiting symptoms.

Case Presentation

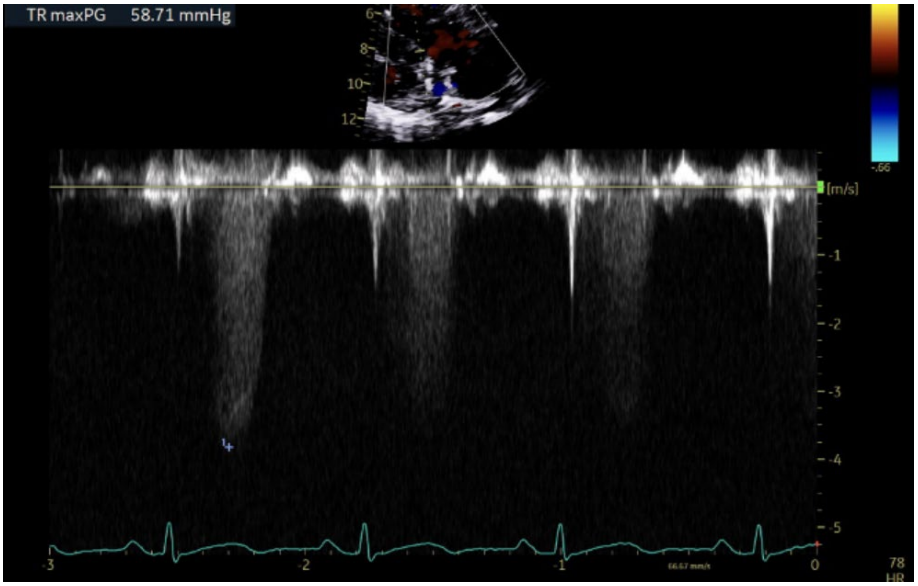
77-year-old female past medical history of hypertension, acid reflux, diabetes type 2, 25-pack year smoking history presented to ER for assessment for progressive 2-month nausea and vomiting that interfered with activities of daily living. Outpatient cardiac work up 3 months ago was unremarkable. Patient denied fevers, chills, weight loss, hemoptysis, hematemesis, recent travels, or recent illness. TTE showed elevated pulmonary pressures and severe tricuspid regurgitation. CTA chest showed mass compression of distal SVC and right inferior pulmonary vein with mediastinal and right hilar lymphadenopathy. Upper esophageal ultrasound and found a large left atrial mass. TEE showed large nonhomogeneous infiltrative mass with irregular borders in left atrium with obstruction of left and right pulmonary veins drainage. No mitral valve involvement. Pleural serology unremarkable for autoimmune and infectious causes. Patient subsequently underwent transeptal biopsy using intracardiac echocardiogram. Pathology reports left atrial mass as Leiomyosarcoma/Rhabdomyosarcoma, and patient subsequently transferred to a tertiary hospital for higher level of care with dedicated multidisciplinary sarcoma teams.

Discussion

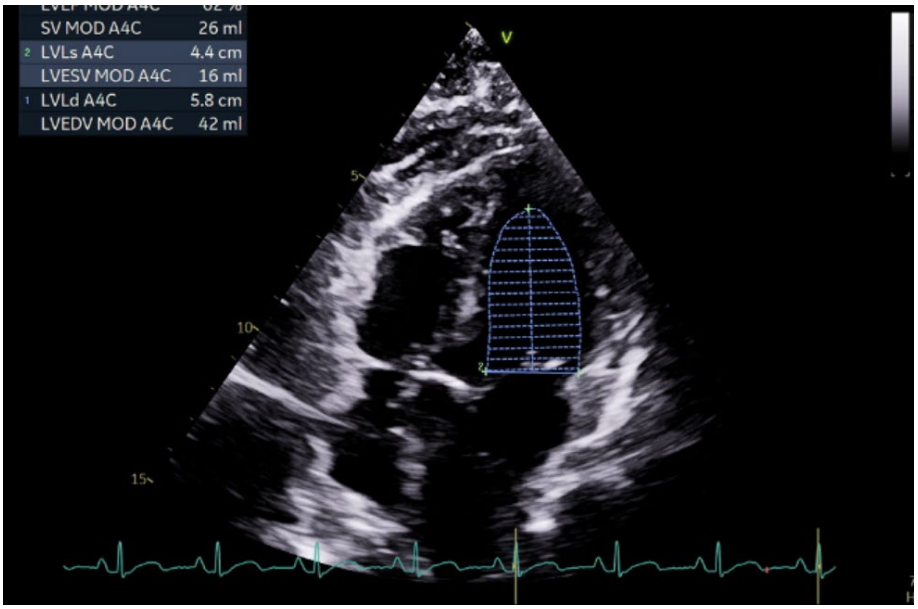
Primary cardiac tumors are rare and only 5-6% of these tumors are malignant. Among all malignant primary cardiac tumors, leiomyosarcomas account for less than 1%2. It is a highly aggressive condition, and the incidence is twice as common in females. The presentation of primary cardiac tumors varies, and the classic triad includes symptoms of intracardiac obstruction, systemic embolization signs, and systemic or constitutional symptoms. Here we present an unusual case of primary cardiac leiomyosarcoma in a patient presenting with 2 months of progressive nausea and vomiting. Initial assessment with TTE is critical in establishing the location, mobility, size, and pericardial condition of the tumor. TEE and three-dimensional echocardiography can be used for a more accurate evaluation of the mass. Pathology and immunohistochemistry confirm the diagnosis. These tumors have a poor prognosis, with reported mean survival time of 6 months without treatment3. Tumor resection with wide margins is the primary objective of treatment, and surgical intervention can be complicated by hemodynamic compromise due to mass effect, cardiac arrhythmia, and embolism. Patients with complete resections have an average survival rate of 24-34 months, while those with incomplete resections have a shorter survival time of 6-12 months. This is a rare,

aggressive condition with a poor prognosis. Early diagnosis and evaluation with cardiac imaging and histopathological confirmation is crucial for patient outcomes, and complete resection of the neoplasm is the preferred treatment.

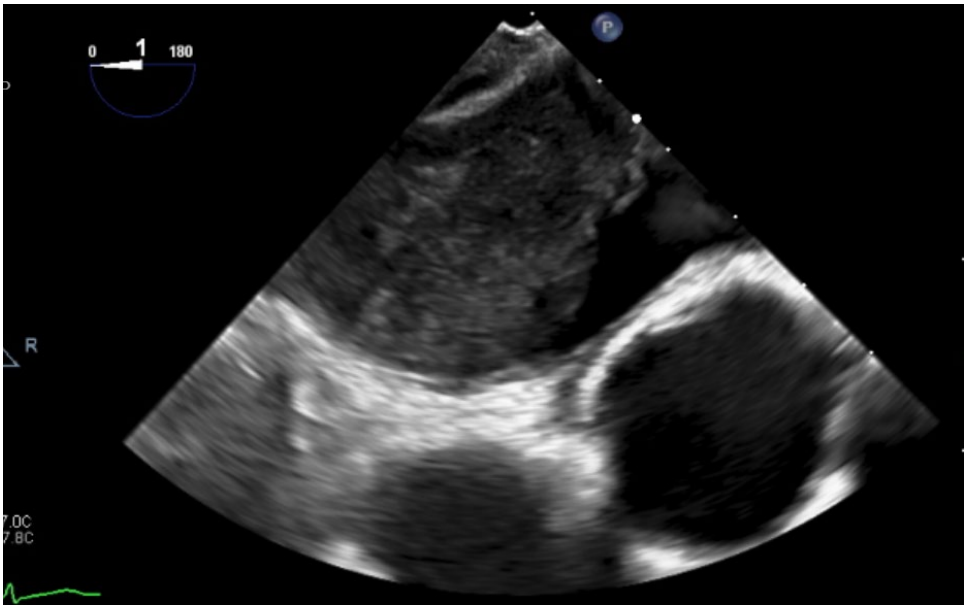
Images



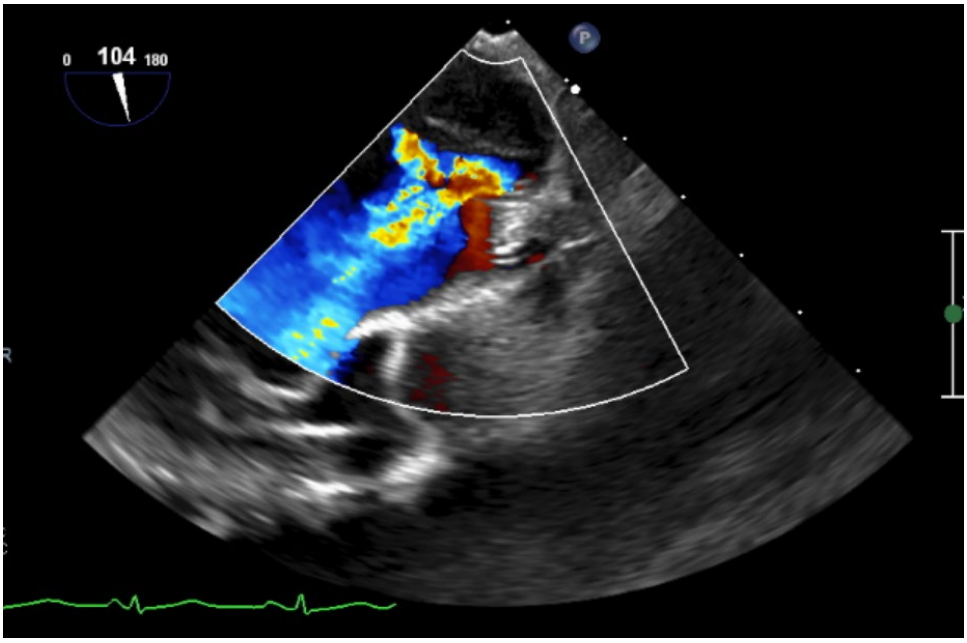
TTE measurement of tricuspid velocity, demonstrating elevated tricuspid velocity



TTE measurement in 4 chamber view. The RV is similar in size to the LV, suggesting pulmonary hypertension.



TEE measurement in mid-esophageal 4-chamber view, demonstrating left atrial mass behind the aorta.



TEE measurement of left pulmonary vein demonstrating turbulent flow caused by an obstructing mass.

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#FR 1 – Kiosk 4 – Poster 5

Acute RV Failure Due to Right Coronary Ostial Obstruction After Commando Procedure

Presenting Author: Melissa J. Kimlinger, MD, Anesthesiology Resident, Massachusetts General Hospital

Introduction

The Commando involves aortic and mitral valve replacement with reconstruction of the aorto-mitral curtain. It is typically performed for patients with infective endocarditis, extensive fibrous calcification – often related to chest radiation – or patient-prosthesis mismatch. Due to surgical complexity, prolonged cardiopulmonary bypass times, and the medically high-risk population in whom it is performed, the procedure carries substantial perioperative risk. A thorough understanding of the surgical anatomy, operative steps, and vigilant echocardiographic evaluation is essential for identifying and managing unexpected complications.

Case Presentation

A 38-year-old female with a history of aortic and mitral valve stenosis, status post aortic and mitral valve replacement, presented with bacterial endocarditis and a recurrent mitral valve vegetation (Figure, Panel A) and root pseudoaneurysm beneath the right coronary artery (RCA) ostium. She underwent redo sternotomy for a Commando procedure. Induction of anesthesia proceeded uneventfully. Monitors included arterial line, pulmonary artery catheter, and transesophageal echocardiography (TEE). Redo sternotomy and initiation of cardiopulmonary bypass were uneventful and bioprosthetic valve replacements, aorto-mitral curtain reconstruction, and root abscess debridement with bovine pericardial patch were performed. She was weaned from cardiopulmonary bypass and decannulated without complication. During closure, the patient developed escalating vasopressor and inotropic requirements, and TEE revealed severe right ventricular (RV) dilation with systolic and diastolic septal bowing (Panel B, C). Assessment of the RCA inflow was limited by acoustic shadowing from the bioprosthetic mitral valve however given the pseudoaneurysm's proximity to the RCA ostium, there was concern for RCA obstruction and an off-pump right internal mammary artery-RCA bypass was performed. Restoration of RCA flow resulted in improvement in RV function and reduced hemodynamic requirements (Panel D). Postoperatively, the patient developed a sinus pause requiring 1 minute of chest compressions and a temporary pacemaker which was later transitioned to a leadless pacemaker.

Discussion

Patients undergoing Commado procedures are vulnerable to myocardial ischemia, ventricular dysfunction, and conduction system injury due to the operative area's proximity to the coronary arteries and conduction system. Familiarity with each patient's anatomy and the specific surgical steps – including adjunctive repairs such as RVOT pseudoaneurysm patch – is essential, as these additional interventions can predispose patients to complications such as coronary obstruction, fistula formation, or acute RV ischemia. Up to 50% of patients demonstrate at least mild postoperative RV dysfunction following Commando procedure, and intraoperative TEE is critical for differentiation of ischemia, acute RV failure, or elevated afterload states. Sudden changes in hemodynamic or vasopressor requirements should immediately

prompt a focused TEE examination and multidisciplinary discussion. Postoperative vigilance is equally important. Conduction abnormalities may evolve over days due to surgical inflammation, edema, or ischemia, with up to one-third of patients ultimately requiring pacemaker placement.

Images

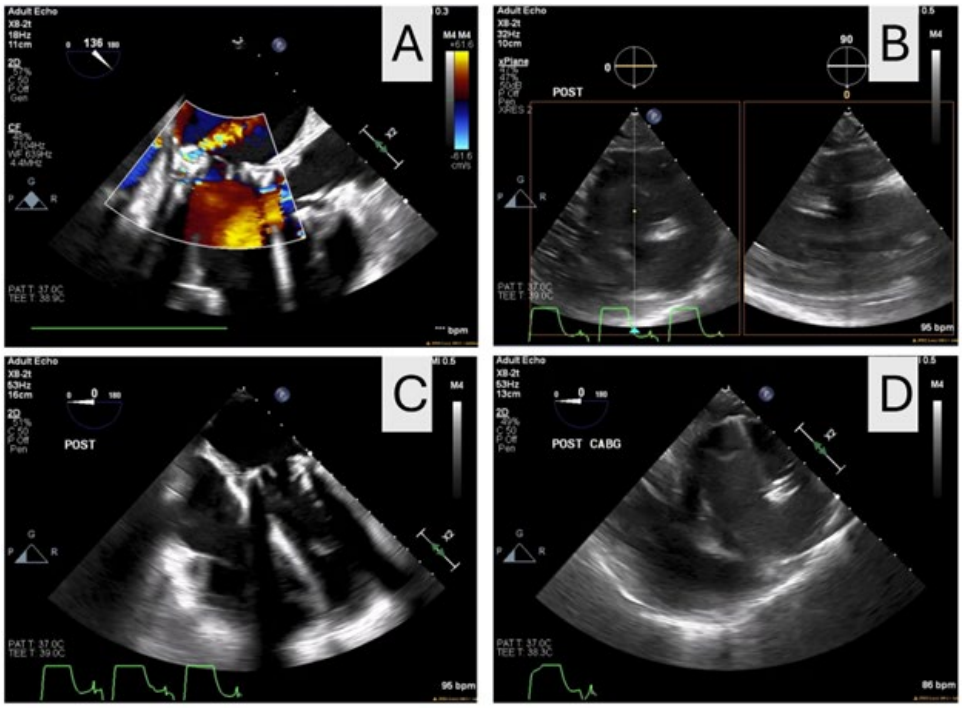


Figure 1: Intraoperative TEE Images. A) Preoperative long axis view of mitral valve fistula B) Post-cardiopulmonary bypass long and short axis views of RV dilatation and failure C) Four chamber view of RV dilation and failure D) Post RIMA-RCA bypass view of resolution of RV dilation

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#FR 1 – Kiosk 4 – Poster 6

Perioperative Interruption of Pacemaker Integrity in the Setting of Pacemaker Dependency

Presenting Author: Megan Koenig, MD, Resident Physician, University of Florida

Introduction

A permanent pacemaker (PPM) is one type of cardiac implantable electronic device used in patients with significant bradyarrhythmia. PPM dependency is defined as the absence of adequate intrinsic rhythm to maintain hemodynamic stability without device support. PPM revision and explanation procedures have significantly increased over the past decade due to device malfunction or infection. With the rising prevalence of lead extraction procedures, it is imperative to establish standardized protocol or management strategies in the perioperative setting, particularly in patients who are fully depended on PPM function.

Case Presentation

A 67-year-old male with history of hypertension, coronary artery disease, and atrial fibrillation underwent PPM implantation due to complete heart block and has remained 100% pacemaker dependent. He presented to an outside hospital with bacteremia, and transesophageal echocardiogram (TEE) revealed vegetations on his device leads. He underwent a thrombectomy of the vegetations before being transferred to our facility for removal of the infected device. Transcutaneous pacing pads were applied to the patient and a temporary screw-in pacing lead was placed prior to extraction of the infected lead. However, the operation room (OR) team did not ensure the temporary pacing lead worked with the pacer box prior to removal. Due to concern of electrocautery interference with the permanent pacer, the surgeon removed the generator from the leads which resulted in immediate asystole. Chest compressions were initiated while external pacing was attempted; however, transcutaneous pacing demonstrated intermittent capture and was unable to maintain hemodynamic stability. As the surgical team attempted to place a transvenous pacing lead, the appropriate adaptor cable was located and connected to the existing screw-in lead, restoring circulation and hemodynamic stability. The lead extraction procedure then proceeded without further complications.

Discussion

Whenever a patient is PPM dependent, any disruption in PPM function would place the patient at significant risk. It is essential to prepare other temporary pacing modalities and to provide alternative circulatory support during removal of an infected device. In this case, the OR team initially applied external pacing pads and a screw-in lead as backup support in anticipation of interrupted PPM function. Failure of transcutaneous pacing may result from operator error, improper pad placement, inadequate skin contact, insufficient output current, or patient factors such as obesity. Emergent placement of a transvenous pacing lead may be time consuming, and occasionally the lead may become dislodged during permanent lead extraction, making it a less attractive first option. A temporary screw-in lead was an effective method for maintaining cardiac rhythm, however requires the appropriate adaptor cable to connect with the pacing box. In this case, interruption of circulation was minimized through prompt manual chest compressions and

systematic evaluation of alternative pacing strategies. This event also highlighted the important of clear communication among the OR team and need for a predefined plan to ensure reliable means of temporary pacing support in pacemaker-dependent patients.

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#FR 1 – Kiosk 4 – Poster 7

Pulmonary Embolism to Alveolar Hemorrhage: Two sides of an Open Embolectomy

Presenting Author: Tyler J. Fraga, MD, Resident, Medstar Georgetown University Hospital

Introduction

A 78-year-old female with no significant past medical history was found to have a saddle pulmonary embolism, severely dilated RV with RV strain and small mobile mass in the RA. While coming off of cardiopulmonary bypass (CPB) following a successful open embolectomy, large volume hemorrhage was noted out of the endotracheal tube requiring massive transfusion and cannulation for central VA ECMO.

Case Presentation

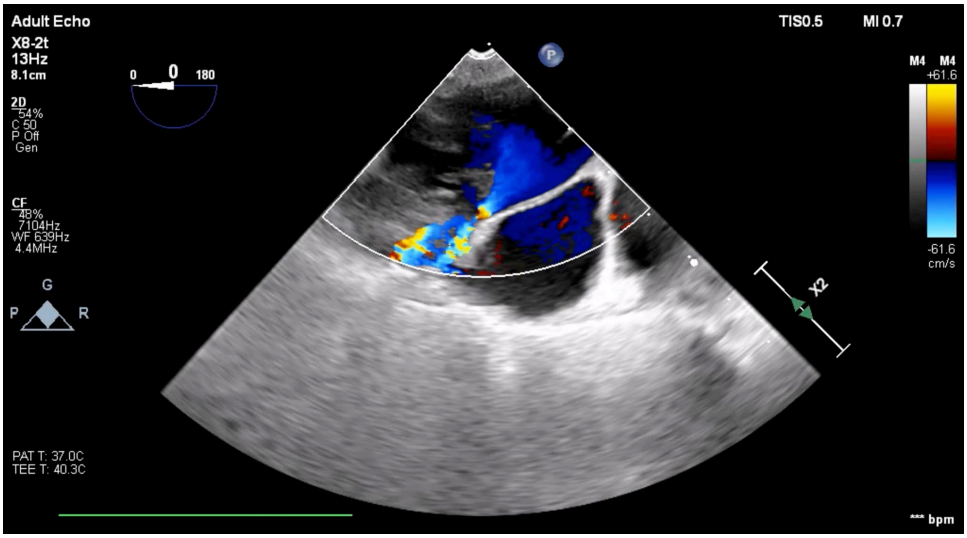
A 78-year-old female presented with generalized weakness. Vital signs showed tachycardia and hypotension prompting sepsis workup including CTA CAP. Imaging was significant for bilateral pulmonary emboli with saddle pulmonary embolism and RV strain. Due to risk of clot dislodging, IR was unable to perform thrombectomy and the patient was transferred for open embolectomy. Prior to induction, high doses of epinephrine and dobutamine infusions were started for inotropic support. GA was induced with Etomidate, Fentanyl and Versed. Endotracheal Milrinone was administered upon airway securement. Cannulation for CPB and open embolectomy proceeded in standard fashion. After successful clot evacuation, the patient was weaned from CPB. At this time, large volume bleeding was noted out of the ET tube into the breathing circuit with concern for alveolar hemorrhage vs pulmonary artery injury. CPB was re-initiated and a massive transfusion protocol was initiated with a Belmont Rapid Transfuser. Using ROTEM, the patient's coagulopathy and acute blood loss was managed with 13 PRBC, 8 FFP, 4 Cryo, 2 Platelets and 1000 units of KCentra. The decision was then made to cannulate for central VA ECMO due to ongoing RV dysfunction and inability to ventilate/oxygenate. Once on ECMO, the patient was separated from CPB and Protamine reversal was administered with resolution of alveolar hemorrhage. The patient was then transported to the CVICU. She returned to the OR for sternal closure on POD 3, de-cannulated from ECMO on POD 7, and extubated on POD 10. Her remaining hospital course was complicated by pericardial effusion requiring surgical washout and unstable AFib with RVR requiring cardioversion x 3. The patient decided to transition to comfort care and passed on POD 17.

Discussion

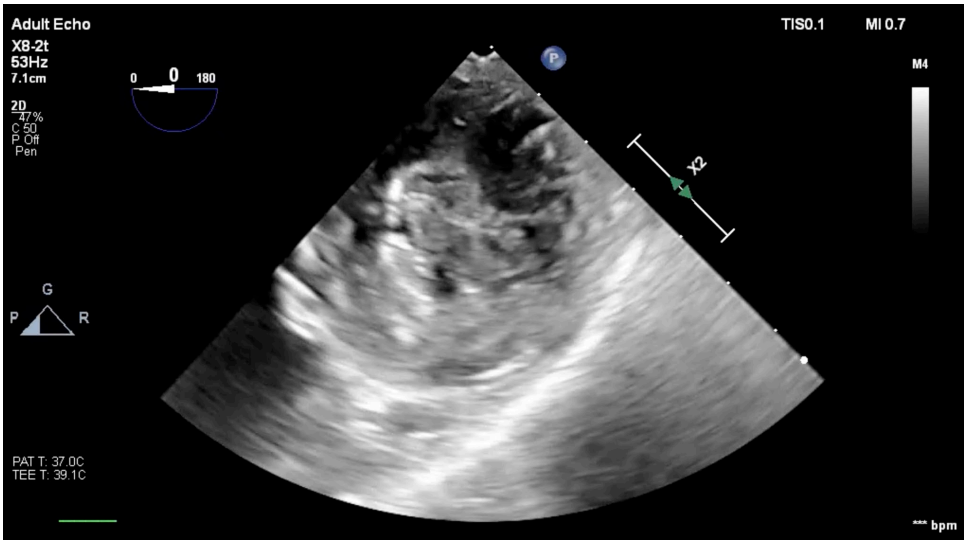
This case posed two unique challenges in management. First was supporting the RV during induction. For patients with severe RV dysfunction, Endotracheal Milrinone after induction is a novel treatment as it increases inotropy, and reduces PVR and subsequent RV afterload. By administering Milrinone endotracheally, its effects are confined to the Cardiopulmonary system and systemic effects are limited [1]. Conversely, intravenous administration will reduce SVR, dropping preload resulting in increased vasopressor requirements and worsening RV dysfunction. The second challenge was management of acute alveolar hemorrhage which is a rare manifestation of pulmonary reperfusion injury. The combination of obstructive saddle PE and time on CBP caused pulmonary ischemia and local inflammation resulting in cell

death and increased vascular permeability [2]. As the lungs are reperfused, oxygenated blood generates reactive oxygen species further worsening vascular permeability and bleeding [3]. Supportive treatment includes correcting coagulopathy, reversing anticoagulation, corticosteroids and VA ECMO.

Images



Pulmonary Embolism in the Right Pulmonary Artery.



Left ventricle post induction.

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#FR 1 – Kiosk 5 – Poster 1

Amulet Placement After Incomplete Closure with a Watchman

Presenting Author: Lueke Anderson, DO, Fellow, University of Nebraska Medical Center

Introduction

Atrial Fibrillation is the most common cardiac arrhythmia and is a major risk factor for stroke, with the left atrial appendage being the most likely location for clot formation in patients with non-valvular AF.2,3 Oral anticoagulation is the mainstay of treatment for patients with AF to prevent embolic stroke but intolerance or contraindications, most commonly bleeding or fall risk, may necessitate alternative therapies. Percutaneous left atrial appendage occlusion (LAAO) using devices such as the Watchman or Amulet provides stroke risk reduction for these patients2. This case will discuss both options as well as show an example of a patient who required both devices to adequately seal her left atrial appendage.

Case Presentation

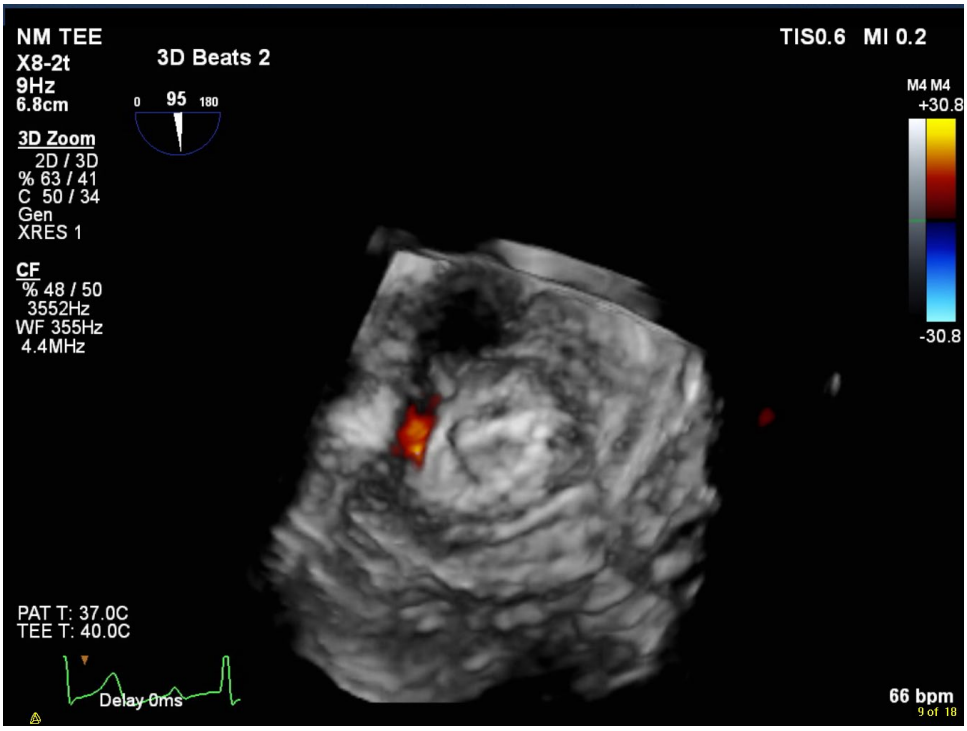
A 77 y.o. year old female with a current and past medical history of atrial fibrillation (on Coumadin, CHA2DS2-VASc Score 6), HTN, CVA, CKD 3, multiple sclerosis, dizziness, imbalance and adrenal insufficiency. She initially underwent LAAO with a 27 mm Watchman FLX due to high risk of falls related to dizziness/imbalance. Follow-up imaging showed a persistent peridevice leak in the lateral landing zone that remained unresolved for over a year. TEE revealed a secondary lobe that was missed on the initial evaluation. A decision was made to close the secondary lobe by deploying an Amulet device over the existing watchman. 16-mm Amulet was successfully implanted, achieving complete occlusion of the accessory lobe. 45-day imaging showed both devices being well seated with no thrombus or peridevice leak. This case highlights a rare and technically challenging situation: the sequential use of two different LAA occlusion devices to achieve complete seal.

Discussion

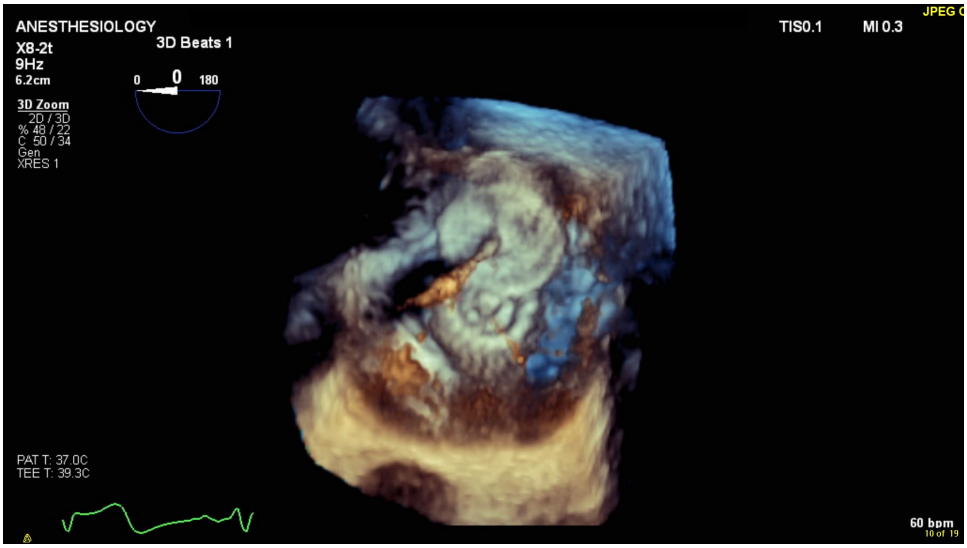
LAAO is an established option for stroke prevention in patients unable to maintain long-term anticoagulation. Device selection varies widely by operator and institution because Watchman and Amulet differ in size ranges, anchoring mechanisms (dual-row anchors vs. stabilizing wires), and release criteria (PASS vs. CLOSE) 2,3. Careful pre-procedure imaging is essential, particularly in patients with complex or multilobed appendage anatomy 2. The Watchman platform includes the Watchman FLX and Watchman FLX Pro, available in sizes from 20 mm to 40 mm for the FLX Pro. The Amulet device ranges from 16 mm to 31 mm. The Watchman secures in place using dual row anchors, whereas the Amulet relies on stabilizing wires. Release criteria also differ between the systems, with the Watchman using the PASS criteria and the Amulet using the CLOSE criteria2,3. Previous reports have described sealing bilobed appendages using more than one device, either in staged procedures or during the same intervention. Although such cases exist, this case is notable because it demonstrates successful closure using two different device platforms. A Watchman FLX sealed the primary lobe, and an Amulet was subsequently used to close an unrecognized

secondary lobe. This dual platform approach highlights the importance of tailoring device selection to individual anatomy and illustrates a novel strategy for managing challenging multilobed LAA morphology.

Images



3D image showing peridevice leak prior to closure of secondary lobe.



3D image showing amulet being placed over secondary lobe and on top of pre-existing watchman device.

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#FR 1 – Kiosk 5 – Poster 2

When Surgery Is Possible but Survival Is Uncertain: Navigating Ethics and Decision Making in a High-Risk Coronary Artery Bypass Surgery After Stroke, Sepsis, Cardiac Arrest, and Multisystem Failure

Presenting Author: Connor K. Gwinn, M.D., Anesthesia Resident, Detroit Medical Center

Introduction

Patients who present with overlapping sepsis, neurologic injury, and severe cardiac disease often decline rapidly and force difficult decisions about when, or even if high-risk surgery is appropriate. We describe a case involving ruptured appendicitis, an acute cardioembolic stroke, cardiac arrest, and profound biventricular failure in a patient ultimately presenting for CABG. This case highlights both the anesthetic challenges and the ethical decision making required when caring for a patient in multisystem failure.

Case Presentation

A 60-year-old man with atrial fibrillation, HFrEF, CAD, COPD, DM2, and HTN presented with abdominal pain and was found to have ruptured but contained appendicitis. He was then diagnosed with an NSTEMI, QTc >520 msec, and EF 25–30%. Cardiac catheterization revealed severe multivessel disease: ostial LM 40%, proximal LAD 85%, LCx 80% with 95% subtotal occlusion, and RCA 95%. CABG was recommended once he was stable. Prior to surgery, he deteriorated significantly. He developed AFib with RVR, sepsis, and a left temporal cardioembolic stroke with new right-sided motor deficits. While being evaluated for worsening neurologic status, he developed wide-complex VT that progressed to TdP and then to PEA arrest, requiring 31 minutes of ACLS. After ROSC, he developed shock liver, anuric renal failure requiring iHD, recurrent upper GI bleeding requiring transfusion, and worsening cardiogenic shock. Coronary revascularization offered the best chance of meaningful recovery. However, his ischemic stroke placed him at risk of hemorrhagic conversion with CPB heparinization (1). Ongoing GI bleeding further increased his risk of hemorrhage. Balancing these competing concerns, his surgery was delayed eight days to reduce the chance of catastrophic neurologic or abdominal hemorrhage. By the time he reached the OR, he required IABP support, CRRT, multiple vasopressors, and demonstrated an EF of 10–15%. His STS-PROM risk score (25–40%) reflected extreme physiologic vulnerability, and current research suggests that traditional scoring systems underestimate mortality in such high-risk CABG populations (2). Induction focused on maintaining perfusion and limiting arrhythmogenic triggers. He was cardioverted after sternotomy and underwent CABG x 3 with left atrial appendage ligation. Intraoperative TEE showed severe biventricular dysfunction with only modest improvement on inotropes. Postoperatively, his shock worsened and vasopressor needs escalated. After another cardiac arrest, the care team and family agreed to forego further escalation. When the IABP was paused, his underlying rhythm demonstrated PEA, and time of death was pronounced.

Discussion

This case demonstrates how multiple insults can push operable patients beyond the threshold of meaningful recovery. High-risk CABG literature emphasizes that evidence guiding surgery in patients with extreme physiologic compromise remains limited, and operating simply because surgery is possible does

not guarantee outcome benefit (3). This case underscores the importance of clear communication, realistic prognostication, and thoughtful guidance for families when weighing high-risk cardiac surgery against the likelihood of meaningful survival.

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#FR 1 – Kiosk 5 – Poster 3

Supermassive Transfusion and Intracardiac Thrombus during Adrenalectomy and Inferior Vena Cava Thrombectomy

Presenting Author: Kyra Rodi, MD, Adult Cardiothoracic Anesthesiology Fellow, University of Colorado

Introduction

Cardiac anesthesiologists must be prepared for a range of intraoperative complications, including massive transfusion, ventricular failure, and intracardiac thrombosis. This is an exceptionally complex case of combined open adrenalectomy and inferior vena cava (IVC) thrombectomy complicated by all three. Mortality following open IVC thrombectomy or nephrectomy is estimated at 6–7%, while the mortality associated with transfusion of more than 50 units of packed red blood cells exceeds 50%. The development of thrombus within the right atrium (RA), right ventricle (RV), and pulmonary artery (PA) further increases mortality risk. The reported mortality for RV thrombus and concomitant pulmonary embolism ranges from 16–29%, increasing to 42% in patients with hemodynamic instability. The confluence of all three complications is exceedingly rare, and no mortality rate has been reported. Management options for RV thrombus include systemic thrombolysis, surgical embolectomy, catheter-directed therapies, anticoagulation, and veno-arterial extracorporeal membrane oxygenation (VA-ECMO).

Case Presentation

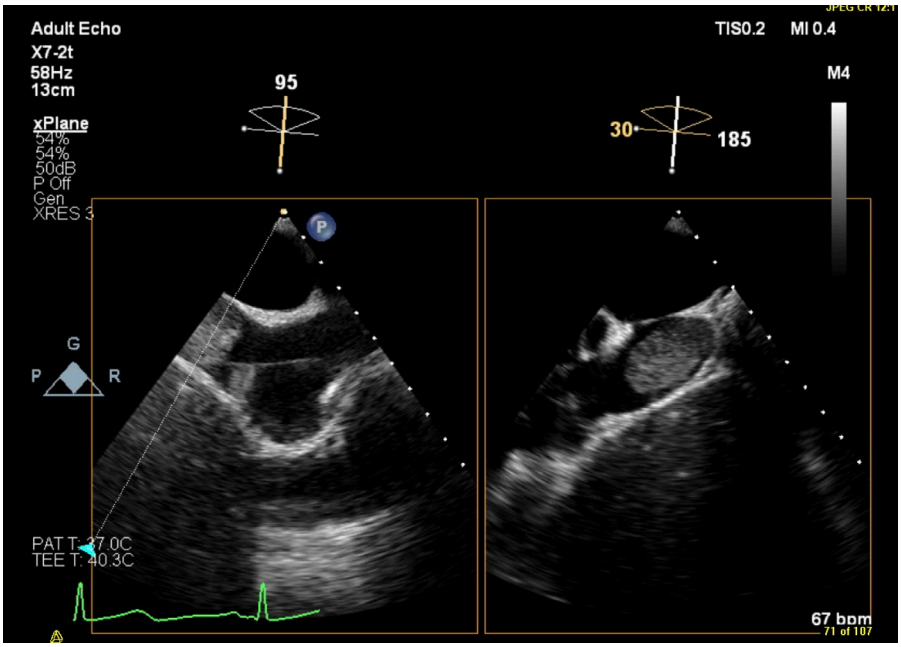
A 34-year-old female with adrenal cortical carcinoma and associated IVC thrombus extending into the RA presented for open right adrenalectomy, IVC thrombectomy. Upon initial dissection, liver congestion led to significant bleeding requiring veno-veno (VV) bypass initiation. Hepatic vein injury led to uncontrolled bleeding requiring significant transfusion (1L per minute through 2 rapid transfusers). This led to RV failure with akinetic RV and associated thrombus in RA, RV, and PA. Cardiac surgery was called for sternotomy and emergent initiation of full cardiopulmonary bypass. Prior to initiation of cardio-pulmonary bypass (CPB) the patient had a potassium of 8 mmol/L and non-perfusing arrythmias, requiring manual cardiac massage. After initiation of CPB, transesophageal echocardiogram (TEE) showed no residual clot in the RV and returned function. Due to persistent RV dysfunction and hypoxia with massive resuscitation, the decision was made to convert to central VA ECMO. The procedure concluded with over 115 liters of blood loss and 368 units of product intraoperatively.

Discussion

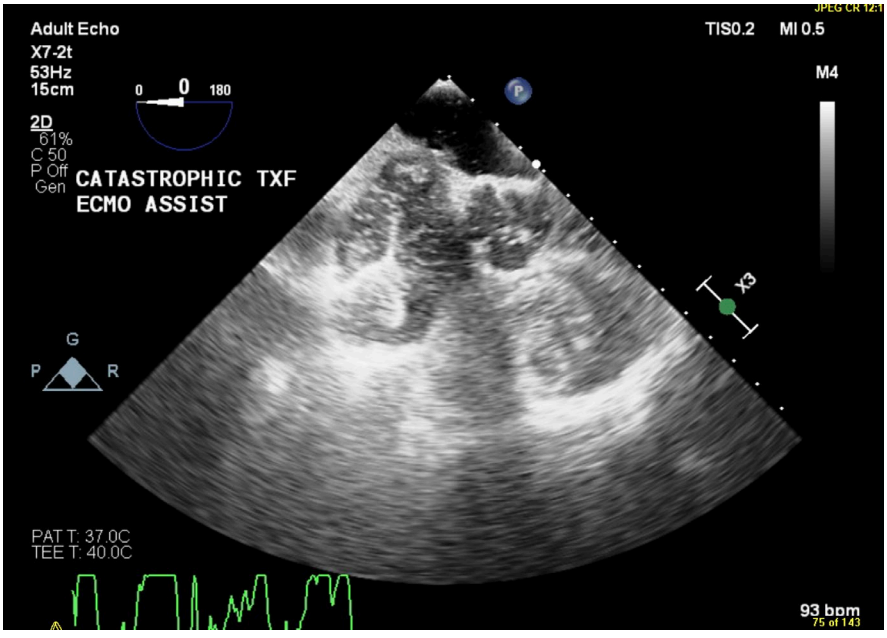
Right sided thrombus is a rare but known complication of massive transfusion, and there are multiple treatment options. Systemic tissue plasminogen activator (TPA), full anticoagulation and mechanical thrombectomy are all options intraoperatively. Both TPA and anticoagulation can exacerbate previously significant bleeding, though can result in resolution of right sided clot. Aggressive resuscitation and support are required with the hemodynamic instability of these patients, and they may require VA ECMO or full cardiopulmonary bypass, along with inotropic support and inhaled pulmonary vasodilators. Other concomitant problems associated with catastrophic transfusion requirements include hyperkalemia,

arrythmias, coagulopathy, acute renal failure, infection, transfusion-associated lung injury (TRALI), and transfusion associated circulatory overload (TACO) – all of which this patient experienced. In cases where supermassive transfusion is expected, pre-procedural planning is vital amongst a multidisciplinary team.

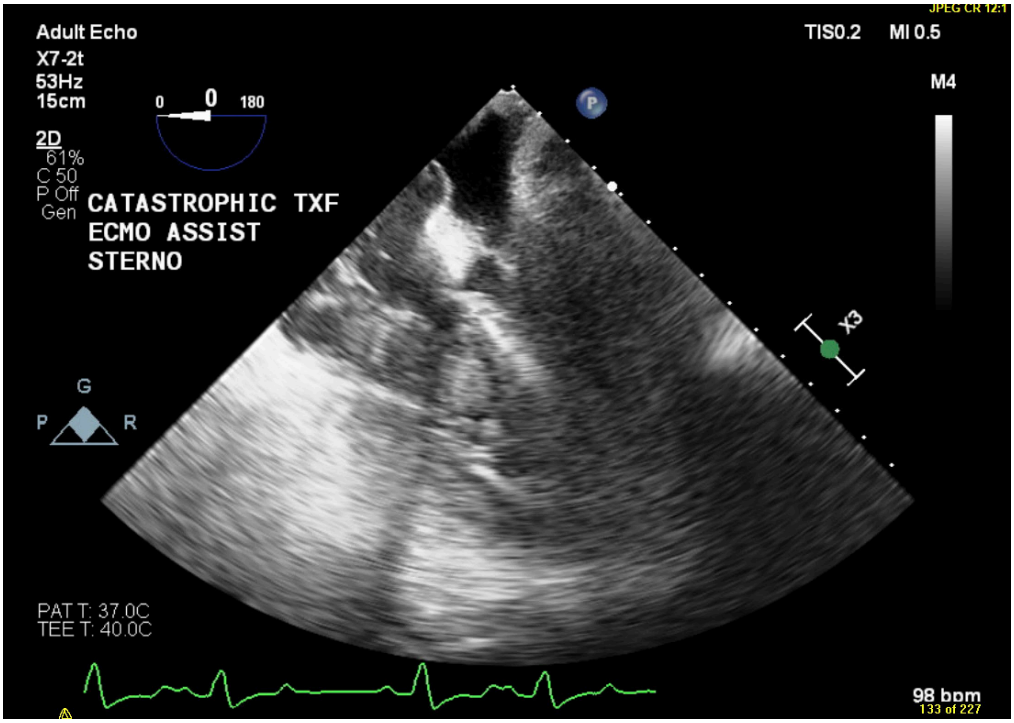
Images



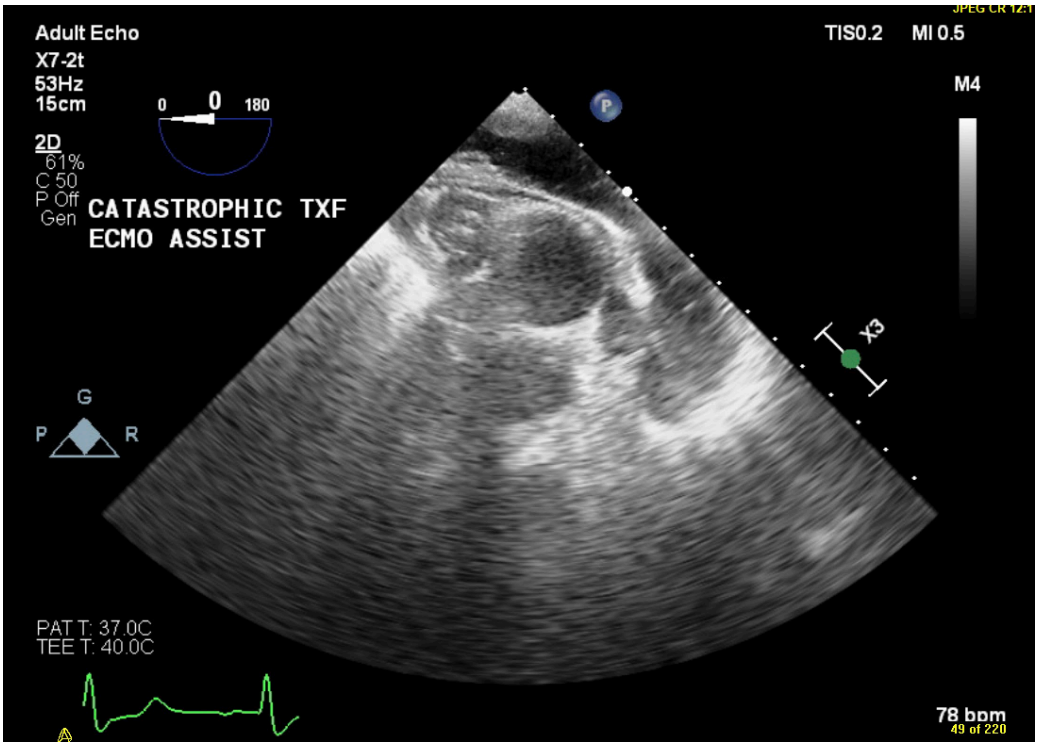
IVC thrombus



RV thrombus 1



RV thrombus 2



PA thrombus

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#FR 1 – Kiosk 5 – Poster 4

I Can't Breathe: Respiratory Failure After Total Artificial Heart Placement

Presenting Author: Chad Q. Zhao, MD, Fellow, University of California San Francisco

Introduction

We present a case of total artificial heart implantation (TAH) complicated by severe hypoxemia. TAH implantation remains an important tool in bridging patients with severe biventricular failure to a heart transplant. These patients frequently suffer from the sequelae of acute-on-chronic heart failure decompensation requiring special care and consideration from anesthesiologists.

Case Presentation

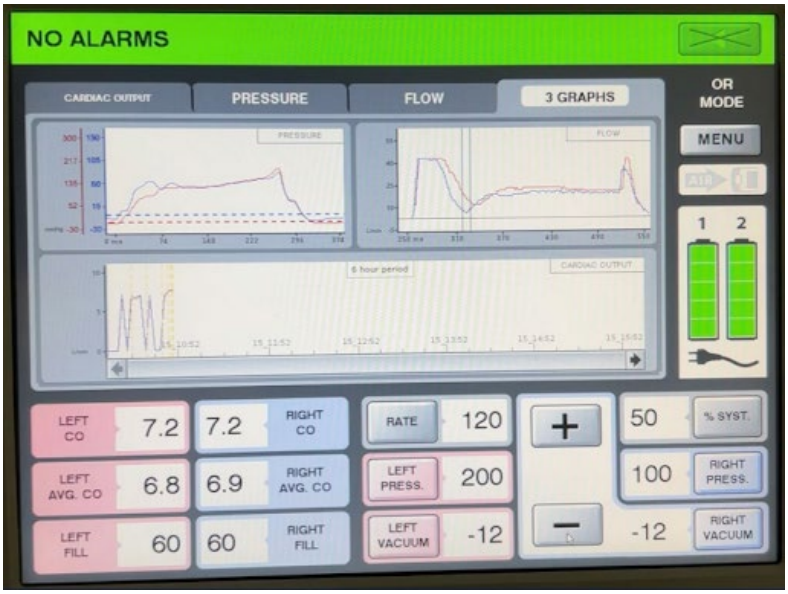
A 59 y/o female who presented with 5 days of intermittent chest pain and dyspnea concerning for NSTEMI who was found to have severe aortic stenosis and heart failure (EF35-40%). She initially underwent aortic valve replacement complicated by intraoperative ventricular fibrillation (Vfib) arrest requiring central ECMO. Her postoperative course was further complicated by vfib arrest following central VA-ECMO decannulation requiring urgent peripheral VA-ECMO cannulation. The patient was not a candidate for heart transplant due to pre-capillary pulmonary hypertension (PVR 5.29 WU) and was not a candidate for heart-lung transplant due to body habitus (BMI > 35) and severe deconditioning. She was eventually approved and underwent Syncardia TAH. Her intraoperative course was complicated by significant pulmonary edema after weaning from cardiopulmonary bypass (CPB) with inability to improve SpO2 above 70%. The decision was made to place the patient on VV-ECMO support. Her postoperative course was complicated by sustained respiratory failure requiring prolonged VV-ECMO, renal failure requiring CRRT, pneumonia, small bowel obstruction and eventually death.

Discussion

TAH implantation requires careful consideration of the patient & procedure by the anesthesiologist. Pre-operatively, the patient's capacity to oxygenate & ventilate was masked by high-flows on peripheral VA-ECMO. However, the anesthesiology and mechanical team suspected poor pulmonary reserve due to opacifications found on serial chest x-rays. Prior to separation from CPB, efforts were made to optimize oxygenation. Furthermore, during weaning from CPB, significant negative pressure was applied to the neo-left ventricle to reduce formation of pulmonary edema. Despite these measures, the patient became hypoxic. Cannulating for VV-ECMO for hypoxia is of extreme risk in patients with a TAH. If a wire is caught in the mechanical tricuspid prosthesis, the TAH will experience catastrophic failure. Our use of TEE was instrumental in confirming appropriate wire position in the right atrial remnant while ensuring the wire did not interact with the mechanical prosthesis. In addition to the anesthesiology team's concern with oxygenation we were worried about potential renal dysfunction. TAH patients lose ventricular tissue, the source of BNP, theorized to be strongly associated with natriuresis. In the absence of BNP, many TAH patients suffer from renal dysfunction and require CRRT early in their post-operative course. We placed a dialysis catheter prior to TAH implantation to avoid any potential issues placing this catheter post-

operatively due to wire interference with the mechanical prosthesis. In our patient, renal dysfunction was significant and urgent CRRT was needed due to hypoxia secondary to volume overload and pulmonary edema. The dialysis catheter ultimately facilitated her immediate post-operative management.

Images



Companion 2 Driver System Display Screen

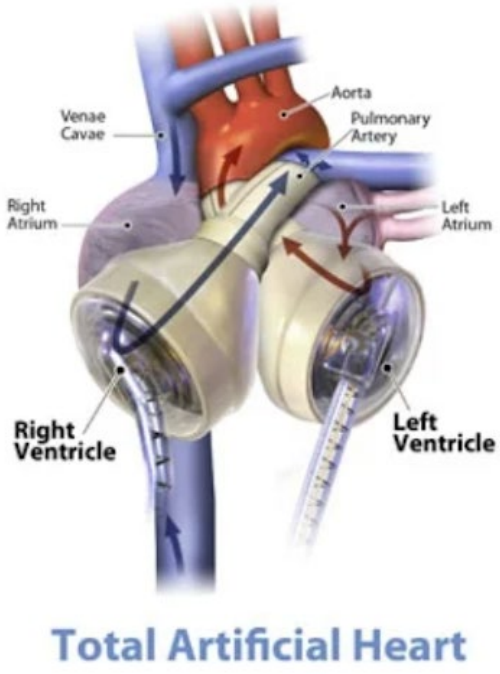


Diagram of total artificial heart



CT Angiogram of total artificial heart

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#FR 1 – Kiosk 5 – Poster 5

Conflicting Hemodynamic Objectives in Open Abdominal Aortic Aneurysm (AAA) Repair with Residual Aortic Dissection

Presenting Author: Melissa L. Tu, MD, Adult Cardiothoracic Fellow, University of Michigan

Introduction

Coexisting aortic pathologies create conflicting hemodynamic priorities during major vascular surgery. We present a case of open AAA repair in a patient with persistent type A dissection propagation, where blood pressure management required balancing protection of the proximal aorta with preservation of distal organ perfusion in the setting of cross clamp physiology.

Case Presentation

A 67-year-old woman with hypertension, chronic kidney disease, prior infrarenal EVAR, and repaired type A dissection with preserved native arch presented with persistent dissection and progressive infrarenal aneurysm enlargement due to false-lumen perfusion, concerning for a type Ia endoleak (Figure 1). She underwent open AAA repair with partial EVAR explantation. Baseline hemodynamic strategy focused on minimizing shear stress on the proximal aorta to avoid dissection propagation toward the arch and head vessels. The anesthetic plan incorporated conflicting goals: preventing proximal hypertension while maintaining adequate perfusion and balancing the physiologic effects of aortic cross clamping which increase afterload. Prior to surgical intervention, arterial and central access were obtained. In addition to standard ASA monitors, we included cerebral oximetry and arterial access on the opposite arm to monitor for any differential changes indicating dissection advancement. After induction, we maintained systolic blood pressure (SBP) < 130 mmHg and hemoglobin >10g/dL to minimize further kidney injury and optimize cerebral and end-organ oxygen delivery. Prior to aortic cross-clamping, we titrated infusions to target SBP < 80 mmHg to minimize the risk of aortic rupture and blood loss during the high-afterload phase. Judicious fluid administration with reliance on blood products supported hemodynamics during surgical bleeding and in anticipation of cross clamp release. Following clamp removal, SBP gradually increased requiring clevidipine and esmolol to maintain a SBP target of 90–110 mmHg. Cerebral oximetry remained stable, and the patient tolerated the remainder of the procedure without hemodynamic instability and correlating arterial line measurements.

Discussion

This case highlights the complex interplay between proximal aortic protection and distal perfusion requirements in patients with combined dissecting and aneurysmal disease. There was extensive discussion between anesthesiology, cardiac surgery and vascular surgery to plan for hemodynamic challenges. Standard cross-clamp physiology—often requiring aggressive afterload support—must be modified when proximal hypertension risks further dissection advancement. Cerebral oximetry proved valuable in guiding real-time adjustments, especially given the absence of echocardiography-derived lumen flow assessment. Managing fluid limitation, renal vulnerability, and competing SBP targets required continuous coordination

between surgical and anesthetic teams. Careful, dynamic blood pressure management is essential in patients with persistent type A dissection undergoing AAA repair. We found the integration of cerebral perfusion monitoring, tailored vasoactive strategies, and cautious resuscitation were key to successfully navigate these opposing physiologic demands.

Images



Figure 1. Computed tomography and three-dimensional imaging depicting combined dissection and aneurysmal disease of aorta.

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#FR 1 – Kiosk 5 – Poster 6

When Lightning Strikes Twice: Navigating Multiple Cardiac Crises in a Single Operation

Presenting Author: Navoda Mirihagalle, MD, Resident Physician, University of Wisconsin

Introduction

This case unfolded over a single 24-hour period in which multiple rare, high-acuity events fundamentally altered the expected surgical course. A 58-year-old woman presented to the operating room for bilateral lung transplantation. Unbeknownst to the operative teams, the sequence of intraoperative events would ultimately challenge the limits of cardiothoracic surgery, anesthesiology, and critical care.

Case Presentation

A 58-year-old female underwent bilateral lung transplantation via clamshell incision with central ECMO cannulation. Following explant of native lungs and implantation of donor lungs, targeted hemodynamics were achieved in preparation for routine ECMO decannulation. However, intraoperative TEE revealed a new ascending aortic dissection, originating at the cannulation site and extending distally. After multidisciplinary assessment, emergent repair was deemed necessary for this iatrogenic injury. The patient was placed on cardiopulmonary bypass (CPB), cooled to 24 °C, and underwent circulatory arrest with selective antegrade cerebral perfusion for total arch replacement. Reperfusion, rewarming, and CPB separation were achieved, but the patient demonstrated marked vasoplegia and significant coagulopathy. During ongoing hemostatic resuscitation guided by TEG, the patient developed sudden, extensive intracardiac thrombosis with concurrent acute biventricular failure. Thrombus occupied the right atrium and ventricle, pulmonary artery, mitral valve, and aortic root, with associated anterior and lateral wall akinesis. With obstructed coronary flow confirmed, the patient was emergently returned to CPB for left main coronary bypass and central ECMO cannulation. After successful ECMO initiation, she was transferred to the ICU but ultimately died from refractory multi-organ failure.

Discussion

This case illustrates a cascade of rare, catastrophic complications in a patient already undergoing high-risk thoracic surgery. The iatrogenic ascending aortic dissection immediately following ECMO decannulation necessitated complex total arch repair, markedly increasing systemic inflammatory load, coagulopathy, and physiologic stress. The combination of recent lung transplantation, prolonged CPB exposure, hypothermia, circulatory arrest, and metabolic derangements likely contributed to profound coagulation dysfunction. This milieu set the stage for the second major event: fulminant intracardiac thrombosis leading to abrupt myocardial ischemia and hemodynamic collapse. The requirement for repeated CPB runs and subsequent ECMO further amplified the cycle of inflammation, vasoplegia, bleeding, and thrombosis. The case highlights the extreme challenges of managing sequential aortic and coronary catastrophes in the immediate post-transplant setting and underscores the importance of rapid multidisciplinary decision-making in ultra-high-risk cardiothoracic scenarios.



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#FR 1 – Kiosk 5 – Poster 7

Beating Heart Isolated Tricuspid Valve Replacement for Carcinoid Heart Disease

Presenting Author: Vidhi Patel, MD, Fellow, Icahn School of Medicine at Mount Sinai

Introduction

Carcinoid tumors are a subset of neuroendocrine tumors (NETs), with approximately 90% originating from the gastrointestinal tract. When these tumors secrete vasoactive substances such as serotonin and bradykinin, they give rise to carcinoid syndrome, characterized by facial flushing, diarrhea, wheezing, and bronchospasm. The incidence of NETs is estimated at 2.5–5 per 100,000 individuals, and 30–40% of these develop carcinoid syndrome1. Carcinoid heart disease (Hedinger syndrome) occurs in up to 50% of patients with carcinoid syndrome, typically involving right-sided cardiac valves. Metastatic foci can overwhelm the liver's capacity to metabolize these substances, while the lungs inactivate these substances, sparing the left heart. We present a case of a 71-year-old man with metastatic carcinoid syndrome who underwent isolated tricuspid valve replacement for carcinoid heart disease.

Case Presentation

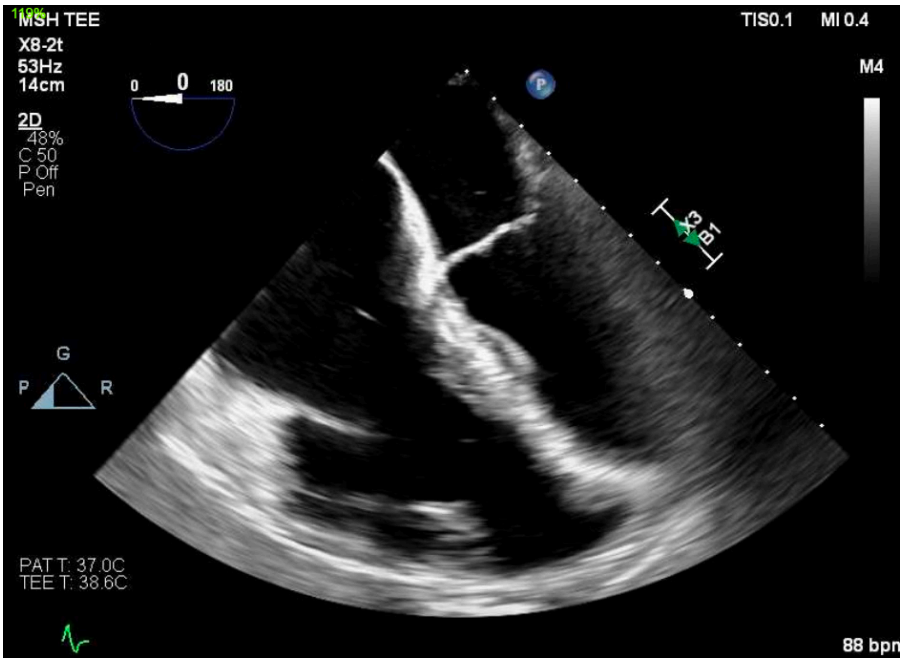
A 71yo male with known metastatic carcinoid disease, presented to us for worsening dyspnea. He was followed by hematology outpatient and was treated with octreotide every 4 weeks. A year prior, he had undergone percutaneous pulmonary valve replacement for pulmonary regurgitation. The patient had a known history of ascites for which he was also undergoing paracentesis. His plasma 5-HIAA was 210ng/mL and he had a serum serotonin of 1295ng/mL. Pre-operative TTE showed frozen open thickened tricuspid leaflets, moderate RV dysfunction and a well seated pulmonic prosthesis. The patient was admitted preoperatively and was started on an octreotide drip that was uptitrated perioperatively 2. Prior to induction he was also premedicated with hydrocortisone, diphenhydramine and zofran to further mitigate the peri-operative hormone release. Intraoperative TEE showed torrential TR with leaflets frozen open (Figure 1) , annulus of 4.41cm and complete flow reversal in the hepatic veins (Figure 2) . He underwent a beating heart isolated tricuspid valve replacement. Post bypass TEE showed a well seated valve with a mean gradient of 1mmhg. The flows in the hepatic vein had normalized (Figure 3). The patient was admitted to ICU and had an overall uncomplicated post-op course. He was weaned from the octreotide drip and resumed his monthly outpatient injections.

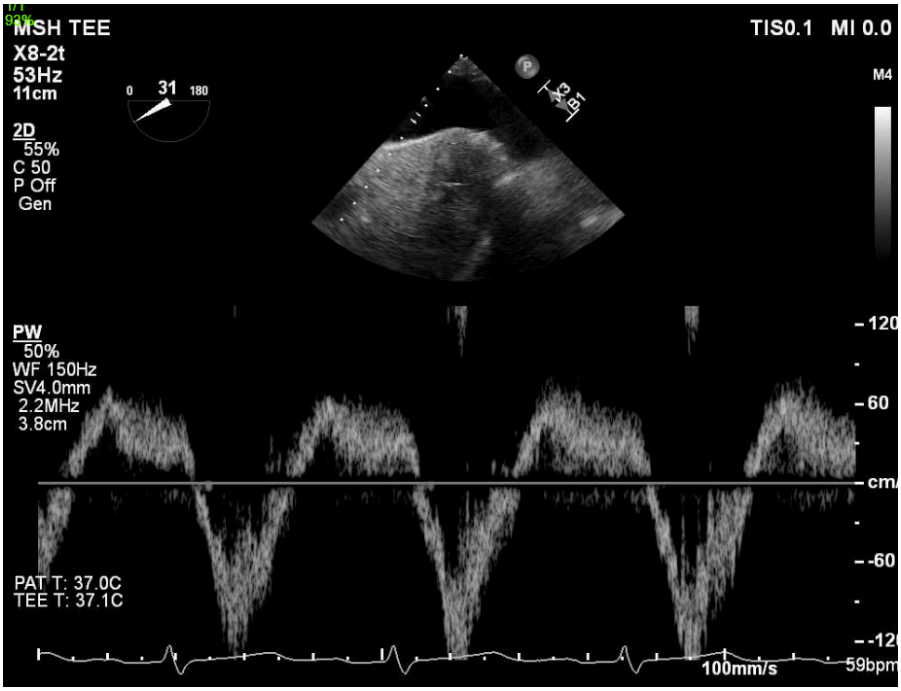
Discussion

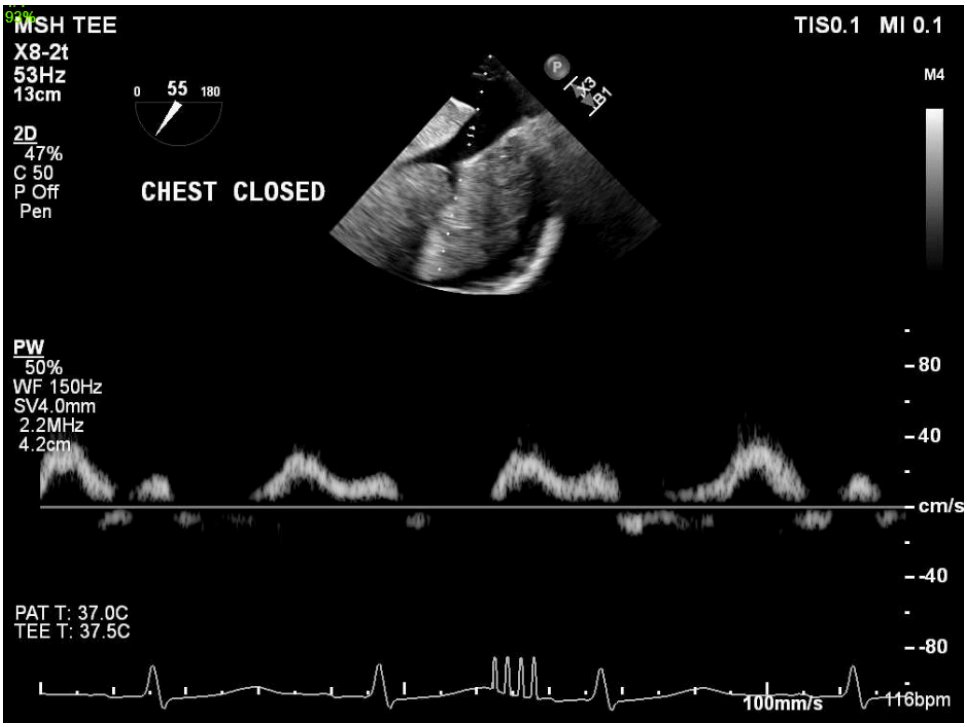
Neuroendocrine tumors that secrete vasoactive peptides, predominantly serotonin and bradykinin, can lead to carcinoid syndrome. Patients with metastatic carcinoid disease are also at risk of carcinoid crisis, characterized by the sudden, massive release of vasoactive mediators resulting in severe hemodynamic instability. Both anesthesia and surgical manipulation can precipitate this life-threatening event3. Effective perioperative management requires a multidisciplinary approach involving anesthesiology, cardiology, hematology, and surgery. Preventive strategies include premedication and continuous octreotide infusion to suppress hormonal release. In addition, isolated tricuspid valve replacement for carcinoid heart disease is

uncommon; recent studies suggest that a beating-heart approach may be associated with improved postoperative outcomes. This case underscores the importance of coordinated, multidisciplinary perioperative care and tailored surgical strategy in optimizing outcomes for patients with carcinoid heart disease.

Images




MSH TEE
X8-2t
53Hz
11cm
2D
55%
C 50
P Off
Gen
TIS0.1 MI 0.0
M4
PW
50%
WF 150Hz
SV4.0mm
2.2MHz
3.8cm
PAT T: 37.0C
TEE T: 37.1C
100mm/s
59bpm


MSH TEE
X8-2t
53Hz
13cm
2D
47%
C 50
P Off
Pen
CHEST CLOSED
TIS0.1 MI 0.1
M4
PW
50%
WF 150Hz
SV4.0mm
2.2MHz
4.2cm
PAT T: 37.0C
TEE T: 37.5C
100mm/s
116bpm

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#FR 1 – Kiosk 6 – Poster 1

Perioperative management of late adulthood diagnosis and repair of tetralogy of Fallot

Presenting Author: Peter Sloane, MD, Resident physician, University of Chicago, Department of Anesthesia & Critical Care

Introduction

Unrepaired tetralogy of Fallot (TOF) presenting in late adulthood is rare and reflects tenuously balanced anatomic severity, physiologic adaptation, and associated congenital anomalies. Adults typically present with heart failure, not cyanosis, due to chronic RV pressure overload, infundibular hypertrophy, secondary tricuspid regurgitation, and increased pulmonary collateral flow. Herein we describe the perioperative anesthetic management of a 65-year-old woman with undiagnosed TOF, residual atrial septal defect (ASD), and suspected 22q11.2 deletion syndrome undergoing definitive repair.

Case Presentation

She presented with biventricular failure with a large low-velocity bidirectional membranous VSD, severe

pulmonic stenosis and regurgitation, severe tricuspid regurgitation. RV dilation and hypertrophy, moderate aortic regurgitation, and a residual ASD. History included developmental delay and childhood cleft lip/palate and ASD repairs. Concern for syndrome-associated airway anomalies warranted GlideScope-assisted fiberoptic intubation; anatomy was normal. TEE confirmed significant RV dysfunction and the VSD. Pre-bypass management supported pulmonary blood flow across a dynamic RVOT obstruction: augmenting preload in a stiff RV, increasing in SVR, and minimizing PVR through vasopressin, low PEEP, and high FiO_2. Repair included VSD patch closure, RVOT reconstruction with subpulmonary resection and bioprosthetic pulmonic valve, tricuspid valve Alfieri repair, and primary ASD closure. Post-bypass management augmented preload, maintained sinus rhythm with a high-normal heart rate, minimized PVR, and supported RV contractility with epinephrine, milrinone, inhaled nitric oxide, and gentle ventilation. Post-bypass TEE demonstrated a well-functioning pulmonic prosthesis, trace TR, residual small high-velocity VSD jet, and moderate RV dysfunction. ICU course featured RV dysfunction prompting right heart catheterization and imaging revealing VSD patch dehiscence requiring emergent revision. She also suffered atrial fibrillation, acute kidney injury, and respiratory failure due to pneumonia requiring tracheostomy ahead of discharge post-operative day 27 to long-term care facility.

Discussion

Late TOF repair confers substantial perioperative risk due to chronic RV pressure overload, volume redistribution from systemic-to-pulmonary collaterals, residual shunts, and limited biventricular reserve. Survival into the seventh decade reflects a balanced defect: fixed RVOT obstruction limiting pulmonary circulation without cyanosis, combined with gradual development of bronchopulmonary collaterals sustaining inefficient pulmonary flow. Relief of longstanding RVOT obstruction exposes the RV to an abruptly increased pulmonary runoff, resulting in acute RV volume loading and impaired ejection. Improved RV pressures pathologize residual VSDs favoring high-velocity left-to-right shunts, increasing LV volume load and pulmonary flow without meaningful decompression. Tricuspid repair, optimized preload, controlled reduction of PVR, and inotropic support are essential to maintain forward flow in this setting. This case highlights the complex interplay of congenital anatomy, ventricular adaptation, and perioperative physiology in managing late-presenting TOF.

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#FR 1 – Kiosk 6 – Poster 2

Protamine Reaction Post-Coronary Artery Bypass Grafting Causing Graft Failure Resulting in Veno-Arterial Extracorporeal Membrane Oxygenation and Stent Placement

Presenting Author: Laurel A. Mouer, DO, Anesthesiology Resident, University of Virginia

Introduction

Systemic anticoagulation is a requirement for cardiac surgery on Cardiopulmonary bypass (CPB). Heparin has been the drug of choice since the advent of CPB [1]. Protamine, the reversal agent for heparin, comes with an array of side effects. These range from histamine-mediated hypotension to heparin-protamine complex complement activation with severe pulmonary vasoconstriction. Up to 13% of patients can have some form of protamine reaction, with catastrophic pulmonary hypertension incidence around 0.6%[2]. This case is an example of successful management of a catastrophic protamine reaction.

Case Presentation

A 68-year-old female with a history of stroke and 100% right internal carotid stenosis presented with chest pain and was diagnosed with a myocardial infarction. Her catheterization found diffuse disease with 80% stenosis at the proximal LAD, 90% stenosis at the proximal LCx, and 80% stenosis in the proximal RCA. She underwent a 3-vessel CABG utilizing CPB. Baseline TEE showed a mildly dilated LV with an ejection fraction of 45-50%, no RV dysfunction and no valvular pathologies. CBP was initiated with no complication. The LIMA bypass graft was placed at the mid-LAD and the obtuse marginal (OM) 1 and 2 vein grafts were placed at the mid and distal OM. The patient was weaned off CPB and was slowly given protamine. Minutes after protamine administration, the patient had profound hypotension. The surgical team noted her pulmonary artery was firm, and her right ventricle was grossly dysfunctional. TEE confirmed severely depressed function with LVEF of 20%, severe apical anterior-lateral hypokinesis, moderate RV failure, and severe mitral regurgitation. Her hypotension was refractory to medical management, and she was re-cannulated for CPB. An IABP was placed for mechanical support. Cardiology was notified that there was a concern that her bypass grafts had thrombosed due to the low-flow state caused by the protamine reaction. The decision was made to leave her chest open and transition from CPB to VA ECMO. The patient was transported to the catheterization lab and her proximal LCx was stented. A cangrelor infusion was initiated to prevent thrombosis of the new stent. Repeat TEE showed significant improvement in regional wall motion abnormalities with an LVEF of 55-60%, mild RV dysfunction, and no valvopathies. The patient was weaned off ECMO before going to the ICU. She continued to receive blood products overnight due to coagulopathy from cangrelor. Her chest was closed the following morning. Her IABP was removed on POD3, and she was extubated on POD4 with a baseline neuro exam.

Discussion

Protamine is considered a “necessary evil” when managing post-CPB coagulopathy. In some cases, heparin alternatives like bivalirudin are an acceptable choice. Emergent re-cannulation in the setting of a severe protamine reaction is still an indication for heparinization. The addition of antiplatelet therapy is indicated

for prevention of stent thrombosis but will likely worsen post-op coagulopathy. This case is an example of an effective multidisciplinary approach to a severe protamine reaction with the sequelae of acute biventricular failure and ACS requiring stenting after CABG, and subsequent coagulopathy from cangrelor.

Images

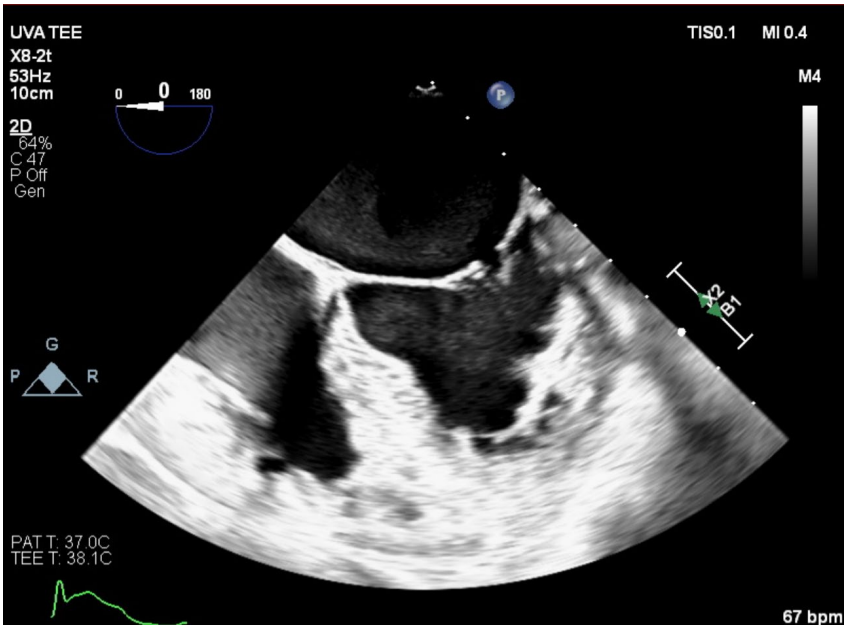


Figure 1. Normal RV in diastole before procedure

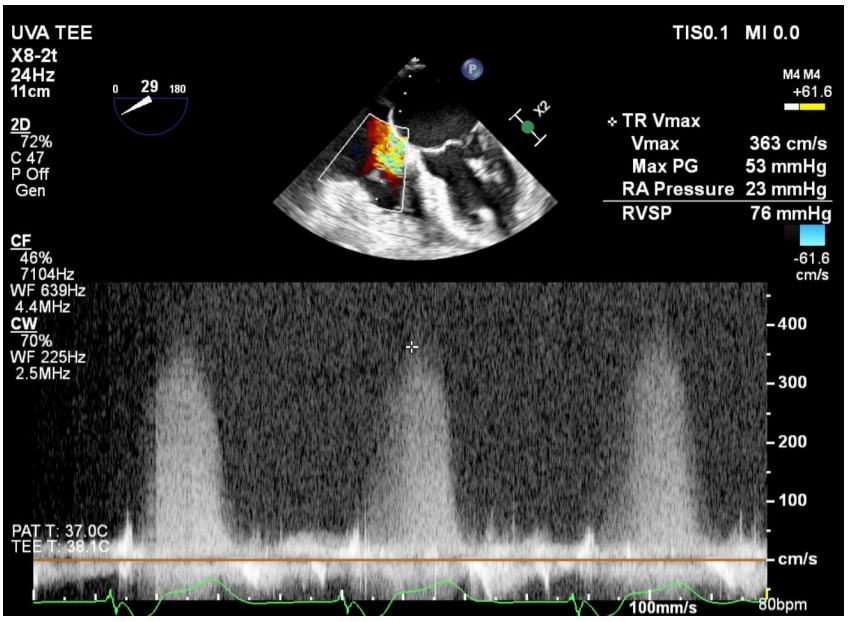


Figure 2. Acute RV failure during protamine reaction with PASP of 76

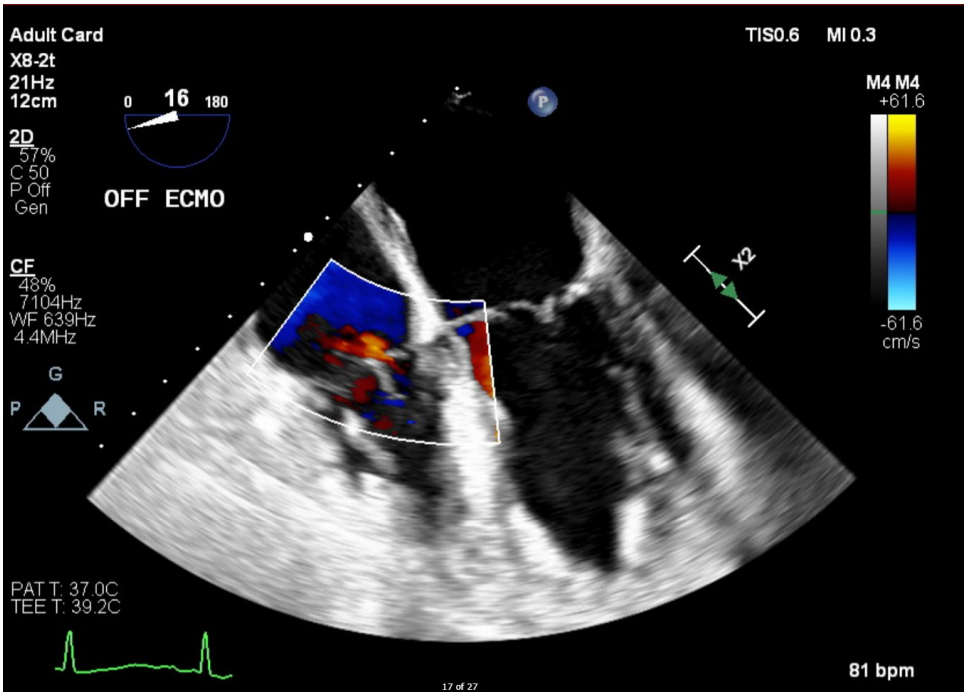


Figure 3. Post-stent placement, off ECMO with normal RV function and trace TR

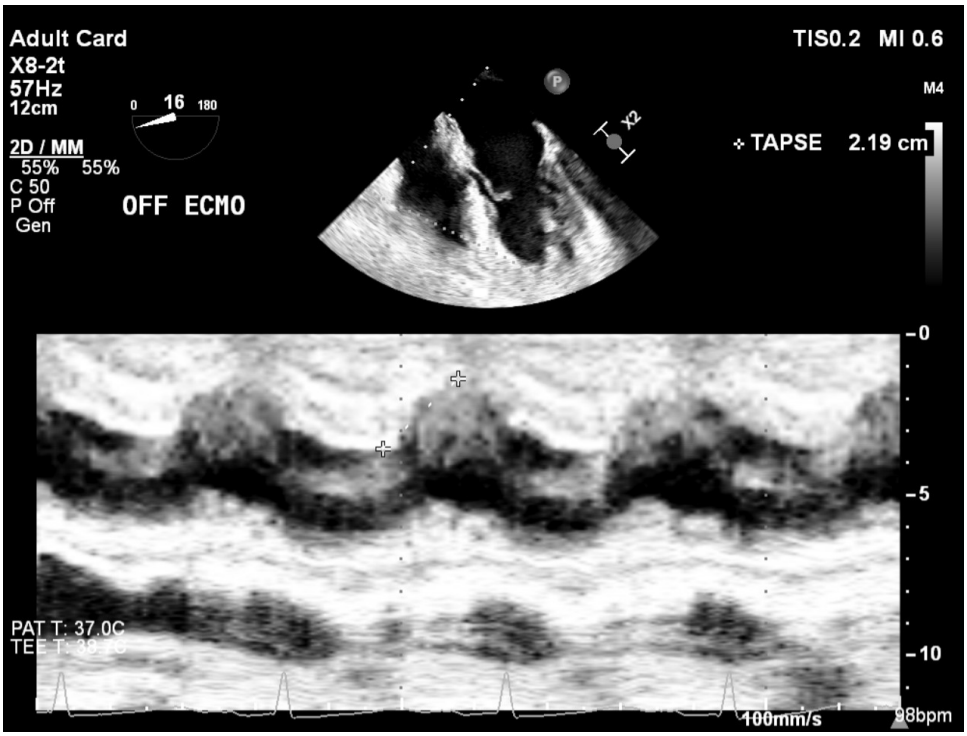


Figure 4. Normal TAPSE post-stent and off ECMO

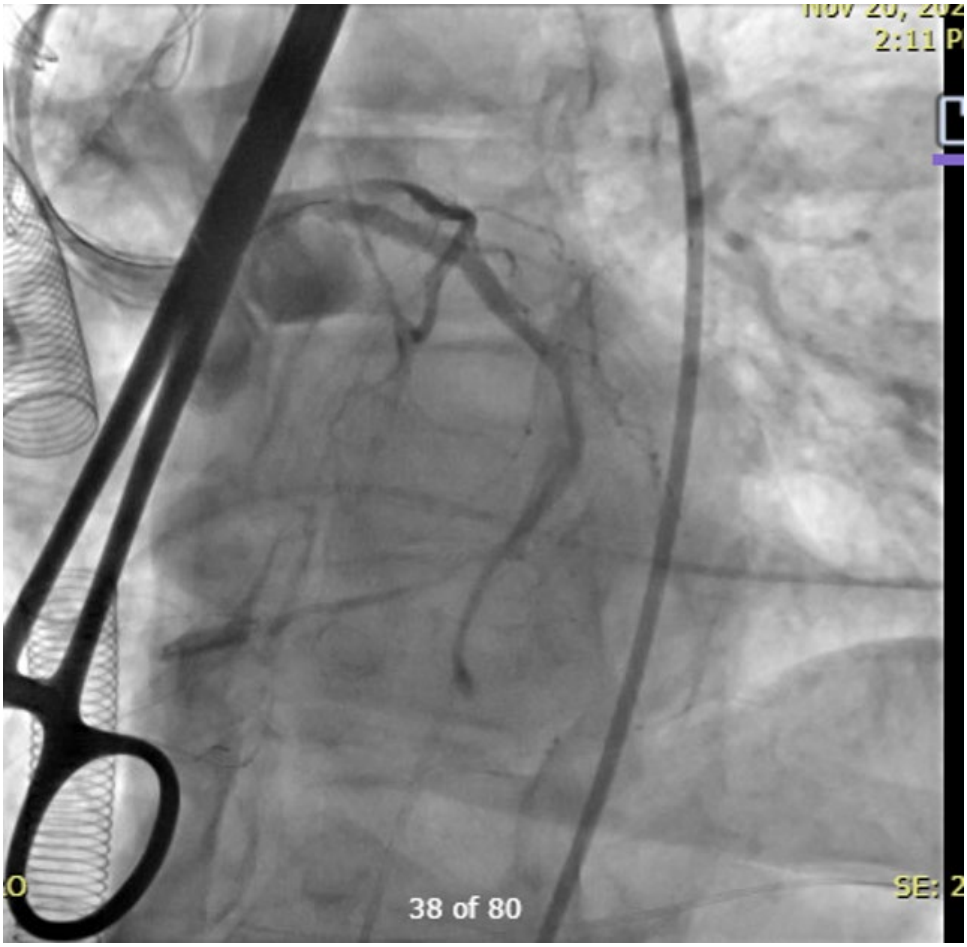


Figure 5. LCx post-stent with patent grafts

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#FR 1 – Kiosk 6 – Poster 3

Iatrogenic Aorta-Atrial Fistula During Combined BASILICA/LLAMACORN TAVR

Presenting Author: Robert Roth, MD, Resident, Dartmouth Health

Introduction

Coronary obstruction is a rare, but serious complication in patients with high-risk anatomic features undergoing transcutaneous aortic valve replacement (TAVR). Bioprosthetic or Native Aortic Scallop Intentional Laceration to Prevent Iatrogenic Coronary Artery Obstruction (BASILICA) is a novel transcatheter technique to minimize the risk of coronary artery obstruction in these high-risk patients undergoing TAVR. The aim is to cut the aortic valve leaflets so that they do not cover the coronaries1. Newer techniques in a similar vein to BASILICA have emerged such as the LLAMACORN technique (Leaflet Laceration with Balloon Mediated Annihilation to Prevent Coronary Obstruction with Radiofrequency Needle). The key difference to this technique is instead of cutting the aortic valve lengthwise, a hole is made in the leaflet and a balloon is expanded to obliterate the leaflet prior to TAVR expansion2.

Case Presentation

The patient is a 73-year-old male with a past medical history notable for hypertension, diastolic heart failure (EF 60%) and SAVR in 2018. He underwent a TAVR-in-SAVR procedure for severe aortic stenosis and intra-valvular aortic insufficiency. His workup was notable for bilateral coronary heights of 7.6 cm. He was deemed high risk for coronary obstruction after TAVR placement so a BASILICA/LLAMACORN procedure was planned. The patient had a preinduction arterial line and was induced with propofol and rocuronium. Transesophageal echocardiography was used throughout the procedure. Isoflurane was used for maintenance of anesthesia. The patient required hemodynamic support with epinephrine and vasopressin. The interventionalist team began with the BASILICA procedure on the left coronary cusp. During one of the attempts to puncture the left coronary cusp, it was quickly noted that the traversal wire was in the left atrium on TEE. Once the interventionalist team had finished with the left coronary leaflet, they moved to perform LLAMACORN on the right coronary cusp. Nearly an hour was spent trying to successfully cross the right coronary cusp, during which there was significantly more aortic insufficiency due to the transected left coronary leaflet. Once the interventionalist team was able to balloon the right coronary leaflet, a 26 mm Medtronic Evolut valve was deployed. TEE imaging of the previously noted aortic-atrial fistula showed that the new valve occluded the previously seen jet. Additionally there were no wall motion abnormalities or EKG changes.

Discussion

This case highlights a novel method of reducing the risk of coronary obstruction of a TAVR-in-SAVR valve in a patient with high risk anatomy. This case also highlights the importance of direct TEE visualization of the interventionalist's wire, as the team had inadvertently crossed into the left atrium. The interventionalists could not see this traversal into the left atrium on fluoro, so close communication and visualization was key. Had they extended further with their traversal catheter, without being alerted via TEE, it would have likely

traversed the atrium and damaged the posterior mitral annulus. Finally the patient likely only tolerated the extended period of time it took to obliterate the right coronary leaflet due to cardiac remodeling from his severe AI and his excellent baseline functional status.

Images



Figure 1: Accidental traversal of the wire into the left atrium

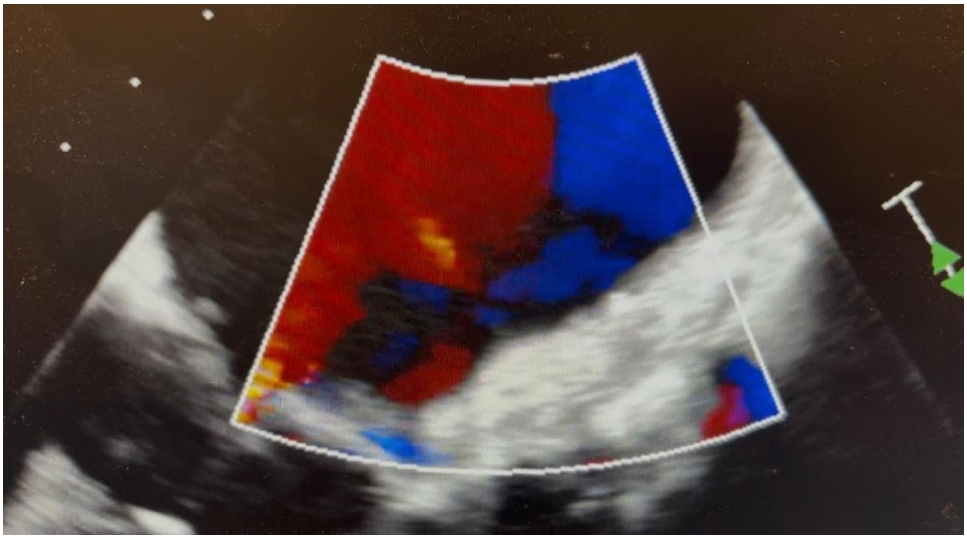


Figure 2: Notable jet originating from the aorta to the left atrium



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#FR 1 – Kiosk 6 – Poster 4

Total Intravenous Anesthesia for En-Bloc Bilateral Lung Transplantation with Tracheal Anastomosis and Omental Flap in a VV-ECMO–Bridged Patient with Familial Malignant Hyperthermia Risk

Presenting Author: John Boyle, MD, Resident, Penn Medicine

Introduction

Bilateral orthotopic lung transplantation (BOLT) is typically completed with bilateral bronchial anastomoses and often with intraoperative Venoarterial (VA) extracorporeal membrane oxygenation (ECMO) support. Conversely, en-bloc lung transplantation (LT) with tracheal anastomosis, performed during the advent of bilateral LT, is now rare due to high risk of tracheal dehiscence and need for cardiopulmonary bypass (CPB). Additionally, patients who have a risk for malignant hyperthermia (MH), precluding volatile agents, can be clinically challenging to manage while on CPB. In this case we present a patient placed on preoperative venovenous (VV) ECMO support as a bridge to en-bloc LT with tracheal anastomosis and omental flap with a totally intravenous anesthetic (TIVA) on CPB due to a family history of MH.

Case Presentation

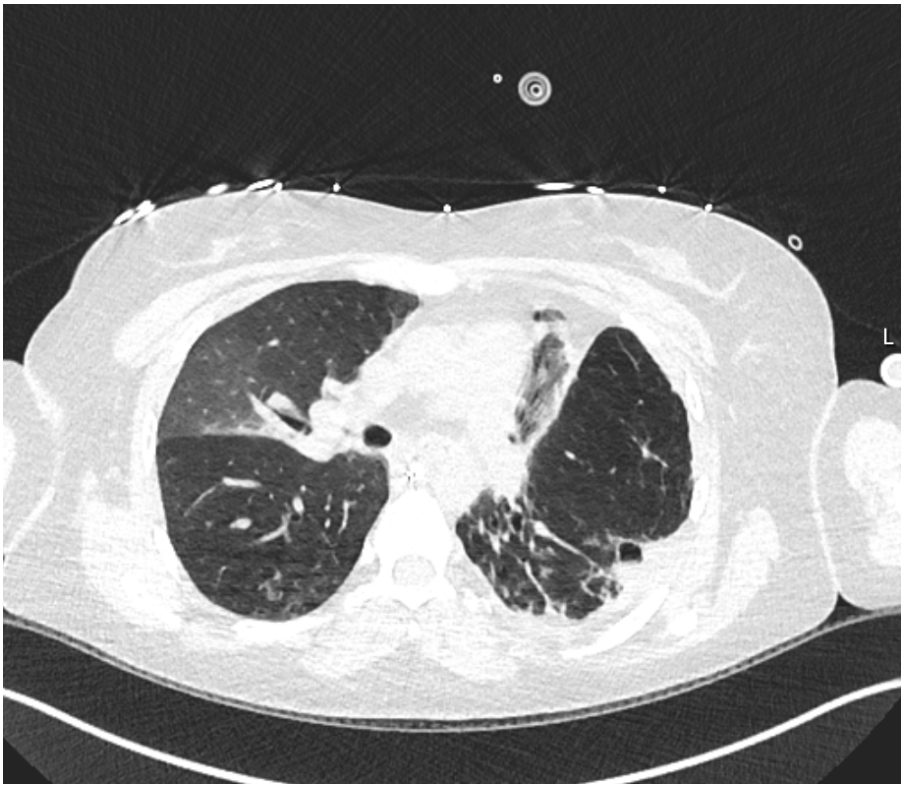
A 23-year-old female with idiopathic familial recurrent multifocal airway stenosis, bronchiectasis, and hypogammaglobulinemia developed severe hypercapnic respiratory failure in the bronchoscopy suite during an attempt at endoluminal bronchial dilation. She was urgently placed on emergency VV ECMO as a bridge to decision. She was unable to be weaned from VV ECMO support and after careful evaluation listed for LT. Severe proximal bronchial stenosis precluded bronchial anastomosis and therefore, she underwent en-bloc LT with tracheal reconstruction above the carina. In order to protect the anastomosis a rotational omental flap was used. Due to the aggressive nature of retraction required for placement of the en-bloc lung as well as the high risk of hemodynamic instability she was placed on CPB for the procedure. The exisiting VV ECMO cannulas were spliced together as drainage and an ascending aortic cannula was placed for arterial flow for cardiopulmonary bypass. Pre-bypass TEE showed normal LV function (EF 55%) and mild RV dysfunction. A fentanyl and propofol based TIVA was utilized in context of her risk for MH. In order to monitor the oxygen saturation, bilateral cerebral oximetry as well as the bispectral index were applied to the forehead. Post-implant TEE confirmed patent pulmonary anastomoses, preserved LV function, and mild-to-moderate RV dysfunction. Extracorporeal support was weaned with epinephrine, norepinephrine, vasopressin, and inhaled nitric oxide. Due to graft edema, the chest was left open for delayed closure, and airway ischemia gradually improved postoperatively. The patient was successfully decannulated from VV ECMO, the chest was closed and tracheostomy was completed.

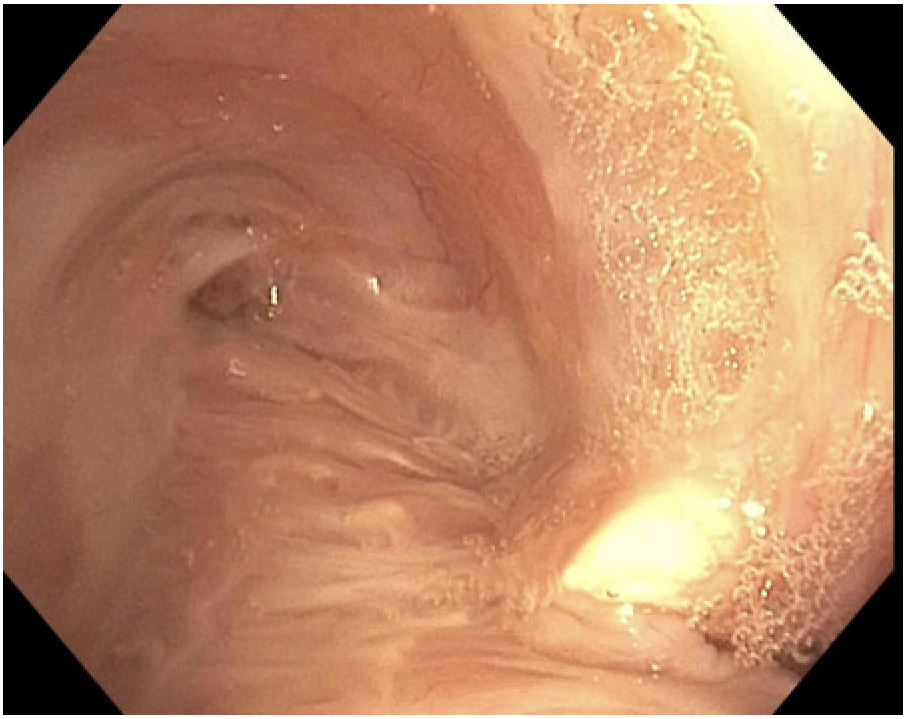
Discussion

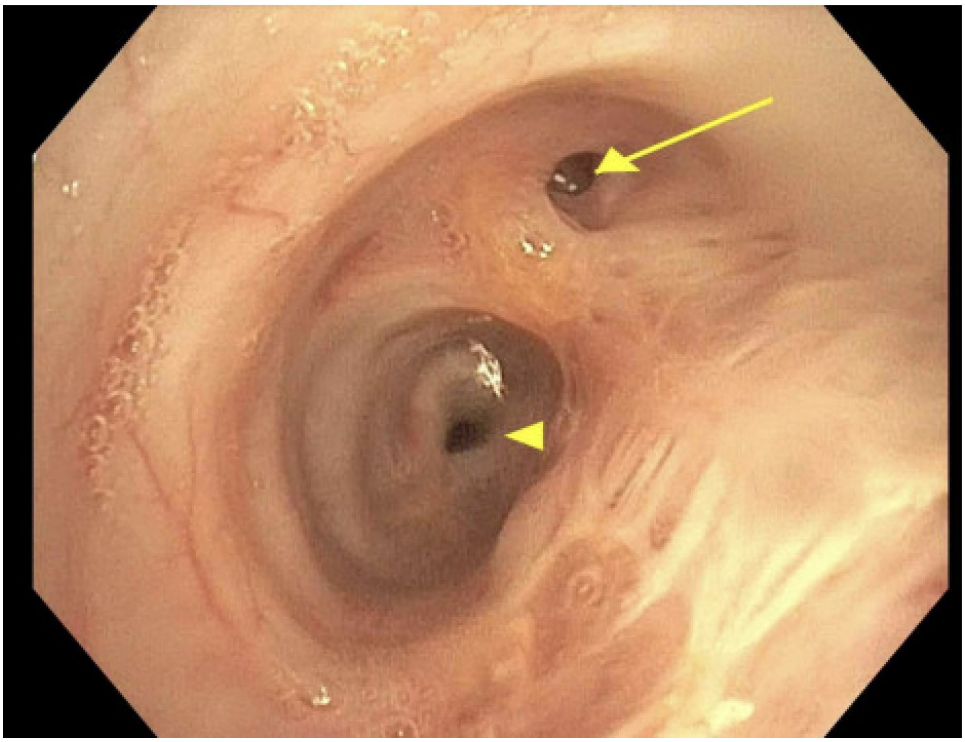
This case illustrates anesthetic management of lung transplantation under TIVA in an MH-risk patient. Avoiding volatile agents required careful IV titration and BIS-guided depth assessment. NIRS provided reliable monitoring of cerebral oxygenation during VA-ECMO and CPB, ensuring early detection of cerebral ischemic insult. The en-bloc transplant and omental flap highlight rare surgical strategies to mitigate

tracheal ischemia. This case underscores the need for multidisciplinary vigilance and innovation in complex cardiothoracic anesthesia.

Images







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#FR 1 – Kiosk 6 – Poster 5

Left Coronary Artery Thrombus Identified with Intraoperative Transesophageal Echocardiogram

Presenting Author: Matthew Saunders, MD, Fellow, University of Alabama at Birmingham Department of Anesthesiology

Introduction

Coronary Artery Bypass Graft (CABG) is the most common open-heart surgery performed each year. The recommended use of intraoperative transesophageal echocardiogram (TEE) by national organizations has varied over the years1. In 2021, only 66.4%1 of CABGs were performed with intraoperative TEE. Use of intraoperative TEE has been shown to improve patient outcomes and is linked to lower operative mortality2. Intraoperative TEE during CABGs enables the surgical team to make informed decisions by evaluating valve function and graft function2. However, intraoperative TEE carries risks of esophageal injury and related mortality and morbidity1. There is also an increase in unplanned procedures when TEE is used during isolated CABGs, but some may argue this uncovers previously unknown valvular pathologies. This case highlights how surgical plans can change based on intraoperative TEE findings.

Case Presentation

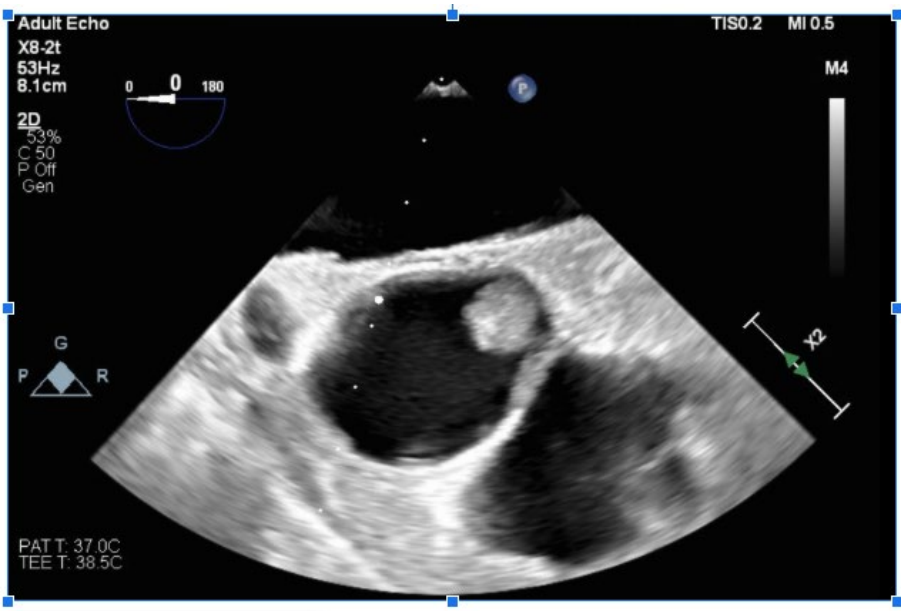
A 62-year-old male presented with chest pain and was diagnosed with a myocardial infarction. His left heart catheterization revealed a 90% occlusive, mobile eccentric thrombus in the left main coronary artery and a 100% occlusive lesion in the mid LAD. The remainder of his coronary vasculature was patent. An intra-aortic balloon pump was placed, and he was transferred to our hospital for surgical management. The patient was brought to the operating room for an off-pump CABG. His baseline TEE confirmed severely reduced systolic function as well as an echogenic mass in the aortic root. It was situated quite close to the aortic valve. It did not appear to be a vegetation related to the valve itself but rather associated with the ostium of the left coronary artery. After discussion with the surgical team, the decision was made to surgically remove the suspicious lesion. Cardiac bypass was initiated, and an aortotomy was performed. The surgical team identified a thrombus entering the left coronary artery. It was removed manually and with suction. A retrograde coronary sinus catheter was used to determine if the left coronary artery was now patent. There was no flow retrograde through the artery, so the team proceeded with placing a bypass graft. Before coming off bypass, an impella was placed for mechanical support. The patient required significant vasoactive support but left the OR in stable condition. He was able to be extubated and had his vasoactive medications weaned. His heart function did not recover. Eventually, an LVAD was placed after which he required increasing support. The patient unfortunately passed away after his family decided to withdraw care.

Discussion

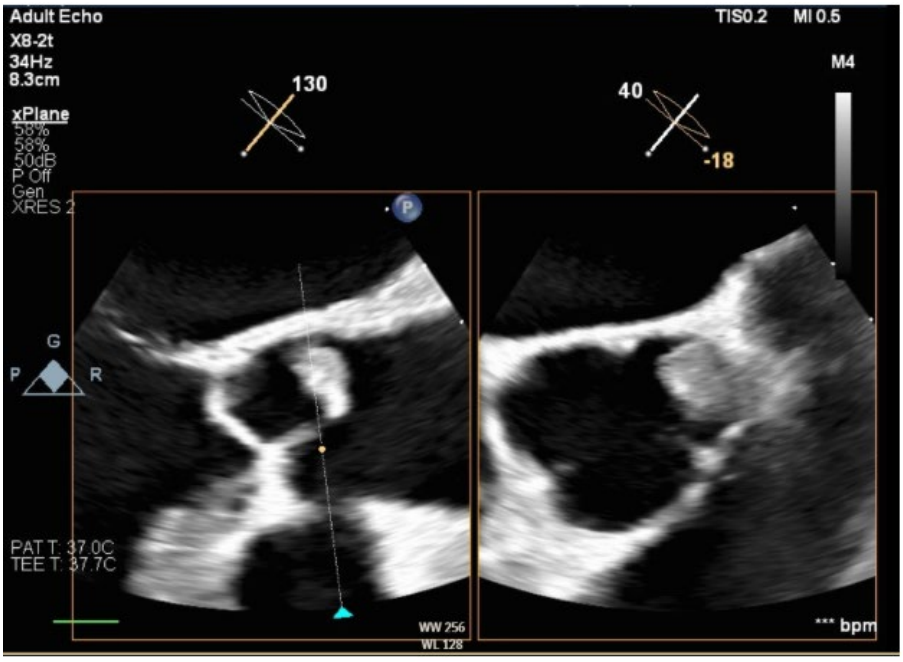
Intraoperative TEE plays a crucial role in surgical planning and patient outcomes. Although not always essential for every cardiac case, intraoperative TEE enables real-time assessment and guides both surgical and anesthetic decisions. This case illustrates that without proper diagnosis via TEE, this patient would not have received the correct surgical intervention. Intraoperative TEE is an important tool in the cardiac

anesthesiologist's toolkit that promotes flexibility and allows for quick responses to the changing nature of cardiac surgery.

Images



Midesophageal aortic valve short axis view showing lesion in the left coronary sinu above the aortic valve.



X-plane imaging show the lesion in the midesophageal aortic valve long axis and short axis. The lesion appears to originate from near the ostium of the left coronary artery.

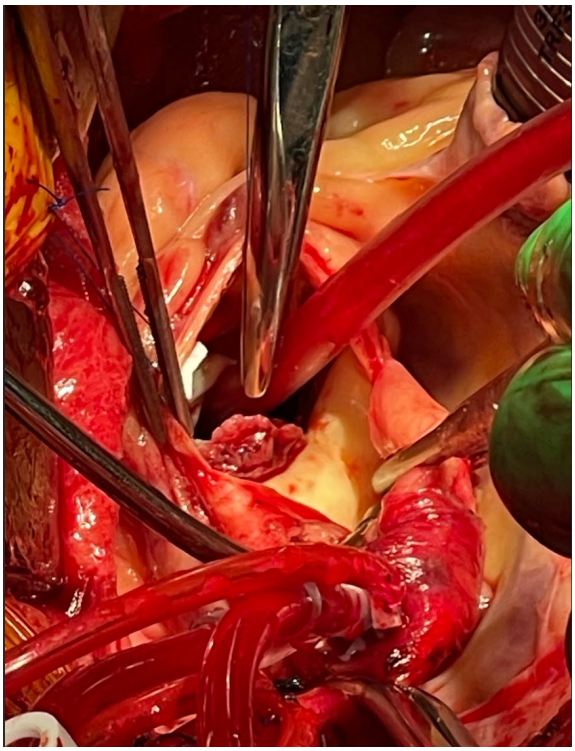
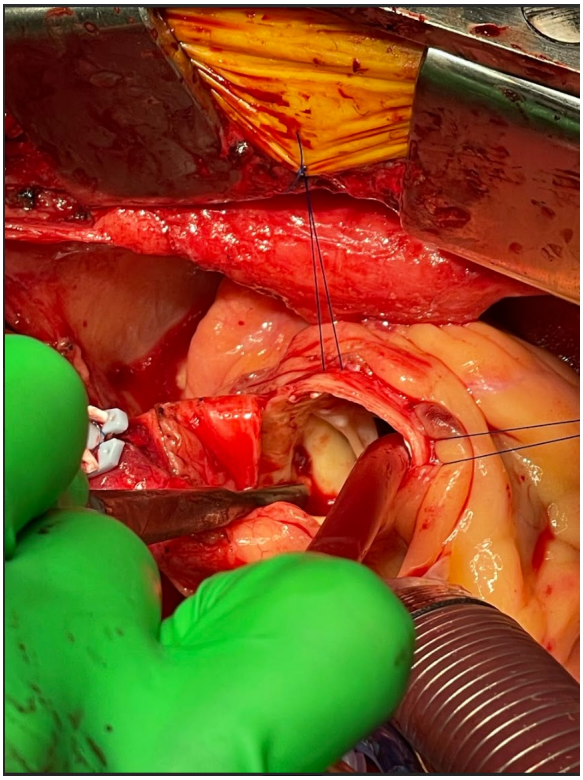


Image of the lesion looking through the aortotomy.



Remainder of the lesion within the left coronary artery after removal of portion seen on TEE.



Largest portion of the lesion after removal.

References

Considerations of Intraoperative Transesophageal Echocardiography During Adult Cardiac Surgery: A Scientific Statement From the American Heart Association Lisa Q. Rong, MD, FAHA, Chair, Linda Shore-Lesserson, MD, FAHA, Vice Chair, Kiran Belani, MD, Abimbola Faloye, MD, Enrique Garcia-Sayan, MD, Jennifer Lawton, MD, FAHA, Timothy Maus, MD, Wanda Miller-Hance, MD, FAHA, Alina Nicoara, MD, Richard Sheu, MD, on behalf of the American Heart Association Council on Cardiovascular Surgery and Anesthesia; and Council on Basic Cardiovascular Sciences

Metkus TS, Thibault D, Grant MC, Badhwar V, Jacobs JP, Lawton J, O'Brien SM, Thourani V, Wegermann ZK, Zwischenberger B, Higgins R. Transesophageal Echocardiography in Patients Undergoing Coronary Artery Bypass Graft Surgery. J Am Coll Cardiol. 2021 Jul 13;78(2):112-122. doi: 10.1016/j.jacc.2021.04.064. Epub 2021 May 3. PMID: 33957241; PMCID: PMC8876254.

#FR 1 – Kiosk 6 – Poster 6

Ventricular Fibrillation During Normothermic HIPEC: When Heat Isn't the Culprit

Presenting Author: Mohammed Rashid, MD, Resident, Advocate Illinois Masonic Medical Center

Introduction

Hyperthermic intraperitoneal chemotherapy (HIPEC) is associated with significant metabolic and hemodynamic stress. Cardiac arrhythmias occur in 3–5% of cases, typically as benign supraventricular tachyarrhythmias. Ventricular arrhythmias are exceedingly rare, with only two published cases linked to cisplatin-based HIPEC under marked hyperthermia (>40 °C). We present, to our knowledge, the first reported case of ventricular fibrillation (VF) during normothermic Mitomycin-C HIPEC, highlighting the diagnostic challenge and physiologic mechanisms involved.

Case Presentation

An 83-year-old woman (EF 38%) with coronary artery disease, LBBB, hypertension, and hypothyroidism underwent cytoreductive surgery followed by HIPEC using Mitomycin-C. Baseline labs were normal. General anesthesia was induced with etomidate and maintained with volatile anesthetics. A pre-induction arterial line and bladder temperature probe were used. During perfusion, the perfusate temperature was 41–42 °C while core temperature remained ≤ 38.5 °C. Ten minutes before the event, 40 mEq of sodium bicarbonate was administered slowly for mild metabolic acidosis. Near the end of perfusion, while the abdomen was being agitated to redistribute perfusate, the arterial line tracing became flat and $EtCO_2$ fell from 33 → 18 mmHg. Despite movement artifact, the team recognized VF within seconds and stopped agitation. Intraoperative ACLS was initiated immediately: chest compressions were started, amiodarone 300 mg IV was given, and defibrillation (200 J biphasic) was delivered within 2 minutes. ROSC occurred at 4 minutes. Post-resuscitation ABG showed normal pH with K^+ 3.1 mmol/L and normal calcium. Potassium replacement was initiated. A chest X-ray prior to extubation showed no acute findings. The patient was extubated at case end, neurologically intact, and transferred to the ICU. No recurrent arrhythmias occurred.

Discussion

Ventricular fibrillation during HIPEC is extraordinarily rare, particularly under normothermic conditions. In this case, transient hypokalemia—likely potentiated by recent bicarbonate administration—may have reduced myocardial stability, lowering the fibrillation threshold. Mechanical abdominal agitation could have triggered transient autonomic imbalance or myocardial stretch, while perfusate-related metabolic stress or localized temperature gradients further increased electrical irritability. Despite significant ECG artifact, early recognition through multimodal monitoring (arterial waveform and capnography) allowed immediate intervention and full recovery. This case emphasizes that VF can occur during HIPEC even without hyperthermia or platinum agents, underscoring the importance of vigilant metabolic surveillance, awareness of medication-induced electrolyte shifts, cautious abdominal manipulation, and readiness for intraoperative resuscitation.



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#FR 1 – Kiosk 6 – Poster 7

When ECMO is a No-Go

Presenting Author: Samantha C. Olivero, MD, Fellow, Ochnser Medical Center

Introduction

Extracorporeal membrane oxygenation (ECMO) is used in complex thoracic surgery when one-lung ventilation or conventional ventilation strategies fail to provide adequate gas exchange. ECMO candidacy must also account for perioperative factors such as anticoagulation, airway interruption, and surgical positioning which alter the relevance of traditional contraindications. Current evidence identifies anticipated non-recovery as the only absolute contraindication; conditions such as prolonged mechanical ventilation, coagulopathy, frailty, neurologic injury, and multi-organ dysfunction represent relative contraindications with heightened risk in thoracic surgery.

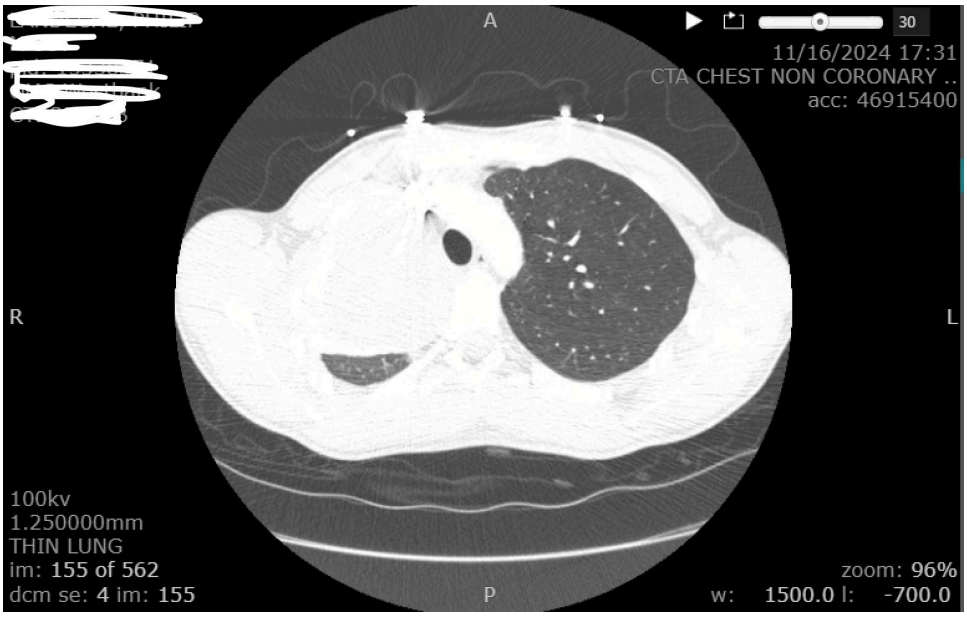
Case Presentation

A 36-year-old male with a history of mixed germ-cell testicular carcinoma (s/p orchiectomy, 2021) presented in acute hypoxic respiratory failure with oxygen saturations in the 60s, requiring intubation. CT imaging revealed a 9.5 × 8 × 7 cm right apical lung mass invading the right mainstem bronchus with mediastinal shift and RUL/RML atelectasis. Cardiac surgery declined VV-ECMO due to presumed metastatic disease and low life expectancy. Thoracic surgery planned urgent airway debulking. During transfer from the ICU bed to OR table, the patient acutely desaturated. Left-lung ventilation was possible, but oxygenation could not be achieved; $ETCO_2$ fell to zero. ECMO was urgently requested. The patient progressed from respiratory arrest to cardiac arrest, requiring 4–5 minutes of CPR before ECMO support was established. Initial TEE showed RV enlargement with mild dysfunction, worsening briefly post-cannulation, then improving within 20 minutes of ECMO initiation. Following stabilization on VV-ECMO, the surgery proceeded without complication and the patient ultimately made a full recovery.

Discussion

In thoracic surgery, ECMO contraindications must be evaluated within a perioperative environment that magnifies bleeding risk, airway disruption, and cannulation challenges. Prolonged mechanical ventilation, coagulopathy, frailty, neurologic injury, and multi-organ dysfunction—identified as relative contraindications in the literature—carry elevated procedural risk in the thoracic OR. Metastatic malignancy is also considered a relative contraindication because it often reflects irreversible systemic disease without a viable pathway to recovery or decannulation. In such cases, ECMO may prolong critical illness without altering overall prognosis, making the risks of anticoagulation, cannulation, and resource-intensive support ethically disproportionate to the benefit. However, for this patient, the immediate threat of profound hypoxemia and impending cardiopulmonary arrest created a scenario in which the urgent life-saving potential of ECMO outweighed concerns regarding long-term oncologic prognosis, supporting emergent cannulation despite metastatic disease.

Images





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#FR 1 – Kiosk 7 – Poster 1

Atrioventricular Groove Disruption After Mitral Valve Replacement Successfully Managed With VA-ECMO Support

Presenting Author: Ricardo Kyoiti Sant Anna, Fellow, University of Western Ontario

Introduction

Atrioventricular groove disruption (AVGD) is an uncommon yet devastating complication, most often occurring after mitral valve procedures, especially replacement. Reported mortality has traditionally been close to 90%, and the literature provides limited guidance on optimal treatment approaches. In this report, we describe a case effectively treated through external repair combined with postoperative VA-ECMO support.

Case Presentation

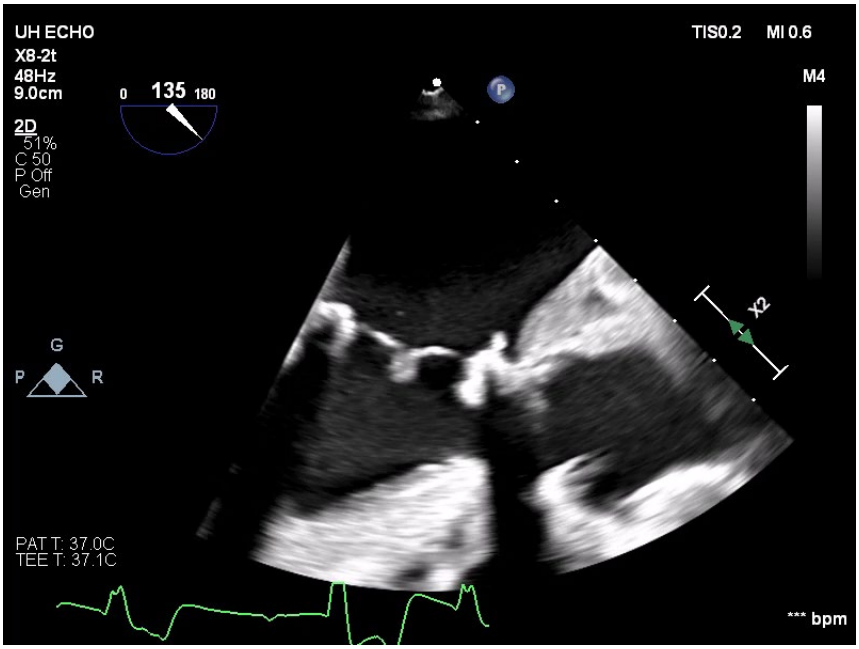
A 64-year-old woman was transferred to our university hospital for evaluation of severe mitral regurgitation, pulmonary hypertension (PSAP 80 mmHg), and RV dysfunction. She reported fatigue and dyspnea (NYHA III). TEE showed mitral annular calcification, anterior leaflet prolapse, and restricted posterior leaflet motion. LV function was normal (LVEF 60–65%), and the RV was dilated with mild–moderate dysfunction. Comorbidities included hypertension, dyslipidemia, type 2 diabetes, and smoking. Coronary angiography showed 95% LCx and 80% RCA stenosis. Surgery planned: mitral valve replacement and CABG. On the day of surgery, intraoperative TEE confirmed prior findings. CABG was performed (radial graft to OM and LITA to LAD), followed by mitral valve replacement with a 27-mm Edwards Mitris valve. After unclamping, TEE showed severe MR with restricted leaflet motion. Bypass was resumed, sutures revised, and a 29-mm Mosaic valve implanted. After the second unclamping, vasopressor needs rose. TEE revealed a clot near the aortic–mitral curtain and annular bruising, raising concern for AV groove disruption (AVGD). Bleeding was manageable, so the team packed and reversed anticoagulation. Due to worsening hemodynamics, central VA-ECMO was started. She was transferred to the cardiovascular ICU with a MAP target of 60 mmHg. Serial TEE every 48 hours showed progressive clot organization. She was decannulated from ECMO on postoperative day 5 and extubated on day 7.

Discussion

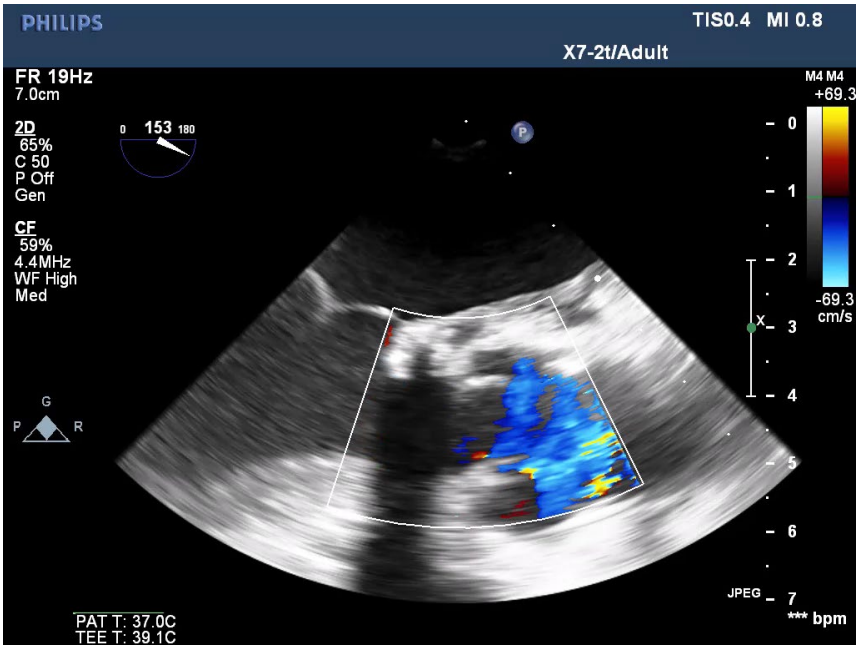
Atrioventricular groove disruption (AVGD) is classically categorized as Type I, II, or III, according to the anatomic site of rupture. Type I, the most common, involves the posterior mitral annulus and adjacent LV

wall and is strongly associated with mitral valve replacement in the presence of severe annular calcification or tissue fragility—features present in our patient. This classification is clinically relevant because Type I disruptions may be managed with external repair or patch reinforcement, whereas Types II and III, which extend laterally or anteriorly, often require more complex reconstruction. In our case, the intraoperative finding of annular bruising and clot formation near the aortic–mitral curtain suggested a limited posterior disruption consistent with Type I. Because the bleeding remained controllable, a conservative external approach combined with anticoagulation reversal was chosen. Hemodynamic instability prompted initiation of VA-ECMO, which served as a crucial adjunct by unloading the heart and allowing stabilization while the AV groove organized. This strategy aligns with reported experiences, although evidence remains limited to case reports.

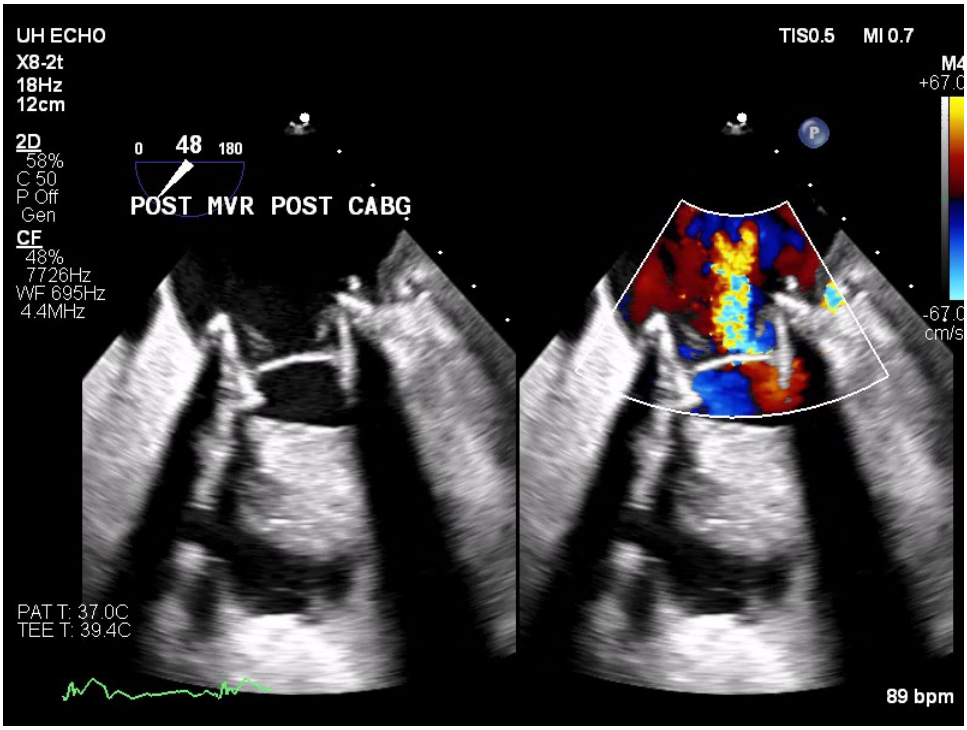
Images



Intraoperative TEE - Hematoma in the AV groove



TEE 3º day PO Patient in VA ECMO - clot in the AV groove with no flow



Intraoperative TEE - central severe MR and para valvular leak

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#FR 1 – Kiosk 7 – Poster 2

Cangrelor based anticoagulation strategy in a case of high-risk PTE for severe CTEPH and suspected HIT

Presenting Author: Muhammad Arif, MD, Fellow, Adult Cardiothoracic Anesthesiology, Temple University Hospital

Introduction

Heparin induced thrombocytopenia (HIT) is an immune mediated complication of heparin exposure in which antibiotics against heparin–platelet factor 4 complex trigger widespread platelet activation, which can induce a pro-thrombotic state despite thrombocytopenia. Currently the mainstay for anticoagulation for procedures requiring CPB is unfractionated heparin. A suspected or confirmed diagnosis of HIT makes heparin contraindicated and alternative anti-coagulation strategies are required. This becomes even more complicated in cases of PTE because of the required circulatory arrest. In cases of HIT requiring CPB, the main alternative anticoagulation is bivalirudin. However, morbidity from PTE with this strategy is high given the risk of thrombosis with bivalirudin due to its metabolism by plasma proteases during circulatory arrest, and for post-CPB bleeding as there is no reversal agent.

Case Presentation

A 29-year-old female with a medical history including asthma, and recurrent DVT/PE was admitted to an

outside hospital with presumed worsening asthma symptoms. After failure to improve with bronchodilators and steroids, she was diagnosed with severe pulmonary hypertension and cor pulmonale. Her course was complicated by thrombocytopenia and a presumed diagnosis of HIT. She was transferred to our tertiary center for higher level of care. On admission, she was in cardiogenic shock eventually requiring VA ECMO. Preoperatively, a TTE demonstrated an LVEF of 20 - 25% and a severely RV dilatation and dysfunction. She had PA pressures of 99/40 mmHg and PA saturation of 36%, and a platelet count 40,000. Intraoperatively, a novel anticoagulation strategy was used to manage the risk of thrombosis and bleeding from HIT. Cangrelor was used to inhibit platelets before the administration of heparin, preventing activation of platelets by any potential HIT antibody/heparin/P4 complexes. Platelet inhibition was confirmed by a point-of-care P2Y12 assay. P2Y12 reaction units fell from a baseline of 193 to 83 after cangrelor administration, indicating effective blockade of platelets. Then, unfractionated heparin was administered to achieve target ACT before onset of CPB and deep hypothermic circulatory arrest. Upon weaning CPB, protamine was administered to neutralize circulating heparin, then the cangrelor infusion was discontinued. She was decannulated from VA ECMO at the end of the procedure and extubated two days later. She suffered a PEA arrest 8 days later. Despite heroic resuscitative efforts and VA ECMO, she suffered anoxic brain injury and was transitioned to comfort care.

Discussion

This case demonstrates the feasibility of a cangrelor and heparin anticoagulation strategy for CPB in a patient with suspected HIT requiring circulatory arrest. Cangrelor has a more rapid onset, shorter elimination half-life, and allows anticoagulant effect of heparin on CPB without activation of platelets by heparin PF4 complexes.This is superior to bivalirudin which forgoes the continuous anticoagulant affect of heparin on CPB and can have a longer elimination half life especially in dialysis patients reaching 3.5 hours. This case had an eventual fatal outcome postoperatively due to the advanced and severe nature of the underlying disease, however the technical result of the surgery was successful.

References

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#FR 1 – Kiosk 7 – Poster 3

Knot the Ideal Situation: Pulmonary Artery Catheter Causing Chordae Tendineae Rupture

Presenting Author: Hernan A. Arias-Ruiz, MD, Resident, Jersey Shore University Medical Center

Introduction

Pulmonary Artery Catheter (PAC) plays an invaluable role in perioperative care by providing continuous hemodynamic monitoring. Despite their diagnostic utility, mechanical complications can occur. Knotting of PACs is a rare complication that can result in significant challenges, such as valvular or vascular injury. We present a case of a knotted PAC that led to the rupture of the tricuspid valve chordae tendineae in a patient undergoing mitral valve replacement. This case underscores the risks associated with PA catheter manipulation, which can result in knot formation, entrapment, and potential chordal or vascular injury. Additionally, it highlights the importance of early recognition of resistance during PAC withdrawal and emphasizes the value of intraoperative transesophageal echocardiography (TEE) for prompt diagnosis.

Case Presentation

In the operating room, a left radial arterial line was first placed, followed by the induction of general anesthesia and intubation. A left internal jugular vein introducer and pulmonary artery (PA) catheter were then inserted, with correct placement confirmed by pressure tracing changes. The procedure proceeded uneventfully until bicaval decannulation. At this point, the PA catheter was withdrawn 5-10 cm to rule out accidental suturing to the decannulation site. After confirming that the PAC was free, multiple unsuccessful attempts were made to advance the catheter back into the PA, showing an erratic PAC waveform. Therefore, the decision was made to withdraw the PAC, during which resistance was encountered. A second attempt resulted in similar resistance, at which point the surgeon confirmed that the PA catheter was not sutured into the SVC. A third attempt was made to withdraw the catheter, at which point a loss of resistance was felt. Subsequently, TEE revealed new-onset severe tricuspid regurgitation (TR) with chordal rupture. Cardiopulmonary bypass (CPB) was resumed for tricuspid valve repair (TVR), and surgical exploration revealed a knot at the catheter's tip within the right atrium (Figure 1). The ruptured chord was excised, and plication sutures were placed. Repeat TEE revealed only mild residual tricuspid regurgitation.

Discussion

Pulmonary artery catheter complications are well-described. Damage to cardiac structures, although rare, remains a critical complication of this technique1. Knot formation is believed to occur when the catheter loops within the right atrium or ventricle, or advances into a dilated heart chamber. Also, changes in heart anatomy following CPB may predispose the catheter to looping or twisting. During catheter retrieval, if resistance is encountered, all traction should be ceased immediately. Intraoperative TEE can provide rapid assessment of catheter location and valve integrity, while fluoroscopy can be used to confirm the presence of a knot and guide retrieval planning. The management strategy depends on the location and mobility of the knot. For proximal knots, an effective maneuver involves tightening the knot against the insertion sheath and removing both the sheath and catheter altogether2. However, for distal knots, more invasive techniques,

such as retrieval baskets, guidewire loop snares, or even surgery, may be necessary to safely remove the catheter3.

Images



Figure 1. Knot in distal aspect of Pulmonary Artery (PA) catheter.

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#FR 1 – Kiosk 7 – Poster 4

Right Atrial Myxoma Resection: New-Onset Tricuspid Regurgitation Requiring a Second Bypass Run

Presenting Author: Liem Nguyen, DO, University of Iowa, Department of Anesthesia

Introduction

Atrial myxoma is the most common benign cardiac tumor, with right-sided lesions accounting for approximately 20%. Sessile right atrial myxomas often adhere to the fossa ovalis and may cause obstructive physiology upon induction of general anesthesia due to the acute reductions in preload and systemic vascular resistance. Excision may create iatrogenic septal defects or unmask tricuspid pathology due to leaflet contact or geometric distortion. This case describes new-onset moderate tricuspid regurgitation (TR) following right atrial myxoma excision, requiring a second cardiopulmonary bypass (CPB) run.

Case Presentation

A 51-year-old woman with no significant medical history was found to have a large right atrial mass during evaluation of syncope from multiple pulmonary emboli, with the most proximal clot located in the left main pulmonary artery (PA). After two months of therapeutic anticoagulation, repeat CT showed stable clot

burden. TTE and cardiac MRI demonstrated normal biventricular function and a large T2-hyperintense right atrial mass consistent with myxoma. Planned surgical intervention included sternotomy, right atriotomy for mass excision, and pulmonary embolectomy. Induction was performed with ketamine and inhalational anesthetic to maintain spontaneous ventilation and preserve preload. A pulmonary artery catheter was avoided due to embolization risk. Post-induction TEE confirmed a sessile mass firmly adherent to the fossa ovalis with no atrial septal defect. Significant systolic contact between the mass and the septal tricuspid leaflet was visualized in the mid-esophageal four-chamber and modified bicaval views. The left main PA clot was also identified. CPB was initiated uneventfully. The mass was excised, and the resultant septal defect repaired with a pericardial patch. The left main PA clot was removed through a PA incision. After separation from CPB, intrinsic rhythm and normal biventricular function returned. TEE showed no residual mass and an intact septum with a negative bubble study. A systematic post-CPB exam revealed new moderate TR with a central regurgitant jet (vena contracta 0.478 cm), without hepatic vein systolic reversal or right atrial dilation. Given the clinical significance of moderate TR, CPB was reinitiated for valve inspection. Although no structural abnormality was identified, tricuspid annuloplasty was performed. Post-CPB TEE demonstrated complete resolution of TR.

Discussion

New-onset TR in this setting may result from previously obscured regurgitation due to tumor and leaflet interaction, geometric distortion from septal patch repair, or loss of mass-related tethering. Moderate TR following CPB is clinically significant. In a large cohort study done at MGH, which was published in 2023, moderate TR was associated with a 2- to 3-fold increase in mortality and major morbidity during the in-hospital and subsequent follow-up period. Early recognition with TEE and timely surgical intervention is therefore essential. This case underscores the importance of comprehensive intraoperative TEE evaluation. Pre-excision leaflet assessment allowed for comprehensive evaluation post-CPB, while repeat exam was critical despite normal pre-CPB findings.

Images

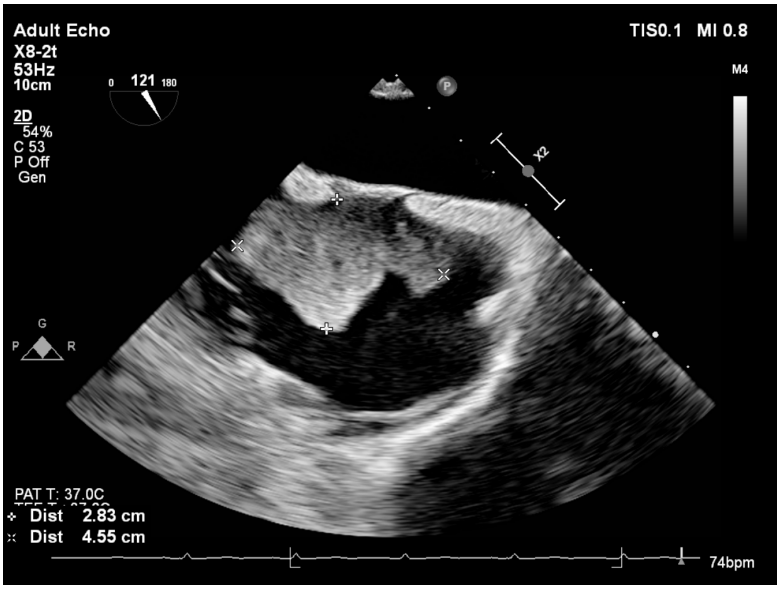


Figure 1. Pre-CPB Mid-Esophageal Bicaval View Large sessile right atrial myxoma firmly adherent to the fossa ovalis. No atrial septal defect is present. Mass \contacts the tricuspid leaflet during systole.

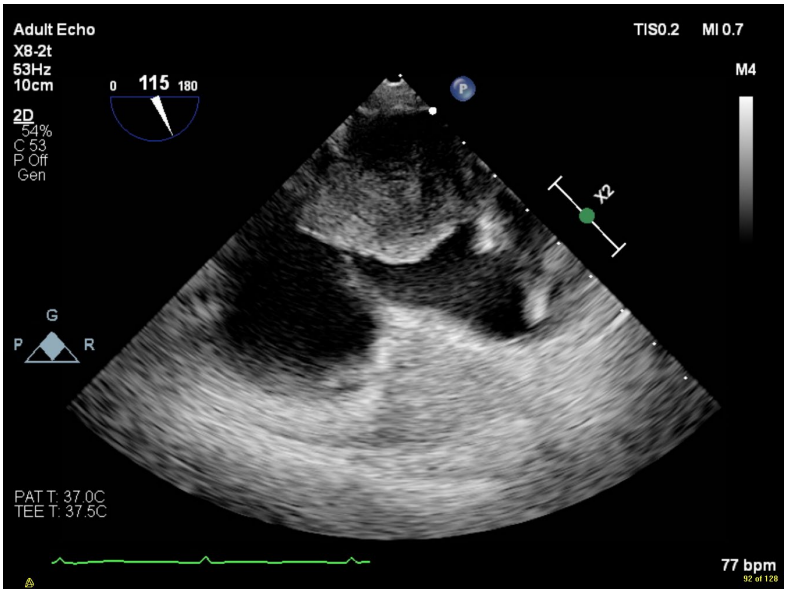


Figure 2. Pre-CPB Modified Bicaval View Systolic contact between the right atrial myxoma and the leaflet of the tricuspid valve, potentially masking preexisting regurgitation or directly related to post-CPB tricuspid valve pathology

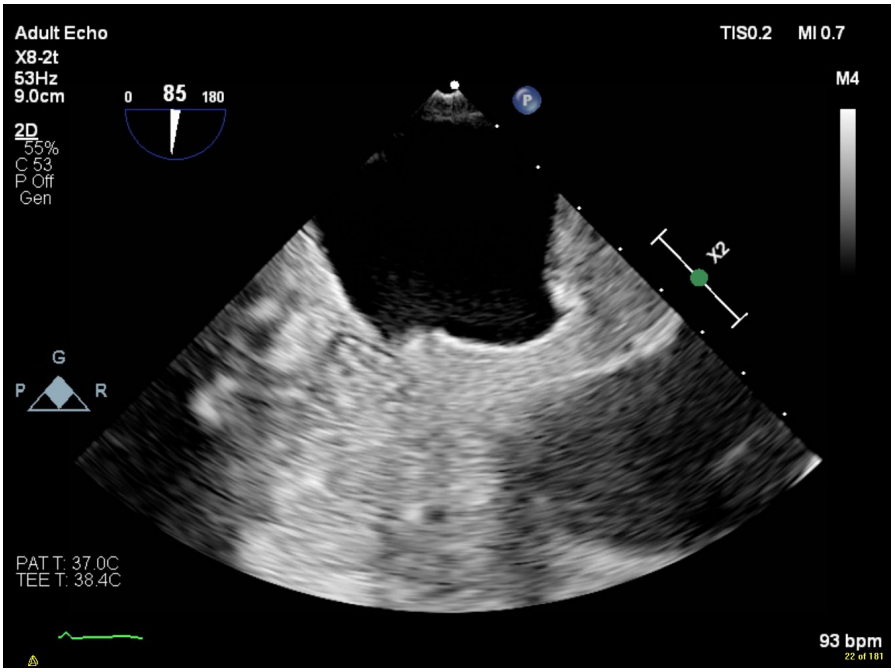


Figure 3. Post-CPB Mid-Esophageal Bicaval view No remnant of large right atrial mass post excision. No intra-atrial septal defect post patch repair. Negative bubble study.

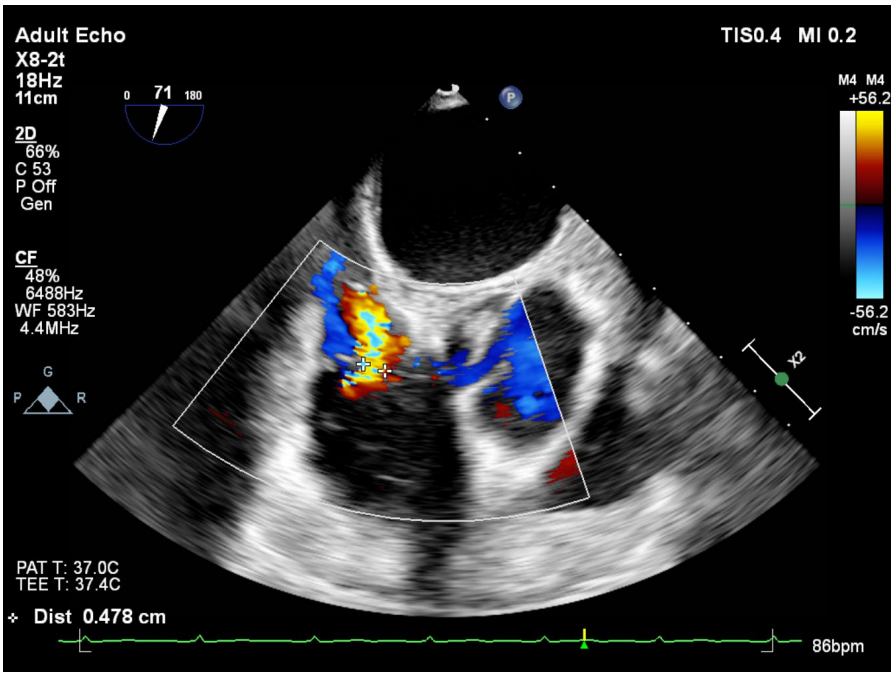


Figure 4. Post-CPB Mid-Esophageal RV inflow-outflow with Color Doppler New moderate tricuspid regurgitation with a central jet with vena contracta measured at 0.478 cm. No hepatic vein systolic reversal. TR resolved following tricuspid ring annuloplasty.

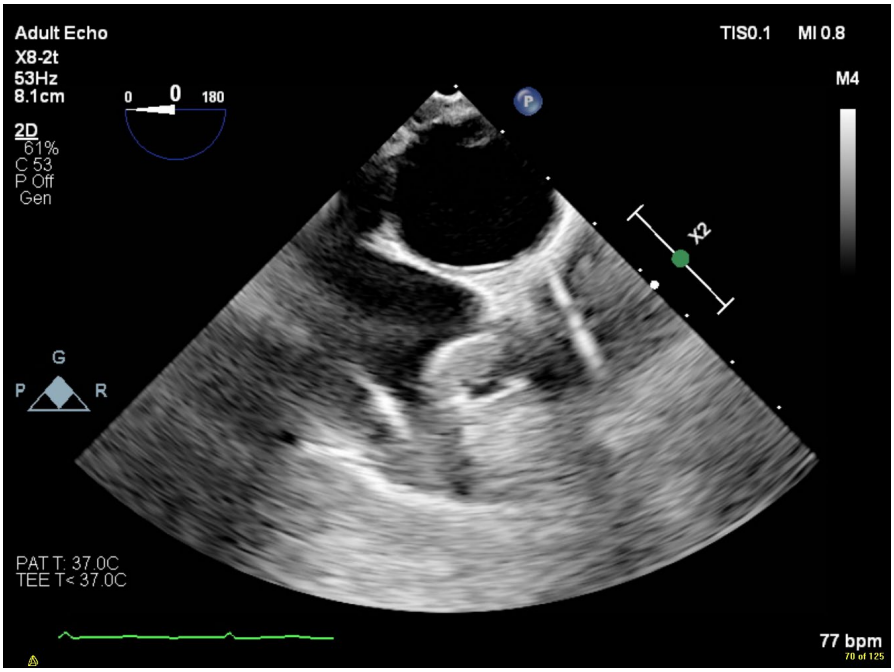


Figure 5. Visualization of left main PA clot prior to embolectomy

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#FR 1 – Kiosk 7 – Poster 5

Common concerns: Combined cardiac and liver transplantation for NICM and secondary cirrhosis

Presenting Author: Rasheeq Nizam, MD, Adult Cardiothoracic Anesthesiology Fellow, Washington University in St. Louis

Introduction

Combined heart and liver transplantation is most often seen for severe amyloidosis. Rarely, severe non-ischemic cardiomyopathy (NICM) may lead to cirrhosis due to congestive and ischemic hepatopathy. Secondary cirrhosis presents additional challenges, such as coagulopathy, hepatopulmonary syndrome, and vasoplegia while exacerbating pulmonary hypertension (pHTN) with superimposed portopulmonary hypertension (ppHTN). While this underlines the importance of RV support, the overlapping anesthetic goals of cardiac and liver transplantation also present opportunities to optimize the patient's physiology.

Case Presentation

Patient JW is a 50-y.o.m. with PMH NICM s/p ICD in 2006 for VF arrest admitted electively for heart and liver transplantation. RHC showed elevated filling pressures and severe pHTN, and TTE showed LVEF 17% with severe LV dilation. Hospital course included IABP placement w/ several replacements and rupture, microaxial pump (Impella 5.5, Abiomed) placement, VA ECMO cannulation, ECMO decannulation and RVAD (CentriMag, Abbott; RfV and R IJ-PA) placement, as well as progression of cirrhosis, renal failure, and ischemic colitis. CT demonstrated mild pulmonary edema and cirrhosis. Pre-operative labs notable for Hgb 9.1, LDH 480, INR 1.4, BUN 33, TBili 4.6, DBili 1.9, Alk phos 207, ALT 50, AST 70. On arrival to OR, JW was supported on epi 0.15 mcg/kg/min, with Impella at P5 and RVAD at 3600 RPM. General anesthesia was induced, JW was intubated and L radial arterial line, L IJ quad-lumen CVC, and R IJ introducer + PAC were placed. MAP 80s and mPAP 1/4 systemic. TEE demonstrated LVEF 10-20%, moderately decreased RV function, severe MR, severe TR, and moderate bilateral pleural effusions. CPB was initiated, including advancement of RVAD femoral venous cannula for IVC drainage, Impella was removed, heart transplantation was performed, RVAD outflow cannula was readvanced, and IVC cannula was retracted to femoral vein for reinitiation of RVAD. Epi 0.1mcg/kg/min and iNO at 20ppm were initiated. CPB was terminated, and epicardial pacing wires were placed, with pacer set to AAI 100. Liver transplantation then proceeded. During reperfusion, JW became hypotensive, requiring dual pressors. JW received 8u pRBC, 6u FFP, 2u Plt, 2 pools cryo, 1750 albumin and 3L crystalloid. Incisions were closed and JW was handed off in the ICU intubated and supported on epi 0.1 mcg/kg/min, norepi 0.12 mcg/kg/min, vaso 0.08 u/min, and RVAD 4500 RPM. JW was extubated POD 1, RVAD was weaned and decannulated POD 7, and JW transferred to the floor POD 12. JW returned POD 13 after emergent ex-lap for hepatic hematomas and lacerations, then again transferred to floor POD 25 and discharged POD 39, hospital day 148.

Discussion

In the setting of preexisting, likely multifactorial pHTN, RV support is vital before and after heart transplantation. While ppHTN is often a contraindication to liver transplantation, in this case it supported

the decision to pursue RVAD support. In addition, the combination of epinephrine infusion, iNO and AAI pacing at 100 bpm served to limit the usual hemodynamic effects of hepatic reperfusion including RV failure, hypotension, and bradycardia, and in turn protected both of the newly transplanted organs.

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#FR 1 – Kiosk 7 – Poster 6

Triple-Envelope Mitral Inflow Profile: Chaos in Hypertrophic Obstructive Cardiomyopathy

Presenting Author: Nicholas R. Phillips, MD, Fellow, University of California - San Diego

Introduction

The phenomenon of a "double envelope" mitral inflow continuous wave doppler profile is well documented. While some have proposed reverberation or "ghosting" artifact as an explanation[1], others have argued that the double envelope results from jet impingement on the ventricular septal wall, leading to sharp deceleration of flow, a pressure spike in the surrounding blood, and acceleration outward along the wall[2]. Another physiological postulate for a double envelope is vortex formation within the left ventricle (LV) which in states of health manifests as two predominant vortices[3]. We present a case of ventricular septal myectomy for hypertrophic obstructive cardiomyopathy (HOCM), where we encountered a triple-envelope in both mitral inflow as well as ventricular outflow profiles on continuous wave doppler (CWD), and attempt to provide an explanation.

Case Presentation

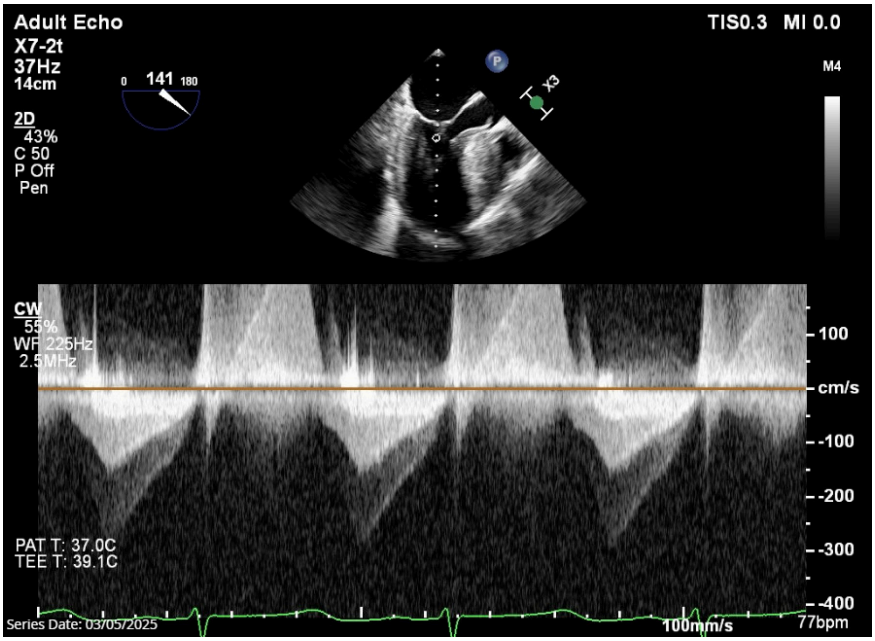
A 35-year-old woman with a history of HOCM secondary to MYBPC3 mutation, multiple syncopal episodes, and prior ICD placement presented for a septal myectomy. General anesthesia was induced after gaining radial arterial access. Following intubation, a central venous introducer and transesophageal echocardiography (TEE) probe were placed. The preoperative TEE exam revealed severe LV hypertrophy with a small LV cavity, a normal right ventricle, moderate tricuspid regurgitation associated with ICD leads, and moderate mitral regurgitation (MR) with systolic anterior motion (SAM) of the mitral valve leading to dynamic LV outflow (LVOT) obstruction. The interventricular septum was hypertrophied to 2.5cm. CWD revealed a triple-envelope mitral inflow profile over repeated acquisitions and views (figure 1). Color-flow doppler revealed diastolic flow convergence near the base of the papillary muscles. Strain analysis was notable for left ventricular dyssynchrony. The case proceeded uneventfully. The postoperative TEE revealed reduction in septal thickness to 1.8cm, improvement of SAM, and decreased LVOT flow turbulence. The LVOT peak pressure gradient decreased from 38 mmHg to 12 mmHg and peak velocity decreased from 3.1 m/s to 1.7 m/s.

Discussion

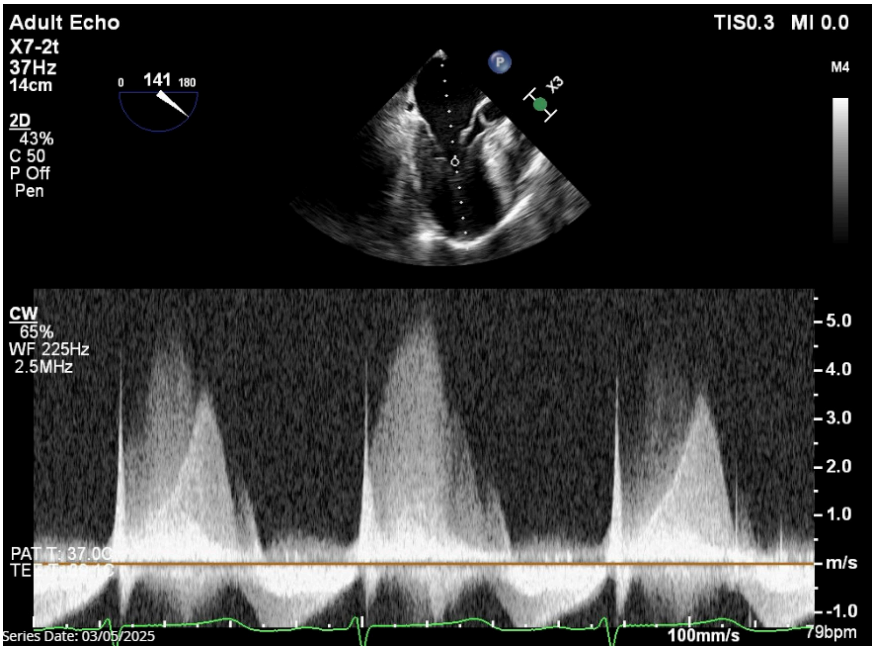
Intracardiac vortex formation and preservation of kinetic energy have been described in states of health[3]. These phenomena are disrupted, often at the expense of greater energy dissipation, in various disease states, including hypertrophic cardiomyopathies[3]. We believe the triple envelope demonstrated here reflects multiple time-discordant regions of flow alteration within the diseased ventricle. We propose artifact as a less likely explanation given the demonstration of the triple envelope over multiple plane angles and acquisitions, and at appropriate gain settings. Indeed, under similar imaging conditions, an asymmetric systolic triple envelope was additionally demonstrated (figure 2). Our case, founded on the vorticial theory of multiple doppler envelopes, suggests that differential diastolic relaxation related to wall asymmetry may

result in intraventricular obstruction to flow at multiple sites and time points. Conventional parameters of diastolic evaluation thus may underestimate the degree of filling abnormality. In the future, this phenomenon may benefit from further interrogation using vector flow mapping and vorticial analysis.

Images



CWD of mitral inflow demonstrating triple envelope



CWD during systole

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#FR 1 – Kiosk 7 – Poster 7

LVAD decommissioning with outflow graft plug via percutaneous approach

Presenting Author: Dominic Carusillo, M.D., Cedars Sinai Medical Center

Introduction

Left ventricular assist devices (LVADs) are indicated for patients with advanced heart failure as they improve survival, functional status, and quality of life in these populations. For select patients who have received LVAD therapy but have device-related complications requiring removal, myocardial recovery, or end-of-life care when continued support is no longer desired, the LVAD is traditionally removed via open surgical explantation, which carries risks inherent to open surgical approaches and cardiopulmonary bypass. Alternatively, the device may be decommissioned via LVAD deactivation and outflow graft occlusion, which can be done via minimally invasive surgical approaches or percutaneously. In this case, we illustrate an example of a successful LVAD decommissioning via percutaneous approach.

Case Presentation

38-year-old male with PMH of non-ischemic dilated cardiomyopathy status post CRT-D and HeartMate3 implantation with chronic LVAD driveline infection. Due to dissatisfaction with quality of life on LVAD therapy, the patient transected the percutaneous driveline. This resulted in retrograde flow into the LV through the nonfunctioning outflow graft, requiring percutaneous plugging of the LVAD graft two weeks later. During this procedure, a Sentinel protection device was used prior to placement of a 22mm and 18 mm Amplatzer plug in the mid and proximal cannula, respectively. No residual flow was seen on angiogram or echocardiogram and post-procedural LVEF was 35%. While there were no immediate procedural complications, the patient ultimately underwent device explantation one year later due to persistent infection.

Discussion

When LVAD therapy is no longer required, open surgical explantation is the traditional approach to deactivation. This involves chest re-entry and cardiopulmonary bypass to facilitate complete device and graft removal. This approach carries significant procedural risks such as higher bleeding risk, possible mediastinal dissection, and complications related to apical ventriculoplasty.2 Because of this, LVAD decommissioning is appealing as it does not entail device removal and current available data shows LVAD decommissioning for myocardial recovery has a similar risk for recurrent heart failure as complete device explantation.1 However, when a continuous-flow LVAD is turned off but the outflow graft remains patent, the pressure gradient between the aorta and the left ventricle can drive blood backward through the device, resulting in retrograde flow, which increases left ventricular volumes and workload. To prevent this, ligation or occlusion of the outflow graft is necessary. Two options to accomplish this exist: surgical ligation via thoracotomy and percutaneous occlusion via endovascular plug placement. The benefit of the latter is the complete avoidance of chest re-entry and appears to have a more favorable procedural risk profile.2 Limited data shows these minimally invasive approaches have equivalent long-term outcomes to open surgical

explantation with regards to rates of cerebrovascular accidents, infection, and mortalility.3 As demonstrated in this case, percutaneous outflow graft plug placement appears to be a safe and effective approach to LVAD therapy discontinuation in select patients.

Images

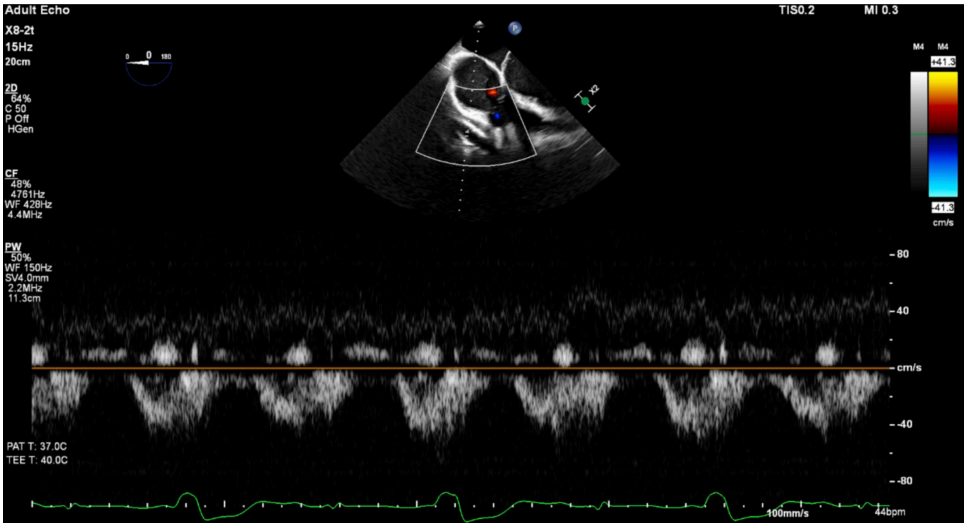


Figure 1. Mid-esophageal four chamber view with pulsed wave doppler in the LVAD outflow graft demonstrating retrograde flow.

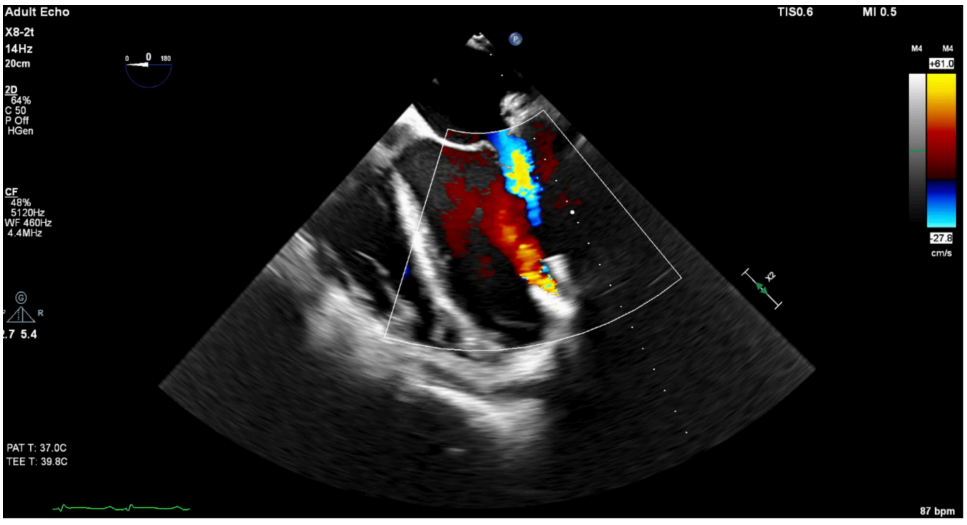


Figure 2. Mid-esophageal four chamber view with Color doppler in LVAD inflow cannula demonstrating retrograde flow into left ventricle.



Figure 3. Post-intervention angiogram demonstrating 18mm Amplatzer plug in the proximal cannula, 22mm Amplatzer plug in the mid cannula, and no residual retrograde flow.

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#FR 1 – Kiosk 8 – Poster 1

Redirecting the Flow: Concurrent TAVR and Closure of a Left-Atrial Draining Persistent Left Superior Vena Cava

Presenting Author: Theodore J. Trumble, MD, Resident Physician – Anesthesiology, University of Kentucky

Introduction

Persistent left superior vena cava (PLSVC) is the most common congenital anomaly of thoracic venous return, occurring in 0.3–0.5% of the general population and 4.5% of patients with congenital heart disease (1). Most PLSVCs drain into the right atrium (RA), but 10–20% drain into the left atrium (LA) (1), which can result in cyanosis, arrhythmias, and paradoxical embolism (2). PLSVC also carries important anesthetic and procedural implications. We present a case in which PLSVC with LA drainage was discovered in a patient with bicuspid aortic valve (BAV) stenosis requiring intervention.

Case Presentation

A 58-year-old man with BAV and moderate-to-severe aortic stenosis (AS), right aortic arch, PLSVC draining to the LA, and remote chest radiation for Hodgkin's lymphoma presented with exertional dyspnea. Air embolism detected on cardiac CT prompted catheterization for angiography and possible PLSVC embolization. Left heart catheterization was performed using a 6-Fr femoral arterial catheter and a 4-Fr catheter advanced from the left internal jugular vein through the PLSVC into the LA and across the mitral valve to the left ventricle. Simultaneous aortic and LV pressures confirmed severe AS. Embolization was deferred due to the newly recognized need for aortic valve intervention. Surgical aortic valve replacement was deemed high risk because of prior chest radiation and the PLSVC's impact on cardiopulmonary bypass cannulation. After multidisciplinary discussion, the patient was scheduled for transcatheter aortic valve replacement (TAVR) with concurrent PLSVC embolization. Under general anesthesia, TAVR was performed with femoral and left IJ access and TEE guidance. A 29-mm Sapien valve was successfully deployed. Via the right femoral vein, an 8-Fr sheath was advanced to the LA fistula. An 18-mm Amplatzer II plug was placed but obstructed bridging veins, necessitating replacement with a 16-mm plug. TEE confirmed appropriate positioning with preserved collateral drainage.

Discussion

PLSVC typically drains into the RA via the coronary sinus; however, LA drainage occurs in up to 20% of cases (1). Diagnosis is usually incidental but can be made with echocardiography, CT, or MRI. Up to 40% of patients have associated congenital anomalies, including ASD, VSD, BAV, and coarctation (3). LA-draining PLSVC carries risks of hypoxemia, embolic events, and important anesthetic considerations (Table 1). These considerations include altered cardiopulmonary bypass strategies with a relative contraindication to retrograde cardioplegia. In some cases, the PLSVC may be clamped but additional collateral flow will need to be monitored. Management options include percutaneous closure, surgical ligation, or transcatheter stenting. In this case, TAVR with simultaneous transcatheter closure successfully redirected venous return

through bridging collaterals.

Images

Anesthetic Implications and Complications	Management
Potential for desaturations and cyanosis	Surgical or catheter-based intervention
Increased risk for arrhythmias, cerebral abscess, and thromboembolic events	Surgical or catheter-based intervention
Difficulty with central venous line and swan-ganz placement	Avoidance of utilizing LIJ or L subclavian for central access
Increased risk of associated congenital anomalies (esophageal atresia, VSD, ASD, TOF, etc.)	Investigate with additional imaging
Relative contraindication for retrograde cardioplegia	May need to clamp PLSVC if retrograde cardioplegia is required (hemiazygos vein may still provide collaterals)
Difficulty with placement of pacemakers/AICD	Tortuous course may increase difficulty and increased risk of arrhythmias, cardiogenic shock, tamponade, and coronary sinus thrombosis

Table 1: Anesthetic Implications, complications, and management of PLSVC



Figure 1: Demonstration of successful deployment of vascular plug with contrast indicating venous flow through bridging vein from LSVC

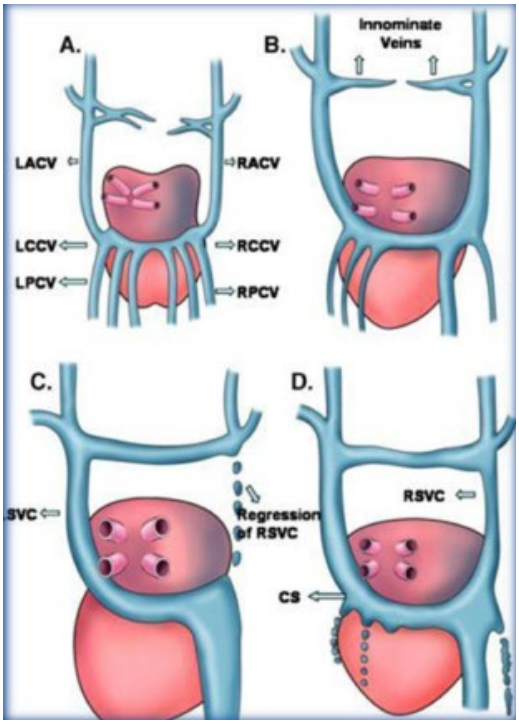


Figure 2: Developmental anatomy of LSVC from posterior view. (A): Embryological cardinal veins (B): Development of innominate vein (C): Abnormal regression of RSVC (D): PLSVC

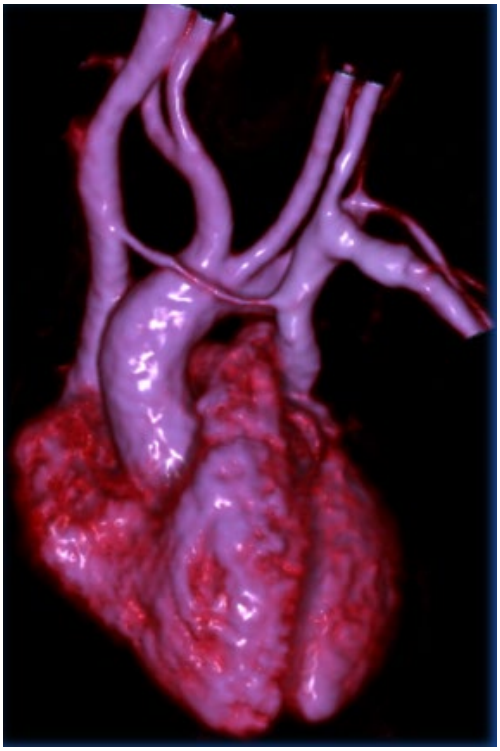


Figure 3 (Left): Anterior view of 3D cMRI demonstrating PLSVC with bridging vein.

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#FR 1 – Kiosk 8 – Poster 2

Successful Perioperative Management of Carcinoid Heart Syndrome During Tricuspid Valve Replacement Surgery

Presenting Author: Derek Wilson, MD, University of Michigan

Introduction

Patients with carcinoid heart syndrome pose a unique set of challenges and considerations for anesthetic management during invasive cardiac surgery.

Case Presentation

A 73-year old female with known GI neuroendocrine tumor (NET) with hepatic metastases, symptomatic carcinoid syndrome, carcinoid heart disease leading to severe tricuspid regurgitation and mild pulmonic regurgitation, NYHA class III heart failure symptoms, mild pulmonary hypertension, asthma, CKD stage 3, pancytopenia, patent foramen ovale (PFO) presented for tricuspid valve replacement and a PFO closure. An octreotide infusion was initiated upon entering the operating room, and general anesthesia was induced without major hemodynamic compromise. Intraoperative hypotension was treated with octreotide boluses. Post-bypass the patient was coagulopathic requiring fresh frozen plasma, platelets, packed red cells, and concentrated factor VII to achieve hemostasis. She did not experience any cutaneous changes or bronchospasm events intraoperatively. The operation and postoperative course were otherwise uncomplicated.

Discussion

NETs commonly produce large amounts of vasoactive substances, most notably serotonin. In normal circumstances, the vasoactive substances are inactivated by the liver, lungs, and brain. However, in our patient with liver metastasis, these substances bypass the liver leading to right sided valve deterioration. The vasoactive mediators classically form fibrous plaques causing tissue distortion. The valvular disease can lead to stenosis, regurgitation, or both resulting in pulmonary hypertension and eventually right ventricular failure. In patients with a PFO, there is a greater chance that left-sided structures may be affected as well. Pain, direct manipulation of tumors, trans esophageal manipulation, and induction of anesthesia can trigger the release of large amounts of vasoactive substances causing profound hemodynamic instability most often in the form of refractory hypotension(1). Classically carcinoid crises are thought to be exacerbated by catecholamines so typical treatment for hypotension including epinephrine, ephedrine, and norepinephrine, are relatively contraindicated(2). Some data suggest the crises can be avoided by pre administration of octreotide. Other data show that octreotide (bolus and infusion) may be less efficacious than typical vasopressors (phenylephrine, and vasopressin), and crystalloid boluses(3). Intraoperatively it can be difficult to quickly differentiate the etiology of hypotension. Octreotide has a relatively small side effect profile so we elected to treat hypotension with octreotide as well as the standard of practice including crystalloid administration and vasopressors(1).

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#FR 1 – Kiosk 8 – Poster 3

Threading the Needle: Managing Ischemia During RV-PA Conduit Revision in a Patient with Single Coronary Anatomy

Presenting Author: Diane L. Wei, M.D., Resident, Brigham & Women's Hospital

Introduction

Tetralogy of Fallot (TOF) is a congenital heart disease characterized by four interrelated structural defects that collectively impair pulmonary blood flow and systemic oxygenation. 69% to 91% of TOF patients survive to adulthood—often with preserved functional status. However, late morbidity persists, with the majority experiencing a major complication by the 50-year post-repair point. Common surgical reinterventions include pulmonic valve interventions, RV-PA conduit replacement, arrhythmia surgery, and repair of residual lesions. Technical complexity increases in reoperations due to adhesions and distorted anatomy, increasing the risk of bleeding, injury to cardiac structures, and longer operative times.

Case Presentation

A 44-year-old female with a history of atrial flutter, atrophic right kidney, intraoperative awareness, and repaired TOF presented for pulmonic valve replacement for severe pulmonic insufficiency and stenosis and frequent heart failure exacerbations. She had prior RVOT interventions, and this would be her third sternotomy. Her anatomy was notable for a single coronary artery (Figure 1) and the presence of two RV outflow tracts, the native RVOT and an RV-PA conduit (Figure 2). Intraoperative course was notable for injury to the RCA during sternotomy. Inferior ST-segment elevations, which corresponded to inferoseptal wall motion abnormalities on TEE, were noted. This precipitated hemodynamic instability requiring vasopressor support and fluid resuscitation. Management of mixed cardiogenic and hypovolemic shock in the setting of ongoing coronary ischemia was further complicated by poor vascular access, leading to an inability to obtain femoral access for emergent cardiopulmonary bypass for 90 minutes. On bypass, the native RVOT was closed with a bovine patch, the caliber of the main PA was expanded with a bovine patch, and a bioprosthetic PV was placed in the RVOT proximal to the RV-PA conduit. Next, the patient was cooled to 28° C, and circulatory arrest was achieved with systemic potassium due to difficulty dissecting out the ascending aorta for cross-clamp and anterograde cardioplegia. This facilitated repair of the RCA. The patient was transferred to the ICU in stable condition and discharged on post-op day 14.

Discussion

TOF patients with prior RV-PA conduit repairs are prone to conduit stenosis requiring serial interventions on the conduit and pulmonic valve. The presence of single coronary anatomy increases the complexity and risk, requiring careful preoperative planning. In this case, we observe how the known complications of reoperative sternotomy carry increased risk of coronary ischemia. Management of mixed cardiogenic and hypovolemic shock in the setting of prolonged coronary ischemia involves the use of inotropes, alpha-agonists, rapid transfusion of blood products, and efforts to reduce both cardiac wall tension and oxygen demand. Surgical strategies include expeditiously initiating CPB to repair or bypass the injured coronary

artery. Notably, manual hemostasis of the injured coronary artery may compromise myocardial oxygen delivery. Thoughtful management of TOF patients considers how existing cardiac structural disease modifies complication risk.

Images

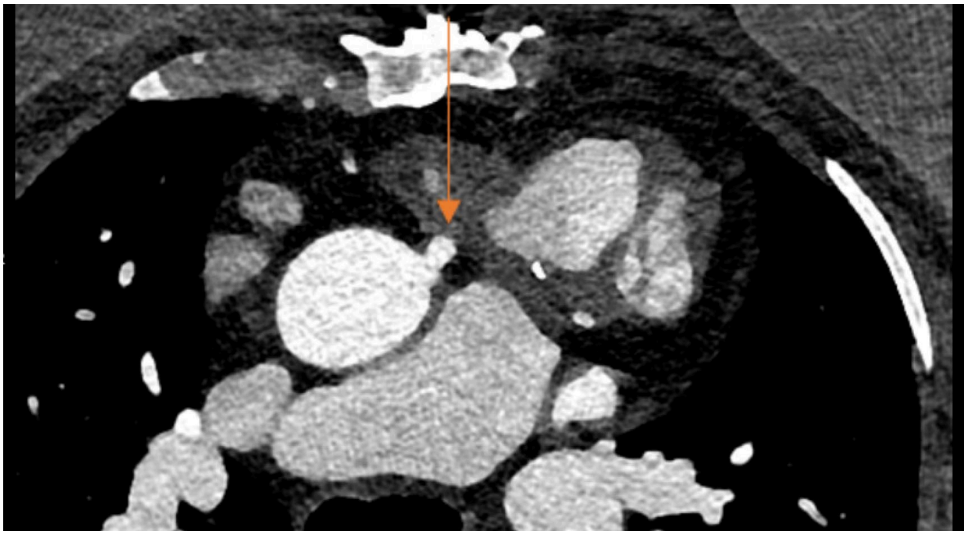
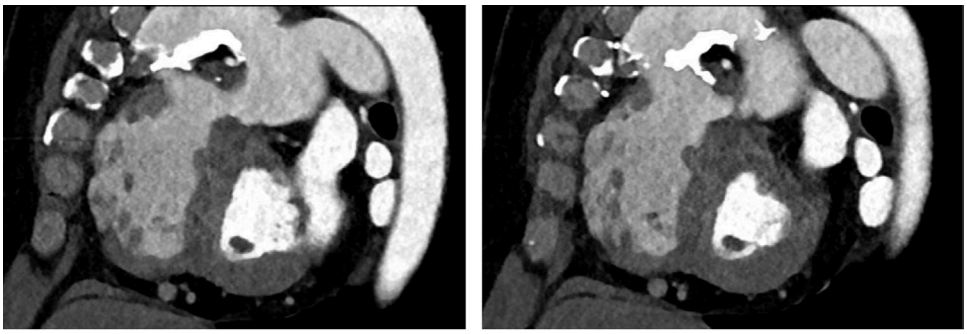


Figure 1: Axial CTA demonstrating single coronary artery.



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#FR 1 – Kiosk 8 – Poster 4

Better Late Than Never: Delayed Tetralogy of Fallot Repair In A Ten-Year-Old Patient

Presenting Author: Joseph A. Zegar, III, MD, PharmDs, Resident Physician, University of Kentucky

Introduction

Tetralogy of Fallot (TOF) is a cyanotic congenital heart lesion characterized by the combination of a ventricular septal defect (VSD), right ventricular outflow tract (RVOT) obstruction, overriding aorta, and right ventricular (RV) hypertrophy. However, there can be significant anatomic variation.1 Most patients undergo repair at 3-6 months of age, but it may be done up to 2 years of age depending on anatomy, growth, and clinical stability.2 Late repairs, defined by age older than 5 years, are typically seen in resource limited countries. We present an unusual case of a 10-year-old male who presented to our institution for repair of TOF.

Case Presentation

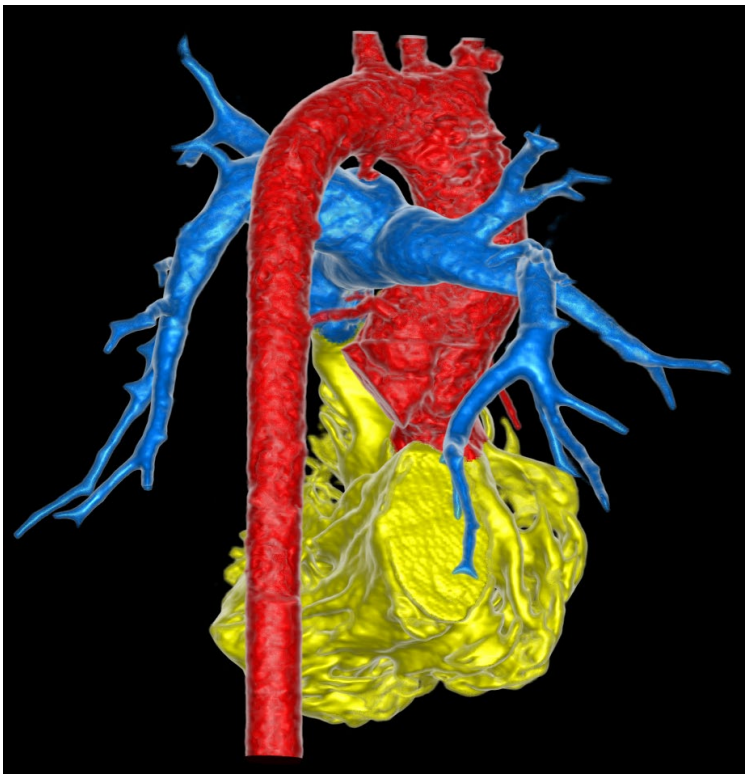
Our patient was a 10-year-old male with TOF. He was initially diagnosed in Mexico but was not referred for surgical repair until he emigrated to the United States. At presentation he reported fatigue and episodes of cyanosis. Labs noted polycythemia and vitals demonstrated hypoxemia in the 70's. Echocardiography revealed a large VSD, RVOT obstruction, hypoplastic pulmonary valve, and a small patent ductus arteriosus (PDA). The patient had an IV induction of general anesthesia. An arterial line and central line were then placed. Prior to bypass, the patient had multiple episodes of cyanosis which improved with phenylephrine, esmolol, and volume. The patient underwent VSD repair, creation of an atrial fenestration, resection of subpulmonic RV muscle band, and PDA ligation. Due to its small size, the pulmonic valve was also excised, and a transannular patch was placed to augment the RVOT. After bypass, there was coagulopathy despite adequate reversal of protamine which improved with administration of platelets and fresh frozen plasma. The case ended uneventfully.

Discussion

The timing of TOF repair varies, but most are done by age one. Our patient is unusual as he presented at age 10 for repair. He likely lasted until then in part due to his PDA providing collateral pulmonary blood flow as his RVOT was effectively banded by infundibular hypertrophy. Severe variants of TOF require urgent repair

before 1 month of age due to severely limited pulmonary blood flow. Such cases may require aortocaval shunt or palliation via the single ventricle pathway.2 Most patients are repaired at 4-12 months to limit RV hypertrophy, avoid a transannular patch, and preserve pulmonary valve function. Unrepaired TOF patients face physiologic consequences related to chronic cyanosis, such as secondary erythrocytosis, coagulopathy, and arrythmias. Interestingly, outcomes are similar in older patients, although they may have long-term issues such as RV failure.3 Anesthetic management in unrepaired TOF patients focuses on avoiding a 'Tet spell.' This includes maintaining systemic and pulmonary vascular resistance, preventing tachycardia, and ensuring adequate preload to minimize right-to-left shunting. Intraoperatively, Tet spells manifest by sudden desaturation, tachycardia, hypotension, and decreased end-tidal CO_2. Management includes use of phenylephrine, deepening anesthesia, volume resuscitation, and beta-blockade to relieve infundibular spasm, thus improving pulmonary blood flow. While uncommon, TOF repair in older patients can still be done safely by understanding their unique anatomy and being prepared for common intra-operative challenges.

Images



A 3D reconstruction of our patient's cardiac CTA, revealing a PDA that connects with the left pulmonary artery.

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#FR 1 – Kiosk 8 – Poster 5

Inter-atrial Septal Thrombus Formation Detected on Transesophageal Echocardiogram during Insertion of WATCHMAN Device: CASE REPORT

Presenting Author: John Carlet, D.O, Resident, St. Mary's General Hospital

Introduction

Spontaneous thrombus formation during the insertion of a WATCHMAN device in patients undergoing percutaneous left atrial appendage closure (LAAC) is a rare complication. The intraoperative incidence of device-related thrombus has not been well defined, and current literature only reports thrombus detection months following the procedure1. Moreover, interatrial septal thrombus has been described only once[2].

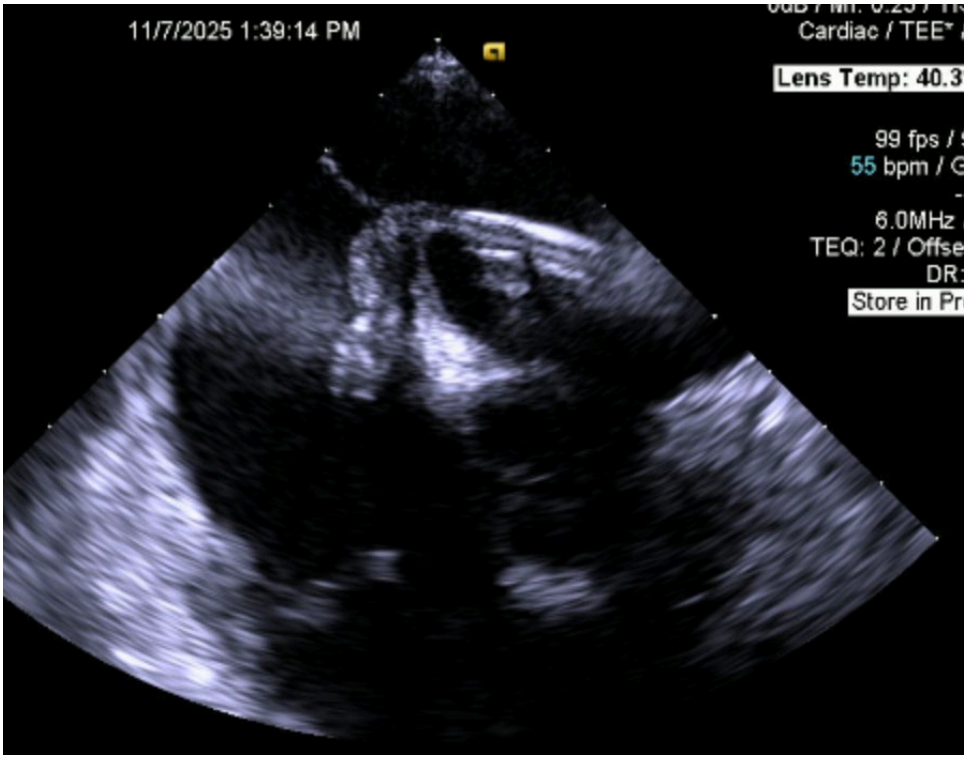
Case Presentation

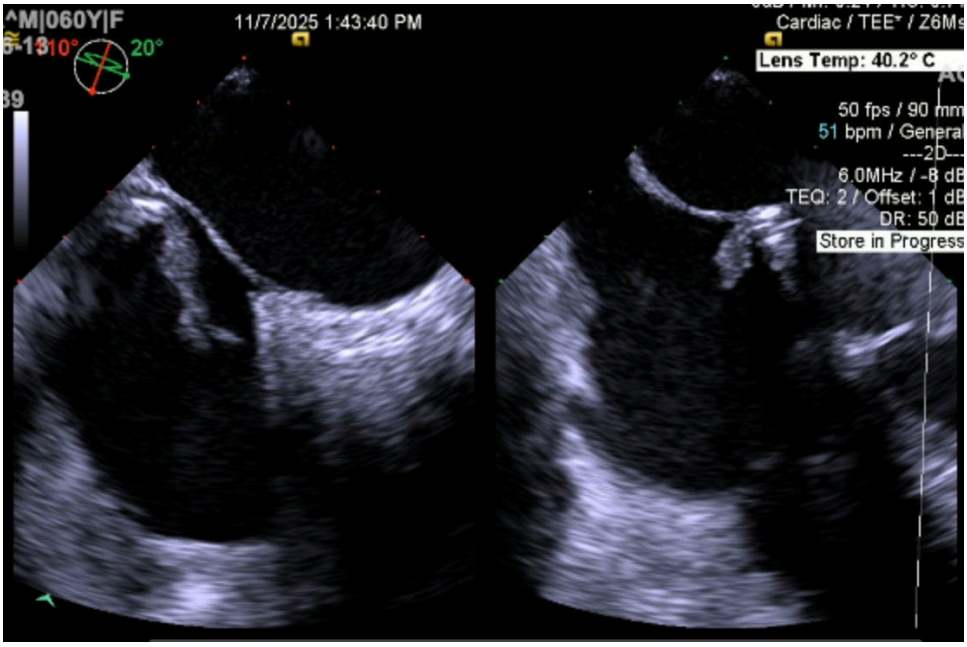
This case reports on a 60-year-old female who was undergoing left atrial appendage closure with WATCHMAN device placement under continuous transesophageal echocardiogram (TEE) guidance. The procedure was complicated by the presence of a filamentous thrombus attached to the sheath in the right atrium at the interatrial septum. The patient's medical history included paroxysmal atrial, chronic diastolic congestive heart failure, hypertension, type 2 diabetes mellitus, and diverticulitis. Consistent with current recommendations for thrombus prevention and management, a heparin bolus of 6,000 units and continuous infusion of 10.5 units/kg/hr were administered prior to transeptal puncture3. Upon detection of the clot, an immediate heparin bolus was administered to maintain the activated clotting time at therapeutic levels. A total of 14,000 additional units of heparin were administered following detection of the thrombus. Due to the decision for ongoing anticoagulation, protamine was withheld. The WATCHMAN device was successfully and optimally positioned and sealed. Post-procedural management emphasized short-term anticoagulation with close monitoring for both bleeding and thrombus resolution.

Discussion

Intraoperative thrombus formation during WATCHMAN device implantation is highly uncommon, as most device-related thrombi are detected on follow-up imaging rather than during the procedure. In this case, thrombus formation was likely attributable to factors such as sheath manipulation, transient blood stasis, and the patient's underlying prothrombotic conditions. Because filamentous clot formation on the interatrial septum has been reported only once, there is a gap in the literature, underscoring the need for vigilance during transseptal access and catheter exchanges. This case highlights the importance of clinical preparedness in managing such complications and the utility of TEE in guiding complex interventions. Continuous TEE surveillance was crucial for immediate detection and rapid adjustment of anticoagulation, potentially preventing further propagation and enabling successful completion of the procedure. TEE serves as an effective real-time diagnostic and monitoring adjunct, essential not only for catheter guidance but also for the simultaneous surveillance of potential complications during LAAC procedures.

Images





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#FR 1 – Kiosk 8 – Poster 6

A Tight Squeeze: High-Risk Bilateral Pulmonary Vein Stenting in a Patient with Complete Pulmonary Vein Occlusion After Atrial Fibrillation Ablation

Presenting Author: Andres Gonzalez, MD, University of Maryland School of Medicine

Introduction

Pulmonary vein stenosis (PVS) is a known but rare complication following radiofrequency (RF) catheter ablation for atrial fibrillation (AF). Due to proximity of PV ostia to the area of ablation in the left atrium, they are susceptible to thermal injury during RF ablation, which can lead to inflammation, endothelial injury and fibrosis causing PVS. Due to procedural improvement, prevalence of severe PVS has drastically decreased from 42.4% to about 2% after 2000.[1] Many with severe PVS may have few symptoms or present with subtle non-specific pulmonary symptoms leading to a misdiagnosis.[2]

Case Presentation

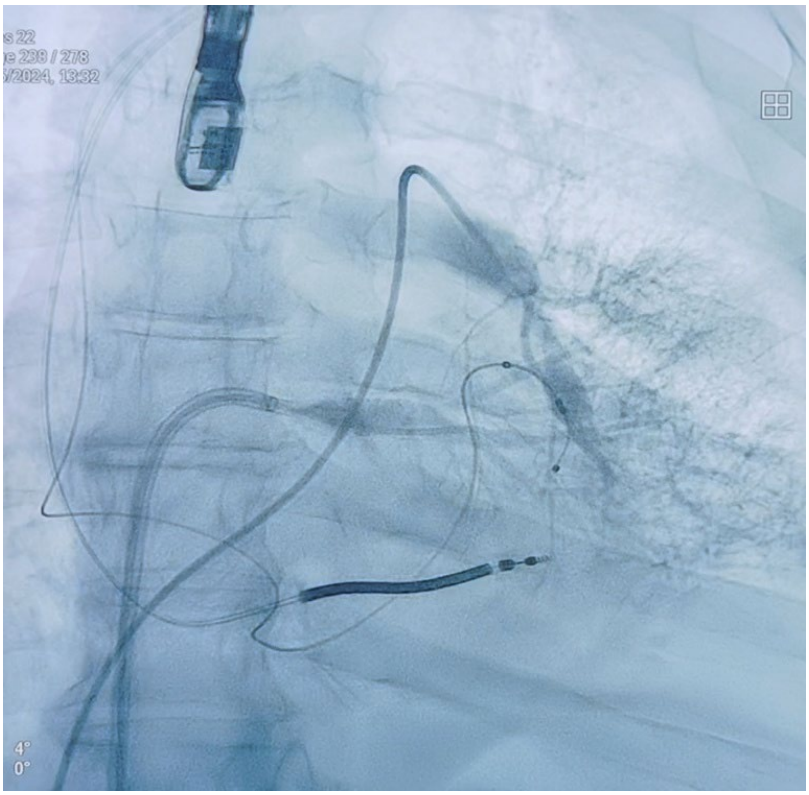
We present a 65-year-old man with history of heart failure and recovered ejection fraction, pulmonary hypertension (PH), recurrent pleural effusions, sleep apnea and AF. He had undergone 2 unsuccessful RF ablations in the past 2-3 yrs followed by pacemaker/ICD implantation. A few months prior, he presented with acute shortness of breath and hemoptysis. Trans-esophageal echocardiogram (TEE) and CT angiogram revealed complete stenoses of both left pulmonary veins. He presented to the cardiac catheterization lab for stenting of his left PVs. Due to anticipated challenging induction given SOB from pulmonary edema and pleural effusions, pre-induction arterial line was placed. Induction with fentanyl, lidocaine, propofol, rocuronium and vasopressor support with phenylephrine was uneventful. Femoral central venous access was provided by the surgeon. Blood was available in the room in event of intra-operative catastrophe. A TEE machine was also readily available if needed. Intra-operatively, he required phenylephrine infusion for support, but course was otherwise unremarkable. Following stent deployment, TEE and fluoroscopic angiography showed successful stent re-canalization of the left PVs with restoration of flow. He was successfully extubated at the end of the case.

Discussion

Since many patients now undergo RF ablation for AF, it is essential for anesthesiologists to be attentive to the serious albeit rare complication of PVS and its management. Anesthetic management of PVS requires understanding of its pathophysiological adaptations as well as the circumstances and medications that may modify management. Stenosis of the pulmonary veins leads to increased pulmonary venous pressures, which can result in pulmonary edema and PH. As PH progresses, the RV gets increasingly strained, elevating the risk of RV failure. Hypoxia, hypercarbia, acidosis, elevated airway pressures, and sympathetic surges can all promptly elevate PVR and should be avoided. In patients with severe PH, inhaled nitric oxide or prostacyclins can be used to help offload RV strain. Stable blood pressure is crucial since PVR and the RV are sensitive to systemic hemodynamics. Vasopressin and norepinephrine are excellent options as they maintain systemic vascular tone without worsening PVR. Re-perfusion pulmonary edema is often seen due

to the sudden restoration of flow to the chronically obstructed vessel.3 This presents with hypoxemia due to V/Q mismatch and pink, frothy secretions. Also, post-reperfusion, the left heart will see an acute increase in preload which could lead to acute heart failure or malignant arrhythmias. This procedure should be performed at a tertiary care center where ECMO is available.

Images



Retrograde flow of contrast from left pulmonary veins into the left lung vasculature, demonstrating successful recanalization.

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#FR 1 – Kiosk 8 – Poster 7

Cold Antibodies, Warm Heart: Navigating Anti-M Before Bypass

Presenting Author: Sheron Mehak, Resident Physician, Robert Wood Johnson Medical School

Introduction

Cold-reactive antibodies are an important consideration in cardiovascular surgery because hypothermia during cardiopulmonary bypass (CPB) can enhance temperature-dependent erythrocyte agglutination. Anti-M antibodies, typically naturally occurring Immunoglobulin M alloantibodies, may bind more strongly to M-antigen–positive red blood cells at lower temperatures and raise concerns for perioperative hemagglutination or hemolysis. Patients with anti-M undergoing hypothermic procedures are therefore often evaluated with a cold agglutinin test to assess for broader cold autoantibodies that could complicate bypass. A negative result helps confirm that no high-titer, clinically significant cold autoantibodies are present, although transfusion planning must still account for the alloantibody. We describe a case of coronary artery bypass grafting (CABG) in a patient with anti-M antibody activity who underwent a successful operation after targeted perioperative evaluation.

Case Presentation

A 67-year-old female with multivessel coronary artery disease was scheduled for elective CABG. On the day of surgery, routine blood bank screening identified anti-M alloantibodies, and the case was delayed when compatible M-antigen–negative units were not immediately available. Concern arose that the antibody could demonstrate cold reactivity, creating risk during hypothermic cardiopulmonary bypass, and the operation was cancelled for further evaluation. A cold agglutinin test with reflex thermal-amplitude assessment was performed to determine whether the antibody reacted at CPB-relevant temperatures and to guide consideration of alternative strategies, including warm cardioplegia, off-pump revascularization, or percutaneous coronary intervention (PCI). The test was negative, indicating no broad cold-reactive autoantibodies, and compatible blood products were obtained. Two days later, she underwent routine CABG ×2 without complications. CPB proceeded with mild hypothermia to 34 °C, with 77 minutes of aortic cross-clamp time and 97 minutes of total CPB time with successful outcomes.

Discussion

This case demonstrates the perioperative impact of unexpected red cell antibodies in cardiac surgery and the need for rapid, multidisciplinary coordination. Although anti-M is often benign, some IgM variants may show cold reactivity and create risk during cooling. In patients with clinically meaningful cold agglutinins, exposure to very cold solutions such as 4 °C cardioplegia can trigger agglutination or complement-mediated hemolysis, potentially impairing myocardial perfusion. The unexpected identification of anti-M therefore required timely assessment of thermal behavior, procurement of antigen-negative blood, and consideration of alternative approaches such as warm cardioplegia, normothermic or off-pump techniques, or PCI. Cardiac anesthesiologists play a central role in this evaluation by coordinating immunohematologic testing, guiding temperature strategies, and aligning perfusion and transfusion planning. The negative cold agglutinin test permitted routine mild hypothermia, underscoring the value of early recognition and structured perioperative planning when unexpected alloantibodies are discovered on the day of surgery.

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#FR 1 – Kiosk 9 – Poster 1

As Stubborn as A Mule: The Tale of Intraoperative Heparin Resistance

Presenting Author: Chukwudubem Obianagha, DO, MBA, MPH, Anesthesiology Fellow, Cleveland Clinic Foundation

Introduction

Patients with heparin resistance (HR) undergoing cardiopulmonary bypass (CPB) are at an increased risk for bleeding, thrombosis, renal injury, stroke, myocardial infarction, and mesenteric ischemia [1]. Here, we present a case of HR due to severe antithrombin III (ATIII) deficiency. In this case, our institutional HR protocol was insufficient, and therefore demanded multidisciplinary decision-making to overcome the problem.

Case Presentation

This is a case of a 49-year-old (76kg, BMI: 26) who presented for robotic mitral valve repair due to severe mitral regurgitation. The patient's perioperative period was uneventful until heparinization for CPB. Our institutional protocol was followed - a bolus of heparin 400U/kg was administered, and an activated clotting time (ACT) was obtained 3 minutes later which resulted as 302s. When the ACT is short of 480s, the protocol recommends administration of a second heparin bolus (200IU/kg); subsequent repeat ACT was 343s. Next, one vial (500IU) of ATIII was administered. The ACT continued to be subtherapeutic even after two vials of ATIII (1000IU), additional 10,000U of heparin, and measurement of the ACT with a separate device. At this time, the patient's heparin resistance extended beyond the institutional algorithm. Hematology was consulted and advised to administer an additional 1000 units of ATIII and 2 units of FFP for presumed severe ATIII deficiency and an additional 200IU/kg of heparin from a different vial source which finally achieved an ACT of 567s. Retrospectively, intraoperative coagulation tests revealed: elevated Factor VIII, normal thromboelastography with heparinase, elevated PT/PTT and Heparin anti-Xa after heparin: 600IU/kg and ATIII: 1500IU were administered. Postoperatively, hematologic consultation revealed no known family history of hematologic disease but a history significant alcohol use. The patient had reoperation for bleeding and was discharged on postoperative day 7.

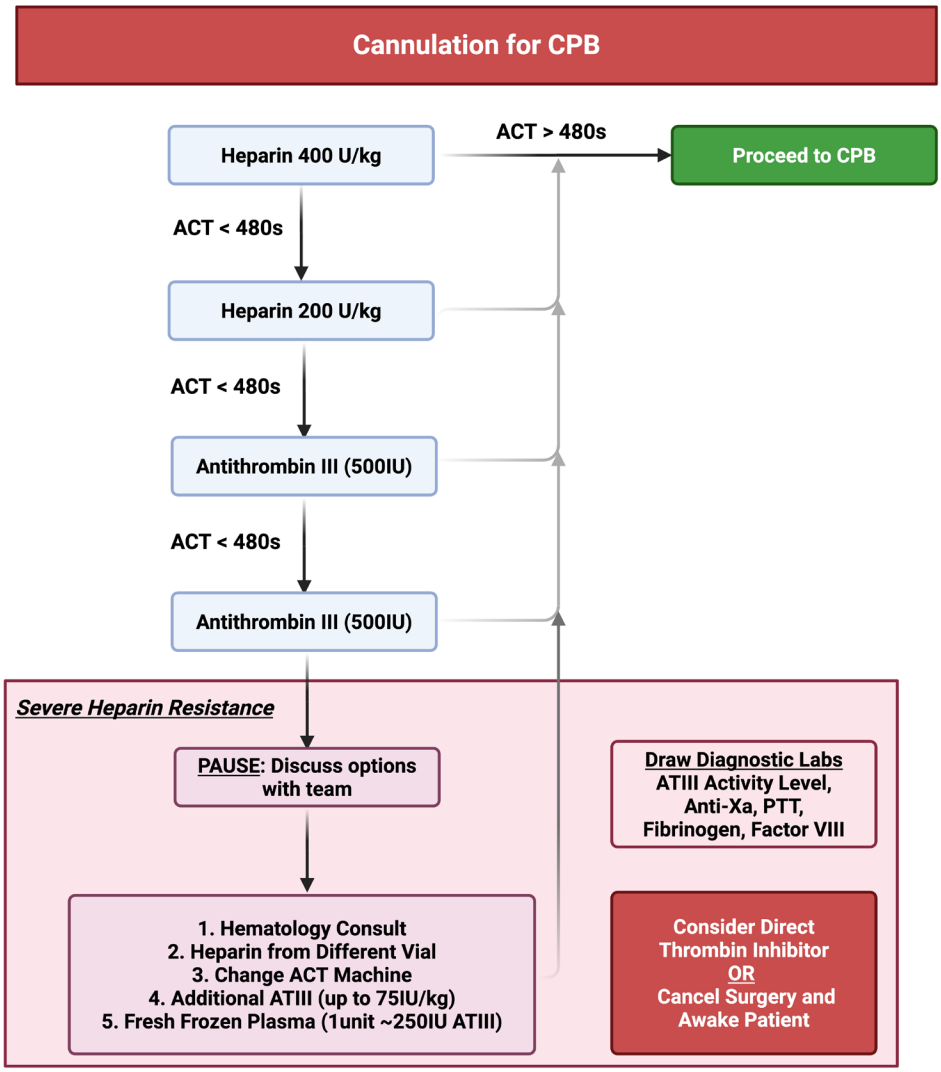
Discussion

Unfortunately, there are no established consensus protocols to define HR and treatment. HR can take two forms: (1) increased levels of heparin binding proteins (2) ATIII deficiency (2a) acquired or (2b) inherited [2].

Patients with alcohol use disorder and cirrhosis have been shown to have lower levels of ATIII, [3] which could explain our intraoperative course. While ATIII has been an effective means of correcting HR, the dosing and target levels of synthetic ATIII concentrate are poorly defined. ATIII deficiency in severe cases may require up to 75U/kg of synthetic ATIII to achieve adequate supplementation [2]. Fresh frozen plasma may be a less costly alternative to ATIII supplementation; however, each unit FFP is equivalent to only 250IU of synthetic ATIII and transfusions carry risk of complications including transfusion related acute lung injury (TRALI) and immunologic reactions [1]. Here, we propose an updated clinical guide to significant HR encountered intraoperatively (Figure 1). This case highlights the need for cardiothoracic anesthesiologists to develop expertise in HR and lead the intraoperative management of this complex disease state.

Images

Severe Heparin Resistance Treatment Algorithm



Heparin Resistance Algorithm

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#FR 1 – Kiosk 9 – Poster 2

Precision Under Pressure: Valve-Sparing Repair of a Giant Pulmonary Artery Aneurysm

Presenting Author: Karyn E. Barrett, MD, Stanford University School of Medicine

Introduction

Pulmonary artery (PA) aneurysms are a rare phenomenon (0.007% prevalence) scantly described in literature with varying definitions and no consensus on surgical repair indictations.[1] Some accepted guidelines for surgical repair include size greater than 60mm, significant pulmonary valve insufficiency, development of clinical symptoms such as dyspnea, impending signs of rupture, pulmonary hypertension (pHTN), and compression of adjacent structures.[1] There are no evidence-based recommendations for anesthetic care and there is limited literature on the anesthetic implications of the disease, but it is suggested to minimize wall stress to decrease the risk of dissection or rupture.[1]

Case Presentation

A 66-year-old woman with pHTN, HTN, OSA, obesity, Ortner's syndrome, and chronic exertional dyspnea was incidentally found to have an 80mm main PA aneurysm extending into the right (57mm) and left (45mm) branches on CT scan (Fig. 1). Pre-operative TTE showed a normal pulmonic valve with mild-moderate regurgitation, mildly reduced right ventricular (RV) systolic function, and a moderate pericardial effusion (Fig. 2 and 3). Right heart catheterization revealed mean PAP of 41mmHg, improving to 29mmHg with nitric oxide challenge. With no history of connective tissue disease, the aneurysm was attributed to pHTN. She was urgently scheduled for surgical resection and PA reconstruction given the symptomatology and giant size of PA aneurysm. Based on the preoperative imaging and intraoperative TEE the PA was noted to taper to normal size at the pulmonic root and the pulmonary valve remained intact, allowing for a valve sparing PA replacement and reconstruction of right and left pulmonary arteries. Due to increased risk of PA rupture with manipulation, we elected to forgo PA catheter placement as TEE images displayed RV systolic function was only mildly decreased. Patient tolerated induction and bypass periods well and was weaned on epinephrine and inhaled nitric oxide at 20ppm. Post-operative TEE revealed normal biventricular function and trace pulmonary regurgitation. Patient was transferred to ICU and extubated the same day. Her chronic hoarse voice started to improve on POD1 and discharged home on POD9.

Discussion

Given the giant size of this PA aneurysm and clinical symptoms, surgical repair was indicated. Due to the rarity of PA aneurysms anesthetic management lacks consensus. Literature suggests avoiding increases in pulmonary vascular resistance to prevent an increase in RV afterload and aneurysm wall tension thereby increasing the risk of rupture.[2] We designed a strategy that avoided abrupt increases in pulmonary vascular resistance, maintained RV perfusion, and avoided aneurysm rupture. A hemodynamically stable induction prevented sympathetic surges, hypoxia, and hypercarbia. TEE served as our primary monitor for RV function, PA anatomy, and volume status. RV dysfunction was managed proactively with inhaled vasodilators, vasoactive support, and careful volume management. Given the rarity and complexity, multidisciplinary

planning was essential to ensure safety of the patient and success of the operation. Improved reporting of these rare cases is needed to better define surgical repair indications, optimized anesthesia care, and long-term outcomes.

Images

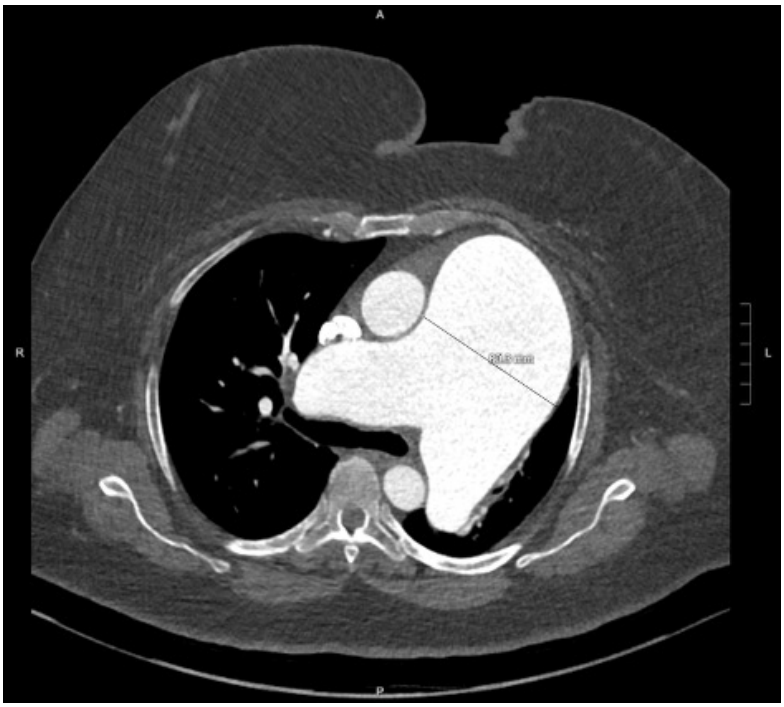


Figure 1: Computed tomography angiography demonstrating giant aneurysm of main and branch pulmonary arteries

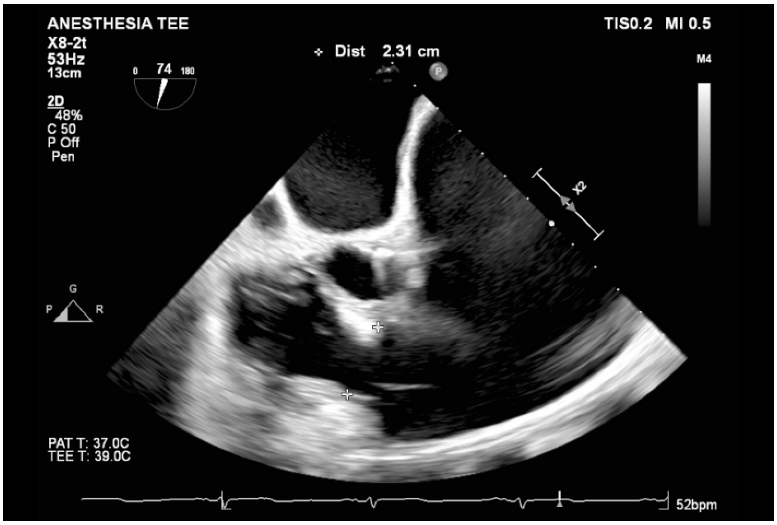


Figure 2: TEE image of a modified RV Inflow-Outflow view demonstrating a preserved pulmonary valve diameter of 2.31cm and giant pulmonary artery aneurysm

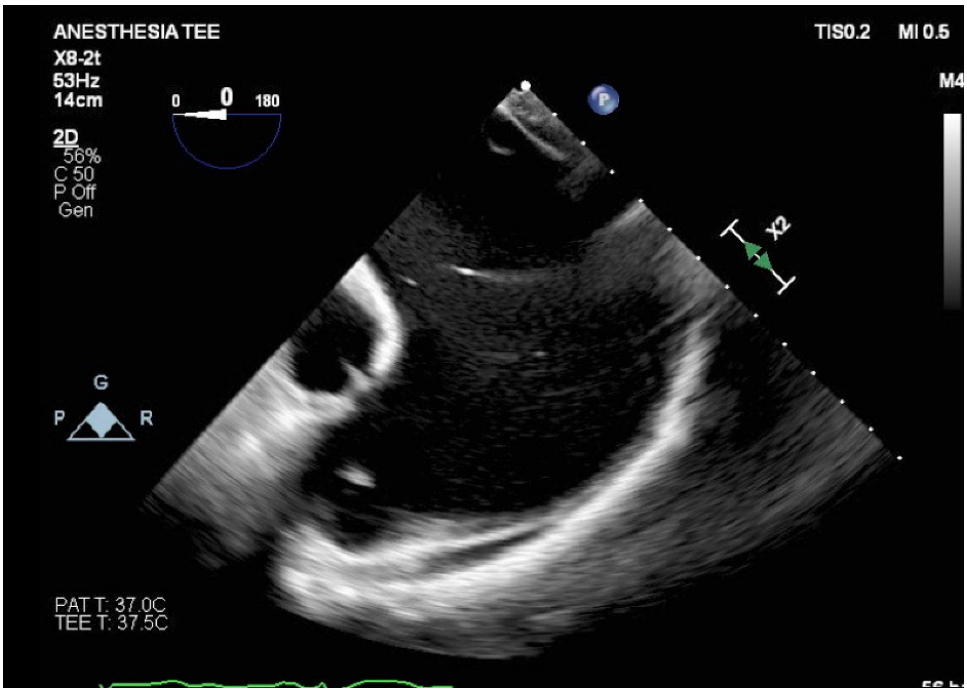


Figure 3: TEE image of mid-esophageal ascending aorta short axis demonstrating giant PA aneurysm

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#FR 1 – Kiosk 9 – Poster 3

Aorto-Left Atrial Fistula Following Aortic Valve Replacement

Presenting Author: Sukruta Pradhan, MD, Adult Cardiothoracic Anesthesia Fellow, University of Louisville

Introduction

Aorto-atrial fistulas (AAF) are complex conditions which present unique challenges due to continuous blood blow from the aorta into the left atrium in all phases of the cardiac cycle.[1] Unfortunately, low incidence has precluded optimal therapeutic strategies. We present a patient who developed an AAF after a complex aortic valve replacement (AVR) surgery.

Case Presentation

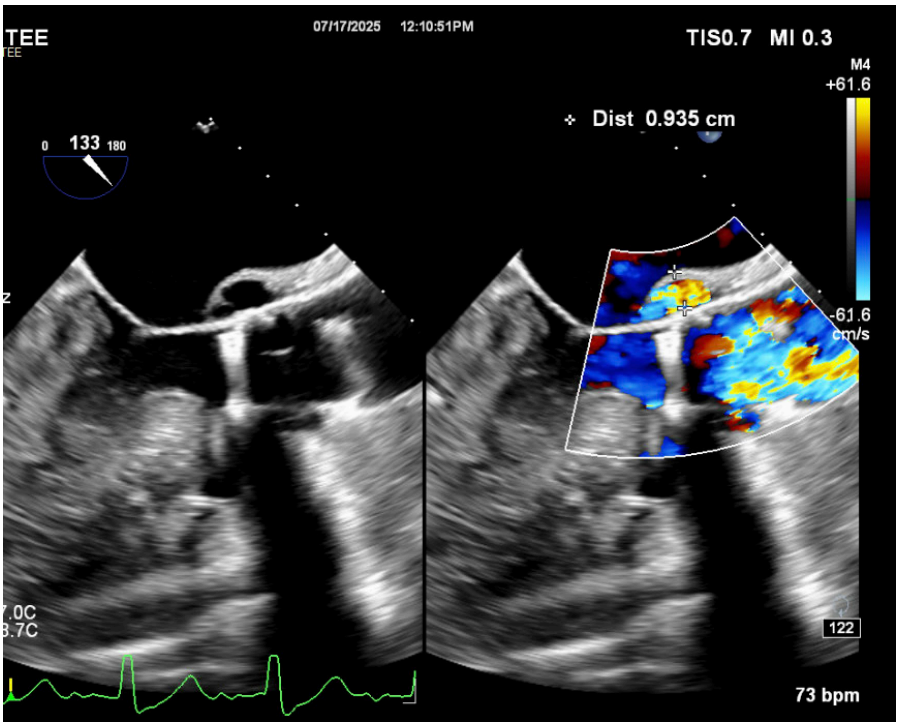
A 59-year-old male with severe bioprosthetic valve aortic stenosis presented for redo AVR. His native aortic valve had been repaired once followed by a bioprosthetic aortic valve insertion 10 years prior. Following cross-clamp removal, an AAF was identified in the aortic root to the left atrium. Due to multiple previous surgeries on the aortic annulus, there was inadequate native tissue to close the defect well. Given that the patient was hemodynamically stable, and that the AAF had not increased in size over time, a decision was made to conclude the case and move the patient to the ICU for recovery, rather than subject the patient to another cardiopulmonary bypass run. The patient was successfully extubated and the AAF was managed pharmacologically with afterload reducers and diuretics. Unfortunately, over the next few weeks the patient's condition deteriorated secondary to heart failure. A percutaneous closure of the AAF was attempted but abandoned due to the severity of the jet across the fistula. The patient eventually required repeat surgical intervention with CPB, including a repeat AVR and bovine pericardial patch to close the AAF. No residual fistula was visualized when separating from bypass and the patient recovered well.

Discussion

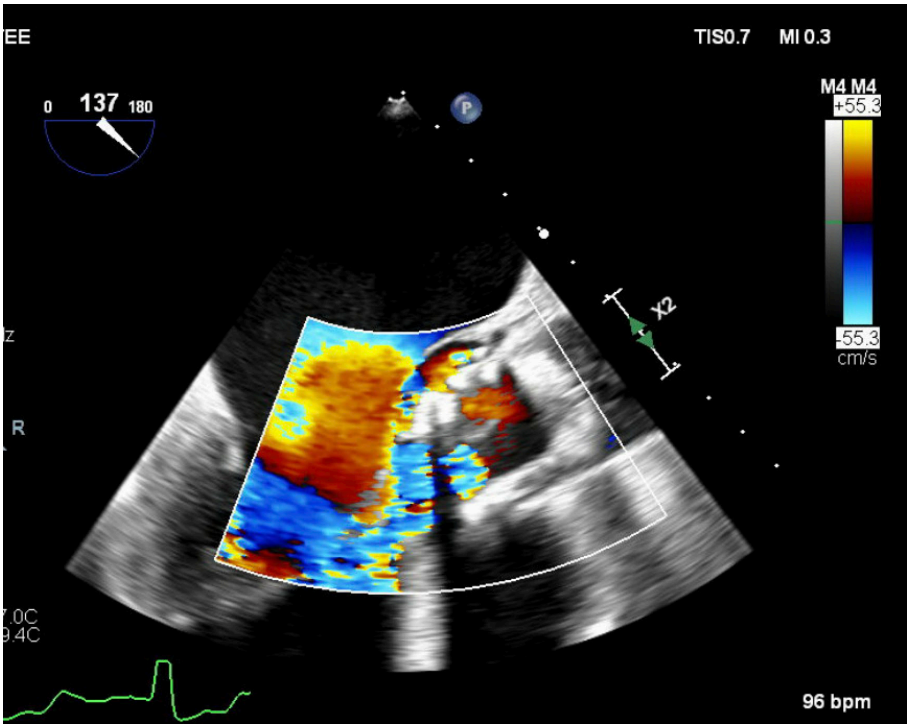
Due to the low incidence rate of AAF, there are no consensus guidelines regarding treatment strategies. However, expert opinion from previous case reports leans towards correction of the AAF either surgically or using a percutaneous closure device.[2,3] This patient had previously undergone numerous surgeries on his aortic valve, which had led to the denudation of tissue at the annulus. Surgical intervention would have not only required a very complex repair but also would have subjected the patient to additional CPB and ischemic time. Numerous measurements were taken of the AAF with TEE and they all remained stable. The remainder of the echo was also improved from baseline. Hence, the decision to try pharmacologic therapies instead of pursuing immediate surgical intervention was made. During the reoperation, the AAF was easily identified, and it had increased in size in comparison to the previous echo. The second surgery presented with more challenges anesthetically due to bleeding and vasoplegia. Following a successful bovine patch repair and re-do AVR, the fistula was no longer visible on TEE. Ultimately the patient did well. This case highlights that although AAF may appear stable initially, they tend to worsen with time. Echocardiography is thus an invaluable tool and helps with early detection. Treatment strategies should be individualized to

patients depending on their unique clinical scenario.

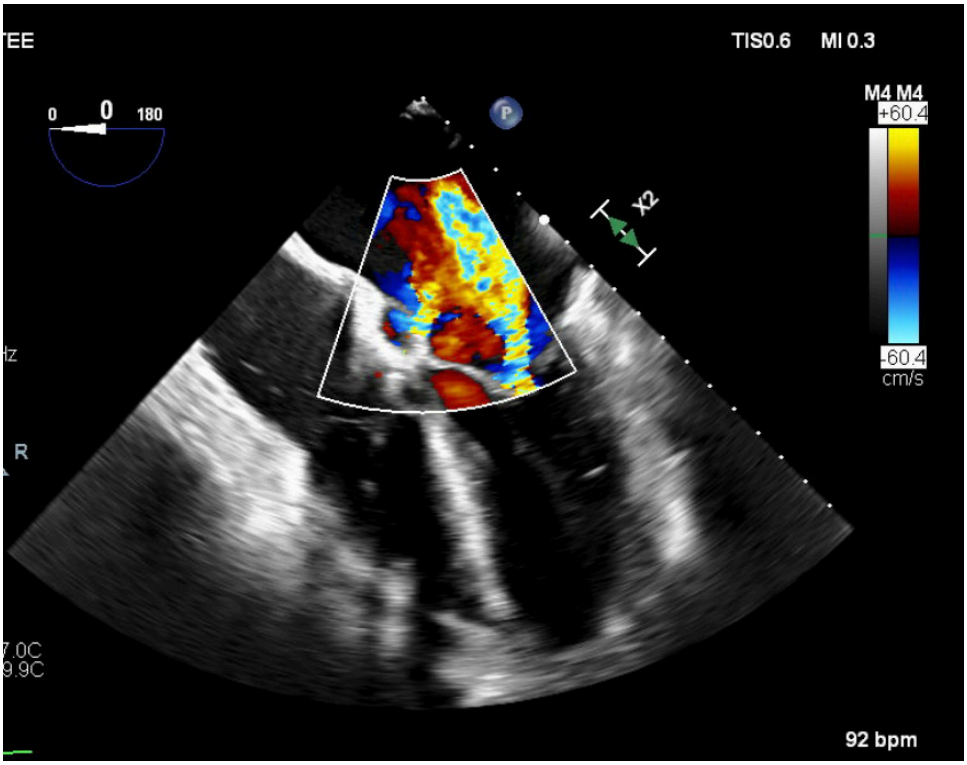
Images



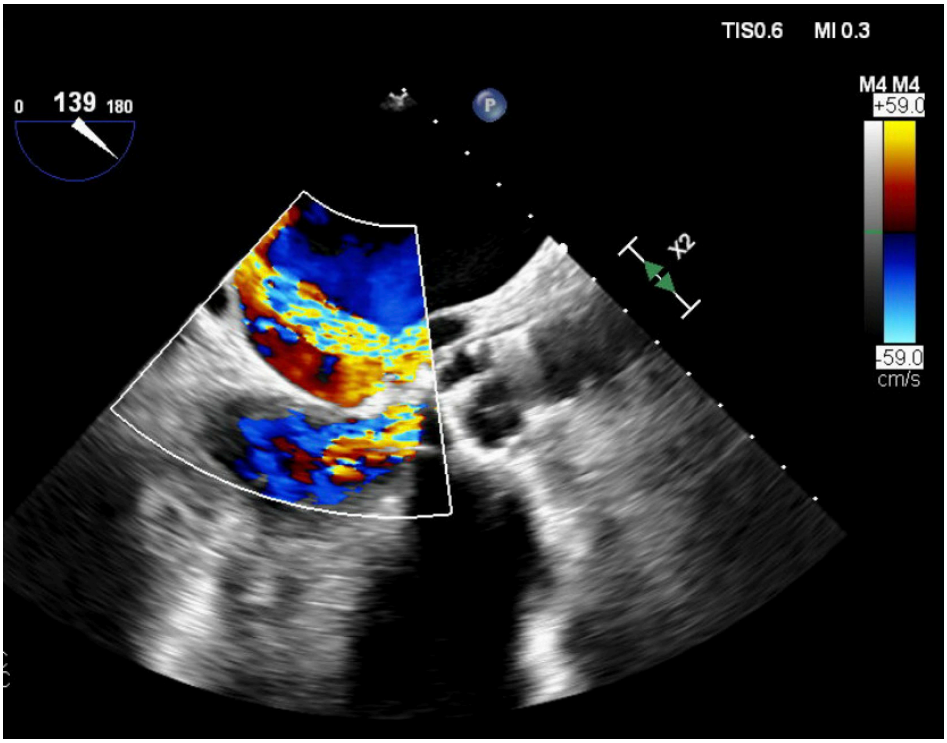
Color compare images showing Left atrial - aorto fistula formation following complex re-do AVR surgery



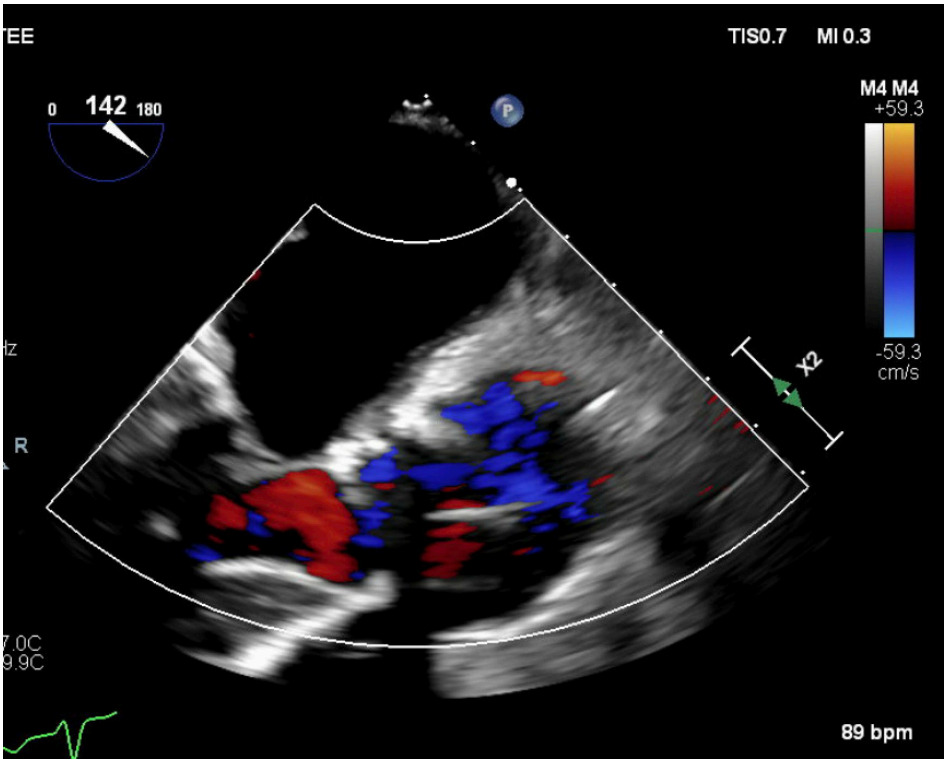
ME AV LAX view demonstrating Left atrial - aorto fistula



ME 5 chamber view showing Left atrial-aorto fistula and mitral regurgitation



ME LAX view showing worsened AAF when the patient presented for reoperation



ME LAX view showing disappearance of the AAF following patch closure

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#FR 1 – Kiosk 9 – Poster 4

Complex Fontan Takedown with Biventricular Conversion Complicated by Conduit Obstruction, RV Failure, and Distributive Shock

Presenting Author: Natalia Roszkowska, MD, Resident, Vanderbilt University Medical Center

Introduction

Fontan takedown with conversion to biventricular circulation is rare and technically difficult, performed only in select patients with borderline two-ventricle anatomy. These surgeries are high risk, largely because of distorted anatomy and multiple prior sternotomies. Long-standing single-ventricle physiology also adds its own set of challenges. We present a medically challenging case of Fontan takedown with biventricular repair complicated by a prolonged cardiopulmonary bypass run (CPB), RV-PA conduit obstruction, right coronary artery (RCA) impingement, and acute RV failure, requiring multisystem support.

Case Presentation

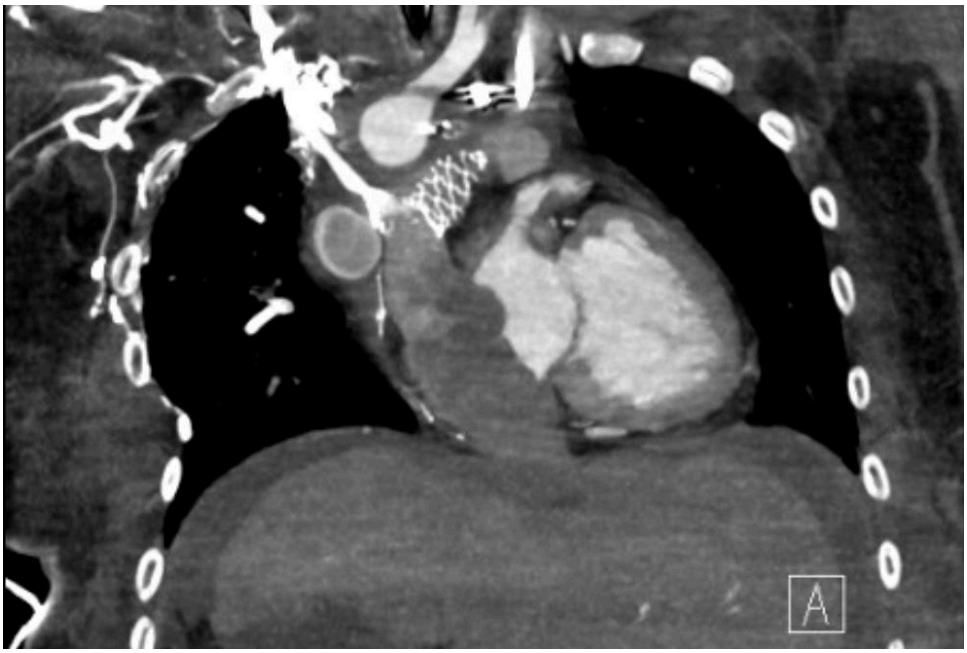
A patient with complex congenital heart disease, including right-dominant unbalanced AVSD and prior extracardiac Fontan, underwent planned Fontan takedown with biventricular repair. After multiple re-entry adhesions were dissected, CPB was initiated. The repair involved the removal of the extracardiac Fontan conduit, creation of a pulmonary venous baffle, and placement of a 10 mm RV–PA conduit. Given the extensive scarring, anatomical changes and repeated revision, CPB time exceeded five hours. During rewarming, the patient developed escalating RV pressures and low cardiac output. TEE suggested RV–PA conduit obstruction which prompted reinitiation of CPB. On exploration, the team noted kinking and narrowing of the proximal conduit, requiring complete revision and re-anastomosis. After separation from CPB, patient developed a persistent new arrhythmia, leading to additional re-exploration of sutures and identification of a small coronary branch dissection. Patient's postoperative course was further complicated by an emergent catheterization on day 7 which demonstrated dynamic RCA compression from postoperative tissue edema and patch reconstruction. Surgical re-exploration was performed, and the area of impingement was relieved with immediate hemodynamic improvement. Postoperatively, the patient's clinical course complicated by distributive shock, and oliguria. Progressive acute kidney injury and acidemia prompted initiation of continuous renal replacement therapy (CRRT).

Discussion

Biventricular conversion after a failing Fontan remains a high-risk intervention and is generally reserved for patients with anatomy that appears. High-volume centers report 5-year reoperation-free survival in the 80–90% range for elective cases, with significantly worse outcomes in those presenting with advanced Fontan failure or cirrhosis. This case underscores the technical demands of these conversions. Multiple CPB runs, RV–PA conduit revision, and relief of RCA impingement illustrate the complex intraoperative decision-making. Acute kidney injury requiring CRRT is a frequent complication in prolonged bypass and hemodynamic instability, with studies reporting up to 50% incidence in Fontan reoperations. Similarly, distributive shock and RV dysfunction requiring high-dose inotropes are common. However, biventricular

conversion can improve long-term outcomes by restoring more physiologic circulation and potentially mitigating Fontan-associated complications, such as liver disease. Our patient's postoperative course demonstrates possible risks and complications of this approach with the need for surgical expertise and aggressive post-operative rehabilitation.

Images



Complex Fontan Takedown With Biventricular Conversion Complicated by Conduit Obstruction

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#FR 1 – Kiosk 9 – Poster 5

Management of left ventricular rupture during mitral valve replacement surgery

Presenting Author: Olivia Garrett, DO, Resident Physician, University of Kentucky

Introduction

Left ventricular rupture is a rare complication following mitral valve replacement with a high mortality rate of between 50-93% (1). In this case report, we discuss our successful repair of mid left ventricular rupture following mitral valve replacement for severe rheumatic mitral stenosis.

Case Presentation

A 65-year-old female presented to the emergency room with shortness of breath and new onset atrial fibrillation requiring defibrillation. Workup included an echocardiogram which showed an ejection fraction (EF) of 60-65%, enlarged left atrium, moderate mitral regurgitation, severe mitral stenosis with mean gradient of 13 mmHg, and characteristics of rheumatic disease. She presented for elective surgery and underwent extensive biatrial maze procedure, exclusion of the left atrial appendage, and mitral valve replacement with a 33 mm St. Jude valve. Operative report noted extensive calcification of the mitral valve annulus, leaflets, and chordae, with severe shortening and thickening of mitral valve chords. Initial weaning off cardiopulmonary bypass (CPB) was successful with epinephrine at 0.05 mcg / kg / minute. During administration of protamine, bleeding was noted from the back of the pericardium. Protamine was stopped, additional heparin was administered, and CPB was initiated. A rupture was noted in the mid left ventricle away from the atrioventricular (AV) groove and away from the coronary arteries on the epicardium. The location of her coronary arteries was reviewed in relation to the location of the rupture and found to be favorable for eternal repair without necessitating a bypass. As such, the rupture was repaired externally with multiple pledgetted sutures, covering the repair with BioGlue and with felt. She was successfully weaned from CPB without inotropes. Transesophageal echocardiogram post-bypass showed a well-seated mechanical valve with no perivalvular leak and mean gradient of 2 mmHg. There were no regional wall motion abnormalities. On postoperative day (POD) 2 patient was extubated and started on warfarin. She was discharged POD 14. Her echocardiogram at 3 months post-operation showed an EF of 40% and a mean gradient of 2 mmHg across the mechanical mitral valve.

Discussion

Atrioventricular groove disruption (AVGD) is a rare but often lethal complication of mitral valve surgery occurring in less than 1% of mitral valve operations with mortality rates approaching 75%. AVGD can occur at three different locations: type I is disruption of the AV groove, type II is at the base of the papillary muscles and type III is mid-ventricular wall rupture. Type I is the most complex to repair however all types carry high mortality rates. Risk factors for AVGD include chronic rheumatic heart disease, female sex, advanced age, mitral valve (MV) annular abscess due to infective endocarditis, extensive MV apparatus calcifications, and small left ventricular size. Postoperatively tight blood pressure control is paramount with a preference for

afterload reduction and inotropes must be avoided if possible. When initiating anticoagulation, the risk of bleeding must be balanced with the risk of valve thrombosis.

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#FR 1 – Kiosk 9 – Poster 6

Breaking Out: Conduit Erosion Through the Sternum After Rastelli Repair

Presenting Author: Jean Y. Yang, MD, Cardiothoracic Anesthesia Fellow, Cedars Sinai Medical Center

Introduction

Patients with complex congenital heart disease, including those who undergo Rastelli repair, often require lifelong surveillance and multiple re-interventions due to conduit degeneration, valve dysfunction, arrhythmia, and endocarditis. Transcatheter pulmonary valve replacement has delayed surgical reoperation, but complications such as infection, stenosis, and regurgitation persist. Rarely, RV–PA conduits may cause structural erosion of adjacent tissues, including the sternum, creating substantial risk during repeat sternotomy. We report a case of complete sternal erosion from an RV–PA conduit in a patient with prior Melody valve endocarditis.

Case Presentation

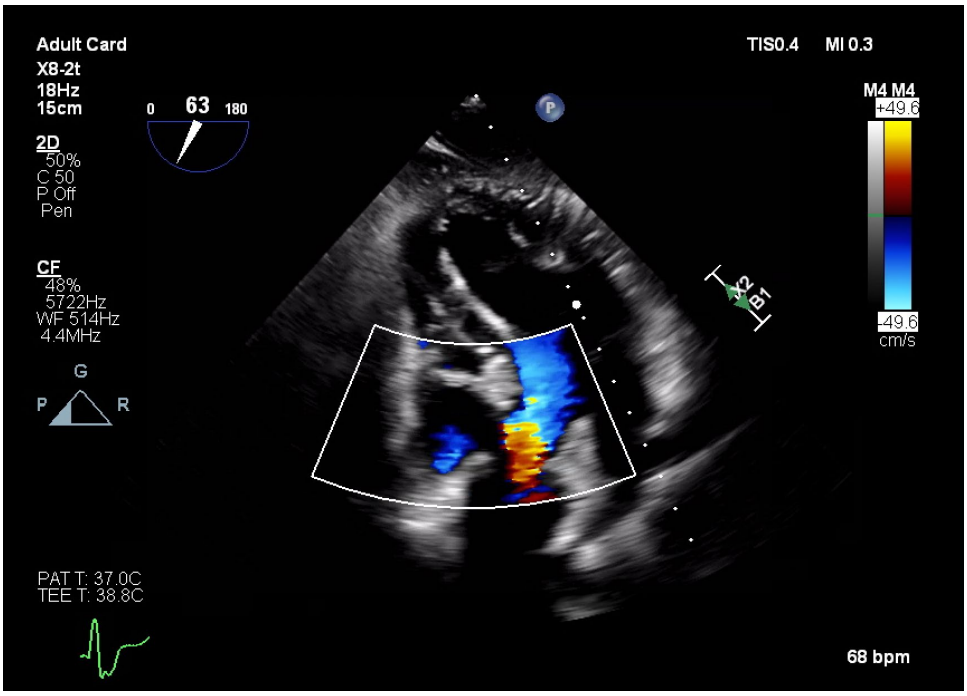
A 37-year-old woman with complex congenital heart disease s/p Rastelli repair, RV–PA conduit, and Melody valve presented after being lost to follow-up with dyspnea, weight loss, and recurrent endocarditis. During preoperative evaluation for planned conduit revision, CT imaging revealed erosion of the conduit into the posterior sternum, prompting urgent surgery. She underwent RV–PA conduit replacement on August 15, 2025. Due to anticipated high-risk resternotomy, cardiopulmonary bypass was established via peripheral right groin cannulation. On sternal entry, the conduit was found to have completely eroded through the sternum. During careful dissection, the conduit was inadvertently entered, resulting in significant bleeding controlled with pump suction. Dense adherence to the sternum and an associated bony defect were noted. A 25 mm MagnaEase bioprosthetic pulmonary valve sewn into a 28 mm Gelweave conduit was implanted. Limited pectus advancement flaps were used to cover the sternal defect. Post-bypass TEE showed normal biventricular function and a well-functioning valve. The patient was extubated on POD1 and discharged two days in stable condition.

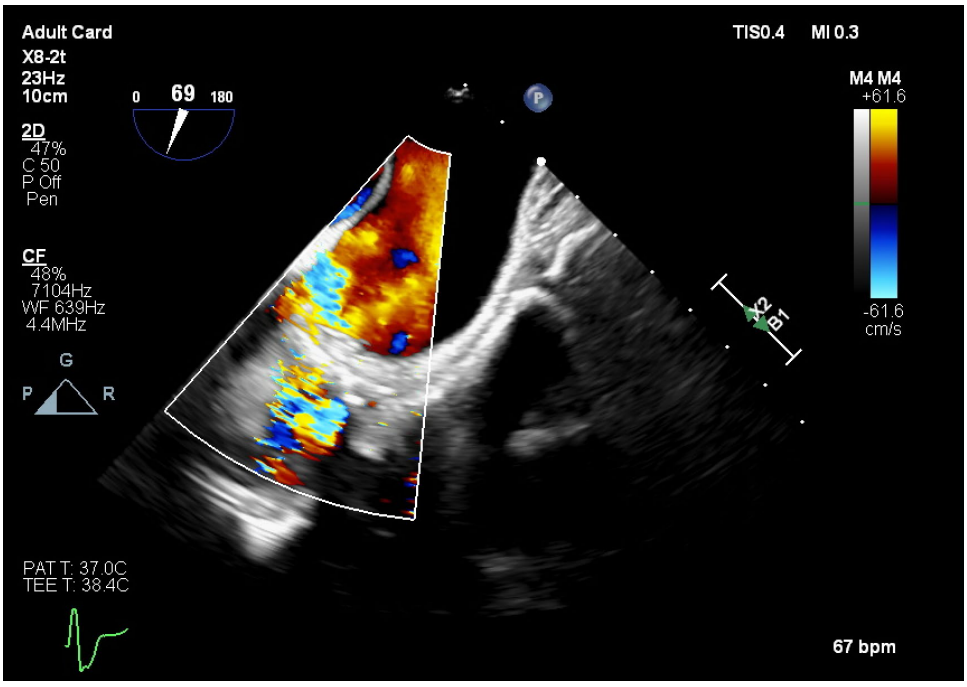
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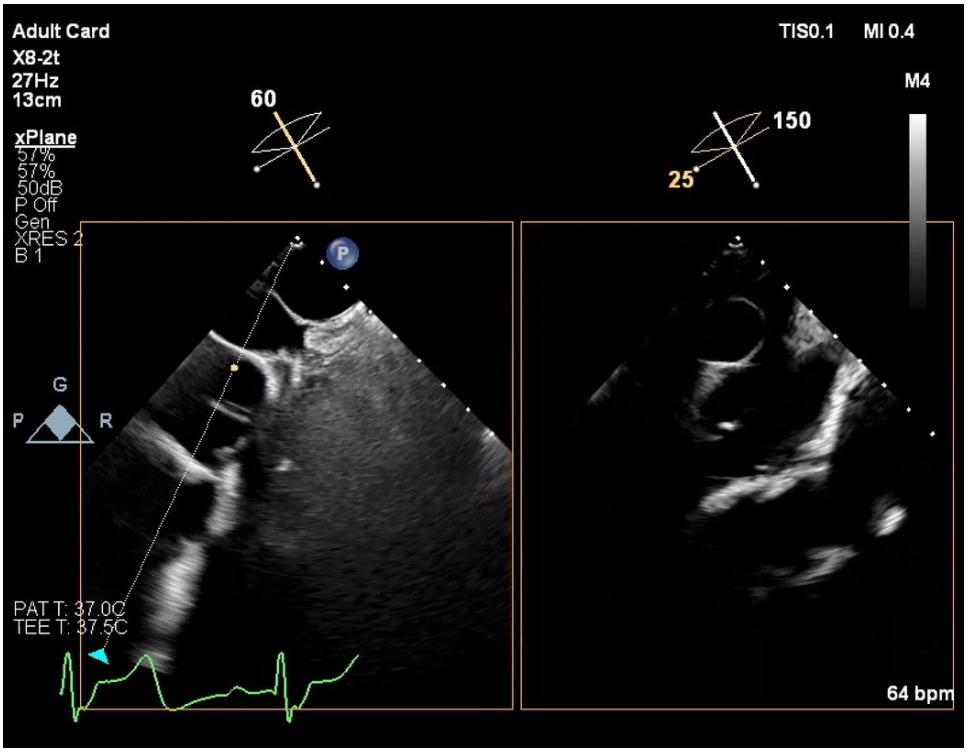
RV–PA conduits in patients with prior sternotomies may become tightly adherent to or erode into the sternum, creating substantial risk during reoperative congenital cardiac surgery. Although full-thickness sternal erosion is rare, preoperative CT imaging is essential for identifying abnormal conduit–sternal relationships and planning safe reentry. Imaging has been shown to alter surgical strategy in a substantial proportion of reoperative cases.

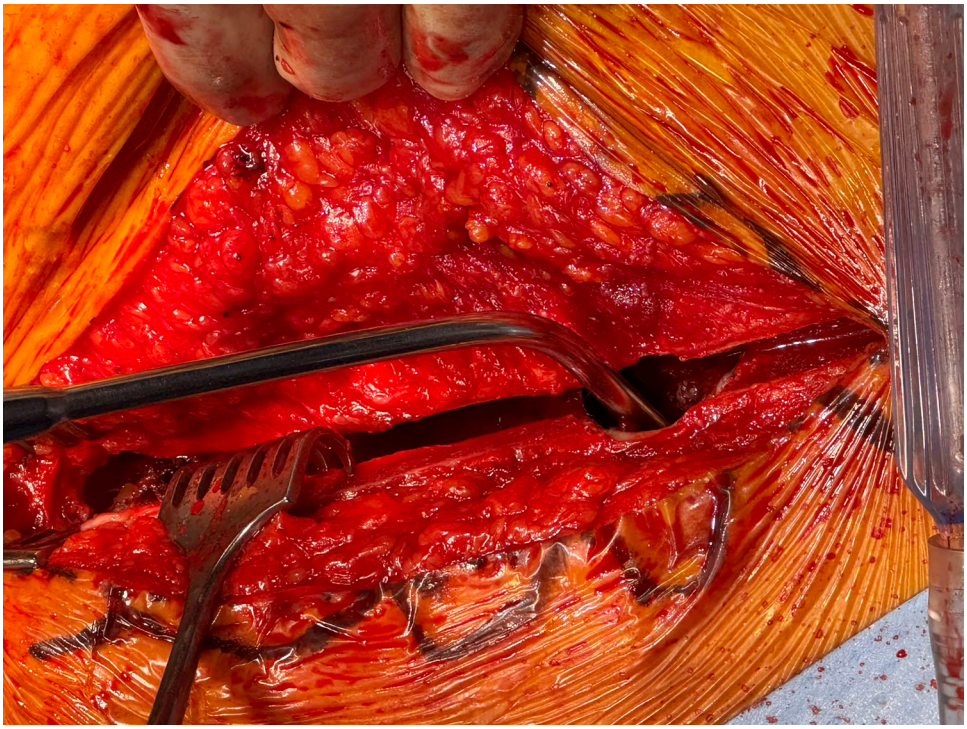
Peripheral cannulation before resternotomy is a key protective strategy, allowing rapid initiation of cardiopulmonary bypass in the event of injury. In this case, establishing bypass prior to sternal entry was critical; inadvertent entry into the conduit during dissection could have resulted in catastrophic hemorrhage without extracorporeal support. This case highlights the importance of preoperative imaging and early peripheral cannulation in high-risk reoperations involving RV–PA conduits.

Images

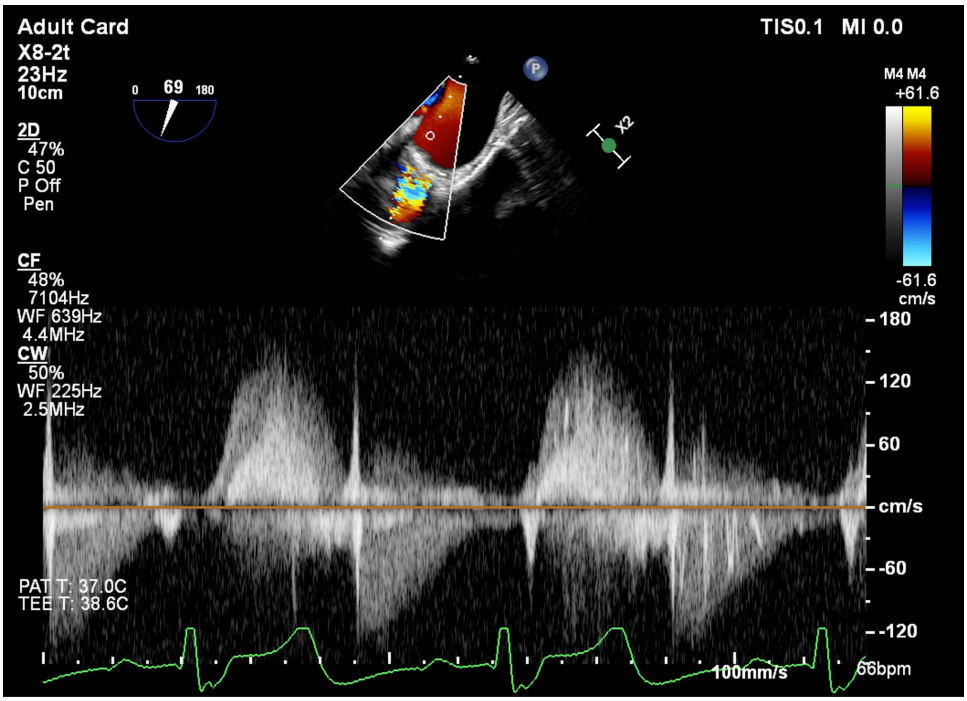

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#FR 1 – Kiosk 10 – Poster 1

Robotic Cholecystectomy in a Patient with Chronic Thromboembolic Pulmonary Hypertension (PVR 12): Anesthetic Management in High-Risk Cardiac Pathophysiology

Presenting Author: Renira Rugnath, MD, Resident Physician, The University of Mississippi Medical Center, Department of Anesthesiology

Introduction

Chronic thromboembolic pulmonary hypertension (CTEPH) presents a uniquely challenging perioperative physiology due to chronically elevated PVR and fixed pulmonary vascular remodeling. Our patient had severe PH with a PVR of 12 Wood units, chronic RV failure, marked RV dilation, and severe tricuspid regurgitation. Noncardiac surgery, particularly laparoscopic or robotic procedures requiring pneumoperitoneum and steep positioning, poses significant risk of acute RV failure. We describe the anesthetic considerations for a robotic cholecystectomy in a patient with severe CTEPH in whom physiological optimization is limited by failing pathology.

Case Presentation

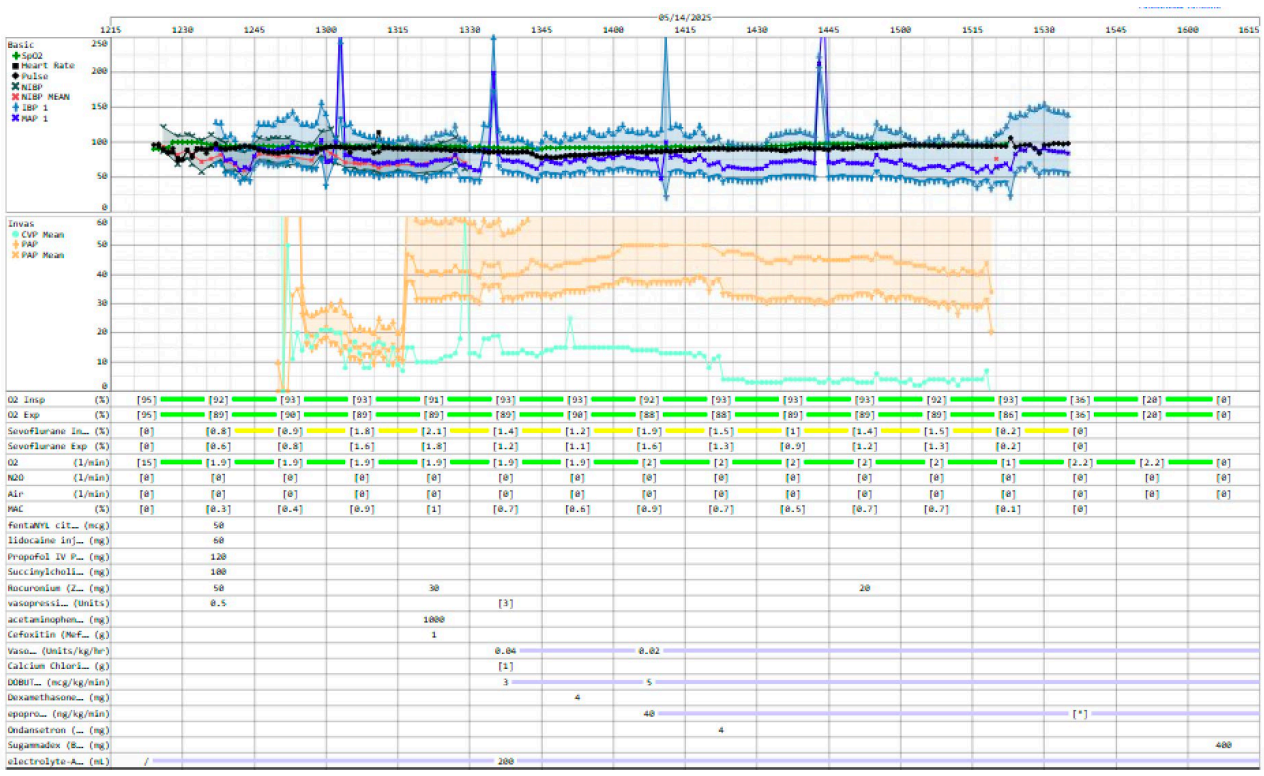
A 66-year-old man with WHO Group 4 PH from CTEPH (PVR 12, mPAP 45 mmHg, preserved LV function), chronic RV failure, severe RV dilation (FAC 25%), severe TR, CKD3b, and baseline SpO_2 92% on 3L NC presented for robotic cholecystectomy. His hemodynamics revealed severe PH, consistent with inability to acutely reduce pulmonary pressures perioperatively. Home PH therapy included treprostinil SQ pump, ambrisentan, and riociguat. Anesthetic planning emphasized maintaining RV perfusion, avoiding PVR elevation, and supporting contractility. Induction used fentanyl, lidocaine, propofol, and succinylcholine. Monitoring included arterial line, central venous catheter, pulmonary artery catheter, and TEE. Sevoflurane and FiO_2 0.8–0.95 were used to avoid hypoxia, hypercarbia, and acidosis. Pneumoperitoneum was established slowly with continuous hemodynamic assessment. Intraoperatively, the patient demonstrated rising RV pressures with insufflation and RV dilation on TEE. Vasopressin (0.02–0.04) provided systemic support with minimal PVR impact. Dobutamine (3) improved RV contractility; epinephrine was weaned early. Inhaled Veletri improved oxygenation and RV afterload. PAP remained 35–45 mmHg, CVP 10–12 mmHg, and SvO_2 ~78%. LV function remained normal; RV remained severely dilated but compensated. The patient was admitted to the CVICU on propofol, epinephrine 0.2, dobutamine 5, and inhaled Veletri 50. He was extubated successfully the same day. By POD1, all vasoactive support was discontinued; he remained on 3 L NC, was tolerating diet, and had normal hepatic labs. He was discharged with home oxygen.

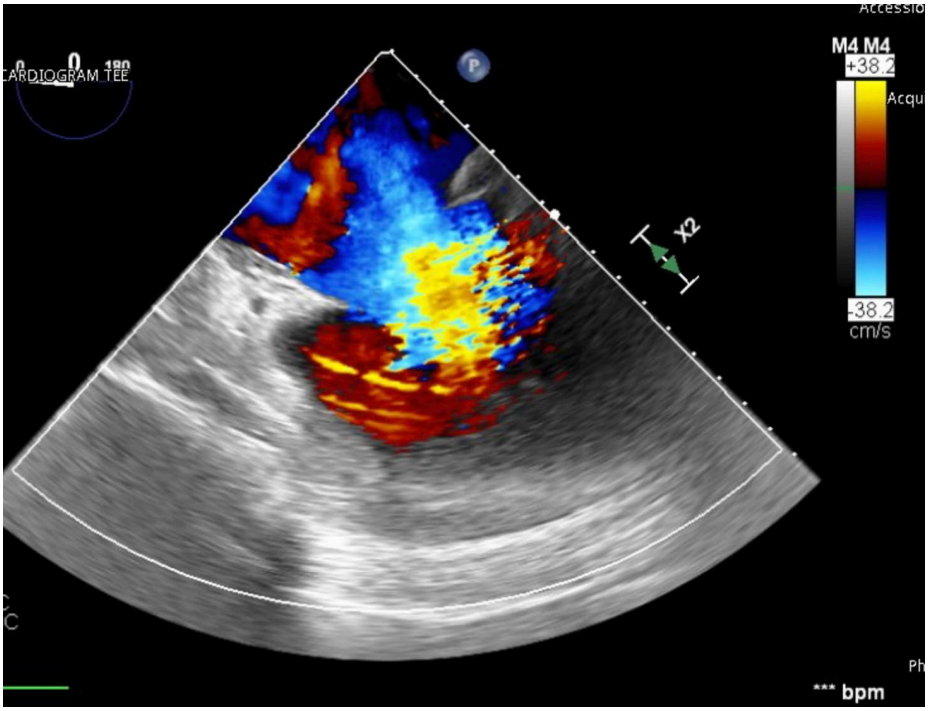
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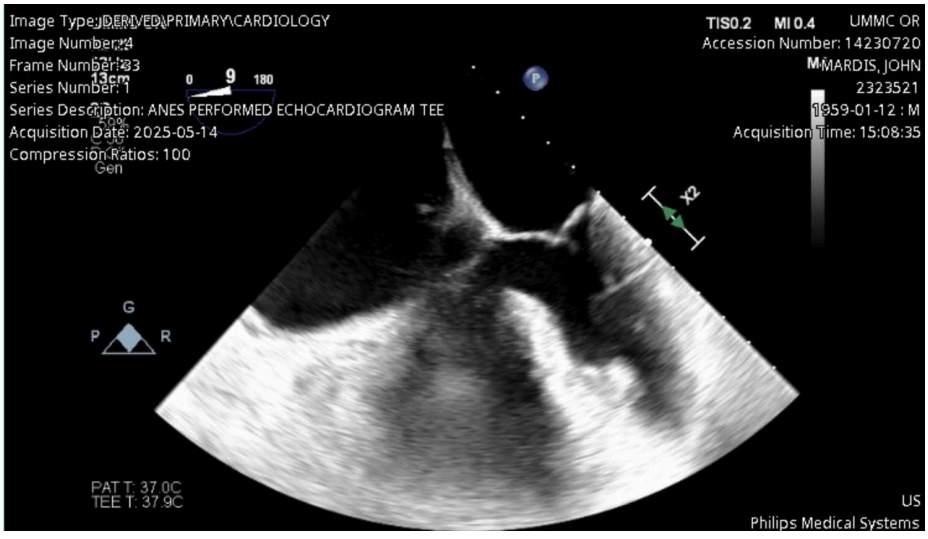
Patients with markedly elevated PVR from CTEPH are at extreme risk during pneumoperitoneum, as increased intrathoracic and abdominal pressure can further raise RV afterload and precipitate decompensation. Continuous PA catheter and TEE monitoring enabled early detection of RV strain and real-time titration of vasopressors to maintain RV-systemic coupling. Although laparoscopy and robotics impose physiologic stressors that challenge RV function, the robotic approach was intentionally chosen over open

surgery to reduce postoperative splinting, hypoventilation, atelectasis, pneumonia, and opioid requirements, all complications poorly tolerated in a PH patient with chronic RV failure. Minimizing postoperative pulmonary complications was felt to outweigh the intraoperative risks. This case demonstrates that even patients with severe PH can safely undergo robotic abdominal surgery when a multidisciplinary team applies RV-protective anesthetic strategies, PA catheter and TEE-guided hemodynamics, and early postoperative optimization.

Images







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#FR 1 – Kiosk 10 – Poster 2

Anesthetic Management for Bloodless Resection of Giant Pleomorphic Liposarcoma with Functional Tricuspid Stenosis and Profound Thrombocytopenia

Presenting Author: Joshua D. Irby, MD, Cardiothoracic Anesthesiology Fellow, University of Mississippi Medical Center

Introduction

Primary cardiac tumors are rare, reportedly occurring in 0.001% to 0.3% of patients. Despite the need for timely recognition, symptomatology is often vague and non-specific, dependent upon tumor size, location, mobility, and encroachment upon other structures.[1] Prevention of lethal complications, such as embolization, valvular obstruction, cardiovascular collapse, and mechanical shearing of blood components presents unique anesthetic and hemodynamic challenges. Herein, we present a case highlighting the anesthetic management of a large, space-occupying right atrial (RA) mass with subtotal obstruction of transtricuspid right ventricular (RV) inflow confounded by transesophageal echocardiography (TEE) assessment limitations and thrombocytopenia.[2]

Case Presentation

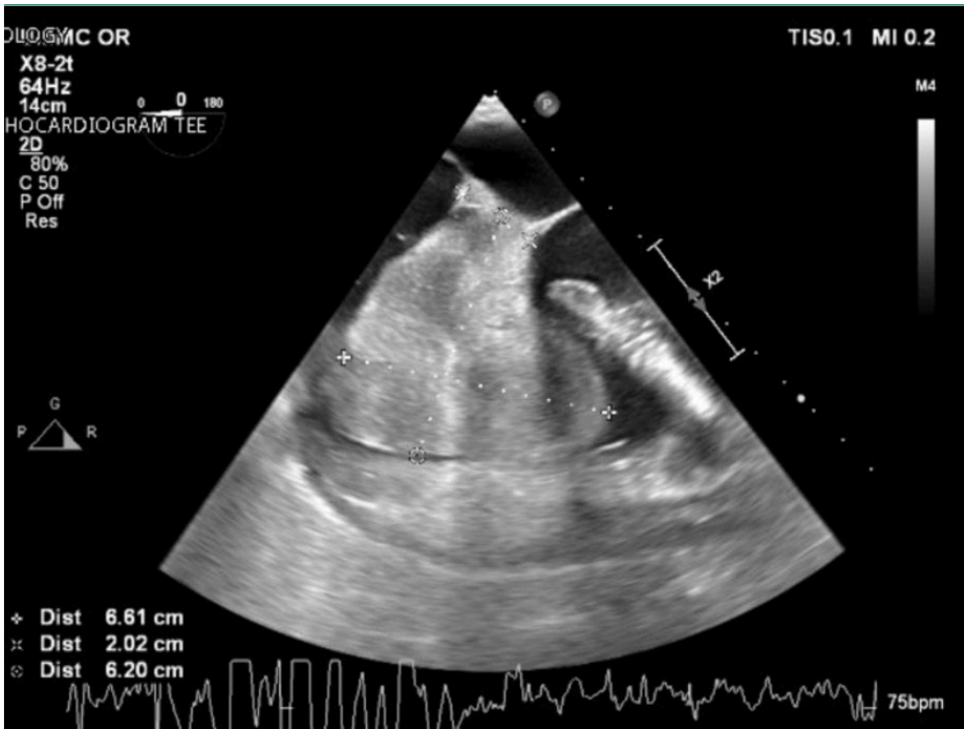
A 59-year-old male with a history of tobacco use and hypertension was admitted to our hospital with worsening exertional dyspnea and profound thrombocytopenia. Initial workup revealed a large RA mass and immature platelet (PLT) fraction, suggestive of peripheral destruction. With multidisciplinary collaboration involving hematology, patient underwent early PLT and intravenous immunoglobulins infusion, diuresis, and surgical resection. Intraoperatively, the patient had significant variation in systolic blood pressure (SBP) with bed changes, indicative of preload dependency. After fluid administration and preoxygenation, general anesthesia was induced via slow, controlled induction and institution of mechanical ventilation to avoid drastic changes in venous return. To avoid mass dislodgement, guidewire for central line was carefully inserted under TEE guidance and swan was avoided. TEE showed a large RA mass occupying almost the entire tricuspid annulus and RV cavity. Relying on the assumption that the RV would be sensitive to preload restoration post-resection, additional volume was minimized, 2mg milrinone bolus was given just prior to CPB separation, and inotropic infusion was initiated to optimize RV support and successfully separate from CPB. To mitigate bleeding risk, desmopressin was given, cell salvage minimized, and PLTs were transfused. ICU stay was plagued by vasoplegia. No blood was given. Patient was ultimately discharged home on POD5.

Mass was later histologically identified as pleomorphic sarcoma.

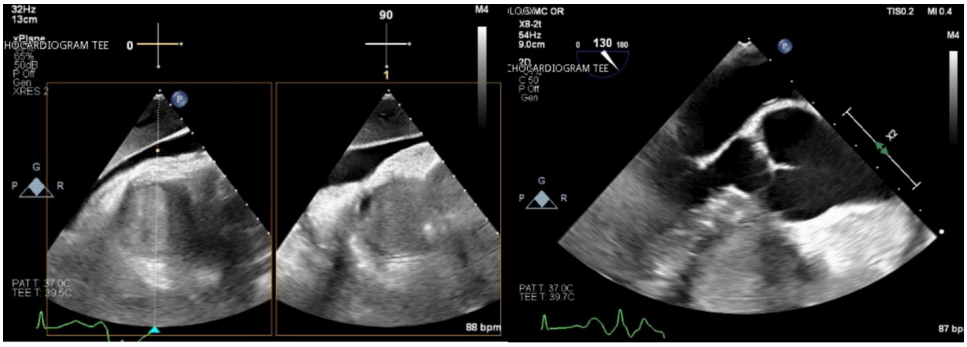
Discussion

With large RA masses, management must address preload dependence, obstructive physiology, embolization risk, and clinical hemodynamic assumptions when complete evaluation is limited. Pre-induction assessment of preload dependency is easily replicated by gravitational effects of bed changes on SBP. Fluid administration and avoidance of tachycardia and sudden increases in intrathoracic pressure are key to optimize RV inflow, cardiac output, and LV filling.[3] Swan avoidance and TEE utilization during guidewire/cannula insertion can be considered to minimize embolization risk. TEE evaluation for pre-existing tricuspid valve pathology and RV function is often limited, and without a swan, it is often necessary to rely on clinical anticipatory assumptions for successful CPB separation. This case underscores the importance of multidisciplinary preoperative optimization, TEE usefulness and limitations, and a tailored anesthetic strategy to avoid hemodynamic compromise in these patients.

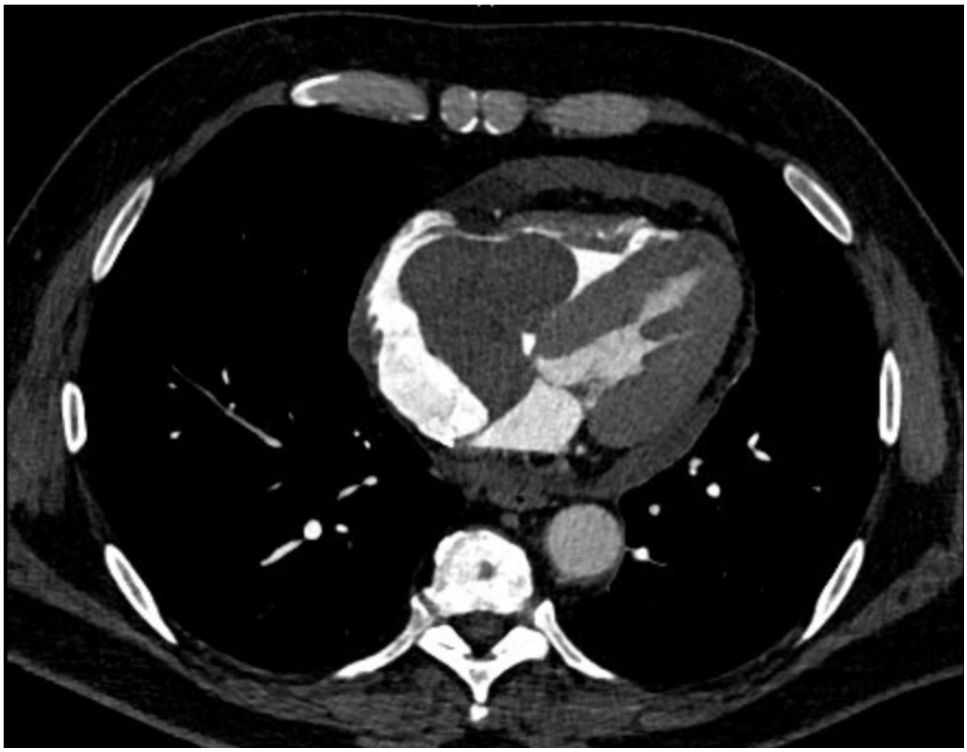
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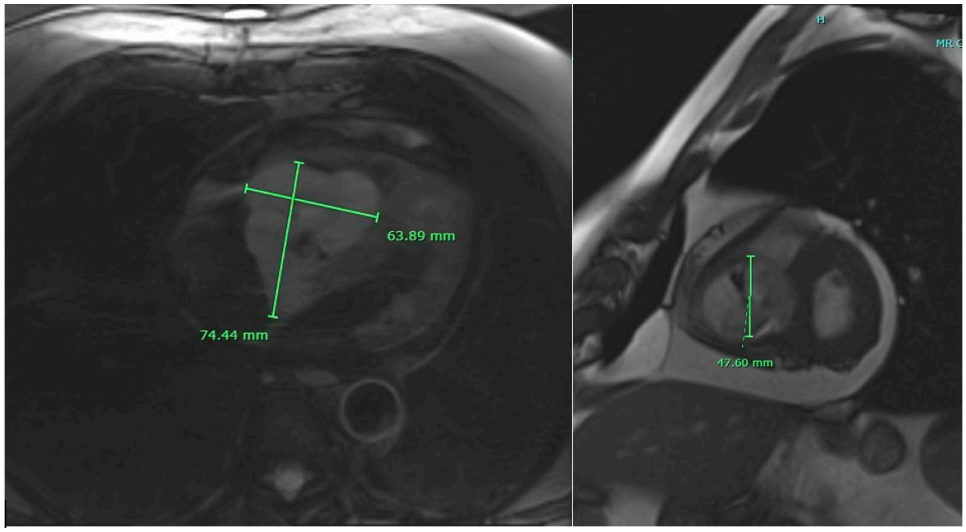
Intraoperative TEE showing right atrial mass (6.6 × 6.2 cm) extending into the right ventricle through the tricuspid valve.



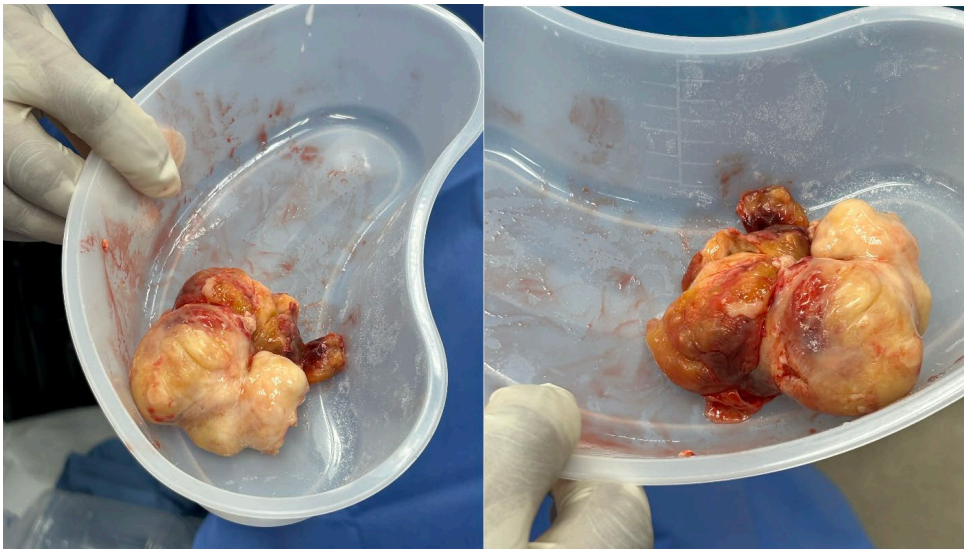
Intraoperative TEE showing mass spanning almost the entire right ventricular outflow tract.



Computed tomography scan showing an intracardiac mass in the right atrium extending into the right ventricle.



Cardiac MRI showing axial and sagittal views of intracardiac mass.



Resected cardiac mass.

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#FR 1 – Kiosk 10 – Poster 3

Cardiac Arrest After Induction in a Patient with Pulmonary Embolism and Clot-in-Transit: The Role of ECMO in Perioperative Rescue

Presenting Author: Camila Magalhaes Funatsu, MD, Anesthesiology Resident, University of Miami / Jackson Health System

Introduction

Pulmonary embolism (PE) with right heart thrombus-in-transit represents a critical perioperative challenge with mortality exceeding 40%. The combination of RV dysfunction, pHTN, and hemodynamic instability places these patients at extreme risk during induction of anesthesia. ECMO has emerged as a lifesaving adjunct when rapid hemodynamic collapse occurs. We present a case of cardiac arrest immediately following induction for planned thrombectomy, successfully managed with emergent institution of VA ECMO and surgical pulmonary embolectomy.

Case Presentation

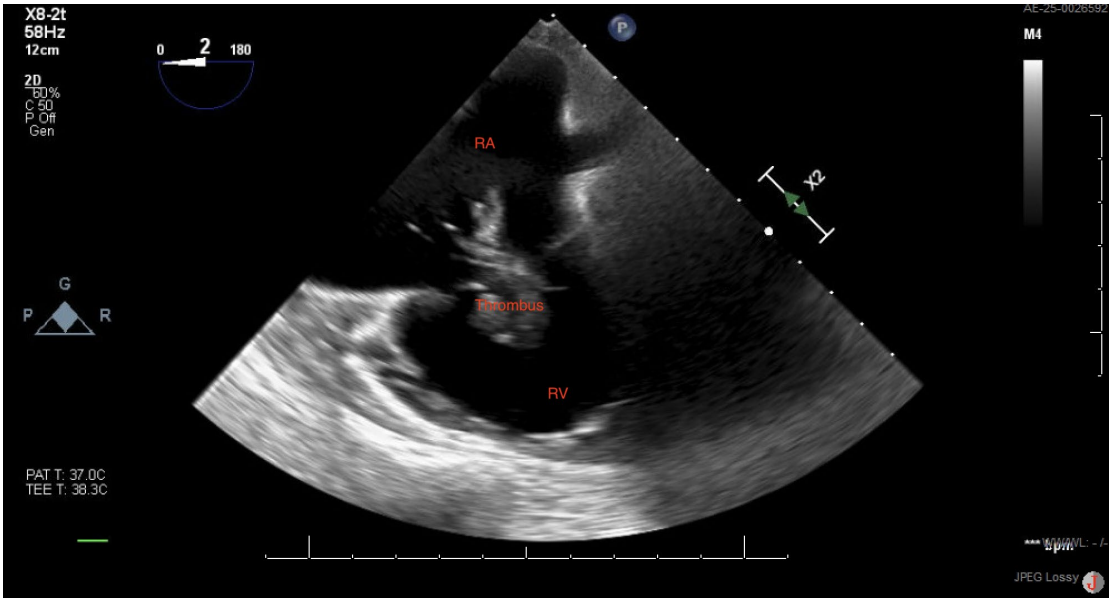
A 55 y/o obese woman with HTN presented with acute dyspnea and presyncope, found to have left popliteal/femoral vein thrombosis, a large saddle PE with severe RV dilation/dysfunction, and a large mobile RA thrombus. She was transferred from the Virgin Islands for higher-level care and scheduled for thrombectomy. Preoperatively, she was hemodynamically stable on room air without vasopressor support. Anesthesia was induced with lidocaine, fentanyl, propofol, and succinylcholine. Intubation was uneventful, but shortly thereafter the patient developed pulseless VTach/VFib. ACLS was initiated, but profound cardiovascular collapse ensued. The cardiothoracic surgery team proceeded with emergent sternotomy, internal cardiac massage was continued, and central VA cannulation was rapidly obtained. ECMO was initiated, restoring perfusion and hemodynamic stability. Full heparinization and conversion to CPB were achieved. The RA was opened, revealing extensive thrombus within the RA and RV trabeculations, which were evacuated. Inspection of the pulmonary arteries revealed large thrombi at the main bifurcation extending into segmental branches bilaterally. The emboli were carefully extracted under low-flow CPB, ensuring clearance of all branches. After complete embolectomy, the patient was rewarmed, weaned from CPB, and transitioned back to VA ECMO via central aortic and right femoral venous cannulation. The chest was partially closed due to coagulopathy. The patient was transferred to CVICU. Postoperative course was complicated by a left hemothorax requiring washout and subsequent conversion to tunneled central cannulation. Gradual RV recovery was observed on serial TEE. She was decannulated on POD 10, weaned from mechanical ventilation, and discharged to long-term rehabilitation.

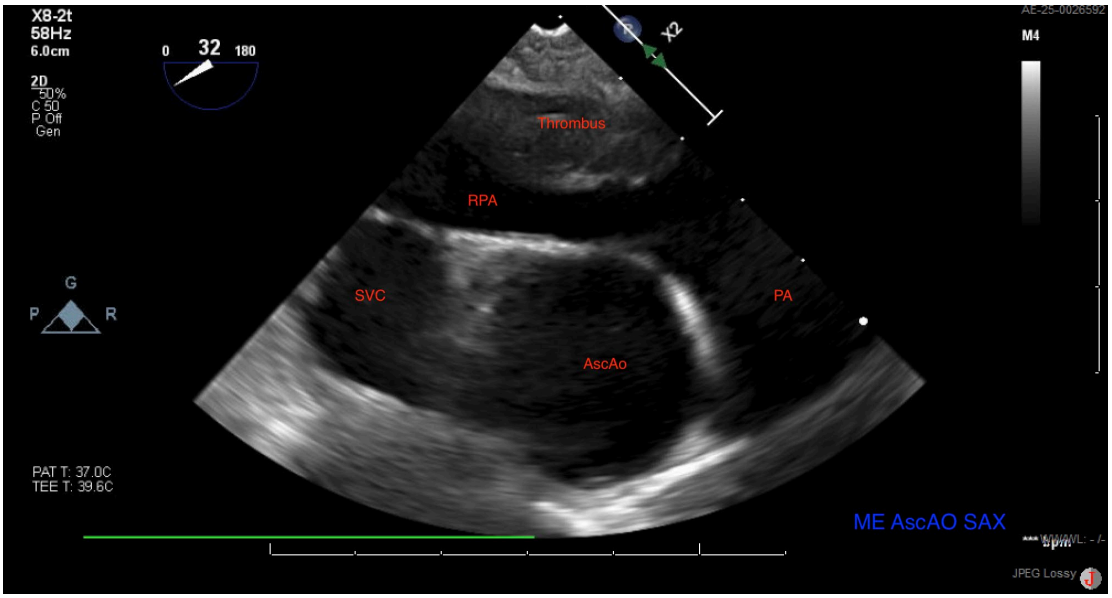
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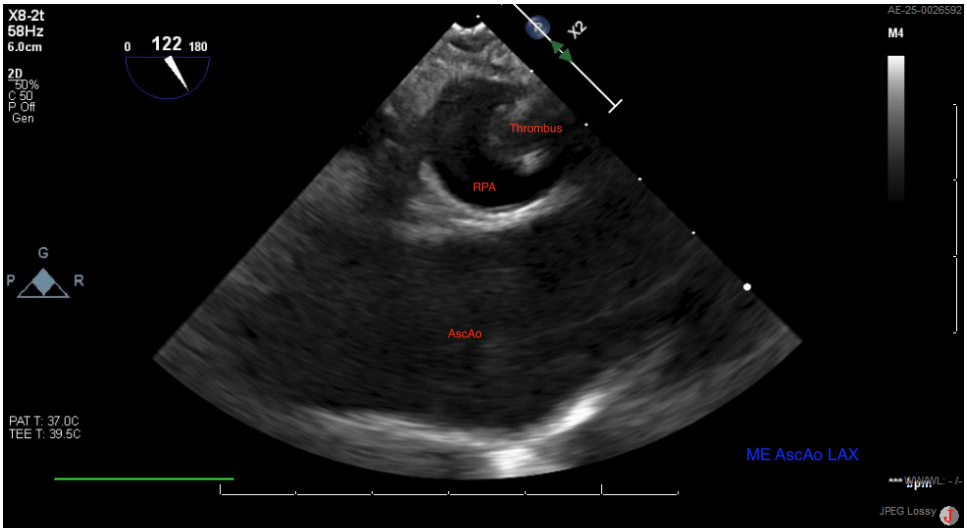
This case highlights the hemodynamic vulnerability of patients with acute PE and clot-in-transit during induction. Minimal reductions in preload, afterload, or contractility can trigger cardiovascular collapse. ECMO provides an immediate means of stabilizing perfusion and oxygenation while enabling definitive surgical intervention. Early coordination between anesthesia, cardiac surgery, and perfusion teams allowed

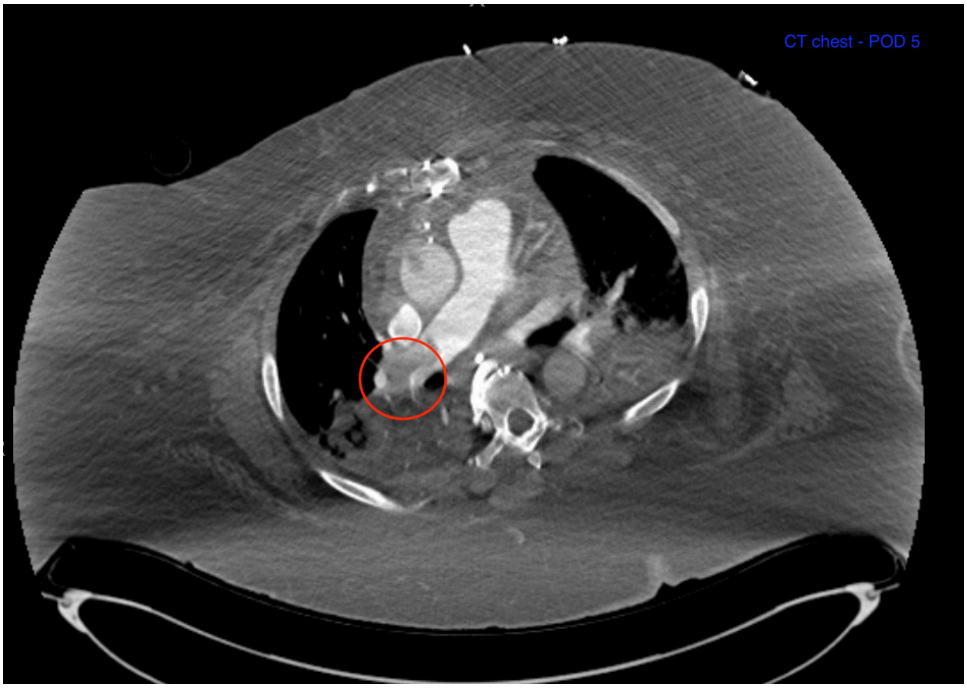
rapid initiation of ECMO and restoration of circulation. Elective pre-induction femoral cannulation for standby ECMO may have mitigated peri-induction arrest risk. Anesthetic management of patients with PE and clot-in-transit requires planning and readiness for rapid deterioration. Emergent ECMO initiation can be lifesaving when catastrophic collapse occurs. This case illustrates the importance of multidisciplinary coordination and the vital role of ECMO as both a bridge to embolectomy and recovery of RV function.

Images









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#FR 1 – Kiosk 10 – Poster 4

Severe Pulmonary Arterial Hypertension in the Multigravid Patient

Presenting Author: Nicole Zanolli, MD, Resident Physician, University of Pennsylvania

Introduction

Severe pulmonary arterial hypertension (PAH) requires preparation and complex perioperative management. When coupled with the physiologic changes of pregnancy, it poses a unique challenge for anesthesia providers. Here we describe the perioperative management of a patient with severe PAH presenting for urgent C-section.

Case Presentation

A 34-year-old multiparous woman with severe PAH, OSA, and lupus was admitted at 30 weeks for worsening RV function and increasing PA pressures. Upon admission, RHC showed RVSP 90 mmHg, mean PA 57 mmHg, PCWP 15mmHg, CI 2.4, and PVR 8.4 Wood units. TTE was notable for moderately decreased RV function. She continued her home tadalafil and was initiated on IV treprostinil after PICC placement. The interdisciplinary team, consisting of cardiology, OB, pediatrics, cardiac surgery and cardiac anesthesia, targeted expectant management with delivery at 34 weeks. However, on Day 6 of admission, the patient went into pre-term labor. She was treated temporarily with tocolytics and administered IV betamethasone for fetal lung maturity and IV magnesium for fetal neuroprotection. A central line was placed prior to the OR on Day 7. In the OR, an arterial line and a 14G PIV were placed. Continuous fetal heart rate monitoring was established. Inhaled NO was administered via HFNC. Neuraxial anesthesia was planned, and an epidural was placed using a dural puncture (DPE) technique to confirm the catheter's location, and to avoid abrupt hemodynamic changes associated with intrathecal doses. The epidural was slowly dosed with 1.8% lidocaine with epinephrine and bicarbonate until a T4 level was achieved. In the interim, peripheral ECMO placeholders were inserted. For blood pressure and uteroplacental perfusion as well as RV support during titration of the epidural and throughout the case, vasopressin, epinephrine, and norepinephrine infusions were started. Delivery proceeded uneventfully, and oxytocin was administered via an IM route for both more gradual hemodynamic changes and avoidance of a large fluid bolus. Postoperatively, she was monitored in the cardiac care unit and weaned from the inhaled nitric oxide, inotropes, and vasopressors over 2 days. TTE showed stable biventricular function. She was discharged on IV treprostinil and diuretics.

Discussion

Interdisciplinary communication was vital to the successful management of this critically ill gravid patient and her baby. Anesthetic management focused on RV support, stable hemodynamics, and avoiding additional increases in PVR, while providing optimal analgesia. Our gradually titrated DPE allowed our team to reliably confirm catheter location and also to avoid hemodynamic changes associated with spinal doses or large epidural boluses.1 Additionally, avoiding positive pressure ventilation was important due to its potentially negative impact on RV preload and PVR.1 Volume optimization was also a key consideration given the fluid shifts occurring during and after delivery. Preparation for the possibility of both massive

hemorrhage and autotransfusion that occurs following delivery was necessary. Placeholders would also allow rapid initiation of ECMO for RV failure.2 Postpartum ICU monitoring was critical as many of the hemodynamic and fluid shifts occur in the first days to week postpartum.

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#FR 1 – Kiosk 10 – Poster 5

Throwing a Clip into the Band: A Case of Salvage Mitral Transcatheter Therapy Following Failed Surgical Intervention

Presenting Author: Douglas Choe, MD, Beth Israel Deaconess Medical Center

Introduction

Mitral valve repair (MVr) is the preferred treatment modality for degenerative mitral regurgitation (MR). Compared to MV replacement, repair has been shown to have lower operative mortality, improved long-term survival, and similar risk of reoperation.1 Among MVr techniques, repair using a flexible posterior band, as opposed to a complete semi-rigid ring, has become more common due to comparable repair success with decreased surgical risk and hemodynamic benefits related to preservation of annular geometry and motion.2 Although repair failure requiring reoperation is uncommon, repeat mitral valve surgery is associated with elevated complication rates and operative mortality. Mitral valve transcatheter edge-to-edge repair (MV TEER) has been shown to be a viable, less invasive alternative to repeat surgery.3 We present a case of an attempted MVr with partial annuloplasty band complicated by severe recurrent MR and multi-pressor shock requiring an intra-aortic balloon pump (IABP) who was ultimately treated with MV TEER. This case involved complex procedural decision-making guided by intraoperative transesophageal echocardiography (TEE).

Case Presentation

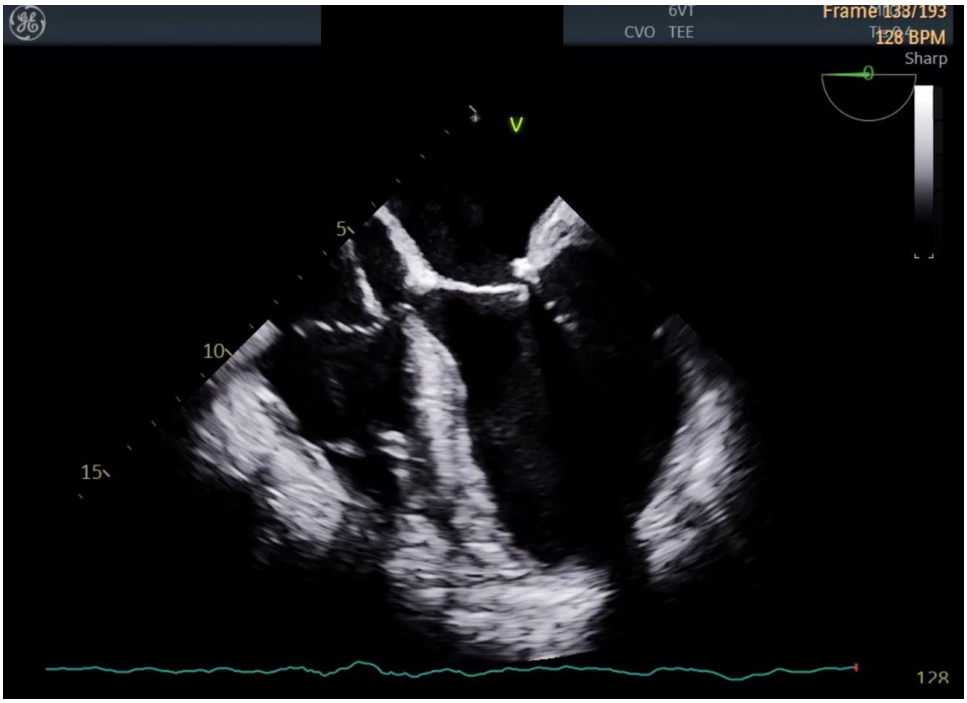
75-year-old gentleman with severe MR due to P2 flail and severe left ventricular dysfunction underwent surgical MVr at an outside hospital with a 28mm partial annuloplasty band and P2 resection. The postoperative course was complicated by repair failure with recurrence of MR and postcardiotomy shock requiring IABP support. He was weaned off support over two weeks. Unfortunately, he developed recurrent cardiogenic shock due to severe MR. IABP was again placed and he was transferred to our institution for evaluation. He was deemed prohibitive risk for redo surgery. Following multidisciplinary team discussion, he was brought to the cath lab for TEE examination and salvage transcatheter therapies if feasible. TEE demonstrated complete repair failure with severe MR. Given the presence of a partial annuloplasty band, he was not a candidate for a transcatheter valve-in-ring. We decided to proceed with MV TEER with the MitraClip system. TEE showed minimal residual posterior mitral leaflet, and attempts to grasp this tissue with the clip were unsuccessful. Ultimately, the procedural team secured the clip to the anterior mitral leaflet and partial annuloplasty band at the A2-P2 coaptation point with immediate 3-grade reduction in MR severity. The MitraClip remained stable following deployment with mild residual MR and a mean gradient of 2 mmHg through the mitral valve.

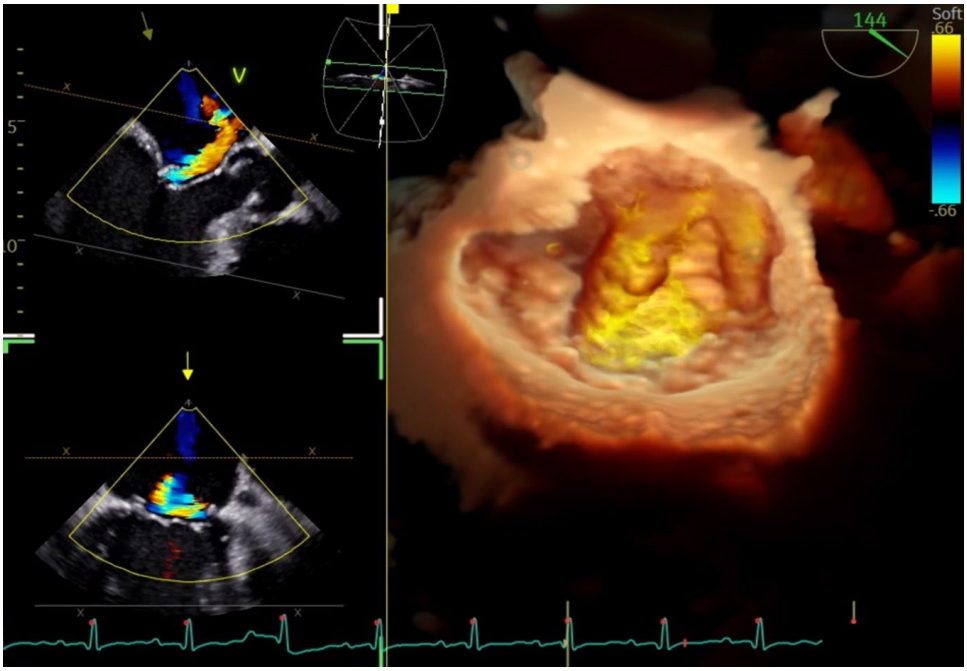
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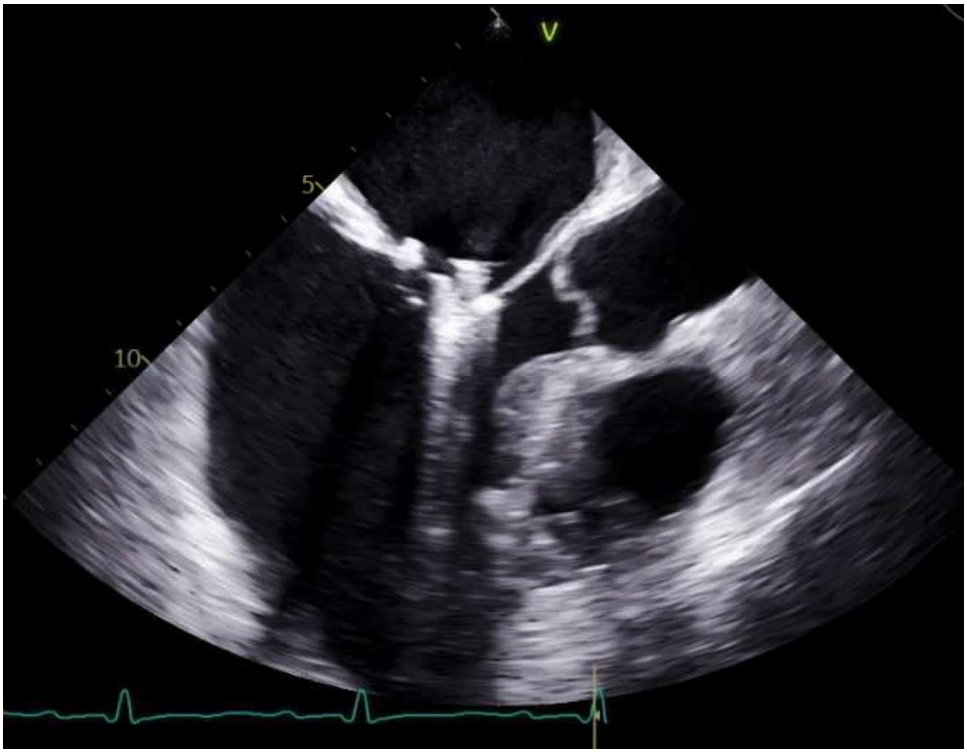
In the setting of mitral valve repair failure with a partial annuloplasty band, MV TEER is a viable salvage option. Transcatheter valve-in-ring may be considered in the setting of a complete annuloplasty ring. Here we demonstrate that a partial annuloplasty band is a feasible alternate grasping site if there is inadequate

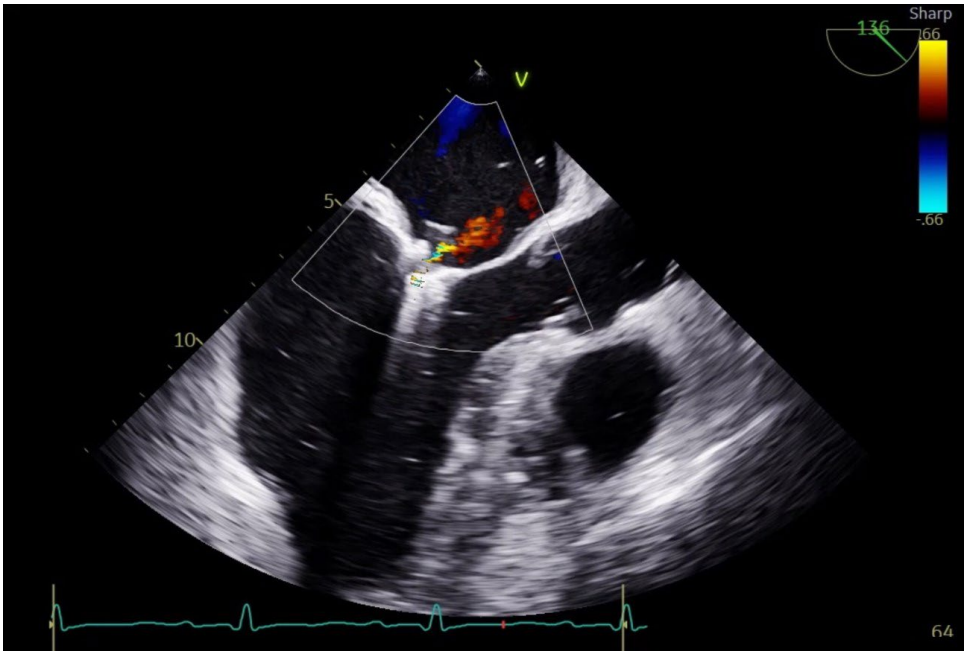
residual posterior mitral leaflet following surgical resection. This patient also had an unexpected finding of atrial septal defect due to a trans-septal approach to MVr instead of the more common left atriotomy. Given predominantly left to right flow on doppler assessment, the ASD was left open to facilitate future percutaneous procedures.

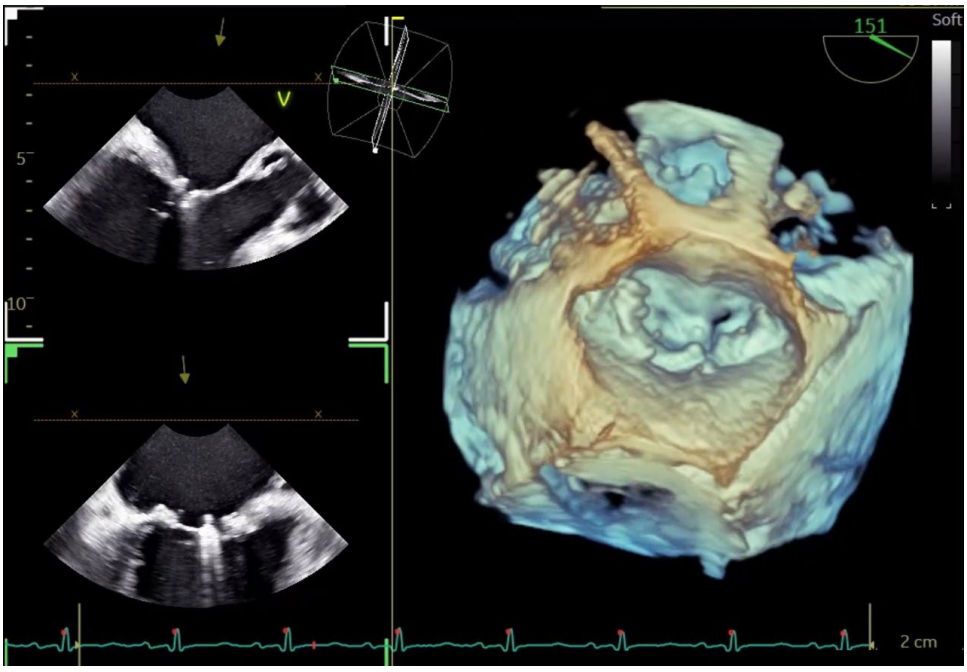
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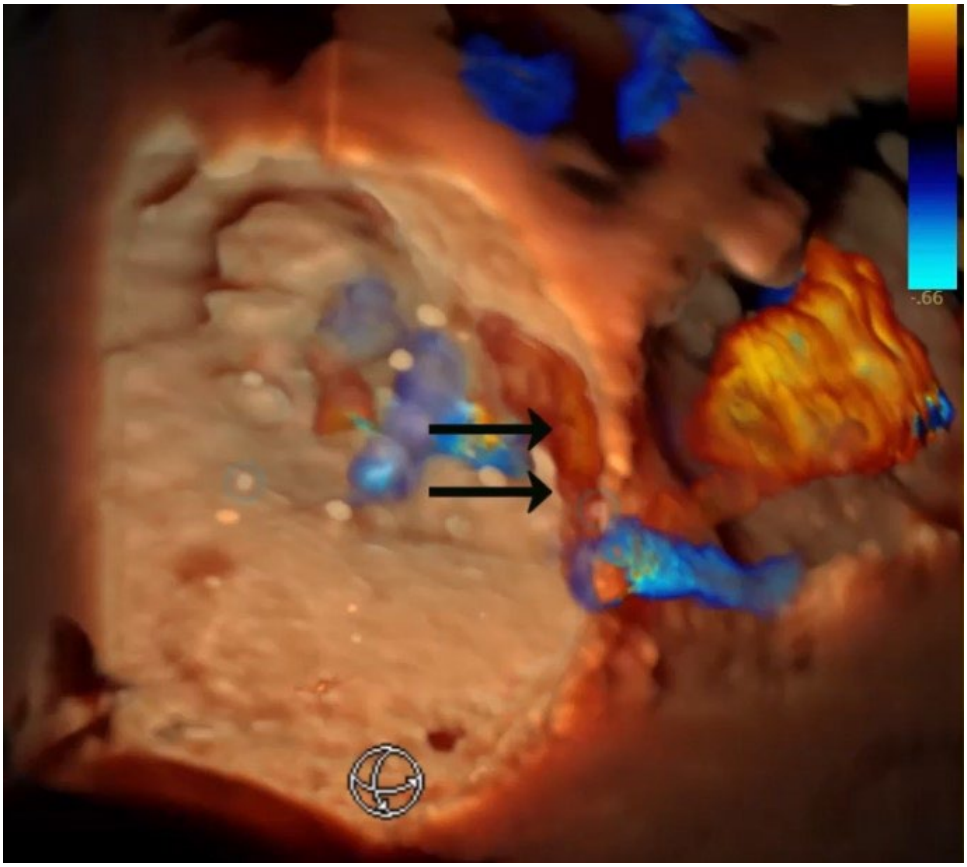



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#FR 1 – Kiosk 10 – Poster 6

Wait, What's That Vessel? An Anomalous Coronary Sinus Diagnosed on Intraoperative TEE

Presenting Author: Kate Frost, MD, Obstetric Anesthesiology Fellow, Brigham and Women's Hospital

Introduction

Congenital anomalies of the coronary sinus (CS) can occur either in isolation or in association with other congenital heart defects. There are four main types of CS anomalies – 1) variations in caliber, shape, and course; 2) enlargement; 3) absence; 4) atresia of the CS ostium (1). We present a case of a congenitally anomalous CS that was both enlarged and associated with persistent left superior vena cava (PLSVC), as well as with an atretic ostium and anomalous connection to the left atrium (LA), all of which was first diagnosed on intraoperative TEE.

Case Presentation

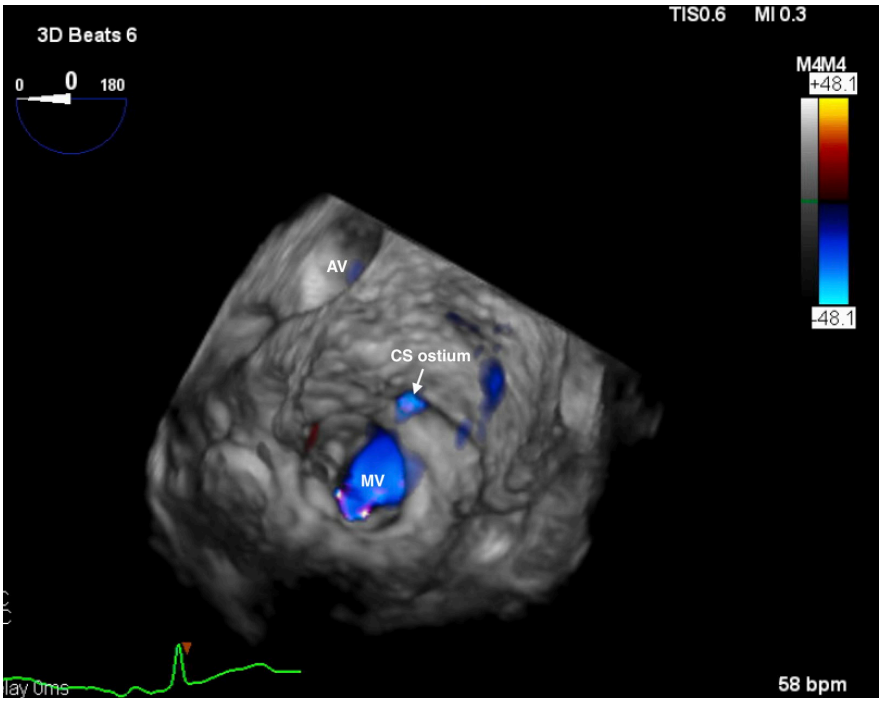
A 63-year-old woman with moderate-to-severe rheumatic mitral stenosis (MS) and moderate mitral regurgitation (MR) presented to the OR for endoscopic mitral valve replacement (MVR). A dilated CS with associated PLSVC was noted on pre-operative CT scan. A left arm PIV was placed and bubble study performed, but no bubbles were seen in the right atrium (RA) or other intracardiac locations. On further investigation, the dilated CS terminated in a smaller caliber vessel that continued along the interatrial septum and opened into the anteromedial LA, just above the medial mitral valve commissure (Figure 1). Flow through the CS appeared to be retrograde in nature, originating from the LA and flowing backwards through the CS (Figure 2). The pre-operative CT scan was further investigated and radiology was contacted, both of which confirmed the anomalous CS. At this time, the case was aborted given the concern that the flow could reverse and become right-to-left shunt with the relief of the high LA pressures following MVR. After further imaging and consultation with the congenital cardiac surgeons, she came back to the OR three days later for an endoscopic MVR with unroofing of the CS to RA and patch closure of anomalous ostium. Following surgery, the CS emptied into the RA and there was no further connection to the LA appreciated (Figure 3).

Discussion

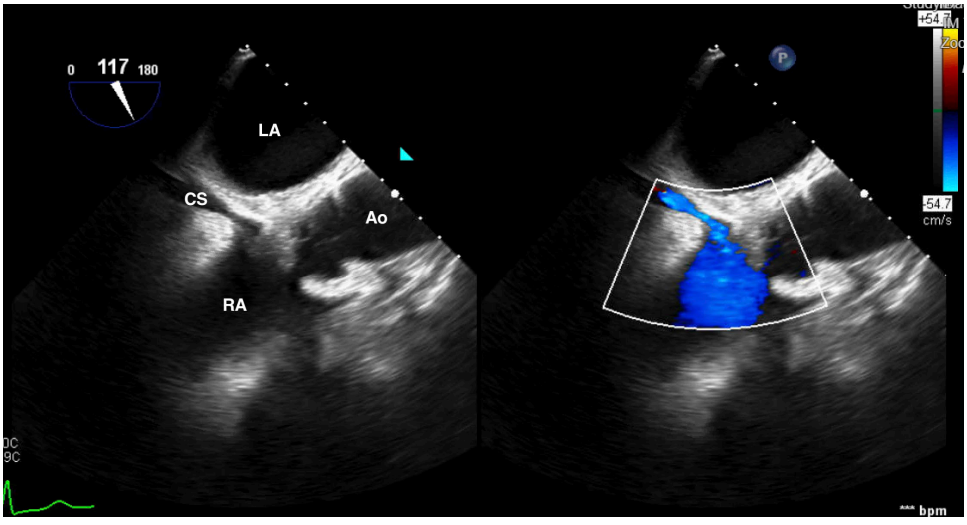
Congenital anomalies of the CS can occur in isolation or with other congenital heart defects, and are of varying hemodynamic significance. Patients with a PLSVC draining into the CS classically have a dilated CS and have a positive bubble study with injection into a left upper extremity PIV. In this case, however, a negative bubble study and an unusual small caliber vessel led to discovery of an anomalous connection to the LA. Therefore, a negative bubble study in the setting of known PLSVC must be carefully investigated. Additionally, the CS had unexpected retrograde flow with blood flow going from the LA back into the coronary sinus, which was likely due to the high LA pressures in the setting of both moderate-severe MS and moderate MR. We suspect that the patient had a baseline left-to-right shunt with blood flowing retrograde from the LA through the CS to the PSLSV and ultimately emptying back into the RA. Careful consideration was given to the potential consequences of proceeding with an MVR and relieving the high LA pressures, and

the creation of a right-to-left shunt of unknown hemodynamic and clinical significance. This case demonstrates the importance for cardiac anesthesiologists to carefully and thoroughly investigate unknown vessels discovered on intraoperative TEE, as well as consider their implications to the planned surgical procedure.

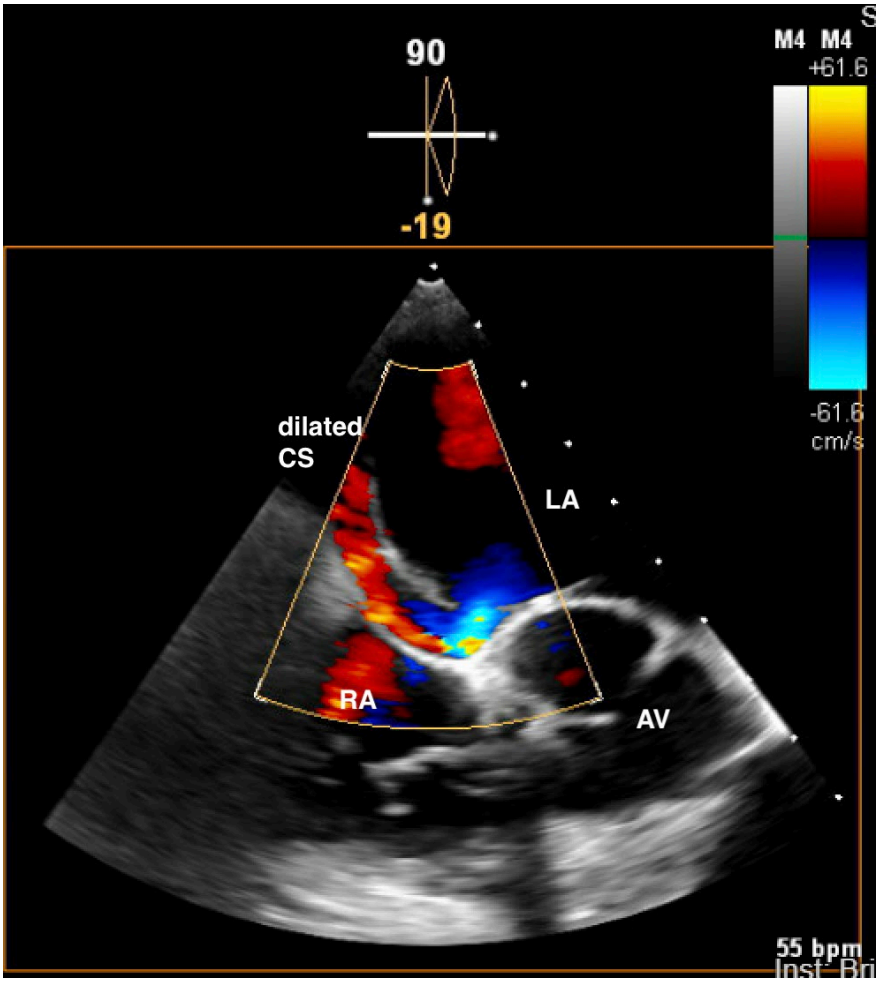
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3D TEE image looking from the left atrium (LA) down towards the mitral valve (MV), demonstrating the coronary sinus (CS) osmium opening into the anteromedial LA, just above the medial commissure of the MV.



Color compare TEE image showing coronary sinus emptying into right atrium following surgical procedure.



Color doppler TEE image showing retrograde flow from the left atrium into the coronary sinus.

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#FR 1 – Kiosk 10 – Poster 7

Considerations in a Complex Post-Myocardial Infarction Ventricular Septal Defect Repair

Presenting Author: Melissa Pelkey, MD, Resident Physician, Mayo Clinic

Introduction

A post-myocardial infarction ventricular septal defect (post-MI VSD) is rare with < 0.3% incidence, yet mortality rates exceed 40% even with intervention (1). Delayed presentation, recognition, or management plays a critical role in outcome. Patients present with acute onset heart failure and cardiogenic shock. Initial management consists of shock support and LV afterload reduction, usually with temporary mechanical support. Current guidelines favor delaying intervention when feasible to allow scar formation and improve tissue stability. We present a case where a patient presented with post-MI VSD requiring both surgical and percutaneous repair.

Case Presentation

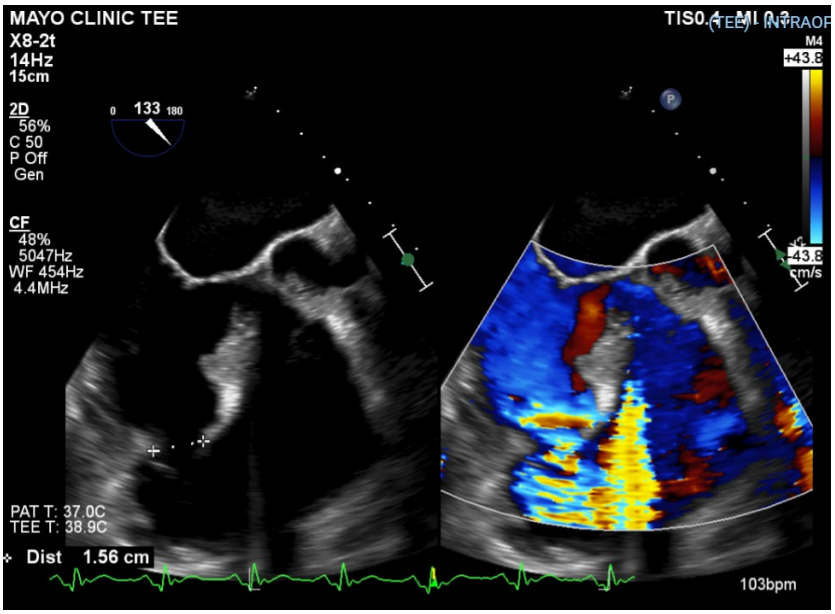
A 55 year old incarcerated male was transferred to Mayo Clinic AZ CTICU in SCAI Stage D cardiogenic shock requiring an intra-aortic balloon pump due to post-MI VSD. He initially presented to an outside hospital after 1 week of chest pain and angiography revealed 100% occlusion of the RCA without collaterals; mechanical aspiration thrombectomy with balloon angioplasty was performed. At transfer he had biventricular heart failure, left to right shunting through the VSD (Image 1), and early multi-system organ failure (shock liver and AKI). Surgical VSD repair was deferred to allow for scar formation. An Impella was placed for hemodynamic support along with dobutamine and clevidipine drips for inotropy and afterload reduction. On hospital day 6, he underwent surgical VSD closure. Induction of general anesthesia and cardiopulmonary bypass initiation were uneventful. The repair was challenging due to a large, posterior defect with extensive necrosis. After a prolonged OR course, a small residual VSD remained (Image 2). He required dobutamine, epinephrine, norepinephrine and vasopressin after weaning from bypass. Cardiogenic shock continued in the ICU, requiring dialysis for volume overload and ongoing pressor and inotropic support. On post-op day 21, TEE revealed a large residual VSD with left to right shunt (Image 3). He was a poor surgical candidate and interventional cardiology closed the defect with an Amplatzer device (Image 4). Hemodynamic goals for this procedure focused on optimizing shunt flow through afterload reduction; however induction of general anesthesia required a norepinephrine drip which was weaned at emergence. Dobutamine was continued postoperatively and slowly weaned. He was discharged on post-op day 30.

Discussion

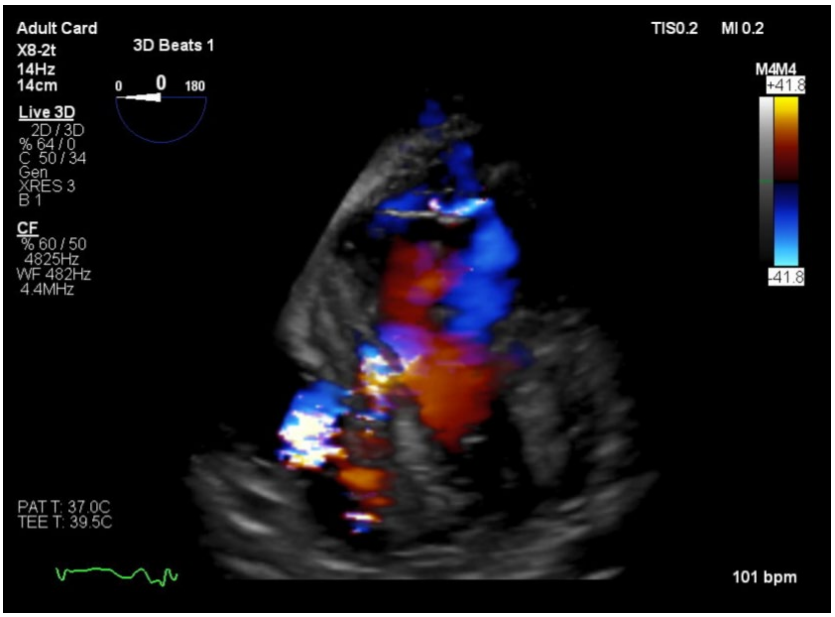
The availability of percutaneous VSD repair creates a crucial decision on the approach for VSD closure. Anesthesia providers play a key role in this decision as percutaneous repair may be preferable in patients requiring mechanical hemodynamic support or with multiorgan failure. Evidence is limited to retrospective studies with no clear superior option for repair (2). In cases like this, percutaneous closure may serve as a bridge, delaying definitive repair to allow for better tissue conditions. Another important factor in this case may have been delayed presentation due to the patient being incarcerated. Cardiovascular disease is a

leading cause of death in this population and a recent review showed high mortality due to lack of medical treatment in prison (3). Any systemic delay of care can greatly impact the development and outcome of a post-MI VSD.

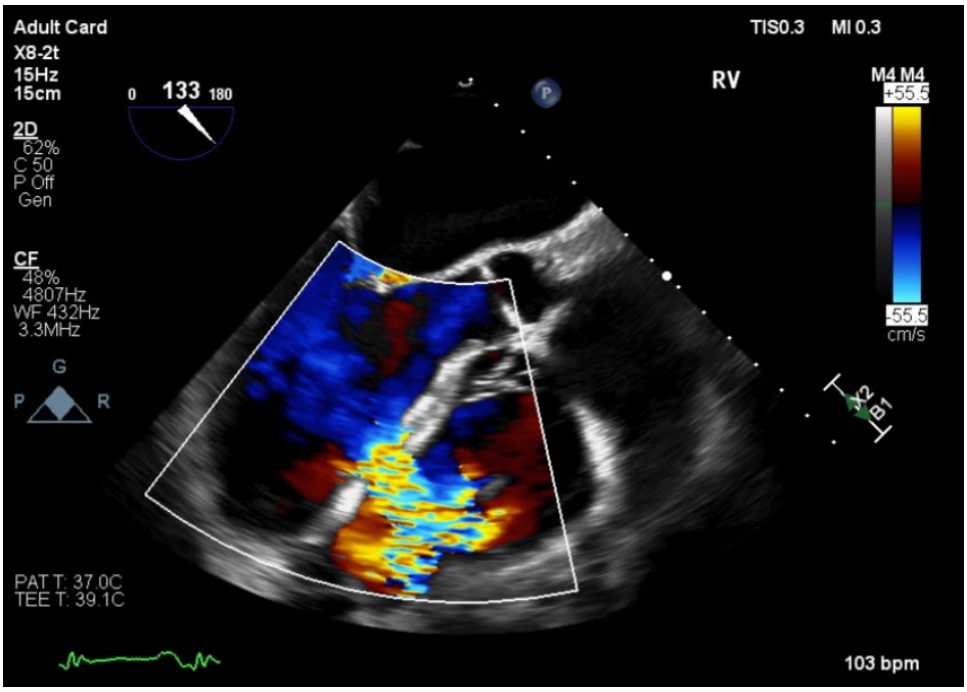
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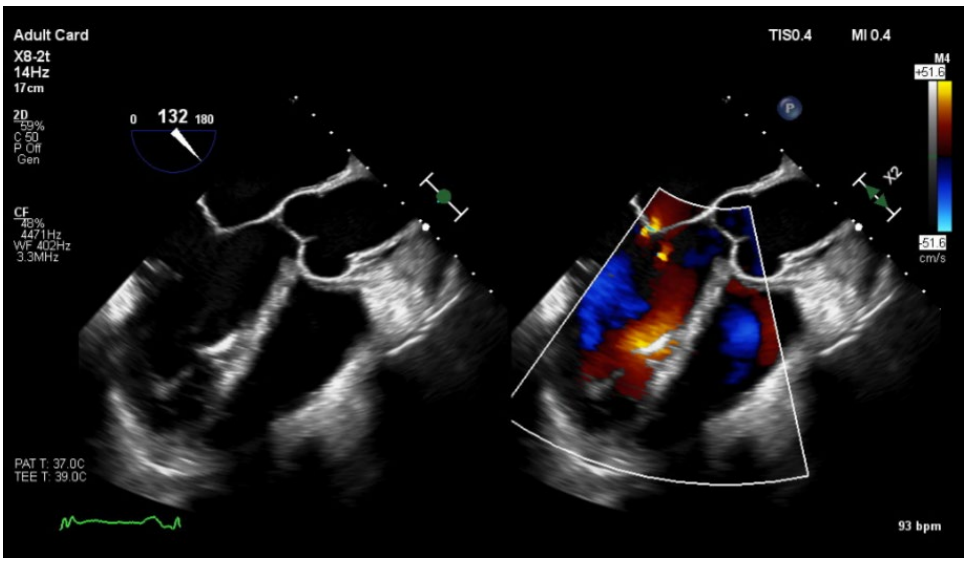
TEE image of 1.56 cm VSD with color Doppler demonstrating left to right shunt acquired at patient presentation to Mayo Clinic hospital.



3D TEE image of residual 8 x 8 mm VSD with left to right shunt on color Doppler in the immediate intraoperative setting status-post surgical closure.



TEE image of residual VSD on post-operative day 21; enlarged to 1.2 cm with left to right shunt on color Doppler.



TEE image of interventricular septum status-post VSD closure with Amplatzer device with color Doppler demonstrating lack of peri-device interventricular shunt.

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#FR 1 – Kiosk 11 – Poster 1

Veno-Arterial Extracorporeal Membrane Oxygenation assisted supracervical hysterectomy in a patient with Chronic Thromboembolic Pulmonary Hypertension – A Case Report

Presenting Author: Brian V. Entler, M.D., M.S., Fellow- Adult Cardiothoracic Anesthesia, Thomas Jefferson University Hospital

Introduction

Chronic thromboembolic pulmonary hypertension (CTEPH) is the result of venous emboli causing obstruction of the pulmonary vasculature.1 This leads to increased pulmonary vascular resistance (PVR), pulmonary hypertension (pHTN), and subsequent right heart failure (RHF).2 Induction of anesthesia, therefore, requires careful consideration to avoid increased PVR and hemodynamic instability. Veno-Arterial Extracorporeal Membrane Oxygenation (VA-ECMO) is a tool commonly employed in the treatment of CTEPH , as it reduces right heart volume, pulmonary artery pressure (PAP), and allows for recovery of ventricular function and improved cardiac output.3 Nonetheless, the association of pHTN and RHF with poor intraoperative outcomes requires real time cardiac monitoring.2 Thus, transesophageal echocardiography (TEE) has long been established in patients with CTEPH to assess biventricular function, PAP, and severity of Tricuspid Regurgitation (TR).2 Previously, case reports have demonstrated hysterectomies in patients with CTEPH only after pulmonary angioplasty or endarterectomy (PEA).2 Here, we describe the use of temporary VA-ECMO for hemodynamic support during an urgent hysterectomy.

Case Presentation

45y.o. G1P1001 with a history of adenomyosis status-post uterine artery embolization, recurrent pulmonary embolism on apixaban, and severe pHTN (systolic PAP 111). She re-presented with acute on chronic blood loss anemia (Hgb 5.4) with concerns for uterine necrosis and infection (WBC 28.1). Due to concern for cardiopulmonary collapse, despite a hemodynamically stable induction with etomidate, fentanyl, and rocuronium, VA-ECMO was initiated prior to surgical intervention. Initial TEE showed EF 28% with a dilated and elongated RV (Fig.1). The RV free wall was dyskinetic with a severely dilated RA (6cm) and there was moderate, central TR (Fig.2). Once ECMO was initiated, pHTN significantly improved (sPAP 70) and the gynecology team was then able to extract a 26-week sized necrotic uterus without significant blood loss (est.<500mL) or hemodynamic instability. The post-operative course was uneventful without morbidity or mortality. On POD 4, the patient was decannulated from VA-ECMO and she was successfully discharged home on POD 14.

Discussion

Management of patients with CTEPH is challenging, as severe pHTN and RHF can be further exacerbated by induction of anesthesia. In many cases, patients with known CTEPH and severe pHTN are medically optimized with definitive treatment being surgical intervention and PEA.2 Our patients' re-presentation, however, was urgent and she was deemed not a candidate for PEA or balloon angioplasty. VA-ECMO is not well documented as an adjunct therapy in noncardiac surgery, such as gynecology. Prior to initiation of

ECMO, we demonstrated findings consistent with the pre-op diagnoses of severe pHTN and RHF. Many of these TEE findings improved with the initiation of VA-ECMO and were sustained upon removal of the uterus (Fig. 3 & 4). Shortly after the procedure, opioid sparing analgesia allowed for early decannulation of ECMO. As demonstrated in the current case, VA-ECMO can be a useful tool to support the right heart in patients with CTEPH for urgent and/or emergent non-cardiac surgery.

Images

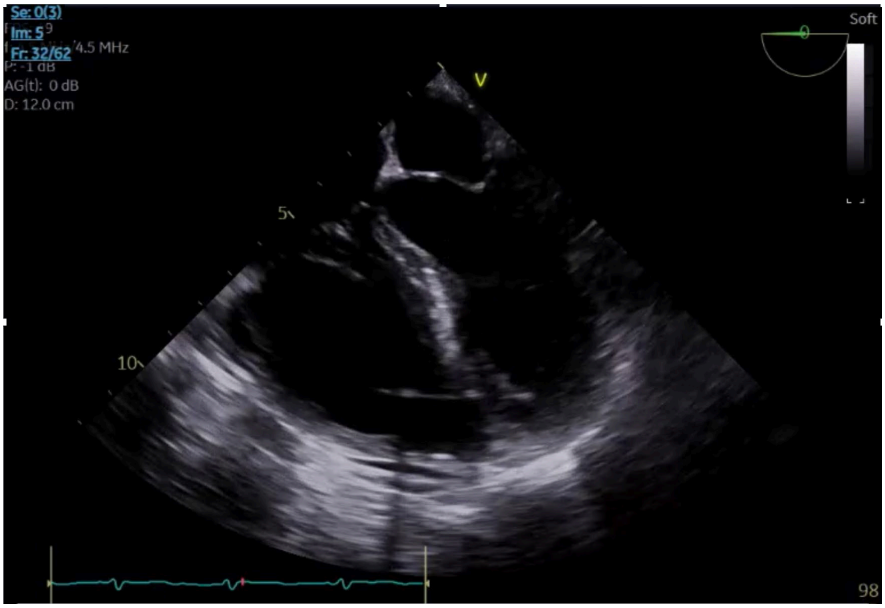


Figure 1. Dilated and elongated RV with bowing of interventricular septum prior to initiation of VA-ECMO.

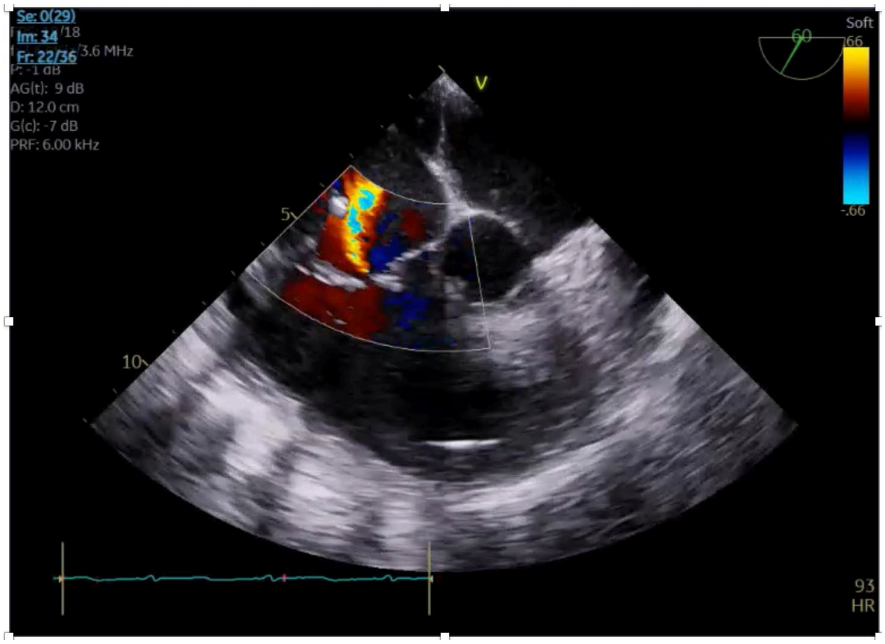


Figure 2. Moderate, central, TR (vena contracta, VC=0.52cm) with a PAC in situ.

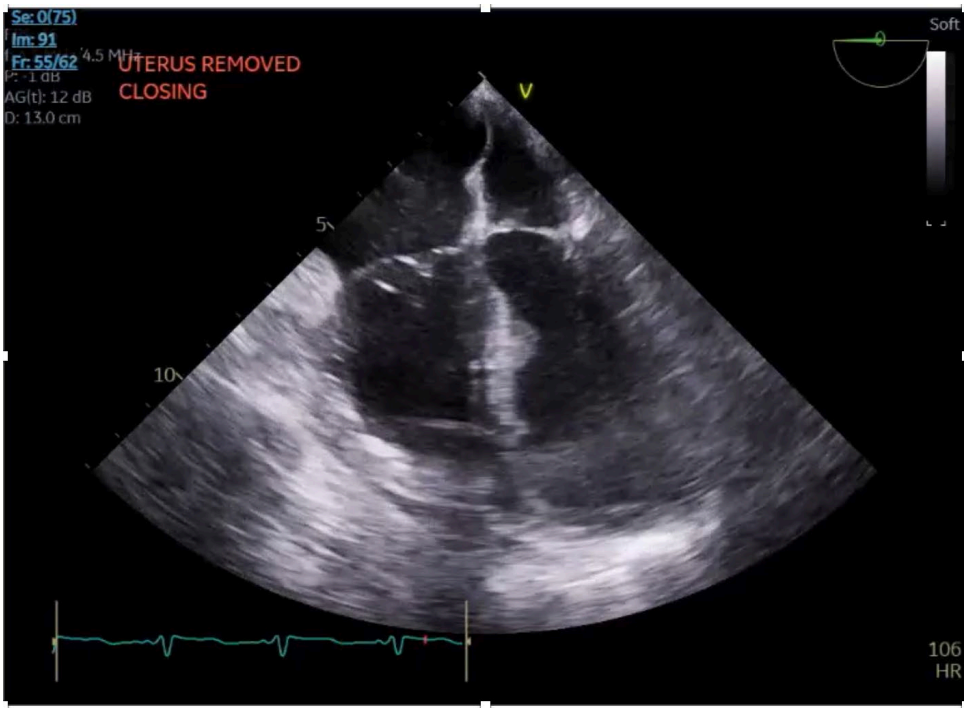


Figure 3. Improved RV function, with less bowing of interventricular septum, status post VA-ECMO initiation and hysterectomy.

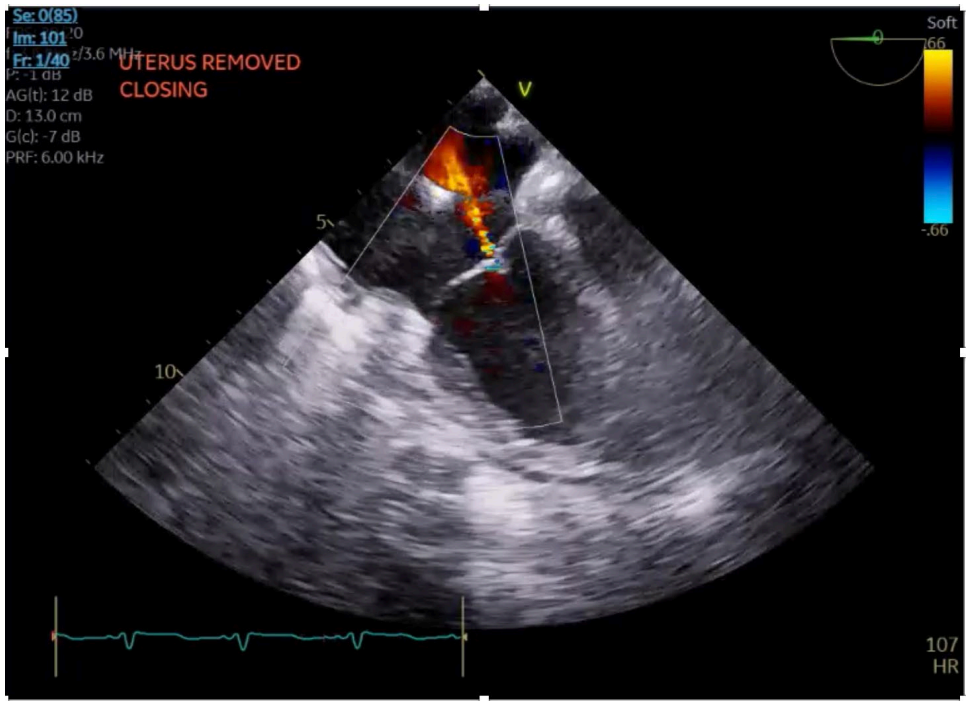


Figure 4. Improved TR status post VA-ECMO initiation and hysterectomy.

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#FR 1 – Kiosk 11 – Poster 2

Overcoming hypoxemia, vasoplegia and coagulopathy during emergent reoperation for aortic and mitral valve endocarditis

Presenting Author: Carson Brown, Resident, MaineHealth Maine Medical Center - Portland

Introduction

Patients presenting to the cardiac operating room in a mixed shock state with infective endocarditis are at high risk for perioperative hemodynamic instability, ventilatory challenges and complications.1-3 Our patient underwent a successful emergent redo sternotomy, mitral valve replacement, aortic valve replacement and Wheat procedure complicated by profound vasoplegia, severe post bypass hypoxemia and coagulopathy. We highlight our stepwise approach to treating refractory vasoplegia, the utilization of inhaled nitric oxide to treat hypoxemia and proactive management of coagulopathy to achieve an acceptable outcome for our patient.

Case Presentation

A 37 year old male with history of coarctation of the aorta treated with balloon angioplasty as an infant and aortic root dilatation treated with a Wheat procedure and aortic valve replacement 3 years prior was admitted to the intensive care unit with acute hypoxemic respiratory failure, aortic and mitral valve endocarditis and mixed cardiogenic and septic shock. Recent history was notable for intravenous drug use and blood cultures were positive for Streptococcus sanguinous. The patient was brought emergently to the operating room with an arterial line in place on supplemental oxygen via nonrebreather mask. TEE demonstrated vegetations on the aortic and mitral valves with associated severe aortic insufficiency and mitral regurgitation and a pseudoaneurysm originating at the left/non commissure. After initiation of cardiopulmonary bypass we were unable to achieve a mean arterial pressure of 65 mmHg despite escalating doses of phenylephrine, norepinephrine and vasopressin. Stress dose steroids, methylene blue, Angiotensin II and hydroxycobalamin were utilized to achieve adequate systemic vascular resistance. In anticipation of post bypass coagulopathy, 4 units of fresh frozen plasma were administered during rewarming and platelets and cryoprecipitate were prepared for transfusion following protamine administration. After successful completion of the planned surgical procedure the patient was weaned from cardiopulmonary bypass. TEE revealed adequate biventricular function and well functioning bioprosthetic valves in the aortic and mitral positions, however, despite typical ventilation maneuvers, adequate oxygenation could not be achieved. Bronchoscopy was performed and 250 ml of serosanguinous fluid was evacuated from the right and left bronchial trees. Inhaled nitric oxide was applied and oxygen saturations improved to the mid 90s, allowing the patient to transition to the intensive care unit without mechanical circulatory support.

Discussion

Management of patients with multifactorial shock in the setting of infective endocarditis can pose significant challenges in the operating room, particularly in patients undergoing technically complex surgeries. This case highlights a stepwise approach to managing refractory vasoplegia, the role of inhaled nitric oxide to

treat refractory hypoxemia and early goal directed blood product transfusion to achieve a successful patient outcome.

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#FR 1 – Kiosk 11 – Poster 3

When CABG Turns into Arch Surgery: Managing a Cannulation Injury, Multiple Arrests, and a Remarkably Preserved Neurologic Exam

Presenting Author: Jacob D. King, DO, Resident, Mayo Clinic Florida

Introduction

Cannulation-site aortic injuries during cardiac surgery are rare but may rapidly escalate a planned CABG into complex aortic reconstruction. Patients with ascending aortic ectasia are at higher risk for fragile tissue and bleeding at cannulation sites. When hemostasis cannot be achieved, deep hypothermic circulatory arrest (DHCA) and arch replacement may be required. Neurologic injury, often multifocal and embolic, is common after aortic manipulation, with up to 70% of patients developing new diffusion-positive MRI lesions postoperatively.[1] This case describes urgent CABG converting to ascending and Zone 2 arch replacement after cannulation-related bleeding, followed by delayed multifocal embolic stroke with meaningful recovery.

Case Presentation

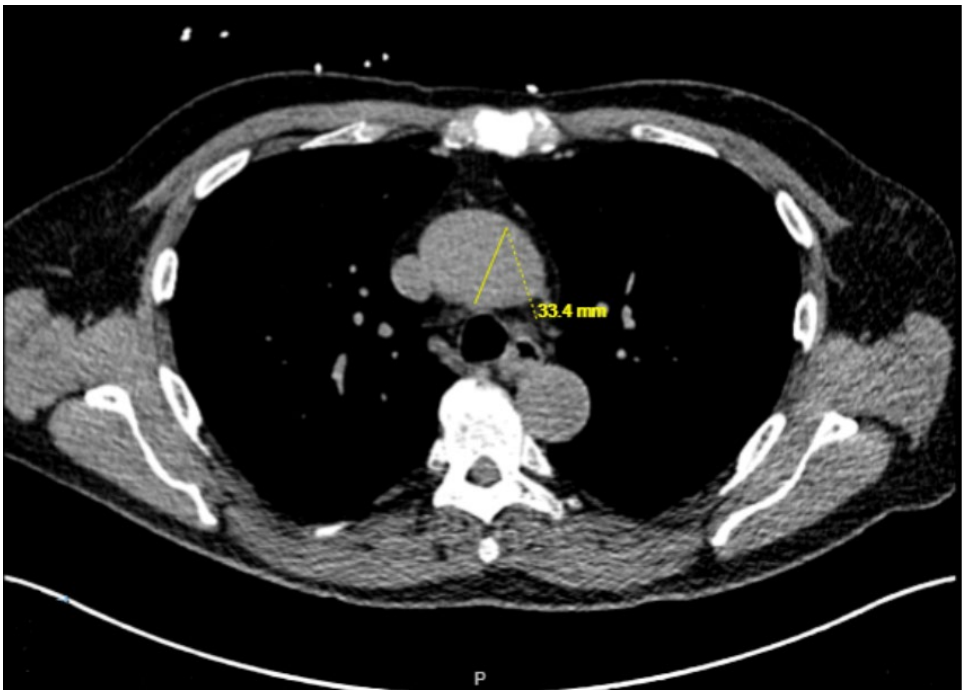
A 72-year-old man with NSTEMI, severe multivessel CAD, and ascending aortic ectasia underwent urgent CABG ×3 (LIMA–LAD, SVG–OM, SVG–PDA). After coming off bypass, the ascending aortic pursestring was non-hemostatic. A physician-modified Dacron graft was used off-label, adapting its side-arm and skirt to patch the cannulation-site tear. Persistent bleeding and a new innominate injury required femoral cannulation, DHCA, and conversion to ascending and Zone 2 arch replacement with reimplantation of the innominate and left carotid arteries (circulatory arrest 77 minutes). No preoperative cerebrovascular imaging had been obtained, and cerebral oximetry showed marked asymmetry. Initially the patient followed commands. On POD 3 he developed acute neurologic decline with facial droop, hemiparesis, and aphasia (NIHSS 19). CT showed no hemorrhage. MRI on POD 5 revealed a left PICA infarct with numerous additional infarcts across ACA, MCA, PCA, and cerebellar territories, consistent with a central embolic process after aortic reconstruction. Numerous microhemorrhages and superficial siderosis were also present. He developed AKI (creatinine peak 3.03) that later improved. Dysphagia progressed from NPO to regular diet. By POD 7–11 he was hemodynamically stable, more interactive, and improving in therapy. He was discharged to inpatient rehabilitation with continued neurologic recovery.

Discussion

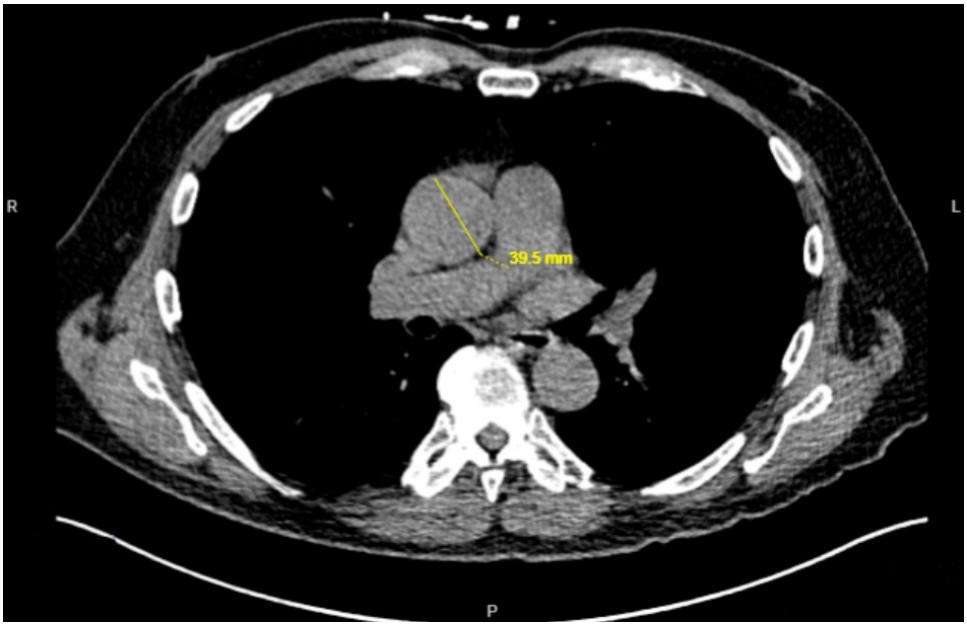
Cannulation-site tears are uncommon but recognized in patients with ectatic or degenerative aortas and may require rapid conversion to arch repair and circulatory arrest.[2],[3] The patient's postoperative infarct pattern is typical of neurologic injury after aortic surgery: multifocal, embolic, and frequently involving the cerebellum, especially the PICA territory. Up to 70% of patients have new infarcts after arch procedures.[1] Emboli may arise from friable aorta during cannulation, decannulation, or graft suturing.[3] Postoperative cerebral microbleeds are also common, occurring in over 70% of patients after cardiac surgery and likely reflecting microemboli or small-vessel stress.[4] Although superficial siderosis raised concern for underlying small-vessel disease, the distribution favored procedure-related injury. Despite prolonged DHCA, major

aortic reconstruction, and significant cerebral oximetry asymmetry, the patient experienced meaningful neurologic recovery. This case underscores the risk of embolic brain injury during complex aortic surgery and the need for close postoperative neurologic surveillance.

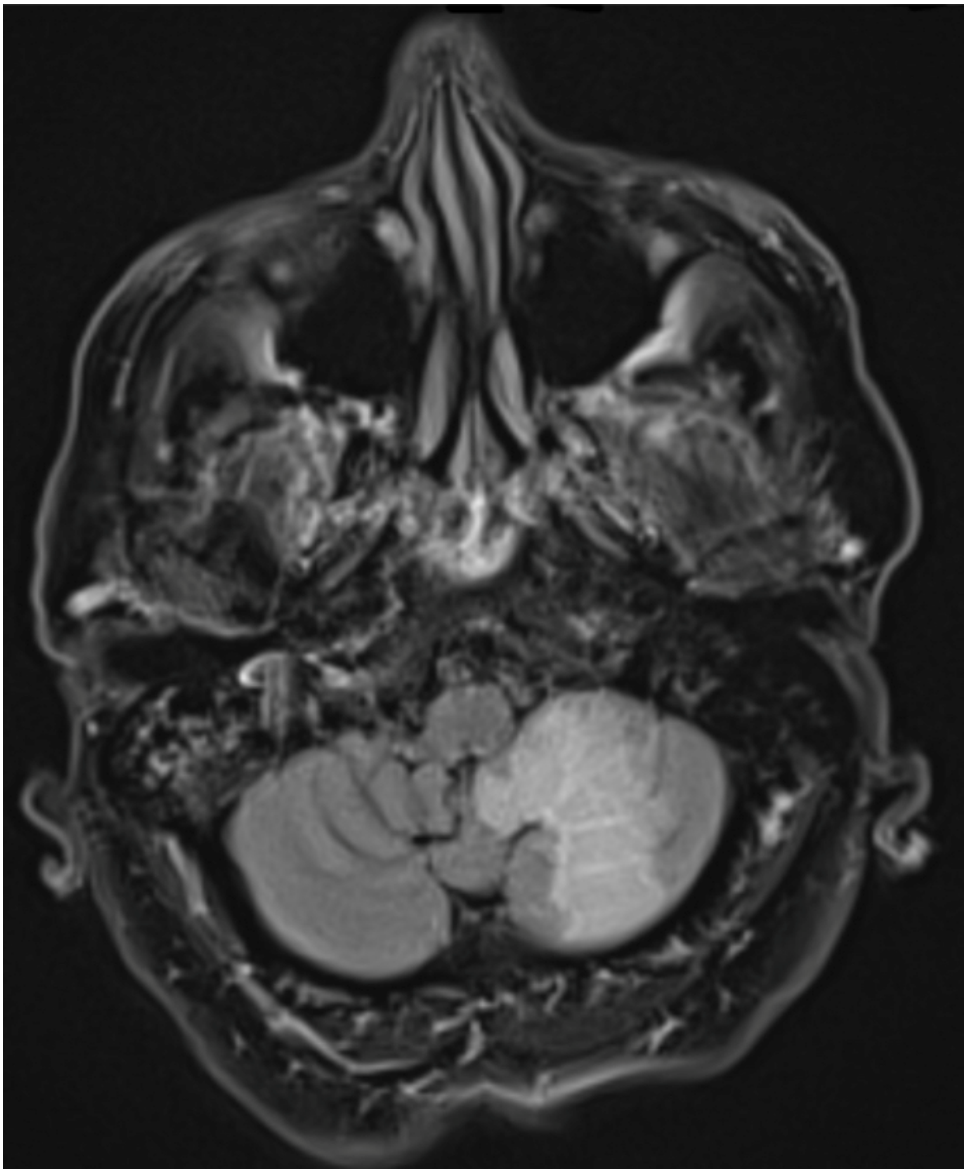
Images



CT Chest: Ectatic ascending aorta prior to surgery.



CT Chest: Ectatic ascending aorta prior to surgery.



MRI Brain: Left PICA infarct with multifocal embolic lesions.

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#FR 1 – Kiosk 11 – Poster 4

Use of Amplatzer Septal Occluder to Treat Cardiogenic Shock Secondary to Retrograde Flow through a Nonfunctioning Left Ventricular Assist Device

Presenting Author: Ariana Elder, MD, Northwestern Medicine

Introduction

Left ventricular assist devices (LVADs) have transformed advanced heart failure treatment but carry serious risk such as pump thrombosis or failure, with an incidence of 0.04% to 0.09%.[1] These conditions are often managed with anticoagulation and/or pump exchange, making treatment challenging for those with anticoagulation contraindications. In these patients, percutaneous closure devices such as the Amplatzer Occluder may offer an alternative, minimally invasive transcatheter approach. While mostly used for stroke-risk reduction and septal defect closure, Amplatzer use is also applied to LVAD-associated aortic insufficiency2 and LVAD decommissioning.3 This case highlights the novel use of an Amplatzer Septal Occluder in treating retrograde flow-induced cardiogenic shock in a patient with LVAD pump failure unable to receive anticoagulation or pump exchange.

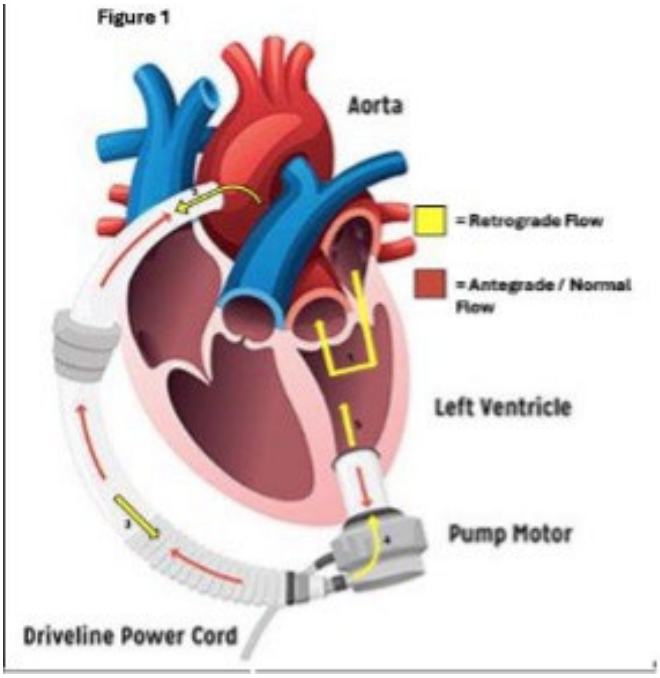
Case Presentation

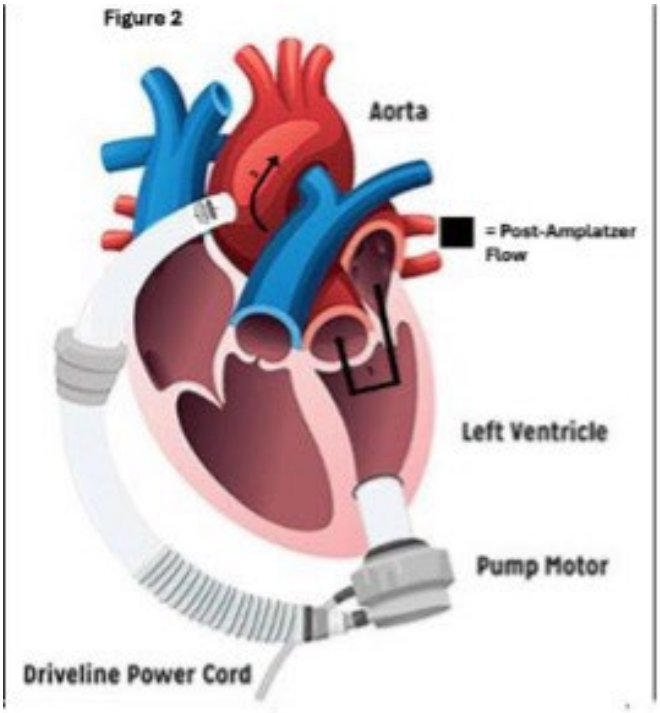
A 60-year-old male received a HeartMate III LVAD for chemotherapy-induced heart failure. Post-operative course was unremarkable until an acute subdural hematoma developed 15 months later, necessitating an anticoagulation hold. Once stable, a stepwise reinitiation of anticoagulation resulted in hematoma progression, rendering him ineligible for further anticoagulation. Despite this, the LVAD functioned well and transthoracic echocardiogram (TTE) showed preserved function without valvular disease. Five days following discharge, the patient was readmitted in cardiogenic shock with LVAD pump malfunction refractory to controller and cable exchange. TTE showed reduced biventricular function and aortic valve opening without insufficiency. Outflow graft angiography demonstrated retrograde flow through the nonfunctioning LVAD. At prohibitive risk for anticoagulation given the hematoma, the patient underwent percutaneous LVAD outflow graft occlusion to prevent recirculation. An Amplatzer Septal Occluder was deployed in the outflow graft, halting retrograde flow and decommissioning the LVAD. Hemodynamics improved and the patient was discharged home on hospice the next day, where he died two months later.

Discussion

In this patient, flow from the left ventricle was no longer directed through the malfunctioning LVAD into the aorta. Rather, blood was ejected through the aortic valve, traveling retrograde down the nonfunctioning, lower-pressure LVAD outflow graft and back into the left ventricle, leading to recirculation and progression to cardiogenic shock. This aberrant flow pathway is depicted below (Figure 1). Given the patient's absolute contraindication to anticoagulation, Amplatzer occlusion of the LVAD outflow graft treated the shock state and palliated the LVAD malfunction. As illustrated in Figure 2, graft occlusion eliminated retrograde flow, prevented recirculation into the native heart, and allowed the patient to return home on hospice. While Amplatzer devices have been used for LVAD-associated aortic insufficiency and decommissioning, there are no cases to our knowledge that use this approach to palliate cardiogenic shock from retrograde flow through a nonfunctioning LVAD, underscoring case novelty and device utility.

Images





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#FR 1 – Kiosk 11 – Poster 5

Complex Hypoxemia in an Adult with 1.5-Ventricle Glenn Physiology: Challenges in Anesthetic and Therapeutic Planning

Presenting Author: Andrew K. Russell Cheung, DO, Adult Cardiothoracic Anesthesiology Fellow, University of Nebraska

Introduction

Persistent hypoxemia in adults with 1.5-ventricle physiology presents a diagnostic and perioperative challenge. Venovenous collaterals, intrapulmonary shunting, and altered Glenn pathway flow may coexist, and determining the dominant mechanism is essential not only for anesthetic management but also for guiding potential therapeutic interventions. Interpretation is particularly difficult when pulmonary venous oxygen levels cannot be measured, which is common in Glenn anatomy due to technical challenges, risk of arrhythmias, or catheter-induced trauma. Complex congenital physiology requires multidisciplinary collaboration with multiple imaging modalities for safe anesthesia and interventions[1,2].

Case Presentation

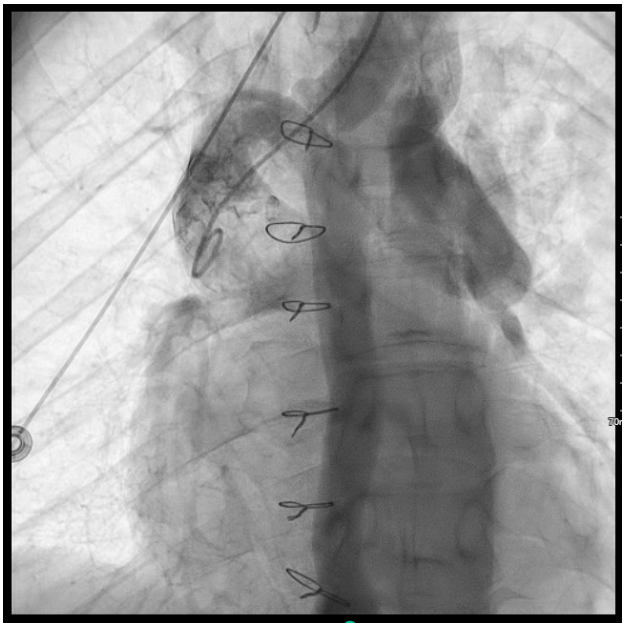
A 26-year-old male with Ebstein's anomaly status-post 1.5-ventricle repair (tricuspid valvuloplasty, RA/RV plication, ASD closure, bidirectional Glenn, and RA isthmus cryoablation) presented with progressive cyanosis and exertional intolerance. Baseline saturations were in the mid-70s. He was taken for general anesthesia for diagnostic cardiac catheterization and transesopahgeal echocardiography (TEE), with consideration for possible intervention. RHC demonstrated mildly depressed cardiac output (4.3L/min), mildly elevated biventricular diastolic pressures, mildly elevated mean pulmonary artery pressures with pulsatility extending into the innominate vein, and small branch pulmonary arteries. A large decompressing azygous vein and venovenous collateral from the innominate vein drained predominantly to the IVC, providing a pathway for systemic venous desaturation. Pulmonary venous saturations could not be obtained, limiting calculation of pulmonary blood flow and assessment of oxygenation efficiency. Hyperoxia testing was difficult to interpret; however, the patient's oxygen saturation increased to 89-92% with only 25-28% FiO_2. Test occlusion of the azygous vein produced no significant change in saturations or pulmonary artery pressures, suggesting collateral flow was not the sole driver of hypoxemia. TEE confirmed normal pulmonary venous drainage, a dysplastic Ebstenoid tricuspid valve with moderate regurgitation, a markedly dilated and dysfunctional RV, and no intracardiac shunt. Agitated saline injections demonstrated late bubbles (4-5 beats), consistent with mixed venovenous and intrapulmonary shunting. After diagnostic analysis, the patient was extubated and recovered without complication.

Discussion

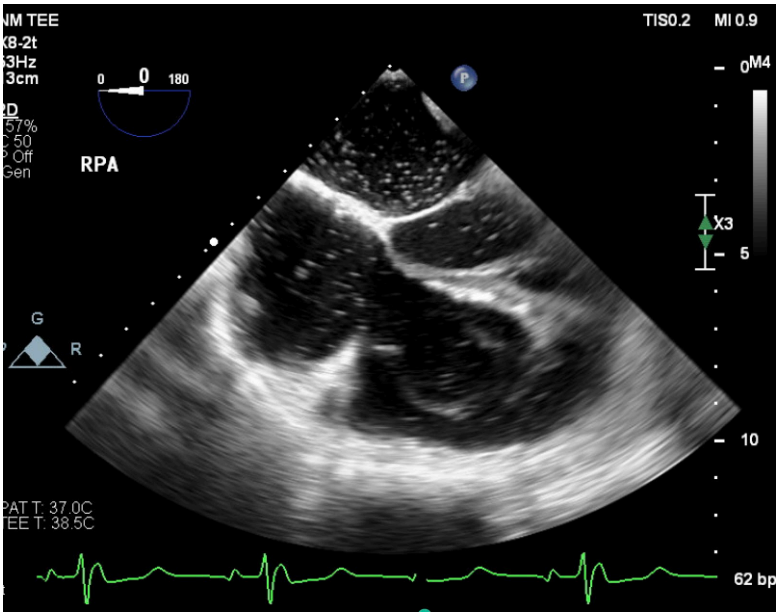
This case illustrates the complexity of evaluating hypoxemia in adults with 1.5-ventricle physiology, where Glenn flow, venous collaterals, and intrapulmonary shunting interact. Inability to sample pulmonary veins limited the quantitative assessment of pulmonary blood flow and oxygenation. The patient's robust response to low dose supplemental oxygen suggested a significant intrapulmonary component, while

systemic venous collaterals and attenuated Glenn flow indicated reduced effective pulmonary blood flow. For anesthesiologists, this case emphasizes the importance of integrating hemodynamics, TEE, and shunt physiology, and highlights how missing pulmonary venous data can impact both perioperative interpretation and planning for potential therapeutic interventions. Further management will rely on multidisciplinary review and consideration for staged intervention.

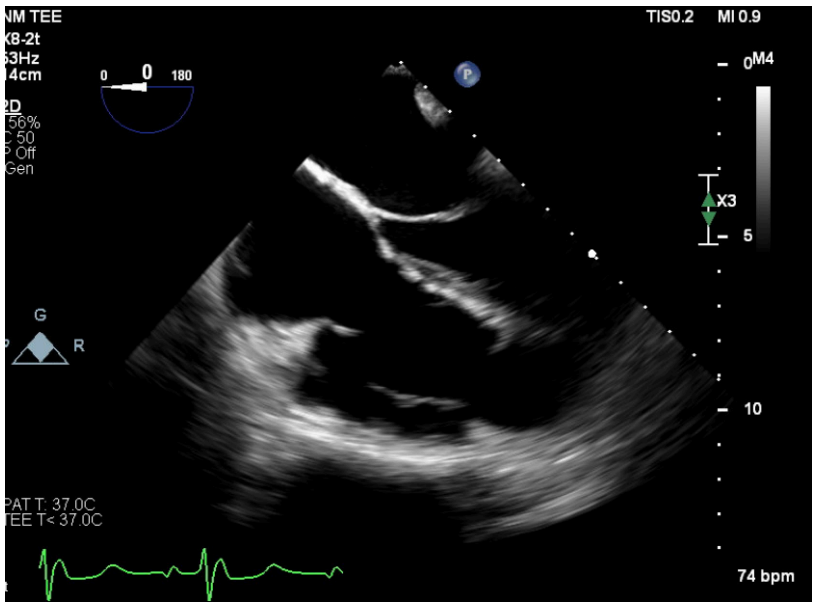
Images



Fluoroscopy Image of Azygous Collateral



Right pulmonary artery bubble study showing simultaneous LA/RA bubbles



Mid esophageal 4 chamber

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#FR 1 – Kiosk 11 – Poster 6

Severe Hypercarbia During Endoscopic Saphenous Vein Graft Harvesting

Presenting Author: Ralph Rose, Resident Physician, Cedars Sinai Medical Center

Introduction

Endoscopic vein harvesting (EVH) of the saphenous vein has become the standard technique for obtaining conduit for coronary artery bypass grafting (CABG) because it results in fewer wound complications, reduced postoperative pain, and shorter hospital stays (1). During EVH, carbon dioxide (CO_2) insufflation is used to create a subcutaneous tunnel to facilitate vein harvesting (1). Systemic absorption of CO_2 can occur, potentially causing hypercapnia. Here, we present a rare case of severe hypercapnia leading to acute respiratory acidosis during CABG associated with EVH.

Case Presentation

A 60–year–old male with triple-vessel coronary artery disease was uneventfully anesthetized for CABG surgery using standard anesthetic techniques. A baseline arterial blood gas (ABG) prior to incision revealed normal values. CO_2 insufflation for endoscopic vessel harvesting was initiated at a flow rate of 2-5 L/min, resulting in a pressure of 10 – 15 mmHg. The end–tidal CO_2 ($ETCO_2$) began to rise progressively and ultimately peaked at 94 mmHg, without improvement despite increasing minute ventilation (Figure 1). An ABG demonstrated severe respiratory acidosis (pH 7.16; $PaCO_2$ 81; PaO_2 402 (FiO_2 1) (Figure 2). The patient's temperature remained normal. Cardiac function remained unchanged, and no air was visualized in the right atrium or inferior vena cava on transesophageal echocardiography. Hemodynamics remained stable. There was no evidence of a hypermetabolic state, such as malignant hyperthermia or CO_2 embolism. At this point, there was a suspicion that the CO_2 being used for the EVH could contribute to the high $ETCO_2$. The surgeon was advised to decrease CO_2 insufflation pressure and expedite the EVH. There was a remarkable improvement in the $ETCO_2$, which returned to pre-insufflation levels shortly after EVH following cessation of CO_2 insufflation. The patient then underwent 3-vessel coronary artery bypass graft surgery without further complications.

Discussion

Severe hypercarbia during EVH is rare but can result from increased CO_2 absorption either from increased venous absorption of the gas or from entry of CO_2 through the open end of injured or cut veins during dissection (3). In this case, higher tunnel pressures, prolonged harvest, and closed EVH systems contribute to higher CO_2 absorption (3). It's crucial to rule out other potential catastrophic events, including hypermetabolic state, malignant hyperthermia, or CO_2 embolism (2). In summary, we describe a rare case of significant hypercarbia during EVH. It is important to closely monitor $ETCO_2$ during EVH for early detection of hypercarbia and timely management, including effective communication with the surgical team to promptly adjust insufflation settings and modify the harvesting technique.

Images

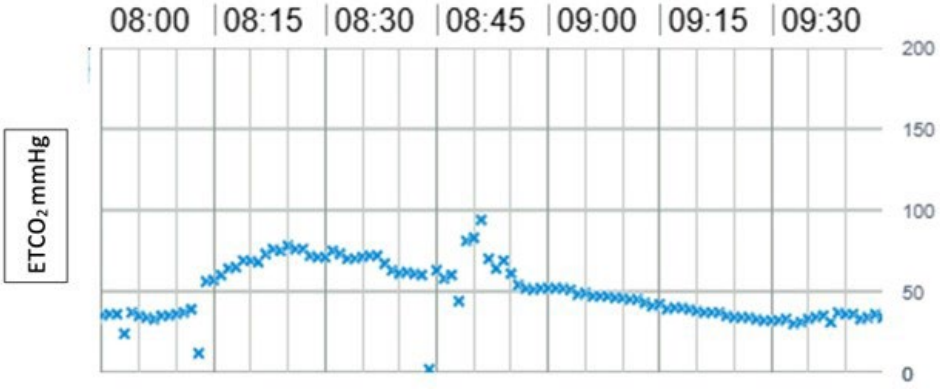


Figure 1. Uptrend of ETCO2 during EVH with peak at 94 mmHg

ARTERIAL BG	07:48	08:36	10:14
FiO2-Arterial (POC)	100	100	100
pH-Arterial (POC)	7.45	7.16	7.47
PCO2-Arterial (POC)	36	81	35
PO2-Arterial (POC)	402	473	580
HCO3-Arterial (POC)	25.3	29.0	26.5
Base Excess-Arterial (POC)	1	0	2
O2 Saturation-Arterial (POC)	100	100	100

Figure 2. Intraoperative ABGs reviewed respiratory acidosis due to severe hypercarbia of PaCO2 of 81 during EVH, which normalized after cessation of CO2 insufflation

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#FR 1 – Kiosk 12 – Poster 1

Normothermia During Cardiopulmonary Bypass in a Patient with Cold Agglutinin Disease

Presenting Author: Handeul (Hanna) Yoon, DO, PGY-1, Resident Physician, Cedars Sinai Medical Center

Introduction

Cold agglutinins are autoantibodies that cause hemagglutination of red blood cells (RBC) and extravascular hemolysis in cold temperatures, most commonly in the periphery (e.g. hands, feet, nose, ears).[1] Cold agglutinin disease poses a significant concern for those undergoing cardiopulmonary bypass (CPB), which often involves intraoperative hypothermia and cold cardioplegia to protect vital organs by reducing metabolic demand. Although there are no specific guidelines for a target temperature for intraoperative hypothermia,[2] it is imperative to protect against hemagglutination by avoiding intraoperative hypothermia.[3] We present a patient with cold agglutinin disease who successfully underwent CPB with normothermia.

Case Presentation

A 74-year-old patient with history of tobacco use disorder, atrial fibrillation on apixaban, peripheral artery disease, and chronic anemia requiring blood transfusion was transferred to our facility for higher level of care for surgical intervention of severe aortic valve stenosis and multivessel coronary artery disease. Hematologic workup for chronic anemia of unknown origin revealed a diagnosis of cold agglutinin disease (results in Table 1), and RBC agglutination seen on peripheral blood smear. The patient was uneventfully anesthetized using standard anesthetic techniques. A fluid warmer and Bair Hugger warming device were applied at the beginning of the case. Additionally, the surgical team elected to maintain normothermia during CPB while administering cold cardioplegia to maximize myocardial protection, with meticulous monitoring for evidence of hemagglutination. During CPB, cardiac arrest was achieved with antegrade cold cardioplegia, followed by intermittent direct coronary ostial administration of cold cardioplegia while maintaining a systemic temperature of 36°C. CPB duration was 227 minutes with an aortic cross-clamp time of 193 minutes. The patient was weaned from CPB after completing two-vessel coronary artery bypass grafting and aortic valve replacement. The patient was extubated six hours postoperatively with intact neurologic exam. On POD1, the patient demonstrated signs of anemia and hemolysis with dusky extremities and RBC agglutination. However, the symptoms quickly resolved with actively warming up the patient and all fluids administered postoperatively were warmed. Possible trigger for agglutination included administration of non-warmed fluids and blood products perioperatively versus surgery itself. By POD2, haptoglobin levels were normal, total bilirubin decreased, and hemoglobin levels were stable.

Discussion

Standardized guidelines for temperature management and cardioplegia for CPB for cold agglutinin disease remain limited. A few reported methods of cardioplegia during CPB for cold agglutinin disease are listed in Table 2. As the quantitative level of antibody titers is not a reliable predictor of hemagglutination or hemolysis, significant reactions may occur at low titers. This case report and other similar reports demonstrate a need for standardized guidelines regarding intraoperative temperature management during

CPB.

Images

Test	Result
Antibody Screen	Negative
Direct Coombs, Poly	Positive
Direct Coombs, IgG	Negative
Direct Coombs, Complement	Positive
Cold Agglutinin Titer	320 (Abnormal)

Table 1. Laboratory results of our patient indicating cold agglutinin disease.

	Method
A	Complete coronary and cardiac washout with intermittent cold crystalloid cardioplegia in conjunction with systemic hypothermia
B	Intermittent antegrade infusion of warm cardioplegia with continuous warm retrograde hyperkalemic infusion
C	Cold crystalloid-only cardioplegia to achieve cardiac arrest with cold crystalloid cardioplegia every 15-20 minutes

Table 2. Cardioplegia methods for myocardial protection during CPB in patients with cold agglutinin disease.

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#FR 1 – Kiosk 12 – Poster 2

When the Right Side Goes Wrong: Anesthetic Management of Carcinoid Heart Disease in a Cardiac Surgical Patient

Presenting Author: Steven Pham, DO, Fellow, New York University Langone Health

Introduction

Carcinoid tumors are rare neuroendocrine malignancies arising from enterochromaffin cells. They can secrete substances such as serotonin and histamine, which are normally inactivated by the liver. When hepatic metastases occur, these substances can reach the systemic circulation, resulting in carcinoid syndrome, classically characterized by flushing, diarrhea, and wheezing. Carcinoid heart disease (CHD) develops in 70% of patients with carcinoid syndrome, predominantly affecting the right heart due to pulmonary metabolism of the mediators before reaching the left heart. Perioperative management in patients with CHD is challenging due to the risk of carcinoid crisis, characterized by profound hemodynamic instability, arrhythmias, and bronchospasm. We present a case detailing the anesthetic considerations in a patient with advanced CHD undergoing surgical tricuspid and pulmonary valve replacement with TEE playing a crucial role in anesthetic and surgical management.

Case Presentation

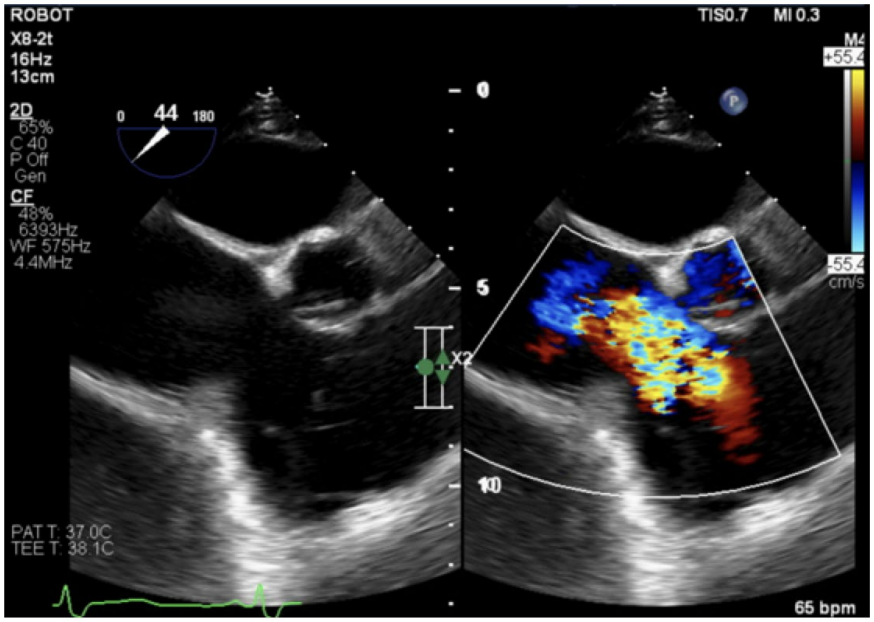
A 71-year-old female with a PMH of carcinoid syndrome, HTN, and DM2 presented with progressive fatigue and shortness of breath. TTE was notable for massive tricuspid regurgitation (TR) and severe pulmonic regurgitation (PR) in the setting of CHD. The patient was assessed in the interventional cardiology, cardiothoracic surgery, and oncology clinics and after multidisciplinary discussions, the decision was made to proceed with surgical tricuspid and pulmonic valve replacement. Prior to induction, all members of the team were made aware of histamine-releasing or sympathomimetic offending agents and triggers and the signs to be aware of in the event of a perioperative carcinoid crisis. Intra-operatively, the patient was preemptively administered diphenhydramine, famotidine, dexamethasone, and octreotide. Anesthetic induction was conducted cautiously and tailored to the patient's underlying cardiac disease, ensuring hemodynamic stability throughout. The tricuspid and pulmonic valves were successfully replaced with bioprosthetic valves with TEE confirmation. The patient was weaned from cardiopulmonary bypass and extubated in the operating room with minimal inotropic support. She was continued on the octreotide infusion post-operatively.

Discussion

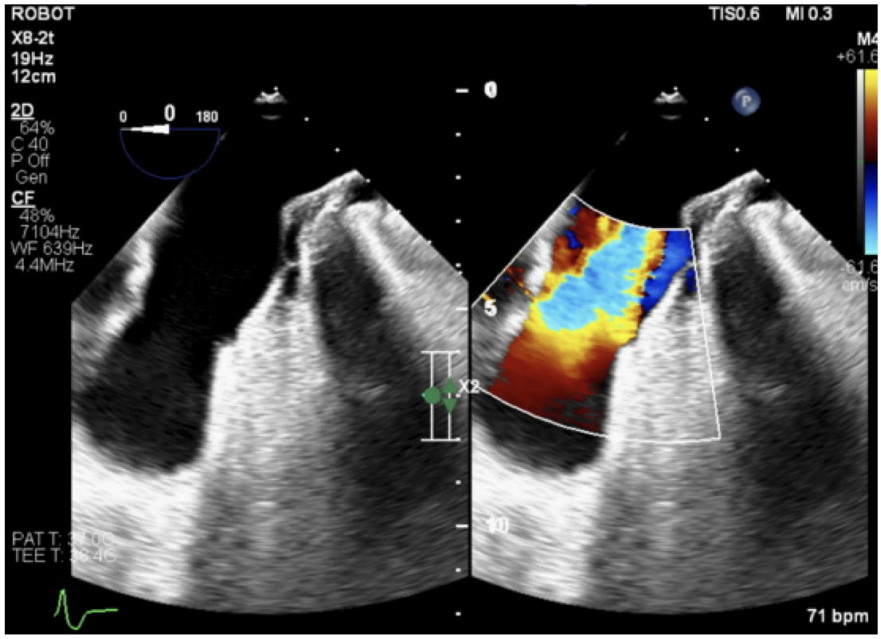
Multidisciplinary collaboration, meticulous perioperative planning, vigilant anesthetic management, and TEE guidance were all essential in contributing to this successful surgery. Perioperative infusion of octreotide effectively inhibits the release of serotonin and other vasoactive peptides, significantly reducing the risk of crisis. Triggers such as sympathomimetics and histamine-releasing agents should be avoided whenever possible, as they can exacerbate mediator release and provoke hemodynamic instability. The anesthesiologist must also be aware of the anesthetic implications associated with TR and PR.

Intraoperative TEE is crucial in the management of these cases providing real-time evaluation of volume status, contractility, and valve function prior to and post-surgical valve replacement. Our case highlights the unique challenges managing CHD perioperatively during cardiac surgery. The collaborative multidisciplinary effort played a crucial role in developing a tailored approach to mitigate the risk of carcinoid crisis and its clinical detriment

Images



Massive Tricuspid Regurgitation



Severe Pulmonic Regurgitation

CATEGORY	TRIGGER	REASON
Catecholaminergic medications	Epinephrine, dopamine	Can trigger mediator release
Histamine-releasing opioids	Morphine, meperidine	Histamine surge → hypotension
Anesthetic agents	Succinylcholine, ketamine	Can provoke catecholamine or mast cell activation
Beta agonists	Epinephrine, high-dose albuterol	Can trigger mediator release
Glucagon	Glucagon bolus/infusion	Can increase catecholamines

Triggers to Avoid to Prevent Carcinoid Crisis

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#FR 1 – Kiosk 12 – Poster 3

Failed Transcatheter Edge-to-Edge Mitral Valve Repairs Requiring Open Mitral Valve Replacement in an Elderly Patient

Presenting Author: Miriam Kaufman, MD, Resident, Johns Hopkins University

Introduction

Transcatheter edge-to-edge mitral valve repair (M-TEER) is increasingly used for degenerative mitral regurgitation (MR) in high-risk surgical candidates. While procedural success is high, complications such as partial clip detachment (up to 11%), device embolization (up to 0.1%), leaflet injury (up to 2%) may be severe [1]. Repeat TEER attempts are uncommon and can complicate surgical repair due to leaflet damage [2]. We present an 82-year-old female with three failed Pascal Ace mitral clips placed over two procedures, eventually requiring open mitral valve replacement (MVR).

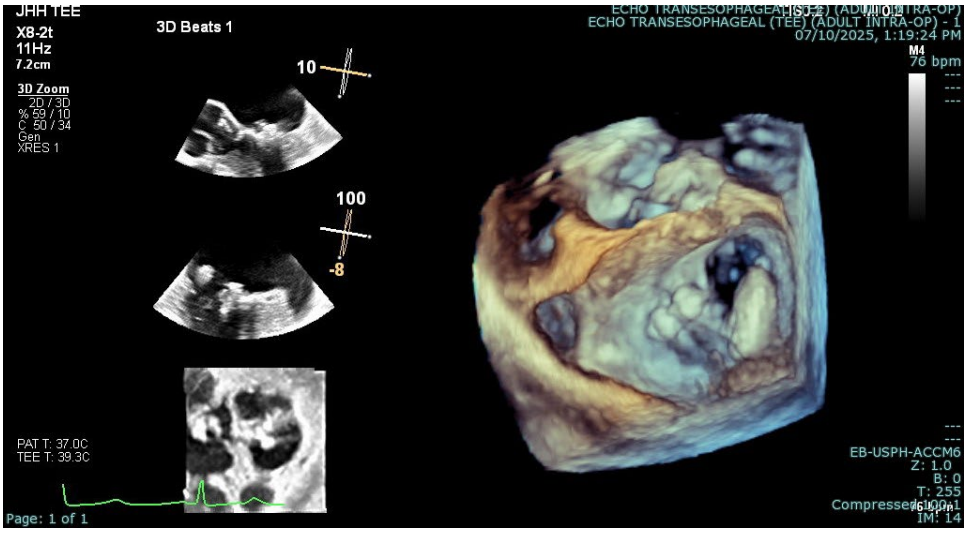
Case Presentation

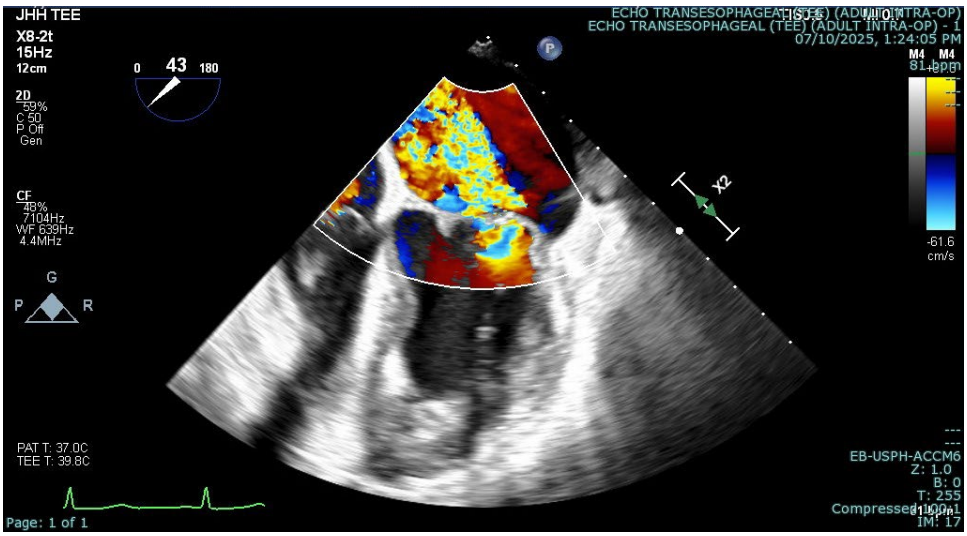
An 82-year-old woman with severe MR due to posterior leaflet prolapse underwent TEER with the Pascal Precision system, receiving two Ace devices in an A2–P2 and A1–P1 configuration. She presented one month later with worsening dyspnea and was found to have detachment of both devices from the anterior leaflets. TEE findings showed torrential MR due to detachment of the device from the A2 scallop, resulting in anterior leaflet destruction, flail of the A2 scallop, and severe P2 flail. The device on P2 was highly mobile, prolapsing into the left atrium during systole, while the P1 device was well seated but did not capture A1. She underwent a subsequent procedure during which an additional clip was placed just medial to the A1–P1 clip. However, she continued to have severe MR (mean transmittal gradient 8mmHg) and persistent heart failure symptoms despite maximally tolerated medical management and the decision was made to pursue surgical mitral valve replacement. All devices were explanted, and the flail P2 segment was resected. A 27 mm Mosaic bioprosthetic mitral valve was implanted. Postoperative TEE revealed a well-seated valve with a mean gradient 5 mmHg and trace MR. The patient's recovery was uneventful; she was extubated on postoperative day one and discharged home on day seven. At follow-up, she reported marked symptomatic improvement.

Discussion

This case highlights the mechanical and anatomical challenges associated with repeat TEER procedures. Single-leaflet detachment and leaflet tear are recognized, although uncommon, complications, often related to degenerative tissue and clip tension. Repeat device placement can exacerbate leaflet injury, rendering further percutaneous repair ineffective. This is especially problematic in high-risk surgical candidates. Surgical conversion for valve repair after multiple TEER failures is technically challenging at best due to leaflet fibrosis and annular distortion. In some cases, repair may be impossible, necessitating surgical replacement. Early recognition of device failure and multidisciplinary shared decision-making are essential to optimize outcome.

Images





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#FR 1 – Kiosk 12 – Poster 4

Primary Intracardiac Spindle Cell Sarcoma

Presenting Author: Mohammad-Saad M. Siddiqui, MD, Adult Cardiothoracic Anesthesiology Fellow, University of Cincinnati

Introduction

Primary cardiac tumors (PCTs) are very rare and the vast majority, approximately 90%, are benign3. Malignant PCTs have an incidence of 0.001% up to 0.03%, though the overall incidence of these tumors has increased in the past decade2,3. With only roughly a dozen cases described in the literature, there is a dearth of research on the best practice regarding management in these cases. Regardless of malignancy, these tumors can have significant hemodynamic effects depending on location and size. In describing this particular case, we hope to expand the available literature and provide further insight into anesthetic management of these cases.

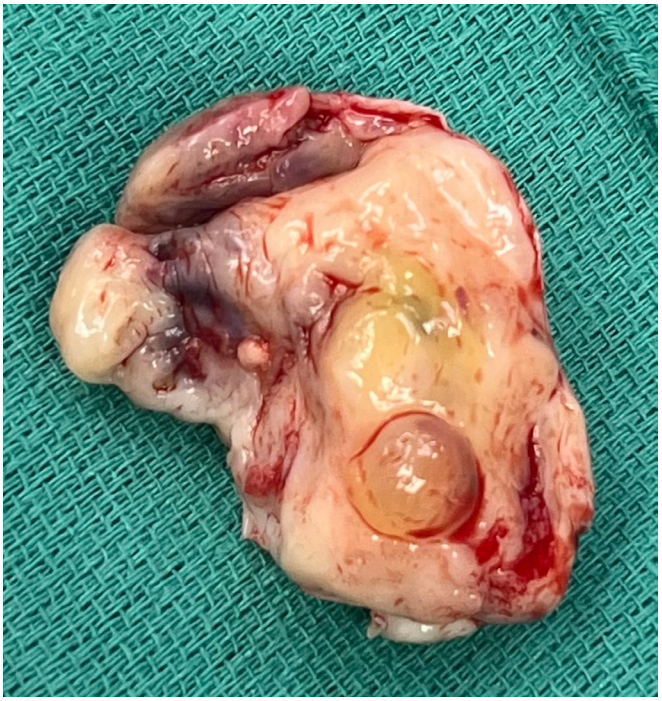
Case Presentation

A 37-year-old woman with four prior pregnancies but no other significant medical history presented with progressive dyspnea over the past year. Initial workup included a CTPA which revealed subsegmental PE. TTE revealed a large intracardiac mass within the left atrium and significant mitral regurgitation. She was referred to cardiac surgery for further workup. Her cardiac MRI revealed a large heterogenous mass with a necrotic core concerning for malignancy. A full body PET scan showed significant radiotracer uptake only within the mass, which indicated potential for primary cardiac tumor. She was then scheduled for mitral valve replacement vs repair and removal of the intracardiac mass with biopsy. Preoperative planning included review of pertinent imaging and lab work. A brachial arterial line and defibrillator pads were placed prior to slow induction with standard monitors. Additional IV access, central line, and PA catheter were placed before surgical prep. Intraoperative TEE was notable for severe mitral regurgitation and stenosis due to the mass and a dilated mitral annulus. The mass was removed without disruption of the mitral valve, which was repaired successfully, leaving no regurgitation or stenosis. A frozen sample was sent to pathology during the case and reported to be malignant, concerning for spindle cell sarcoma. She was successfully weaned off cardiopulmonary bypass and taken to the cardiac ICU on low dose milrinone. She ultimately had an uneventful recovery.

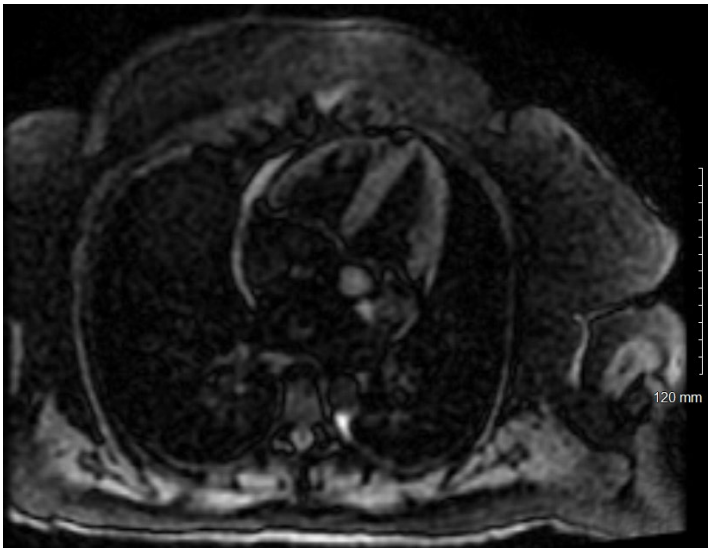
Discussion

This patient's presentation displays some of the typical symptoms associated with cardiac masses – valve regurgitation, dyspnea, and pulmonary embolism. Based on chart review, she did not display any constitutional symptoms associated with malignancy at the time of her presentation. The progression of her workup from TTE, to cardiac MRI, PET scan, and TEE were standard practice described in other case studies. In a small study, F-FDG-PET scans had a 100% sensitivity and 92% specificity for differentiating benign vs malignant cardiac masses2. It is also useful in cancer staging and determining myocardial or pericardial involvement for surgical planning2. Intraoperative management centered around maintaining normal hemodynamics and avoiding arrhythmias given that obstruction and arrhythmia can be fatal even with a benign mass. Of the malignant PCTs, sarcoma is the most common and is associated with rapid growth, central necrosis, and involvement of at least one cardiac chamber2. Prognosis for primary cardiac sarcoma is poor with a 5-year survival rate of 14%, with better outcomes for patients treated with chemotherapy and radiation2.

Images



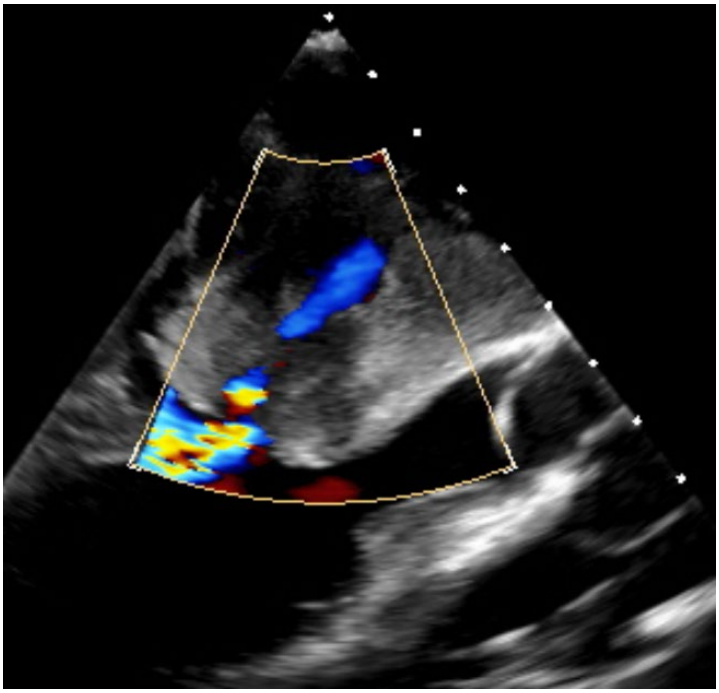
Largest gross specimen of mass



Cardiac MRI with mass heterogeneity



Intraoperative TEE image of ME 4 chamber view of the mass



Intraoperative TEE image of ME LVOT LAX view of the mass with color doppler

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#FR 1 – Kiosk 12 – Poster 5

Placement of Venous-Arterial Extracorporeal Membrane Oxygenation and Intra-Aortic Balloon Pump for cardiac recovery in severe cardiogenic shock secondary to Transfusion Associated Circulatory Overload

Presenting Author: William Chan, MD, Loyola University Medical Center

Introduction

Transfusion-associated circulatory overload (TACO) is a leading cause of transfusion-related mortality, characterized by pulmonary hydrostatic (cardiogenic) edema within 6 hours of transfusion. Signs include respiratory distress, left ventricular dysfunction/failure, pulmonary edema, and elevation of brain natriuretic peptide (1). This case highlights the use of VA-ECMO and IABP in severe cardiogenic shock from TACO.

Case Presentation

A 48-year-old male with PMH of hepatitis B related cirrhosis was admitted to the ICU following orthotopic liver transplantation. Post operatively, the patient had high volume sanguinous output from his operative drains with profound anemia, hypotension, and worsening hypoxia on mechanical ventilation. Massive Transfusion Protocol was initiated and patient was resuscitated. He then developed a rash, with persistent hypotension, increasing vasoactive requirements, and subsequently went into PEA arrest with ROSC obtained after CPR. With a concern for transfusion associated reaction, he was treated with diphenhydramine. Initial workup showed pulmonary edema with bilateral infiltrates on CXR and no acute pathology on bedside TTE. The patient was brought back to the operating room for re-exploration given hemodynamics. No active bleeding was found, however, intra-operative bronchoscopy showed significant secretions suggestive of flash pulmonary edema. TEE showed cardiogenic shock with severe global right and left ventricular dysfunction (EF 10%). Patient was then emergently placed on VA-ECMO and Intra-Aortic Balloon Pump (IABP) for cardiac support. Given the patient's presentation, it was determined that the abrupt cardiopulmonary decompensation was likely due to TACO due to evidence of pulmonary edema and fluid overload acutely exacerbated by transfusion. Initial post-operative TTE showed severe LV global hypokinesis with ejection fraction (EF) of 10-15%. A left heart catheterization was also performed with no evidence of coronary artery disease or ischemia suggesting that heart dysfunction may have been a result of myocardial stunning after severe cardiogenic shock and cardiac arrest. Over the next few days, vasopressor requirements dramatically decreased and serial TTEs showed improvement of biventricular function with an EF of 55%. Patient subsequently underwent VA ECMO decannulation and IABP removal.

Discussion

VA-ECMO is a temporary form of mechanical cardiopulmonary support that provides extracorporeal gas exchange in patients with acute cardiorespiratory failure (3). Given the rapid transfusion needs and patient's clinical picture of anuric AKI and pulmonary insufficiency with worsening hypoxia, this patient likely had an increased risk for developing TACO (2). Intraoperative TEE allowed for swift diagnosis of severe cardiogenic shock and prompt action to initiate VA-ECMO and IABP as a bridge to recovery. In recent literature review,

there are no reports on the use of VA-ECMO to overcome severe cardiogenic shock in patients with TACO. This case suggests that VA-ECMO can be used as a therapeutic intervention alongside an IABP for recovery of cardiac function.

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FR 1 – Kiosk 12 – Poster 6

BrioVAD placement: An INNOVATE trial experience for a novel LVAD

Presenting Author: Alex J. Horowitz, DO, MS, Adult Cardiothoracic Anesthesiology Fellow, University of Florida College of Medicine, Department of Anesthesiology

Introduction

End stage NYHA IV heart failure (HF) is a terrible condition with a poor prognosis and quality of life. Cardiac transplant is typically the definitive treatment. Few organs are available nationally, thus a means to temporarily support the function of the heart is necessary. Left ventricular assist devices (LVADs) are mechanical circulatory support devices designed to offload the work of the LV. They have been demonstrated to improve the quality of life in patients with NYHA IV HF and may serve as a bridge to cardiac transplantation.1 Increasingly, durable LVADs may be used for destination therapy, obviating the need for transplant due to incremental improvements in the engineering of these devices. Staying current with evolving LVAD technology is essential, as each new device brings unique management considerations that impact patient safety and outcomes. We will present our institution's first experience with implantation of the BrioVAD.

Case Presentation

A 33-year-old-gentleman with a history of chronic systolic heart failure due to nonischemic dilated cardiomyopathy was admitted to the intensive care unit (ICU) for volume and hemodynamic optimization prior to planned LVAD implantation. The cardiac surgery team implanted a BrioVAD via a median sternotomy approach on CPB. Postoperatively, the patient remained intubated on inodilation support for transport to the ICU.

Discussion

LVADs are implanted mechanical circulatory assist devices that are implanted to offload the LV. During placement, an inflow cannula is placed in the LV apex. An outflow cannula is placed in the proximal ascending aorta. A driveline is connected to the pump, tunneled through the skin, and connected to an external battery pocket and controller. Some LVADs may intermittently allow autoejection of blood through the AV and LVOT to simulate pulsatility, in addition to offloading the work of the LV. The HeartMate 3 (HM3) is the current industry standard LVAD. Complications of LVADs include thromboembolic stroke, hemorrhage, infection, myocardial infarction, and right heart failure. BrioVAD is a novel investigational LVAD designed to reduce surgical invasiveness, improve patients' QoL, and decrease the risk of some common complications. The Investigation of a Novel, MagNetically Levitated VAD for the Treatment of RefractOry Left Ventricular HeArT FailurE Clinical Trial (INNOVATE) trial is a multicenter, randomized control trial to investigate the effectiveness of the BrioVAD relative to the HM3. Several proposed benefits of the BrioVAD exist. The BrioVAD features a smaller pump than the HM3. This reduces the need for median sternotomy, and increases the likelihood of successful implantation with less invasive surgical approaches such as mini-sternotomy and left thoracotomy. These techniques are associated with a smaller incidence of

postoperative right heart dysfunction.2 The pump has a fully magnetically levitated rotor, a long-blade impeller, and optimized blood-flow pathway to minimize blood trauma, reduce hemolysis, and preserve vulnerable blood components like vWF multimers. The flexible driveline may further reduce patient discomfort, improve patient quality of life, and improve healing postoperatively with a corresponding decrease in infections.3

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#FR 1 – Kiosk 12 – Poster 7

The Three Shunt Problem: Ruptured Sinus of Valsalva Aneurysm, Outlet Ventricular Septal Defect, and Patent Foramen Ovale

Presenting Author: Karam Atli, MD, Fellow, Stanford University School of Medicine

Introduction

Sinus of Valsalva aneurysms (SOVA) are rare congenital lesions which often coexist with ventricular septal defects (VSDs).[1] The additional presence of a patent foramen ovale (PFO) creates a unique triple shunt physiology, where changes in systemic or pulmonary vascular resistance can alter shunt direction, cardiac output, and systemic oxygenation. We present the case of a patient with a ruptured SOVA, outlet VSD, and PFO undergoing repair, highlighting the anesthetic challenges of interacting shunts and the role of intraoperative echocardiography in perioperative decision-making.

Case Presentation

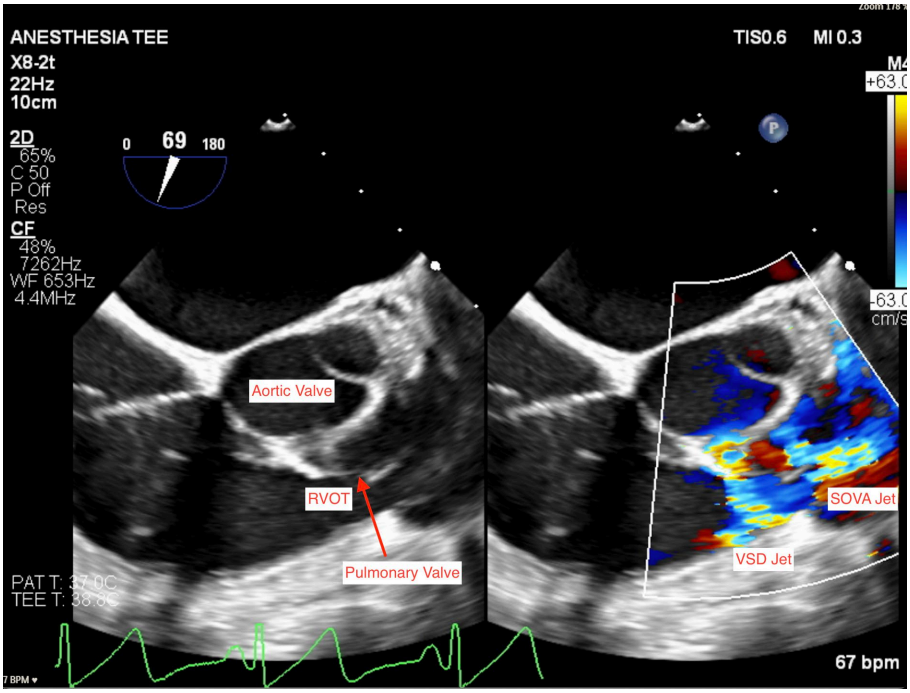
A 40-year-old woman with hypothyroidism and a restrictive outlet VSD diagnosed at birth underwent routine surveillance echocardiography which revealed a ruptured right coronary sinus SOVA. She presented for patch closure of the outlet VSD, SOVA repair, and PFO closure. Anesthetic considerations included the potential for increased left-to-right shunting through the VSD and ruptured SOVA, right-to-left shunting through the PFO, paradoxical embolism, pulmonary over-circulation, and reduction in cardiac output with fluctuations in pulmonary and systemic vascular resistances. A pre-induction arterial line was placed, general anesthesia was induced, and central venous access was obtained with careful placement of a pulmonary artery catheter under transesophageal echocardiography (TEE) guidance. Pre-cardiopulmonary bypass (CPB) TEE demonstrated normal biventricular size and function, an outlet VSD with left-to-right systolic flow (peak gradient 73 mmHg), a ruptured right coronary sinus SOVA with continuous left-to-right flow into the right ventricular outflow tract (RVOT) and a highly eccentric jet directed through the pulmonic valve towards the anterior main pulmonary artery, a PFO with left-to-right flow, mild mitral regurgitation, mild pulmonary regurgitation, and trace tricuspid regurgitation. Cardiopulmonary bypass was initiated to allow for sinus of Valsalva repair with patch closure, VSD repair with patch, PFO closure, and right coronary cusp free margin shortening. Post-CPB TEE on epinephrine 0.03 mcg/kg/min showed normal biventricular function with no residual VSD or SOVA flow, no interatrial shunt after PFO closure, and new mild to moderate eccentric aortic regurgitation directed toward the anterior mitral leaflet due to residual mild right coronary cusp prolapse. All other valves were unchanged. The patient was weaned from CPB uneventfully and was discharged from the hospital one week later.

Discussion

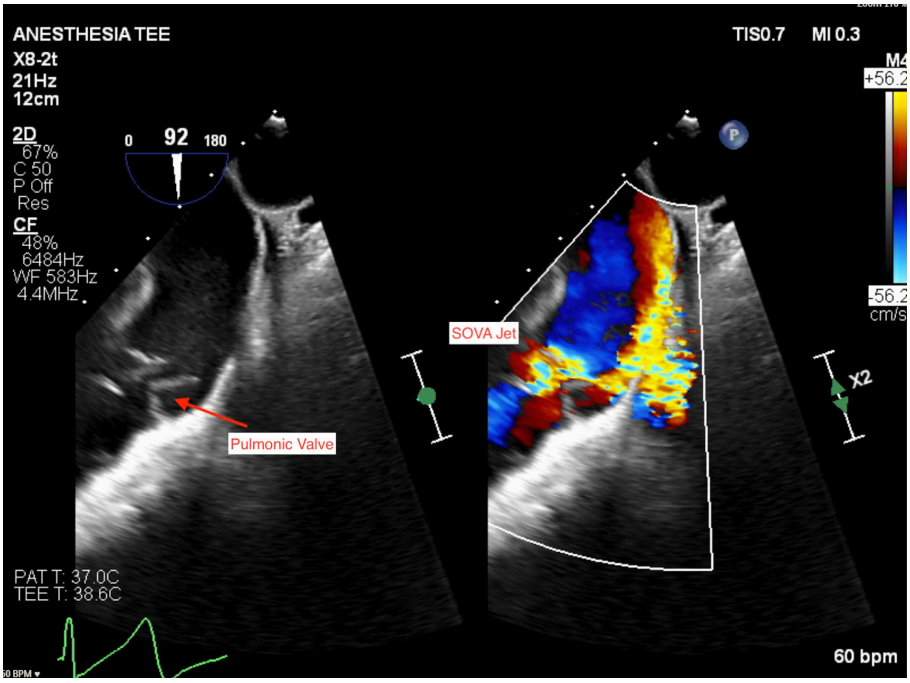
This case highlights the complexity that can arise in anesthesia for adults with congenital heart disease. In this case of multi-shunt physiology, there was the interplay of three simultaneously interacting shunts. The anesthetic strategy emphasized avoiding acute increases in SVR and PVR, air-avoidance techniques, and monitoring for pulmonary over-circulation. TEE was important for defining location of the lesions, guiding

surgical approach, and confirming complete repair. This case highlights the importance of considering individualized, physiology-driven management in complex congenital lesions.

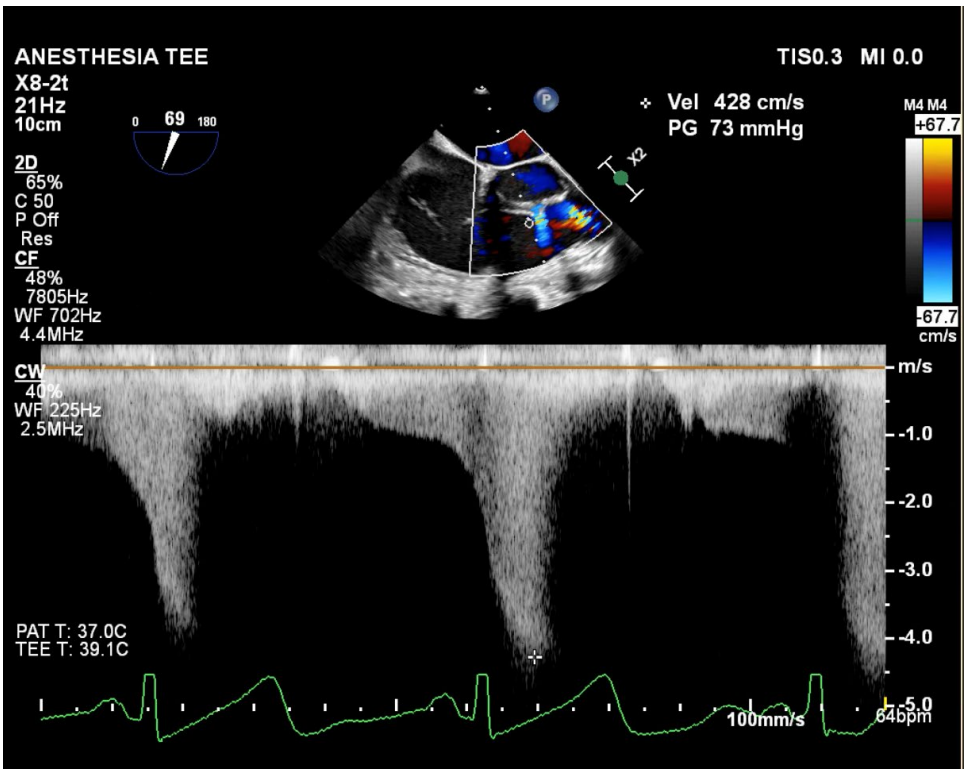
Images



Right Ventricular Inflow-Outflow TEE view showing VSD and ruptured SOVA jets.



Upper esophageal aortic arch short axis view showing ruptured SOVA jet.



Continuous wave doppler of VSD jet with peak gradient of 73 mmHg.

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#FR 1 – Kiosk 13 – Poster 1

Inadvertent Malpositioning of Paravertebral Catheter in Thorax for Robotic Mitral Valve Repair: A Case Series

Presenting Author: David Mariano, MD, Cedars Sinai Medical Center

Introduction

Ultrasound-guided paravertebral blocks have been proven to be an effective method for intraoperative and postoperative analgesia in patients undergoing thoracotomies. However, an ultrasound-guided paravertebral block is one of the most challenging techniques in regional anesthesia. In this case series, we present two cases of malpositioned of paravertebral catheters that were found intraoperatively during a robotic mitral valve repair surgery.

Case Presentation

A 61-year-old male, BMI 26.3, with a history of nonrheumatic mitral valve regurgitation presented for a robotic mitral valve repair with mini thoracotomy. Prior to the induction of anesthesia, an ultrasound-guided

T5-T6 paravertebral catheter was smoothly placed for postoperative analgesia. During the procedure, the paravertebral catheter was found to be in the thoracic cavity by the surgical team during closure. Given intraoperative need for heparinization, the catheter was not immediately removed due to suboptimal coagulation status. The catheter was removed and successfully replaced postoperatively once the patient's INR and PTT normalized on POD0. The patient's chest tube was removed on POD4, and the paravertebral catheter was subsequently removed without complications. A 67-year-old male, BMI 24.4, with a history of severe mitral valve prolapse with severe MR and PFO presented for a robotic mitral valve repair, left atrial appendage closure, and PFO closure. An ultrasound guided T3-T4 paravertebral catheter was placed preoperatively without difficulty. Intraoperatively, the paravertebral catheter tip was noted to be in the right thoracic cavity. The decision was made to keep the catheter retained in place until postoperative normalization of INR, after which a replacement catheter was placed at T6-T7 on POD0. This replacement paravertebral catheter was subsequently removed on POD4 after chest tube removal without complications.

Discussion

Paravertebral blocks and catheters are known to be an effective regional anesthetic technique for postoperative analgesia in patients undergoing thoracotomies, with both somatic and sympathetic nerve coverage. Previous studies have demonstrated that the paravertebral block provides superior analgesia to intravenous analgesia and is comparable to thoracic epidural analgesia with a better safety profile.1 In one randomized prospective trial, a paravertebral block was found to reduce postoperative pain and postoperative narcotic usage, with a higher reported satisfaction with anesthetic care after robotic mitral valve repair.2 With ultrasound guidance, it can be easier to directly visualize key landmarks. However, paravertebral block and catheter placement are not without risk. These risks include pleural puncture (0.8 - 1.1%), pneumothorax, vascular injury, nerve injury, and paravertebral catheter migration.3 Numerous factors can increase the risk of catheter misplacement, including variable patient anatomy, non-optimal body habitus, and lack of operator expertise. Therefore, it is critical for anesthesiologists placing paravertebral catheters to consider the possibility of migration into inappropriate regions after placement and when troubleshooting catheter failure in the postoperative period.

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#FR 1 – Kiosk 13 – Poster 2

Saddle Pulmonary Embolus from Heparin-Induced Thrombocytopenia after LVAD Implantation

Presenting Author: Farhan S. Raza, M.D., Fellow, University of Kansas Medical Center

Introduction

Placement of a left ventricular assist device (LVAD) represents short and long-term therapy for patients suffering from advanced heart failure. LVAD placement carries significant perioperative risk and introduces the need for lifelong anticoagulation. Complications include pump thrombosis, driveline infection, postoperative bleeding, stroke, and right heart failure. Heparin-induced thrombocytopenia (HIT) is another possible complication and is associated with worse outcomes following cardiac surgery.

Case Presentation

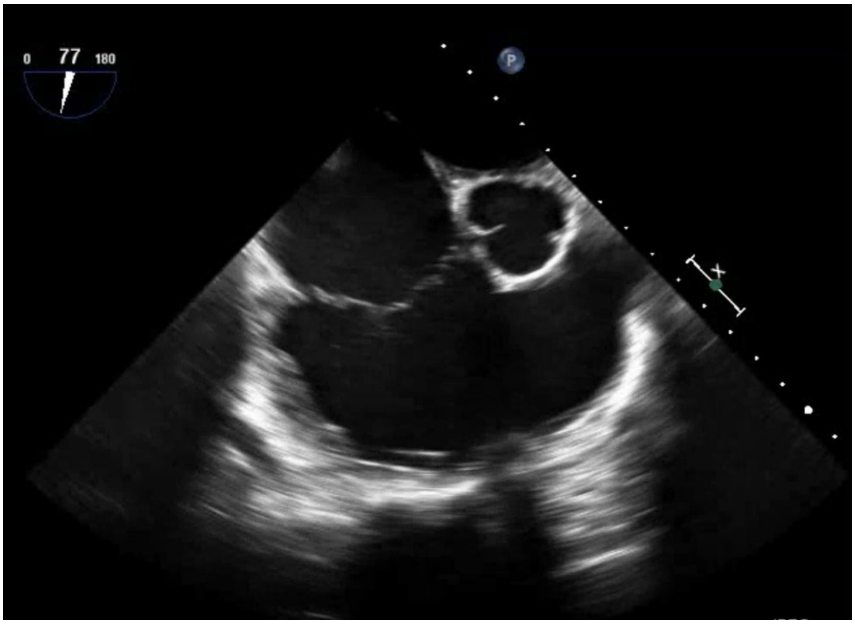
A 29-year-old man presented to a nearby hospital reporting progressive dyspnea in the setting of a recent upper respiratory infection. There, he was found to be in acute heart failure requiring vasopressors and inotropes. His clinical course progressed to INTERMACS 1 cardiogenic shock; a temporary left heart microaxial pump was placed, and he was transferred to our institution. Here, the patient underwent LVAD placement (HeartWare™) involving systemic heparinization. Postoperatively, he was started on warfarin with appropriate response. Two days later, he became hypotensive and had a syncopal event. Chest CT revealed a saddle pulmonary embolus, and thrombus in the internal jugular vein and superior vena cava. The patient underwent emergent thrombectomies requiring cardiopulmonary bypass (CPB). Upon entry of the mediastinum, the already-dilated right ventricle (RV) continued enlarging, leading to a suction event. Shortly thereafter, the patient developed frank RV failure, necessitating cardiac massage and emergent cannulation for CPB. The surgery was successful, though there was an iatrogenic type B aortic dissection from the cannulation attempt. This was diagnosed via transesophageal echocardiography. It was ultimately managed conservatively and continues to be surveilled. Postoperatively, the patient underwent an IVC filter placement. Leading up to his thrombotic event, it was noted that the platelet counts had steadily decreased to a nadir of 31,000/μL - a four-fold decrease over the previous week. Molecular testing confirmed a diagnosis of HIT, which is thought to be the cause of his thrombi. He ultimately recovered, was discharged on postoperative day 46, and remains stable on warfarin therapy. Follow-up HIT testing has been negative.

Discussion

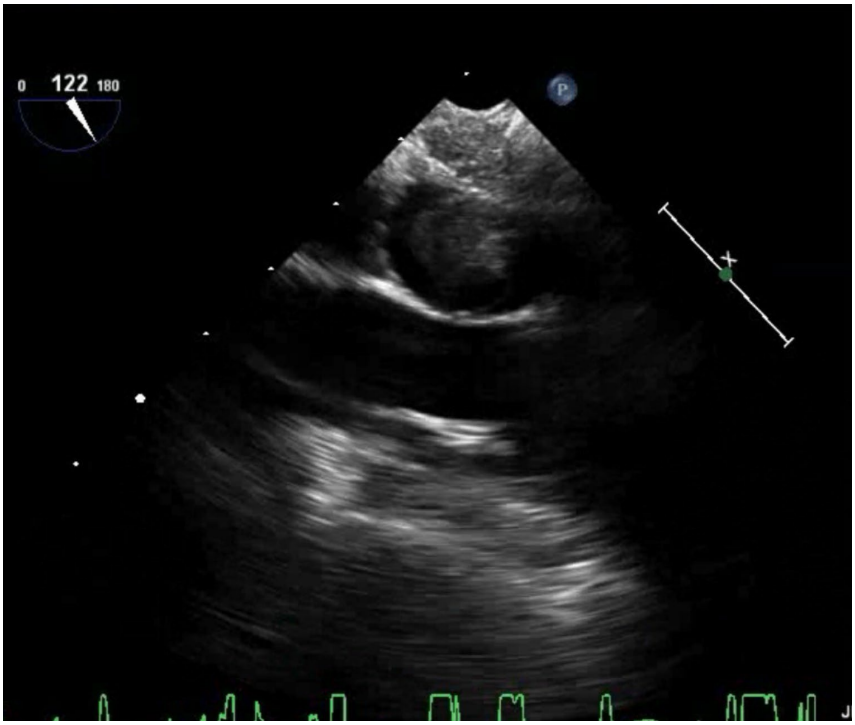
Heparin is the anti-clotting agent of choice for surgeries involving CPB due to its reversibility. HIT is an immune response to complexes formed by heparin and platelet factor 4 resulting in thrombocytopenia. HIT usually develops 5-14 days following heparin exposure. It leads to platelet activation and aggregation, which can cause thrombosis. In patients with high pre-test probability, the diagnosis can be confirmed via antibody detection (ex: serotonin release assay). There are no clear guidelines regarding anticoagulation strategies in patients with HIT undergoing CPB. Management options include intraoperative anticoagulation with direct thrombin inhibitors (such as bivalirudin), giving heparin with intravenous antiplatelet agents, and plasmapheresis. It may also be reasonable to delay surgery until antibodies have cleared. The American

College of Chest Physicians suggests administering bivalirudin in these patients (Grade 2C), though this can increase bleeding risk and it does not have a reversal agent.

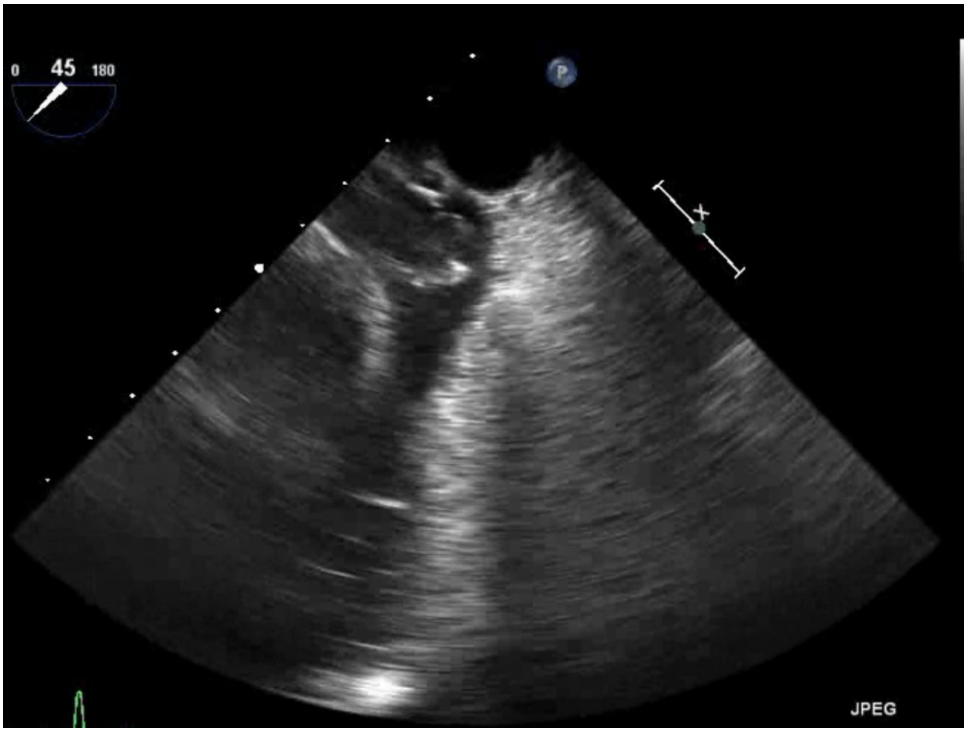
Images



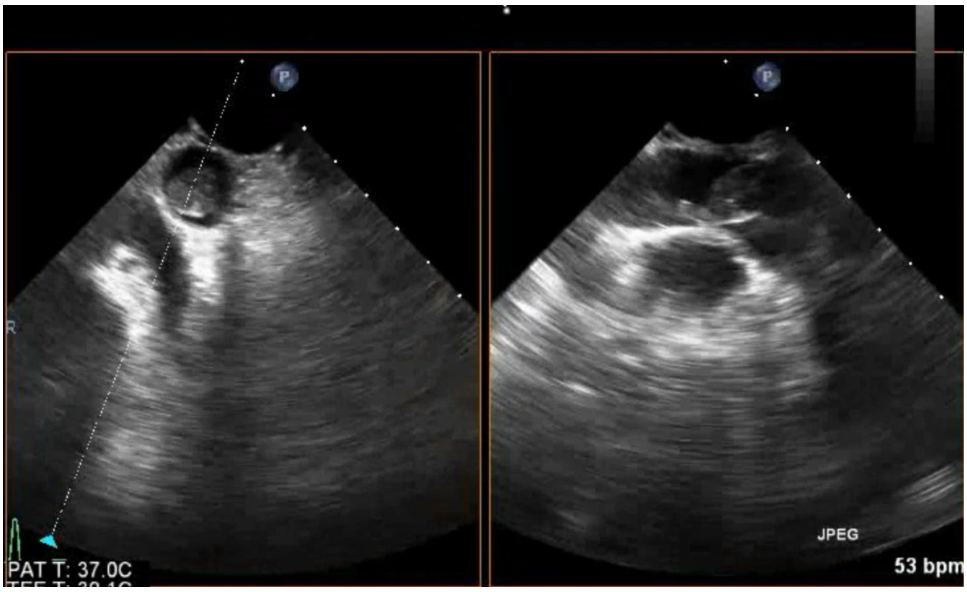
ME RV inflow-outflow showing RV dilation in the setting of PE



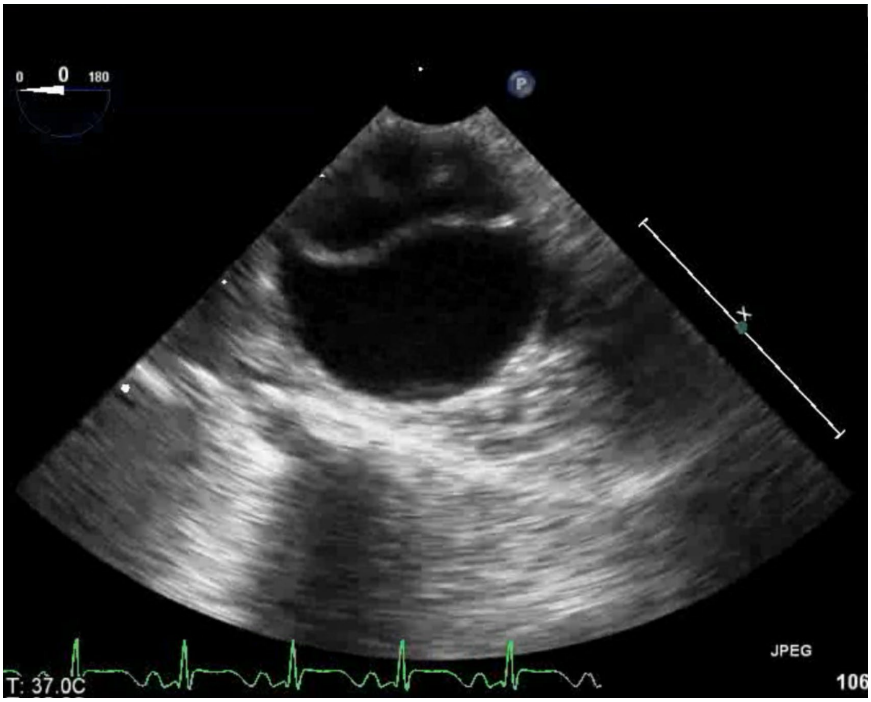
ME ascending aortic LAX showing PE



ME asc aortic SAX showing PE



UE aortic arch SAX with X-plane



Desc aortic SAX showing intimal tear post CPB

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#FR 1 – Kiosk 13 – Poster 3

Euglycemic Diabetic Ketoacidosis After Emergent Non-Cardiac Surgery in a Remote Heart-Transplant Recipient on SGLT2 Inhibitor Therapy

Presenting Author: Dominique Levy, MD, Cedars Sinai Medical

Introduction

Sodium–glucose cotransporter-2 (SGLT2) inhibitors are increasingly used in transplant recipients for cardiometabolic benefit but pose a perioperative risk for euglycemic diabetic ketoacidosis (EDKA). Recognition is challenging because glucose levels remain normal and early physiologic warning signs may be blunted in heart-transplant patients due to cardiac denervation. Existing reports of SGLT2i-associated EDKA in heart-transplant recipients are limited to the peri-transplant period or cardiac surgery. Whether long-term, clinically stable transplant recipients remain vulnerable during unrelated emergent surgery is not well understood.

Case Presentation

A 47-year-old man, 10 years post–orthotopic heart transplant with normal graft function, presented with perforated appendicitis. His most recent doses included empagliflozin one day prior and semaglutide seven days prior. Preoperative labs showed hemoconcentration (Hgb 16.7), leukocytosis (WBC 12.8), and high anion-gap metabolic acidosis (Na 131, CO_2 19, anion gap 22) despite glucose 105–123 mg/dL. Beta-hydroxybutyrate was 2.6 mmol/L; urinalysis showed 4+ glucosuria and 1+ ketonuria. He underwent urgent robotic appendectomy with arterial-line monitoring. Postoperatively, acidosis progressed (pH 7.30→7.26; HCO_3 19.5→17.7) while vitals remained stable (HR 98–115; BP 113/82), consistent with denervated transplant physiology. He was admitted to the SICU with presumed EDKA and treated with insulin infusion, D5W-NS, and electrolyte repletion. Acidosis resolved within 48 hours. Additional issues included ileus and an incidental right-middle-lobe pulmonary embolism requiring anticoagulation. He was discharged on postoperative day 8 with permanent discontinuation of empagliflozin.

Discussion

This case demonstrates that EDKA can occur more than a decade after heart transplantation during non-cardiac surgery, expanding the clinical spectrum of SGLT2 inhibitor–associated metabolic complications. The patient's euglycemia and stable hemodynamics delayed recognition, highlighting a diagnostic blind spot when caring for denervated transplant recipients. The combination of recent SGLT2 inhibitor exposure, fasting, infection, and surgical stress created ideal conditions for ketogenesis despite normal glucose. Early measurement of beta-hydroxybutyrate and routine anion-gap assessment are critical when SGLT2 inhibitors are involved. Current perioperative guidelines to stop SGLT2 inhibitors ≥3 days before elective surgery may not fully protect high-risk transplant patients presenting emergently. To our knowledge, this is the first reported case of SGLT2 inhibitor–associated EDKA in a remote heart-transplant recipient undergoing emergent non-cardiac surgery.

Images

FIGURE 1. Perioperative Timeline – Remote Heart-Transplant Recipient with EDKA

9/8/25
• Last dose of empagliflozin (SGLT2 inhibitor)

9/9/25
• RLQ abdominal pain → CT: perforated appendicitis
• Labs: AG 22, CO2 19, Glucose 105–123 (euglycemic)
• β-hydroxybutyrate 2.6 mmol/L; UA: 4+ glucose, 1+ ketones
• Urgent robotic appendectomy; arterial line inserted

9/10/25
• Worsening metabolic acidosis: pH 7.30 → 7.26; HCO3 19.5 → 17.7
• Hemodynamics stable (HR 98–115; BP 113/82) — denervated heart
• SICU admission → EDKA diagnosed
• Insulin infusion + D5W-NS initiated

9/11–9/12/25
• Ileus; mild wound infection

9/13/25
• Incidental right-middle-lobe pulmonary embolism → anticoagulation

9/14–9/16/25
• Resolution of EDKA; normalization of pH and bicarbonate

9/17/25
• Discharged home
• Empagliflozin permanently discontinued

FIGURE 1. Perioperative Timeline – Remote Heart-Transplant Recipient with EDKA

TABLE 1. Key Laboratory Data Demonstrating Euglycemic DKA

Parameter	Value Range	Interpretation
Glucose	105–123 mg/dL	Normal despite severe acidosis
Anion Gap	22	High, consistent with AGMA
CO2 (HCO3–)	17.7–19.5 mmol/L	Markedly low
pH	7.26–7.30	Metabolic acidosis
β-Hydroxybutyrate	2.6 mmol/L	Elevated ketones
Urinalysis	4+ glucosuria; 1+ ketonuria	Confirms ketogenesis
Hemoglobin	16.7 g/dL	Hemoconcentration

TABLE 1. Key Laboratory Data Demonstrating Euglycemic DKA.png

TABLE 2. Hemodynamics and ABG Progression

Time	pH	HCO3	CO2	AG	Glucose	HR	BP	Interpretation
9/09/25 23:42	7.30	19.5	40	22	105–120	98–110	113/82	Early acidosis; euglycemia
9/10/25 00:24	7.26	17.7	40	22	122	102–115	Stable	Worsening; masked by denervation
9/10/25 01:12	7.27	18.5	41	—	—	100–112	Stable	Persistent acidosis
9/10/25 SICU	7.2–7.3	17–19	Normal	High	105–123	98–115	Stable	Meets EDKA criteria
9/11–12/25	Improving	19–21	Normal	Closing	110–130	90–110	Normal	Response to therapy
9/14–16/25	Normal	22–24	Normal	Normal	100–115	90–105	Normal	Resolution

TABLE 2. Hemodynamics and ABG Progression

TABLE 3. Medication Timeline

Date	Medication	Dose/Details	Relevance
9/2–9/8/25	Semaglutide	1 mg weekly	Delayed gastric emptying; ileus risk
9/8/25	Empagliflozin	10 mg	**Primary trigger; promotes ketogenesis**
Baseline	Tacrolimus	1 mg AM / 0.5 mg PM	Maintains graft; no direct EDKA impact
Baseline	Mycophenolate	250 mg BID	↑ infection risk
Peri-op	Antibiotics	Appendicitis regimen	No metabolic impact
9/10/25	Insulin infusion	ICU protocol	Reverses EDKA
9/10/25	D5W-NS fluids	Continuous	Stops ketogenesis
9/13/25	Apixaban	PE treatment	No metabolic effect

TABLE 3. Medication Timeline

TABLE 4. Differential Diagnosis Comparison

Feature	EDKA	Classic DKA	Starvation Ketosis	Lactic Acidosis
Glucose	**Normal**	High	Normal–low	Normal–mildly high
Ketones	**High**	High	Mild	Absent
pH	**Low**	Low	Normal–mild ↓	Low
Bicarbonate	**Low**	Low	Mild ↓	Low
Anion Gap	**High**	High	Mild ↑	High
Trigger	**SGLT2i + fasting**	Insulin deficiency	Fasting	Shock/ischemia
Hemodynamics	**Stable**	Tachycardic	Stable	Often unstable
Hallmark	**Ketosis + euglycemia**	Hyperglycemia	Fasting	↑ Lactate

TABLE 4. Differential Diagnosis Comparison

TABLE 5. Anesthetic Implications & Perioperative Pearls

Issue	Key Insight	Anesthetic Impact
Denervated Heart	No tachycardic response	Vitals unreliable → labs critical
SGLT2i Mechanism	Promotes ketogenesis	Hold ≥3 days electively
Euglycemia	Glucose normal	Do NOT exclude DKA; check β-hydroxybutyrate
Ventilation	Compensatory hyperventilation	Avoid suppressing ventilation
Volume Status	Hemoconcentration	Aggressive fluids
GL		

TABLE 5. Anesthetic Implications & Perioperative Pearls

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#FR 1 – Kiosk 13 – Poster 4

Intraoperative Coronary Sinus Injury: Case Series Review of Risk Factors and Transesophageal Echocardiogram Assessment

Presenting Author: Quinton Riter, DO, Fellow, Cleveland Clinic Foundation

Introduction

Coronary sinus injury (CSI) is a rare but potentially life-threatening complication of retrograde cardioplegia (direct and indirect cannulation), with an incidence of 0.06–0.6%.(3) It is often caused by mechanical stress from high cardioplegia pressures, challenging or mispositioned cannula placement, balloon overinflation, or excessive cardiac manipulation. These mechanisms can tear, dissect, or perforate the compliant, low-pressure coronary venous sinus.(3) As cardiac anesthesiologists, prompt recognition and assessment of CSI is critical for guiding management. We present a case series of 4 intraoperative CSIs, highlighting key transesophageal echocardiography (TEE) findings and management considerations.

Case Presentation

All cases were performed under general anesthesia with TEE, invasive arterial monitoring, and internal jugular central venous access. Direct or indirect retrograde cardioplegia cannulas were placed prior to cardiopulmonary bypass for diastolic cardiac arrest by 4 different surgeons over a short time span (months). (For details, see table 1)

Discussion

CSI is an uncommon complication of retrograde cardioplegia cannulation. Risk factors include delivery pressures > 40 mmHg, difficult cannula placement, cannula malposition, operator inexperience, and

excessive cardiac manipulation. (1,3) Early recognition is essential to guide timely management and prevent further morbidity. TEE can aid in the detection of CSI and in assessing its hemodynamic significance by evaluating mitral inflow and pulmonary vein flow, identifying structural abnormalities, and monitoring interval changes—particularly after correction of coagulopathy. In parallel, continuous vigilance for increased surgical bleeding, acute tamponade, conduction disturbances, and hemodynamic instability is warranted, as these may indicate ongoing coronary sinus disruption, atrial ventricle groove instability, expanding hematoma formation, or development of false lumens. The coronary sinus is visualized with the TEE mid esophageal (ME) 4 chamber, ME 2 chamber, and modified bicaval views which can also be used to guide cannula placement. The ME 4 chamber, ME commissural, ME 2 chamber, and ME aortic long axis assess for encroachment on left atrium (LA), LA collapse, and pseudo-mitral stenosis physiology which may be the first indications of CSI.(2) Evaluation of lesion progression, injury location, and coronary sinus patency guides management. Mitral inflow obstruction, hemodynamic instability, or an expanding hematoma may warrant surgical repair. Coronary sinus injuries can be managed with direct suturing, hemostatic packing agents, or venous/pericardial patch techniques, though repair is often challenging given the coronary sinus' posterior location.(1) Stable abnormalities from CSIs may be managed conservatively with serial monitoring. Treatment centers on correcting coagulopathy to promote thrombosis within the low-pressure system and on careful preload optimization. Adequate preload helps maintain LA pressure and prevents hematoma-related LA collapse and functional mitral stenosis, whereas excessive preload may worsen coronary sinus dilation or propagate LA dissection.(1) In summary, TEE allows for prompt recognition and tailored management of CSI resulting in improved patient care.

Images

Table 1: Summary of Cases.	Column1	Column2	Column3	Column4	Column5	Column6	Column7	Column8	Column9
	Age	Comorbidities	Surgery	CPB time	Method of Cardioplegia	Main TEE finding	Further Evaluation of CSI w/w.o TEE	Follow Up Echocardiography Results	Special Considerations
Case 1	72yoF	Hodgkin lymphoma s/p radiation c/b radiation induced heart disease with moderate AI/AS, moderate-severe MR with mild stenosis, and moderate TR	AVR, MVR, TVr	123 minutes	Antergrade and direct retrograde	Hematoma noted tracking along inferior and lateral walls of left atrium without expansion after protamine	(1) No mitral inflow obstruction (2) The visual component of the hematoma appeared decreased in size after protamine & chest closure with no wall motion abnormalities on TEE	Hospital course complicated by problems associated with patient's radiation history including dysphagia (EGD, corpak) and AFib eventually requiring PPM. Follow up POD8 echo showing resolution of hematoma.	Surgery noted as complex due to radiation heart disease with calcification of aortomitral curtain
Case 2	71yoF	MI s/p PCI to LAD (1/2019) and distal ramus, LCx (1/2021), HTN, HLD, Postop AF (Self converted)	CABG (LIMA-LAD, SVG-PDA, SVG-Ramus)	89 minutes	Antergrade and indirect retrograde	Bi-directional flow between the RA and false lumen located in posterior LA following CPB, most likely due to coronary sinus injury induced LA dissection	(1) No mitral inflow obstruction (2) No hemodynamic instability following cessation of CPB (3) False lumen initially originating from posterior wall of the LA, which expanded anteriorly despite protamine and chest closure	Discharged POD5. POD12 TTE no signs of residual LA dissection secondary to coronary sinus injury	[1] Surgical team noted resistance with placing the retrograde cannula. A total of 3 attempts were made for final placement [2] Manipulation of the heart to access the posterior coronaries for grafting may facilitate retrograde cannula migration/misplacement
Case 3	70yoF	Thoracic aortic aneurysm, Afib and MV prolapse	David procedure, b/l PVI & LAAC	153 minutes	Antergrade and indirect retrograde	Large hypoechoic cavity in the posterolateral left atrial wall, which reduced in size after discontinuation from CPB. A low velocity flow was noted in the cavity. The hypoechoic cavity measured 3.5 cm X 1.1 cm	(1) No mitral inflow obstruction (2) No longer flow through the false lumen which eventually clotted off after protamine administration	Discharged POD7. POD4 TTE no signs of false cavity with flow	
Case 4	75yoM	HFrEF (LVEF 20%), CAD with MVD, pAF/AFL s/p failed ablation (Eliquis), HTN, HLD, Hypothyroidism, and CKD	HM3 LVAD placement, CABG, MAZE, and LAAL	131 minutes	Antegrade and direct retrograde	Following cessation of CPB, LA & LV noted to be compressed by a coronary sinus hematoma but with adequate HM3 flow. No mitral inflow obstruction.	Lateral wall hematoma decompressed and closed with interrupted pledgeted sutures. No mitral inflow obstruction.	Postoperative course c/b PDA laceration which was repaired. POD14 TTE noting residual lateral wall hematoma indenting into the LV cavity without hemodynamic effects.	(1) Manipulation of the heart to access the posterior coronaries for grafting may facilitate retrograde cannula migration/misplacement (2) Hematoma noted on postoperative TTE may be confounded by PDA laceration but nonetheless, no hemodynamic consequences from the hematoma.
Abbreviations: s/p- status post, c/b- complicated by, MVD- mitral valve disease, AF-atrial fibrillation, AFL- atrial flutter, PVI- pulmonary vein isolation, LAAC- left atrial appendage clip, CPB- cardiopulmonary bypass, HM3- Heartmate 3, POD- post operative day									

Table 1.

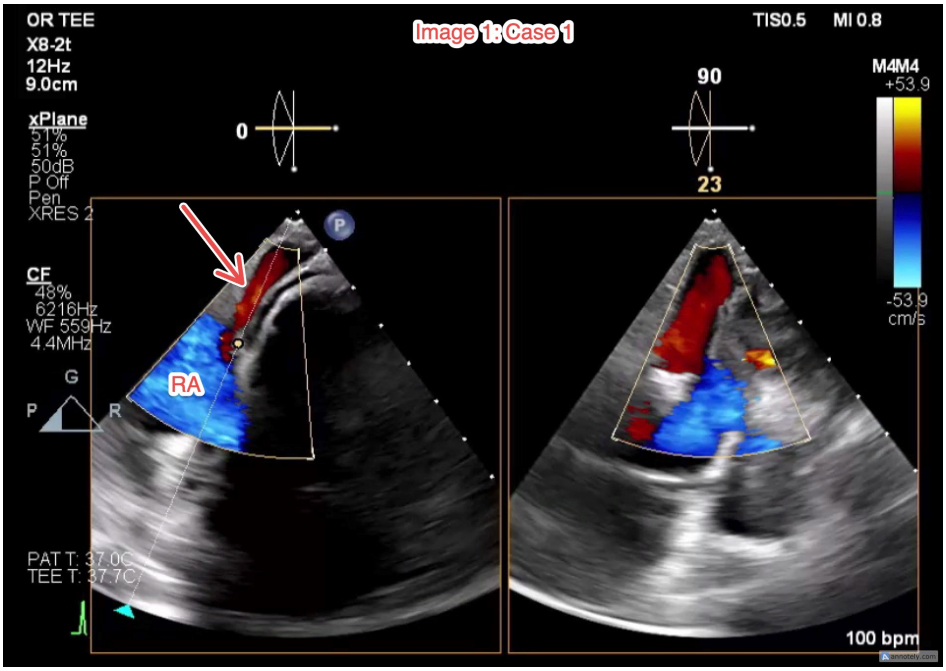


Image 1: Case 1- Arrow indicating hematoma overlaid with color flow.

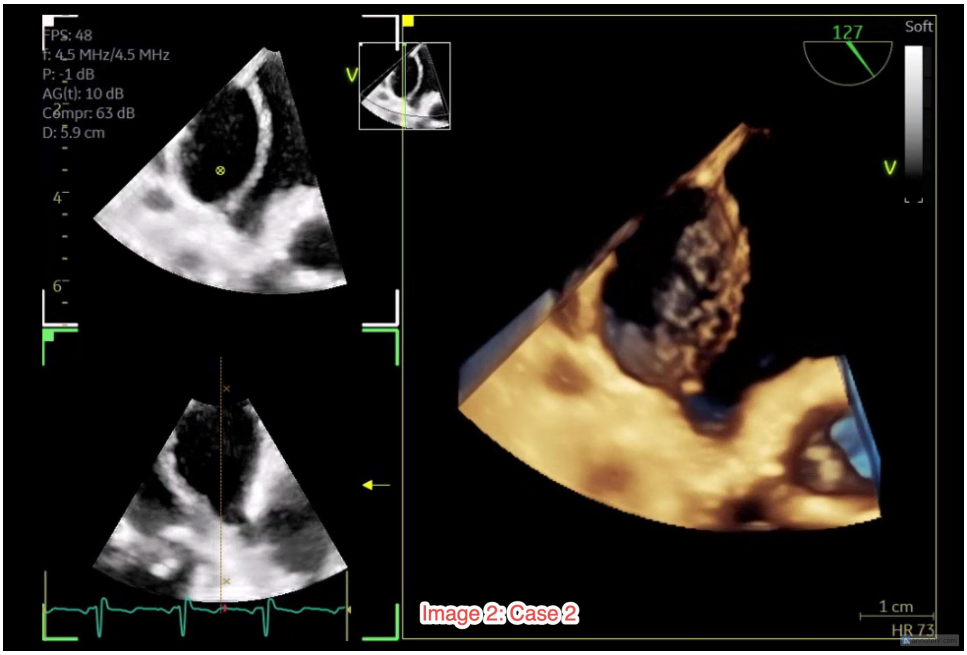


Image 2: Case 2- 3D of false lumen/ hematoma.

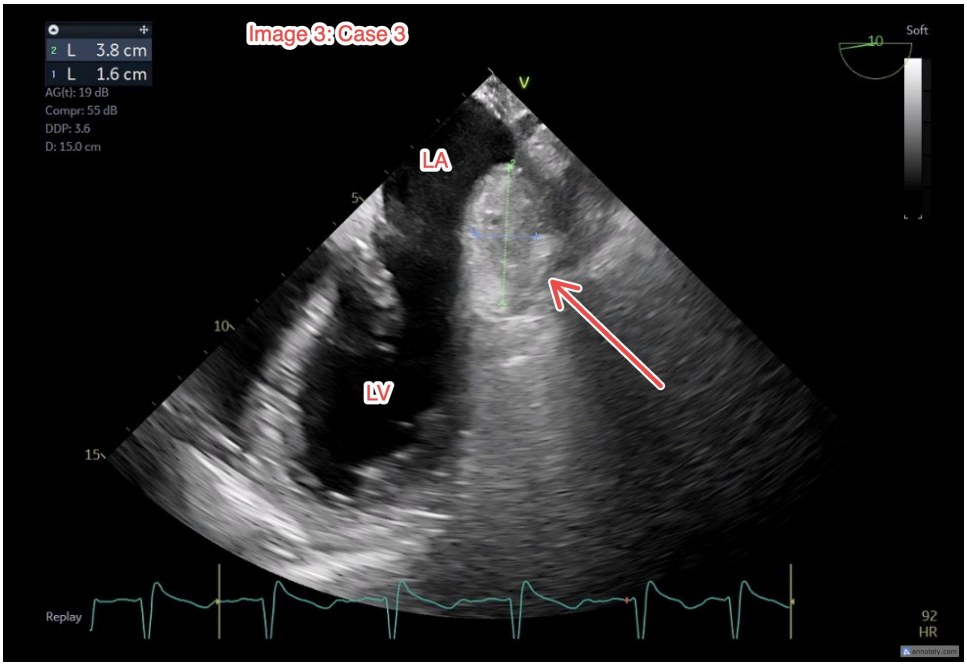


Image 3: Case 3- Arrow indicating hematoma in lateral wall.

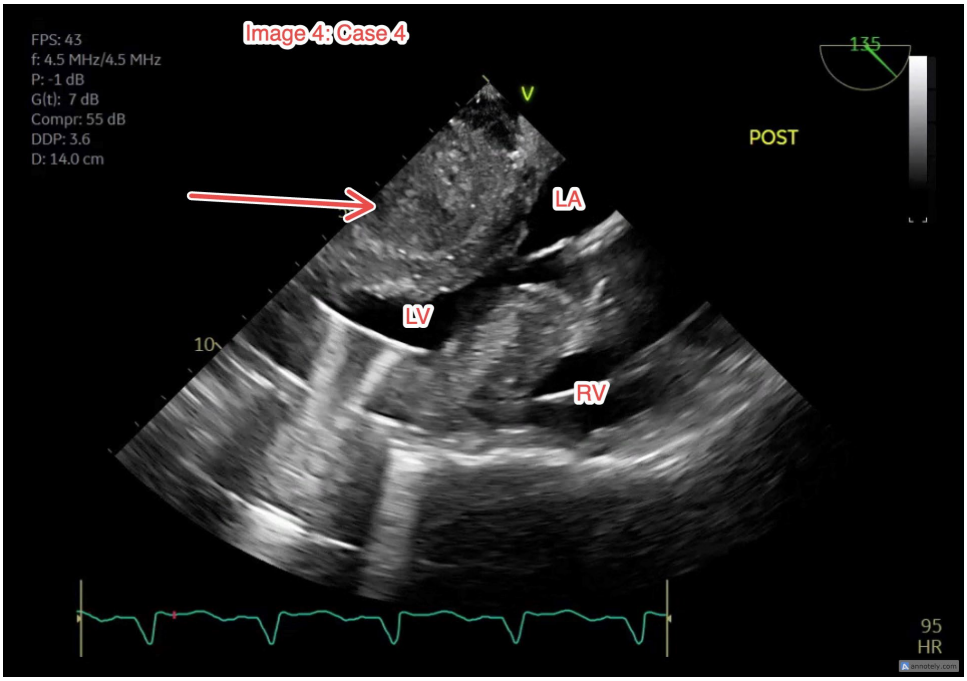


Image 4: Case 4- Arrow indicating hematoma causing LA/LV compression.

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#FR 1 – Kiosk 13 – Poster 5

Management of cardiac catheterization complication cardiac tamponade in a patient with hypertrophic obstructive cardiomyopathy

Presenting Author: Blaire Lutes, D.O., Anesthesiology Resident, University of North Carolina Medical Center

Introduction

Hypertrophic obstructive cardiomyopathy (HOCM) in patients undergoing cardiac surgery requires special consideration in anesthetic management. Left ventricular outflow tract (LVOT) obstruction from asymmetric septal hypertrophy poses significant risk of cardiovascular collapse with the various hemodynamic changes that can occur during anesthesia.[2] This case highlights the management of a patient with newly diagnosed HOCM who developed cardiac tamponade following accidental pericardial puncture during cardiac catheterization.

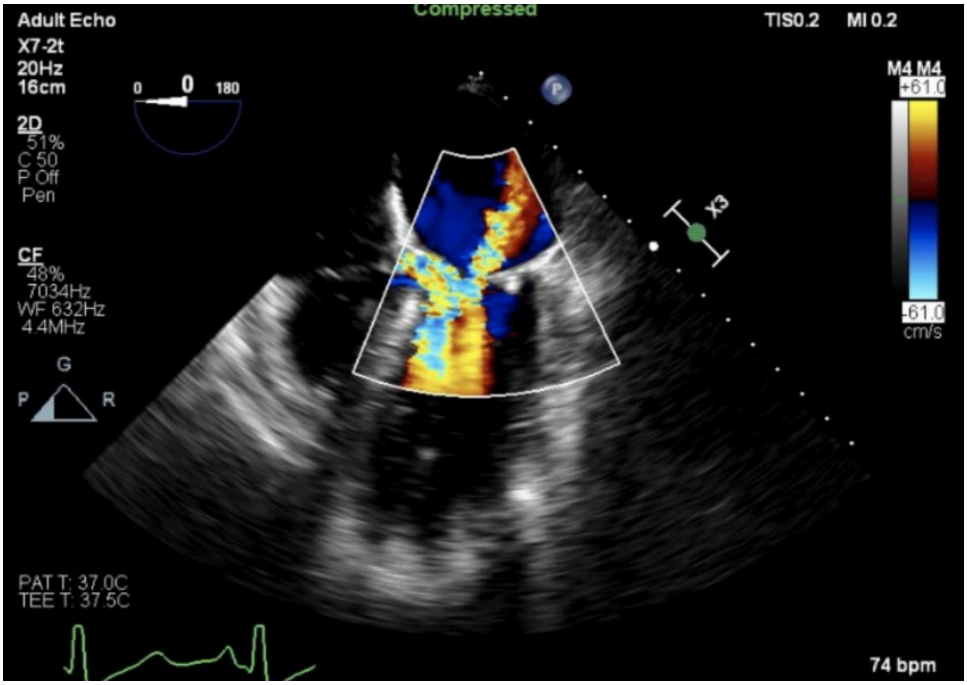
Case Presentation

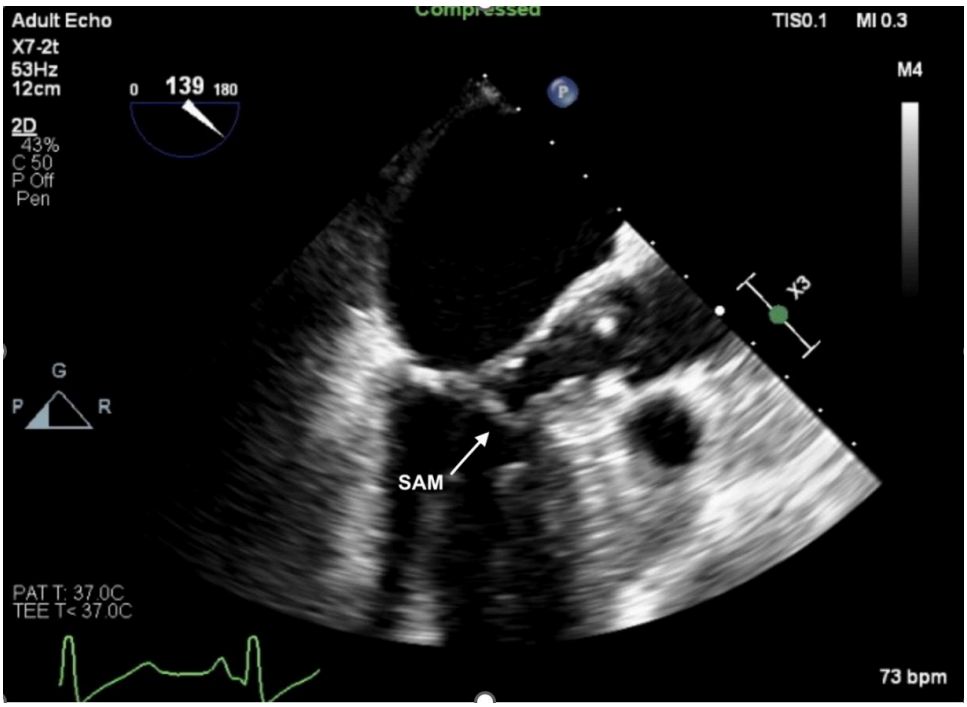
75 year-old female with history of hypertension and coronary artery disease with prior stent was found to have new aortic stenosis as well as imaging concerning for severe HOCM. She presented for left heart catheterization to further evaluate her left ventricular outflow tract (LVOT). At the end of the procedure, the patient developed acute chest pain and hypotension, with Point-of-Care ultrasound showing a large pericardial effusion with tamponade physiology. Her symptoms initially improved following urgent pericardial drain placement, however repeat angiography showed persistent extravasation into the pericardial space. Cardiothoracic anesthesiology and surgery emergently presented to the catheterization lab. The patient was transported to the cardiac operating room in preparation for sternotomy. Anesthesia was carefully induced with maintenance of spontaneous respirations, utilizing phenylephrine and esmolol boluses to prevent tachycardia. Preload was maintained with liberal fluid boluses and transfusion of blood products. Phenylephrine was used as the first-line vasopressor to maintain after load and keep heart rate low. Following intubation, rocuronium was given once patient stability was confirmed. Intraoperative transesophageal echo (TEE) showed classic HOCM characteristics with basal septal hypertrophy, systolic anterior motion of mitral valve with posteriorly-directed mitral regurgitation jet, and an elevated LVOT gradient. Exploratory sternotomy was performed off bypass pump with identification and repair of the left ventricular wall injury, leading to eventual stabilization of patient hemodynamics. The patient remained intubated at the end of the case and was brought to the ICU.

Discussion

This case highlights the need for emergent intervention following a complication from cardiac catheterization, as well as the unique hemodynamics management of a patient with HOCM during off-pump cardiac surgery. Myocardial perforation is a known risk of heart catheterization and requires urgent diagnosis and intervention. Cardiac tamponade in patients with HOCM poses a unique challenge, as the hemodynamics goals for each contract one another. While a slow to normal heart rate is preferred in HOCM, tachycardia helps maintain adequate cardiac output in tamponade, preventing ventricular collapse.[1] In this case, the anesthesia team opted to decrease heart rate and maintain after load to relieve the LVOT obstruction as the pericardial effusion was continuously drained and auto-transfused back to the patient. The reduced ventricular compliance seen in HOCM may lower the threshold for cardiovascular collapse in the setting of pericardial effusion, as seen here.

Images





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#FR 1 – Kiosk 13 – Poster 6

Thoraflex Graft for Redo Sternotomy Complex Aortic Arch Dissection: Anesthetic Perspective

Presenting Author: Ramon Jauregui, MD, Anesthesia Resident, University of Arizona

Introduction

Aortic dissection, first described over 200 years ago, continues to present major challenges in the medical field. Advancements in cardiopulmonary bypass and modern imaging have progressively improved outcomes, and newer technologies such as the Thoraflex hybrid graft offer further potential benefit in treating complex aortic arch and descending aortic pathology.

Case Presentation

The patient was a male in his 50s with a history of ascending aortic dissection status post proximal ascending aorta repair seven years prior to presentation, as well as polysubstance abuse. He presented with acute-onset chest pain. Imaging demonstrated a Stanford type A dissection originating distal to the prior graft and extending into the infrarenal abdominal aorta, the right common iliac artery, and posteriorly into the right internal iliac artery. The decision was made to proceed with surgical repair using the Terumo Thoraflex hybrid graft. Key anesthetic challenges in this case included neuroprotection, management of rapid hemodynamic changes, and transesophageal echocardiography (TEE) guidance. Neuroprotection was provided with antegrade cerebral perfusion and monitored using cerebral oximetry. Marked hemodynamic variability occurred during multiple phases including cardiopulmonary bypass and brain-only perfusion during circulatory arrest requiring vasopressor support to limit hypoperfusion and reduce the risk of spinal cord ischemia. TEE guidance was especially important and technically challenging given the patient's prior aortic repair, and it played a critical role in confirming true-lumen cannulation and appropriate stent deployment. Anesthetic management proceeded without major adverse events, and the patient was transported to the ICU in stable condition. He was successfully extubated on postoperative day seven and discharged home, ambulating on room air.

Discussion

Traditionally, arch and descending aortic pathology have been treated by combining commercially available arch grafts with separate endovascular prostheses. The Thoraflex hybrid graft, FDA-approved in 2022, integrates an arch graft and descending aortic stent into a single device that can be implanted simultaneously. By eliminating the need for an additional anastomosis, this approach has been associated with reduced aortic cross-clamp and circulatory arrest times.[1] It also supports improved aortic remodeling and provides a stable landing zone for future endovascular interventions, particularly important in dissections extending into the distal descending aorta, as seen in this patient.[2] Furthermore, the device may help reduce graft migration and Type Ia endoleak[3]. While offering several clear advantages, this hybrid technique maintains comparable rates of operative mortality, stroke, and paralysis to conventional total arch replacement with a non-hybrid frozen elephant trunk.

Images



Stanford type A aortic dissection involving the aortic root with extension into the infrarenal abdominal aorta

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#FR 1 – Kiosk 13 – Poster 7

Primary Graft Failure After Bilateral Lung Transplant in Setting of Pulmonary Vein Stenosis and Pulmonary Hypertension

Presenting Author: Anthony Boateng, MD, Cardiac Anesthesia Fellow, University of Florida

Introduction

Primary graft failure occurs in 15-25% of lung transplants, characterized by acute lung injury that occurs within the first 72 hours, presenting with pulmonary edema and hypoxemia. Given its high prevalence, supportive therapy remains the standard of practice. Because the innate immune system plays a pivotal role in graft failure, routine immunosuppression is recommended. Proposed major risk factors for primary graft failure include excessive use of vasoactive drugs, fluids overload, and elevated peak flow velocities of the anastomotic pulmonary veins. We present a patient who underwent a bilateral lung transplantation with concomitant pulmonary hypertension and demonstrated concerning pulmonary venous peak velocities, resulting in need for urgent VV ECMO implantation within hours of ICU arrival.

Case Presentation

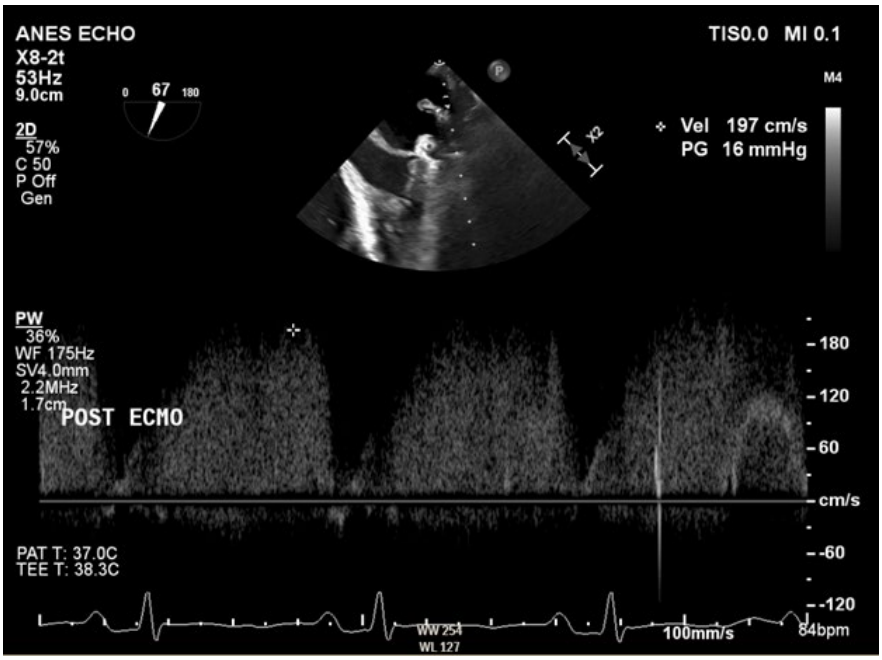
A 71-year-old female with a history of tobacco use, pulmonary fibrosis and pulmonary hypertension presented for bilateral orthotopic lung transplantation. On the day of surgery, she required high flow nasal cannula and inhaled nitric oxide. She underwent intubation with double lumen endotracheal tube, placement of a left brachial arterial line and a left internal jugular central venous catheter. The immunosuppressants were administered according to transplantation protocol. Under one lung ventilation and central VA ECMO support, the right lung transplantation was completed, followed by the left lung transplantation. Patient was weaned from ECMO after 272 minutes. Postoperative TEE exam demonstrated preserved LV systolic function and unchanged RV dilation with depressed systolic function. Peak velocities of the anastomotic sites of the left and right upper pulmonary veins was 197cm/s and 97.5cm/s, respectively. She received 5 units of packed red blood cells, 1.4L of crystalloid and 1.5L of 5% albumin. She was successfully transferred to the ICU intubated with propofol and norepinephrine infusion. Over the following 14 hours, she developed hypotension and hypoxemia which was refractory to inotropic support and escalating mechanical ventilation, ultimately requiring VV ECMO cannulation for grade 3 primary graft dysfunction. Her hospital course required further management of her RV failure and tracheostomy placement. She was decannulated from ECMO 6 days later.

Discussion

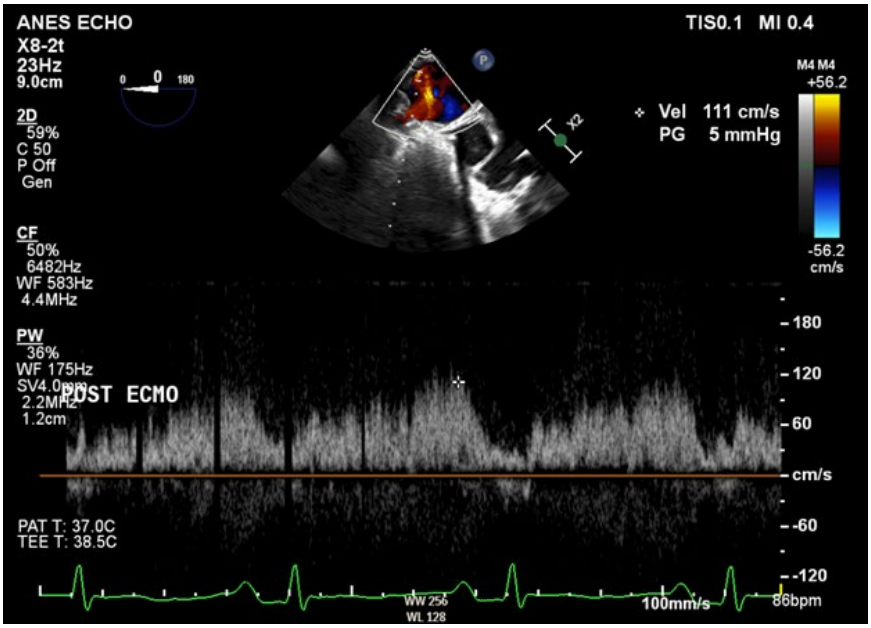
The increased risk of graft failure in our patient included female gender, pulmonary hypertension, and post anastomotic elevation of pulmonary vein peak systolic flow velocities. It is generally accepted that pulmonary venous peak systolic velocity should not exceed 100cm/s. When elevated velocities are present, they may indicate an increased risk of graft failure. However, these findings can be confounded by rapid pulmonary blood flow into the compliant donor lungs in the presence pulmonary hypertension. Moreover, pulmonary hypertension can amplify abrupt change in pressure and flow creating mechanical stress on the

donor endothelium. Combined with the ongoing inflammatory process, these factors further exacerbate the risk of reperfusion injury and graft dysfunction. This case highlights the importance of anesthesiologists understanding this mechanism of reperfusion injury and developing strategic perioperative plans to mitigate potential complications and associated morbidity.

Images



Left pulmonary vein spectral doppler



Right pulmonary vein spectral doppler

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#FR 1 – Kiosk 14 – Poster 1

A Case of Extreme Heparin Resistance in a Patient with a Left Atrial Mass

Presenting Author: Tanya E. Sorensen, Anesthesiology Resident, University of Virginia

Introduction

Unfractionated heparin (UFH) is a mixture of variably sized negatively charged polymers used for anticoagulation for surgeries requiring cardiopulmonary bypass (CPB). UFH exerts its effect by binding to antithrombin (AT) which binds to thrombin and factor Xa leading to inhibition of clot formation. The dose of UFH ranges from 300-400 IU/kg with a goal activated clotting time (ACT) of 400-480 seconds prior to going on CPB. However, some patients may be resistant to UFH and require additional measures to achieve the goal ACT.

Case Presentation

A 46-year-old, 90 kg woman was scheduled for an excision of a left atrial mass and left atrial appendage ligation via minimally invasive approach (right thoracotomy). Her past medical history included a right MCA stroke, spontaneous renal vein thrombosis, and isolated FVIII elevation (maximum value 340%) for which she was taking Eliquis. Before her surgery her CRP was elevated at 5 mg/dL (normal < 0.5 mg/dL). Her sedimentation rate was also elevated at 70 mm/h (normal < 30 mm/h). Other hypercoagulability workup including Protein C, Protein S, antiphospholipid antibody syndrome, Factor V Leiden, and prothrombin gene mutation were all negative. Baseline platelet count was 489 x 10 9 /L. Hemosonics Quantra was performed just after induction and CS was 47.1 hPa, FCS was 6.7 hPa, and PCS was 40.4 hPa suggesting hypercoagulability. Prior to heparinization for CPB the baseline ACT was 97 seconds. An initial UFH bolus of 29,000 IU was given and the ACT climbed to 368 seconds. Given that this was below the target ACT, an additional 10,000 IU was administered and the repeat ACT was 368 seconds. At this time 1,130 IU of AT concentrate and another 20,000 IU of UFH were given and the subsequent ACT was 350 seconds. In order to obtain our institution's target ACT of >480 seconds an additional 1,668 IU of AT and 25,000 IU of UFH was given. The total UFH dose was 822 IU per kg and total AT dose was 31 IU per kg. After all UFH and AT were given the ACT finally climbed to 834 seconds and CPB proceeded uneventfully. Heparin was reversed after CPB with 450 mg of protamine and the post-protamine ACT was 118 seconds. Chest tube output in the first 24 hours after surgery was 605 mL and the patient was not transfused during or after surgery. The patient had no thrombotic complications during the perioperative period.

Discussion

The etiology of UFH resistance is often multifactorial. Resistance may be attributed to AT deficiency (acquired or congenital), binding negatively charged UFH molecules to positively charged proteins (e.g. platelet factor 4, fibrinogen, endothelial cells, etc.), elevated prothrombotic molecules (factor VIII, etc.), or hypovolemia. This patient demonstrated extreme heparin resistance which may be attributable to increased factor VIII. Heparin resistance is often overcome with additional boluses of UFH to reach the desired ACT, although this is not without risk as the patient can become re-heparinized after reversal with protamine due

to the high reservoir of bound UFH. Antithrombin concentrate is a safe option to treat heparin resistance due to low AT. The dose required is generally 500-1000 IU. If AT concentrate is not available, fresh frozen plasma may be used but the volume required may be prohibitive (1 mL FFP provides 1 IU AT).

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#FR 1 – Kiosk 14 – Poster 2

Severe Peripheral Artery Disease with Two Types of Mitral Regurgitation: Interpreting Monitors and Balancing Management

Presenting Author: Justin Fleischer, DO, Resident Physician, Dartmouth-Hitchcock Medical Center

Introduction

We describe the procedural & hemodynamic complexities involved in management of a patient with severe mitral regurgitation in conjunction with severe peripheral artery disease (PAD) undergoing spleno-renal bypass procedure.

Case Presentation

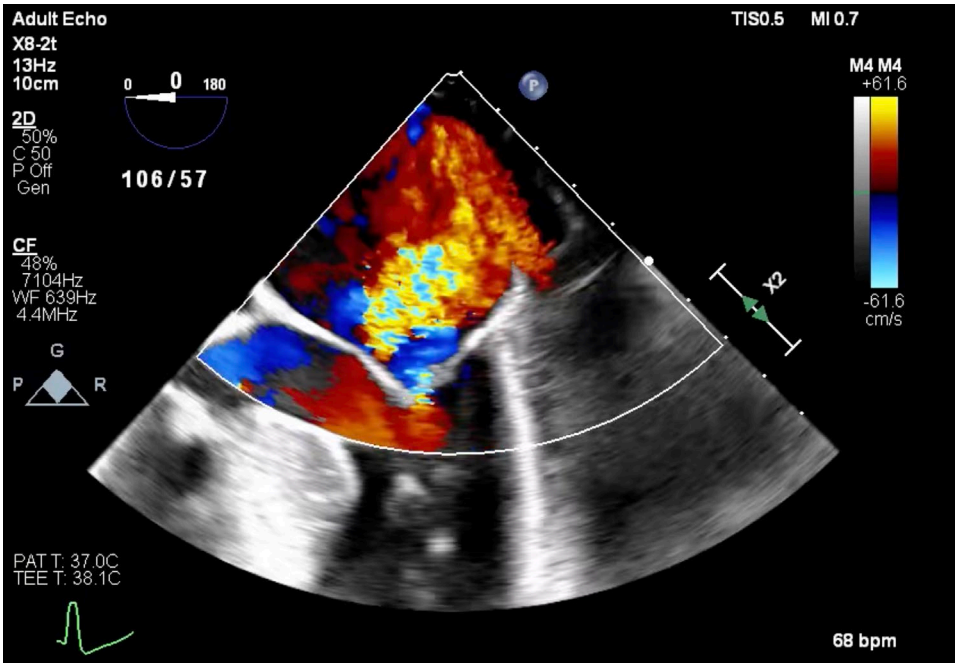
Patient is a 62-year-old female with a history of severe mitral regurgitation, heart failure with preserved ejection fraction, severe PAD including near total occlusion of infra-renal aorta and bilateral brachiocephalic atherosclerotic disease (total occlusion of right), anemia, stage IV CKD, COPD & T2DM. She was initially admitted for recurrent flash pulmonary edema & evaluation of her infrarenal calcific aortic disease for potential surgical intervention. She was admitted to the cardiology service for optimization. Despite this, she had multiple episodes of flash pulmonary edema triggered by severe hypertension, requiring positive pressure ventilation. Cardiac catheterization showed significant discrepancies between peripheral and central pressures: left radial artery BP 78/48 & aortic pressure 206/75. She arrived to the OR on BIPAP. Given her vasculature, we placed a preinduction left radial arterial line, knowing that the central SBP could be approximately 120 greater. The patient was intubated in an upright position to prevent an acute increase in venous return. Following intubation, we placed a central venous catheter with a PA catheter to monitor filling pressures. We slowly laid the patient flat without any significant hemodynamic changes and were able to de-escalate the Fio2 while maintaining PEEP. Our hemodynamic goals were to maintain euvolemia & balance of perfusion to organs distal to the stenosis (using MAP from the arterial line, lactate & urine output) but also prevent pulmonary edema from high afterload (using PAOP & PAD). Milrinone was started to promote forward flow, lusitropy given the grade 3 diastolic dysfunction & maintain higher HR. After the bypass was completed, heparin was reversed with a slow bolus of protamine & despite matching blood loss with PRBC transfusion, the patient's blood pressure was dropping with an increase in PAPs despite escalating doses of pressor. The surgery team was informed of the situation & were able to palpate brisk aortic pressure and femoral pulses. Additionally, there was no decrease in end-tidal CO2 throughout this hypotensive period, making global hypoperfusion less likely. Given an unclear clinical picture, a TEE probe was then placed. TEE showed both ventricles being volume down with systolic anterior motion of the mitral valve causing severe MR. Given these findings, the patient was resuscitated and the milrinone was stopped. We were able to de-escalate pressors without escalating ventilator settings & transported the patient to the cardiac care unit.

Discussion

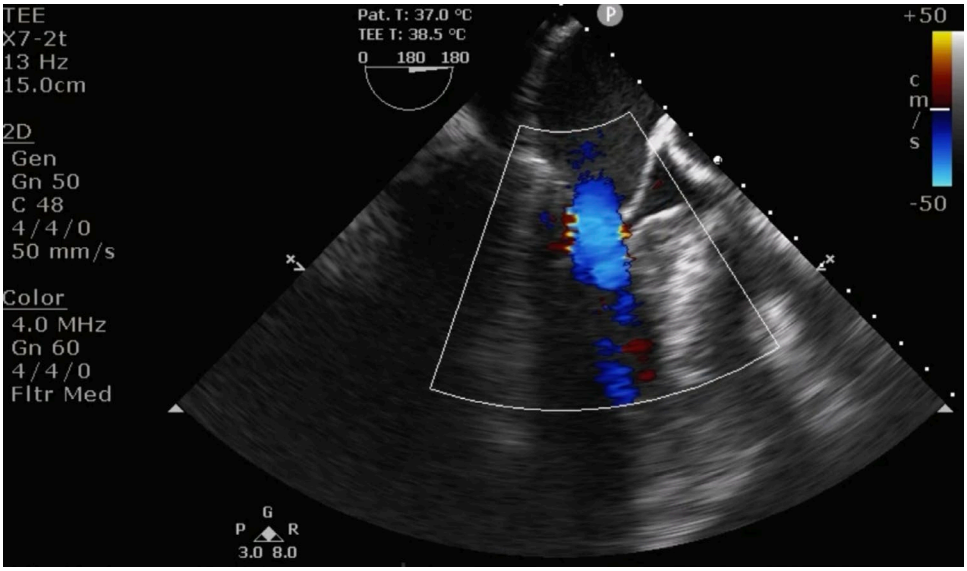
Patients with severe vascular disease are among the sickest we get to take care of as anesthesiologists. The unreliability of peripheral blood pressure monitoring from diffuse arterial disease make their care even more

challenging. The final curve ball was developing mitral regurgitation from SAM, which is managed completely different from “classic MR”. This case stresses the importance of knowing how to correctly interpret your monitors, their pitfalls and when to escalate when things are not correlating clinically.

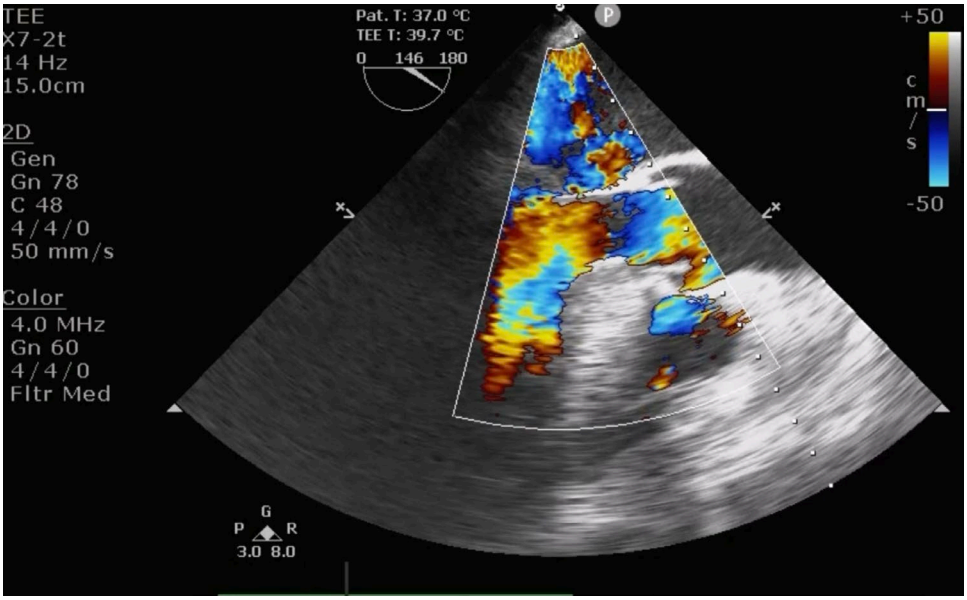
Images



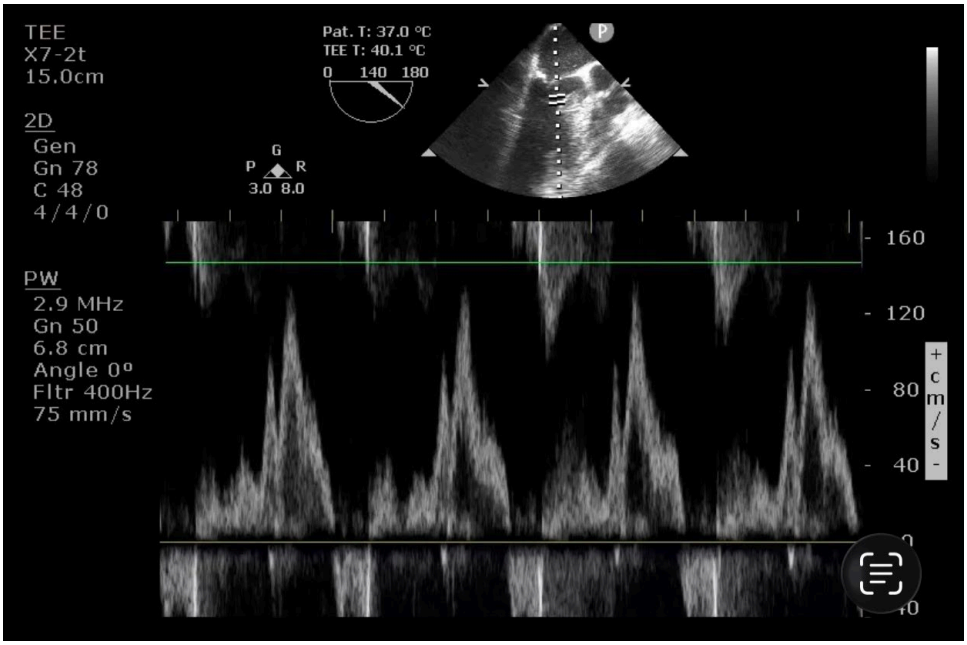
Patient's baseline severe MR



Mitral leaflet obstructing LVOT



LVOT flow during systole



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#FR 1 – Kiosk 14 – Poster 3

Acute Right Ventricular Dysfunction from Intracardiac Thrombosis and Disseminated Intravascular Coagulation During Liver Transplantation

Presenting Author: Andrew Hopkins, MD, Resident Physician, Ohio State University Medical Center

Introduction

Acute right ventricular (RV) dysfunction during orthotopic liver transplantation (OLT) is an uncommon but well recognized complication, usually occurring with donor liver reperfusion. Intracardiac thrombosis (ICT) is a rare cause of RV failure and is commonly associated with disseminated intravascular coagulation (DIC). We present a case of pre-anhepatic RV failure from ICT and DIC.

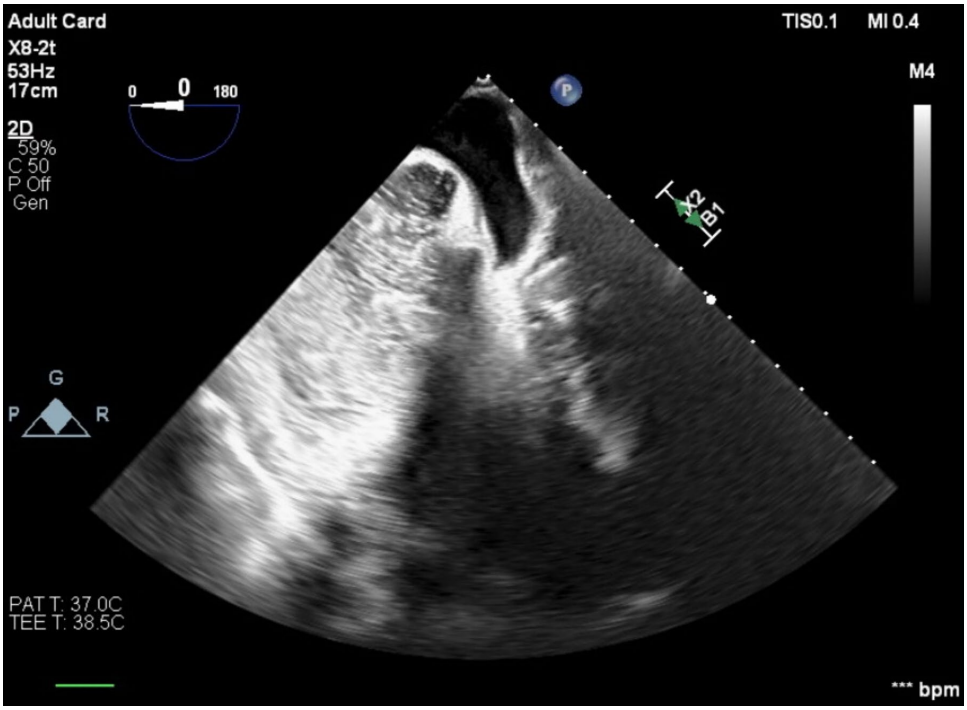
Case Presentation

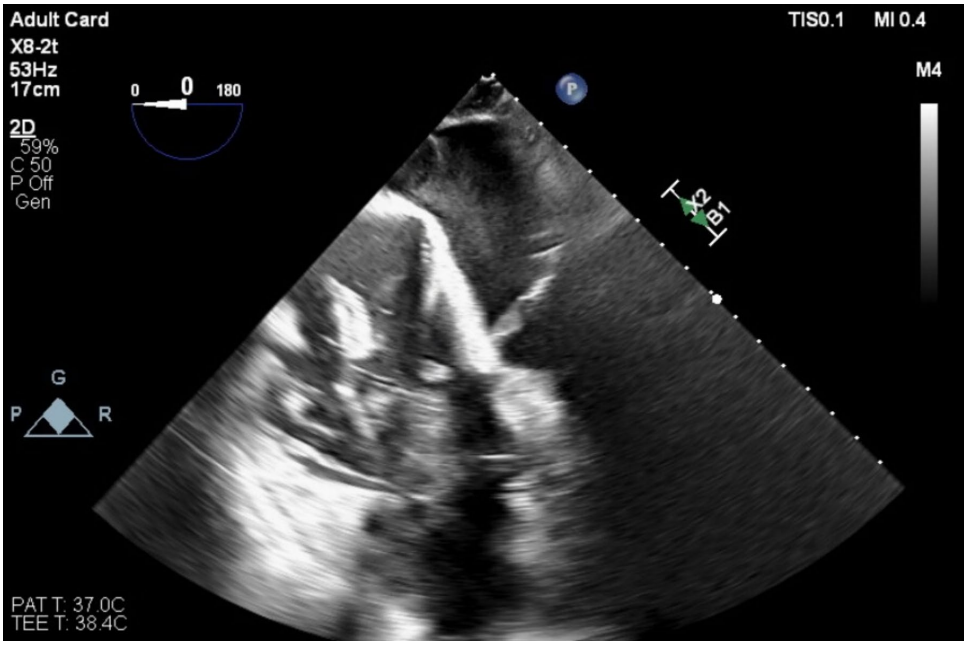
A 70-year-old male with cryptogenic cirrhosis (MELD 14), ascites, hepatic encephalopathy and coronary artery disease with multiple stents, including a recent RCA stent, for DCD OLT. Preoperative evaluation showed normal biventricular size and function and normal pulmonary hemodynamics (PAP 31/11 [19] mmHg; PVR 1.16 WU). Induction, intubation, and invasive monitor placement were uneventful; initial TEE confirmed normal biventricular function. During mobilization of the liver from the vena cava, the patient became profoundly hypotensive—TEE showed large thrombus in the right atrium, right ventricle, and pulmonary trunk. Epinephrin and vasopressin boluses, as well as 10,000 units of heparin, were given. Systemic pressures improved, and the thrombus lysed over the following 10 minutes; however, PA pressures rose from 29/24 (28) to 64/37 (49) and CVP rose from 14 to 33. Post-lysis TEE revealed severe RV dysfunction, so epinephrine infusion and inhaled epoprostenol were added. The ROTEM immediately following clot dissolution was consistent with DIC. Aminocaproic acid, fibrinogen concentrate, and prothrombin complex concentrate were given. After reperfusion, RV dysfunction continued to improved and epoprostenol was discontinued upon completion of the case. The abdomen was temporarily closed, and the patient was transferred to the ICU. He returned to the OR on POD 1 for closure, which he tolerated well. Follow-up TTE demonstrated low-normal RV systolic function. The remainder of his hospitalization was uneventful; he was discharged on POD 14.

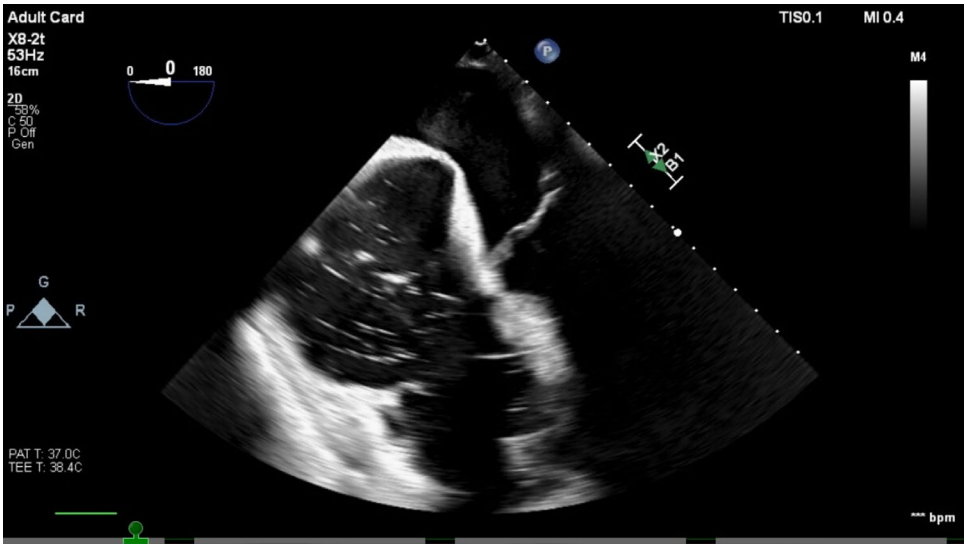
Discussion

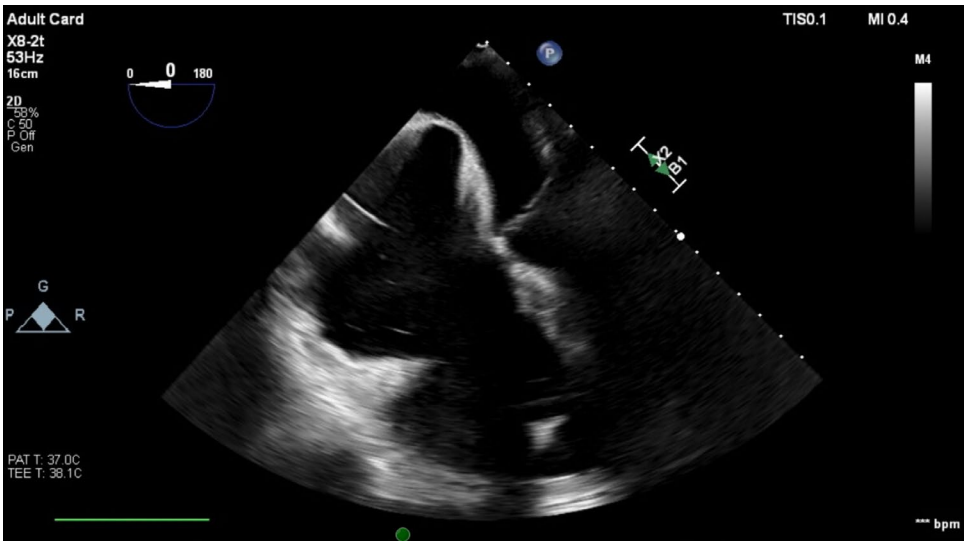
Severe RV dysfunction is rare during OLT and usually occurs during the anhepatic or reperfusion phases. Patients with a high MELD score or pulmonary hypertension are more likely to be affected. This case is notable for pre-anhepatic RV dysfunction related to ICT with subsequent DIC which required massive transfusion. TEE monitoring was crucial in the rapid diagnosis and management of this patient, which can be challenging with competing priorities. Treatment for DIC may include large volume transfusion, even when guided by viscoelastic testing, while the management of RV dysfunction Requires volume restriction and hemodynamic support.

Images


Adult Card
X8-2t
53Hz
17cm
2D
59%
C 50
P Off
Gen
TIS0.1
MI 0.4
M4
PAT T: 37.0C
TEE T: 38.5C
*** bpm


Adult Card
X8-2t
53Hz
17cm
2D
59%
C 50
P Off
Gen
TIS0.1
MI 0.4
M4
PAT T: 37.0C
TEE T: 38.4C





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#FR 1 – Kiosk 14 – Poster 4

Percutaneous Mechanical Aspiration of an Unknown Intracardiac Mass – Consequences and Considerations

Presenting Author: Daniel Mattimore, MD, Adult Cardiothoracic Anesthesiology Fellow, Duke University Hospital

Introduction

Percutaneous mechanical aspiration (PMA) devices are minimally invasive suction catheters designed for debulking intracardiac masses, including vegetations and thrombi. For patients who are not surgical candidates, there are three general indications of PMA: bridge to surgical decision, adjunctive management, or destination therapy.[1] From 2003 to 2017, hospitalizations for cardiovascular implantable electronic device -associated infective endocarditis (CIED-IE) have increased five-fold, a trend driven by an aging population and a growing number of implanted devices.[2] Correspondingly, the use of PMA has risen due to the higher incidence of CIED lead masses and endocarditis. Here we present a case of PMA of an intracardiac mass resulting in acute right ventricular (RV) failure and later death.

Case Presentation

A 62-year-old female with history cardiomyopathy atrial septal defect (ASD) status post internal cardiac defibrillator (ICD) and interatrial occlusion device placement presented with cough and progressive shortness of breath. Workup was significant for leukocytosis, subsegmental pulmonary emboli, and a 2.2 cm mass attached to the ICD lead and interatrial occlusion device in the right atrium and normal RV function with trace tricuspid regurgitation. Blood cultures and infectious PCRs were notably negative. She was deemed not a surgical candidate due to the unknown etiology of the mass. She underwent PMA aspiration of the thrombus with a Penumbra Lightning 12 system under general anesthesia. Approximately 65% of the mass was removed but the procedure was complicated acute RV failure concerning for embolization of mass and PEA arrest with ROSC after 3 minutes for which the patient was placed on VA-ECMO. Pathology results from the aspirated material revealed Aspergillosis as the causative organism. The patient transitioned to comfort care during her postoperative course due to multisystem organ failure.

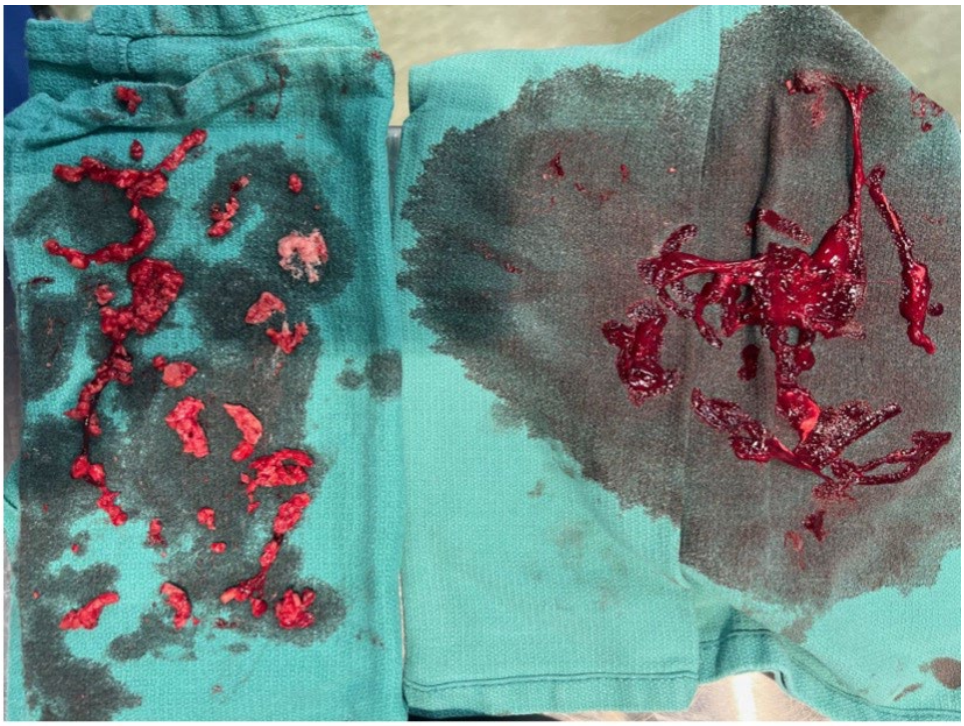
Discussion

There are several key learning points in this case deserving of attention. First, it was unknown whether this patient's intracardiac mass was thrombus or IE prior to PMA intervention. Establishing a diagnosis would stratify risk-benefit discussions for invasive procedures as fungal endocarditis carries a mortality rate of over 50%.[3] Here, multidisciplinary communication between anesthesia, surgery and cardiology may have yielded a more conservative management strategy such as performing a biopsy of the mass first to establish a formal diagnosis before attempting PMA or other intervention. Second, anesthesiologists must be familiar with the anesthetic considerations for PMA procedures, particularly given the many different devices available. These risks include fluid shifts, acute RV failure, malignant arrythmias, and worsening sepsis. This is especially critical since many of these procedures are performed outside of main operating room areas, where access to mechanical circulatory support is often limited.

Images



Midesophageal RV inflow-outflow image depicting right atrial thrombus adherent to a CIED lead



Aspirated material from procedure. Roughly 60% of the mass was aspirated.

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#FR 1 – Kiosk 14 – Poster 5

Use of a Novel Open-Chest Management System as a Bridge to Hemodynamic Recovery Following Heart Transplantation

Presenting Author: Adriana McMichael, MD, ACTA Fellow, Duke University - School of Medicine

Introduction

Open-chest management after orthotopic heart transplantation (OHT) may be required for cases of severe graft edema, right ventricular (RV) failure, hemodynamic instability, or coagulopathy (1). The main challenge is optimizing ventricular filling and intrathoracic compliance while protecting the graft from compression and contamination. Modern systems, such as the BlueBridge open-chest management system (OCMS), provide controlled decompression and maintain sterility, offering a physiologic alternative to delayed sternal closure with packing (2). This case highlights the device's role as a bridge to recovery in a patient with an oversized donor graft who developed early post-transplant low cardiac output syndrome.

Case Presentation

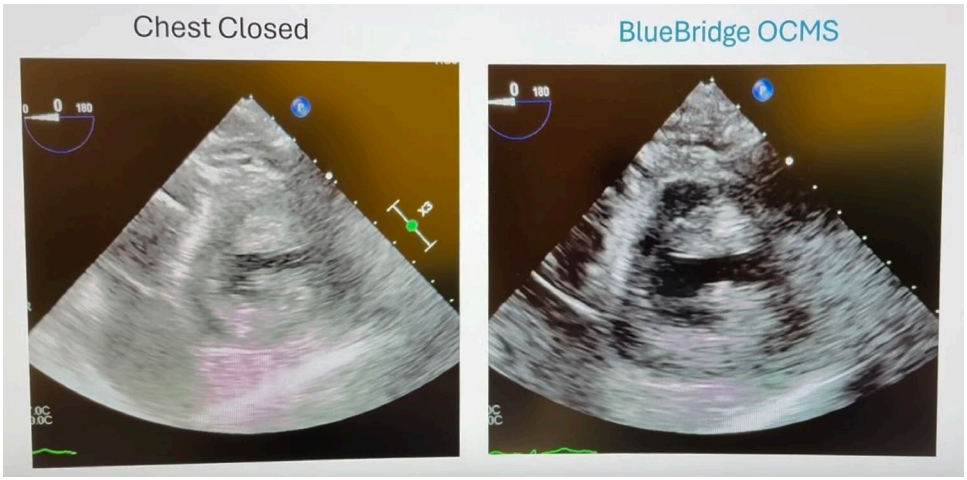
A 33-year-old male with end-stage hypertrophic obstructive cardiomyopathy and prior embolic right coronary infarction underwent OHT. Intraoperative TEE showed mild left ventricular (LV) systolic dysfunction and normal RV function. Hemodynamics were adequate but with a borderline cardiac index (>2 L/min/m^2), and the chest was closed cautiously. Within hours of ICU arrival, he developed worsening hypotension, elevated central venous pressure, hypoxemia, and severe pulmonary edema. The etiology was unclear, prompting emergent return to the operating room for exploration with possible veno-arterial ECMO cannulation. Repeat TEE demonstrated a very small, hypercontractile LV and a dilated RV with systolic dysfunction: findings consistent with extrinsic LV compression and secondary RV overload. Upon reopening the chest, hemodynamics improved immediately: central venous pressure decreased, mean arterial pressure rose, and cardiac index increased. The presumed mechanism was donor-recipient size mismatch with mediastinal edema causing functional pulmonary vein stenosis. A BlueBridge OCMS was placed to maintain decompression (2). Ventricular filling and gas exchange improved promptly. After 48 hours, the patient returned to the operating room; mediastinal edema had improved, the device was removed, the chest was closed, and graft function remained stable.

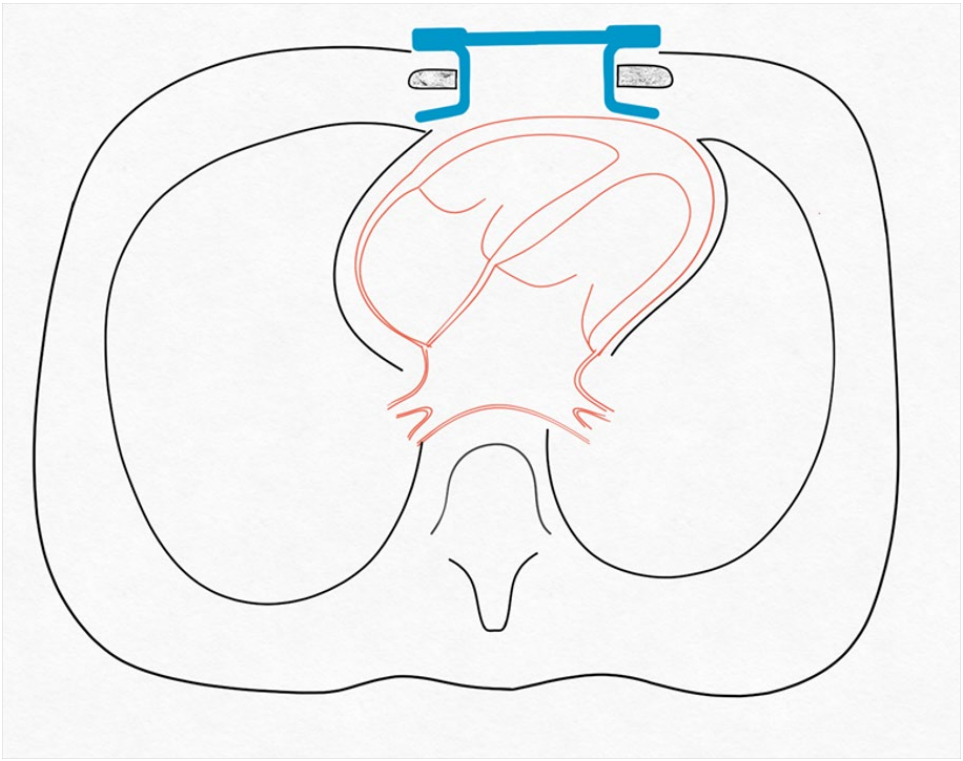
Discussion

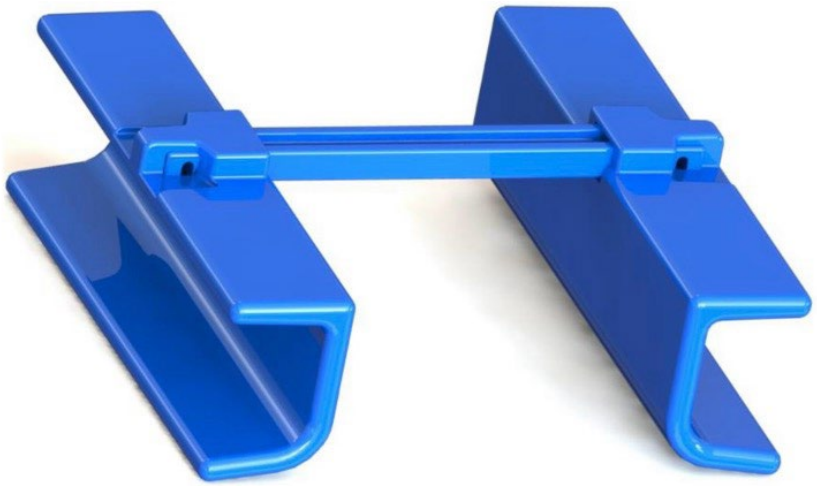
This case demonstrates a reversible episode of early post-transplant cardiogenic shock resulting from mechanical constraint rather than intrinsic graft dysfunction. The TEE pattern of a hyperdynamic, under-filled LV with RV dilation identified extrinsic compression with presumed functional pulmonary vein stenosis as the cause. Recognition of this physiology allowed a straightforward mechanical correction. Traditional open-chest techniques using packing may limit uniform decompression and sterility. The BlueBridge OCMS provides stable sternal separation, uniform pressure relief, and protection of the sternum and intrathoracic organs, while maintaining adequate intrathoracic volume (2). Its use exemplifies structured open-chest management to restore hemodynamic stability in cases of donor-recipient size mismatch or significant

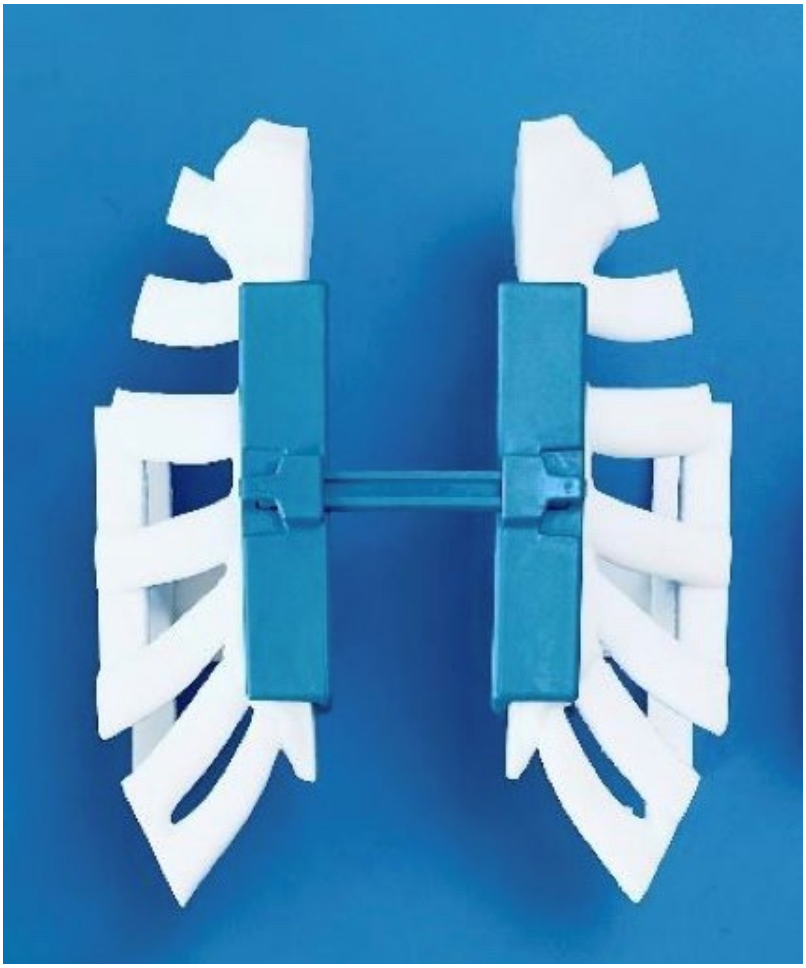
mediastinal edema (3). Early echocardiographic assessment and multidisciplinary management are essential for timely recognition and management of reversible hemodynamic compromise. OCMSs can be lifesaving in complex OHT recipients, serving as a temporary bridge until thoracic compliance and hemodynamics normalize.

Images









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#FR 1 – Kiosk 14 – Poster 6

Myocardial Protection with a Patent Internal Mammary Artery: A Tale of Refractory Ventricular Tachycardia and Cardiogenic Shock

Presenting Author: Monica Nagalla, MD, Resident Physician, Northwestern Feinberg School of Medicine

Introduction

Myocardial protection during cardiopulmonary bypass (CPB) is essential in maintaining heart muscle integrity during cardiac surgery. In patients with a patent internal mammary artery (IMA) to LAD graft, and undergoing a procedure where cardiac arrest with an aortic cross clamp is needed, myocardial protection is more challenging. Techniques that have been successful include clamping of the IMA, leaving the IMA open with moderate hypothermia, and deep hypothermic circulatory arrest[1]. Despite use of any of these measures, myocardial damage can still occur to varying degrees. Consequences of inadequate myocardial protection include refractory ventricular tachycardia (VT) and cardiogenic shock, and efforts must be made to identify and address all contributing factors.

Case Presentation

A 68-year-old male with history of CAD, MI complicated by cardiogenic shock requiring an intra-aortic balloon pump and CABGx2 4 years prior, HTN, pAfib, HFpEF with LVEF 55-60%, COPD, OSA, and ascending aneurysm presented for a redo sternotomy, ascending aorta and hemiarch replacement, and CABGx3. The patient was successfully placed on cardiopulmonary bypass. After aortic cross clamp, administration of cardioplegia, and moderate hypothermia to 24°C, electrical activity was drastically reduced, however total electrical quiescence was not achieved. At this time, bleeding was noted from IMA and was controlled. On removal of the aortic cross clamp the patient developed refractory VT despite optimal medical management. An unrecognized RCA injury was then discovered, and SV grafts were used to bypass the RCA and acute marginal artery. VT persisted and the decision was made to perform SVG bypass to IMA/LAD without resolution of VT. The patient was transitioned to central VA ECMO and taken to the ICU. VT resolved in the ICU but the patient required extensive transfusion, had a massive pulmonary embolism, and the decision was made to withdraw care.

Discussion

Patients with inadequate myocardial protection during open heart surgery have been found to require increased intraoperative inotropic support, mechanical support, and other assist devices post operatively[2]. Additionally, inadequate myocardial protection increases the risk of arrhythmias, secondary to ischemia-reperfusion injuries and direct injury to the conduction system. Importantly, persistent arrhythmia due to inadequate cardiac protection may not immediately resolve with restoration of flow and may even get worse. In severe cases, additional measures such as temporary pacing wires, and mechanical circulatory support may also be needed[3]. During our case, multiple confounding variables led to inadequate cardioprotection resulting in refractory VT and cardiogenic shock. The presence of the patent IMA was thought to be the reason for the inadequate cardioprotection. However, the continued electrical activity after combined

cardioplegia and hypothermia, and repair of the IMA, should have prompted the investigation into other potential causes. Delayed identification of the RCA injury likely contributed to inadequate myocardial protection during this case with a long CPB run. The refractory VT in our case was unresponsive to return of perfusion, pharmacologic intervention, defibrillation attempts, and ultimately required ECMO support.

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#FR 1 – Kiosk 14 – Poster 7

Overcoming Significant Hemodynamic Challenges During TAVR Using Intraprocedural VA-ECMO and Remimazolam

Presenting Author: Laura Furtado Pessoa de Mendonca, M.D., Resident, Mayo Clinic

Introduction

Although transcatheter aortic valve replacement (TAVR) is less invasive than surgical valve replacement, it can still provoke hemodynamic instability, especially in patients with severe multivalvular disease and cardiogenic shock. In patients requiring high-dose inotropes, TAVR may be the only feasible short-term option. Temporary mechanical circulatory support may be considered; however, severe aortic regurgitation (AR) and stenosis (AS) often preclude devices such as Impella or intra-aortic balloon pump (IABP). We present the anesthetic management of a patient with cardiogenic shock and severe AS who underwent TAVR with prophylactic intraprocedural veno-arterial extracorporeal membrane oxygenation (VA-ECMO) using high-flow nasal cannula (HFNC) and remimazolam.

Case Presentation

A 62-year-old male with coronary artery disease, type 2 diabetes, hypertension, hyperlipidemia, and end-stage renal disease on peritoneal dialysis presented with progressive dyspnea. Point-of-care ultrasound revealed apical ballooning of the left ventricle; NT-Pro BNP was >70,000 pg/mL and troponin was elevated. He was admitted for non-ST-elevation myocardial infarction, heart catheterization showed right-dominant circulation without obstructive disease. Transthoracic echocardiogram (TTE) showed an ejection fraction (EF) of 15–20%, severe low-flow/low-gradient AS, severe AR, and moderate–severe mitral regurgitation. He rapidly progressed to cardiogenic shock with lactate 14.5 mmol/L, requiring ICU transfer with dobutamine infusion, continuous renal replacement therapy, and escalating vasopressor support (norepinephrine, epinephrine, vasopressin) to maintain mean arterial pressure > 65 mmHg. After multidisciplinary evaluation, TAVR was deemed viable. Avoiding positive-pressure ventilation was critical to prevent worsening afterload and maintaining cardiac output in a patient requiring high-dose vasopressors. Remimazolam (20mg total) was used to provide hemodynamically stable sedation while maintaining spontaneous ventilation with HFNC. The patient underwent a successful TAVR with a 26-mm Edwards Sapien valve under prophylactic intraprocedural VA- ECMO support with peripheral cannulation of both femoral arteries and veins. Vasopressors were weaned during the procedure, and he returned to the ICU on dobutamine infusion. Follow-up TTE showed improved EF to 50–55%, and well-functioning bioprosthesis. He was later weaned off dobutamine and discharged on guideline-directed medical therapy and peritoneal dialysis.

Discussion

Intraprocedural VA-ECMO combined with HFNC and remimazolam enabled safe anesthetic management in severe cardiogenic shock. The inability to use Impella or IABP made VA-ECMO necessary, despite risks of limb ischemia and bleeding. TAVR often induces abrupt hemodynamic shifts, reducing cardiac output – especially during rapid pacing and valve deployment. VA-ECMO maintained continuous systemic perfusion

throughout these critical steps. Minimal sedation with natural airway avoided significant vasodilation and cardiovascular depression associated with general anesthesia. Remimazolam's rapid onset, short half-life, and favorable cardiovascular profile, allowed stable sedation without compromising perfusion.

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#FR 1 – Kiosk 15 – Poster 1

Should an Incidentally Found Patent Foramen Ovale Be Closed During Lung Transplantation?

Presenting Author: Alexander Sumarli, MD, Cardiac Anesthesia Fellow, Cedars Sinai

Introduction

Patent foramen ovale (PFO) is present in 20-34% of adults and is usually benign, with a prevalence that decreases with age. Although commonly found incidentally during an echocardiographic study, only certain conditions necessitate the closure of the shunt, which includes significant right to left shunting and secondary prevention of cryptogenic stroke in patients with high risk PFO features and morphology. This is a case of a patient post-operative from lung transplantation, who developed massive intracardiac thrombi and paradoxical embolus across a previously unidentified PFO.

Case Presentation

A 45-year-old woman with SLE, Sjögren's disease, and ILD from hypersensitivity pneumonitis on the lung transplant list presented with acute on chronic respiratory failure in the setting of acute metapneumovirus infection and pneumothorax. Pre-op echocardiography suggested cor pulmonale with severe tricuspid regurgitation. After treatment of her acute conditions, she subsequently received an offer for bilateral lung transplantation and underwent the procedure under VA ECMO support. Intraoperative TEE, reviewed by multiple providers, showed an intact interatrial septum without PFO, and a bubble study was negative within 3–5 cardiac cycles. Her intraoperative course was uneventful; she was weaned off of VA ECMO on low dose epinephrine due to moderately reduced RV function. Her initial postoperative course was notable for AKI requiring CRRT, and inability to wean from mechanical ventilation, ultimately requiring tracheostomy. Two weeks later, she developed new-onset atrial fibrillation with rapid ventricular response during hemodialysis and new back pain. A code blue was called for hypotension and unresponsiveness. POCUS revealed intracardiac thrombi in both the left and right sides. She underwent pulmonary thrombectomy, during which a 20 cm thrombus extending from the right atrium to the pulmonary artery was removed. TEE at that time demonstrated a PFO, likely due to markedly elevated right atrial pressures. She was placed on VA ECMO. A head CT revealed a large pontine and midbrain hemorrhage. After a family meeting, she was transitioned to comfort care.

Discussion

Routine closure of PFO during lung transplantation does not demonstrate a clear benefit in reducing postoperative stroke or mortality1. Closure may be considered in cases with significant right to left shunting. Although PFO may carry a risk of paradoxical embolism, no significant differences in 90-day mortality has been found in patients with repaired versus unrepaired PFO1. Although percutaneous device closure of a PFO is considered a low risk procedure, it nevertheless carries a 4-6% risk of procedural complications and < 1% risk of new onset atrial fibrillation2. Although this case demonstrates the unlikely catastrophic outcome of a paradoxical embolus, the evidence does not support the routine closure of incidentally found PFO. The case, however, does highlight the importance of meticulous TEE evaluation of the interatrial

septum with color doppler and bubble study. Given the false negative rate of PFO detection on TEE of 10-11%3, other modalities described in the literature can also be considered if a PFO is suspected, such as contrast transcranial Doppler (TCD) or a cardiac CT.

Images



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#FR 1 – Kiosk 15 – Poster 2

Circumferential Intimal Avulsion During Transaxillary TAVI: Early Recognition is Key

Presenting Author: Ally Citro, MD, Resident Physician, Department of Anesthesiology, University of Miami / Jackson Health System

Introduction

Vascular injury, including acute aortic dissection, is a recognized complication of transcatheter aortic valve implantation (TAVI). We report a rare case of en bloc arterial intimal avulsion during transaxillary TAVI that closely mimicked an ascending aortic dissection. Early recognition is essential to guide appropriate intervention and avoid misclassification.

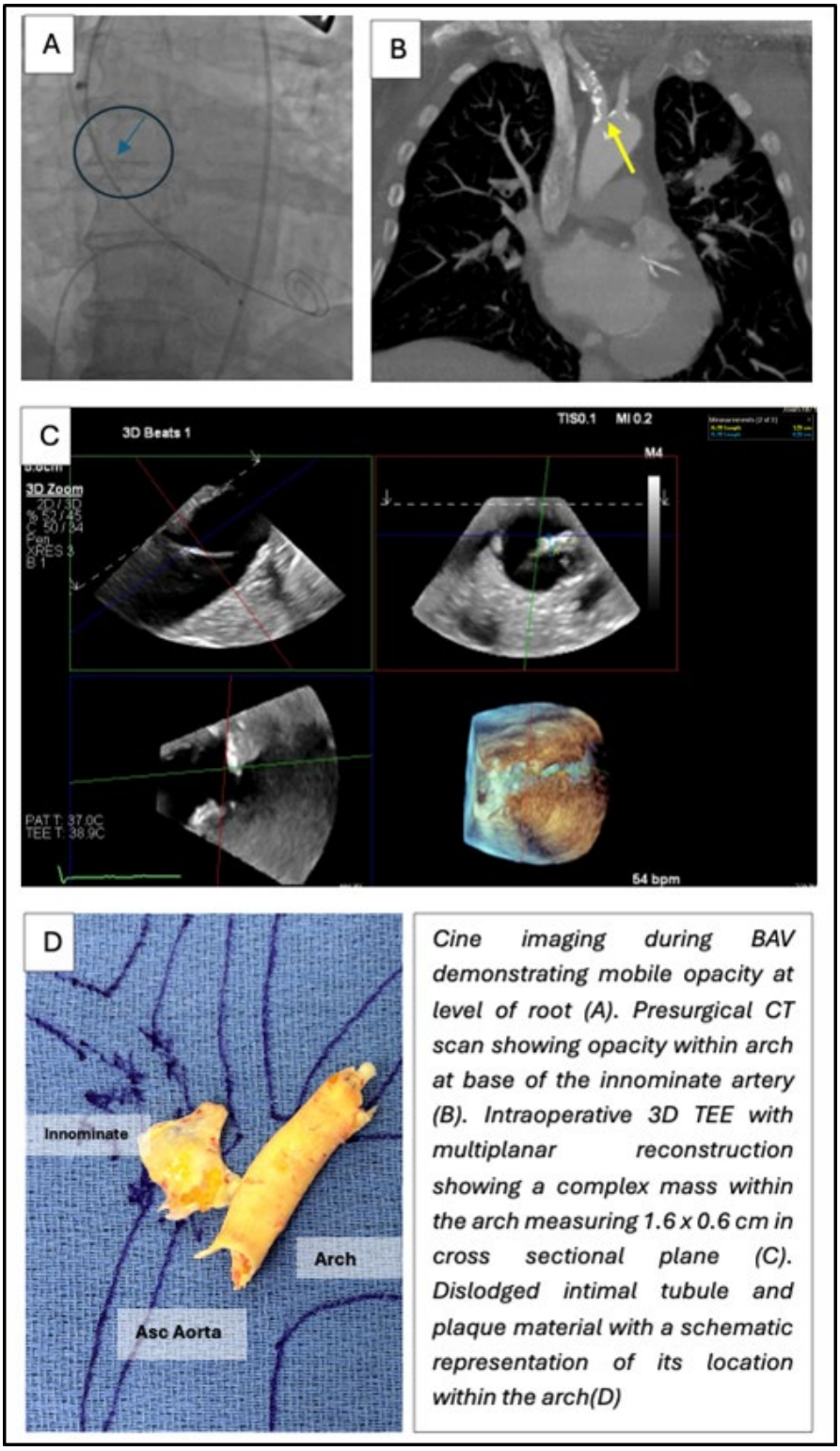
Case Presentation

A 75-year-old man with severe vascular disease and aortic stenosis presented for elective TAVI via the left axillary approach under general anesthesia. After uncomplicated placement of a 14-Fr eSheath, balloon aortic valvotomy was performed under rapid pacing. During balloon inflation, a previously unseen mobile radiopacity at the sinotubular junction (STJ) was noted, raising concern for an acute type A dissection (Figure A). A transesophageal echocardiography (TEE) probe was introduced which demonstrated a corresponding echodensity. During withdrawal of the delivery wire and sheath, the radiopacity embolized to the aortic arch and was no longer visualized at the STJ by fluoroscopy or TEE, significantly altering our diagnostic suspicion. Attenuation of the right radial arterial waveform was also noted at this stage despite stable central aortic pressure. The procedure was halted, and conversion to surgical management including aortic valve replacement was planned. Given the patient's stability and ongoing uncertainty, a CT was obtained prior to surgery. CT excluded aortic dissection and revealed elongated filling defect and plaque like material along the aortic arch from the innominate to the left subclavian artery (Figure B). Intraoperative TEE showed echogenic material (1.6 × 0.6 cm with a filamentous component) along the superior mid-arch (Figure C). After initiation of cardiopulmonary bypass (CPB) and hypothermic circulatory arrest, the aortic arch was opened, revealing a fully dislodged intimal tubular "cast" originating from the left subclavian artery. A second free-floating intimal fragment was retrieved from the base of the innominate artery (Figure D). No repair of the subclavian artery or aorta was required. A bioprosthetic aortic valve was implanted, and the patient was weaned from CPB and extubated neurologically intact on postoperative day three.

Discussion

En bloc arterial intimal avulsion is an exceedingly rare complication of large-bore access and can mimic other vascular complications. Intimal avulsion produces circumferential delamination that may detach as a tubular cast and migrate distally, requiring surgical retrieval. Awareness of this uncommon complication, and prompt recognition via multimodality imaging and clinical signs are essential, as misclassification may delay or misdirect definitive management.

Images



Cine imaging during BAV demonstrating mobile opacity at level of root (A). Presurgical CT scan showing opacity within arch at base of the innominate artery (B). Intraoperative 3D TEE with multiplanar reconstruction showing a complex mass within the arch measuring 1.6 x 0.6 cm in cross sectional plane (C). Dislodged intimal tubule and plaque material with a schematic representation of its location within the arch(D)

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#FR 1 – Kiosk 15 – Poster 3

New Dynamic LVOT Obstruction Unmasked: Postoperative Systolic Anterior Motion after Subaortic Membrane Resection

Presenting Author: Anjana M. Kumar, MD, Cardiothoracic Anesthesiology Fellow, Northwestern Medicine

Introduction

Systolic anterior motion (SAM) of the mitral valve is a dynamic phenomenon typically associated with hypertrophic obstructive cardiomyopathy, but it can also arise in other contexts where altered left ventricular outflow tract (LVOT) geometry or loading conditions exert force on the mitral valve.[1] Although subaortic membrane resection is performed to relieve fixed LVOT obstruction, changes in septal contour can paradoxically precipitate new-onset SAM. We report a case of newly developed SAM following subaortic membrane resection identified immediately on post-bypass echo, requiring septal myectomy and papillary muscle repositioning. This case highlights a rare complication of subaortic membrane resection and the utility of echocardiography to inform further surgical management.

Case Presentation

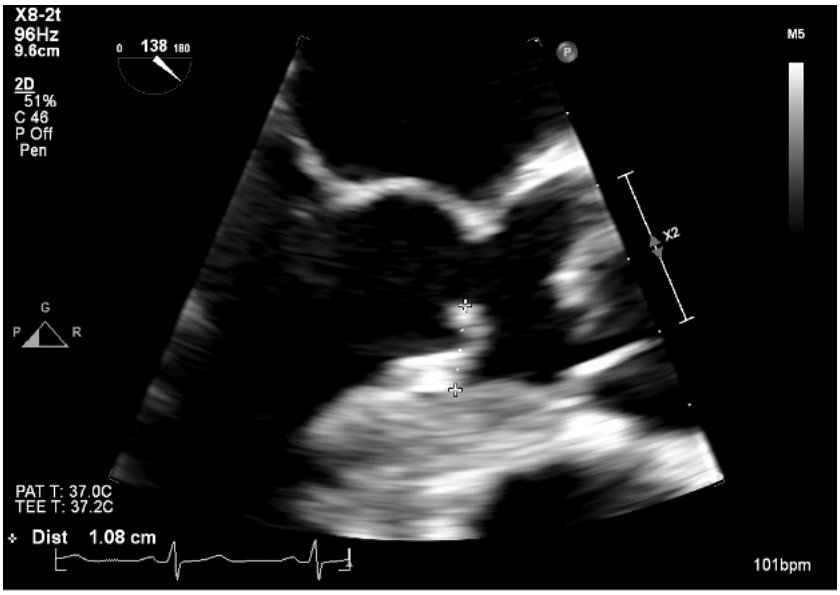
An asymptomatic 42 year old man with subvalvular aortic stenosis presented for elective mini thoracotomy subaortic membrane resection after surveillance echos showed a worsening LVOT gradient. He was induced under general anesthesia and intubated uneventfully. His pre-bypass transesophageal echo (TEE) showed normal biventricular function and no significant valvular abnormalities. It was notable for a subaortic membrane with subvalvular stenosis and a mean gradient of 30 mmHg through the LVOT, consistent with the preoperative echo. While there was basal septal hypertrophy, no SAM was noted. The patient underwent an uneventful subaortic membrane resection via a minimally invasive approach. Upon weaning from bypass, TEE revealed new severe mitral regurgitation with a posteriorly directed eccentric jet related to new chordal SAM. There was a persistently elevated gradient through the LVOT (19 mmHg). The surgical team then converted to a full sternotomy for myectomy with repositioning of the papillary muscles. The second post-bypass echo showed a reduction in the mean gradient through the LVOT to 7 mmHg, and trivial central mitral regurgitation with no further evidence of SAM. The patient was extubated on postoperative day 1 and discharged on postoperative day 3.

Discussion

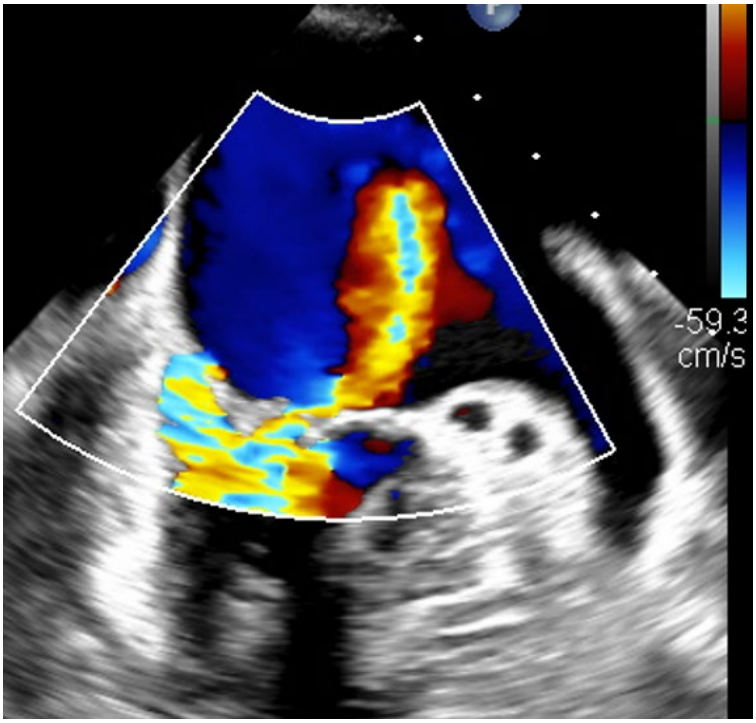
New-onset SAM of the mitral valve following subaortic membrane resection is an uncommon but clinically significant postoperative complication. In this case, the relief of fixed obstruction likely altered the balance of forces within the LVOT, unmasking a predisposition toward abnormal mitral leaflet motion.[2] Further, sudden reduction in afterload may enhance basal hypercontractility, further narrowing the LVOT in this patient with a thickened septum at baseline. Other anatomic factors, such as displacement of papillary muscles in this patient, may not cause obstruction in the presence of a fixed membrane but become pathologic post-resection. This case underscores the need for thorough preoperative assessment of the LVOT and mitral apparatus, particularly in patients undergoing minimally invasive surgery for fixed subaortic

obstruction. Identifying subtle morphological risk factors may inform operative planning and anticipatory hemodynamic management.

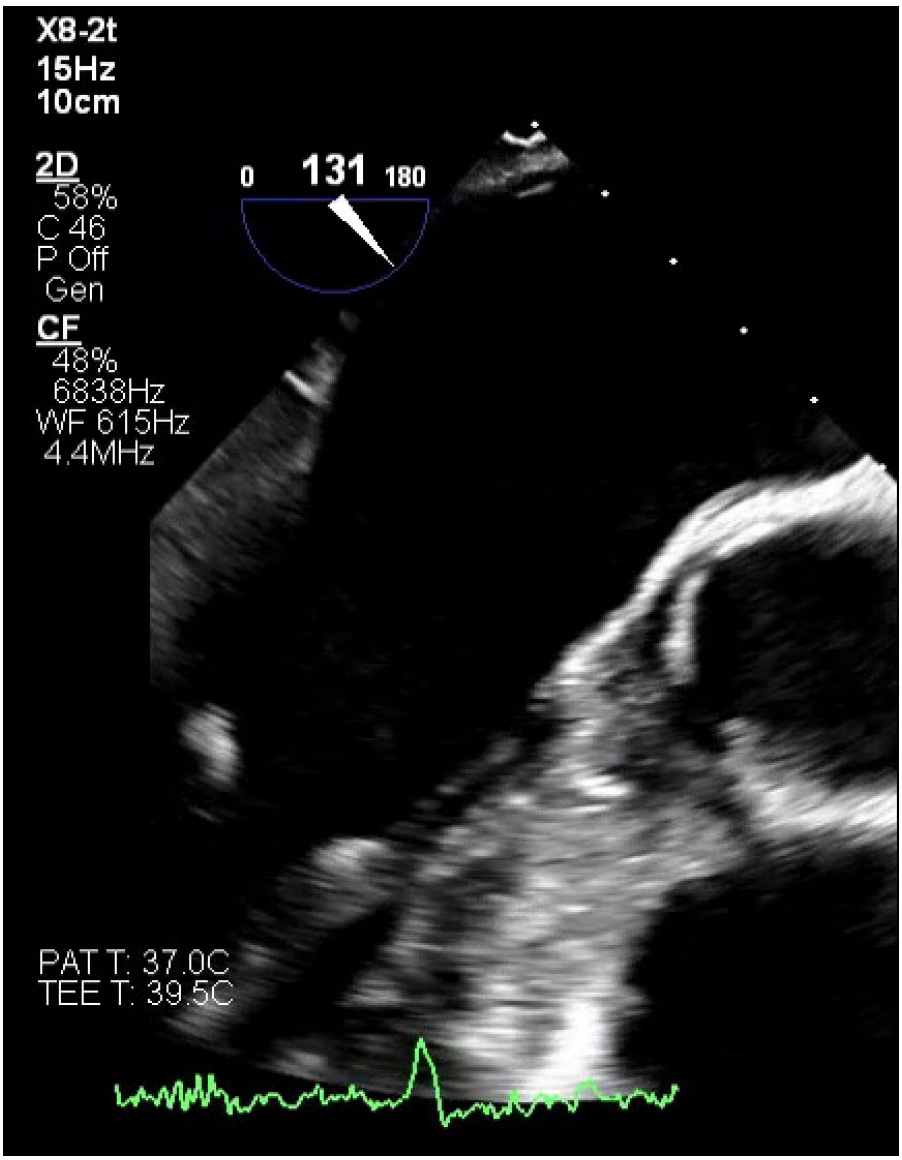
Images



Pre-bypass subaortic membrane with underlying basal septal hypertrophy



New eccentric mitral regurgitation following subaortic membrane resection



Chordal SAM in long axis

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#FR 1 – Kiosk 15 – Poster 4

Anesthetic Management of Aorto-Innominate Bypass Graft

Presenting Author: Michelle Dickinson, MD, Adult Cardiothoracic Anesthesiology Fellow, Northwestern McGaw Medical Center

Introduction

Operative intervention of the aortic arch vessels is indicated for occlusion or severe stenosis that results in symptomatic cerebral or extremity ischemia. Although this presents primarily as a vascular case, the cardiac anesthesiologist must be prepared to advocate for modifications to the surgical plan and additional resources as intraoperative conditions evolve.

Case Presentation

A 73-year-old woman with innominate artery stenosis presented for aorto-innominate bypass surgery. CTA of the head and neck showed severe stenosis of the proximal innominate artery. Cardiac workup revealed normal biventricular function, no significant valvular abnormalities, and mild CAD. Due to heavy calcification of the innominate artery, vascular surgery recommended an open bypass graft with hemisternotomy. In the operating room, bilateral radial arterial lines were placed. The right line initially read a MAP 20-40 mmHg lower than the left. Central access was obtained, and to ensure adequate cerebral perfusion during clamping of the innominate artery, cerebral oximetry was monitored and MAP was maintained >80 mmHg. A 10mm Gelweave graft was anastomosed end-to-side to the ascending aorta using a side-biting aortic clamp, and then end-to-end to the distal innominate artery. The proximal innominate stump was oversewn. Following the procedure, the arterial lines correlated within 5 mmHg. The cerebral oximetry readings continued to correlate within 3-4%. The patient was extubated in the operating room, recovered in the ICU, and was discharged to an acute rehab facility.

Discussion

Supra-aortic trunk vessel stenosis can be treated endovascularly, via open extra-anatomic bypass, or via open transthoracic aortic bypass. In this case, the heavily calcified innominate lesion required open bypass. Patient-specific factors, surgeon comfort, and need for concomitant cardiac surgery can all determine the optimal repair technique2. While endovascular approaches are associated with lower risk of stroke or death

(about 1.5% over 1 year), the risk of restenosis approaches 9%. Open revascularization is associated with a higher risk of stroke or death (7%), however with lower restenosis incidence (2.6%)3. To avoid cardiopulmonary bypass, these cases are typically performed with a side-biting aortic clamp. This requires an adequately sized aorta with an appropriate region of disease-free wall for the clamp to be applied. In the absence of this, or in the setting of concomitant cardiac disease requiring intervention, CPB may be necessary to facilitate complete aortic cross-clamping. CPB is also indicated when there is uncontrolled aortotomy bleeding from the side-biting clamp, or in the setting of aortic complications such as dissection or rupture. Given the potential for conversion to CPB, these cases should be performed with central access and a perfusionist on standby. Outside of aortic complications and bleeding, the primary intraoperative risk in these cases is ischemic stroke from clamping extracranial cerebral vessels or from dislodged atheroma1. During this case, MAP was kept >80 mmHg and cerebral oximetry was measured continuously. Other techniques include measuring stump pressure or using evoked potentials. Early extubation facilitates rapid neurological assessment.

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#FR 1 – Kiosk 15 – Poster 5

Ventricular Assist Device in Patients with Congenital Heart Disease

Presenting Author: Reem Almuqati, MD, cardiothoracic anesthesia fellow, Cleveland clinic

Introduction

Mechanical circulatory support in patients with congenital heart disease remains challenging and requires significant multidisciplinary planning. Congenital heart disease often has wide variations in anatomy and unique physiology that require careful surgical planning. Here, we present a case of a patient with congenital heart disease that underwent Left Ventricular Assist Device (LVAD) as a destination therapy.

Case Presentation

This is a case of a 20-year-old male with a history of a double inlet left ventricle who underwent bidirectional Glenn procedure as a child and presented in severe cardiogenic shock and respiratory failure. The patient suffered an in-hospital PEA arrest and was cannulated with peripheral venoarterial extracorporeal membrane oxygenation (VA-ECMO). Subsequent workup for heart transplantation revealed high panel reactive antibodies (PRAs), and he was deemed ineligible for heart transplantation. Thus, the patient underwent completion of the Fontan procedure and placement of a left ventricular assist device (LVAD, HeartMate III) to facilitate wean from VAECMO and for management of his advanced heart failure physiology. At time of the LVAD implantation, there was considerable concern for circulatory overload of the new Fontan circulation. Thus, after multidisciplinary discussion with the anesthesia and surgical team, a 5mm fenestration was created in the Fontan conduit. His postoperative course was complicated by mixed hypercarbic hypoxemic respiratory failure and extended stay in the intensive care unit.

Discussion

Here, we presented a unique case of destination therapy MCS in a patient with congenital heart disease. His presentation was unusual because routine pediatric operative management dictates conversion of the Glenn circulation to a Fontan in early childhood; however, this patient was lost to follow-up and survived early childhood with a Glenn circulation. This case has many learning points for adult cardiac anesthesiologists less familiar with intricate details of congenital heart disease. Firstly, induction of anesthesia in an unstable patient with a Glenn procedure should be done through femoral access, as the inferior vena cava connects directly to the heart and systemic circulation while the superior vena cava drains directly into the pulmonary artery leading to delayed effect of administered medication. Second, the decision to create a Fontan fenestration must be done in conjunction with the surgical and anesthesia team. The teams should weigh the risk of circulatory overload caused by the LVAD while also considering that a fenestration will lead to hypoxemic shunt physiology. LVAD speed and flow rate should be titrated to the desired effects of ventricle decompression, while maintaining optimal central venous pressure. Many centers have adopted implantation of LVAD in patients with congenital heart disease, and it is imperative for the adult cardiac anesthesiologist to be familiar with this complex physiology.

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#FR 1 – Kiosk 15 – Poster 6

Dissecting the Use of the Endoaortic Balloon for Minimally Invasive Mitral Valve Surgery

Presenting Author: Ricki Pad, Cardiothoracic Anesthesiology Fellow, University Hospitals Cleveland Medical Center

Introduction

Minimally invasive mitral valve surgery (MIMVS) emerged in the mid-1990s as an alternative to conventional median sternotomy. MIMVS is associated with reduced postoperative pain, shorter hospitalizations, and faster return to baseline function. Aortic occlusion in MIMVS may be achieved using either an external aortic clamp (EAC) or endoaortic balloon occlusion (EABO). Although EABO facilitates a minimally invasive approach, it carries unique risks including aortic dissection and balloon migration.

Case Presentation

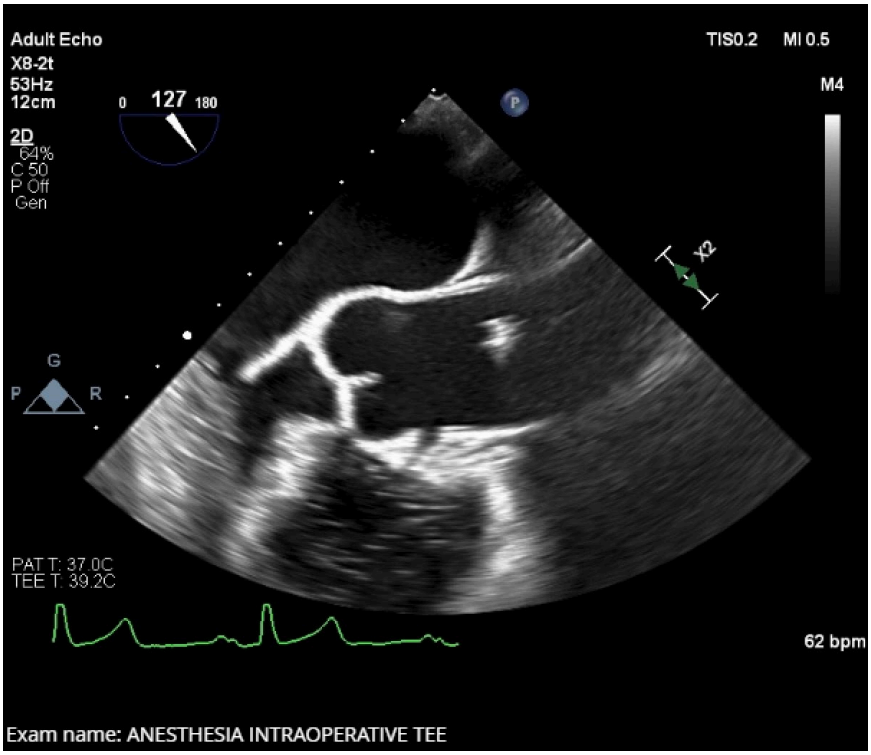
A 68-year-old male with hypertension, atrial fibrillation, and newly diagnosed moderate-to-severe mitral regurgitation presented for robotic-assisted mitral valve repair. During the presurgical briefing, the operative plan included use of the IntraClude endoaortic balloon occlusion device for cardiopulmonary bypass. Prior to induction, a left brachial arterial line was placed, followed by intubation with a double-lumen endotracheal tube. Cerebral oximetry and a contralateral arterial line were utilized to detect balloon migration. The balloon was positioned under transesophageal echocardiography (TEE) guidance at the sinotubular junction, with root pressure monitored through the device. Approximately 15 minutes after placement, the aortic root pressure waveform was lost. TEE demonstrated a large aortic root hematoma and a new dissection flap extending from the ascending aorta through the descending thoracic aorta, with involvement of the left subclavian artery. The procedure was emergently converted to median sternotomy. The ascending aorta was replaced with a 28-mm graft, and mitral valve repair was completed with 2 neo-cords and placement of a 34-mm annuloplasty ring. The patient was transferred to the cardiothoracic intensive care unit in stable condition. Postoperative CT imaging confirmed dissection originating in the proximal ascending aorta with propagation to the left subclavian and bilateral iliac arteries. No additional surgical intervention was required. The patient was discharged on postoperative day 16 and demonstrated full recovery at 1-month follow-up.

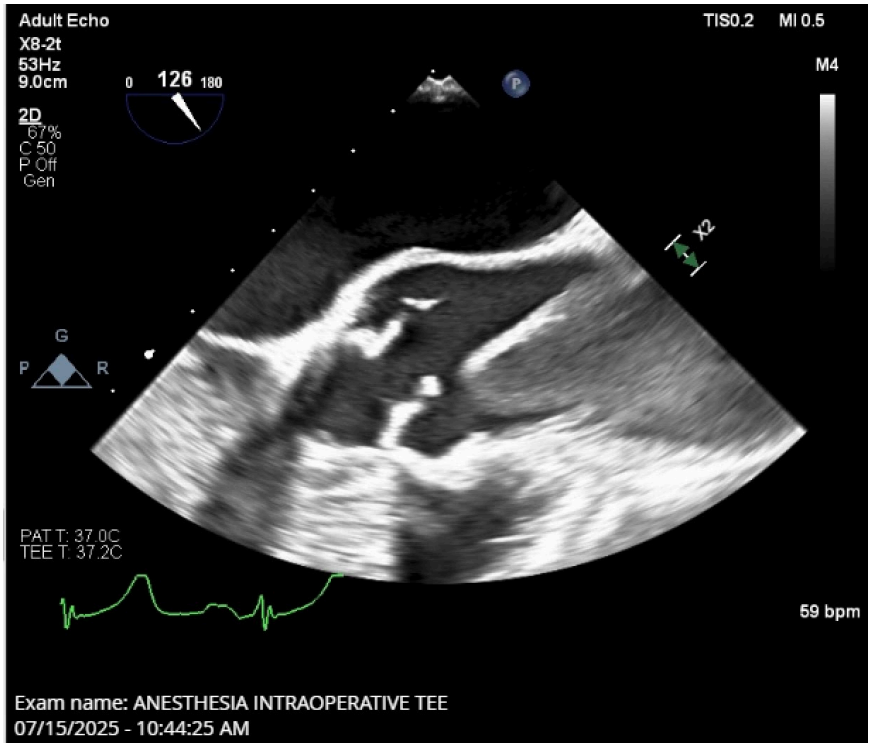
Discussion

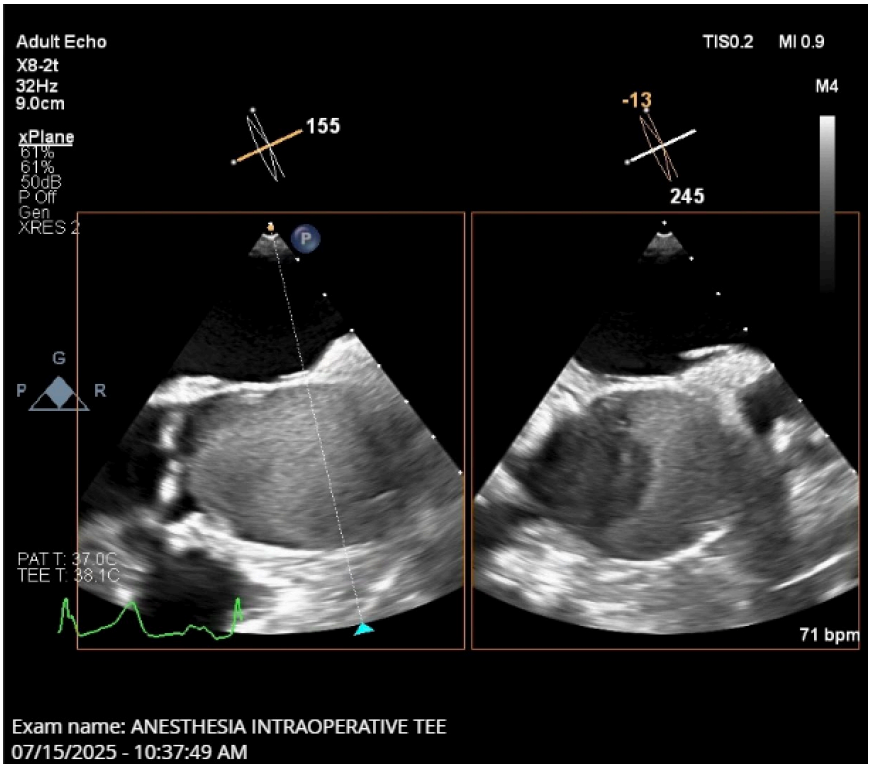
The endoaortic occlusion balloon includes three lumens—one for balloon inflation, one for cardioplegia delivery and venting, and one for monitoring aortic root pressure. Although EABO offers an alternative when external clamping is not feasible, current evidence demonstrates an increased risk of iatrogenic aortic dissection compared with EAC. A meta-analysis by Kowalewski et al. identified no difference in 30-day mortality between EABO and EAC across 15 studies; however, EABO demonstrated a significantly higher rate of aortic dissection. Rates of stroke and acute kidney injury did not differ significantly, and EABO was associated with longer cardiopulmonary bypass and cross-clamp times. EABO remains an effective strategy for aortic occlusion during MIMVS but requires meticulous monitoring and heightened awareness of

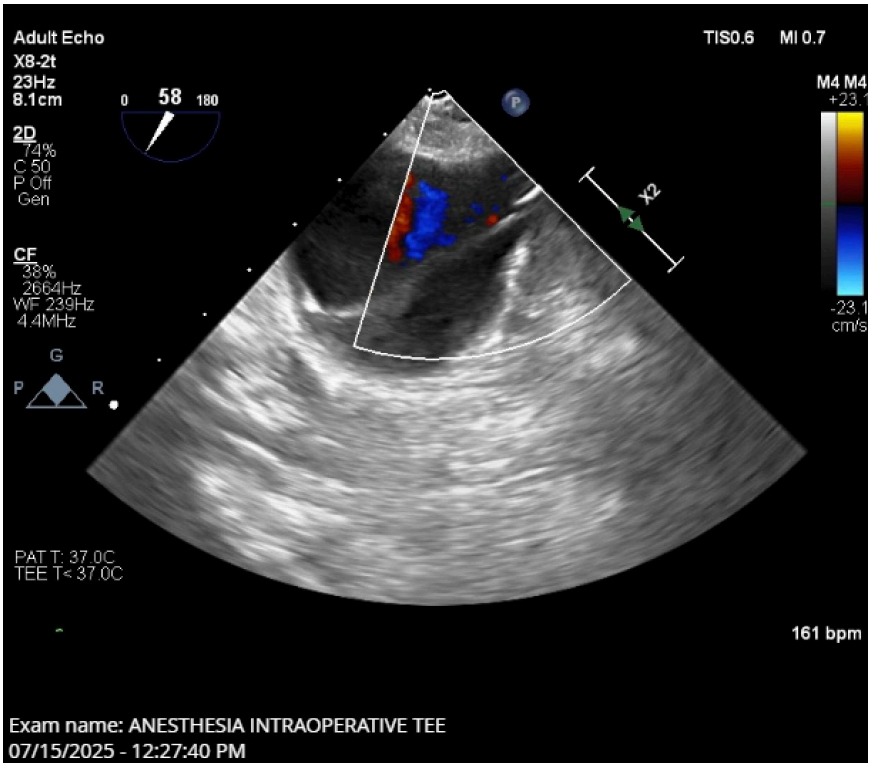
potential vascular complications. Early recognition via vigilant hemodynamic and echocardiographic assessment is essential to reducing morbidity and facilitating rapid management

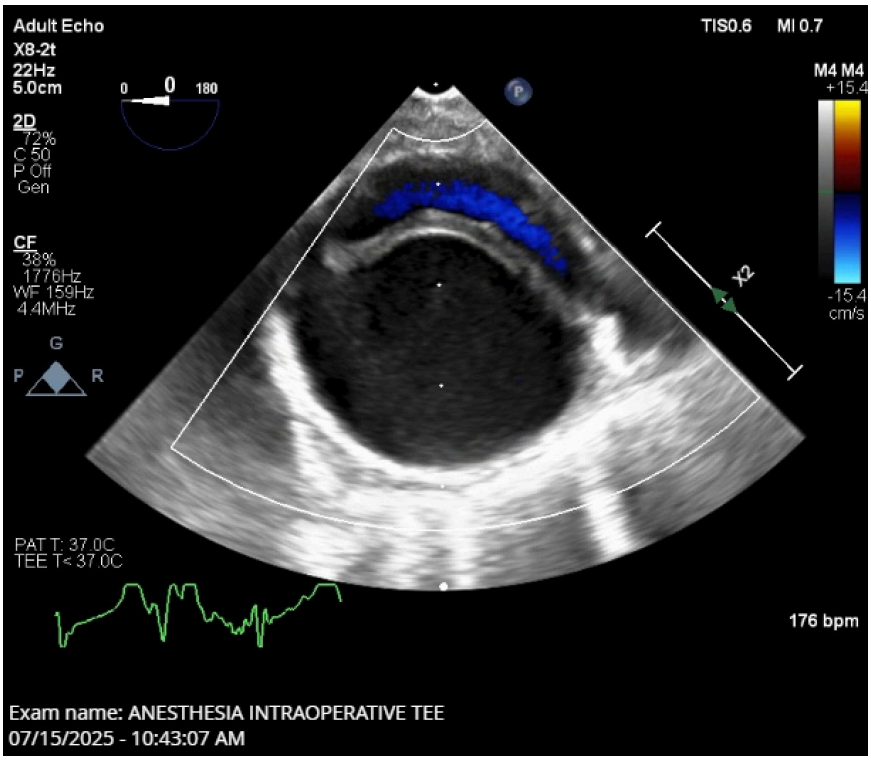
Images




Adult Echo
X8-2t
53Hz
9.0cm
2D
67%
C 50
P Off
Gen
126
TIS0.2 MI 0.5
M4
PAT T: 37.0C
TEE T: 37.2C
59 bpm
Exam name: ANESTHESIA INTRAOPERATIVE TEE
07/15/2025 - 10:44:25 AM


Adult Echo
X8-2t
32Hz
9.0cm
xPlane
61%
61%
50dB
P Off
Gen
XRES 2
155
-13
245
TIS0.2 MI 0.9
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PAT T: 37.0C
TEE T: 38.1C
71 bpm
Exam name: ANESTHESIA INTRAOPERATIVE TEE
07/15/2025 - 10:37:49 AM


Adult Echo
X8-2t
23Hz
8.1cm
2D
74%
C 50
P Off
Gen
CF
38%
2664Hz
WF 239Hz
4.4MHz
TIS0.6 MI 0.7
M4 M4
+23.1
-23.1
cm/s
PAT T: 37.0C
TEE T< 37.0C
161 bpm
Exam name: ANESTHESIA INTRAOPERATIVE TEE
07/15/2025 - 12:27:40 PM


Adult Echo
X8-2t
22Hz
5.0cm
2D
72%
C 50
P Off
Gen
CF
38%
1776Hz
WF 159Hz
4.4MHz
TIS0.6 MI 0.7
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+15.4
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cm/s
PAT T: 37.0C
TEE T< 37.0C
176 bpm
Exam name: ANESTHESIA INTRAOPERATIVE TEE
07/15/2025 - 10:43:07 AM



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#FR 1 – Kiosk 15 – Poster 7

Perioperative Management of Vasoplegic Shock During HeartMate 3 Implantation in an Advanced Heart Failure Patient Requiring Inotropic and Intra-aortic Balloon Pump Support

Presenting Author: Kaber Karimi, MD, Fellow Physician, New York University

Introduction

Management of patients undergoing LVAD implantation presents one of the most difficult challenges in cardiac anesthesia, particularly when complicated by severe biventricular dysfunction, inotrope dependence, mechanical circulatory support, and anticipated vasoplegia. Optimization of right ventricular (RV) function, mitigation of afterload shifts, de-airing and weaning from cardiopulmonary bypass (CPB) are critical determinants of successful HeartMate 3 implantation. This case highlights perioperative anesthetic challenges in a patient with advanced nonischemic cardiomyopathy on milrinone and intra-aortic balloon pump (IABP) support who underwent Heartmate3 implantation and developed significant post-CPB vasoplegia along with challenges de-airing the heart

Case Presentation

57-year-old male with end-stage heart failure due to nonischemic cardiomyopathy presented in cardiogenic shock requiring milrinone and IABP support. He was admitted with decompensated heart failure and was offered a HeartMate3. Pre-bypass TEE revealed severe left ventricular failure and severe central mitral regurgitation originating around a prior MitraClip, along with moderate central tricuspid regurgitation without hepatic vein flow reversal. Based on these findings, the decision was made to proceed with isolated LVAD placement. Following cross-clamp removal, an ascending aortic vent was placed. Prolonged de-airing was required before adequate cardiac decompression was achieved. After complete de-airing, the patient was separated from CPB. RV function was initially adequate; however, the patient experienced recurrent hypotensive episodes despite epinephrine and dobutamine support. Epinephrine doses were increased and vasopressin was initiated to treat vasoplegia, which was suspected to be related to prolonged milrinone exposure and the underlying shock state. Final LVAD flows were 2.8–3.0 L/min at 5100–5200 RPM. There were no intraoperative complications aside from significant vasoplegia however weaning off CPB and considerations for RVAD supported were a significant part of discussion before leaving the OR

Discussion

This case illustrates several anesthetic considerations in LVAD implantation. Peri-operative TEE assessment was pivotal in guiding the surgical decision to avoid mitral or tricuspid intervention, given preserved RV preload indices. The need for extensive de-airing emphasized the anesthetic role in coordinating venting strategies, maintaining adequate ventilation, and continuously monitoring with TEE to prevent embolic events. The impact of increased workload on RV function further complicated management. Hemodynamic instability following CPB was attributed to vasoplegia, a recognized risk in patients on chronic inotropes and mechanical support. Early vasopressin supplementation and escalation of catecholamines allowed maintenance of systemic vascular resistance while preserving RV output in the setting of new LVAD support.

Achieving appropriate pump flows while balancing RV performance highlighted the interplay between vasoactive therapy, volume status, and afterload management. This case underscores the importance of anticipatory anesthetic planning, TEE-guided surgical decision-making, and tailored vasoactive therapy during LVAD implantation.

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#FR 1 – Kiosk 16 – Poster 1

Case of Severe Acute Mitral Regurgitation and Hemorrhagic Shock after AMULET Device Embolization

Presenting Author: Nicholas S. Cho, MD, Anesthesiology Resident, Virginia Mason Medical Center

Introduction

Percutaneous LAA occlusion devices such as the WATCHMANTM and AmplatzerTM AmuletTM continue to be a viable alternative for stroke prevention in the setting of atrial fibrillation. Multidisciplinary discussion and shared patient decision making is incorporated in optimizing the risk assessment of each individual patient for stroke prevention with CHA2DS2VASc score and bleeding risk with HASBLED score. Percutaneous LAAO device utilization continues to increase as large RCT studies such as PROTECT-AF and PREVAIL show non-inferiority in long-term primary outcomes of stroke prevention. However, intraoperative management of device deployment and subsequent complications continue to evolve in technique and device development.

Case Presentation

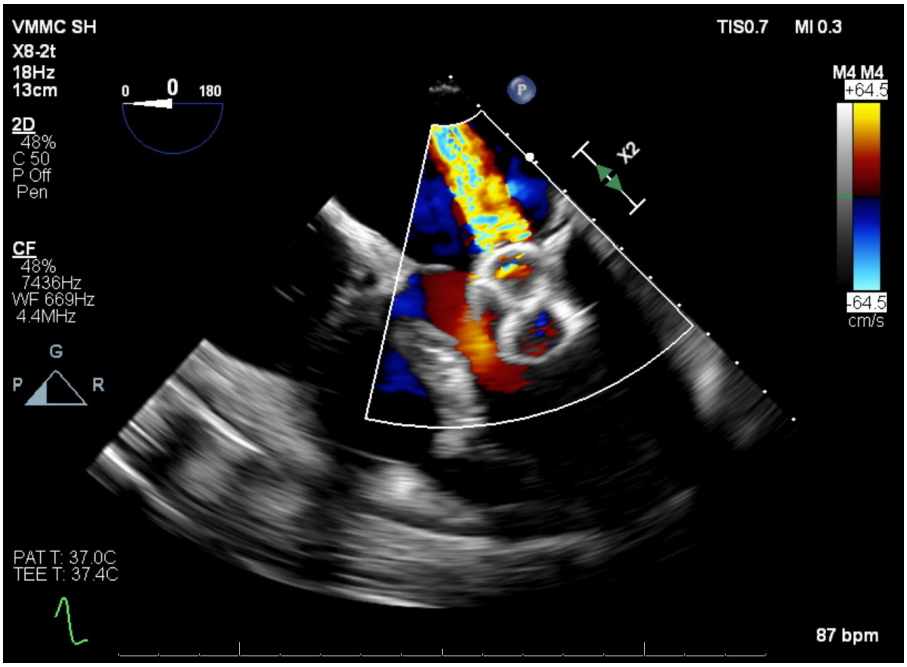
84-year-old female with a past medical history of paroxysmal atrial fibrillation on Eliquis, frequent falls, HFpEF, moderate mitral regurgitation, mild CAD, hypertension, and cerebral aneurysms presented for routine implantation of Watchman placement. General endotracheal anesthesia and TEE was utilized during this procedure. Initially, a 24 mm Watchman device was used, but after multiple repositioning maneuvers a persistent leak was evident. After much discussion, it was replaced with a 28 mm AMULET device, which was successful and without complication. Two hours later, the patient had severe hypotension and nausea. TTE showed a dislodged AMULET device was entangled in the mitral valve and the patient was promptly taken back to cath lab. A RAPTOR grasping device was utilized to capture the embolized device and then released into the left atrium with attempts to pin the device and recapture it. Eventually, it was successfully removed via the femoral vein. However, venogram showed significant IVC injury. A Coda balloon was inserted and an IVC stent was successfully placed. On further TEE evaluation, severe mitral regurgitation was noted. A MitraClipTM device was attempted but eventually aborted due to poor tissue quality and inability to grasp the previously damaged leaflets. The septal defect was closed and an IABP was inserted. Unfortunately, the patient continued to fail maximum medical therapy and passed approximately 24 hours later.

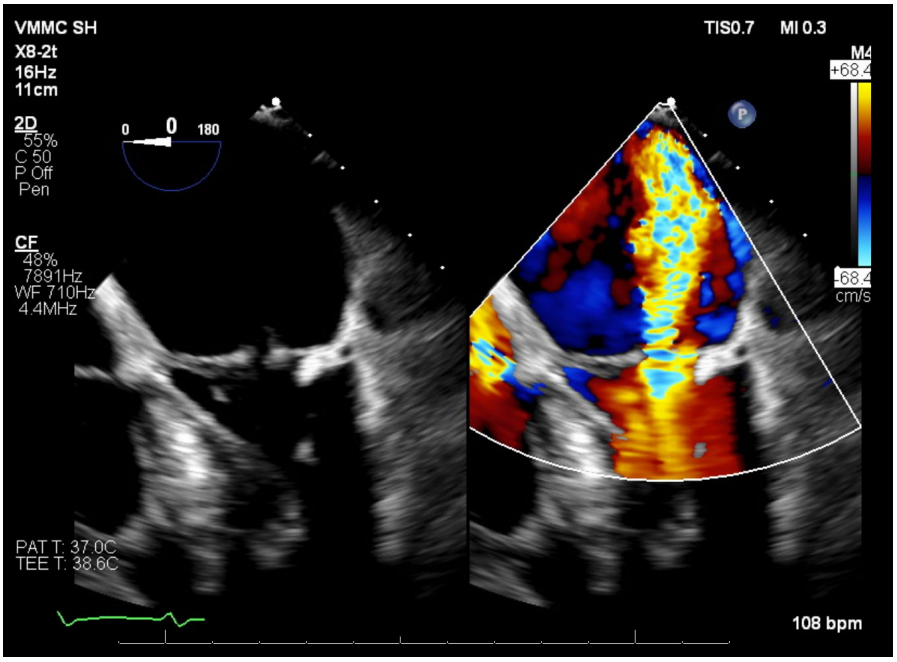
Discussion

While LAAO device placement has become safer, this patient's intraoperative course elucidates the intraprocedural risks of device placement. Due to the rarity of the embolic events, small multi-centered studies, case reports, and expert opinion provide insight into management of device embolization. Unfortunately, there were several re-deployments of the initial 24mm Watchman. Naturally, tissue integrity of the thin left atrial walls can be postulated as compromised, making subsequent deployments at higher risk of migration or embolization. Aborting and allowing wound healing time in between attempts could also be considered. Proper sizing and landing of LAAO devices are undoubtedly imperative for success and

decreasing embolization risk as both over and under sizing has been postulated as reasons for device embolization. Vigorous tug tests have also been implicated in device embolization. Given the rarity of device embolization events we hope this clinical case contributes to discussion of management and further awareness of complications.

Images





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#FR 1 – Kiosk 16 – Poster 2

Multidisciplinary decision making in management of paravalvular leaks following mitral and aortic valve replacement

Presenting Author: Rachel A. Gioscia-Ryan, MD, PhD, Clinical Fellow, Brigham and Women's Hospital

Introduction

We present a patient with severe calcific mitral and aortic stenosis (MS; AS) who underwent mitral and aortic valve replacement (MVR; AVR) with paravalvular leaks (PVL) of the prosthetic mitral valve discovered on weaning from cardiopulmonary bypass (CPB). After intraoperative discussion among surgical, interventional cardiology and anesthesia teams—guided by real-time transesophageal echocardiography (TEE)—a decision was made not to return to CBP and to pursue percutaneous PVL repair.This case illustrates considerations for anesthetic management of severe mixed valvular pathology (MS; AS), highlights complexities of management of PVL in the setting of MAC and emphasizes the importance of multidisciplinary collaboration and the role of TEE for intraoperative decision making.

Case Presentation

A 67-year-old male with gout arthritis on steroids and severe MS and AS presented for MVR and AVR. Anesthesia induction was performed via titration of midazolam, fentanyl and etomidate, with continuous epinephrine infusion. Pre-CPB TEE showed normal biventricular function, severe calcific AS and MS with significant mitral annular calcification (MAC; Fig 1). The surgical procedure consisted of mechanical MVR (25/33 mm On-X) and AVR (21 mm TopHat). When weaning from CPB, TEE revealed anteromedial and posterolateral PVLs of the mitral prosthesis with 3D TEE used for localization (Fig 2). The valve was stable without stenosis and the patient remained hemodynamically stable. The PVLs were discussed with the surgeon, and benefits of returning to CPB for repair were considered against risks of worsening PVL in setting of severe MAC and extending time on CBP (already 283 min). Interventional cardiology was consulted intraoperatively, with TEE used to provide real-time imaging. Ultimately, it was felt that the PVL could be addressed via transcatheter approach and the decision was made to close and proceed to the intensive care unit. The patient was discharged on postoperative day 9 with no evidence of hemolysis and a plan to return for transcatheter repair one month later.

Discussion

Key tenets of anesthetic management for severe MS and AS are maintenance of cardiac output and avoidance of myocardial ischemia; a gradual, mixed induction technique with concomitant inotropic support was selected to achieve these goals. Incidence of PVL is reported as 6-17% following MVR. MAC and mechanical valves— both present in this case—increase the risk for PVL. Intervention to repair PVL is indicated when the lesion causes heart failure symptoms or significant hemolysis and can be accomplished via surgical and transcatheter approaches, both of which carry attendant risks. Notably, surgical re-repair is difficult and complex in the setting of MAC, with risk of worsening PVL or causing damage such as atrioventricular groove disruption. As illustrated here, TEE is the modality of choice for assessing PVL and

facilitates precise localization. Providing this information to the interventional team was integral to decision making. Professional societies emphasize the importance of individualized risk-benefit assessment and multi-disciplinary approach when considering PVL repair; this was exemplified in this case wherein expertise from multiple teams was employed to determine a course of action.

Images

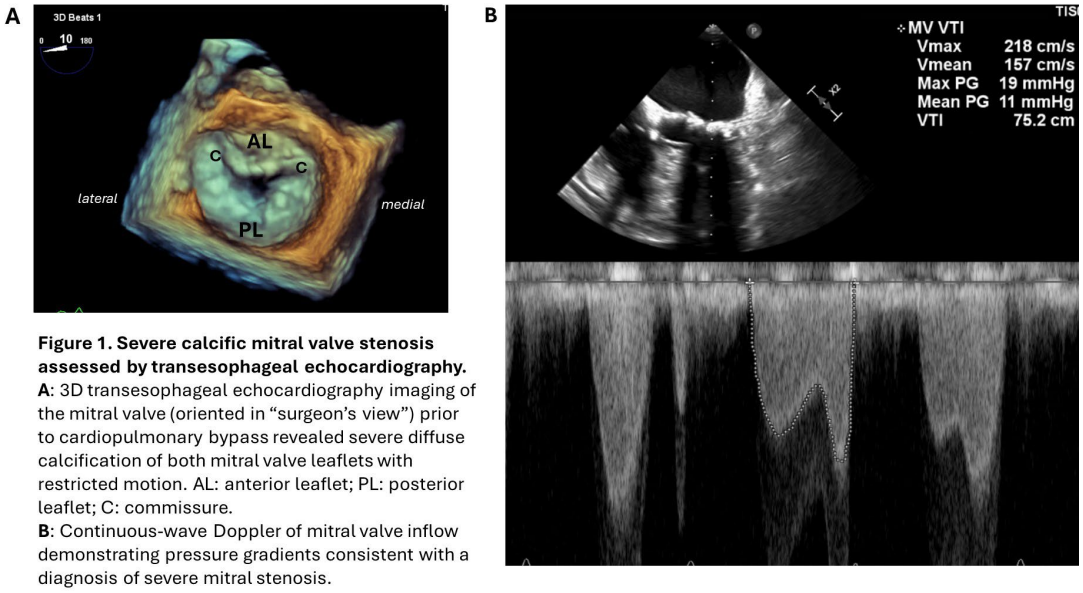


Figure 1. Severe calcific mitral valve stenosis assessed by transesophageal echocardiography.
A: 3D transesophageal echocardiography imaging of the mitral valve (oriented in "surgeon's view") prior to cardiopulmonary bypass revealed severe diffuse calcification of both mitral valve leaflets with restricted motion. AL: anterior leaflet; PL: posterior leaflet; C: commissure.
B: Continuous-wave Doppler of mitral valve inflow demonstrating pressure gradients consistent with a diagnosis of severe mitral stenosis.

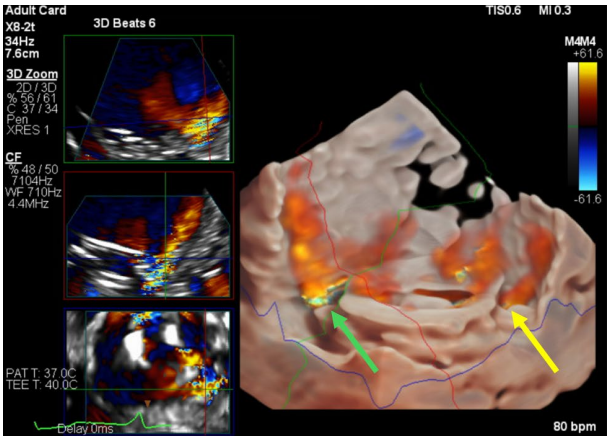


Figure 2. Localization of paravalvular leaks using 3D transesophageal echocardiography.
Intraoperative 3D imaging with multiplanar reconstruction of the prosthetic mitral valve enabled precise localization of paravalvular leaks (posterolateral, green arrow, EROA 0.30 cm^2; anteromedial, yellow arrow, EROA 0.12 cm^2) in real time to facilitate multidisciplinary decision-making regarding management strategy.
EROA: effective regurgitant orifice area

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#FR 1 – Kiosk 16 – Poster 3

Cesarean Section in Parturient with Severe Peripartum Cardiomyopathy

Presenting Author: Andrea Martinez, MD, Resident, Northwell Health

Introduction

We present the management of a parturient with severe PPCM (LVEF 22%), CardioMEMS, and CRT-D undergoing C-section using a slow-titrated epidural.

Case Presentation

A 28-year-old G3P2 with a history of peripartum cardiomyopathy (LVEF 22%), pulmonary embolism (on prophylactic Lovenox), and GDMA, s/p CRT-D and CardioMEMS placement following her first pregnancy, was admitted at 35w6d for cardiac optimization prior to scheduled primary cesarean section for breech presentation and bilateral salpingectomy. Admission TTE demonstrated an LVEF of 22%, PASP 35 mmHg, and no valvular pathology. Interrogation of the CardioMEMS revealed elevated pulmonary artery diastolic pressures, prompting aggressive diuresis. On the day of surgery, a lumbar epidural was placed. Surgical anesthesia was achieved using a slow titration of 2% lidocaine with epinephrine (1:200,000) in 5 mL increments to blunt sympathetic swings. A magnet was applied to the CIED to suspend antitachycardia therapies, and external defibrillator pads were applied. The patient delivered a vigorous infant. Maternal hemodynamics remained stable throughout autotransfusion. She was transferred to the CCU, downgraded on POD #1, and discharged home on POD #4 with close cardiology follow-up.

Discussion

This case illustrates the successful anesthetic management of a parturient with severe Stage IV heart failure (LVEF 22%) secondary to peripartum cardiomyopathy (PPCM), utilizing invasive hemodynamic guidance and a titrated neuraxial technique. The patient's history of prior PPCM necessitated a strategy prioritizing afterload reduction without compromising coronary perfusion pressure. The utilization of the CardioMEMS provided a unique advantage in the pre-anesthetic phase. Unlike traditional reliance on clinical signs or TTE surrogates, this allowed for direct, ambulatory monitoring of pulmonary artery diastolic pressures (PADP), serving as a surrogate for PCWP. This data facilitated precise diuretic optimization prior to the operative autotransfusion burden, crucial for a ventricle with limited contractile reserve. Intraoperatively, the decision to proceed with a graded epidural rather than a Combined Spinal-Epidural (CSE) or General Anesthesia (GA) was strategic. While GA allows for transesophageal echocardiography (TEE) guidance, a careful, slow induction of anesthesia would be contraindicated since the patient is considered full stomach with poor pulmonary reserve. Conversely, a single-shot spinal risks precipitous sympathectomy and SVR collapse, which is poorly tolerated in fixed-output states. The "slow-loading" epidural technique (5 mL lidocaine/epi boluses) allowed for a controlled reduction in SVR, effectively reducing LV afterload while allowing time for compensatory reflex vasoconstriction or vasopressor titration to maintain MAP. Successful management relied on a coordinated multidisciplinary approach involving obstetrics, cardio-obstetrics, and anesthesia

for optimization. Furthermore, cardiothoracic surgery backup was secured for immediate mechanical support if required.

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#FR 1 – Kiosk 16 – Poster 4

Combined CABG, Liver Transplant, and Double Lung Transplant in a Patient with IgG4 Related Disease

Presenting Author: Christopher J. Scheiber, MD, Vanderbilt University Medical Center

Introduction

Although experiments with transplantation have been occurring since the 19th century, it was not until the introduction of cyclosporine in 1983 that the risk of rejection was greatly reduced. Since then, the field of organ transplantation has greatly expanded to include combination procedures such as the Combined Lung and Liver Transplant (CLLT). CLLT is one of the rarer transplant operations and was utilized in only 25 patients in the United States in 2024, and only 22 in 2023 [Reference 1]. Many of these individuals suffer from cystic fibrosis or other disease affecting both organ systems [Reference 2]. CLLT raises several challenges for the intra-operative anesthesia team, most notably being the delicate balance between the resuscitation of a coagulopathic liver transplant candidate and the fragile nature of freshly transplanted lungs. In this case presentation, we will describe the anesthetic management of a combined coronary artery bypass graft (CABG), double lung and liver transplant.

Case Presentation

The patient was a 61 yo M with prior medical history notable for hypertension and a history of tobacco use. He was found to have interstitial lung disease due to Immunoglobulin G-4 related disease (IgG4) and short telomeres syndrome. At the time of this case, he required up to 3 liters/min of oxygen via nasal cannula at rest and 5 liters/min with activity. Due to this diagnosis, he was listed for double lung transplantation. During his evaluation for transplant, he was found to have coronary artery disease as well as cirrhosis. Therefore, following multidisciplinary team discussions it was determined to best optimize his chance of success that a combined lung transplant, liver transplant and coronary artery bypass graft be performed. At the time of the procedure, the patient was first cannulated for ECMO by both the anesthesia and cardiac surgery teams. Single vessel CABG was then completed in a heart beating fashion. Followed by liver transplant with a piggyback caval anastomosis. The lung transplant was then performed, and the patient was able to be separated from ECMO at the conclusion of the procedure on 60% FiO2 with supplemental inhaled nitric oxide. The patient was then transported to the ICU off all pressor support.

Discussion

Combined organ transplants require thorough planning as they are a complicated procedure and involve the allocation of multiple organs to a single recipient. However, in cases where single organ transplantation is insufficient for patient survival, a multi-organ approach must be utilized. There were several aspects of this case which our team felt optimized the patient's outcome, the first being the order in which the procedures were done. The CABG was completed first as to prioritize cardiac perfusion. Then the liver transplant completed before the lungs to reduce the exposure of donor lung tissue to the high-volume resuscitation. Another key decision was to utilize VA ECMO over full cardiopulmonary bypass (CPB) as to mitigate the negative effects of CPB compared to ECMO. And finally, the use of VA ECMO during the liver transplant

procedure along with a piggyback caval anastomosis mitigated any major hemodynamic alterations throughout this stage of the procedure.

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#FR 1 – Kiosk 16 – Poster 5

Right Extracardiac Mass Post-CABG: Limitations of Vacuum-Assisted Aspiration

Presenting Author: Chase Thompson, MD, Anesthesia Resident, University of Florida College of Medicine-Jacksonville

Introduction

Right-sided masses after CABG are challenging to manage, and redo sternotomy carries high risk. Although AngioVac offers a less invasive option, success depends on mass characteristics. We present a post-CABG patient in whom transcatheter aspiration failed, requiring surgical intervention.

Case Presentation

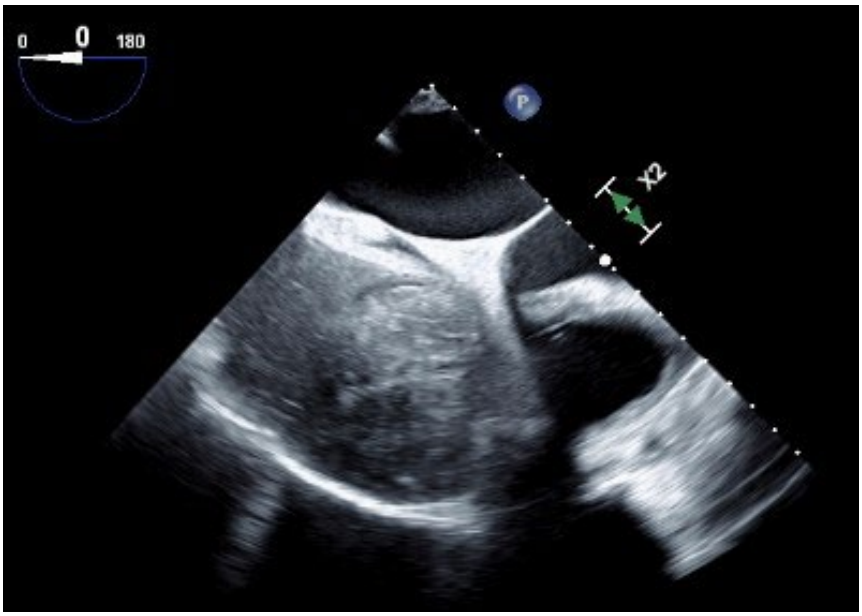
A 67 year old male with severe multivessel coronary artery disease, recent non–ST-segment myocardial infarction, hypertension, ischemic cardiomyopathy, heart failure with reduced ejection fraction (20–25%), new-onset atrial flutter, and type II diabetes mellitus presented for mediastinal washout due to a right cardiac mass. Approximately 30 days prior, he had undergone four-vessel coronary artery bypass grafting, complicated by cardiogenic shock requiring Impella support and a ventricular tachycardia arrest. After discharge with a wearable cardioverter-defibrillator, he re-presented within two weeks with decompensated heart failure. Evaluation revealed an ejection fraction of 20–25%, an 8.3 × 6.8 cm right atrial mass concerning for thrombus, and a small loculated pericardial effusion on limited transthoracic echocardiography. Transcatheter debulking with a Penumbra device was attempted but unsuccessful. A subsequent AngioVac procedure was aborted when transesophageal echocardiography suggested the mass was extracardiac and compressing the right atrium rather than intracavitary. Additional imaging confirmed the extracardiac mass as well as a right subsegmental pulmonary embolus. He was classified as ASA Physical Status 4–emergent. General anesthesia was induced intravenously, and the patient was endotracheally intubated. Intraoperatively, he required only low-dose epinephrine for inotropic support. He remained intubated for transport to the cardiovascular intensive care unit, where his postoperative course was unremarkable. He was later extubated and discharged.

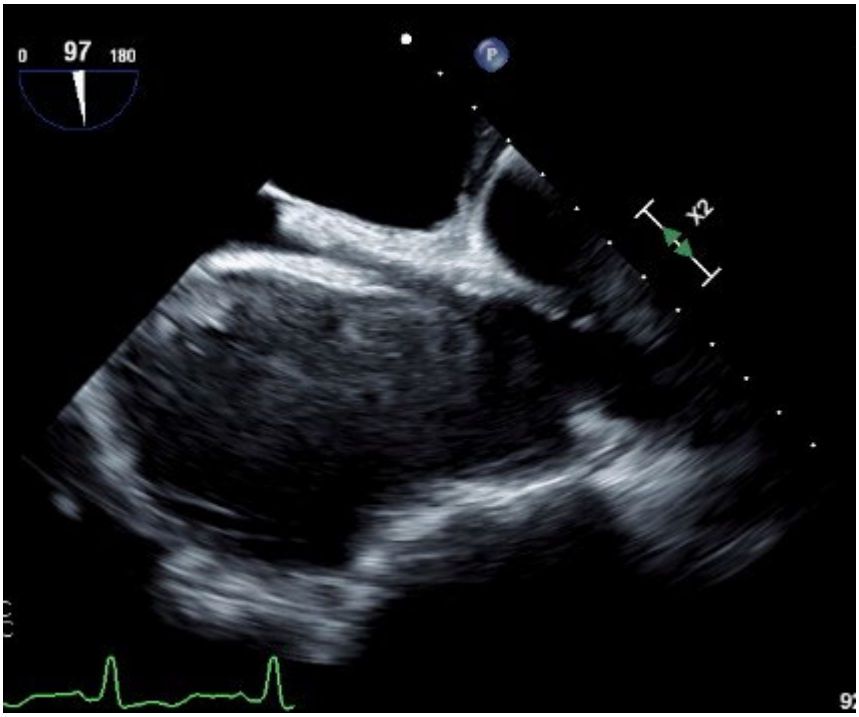
Discussion

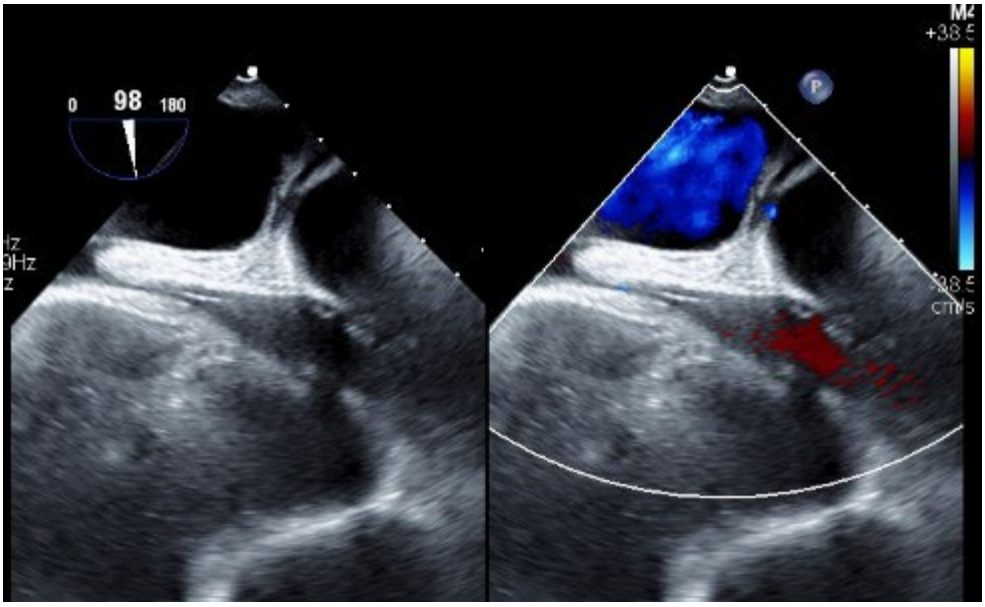
Redo sternotomy after CABG is typically performed for bleeding, tamponade, or hemodynamic instability but carries a high mortality rate of 9.5–22% and significant postoperative morbidity.[1] As a result, transcatheter vacuum-assisted aspiration has become an appealing less invasive option for managing intracardiac and intravascular masses in high-risk patients. The AngioVac system uses a venovenous extracorporeal circuit with fluoroscopic and transesophageal echocardiographic guidance. In the RAPID registry, AngioVac demonstrated success rates of 73.6% for caval thromboemboli, 58.5% for right heart masses, 60% for catheter-related thrombi, and 57.1% for pulmonary emboli.[2] Failure often relates to mass morphology, size, location, and chronicity. Large or extracardiac masses, such as in this case are less amenable to aspiration. Acute thrombi have higher success rates (83%) compared with chronic thrombi (33%), likely due to greater friability.[3] In this patient, transesophageal echocardiography was crucial in identifying the mass as

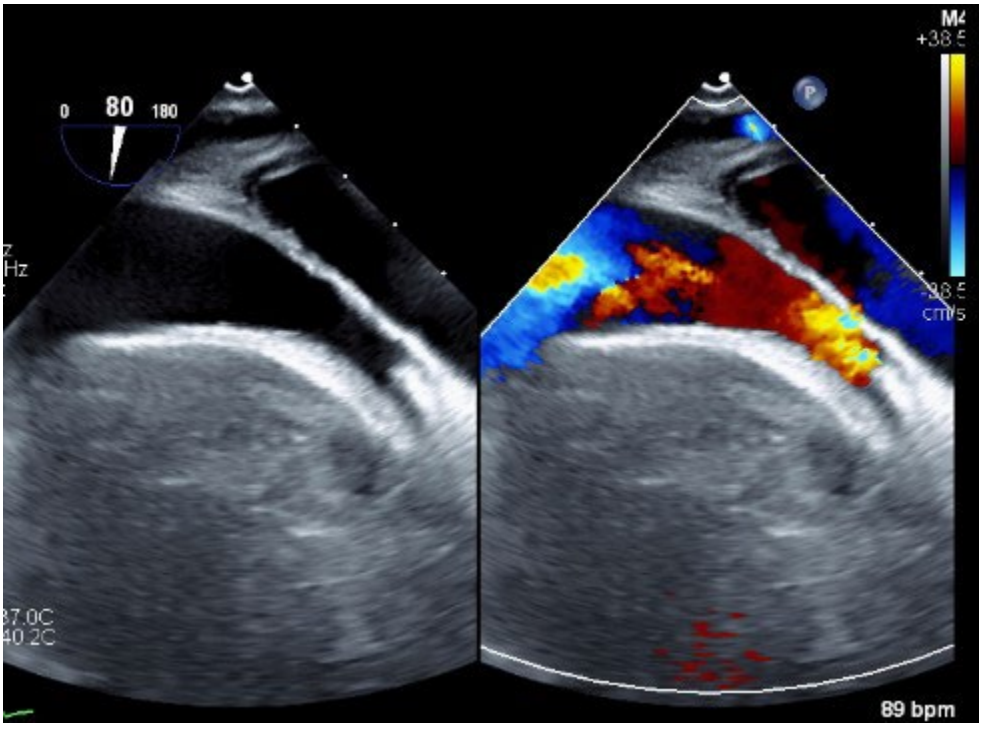
extracardiac. Its smooth borders, proximity to recent CABG, and prior nondiagnostic aspiration raised suspicion for an atypical etiology. This case emphasizes the importance of detailed echocardiographic evaluation when managing right-sided masses.

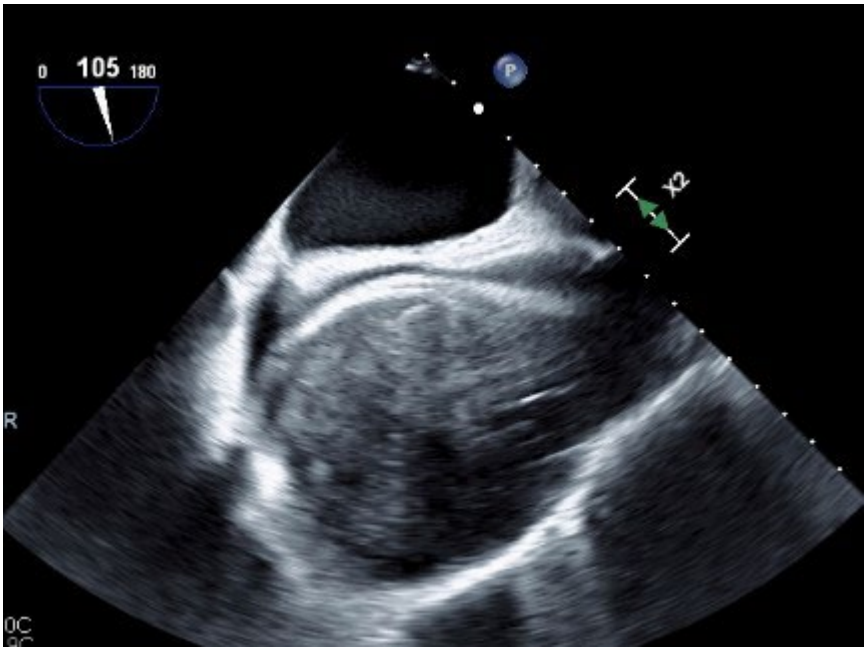
Images






0
98
180
cm/s


0
80
180
cm/s
89 bpm



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#FR 1 – Kiosk 16 – Poster 6

Peripartum management of mitral stenosis, a case report

Presenting Author: Cole Rinehart, MD, Resident Physician, University of Pennsylvania

Introduction

Mitral stenosis is a prevalent valvular disease in developing countries. It is a particularly complex lesion in the peripartum period that requires multidisciplinary discussion and planning. It can adversely impact maternal-fetal outcomes. Additionally, the physiologic changes of pregnancy may worsen previously asymptomatic mitral valve disease. Management of mitral valve disease in pregnancy can range from non-invasive monitoring and medical management to invasive monitoring and procedures including open mitral valve replacement and balloon valvuloplasty (PMBC)[1].

Case Presentation

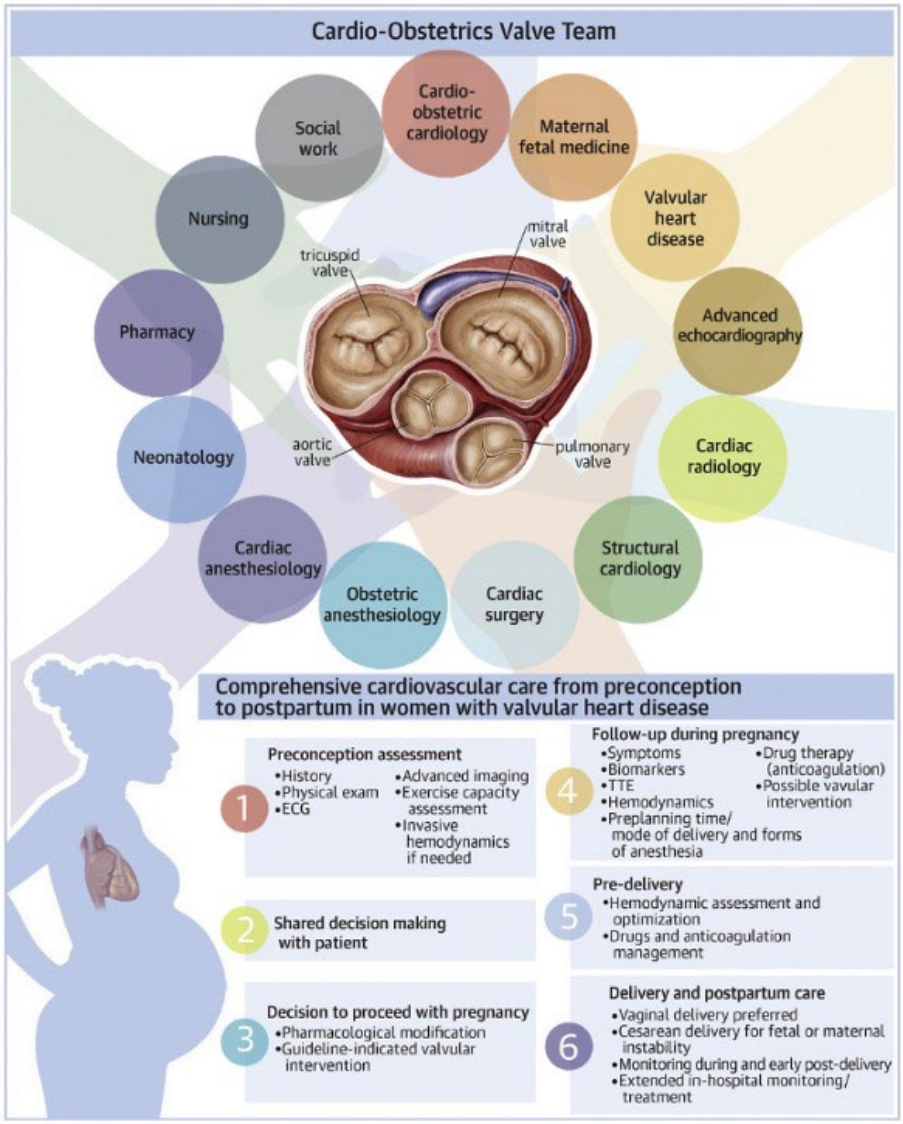
A 29 yo G2P0 with a congenital parachute mitral valve with severe mitral stenosis, bicuspid aortic valve with mild to moderate aortic stenosis, and chronic hypertension presented at 38 weeks and 1 day gestation (GA) for induction of labor due to superimposed preeclampsia with severe features. The patient was noted to have a murmur at birth and was followed through 18 years of age for a congenital parachute mitral valve. She was lost to follow up and reestablished care at 28 yo, at which time a transthoracic echocardiogram (TTE) was notable for moderate mitral stenosis (valve area 1.7 cm^2, mean gradient 7 mmHg). She was evaluated by Cardiology at 14 weeks GA and the decision was made not to pursue ballon valvuloplasty or other intervention given she was asymptomatic and her valve area was greater than 1.5 cm^2. TTE at 28 weeks GA was notable for severe mitral stenosis (valve area 1.1 cm^2, mean gradient 15 mmHg), bicuspid aortic valve with mild to moderate aortic stenosis (valve area 1.7cm^2, mean gradient 20 mmHg, peak velocity 275 cm/s), ejection fraction of 68%, and normal right ventricular size and function. Loop diuretics and beta blockade were both initiated. On presentation, a multidisciplinary discussion was had with Obstetric Anesthesia, Cardiac Anesthesia, Obstetrics, and Cardiology (Figure 1[3]). Early epidural analgesia was initiated and slowly titrated. The patient was monitored on continuous telemetry and continued loop diuretics and beta blockade with strict fluid management. Invasive monitoring with an arterial line and/or pulmonary artery catheter was considered but not performed. The patient had a spontaneous vaginal delivery and was discharged from the hospital five days after presentation.

Discussion

This abstract highlights challenges in both management and monitoring of a patient with severe mitral stenosis in the peripartum period. Multi center studies have shown a higher incidence of maternal morbidity and uteroplacental insufficiency, compromising the developing fetus[1]. Our case involves one patient who successfully had a spontaneous vaginal delivery with late medical management, slow and controlled initiation of neuraxial anesthesia, diligent non-invasive monitoring, and multidisciplinary teamwork. In other countries, the high risk of developing pulmonary edema during vaginal delivery makes planned cesarean section favorable[2]. Medical management should be initiated early[3]. Invasive monitoring of arterial and

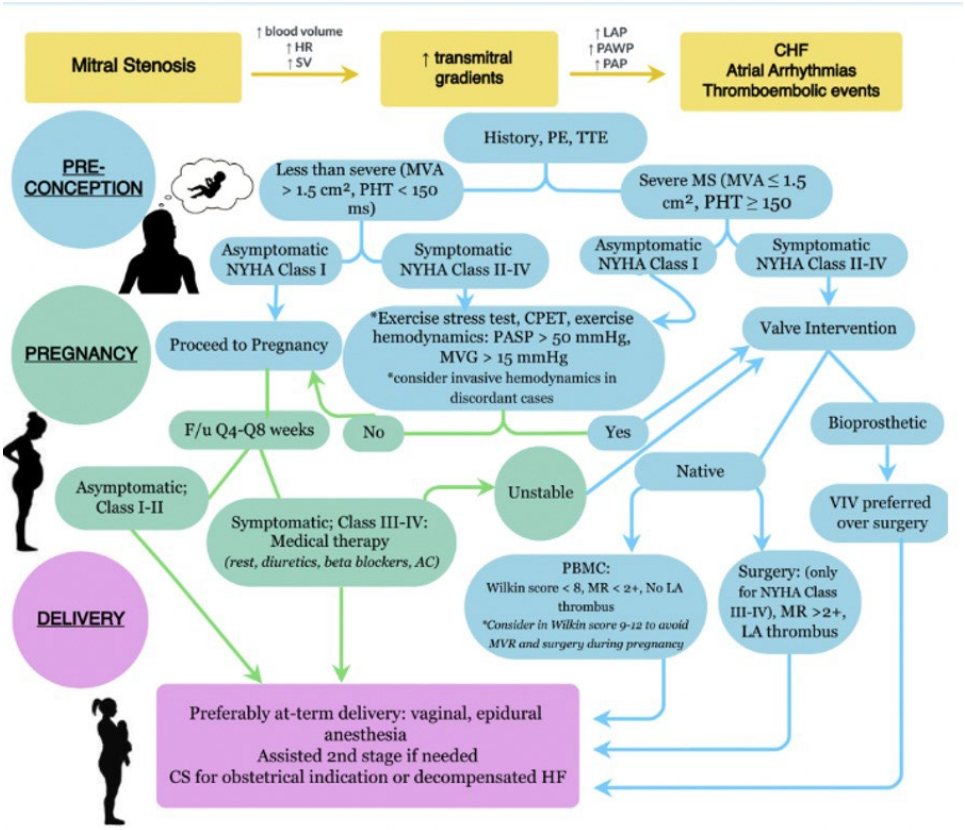
pulmonary artery pressures can be implemented to closely manage patient hemodynamics and volume status. The discussion for PMBC should be prior to conception, however it can be done after 20-weeks GA to minimize fetal radiation exposure (Figure 2[3]).

Images



Elkayam U, et al. JACC Adv. 2022;1(2):100022.

Elkayam, U, Bansal, P, Mehra, A. Catheter-Based Interventions for the Management of Valvular Heart Disease During Pregnancy. JACC Adv. 2022 Jun, 1 (2) . https://doi.org/10.1016/j.jacadv.2022.100022



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#FR 1 – Kiosk 16 – Poster 7

Sewing It Right: Robotic Two-Patch Fix for Right-Sided Partial Anomalous Pulmonary Venous Connection —An Anesthetic Perspective

Presenting Author: Nicholas Koenig, MD, West Virginia Universtiy Department of Anesthesiology

Introduction

Partial anomalous pulmonary venous return (PAPVR) with superior sinus venosus atrial septal defect (ASD) presents diagnostic and hemodynamic challenges, especially when combined with severe aortic insufficiency (AI) from bicuspid aortic valve disease.[1] Because PAPVR and sinus venosus defects are often missed on transthoracic echocardiography (TTE), transesophageal echocardiography (TEE) is essential for identifying true anatomy and associated congenital lesions.[2] Intraoperatively, TEE guides cannulation strategies and assesses the adequacy of complex repairs.[3] We aim to highlight the anesthetic and TEE considerations during robotic two-patch repair of PAPVR and sinus venosus ASD with concomitant aortic valve replacement via right mini-thoracotomy.

Case Presentation

Our patient is a 30-year-old man with a history of bicuspid aortic valve with severe AI, with worsening dyspnea, LV dilation, and reduced LV systolic function who was found to have previously undiagnosed PAPVR and superior sinus venosus ASD on preoperative cardiac CT angiography, despite years of routine TTE surveillance. General endotracheal anesthesia with a double lumen tube was used along with standard monitors, an arterial line, left sided central venous access and TEE. Intraoperative TEE demonstrated mildly dilated LV with LVEF 45%, bicuspid aortic valve with cusp prolapse and severe AI, a 1.5 cm superior sinus venosus ASD with bidirectional shunting, PAPVR draining into the SVC, and dilated right atrium. An 18 French SVC cannula was placed under TEE guidance proximal to the PAPVR drainage site. Additional cannulation included the right common femoral vein and artery with a distal perfusion catheter. After aortic cross-clamping, myocardial protection was achieved with cold direct ostial cardioplegia. Robotic-assisted mechanical aortic valve replacement was performed, followed by two-patch repair of the PAPVR and ASD with SVC augmentation using bovine pericardium. Post-repair TEE showed normalized biventricular function (LVEF 55%), a well-seated mechanical valve with normal leaflet motion and an 8 mmHg mean gradient, no paravalvular leak, complete rerouting of the right superior pulmonary vein into the left atrium, and full ASD closure without residual shunting. He was transported to the ICU intubated and extubated shortly after arrival without inotropic support.

Discussion

This case underscores the importance of TEE in hybrid congenital–acquired cardiac surgery. Preoperatively, TEE confirmed anomalous venous drainage, quantified AI severity, and excluded additional defects, which was essential since PAPVR and sinus venosus ASD had been missed on prior TTEs. Intraoperatively, TEE guided precise SVC cannula placement relative to the PAPVR drainage site, optimized venous return, and confirmed adequate venting and de-airing during minimally invasive bypass. Post-repair, TEE verified

mechanical valve function, successful PAPVR rerouting, and complete ASD closure—key elements of hemodynamic assessment. Real-time echocardiographic guidance is essential in managing complex congenital acquired cardiac pathology.

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#FR 1 – Kiosk 17 – Poster 1

Percutaneous Thrombectomy for Complete Left Ventricular Assist Device (LVAD) Occlusion Guided by Intraprocedural Transesophageal Echo (TEE)

Presenting Author: Daniel R. Evans, MD MSc MSc, Resident, Brigham and Women's Hospital

Introduction

Anticoagulation management in patients with left ventricular assist devices (LVADs) is frequently complicated by devastating bleeding and thrombotic events.

Case Presentation

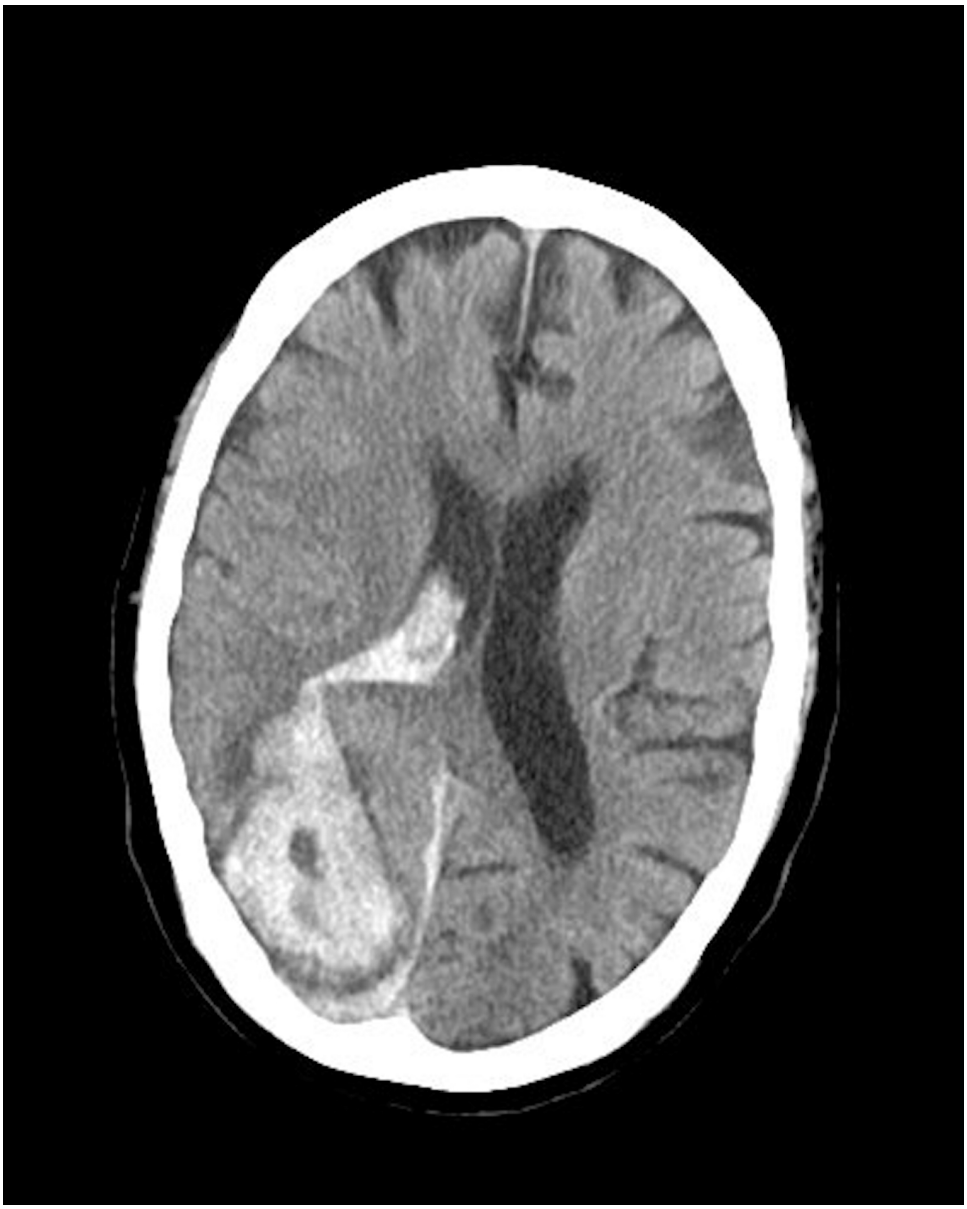
A 71-year-old man with end-stage nonischemic cardiomyopathy underwent implantation of a HeartMate 3 (HM3) LVAD. Postoperatively, he developed paroxysmal atrial fibrillation (PAF) and limited aortic valve opening with thrombus on the noncoronary cusp. He was discharged to rehab after 20 days with normal LVAD flows. One week later, he presented with left hemianopsia and PAF. CT demonstrated a large right occipital intraparenchymal hemorrhage with 3 mm midline shift (Fig. 1). Anticoagulation was reversed with vitamin K and K-Centra®. Neurologic examination showed left visual field deficit with preserved motor and sensory function and normal LVAD flow. He was admitted to the ICU for neurologic and anticoagulation management, then transferred to the floor. He was readmitted to the ICU after an episode of ventricular tachycardia, with TTE demonstrating a larger thrombus in the aortic root. He was managed medically and discharged several days later. While enroute home, he developed sudden LVAD failure and represented in cardiogenic shock with no LVAD support. CT demonstrated a large thrombus obstructing the LVAD inflow cannula. He was not a candidate for LVAD exchange. After goals-of-care discussions, the patient and family agreed to an off-label attempt at percutaneous thrombectomy to restore LVAD flow. He was emergently cannulated for VA-ECMO, intubated, and taken to the catheterization lab. TEE demonstrated a dilated left ventricle with a large thrombus over the LVAD inflow cannula (Figs. 2–3). Imaging was challenging due to ventricular dilation and metallic artifact. A Sentinel cerebral protection device was deployed. Under TEE guidance, transseptal puncture was performed and the thrombectomy cannula advanced into the left atrium. Using 2D and 3D TEE, the cannula was advanced into the left ventricle and positioned over the thrombus (Fig. 4). Although the thrombus was extracted (Fig. 5), the LVAD did not restart. Suspecting residual thrombus within the inflow cannula, a flexible wire was advanced through a new transseptal puncture into the LVAD inflow under TEE guidance. Upon wire passage, the LVAD abruptly restarted (Fig. 6), likely due to displacement of residual thrombus and restoration of flow, with left ventricular decompression confirmed on imaging. All devices were removed, the septostomy was closed, and the patient returned to the ICU. The following day, LVAD flows remained stable and ECMO support successfully weaned. He was extubated the following day, listed for heart transplantation, and transplanted one month later.

Discussion

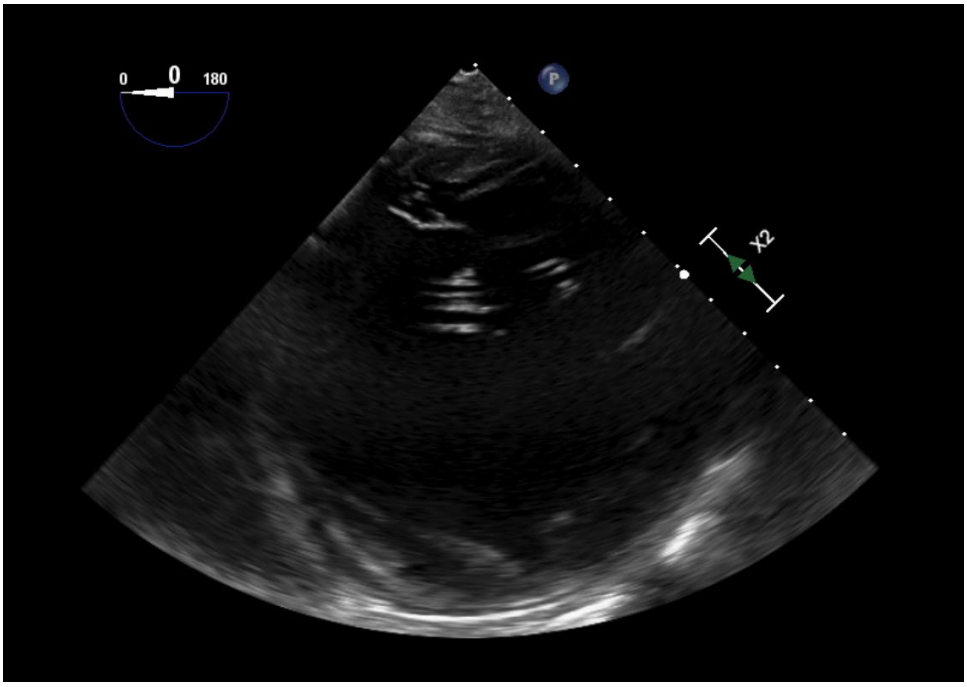
Anticoagulation management in LVAD patients is associated with high risks of both bleeding and thrombosis. This case highlights the successful off-label use of percutaneous thrombectomy for LVAD

inflow cannula obstruction causing pump failure, with TEE providing essential procedural guidance. Percutaneous thrombectomy may be considered in select high-risk patients after careful evaluation of risks and benefits.

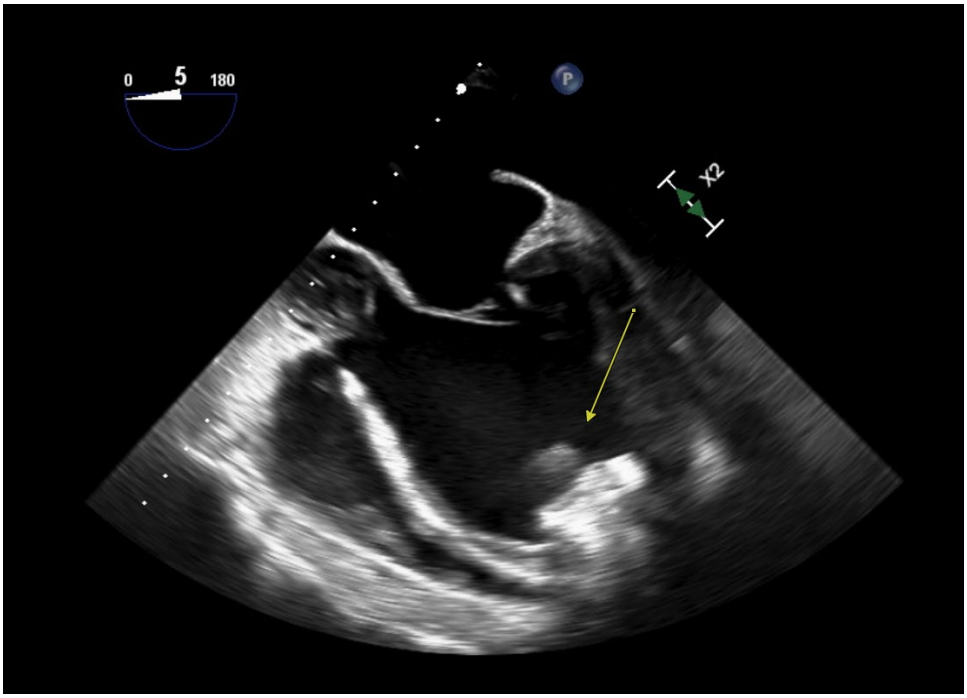
Images



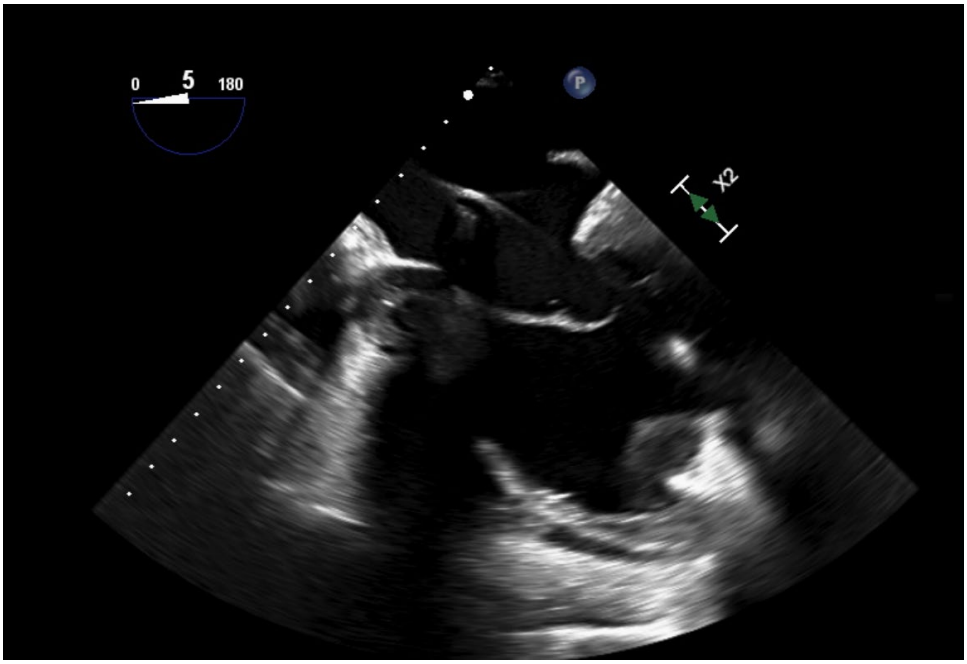
Head CT demonstrating a large right occipital intraparenchymal hemorrhage.



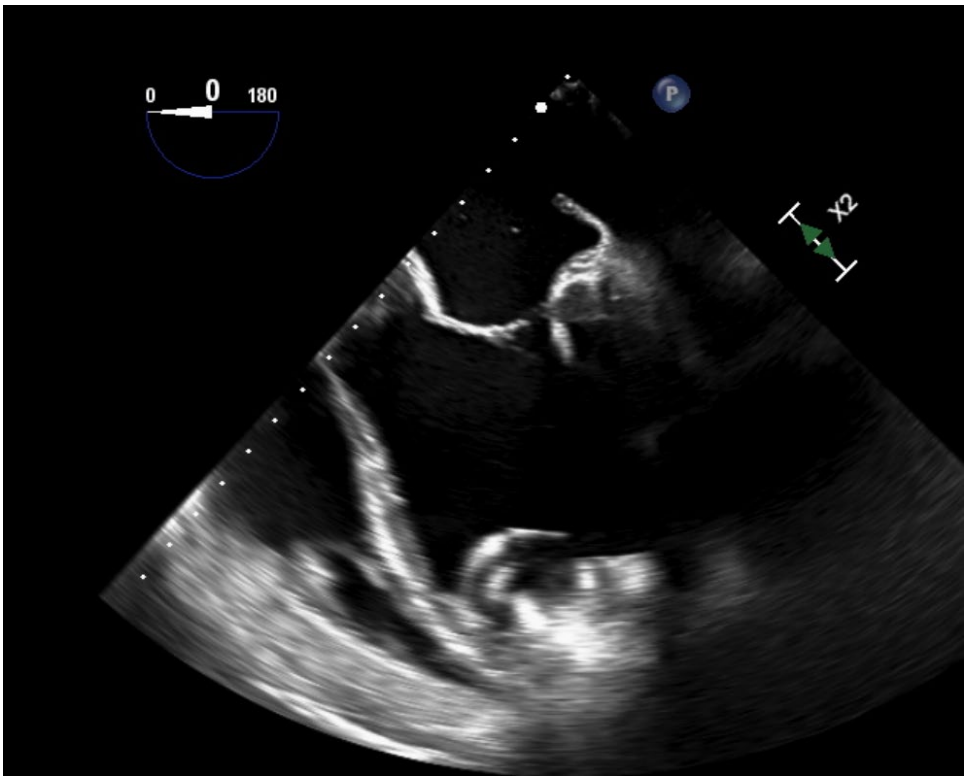
TEE image demonstrating dilated left ventricle.



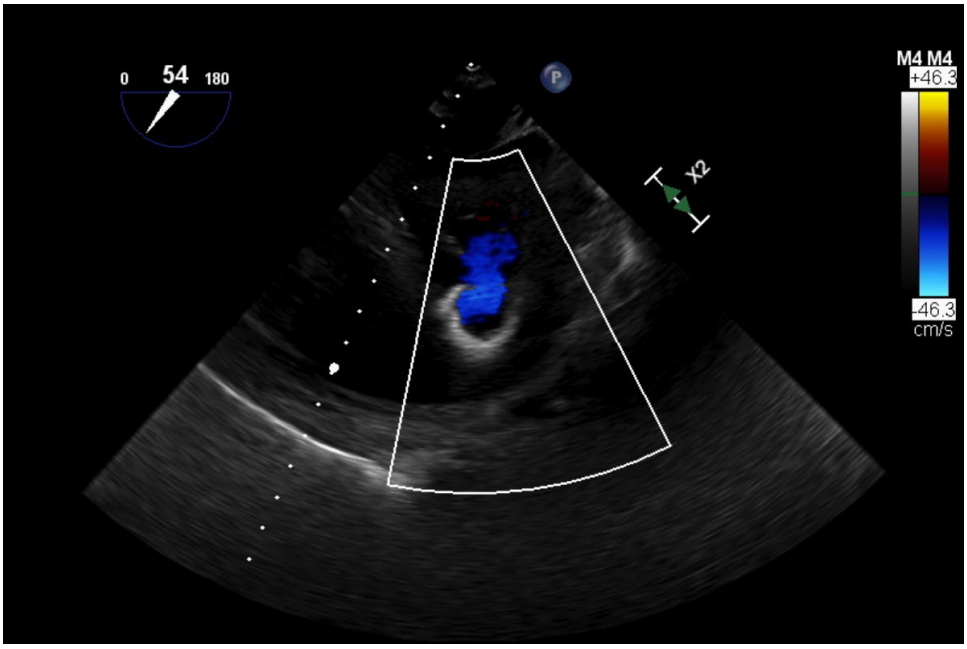
TEE image demonstrating thrombus obstructing LVAD inflow cannula.



TEE image demonstrating thrombectomy cannula and LVAD inflow thrombus.



TEE image demonstrating LVAD inflow cannula after removal of thrombus.



TEE image with Doppler demonstrating resumption of flow through LVAD inflow.

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#FR 1 – Kiosk 17 – Poster 2

VA ECMO as a Bridge to Combined Pulmonary Thromboendarterectomy and Mitral Valve Replacement

Presenting Author: Jenny Huang, MD, University of Texas Southwestern Medical Center

Introduction

Patients with concurrent chronic thromboembolic pulmonary hypertension (CTEPH) and left heart disease have an increased risk of morbidity and mortality. While CTEPH is characterized as precapillary pulmonary hypertension (PH) by chronic obstruction of pulmonary arteries with organized clot, left-sided valvular disease contributes to postcapillary PH with elevated left ventricular pressures.1 With this complex pathophysiology, we present the case of a patient with CTEPH and severe mitral stenosis (MS) on VA-ECMO as a bridge to definitive surgical therapy.

Case Presentation

The patient is a 25-year-old female with no past medical history who was transferred from an outside hospital with worsening shortness of breath since having COVID-19 two years ago. Imaging was notable for severe CTEPH. TTE showed severe MS (mean gradient 23 mmHg, valve area 0.4 cm2), suprasystemic pulmonary artery pressures (PAP), and right ventricular (RV) failure (Figure 1). Due to worsening shock, VA ECMO was initiated. Right heart catheterization demonstrated mean PAP 78 mmHg, PCWP 30 mmHg, and CI 2 L/min/m2. She was taken to the OR the next day for bilateral pulmonary thromboendarterectomy (PTE) and concurrent mitral valve replacement (MVR). The bilateral PTE was completed under deep hypothermic circulatory arrest for a total of 55 minutes (Figure 2). During rewarming, she received a mechanical mitral valve. Intraoperative TEE showed LVEF 55-60% on epinephrine 0.09 mcg/kg/min and dopamine 4 mcg/kg/min, moderate-severe RV dysfunction, and a well-seated mechanical valve (mean gradient 2 mmHg; Figure 3). She also required vasopressin 0.05 units/min and inhaled nitric oxide 40 ppm. Swanz Ganz thermodilution demonstrated a CI 2.39 L/min/m2, SVR 1274 dynes/sec/cm-5, and PVR 329 dynes/sec/cm-5. Hemodynamics in the operating room were favorable for ECMO decannulation. However, due to persistent RV depression and elevated mean PAP 42 mmHg, she was transported to the ICU with an open chest for hemodynamic stabilization. Her sternum was closed 2 days later. Postoperative TTE demonstrated LVEF 68% and mild RV dysfunction. She was started on sildenafil and warfarin. Postoperative course was complicated by left external iliac thromboembolectomy for acute limb ischemia, subarachnoid hemorrhage while bridging to warfarin, and pericardial effusion requiring a pericardial drain. She was discharged to inpatient rehabilitation after 29 days of hospitalization.

Discussion

This case demonstrates successful ECMO bridging to combined PTE and MVR. Combined pre- and postcapillary PH is associated with increased morbidity and mortality and likely contributed to her severe RV failure and cardiogenic shock.2 Prior studies showed that perioperative survival in combined PTE and cardiac procedures was similar to PTE alone.3 This case reinforces the importance of considering mixed pathology in patients with pulmonary hypertension and the role of ECMO as a bridge to definitive therapy.

Images

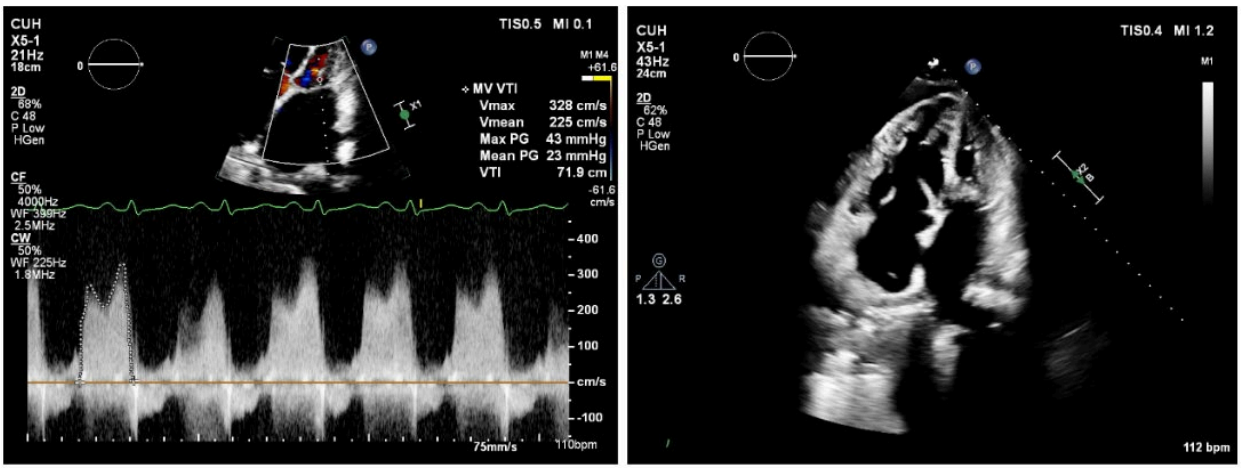


Figure 1: Preoperative TTE showing severe MS (mean gradient 23 mmHg) and enlarged RV.

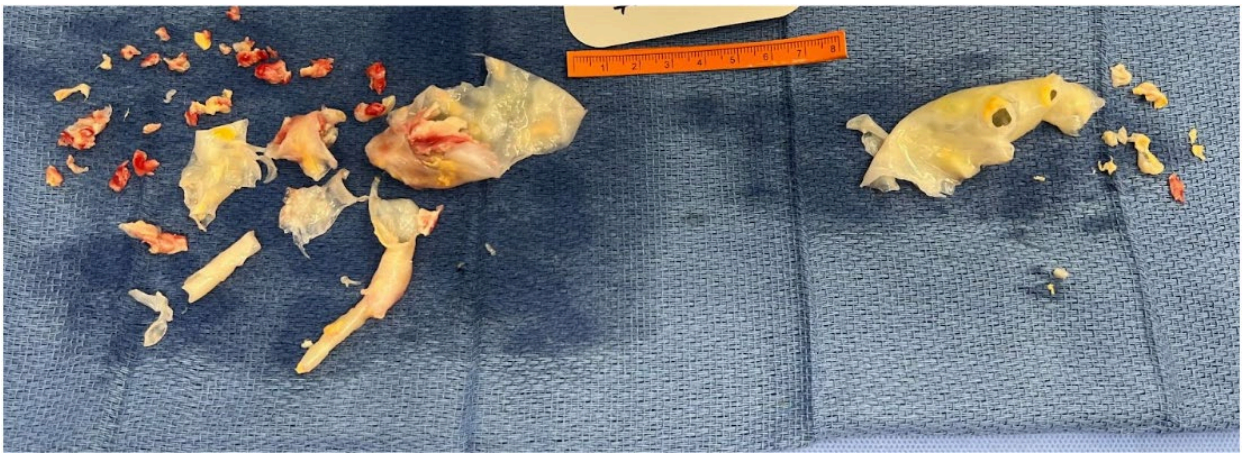


Figure 2: Excised clot from bilateral PTE

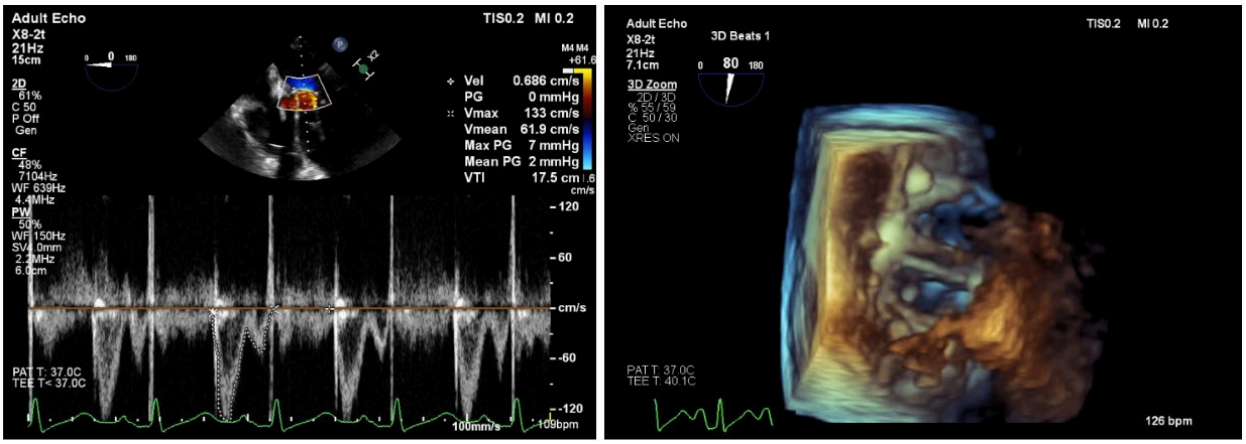


Figure 3: Intraoperative TEE showing a well-seated mechanical mitral valve with no evidence of significant leak and a mean gradient of 2 mmHg.

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#FR 1 – Kiosk 17 – Poster 3

Anesthetic Management for Pulmonary Thromboendarterectomy in the Setting of Suprasystemic Pulmonary Artery Pressures

Presenting Author: Alexandra Arendt, DO, Fellow, Adult Cardiothoracic Anesthesiology, Temple University Hospital

Introduction

CTEPH is a rare and complex disease of chronically elevated mean pulmonary arterial pressure. This elevated mPAP leads to an elevation of PVR and promotes a state or low oxygen tension promoting inflammation, coagulation, thrombosis, and scar formation in the pulmonary vasculature. Over time, these lesions exacerbate flow obstruction, ultimately resulting in right heart failure. PTE, where the built-up thromboembolic material is surgically removed from the pulmonary arteries, is the treatment of choice for CTEPH. Anesthetic management of these patients is complex, and induction of anesthesia may result in catastrophic hemodynamic compromise secondary to myocardial depression, hypotension, hypoxia, and hypercarbia in the setting of elevated right-sided pressures and PVR.

Case Presentation

63-year-old female with history of HTN, HLD, DM2, GERD, and hypothyroidism with CTEPH, acute on chronic right heart failure, and acute on chronic PE presented for PTE after experiencing worsening dyspnea over the course of a year. Her preoperative testing by TTE demonstrated a preserved LVEF 65% with a severely dilated and dysfunctional RV and severe TR. Hemodynamic measurements showed a systemic pressure of 122/81(110) mmHg with near-systemic PA pressures of 110/43(69) mmHg and an PVR of 22WU. Anesthetic plan: We placed a pre-induction brachial arterial line, and induction was performed under continuous TTE monitoring [Image 1]. Induction was achieved with midazolam, fentanyl, and sevoflurane with concomitant infusion and bolus doses of epinephrine. The surgical team was scrubbed in and prepared for emergent initiation of ECMO via femoral cannulation. She was intubated with a single lumen endotracheal tube under direct laryngoscopy. Following induction and intubation, a TEE probe was placed as well as a central line and pulmonary artery catheter. Her initial pulmonary artery pressures following PAC placement were 133/68 (91) compared to systolic arterial pressure of 114/69 (83) [image 2]. TEE findings intraoperatively demonstrated a severely dilated and hypertrophic right ventricle with intraventricular septal flattening in both systole and diastole, indicating right ventricular volume and pressure overload [Image 1]. Her intraoperative course was uneventful. Total CPB time was 171 minutes with aortic cross-clamp time of 98 minutes and circulatory arrest time of 57 minutes (20min right side, 25 min left side). Postoperative hemodynamics showed an almost 50% reduction in right heart pressures with PA 50/26/35 and preserved LV function. She was extubated on POD1 and transferred out of ICU on POD2. She remained on heparin infusion and subsequently transitioned to warfarin for substantial acute clot noted intraoperatively.

Discussion

This was a complex case of a patient undergoing PTE for CTEPH in the setting of right heart pressures

exceeding that of systemic circulation. The combination of anxiolysis, a combined intravenous and inhalational technique with inotropic support, careful monitoring with pre-induction invasive arterial blood pressure monitoring and TTE, and close collaboration with the perfusion and surgical teams helped us to provide a safe anesthetic for this patient without major alterations to hemodynamics.

Images

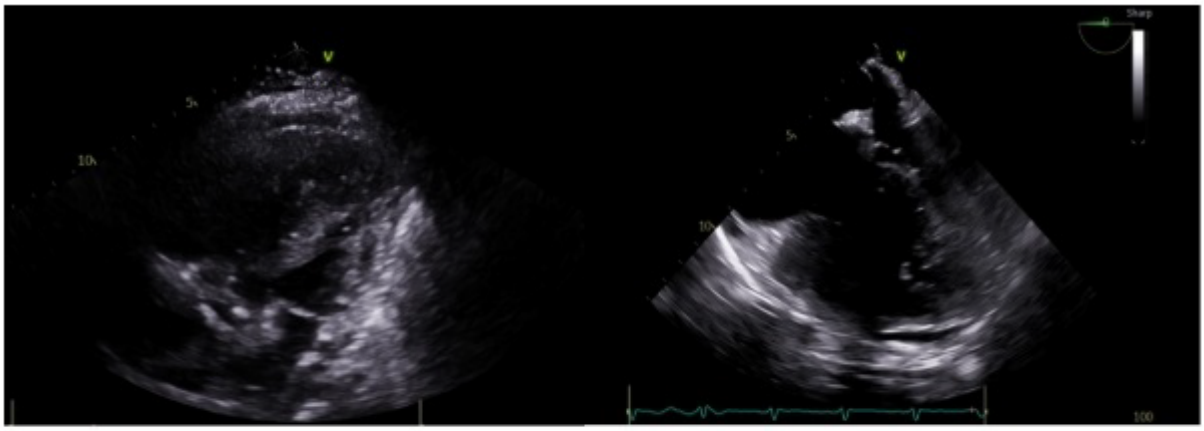


Image 1: Apical 4-Chamber view of TTE performed during induction (right) and ME-4 Chamber view of TEE performed following induction (left) demonstrating severe dilation of the right ventricle and right atrium compared to the left.



Image 2: Intraoperative monitoring showing systemic arterial pressure (red) and pulmonary artery pressure (yellow).

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#FR 1 – Kiosk 17 – Poster 4

Lyme Carditis in a Transplanted Heart: A Rare Cause of Early Conduction Failure

Presenting Author: Brian J. DelPino, D.O., Cardiac Anesthesiology Fellow, Duke

Introduction

Cardiac involvement in Lyme disease is uncommon and usually presents as transient conduction disease or myocarditis; Lyme endocarditis is extremely rare and, to our knowledge, has not been reported in a donor cardiac allograft. Early conduction disturbances after orthotopic heart transplantation (OHT) are usually attributed to surgical trauma, ischemia–reperfusion injury, or rejection. We describe presumed donor-derived Lyme carditis in a transplanted heart presenting with complete heart block, fever, and tamponade, diagnosed on routine surveillance biopsy.

Case Presentation

A 29-year-old man with arrhythmogenic right ventricular cardiomyopathy, severe right ventricular dysfunction, recurrent ventricular arrhythmias, and an implantable cardioverter-defibrillator underwent donation-after-circulatory-death OHT with uncomplicated implantation. He received basiliximab induction with steroids, mycophenolate, and tacrolimus. Extubated on postoperative day (POD) 0, he developed fevers, leukocytosis, and atrial flutter with slow ventricular response, progressing to complete heart block on POD 5. Despite normal graft function on echocardiography, he required epicardial pacing and inotropes. Crossmatch was negative, no donor-specific antibodies were detected, and his POD 8 biopsy showed no cellular or antibody-mediated rejection. Because the degree of conduction disease was unexplained, electrophysiology and transplant infectious diseases were consulted. Donor chart review revealed recent “deer camp” exposure. Lyme testing was sent, and Warthin–Starry staining was added to the biopsy. Before results returned, he underwent cardioversion, briefly entered sinus rhythm, then developed hemodynamic collapse with echocardiographic and catheter-based evidence of tamponade. He returned to the operating room, where ~350 mL of sanguineous pericardial fluid and clot was evacuated without a clear bleeding source. He stabilized but remained pacer-dependent with persistent leukocytosis and chills; serial blood cultures were negative. On POD 12, Warthin–Starry staining from the initial biopsy demonstrated spirochetes consistent with *Borrelia* species; initial serologies were negative. He was diagnosed with severe Lyme carditis of the allograft and started on IV ceftriaxone with a short doxycycline course. Mycophenolate was reduced. Over the next 1–2 weeks, leukocytosis resolved and intermittent intrinsic sinus rhythm returned, though high-grade block persisted and a permanent pacemaker was placed before discharge. Graft function remained normal and later biopsies showed no rejection.

Discussion

This case demonstrates donor-derived Lyme carditis as a rare cause of early complete heart block after OHT. Clues included conduction disease out of proportion to graft function, a negative rejection evaluation, and donor epidemiologic risk only recognized on review. Diagnosis required special staining of a routine biopsy, guiding antimicrobial therapy and immunosuppression adjustments. The tamponade episode

without a surgical source may have reflected inflamed allograft tissue in the setting of carditis and anticoagulation. Clinicians should consider tick-borne infection when early conduction abnormalities and hemodynamic instability after OHT remain unexplained.

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#FR 1 – Kiosk 17 – Poster 5

Deployment of percutaneous vascular plug devices to address severe prosthetic pulmonic valve paravalvular leak

Presenting Author: Olesia Merinova, MD, PhD, Fellow, University of Alabama at Birmingham

Introduction

Pulmonary valve replacement (PVR) is a commonly performed intervention for late pulmonary regurgitation (PR) in patients with previously repaired Tetralogy of Fallot (TOF) [1]. A significant late complication of PVR is paravalvular leak (PVL), a defect that re-imposes right ventricular volume overload and can lead to heart failure and anemia due to hemolysis. Transcatheter PVL closure has emerged as a less invasive alternative to reoperative open surgical valve replacement [2]. This case contributes to the limited literature reporting successful percutaneous closure of prosthetic pulmonary valve PVL.

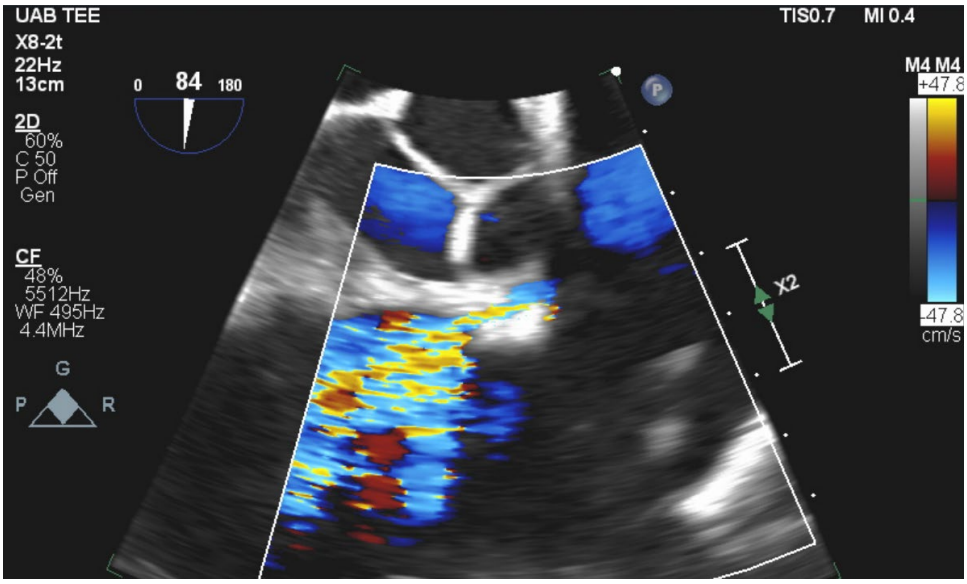
Case Presentation

A 34-year-old female with a history of repaired TOF in childhood followed by implantation of a 29 mm bioprosthetic valve in the native pulmonic position 4 years prior for endocarditis-related dehiscence, presented with progressive shortness of breath. Echocardiography demonstrated severe right ventricular enlargement and a severe prosthetic pulmonic PVL. Given her high risk for open reoperation, she was referred for percutaneous transcatheter closure of the PVL. The procedure was performed under general anesthesia with TEE and fluoroscopic guidance. Two 16 mm Amplatzer AVP-II vascular plugs (Abbott, Abbott park, IL) were successfully deployed across in stable “multi-plug” fashion and the large defect was closed. TEE demonstrated a reduction of the PVL from severe to mild. The patient reported a complete resolution of her shortness of breath after conclusion of the procedure and was discharged in good condition on dual anti-platelet therapy. A TTE performed on POD 30 demonstrated that both plugs remained in a stable position with mild residual PVL and a maximal transvalvular gradient of 17.2 mmHg.

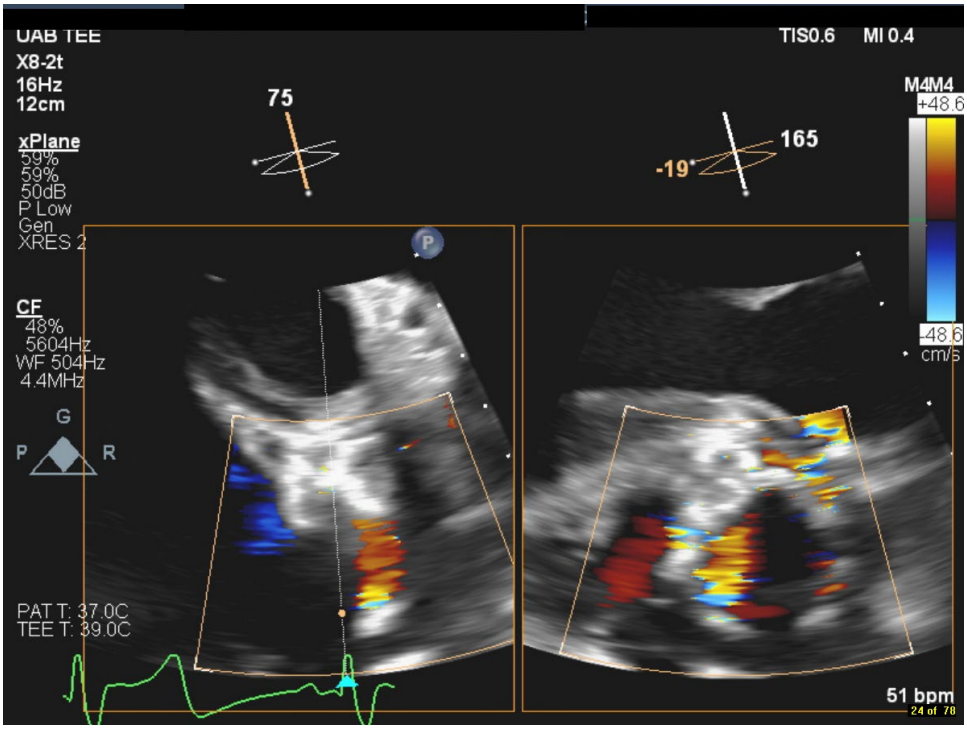
Discussion

This case demonstrates the successful transcatheter closure of a severe prosthetic pulmonic PVL. PVL is a potential complication of transcatheter PVR in the adult congenital heart disease population that can lead to substantial symptomatic burden and morbidity. This report highlights the feasibility of adapting techniques and devices— such as the "multi-plug" use of AVP-II occluders—from the more common left-sided PVL occlusion procedures to the unique, low-pressure, and often complex post-surgical right-sided procedures. This report contributes to the sparse literature and supports transcatheter closure as a viable, effective, and low-morbidity alternative to high-risk redo-surgery for this challenging clinical problem [3]. Management of complex pulmonic PVL with deployment of vascular plug devices is feasible and can achieve durable results.

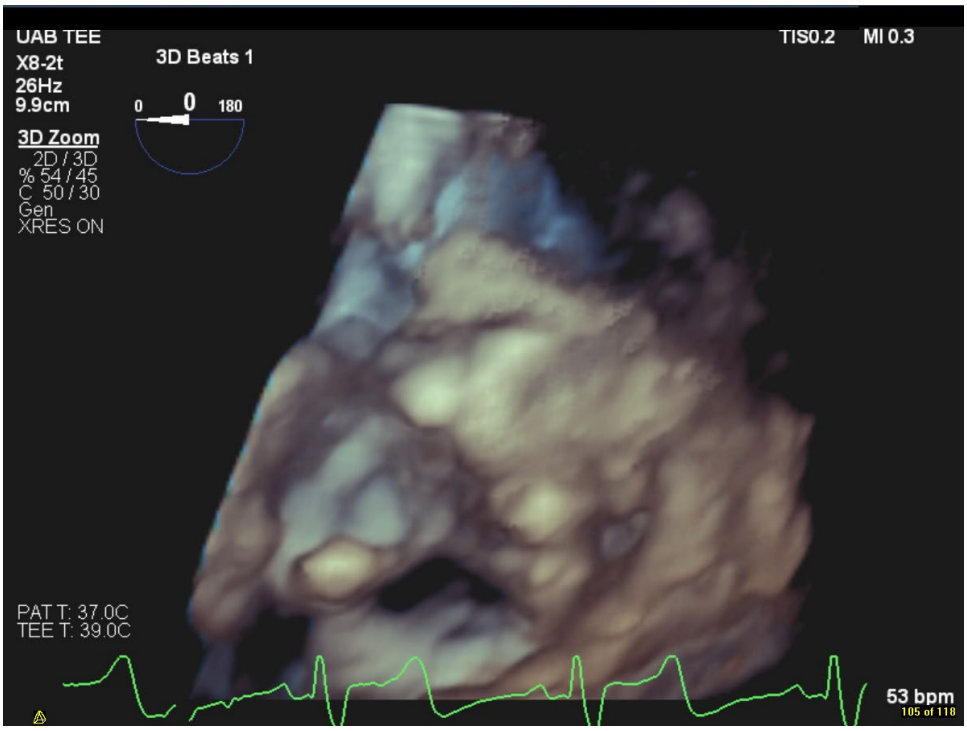
Images



Pre-intervention right ventricle inflow-outflow view of the prosthetic PV severe PVL.



Post-intervention right ventricle inflow-outflow view with x-plane through the prosthetic PV demonstrating residual PVL shortly after deployment of two Amplatzer vascular plugs. Reduction of PVL from severe to mild appreciated.



Post-interventional 3D of prosthetic PV with two 16mm Amplatzer AVP-II vascular plugs appreciated.

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#FR 1 – Kiosk 17 – Poster 6

Pulmonary Artery Obstruction after Aortic Repair

Presenting Author: Daniel P. Schneider, MD, Anesthesiology Resident, Medical College of Georgia

Introduction

The right ventricle's distinct anatomy—including its three segments, trabeculated structure, and predominantly longitudinal contraction—creates unique mechanical behavior and a sensitivity to pressure loading that differs from the left ventricle. Its thin, crescent-shaped geometry and shared septal-pericardial interactions further influence both normal physiology and disease manifestations. Echocardiography, guided by ASE standards, serves as the cornerstone for assessing these features, enabling accurate hemodynamic evaluation and reliable monitoring of right-sided pathology such as pulmonary hypertension. Right ventricular failure (RVF) typically presents as an inability to separate from cardiopulmonary bypass (CPB) or as low cardiac output syndrome, often leading to multi-organ dysfunction in the postoperative period. We describe here the perioperative considerations and challenges in a patient who developed severe RV dysfunction after Type A aortic dissection repair, ultimately requiring veno-arterial extracorporeal membrane oxygenation (VA ECMO).

Case Presentation

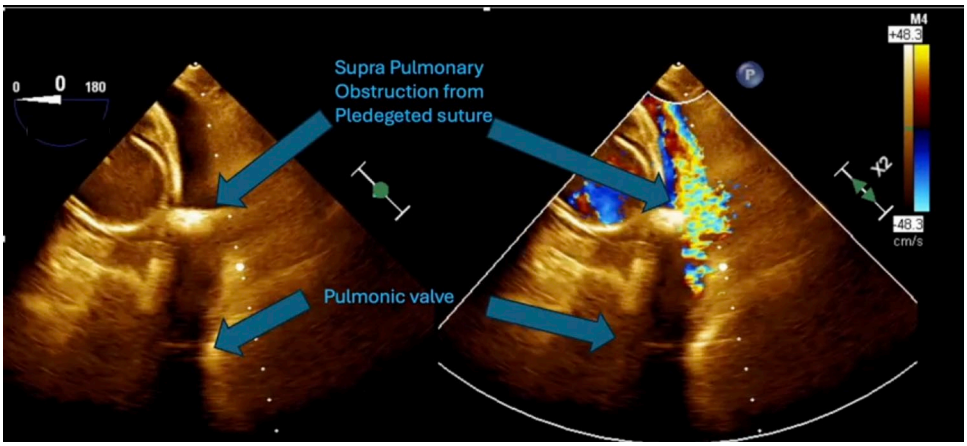
A 57-year-old female with a history of hypertension and tobacco use presented with severe chest pain. CT angiography revealed a Type A aortic dissection extending from the aortic root to the left subclavian artery. She underwent emergent aortic repair using deep hypothermic circulatory arrest. Following cardiopulmonary bypass, the patient was difficult to wean due to biventricular failure, requiring high-dose inotropes and an intra-aortic balloon pump, which was subsequently upgraded to an Impella 5.5. Ultimately, she was placed on VA ECMO for refractory shock and severe RV failure, and her chest was left open. Over the ensuing course, she remained unable to be weaned from ECMO. Transesophageal echocardiography (TEE) demonstrated a dilated RV with preserved left ventricular function. Left heart catheterization showed a patent right coronary artery with good flow. Severe tricuspid regurgitation was noted, along with a 30–35 mmHg gradient between Doppler-derived RV systolic pressure and direct pulmonary artery catheterization measurements. Further evaluation revealed supravalvular pulmonary artery obstruction, with a differential diagnosis including pulmonary embolism versus iatrogenic stenosis from the initial surgical repair. Upon re-exploration, surgical assessment identified pulmonary artery injury with a pledgeted repair causing stenosis just above the pulmonic valve. The pledgets were removed and a pericardial patch repair was performed. Following this intervention, the patient's RV function improved significantly, enabling successful ECMO decannulation and rapid reduction in inotropic and vasopressor support. She was discharged from the ICU three days later.

Discussion

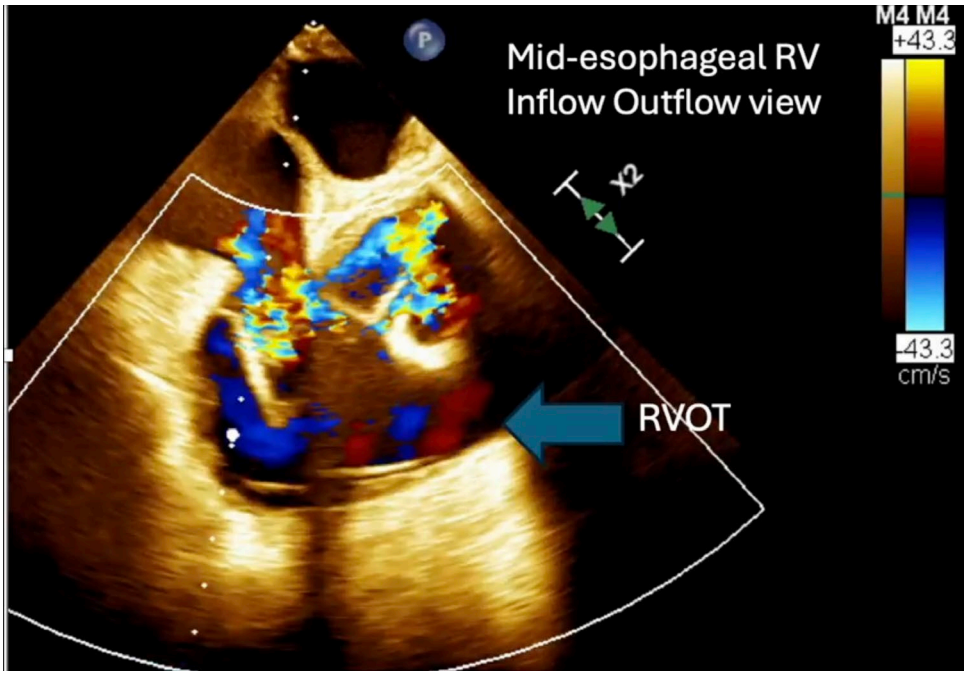
RV failure can arise from multiple etiologies, including volume overload, pressure overload, myocardial disease, and pericardial pathology. In this case, RV failure manifested as an inability to wean from ECMO

due to pressure overload from a fixed pulmonary artery obstruction caused by surgical repair. A high degree of clinical suspicion—combined with a thorough TEE examination—was crucial in identifying this life-threatening lesion.

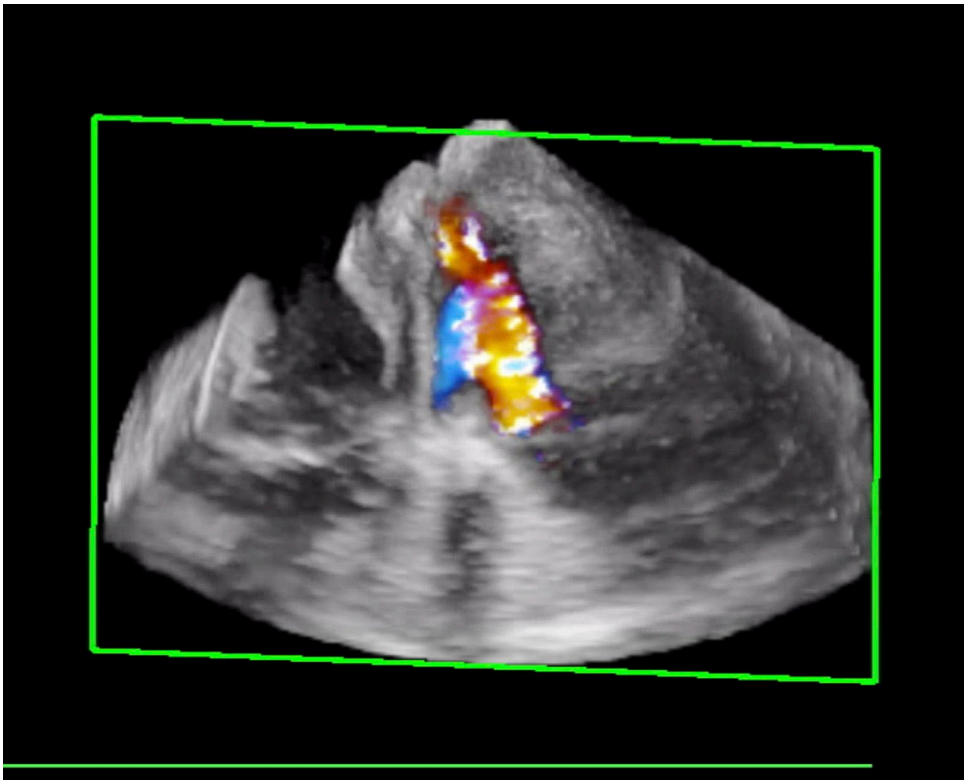
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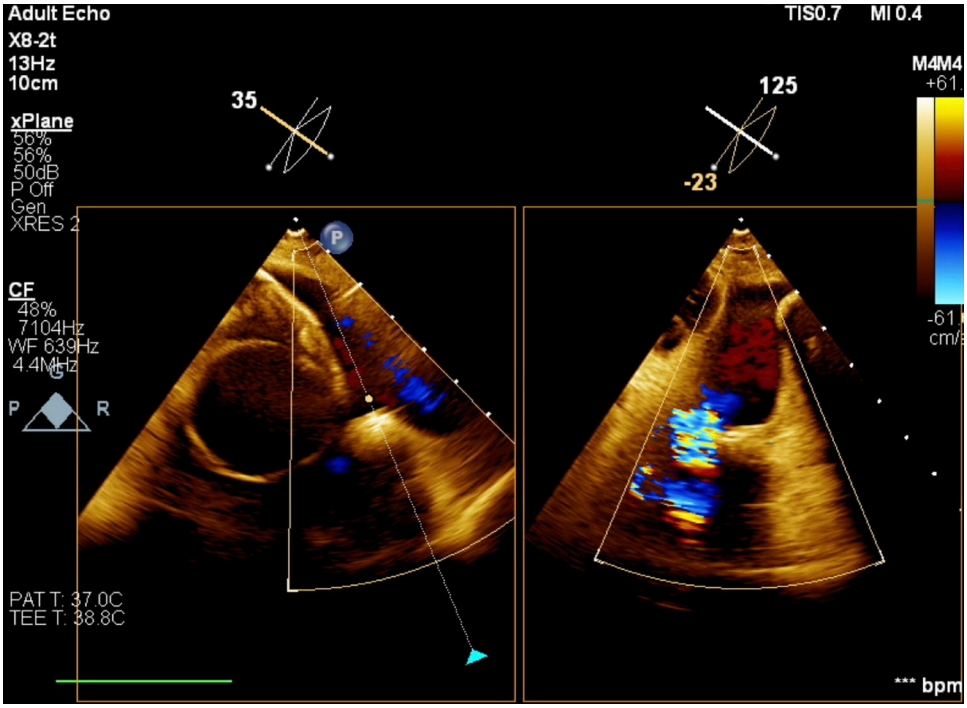
Ascending Aorta Short Axis



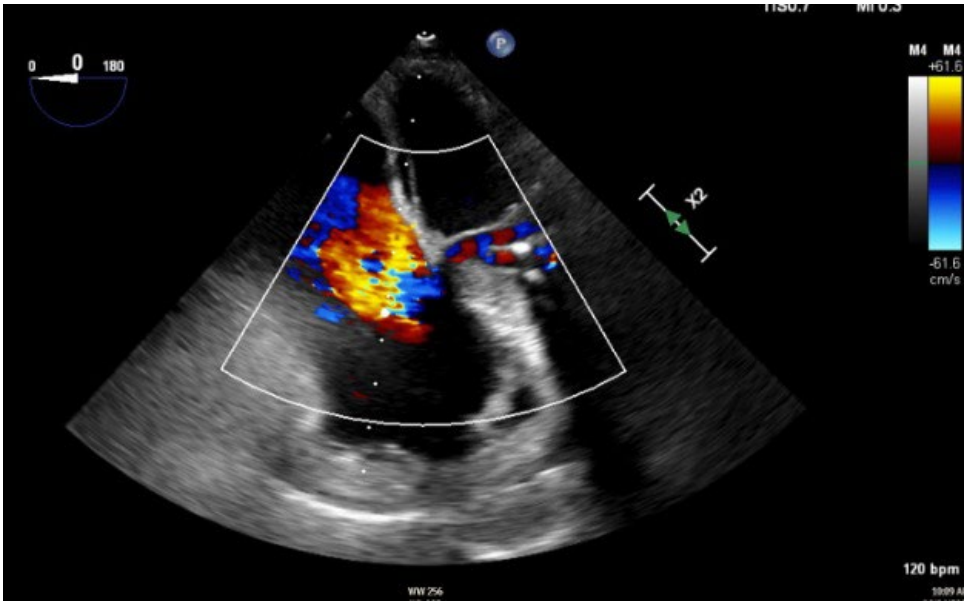
Mid-esophageal RV Inflow Outflow



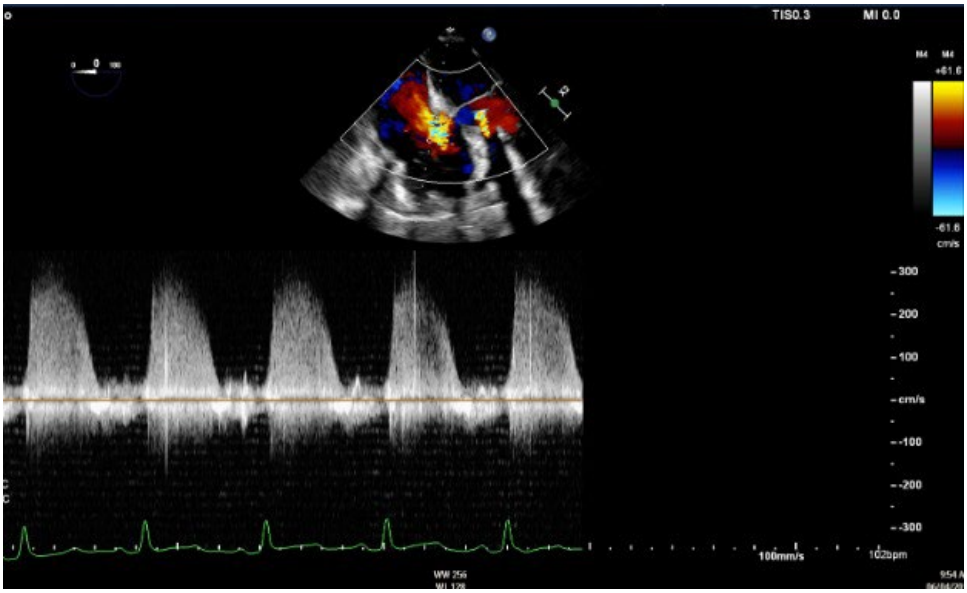
3D Rendering of Flow Acceleration in Pulmonary Artery



Bi-plane of Supra-pulmonic valve obstruction



RV Dysfunction



Tricuspid regurgitation

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#FR 1 – Kiosk 17 – Poster 7

Between a Rock and a Hard Place: Supporting the Right Ventricle in the Context of Systolic Anterior Motion of the Mitral Valve

Presenting Author: Jayce J. Breig, MD, Fellow, Cleveland Clinic

Introduction

Tricuspid annuloplasty (TA) during mitral valve repair (MVr) in patients with tricuspid regurgitation (TR) is associated with decreased progression of TR, reoperation for TR, and death (1). However, TA is not without risk. Here we present a case of severe right ventricle (RV) dysfunction after MVr and TA complicated by inotrope induced systolic anterior motion (SAM) of the mitral valve.

Case Presentation

This is a case of a 67-year-old male presenting with worsening fatigue in the context of severe mitral regurgitation (MR) who underwent elective robotic MVr and TA. Intraoperative transesophageal echocardiography (TEE) revealed normal biventricular function, prolapse of the P1 and P3 segment of the mitral valve, and moderate TR with a dilated annulus. The patient was deemed to be at low risk for SAM after repair, based on C-sept measurement, anterior to posterior leaflet height, and left ventricular (LV) size. The surgical approach included a P2 resection, sliding repair, and ring annuloplasty. TA was then performed. As cardiopulmonary bypass (CPB) was weaned, the patient immediately developed severe hypotension. On TEE, the RV was severely dilated and hypokinetic despite attempts to improve function with inotropes and vasopressors. Surgically, there was concern that the TA distorted the right coronary artery (RCA), and a decision was made to remove the TA. With the next attempt to separate from CPB, inotropes and vasopressors were continued; however, hypotension persisted, and the RV remained severely dysfunctional. With significant use of inotropes and vasopressors, the patient developed obstructive SAM. The team decided to return to CPB and repair the mitral valve with posterior neochords. In the final separation from CPB, the patient remained on a stable dose of inotropes and vasopressors, with interval improvement in hemodynamics. TEE demonstrated a resolution of SAM, and the RV improved to moderate dysfunction with trivial TR. The patient's ICU stay was complicated by prolonged intubation, prolonged wean of inotropic support, and renal failure.

Discussion

This case highlights the complexity of tricuspid annuloplasty during mitral repair. RCA injury in TA can occur from direct obstruction or kinking of the artery from changes in annular shape (2). Patients with atherosclerotic arteries or dilated annuli may be at higher risk for RCA injury (2). To support RV failure, inotropes are initiated which may precipitate SAM and circulatory failure. Management of RV failure in the setting of SAM is complex, as the pathologies require competing goals. The RV is unable to deliver adequate preload to the LV. Meanwhile, inotropic support increases heart rate and decreases LV filling time. While SAM occurs, cardiac output is reduced and RV perfusion suffers. Multiple medical and surgical interventions could be considered. A proposed decision tree (Figure 1) could assist teams in perioperative decision

making. While it is recommended that TR be addressed during left-sided heart surgery (class 2a recommendation [3]), complications may arise from an additional surgical procedure.

Images

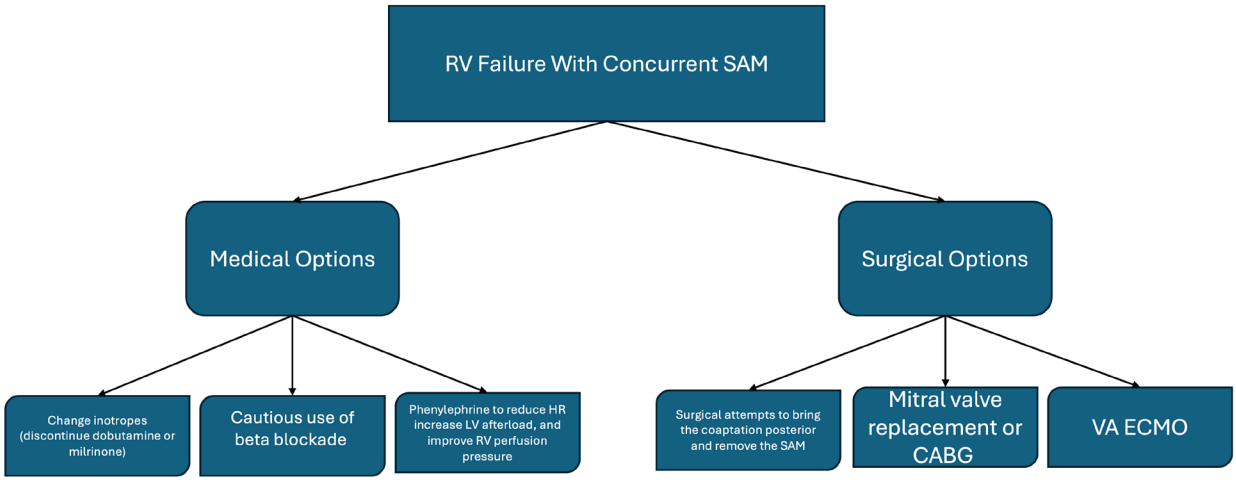


Figure 1. Decision tree for teams facing RV failure and SAM.

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#FR 1 – Kiosk 18 – Poster 1

From Stent to Sternotomy: Anesthesia for Sequential Ascending and Descending Aortic Dissection Repair

Presenting Author: Lucy Zhuo, M.D., Resident, University of Rochester Strong Memorial Hospital

Introduction

Complex aortic pathology necessitating staged surgical repair presents exceptional perioperative risk. We describe the anesthetic management of a critically ill patient presenting with a prior Thoracic Endovascular Aortic Repair (TEVAR) failure, an uncommon coexisting ascending and descending aortic dissection, and evidence of impending mesenteric malperfusion requiring two high-risk procedures: urgent endovascular visceral stenting followed by definitive open aortic arch replacement. This case highlights the intricate anesthetic decision-making required for safe progression through multidisciplinary staged repair.

Case Presentation

A 41-year-old male with a history of hypertension, Type B aortic dissection (with prior emergent TEVAR) requiring left subclavian to carotid transposition, and pacemaker for bradycardic arrest, presented with severe acute abdominal pain several days following a recent uncomplicated distal TEVAR extension for an endoleak. Imaging was concerning for both acute mesenteric ischemia secondary to a mobile dissection flap and an occult Type Ia endoleak. To restore visceral perfusion and mitigate the risk of acute malperfusion during the anticipated cardiopulmonary bypass (CPB) required for arch repair, the patient underwent urgent additional TEVAR extension and stenting into the infrarenal abdominal aorta. Anesthetic management for this initial procedure involved general anesthesia with standard induction and post-induction placement of a radial arterial line. The case was uncomplicated, and the patient was successfully extubated. Several days later, the patient underwent staged Zone 2 Aortic Arch Replacement with debranching of the left common carotid and innominate artery, requiring CPB and deep hypothermic circulatory arrest (DHCA). Patient required general anesthesia, post-induction central line, arterial line placement, and high-dose pressor infusions (phenylephrine, norepinephrine, vasopressin). Post-operatively, the patient was transported to the cardiovascular intensive care unit, and he was successfully discharged days later without complications.

Discussion

This case exemplifies the challenges of managing complex, staged aortic repairs in critically ill patients. The initial urgent TEVAR extension was an essential risk-reduction strategy, mitigating the catastrophic complication of acute mesenteric ischemia during the subsequent arch repair that would require CPB. For the endovascular case, the focus was on strict blood pressure control via continuous arterial monitoring to optimize the stent landing zone and minimize forces on the fragile aorta. Success for the open arch case was dependent on successful infrarenal aortic stent placement. Proactive hemodynamic optimization was paramount for decreasing long-term ischemic complications, involving meticulous titration of multi-agent vasopressor infusions and advanced monitoring to maintain cerebral perfusion during DHCA. The successful outcome underscores the necessity of multidisciplinary planning and individualized, proactive

anesthetic titration as essential components for optimizing safety and outcomes in patients with rare, evolving, and severe aortic dissection requiring staged, technically complex procedures.

Images

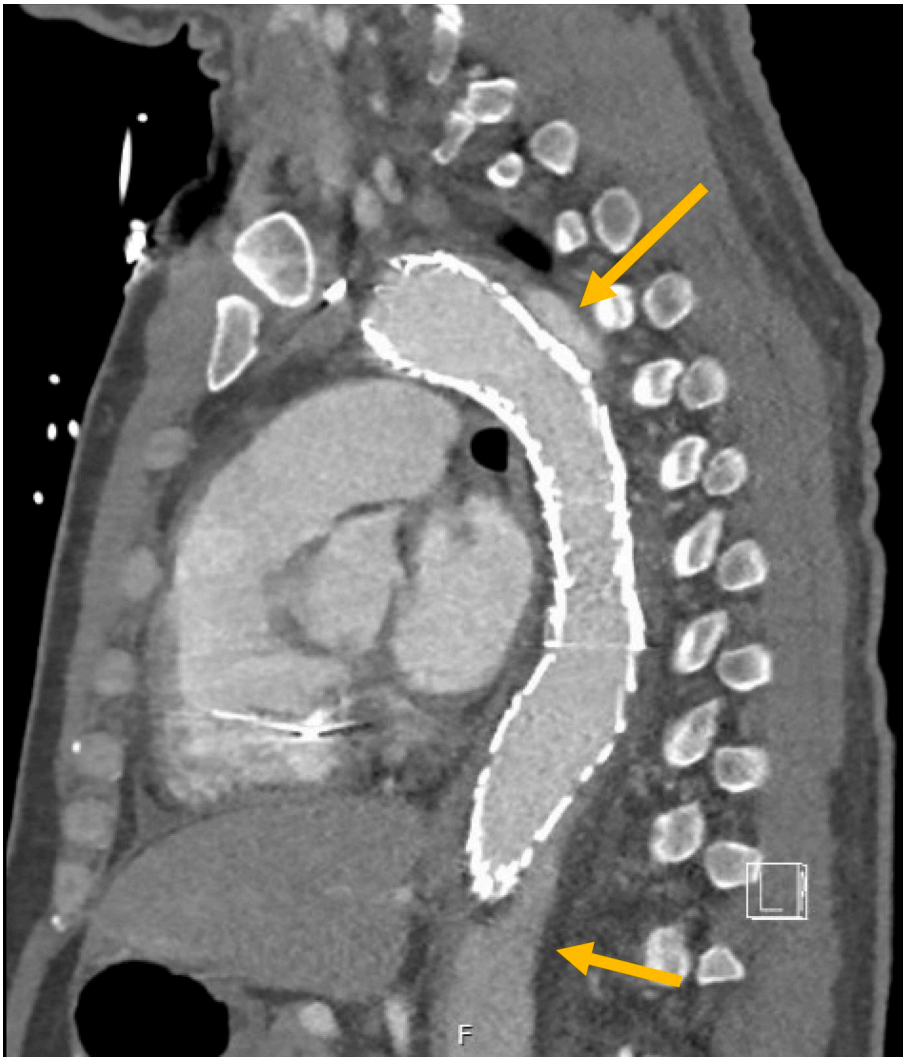


Figure 1. Pre-operative CT chest angiogram, arrows pointing to areas of aortic dissection

References

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#FR 1 – Kiosk 18 – Poster 2

Post-Myocardial Infarction Ventricular Septal Defect: A Complex Case Report

Presenting Author: Matthew A. Diaz, M.D., Resident physician, Jackson Memorial Hospital

Introduction

Post-myocardial infarction (MI) ventricular septal defects (VSD) are rare (~ 0.2 of acute MI) but highly lethal, with mortality approaching 100% without surgical repair. This case highlights anesthetic management for urgent VSD repair following inferior MI complicated by severe pulmonary hypertension and biventricular dysfunction.

Case Presentation

A 64 year old female with a history of CAD with prior anterior STEMI and LAD stent, hypertension, diabetes, and tobacco use presented to the ED with chest pain and shortness of breath. ECG revealed an inferior STEMI. She underwent emergent cardiac cath, revealing a 100% mid-RCA occlusion, moderate LAD/LCx disease. The RCA was stented successfully and an intra-aortic balloon pump (IABP) was placed for hemodynamic support. Post-PCI, TTE showed reduced LV function (30-35%), with inferior akinesis, a large muscular VSD, moderate MR, a severely dilated RV with severely reduced systolic function, moderate TR, and evidence of severe pulmonary hypertension (RVSP ~ 80 mmHg). Due to worsening biventricular function and a large VSD, urgent repair was planned. In the OR, an awake radial arterial line and PA catheter were placed. Milrinone, vasopressin, and inhaled nitric oxide were started before induction. General anesthesia was induced. Pre-bypass TEE revealed an inferior septal aneurysm with connection to a large muscular VSD. A moderately dilated LV causing moderate functional MR. LVSF was moderately reduced (40-45%). The RV was severely dilated with severely reduced systolic function. (RVSP ~ 40-45 under anesthesia). Surgery was performed via median sternotomy, central aortic cannulation, and bicaval venous cannulation. CPB was instituted, and repair was performed via a posterior left ventriculotomy with closure of the VSD using a bovine pericardial patch (exclusion technique). Post-repair TEE revealed complete closure of the VSD with no residual shunt. The inferior septal aneurysm's size was reduced, improved LV and RV size and function. Postoperatively, the Pt remained stable on IABP and low dose inotropes. The IABP was removed on POD1. Iontropes were weaned by 36 hours. The patient was discharged on POD6.

Discussion

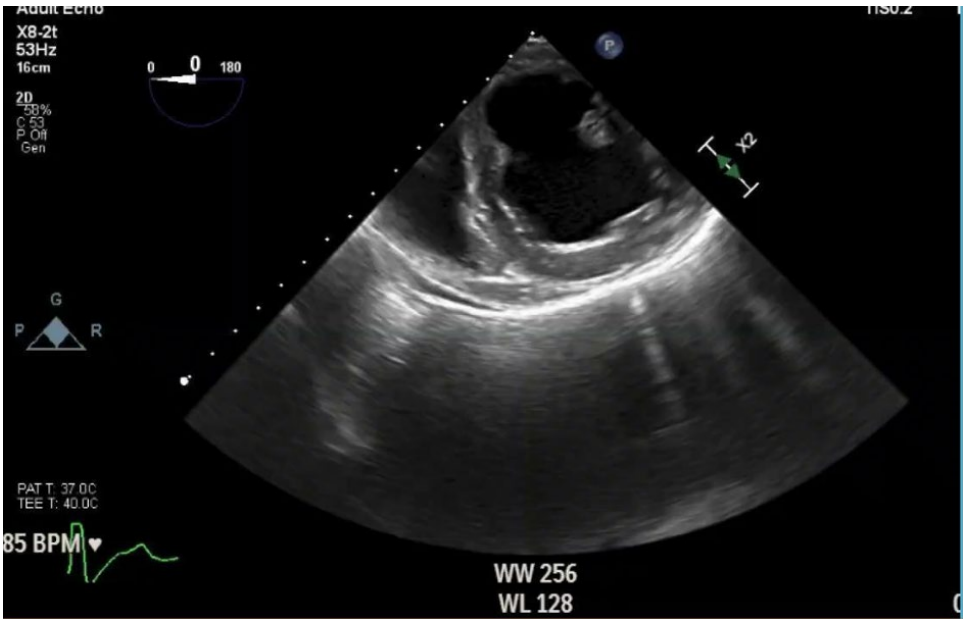
Post MI VSD's pose significant anesthetic challenges. VSD size, shunt magnitude, RV function and pulmonary hypertension must be optimized perioperatively. This patient's severe pulmonary hypertension and biventricular dysfunction which greatly increased her anesthetic and surgical risk. The timing of repair required balancing hemodynamic instability with the benefit of allowing tissue to stabilize. Surgery occurred 15 days post-MI, permitting partial scar formation. Anesthetic goals were to decrease LV/RV afterload, maintain cardiac output and coronary perfusion, and support RV function. Strategies included hemodynamic line placement, continuation of the IABP, use of inodilators (milrinone) and pulmonary-sparing vasopressors (vasopressin), maintaining adequate preload, and avoiding hypoxia, hypercarbia, and

acidosis. Inhaled nitric oxide provided selective pulmonary vasodilation. TEE was critical for defining the defect, quantifying shunt severity, guiding surgical repair, and confirming successful closure.

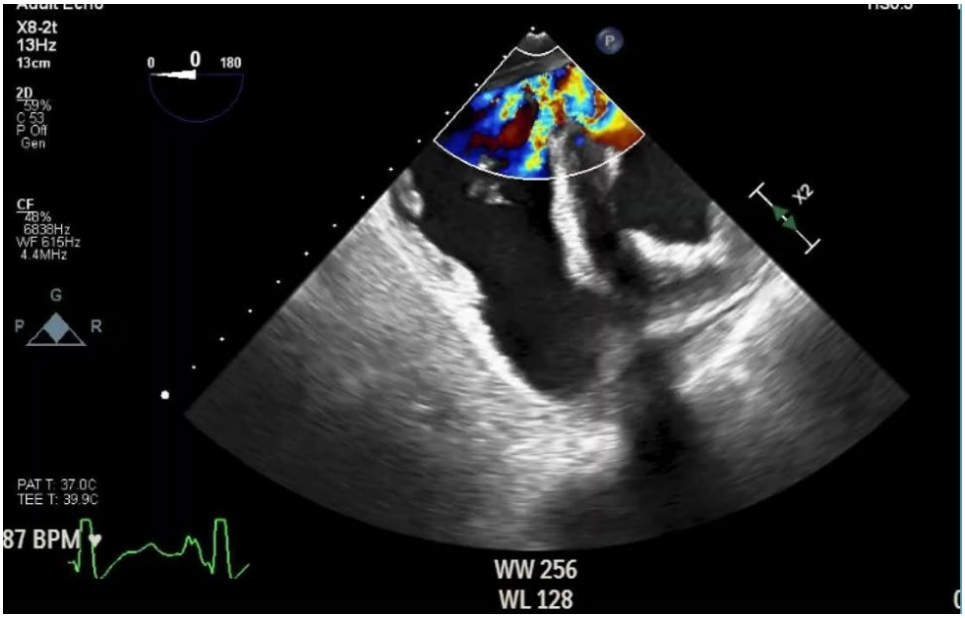
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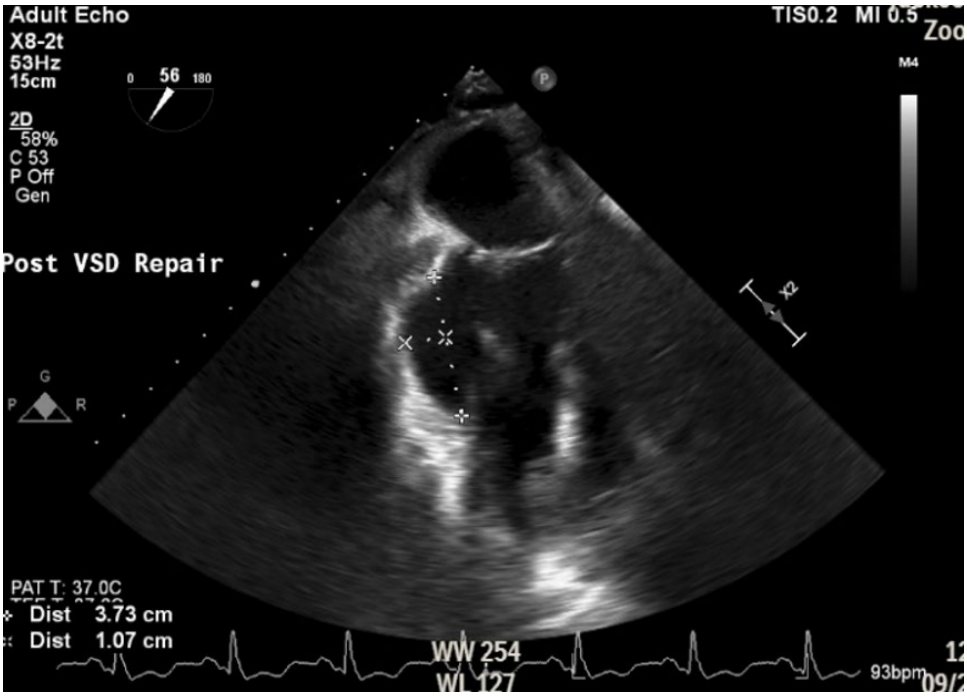
Pre-Repair TEE



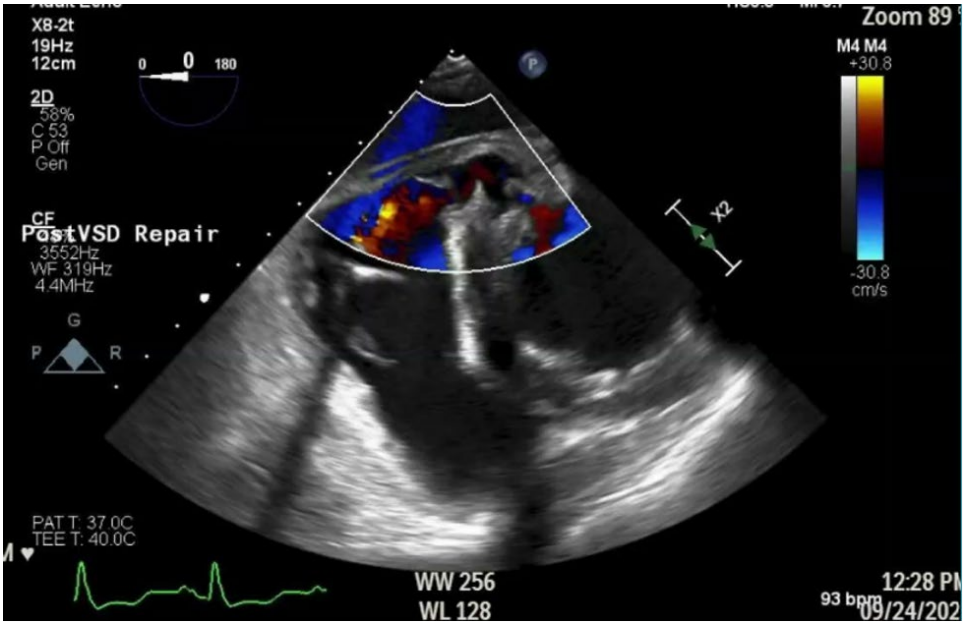
Pre-Repair TEE



Pre-Repair TEE



Post-Repair TEE



Post-Repair TEE

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#FR 1 – Kiosk 18 – Poster 3

Weathering the Storm: Successful Multi-Modal Management of Refractory Ventricular Tachycardia

Presenting Author: Morgan Sexton, MD, Resident Physician, Brigham & Women's Hospital

Introduction

Electrical storm (ES) is defined as three or more sustained ventricular arrythmias in less than 24 hours[1]. Refractory ventricular tachycardia occurs when VT cannot be aborted with typical management. In patients with ES and refractory VT, there are rescue options available including regional anesthesia and mechanical circulatory support. We present a case requiring complex multi-modal treatment for refractory ES.

Case Presentation

A 56-year-old man with ischemic cardiomyopathy presented to an outside hospital with an NSTEMI and NSVT. Coronary angiogram showed RCA plaque rupture but no intervenable target. An ICD was placed, and he received multiple discharges for VT. He was sedated, intubated and transferred for high-risk ablation. During the ablation, he was initially stable but began having increased ectopy. He became hypotensive, and epinephrine was started. The rhythm devolved into refractory VT. Lidocaine (80mg) and amiodarone (150mg) were bolused multiple times, and infusions were started. Electrolyte abnormalities were treated. Over 30 attempts at anti-tachycardia pacing and cardioversion were unsuccessful. The patient lost pulses, and ACLS began with chest compressions, epinephrine (1mg) administration and defibrillation. After ROSC, he remained in VT. Regional anesthesiologists performed emergent stellate ganglion blocks with 5mls 0.5% ropivacaine bilaterally. At the same time, VA ECMO was started, and an Impella was placed for LV decompression. After these interventions, the patient converted to sinus bradycardia with ectopy. The patient was transported to the ICU, where bilateral stellate ganglion catheters were placed. He re-presented for VT ablation 4 days later while on ECMO without success.

Discussion

ES can present as a patient receiving multiple shocks from their ICD or as sustained arrythmia causing hemodynamic instability. Worsening ischemia, increased wall stress, drug toxicity and electrolyte derangements are all triggers to address. ICD interrogation is warranted in patients with ICDs. First line therapies for acute VT include anti-tachycardia pacing, cardioversion, and anti-arrhythmics such as amiodarone, procainamide, or lidocaine. Beta blockers are used but limited in hypotension and severely reduced EF. Sympathetic blockade is the next line of therapy. This includes sedation, general anesthesia, and procedures like thoracic epidurals, stellate ganglion blocks and surgical sympathectomy. Stellate ganglion blocks can reduce the number of episodes of VT in refractory ES[2]. Unlike thoracic epidurals, these blocks can be performed safely with anticoagulation, and they are more feasible than surgical sympathetic denervation in acute VT. In refractory unstable VT, MCS may be necessary. VA ECMO, IABPs, and Impellas unload the LV and increase myocardial perfusion - helpful in restoring sinus rhythm[3]. VA ECMO can replace native cardiac output and bridge hemodynamics until patients can receive definitive care, including catheter ablations, durable LVADs, or heart transplantation. Our patient required all three lines of ES treatment

(Figure 1): electrical conversion and anti-arrhythmics, sympathetic blockade, and MCS. Cardiac anesthesiologists must be familiar with these treatment options so that they can provide tailored care to these tenuous patients.

Images

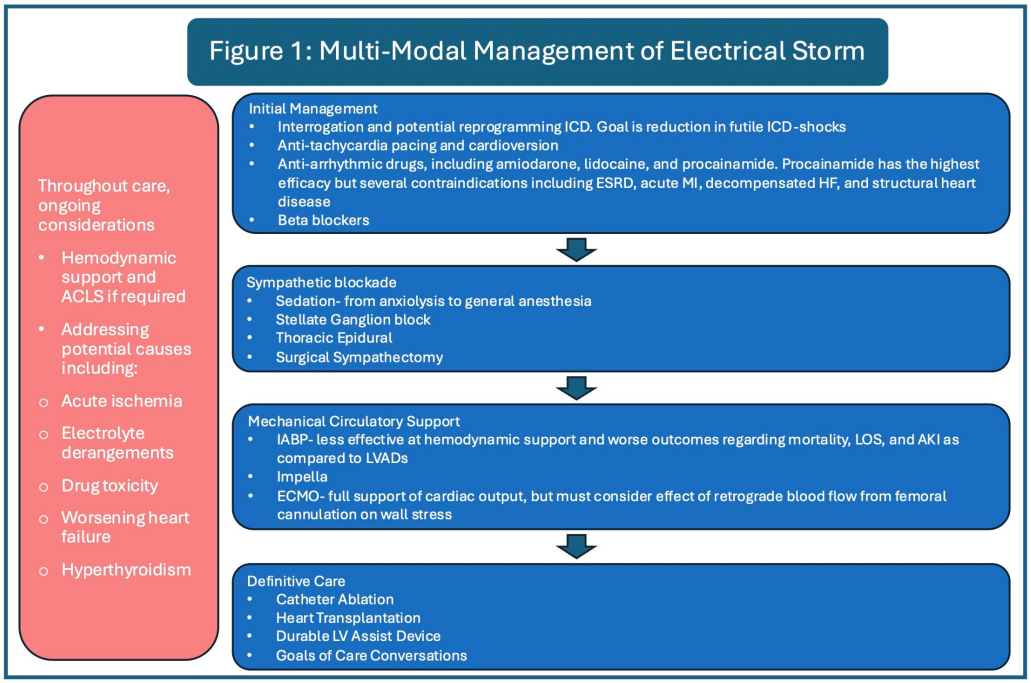


Figure 1: Multi-Modal Management of Electrical Storm

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#FR 1 – Kiosk 18 – Poster 4

A Rare Complication of AtriClip: Intraoperative TEE Diagnosis and Surgical Rescue of Left Main Coronary Compression

Presenting Author: Biodun Adeniyi, MD, MS, Resident Physician, University of Iowa Health Care

Introduction

The AtriClip device is widely used for left atrial appendage (LAA) occlusion to reduce cardioembolic stroke risk in atrial fibrillation. Although generally safe, rare but life-threatening complications such as left main coronary artery (LMCA) compression have been reported. Contractor et al. described four coronary injuries, including one fatality, among ~270,000 devices implanted in the U.S. between 2015 and 2021 (1). With more than 550,000 implants worldwide, only a handful of LMCA compression cases have been documented (2,3). Recognition is challenging during minimally invasive valve surgery, where visualization is limited and ECG interpretation often unreliable. In this setting, intraoperative transesophageal echocardiography (TEE) is indispensable, providing real-time ventricular assessment, detecting ischemia, and guiding interventions.

Case Presentation

A 61-year-old female with hypertension, atrial fibrillation (on rivaroxaban), and severe mitral regurgitation underwent minimally invasive mitral valve repair with concomitant LAA closure using the AtriClip system. The procedure was performed under cardiopulmonary bypass (CPB) via right mini-thoracotomy, with cross-clamp and bypass times of 114 and 153 minutes, respectively. After rewarming and de-airing, sinus rhythm was restored. TEE revealed preserved right ventricular function but new global left ventricular (LV) hypokinesis. Epinephrine was started, but the patient could not be weaned from CPB. The dose was increased and milrinone added, resulting in slight LV improvement and separation from CPB. ECG showed sinus tachycardia, but ST-segment analysis was limited by low voltage from thoracotomy lead placement. After 25 minutes off CPB, the patient developed both worsening hemodynamic instability and global LV hypokinesis on TEE. An intra-aortic balloon pump (IABP) was inserted via the left femoral artery. Despite this, the patient remained hypotensive and developed ventricular fibrillation, prompting emergent re-initiation of CPB. Persistent wall-motion abnormalities on TEE and recurrent arrhythmia raised concern for coronary obstruction. A sternotomy was performed and the aortic root inspected. The AtriClip was found compressing the LMCA and removed. Wall-motion abnormalities resolved immediately on TEE. Intraoperative angiography confirmed normal coronaries. The IABP was discontinued, and the patient was successfully weaned from CPB.

Discussion

This case highlights LMCA compression as a rare but devastating complication of AtriClip placement. TEE was the critical diagnostic modality: it detected LV dysfunction at each stage, guided escalation (repeat CPB, IABP insertion, sternotomy), and confirmed recovery after clip removal. Mitsuishi et al. (2) described percutaneous intervention as a strategy, but Nakanaga et al. (3) emphasized sternotomy with clip removal as the most definitive rescue, consistent with our case. Careful clip deployment and awareness of the LAA–

LMCA relationship are essential, but vigilant intraoperative TEE provides the safeguard for early recognition and intervention. This case underscores the anesthesiologist's key role in preventing catastrophic outcomes during LAA closure, particularly in minimally invasive settings where visualization is limited and ECG analysis is difficult.

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#FR 1 – Kiosk 18 – Poster 5

Navigating Late Ebstein's Anomaly Correction – A Case-Based Discussion

Presenting Author: Katherine E. Kent, MD, Fellow, Stanford Health Care

Introduction

Ebstein's anomaly (EA) is a rare congenital heart defect (CHD) occurring in 1 to 5 per 200,000 live births [1]. EA results from insufficient delamination of the posterior and septal leaflets of the tricuspid valve (TV) leading to varying degrees of apical displacement, TV incompetence, and right ventricular (RV) dysfunction [1]. It is often associated with other CHD lesions. Because of its rarity and heterogenous clinical presentation, optimal management has been difficult to define.

Case Presentation

A 48 year old female with CREST and Sjogren's syndrome presented with persistent cyanosis of her hand. An echocardiogram was obtained which showed EA with severe tricuspid regurgitation (TR) and left ventricular (LV) noncompaction with normal function. She was referred for surgery and then lost to follow up due to insurance reasons. In the intervening years she developed worsening complications including decompensated heart failure, atrial fibrillation, chronic kidney disease, GI bleeds, cirrhosis, and frailty. She presented for surgery at age 56. A TV bioprosthetic replacement with left atrial appendage oversew, epicardial lead placement and right atrial (RA) plication was performed (Figure 1). Her preoperative echocardiogram (TEE) was notable for normal LV function, massive RA dilation with an atrialized RV, mild to moderate RV dysfunction and torrential TR (Figure 2). Central venous pressure (CVP) V waves were seen to 40 mmHg. Given concern for inability to separate from bypass, she was optimized with inhaled nitric oxide, isoproterenol and epinephrine while her underlying coagulopathy was carefully managed. TEE post-repair showed severe RV dysfunction and dilation, no residual TR, and hyperdynamic LV function with near normalization of her CVP. She was extubated within hours of arriving to the ICU. Her post operative course was significant for volume overload necessitating continuous renal replacement therapy, prolonged inotropy wean with ongoing RV dysfunction, and pacemaker placement.

Discussion

Late EA repair is a challenging entity as patients are often markedly symptomatic by the time they present for surgery [1]. Indications for repair depend on underlying symptomatology (fatigue, decreased exercise tolerance, cyanosis, exertional dyspnea) and extent of the anomaly (severe TR, RV enlargement, favorable valve anatomy, etc.) [2]. Careful perioperative screening involves exercise stress testing, cardiac imaging, catheterization, and electrophysiologic studies to determine the appropriate surgical approach for each individual patient [2]. Ideally, severe EA is repaired early in life before maladaptive remodeling occurs. The cone repair which reconstructs the TV by mobilizing leaflets and creating a cone shape to the true atrioventricular junction thereby restoring flow and allowing the valve to grow with the patient has become a cornerstone approach due to superior outcomes and durability [2]. However this approach is not appropriate in older patients with more advanced disease (severe RV +/- annular enlargement, intracardiac

repair, ASD, etc.), as crossclamp (XC) and bypass times become deleterious to RV function [2]. In these patients, as was done in this case, TV replacement and selective plication is chosen to maximize function and minimize bypass/XC times [2].

Images

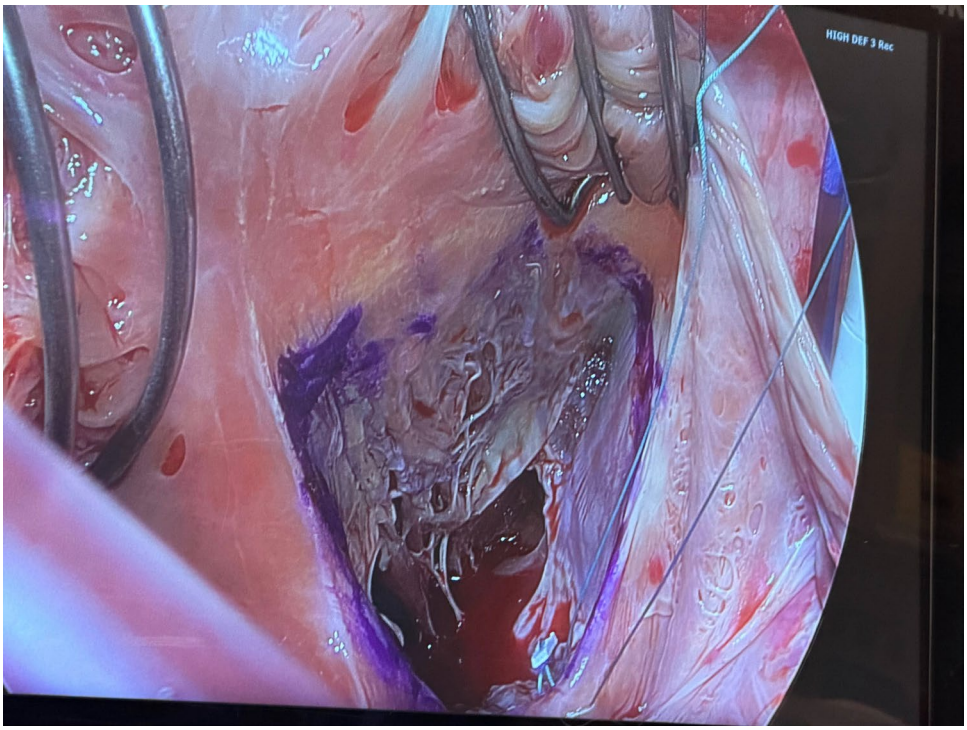


Figure 1: Tricuspid valve annulus and small right ventricle. At right side of screen you can see the diminutive septal leaflet.

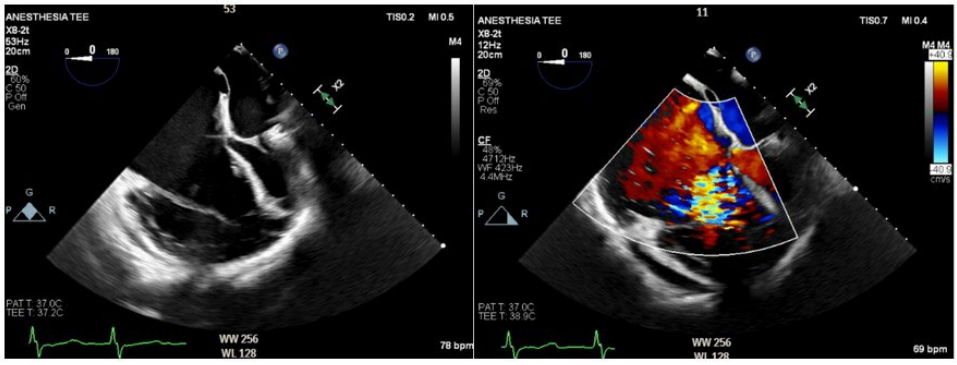


Figure 2: Transesophageal echocardiogram characterizing Ebstein's Anomaly and resultant regurgitation.

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#FR 1 – Kiosk 18 – Poster 6

Mobile Left Atrial Mass with Conflicting Preoperative Imaging: Intraoperative TEE Clarifying Diagnosis and Guiding Management

Presenting Author: Jeremy Tang, MD, PhD, Adult Cardiothoracic Anesthesia Fellow, Rutgers Robert Wood Johnson Medical School

Introduction

Left atrial masses are uncommon findings with a differential that includes thrombus, myxoma, and less frequently other benign or malignant tumors. Accurate preoperative characterization is important because management strategies differ substantially depending on etiology, mobility, and anatomic relationships. Multimodal imaging such as transesophageal echocardiography (TEE) and cardiac MRI provides critical diagnostic information, though overlap in tissue characteristics may limit definitive classification. Mobile left atrial lesions also introduce specific anesthetic risks, including potential for embolization or inflow obstruction during induction or cardiopulmonary bypass (CPB) initiation. We present a case of an incidentally discovered left atrial mass with indeterminate imaging features in which perioperative TEE played a central role in guiding anesthetic management and operative decision-making.

Case Presentation

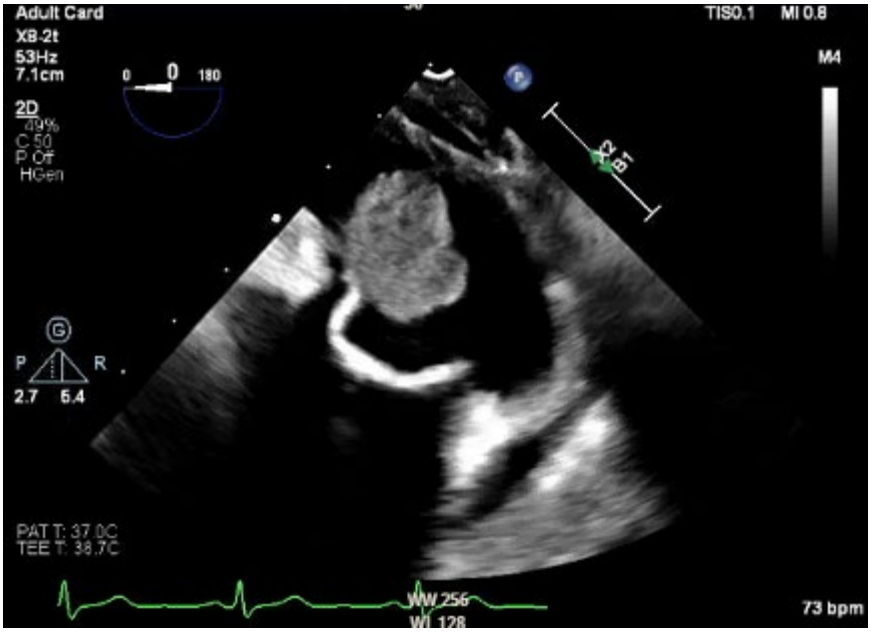
A 52-year-old female with hyperparathyroidism and osteopenia underwent preoperative imaging prior to parathyroidectomy, which revealed a possible left atrial mass. TEE demonstrated a mobile, echogenic lesion arising from the posterior atrial roof measuring 1.6 × 1.8 × 2.3 cm, intermittently contacting an aneurysmal interatrial septum. Cardiac MRI identified an irregular 21.7 × 13.6 mm mass near the right upper pulmonary vein with early gadolinium characteristics suggestive of thrombus but central late enhancement raising concern for necrosis, making thrombus versus myxoma difficult to distinguish. Given the lesion's mobility and proximity to pulmonary venous inflow, cardiothoracic surgery recommended surgical excision. A right mini-thoracotomy approach was undertaken. Anesthetic management emphasized minimizing hemodynamic fluctuations that could alter mass position or risk embolization. Intraoperative TEE performed by the anesthesia team confirmed a 1.9 × 1.0 cm mobile mass prolapsing into the left atrium during diastole and unexpectedly identified a patent foramen ovale (PFO), which was subsequently incorporated into the surgical plan for closure. Standard CPB techniques were used, and the intraoperative course was uncomplicated. Post-CPB TEE verified complete mass resection and successful PFO closure. The patient recovered uneventfully and was discharged on postoperative day four. Gross pathology favored myxoma; final histopathology remains pending.

Discussion

This case demonstrates key anesthetic considerations in managing mobile intracardiac masses of uncertain etiology. Diagnostic ambiguity between thrombus and myxoma is common despite advanced imaging, making real-time assessment of mobility, attachment, and hemodynamic impact more important than definitive tissue diagnosis for perioperative planning. Intraoperative TEE was essential not only for

confirming mass morphology and behavior but also for identifying an occult PFO that carried embolic implications and modified the surgical approach. Hemodynamic control during induction and CPB initiation is critical in preventing mass migration or embolization. This case reinforces the importance of TEE-guided anesthetic management in achieving safe resection of left atrial masses when preoperative characterization is inconclusive.

Images



TEE of Left Atrial Mass

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#FR 1 – Kiosk 18 – Poster 7

Diagnostic Advantage of 3D TEE over 2D imaging on detection of a P1 Fibroelastoma

Presenting Author: Ujalashah Dhanani, MD, Resident Physician, The Texas Heart Institute at Baylor College of Medicine

Introduction

Two-dimensional transesophageal echocardiography (2D TEE) has been a valuable tool for intraoperative evaluation of valvular disease. However, its single-plane views can obscure complex lesions, especially mitral valve (MV) pathology. Therefore, three-dimensional (3D) TEE has become an essential tool for evaluating mitral valve pathology. It is beneficial for complex conditions such as mitral valve prolapse or flail. 3D TEE provides a superior, comprehensive, and more accurate view of the valve's anatomy and function compared to 2D imaging.

Case Presentation

A 53-year-old male with a history of Hodgkin's lymphoma presented with severe dyspnea on exertion, and TEE showed severe mitral regurgitation due to P1 prolapse. The patient was scheduled for a minimally invasive mitral valvuloplasty. The initial intraoperative 2D TEE examination showed a possible P1 flail (Figure 1), but the visualized MR jet was not consistent with this pathology. Subsequent real-time 3D TEE evaluation revealed a different diagnosis. The 3D "en-face" view from the left atrial perspective clearly demonstrated a 6mm x 3mm mobile mass attached to the atrial surface of the P1 segment of the posterior leaflet (Figure 2). This mass prolapsed into the left atrium during systole and mimicked a flailed P1 segment on the 2D plane view. Direct visualization of the mass on the posterior leaflet by the surgeon revealed that it was, in fact, a fibroelastoma, and not a flail segment of the posterior leaflet (Figure 3). Based on the new diagnosis, the surgical plan was changed from P1 repair to a simple resection of the mass with an annuloplasty ring.

Discussion

While 2D TEE remains a valuable tool for common pathologies when performed by experienced operators (Mukherjee et al., 2011), this case illustrates its limitations when encountering an atypical lesion. The intraoperative 2D TEE findings were contradictory: the 2D image suggested P1 flail, but the central MR jet was seen instead of the expected anterior jet from P1 flail. This ambiguity is a known limitation of 2D imaging, which requires mental reconstruction to synthesize multiple, disparate planes. The P1 scallop is a recognized area of difficulty for 2D TEE, where diagnostic accuracy is often reduced. One study of 42

patients demonstrated 3D TEE's superior accuracy in identifying pathology of the P1, A2, and A3 scallops, which are challenging to assess with 2D TEE (Grewal et al., 2009). Beyond simple localization, the “surgical view” obtained with 3D TEE creates a shared understanding between the anesthesiologist and surgeon, which is crucial for real-time decision-making (Tsang W et al., 2013). Real-time 3D TEE is therefore a powerful complement to 2D TEE, offering intuitive spatial understanding that improves diagnostic precision. As this case demonstrates, its intraoperative use can significantly alter surgical planning, preventing misdiagnosis and improving outcomes in complex mitral valve surgery.

Images

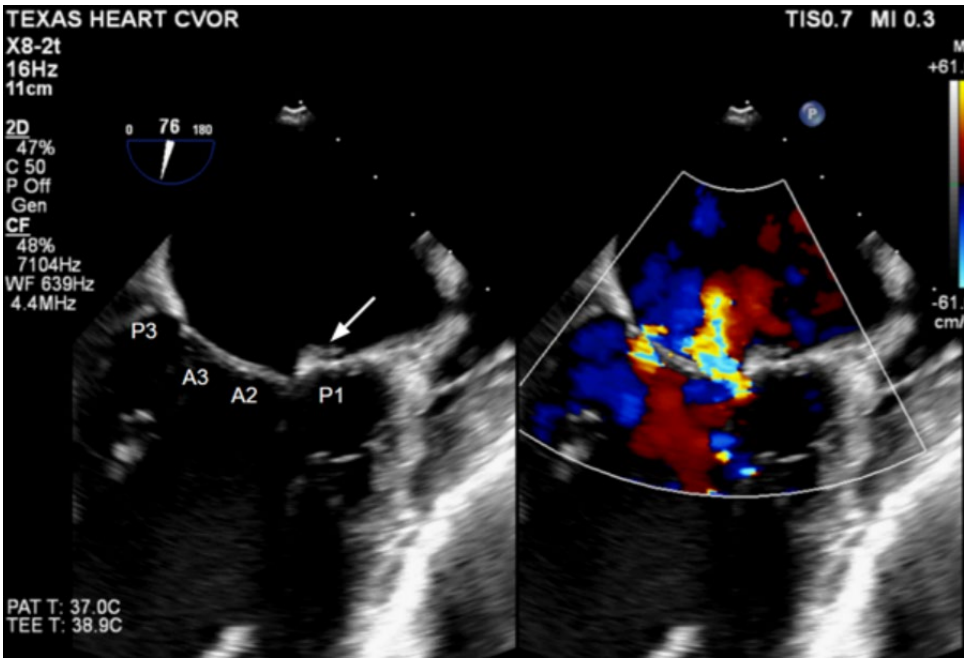


Figure 1. ME Mitral Commissural view showing a possible P1 flail (White arrow).

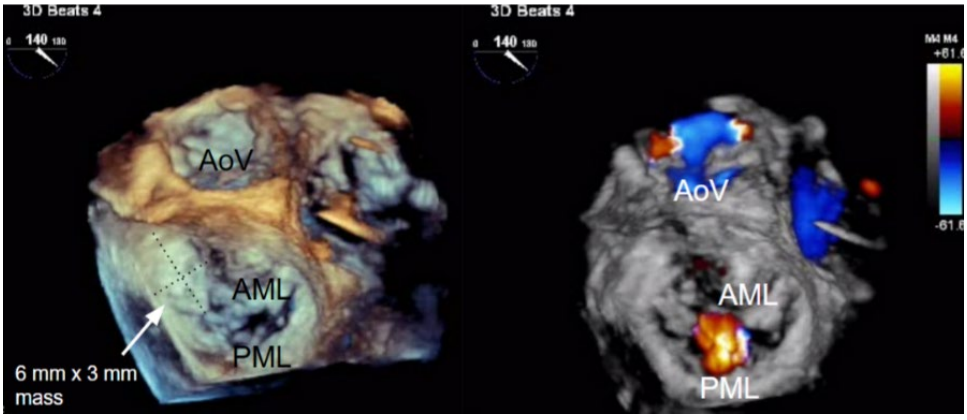


Figure 2. 3D TEE showing a 6mm x 3mm mobile mass (White arrow) on P1 with central jet AI.

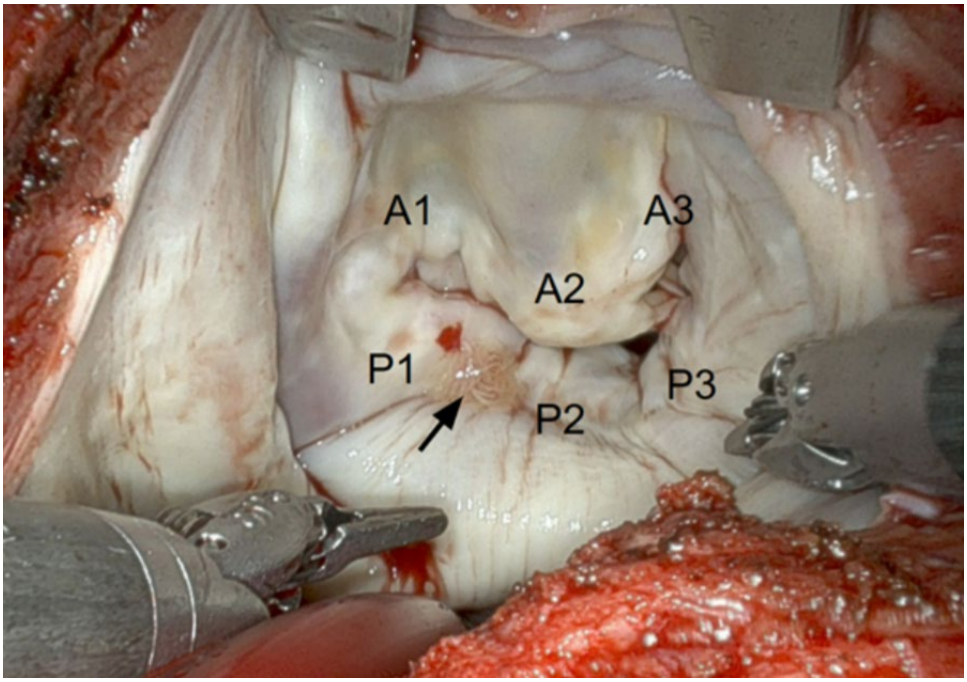


Figure 3. Surgeon's view of the mitral valve. Fibroelastoma (Black arrow) attached to the P1 leaflet of the mitral valve.

References

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#FR 1 – Kiosk 19 – Poster 1

Assessment and Management of Pulmonary Angiosarcoma: Pulmonic Valve Replacement and Pulmonary Thromboendarterectomy

Presenting Author: Matthew Geiser, MD, Fellow, Duke

Introduction

Pulmonary angiosarcoma is a rare and aggressive cancer with often nonspecific symptoms and a poor prognosis. Tumors can originate in the lungs or pulmonary arteries with treatment options including surgery, radiation, and chemotherapy [1]. When the tumor arises from the main pulmonary artery, pulmonic valve or supravalvular stenosis can occur precipitating right heart dysfunction. We present a case of pulmonary artery angiosarcoma, the surgical removal and anesthetic management, and the echocardiographic assessment of pulmonary valve obstruction.

Case Presentation

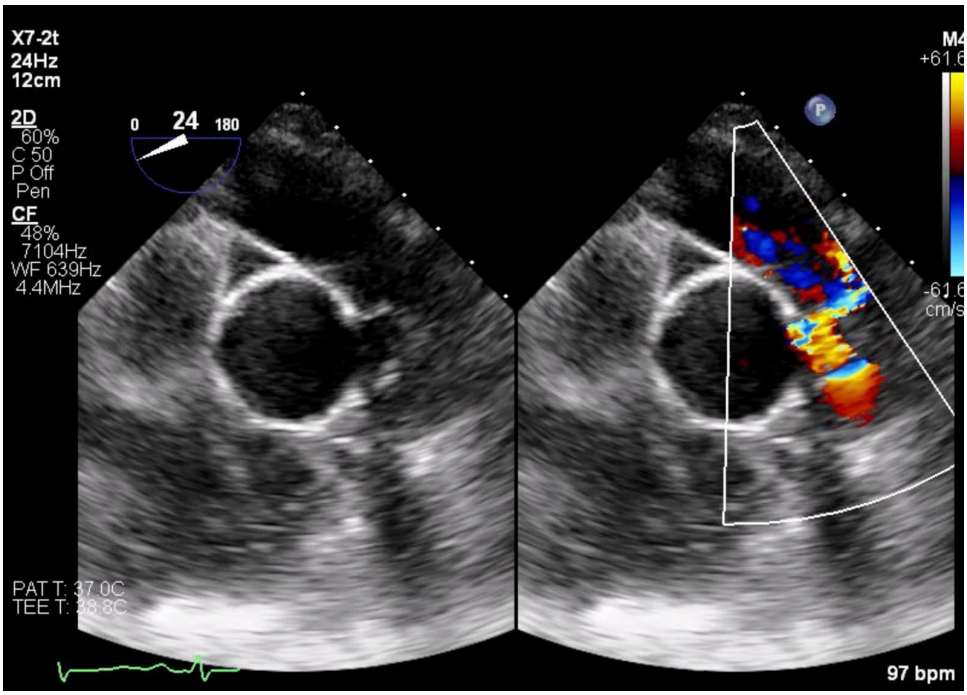
A patient presented to an outside hospital with a history of shortness of breath and fatigue. Transthoracic echocardiogram and MRI imaging showed rapidly progressing severe pulmonic valve / supravalvular stenosis (peak gradient 110 mmHg) and severe right ventricular (RV) dysfunction with a concern for angiosarcoma involving the pulmonary artery. Patient was transferred to our institution for consideration of surgical resection. Patient underwent pulmonic valve replacement, repair of pulmonary artery stenosis, and pulmonary thromboendarterectomy (PTE). Given her severely stenotic pulmonic valve and poor RV function, induction goals were to avoid increases in pulmonary vascular resistance while preserving hemodynamic stability. After initiating vasopressors, an induction with opioids, etomidate, and high dose muscle relaxant was performed uneventfully. Intraoperative transesophageal echocardiography (TEE) showed severe RV with preserved LV function. Flow acceleration was seen in the main pulmonary artery distal to the pulmonic valve, but the valve was difficult to visualize on TEE. The pre cardiopulmonary bypass (CPB) period was otherwise uneventful. Once on CPB, there was noted to be a near occlusive mass involving the pulmonary root and right ventricular outflow track with obliteration of the pulmonic valve. Initial attempts to separate from CPB were unsuccessful due to severe RV dysfunction and hemodynamic instability despite escalation of inotropes, pressors, and initiating iNO. This necessitated VA ECMO deployment. Patient was brought to the ICU with an open chest on VA ECMO with TEE showing severe RV dysfunction. Final pathology was positive for primary pulmonary angiosarcoma.

Discussion

This is a rare case of pulmonary artery stenosis secondary to pulmonary angiosarcoma involving both the valve and pulmonary artery. Pulmonic stenosis is often a congenital disease and less commonly presents in adults. Anesthetic management of patients with pulmonic stenosis in the presence of severe RV dysfunction presents the hemodynamic challenges discussed above, as well as the issues inherent in PTE surgery, including need for transfusion and reperfusion injury. Obtaining an image of the pulmonary valve is more challenging with TEE as it is the most anterior of the heart valves and therefore farthest away from the probe.

Additionally, this can make doppler assessment challenging as alignment can vary based on patient anatomy. Utilization of multiple views including mid-esophageal right ventricular inflow-outflow and ascending aortic short-axis, upper-esophageal aortic arch short-axis, and transgastric basal RV view [2] can be used to assess the etiology and severity of pulmonic stenosis.

Images



References

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#FR 1 – Kiosk 19 – Poster 2

Intraoperative Rescue of False-Lumen Frozen Elephant Trunk Deployment

Presenting Author: Filip Polenakovik, MD, Cardiothoracic Anesthesia Fellow, University of Alabama at Birmingham Department of Anesthesiology and Perioperative Medicine

Introduction

The frozen elephant trunk (FET) technique has become an important strategy for managing complex aortic arch and descending thoracic aortic pathology. Despite its advantages, inadvertent deployment of the stent-graft component into the false lumen remains a rare but serious technical complication associated with significant morbidity if not promptly recognized (1). This case describes the successful intraoperative management and rescue of a FET extension deployed into a false lumen using the assistance of TEE and intravascular ultrasound guidance.

Case Presentation

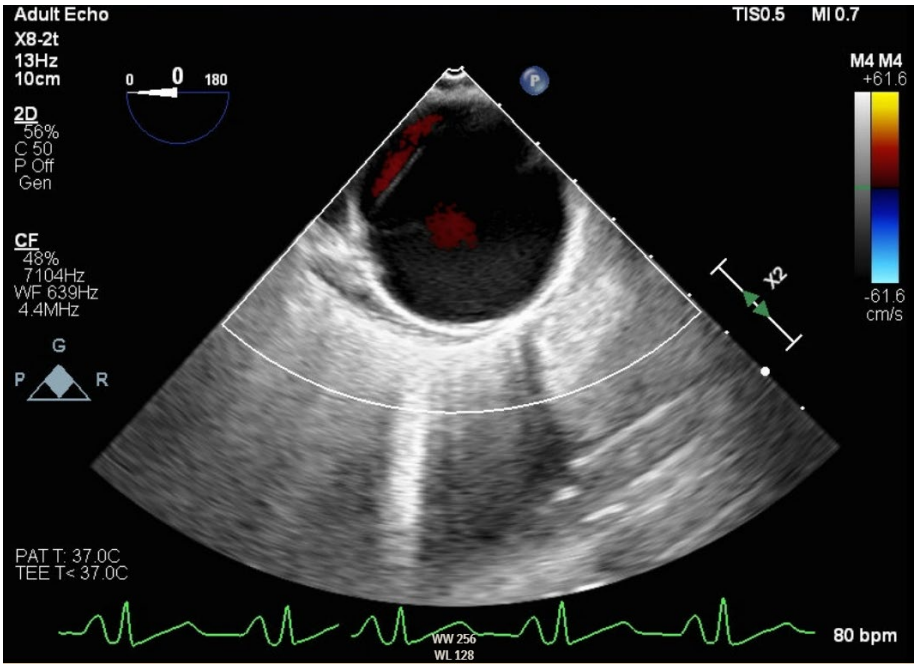
A 49-year-old male with a history of type A aortic dissection repair, ascending aorta and hemiarch replacement with FET extension presented for a repeat sternotomy, total arch replacement with FET extension. Initial imaging revealed the dissection with two definitive lumens identified in the descending thoracic aorta distal to the pre-existing stent graft (image 1). Circulatory arrest proceeded uneventfully with deployment of the new FET into the old FET. TEE at this time demonstrated appropriate positioning of the stent within the old FET, but the true lumen in the descending aorta appeared compressed which raised concern for false lumen stent deployment. After arch reconstruction was completed and cardiopulmonary bypass was discontinued, a retrograde wire advanced from the femoral artery did not reach the FET, confirming false lumen stent deployment (image 2). Under direct TEE guidance and with the help of a Rosh-Uchida sheath, a fenestration was created from the true lumen into the false lumen (image 3). A TEVAR extension stent was then placed over a guidewire and TEE confirmed adequate overlap of the extension into the FET proximally and sufficient length within the true lumen; IVUS was used to rule out occlusion of the mesenteric vessels. TEE imaging confirmed now predominant flow into the true lumen (image 4). The remainder of the procedure was uneventful, and the patient recovered without neurologic deficit or end-organ malperfusion and was discharged home on post-op day 5.

Discussion

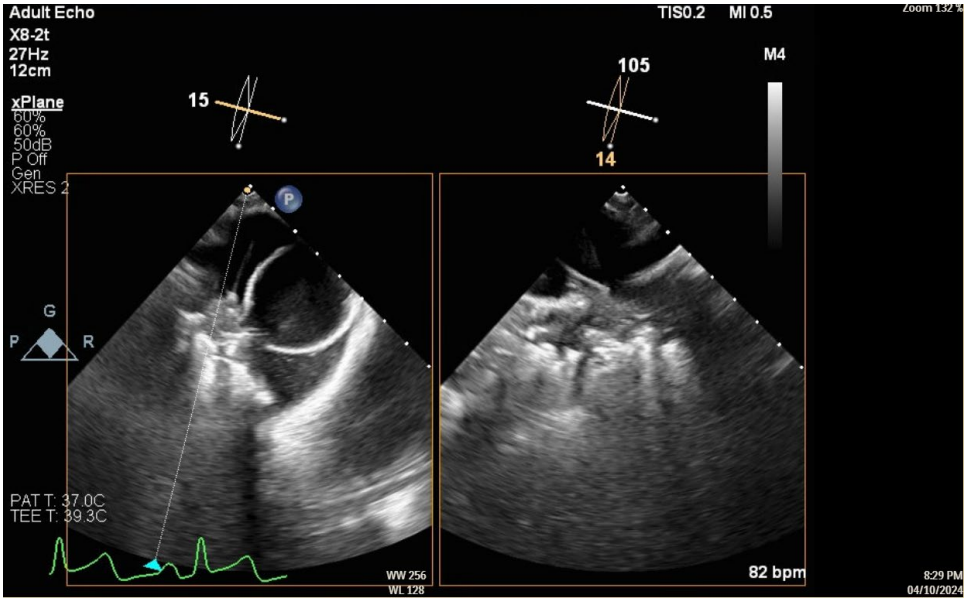
False lumen FET-extension deployment can have devastating consequences including visceral organ and

spinal cord malperfusion, left heart strain, intracranial hypertension, and aortic pressurization and rupture (2,3). Contributing factors may include complex chronic dissection morphology, compressed or slit-like true lumen, or inadequate intraluminal guidance. Some recent data suggests that anterograde placement was also a significant risk factor in false lumen deployment, indicating the need for higher vigilance and awareness when retrograde access is not used or possible (2). Current clinical data suggests that early detection/intervention and utilizing TEE and/or IVUS guidance (versus aortography alone) are critical to preventing catastrophic malperfusion (2). This case highlights the role of intraoperative TEE in the decision-making process for both anesthetic and surgical management.

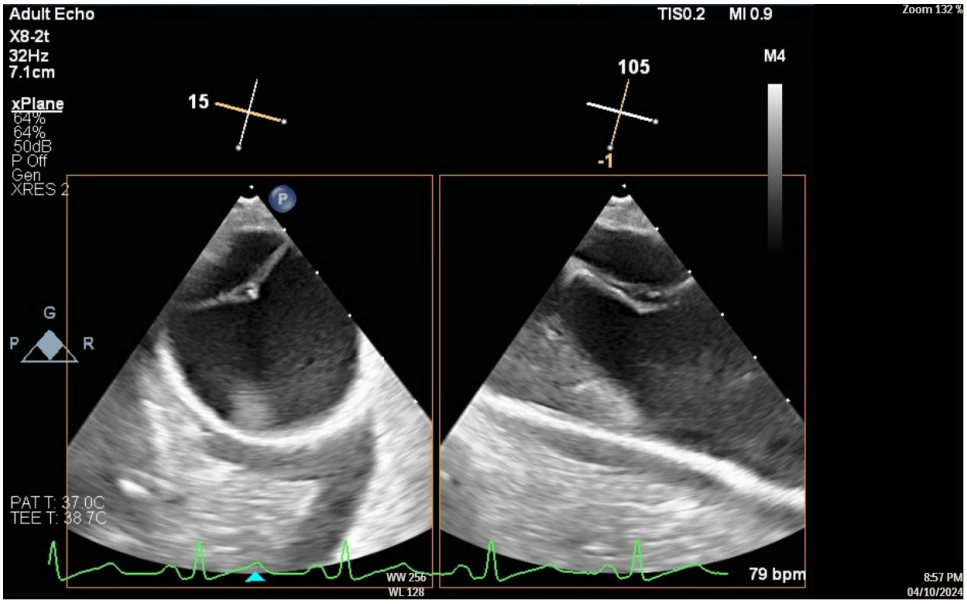
Images



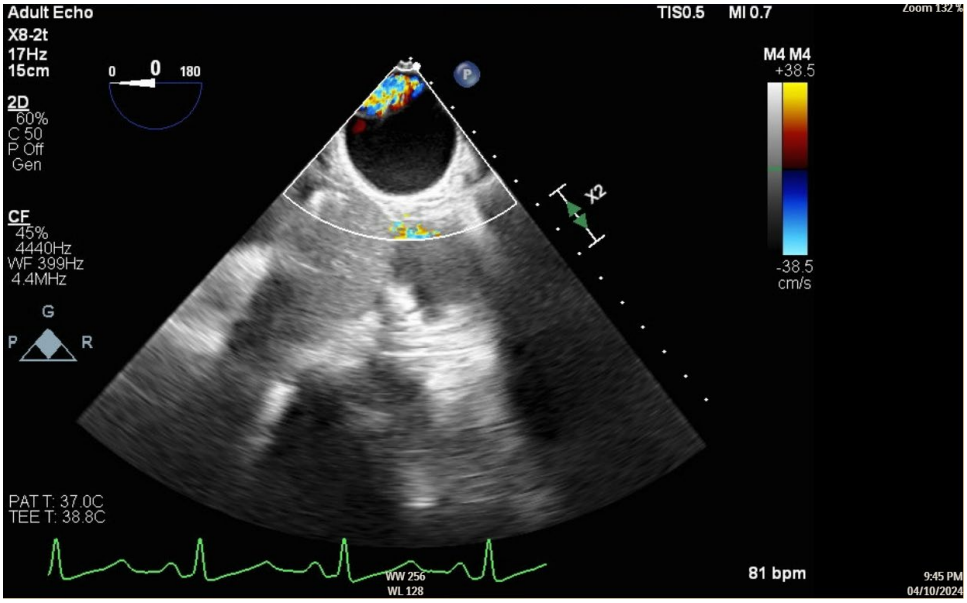
Dissection in the distal thoracic aorta with the smaller true lumen (TL) and larger false lumen (FL) visualized



Retrograde wire seen in the TL outside of FET extension, confirming false lumen deployment of stent graft



Retrograde wire traversing fenestration and connecting the true lumen to false lumen



Color flow doppler confirming predominant flow now in the true lumen following re-deployment

References

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#FR 1 – Kiosk 19 – Poster 3

Veins, Drains, and Bypass: Management of a Critical Complication in the Cath Lab

Presenting Author: Joseph Israeli, MD, Department of Anesthesiology, Perioperative Care, and Pain Medicine, NYU Langone Health

Introduction

Cardiac cath lab interventions may abruptly deteriorate. The anesthesiology team's role in helping diagnose and skillfully manage unexpected complications is crucial.

Case Presentation

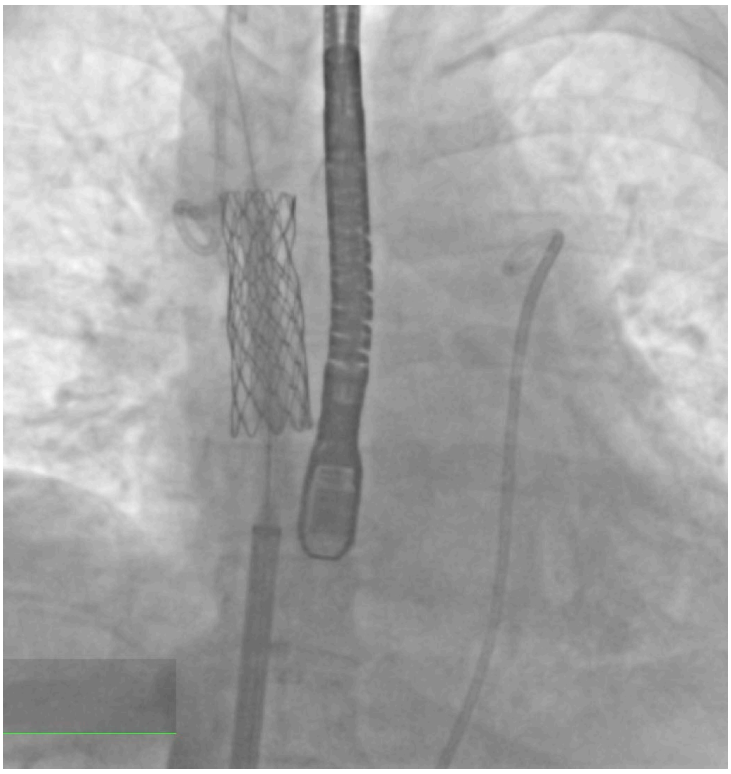
A 54-year-old woman with acquired superior vena cava (SVC) syndrome from long-term port placements presented for SVC recanalization. Perioperative preparations included having available: four units of blood, a cardiothoracic (CT) surgeon, additional staff, and a primed cardiopulmonary bypass (CPB) machine. Draping allowed for easy sternal and femoral access in case of sternotomy or CPB. General anesthetic induction and intubation were uneventful. During serial dilation of the atretic SVC segment, a growing pericardial effusion was visualized with TEE. Systolic blood pressure declined, and pulse pressure narrowed. Treatment included intravascular fluid administration, pericardiocentesis (which aspirated disproportionately large volumes of blood to the expected SVC injury), auto-transfusion of the aspirated blood, and SVC stent placement. Angiography confirmed SVC leak resolution. However, TEE demonstrated unchanged effusion. Soon after, MAP dropped to the 40s and the anesthesiology team called a MTP, with suspicion for continued bleeding. Cardiac resuscitative medications were given. TEE confirmed inadvertent pericardial drain placement into the RVOT/MPA, and angiogram showed that the pigtail was in the left PA. Severe hypotension and bradycardia followed; a brief code ensued involving chest compressions while initiating peripheral cardiopulmonary bypass via the cannulated femoral artery and vein. CT surgery performed a midline sternotomy to remove the pericardiocentesis catheter and repair the RV, which had a 5 mm perforation with pulsatile bleeding. The sternotomy was then closed, and the patient was decannulated. Bypass time was 47 minutes, without cardiac arrest. TEE showed no pericardial effusion and good biventricular function. The patient was brought to the ICU intubated and then extubated the next day. Postoperative echocardiogram resembled the post-bypass TEE. She was discharged home 5 days later.

Discussion

Perioperative preparations for potential blood loss, conversion to open surgery, and the need for mechanical circulatory support helped mitigate delays in proper care. However, the inadvertent pericardiocentesis catheter placement into the pulmonary artery through the RVOT added a unique challenge. When the big picture did not add up (aspiration of blood out of proportion to expected bleeding, continual hemodynamic collapse), quick intraoperative decision making was key. Alternate modalities to radiography were used for diagnosis (TEE initially), and prompt treatment (volume, calcium, epinephrine, leading a code, and establishing bypass conditions) allowed for definitive surgical repair and patient survival. Post-repair assessment allowed the anesthesiology team to clear the patient to leave the operating room. Our case is

exceptionally rare due to the catheter's course. Entering the main pulmonary artery is rare, and extending into the left pulmonary artery is even rarer. No cases describing this were located, but iatrogenic RV perforation during pericardiocentesis (catheter entering RV) (1), and a course from the liver through the IVC to the SVC have been reported (2).

Images



Complications of Pericardiocentesis [3]

Acute Cardiac	Cardiac Perforation	Arrythmia	Coronary Artery Laceration	Vasovagal Reaction
Subacute Cardiac	Infection/Sepsis	Post Cardiac Injury Syndrome (Pericarditis)	Effusion Recurrence (with or without constrictive pericarditis)	Pericardial Thrombus Formation
Acute Cardiopulmonary	Hemothorax	Pneumothorax	Pulmonary Edema (Pericardial Decompression Syndrome)	Hypoxemia (Right to Left Shunting via Septal Defect from RV issues after sudden unloading)
Subacute Cardiopulmonary	Infection/Sepsis	Pleuropericardial Shunt	Pleural Effusions	Pulmonary Thromboembolism (rare, from rapid CVP change)

References

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#FR 1 – Kiosk 19 – Poster 4

A Hole New Problem: Left Ventricle Perforation During Paravalvular Leak Closure, A Case Report

Presenting Author: Angel Pagan, MD, Resident, University of Cincinnati

Introduction

Transcatheter (TC) cardiac interventions have significantly increased over the past decade. While recent literature supports the safety and efficacy of TC approaches, associated complications including paravalvular leak (PVL), conduction disturbances, and rare but life-threatening cardiac perforation can occur[1]. Despite early detection and treatment, perforation can progress to tamponade and challenging anesthetic implications. We present a case of cardiac tamponade following left ventricle (LV) perforation during an attempted PVL closure in a medically complex patient and the importance of an intraoperative team-based care model.

Case Presentation

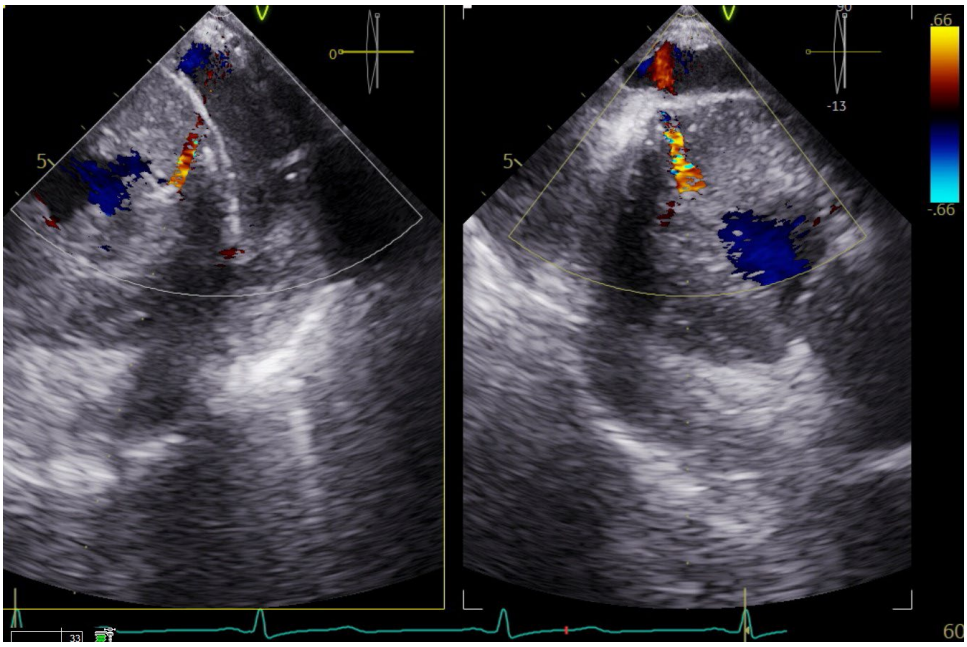
A 77-year-old male with PMHx of severe aortic insufficiency s/p bioprosthetic aortic valve replacement AVR (2013), complicated by recent PVL s/p redo AVR (2025), coronary artery disease s/p thrombectomy, and left lower lobectomy for adenocarcinoma was undergoing evaluation of new hemolytic anemia. Cardiac workup revealed new aortic valve dehiscence with severe paravalvular regurgitation. After multidisciplinary discussions, TC closure was selected given his comorbidities. The procedure was complicated by rapid development of a large pericardial effusion with LV chamber collapse noted on intraoperative TEE with tamponade physiology. Further evaluation revealed an inferolateral myocardium perforation. Emergent pericardial drain placement with continuous aspiration and reinfusion via a femoral venous line stabilized hemodynamics while femoral access was obtained to urgently cannulate for cardiopulmonary bypass. As a result of two prior sternotomies, redo sternotomy posed unique challenges and dissection down to the pericardium took 3 hours. After myocardial access was obtained, the inferolateral perforation was confirmed, and an additional apical LV perforation was identified and surgically repaired. Following closure, he was transferred to the ICU and recovered gradually without long-term sequelae.

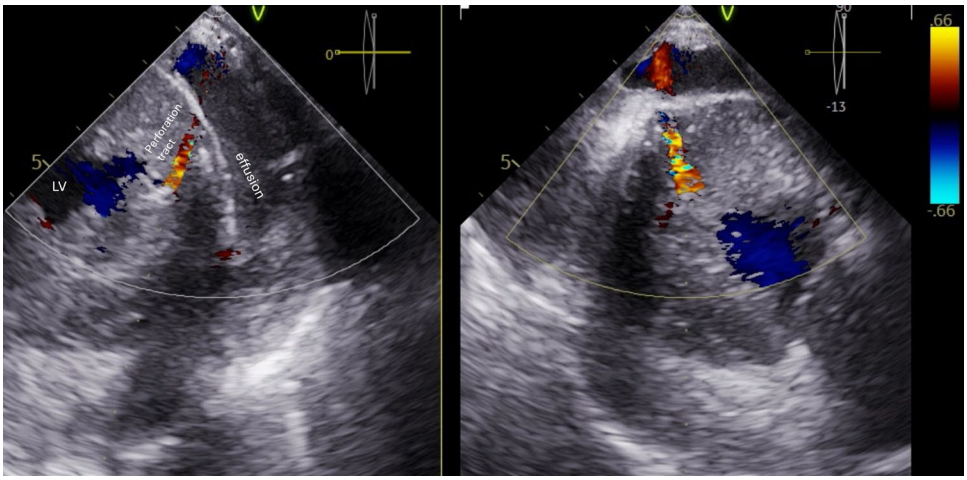
Discussion

Iatrogenic myocardial or central vessel perforation is a rare but often fatal complication of TC procedures with a morality as high as 25% even with immediate intervention [1]. In this case, immediate manual aspiration via pericardial drain provided transient hemodynamic improvement, though rapid reaccumulation caused dramatic swings. Anesthetic management required careful support of RV function by maintaining heart rate and contractility, while managing rebound hypertension post-tamponade [2]. Optimal treatment for PVL includes either a TC or surgical approach guided by a multidisciplinary heart team in collaboration with the patient. In this case, the decision was made to proceed with transcatheter closure due to the increased morbidity and mortality associated with a third redo sternotomy. Upon identification of the LV perforation, emergent surgical conversion became the only option to prevent further collapse. Surgical repair was significantly complicated by prior sternotomies which increased difficulty due to adhesions and

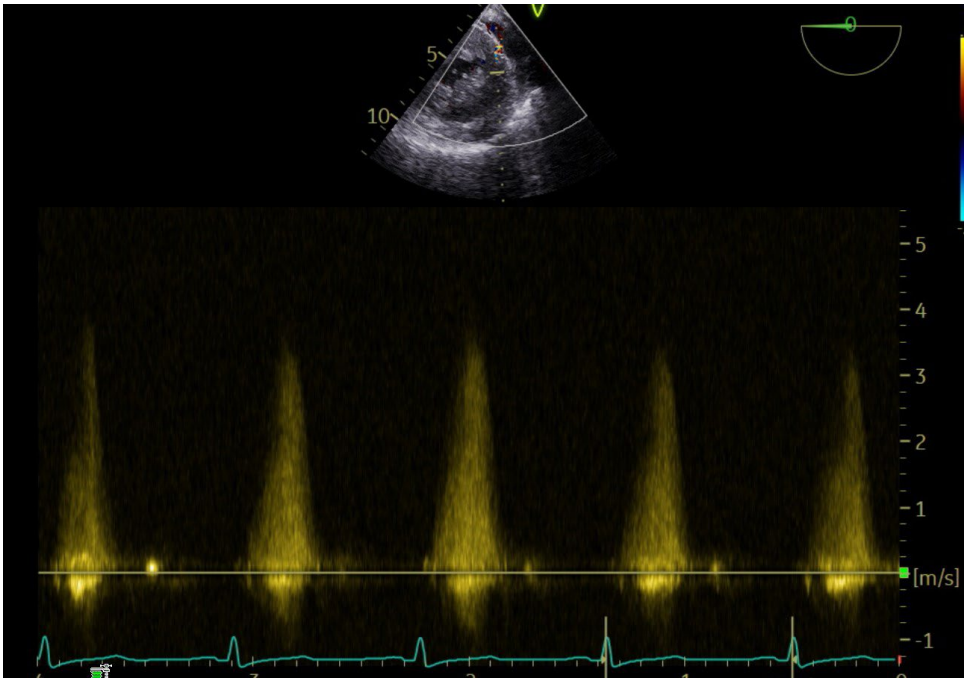
distorted mediastinal anatomy [3]. Despite these challenges, the LV perforations were successfully repaired and the patient recovered. This case highlights the importance of real-time collaboration among intraoperative teams including anesthesia, cardiac surgery, cardiology, perfusion, and nursing.

Images






Apical perforation
Inferolateral perforation
seen on TEE



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#FR 1 – Kiosk 19 – Poster 5

A Shocking Twist

Presenting Author: Theodore G. Black, MD, Fellow, Vanderbilt University Medical Center

Introduction

Shock treatment depends on accurate identification of the underlying etiology, so that derangements can be reversed and adequate tissue perfusion restored. This is increasingly difficult if mixed shock states develop, often with competing interventions. Patients may develop heart failure symptoms or even cardiogenic shock due to initial therapy or from progression of the underlying insult. One study found that 20.5% of patients with sepsis developed cardiac dysfunction (LVEF < 55%) and 21.7% of those with dysfunction developed cardiogenic shock (CI < 2.1). The strongest predictor of developing cardiac dysfunction was prior dysfunction [1].

Case Presentation

26yoF, G4P4 and 10mo post-partum, and no other medical history presented with an ovarian torsion. Following reduction, she re-presented with hemoperitoneum. Post evacuation and salpingo-oophorectomy she was transferred to VUMC SICU with ongoing vasopressor requirement. History and evaluation with positive FAST, hyperdynamic POCUS, and collapsable IVC prompted concern for hypovolemic/distributive shock. Following resuscitation and initiation of antibiotics, hemodynamics improved with reduced vasopressor requirement. Abdominal CTA revealed no bleeding. She later developed hemodynamic lability, agitated delirium, and mild hypoxia. Repeat POCUS revealed LV dilation with mild systolic dysfunction, adequate CO/CI, and low SVR, with small bilateral pleural effusions. The next morning, the decision was made to perform emergent laparotomy given worsening abdominal exam and acidosis. Intra-op TEE for hemodynamic instability showed profound biventricular dysfunction. Despite addition of inotropes, she had worsening acidosis and was placed on peripheral VA ECMO. Intra-op findings were significant for no bleeding, mesenteric and visceral congestion, and murky ascites. She had a complicated hospital course including a left brachial artery thrombectomy, CFA to SFA dissection requiring stenting, and S. pyogenes surgical site infection. However, after a 5-day course on VA-ECMO, she was weaned from MCS and inotropic support with recovery of function.

Discussion

With no predisposing risk factors, she ultimately developed a mixed cardiogenic and distributive shock state. Etiology remains inconclusive but was likely due to septic cardiomyopathy given the rapid recovery following treatment of the underlying cause. It is important to note that VA-ECMO is controversial in the setting of sepsis alone and requires special considerations for patients with mixed shock. Concerns with VA-ECMO in the context of sepsis include high flow requirements, coagulopathy, pharmacologic consequences, and possible nidus of infection. High flow requirements require substantial preload to avoid access insufficiency. The retrograde flow increases afterload and myocardial oxygen demand. Both contribute to vascular congestion, edema, and reduced LV function. The mechanical requirement to

maintain flow also predisposes to hemolysis. Sepsis and ECMO circuits are associated with hypercoagulable states and microthrombi, consumptive coagulopathy, and thrombocytopenia which is an independent predictor of mortality in sepsis. Pharmacologically, the ECMO circuit can lead to lower serum concentration of necessary antibiotic therapy [2].

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#FR 1 – Kiosk 19 – Poster 6

Emergent Extracorporeal Membrane Oxygenation for Intraoperative Pulmonary Embolism With Decannulation Despite Persistent Pulmonary Artery Thrombus

Presenting Author: Samar Ayoub, MD, Resident, University of Virginia

Introduction

Pulmonary embolism (PE) is a feared intraoperative complication, and can be difficult to recognize under general anesthesia. Diagnosis requires high clinical suspicion and is one of exclusion.[1] Potential signs are nonspecific - tachycardia, hypoxia, and hypotension - and early indications involve a sudden drop in end-tidal carbon dioxide (CO_2), paired with an elevated $PaCO_2$.[1] Bedside transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) are effective methods to identify clot in transit or right heart strain, though may be challenging in a prone patient. The following case discusses the diagnosis and complex management of an intraoperative PE during a prone thoracic spine surgery, and its sequalae – emergent extracorporeal membrane oxygenation (ECMO), thrombectomy, decannulation, and repeat thrombectomy.

Case Presentation

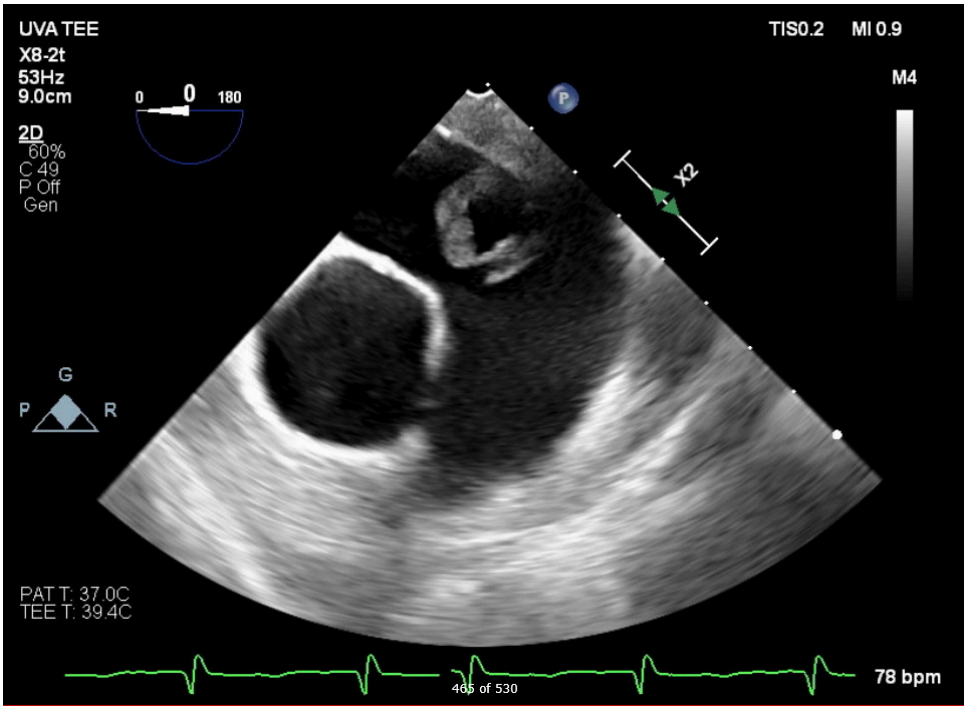
A 74-year-old male with gait imbalance and left sided weakness arrived from home for an elective thoracic spine decompression, instrumentation and fusion. Induction was uneventful, but the patient required progressively increasing vasopressor support to maintain neurosurgical mean arterial pressure (MAP) goals. After case completion and flipping supine, the patient acutely decompensated with end-tidal CO_2 decreasing to 20, and MAP dropping to 40. Bedside TTE was immediately conducted, showing a large, mobile right atrial thrombus intermittently crossing the tricuspid valve. A pulmonary embolism response team was activated, systemic thrombolytics were given, and ECMO was consulted. The patient progressed to PEA arrest, requiring four minutes of CPR. TEE demonstrated migration of the thrombus to the main and right pulmonary artery and a new, large pericardial effusion with impending tamponade physiology. A pericardial drain was placed, venoarterial ECMO (VA-ECMO) initiated, and the patient was transferred to the cardiac ICU for acute hemodynamic management. CT imaging that evening confirmed a saddle PE with right heart strain. The patient underwent urgent interventional radiology thrombectomy with successful clot removal confirmed by TEE intraoperatively. On postoperative day (POD) 3, he returned to the OR for ECMO decannulation. TEE at that time revealed another large, mobile thrombus at the bifurcation of the pulmonary artery with extension into both right and left branches. Cardiac surgery proceeded with decannulation and treated the recurrent PE with heparin. Hematology was consulted, and opined that the saddle PE was additional clot in transit from the lower extremities, not a new clot. Repeat CT angiography confirmed persistent saddle PE extending into multiple lobar and segmental branches. Repeat thrombectomy was successfully performed two days later.

Discussion

This case demonstrates a catastrophic intraoperative event and the importance of utilizing readily available

noninvasive methods of diagnosis. It also highlights the challenges when such imaging is limited by surgical position. Prompt recognition and multidisciplinary discussion with anesthesiology, neurosurgery, and cardiothoracic surgery were vital in an evolving, critical situation. Furthermore, this case underscores the risk-benefit considerations involved in balancing hemodynamic instability, anticoagulation, and surgical recovery in the postoperative period.

Images



Intraoperative TEE demonstrating large thrombus at the bifurcation of the pulmonary artery

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#FR 1 – Kiosk 19 – Poster 7

A Medically Challenging Case of Aortic Valve Disease and Ascending Aortic Aneurysm in a 21-Year-Old Male

Presenting Author: Travis Lariviere, D.O., Fellow, Vanderbilt University Medical Center

Introduction

Management of ascending aortic aneurysm in a young patient with a bicuspid aortic valve (BAV) and aortic stenosis presents several challenges. The decision between aortic valve repair versus replacement has long-term implications for durability, anticoagulation, and quality of life. Preserving the native valve avoids lifelong anticoagulation but success depends on valve morphology and cusp pathology. Mechanical valve placement provides predictable outcomes but requires anticoagulation. The Ross procedure—replacing the diseased aortic valve with the patient's own pulmonic valve—offers a durable solution without anticoagulation. This case involves a 21-year-old male with BAV and ascending aortic aneurysm, highlighting considerations in valve preservation versus replacement.

Case Presentation

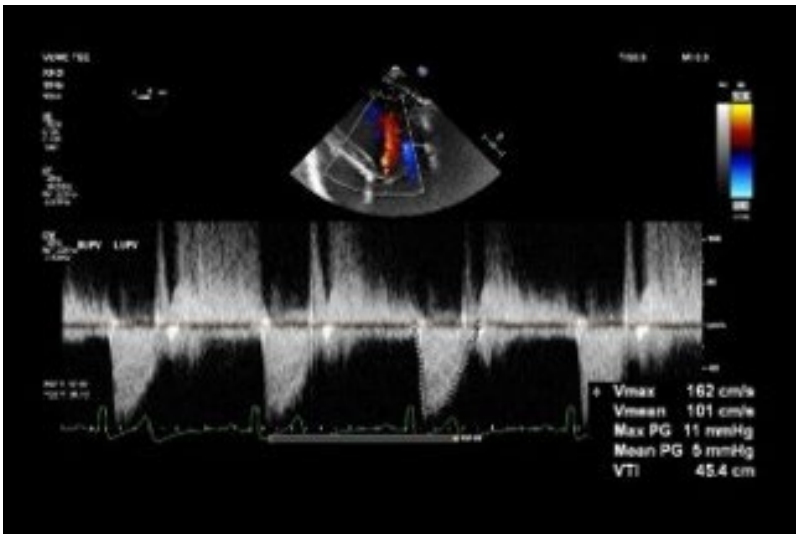
The patient is a 21-year-old male with a known ascending aortic aneurysm and BAV with mild stenosis and moderate regurgitation. His ascending aortic aneurysm had increased from 4.4 cm to 4.9 cm over the prior year. He was otherwise healthy and works in manual labor without functional limitations. His family history was significant for BAV and a family member who died at 30 years of age from an aortic dissection. Genetic testing for aortopathies was negative. In the operating room, a pre-induction arterial line was placed, followed by anesthesia induction and intubation. A central line and pulmonary artery catheter were inserted. Pre-procedure TEE showed an ascending aorta measuring 4.6 cm with incomplete fusion of the left and right AV cusps, mild aortic stenosis, and moderate aortic insufficiency with a single central regurgitant jet. The surgeon replaced the ascending aorta, while preserving the aortic root, and plicated the aortic valve leaflets. After weaning from cardiopulmonary bypass TEE revealed a mean gradient of 17 mmHg across the repaired valve, and worsened AI with now two regurgitant jets and an increased effective regurgitant orifice. The decision was made to resume CPB and proceed with the Ross procedure. Following separation from CPB, an assessment of the aortic position valve revealed no AI and insignificant AS. The patient was brought to the ICU on a nicardipine infusion. On postoperative day four he was discharged without anticoagulation.

Discussion

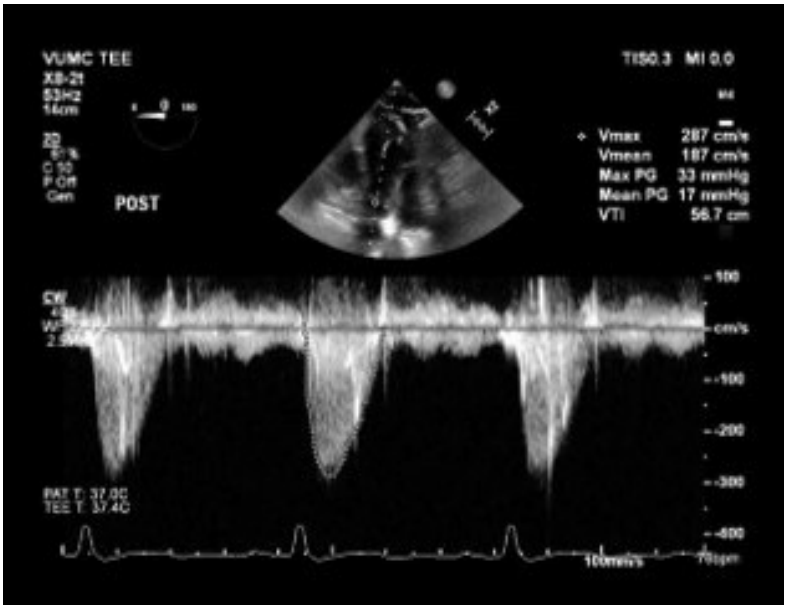
A successful repair of the AV would have been the preferred management for this patient, however, the presence of residual regurgitation and worsening stenosis led to conversion to a Ross procedure. The Ross offered the benefits of improved valve function and the avoiding anticoagulation required for mechanical valves. Risks of the Ross procedure include autograft dilation, progressive root enlargement, and the potential for reintervention. Based on a study of long-term outcomes by Aboud and colleagues, this patient's pre-existing AI placed him at higher risk of reintervention but similar long-term survival as other patients following Ross. In this case, a technically demanding procedure was worth the risk and prevented lifelong

anticoagulation.

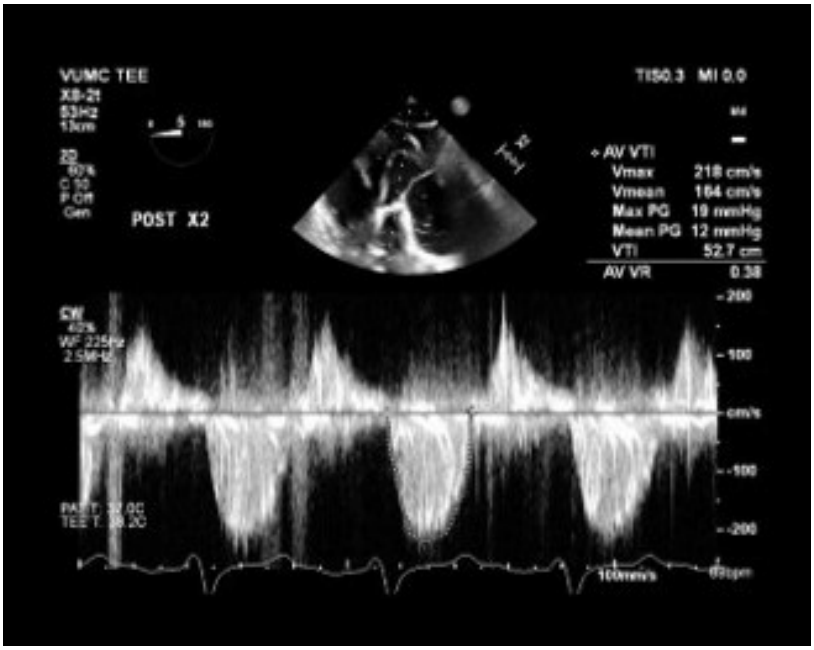
Images



Continuous wave doppler of aortic valve prior to repair. Mean PG 5 mmHg.



Continuous wave doppler of aortic valve status post repair. Mean PG 17 mmHg.



Continuous wave doppler of aortic position valve status post Ross procedure. Mean PG 12 mmHg.

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#FR 1 – Kiosk 20 – Poster 1

Anesthetic Considerations for Combined Heart and Liver Transplantation in a Pediatric Patient

Presenting Author: Tina Jing, MD, Resident, New York Presbyterian - Columbia

Introduction

Combined heart and liver transplantation (CHLT) is rare with a total of 719 performed in the USA between 1988 and 2025, with only 38 (5.3%) age less than 18[1]. Pediatric CHLT is usually for complex congenital heart disease[2], a group with unique perioperative considerations. We report a successful CHLT in a 14-year-old boy with a failing Fontan and associated liver disease (FALD). Successful performance of this operation required comprehensive planning and close coordination.

Case Presentation

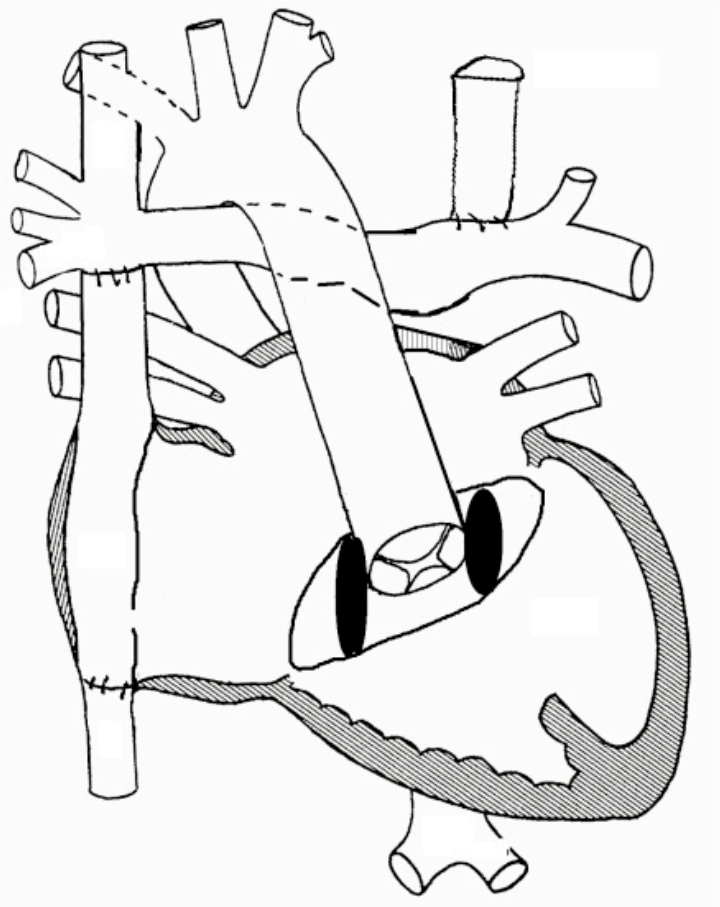
A 14-year-old male patient with a history of heterotaxy (RA Isomerism) complex single ventricle s/p bidirectional Glenn and intracardiac-extracardiac fenestrated Fontan, mechanical tricuspid valve replacement and FALD presented for CHLT (Fig 1). He was maintained on milrinone (0.5 mcg/kg/min), sildenafil, oral amiodarone, and oral bumetanide. Intravenous midazolam was dosed for anxiolysis. Worsening anxiety during arterial line insertion was treated with nitrous oxide. Induction of anesthesia, endotracheal intubation, central line placement, and insertion of transesophageal echocardiogram (TEE) probe were performed without issues. Epinephrine was started at 4mcg/kg/min. TEE demonstrated a single ventricle with intact fenestrated Fontan and a mechanical AV valve with normal function (Figure 1). Initiation of cardiopulmonary bypass, heart explant, and donor heart implant were performed without complication. The patient was weaned from bypass onto ECMO, and dobutamine and inhaled nitric oxide were started for inotropy and right ventricular afterload reduction. Transfusion requirements were modest. TEE demonstrated mildly reduced biventricular function. After satisfactory hemostasis and with the chest open, the liver transplant teams assumed care. Resuscitation was guided by TEE for a goal CVP 8-10 mmHg. Portal vein and caval clamping were well tolerated. Hypotension and bradycardia at reperfusion were treated with epinephrine. After reperfusion, right ventricular function was decreased, associated with transient increase in CVP to 20 mmHg, low portal flows, and liver congestion. This improved with ligation of the left renal vein in the setting of multiple splenorenal shunts. Cardiac anesthesiology team resumed care for ECMO weaning and chest closure. The patient was extubated POD1 in the adult ICU and weaned off vasoactive support on POD3. He was then transferred to the pediatric ICU and discharged home on POD22.

Discussion

The surgical complexity of CHLT requires a comprehensive plan with input from all specialists. Because these cases are few, these operations should be performed at centers with deep experience, with specialists who can identify and balance competing goals. ECMO allows for reduction of right ventricular work during the hemodynamic perturbations of liver transplant. Promptly addressing splenorenal shunts was critical. The operations were performed at the adult hospital with experienced teams. The pediatric team guided immunosuppression, medication dosing strategies, and psychosocial care. Hospital and

clinical leadership vetted the plan. The cardiac anesthesiology team remained available for assessment and recommendations throughout the liver transplantation. As the number of CHLT continues to rise globally, there is a growing need for clear guidelines and practices.

Images



Pre-transplant anatomy including heterotaxy (right atrial isomerism), complex single ventricle, and bilateral superior vena cava s/p intracardiac-extracardiac fenestrated Fontan, common atrioventricular valve replacement (mechanical)

References

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#FR 1 – Kiosk 20 – Poster 2

Optimizing Anesthetic Care for Severe Right-Sided Carcinoid Heart Disease: A Case Report

Presenting Author: Parthik D. Patel, MD, University of Pennsylvania

Introduction

Carcinoid syndrome can lead to progressive right-sided valvular dysfunction due to the fibrotic effects of serotonin and other vasoactive mediators on the endocardium.[1] Severe tricuspid and pulmonic valve regurgitation or stenosis may develop, resulting in right-sided heart failure and often necessitating surgical intervention.[2] Peri-op management is challenging due to the risk of hormonal instability and intra-op carcinoid crisis, particularly during induction, sternotomy, and weaning from cardiopulmonary bypass.

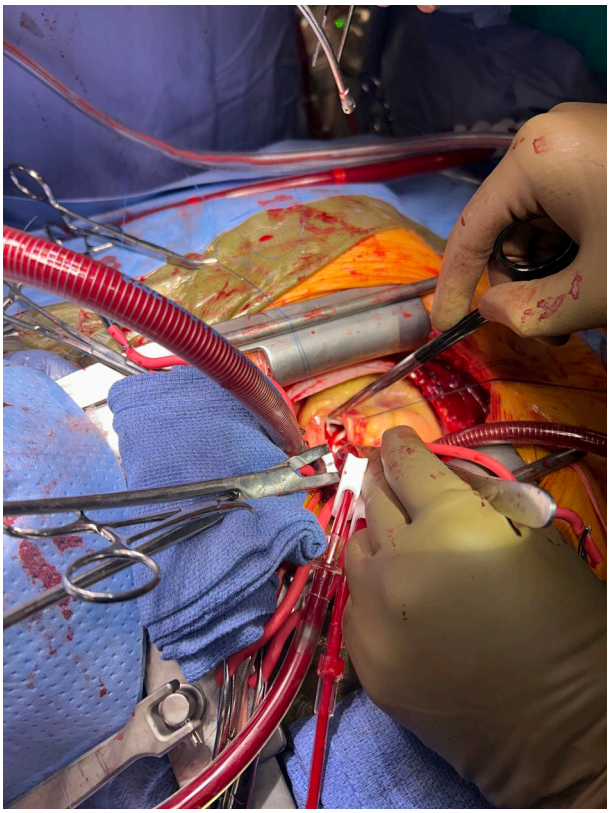
Case Presentation

A 50-year-old female PMH of multiple sclerosis (not on treatment) was found to have a neuroendocrine tumor during screening colonoscopy. She reported a 1-2-month history of intermittent full body flushing, diarrhea, and abdominal cramping. CT showed lesions in both lobes of the liver as well as an abnormality at the ileocecal junction. She underwent multiple surgeries and debulking, was started on monthly lanreotide, and had two chemoembolization procedures for tumor burden of the liver. She reported dyspnea on exertion and limited exercise tolerance a month after debulking surgery. TTE showed normal biventricular function, severe TR, and severe PR. Cardiac cath showed non-obstructive CAD, as well as pronounced v-waves on CVP tracing. She was recommended for tricuspid and pulmonic valve replacement. She was given her last lanreotide injection one-week prior to surgery. Pre-induction the patient was given a 50-µg bolus of octreotide, followed by infusion at 50 µg/hr that was continued into the postop period. Induction was uneventful. Intraop TEE demonstrated severe TR with restricted septal leaflet and thickening/retraction of all three pulmonic leaflets. Patient then underwent PVR and TVR. She came off bypass with no vasoplegia. TEE showed normal biventricular function with well-seated valves and appropriate gradients. Complete heart block (CHB) was noted post-bypass, and epicardial leads were placed. She was transferred to the ICU on minimal support. She was extubated within 6 hours. Inopressor support was weaned off slowly and uneventfully. Octreotide infusion was continued for 24 hours postop prior to being discontinued. Due to persistent CHB, a dual chamber pacemaker was placed on postop day 3.

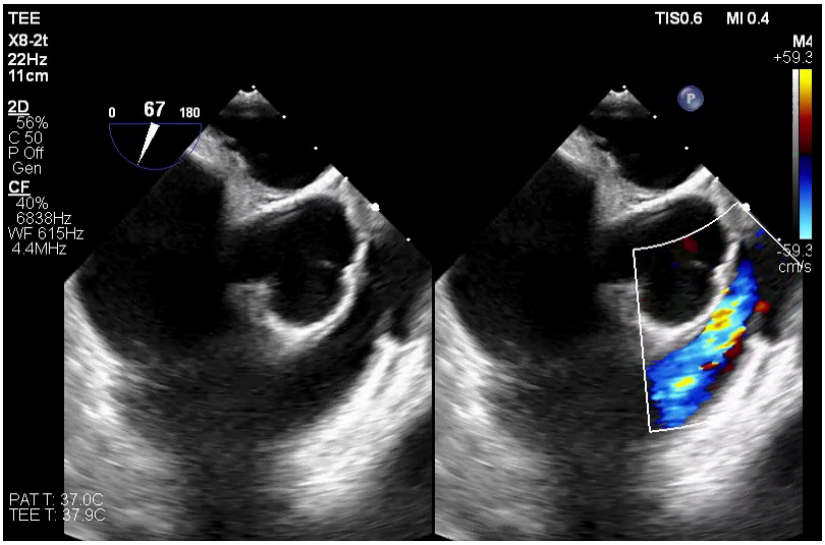
Discussion

Management of Carcinoid Heart disease (CHD) in the periop period must reflect consensus and guideline-based practice, multidisciplinary coordination, evidence-based use of somatostatin analogs, and careful hemodynamic control. Current guidelines recommend all patients with neuroendocrine tumors, especially those with CHD or high biochemical burden, receive periop prophylaxis with IV Octreotide beginning at least 12 hours before anesthesia and maintained during and after surgery.[3] Infusions start at 50–100 µg/h with upward titration depending on symptoms or hemodynamics. While our patient did well with a bolus and infusion, this case demonstrates the need for institutional adoption of a standardized protocol. In addition to IV octreotide, anesthetic management focuses on avoiding drugs known to provoke histamine release or sympathetic surges.[2]

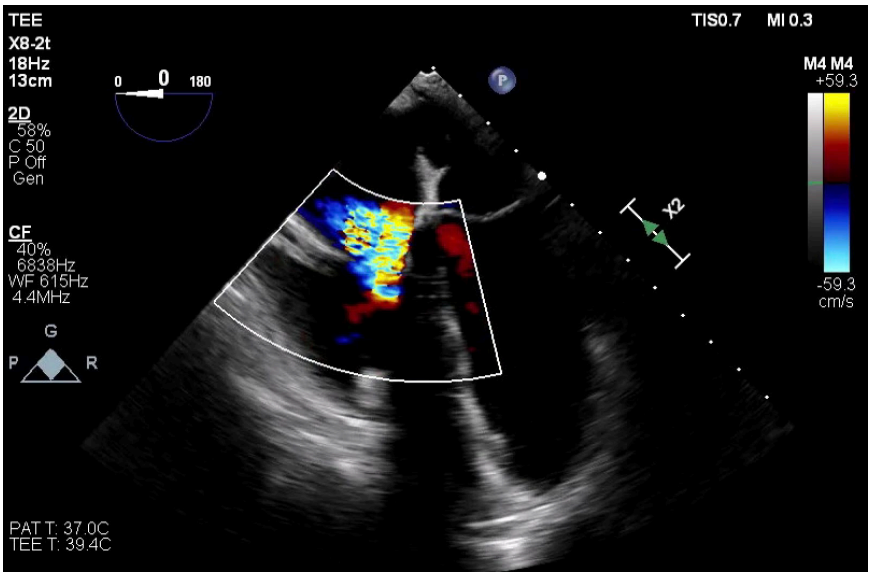
Images



Intraoperative image of pulmonic valve with thickened and reatracted leaflets.



Tranesophageal echocardiography image of severe pulmonic insufficiency.



Tranesophageal echocardiography image of severe tricuspid valve insufficiency with septal leaflet restriction.

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#FR 1 – Kiosk 20 – Poster 3

Impella CP-Shaft Thrombosis and ECMO: Double the Trouble

Presenting Author: Katherine Dudley, MD, UC San Diego

Introduction

Cardiogenic shock (CS) describes a state of reduced myocardial function that results in decreased cardiac output and end-organ hypoperfusion/hypoxia. While vasopressor and inotropic support have historically been the mainstays of therapy, recent evidence suggests that early initiation of mechanical circulatory support (MCS) improves mortality[1]. Both VA-ECMO and Impella are increasingly employed in severe CS. When used together—termed ECPella—these devices unload the left ventricle (LV) and augment systemic perfusion while awaiting myocardial recovery. We describe a case of Impella CP–associated thrombus in the descending thoracic aorta identified by intraoperative transesophageal echocardiography (TEE) in a patient receiving ECPella support.

Case Presentation

An otherwise healthy 41-year-old female, who presented with nausea, was discovered to have heart failure with reduced ejection fraction. Initial transthoracic echocardiogram (TTE) revealed severely reduced LV systolic function with ejection fraction of 15-20%, with mild right ventricular dysfunction. Despite maximal medical therapy, her clinical status worsened, and she was initiated on femoral VA-ECMO and femoral Impella CP. Clinical suspicion for heparin induced thrombocytopenia (HIT) let to bivalirudin use for systemic anticoagulation. Persistent CS prompted a plan to upgrade to an Impella 5.5. Intra-operative TEE revealed spontaneous echo contrast and a mobile thrombus (1.2 x 0.3cm, figure 1) within the descending thoracic aorta, adherent to the Impella shaft. Given the risk of thrombus embolization with Impella removal, the procedure was aborted. The patient remained in the ICU on systemic anticoagulation and thrombus resolution was monitored with serial TEE. On day 16 of MCS, TEE demonstrated significantly reduced thrombus burden (0.3 x 0.3cm, figure 2). The Impella CP was removed without complication and successfully upgraded to Impella 5.5.

Discussion

ECPella use has been associated with lower mortality compared with VA-ECMO alone[1]; however, it is not without some unique risks. In a study by Cho et al., 13 of 98 patients (13.3%) treated with ECPella developed descending thoracic aortic thrombi, detected by TEE or CTA. This phenomenon is likely related to flow stasis at the mixing zone of antegrade Impella flow and retrograde arterial VA-ECMO flow. The risk of thrombus formation is also elevated due to bivalrudin degradation in this zone. Perioperative echocardiographers should carefully evaluate the Impella shaft within the descending thoracic aorta to avoid embolization during device removal. Embolic shower upon removal of femoral Impella can be directed cranially in the setting of retrograde flow from ECMO, thus causing catastrophic occlusive complications and stroke. Our case emphasizes the use of perioperative TEE assessment for thrombus and reviews the clinical decision-making should one be found. Ultimately, vigilance is the key to preventing embolic complications and

improving procedural safety.

Images



Figure 1. Mobile thrombus (1.2 x 0.3cm, figure 1) within the descending thoracic aorta, adherent to the Impella shaft

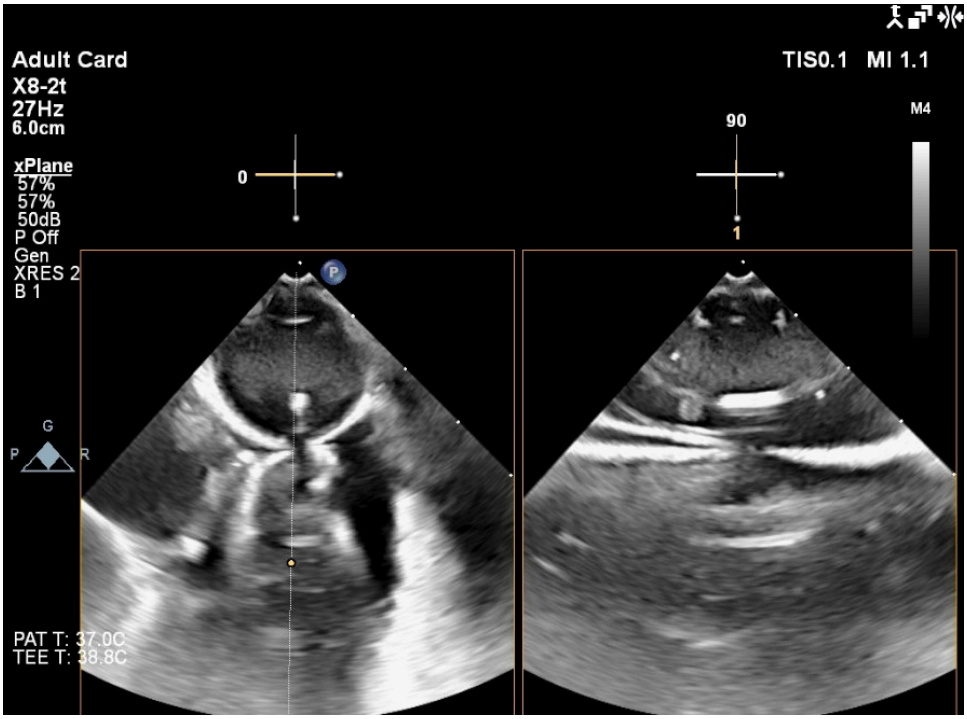


Figure 2. TEE demonstrated significantly reduced thrombus burden (0.3 x 0.3cm).

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#FR 1 – Kiosk 20 – Poster 4

Elevated Ischemic Stroke Risk During Multivessel Coronary Artery Bypass Graft

Presenting Author: Evan R. Birmingham, University of Kentucky Department of Anesthesiology

Introduction

The risk of stroke after coronary artery bypass grafting (CABG) can be influenced by many factors in a patient's history including age, hypertension, diabetes mellitus, renal disease, peripheral vascular disease, atrial fibrillation, and carotid artery stenosis[1]. Other additive but previously unrecognized factors that may be visualized on intraoperative imaging include atherosclerosis of the ascending aorta and cardiac fibroelastomas. Perioperative strokes can lead to permanent disability, increased perioperative mortality, and prolonged hospital length of stay. This case demonstrates precautions able to be used in patients undergoing CABG who possess multiple risk factors for periprocedural stroke.

Case Presentation

This patient is an 81-year-old female who was transferred to the University of Kentucky from an outside hospital for surgical intervention of severe multivessel coronary artery disease (CAD). Medical history was pertinent for hypertension, hyperlipidemia, type 2 diabetes mellitus, and chronic kidney disease. Coronary angiography revealed severe 90-95% stenosis of the distal left main and severe stenosis in the ostial and proximal section of the OM1. Transthoracic echocardiogram (TTE) revealed global hypokinesis of the left ventricle, LVEF estimated 45-50%, and grade II diastolic dysfunction. Notably, bilateral carotid duplex revealed severe occlusive plaque in the right internal carotid artery (ICA) with no flow present by pulse wave or color doppler, consistent with total occlusion. Left ICA showed hemodynamically significant stenosis of 50-69%. It was discussed with the surgical team that based on the severity of this patient's CAD we would proceed with the scheduled CABG. Plan was to take precautions to maintain cerebral prefusion including perioperative mean arterial pressure (MAP) goals >80mmHg, as well as close cerebral pulse oximetry monitoring. Additionally, this case was complicated by the intraoperative detection by transesophageal echocardiography (TEE) of multiple fibroelastomas (Image 1) on the aortic valve. Cardiology was consulted to evaluate and confirm the TEE images. The fibroelastomas were resected and repeat TEE verified the absence of any additional masses. Exclusion of left atrial appendage was achieved via clipping and the patient was taken to the ICU after completion of procedure with no apparent perioperative complications. The patient was extubated post operative day 1 without incident but proceeded to evolve an ischemic stroke during recovery.

Discussion

The risk of stroke after CABG has many variable contributors but has been shown to approach 5%. The severity of this patient's disease precluded PCI and prevented off pump CABG, both of which have decreased peri-CABG stroke risk. Risk of fibroelastoma related ischemic events has been shown to be 6% at 1-year[2]. The strategies utilized in the case to minimize peri-CABG stroke risk included multidisciplinary discussion and risk factor targeted therapy. This case illustrates an example of complex decision making,

multidisciplinary communication, and risk stratification of a patient with several risk factors for stroke after CABG.

Images

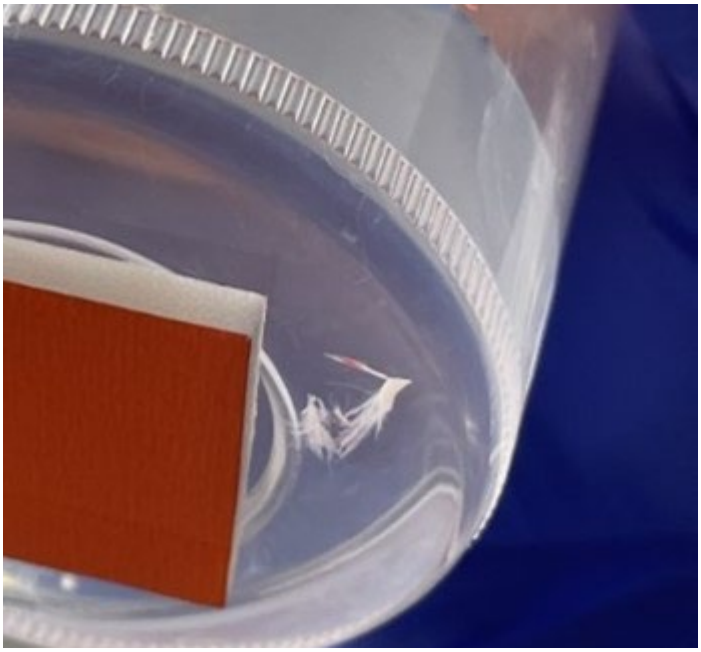


Image 1. Fibroelastomas removed from aortic valve

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#FR 1 – Kiosk 20 – Poster 5

Utilization of 3D and Advanced Echocardiography Techniques to Evaluate Possible Paravalvular Leak

Presenting Author: Bret G. DeGraaff, MD, Resident Physician, University of Washington Department of Anesthesiology and Pain Medicine

Introduction

Paravalvular leak (PVL) is defined as flow between a native valve annulus and the prosthetic sewing ring and is almost always pathologic. Incidence of early PVL after surgical mitral valve replacement ranges from 1.9% to 8%.1, 2 Quantification and characterization of PVL can be challenging with mechanical mitral valve replacement due to acoustic shadowing and the presence of intravalvular "washing jets." It is therefore imperative to have a systemic approach for interrogating the valve post replacement including multiple views and utilization of 3D echo techniques. Here we present a patient who was noted to have a jet which initially was concerning for PVL, however, 3D echo ultimately demonstrated the jet to be intravalvular. Follow-up echocardiogram confirmed the lack of PVL.

Case Presentation

The patient is a 24-year-old female with a history significant for rheumatic heart disease presented with several months of fatigue and shortness of breath. Transthoracic echocardiogram (TTE) showed severe aortic insufficiency (AI) and mitral regurgitation (MR). The patient proceeded to the operating room with an uneventful anesthetic course and transesophageal echocardiogram (TEE) confirming severe MR and AI with flow reversal in the abdominal aorta. Pre-operative valvular dysfunction can be seen in Figure 1. The patient underwent uncomplicated mechanical aortic and mitral valve replacement. An aortic root enlargement was performed due to a small aortic annulus. There were no difficulties in separating the patient from cardiopulmonary bypass (CPB) with moderate dose inotropic support. Postoperative TEE showed well-functioning aortic and mitral valves; however, there was a concern for an eccentric regurgitant jet in the mitral valve position. Upon initial evaluation, it was not consistent with a typical washing jet and it was difficult to discern the origin of the jet. Given the concern of PVL, a thorough evaluation of the valve was performed. Doppler showed a LV-LA pressure gradient suggesting MR and using multiple views in 3D imaging, the jet was determined to be intravalvular (Figure 2). With the possibility of PVL ruled out, it was decided that reestablishment of CPB was not indicated, and the procedure was completed. There was no evidence of residual MR or PVL on follow up TTE.

Discussion

This case demonstrates the importance of 3D imaging and advanced echocardiography techniques intraoperatively during valvular surgery. Significant PVL is pathological and requires reintervention. Intraoperative evaluation of PVL via TEE is critical to correct significant leaks prior to leaving the operating room. Utilization of enhanced imaging skills can save patients from unnecessary additional CPB runs and re-interventions.

Images

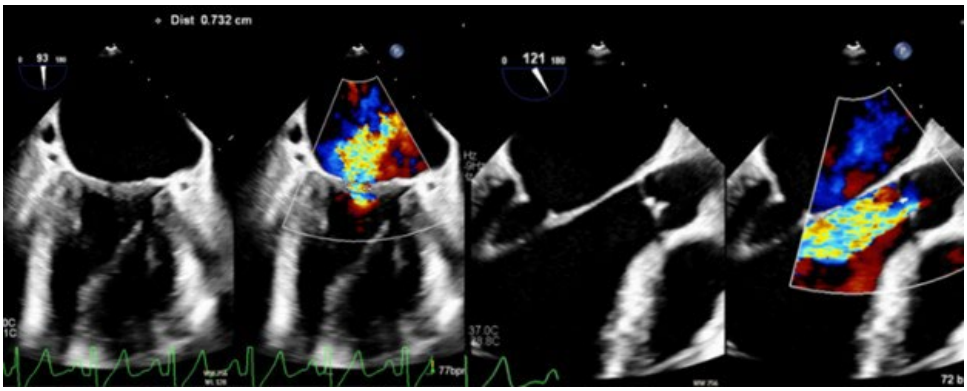


Figure 1: Pre-operative severe mitral regurgitation and aortic insufficiency.

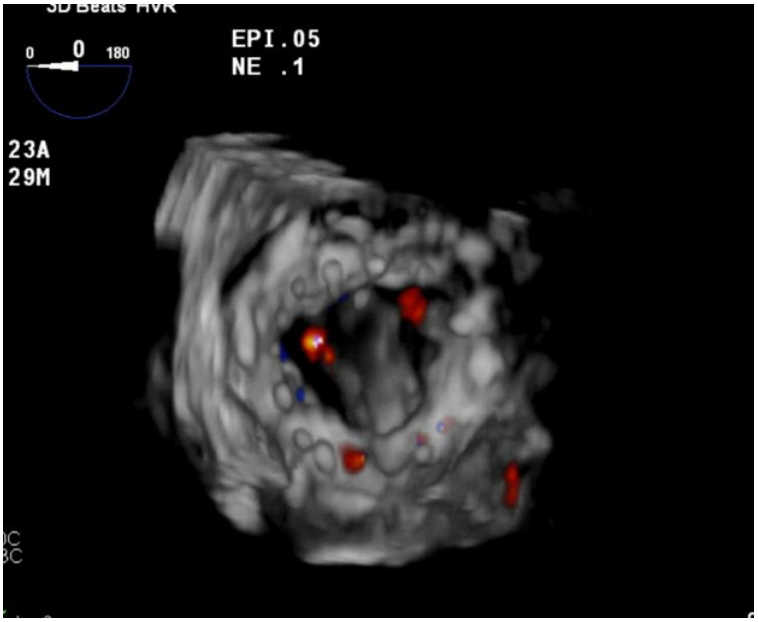


Figure 2: 3D ECHO image showing the intravalvular mechanical mitral valve jet.

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#FR 1 – Kiosk 20 – Poster 6

Mobile Ascending Aortic Thrombus with Systemic Embolization: Anesthetic and Surgical Considerations

Presenting Author: Charles A. McLendon, III, Fellow, Northwestern Memorial Hospital

Introduction

Aortic pathology is likely a significant source of cerebral and systemic embolization. Protruding mobile aortic thrombi are rare and have only been described in case reports. These thrombi likely carry a high risk for cerebral embolization, and early treatment is essential to avoid compounding ischemic events. Here, we present a case of a patient with a mobile protruding ascending aortic thrombus with visceral emboli requiring emergent surgical intervention.

Case Presentation

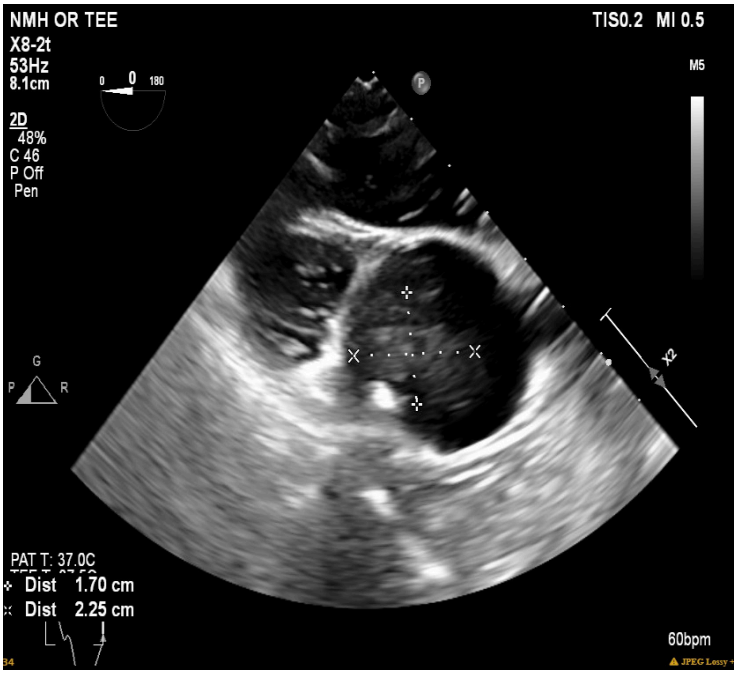
A 65-year-old man with coronary artery disease, hypertension, hyperlipidemia, diabetes, tobacco use, hypothyroidism, and prostate cancer presented with acute right-sided flank pain. CT angiogram revealed a 1.8cm mobile intraluminal mass in the ascending aorta. Splenic and renal infarcts were noted and attributed to his flank pain. The differential diagnosis included thrombus and vegetation, so antibiotics and anticoagulation were started. Given the size, mobility, and location of the mass with systemic embolization, it was deemed necessary to surgically remove the mass. To avoid shear stress on the mas, induction was aimed at avoiding abrupt hypertension and tachycardia. Cardiopulmonary bypass (CPB) with right axillary artery cannulation was selected to minimizing disturbance of the mass and facilitate anterograde cerebral perfusion (ACP) under circulatory arrest. The mass along with ascending aorta and hemi-arch were resected and replaced with a graft. Final pathology demonstrated protruding, calcified atherosclerotic plaque with superimposed thrombus.

Discussion

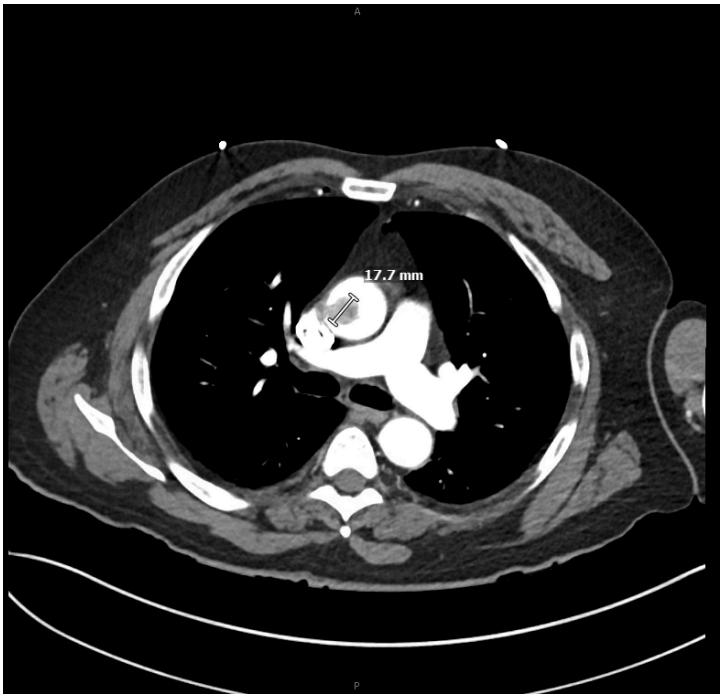
Cerebral embolic events have severe morbidity and mortality, and aortic sources may represent a significant burden for this disease. Aortic atheroma is a known risk factor for embolic stroke and may be a common source of embolism in cryptogenic strokes. However, thrombus formation in a non-aneurysmal thoracic aorta is rare due to the high blood flow.[1] One study estimated the incidence to be as low as 0.45%.[2] The pathogenesis is unclear but may involve vulnerable plaque rupture in the setting of systemic inflammation and prothrombotic states. Final diagnosis is made via histopathologic analysis. There are no treatment guidelines, but options include systemic anticoagulation, endovascular thrombectomy, and surgical excision with or without segmental aortic resection.[3] In high-risk patients, medical management may be best for asymptomatic patients while endovascular aspiration thrombectomy may be effective for symptomatic patients. In symptomatic patients who can tolerate it, surgical thrombectomy may be the best option. Although no guidelines exist, strategies to reduce intraoperative embolic risk in surgical thrombectomies may include: 1) gentle induction to avoid shear stress; 2) cannulation sites that avoid plaque disruption (ex. Right axillary); and 3) cerebral monitoring and ACP during circulatory arrest to maintain cerebral perfusion.[3]

Although ascending aortic thrombus is rare, its potential for catastrophic embolization underscores the need for early recognition and treatment.

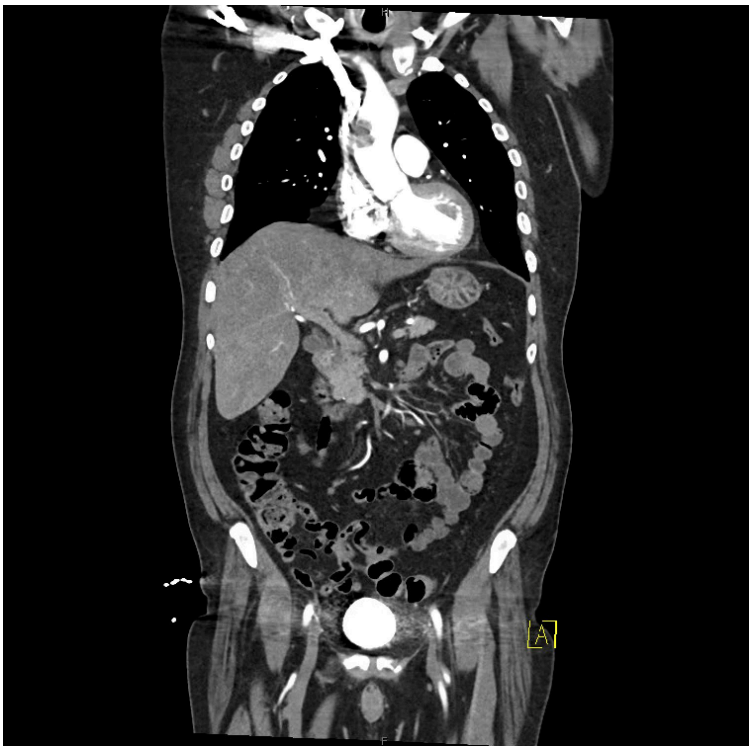
Images



TEE: mid-esophageal great vessel view of ascending aortic mass



CTA: transverse view of the ascending aortic mass



CTA: Coronal view of the ascending aortic mass

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#FR 1 – Kiosk 20 – Poster 7

Anesthetic Management for Surgical Aortic Valve Replacement in a Patient with a Unicuspid Aortic Valve, Severe Critical Aortic Stenosis, and Congestive Heart Failure: A Case Report

Presenting Author: Cameron L. Holguin, MD, Resident Physician, University of Texas Medical Branch, Department of Anesthesiology

Introduction

Unicuspid aortic valves (UAVs) are rare congenital anomalies with a prevalence of~0.02%. UAVs can be divided into two types based on leaflet opening: acommissural or unicommissural. Acommisural typically presents with symptoms of aortic stenosis (AS) at birth. Unicommissural UAVs are more common and less severe, typically presenting with symptoms between the 3rd and 5th decades of life.2,3 UAVs can be difficult to identify on transthoracic echocardiography (TTE).1,2 Aortic balloon valvuloplasty, surgical valvotomy, commissurotomy, Ross procedure, surgical valve repair, or surgical replacement are the most common treatment modalities.1,2 While transcatheter aortic valve replacement for UAV has been described in case reports, there is a high risk of paravalvular leak and device malposition due to their asymmetric shape.3

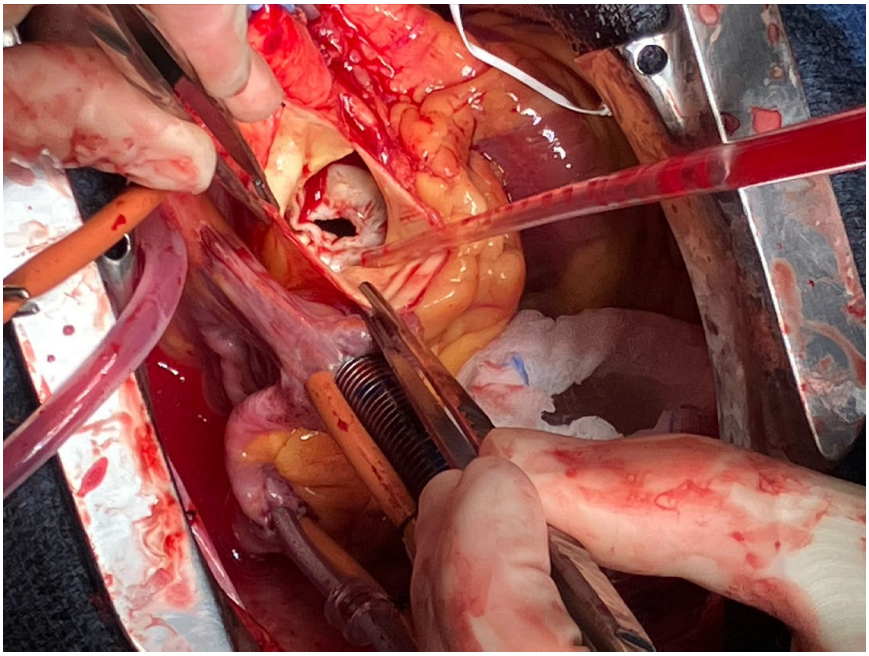
Case Presentation

We present the case of a 52-year-old male with past medical history significant for methamphetamine use who presented with increasing SOB and bilateral leg swelling. Laboratory studies on admission revealed elevated troponin and BNP. Chest x-ray revealed worsening pulmonary congestion and a right lower lobe consolidation. TTE and left heart catheterization were performed and revealed severe critical aortic stenosis, moderate aortic insufficiency, grade III diastolic dysfunction, and reduced ejection fraction (EF) of 20–25% with normal coronary arteries. After being treated for pneumonia, the patient was scheduled for surgical aortic valve replacement (SAVR). Peri-operative management began with the placement of a pre-induction arterial line. After induction of general anesthesia and endotracheal intubation, a central line was placed. Intraoperative transesophageal echo (TEE) was then performed. Findings included a unicommissural, unicuspid aortic valve with moderate calcification, severe aortic insufficiency, low-flow low-gradient critical AS, and global hypokinesis with an EF of 10–15%. Surgical excision of the patient's native valve confirmed the diagnosis of UAV. The patient required significant support with vasoactive medications following termination of cardiopulmonary bypass, including high dose norepinephrine, high dose vasopressin, epinephrine, and milrinone. Post-operative TEE showed improved EF (~20%) with inotropic support and a well-seated bovine aortic valve. The patient was extubated on post-operative day (POD) 0 and was weaned off vasopressors by POD 2. Invasive lines were removed by POD2 and the patient began mobilization with PT on POD3. He required external pacing support until POD3. He was able to be weaned from inotropic support on POD4 and was subsequently downgraded from the ICU. His post-operative course was complicated by atrial fibrillation with rapid ventricular response and acute kidney injury. His atrial fibrillation was controlled with metoprolol and digoxin. His kidney injury improved over time. He was deemed stable for discharge on POD6.

Discussion

UAV are rare congenital defects that may present later in life with symptoms of aortic stenosis. Surgical management is necessary, with high risk of perioperative hemodynamic instability and conduction disturbances—often requiring pacing or permanent pacemaker placement.

Images







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#FR 1 – Kiosk 21 – Poster 1

Perioperative management of patients with sickle cell disease undergoing cardiac surgery on cardiopulmonary bypass

Presenting Author: Jasmine M. Kim, MD, Fellow, University of Michigan

Introduction

Sickle cell disease (SCD) is the most common inherited blood disorder. SCD is characterized by abnormal hemoglobin (HbS) that polymerizes under low oxygen tension when homozygous for HbS[1]. These sickled cells then cause microvascular occlusion, reperfusion injury, and endothelial dysfunction[1]. Triggers for sickle cell crises such as hypothermia, hypoxia, acidosis, and low flow states are also conditions often associated with cardiopulmonary bypass (CPB)[2]. The management of patients with SCD undergoing cardiac surgery can therefore pose significant challenges. We present a case of a patient with SCD who underwent tricuspid valve repair on CPB.

Case Presentation

The patient was a 31-year-old male with tricuspid valve endocarditis in the setting of long-standing central line placement for monthly exchange transfusions. He was found to have residual thrombus on his tricuspid valve following catheter thrombectomy and severe tricuspid regurgitation (TR). He presented for tricuspid valve repair. Preoperatively, he underwent partial exchange transfusion with resulting HbS level of 6.3% (normal < 10%). Intraoperatively, general anesthesia was induced without complication. Transesophageal echocardiography revealed normal biventricular function and moderate to severe TR. A complete exchange transfusion was performed before initiation of CPB. The CPB circuit was primed with five units of packed red blood cells, five units of fresh frozen plasma, and heparin 400 U/kg. The patient was exsanguinated without any flow initially out of the aortic cannula. The blood was deposited into a discard cardiotomy reservoir until loss of arterial line pulsatility (Image 1). The CPB prime was then administered anterograde via the aortic cannula. Immediately after the exchange transfusion, midazolam, fentanyl, rocuronium, and antibiotics were re-dosed. The patient was kept at a temperature of 37 degrees Celsius. The aortic cross clamp was applied, and the heart was arrested with warm crystalloid cardioplegia. The perfusionist ensured high flows and avoidance of acidosis for the duration of CPB. Prior to separation from CPB, hemoconcentration was utilized. The patient separated from CPB without complication. He was taken to the intensive care unit on a norepinephrine infusion. He was extubated the next day.

Discussion

This case highlights key management considerations for patients with SCD undergoing cardiac surgery. Preoperatively, partial exchange transfusion should be utilized to lower HbS levels to mitigate the risk of sickling intraoperatively. Intraoperatively, special care should be taken by the anesthesiologist and the perfusionist to avoid conditions that could precipitate a sickle cell crisis such as hypothermia, acidosis, hypoxia, and hypovolemia. Complete exchange transfusion is a unique procedure that requires careful communication between the anesthesiologist and perfusionist. Intravenous anesthetics, paralytic,

antibiotics, and systemic heparin must be re-dosed after completion of exchange transfusion. Special CPB considerations the anesthesiologist should also be aware of include the use of crystalloid-only cardioplegia and caution with hemoconcentration as deoxygenation during transit of the hemoconcentrator can induce sickling.

Images



Image 1: Completed intraoperative exchange transfusion.

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#FR 1 – Kiosk 21 – Poster 2

Post Transcutaneous Aortic Valve Replacement paravalvular leak management; The role of advanced 3-Dimensional echocardiography

Presenting Author: Connor B. Milone, M.D., Resident, Dartmouth Hitchcock Medical Center

Introduction

Paravalvular leak (PVL) after transcutaneous aortic valve replacement (TAVR) is a feared complication with significant impact on post TAVR outcomes. PVL ^{3} mild was associated with increased cardiovascular and all-cause mortality in 2 metanalyses of >45,000 patients[1]. Accordingly, prevention and early detection of TAVR associated PVL is critical. Several modalities can be utilized to localize, define severity and treat PVLs[2] (table 1). This is a report of a patient presenting for vascular plug occlusion of a moderate to severe post TAVR PVL where quantitative 3D echocardiography, grey scale optimization, and advanced software (GlassVue™) provided a more precise definition of the paravalvular pathology and proved essential in guiding the appropriate intervention.

Case Presentation

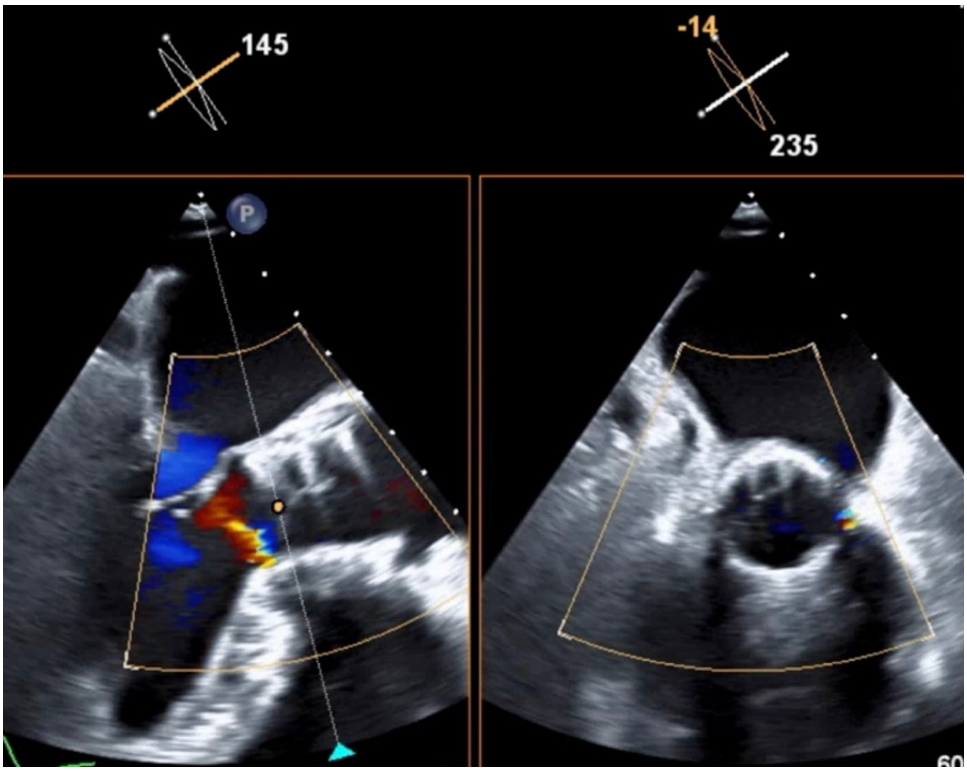
This is a 91-year-old man who underwent a TAVR procedure with a #26 balloon expandable valve (BE). The procedure was uncomplicated and post TTE showed peak/mean gradients (P/MG) of 9/4 mm Hg with trace PVL. The patient had progressive exertional dyspnea 4 weeks later. Repeat TTE demonstrated AV P/MG of 22/13 mmHg and a moderate PVL. TEE was completed and showed a potential moderate to severe anterior PVL encompassing ~ 50% of the AV circumference. Decision was made to pursue percutaneous vascular plug under general anesthesia with TEE guidance. Intraoperatively, TEE imaging in 2D color flow showed an anteriorly originating eccentric jet of mild to moderate severity by visual assessment (figure 1). Further interrogation, utilizing 3D color MPR with “hide greyscale” function and GlassVue™ technology at a higher transparency setting, allowed the visualization of 3 complex PVL jets (figure 2). There were 2 posterior serpiginous jets at 11 and 1 o’clock position on the enface view and a third anterior jet emerging at 4 to 6 o’clock whose origin was difficult to visualize due to the acoustic shadowing from the valve frame. To assess the severity of aortic insufficiency, 3D summative effective regurgitant orifice area (EROA) is a feasible option, albeit time consuming and subject to image quality limitations and user experience. To define the regurgitant severity we resorted to a volumetric method; the difference in stroke volume between right and left ventricular outflow tracts. The patient had no pulmonic or mitral valve pathology and was in an atrial paced rhythm. This analysis yielded AV regurgitant volume of 19 ml and regurgitant fraction of 28% which falls in the mild to moderate category. Our assessment of AI may have been underestimated secondary to altered loading conditions (general anesthesia and positive pressure ventilation). Based on these findings, we decided to abort the vascular plug placement, and the patient received a valve-in-valve 26 BE TAVR the following day.

Discussion

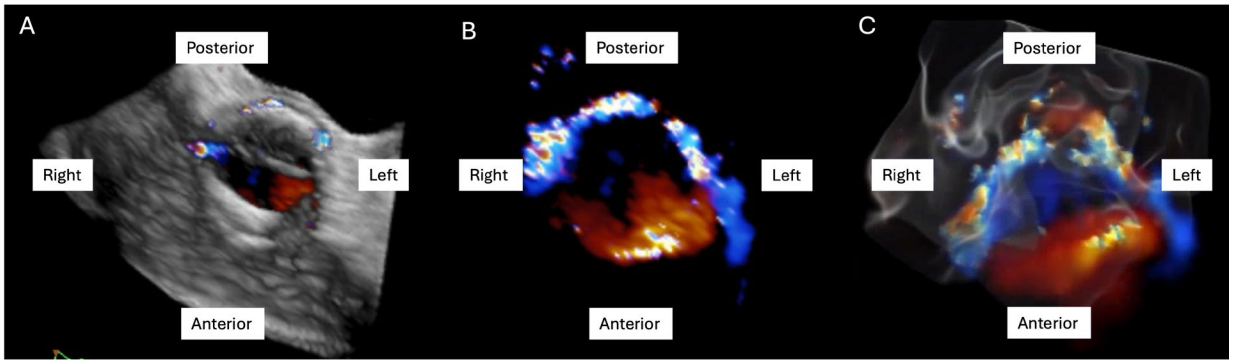
The presence of post TAVR PVL ^{3} mild severity can increase all-cause mortality. PVL repair includes plug,

valve in valve TAVR, and surgical replacement[3]. 2D assessment can be limited by acoustic shadowing from the TAVR frame. In our case, 3D assessment with image optimization demonstrated three separate PVL, which limits the therapeutic benefit of a plug device and thus 3D echocardiography and quantitative echocardiography helped decide the optimal intervention for this patient.

Images



TEE imaging in 2D and 2D color showed an anteriorly originated posteriorly directed eccentric jet of mild to moderate severity by visual assessment.



We visualized 2 posterior narrow and high velocity jets originating at 11 and 1 o'clock on the en-face view with a serpiginous course emerging at 9 and 3 o'clock on the LVOT side, respectively. A third wider jet

emerged on the LVOT side from 4 to 6 o'clock, but origination was difficult to visualize due to shadowing. A. 3D with color, B. 3D with color with "Hide Grey Scale" function, C. GlassVueTM enhanced 3D

Table 1. Various modalities for diagnosis and quantification of post TAVR PVL

Modality	Specific Features	Strengths	Limitations	Clinical Utility
TTE (Transthoracic Echo)	2D/Color Doppler; initial screening; assesses LV size/function, gradients	Widely available; noninvasive; good for anterior leaks and overall cardiac assessment	Acoustic shadowing from prosthesis; suboptimal for posterior leaks; limited spatial resolution	First-line for initial assessment, severity grading, and LV response
TEE (Transesophageal Echo)	2D/3D imaging; multiplanar; color Doppler; direct visualization of sewing ring	Superior localization and characterization; essential for procedural planning	May miss anterior defects; acoustic shadowing; semi-invasive	Gold standard for PVL localization, sizing, and guiding intervention
3D TEE	Multiplanar reconstruction; direct vena contracta measurement	Precise defect sizing and location; improves device selection for closure	Image quality affected by artifacts; less validated for aortic than mitral PVL	Best for procedural planning and device sizing, especially for percutaneous closure
Cardiac CT	High-resolution anatomical imaging; planimetry of regurgitant orifice	Excellent for anatomical detail, defect morphology, and pre-procedural planning	Lacks flow quantification; metal artifacts; may miss small leaks	Adjunct when echo is inconclusive or for complex anatomy; device sizing and procedural guidance
CMR (Cardiac MRI)	Phase-contrast imaging; quantifies regurgitant volume/fraction	Accurate quantification of regurgitant volume; unaffected by acoustic shadowing	Limited availability; prosthetic artifacts; less spatial detail	Useful when echo is inconclusive or discordant; reclassifies severity; guides management
Cardiac Catheterization	Hemodynamic assessment; angiography	Direct measurement of pressures; visualizes regurgitation	Invasive; rarely needed; limited anatomical detail	Reserved for cases with inconclusive noninvasive imaging or for hemodynamic assessment

TAVR: Transcutaneous Aortic Valve Replacement, PVL: Para-Valvular Leak

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#FR 1 – Kiosk 21 – Poster 3

A Case of Recurrent Refractory Carcinoid Crisis Requiring ECMO

Presenting Author: Azhar Dalal, MD, Indiana University School of Medicine

Introduction

Carcinoid crisis is a life-threatening complication of metastatic neuroendocrine tumors, caused by abrupt mediator release leading to bronchospasm, vasoplegia, and cardiovascular collapse. Even with recommended prophylaxis, crises remain difficult to predict or prevent. We present a case in which crisis prevention was ineffective, and two attempts at tricuspid valve surgery were aborted due to refractory carcinoid crisis requiring ECMO support.

Case Presentation

A 66-year-old female with metastatic carcinoid tumor on monthly octreotide presented with volume overload and symptomatic right-sided heart failure. Baseline symptoms were mild, limited to intermittent diarrhea, without flushing or prior bronchospasm. TTE showed EF 57%, moderate RV dilation, severe TR, and pulmonary hypertension. After diuresis, she was scheduled for tricuspid valve replacement.

Attempt #1:

A prophylactic 250 mcg octreotide bolus was given before induction. Immediately afterward, she developed severe bronchospasm and profound hypotension progressing to PEA arrest. ROSC was achieved with epinephrine (100 mcg boluses followed by two 1 mg doses) and 100 mcg octreotide. She required epinephrine, norepinephrine, and vasopressin infusions, as well as albuterol and 100 mg hydrocortisone. Blood pressure remained highly labile despite these measures. After a 500 mcg octreotide bolus and initiation of a 200 mcg/h infusion, her hemodynamics gradually stabilized, allowing vasopressor weaning.

Attempt #2 (10 days later):

Surgery was reattempted after 24 h of IV octreotide at 500 mcg/h plus prophylactic cyproheptadine, hydrocortisone, ondansetron, diphenhydramine, and famotidine. Immediately after induction and intubation, she again developed severe hypercapnic, hypoxic respiratory failure, followed by PEA arrest. ROSC was achieved after 1 mg epinephrine, but profound hypotension and bronchospasm persisted despite repeated octreotide boluses. VV-ECMO was initiated; worsening cardiogenic shock in the ICU required conversion to VV-A ECMO. She was eventually decannulated and weaned off vasopressors but was no longer a surgical candidate and was transitioned to hospice.

Discussion

Despite minimal baseline symptoms, this patient experienced two severe, refractory carcinoid crises during separate surgical attempts, even with maximal optimization and recommended prophylaxis. Vasopressors offered limited benefit, and octreotide boluses did not reliably reverse cardiovascular collapse. This case underscores the unpredictable nature of carcinoid crisis, the limited reliability of prophylaxis, and the

importance of early preparation for rapid deterioration, including immediate access to continuous octreotide, stress-dose steroids, aggressive bronchodilator therapy, and mechanical support such as ECMO. Induction and airway manipulation appear to be particularly high-risk periods; minimizing triggers, ensuring adequate anesthetic depth, and maintaining stable ventilation may reduce risk but cannot fully prevent crisis.

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#FR 1 – Kiosk 21 – Poster 4

Robotic TECAB and Osteogenesis Imperfecta; Unexpected Anesthetic Challenges

Presenting Author: Max Kohen, MD, Fellow, Yale New Haven Hospital

Introduction

Totally endoscopic coronary artery bypass (TECAB) offers a sternal-sparing alternative for patients at high risk of sternotomy complications. However, one-lung ventilation, limited access, and reliance on endovascular or percutaneous myocardial protection create major anesthetic challenges—especially when unexpected valvular disease is present. Abrupt loss of lung compliance during robotic cardiac surgery warrants concern for unilateral pulmonary edema (UPE), a rare complication linked to lung collapse, reperfusion injury, and inflammatory capillary leak. Rapid evaluation and readiness for extracorporeal support are critical.

Case Presentation

A 61-year-old man with hypertension, asthma, osteogenesis imperfecta with cervical fixation, prior PCI, and smoking history presented with stable angina. Angiography showed >90% LAD–diagonal stenosis, diffuse circumflex restenosis, and chronic RCA occlusion. Preoperative TTE demonstrated mild AS, mild–moderate AR, trace MR, and mildly reduced RV function. To reduce fracture and nonunion risk, robotic TECAB using a radial-artery Y-graft to the LIMA was planned. A right radial arterial line was placed pre-induction; intubation, TEE, and left bronchial blocker placement were performed with video and fiberoptic assistance to protect the cervical spine. A femoral arterial line was added for aortic-territory monitoring. Intraoperative TEE unexpectedly revealed moderate AS, severe AR, and moderate–severe MR; percutaneous retrograde cardioplegia was selected. Two right IJ sheaths provided venous access and coronary-sinus cannulation. Severe calcification and abnormal heart orientation limited TEE windows and complicated coronary-sinus catheter placement. Radial harvest difficulty and intrathoracic anatomy led to grafting of the diagonal and LAD. Prior to CPB weaning, lung compliance abruptly collapsed with tidal volumes < 200 mL. Bronchoscopy showed no obstruction, and TEE confirmed preserved function without effusion or pulmonary venous blockage. Recruitment and bronchodilation were ineffective, raising concern for UPE or reperfusion injury. VV-ECMO was prepared but avoided when ventilation improved after switching to an ICU ventilator. The patient was transferred to the ICU and recovered.

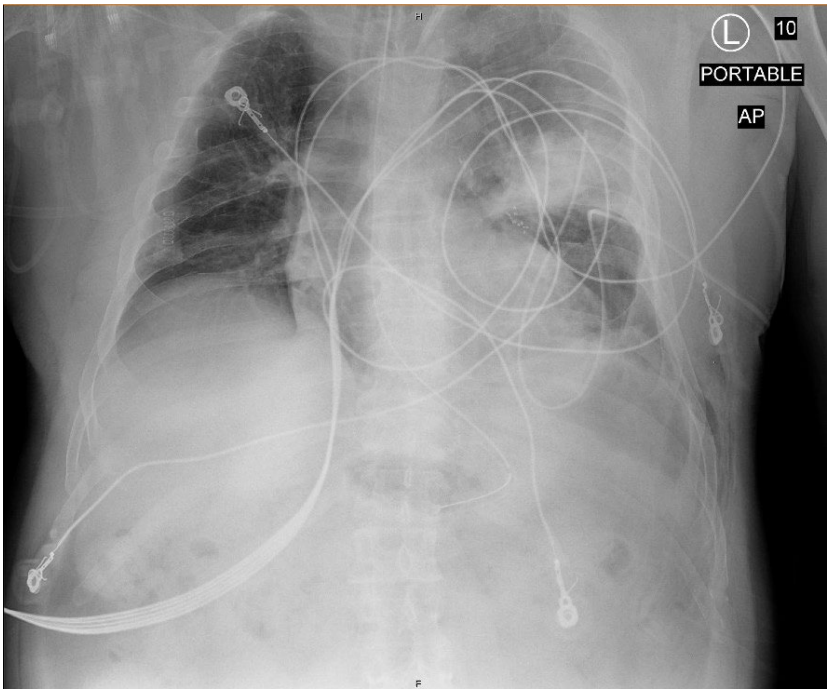
Discussion

Key challenges in this TECAB case included:

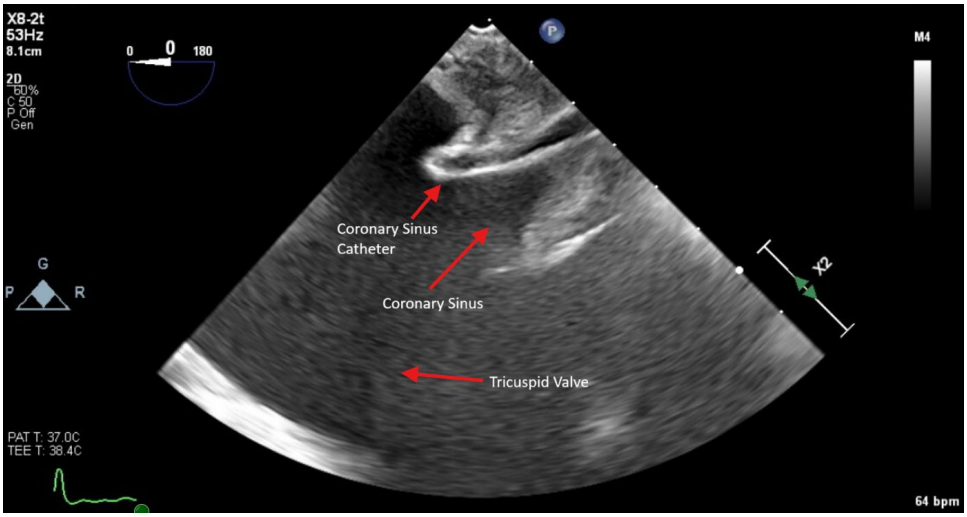
- Myocardial protection with severe AR: Antegrade delivery risks LV distention; retrograde cardioplegia was technically difficult, and endovascular balloon occlusion would worsen regurgitation.
- Robotic constraints: One-lung ventilation, CO_2 insufflation, and limited access increased hemodynamic stress, impaired oxygenation, and reduced rescue options, raising UPE risk.
- Osteogenesis imperfecta: Fragile bones, restrictive mechanics, and cervical fixation contributed to a difficult airway and decreased pulmonary reserve.

- Post-CPB respiratory failure: Sudden compliance loss with preserved cardiac function suggested noncardiogenic lung injury. Rapid exclusion of mechanical causes and ECMO readiness were essential; improvement after ventilator change highlights the need for adaptable strategies in minimally invasive cardiac surgery.

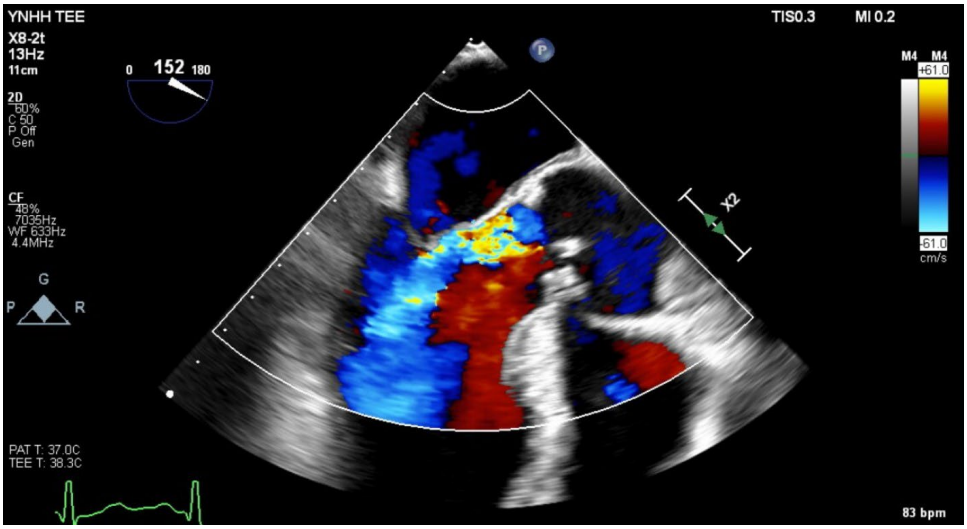
Images



Post Op Day 0 AP CXR



Placement of Coronary Sinus Catheter



Representative still frame color doppler evaluation of Aortic Regurgitation

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#FR 1 – Kiosk 21 – Poster 5

Combined Heart-Lung Transplantation for Interstitial Lung Disease with Acute Heart Failure

Presenting Author: Madelinn Rice, MD, Vanderbilt University Medical Center

Introduction

Combined heart-lung transplant became a therapeutic option for patients with cardiopulmonary disease since the first successful surgery in 1981. These patients present unique management challenges of both end stage pulmonary and cardiac disease throughout their operative course.

Case Presentation

A 52-year-old male with interstitial lung disease was transferred from an outside hospital for expedited lung transplant evaluation secondary to disease progression. Echocardiogram on admission demonstrated a newly reduced left ventricle systolic function, with heart catheterization significant for severe pulmonary hypertension. He clinically deteriorated with worsening left and right sided systolic function, requiring VA ECMO support. Turndown studies in the ICU demonstrated LV recovery, suggesting stress-induced cardiomyopathy. An ECMO clamping trial demonstrated a cardiac index of 2-3, as well as significant increases in CVP and PCWP. In the setting of moderate RV failure with tricuspid regurgitation, it was determined he would not recover from his stress-induced cardiomyopathy, and he was listed via exception for combined transplant. Four days later, he was transplanted. His intra-operative course was complicated by hemorrhage, vasoplegia and primary graft dysfunction which required continued VA ECMO support post-operatively. He also developed renal failure, hypoxic respiratory failure, and donor-associated infections. Despite a challenging hospital course, he was discharged to rehab without any supportive devices on post-op day 43.

Discussion

Combined heart-lung transplant is a curative treatment for patients with end-stage pulmonary and cardiac disease. Improvements in lung transplant outcomes have reduced the population requiring combined organ transplant to patients with end stage lung disease and concomitant left ventricular failure and/or irreversible right ventricular failure. As the congenital heart disease population ages into adulthood, the need for combined heart-lung transplants is anticipated to increase. Co-existing end stage pulmonary and cardiac disease create a unique and challenging operative course. Optimizing patients pre-operatively frequently requires mechanical circulatory support. Careful, controlled induction of anesthesia is necessary in the setting of severe pulmonary hypertension. Intra-operatively, the complete dissection of the middle mediastinum exposes patients to increased hemorrhage risk. Congenital heart disease patients are known to develop aortopulmonary collaterals, further complicating appropriate hemostasis during dissection. Most centers choose to re-perfuse for a prolonged period, which leads to longer cardiopulmonary bypass time. The dissection of the heart and lungs increases the risk of phrenic nerve, vagus nerve, and thoracic duct injury. Post-operatively, these manifest as prolonged mechanical ventilation support, high aspiration and gastroparesis risk, and chylothorax. Primary graft dysfunction is also more common than single organ

transplantation. While lung dysfunction is more common, cardiac dysfunction is a leading cause of mortality. Combined heart-lung transplants provide a critical therapy for a high-risk patient population with limited therapeutic options.

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#FR 1 – Kiosk 21 – Poster 6

Cardiovascular Collapse: Dynamic Obstruction from a Large Left Atrial Myxoma

Presenting Author: Whitney Wolaver, MD, Cardiothoracic Anesthesiology Fellow, Medical University of South Carolina

Introduction

Primary cardiac malignancies represent a rare subset of cancers, with an incidence ranging from 0.001% to 0.03%. Metastatic lesions from other malignancies represent the most common cause of cardiac tumors. Lung cancer, breast cancer, and hematologic malignancies have the highest rates of cardiac metastasis[1]. Cardiac myxomas make up approximately 50% of primary, benign cardiac tumors with a predilection for the left atrium and attachment near the fossa ovalis. Clinical manifestations of heart failure symptoms, arrythmias, and stroke result from the obstructive and embolic nature of these intracardiac tumors necessitating urgent surgical intervention[2]. Interference of the mitral valve by left atrial myxomas can produce a "ball-valve" physiology with left ventricle inflow obstruction. This poses a dynamic anesthetic challenge to avoid acute hemodynamic deterioration.

Case Presentation

A 50-year-old male with no significant cardiopulmonary history presented as transfer from an outside hospital with shortness of breath. Advanced cardiac workup demonstrated a large, pedunculated mass measuring 7.1 x 3.5cm with attachment to the interatrial septum near the fossa ovalis, consistent with a myxoma. Pre-operative TEE findings included a normal LV EF of 60-65%, severely enlarged left atrium with mild-moderate MR, and protrusion of the mass through the MV during diastole. After surgical and anesthetic planning, the patient was brought to the operating room for median sternotomy and mass excision under cardiopulmonary bypass. After arterial line placement and pre-oxygenation, general anesthesia was induced with 4mcg/kg fentanyl, 1mg/kg lidocaine, and 1mg/kg propofol. The patient experienced a precipitous drop in mean arterial pressures to 30mmHg requiring a crystalloid bolus, norepinephrine bolus, and a high-dose norepinephrine infusion to establish hemodynamic recovery. During this time, the cardiac surgery team was notified to expedite surgical preparation and sternotomy. The case proceeded uneventfully, and the patient was discharged on post-operative day three.

Discussion

"Ball-valve" physiology from an intracardiac tumor can be dynamic in the setting of hemodynamic changes from induction of general anesthesia. Pre-operative echocardiography demonstrated mild-moderate mitral regurgitation secondary to prevention of appropriate leaflet coaptation. Intra-operatively the prolapsed myxoma exhibited stenosis physiology, likely exacerbated by the influence of general anesthesia. Increased forward flow from the LV in the setting of decreased afterload may have contributed to further prolapse of the myxoma and the resulting tachycardia prevented appropriate LV filling. After aggressive preload and afterload augmentation, the patient's hemodynamics improved. The hemodynamic perturbations demonstrated by this case illustrate the importance of understanding the dynamic nature of "ball-valve"

physiology from mobile intracardiac tumors.

Images



Large left atrial myxoma (7cm x 4.5cm x 3cm)



Large left atrial myxoma (7cm x 4.5cm x 3cm)

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#FR 1 – Kiosk 22 – Poster 1

A Case of Mistaken Identity: Endocarditis or Myxomatous Degeneration

Presenting Author: Nicholas Rothbard, DO, Cardiothoracic Anesthesiology Fellow, University of Minnesota

Introduction

The prevalence of infective endocarditis (IE) is increasing in the United States and remains a significant source of morbidity and mortality, with use of intravenous drugs and the presence of cardiovascular device implants as important risk factors[1]. We present a case of a patient with extensive cardiovascular device implants with suspected native mitral valve endocarditis and show the importance of thorough transesophageal echocardiography (TEE) evaluations in affecting surgical decision-making.

Case Presentation

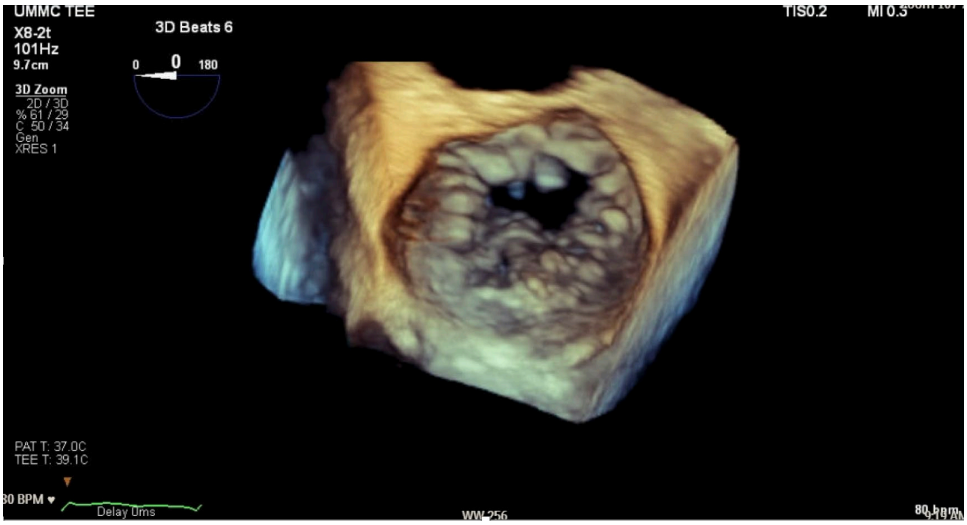
A 78-year-old male status post bioprosthetic aortic valve replacement, ascending aorta and total arch replacement in frozen elephant trunk configuration, left atrial appendage excision 1 year prior at our institution, presented with suspected IE of the native mitral valve. His comorbid conditions included heart failure with reduced ejection fraction (40-45%) and sinus bradycardia with permanent pacemaker. He presented with sepsis progressing to septic shock and was found to have MSSA bacteremia; TEE showed possible vegetations on the native mitral valve with bileaflet billowing but without mitral regurgitation. He was started on Nafcillin and briefly required vasopressor support. The operative plan was repeat sternotomy, repeat aortic root and valve replacement, mitral valve replacement, reconstruction of the aorto-mitral curtain and explant of permanent pacemaker leads. By the day of surgery, blood cultures had been negative for several days. Our initial TEE evaluation was suspicious for native mitral valve endocarditis and was consistent with admission TEE. However, a more comprehensive inspection of the valve in multiple planes and in 3D showed that what was thought to be vegetation was extensive and heterogeneous thickening of anterior and posterior leaflets. Prior echocardiographic images were reviewed; these also showed severe thickening of both mitral valve leaflets. Surgery was canceled as these findings were felt to be more consistent with Barlow's disease. The patient continued IV antibiotics and a repeat TEE a few days later was unchanged. The patient has had no recrudescence of bacteremia several months later.

Discussion

Equivocal echocardiographic findings in a high-stakes situation is one of the most difficult situations cardiothoracic anesthesiologists face. The diagnosis hinged on 3 findings: 1) severe, diffuse—albeit very heterogenous—leaflet thickening, 2) the presence of leaflet billowing and 3) corroboration with prior imaging. Leaflet billowing is suggestive of Barlow's disease, as is the diffuse thickening of both leaflets. Beyond a clinical picture strongly suggestive of IE, there were two things that further complicated diagnosis: 1) there was no MR present, which is atypical for Barlow's disease and 2) a study of TEEs ordered for suspected IE found that masses described as large, multi-lobulated or protruding/pedunculated were more frequently associated with a final interpretation of IE[2]. In this case, a very thorough and careful TEE evaluation prevented an unnecessary and very high risk re-operation, despite the clinical scenario strongly

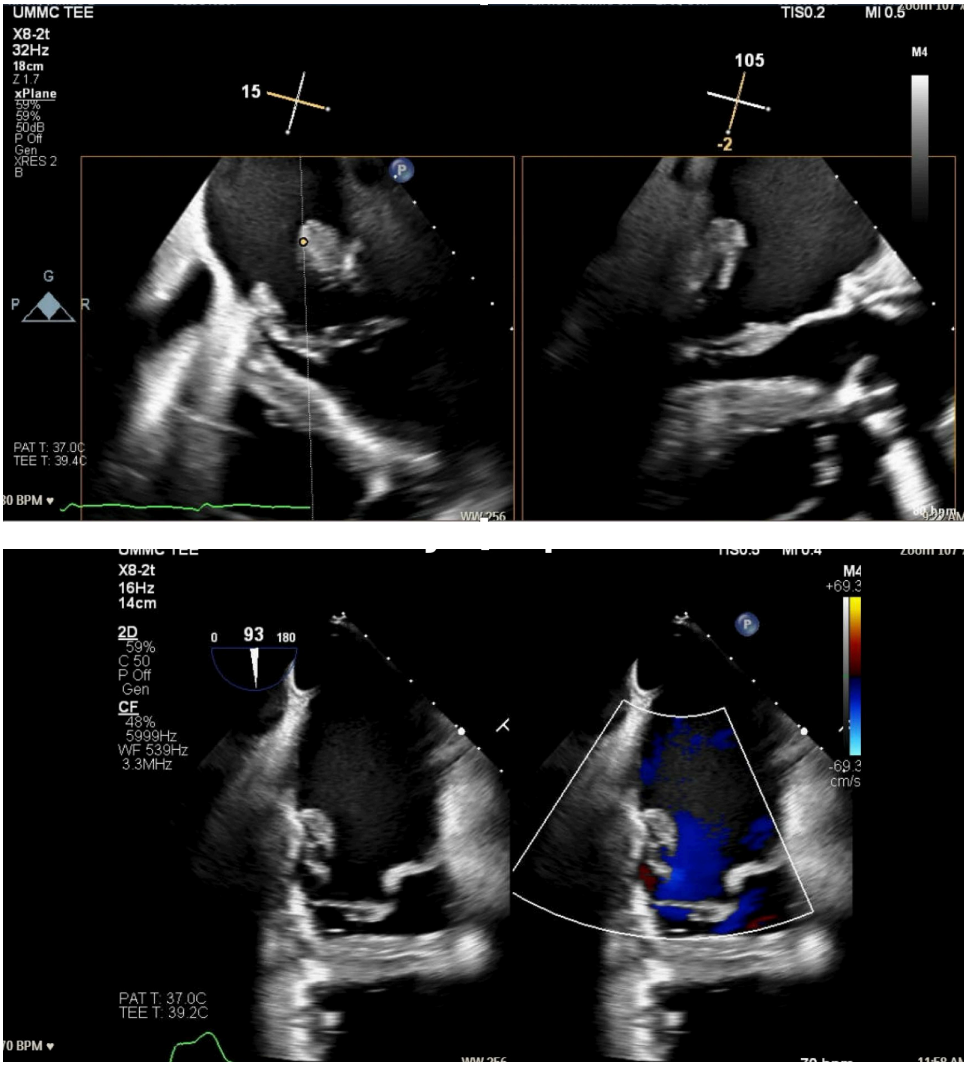
supporting a diagnosis of infective endocarditis.

Images



3D acquisition of mitral valve





TEE 2 years prior to case

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#FR 1 – Kiosk 22 – Poster 2

Iatrogenic Tension Pneumopericardium Leading to Cardiac Arrest in an 18-Year-Old Patient on Veno-Arterial Extra Corporeal Membrane Oxygenation

Presenting Author: Austin S. Anderson, Resident Physician, University of Arizona

Introduction

Tension pneumopericardium is a rare but life-threatening condition in which air accumulates under pressure within the pericardial sac, impairing diastolic filling and reducing cardiac output. Although described after trauma, mechanical ventilation, and cardiac procedures, progression to tension physiology is uncommon (Cummings et al., 2002). Diagnosis is especially challenging in VA-ECMO patients, as extracorporeal flow can mask hallmark signs of tamponade. We present a case of tension pneumopericardium precipitating cardiac arrest in a patient on central VA-ECMO.

Case Presentation

An 18-year-old woman with suspected Ehlers Danlos syndrome, mitral valve prolapse, and recent postpartum status presented after an out-of-hospital cardiac arrest. She was in severe cardiogenic shock with an LVEF of approximately 7% despite maximal inotropes. She underwent emergent central VA-ECMO cannulation via sternotomy and mediastinal chest tubes were placed. A moderate pericardial effusion was noted postoperatively, however, it remained stable on serial exams. To enhance drainage of the pericardial effusion, the surgical team performed forceful mediastinal chest tube stripping. Thirty minutes later, the patient developed ventricular fibrillation progressing to PEA arrest. CVP and PA pressures remained stable—an effect of ECMO support, which can obscure tamponade physiology. Bedside echocardiography demonstrated new intrapericardial air, and chest radiography confirmed pneumopericardium with a radiolucent halo confined to the pericardial sac. Emergent needle pericardiocentesis was performed with release of pressurized air. The patient subsequently developed torsades de pointes requiring magnesium and defibrillation before returning to sinus rhythm. The timing and imaging strongly implicated iatrogenic tension pneumopericardium from aggressive chest tube stripping to be the etiology of the cardiac arrest.

Discussion

Tension pneumopericardium develops when air enters the pericardial space and cannot escape, generating pressure that restricts cardiac filling and output. Immediate decompression is essential to prevent cardiovascular collapse. In VA-ECMO patients, diagnosis can be difficult as extracorporeal support can blunt hypotension, stabilize filling pressures, and may preserve apparent hemodynamic stability. Thus, tamponade may present only with arrhythmia, loss of pulsatility, or ECMO flow disturbances (Adriaansen et al., 2023). Chest tube stripping is controversial, as it produces high negative intrathoracic pressure capable of causing tissue trauma, hemopericardium, or air entrainment through small defects—risks are amplified in patients with mediastinal drains and pre-existing effusions. Evidence shows no clear benefit to routine stripping, while potential harms are increasingly recognized (Day et al., 2006). This case emphasizes the need to (1) maintain suspicion for pericardial air in ECMO patients with sudden decompensation despite

"normal" hemodynamics; (2) avoid aggressive chest tube manipulation when the pericardium is exposed or effusions are present; (3) use bedside echocardiography liberally, as air artifact may be the earliest clue; and (4) treat suspected tension pneumopericardium as a time-critical emergency requiring immediate decompression.

Images

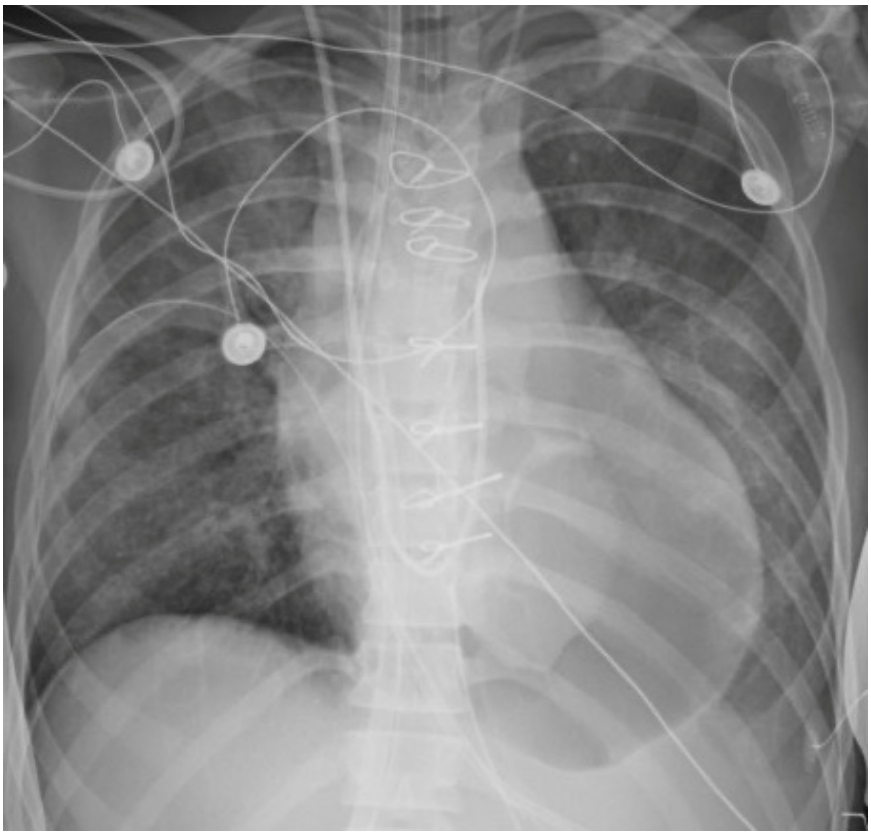


Figure 1. Chest radiograph demonstrating pneumopericardium.

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#FR 1 – Kiosk 22 – Poster 3

Intraoperative Placement of a Right Axillary Impella 5.5 through a Konect Graft for Cardiogenic Shock: A Case Report

Presenting Author: Michael LoBasso, MD, Resident Physician, UCLA Health

Introduction

Cardiogenic shock after cardiothoracic surgery affects 0.2% to 6% of patients and carries an estimated mortality rate of 50% to 80% [1,2]. Axillary insertion of the Impella 5.0 or 5.5 has shown value in patients with left ventricular dysfunction undergoing cardiac surgery [1,3]. Microaxial flow pumps such as the Impella 5.5 (Abiomed) unload the left ventricle, decrease myocardial work, and provide circulatory support to promote recovery and assess residual function [2,3]. Although widely used in the management of cardiogenic shock after cardiac surgery, evidence for Impella use in aortic surgery remains limited. This case report describes the intraoperative placement of an Impella 5.5 through the right axillary artery following a Bentall procedure with a newly placed 23mm aortic valved conduit (Konect graft).

Case Presentation

The patient is a 67-year-old female with severe chronic obstructive pulmonary disease, hypertension, obstructive sleep apnea, fibromyalgia, and chronic opioid use. She had been recently hospitalized for acute hypoxic respiratory failure from a respiratory infection, during which she was incidentally found to have a thoracoabdominal aneurysm with an ascending aorta measuring 6.8 by 6.5 cm, heart failure with an ejection fraction of 40 to 45%, and mild to moderate aortic valve regurgitation. After respiratory optimization, she agreed to proceed with aortic surgery. She underwent a Bentall procedure with placement of a 23 mm Konect graft and hemiarch repair under deep hypothermic circulatory arrest with antegrade cerebral perfusion. Induction of general anesthesia was uneventful. A radial arterial line was placed pre-induction, and a central venous and pulmonary artery catheter were inserted post-induction. Given her low EF and prolonged cardiopulmonary bypass (CPB) time of 195 minutes, separation from CPB proved to be difficult. Transesophageal echocardiography showed inferior and inferoseptal left ventricular hypokinesis with normal right ventricular function. She required escalating inotropic support and experienced recurrent episodes of sudden, profound hypotension with left ventricular dilation concerning for postcardiotomy left ventricular dysfunction. Given her deteriorating hemodynamics, the teams decided to place an Impella 5.5 via the right axillary artery, advancing it across the newly implanted 23 mm Konect graft. This was done under TEE guidance, prior to chest closure. The chest was subsequently closed and she was transported to the cardiothoracic ICU intubated. On POD1 she was extubated. Over the next several days her left ventricular function improved, and the Impella 5.5 was removed in the OR under general anesthesia on POD5.

Discussion

This case highlights the successful use of an Impella 5.5 via the right axillary artery to unload the left ventricle in a patient immediately following a Bentall and hemiarch repair with a newly implanted Konect

graft. This approach allowed for circulatory support of the left ventricle while maintaining transpulmonary flow and allowing for myocardial recovery, and ultimately safe device explantation. Axillary placement of an Impella 5.5 may be a viable option for managing postcardiotomy left ventricular dysfunction in patients undergoing aortic repairs.

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#FR 1 – Kiosk 22 – Poster 4

The Rarest Mediastinal Mass Makes a Splash: Perioperative Management of Saccular Superior Vena Cava Aneurysm Complicated by Pulmonary Embolism and Acute Rupture

Presenting Author: Richard Zack-Guasp, MD, CT Anesthesia Fellow, University of Maryland Medical Center

Introduction

SVC aneurysms are extremely rare with only around 40 cases reported, of which the majority are fusiform subtype. This is the most stable subtype, typically managed conservatively or surgically repaired based on size and growth rate. Saccular aneurysms are considered urgent/emergent in nature given the high risk of perforation secondary to vascular wall thinning and weakening. We present the case of a massive SVC saccular aneurysm associated with acute pulmonary emboli. The perioperative care of our patient was complicated by peripheral vascular injury during emergent arterial line/ECMO cannulation and aneurysm rupture during invasive diagnostic testing.

Case Presentation

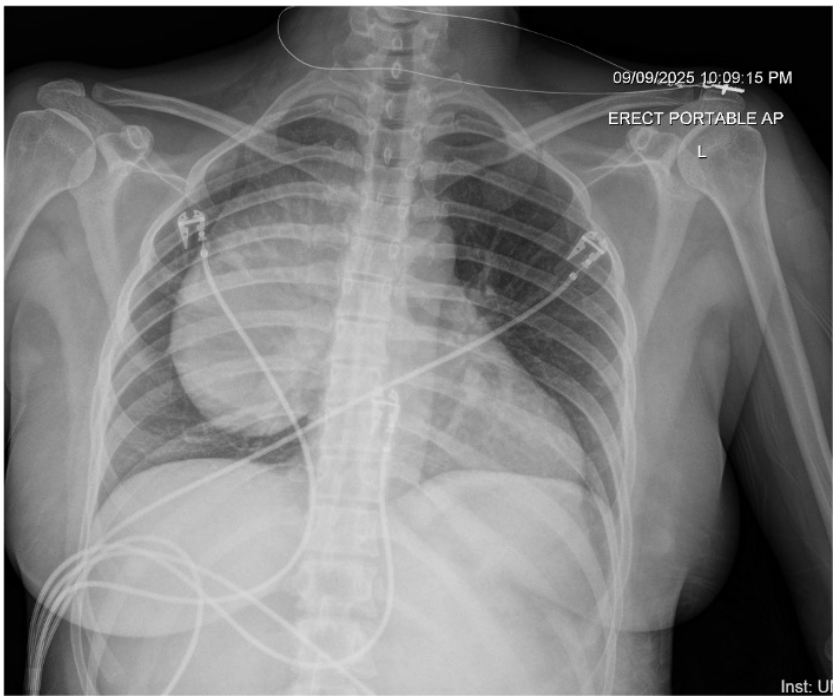
A 40-year-old female presented with abrupt onset of nausea, vomiting, diaphoresis, which led to unresponsiveness. In the ED, she was found to have severe lactic acidosis and elevated troponins. Chest imaging revealed a massive SVC aneurysm. While awaiting institutional transfer, she developed hemodynamic instability with an in-transit RA/RV thrombus identified on bedside TTE. Tenecteplase and vasopressor support temporarily stabilized her. Hemodynamic monitoring was complicated by challenging right femoral arterial access with suspected venous placement. Upon arrival to the CVICU, worsening acidosis and shock prompted emergent VA-ECMO initiation. After stabilization and surgical repair of an expanding femoral hematoma, she underwent planned right heart catheterization to further characterize the aneurysm and assess right sided pressures. Early in the procedure, she became acutely unstable, and fluoroscopy indicated rupture of the aneurysm. Cardiac Surgery was activated for emergent intervention. TEE revealed an underfilled LV and massive right pleural hemorrhage compressing the heart. VA-ECMO was converted to CPB, hemorrhage was controlled, the SVC was repaired with a graft, and bilateral pulmonary embolectomy was performed. Following correction of coagulopathy, she was successfully weaned from CPB. The patient recovered well and was discharged later that week with a plan for aggressive cardiac rehabilitation and medical management.

Discussion

SVC aneurysms are extremely rare and often discovered incidentally. They may be congenital, acquired, pseudoaneurysmal, or related to arteriovenous fistula. Congenital origin was unlikely given prior normal imaging, but patient did endorse sustaining non-displaced rib fractures in a MVA. Despite the large size, what likely caused the acute presentation was the presence of pulmonary emboli, which is a known complication in the setting of stasis within the aneurysmal sac. Important anesthetic considerations include prioritizing lower extremity IV access for effective resuscitation and systemic medication delivery in the event of aneurysmal rupture. Preemptive ECMO cannulation should be strongly considered. Hemodynamic

swings should be avoided, particularly during induction of anesthesia, and blood products be readily available. Despite their rare nature, SVC aneurysms should not be forgotten in our differential list of chest masses, especially since early diagnosis is imperative for the decision-making process of whether emergent surgical intervention is warranted and for tailored patient perioperative management.

Images



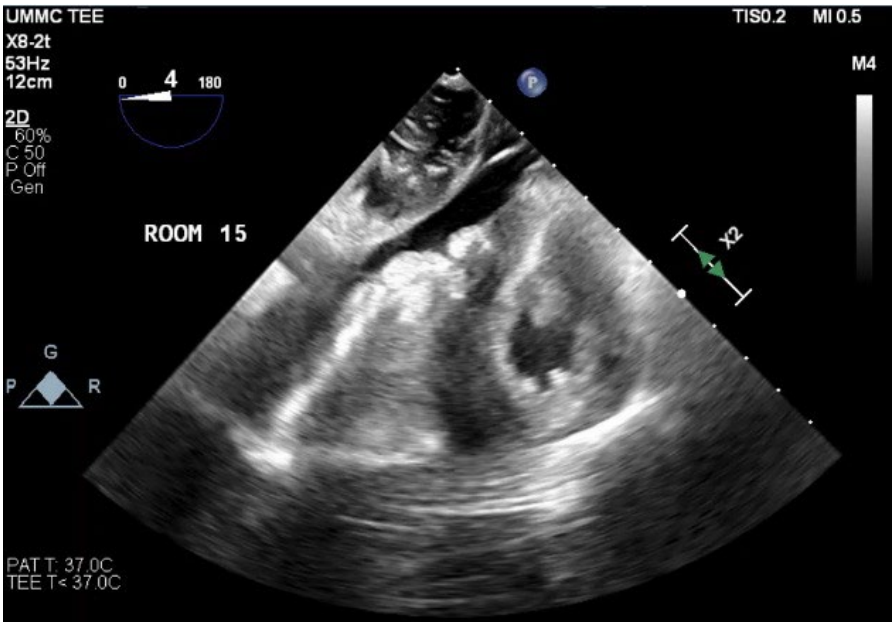
ED Admission CXR.



CTA (PE Protocol) done after CXR to characterize right hemithorax mass



CTA (PE Protocol) showing SVC aneurysm with associated thrombi



TEE transgastric view of undefilled LV and completely collapse RV in setting of tension right hemothorax



Postop CT demonstrating SVC graft and reexpansion of right lung

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#FR 1 – Kiosk 22 – Poster 5

The Rastelli Procedure: Anesthetic Challenges in a Complex Congenital Reoperation

Presenting Author: Bobby T. Houston, M.D., Fellow, Cleveland Clinic Foundation

Introduction

Dextro-Transposition of the Great Arteries (D-TGA) is a congenital defect, often coexisting with ventricular septal defect (VSD) and pulmonary atresia.[1] The Rastelli Procedure provides surgical correction with VSD closure and an extracardiac right ventricle (RV)-to-pulmonary artery (PA) conduit. Survivors often develop conduit degeneration, left ventricular outflow tract (LVOT) and right ventricular outflow tract (RVOT) obstruction, and valvular dysfunction requiring reoperation.[2] We describe the anesthetic challenges during a redo Rastelli procedure

Case Presentation

A 36-year-old male with a history of D-TGA, VSD, and pulmonic valve (PV) atresia was treated with a Blalock-Taussig shunt (BTTS), followed by a Rastelli procedure. He developed RV-PA conduit regurgitation, residual VSD, subaortic stenosis, and mild aortic insufficiency. He received a percutaneous PV replacement, which later resulted in pulmonic stenosis (PS) and regurgitation. He presented for a redo Rastelli procedure, PV replacement, aortic valve (AV) replacement, and ascending aorta replacement. Heart catheterization revealed moderate PS (pressure gradient 57 mmHg) and LVOT gradient of 27 mmHg. Echocardiography showed preserved LV function and mildly reduced RV function. A CT scan revealed major cardiovascular structures located 1 mm behind the sternum. Pre-induction brachial arterial line and PA catheter were placed for hemodynamic monitoring. Anesthesia was induced uneventfully with midazolam, fentanyl, etomidate, and succinylcholine. The left groin was prepared for peripheral cannulation. Intraoperative TEE confirmed a residual VSD, severe subaortic stenosis due to the infundibulum between the native PV and AV, and calcification of the VSD patch. (Fig 1, Fig 2) The RV-to-PA conduit was identified in modified TEE views. (Fig 3) The prosthetic PV was calcified, with moderate regurgitation and moderate stenosis. (Fig 4) During weaning of cardiopulmonary bypass, complete heart block was noted, requiring pacing. There was significant coagulopathy requiring multiple blood products. The postoperative course was uncomplicated and he was extubated on postoperative day 1.

Discussion

Reoperations in adult congenital patients are risky given prior sternotomies and mediastinal adhesions, risking exsanguination upon sternal re-entry. This necessitates alternative cannulation strategies for rapid initiation of CPB, e.g. femoral cannulation. Repaired D-TGA has residual anatomic distortion, affecting the choice of invasive monitoring sites.[3] D-TGA patients often undergo a classic vs. modified BTTS. This patient's right subclavian artery was ligated for a classic BTTS, so the decision was made to place the arterial line on the left arm. This patient had a fixed stenotic lesion from PS. Placement of an awake PA catheter should be considered in patients with right-sided stenotic lesions to monitor RV and PA pressures during induction. Conduction disturbances are common during VSD patch work. Temporary pacing should be anticipated.

Multiple imaging modalities must be thoroughly reviewed in congenital patients to identify unusual anatomic connections and translate these to intraoperative TEE findings. (Fig 5) Non-standard TEE views are often utilized to image congenital cardiac anatomy.

Images

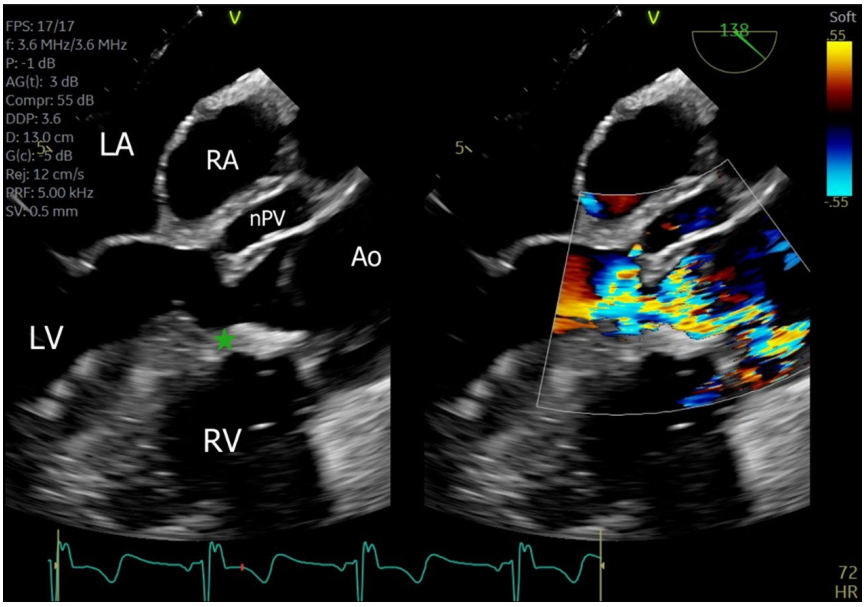


Figure 1. Midesophageal long axis view with color compare demonstrating a narrowed LVOT due to encroachment from the native pulmonic valve infundibulum, and flow turbulence which begins at the location of stenosis. The green star denotes the calcified VSD patch. LA = left atrium, LV = left ventricle, RA = right atrium, RV = right ventricle, nPV = native pulmonic valve, Ao = aorta.

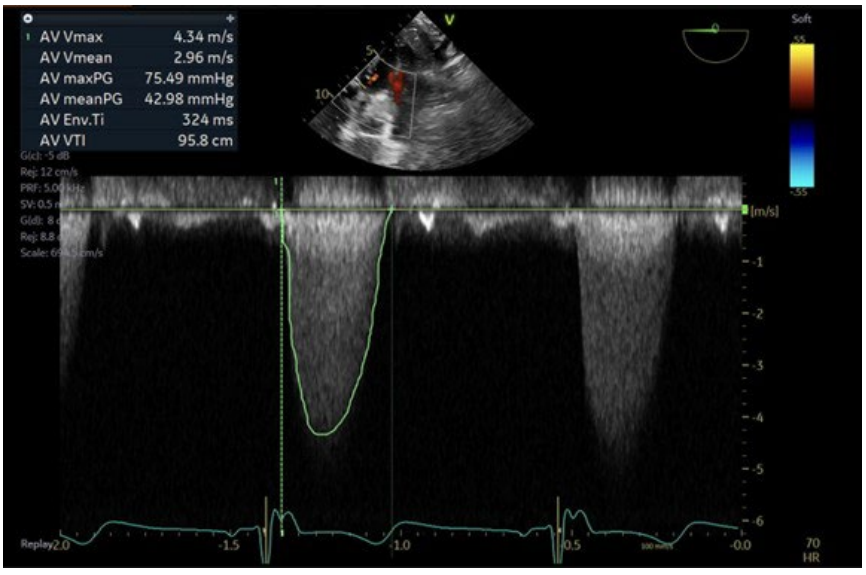


Figure 2. Deep transgastric long axis view with continuous wave Doppler through the AV/LVOT. Peak/mean gradients were 75/42 mmHg, consistent with severe subaortic stenosis.

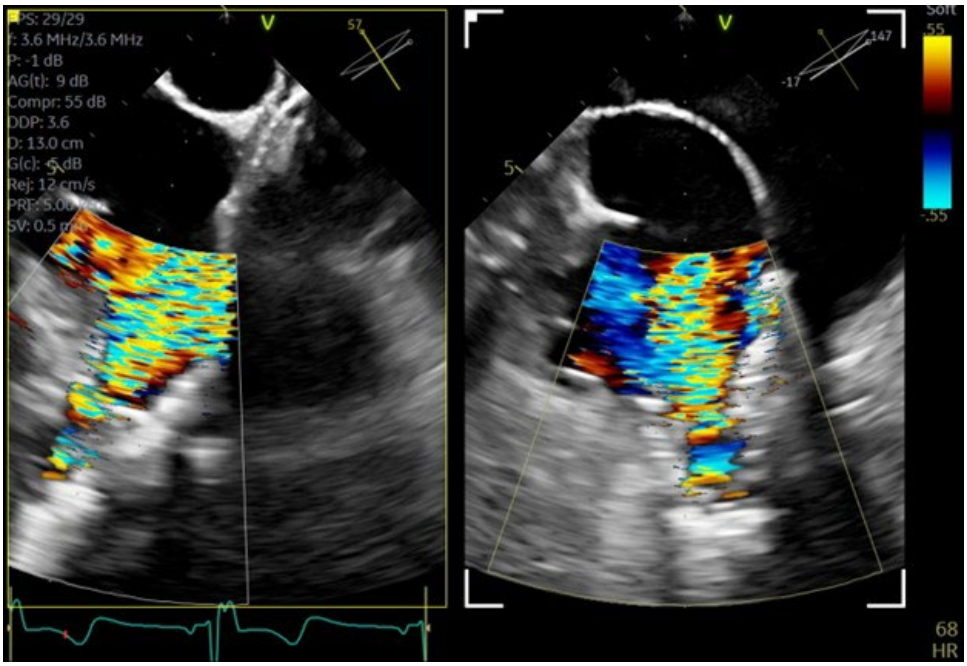


Figure 3. Modified upper esophageal aortic arch short axis view with biplane imaging (equivalent to modified upper esophageal aortic arch long axis view) and color Doppler over the pulmonic prosthesis. Non-standard angles and biplane are used to obtain optimal views of the RV-PA conduit and pulmonic prosthesis.

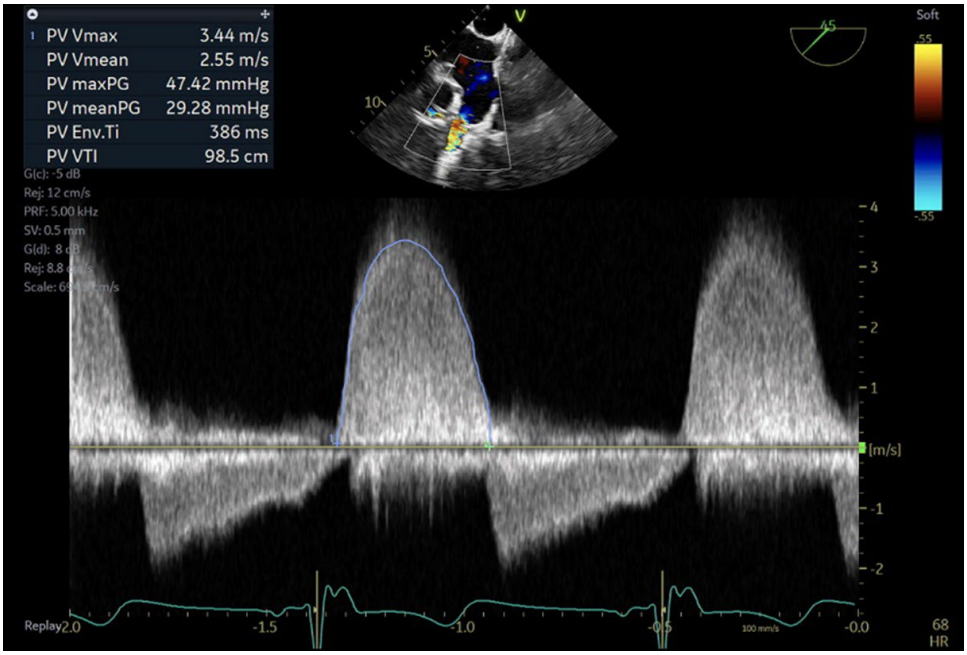


Figure 4. Modified upper esophageal aortic arch short axis view with continuous wave Doppler through the pulmonic prosthesis. Peak/mean gradients across the bioprosthesis (47/29 mmHg) suggest moderate stenosis. Spectral Doppler waveform of pulmonic insufficiency is dense, with a pressure half-time of 150 ms, which is consistent with moderate pulmonic insufficiency.

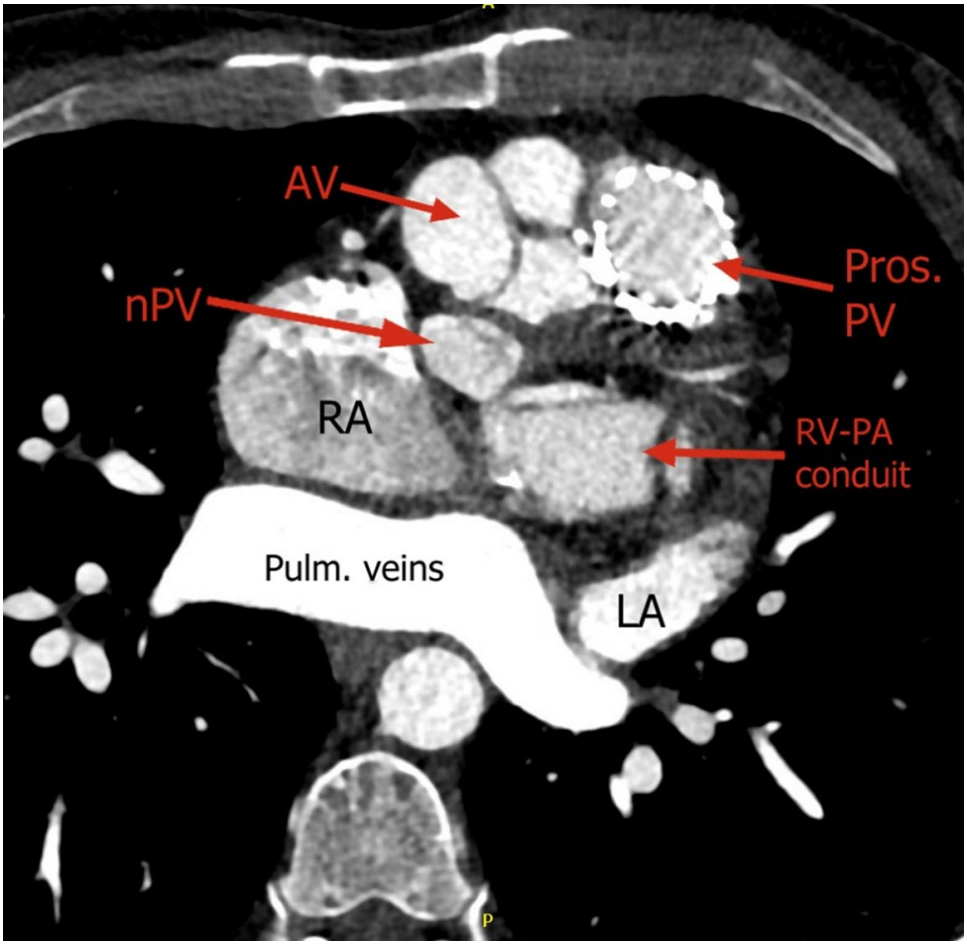


Figure 5. Computed tomography scan of the chest with contrast demonstrating cardiac anatomy of D-TGA and surgical correction with Rastelli procedure. The native oversewn pulmonic valve is seen posterior to the aortic valve. Parts of the RV-PA conduit can be seen and a prosthetic pulmonic valve can be seen in the conduit, to the left of the aortic valve. AV = aortic valve, nPV = native pulmonic valve, pros. PV = prosthetic pulmonic valve, RA = right atrium, LA = left atrium, pulm. veins = pulmonary veins

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#FR 1 – Kiosk 22 – Poster 6

A Puzzling Path to Lung Transplantation: Immunologic and Coagulation Conundrums

Presenting Author: Christine Shen, MD, Anesthesiology Resident, Loyola University Medical Center

Introduction

We present a lung transplant patient with two conundrums: allosensitization and a prothrombotic state. Allosensitization is associated with worse outcomes. Venous thromboembolism (VTE) is a major complication after lung transplantation. Thus, this patient presented with additional risks for lung transplantation and cardiopulmonary bypass.

Case Presentation

A 61-year-old, 61 kg woman with a history of mixed connective tissue disease, interstitial lung disease, carcinoid tumor s/p right VATS, pulmonary artery hypertension with right ventricular dysfunction, iron deficiency anemia s/p IV iron infusion, multiple pulmonary embolisms due to protein C deficiency, and allosensitization presented with acute respiratory failure. After a multidisciplinary discussion that included hematology and transfusion medicine, she was listed for bilateral lung transplantation. She transitioned from warfarin to heparin infusion while awaiting transplantation. Plasmapheresis with fresh frozen plasma (FFP) was started as part of her desensitization protocol, and she underwent bilateral lung transplantation with cardiopulmonary bypass. Tranexamic acid was given through the whole case. Heparin was dosed at 400 U/kg, and protamine at 0.5 mg/100 U heparin. Intraoperatively, she received three units of FFP and three units of pRBCs. Pt resumed intravenous heparin on postoperative day three and monthly IV immunoglobulin (IVIG). Her hospital course was complicated by VV-ECMO and a suboptimal response to plasmapheresis. Despite finishing the desensitization protocol, her anti-HLA titers remained elevated. Her hospital course spanned over five months with eventual expiration.

Discussion

Pre-transplant allosensitization was thought to be associated with worse outcomes after lung

transplantation as it increases the risk for antibody mediated rejection, chronic allograft dysfunction, and higher mortality. It has become less of a barrier with improved antibody detection, the use of virtual crossmatching, and desensitization protocols. Desensitization protocols often include plasmapheresis and IV immunoglobulin to reduce the antibody burden. Albumin or FFP can be used for plasmapheresis. Using albumin can be associated with increased risk of bleeding due to absence of clotting factors. FFP provides clotting factors but is associated with transfusion reactions and viral transmission. Protein C is a vitamin K-dependent protease that regulates thrombin in the coagulation cascade. Thus, protein C deficiency can lead to increased thrombotic risk. Anticoagulation, including warfarin and heparin, can interfere with functional assays used to quantify protein C activity. There are no recommended guidelines for protein C replacement or anticoagulation in the setting of cardiopulmonary bypass. Case reports describe the use of standard and higher doses of heparin with usual ACT monitoring and protamine reversal. Antifibrinolytic usage ranged from none to half to normal. Protein C deficiency is treated with protein C replacement from protein C concentrate, FFP, or prothrombin complex concentrate (PCC). Additional protein C was not administered perioperatively for this patient because she had been receiving FFP with plasmapheresis and intraoperative transfusion of RBCs and FFP was expected due to her small size and anemia.

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#FR 1 – Kiosk 22 – Poster 7

Heyde Syndrome in a Patient Presenting for Transcatheter Aortic Valve Replacement

Presenting Author: Alexander Theodotou, MD, Resident Physician, MedStar Georgetown University Hospital

Introduction

Heyde syndrome is a form of acquired von Willebrand syndrome (vWS) characterized by gastrointestinal bleeding (GIB) and severe aortic stenosis (AS) [1]. It results when high jet velocity across the stenotic valve causes damage to von Willebrand factor (vWF), leading to ADAMTS13-mediated destruction and impaired clot formation. Additionally, the decreased pulsatility of arterial blood flow in severe AS stimulates increased angiogenesis in the GI tract, promoting growth of arteriovenous malformations (AVMs). Though Heyde syndrome is uncommon, occurring in an estimated 1.8% of patients presenting for TAVR, the anesthesiologist caring for patients with AS is likely to encounter this syndrome in clinical practice [2].

Case Presentation

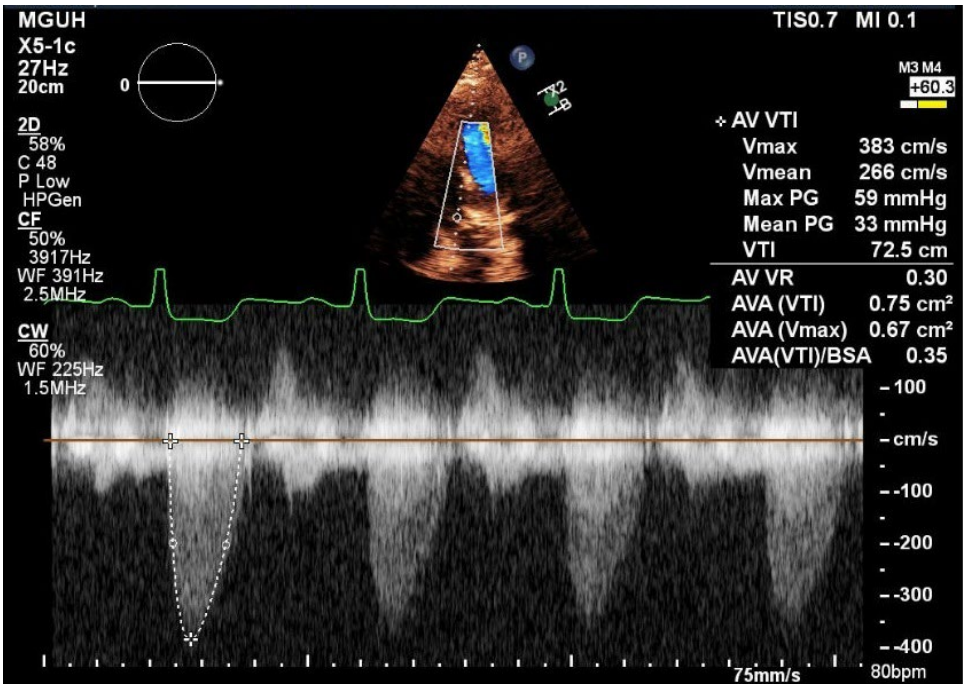
A 68-year-old male with a history of severe AS and lower GIB (previously treated with thalidomide) presented for TAVR. He had been admitted one month prior for severe anemia complicated by NSTEMI, with subsequent enteroscopy and argon plasma cautery of duodenal AVMs. He had also undergone enteroscopy with clipping of duodenal AVMs four years prior. TTE revealed AVA 0.66 cm2, MPG 34 mm Hg, transvalvular aortic velocity (V2) 3.8 m/sec, and DI of 0.29. Initial labs revealed Hb 5.9 g/dL, and TAVR was delayed for transfusion and iron dextran treatment. On the day of TAVR, standard monitors were established, with a femoral arterial line placed by the proceduralist. Sedation was achieved with fentanyl, propofol, and dexmedetomidine. Anticoagulation was achieved with heparin for a target ACT of 250 ms, and this was reversed at the end with protamine. Oxygen was administered by simple face mask with the patient spontaneously breathing throughout. One unit of pRBCs was transfused given his pre-procedural anemia. The patient tolerated placement of a 26mm Evolut valve without significant hemodynamic instability, requiring only a low-dose norepinephrine infusion. Of note, there was no excess bleeding during the procedure or in the immediate post-procedure period. As of November 2025 (7 weeks post-procedure), the patient's follow-up studies had demonstrated appropriate position and function of the prosthetic aortic valve. His anemia had improved, and he had no further hospital admissions.

Discussion

Heyde syndrome presents additional challenges in the already complex anesthetic management of the patient with severe AS. Of particular concern are challenging hemostasis and severe anemia. Preoperative assessment should include a detailed history of GIB. Endoscopic studies and interventions should be reviewed if available. In this case, the patient required transfusion of pRBCs and treatment with iron dextran to address acute on chronic, multifactorial anemia. It is important to recognize that anemia in patients with Heyde syndrome is an indirect sequela of AS, and that definitive therapy for their anemia cannot be achieved without AVR. It is also worth noting that the vWF destruction and decreased pulsatile flow that lead to GIB in Heyde syndrome can also occur in patients with mechanical circulatory support devices, albeit due to the

physiologic effects of the device itself rather than valvular stenosis. Thus, patients with MCS devices may similarly develop GIB and severe anemia [3].

Images



Transthoracic echocardiography measurements of the patient's aortic valve

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#FR 1 – Kiosk 23 – Poster 1

Bridging the Gap: Mechanical Circulatory Support for Management of Post-Infarction VSD

Presenting Author: Michael Berken, MD, ACTA fellow, University of Chicago

Introduction

Post-infarction ventricular septal defect (VSD) is a rare complication of myocardial infarction with high mortality. Repair of ischemic VSDs is a complex procedure that is made more difficult by the necrotic surrounding tissues. A waiting period of several days following infarction is often required, and how to best stabilize patients is a topic of debate. We present a case of utilizing Impella (Abiomed, MA) in a patient with a post-infarction VSD as a bridge to repair.

Case Presentation

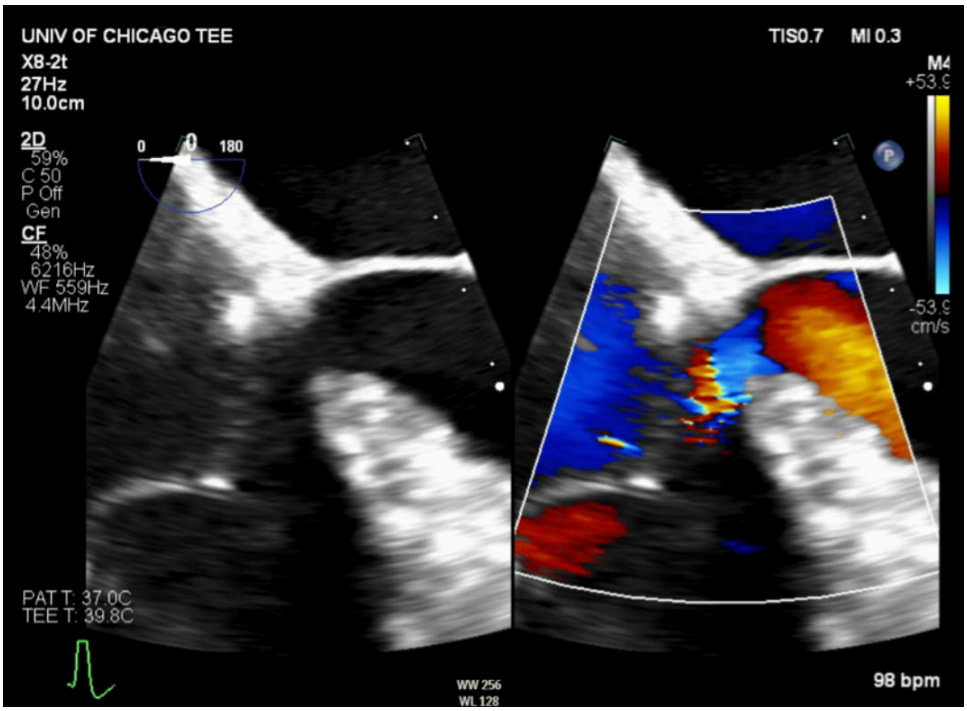
A 67-year-old man presented to an outside hospital with 2 days of shortness of breath and back pain. Imaging revealed a defect in the interventricular septum, and a transthoracic echocardiogram (TTE) revealed a large muscular VSD with left to right shunt, mildly reduced left ventricle (LV) function, and an inferior wall aneurysm. Left heart catheterization revealed a 100% right coronary artery occlusion. An intra-aortic balloon pump (IABP) was placed, and he was transferred to our institution. An axillary Impella was then placed with plans for definitive VSD repair. After ten days of Impella support he underwent interventricular septum reconstruction, LV aneurysm excision with reconstruction of LV free wall, and removal of the Impella. He was extubated on postoperative (POD) 1, transferred to the floor on POD 5, and discharged home on POD 7. Follow up TTE showed a small residual VSD.

Discussion

Post infarction VSDs have become increasingly rare due largely to advancements such as percutaneous coronary intervention (PCI)1. Prior to PCI, 1-2% of AMIs were complicated by post-infarction VSDs while recently the rate is 0.17-0.31%2. Despite this, mortality remains as high as 94% without intervention and 44-55% following repair3. Post-infarction VSDs can cause pulmonary edema and cardiogenic shock due to left to right shunting3. Surgical or percutaneous closure is the gold standard, with data showing a decrease in mortality if performed after seven days3. This period allows for fibrosis to develop around the ischemic defect, providing an anchor for patch material and sutures, and decreases the risk of dehiscence. Treatment modalities focus on afterload reduction, increasing effective LV stroke volume, and decreasing the shunt fraction2. While inotropic agents are often utilized in cardiogenic shock these can worsen shunting.

Mechanical circulatory support (MCS) can also be utilized to achieve these goals. The primary options are IABPs, VA ECMO and percutaneous LVADs such as Impella or Tandem Heart (LivaNova, England). In our case, Impella support was able to directly offload the LV, decrease the shunt, and increase cardiac output1. A benefit of axillary insertion is the ability to extubate and allow for mobilization1. Impella support for post-infarction VSDs does carry theoretical risks of insertion site infection or bleeding and embolization of infarcted tissue1. There is also a risk causing hypoxemia due to a right to left shunt from overly decompressing the LV2. Careful monitoring of oxygenation and echocardiography can identify shunt reversal and the flow can be titrated to minimize this risk2. Moving forward, improved mechanisms for stabilizing these patients may allow for more effective bridging to definitive repair and improve outcomes.

Images



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#FR 1 – Kiosk 23 – Poster 2

Incidental saddle embolus found by trans-esophageal echocardiography during elective coronary artery bypass surgery

Presenting Author: George Zhou, MD, Adult Cardiothoracic Anesthesia Fellow, Westchester Medical Center

Introduction

Saddle pulmonary embolism is a rare, potentially life threatening condition where a blood clot lodges at the bifurcation of the main pulmonary artery(1). While CT pulmonary angiography is the gold standard for diagnosis of pulmonary embolism, echocardiography can be useful in assessing the severity of illness by providing information on right heart strain and pulmonary artery pressures. Here, we present a case in which a patient undergoing elective CABG was incidentally found to have a saddle embolism on intraoperative TEE, allowing immediate surgical intervention.

Case Presentation

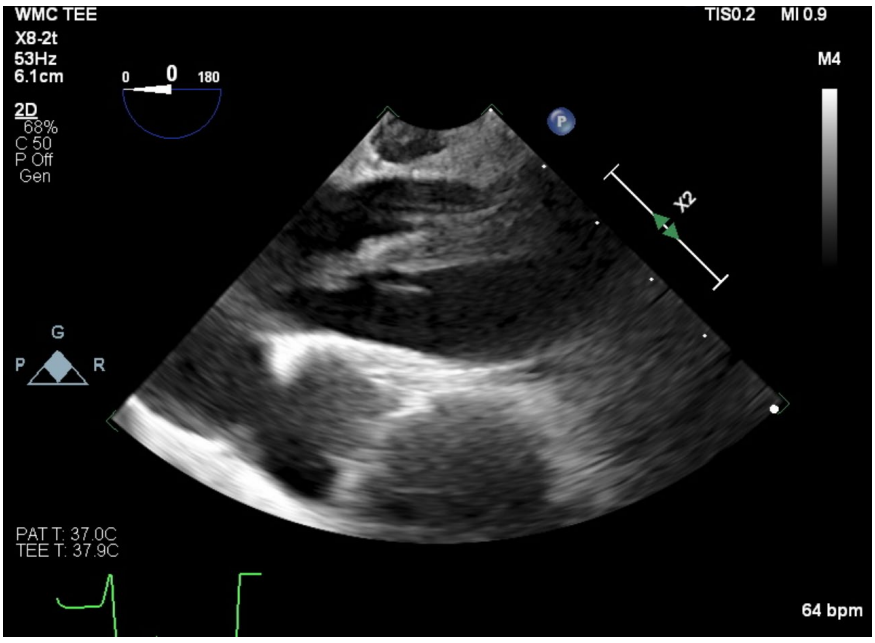
A male patient in his 70's with past medical history of COPD, asthma, hyperlipidemia, gastric ulcer, HFrEF, TBI secondary to MVA, anxiety disorder, anxiety, depression, and PTSD initially presented for a seizure but had an episode of ventricular tachycardia while undergoing ER evaluation and thus underwent a cardiac evaluation. Cardiac catheterization showed multivessel CAD and the patient was transferred for a CABG evaluation. He was started on nitroglycerin infusion for chest pain and low dose heparin infusion. As part of the evaluation lower extremity ultrasound was performed, which showed no evidence of venous thrombosis. On day 1 of admission the patient developed hematuria, and thus low dose heparin was stopped. Urologic workup was unrevealing, so heparin was then resumed on day 3 of admission. The patient presented to the OR 5 days after admission for elective coronary artery bypass grafting. In the operating room, standard monitors were placed and a pre-induction arterial line was placed into the left radial artery. A standard IV induction was performed. The patient was intubated and a TEE probe was passed into the esophagus. In the ME ascending aorta in short axis view, a clot was visible in the right pulmonary artery by the bifurcation of the main pulmonary artery. The echocardiographic exam was otherwise benign, with no evidence of right heart

strain. The RVSP derived from TR was 23 mmHg. The surgeon was notified of the findings. After successful cannulation and initiation of cardiopulmonary bypass, the surgery team incised the main PA longitudinally and removed a large clot, then closed the incision. After this the rest of the planned coronary bypass surgery was performed without incident. The patient was transported to the ICU intubated and sedated in stable condition. During the evening of POD 0 the patient had an IVC filter placed by vascular surgery. He was extubated late in the evening of POD 0 with no issues. On POD 1 he had venous ultrasounds performed demonstrating likely clot in the left peroneal vein and a left posterior tibial vein. He was transitioned from IV heparin to PO apixaban and discharged on POD 7.

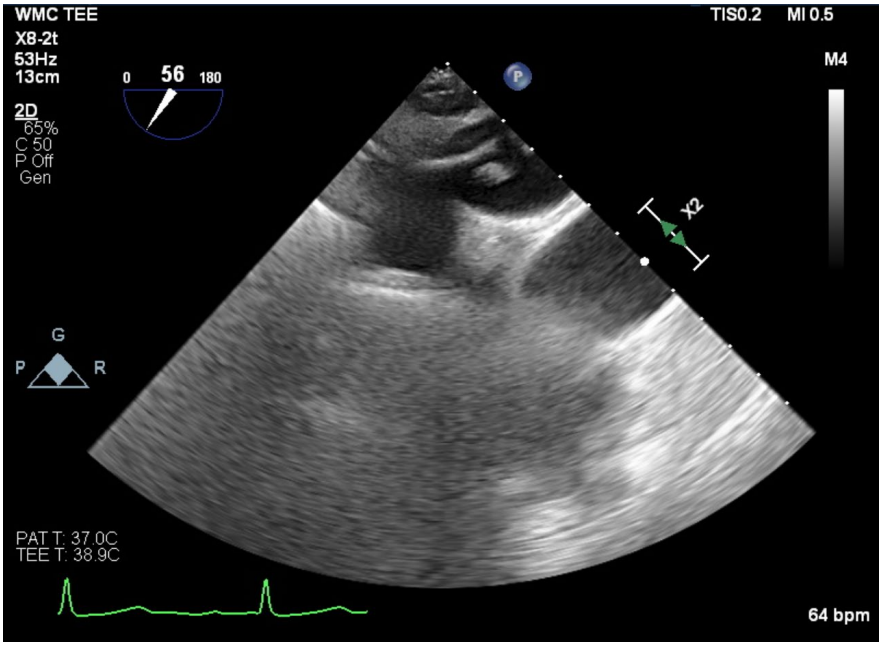
Discussion

TEE provides useful information in cardiac surgery, however it is not always used in isolated CABG surgery. In one database review 46% of isolated CABG procedures were performed without TEE, although TEE was associated with lower odds of mortality(2). The initial negative vein mapping study and use of heparin infusion all decreased our clinical suspicion for PE prior to surgery. This case serves as an example where TEE during routine CABG identified pathology that was previously unknown, allowing immediate surgical intervention.

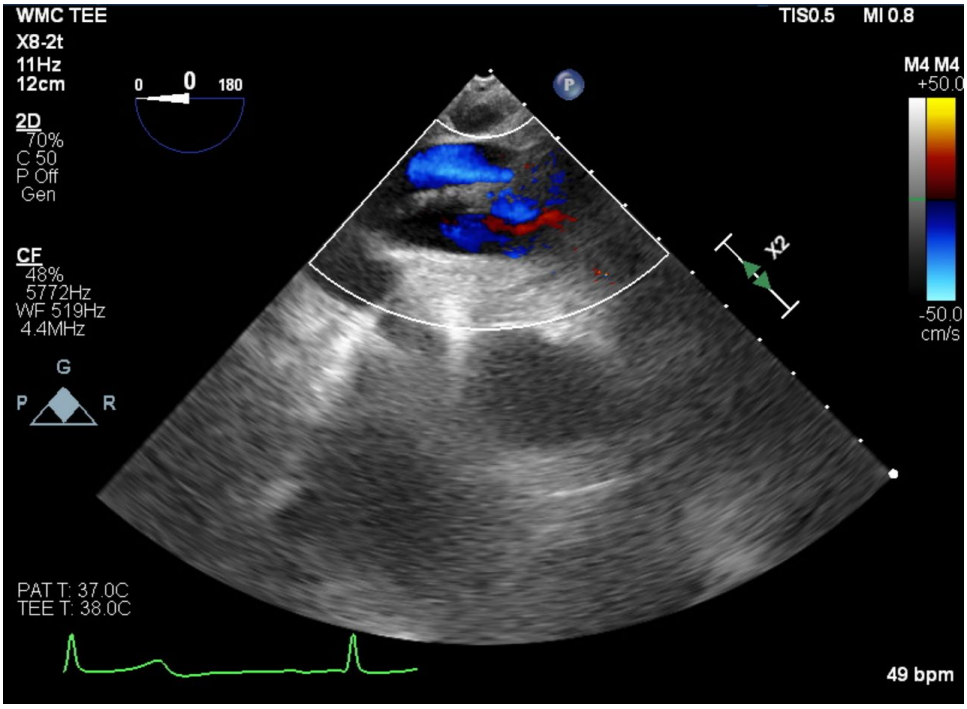
Images



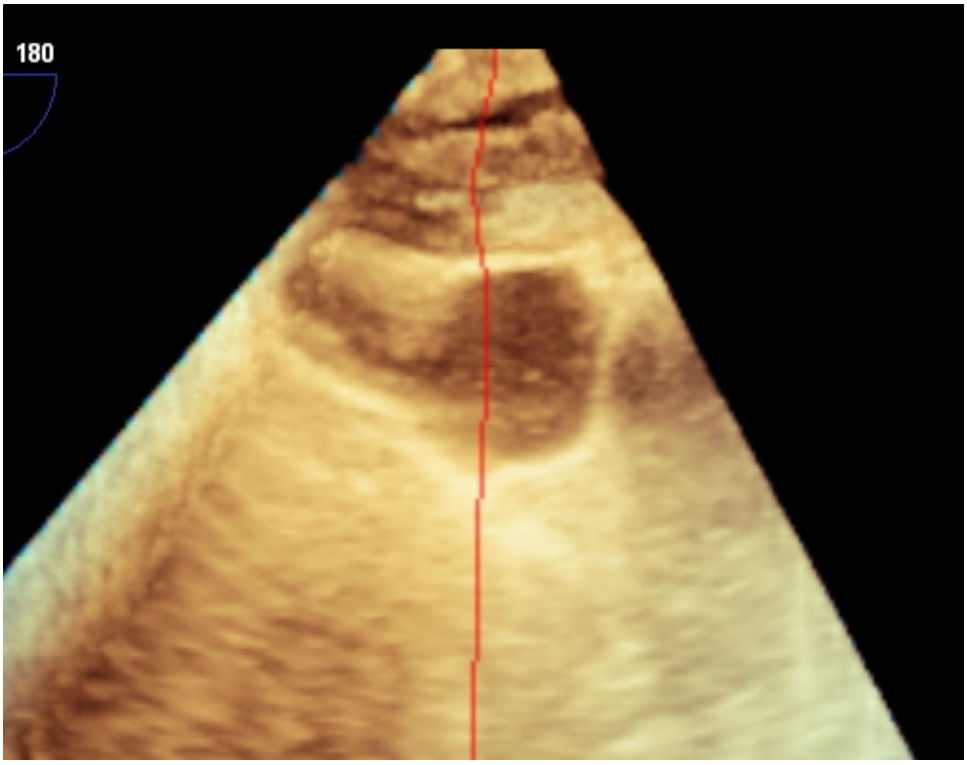
ME Ascending aorta short axis view showing PE at bifurcation of main PA



ME Ascending aorta long axis view showing PE in short axis



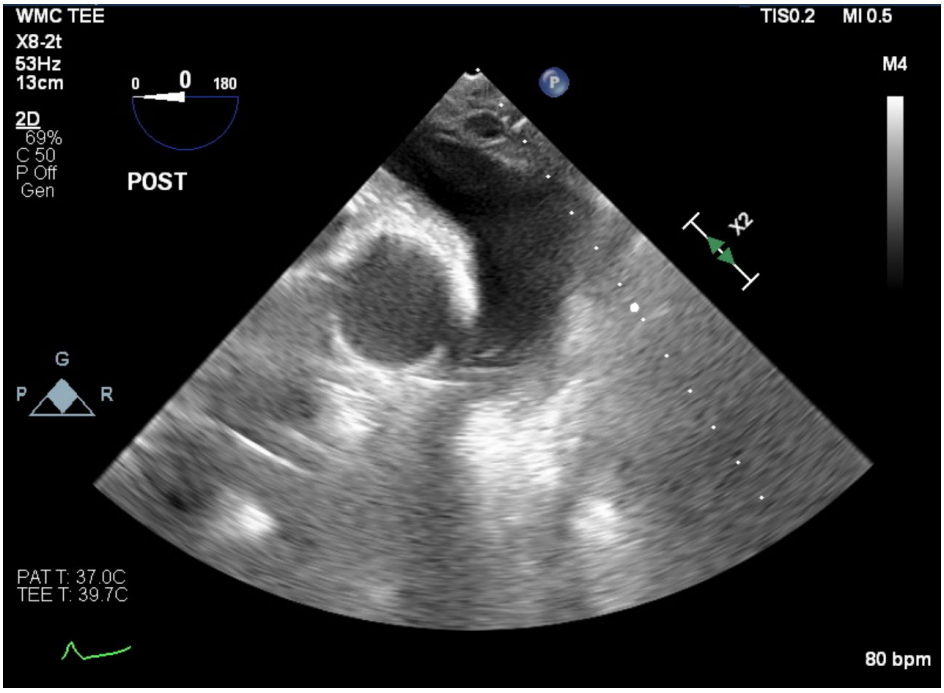
ME Ascending aorta short axis view with color doppler showing flow around the PE



3D TEE image showing PE



Photograph of large clot after removal from pulmonary artery



ME Ascending aorta short axis view after pulmonary embolectomy showing absence of clot

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#FR 1 – Kiosk 23 – Poster 3

Between a Rock and a Hard Place – Approach to Post-Pulmonary Endarterectomy Mechanical Circulatory Support

Presenting Author: Cory Roth, MD, Adult Cardiothoracic Anesthesiology Fellow, Cleveland Clinic Anesthesiology Institute

Introduction

Chronic thromboembolic pulmonary hypertension (CTEPH) is a rare but potentially curable form of pulmonary hypertension resulting from recurrent pulmonary emboli and fibrotic vascular remodeling. Pulmonary thrombo-endarterectomy (PTE) improves hemodynamics, reverses vascular remodeling and improves long term survival. We present a patient developing significant hypoxemia following successful PTE requiring complex extracorporeal life support (ECLS) decision making.

Case Presentation

A 43 year-old woman with morbid obesity post gastric bypass, membranous nephropathy, IVC stenosis and recurrent pulmonary emboli managed with apixaban, was transferred for PTE on veno-arterial (VA) ECLS for right ventricular (RV) failure after catheter thrombectomy with minimal thrombus removal. Pre-op chest Xray showed airspace and interstitial opacification and pre-op TEE showed normal left and moderately reduced RV function with mild tricuspid regurgitation. Peripheral VA ECLS was converted to CPB with addition of SVC inflow and ascending aortic outflow cannulas and the patient underwent bilateral PTE under deep hypothermic arrest. After warming, the patient weaned from CPB on inotropic support with good RV function but experienced decreased ventilatory compliance and refractory hypoxia. The etiology was multifactorial given pre-op acinetobacter pneumonia, pulmonary edema, and distal thromboembolic disease, and the decision to institute ECLS was made. Due to a history of IVC stenosis and RIJ occlusion placement of a second venous cannula for veno-venous (VV) ECLS was not possible. Thus, oxy-right ventricular assist device (RVAD) was placed via existing femoral venous cannula and pulmonary artery graft for return. After the oxy-RVAD initiation (flow 3.0 L/min, sweep 3.0 L/min) serosanguinous endotracheal tube secretions with worsening lung compliance, consistent with massive pulmonary edema, and worsening RV dysfunction occurred. The patient is currently in the ICU awaiting pulmonary recovery on RVAD support.

Discussion

Post PTE management is tailored towards amelioration of ischemia-reperfusion injury by avoiding cardiac output augmentation, avoiding pulmonary vasodilators, adequate surgical hemostasis, and judicious fluid administration. CPB separation unmasked underlying refractory hypoxemia that required respiratory ECLS. The decision on how to support the patient was complex. While VV ECLS may have presented the best option, it was not possible due to IVC and RIJ disease. Reinitiation of VA ECLS has the advantage of protection against reperfusion injury by bypassing pulmonary circulation but risks thrombosis at fresh endarterectomy vascular beds due to endothelial disruption. Oxy-RVAD was a feasible option to provide hypoxemia support. Oxy-RVAD initiation has the advantage of RV off-loading, however, the unintended

consequence in this case was blood return directly into the pulmonary arterial system with high hydrostatic pressure which led to worsening pulmonary edema from reperfusion injury and loss of lung compliance. In this case, competing treatment goals arose from the need to balance RV failure, intrinsic lung disease and anatomic limitations that no MCS configuration could achieve simultaneously.

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#FR 1 – Kiosk 23 – Poster 4

Severe Aortic Insufficiency with Peri-Induction Cardiovascular Collapse: Anesthetic Considerations in Axillary Impella Placement

Presenting Author: Javaughn R. Baker, MD, Fellow Physician, WashU Medicine, Department of Anesthesiology

Introduction

Severe aortic insufficiency (AI) poses major anesthetic challenges due to preload dependence, vulnerability to changes in systemic vascular resistance (SVR) and LV loading, and limited mechanical circulatory support (MCS) options. We report a case of acutely worsened chronic severe AI with peri-induction cardiovascular collapse requiring mechanical support, emphasizing anesthetic management of acute decompensated AI and considerations for Impella placement.

Case Presentation

A 65-year-old man with moderate–severe AI receiving treatment for MRSA infective endocarditis presented for AVR. History included HFpEF, hypertension, class III obesity, diabetes, and OSA on oxygen. Preoperative TEE showed mildly dilated LV with EF 50%, normal RV function, and an eccentric AI jet with vegetation on the right coronary cusp. After induction, he developed profound hypoxemia and hypotension refractory to high-dose vasopressors and inotropes. TEE revealed new severe biventricular dysfunction, torrential AI with minimal forward stroke volume. Near systemic PA pressures were noted. Large bilateral pleural effusions compounded hypoxemia despite maximal ventilatory support. Given refractory instability, review of real-time TEE, PA catheter data, escalating vasopressor and inotropes, the anesthesia, heart failure, and surgical teams agreed that proceeding with AVR under profound shock was prohibitively risky. Collaboratively, the teams decided that axillary Impella 5.5 placement offered the safest path of stabilization before future surgery. After bilateral chest tubes, Impella was placed. Positioning was optimized via measurement of Impella, verification of waveforms on the console, and evaluation of inflow, outflow, and AI jets. Indices of LV loading were used to guide the effectiveness of therapy. Within hours, he was stabilized on a single low-dose inotrope. After aggressive diuresis for surgical optimization, he subsequently underwent AVR with VA-ECMO, decannulation, and was discharged to inpatient rehabilitation on postoperative day 31.

Discussion

Management of severe AI requires preserving forward flow and minimizing regurgitant volume. These patients depend on adequate preload and maintained or slightly elevated SVR to reduce regurgitant flow across the incompetent valve. Induction-related vasodilation can worsen regurgitation, reduce coronary perfusion, and precipitate collapse. Hemodynamic goals include avoiding bradycardia, supporting contractility, preserving afterload, and ensuring sufficient intravascular volume.1 Severe AI is a relative contraindication to Impella use because retrograde flow into the LV may worsen regurgitation.2 Impella-assisted LV unloading was confirmed through real-time multimodal assessment. TEE demonstrated decreased LV end-diastolic dimensions. Hemodynamic data showed reductions in pulmonary artery

pressures at Impella flow of 4.2 L/min. Optimal placement was guided by TEE, confirming inlet positioning below the aortic annulus while avoiding wall contact and mitral entanglement. Color Doppler verified unobstructed LV inflow and laminar aortic outflow. This case highlights the importance of vigilant anesthetic management of severe AI, rapid recognition of peri-induction collapse, and coordinated deployment of MCS when conventional support is insufficient.

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#FR 1 – Kiosk 23 – Poster 5

Two Concurrent Valves via Transcatheter Mitral Valve Replacement as a Rescue for Failed Mitral Transcatheter Edge-to-Edge Repair

Presenting Author: Shelly Gupta, DO, Fellow, Emory University School of Medicine

Introduction

While less invasive, mitral transcatheter edge-to-edge repair (M-TEER) carries a higher risk of recurrent mitral regurgitation compared to that of conventional surgery. However, there are limited non-invasive options for failed M-TEER. We present a case of a patient who underwent emergent transcatheter mitral valve replacement (TMVR) with concurrent deployment of two valves to occupy the mitral orifice status post failed M-TEER. To our knowledge, this case represents the first documented use of this approach.

Case Presentation

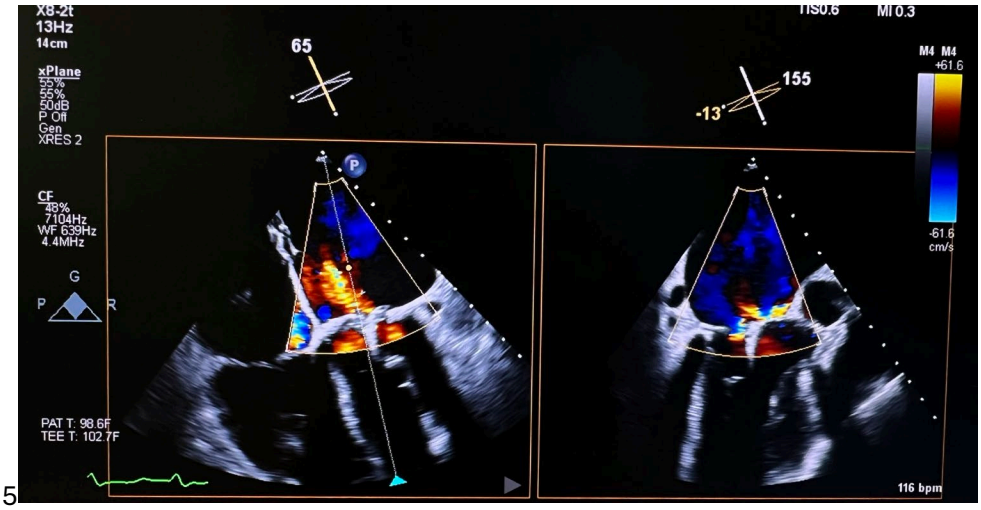
A 86-year-old woman with a history of atrial fibrillation on warfarin, hypertension, pulmonary hypertension, heart failure with preserved ejection fraction, severe tricuspid regurgitation, and non-rheumatic moderate-severe mitral regurgitation presented in cardiogenic shock requiring multiple vasoactive medications and an intraaortic balloon pump (IABP). She had undergone M-TEER complicated by residual severe mitral regurgitation (MR), post-procedural severe mitral stenosis (MS), and cardiac arrest. Two days later, as she was not an operative candidate, an emergent TMVR with CPB support was proposed, involving concurrent deployment of two valves to occupy the medial and lateral orifices created by the original mitraclip. After an uneventful induction of general anesthesia, the patient was cannulated peripherally (RIJ venous, R CFA arterial) and CPB was initiated. A 29 mm SAPIEN valve was deployed simultaneously with a 24 mm CP stent, immediately followed by a 22 mm Melody valve deployed inside the CP stent under TEE guidance. The procedure was uneventful with the patient successfully being weaned from CPB, remaining on chemical and IABP support. She was transferred to the intensive care unit post-operatively. Post-procedurally, there was no residual MS and likely moderate residual MR with the valves in stable position with a mean gradient of 5 mmHg. Postoperatively, she was not anticoagulated secondary to a supratherapeutic INR. On POD 5, a CT head imaging showed a hypodensity in the right parietal lobe concerning for stroke. On POD 6, she was started on a heparin drip; later that day, she was transitioned to comfort care by family.

Discussion

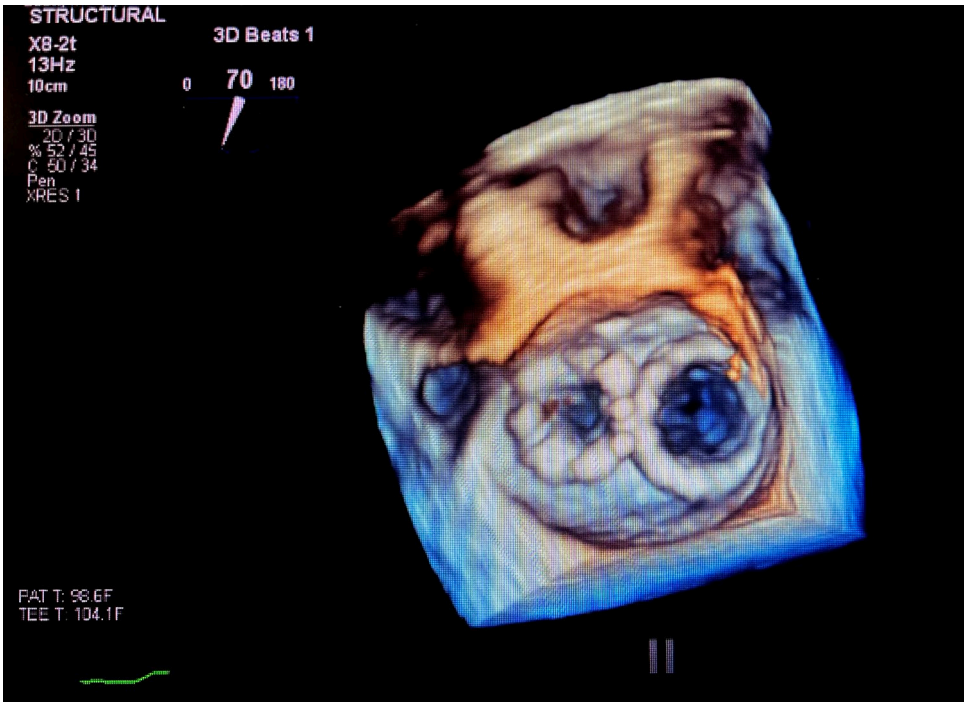
Mitral regurgitation is the most prevalent valvular disease in developed countries with a prevalence of 10% of the total population worldwide. Historically, surgical mitral valve repair or replacement was considered the gold standard. With the advancement of transcatheter approaches to cardiac disease, newer treatment options are available to patients who are poor cardiac surgery candidates (1). Studies have suggested that about 80% of patients who undergo M-TEER achieve an immediate reduction of regurgitation; about 70% of patients experience a significant improvement in quality of life and functional status. However, limited options exist for those patients who experience failed M-TEER besides invasive surgery (2). Our case describes a novel successful approach to refractory severe MR following M-TEER using two transcatheter

valves to occupy the residual medial and lateral mitral orifices. Further investigation must be conducted to evaluate less invasive options for failed M-TEER procedures as percutaneous treatment modalities become more established.

Images

5 

Failed M-TEER



SAPIEN valve in the medial orifice (right), CP stent plus Melody valve in the lateral orifice (left)

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#FR 1 – Kiosk 23 – Poster 6

Mitral Valve Replacement under Cardiopulmonary Bypass in the Pregnant Patient

Presenting Author: Sheddie Gonzalez-Castillo, MD, UVA Health

Introduction

Although rare, cardiac surgery in pregnancy is usually urgent and cannot be delayed. The anesthesiologist must manage both maternal physiology and fetal vulnerability. If possible, the second trimester is optimal, reducing the risks of first-trimester loss and third-trimester preterm labor. Knowledge of the effects of cardiopulmonary bypass (CPB) on the fetus and how to optimize fetal viability and maternal safety is essential in order to achieve acceptable outcomes.

Case Presentation

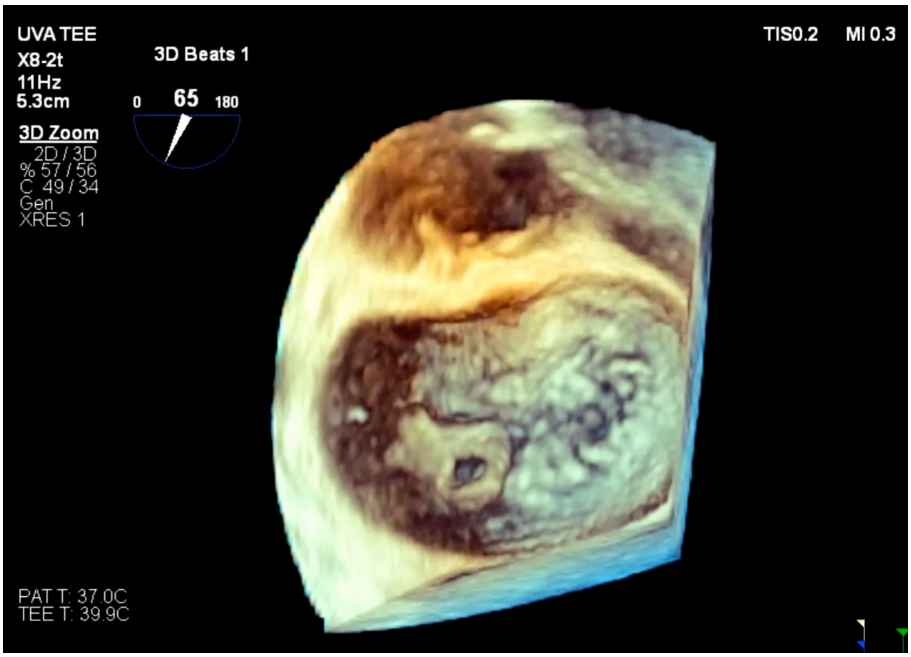
A 26 year old woman who was 15 weeks and 2 days pregnant presented with fever and malaise and found to be bacteremic. TTE showed a 1x1.5cm posterior mitral leaflet vegetation with perforation causing severe regurgitation, consistent with endocarditis. She underwent a mitral valve replacement under CPB. General anesthesia was induced with fentanyl, lidocaine, propofol and rocuronium. She was cannulated in standard fashion and CPB was initiated without complication. She was kept normothermic and CPB flows were kept > 3L/min/m^2. The anesthesia team maintained normotension. She was separated from CPB without complication, and TEE showed a well-seated bioprosthetic valve without leak. Fetal heart tones were checked pre- and postoperatively with no changes. She was extubated in the ICU several hours later and discharged home on POD 5.

Discussion

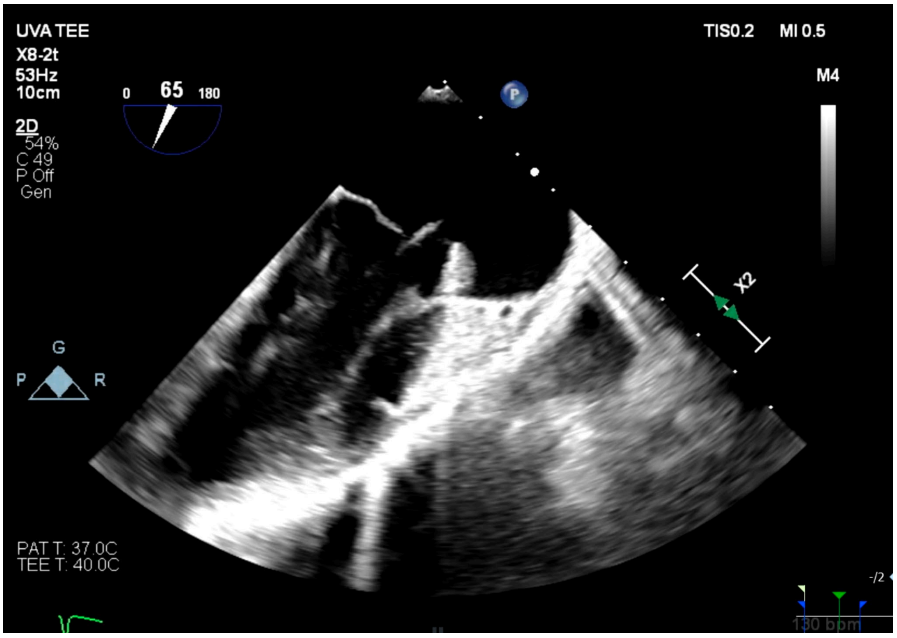
Cardiac surgery and CPB carries substantial fetal risk, with reports of up to 50% mortality. Pursuing cardiac surgery requires a patient-centered multidisciplinary discussion. Loss of pulsatile flow, hypotension, and alterations in acid–base status can precipitate fetal bradycardia, while uterine contractions—strongly associated with fetal morbidity and mortality—increase in frequency with advancing gestational age and are particularly common during the rewarming phase. For these reasons, several fetal-protective strategies have been proposed. Higher pump flow rates (>2.5 L/min/m^2), perfusion pressures >70 mmHg, hematocrits >28%, and normothermia appear beneficial, with case series demonstrating significantly improved fetal survival under normothermic conditions compared to hypothermia. Alpha-stat CO_2 management is preferred, as both maternal hypo- and hypercapnia can detrimentally alter uteroplacental blood flow. Standard-dose heparin is safe because it does not cross the placenta. Pulsatile flow may provide theoretical benefit, though the evidence remains inconclusive. Regardless of strategy, minimizing CPB time, avoiding hypotension, ensuring adequate maternal oxygenation, and maintaining left uterine displacement (if estimated gestational age > 20 weeks) are most critical. Fetal monitoring during these procedures is individualized. In pre-viable pregnancies, pre- and postoperative fetal heart rate (FHR) assessment is sufficient. In viable pregnancies, FHR and contraction monitoring should be performed before and after surgery at minimum. Intraoperative monitoring is reserved for select situations and only when all

prerequisites are met: fetal viability, the physical ability to monitor continuously, the presence of an obstetrician credentialed for emergent cesarean delivery, clear informed consent for emergent C-section, and a surgical plan that can be safely paused for delivery if required.

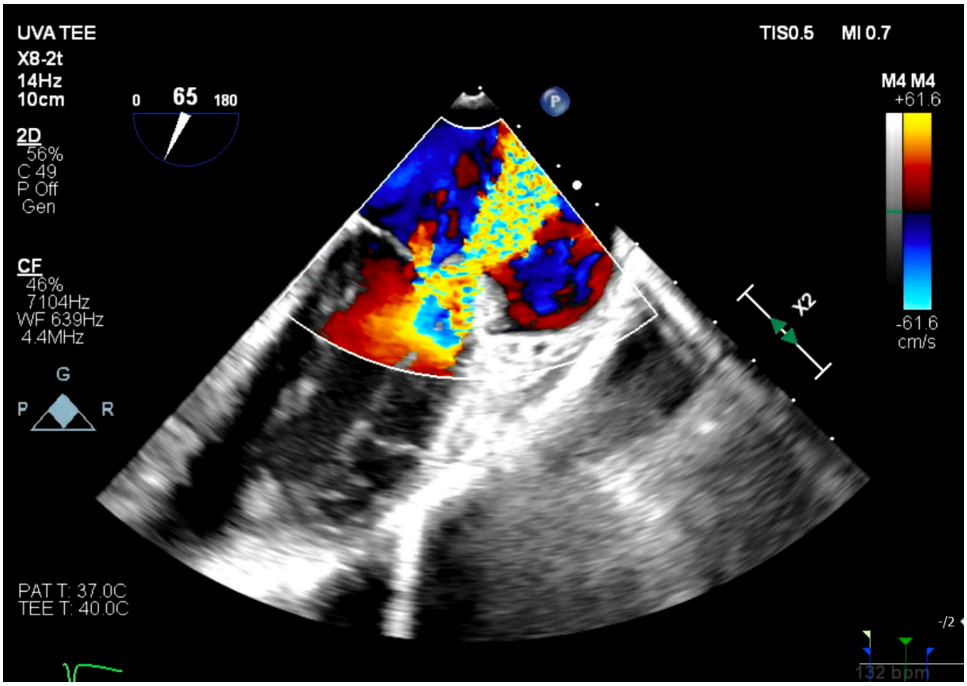
Images



Mitral valve pre-CPB



Commisural view pre-CPB



Commisural view with color

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#FR 1 – Kiosk 23 – Poster 7

Revision Mustard Procedure in an Adult D-TGA Patient: Echocardiographic Findings and Anesthetic Management

Presenting Author: Christopher A. Folgueras, MD, Cardiothoracic Anesthesiology Fellow, New York-Presbyterian Hospital, Columbia Campus

Introduction

Dextro transposition of the great arteries (D-TGA) is a congenital defect in which the aorta arises from the right ventricle and the pulmonary trunk from the left ventricle, creating two parallel circuits that separate oxygenated and deoxygenated blood. The Mustard procedure is a surgery to palliate D-TGA that reroutes systemic venous return, via a baffle sutured between the atria, to the left ventricle; oxygenated blood then returns to the morphologic systemic right ventricle by way of the pulmonary veins through another baffle (2). We describe a patient with a childhood Mustard repair who presented with worsening heart failure symptoms attributed to pulmonary return baffle obstruction, along with key transesophageal echocardiogram findings and anesthetic management.

Case Presentation

A 57-year-old male with prior Mustard procedure in infancy for D-TGA presented with progressive dyspnea on exertion, fatigue, and paroxysmal SVT. TTE showed pulmonary venous baffle obstruction, severe pulmonary artery dilation, and pulmonary venous pathway obstruction with a significant gradient across the pulmonary valve. General anesthesia was induced primarily with midazolam and fentanyl after initiating vasopressor and inotropic support, out of concern for right ventricular dysfunction in the setting of pulmonary return baffle obstruction. Preoperative transesophageal echo (TEE) was performed and revealed a severely dilated, hypertrophied right ventricle with moderately reduced function and posterior bowing of septum. The pulmonary return baffle was identified by withdrawing the probe 2-3cm from the midesophageal 4-chamber view and identifying flow acceleration with color flow doppler. Continuous wave doppler demonstrated a mean gradient (MG) of 10 mmHg. Additional findings were moderate tricuspid regurgitation, severe sub-pulmonic valve stenosis vs left ventricular outflow tract obstruction (MG 79), moderate tricuspid regurgitation, trace mitral regurgitation, trace sub-systemic valve regurgitation, and a severely dilated main pulmonary artery. Operative findings included a calcified Mustard patch with severe obstructions at multiple levels. Both the procedure and separation from bypass were largely uneventful except for brief supraventricular tachycardia that resolved with inotropy wean. The post-operative TEE was notable for improved visualization of the repaired pulmonary return baffle and visualization of laminar flow within the systemic return baffle. Additional findings included improvement of the sub-pulmonic/LVOTO MG to 34 mmHg. Biventricular function was preserved and no worsening of valvulopathies was appreciated. The patient had an uncomplicated ICU course and was transferred to stepdown on POD1.

Discussion

While the Mustard procedure has fallen out of favor for D-TGA palliation, the procedure was performed as

late as 1994 (1). Additionally, there has been some interest in reviving the Mustard procedure for late-stage presentation TGA in low- and middle-income countries (1, 3). With this consideration and given the steadily improving survival of patients with congenital heart disease, an understanding of TEE anatomical nuances and the physiological complications that can arise in this aging population is essential.

Images

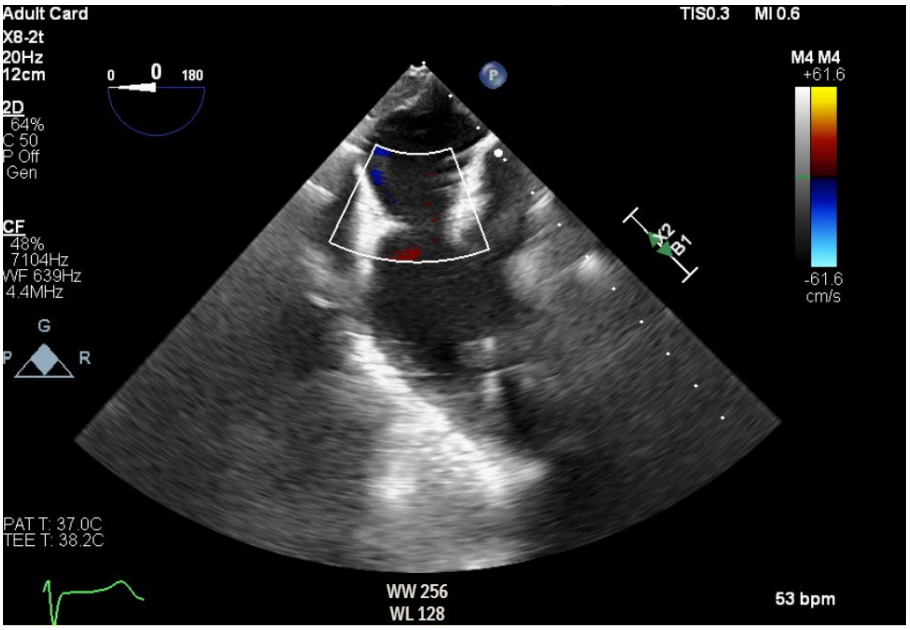


Figure 1: Preoperative image of the pulmonary return baffle. Located at 0 degrees with probe withdrawn 2-3cm from midesophageal four chamber position. Anteriorly, the systemic return baffle can be visualized as well

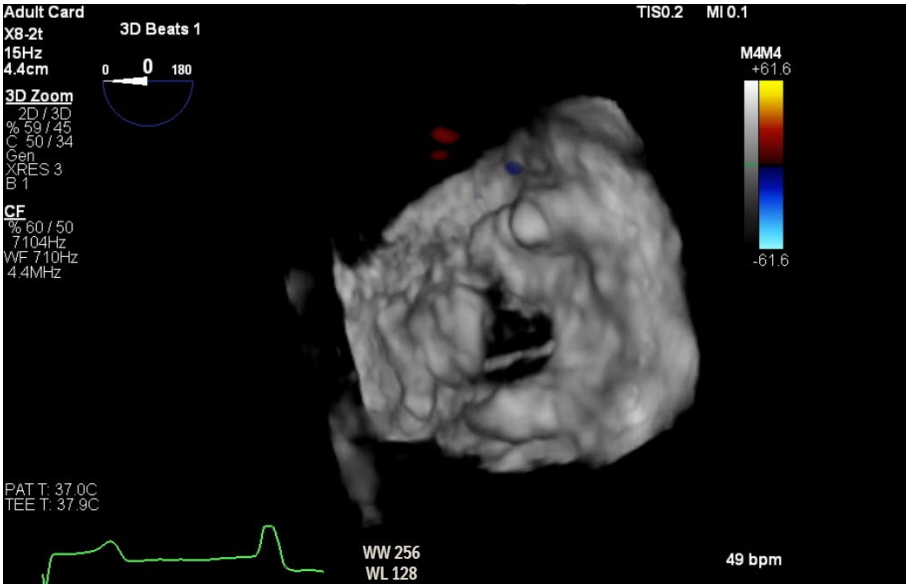


Figure 2: 3D Echocardiographic image of the pulmonary return baffle visualized from the right atrium

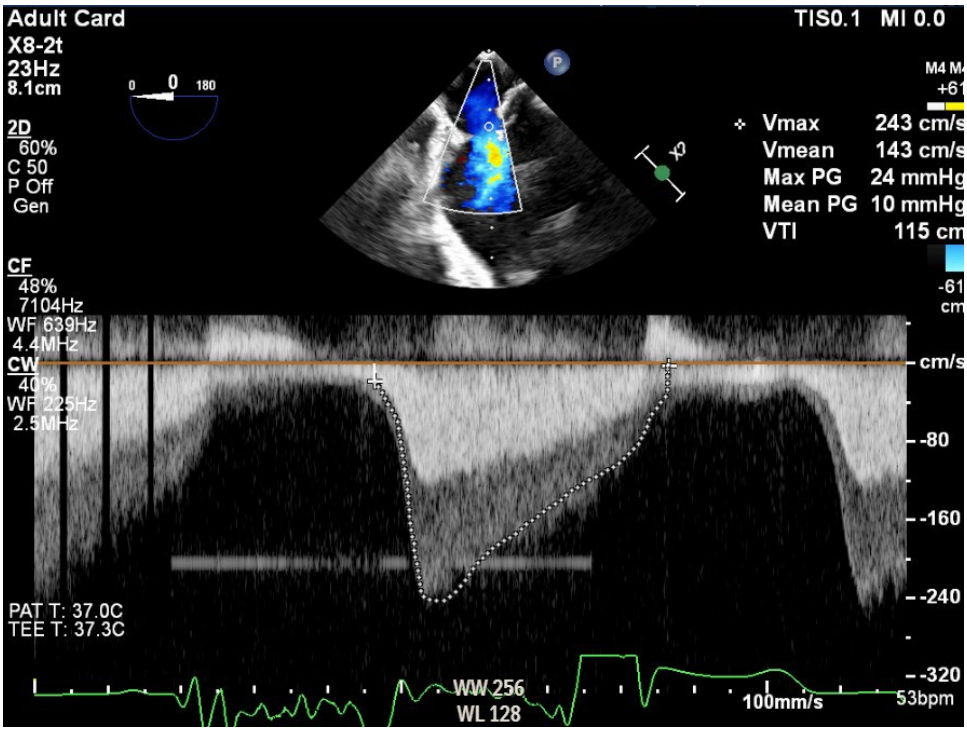


Figure 3: Continuous wave doppler applied through pulmonary return baffle demonstrating a mean gradient of 10mm Hg

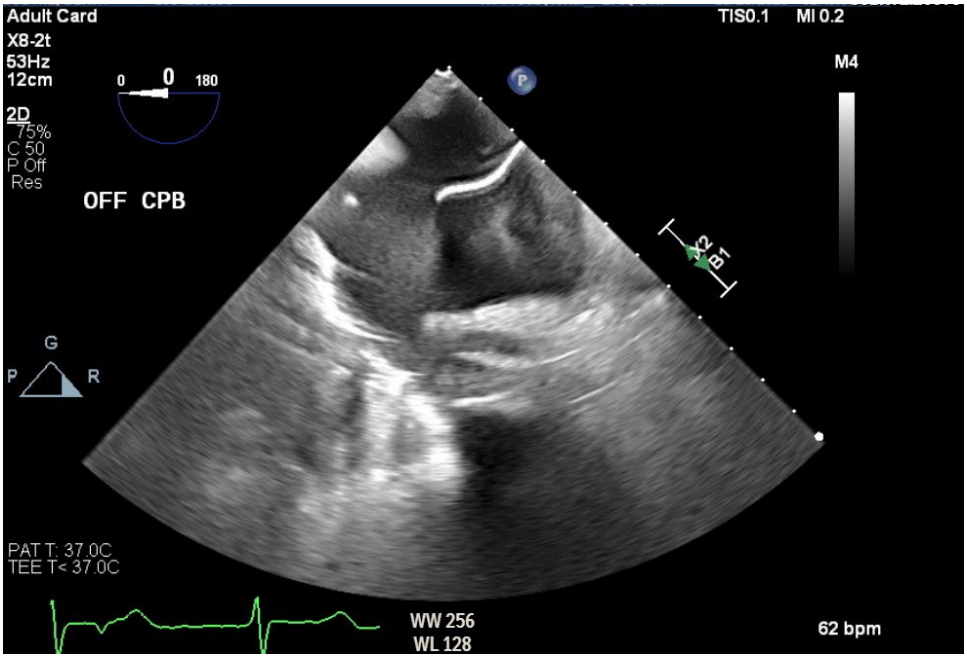


Figure 4: Post-operative image of the reconstructed baffles. The systemic return baffle can be seen much more clearly anterior to the pulmonary return baffle.

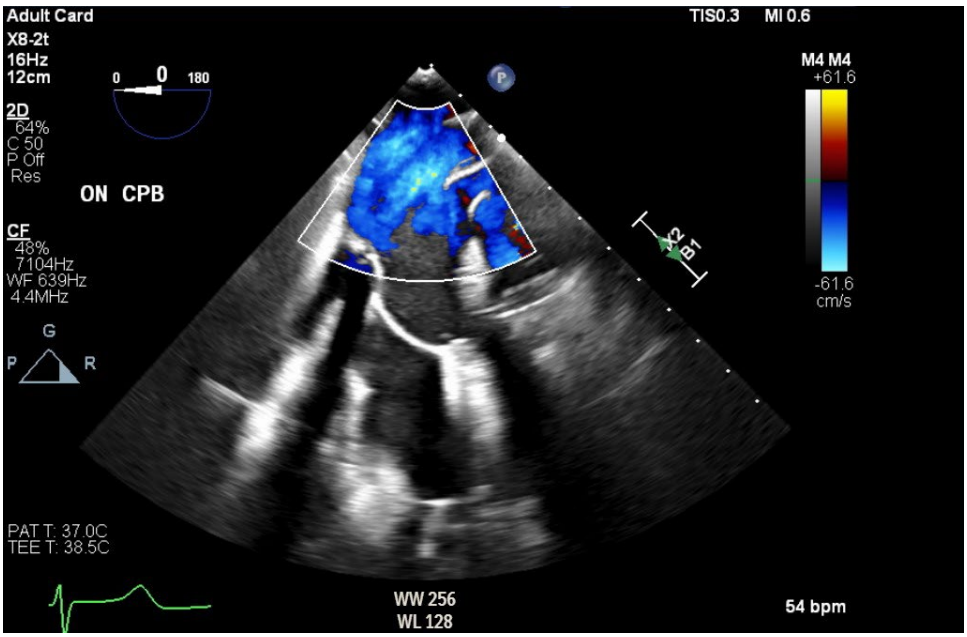


Figure 5: Post-operative image of laminar flow through the reconstructed pulmonary return baffle

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#FR 1 – Kiosk 24 – Poster 1

Septal Myectomy with Preexisting Perimembranous Ventricular Septal Defect Repair and Subaortic Membrane Resection with Mechanical Aortic Valve Replacement

Presenting Author: Patrick Haggerty, MD, Cardiac Anesthesia Fellow, Allegheny General Hospital

Introduction

Subvalvular aortic stenosis (SAS) is a rare disorder seen in infants. In most cases, it involves the presence of a membrane that is typically muscular just below the aortic valve. SAS causes clinically significant obstruction to the blood flow ejecting out of the left ventricle resulting in the development of concentric left ventricular hypertrophy, frequently with a septal bulge. SAS may co-occur with a ventricular septal defect, PAD, coarctation of the aorta, bicuspid aortic valve, abnormal left ventricular papillary muscle, and/or atrioventricular septal defect. We are presenting a case of a patient with PDA and coarctation of the aorta status post repair at 10 days old with bicuspid aortic valve, perimembranous VSD, and subaortic membrane who developed septal hypertrophy.

Case Presentation

A 51 y/o female with history of CHD, non-obstructive CAD, moderate aortic stenosis, hypertension, pulmonary hypertension, and OSA presented for septal myectomy, VSD repair, excision of subaortic membrane, and mechanical AVR versus Ross procedure. Patient initially presented with multiple visits to the ER for atypical chest pain and shortness of breathe. Preoperative TEE showed normal LV systolic function (EF 50-55%) with LV hypertrophy and mild RV systolic dysfunction. The septal knob measured 1.4 cm in the A-P diameter. The aortic valve was bicuspid with low flow low gradient moderate aortic stenosis. A subaortic membrane measuring 0.5 x 1.6 cm was present with flow acceleration across the LVOT. Of note the VSD was not seen in the initial TEE possibly due to turbulent flow from subaortic membrane. The intra-operative TEE showed perimembranous VSD with flow convergence from left to right. The RHC demonstrated normal cardiac ouput index, step-up in oxygen saturation in the pulmonic artery with Qp/Qs 1.36, and severe pulmonary hypertension with PA pressure of 72/32. Induction of general anesthesia and CPB were uncomplicated. Due to condition of LVOT tissue, patient underwent mechanical AVR instead of Ross procedure. Septal myectomy, vsd repair, and excision of subaortic membrane was performed. Inhaled nitric oxide was used to support the RV in the setting of severe pulmonary hypertension while weaning from CPB. The patient was successfully weaned from CPB with minimal vasoactive and inotropic support. The patient was extubated and the postoperative course was uncomplicated.

Discussion

Cardiac output and systolic function are well maintained until severe obstruction develops in SAS.The rate of progression of SAS is slow in adults This patient had a normal cardiac ouput index and normal LV systolic function but started to become symptomatic with atypical chest pain and shortness of breathe. Exertional dyspnea is the most common symptom seen in 40% of symptomatic patients, and it reflects pulmonary venous hypertension that is induced by an increase in left ventricular filling pressure caused by the impaired

diastolic compliance of the hypertrophied left ventricle. The survival in patients who undergo surgery to excise the subaortic membrane is excellent, but these individuals need to be followed up as the left ventricular outflow tract gradient increases slowly over time. Most patients will need re-operation for recurrence at some point in their lifetime.

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#FR 1 – Kiosk 24 – Poster 2

In the Right Place at the Right Time: Pulmonary Artery Catheter and Transesophageal Echocardiography as Predictive Metrics for Right Ventricular Assist Device Placement

Presenting Author: Vincent Franco, MD, Jersey Shore University Medical Center

Introduction

Right ventricular (RV) failure remains a major complication after left ventricular assist device (LVAD) implantation. The ability to predict RV failure post LVAD placement is difficult due to a lack of reliable metrics to capture RV reserve (Vogel et al 2025). Combining pulmonary artery catheter (PAC) data with transesophageal echocardiography (TEE) offers a more complete assessment of RV function as well as a functional and structural assessment. We present a case where integrated PAC and TEE findings identified high RV failure risk and guided prophylactic right ventricular assist device (RVAD) placement at the time of LVAD implantation.

Case Presentation

A 52-year-old man with hypertension, chronic kidney disease and nonischemic cardiomyopathy, secondary to chemotherapy treatment of Ewing Sarcoma at age 14 with NYHA IV presents in SCAI C stage. Echocardiogram demonstrated reduction of left ventricular ejection fraction (LVEF) to 21-25%. He remained dependent on continuous inotropic support, consistent with INTERMACS 3 profile. LVAD implantation was recommended as destination therapy as he was not a candidate for heart transplantation. Preoperative TEE demonstrated moderate RV dilation (4.5 cm), moderate tricuspid regurgitation, an RV FAC of 25%, an RV/LV ratio of 0.75, and moderate RV dysfunction. PAC data revealed a right atrial pressure of 20 mmHg, PAP of 63/34 mmHg, and a PCWP of 26 mmHg, yielding a CVP/PCWP ratio of 0.68, PAPi of 1.45, transpulmonary gradient (TPG) of 18, and PVR of 3 Wood units. His EUROMACS-RVF score was 5. These findings indicated a moderate-to-high risk for postoperative RV failure. Therefore, the decision was made to proceed with prophylactic RVAD placement at the time of LVAD implantation. A hybrid RVAD configuration using an Oxy-CentriMag system (femoral vein–to–pulmonary artery graft) was implanted. Postoperatively, the patient demonstrated rapid recovery: he was extubated 12 hours after surgery, the oxygenator was removed on postoperative day (POD) 1, and the RVAD was successfully weaned and explanted on POD 4 without evidence of acute kidney injury.

Discussion

RV failure (RVF) remains a serious complication after LVAD implantation, driven by altered loading conditions, increased RV strain, and pre-existing RV dysfunction. Predicting RVF is challenging, as individual hemodynamic or echocardiographic markers often underestimate risk when interpreted in isolation. Integrating TEE, PAC data, and validated risk tools such as the EuroMACS score is essential to identify patients who may benefit from prophylactic RVAD support. Key PAC-derived parameters include mean PAP, PAPi, RVSWi, the CVP/PCWP ratio, PVR, and TPG (Table 1). TEE findings that raise concern include an increased RV/LV ratio, reduced RV FAC, and moderate-to-severe RV dysfunction on strain or surrogate

measures despite optimized inotropic support. In this case, the decision to place a prophylactic RVAD avoided the need for emergent postoperative rescue, prevented multi-organ injury, and facilitated early extubation. This highlights the importance of a synergistic evaluation of PAC metrics and TEE assessment to identify high-risk LVAD candidates who would benefit from planned biventricular mechanical support, ultimately improving perioperative stability and long-term outcomes.

Images

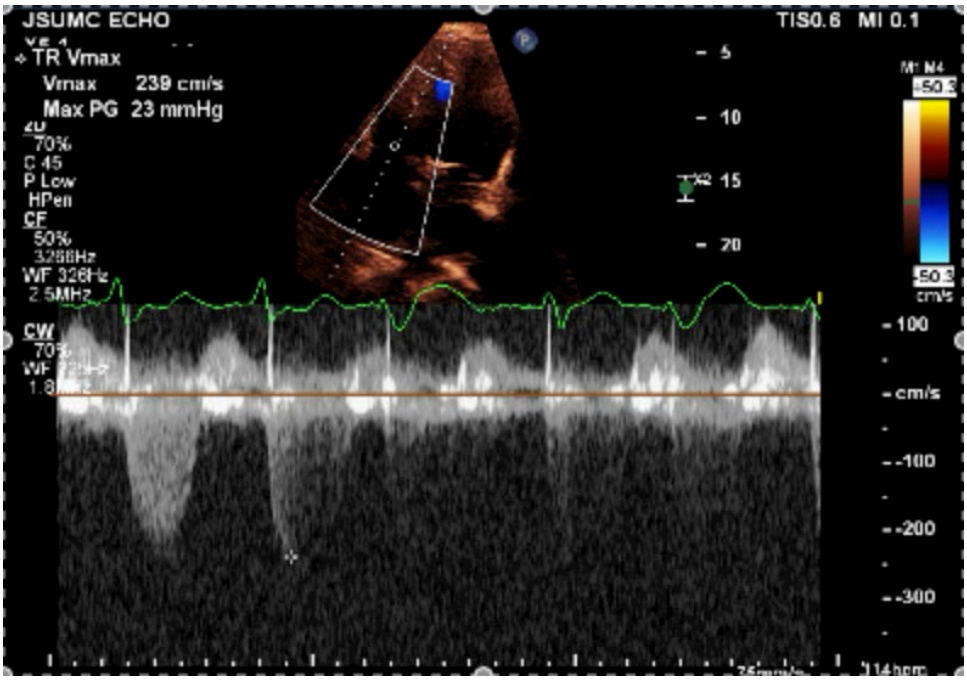


Figure 1. This ECHO image reveals moderate tricuspid regurgitation with a consistent elevated right-sided filling pressure and altered RV function.

Table 3 **Haemodynamic parameters to predict right ventricular failure following left ventricular assist device implantation**

Haemodynamic parameter	*n*	Main findings
Right atrial pressure		
Dandel *et al.*[26]	205	CVP was higher in patients undergoing LVAD and postoperative RV failure compared to those without RV failure (18 vs. 11 mmHg, $p = 0.01$)
Drakos *et al.*[37]	175	Patients following LVAD that reveled RV failure had higher CVP preoperatively (>11.6 ± 6.2 vs. 9.5 ± 5.1 mmHg, $p = 0.023$)
Mean pulmonary artery pressure		
Fukamachi *et al.*[39]	100	Mean pulmonary artery pressure was lower in those patients that following LVAD needed RVAD vs. those that did not (31 ± 5 vs. 38 ± 10 mmHg, $p = 0.015$)
Ochiai *et al.*[40]	245	Mean pulmonary artery pressure was lower in those patients that following LVAD needed RVAD vs. those that did not (33 ± 7 vs. 37 ± 9 mmHg, $p = 0.041$)
RVSWi		
Fukamachi *et al.*[39]	100	RVSWi was lower in those patients that following LVAD needed RVAD vs. those that did not (151 ± 75 vs. 368 ± 245 mmHg × ml/m^2, $p = 0.011$). If preoperative RVSWi was >300 mmHg × ml/m^2, none of the patients required RVAD support
Matthews *et al.*[42]	197	RVSWi ≤450 mmHg × ml/m^2 had an OR 2.32 (95% CI 1.24–4.32) for RV failure following LVAD implantation
Ochiai *et al.*[40]	245	RVSWi 285 ± 196 mmHg × ml/m^2
Kiernan *et al.*[30]	26	RVSWi was lower in those patients that following LVAD developed RV failure vs. those that did not (151 ± 75 vs. 368 ± 245 mmHg × ml/m^2, $p = 0.011$)
CVP/PCWP		
Morine *et al.*[43]	132	RAP/PCWP ratio was higher in those LVAD patients that revealed RV failure compared to those that did not (0.69 ± 0.19 vs. 0.45 ± 0.16, $p < 0.01$)
Nitta *et al.*[44]	70	Patients with preoperative CVP/PCWP >0.8 had an OR 15.0 (95% CI 3.8–59.6) for RVAD support post-LVAD implantation
Shiga *et al.*[45]		A preoperative CVP/PCWP ratio ≥0.5 was associated with an OR 11.39 (95% CI 1.164–111.4) for RVAD support post-LVAD implantation
PAPi		
Lim and Gustafsson[46]	85	In patients undergoing LVAD implantation, PAPi was lower in those that required RVAD compared to those that did not (1.7 ± 0.3 vs. 3.6 ± 0.3, $p < 0.005$)
Morine *et al.*[43]	132	PAPi was lower in patients with vs. without post-LVAD implantation RV failure (1.32 ± 0.46 vs. 2.77 ± 1.16, $p < 0.001$)
Raymer *et al.*[49]	216	The group of patients with LVAD that revealed severe RV failure postoperatively compared to those that did not, had significantly lower PAPi (2.47 ± 2.03 vs. 1.77 ± 1.22, $p = 0.001$)

CI, confidence interval; CVP, central venous pressure; LVAD, left ventricular assist device; OR, odds ratio; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RV, right ventricular; RVAD, right ventricular assist device; RVSWi, right ventricular stroke work index.

Table 1: PAC derived hemodynamic parameters for determining RV failure after LVAD placement

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#FR 1 – Kiosk 24 – Poster 3

Retrograde Surgical Placement of a Thoracic Epidural Catheter in a Patient Undergoing Combined Thoracic and Spinal Surgery

Presenting Author: Vanessa B. Davis, DO, Resident Physician, University of Washington

Introduction

Thoracic epidural analgesia (TEA) is the standard for managing postoperative pain after thoracotomy, but is rarely used in spinal surgery due to risk of infection and interference with neurological monitoring. High thoracic TEA (T1-T2) is avoided due to elevated risk of cardiovascular and respiratory complications. We present a novel case of retrograde thoracic epidural catheter (TEC) placement via direct surgical visualization of T2 laminectomy in a patient undergoing combined thoracic and spinal surgery.

Case Presentation

A 52-year-old man with a 9.5x9.6x13.5 cm left apical lung schwannoma suspected to arise from T2 nerve root (Fig. 1), presented with chronic pleural effusion and compressive left lung atelectasis. The patient underwent left extended posterolateral thoracotomy and T2 laminectomy with transection of left T2 nerve root. Preoperative TEC placement was discussed but deferred due to potential interference with the surgical field. Under general anesthesia, thoracotomy was performed at T4-T5 intercostal space, with chest tube placement at T5-T6. A posterior incision exposed C7-T2 for hemilaminectomy and visualization of the mass at T2 foramen. Due to extensive tumor invasion, complete resection was aborted. Before closure, a TEC was placed by the neurosurgeon under direct vision via T2 laminectomy and threaded caudally ~10cm to T6-T7. The catheter was tunneled and secured at the skin at 18cm (Fig. 2). The patient was extubated without complication. Postoperatively, sensory testing revealed predominate left-sided T4-T7 block. On POD 1, epidurogram confirmed catheter tip at T6-T7 with contrast spread from T3-T9 (Fig. 3). Sensory changes at T1-T2 were attributed to T2 nerve root transection. The patient reported tolerable pain that did not impede recovery, with incentive spirometry volumes of 2500 mL. Table 1 illustrates total postoperative pain regimen. The TEC infusion was increased over POD 1–2 and removed on POD 3, where he was then discharged home with oxycodone, APAP, and methocarbamol. At follow-up on POD 4 and two weeks later, he reported minimal pain, denied taking opioids due to side effects, and had no respiratory or neurological deficits. No complications were reported at 8-month follow-up.

Discussion

Thoracotomy is one of the most painful surgeries, often resulting in postoperative pulmonary complications and delayed recovery.[1] TEA offers superior analgesia, but high thoracic TEA is associated with phrenic nerve blockade and ventilatory depression. TEA is seldom used in spine surgery due to risk of infection and interference with neurological assessment.[2] Some advocate for intraoperative TEC placement under direct visualization to minimize these risks, which has been shown to reduce opioid use, improve pain control, and avoid compromising postoperative exam when using low-dose local anesthetic.[2-3] This case is, to our knowledge, the first report of retrograde TEC placement during combined thoracic and spinal surgery. The

catheter was safely advanced from T2 laminectomy site to T6-T7, providing effective, targeted analgesia while avoiding high thoracic blockade. This technique may be valuable in select patients undergoing complex thoracic and spinal surgery, as it offers a feasible, opioid-sparing alternative without increasing postoperative risks.

Images

	TEA	IV opioids	PO opioids	APAP	Ketamine	Adjuncts
Pre-op	-	-	-	-	-	-
Intra-op	bupivacaine 1/16% at 6ml/hr	Fentanyl 200mcg	-	1gm IV	0.1mg/kg/hr	-
Immediate post-op in PACU	bupivacaine 1/16% at 6-8ml/hr	Hydromorphone 1mg	Oxycodone 10mg	1gm PO	-	-
POD #1	bupivacaine 1/16% at 8ml/hr	-	Oxycodone 40mg	4gm PO	-	Methocarbamol 1000mg
POD #2	bupivacaine 1/16% at 12ml/hr	-	Oxycodone 20mg	4gm PO	-	Methocarbamol 500mg
POD #3	Discontinued	-	-	4gm PO	-	-
Discharge medications	-	-	-	-	-	-

Table 1: This table illustrates the total amount of pain medications the patient received in the perioperative period.

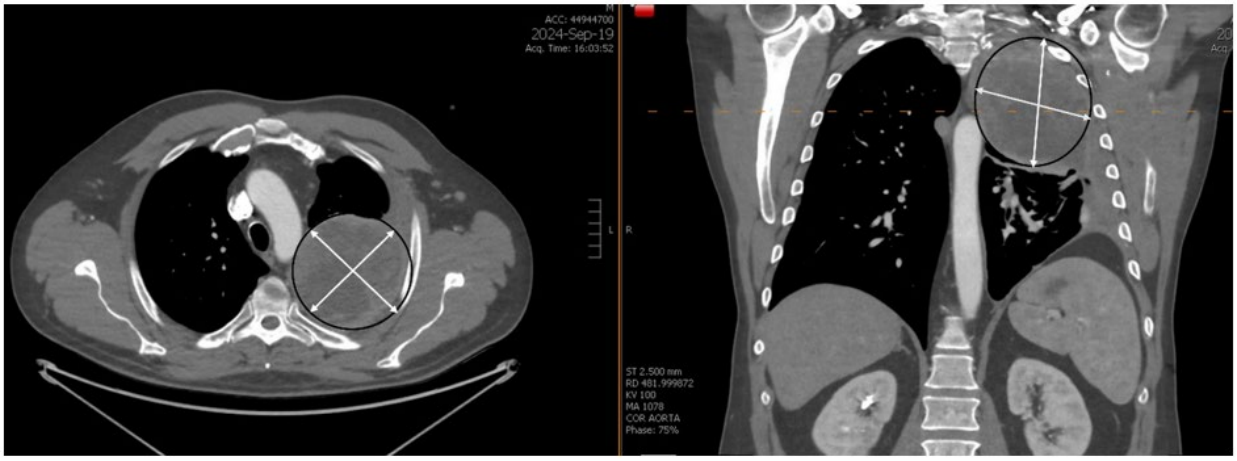


Figure 1: The location and size of left apical lung schwannoma. Black circle outlines the approximate border of the tumor with white arrows showing dimensions of the tumor (9.5cm x 9.6cm x 13.5cm).

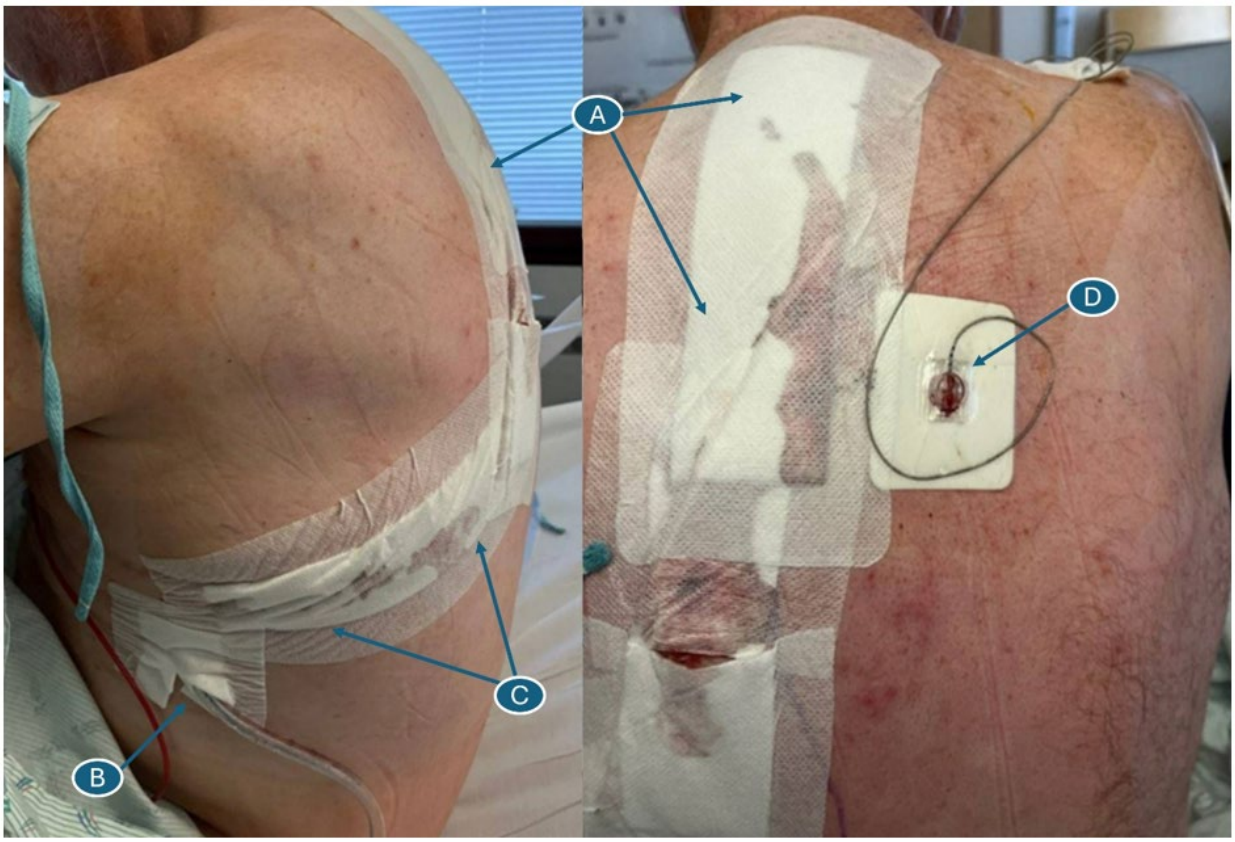


Figure 2: Surgical incisions of combined thoracic and spine surgery, and the exit site of thoracic epidural with sterile dressing. A) spine incision. B) chest tube placement. C) thoracotomy incision. D) epidural exit site.

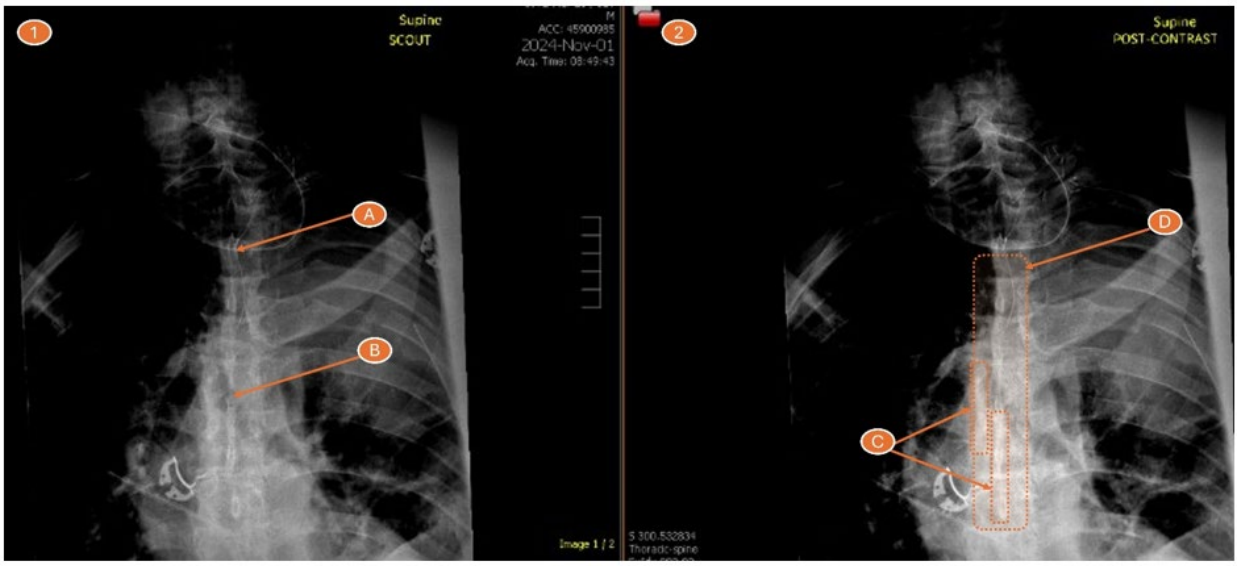


Figure 3: The postoperative epidurogram demonstrating pre- (1) and post- (2) contrast T-spine X-ray. A) epidural catheter insertion site (T1-T2). B) epidural catheter end site (T6-T7). C) concentrated areas of contrast. D) contour of contrast spread within spinal canal between T3-T9.



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#FR 1 – Kiosk 24 – Poster 4

A large right atrial appendage aneurysm: anesthetic management for a rare cardiac surgery

Presenting Author: David N. Lumelsky, MD, Resident, New York Presbyterian/Weill Cornell

Introduction

Right atrial appendage aneurysm (RAAA) is a rare condition with sporadic case reports[1]. While many RAAAs grow to "giant" size, most remain asymptomatic and are diagnosed incidentally[1]. Palpitations with finding of atrial tachyarrhythmia are the most common symptoms leading to diagnosis. While many RAAAs are isolated abnormalities, some may be associated with ASDs or pulmonary hypertension causing elevated RA pressures as an etiology for their formation2. Rare reports also demonstrate that large RAAAs may cause pulmonary complications from mass effect[3].

Case Presentation

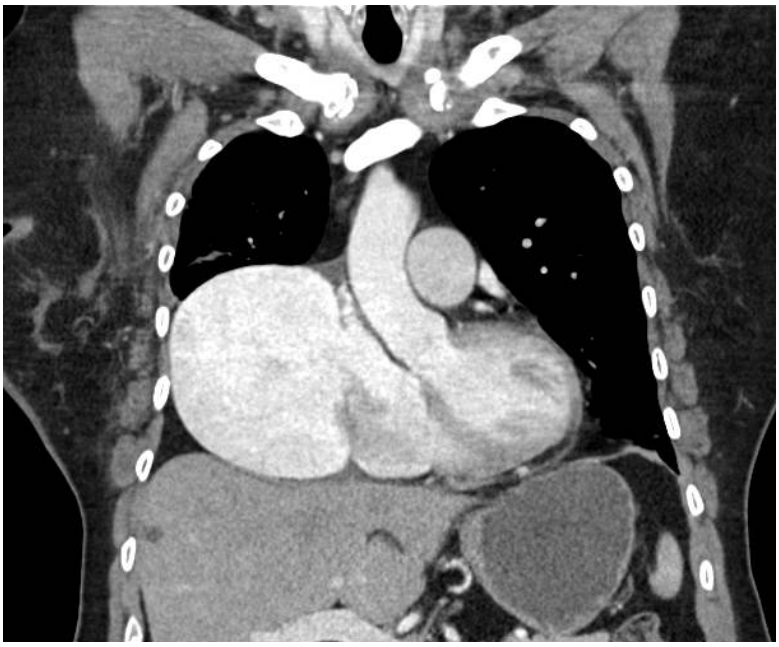
A 61-year-old female undergoing breast cancer staging was found to have a 13cm mass involving the right hemithorax on chest CT. Symptoms included fatigue and dyspnea on exertion. She had normal biventricular function and no valvular disease; R/LHC showed non-obstructive CAD with elevated RA and PA pressures. She was brought to the OR for RAAA resection in rate controlled atrial fibrillation. After an uncomplicated induction, TEE identified a grossly normal RA with an aneurysmal appendage contiguous with an 8.5x9.5x11.5 hypoechoic space with spontaneous echo contrast. A R IJ introducer and PAC were placed under TEE guidance, avoiding the RAAA. Additional IV access was obtained in a lower extremity with an 18G in the L saphenous. Upon entry into the pericardium, a large mass emanated from the lateral RA wall extending rightward into a large pericardial recess created by the aneurysm. Cannulation for CPB was achieved with aortic cannulation followed by femoral venous cannulation; the patient was brought onto full CPB support, followed by followed by SVC cannulation allowing RA isolation. A secundum ASD was identified intraoperatively, not previously seen on any imaging. The patient was separated from CPB without inotropes or vasopressors and was defibrillated once with an amiodarone bolus converting to NSR. Following separation from CPB, the patient was hypoxic, which continued on ICU arrival. Bronchoscopy and airway pressures were unremarkable; she was started on iNO with improvement and extubated to HFNC on POD1. AF recurred on POD3; she was weaned to NC by POD4 and discharged on POD8.

Discussion

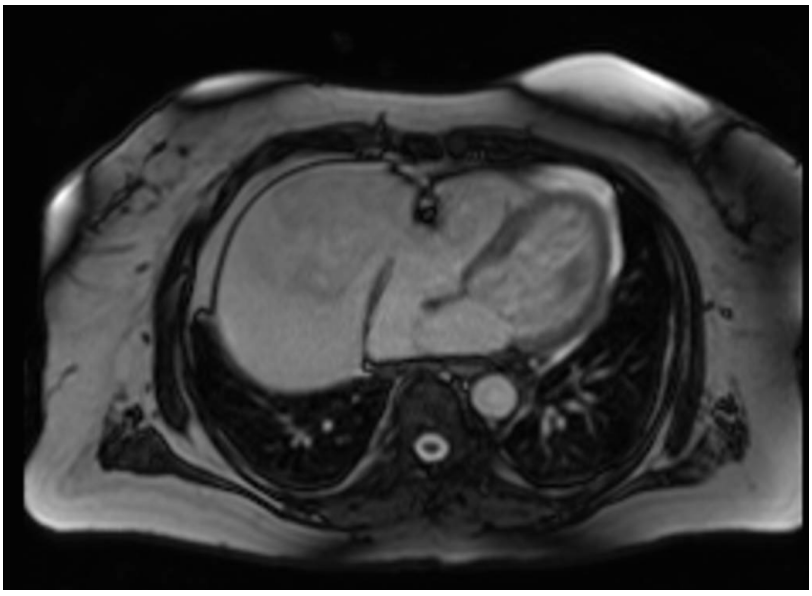
We report a symptomatic large RAAA in a patient with an ASD as a possible etiology for its development and long-standing AF in this setting. Careful central venous and pulmonary artery cannulation can be achieved only under live imaging guidance as the giant RAAA is exceedingly thin walled, and blind PAC advancement into the aneurysm may result in catastrophic rupture. The anesthesiologist should obtain appropriate lower extremity venous access to prepare for uncontrolled RA bleeding during dissection or for planned surgical clamping of the SVC. Similarly, peripheral cannulation strategies for CPB may be used to avoid disturbing the aneurysm, although the IVC cannula would need to be placed with great care, under vision. Large RAAAs should be recognized as functional mediastinal masses compressing thoracic structures. Our patient did

not have respiratory symptoms while supine, and we were able to perform a standard intubation, but a spontaneously ventilating induction should be considered. Additionally, hypoxia as in our patient and previous case reports[3], should be anticipated and treated by the anesthesiologist.

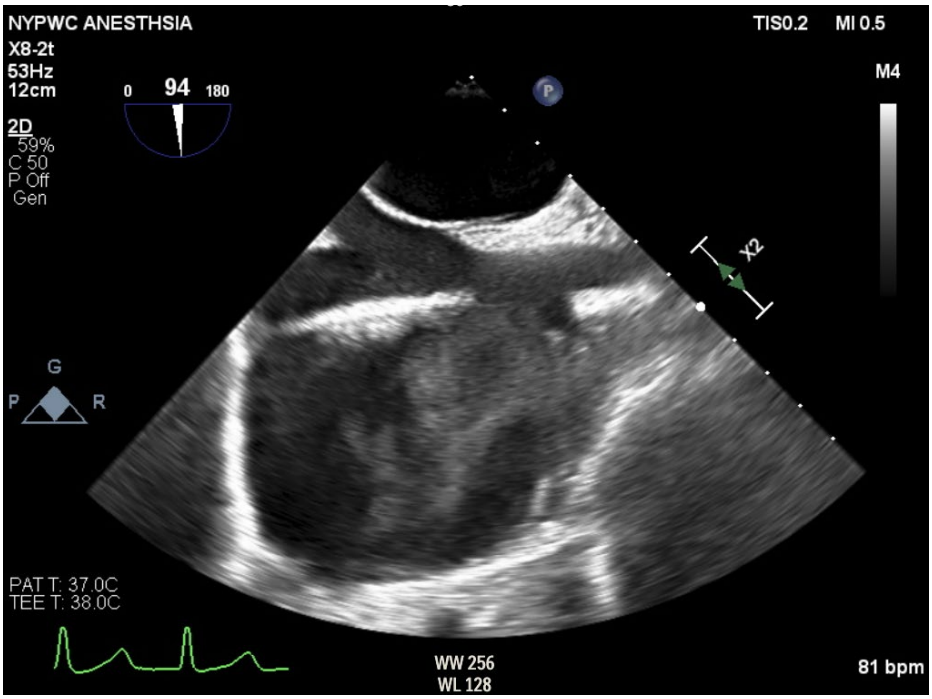
Images



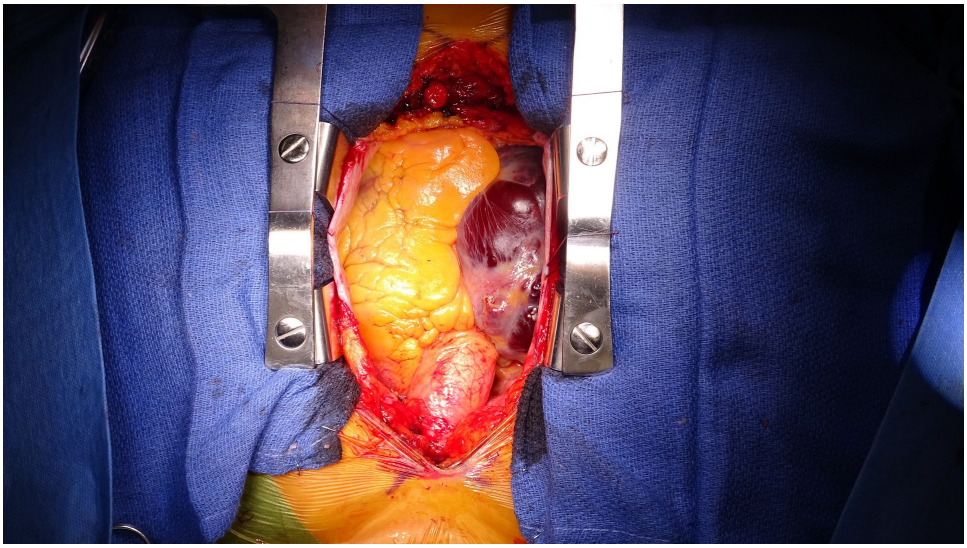
Coronal image of a CT Chest with IV contrast demonstrating the large mass in the right hemithorax consistent wtih right atrial appendage aneurysm.



Axial cardiac MRI image demonstratign the communication of the right atrial appendage aneurysm with the right atrium, also seen is compression of the middle and lower lobe of the right lung.



Intraoperative transesophageal echocardiography image demonstrating the right atrial appendage aneurysm with spontaneous echo contrast. In this image, the well delineated neck of the aneurysm can be seen, allowing TEE guided placement of the PAC.



Intraoperative photograph demonstrating the thin walled RAAA immediately following entry into the pericardium

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#FR 1 – Kiosk 24 – Poster 5

Severe Post-CPB Hypotension from Suspected Anaphylaxis: Importance of Rapid, Multimodal Intraoperative Evaluation

Presenting Author: Jason R. Garrett, DO, Cardiothoracic Anesthesiology Fellow, Indiana University School of Medicine

Introduction

Severe hypotension after cardiopulmonary bypass (CPB) presents a major diagnostic challenge with a broad differential, including ongoing bleeding, mechanical cardiac compression, myocardial stunning, coronary spasm, arrhythmias, mechanical obstruction, and drug-related reactions—most commonly transient protamine effects. Rapid identification of the underlying cause is essential to guide targeted treatment and prevent cardiovascular collapse. We present a case of profound post-CPB hypotension most consistent with an allergic drug reaction and highlight the value of multimodal intraoperative assessment in directing management.

Case Presentation

A 75-year-old man with coronary artery disease and severe aortic stenosis underwent elective aortic valve replacement and CABG. CPB and separation were uneventful, with normal biventricular function on transesophageal echocardiography (TEE) and minimal vasoactive support. Protamine (300 mg) was initiated to reverse heparin, followed shortly by transfusion of a single apheresis platelet unit for surgical oozing. Approximately six minutes after platelet transfusion began, he developed abrupt profound hypotension (MAP < 30 mmHg). He received high-dose epinephrine, norepinephrine, vasopressin, angiotensin II, fluid resuscitation, and intravenous methylprednisolone and diphenhydramine. No rash, urticaria, bronchospasm, hemolysis, or fever occurred. TEE demonstrated preserved biventricular systolic function without right ventricular dilation, and hyperdynamic ventricles consistent with distributive shock. Central venous pressure remained < 10 mmHg. Hemodynamics gradually improved with ongoing resuscitation. Serum tryptase later returned markedly elevated at 87.3 mcg/L. He recovered uneventfully and was extubated on postoperative day one.

Discussion

The combination of sudden severe hypotension, preserved ventricular function, low CVP, and exclusion of surgical causes strongly suggested a drug-related reaction rather than cardiogenic or obstructive shock. TEE rapidly ruled out new regional wall motion abnormalities, acute RV failure, and prosthetic valve issues, supporting a distributive process. Protamine reactions include IgE-mediated anaphylaxis, IgG/complement-mediated pulmonary vasoconstriction with RV failure, and nonimmune myocardial depression. IgE-mediated reactions can elevate serum tryptase. Conversely, hypotensive reactions to platelet transfusion are often bradykinin-mediated or IgE-mediated responses to donor plasma proteins and typically manifest as isolated vasodilatory shock within minutes of transfusion initiation. In this case, the absence of RV dilation, pulmonary hypertension, or rising airway pressures made complement-mediated protamine

pulmonary vasoconstriction unlikely. The markedly elevated tryptase, preserved ventricular function, low CVP, and close temporal association with platelet transfusion supported anaphylaxis as the most likely mechanism, though an IgE-mediated protamine reaction could not be fully excluded. This case reinforces the importance of maintaining a broad differential for post-CPB hypotension and demonstrates how rapid integration of TEE findings, filling pressures, and hemodynamic trends can expedite diagnosis and appropriate intervention.

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#FR 1 – Kiosk 24 – Poster 6

Management of Giant Right Coronary Artery Pseudoaneurysm Undergoing Coronary Artery Bypass Grafting

Presenting Author: Ankit D. Patel, DO, Resident Physician, Baystate Medical Center

Introduction

Coronary artery aneurysms are rare, with giant aneurysms (≥4 cm) representing a high-risk subset associated with thrombosis, rupture, distal embolization, and ischemia. They may arise from atherosclerosis, prior percutaneous coronary intervention, congenital defects, or inflammatory etiologies and often require surgical repair when large or symptomatic (1). For the anesthesiologist, these cases demand meticulous planning, precise hemodynamic control, and close coordination with the surgical team. Key considerations include maintaining myocardial perfusion, avoiding abrupt changes in preload and afterload, and anticipating complications such as aneurysm rupture or distal embolization during induction and initiation of cardiopulmonary bypass. Significant comorbidities—such as advanced CAD, prior stenting, and hepatic dysfunction—further heighten perioperative risk. This case describes the anesthetic management of a giant right coronary artery aneurysm requiring CABG and aneurysm resection after unsuccessful PCI.

Case Presentation

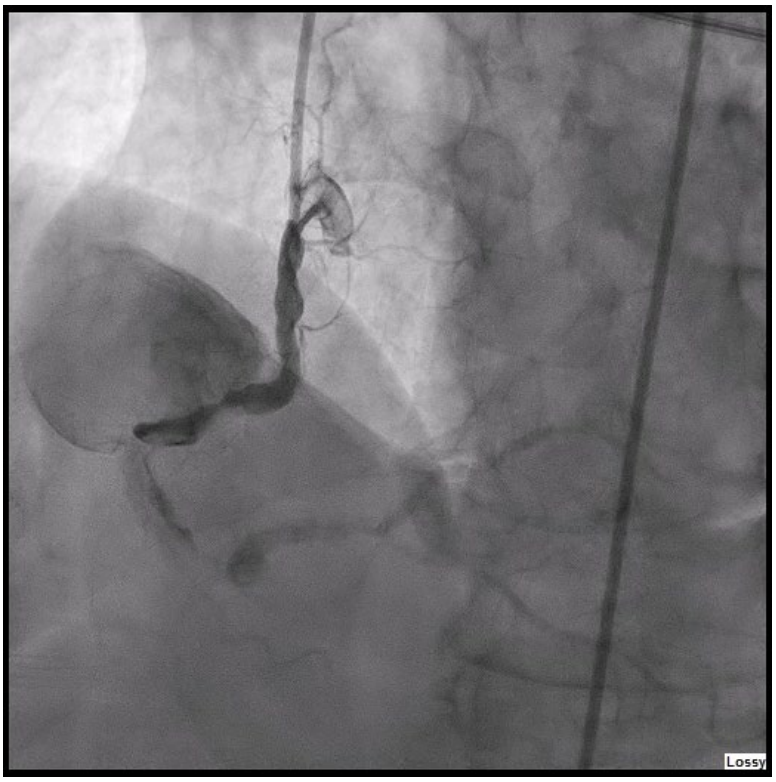
A 61-year-old male smoker with CAD status post PCI ×2 to the mid-RCA after STEMI, hypertension, hyperlipidemia, and alcoholic cirrhosis with esophageal varices underwent lung-cancer screening CT, which incidentally demonstrated an pseudoaneurysmal RCA between prior stents. Subsequent cardiac cath and coronary CT Angiography revealed a large 4.6-cm RCA pseudoaneurysm and chronic total LAD occlusion. TTE was performed 1/2025 but did not initially comment on the pseudoaneurysm, which was able to be visualized on an apical 4-chamber view upon retrospective review of the images. PCI was attempted but unsuccessful, and the patient proceeded to surgery and underwent uneventful CABG ×2 and aneurysm resection with ligation.

Discussion

Giant coronary aneurysms present substantial anesthetic challenges due to their risk of rupture, thrombosis, and dynamic changes in coronary perfusion. Induction is particularly vulnerable: hypotension may reduce perfusion to ischemic territories, whereas hypertension increases aneurysmal wall stress and rupture risk (2,3). Smooth hemodynamic transitions, strict blood-pressure control, and early readiness for CPB are essential. Anesthetic management focused on optimizing coronary perfusion, minimizing wall stress and ensuring preparedness for rapid CPB initiation. Intraoperative TEE allows continuous assessment of ventricular function, regional ischemia, thrombus burden and surgical manipulation of the aneurysm prior to bypass, guiding vasopressor and inotrope titration. Large aneurysms may distort coronary flow or compress adjacent structures, while invasive arterial monitoring—potentially with dual arterial lines—facilitates rapid detection of perfusion changes. This case proceeded without complication but highlights

how lesions like these can be frequently missed on TTE. Multiple ultrasounds prior to surgery did not make any mention of the aneurysm before it was noted on low dose CT scan. It is impossible to know exactly how this happened but was most likely due to overlying of the aneurysm in many views over the left atrium with poor windows that would make it difficult to characterize the lesion as an aneurysm without prior knowledge of it from other imaging modalities such as coronary angiography or CT scan.

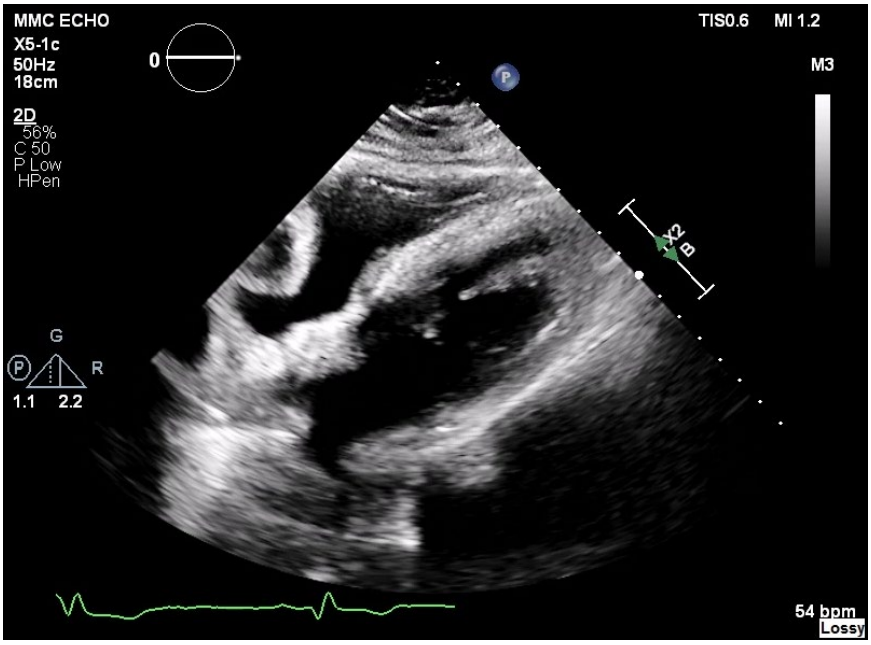
Images



Coronary angiography demonstrating contrast filling giant right coronary pseudoaneurysm with subsequent failed attempt at stenting



Selection of slides from CT coronary angiography with contrast showing right coronary artery aneurysm measuring 43 mm x 40 mm x 46 mm



Transthoracic echocardiogram in subcostal view showing giant right coronary artery aneurysm in the top left, distorting the right atrium



3D reconstruction of coronary vessels including pseudoaneurysm produced from CT heart/coronary with contrast

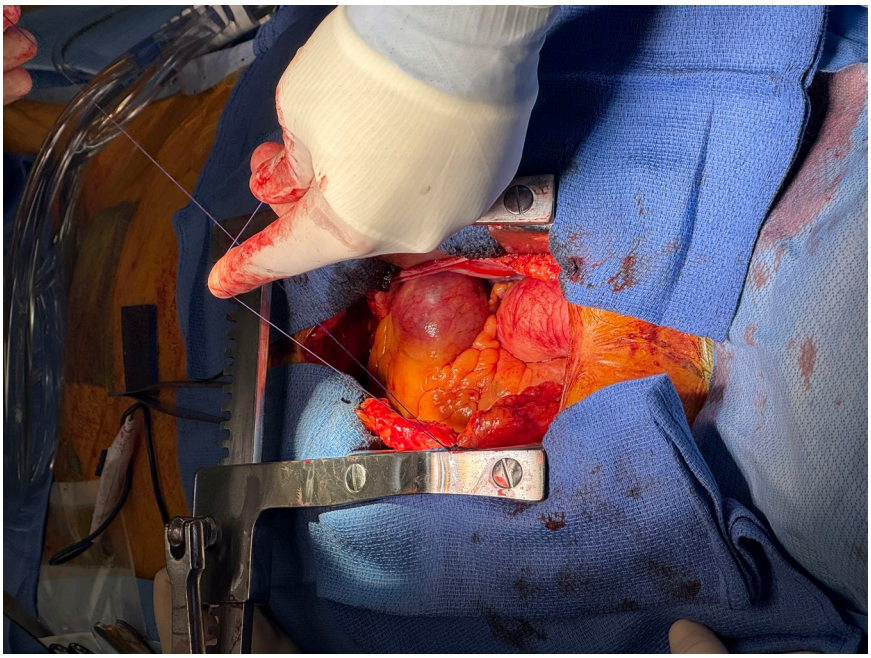


Image obtained during coronary artery bypass grafting showing giant right coronary artery aneurysm directly below surgeon's hand



Image obtained during coronary artery bypass grafting showing giant right coronary artery aneurysm after it was ligated and opened

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#FR 1 – Kiosk 25 – Poster 1

Minimally Invasive Off-Pump Coronary Artery Bypass Surgery in a Patient with an Immature Tracheostomy

Presenting Author: Benjamin R. Weingarten, MD, University of Toronto

Introduction

Minimally invasive cardiac surgery (MICS) has been shown to improve recovery following cardiac surgery and can be an advantageous approach where conventional sternotomy confers increased risk. We present a case of minimally invasive off-pump coronary artery bypass surgery (OPCAB) via mini-thoracotomy in a patient with an immature tracheostomy and highlight the relevant perioperative considerations.

Case Presentation

A 73-year-old male with a history of hypertension, dyslipidemia, and ischemic heart disease underwent hemi-glossectomy, extensive neck dissection and tracheostomy for squamous cell carcinoma of the tongue. On POD2 he developed chest pain with new inferior ST depressions and an elevated troponin. Coronary angiography demonstrated triple vessel disease involving the left main coronary artery and a transthoracic echocardiogram demonstrated a left ventricular ejection fraction of 44%. He subsequently underwent OPCAB via left mini thoracotomy. Access to the heart was facilitated by selective right lung ventilation with a bronchial blocker placed through the tracheostomy and positioned to occlude the left mainstem bronchus under fiberoptic guidance. Transesophageal echocardiography (TEE) was not utilized. His intraoperative course was uncomplicated. A left erector spinae plane (ESP) catheter was placed and produced satisfactory post-operative analgesia.

Discussion

This case highlights unique anesthesia considerations for cardiac surgical patients with recent glossectomy and fresh tracheostomy. Additionally, recent evidence regarding optimal analgesic strategies in MICS is reviewed. Tracheostomy is associated with an increased incidence of sternal wound infection (SWI) in patients with recent sternotomy (OR 2.8, CI 1.904.2)[1]. In contrast, the safety of conventional midline sternotomy in patients with pre-existing immature tracheostomy (< 1-2 weeks old) is unknown. The T modification of sternotomy is an alternative surgical approach in this setting. In our patient with recent glossectomy and flap reconstruction, we used an off-pump, mini-thoracotomy approach to reduce the risk of airway bleeding by reducing heparinization with a lower ACT target. This approach also reduced the risk of tracheostomy disruption or airway injury. In addition, to prevent airway trauma and bleeding, we deferred intraoperative transesophageal echocardiography and inserted a pulmonary artery catheter for invasive hemodynamic monitoring. Epiaortic ultrasound was available as a backup modality. Various lung isolation techniques have been described in patients with tracheostomies, however given our patients immature stoma and altered airway anatomy, a bronchial blocker was deemed safest for selective right lung ventilation[2]. Finally, we would like to highlight the benefits of regional anesthesia techniques in MICS. A recent RCT demonstrated improved quality of recovery and less rescue analgesia in patients who received

ESP catheters for MICS[3]. In our patient, an ESP catheter provided adequate analgesia as part of a multimodal analgesic approach. In summary, our case highlights important considerations for minimally invasive OPCAB in patients with tracheostomy.

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#FR 1 – Kiosk 25 – Poster 2

Anesthetic Management of Severe Polyvalvular Disease for Palliative, Staged Transcatheter Aortic and Mitral Interventions

Presenting Author: Kian Riley, MD, Anesthesiology Resident Physician, Brigham and Women's Hospital

Introduction

Patients with polyvalvular disease (PD) present anesthetic challenges requiring balance of conflicting goals for heart rate (HR), rhythm, preload, and afterload. These challenges intensify during structural heart (SH) procedures, where rapid hemodynamic shifts can significantly impact patients with multiple severe valve lesions.

Case Presentation

A 92-year-old woman presented with newly symptomatic PD underwent transcatheter aortic valve replacement (TAVR) as the first step in a staged approach to improve quality of life. Her history included atrial fibrillation with three cerebrovascular events, atrioventricular block now with a permanent pacemaker, and recent transthoracic echocardiography showing septal-predominant concentric hypertrophy, new rise in right ventricular pressure, severe aortic stenosis (AS), mitral stenosis (MS), tricuspid regurgitation (TR), and moderate-to-severe mitral regurgitation (MR). A multidisciplinary SH evaluation deemed her unsuitable for open cardiac surgery, with an STS- predicted operative mortality of 10% for isolated AVR. The SH team elected to treat her AS first with TAVR under monitored anesthesia. Given the hemodynamic fragility imposed by severe MS, anesthetic goals emphasized minimal sedation, limited pacing runs to 130 bpm, and maintenance of preload and afterload. An arterial line was placed and phenylephrine was administered before each pacing run with excellent effect. A 26-mm self-expandable Evolut FX valve was selected given need for restricted pacing runs and was successfully deployed. Resultant moderate paravalvular leak required an additional pacing run for balloon dilations. The leak improved to mild, and she recovered well with resolution of symptoms. Next steps include valve-in-MAC (mitral annular calcification) transcatheter MVR to address persistent MS and MR, with alcohol septal ablation and LAMPOON to protect the left ventricular outflow tract (LVOT).

Discussion

This case highlights the complexity of severe PD, where optimal management of one lesion may destabilize another. Severe AS requires sinus rhythm and adequate preload; hypotension or tachycardia may precipitate hemodynamic collapse. Severe MS complicates TAVR, as tachycardia or preload shifts from pacing or anesthesia shorten diastolic filling time, raise left atrial pressure, and risk pulmonary edema. Regurgitant lesions favor higher HR and low afterload, necessitating judicious fluid management. Prioritizing the lesion with greatest hemodynamic consequence—here, MS during TAVR—informed the strategy of minimal sedation, short pacing runs, and proactive vasopressor use. Treating AS first improved forward stroke volume and reduced global valvular impedance, creating a safer foundation for future mitral intervention. Persistent MS and MR with extensive MAC now require advanced transcatheter techniques.

MAC complicates anchoring and increases risk of neo-LVOT obstruction. Adjunctive LAMPOON (split of anterior mitral leaflet) and alcohol septal ablation (reduces septal thickness) maximize neo-LVOT for valve-in-MAC implantation. Lessons learned from her TAVR—particularly vasopressor responsiveness and tolerance of pacing—will guide anesthetic planning for this complex second-stage intervention, supporting the overarching goal of symptom palliation.

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#FR 1 – Kiosk 25 – Poster 3

Transcaval Transcatheter Aortic Valve Replacement: When the Plan is to Intentionally Poke a Hole in the Aorta

Presenting Author: Lauren Dwyer, MD, Anesthesiology Resident, University of Virginia

Introduction

Transcatheter aortic valve replacement (TAVR) requires large bore aortic access, typically through a transfemoral arterial approach. However, conditions including severe peripheral artery disease, small or calcified iliofemoral vessels, or prior aortic surgeries may render femoral arterial access unsafe or impossible. Transcaval TAVR achieves large bore access to the aorta via the femoral vein through the creation of an aorto-caval fistula through the retroperitoneum.[1,2] Patient safety and success of this technique relies on multidisciplinary planning and vigilant hemodynamic monitoring.

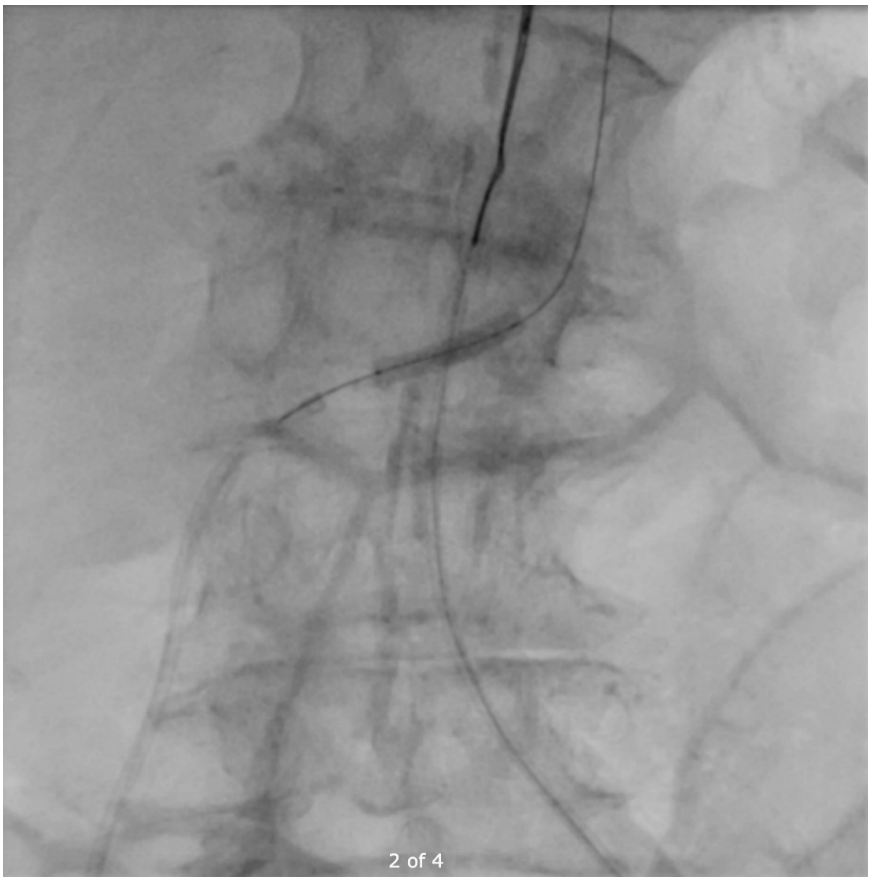
Case Presentation

A 76-year-old female presented for transcaval TAVR with a past medical history of severe aortic stenosis, HFrEF, history of Type A dissection status post hemi arch reconstruction in 2014, hypertension on metoprolol and telmisartan, CAD, chronic LBBB, severe COPD, PAD and chronic kidney disease. Due to her history of aortic dissection and PAD, this patient was not a candidate for transfemoral access. Pre-operative transthoracic echo (TTE) was significant for left ventricle ejection fraction of 60-65%, aortic valve mean gradient of 41mmHg and aortic valve area of 0.55cm2. The case was completed under general anesthesia with a pre-induction right radial arterial line. After discussion with the surgical team, the decision was made to not place a central line. For access, the patient had 18- and 16-gauge IVs in the upper extremities. The patient was maintained on 4 to 8 mcg/min of norepinephrine for a mean arterial pressure goal of greater than 75mmHg and was hemodynamically stable throughout the procedure. The surgeons obtained access in the bilateral femoral veins and left femoral artery. A snare from the left femoral artery was used as a target for the transcaval access and a cautery was used to puncture from the IVC to the aorta at L2/L3. After gaining access, a 4mmx20 mm balloon was used to dilate the track. The 14 fr delivery system was guided across the aorto/IVC track and the patient was rapid ventricular paced at 180 beats per minute while a 26mm Edwards Sapien valve was deployed. Post deployment TTE showed no paravalvular leak and 0 mmHg gradient. The valve delivery system was withdrawn and an 10mmx8mm occlusion device was placed to close the aortic access site. Final images showed a small fistulous connection flowing from the aorta to IVC, but no active extravasation from the aorta or IVC. The patient was extubated and transported to PACU off of norepinephrine. She briefly required a nicardipine infusion for hypertension in pacu. CTA obtained on POD1 showed no extravasation through the occlusion device. The patient's hemoglobin dropped from 13.3 pre-operatively to 9.7 at time of discharge and creatinine remained at her baseline throughout the admission. She was discharged on POD2. TTE one month post procedure showed LVEF of 55% and aortic valve mean gradient of 7mmHg.

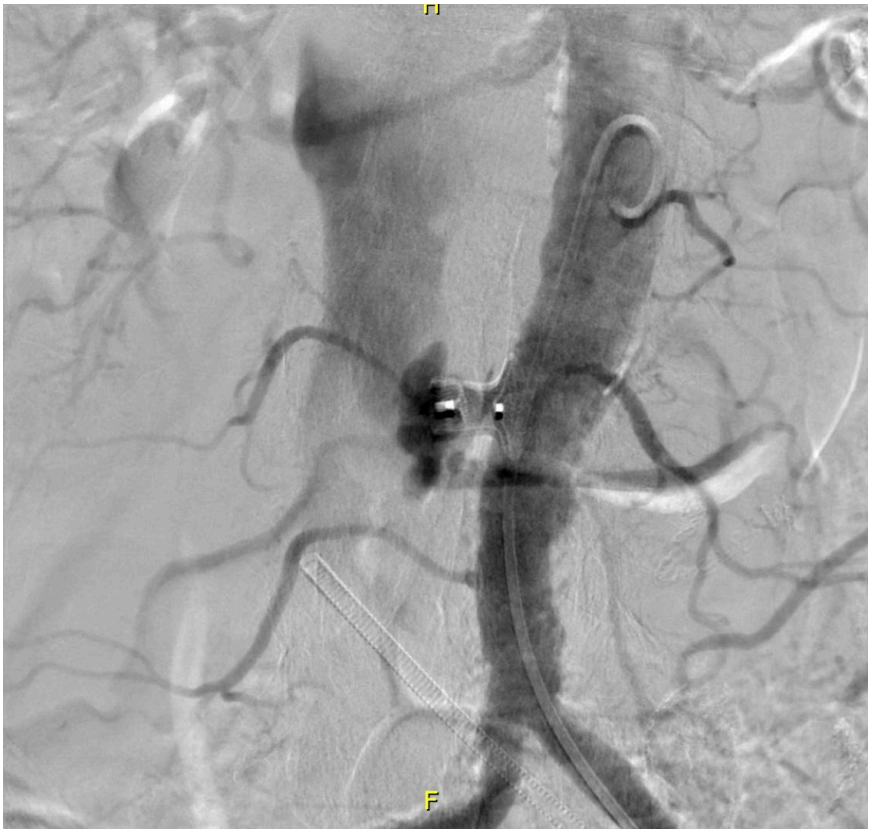
Discussion

This case shows that transcaval TAVR can be a reasonable option for patients with anatomy that makes the routine transfemoral access contraindicated. Open communication with the surgeons, large bore access and pre-induction arterial line were essential for safety in this non routine TAVR.

Images



Fluoroscopic image of the 4mmx20 mm balloon dilating the tract between the IVC and aorta.



Fluoroscopic image after placement of the 10mm x8mm occlusion device that shows a small fistulous connection between the aorta and IVC without active extravasation from the aorta.

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#FR 1 – Kiosk 25 – Poster 4

Post-Operative Diagnosis of Cryoglobulinemia Following Emergent CABG

Presenting Author: Bridget Oei, MD, Anesthesia Resident, University of Vermont

Introduction

This case report describes a patient with Waldenstrom's Macroglobulinemia discovered following a failed left heart catheterization and PCI with subsequent emergent three-vessel CABG. This patient's operative and post-operative recovery were complicated by his underlying hematologic disease.

Case Presentation

An 81 year old male presented with an NSTEMI and had undergone a staged PCI. Following PCI to the LCX he returned to the cath lab for treatment of LAD and RCA disease. During the procedure, the RCA stent became dislodged in the proximal coronary ostia. He was subsequently brought to the OR for emergent aortotomy, retrieval of the stent, and CABG 3 (LIMA-LAD, GSVG-DIAG, GSVG-PDA). Several intraoperative complications occurred. There was significant bleeding from the IJ central line site requiring suture placement for hemostasis. Later in the case, the surgical team noted a clear intraluminal gelatinous material that was expressed from the LIMA prior to anastomosis, and within the retrieved coronary stent. As the patient separated from cardiopulmonary bypass, he was coagulopathic requiring large volume blood resuscitation. He was hemodynamically unstable despite normal biventricular function requiring multiple inotropic and vasopressor agents. The patient was brought to the SICU intubated, in mixed shock. Workup included cold agglutinins, smear, and TEG, and Hematology was consulted. The TEG sample clotted as a clear gelatinous globule, which resolved upon warming the sample, raising suspicion for the presence of a cryoglobulin. The patient was started on steroids and actively warmed. By postoperative day 1, the patient was extubated and off pressors. The following night, he was noted to have aphasia and left facial droop prompting a stroke code. CT angiograph showed small embolic strokes in the subcortical LMCA territory. Within 24 hours, his neurologic exam returned to baseline, and symptoms did not recur. He was transferred to the floor on postoperative day 2, and discharged in stable condition on postoperative day 7. As an outpatient, a bone marrow biopsy confirmed a diagnosis of Waldenstrom's Macroglobulinemia. He has been following with Hematology though he has not qualified for active treatment.

Discussion

This case highlights a rare finding of Waldenstrom's Macroglobulinemia which has a reported incidence of 0.36 per 100,000 in the US[1]. Typical signs include anemia, thrombocytopenia, and hyperviscosity syndrome. Some patients can have concomitant cold agglutinins from IgM cryoglobulins, which can result in end organ ischemia from poorly perfused microcapillary beds[2]. One case report by D'Alosio et al. presents some management strategies for this disease in the setting of cardiac surgery: preoperative plasmapheresis to lower IgM levels, avoidance of hypothermia below 32°C, the administration of warm cardioplegia, use of mannitol to manage hyperviscosity during bypass, tolerance of a slight acidemia to decrease hemoglobin's oxygen affinity and maximize oxygen delivery, and a higher cardiac index target to increase oxygen delivery

and minimize blood viscosity[2]. Ultimately, this case was medically challenging due to the lack of a known preoperative hematologic diagnosis. Despite the potential complications, this patient recovered well from his emergent surgery.

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#FR 1 – Kiosk 25 – Poster 5

Complications of Granulomatosis with Polyangiitis Requiring Extra-Anatomic Aortic Bypass and Neo-Bronchus Creation from Aortic Autograft

Presenting Author: Jaime L. McGrath, DO, Fellow, University of Michigan

Introduction

Granulomatosis with polyangiitis (GPA) is a rare immune-mediated disease characterized by airway granulomas, necrotizing vasculitis, and glomerulonephritis. Untreated, GPA is rapidly fatal, though patients without renal involvement have an estimated 60–70% 10-year survival rate.[1] We present a case of GPA complicated by an aorto-bronchial fistula requiring a staged, multidisciplinary surgical approach and posing significant anesthetic challenges.

Case Presentation

A 30-year-old female with GPA, esophageal strictures, and previous right middle and lower lobectomy for a ruptured pseudoaneurysm between the right pulmonary artery and right mainstem bronchus presented with frank hemoptysis. Imaging demonstrated a left sided aorto-bronchial fistula at the level of the proximal descending aorta prompting emergent transfemoral endovascular aortic repair (TEVAR). Four weeks later, bronchoscopy revealed erosion of the endograft into the left main bronchus (LMB). A multistage surgical plan was formulated: Stage 1, left subclavian artery to left common carotid artery bypass, extra-anatomic bypass from the ascending aorta to the supraceliac aorta with aortic ligation in zone 2 and zone 4 and proximal left common carotid artery ligation for complete exclusion of the stentgraft, and Stage 2, a left thoracotomy for explant of the stentgraft (Figures 1-3), and reconstruction of LMB. Transesophageal echocardiography (TEE) was not possible given severe esophageal stricture; thus a pulmonary artery catheter (PAC) was placed to guide hemodynamic management. Between Stages 1 and 2, she developed severe pneumonia. Due to her likely inability to tolerate lung isolation for the planned airway reconstruction, veno-venous ECMO was initiated. Following stentgraft explant, a gap existed between the proximal and distal left mainstem bronchus. A cook catheter was advanced through the ETT, sewn distally to a sterile Blake drain, then advanced superiorly by the surgical team bridging the bronchial gap (Figure 5). The patient was then extubated in exchange for the Blake drain exiting the airway. The neo-bronchus was then reinforced with remaining aorta (Figures 6-7) and externally reinforced with a latissimus flap before thoracotomy closure. Stage 3 of the operative course required stenting of the neo-bronchus with Blake removal and tracheostomy placement which proceeded without complication (Figures 8-9). The patient had a prolonged recovery in the ICU but was ultimately discharged to home.

Discussion

This case underscores the complexity of extra-anatomic aortic bypass combined with tracheobronchial reconstruction, particularly in the absence of transesophageal echocardiography and in a patient with limited remaining lung parenchyma. Avoiding further airway injury, managing multiple points of potential catastrophic hemorrhage, and supporting oxygenation during loss of functional airway continuity were

central anesthetic challenges. While not novel, the successful utilization of a warm aortic autograft for airway reconstruction is rare and highlights a promising direction in regenerative medicine.[2,3] This case demonstrates how coordinated, multidisciplinary planning enabled survival from a condition previously considered uniformly fatal.

Images

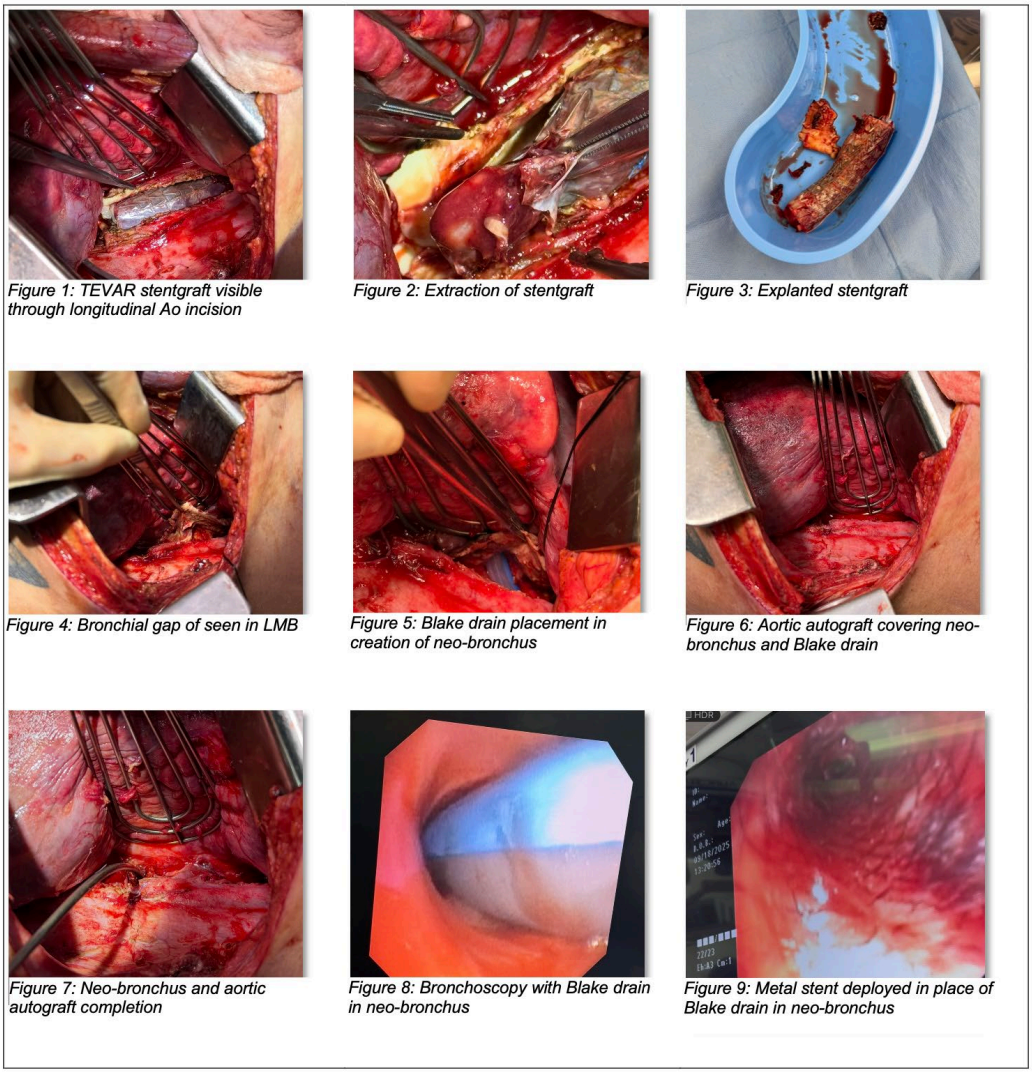


Figure 1: TEVAR stentgraft visible through longitudinal Ao incision

Figure 2: Extraction of stentgraft

Figure 3: Explanted stentgraft

Figure 4: Bronchial gap of seen in LMB

Figure 5: Blake drain placement in creation of neo-bronchus

Figure 6: Aortic autograft covering neo-bronchus and Blake drain

Figure 7: Neo-bronchus and aortic autograft completion

Figure 8: Bronchoscopy with Blake drain in neo-bronchus

Figure 9: Metal stent deployed in place of Blake drain in neo-bronchus

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#FR 1 – Kiosk 25 – Poster 6

Transesophageal Echocardiography Guided Pericardial Release for Left Ventricular Assist Device Cannula Malposition in a Postpartum Patient with Chemotherapy-Induced Cardiomyopathy

Presenting Author: Pavit S. Deol, MD, Cardiothoracic Anesthesia Fellow, University of California San Diego

Introduction

Peripartum and chemotherapy-related cardiomyopathies present unique anesthetic and surgical challenges. As cardiovascular complications of cancer therapy increase, there is growing overlap between cardio-oncology and advanced heart failure management. We describe the use of transesophageal echocardiography (TEE) to identify and correct left ventricular assist device (LVAD) inflow cannula malposition, improving flow and hemodynamics.

Case Presentation

A 36-year-old woman, eight weeks postpartum and recently treated with anthracycline-based chemotherapy for breast cancer, presented in acute biventricular failure and cardiogenic shock. She underwent advanced mechanical circulatory support evaluation, was stabilized with inotropes and an intra-aortic balloon pump, then scheduled for LVAD implantation. After induction of general anesthesia, TEE confirmed severely depressed biventricular function. A HeartMate 3 (HM3) was implanted via sternotomy under TEE guidance, and the patient was separated from cardiopulmonary bypass. Upon sternal closure, hemodynamics deteriorated with escalating vasoactive support and inadequate LVAD flows. TEE demonstrated insufficient left ventricular (LV) unloading with worsening right ventricular function. The inflow cannula was directed toward the mid-inferolateral wall with turbulent flow on color Doppler (Figure 1). Surgical manipulation failed to improve flow or hemodynamics. A lateral pericardial release was then performed to relieve external constraint and facilitate cannula redirection. TEE confirmed immediate realignment toward the mitral valve coaptation zone with restored laminar flow (Figure 2). Left ventricular unloading improved, right ventricular function stabilized, and the sternotomy was closed on stable support. The patient was transported to the ICU and extubated on postoperative day four. She was discharged home on day twenty-four with stable LVAD flows and adequate right ventricular function.

Discussion

Intraoperative TEE is essential for assessing inflow cannula alignment, ventricular function, and guiding corrective maneuvers. Cannula malposition, particularly deviation from the apical-midline axis, impairs unloading, promotes turbulence, and increases adverse outcomes. Narrower coronal angles of HM3 inflow cannula correlate with improved LV unloading and lower rates of heart failure readmission, underscoring the clinical importance of precise alignment.[1] Pericardial release can relieve external constraint on the LV apex, redirect the cannula toward the mitral inflow axis, and improve device flows.[2] In this case, TEE-guided lateral pericardial release restored optimal alignment, enhanced LV unloading, and stabilized right ventricular performance. Comprehensive TEE evaluation is vital not only for diagnosis of malposition, but also for directing intraoperative correction. Peripartum or anthracycline-induced cardiomyopathy may distort

ventricular geometry, warranting vigilance for cannula malalignment, and reinforcing TEE as an indispensable tool in advanced mechanical circulatory support.

Images

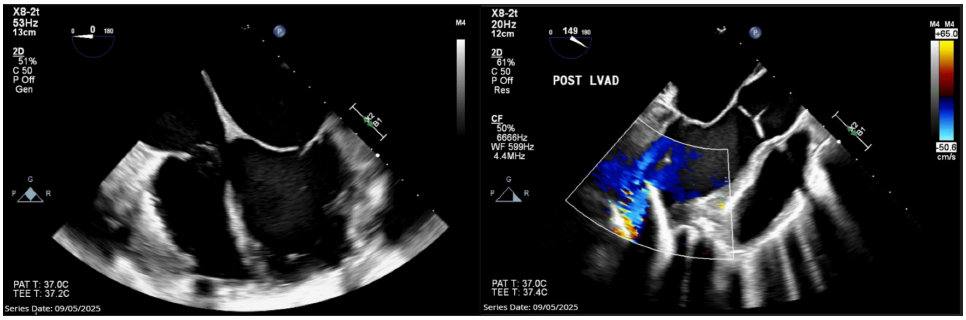


Figure 1: Transesophageal echocardiography at the mid-esophageal position demonstrating biventricular failure (left). After LVAD implantation and closure of the sternum, the cannula can be seen directed towards the mid- inferolateral wall with flow acceleration (right).

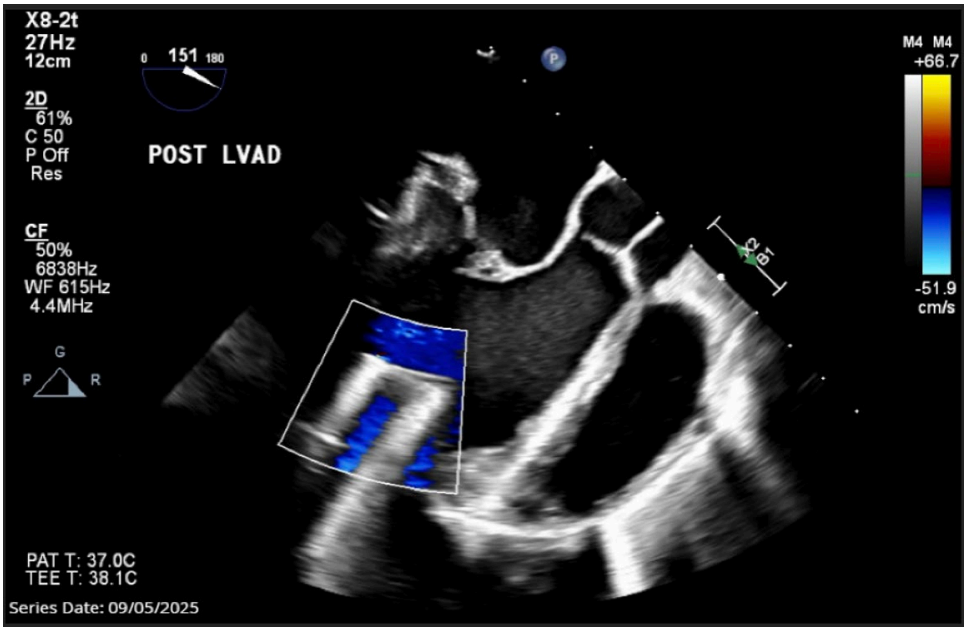


Figure 2: The mid-esophageal long axis TEE view after lateral pericardial release reveals improved angulation and flow of the LVAD inflow cannula.

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#FR 1 – Kiosk 25 – Poster 7

Perioperative Management and Echocardiography for the Cone Reconstruction

Presenting Author: Jungbin A. Choi, MD, Clinical Fellow in Adult Cardiothoracic Anesthesiology, Brigham & Women's Hospital/Massachusetts General Brigham

Introduction

The Ebstein's Anomaly is a congenital cardiac malformation where the tricuspid valve leaflets fail to delaminate from the right ventricular wall, resulting in varying degrees of 'apicalization' of the tricuspid valve and 'atrialization' of the right ventricle (RV). This results in a spectrum of cardiac pathology, including tricuspid regurgitation (TR), right atrial (RA) enlargement, arrhythmia, biventricular failure, and interatrial shunting [1]. Clinical presentation ranges from neonatal cyanotic heart failure to asymptomatic adults. A novel repair for the Ebstein anomaly is the Cone Reconstruction, which involves remobilization and reattachment of the affected leaflets to the true anatomic annulus[2]. To highlight key findings and perioperative considerations, we present the echocardiography and management of an adult with Ebstein's Anomaly undergoing a Cone Reconstruction.

Case Presentation

A 64-year-old man with T2DM, HTN, and Ebstein's Anomaly (diagnosed at age 6, previously asymptomatic) presents with dyspnea and pre-syncope limiting his daily activities. He underwent an extensive preoperative workup. EKG showed a RBBB, but no atrial arrhythmia. He had a TTE and a cardiac MRI which confirmed Ebstein Anomaly, biventricular dysfunction, and severe TR. Coronary angiography was normal, and labs were unremarkable. The patient underwent a cardioprotective induction of general anesthesia with epinephrine for inotropic support. Pre-induction arterial and post-induction central venous access were obtained. TEE confirmed Ebstein anomaly, a moderately dilated RA, and a moderately dilated tricuspid valve annulus apicalized into the right ventricle with severe TR. The RV was dilated with depressed systolic function. The LV had reduced systolic function and a small cavity without hypertrophy (Figure 1). There was no significant valvulopathy otherwise. He tolerated the procedure well. Post CPB, RA pressure was < 15, and a Glenn shunt was not created. TEE showed TR, reduced function of the newly ventricularized portions of the RV wall, and preserved function of the native RV. Post-CPB LV filling improved with low-normal systolic function.

Discussion

Ebstein's Anomaly patients make up < 1% of congenital heart disease but can present for both cardiac and non-cardiac surgery, and anesthesiologists must be prepared to manage their unique physiology[3] (Figure 2). Successful care involves case-specific optimization. For the Cone procedure, TEE is critical to assess the new tricuspid valve apparatus or annular ring and biventricular function before CPB wean. Biventricular failure can present due to parallel pathologic processes. Atrialization of the native RV leads to RA and RV dilatation and systolic dysfunction. The LV can eventually fail due to the Bernheim effect, where RV dilatation prevents filling of the LV, resulting in decreased function. Preoperative diuresis and

pharmacologic inotropic support may be indicated. Due to RA dilatation, atrial arrhythmias are common and must be addressed. Post repair, the new RV may be strained by additional preload, and meticulous fluid management with a CVP goal < 15 is often utilized. High CVP may necessitate creation of a Glenn shunt or ASD to offload the RV.

Images

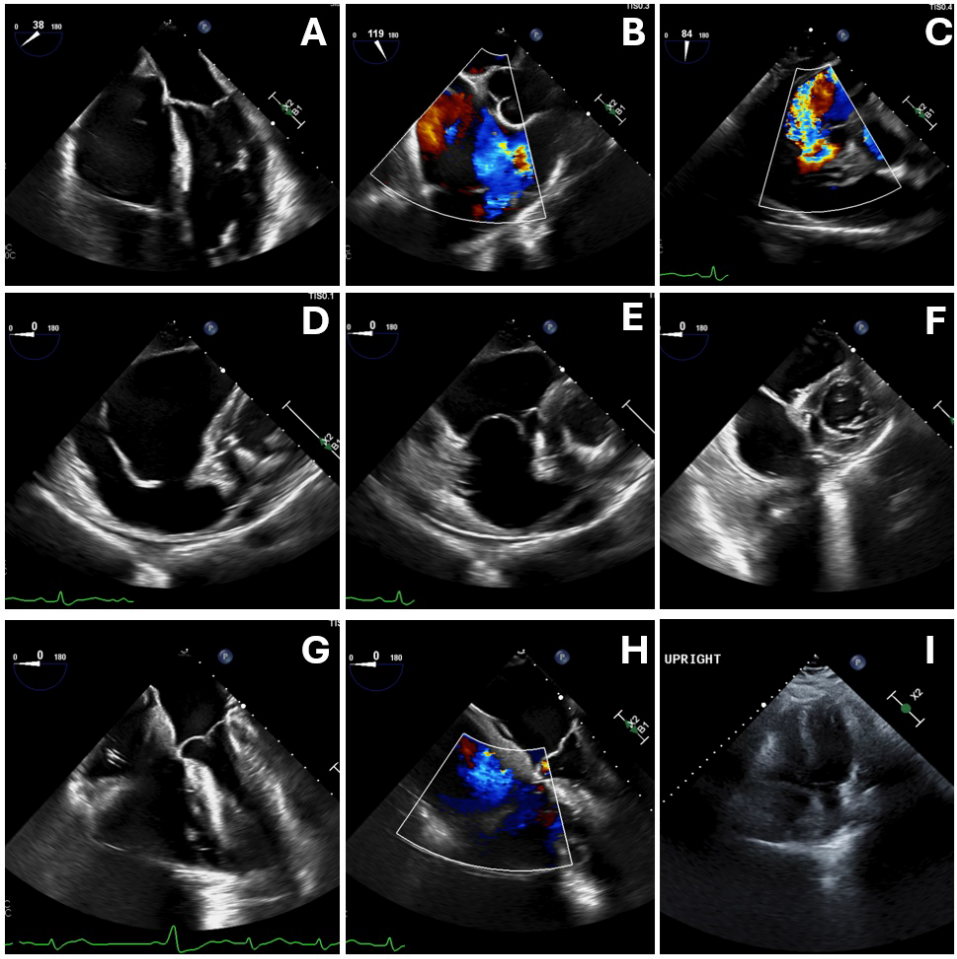


Figure 1: Intraoperative Images of the Cone Reconstruction.
A. Mid-esophageal 4-Chamber view showing Ebstein Anomaly. **B.C.** Showing Severe Tricuspid Regurgitation. **D.E.** Diastolic (D) and Systolic (E) morphology of the Ventricularized Tricuspid Valve, showing significant prolapse and flail during systole. **F.** Transgastric Short-axis view showing small cavity Left Ventricle (2.9cm). **G.** Mid-esophageal 4-chamber view post-procedure showing reconstruction of tricuspid valve at the native annular position. **H.** Significant improvement of tricuspid regurgitation. **I.** One-week post-operative transthoracic apical 4-chamber view showing normal tricuspid valve position.

Figure 1: Intraoperative Images of the Cone Reconstruction.

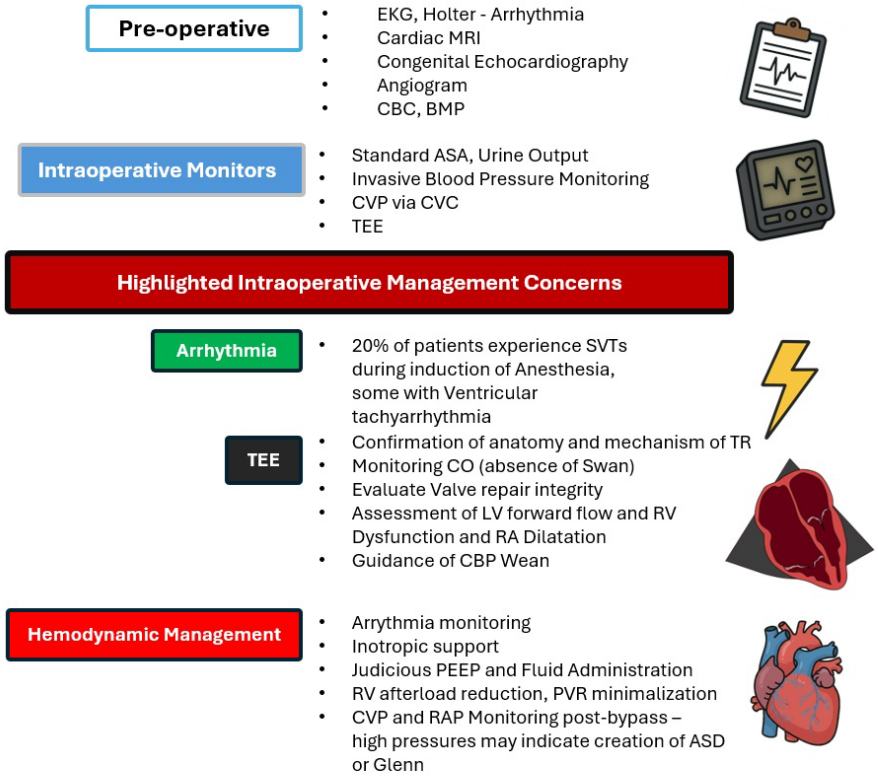


Figure 2: Perioperative Management Highlights of the Cone Reconstruction

Figure 2: Perioperative Management Highlights of the Cone Reconstruction

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#FR 1 – Kiosk 26 – Poster 1

Left Ventricular Pseudoaneurysm After Repeat Mitral Valve Replacement and Suspected AV Groove Separation

Presenting Author: Maciej Czarnecki, MD PhD, Fellow, University of Washington

Introduction

Ventricular wall pseudoaneurysm is a rare complication of myocardial infarction or iatrogenic injury. The propensity for decompensation, either through progression to overt rupture or cardiac tamponade, requires prompt surgical intervention and careful anesthetic management. Here, we discuss a case of suspected AV groove separation after mitral valve replacement leading to ventricular lateral wall pseudoaneurysm.

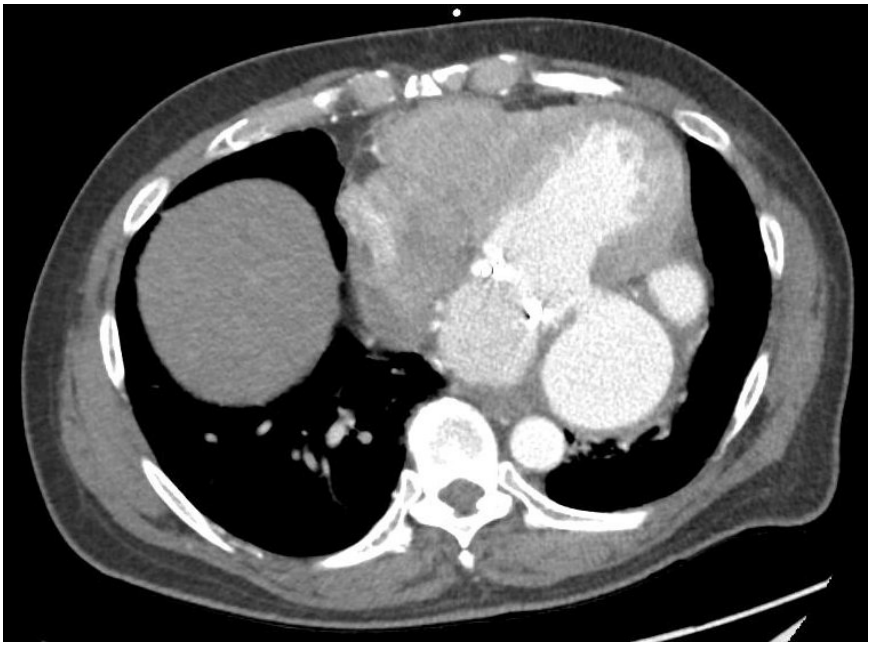
Case Presentation

We present a case of a 61-year-old man with a history of two previous mitral valve replacements, most recently in May 2025, who was admitted with an asymptomatic 14 cm left ventricular pseudoaneurysm. A routine post-operative echo demonstrated bidirectional blood flow between the left ventricle and the posterior pericardial space. Follow up imaging confirmed the suspected diagnosis of ventricular pseudoaneurysm, which on review of prior imaging may have been present post-MVR. The patient was subsequently brought to the OR where he underwent patch repair of the pseudoaneurysm, mitral valve explant, and bioprosthetic replacement. The tissue surrounding the posterior defect was noted to be “quite thin,” concerning for prior contained AV groove separation. On post-bypass TEE, there was concern for trace residual leak, which was ultimately determined to be a vascular structure. His post-surgical course was uncomplicated, and on follow-up imaging, the pseudoaneurysm is noted to be thrombosed with no residual flow.

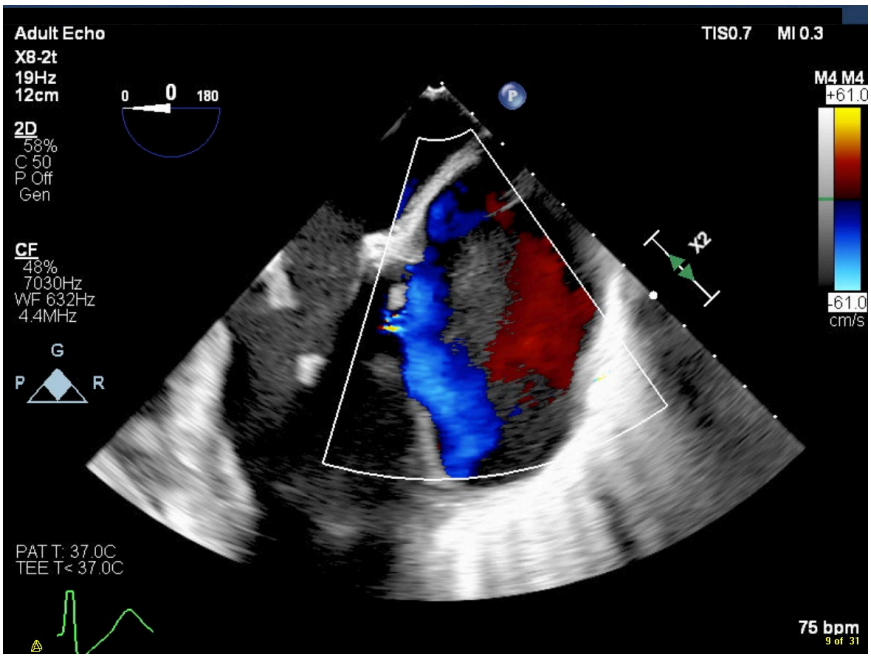
Discussion

Pseudoaneurysm is a rare complication of cardiac injury, estimated to occur in no more than 0.01% to 0.05% patients with myocardial infarction[1]. While ventricular wall rupture is a well reported complication of mitral valve replacement, the exact incidence of pseudoaneurysm in the setting of suspected AV groove dissociation is unknown, with the available literature limited to primarily isolated case reports and historical case series[2,3]. Patients may present with a variety of symptoms ranging from asymptomatic to non-specific cardiac symptoms, or emergently in obstructive shock. Pseudoaneurysm should be managed urgently given the risk of progression to rupture, which is highest during surgical manipulation and changes in intrathoracic pressure. In our case, our patient was likely a delayed presentation after the MVR and remained asymptomatic. Transesophageal echocardiography can be used to delineate between pseudoaneurysm and related conditions, with hallmark findings including presence of bidirectional flow between ventricle and pseudoaneurysm, posterior/lateral wall location, and systolic filling of the pseudoaneurysm sac.

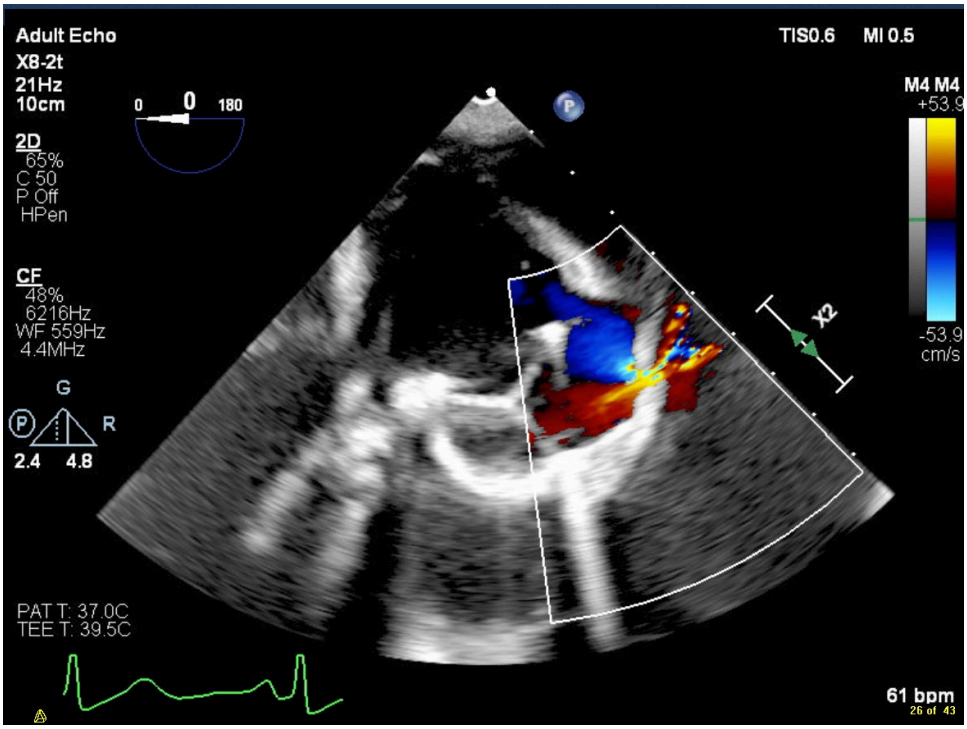
Images



Axial CT angiogram at the level of the bioprosthetic mitral valve demonstrating pseudoaneurysm sac in continuity with left ventricular cavity.



TEE Mid-esophageal 4 chamber view focused on left ventricle and pseudoaneurysm sac. Color doppler demonstrates flow across the aneurysmal neck.



TEE Transgastic short axis at the level of the bioprosthetic mitral valve with pseudoaneurysm at lateral aspect of left ventricle. Color doppler demonstrates systolic flow into pseudoaneurysm.

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#FR 1 – Kiosk 26 – Poster 2

Postoperative Diabetes Insipidus Masked by Vasopressin Infusion in a 76-Year-Old Female Following Deep Hypothermic Circulatory Arrest for Aortic Arch Repair

Presenting Author: Ashesh Trivedi, MD, Mount Sinai Hospital

Introduction

Postoperative diabetes insipidus (DI) is an important but uncommon complication following aortic arch repair. It typically results from transient cerebral ischemia or hypothalamic-pituitary axis disruption caused by cardiopulmonary bypass, deep hypothermic circulatory arrest, or other surgical stressors. DI manifests as excessive urine output and polydipsia due to impaired secretion or renal responsiveness to antidiuretic hormone (ADH). Anesthesiologists and intensivists play a pivotal role in its early recognition and management, as delayed diagnosis can rapidly lead to severe hypernatremia and neurologic morbidity. Vigilant monitoring of fluid balance, urine output, urine specific gravity, and serum sodium/osmolality is essential, particularly in high-risk patients undergoing complex aortic surgery.

Case Presentation

A 76-year-old woman with a family history of aortic dissection presented with acute dyspnea and was found to have an extensive Stanford type A intramural hematoma involving zones 0–5. She underwent urgent ascending aortic valve and total arch replacement with deep hypothermic circulatory arrest with selective antegrade cerebral perfusion. Following initial hemodynamic stabilization and weaning of vasopressin, refractory hypernatremia (peak sodium 173 mmol/L) with polyuria developed. Laboratory evaluation revealed serum osmolality 372 mOsm/kg, urine osmolality 199 mOsm/kg, and urine sodium 8 mmol/L, confirming central diabetes insipidus. Diagnosis was initially delayed because continuous vasopressin infusion for hemodynamic support masked the polyuria and urinary indices typical of diabetes insipidus. After nephrology consultation, desmopressin was initiated and resulted in normalization of serum sodium and resolution of polyuria. The patient ultimately recovered and was discharged on postoperative day 18.

Discussion

Postoperative central diabetes insipidus (DI) is a rare but serious complication after aortic arch surgery, particularly when deep hypothermic circulatory arrest techniques are involved. While receiving vasopressin for hemodynamic support, the patient maintained normal serum sodium and urine output, masking the typical manifestations of DI. Following vasopressin weaning, refractory hypernatremia (peak sodium 173 mmol/L) and polyuria (>300 mL/h) was unmasked. Confirmatory laboratories showed serum osmolality 372 mOsm/kg, urine osmolality 199 mOsm/kg, and urine sodium 8 mmol/L. Desmopressin therapy was initiated after nephrology consultation, resulting in normalization of sodium and resolution of polyuria. This case illustrates a critical diagnostic pitfall: continuous vasopressin infusion for postoperative vasoplegia can fully compensate for deficient endogenous antidiuretic hormone (ADH) secretion, concealing central DI until the infusion is reduced. The presumed mechanism involves transient hypothalamic-pituitary dysfunction induced by cerebral hypoperfusion or ischemia during prolonged CPB and circulatory arrest. Clinicians

managing complex aortic surgery patients must maintain a high index of suspicion for central DI despite vasopressin therapy. Serial monitoring of serum sodium, urine volume, and osmolality—both during and after vasopressor weaning—is essential to ensure timely diagnosis and prevent life-threatening hypernatremia.

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#FR 1 – Kiosk 26 – Poster 3

New Left Anterior Descending Territory Ischemia Post-bypass After Type A Dissection Repair-Diagnosis and Management

Presenting Author: Dimitrios Kampouris, MD, Resident Physician, University of Florida

Introduction

Myocardial ischemia is a known complication in 15% of patients presenting for Type A dissection repair due to coronary involvement.3 New-onset ischemia after repair, presenting as regional wall motion abnormality (RWMA) on trans-esophageal echocardiography (TEE) after cardiopulmonary bypass (CPB) poses a diagnostic and therapeutic challenge with many potential etiologies. We report a case of acute intra-operative ischemia post Type A dissection repair and subsequent management.

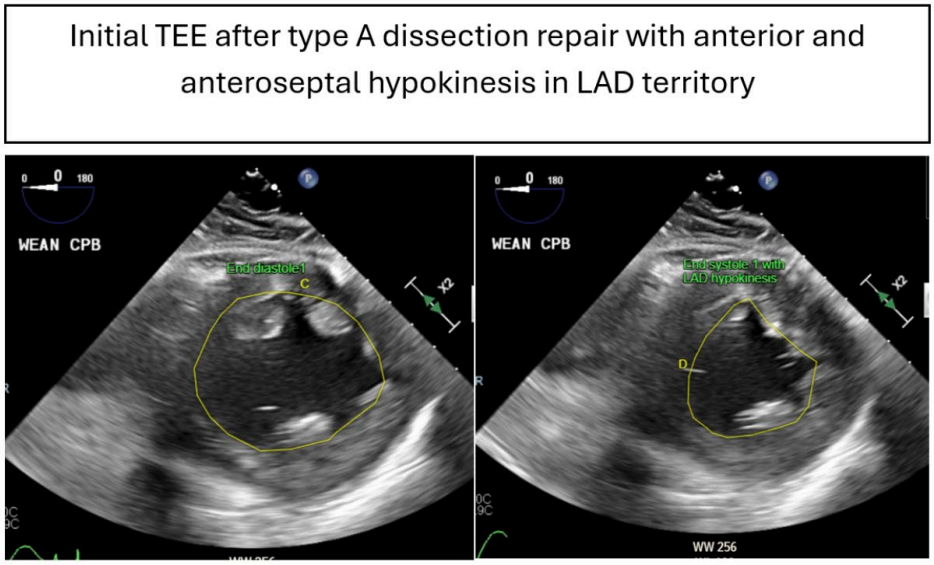
Case Presentation

A 74-year-old man with atrial fibrillation (on apixaban), hypertension, and type 2 diabetes presented for emergent Type A aortic dissection repair after experiencing chest pain. Pre-procedure TEE showed normal biventricular function, no RWMA, dissection flap originating at the root with moderate aortic regurgitation. He underwent aortic root replacement with composite valve graft, coronary re-implantation, ascending and hemiarch replacement. After cross clamp removal, TEE showed severe anteroseptal and anterior hypokinesis. Color flow was seen into both coronaries with no intra-cardiac air. Adequate re-perfusion time was ensured, and mechanical coronary button issues were excluded. While able to separate from CPB with high dose inotropes, isolated left anterior descending (LAD) territory ischemia persisted on TEE suggesting possible acute occlusion. Some coronary artery disease (CAD) noted on preoperative CT scan supported this. CPB was reinitiated to bypass LAD with a saphenous vein graft. TEE post-procedure showed marked improvement in function with resolution of RWMA. He was discharged on POD14 without complications.

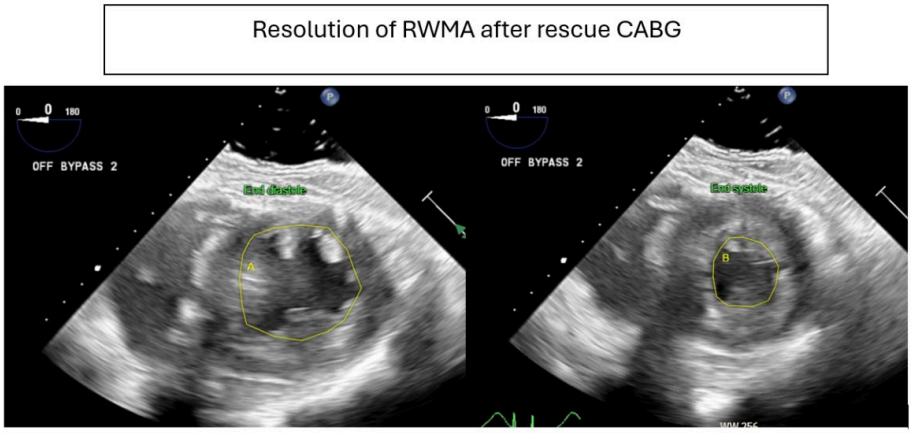
Discussion

Myocardial ischemia resulting in RWMA after aortic root surgery may result from air emboli, mechanical issues with re-implanted coronaries, unidentified coronary dissection, myocardial stunning or acute occlusion from underlying CAD. TEE is crucial in evaluation to identify involved coronary territory and potential etiology. In this case, TEE helped narrow down to LAD territory, excluding air emboli and identifying good flow into coronary buttons. Retrograde dissection and mechanical obstruction were further excluded by direct surgical observations. Isolated LAD hypokinesis with preserved function in remaining areas further excluded other causes, narrowing differential to acute occlusion from pre-existing CAD1,2 Patient's age and coronary disease noted on CT scan supported this diagnosis. While rescue CABG and percutaneous coronary intervention have both been described in literature, rescue CABG in our case resulted in successful treatment avoiding transport of unstable patient to cath lab and need for high dose antiplatelets in the peri-operative period. This case demonstrates a step-wise troubleshooting approach considering the specific operative procedure, echocardiographic evaluation and patient specific risk-factors in successful diagnosis and management of new RWMA after dissection repair.

Images



Initial TEE after type A dissection repair with anterior and anteroseptal hypokinesis in LAD territory



Resolution of RWMA after rescue CABG

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#FR 1 – Kiosk 26 – Poster 4

Off-Pump Coronary Artery Bypass Grafting using Bivalirudin in a Patient with Alpha-Gal Syndrome

Presenting Author: Steven M. Dotzler, MD, PhD, Anesthesiology Resident, Mayo Clinic (Rochester, MN)

Introduction

Alpha-Gal syndrome (AGS), associated with "Lone Star Tick" (*Amblyomma americanum*) bites is an IgE-mediated hypersensitivity reaction to galactose-alpha-1,3-galactose, a carbohydrate found in non-primate mammalian products. AGS predisposes to hives, angioedema, and anaphylaxis on exposures. Implications of AGS on anesthetic management in cardiac surgery are significant, as many perioperative medications including unfractionated heparin are animal-derived and may provoke life-threatening reactions. Few studies exist for AGS in cardiac surgery; most data come from case reports or series.[1] Coronary artery bypass grafting (CABG) typically employs cardiopulmonary bypass (CPB) with heparin for anticoagulation. We present a case highlighting perioperative challenges for a patient with AGS requiring revascularization of severe left main ostial coronary artery disease (CAD), mandating an alternative anticoagulation strategy and surgical approach.

Case Presentation

A 67-year-old male with known AGS, heart failure, and prior drug-eluting stent to proximal left anterior descending artery (LAD) who presented with angioedema and chest pain was found to have severe stenosis of the left main coronary ostium. Surgical consultation determined CABG as the optimal intervention, however AGS precluded heparin due to anaphylaxis risk. After multidisciplinary evaluation with immunology, anesthesia, and cardiothoracic surgery the decision was to perform off-pump CABG using bivalirudin for anticoagulation. Intraoperative management focused on avoidance of multiple common medications that may contain AGS trigger agents (Table 1, data adapted from Leder et al.)[2]— including propofol, hydromorphone, and heparin— premedication with hydrocortisone and diphenhydramine, hemodynamic stability, vigilance for ischemia, and careful titration of bivalirudin. Activated clotting time (ACT) was monitored to ensure adequate anticoagulation without CPB support. The patient underwent successful anastomoses of the left internal mammary artery to LAD and right internal mammary artery to ramus intermedius without hemodynamic compromise, allergic reaction, nor thrombotic complication, and was extubated at case end.

Discussion

This case underscores the complexity AGS imposes on cardiac anesthesia. Alternative strategies for heparin use in AGS have been described where some patients undergo heparin desensitization while others may tolerate heparin with minimal reaction (Nourian et al.).[3] In our case, due to severe left main ostial CAD, we opted for strict heparin avoidance, using bivalirudin and an off-pump CABG strategy to minimize risk of triggering AGS where any reaction precipitating hypotension may be life-threatening. Bivalirudin is synthetic and non-animal derived, offering a viable substitute. Though bivalirudin has predictable pharmacokinetics and short half-life, it lacks a reversal agent which can increase difficulty of managing surgical bleeding. As

awareness of AGS increases, anesthesiologists must anticipate medication restrictions and develop plans tailored to cardiac patients. This case highlights how individualized anticoagulation and off-pump techniques can safely enable revascularization in high-risk patients with AGS while mitigating anaphylaxis risk.

Images

Medication	Formulation	Safety	Potentially Concerning Ingredient
Propofol	IV	Possibly Unsafe	Glycerol
Acetaminophen	Oral	Possibly Unsafe	Gelatin, glycerin, lactose monohydrate, magnesium stearate, stearic acid
Hydromorphone*	IV	Possibly Unsafe	Lactic acid (unspecified form), sodium lactate
Clevidipine	IV	Possibly Unsafe	Glycerin, oleic acid
Milrinone	IV	Possibly Unsafe	Lactic acid
Vasopressin*	IV	Possibly Unsafe	Lactic acid
Enoxaparin	IV, SC	Possibly Unsafe	Potential porcine derivation
Heparin	IV, SC	Possibly Unsafe	Potential porcine derivation
Apixaban	Oral	Possibly Unsafe	Anhydrous lactose, magnesium stearate
Rivaroxaban	Oral	Possibly Unsafe	Lactose monohydrate
Warfarin	Oral	Possibly Unsafe	Lactose monohydrate, magnesium stearate
Aspirin	Oral	Possibly Unsafe	Magnesium stearate
Clopidogrel*	Oral	Possibly Unsafe	Lactose monohydrate
Prasugrel*	Oral	Possibly Unsafe	Lactose monohydrate
Ticagrelor	Oral	Possibly Unsafe	Magnesium stearate

Table 1. Common perioperative medications in cardiac surgery that may be unsafe in Alpha-Gal Syndrome (AGS) due to possible formulation with non-primate mammalian products. *Some formulations may be free from AGS potential trigger ingredients. Intravenous (IV), subcutaneous (SC). Data adapted from Leder et al.(2)

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#FR 1 – Kiosk 26 – Poster 5

Incidental Left Atrial Appendage Mass Altering Surgical Strategy: Transesophageal Echocardiography-Guided Decision-Making During Lung Transplantation

Presenting Author: Andrea Gomez Sanchez, MD, Resident Physician, University of Pittsburgh Medical Center

Introduction

Unexpected intracardiac findings during lung transplantation can significantly alter the operative course and require rapid multidisciplinary coordination. Transesophageal echocardiography (TEE) is critical for detecting high-risk intracardiac pathology and guiding management when surgical exposure is limited. We report the perioperative management of a patient undergoing left single-lung transplantation in whom TEE revealed an incidental, mobile left atrial appendage (LAA) mass on the Coumadin ridge, prompting a change from the standard VA ECMO strategy to full peripheral cardiopulmonary bypass on a beating heart with mass excision before graft implantation.

Case Presentation

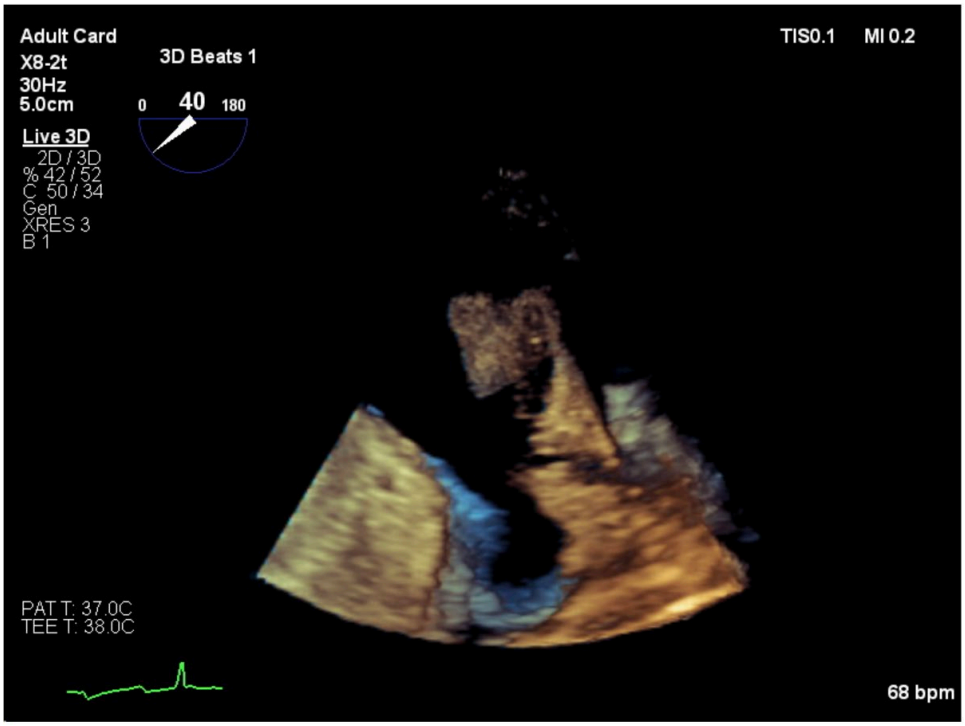
A 72-year-old woman with end-stage idiopathic pulmonary fibrosis and chronic respiratory failure presented for left lung transplantation. Intraoperative TEE revealed a previously undetected 2 × 2 cm irregular, mobile mass attached to the Coumadin ridge near the LAA and pulmonary vein orifices. The anesthesiology and surgical team determined that the mass posed a significant embolic risk during pulmonary vein manipulation. Although VA-ECMO is typically used, the mass's location near the planned atrial cuff anastomosis and the high stroke risk prompted conversion to full peripheral cardiopulmonary bypass on a beating heart with tumor excision prior to graft implantation. Right femoral arterial and venous cannulation were used to initiate bypass. Under continuous TEE guidance, the LAA was opened, revealing a soft, mobile mass consistent with imaging; it was excised and sent to pathology. The friable LAA was repaired with pledgeted sutures, and hemostasis was confirmed. After irrigation and hilar preparation, donor lung implantation proceeded. Bronchial and atrial cuff anastomoses were technically challenging due to deep hilar anatomy, mediastinal shift, and size mismatch but were completed successfully. Following de-airing and reperfusion, the graft demonstrated satisfactory compliance, perfusion, and no anastomotic leak. The patient weaned from bypass without difficulty, requiring brief vasopressor support, IV epoprostenol, and one unit of packed red blood cells. Final TEE confirmed good biventricular function, intact anastomoses, and no residual LAA defect. She was transported to the cardiothoracic ICU in stable condition.

Discussion

This case highlights the critical role of comprehensive TEE during lung transplantation, enabling early detection of an incidental mobile LAA mass with embolic potential and guiding intraoperative decision-making. Because the mass was located on the Coumadin ridge near the intended atrial cuff anastomosis, the surgical team appropriately modified their standard approach, from VA-ECMO to full peripheral CPB, to minimize embolic risk and allow safe resection prior to lung implantation. Beating-heart CPB facilitated tumor removal when limited exposure precluded aortic cross-clamping or antegrade cardioplegia. This case

also underscores the importance of multidisciplinary coordination when managing concurrent complex pathology, including pulmonary hypertension, friable atrial tissue, and technically challenging hilar anatomy. Early recognition and decisive perioperative management enabled successful transplantation with favorable graft function and hemodynamic stability.

Images



Three-dimensional transesophageal echocardiography showing a mobile mass attached to the left atrial appendage on the Coumadin ridge, identified intraoperatively prior to lung transplantation.

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#FR 1 – Kiosk 26 – Poster 6

Cyanide Toxicity from Sodium Nitroprusside During LVAD Surgery

Presenting Author: Jeremiah Fowler, DO, Cardiac Fellow, University of Wisconsin School of Medicine and Public Health

Introduction

Cyanide toxicity is an uncommon intraoperative complication. Although most cases follow ingestion or inhalation, sodium nitroprusside (SNP) is a recognized iatrogenic source. Because presentation is nonspecific, diagnosis relies on clinical suspicion. We describe a case of SNP-induced cyanide toxicity identified and treated during surgery.

Case Presentation

A 48-year-old woman with cardiomyopathy, diabetes, and OSA was admitted with decompensated heart failure and scheduled for HeartMate 3 LVAD implantation. In the ICU a PA catheter was placed. SNP was started on hospital day 9 at 0.8 mcg/kg/min for afterload reduction and titrated to 9.15 mcg/kg/min over eight days. Daily VBGs and CMPs showed normal pH and no anion gap. The night before surgery she became agitated and hypoxic, requiring 2 L nasal cannula oxygen. In the OR, after induction of general anesthesia, she appeared cyanotic despite 100% FiO_2. She developed hypoxia with declining cerebral oximetry and increased filling pressures. ABG revealed of pH 7.32 and lactate of 2.8 mmol/L, consistent with metabolic acidosis. After other causes were excluded, cyanide toxicity was suspected. SNP was stopped before induction due to anticipated vasodilation. Hydroxocobalamin 5 g IV was given, resulting in improved oxygenation and hemodynamics. After stabilization, we discussed whether to proceed with LVAD implantation given concern that hemolysis on bypass could increase cyanide release. However, given her worsening heart failure, the benefit of definitive mechanical support outweighed the risk. Surgery proceeded. During chest entry she had two stable VT episodes treated with cardioversion. Toxicology confirmed hydroxocobalamin as appropriate therapy and recommended continued monitoring without further treatment. A cyanide level drawn intraoperatively later returned positive. Cardiopulmonary bypass was initiated, and the HM3 device was successfully implanted. She separated from bypass on inhaled nitric oxide, milrinone, epinephrine, norepinephrine, and vasopressin. She was transferred to the ICU and extubated on POD 2.

Discussion

Cyanide toxicity is rare and variable in presentation. By inhibiting oxidative phosphorylation, cyanide forces anaerobic metabolism, resulting in lactic acidosis and impaired oxygen utilization. Diagnosis is clinical, as cyanide levels may take days to return. SNP-related toxicity is more likely with doses above 2 mcg/kg/min or prolonged infusion1. Symptoms include headache, confusion, arrhythmia, and cardiovascular collapse. Laboratory findings can show lactic acidosis and elevated venous O_2 saturation. In this patient, daily venous studies were normal, and lactate was not checked until intraoperatively, when it first became elevated. SVO_2 was normal in the ICU but may have been falsely elevated due to impaired cellular oxygen use. Early

recognition, cessation of SNP, and prompt hydroxocobalamin administration were essential to treatment. Hydroxocobalamin is first-line therapy; sodium nitrite and sodium thiosulfate are alternatives but may cause hypotension or methemoglobinemia. Effective interdisciplinary communication supported rapid diagnosis and treatment, allowing safe continuation of surgery. Post-operatively she recovered in the ICU, was extubated on POD 2, and discharged home on POD 24.

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#FR 1 – Kiosk 26 – Poster 7

Inferior Vena Cava Thrombectomy for Leiomyoma

Presenting Author: Benjamin Brunell, MD, PharmD, Cardiac Fellow, UT Health Houston

Introduction

Tumor thrombus extension into the inferior vena cava represents a complex clinical entity typically associated with renal cell carcinoma and other retroperitoneal malignancies. Surgical management requires meticulous preoperative evaluation, precise characterization of thrombus level and extent, and multidisciplinary coordination. Despite advances, IVC tumor thrombectomy remains technically demanding, with substantial variability in surgical strategy depending on tumor level, venous wall invasion, and the presence of metastatic disease.[1] Optimal patient selection, risk mitigation, and long-term oncologic benefit continue to be subjects of debate. Here is presented an IVC tumor thrombectomy for an advanced leiomyoma.

Case Presentation

The patient is a 36-year-old woman with a history of bilateral pulmonary emboli and nephrolithiasis who presented for planned open uterine myomectomy for vaginal bleeding. Her post-operative course was complicated by intrabdominal hemorrhage. CT angiography performed for identifying bleeding source was additionally remarkable for the discovery of a suprahepatic IVC thrombus from leiomyomatosis. This emergent procedure was successful, and her recovery remained uncomplicated. Gynecologic Oncology was consulted and recommended hysterectomy with oophorectomy following recovery from these initial procedures. Following additional workup, the patient was scheduled for a combined hysterectomy and IVC thrombectomy for the suprahepatic thrombus. Upon passing of an echo probe, the thrombus extension was seen to the cavoatrial junction. Consequently, the venous inflow required clamping, which the patient was unable to tolerate, and so venovenous ECMO was employed. Following resection, she was decannulated in the OR and transferred to the ICU, where she was in stable condition. Her recovery was uncomplicated and she was discharged on postoperative day five.

Discussion

This case illustrates an extensive IVC tumor thrombectomy requiring broad multidisciplinary collaboration and intensive operative management. Of note, most evidence details renal cell carcinoma as the thrombus etiology, with leiomyoma a more rare occurrence. Given the extent of tumor burden and areas affected, general surgery was brought in to help mobilize the liver and IVC. With regard to workup, the incidental finding from angiography showing suprarenal extension necessitated vascular surgery for venotomy and resection. At minimum IVC control can be utilized with less extensive tumor burden, however, the procedure may also require cardiopulmonary bypass if more extensive.[2] This case became additionally complex with the extension to the atriocaval junction and resultant instability with clamping. VV-ECMO allowed this operative issue to proceed with relative hemodynamic stability. Broad cooperation and well-resourced institutions are imperative for proper care and resection of this pathology.



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FELLOW AND RESIDENT ABSTRACTS

Poster Session 2

Saturday, April 25, 2026 | 8:45 AM – 9:45 AM

#FR 2 – Kiosk 1 – Poster 1

The Case for Warm Cardioplegia: Intraoperative Discovery of Cold Agglutinin Disease

Presenting Author: Varun J. Adhvaryu, MD, Resident Physician, University Hospitals - Cleveland Medical Center

Introduction

Cold agglutinin disease is a rare disease, primarily IgM antibodies, where at low temperatures cold agglutinins attach to red blood cells causing agglutination, tissue ischemia, and hemolysis (Gabbard 2020). This is of particular concern in cases involving cardioplegia which is traditionally administered at 4°C, which can trigger hemagglutination and increases the risk of hemolysis upon rewarming (Barbara 2013). Here we present a case of a patient who was previously undiagnosed with cold agglutinin disease in which symptoms were noticed intraoperatively, resulting in changes directed by the cardiac anesthesiology team.

Case Presentation

A 66-year-old male with a history of myocardial infarction status post intraaortic balloon pump placement, coronary artery disease, end stage renal disease on dialysis, heart failure with preserved ejection fraction, hypothyroidism, anemia of chronic disease, type 2 diabetes, hypertension, and hyperlipidemia presented for a coronary artery bypass graft x4. Induction of anesthesia was uneventful and there were no significant issues during sternotomy and harvesting of coronary artery grafts. An appropriate ACT was achieved after heparization and cardiopulmonary bypass (CPB) was initiated. However, after the administration of cardioplegia it appeared that clots were seen in the aortic cannula. The cannula was disconnected from the arterial tubing and blood was placed into a bucket and agglutination was seen. At this point, cold cardioplegia (4°C) was discontinued in favor of warm cardioplegia (37°C) and the clotting in the tubing diminished over time. No significant abnormalities were noted on transesophageal echocardiogram and the patient's hemodynamics remained stable. The remainder of the procedure was uneventful, and the patient was discharged home without any complications.

Discussion

This case demonstrates a unique scenario leading to CPB clotting that must be managed appropriately to prevent catastrophic failure of the CPB oxygenator and adverse outcomes for the patient. Common differential diagnoses of clotting in the CPB apparatus include inadequate heparinization, heparin resistance, or factor deficiencies. These are reversible and can be managed with further heparinization, antithrombin III administration, or repletion of factors. In cases with adequate ACTs and no known factor deficiencies, a more insidious cause such as cold agglutinins should be considered. The treatment of this issue involves warming the CPB cardioplegia to prevent further hemagglutination. In patients with a known cold agglutinin disease, preoperative plasmapheresis can be performed. There is a risk of post-operative hemolysis and patients should be closely monitored for signs of hemolytic crisis post-operatively. Additionally, the role of pre-operative testing is important as patients who have exhibited symptoms of cold agglutinin disease such as Raynaud's phenomenon or hemoglobinuria with exposure to cold should be

evaluated for cold agglutinin disease prior to proceeding with elective cardiac surgery. However, the role of routine testing for cold agglutinin disease prior to cardiac surgery is likely not justifiable due to the low prevalence of the disease.

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#FR 2 – Kiosk 1 – Poster 2

Touching the Heart, Triggering the Storm: Carcinoid Crisis During Cardiac Surgery

Presenting Author: Evan Magaliff, MD, Anesthesiology Resident, University of Connecticut

Introduction

A patient with neuroendocrine cancer and carcinomatosis underwent CABG complicated by intra-operative carcinoid crises in the absence of cardiac metastasis or valvulopathy. She required vasopressors, octreotide, steroids, B12, and emergent bypass for treatment to facilitate surgery and bypass weaning. Despite an initial stable post-operative course, she succumbed to complications and died several days later. This case highlights challenges neuroendocrine tumors present for patients undergoing cardiac surgery.

Case Presentation

An 83-year-old female with neuroendocrine cancer and carcinomatosis without cardiac metastasis underwent CABG after workup showed mvCAD and HFrEF without valvulopathy. Prior to surgery, she was maintained on long-acting octreotide with good prognosis, which was continued pre-operatively per endocrinology. On induction, the patient developed carcinoid crisis requiring high-dose vasopressors, octreotide infusion, and methylprednisolone with resolution. She remained stable until the RA was manipulated for cannulation, triggering a histamine storm requiring high dose vasopressors and emergent CPB. The patient underwent uneventful CABG and was separated from bypass with significant hemodynamic lability, which resolved with hydroxocobalamin. She was extubated post-operative day one but developed declining mental status, respiratory failure, lactic acidosis, and abdominal pain requiring reintubation. Imaging showed inflammation and peri-pancreatic head stranding managed conservatively. The patient continued to decline with peritonitis and repeat imaging showed worsening fluid collections and pneumatosis. She underwent exploratory laparotomy revealing diffusely necrotic ischemic bowel deemed non-survivable, transitioned to comfort care, and expired the following day.

Discussion

In this abstract we presented a case where cardiac carcinoid symptoms manifested despite no echocardiographical evidence of cardiac metastasis. Cardiac surgery presents challenges in patients with neuroendocrine tumors, particularly with right-sided metastasis, and requires unique considerations. Pre-operative octreotide infusions can help prevent crises and while several protocols exist, there is no known optimal regimen. Intraoperatively, several factors predispose to acute episodes. Often presenting with RV dysfunction and valvulopathy necessitating inotropes and vasopressors, catecholamines can trigger vasoactive substance release. Thus, non-catecholamine medications for hemodynamic support, as well as early bypass, should be considered. Cardiac metastasis is almost exclusively right sided and manipulation of the right atrium as occurs during venous cannulation can compress the tumor and provoke a crisis. Alternative cannulation strategies such as femoral access should be considered, and the cannulas be kept distal to avoid encroaching on the right atrium. If a central cannulation is required, pre-induction femoral

access can be obtained to facilitate emergent bypass should a crisis occur. We suggest for patients with metastatic carcinoid disease undergoing cardiac surgery, mitigation strategies to prevent carcinoid crises should be initiated, even if there is no echocardiographical evidence of cardiac metastasis or valvulopathy. Early carcinoid heart disease may be present.

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#FR 2 – Kiosk 1 – Poster 3

Right Ventricular Pseudoaneurysm Following Endomyocardial Biopsy in a Post-Orthotopic Heart Transplant Patient

Presenting Author: David M. Immen, MD, Resident Physician, Northwestern University - McGaw Medical Center

Introduction

Endomyocardial biopsies (EMB) are used in post-transplant patients for surveillance of acute rejection. However, overuse of EMBs is not recommended due to associated procedural risks (Heidenreich). A significant complication of EMBs includes cardiac perforation with rates of 0.5% to 0.7% (Yeo). Although perforation most commonly presents as pericardial effusion with tamponade, pseudoaneurysm is a rare but serious sequelae. With the risk of progression to full rupture at stake, recognition and management are crucial.

Case Presentation

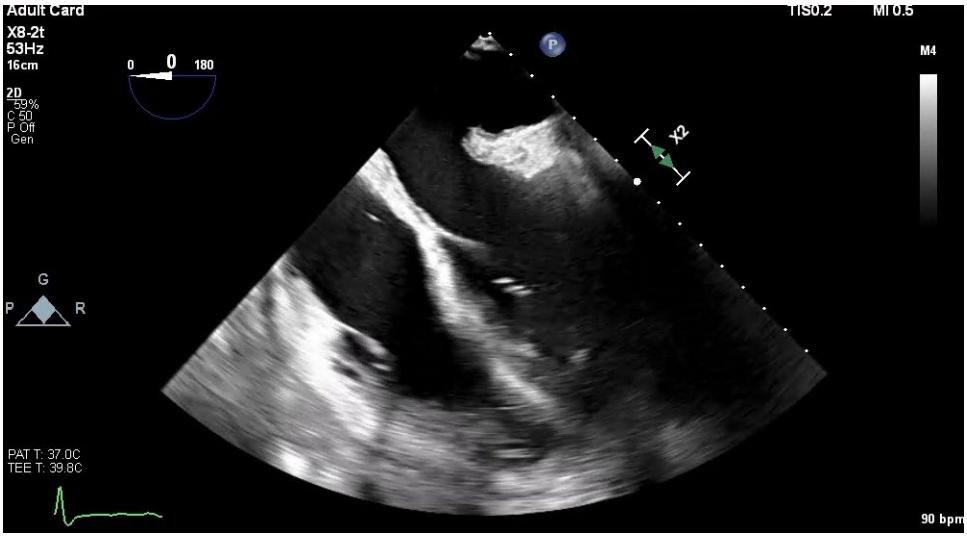
A 63 year-old male with ischemic cardiomyopathy received an orthotopic heart transplant with postoperative course complicated by sternal wound infection. On post-operative day 18 the patient underwent scheduled right heart catheterization with EMB for rejection surveillance. Pathology had no evidence of acute cellular rejection and was negative for acute antibody mediated rejection. On post-operative day 34, an interval transthoracic echocardiogram (TTE) was completed when transitioning immunosuppression regimen. Incidentally, a pseudoaneurysm measuring 4.0cm x 2.3cm of the anterior right ventricle was visualized. This was confirmed on contrasted CT imaging showing a large pseudoaneurysm near the right ventricular outflow tract with possible involvement of coronary vasculature. The patient was taken to the operating room emergently. The patient was hemodynamically stable throughout these sequences of events. General anesthesia was induced and maintained without issue. A complete transesophageal echocardiogram (TEE) exam was completed and no pseudoaneurysm nor obvious outflow tract communication was visualized, however a loculated pericardial effusion was seen. Redo-sternotomy was completed and cardiopulmonary bypass was initiated through central aortic and right femoral vein cannulation. The right ventricular pseudoaneurysm was opened and successfully repaired with sutures. The patient was weaned off bypass and brought to the ICU intubated and sedated.

Discussion

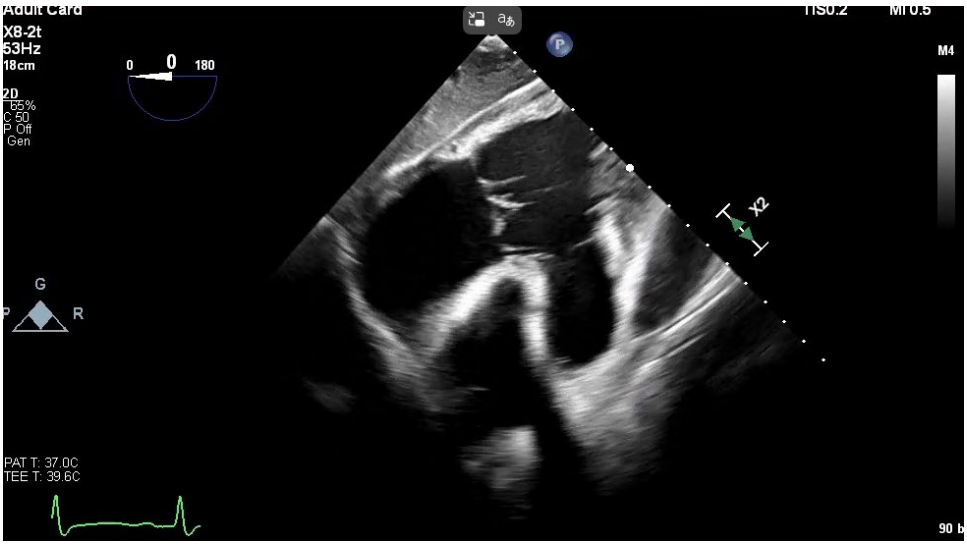
The highest risk of acute rejection occurs during the first six months following transplant, and accordingly EMBs are most frequently performed in the first three to six months post transplant (Seferović). EMB samples are nearly always taken from the right ventricle (RV) as it offers lower complication risks than the left and is more easily accessible via the internal jugular vein. Without any other interventions during the interim period, it is presumed that our patient's pseudoaneurysm was a sequelae of the EMB. Diagnosis relies on imaging, with MRI, cardiac CT and echocardiography all providing diagnostic utility. In our case, this patient's TTE and CT provided cause for emergent surgical repair, however the prominent pseudoaneurysm

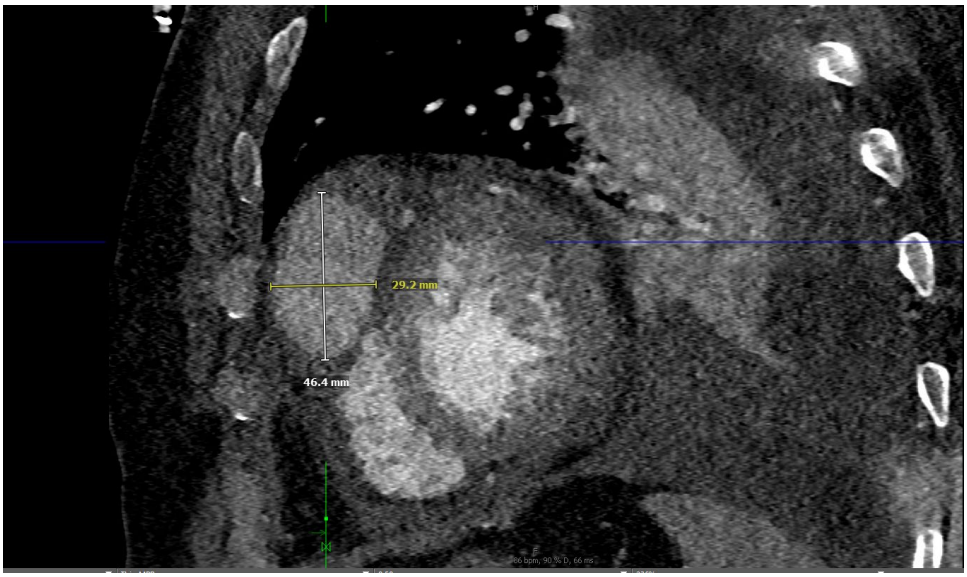
was not visualized during intraoperative TEE. With the high risk of pseudoaneurysmal rupture, surgical repair is standard of care. Percutaneous closure and transthoracic coiling have shown promise for non-surgical candidates. Large or expanding pseudoaneurysms and hemodynamic instability raise urgency for surgical repair. The extensive loculations in the pericardium from our patient's mediastinal infection likely provided some protection from further expansion of the pseudoaneurysm.

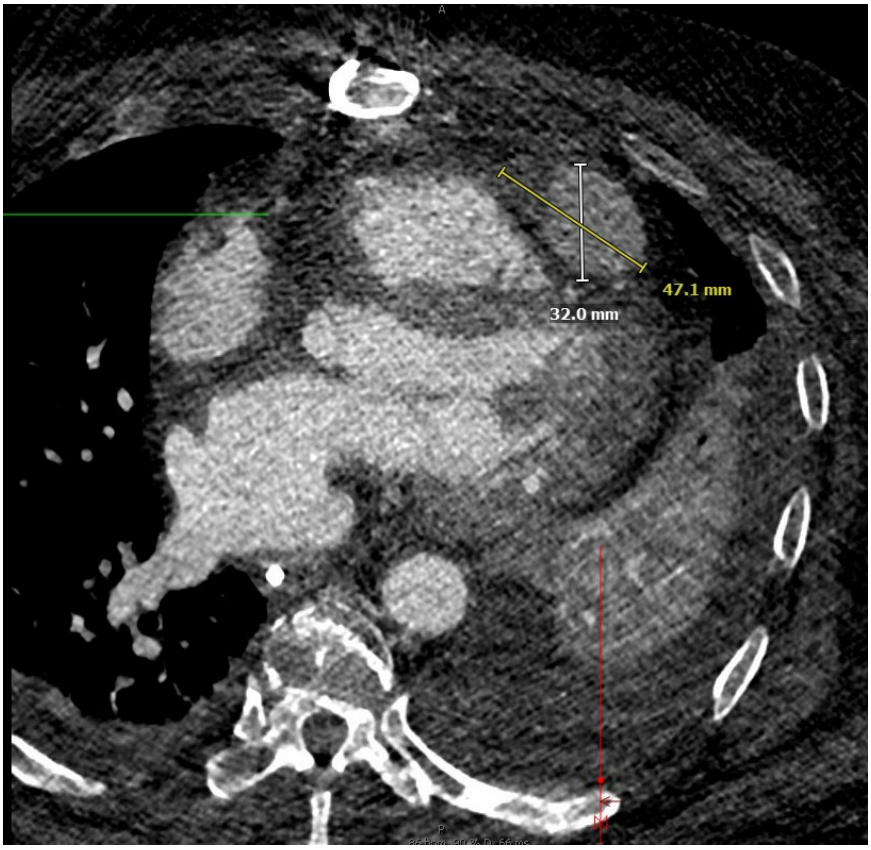
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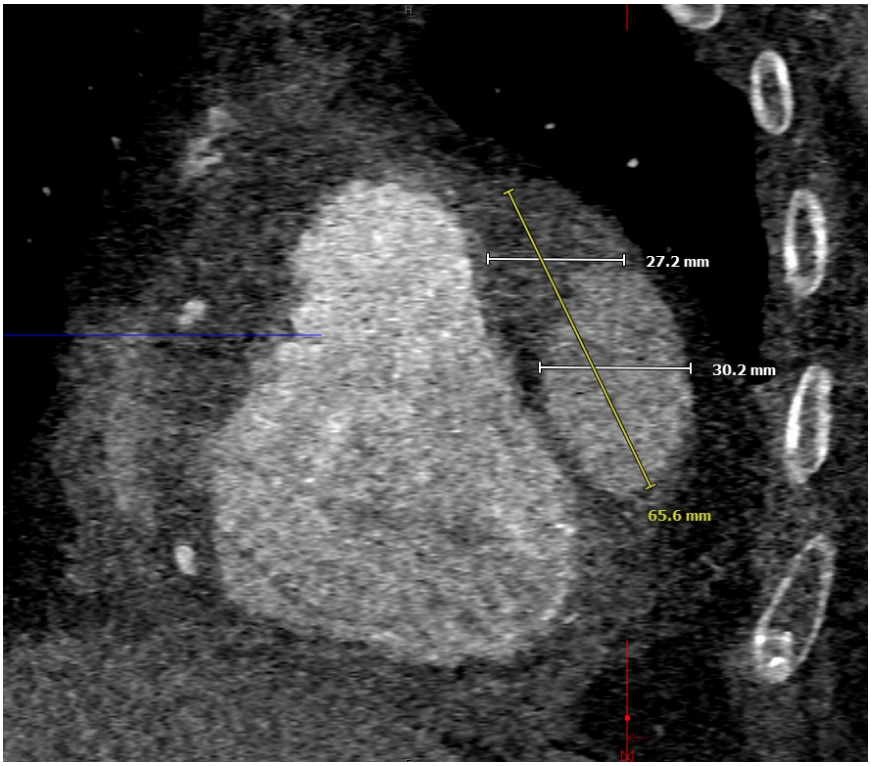





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G
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R
PAT T: 37.0C
TEE T: 39.6C
90 b


29.2 mm
46.4 mm


47.1 mm
32.0 mm



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#FR 2 – Kiosk 1 – Poster 4

When the apex turns deadly: a giant thrombus within a silent aneurysm

Presenting Author: Naema Daino, MD, Medical College of Georgia at Augusta University

Introduction

Ventricular aneurysms are rare complications that can develop months after a myocardial infarction due to weakened myocardium. Ventricular aneurysms can cause chest pain, shortness of breath, palpitations, or murmurs and may lead to heart failure and mural thrombi. While small mural thrombi are relatively common, a large apical thrombus is rare and presents diagnostic and management challenges. We report a case involving a large apical thrombus located within a chronic left ventricular (LV) aneurysm, emphasizing the complexities of perioperative management.

Case Presentation

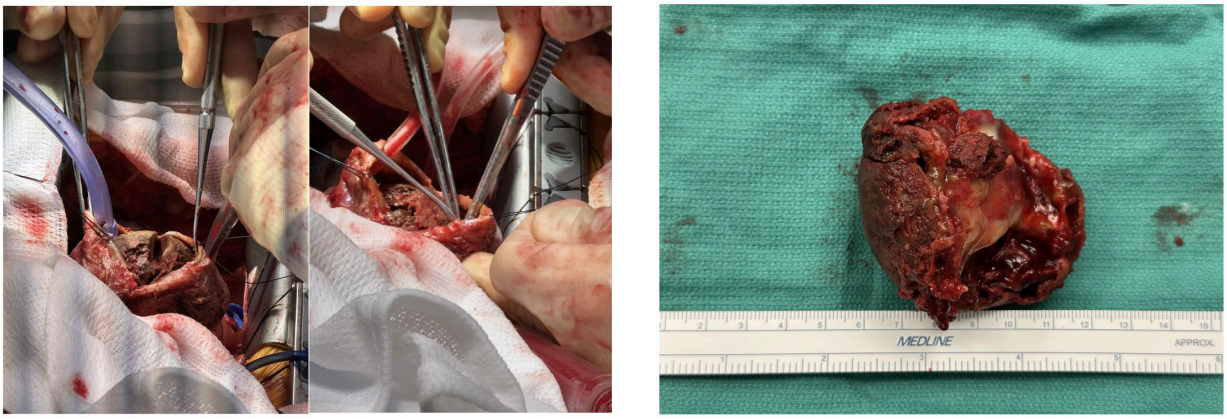
A 49-year-old male with coronary artery disease, a history of three myocardial infarctions treated with five stents, heart failure with reduced ejection fraction, and grand mal seizures presented to the hospital due to syncope, possible seizures, and chest pain for several weeks. Vital signs on admission were stable, with a lactate level of 2.1. The patient's initial echocardiogram revealed a severely enlarged LV chamber, a large dyskinetic apical aneurysm, significantly reduced systolic function (EF 25–30%), mild concentric hypertrophy, and a massive apical thrombus. The apical thrombus had been diagnosed three years earlier, and the patient was subsequently started on 5 mg of Eliquis twice daily. Chest CT showed a large true LV apical aneurysm with a mural thrombus. Brain MRI revealed no acute abnormality. Cardiac MRI indicated delayed enhancement and wall thinning in the basal, inferolateral, inferior, and anterior segments, signaling nonviable tissue. Left heart catheterization identified severe 70% stenosis of the LAD with significant aneurysmal disease, but the sites distal to the stent were unsuitable for surgical grafting, leading to medical management with captopril, metoprolol succinate, dapagliflozin, heparin, and aspirin. The patient was scheduled for ventricular thrombus removal and aneurysm repair using cardiopulmonary bypass. Due to the high-risk nature of disease pathology, a brachial arterial line, left internal jugular central line, and a pulmonary artery catheter were inserted to aid hemodynamic monitoring and vasopressor titration. Continuous transesophageal (TEE) monitoring and cerebral oximetry were used throughout the excision of the thrombus. A left femoral artery line was inserted due to the potential need for a mechanical assist device, and the right femoral vein was cannulated for emergent access due to concern for aneurysm rupture. The surgeons dissected the LV aneurysm, removed a baseball-sized clot, and the cross clamp and pump times were 27 and 61 minutes. Figure 1. The patient was successfully weaned off cardiopulmonary bypass, remained hemodynamically stable, was weaned off milrinone and pressors without issues, and was discharged on postoperative day 4.

Discussion

Left ventricular aneurysms complicated by large apical thrombi pose significant perioperative challenges, requiring coordinated medical, surgical, and anesthetic management due to increased risks of systemic

embolization, hemodynamic instability, and difficulty with myocardial protection during cardiopulmonary bypass. Careful preoperative assessment, including imaging (Chest CT, Cardiac MRI, and catheterization), evaluation of ventricular function, and thrombus morphology, assists in anesthetic planning.

Images



(a) (b)

Figure 1. (a) Left Ventricular Thrombectomy; (b) Mural Left Ventricular Thrombus

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#FR 2 – Kiosk 1 – Poster 5

When Continuity Fails: Grading Aortic Valve Stenosis in the setting of Concomitant Left Ventricular Outflow Tract Obstruction

Presenting Author: Edward Jaffe, MD, Brigham and Women's Hospital

Introduction

Standard quantitative assessment of aortic valve stenosis (AS)—including peak jet velocity, peak and mean pressure gradients, and aortic valve area (AVA) calculation via the continuity equation—relies on spectral Doppler[1]. Specifically, these methods require distinct Continuous Wave Doppler (CWD) and Pulse Wave Doppler (PWD) signals at the aortic valve and left ventricular outflow tract (LVOT), respectively. We present a challenging case of AS with concomitant LVOT obstruction where high-velocity outflow rendered standard Doppler uninterpretable. Clinical decision-making regarding valve replacement was instead guided by three-dimensional (3D) planimetry.

Case Presentation

A 67-year-old female with a history of AS and subaortic membrane-induced LVOT obstruction presented for surgical repair. The operative plan included subaortic membrane resection and possible aortic valve replacement (AVR) based on intraoperative transesophageal echocardiography (TEE) (Figure 1). Initial PWD interrogation of the LVOT demonstrated signal aliasing, precluding measurement of a velocity-time integral (VTI) signal envelope (Figure 2A). CWD across the aortic valve yielded a single parabolic waveform (Figure 2B). This signal could not be reliably attributed to the valve since there was concomitant high-velocity subaortic flow. Consequently, standard hemodynamic metrics were unattainable. To quantitatively grade the AS and assess the LVOT obstruction we instead utilized 3D planimetry with multiplanar reconstruction (MPR) to align orthogonal 2D images. This confirmed a critical AVA of 0.81 cm^2 and an LVOT area of 0.94 cm^2 (Figure 3A & 3B). Given the severely reduced AVA and a small aortic annulus (19mm; BSA 1.88cm^2) the surgical team performed an annular root enlargement and implanted a 23mm mechanical prosthesis in addition to the membrane resection. Post-cardiopulmonary bypass TEE confirmed laminar flow on color Doppler (Figure 4), resolution of PWD aliasing, and an acceptable CWD derived gradient across the prosthetic valve.

Discussion

This case highlights the limitations of standard AS quantification when hemodynamic assumptions are violated. The continuity equation relies on the premises that peak velocity occurs solely at the aortic valve and that LVOT velocity is sufficiently low that PWD aliasing does not occur. In this patient, the subaortic membrane accelerated ejected flow, rendering the LVOT signal unmeasurable and masking the true valvular hemodynamics. Accordingly, 3D planimetry proved essential for verifying severe AS, directly informing the decision to proceed with AVR. Literature suggests 3D planimetry correlates strongly with invasive and Doppler-derived methods. While it may slightly overestimate AVA in mild-to-moderate AS (mean difference ~0.12 cm^2)[2], its accuracy in severe AS is high with nearly identical results to continuity derived

assessments[3].

Images

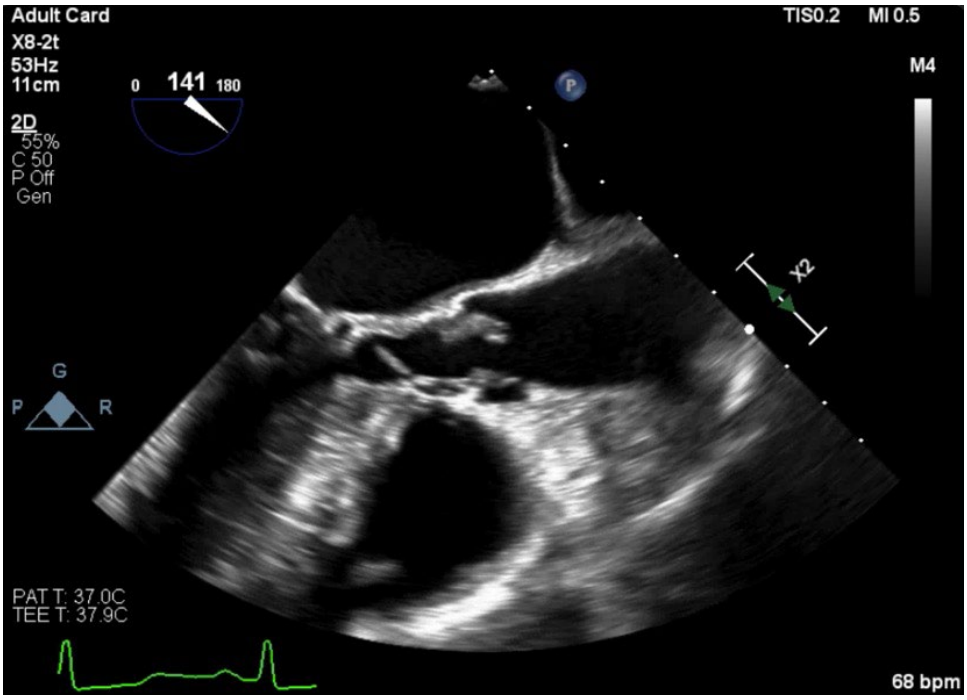


Figure 1: Mid Esophageal Aortic Long Axis View showing sclerotic bicuspid aortic valve as well as subvalvular membrane. Note the septations visible across LVOT.

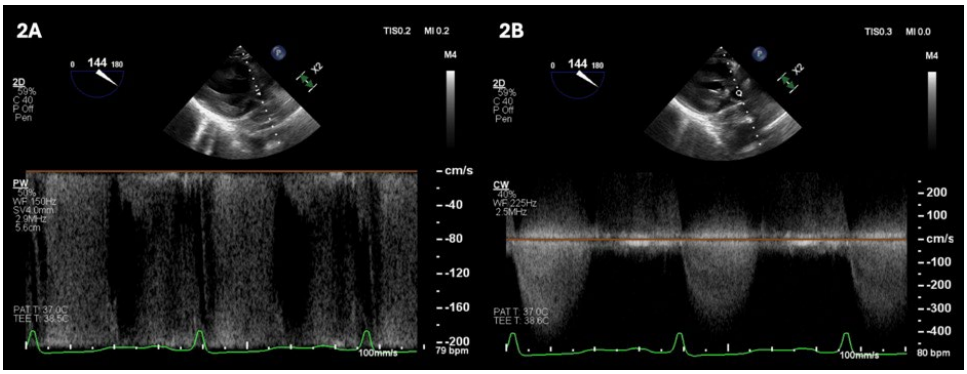


Figure 2A: PWD gated at the LVOT in the transgastric long axis view. Note signal aliasing. Figure 2B: CWD through LVOT in transgastric long axis view. No two discrete envelopes seen.

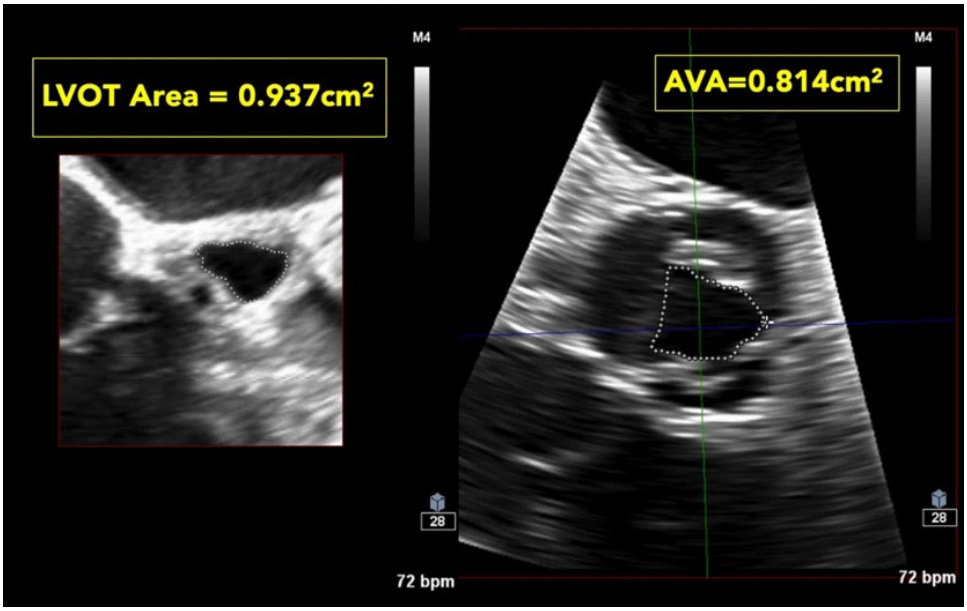


Figure 3A: Multiplanar reconstruction (MPR) tracing of aortic valve area Figure 3B: Multiplanar reconstruction (MPR) of LVOT with smallest area traced

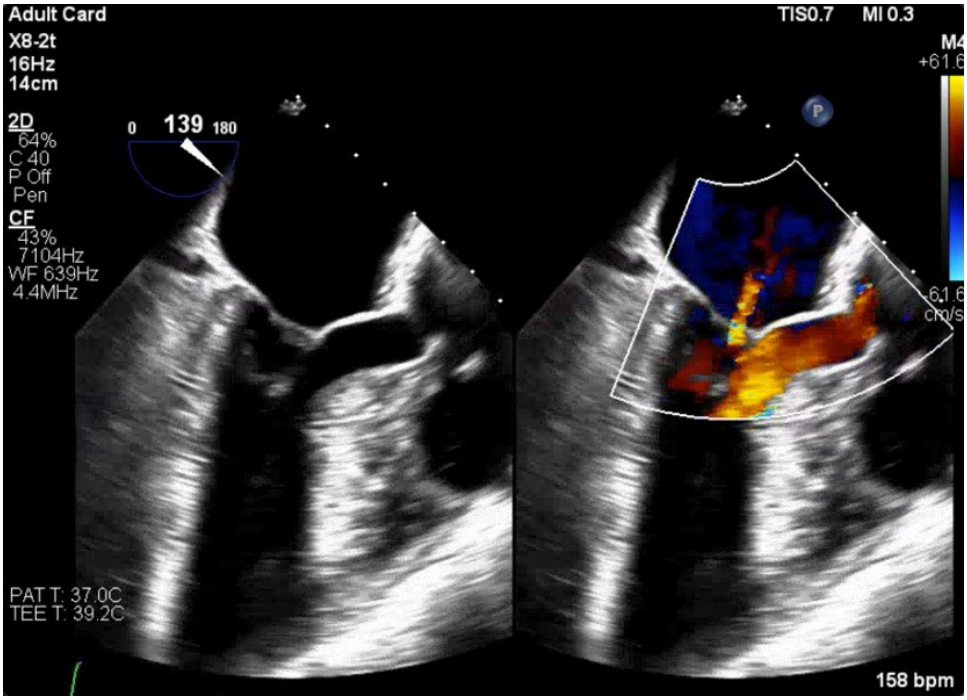


Figure 4: Mid Esophageal long axis view (with color compare) Post-bypass showing open LVOT

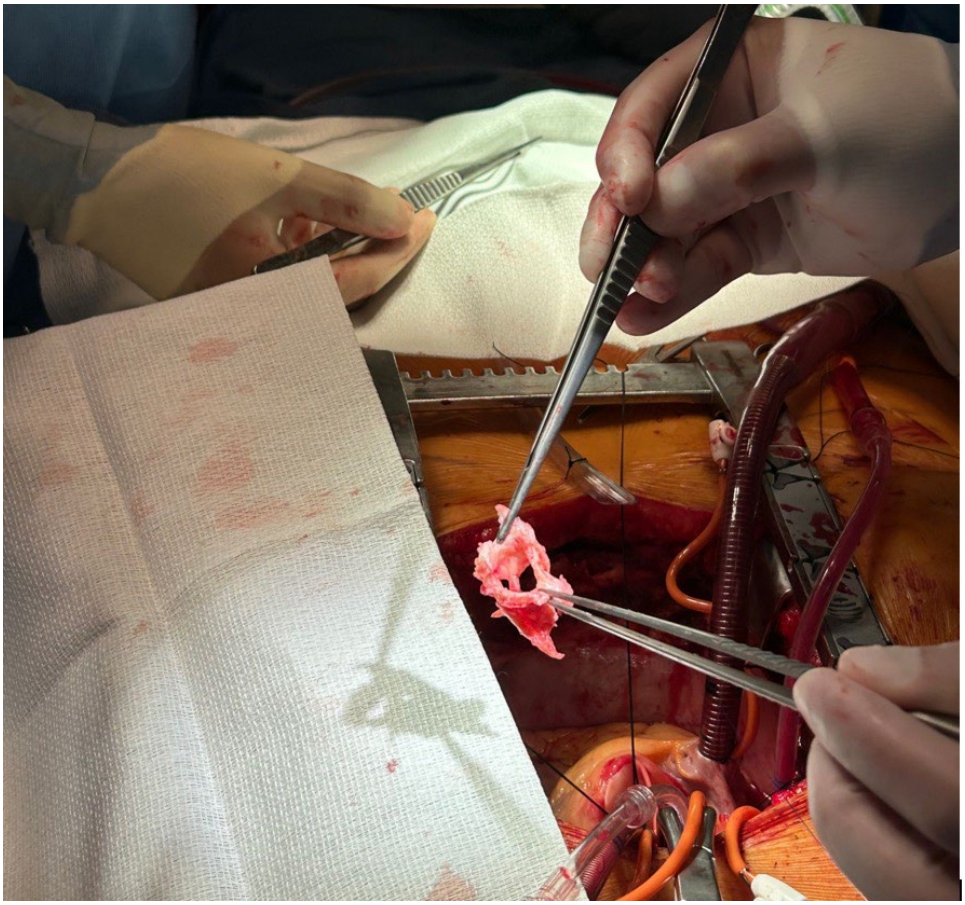


Figure 5: Intra-operative photo of LVOT subvalvular membrane as it is removed from the patient en bloc.

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#FR 2 – Kiosk 1 – Poster 6

Stuck Between a Shock and a Hard Place: Using Rescue Echo to Diagnose Tamponade and Tension Hemothorax on VAV ECMO

Presenting Author: Terrance J. Janes, Jr., MD, Anesthesiology Resident, University of Louisville Department of Anesthesiology

Introduction

Extracorporeal membrane oxygenation (ECMO) is used for patients with advanced cardiac or pulmonary failure[1]. Despite its benefits, ECMO carries significant complications that may rapidly become life-threatening. We present a patient who developed differential hypoxia on V-A ECMO requiring conversion to V-AV support, followed by acute pericardial tamponade, ventricular fibrillation arrest, and tension hemothorax. This case highlights key considerations in recognizing and managing ECMO-related instability

Case Presentation

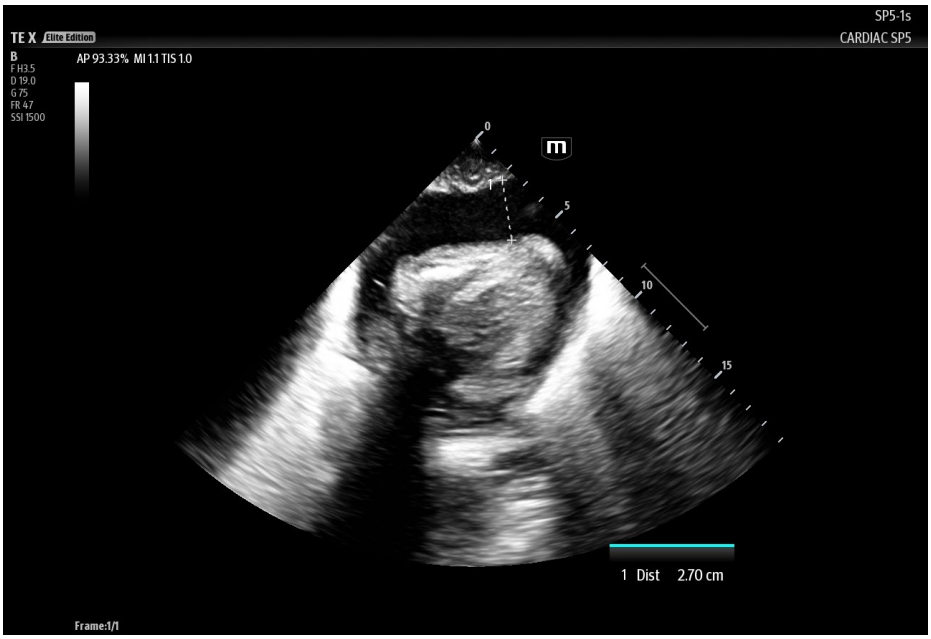
A 37-year-old man with smoking history and pulmonary hypertension was transferred to a tertiary center with acute respiratory failure from pneumonia. Echocardiography showed marked RV dilation and dysfunction, an underfilled LV, and preserved LV systolic function. He was intubated and placed on V-A ECMO early in his course. On ECMO day 4 he developed differential hypoxia and was transitioned to V-AV ECMO in the OR with improved oxygenation and hemodynamics. Eight hours later he developed acutely decreased ECMO flows with a wide-complex rhythm that progressed to pulseless VT arrest and absent ECMO flow. ACLS with compressions was initiated due to inadequate ECMO flow, and ROSC was achieved after multiple defibrillations. Rescue TEE demonstrated a large pericardial effusion with tamponade physiology. Bedside pericardiocentesis with catheter placement led to hemodynamic improvement. The venous return cannula was clamped due to concern it worsened the effusion, and he was returned to V-A ECMO with restored flows and arterial pulsatility. Two hours later, ECMO flows again declined and pulsatility diminished. Bedside POCUS showed a large left pleural effusion. Manipulation of a pre-existing chest tube evacuated 1.5 L of blood, consistent with a tension hemothorax. The patient stabilized and underwent a surgical pericardial window the next morning.

Discussion

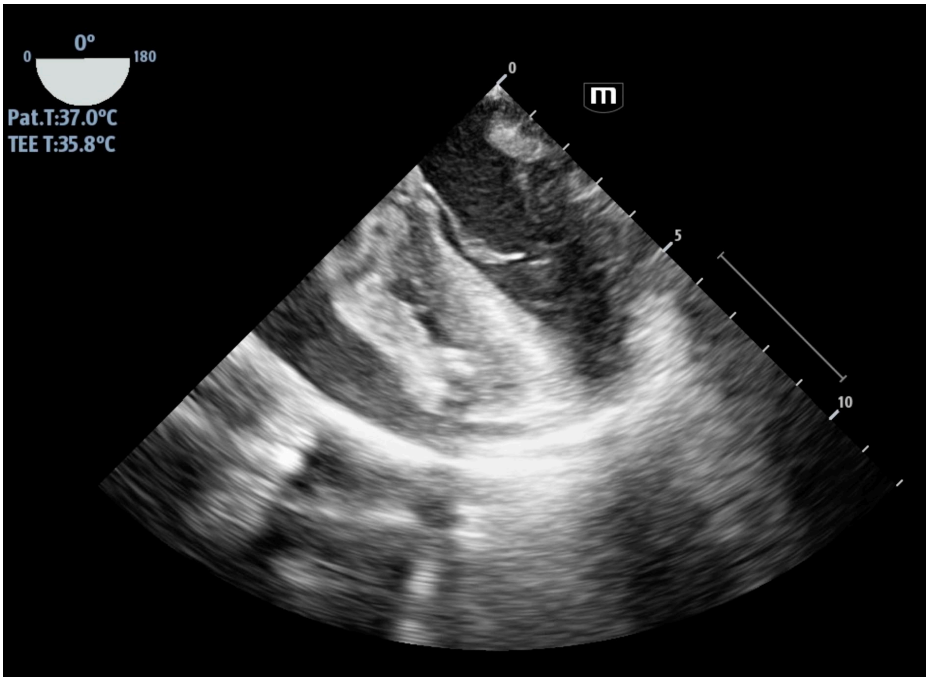
This case demonstrates critical complications of ECMO support. Differential hypoxia in a patient with improving native cardiac output but persistent pulmonary dysfunction is characteristic of “North–South syndrome.” Transitioning to V-AV ECMO is a recognized strategy to restore oxygenated blood to the coronary and cerebral circulations[2]. Other options include conversion to V-V ECMO/RVAD or discontinuation of support. Echocardiography and clinical findings indicated ongoing need for biventricular support, so V-AV ECMO was selected. The subsequent cardiac arrest despite ECMO underscores the need to immediately assess circuit flows during hemodynamic collapse. When flows are inadequate, conventional CPR remains essential while rapidly evaluating reversible causes. Rescue echocardiography identified delayed post-operative tamponade and later a tension hemothorax, each impairing ECMO function. This case emphasizes

rapid troubleshooting of ECMO instability, early use of point-of-care imaging, and awareness of life-threatening complications that may arise even after circuit modification.

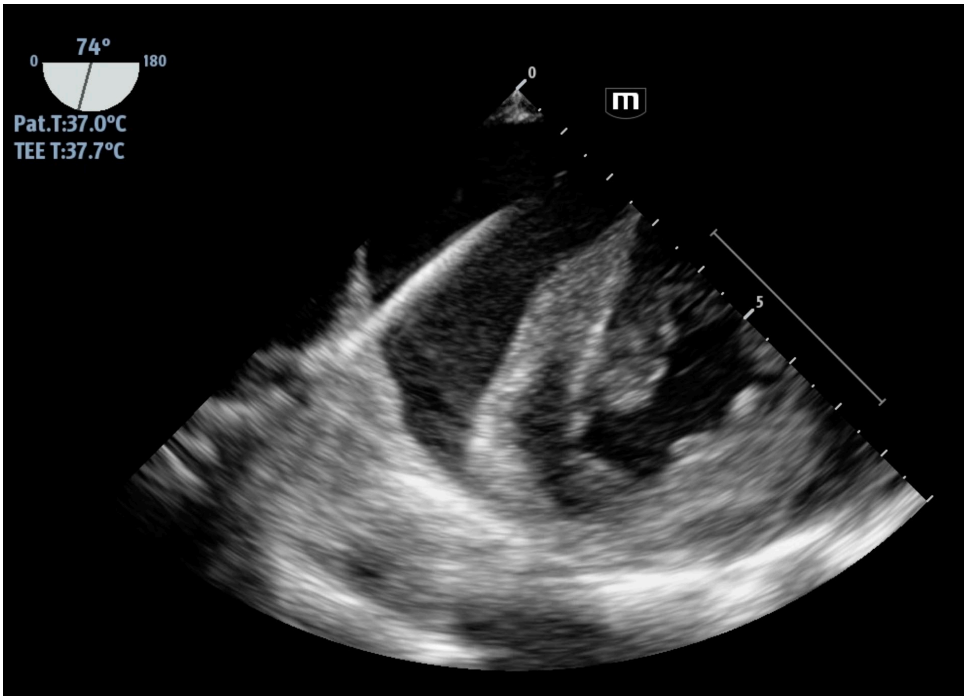
Images



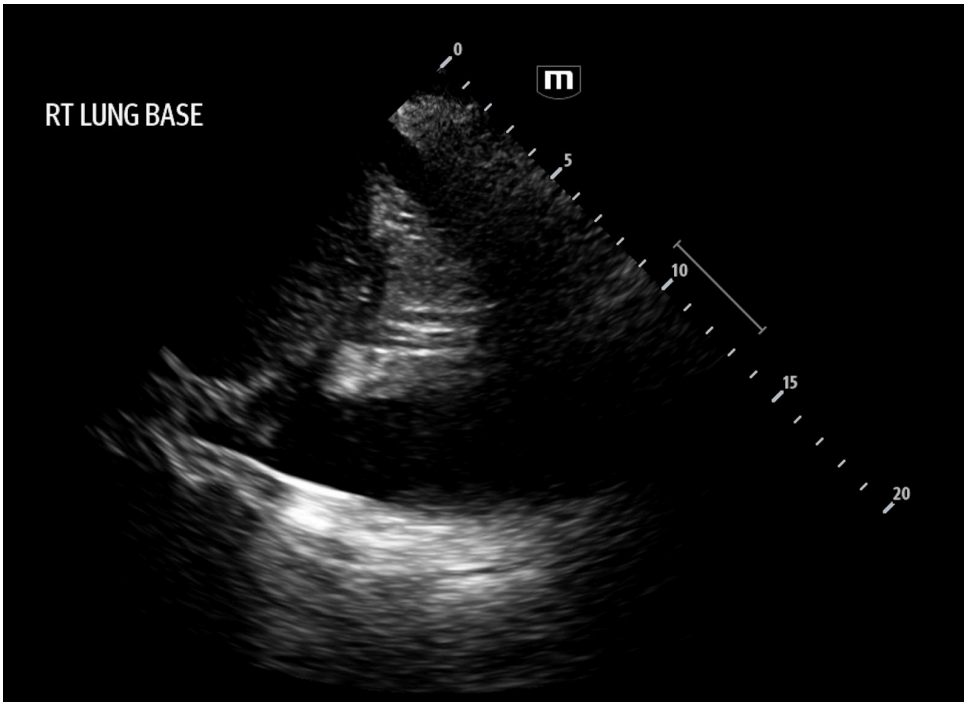
1: Initial TTE (Apical 4 chamber) post ROSC with effusion measurement



2: TEE post ROSC (mid-esophageal 4 chamber) again demonstrating effusion and diastolic RV collapse



3: TEE transgastric short axis during pericardiocentesis to guide wire and catheter placement



4: Right chest lung base showing pleural effusion



5: TTE (Apical 4 chamber) with remnant pericardial effusion (organized clot) after ICU stabilization requiring surgical pericardial window

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#FR 2 – Kiosk 1 – Poster 7

Iatrogenic Pulmonary Vein and Left Atrial Dissection Associated with Left Ventricular Vent Placement in a patient on VA-ECMO for Ventricular Tachycardia Storm

Presenting Author: Joshua E. Insler, Doctor of Medicine (MD), Resident Physician, Department of Anesthesiology, Yale School of Medicine

Introduction

Venoarterial extracorporeal membrane oxygenation (VA-ECMO) provides reliable short-term mechanical support in cardiogenic shock, though it carries the risk of causing left ventricular distension (1). Without adequate decompression, elevated LV pressures can worsen pulmonary edema, impair myocardial recovery, and increase the risk of thrombus formation within the left heart (1,2). The presence of an open chest often predisposes interventional decision making to surgical LV venting. An LV vent is introduced through the right superior pulmonary vein and may be connected to the ECMO venous drainage limb using a Y-connector. Although structural injury from surgically placed LV vents—such as pulmonary vein trauma, left atrial dissection, or malposition—has been described, the current literature contains only a small number of case reports and isolated case series documenting these complications (3). Here we describe a case of right upper pulmonary vein and left atrial dissection following surgical LV vent placement.

Case Presentation

The patient described is an 83 year old male with a past medical history of pulmonary embolism, hypertension, and psoriasis who initially presented to an outside hospital with chest pain and shortness of breath. He was found to have a myocardial infarction from multivessel coronary artery disease which was complicated by cardiogenic shock requiring preoperative intra-aortic balloon pump placement. He underwent coronary artery bypass grafting which was complicated by refractory ventricular tachycardia and ventricular fibrillation requiring emergent peripheral VA-ECMO initiation on post-operative day 3. Peripheral cannulation was complicated by arterial cannula site bleeding leading to return to the operating room for mediastinal exploration and ECMO reconfiguration from peripheral to central arterial cannulation as well as placement of a surgical LV vent. On post-operative day 7 he was taken for Impella 5.5 placement and possible chest closure. Upon removal of the LV vent, a dissection flap was noted on transesophageal echocardiography originating in the right upper pulmonary vein which extended into the left atrium across the posterior aspect terminating near the interatrial septum. The impella was unable to be placed due to vascular calcification and the decision was made to close the chest, return to the ICU for optimization, and to plan for left axillary cutdown and impella placement at a later date.

Discussion

Structural injury from surgically placed LV vents is an uncommon but important complication, particularly when it results in left atrial dissection. Most of the existing evidence consists of isolated case reports, underscoring both the rarity of this event and the limited understanding of its true incidence. The mechanism is thought to involve direct trauma during vent insertion or excessive traction on the pulmonary vein–left

atrial junction, especially in the setting of friable or distended cardiac tissue (3). Early recognition is critical, as left atrial dissection can compromise pulmonary venous return, impair LV filling, and destabilize hemodynamics. This case highlights the need for meticulous surgical technique, routine intraoperative imaging, and heightened vigilance for structural complications when LV venting is introduced.

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#FR 2 – Kiosk 2 – Poster 1

Management of Quadruple Valve Endocarditis

Presenting Author: Talha Saif, MD, CT Anesthesia Fellow, Duke University

Introduction

Infective endocarditis is a well-studied pathology that unfortunately has high rates of in-hospital mortality even in developed countries. Most patients suffering from infective endocarditis typically have a singular culprit valve, usually owing to prior damage or defect of the valve. Treatment takes into consideration many factors, such as: extent of valve damage, severity of valvular function, patient demographics and etcetera. Culminating these factors with expert clinical opinions dictates whether the patient receives a valve repair, biologic or mechanical valve. In this case report, we present an extraordinary case in which a 33 year old female suffered from quadruple valve endocarditis and was successfully treated allowing her to give birth the following year. We will discuss the echocardiographic findings along with the subsequent anesthetic and surgical management that took place.

Case Presentation

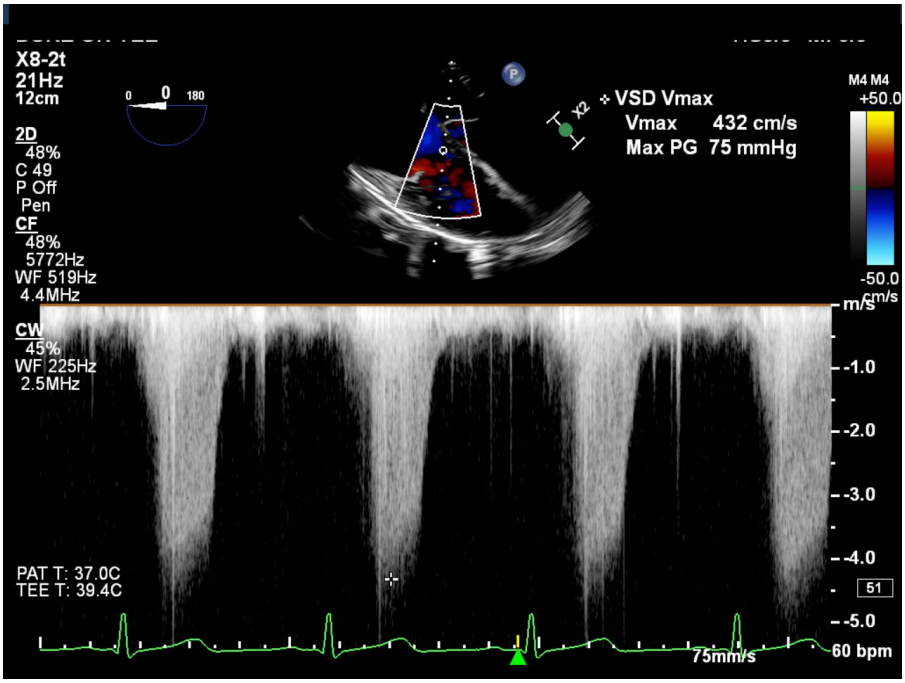
A 33 year old female with PMH significant for Shone Complex status post aortic coarctation repair as a child presented to an outside facility with concerns of bright red per rectum, and several months of fever and chills. A CTA performed at the outside hospital showed evidence of consolidation across both lobes and as well as areas of necrosis in both the kidneys and the lungs concerning for septic emboli. She was transferred to Duke University for further management. Her initial TEE demonstrated evidence of vegetations on the pulmonic, tricuspid, and aortic valves with evidence of severe PI, mild TR, and moderate AI as well as a subaortic membrane. The decision was made to replace the pulmonic, tricuspid and aortic valves via sternotomy. However, the TEE in the OR demonstrated a VSD and vegetation on the mitral valve that were not appreciated on prior studies. Given the severity of regurgitant jets on TEE, the initial decision was to perform the following procedures: tricuspid valve debridement, annuloplasty and cleft closure; mitral valve debridement and cleft closure; subaortic membrane resection with aortic valve repair; pulmonic valve replacement (given severity of PI); and VSD closure. All of the procedures were successful save for the aortic valve repair. Despite the surgeon's best efforts, the aortic valve showed evidence of severe AI on TEE. The aorta was clamped once more, and the decision to do an aortic root replacement was made.

Discussion

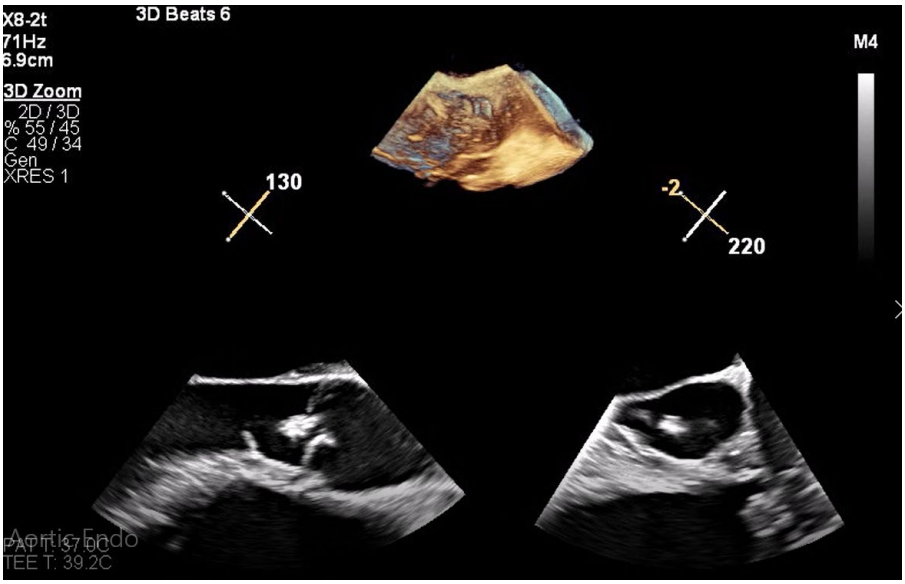
It is believed that the subaortic membrane lead to damaging of the aortic valve, which made it susceptible to infection. The patient was found to have an oral infection that likely seeded the aortic valve. The damaged valve, in addition to the colonization of the bacteria, lead to enough AI which allowed for seeding of the bacteria on the mitral valve. The VSD (which was not noted on prior studies), ultimately lead to the seeding of the tricuspid and pulmonic valves, resulting in quadruple valve endocarditis. This case required complex surgical management given the patient's age (minimizing the amount of prosthesis) and the extent of the disease. Each valve was evaluated for repair candidacy with the hopes of preserving native tissue, but the

patient eventually required having replacement of the aortic and pulmonic valves given the significant pathology noted in both valves.

Images



ME 4 Chamber, View of VSD



ME AV LAX, View of Vegetation on Aortic Valve


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#FR 2 – Kiosk 2 – Poster 2

Striking the Wrong Chord – An Unusual Case of Left Ventricular Outflow Tract Obstruction

Presenting Author: Boris Ladyzhensky, MD, ACTA Fellow, Cleveland Clinic

Introduction

Left ventricular outflow tract (LVOT) obstruction can be due to systolic anterior motion (SAM) of the anterior mitral valve (MV) leaflet, or less commonly, abnormalities of the MV or subvalvular apparatus.1 In order to guide myectomy planning, it is crucial for the transesophageal echocardiography (TEE) exam to evaluate all possible etiologies of LVOT obstruction, such as measurements of septal thickness, pressure gradients within the left ventricle (LV) and LVOT, the presence of SAM, and aberrations of the MV apparatus. We present an unusual case of hypertrophic cardiomyopathy (HCM) with dynamic LVOT obstruction due to redundant chordae tendineae.

Case Presentation

An 82-year-old male with history of bioprosthetic aortic valve and root replacement 18 years ago presented with worsening dyspnea on exertion and presyncope. His transthoracic echocardiogram (TTE) revealed normal biventricular function, severe prosthetic aortic regurgitation, trace mitral regurgitation (MR) without SAM, and concentric LV hypertrophy. He was scheduled to undergo redo aortic valve replacement (AVR). Intraoperative TEE revealed interventricular septal thickness of 2.1 cm, mild centrally-directed MR, papillary muscle hypertrophy, and redundant chordae tendineae. Color Doppler interrogation demonstrated flow acceleration in the LVOT (Fig 1). Spectral Doppler showed peak LVOT gradient of 33 mmHg. 2-D and 3-D echocardiography showed a long, thin, mobile mass prolapsing from the LV into the LVOT during systole (Fig 2, 3). The patient underwent AVR, septal myectomy, and resection of redundant chordae attached to the A2 segment of the MV. The resected chordae measured approximately 8 cm in length (Fig 4). The post-myectomy TEE showed laminar flow in the LVOT, peak LVOT gradient of 11 mmHg, and trace MR. Prior to discharge, TTE showed mild MR without SAM, peak/mean aortic gradients of 7/4 mmHg, and no resting or Valsalva LVOT gradient.

Discussion

Redundant chordae tendineae, or any accessory mitral valve tissue (AMVT), are rare conditions that can lead to significant LVOT obstruction. AMVT is also likely to be associated with other congenital cardiac lesions, and is thus more frequently reported in pediatric patients, and discovered as incidental findings in adults.2 The lesions are typically mobile globular echodensities that prolapse into the LVOT during systole. The anterior MV leaflet is more commonly the origin of AMVT. Spectral and color Doppler should be utilized to localize and quantify the degree of LVOT obstruction. Symptomatic LVOT obstruction occurs at a mean LVOT gradient of 50 mmHg and surgical resection is recommended when the mean gradient exceeds 25 mmHg.2 3-D multiplanar reconstruction as well as real-time 3-D TEE may be useful to fully characterize the lesion dimensions, degree of obstruction, and its relationship and attachments to surrounding structures. Given its association with other congenital abnormalities, a thorough search for other congenital lesions is warranted.

In our case, the redundant chordae were identified on 2-D TEE imaging, and 3-D reconstruction was able to reinforce 2-D findings of chordae prolapse and outflow obstruction. It is important to recognize the rarer causes of LVOT obstruction and exclude significant LVOT obstruction when large AMVT is present.

Images

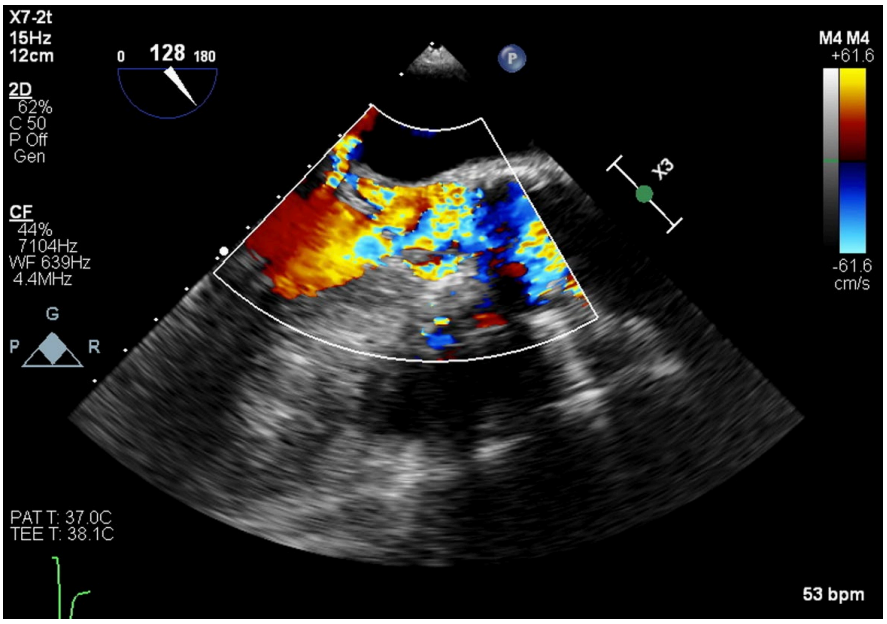


Figure 1. Mid-esophageal long axis view with color Doppler showing turbulent flow acceleration in the LVOT.

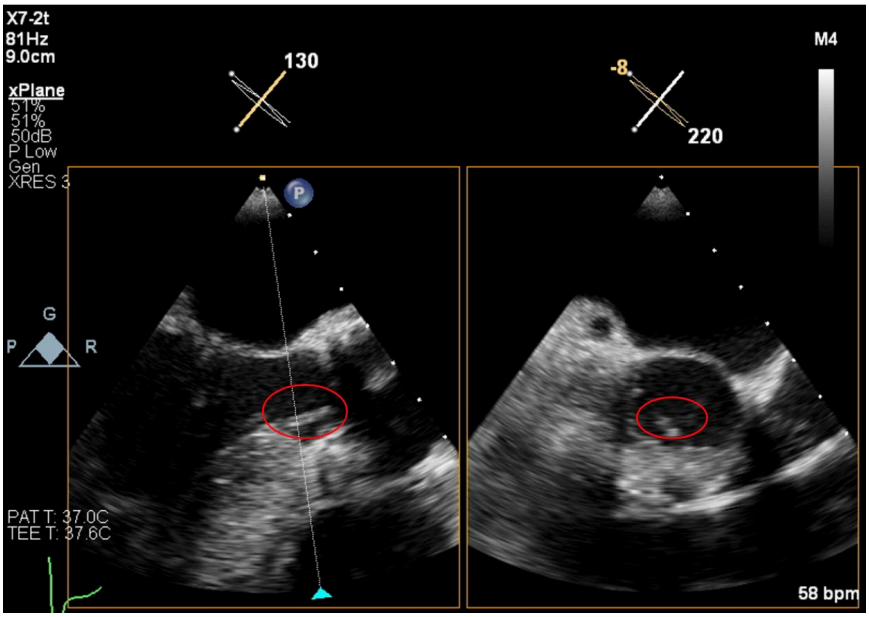


Figure 2. Mid-esophageal long axis view (left) with biplane through LVOT (right) showing mobile mass prolapsing from the LV into the LVOT during systole (circled in red), likely from coiled or ruptured redundant chordae.



Figure 3. 3-D reconstruction of the mid-esophageal long axis view showing mobile mass prolapsing from the LV into the LVOT during systole (circled in red), likely from coiled or ruptured redundant chordae.

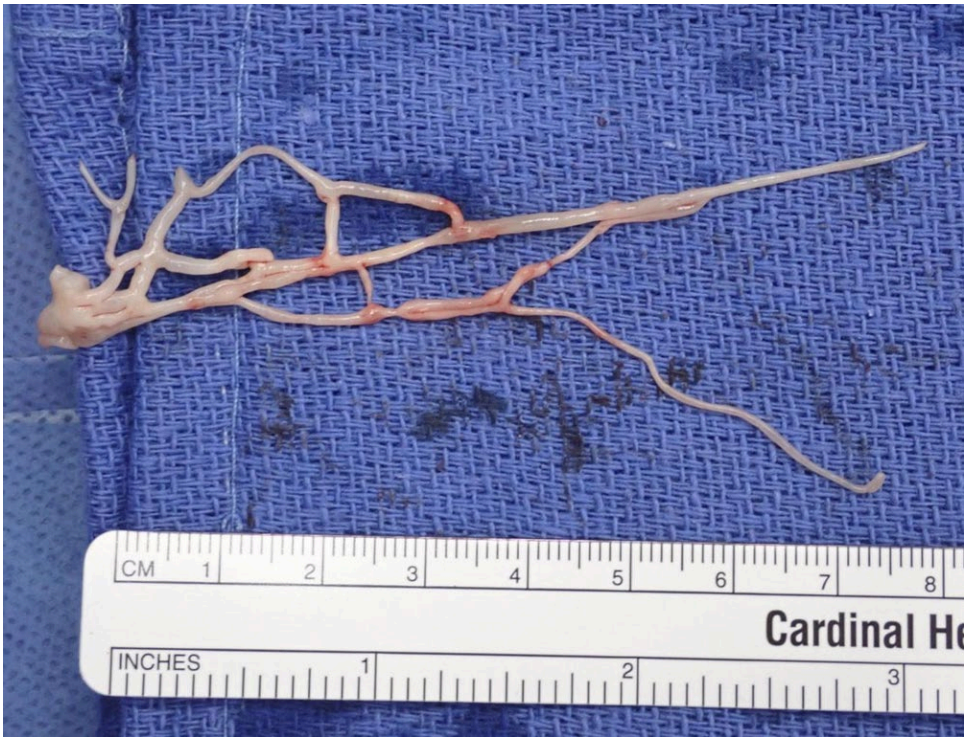


Figure 4. Resected redundant chordae tendineae, measuring approximately 8 cm.

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#FR 2 – Kiosk 2 – Poster 3

A Rocky Start for a Donation After Circulatory Death Heart

Presenting Author: Victoria Hans, D.O., Resident, University of Chicago Medical Center

Introduction

Donation after circulatory death (DCD) heart transplantation is an expanding strategy to increase donor availability and may raise transplant volume by up to 30%. However, DCD hearts experience longer warm ischemic time and carry a higher risk of early graft dysfunction. We describe a DCD heart transplant complicated by immediate severe right ventricular (RV) failure requiring mechanical circulatory support (MCS).

Case Presentation

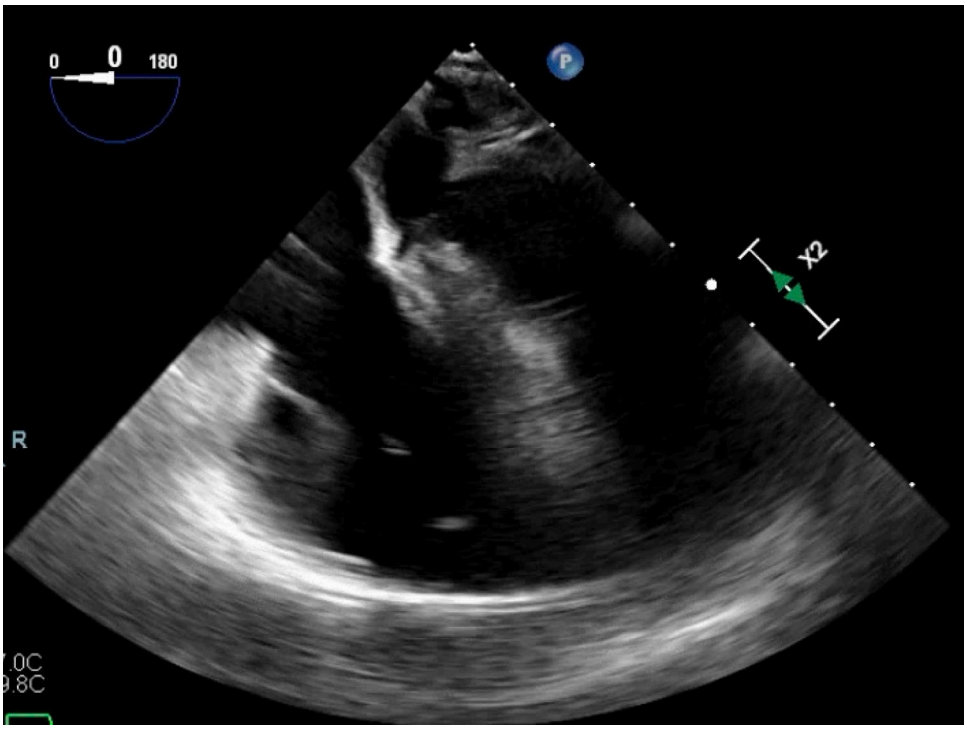
A 64-year-old woman with hypertrophic obstructive cardiomyopathy and longstanding heart failure with reduced ejection fraction was in the ICU on an intra-aortic balloon pump (IABP) and listed as Status 2 for heart transplant. A DCD heart with 206 minutes of ischemic time was accepted. Procurement reported good arrest conditions, closed foramen ovale, normal heart size, and mild tricuspid regurgitation. After uncomplicated sternotomy and initiation of cardiopulmonary bypass (CPB), dopamine 5 mcg/kg/min and dobutamine 5 mcg/kg/min were started in preparation for CPB separation. Following cross-clamp removal and atrial pacing, transesophageal echocardiography (TEE) showed severely reduced RV function and moderately reduced left ventricular function. Epinephrine 0.05 mcg/kg/min was initiated and the IABP was restarted. Partial improvement allowed CPB separation and heparin reversal. Within 30 minutes, RV function worsened despite escalating support, prompting initiation of veno-arterial ECMO (VA-ECMO). By postoperative day (POD) 3, cardiac function recovered sufficiently for decannulation. The IABP was removed on POD 4, and inotropes were discontinued by POD 11. A transthoracic echocardiogram on POD 9 showed EF 59% with mildly reduced RV function.

Discussion

DCD heart transplantation, first attempted in 1967, declined after adoption of donation after brain death (DBD) but has resurged with increasing demand for donor hearts. The first modern U.S. adult DCD transplant occurred in 2019, and DCD hearts rose from 3% of transplants in 2020 to 15% in 2023. In DBD donation, hemodynamic support continues until arrest, minimizing warm ischemia. In DCD, circulatory death must occur before procurement, resulting in longer warm ischemic exposure and limited in situ functional assessment. Primary graft dysfunction (PGD) is severe ventricular dysfunction within 24 hours requiring inotropes or MCS, not explained by surgical issues, pulmonary hypertension, or hyperacute rejection. This patient's hemodynamics and imaging supported early biventricular PGD. Transient PGD is defined by MCS discontinuation within 72 hours. Risk factors include pretransplant ECMO, DCD donation, donor acidemia, and prior sternotomy, two of which applied to our patient. UNOS data show PGD rates of ~5.6% in DBD vs ~10% in DCD hearts, yet 90-day outcomes remain comparable. Critically, patients with severe PGD requiring ECMO can still achieve favorable long-term results when myocardial recovery occurs early, as seen here. As

DCD use expands, anticipating PGD risk and optimizing perioperative management will be essential. Further studies are needed to refine donor and recipient selection and identify modifiable risk factors.

Images



End-diastolic view demonstrating RV failure following CPB separation

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#FR 2 – Kiosk 2 – Poster 4

VAD Insertion in the Systemic Right Ventricle of a Patient with Congenitally Corrected Transposition of the Great Arteries

Presenting Author: Andrew Moreno, MD, Resident Physician, Columbia University Irving Medical Center

Introduction

Congenital heart defects (CHD) are becoming more commonly seen among an adult population of patients undergoing cardiac and non-cardiac surgery. Congenitally Corrected Transposition of the Great Arteries (ccTGA) accounts for < 1% of CHD with a prevalence of ~0.02 to 0.03 per 1000 live births and features an atrioventricular and ventriculoarterial discordance that results in oxygenated blood flow to a systemic right ventricle (RV) [1]. The following is a case of a patient with ccTGA requiring ventricular assist device (VAD) implantation in the setting of systemic RV failure.

Case Presentation

A 47-year-old male with ccTGA (Figure 1) presented with abdominal pain, nausea, and an episode of syncope. Initial work-up was notable for TTE showing biventricular function reduced from baseline and right heart catheterization demonstrating pulmonary hypertension (pHTN, mean PAP 53mmHg), and reduced cardiac index (CI 1.5 L/min/m2) consistent with acute decompensated heart failure. A milrinone infusion was started and an intra-aortic balloon pump was placed with improvement in filling pressures. He then underwent HeartMate 3 VAD insertion. The patient was received in the OR on milrinone 0.2mcg/kg/min. An arterial line was placed and general anesthesia was induced. Inhaled nitric oxide was empirically started at 20ppm after induction with maximum mean PAP of 54mmHg. Pre-operative TEE demonstrated severe systemic RV dysfunction, severe systemic atrio-ventricular (tricuspid) valve regurgitation, and moderate LV dysfunction. The transition to cardiopulmonary bypass (CPB) was done via cannulation of the aorta and left femoral vein. The VAD was inserted into the systemic RV and dobutamine was started at 5mcg/kg/min prior to separating from CPB to support LV function. Post-operative TEE showed moderate to severe reduction of systemic RV function and improved tricuspid valve regurgitation. The VAD inflow cannula was visualized at the RV apex with laminar flow in the ascending aorta (Figure 2). Mixed venous oxygen saturation was 77% with mean PAPs ~20mmHg and CI of 2.2 L/min/m2.

Discussion

Discussion: Patients with surgically or congenitally corrected TGA are now expected to live into the third or fourth decades of life, but they commonly develop sequelae from a systemic RV. Chronic pulmonary congestion from RV failure can predispose these patients to pHTN. This requires careful anesthetic management to optimize systemic RV preload and LV systolic function. Furthermore, the anatomy of the RV makes VAD insertion more challenging as it is often more trabeculated and can readily cause inflow obstruction [2]. Thus, more frequent visualization with TEE to ensure proper cannula positioning is imperative. Other anesthetic concerns include arrhythmias, namely AV block or SVT, resulting from abnormal conduction pathways or AV node position [3]. There is limited data regarding the intraoperative

management and outcomes of patients with ccTGA and VADs. Therefore, metanalyses of the studies that do exist will be necessary as more of these patients present for surgery.

Images

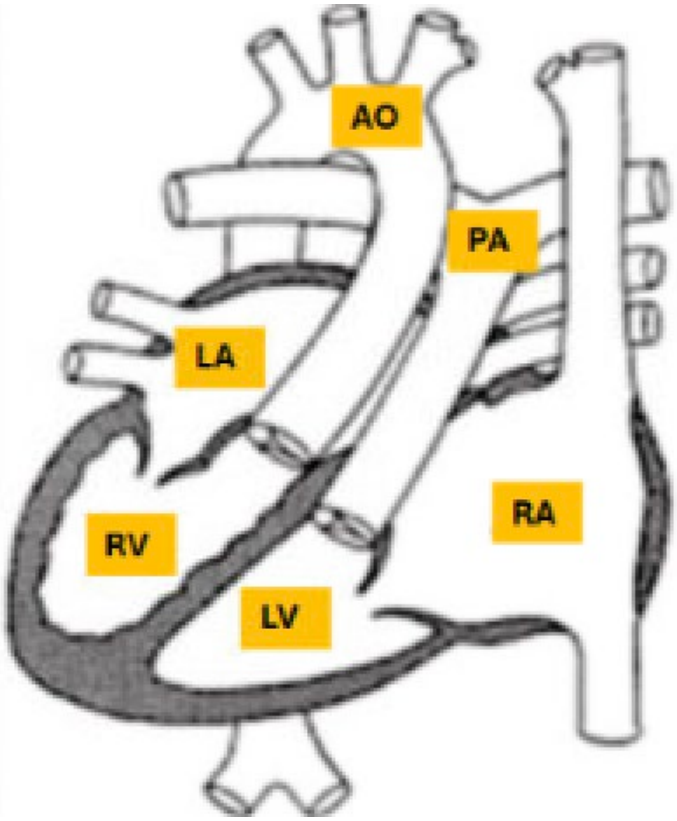


Figure 1. Schematic of the patient's ccTGA anatomy with situs inversus and right-sided right ventricle and aorta (I-D-D). Systemic venous blood returns to the right atrium (RA) and passes through the mitral (bicuspid) valve to the left ventricle (LV) before being pumped to the pulmonary arteries (PA). Oxygenated blood from the pulmonary circulation empties into the left atrium (LA) and traverses the systemic atrio-ventricular (tricuspid) valve into the right ventricle (RV), which then pumps blood to the systemic circulation via the aorta (AO).

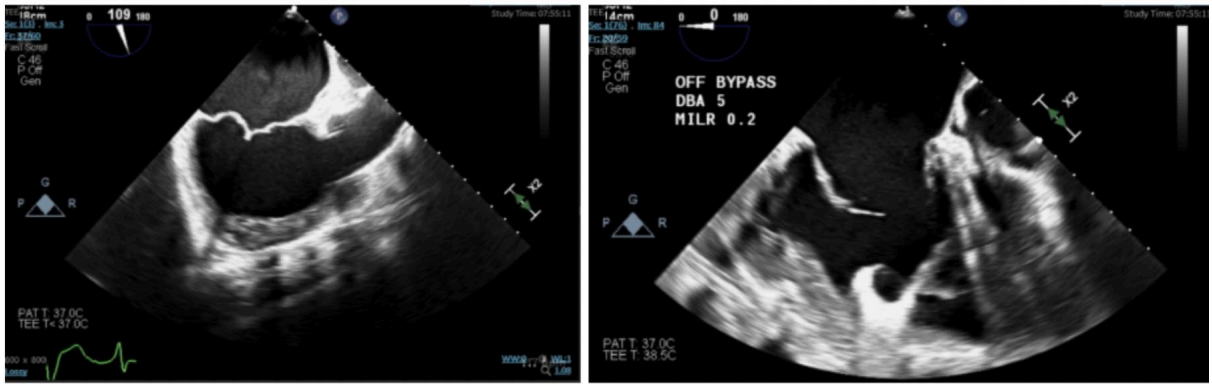


Figure 2. TEE images focused on the systemic right ventricle (RV). Left: Aortic valve long axis review with the systemic RV contiguous into the aorta. Right: HeartMate 3 inflow cannula in the systemic ventricle.

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#FR 2 – Kiosk 2 – Poster 5

Post-Myocardial Infarction Ventricular Septal Defect and Cardiogenic Shock: A Multimodality Mechanical Circulatory Support Bridge to Repair

Presenting Author: Itzel J. Lopez-Hinojosa, MD, University of Michigan

Introduction

Post-myocardial infarction (MI) ventricular septal defect (VSD) complicated by refractory cardiogenic shock carries high mortality. Surgical repair is delayed to allow for tissue demarcation; this requires prolonged hemodynamic stabilization with mechanical circulatory support (MCS). In this case, sequential MCS strategy from intra-aortic balloon pump (IABP) to veno-arterial extracorporeal membrane oxygenation (VA-ECMO) to Impella 5.5 was utilized to optimize a patient for delayed VSD repair.

Case Presentation

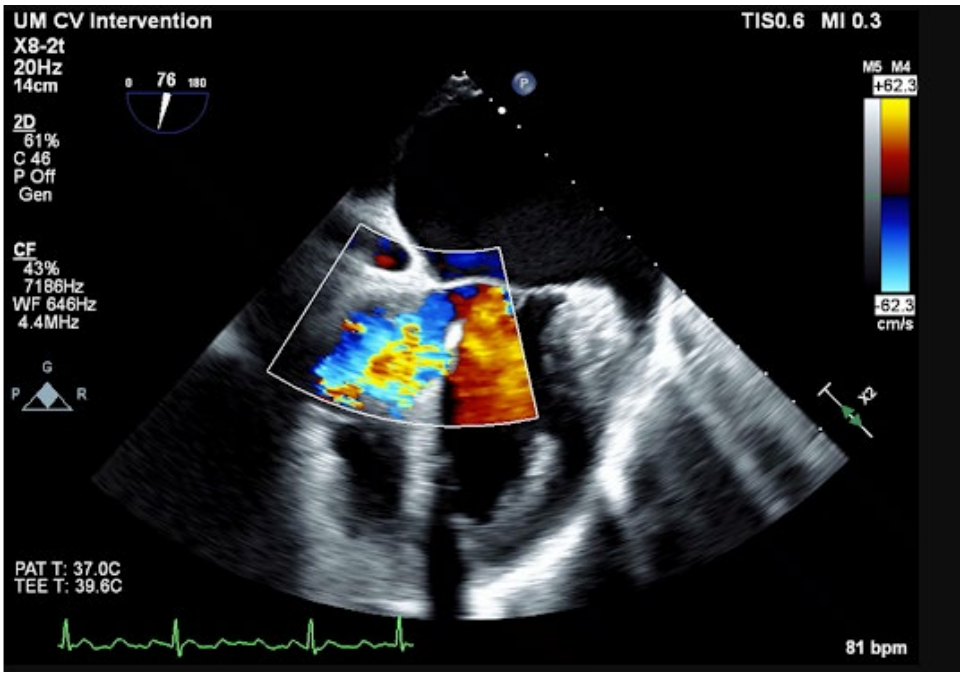
A 66-year-old female presented with an inferior wall MI. Stent placement to an occluded RCA was complicated by ventricular fibrillation arrest and cardiogenic shock, requiring vasopressors and IABP. Imaging confirmed a large VSD. The patient was transferred to our hospital and required immediate VA-ECMO. To allow for stabilization prior to definitive repair with greater mobility and continued ventricular unloading, the patient was transitioned to an Impella 5.5 via the right axillary artery in the OR and IABP and ECMO were discontinued. Intraoperative TEE was used for guidance for placement as well as monitoring of VSD shunt. Her ICU course included managing atrial fibrillation, monitoring for hemolysis, and shunt physiology. On post-MI day 18, she underwent successful two-patch VSD repair, 2-vessel CABG, and Impella removal. Intraoperatively, anesthetic strategies were used to maintain LV function and assess for residual VSD shunt after patch closure. Induction included high-dose opioids to maintain hemodynamic stability and preserve LV function, while taking meticulous care to exclude bubbles from all lines to prevent air embolism. Cangrelor was discontinued preoperatively to minimize bleeding risk; we transfused 5 units pRBC and 2 units plasma. While on cardiopulmonary bypass (CPB), the Impella was removed via the axillary artery. Given the inherent myocardial vulnerability, we weaned from CPB slowly, ready for an immediate return to CPB if needed. Successful weaning required inotropic support with epinephrine and milrinone, and vasopressor support with norepinephrine and vasopressin. Primary intraoperative goals of maintaining satisfactory LV function and confirming complete VSD patch closure were successfully achieved.

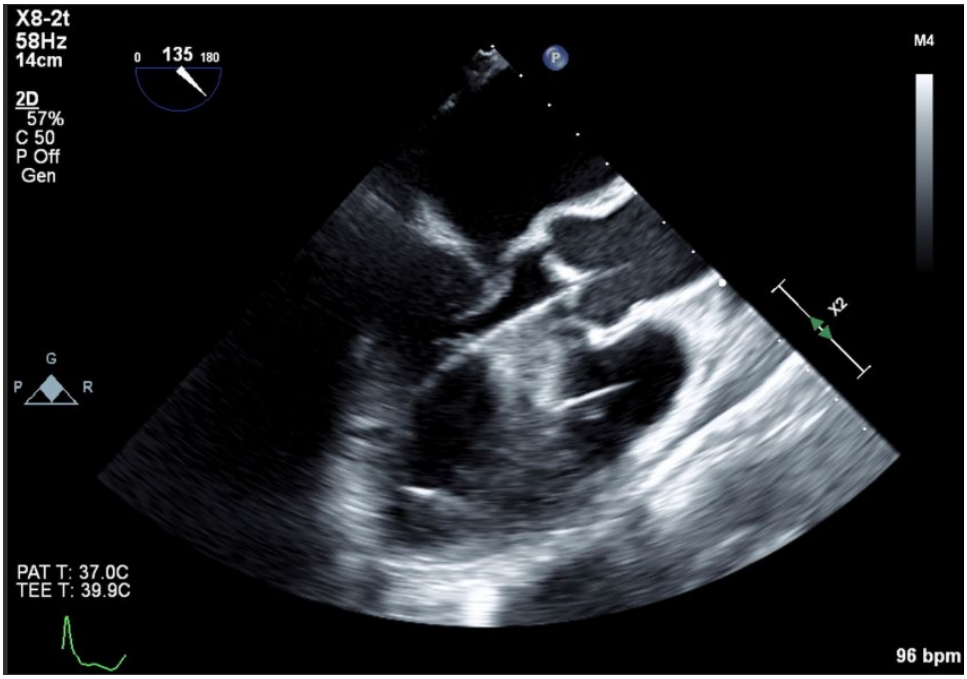
Discussion

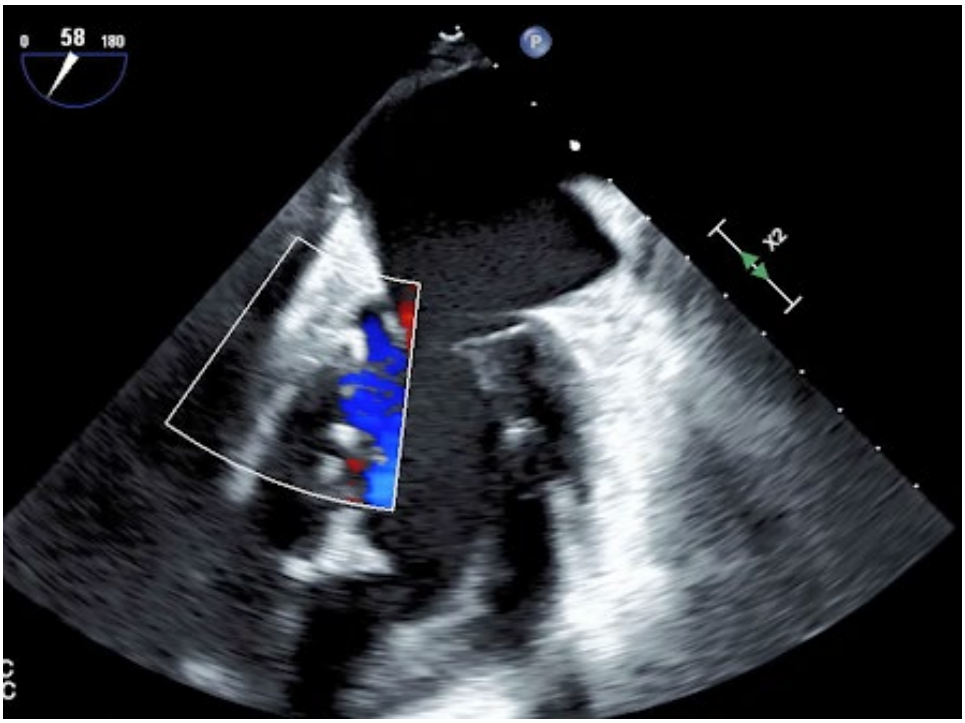
The use of the Impella 5.5 with a large post-MI VSD is a unique, infrequently documented challenge. This case highlights the rationale for a sequential MCS strategy to bridge from VA-ECMO to the less invasive Impella 5.5 for stabilization prior to surgery, promoting myocardial recovery and tissue stability. Surgical outcomes are dramatically improved by delaying repair, ideally by over 2 weeks to allow for tissue healing and demarcation (mortality of ~20% for delayed vs. ~70% for immediate repair). Impella use with a large VSD is complex due to the risks of shunt exacerbation and device-related hemolysis and positioning challenges which has to be carefully managed with echocardiography. The transition from ECMO/IABP to Impella

demanded aggressive hemodynamic management: specifically maintaining MAP with vasopressors and managing rapid changes in LV loading throughout the MCS transition. The definitive anesthetic and operative strategy for VSD repair and CABG prioritized maximizing cardiac stability and was ultimately successful.

Images







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#FR 2 – Kiosk 2 – Poster 6

Navigating the Unexpected: Anesthetic Challenges of Lung Transplantation in the Setting of a Left-Sided Superior Vena Cava

Presenting Author: Cynthia Tan, MBChB, MSc, Cardiothoracic Anesthesia Fellow, NYU Langone Health

Introduction

A persistent left-sided superior vena cava (PLSVC) is one of the most common thoracic venous congenital malformations affecting 0.3-0.5% of the general population. A PLSVC with absent right-sided superior vena cava (RSVC) is even more uncommon, an incidence of 0.07-0.13% of patients. In general, a PLSVC is associated with RSVC. 80-90% of PLSVC drain into the coronary sinus and the remainder drain into the right atrium. A PLSVC is usually asymptomatic and found incidentally in patients. However, it does have multiple clinical implications. PLSVC is associated with a dilated coronary sinus, arrhythmias, cardiac congenital abnormalities, difficulties in pulmonary artery catheterization, and challenges in giving cardioplegia on cardiopulmonary bypass (CPB). At our institution, lung transplantations are primarily done without CPB or ECMO. In all patients undergoing lung transplantation, a pulmonary artery catheter (PAC) is placed to guide management during the perioperative period.

Case Presentation

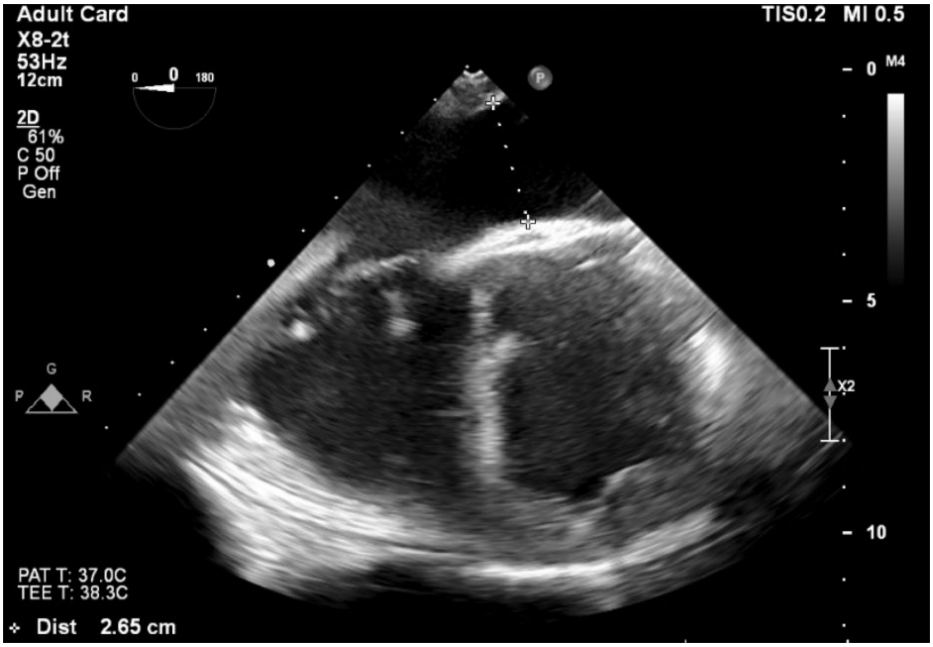
A 66-year-old male with a history of interstitial lung disease on home oxygen, atrial fibrillation, and pulmonary hypertension, presented for a bilateral lung transplant. Pre-transplant CT scan found a left-sided SVC draining into a dilated coronary sinus and no right-sided SVC is present. TTE also found a markedly dilated coronary sinus entering the RA, typical of a persistent left SVC. Following induction and intubation with a left-sided double lumen ETT, a left internal jugular vein central line was placed. There was difficulty with placing the (PAC) due to the patient's anatomy. After discussion between anesthesia and the surgical team, the decision was made to have the surgeon directly cannulate the pulmonary artery for pressure readings. The operative course was further complicated by the decision to cannulate the patient for VA ECMO during the pneumonectomy due to hemodynamic compromise secondary to compression of the PLSVC. The patient was able to be decannulated from VA ECMO at the end of the case and was brought to ICU intubated.

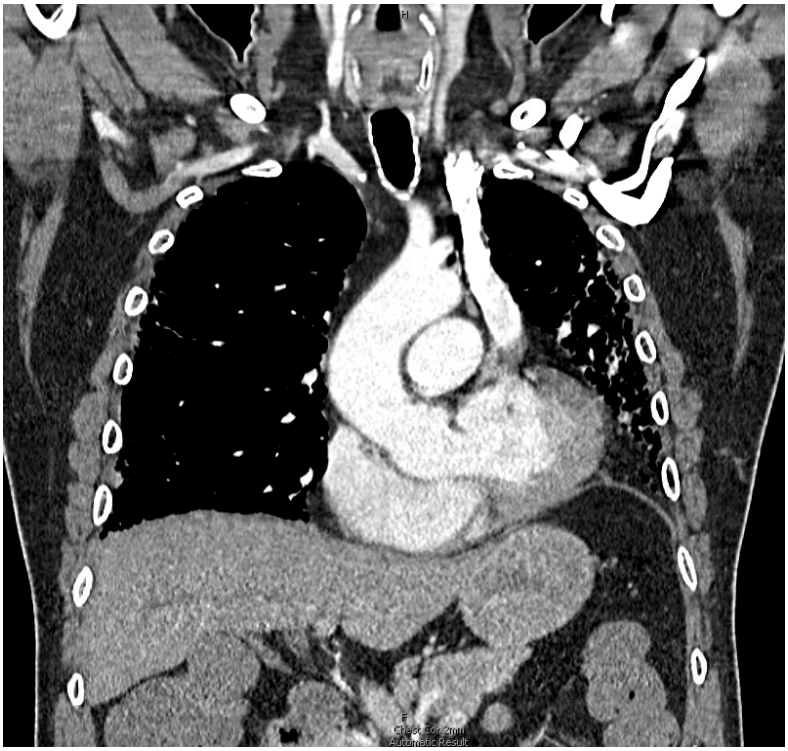
Discussion

An isolated PSLVC in our patient led to a challenging pre- and intra-operative anesthetic management due to the inability to place the PAC and hemodynamic compromise resulting in cannulation for VA ECMO. A PAC is a crucial step of lung transplantation according to the American Association for Thoracic Surgery. Although the pulmonary artery can be directly measured via a pressure catheter inserted into the vessel, it requires the vessel to be exposed after sternotomy. This approach allows for the PA pressure to only be monitored when the vessel is exposed and as a result, does not facilitate pre-sternotomy and post-transplantation management. Lung transplant is often performed without the use of CPB and ECMO if there is no hypoxemia, hypercarbia, or hemodynamic instability. Although the use of CPB and ECMO may facilitate

more favorable surgical conditions, there are risks including bleeding, infection, reoperation and vascular damage. Our case highlights the unique perioperative challenges in a patient with an isolated PSLVC undergoing lung transplant. The collaborative efforts between anesthesia and surgery played a crucial role in guiding this patient safely through the operation.

Images





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#FR 2 – Kiosk 3 – Poster 1

Ebstein Anomaly in Adulthood: Anesthetic Considerations and Intraoperative Surgical Decision-Making

Presenting Author: Anna V. Condacse, MD, Resident, Loma Linda University

Introduction

Ebstein anomaly (EA) is a rare congenital malformation of the tricuspid valve (TV) resulting in tricuspid regurgitation (TR) and right ventricular (RV) myopathy, accounting for < 1% of congenital heart defects.[1] Adult survivors vary widely in severity, yet morbidity and mortality rises with age.[2] Over the past decade, cone repair has become the preferred technique, with significant reduction in TR, remodeling of the RV, and low reoperation rates.[1,2] Although cone repair achieves excellent mid-term results, residual or recurrent TR can occur, particularly in adults with severe RV remodeling. Durability beyond 10 years in adults remains uncertain.[3]

Case Presentation

A 56-year-old female with EA presented for TV repair via cone procedure and pacemaker insertion. Comorbidities included prior Wolff-Parkinson-White ablation, paroxysmal atrial fibrillation, hypertension, obesity, and restrictive lung disease. Transthoracic echocardiography revealed preserved left ventricular function, severe right atrial and ventricular enlargement, apical displacement of septal leaflet (4.4 cm), and severe TR. Electrocardiogram showed sinus arrhythmia, bi-atrial enlargement, and right bundle branch block. Standard monitors were supplemented with a second pulse oximeter, cerebral oximetry, bispectral index, and defibrillator pads. A pre-induction arterial line was placed followed by cardiac induction with fentanyl (150 mcg), lidocaine (80mg), etomidate (8 mg), propofol (20 mg), and rocuronium (80 mg). After intubation, a central line was placed and transesophageal echocardiography (TEE) demonstrated severe TR and massive RV dilation (Figure 1). Cone repair was performed over 180 minutes on cardiopulmonary bypass (CPB). After separation from CPB, the patient was maintained on milrinone (0.4 mg/kg/min), epinephrine (0.2mcg/min), and inhaled nitric oxide (iNO) (20 ppm) with DDD pacing at 100 BPM. Although the TR improved, there was still moderate TR (Figure 2). Multidisciplinary intraoperative consultation between cardiac anesthesia, cardiothoracic surgery, and cardiology teams led to the conversion to TV replacement. A second CPB run (66 minutes) was initiated followed by support with milrinone, epinephrine, vasopressin (0.04 U/min), and iNO. TEE demonstrated improved TV function (Figure 3). The patient was transferred to the intensive care unit intubated with AAI pacing at 90 BPM.

Discussion

Cone repair provides excellent early and mid-term outcomes, yet durability remains uncertain in those with longstanding right heart dilation. Residual TR at discharge occurs in 36% of cone repairs (mean age 16.5 years) versus 5% (P=0.010) after replacement (mean age 40.8 years), with similar 5-year survival, but greater RV deterioration after replacement.[3] RV remodeling after cone repair may improve, yet residual TR may persist in adults with tethered leaflets and atrialized RV.[1] Hemodynamic management prioritizes maintaining

preload and sinus rhythm, while avoiding increases in pulmonary vascular resistance. Given the patient's age, chronic remodeling, and intraoperative TEE findings, valve replacement was chosen to prevent early reoperation and mitigate volume overload, consistent with risk-benefit analyses favoring replacement in adults with residual TR (Table 1).

Images

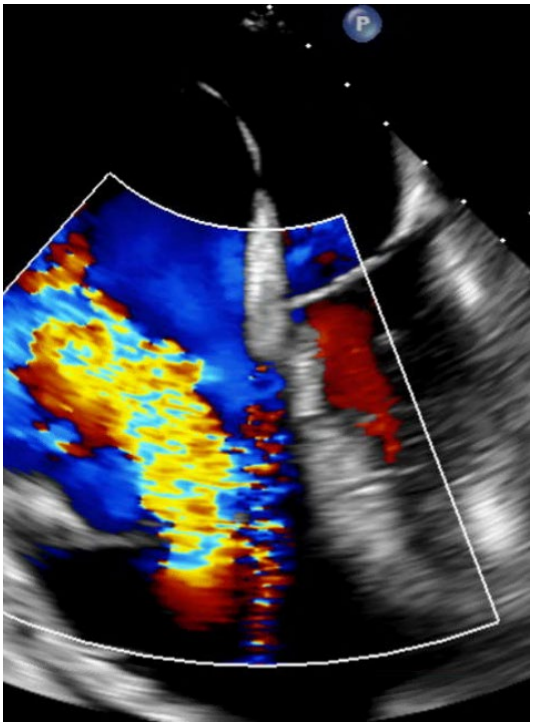


Figure 1. TEE of severe TR prior to cone procedure and CPB

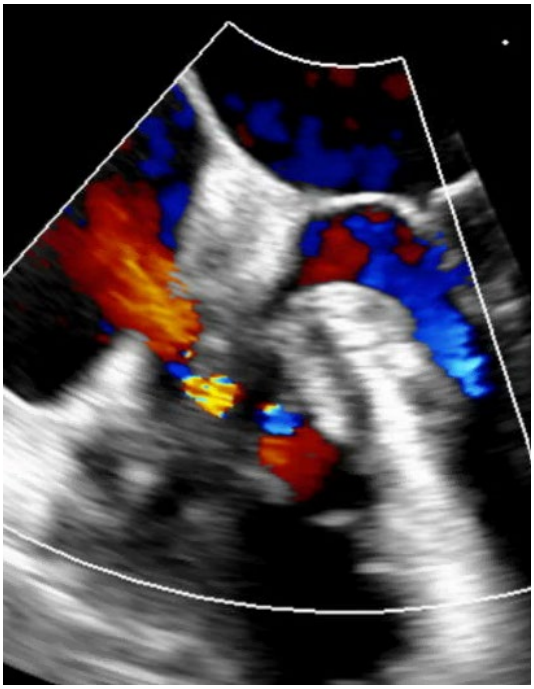


Figure 2. TEE of moderate TR after CPB for cone procedure

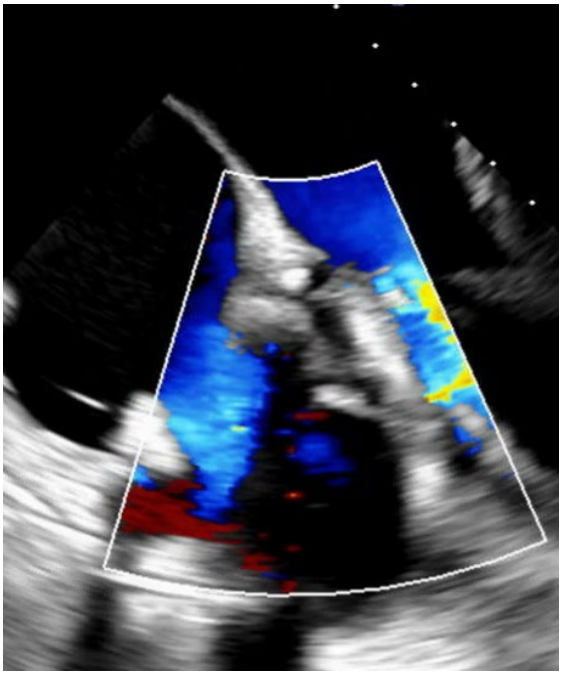


Figure 3. TEE of improved TR after second CPB for TV replacement

Factor	Cone Repair	Tricuspid Valve Replacement
Residual TR at discharge	36% >mild TR (Boyd 2023)	5% (Boyd 2023)
5-year survival	≈96%	≈95%
Reoperation rates	Low, comparable to TVR	Comparable
RV remodeling	Favorable if durable repair	Predictable elimination of volume load
Prosthesis issues	Avoided	Anticoagulation / degeneration risk
Best candidates	Younger patients, less RV dilation	Older adults, severe remodeling

Risk Benefit Analysis Table

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#FR 2 – Kiosk 3 – Poster 2

A Dangerous Detour: Intracardiac Cement Embolization after Spinal Surgery

Presenting Author: Maria Saraf, MD, CT Anesthesiology Fellow, University of Maryland School of Medicine

Introduction

Procedural cement is often used in orthopedic procedures for structural support. However, cement leakage into the vertebral plexus and beyond can lead to various complications, including intracardiac emboli[1]. We present a case of a 61-year-old female who underwent spinal fusion that was complicated by cement embolization through the venous system and into the right atrium.

Case Presentation

A 61-year-old female with history of CAD, aortic stenosis s/p TAVR, COPD, tobacco use and chronic T11 compression fracture presented for an elective T8-L1 fusion with cement augmentation. After an uneventful surgery, the patient was persistently tachycardic with HR 110s. CTA chest done to rule out pulmonary embolism showed extravasated cement extending from the T9 vertebral body to the azygous vein, SVC, and into the right atrium (*Figure 1*). TTE only showed normal biventricular function with trace TR. Cardiac surgery was consulted and the patient presented for removal of foreign body and CABG x2 (for concomitant CAD). After uneventful induction, intubation and radial arterial line placement, a femoral central line was placed given location of the foreign body in the SVC. Intra-op TEE showed a thick, linear echodensity in the SVC and RA that did not traverse the tricuspid valve (*Figure 2 & 3*). After sternotomy and harvesting of LIMA and SV, cannulae were placed in the aorta, proximal SVC and IVC. Incisions were made in the RA and SVC, and cement was removed to the level of the azygous vein which was then ligated (*Figure 4*). Aortic cross-clamp was applied and CABG x2 was completed. After weaning from CPB, TEE showed absence of the linear echodensity. Patient recovered well post-op and was discharged home with PT on POD 14. Repeat imaging 2 months after discharge showed intact spinal hardware with good alignment.

Discussion

Cement extravasation is a common complication of cement-related procedures: 23% of patients have extravasation into central veins while 0.3% have symptomatic intracardiac emboli (ICE)[2]. Fracture anatomy, cement viscosity and cement volume are risk factors most strongly correlated with extravasation[1]. For patients with ICE, conservative management typically consists of surveillance and short-term anticoagulation while more aggressive treatment involves endovascular or open procedures for cement removal with possible repair[2]. While formal guidelines do not exist, patients are risk-stratified according to their hemodynamic stability, embolus characteristics, and complication severity[2]. Thorough preoperative imaging and interdisciplinary consultation then guide individualized management. In our case, the patient's symptomatic presentation, along with the potential for further complications, warranted open removal with concurrent bypass surgery. Careful titration of medications during induction is paramount, as is anticipating potential decompensation from acute, intracardiac mechanical injury. The general anesthesiologist should also consider ICE in the differential diagnosis for sudden intraoperative hemodynamic instability during

cement procedures. Further studies are needed to investigate cement properties and injection techniques that can reduce the initial risk of extravasation and improve patient safety.

Images

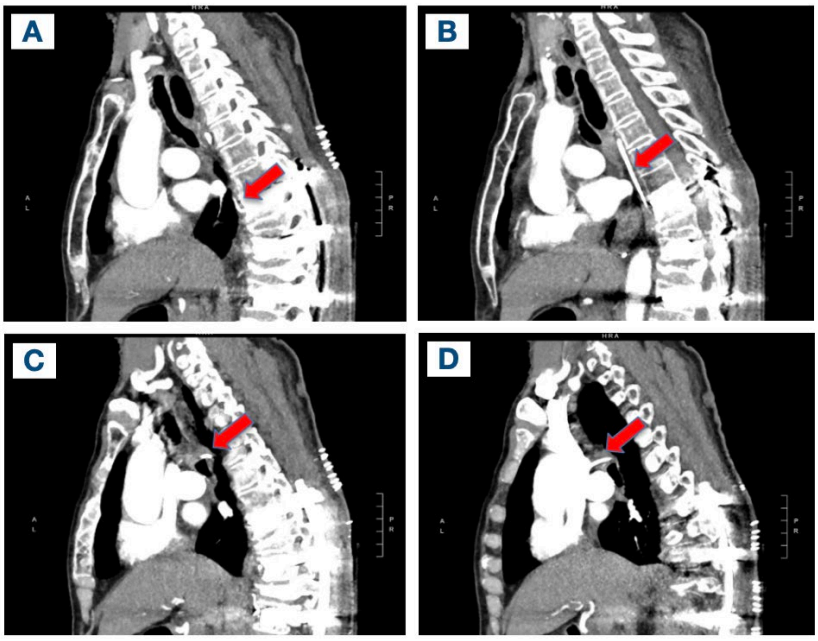


Figure 1: Chest CT scan sagittal views showing cement extravasating from the T9 vertebral body to the azygous vein, SVC, and into the right atrium.

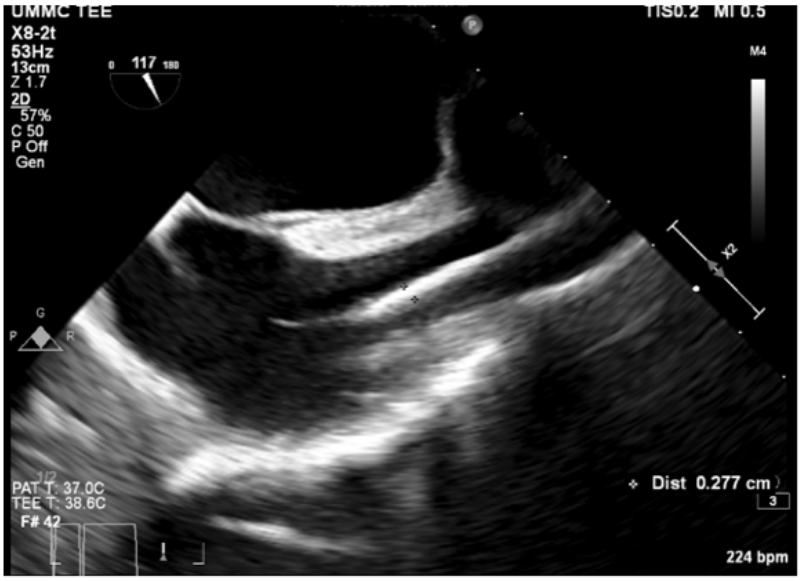


Figure 2: Intra-op TEE bicaval view showing a thick, linear echodensity in the SVC and RA.

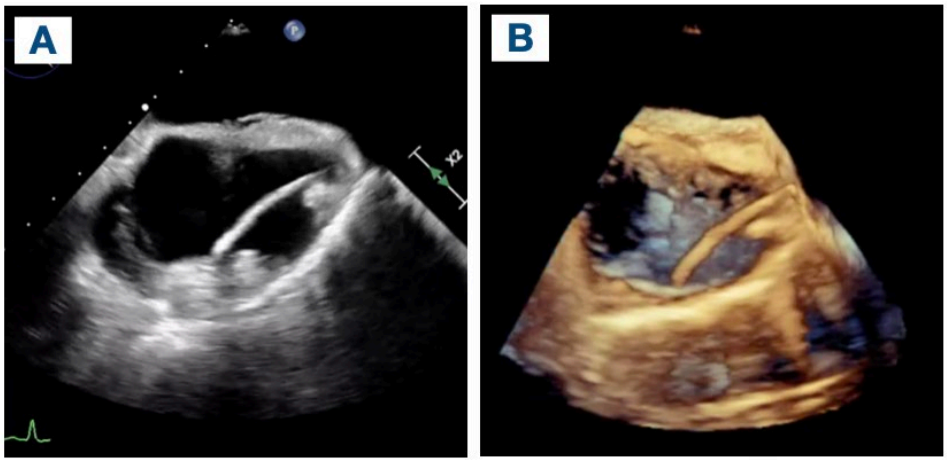


Figure 3: (A) 2D and (B) 3D TEE views of the linear echodensity in the bicaval view.

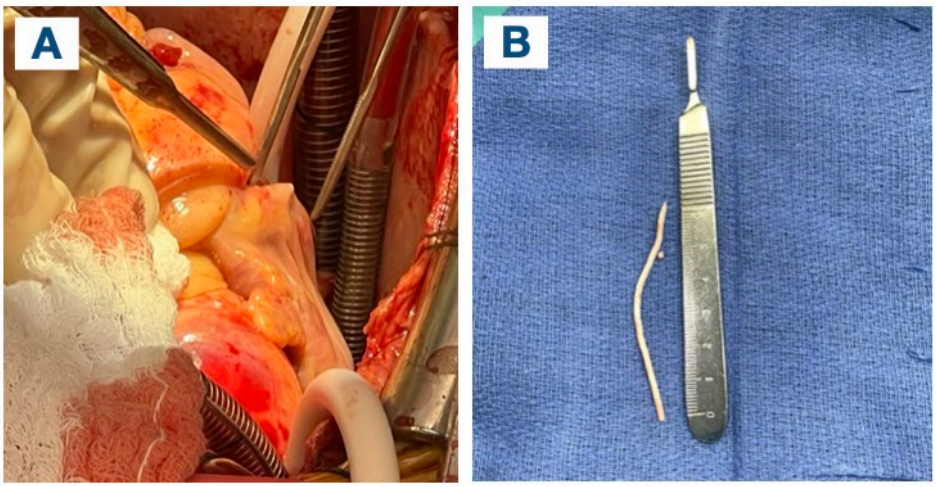


Figure 4: Linear cement seen (A) protruding from the right atrium, and (B) measuring 6cm once excised.

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#FR 2 – Kiosk 3 – Poster 3

Intraoperative TEE in the Assessment of Complex Aortic Root Pseudoaneurysm After Multiple Reoperations

Presenting Author: Aubrey B. Greer, MD, Adult Cardiothoracic Anesthesiology Fellow, MGB - Brigham and Women's Hospital

Introduction

Aortic root pseudoaneurysms are rare but serious complications following aortic surgery. Reported incidence varies widely, from less than 1% to as high as 25% after composite graft repairs, depending on surgical technique and surveillance intensity. They result from suture line dehiscence, infection, or tissue fragility and may lead to paravalvular leak, fistula formation, or rupture. Intraoperative TEE plays a critical role in characterizing anatomy, hemodynamic consequences, and guiding surgical planning.

Case Presentation

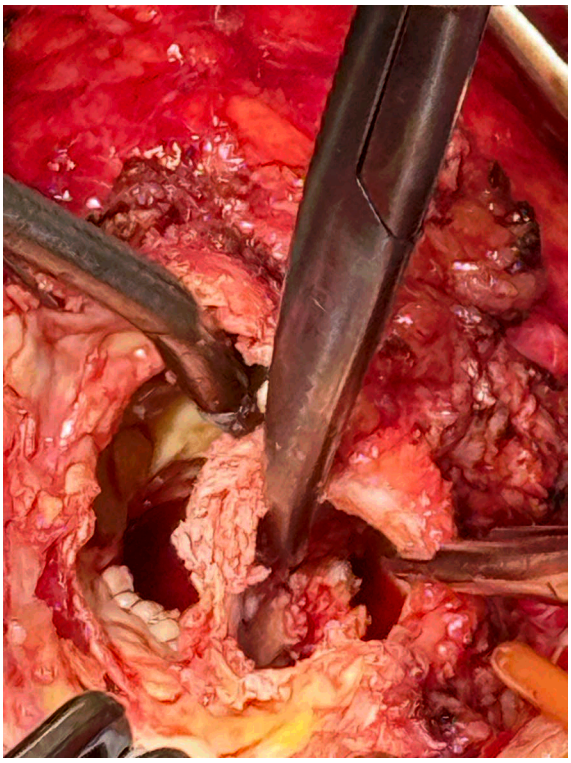
A 60-year-old male with hypertension, atrial fibrillation, and ascending aortic dissection approx. 10 years ago s/p mechanical Bentall presented for evaluation. His course was complicated by graft infection and pseudoaneurysm requiring redo aortic root/hemiarch replacement with a 26mm valved aortic homograft five years later. Operative notes documented coronary sinus dehiscence from the Bentall graft with post-bypass bleeding from the noncoronary sinus necessitating a Cabrol patch. Five years the re-operation with homograft replacement, he presented with symptomatic aortic insufficiency. Preoperative MRA and TTE demonstrated an enlarging pseudoaneurysm distorting aortic valvular architecture with severe aortic insufficiency. The patient underwent redo root, ascending and transverse arch replacement. Intraoperative TEE confirmed severely dilated LV with preserved EF (60%), restrictive filling, and moderately reduced RV function. A 1.5 cm pseudoaneurysm was identified extending approximately 5cm circumferentially from the noncoronary cusp position to the left coronary cusp position, communicating inferiorly with the LVOT. Color Doppler in ME LAX demonstrated severe aortic regurgitation with flow entering the paravalvular pseudoaneurysm in communication with the LVOT. 3D TEE with color localized the defect and complex paravalvular flow throughout the cardiac cycle. Post-CPB TEE demonstrated a well-seated mechanical aortic valve without paravalvular leak and normal biventricular function.

Discussion

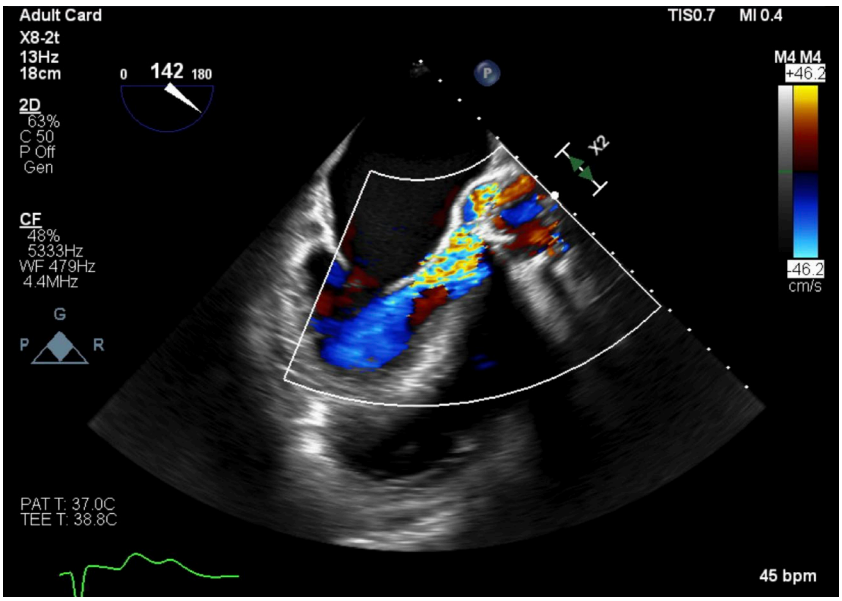
This case illustrates TEE's diagnostic value in complex aortic root pathology. Our findings were consistent with paravalvular/subvalvular leak with communication to a pseudoaneurysm causing severe aortic insufficiency. The Cabrol technique, used for intractable post-operative hemorrhage, involves placing a pericardial patch over the aortic graft space. In this case, the patch site may have became a nidus for pseudoaneurysm formation. TEE was instrumental in delineating pseudoaneurysm dimensions, identifying the leak site, assessing severity, and confirming adequate repair. 3D TEE with color Doppler proved valuable for spatial localization of complex regurgitant flow. This case underscores comprehensive

echocardiographic evaluation in patients with prior aortic surgery presenting with new aortic regurgitation.

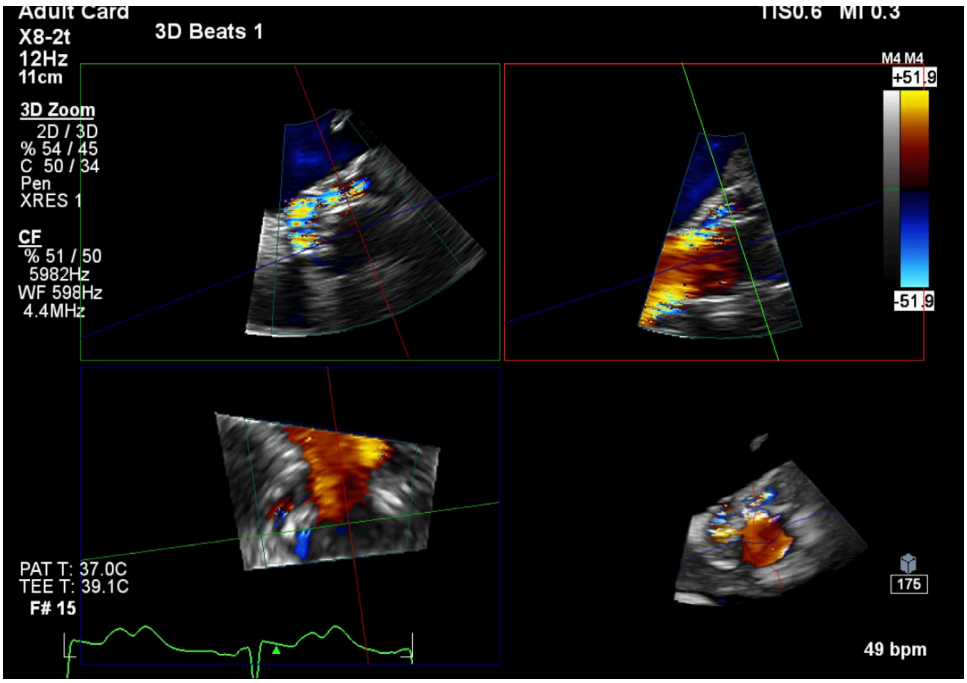
Images



Aortic Pesudoaneurysm. The true lumen is on the left and the pesudoaneurysm (right) is being exposed.



Parasternal long axis with color flow doppler demonstrating paravalvular leak.



Multiplanar reconstruction of aortic root pseudoaneusysm with color doppler.

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#FR 2 – Kiosk 3 – Poster 4

Management of Fontan Physiology and Klippel-Feil Syndrome with Prolonged Remimazolam Infusion for Leadless Pacemaker Placement

Presenting Author: Lai Jiang, M.D., Resident, Mayo Clinic

Introduction

Remimazolam has a very short duration of action, relative hemodynamic stability, and can be rapidly reversed with flumazenil. It is typically administered via boluses for short cases but may have an emerging role as an infusion.

Case Presentation

A 61-year-old male presented for leadless pacemaker placement for increasing epicardial pacing thresholds in the setting of high-grade AV nodal block. His past medical history was significant for dextrocardia, double-outlet RV status-post modified Fontan procedure with a main PA to SVC anastomosis and intra-atrial tube graft from IVC to SVC, and Fontan associated liver disease. He had a history of difficult airway requiring an intubating LMA in the setting of Klippel-Feil syndrome. The patient was brought to the procedural suite and his pacemaker was switched to asynchronous mode. Phenylephrine infusion was available with a low threshold to switch to a PVR sparing agent. An awake arterial line was placed using sedation with a remimazolam bolus. The ECMO team was on standby in anticipation of V-V ECMO rescue therapy. The surgical team established an 8 French catheter in both the left and right femoral veins — left for intracardiac echo and right for pacemaker placement. An adequate level of sedation was achieved with an initial infusion of remimazolam at 0.5 mg/kg/hr for 7 minutes before being decreased to 0.05 to 0.1 mg/kg/hr for most of the procedure due to intermittent transient apnea. Phenylephrine was maintained at 0.3 mcg/kg/min. The procedure was successfully completed without need for positive pressure ventilation or ECMO, while ensuring patient comfort during the seven-and-a-half-hour procedure. The patient was subsequently transported to the cardiac ICU for recovery. He would require baffle fenestration closure the following day due to persistent oxygen saturation around 80% despite HFNC at 60 LPM.

Discussion

Given the difficult airway and potential for profound hypoxic shunt with planned perforation of the intra-atrial graft, V-V ECMO was planned as rescue therapy. Maintaining spontaneous respirations was of utmost importance since Fontan circulation is sensitive to changes in PVR, and thus positive pressure ventilation. The short redistribution half-life and availability of rapid reversibility with flumazenil provided extra layers of safety. Notably, the clearance of remimazolam was likely slightly decreased due to our patient's Child-Pugh Class B status, since non-specific tissue esterase metabolism of this agent occurs predominantly in the liver. Our initial remimazolam infusion at 0.5 mg/kg/hr produced maximum BP effects within 5 minutes – MAP decreased from about 90 mmHg to 75 mmHg – before causing transient apnea. The BP effects remained despite decreasing the infusion to 0.1 mg/kg/hr, suggesting that BP effects manifest first and remain relatively stable despite traversing into deeper planes of anesthesia; this mirrored the results of a

study regarding plasma concentration vs clinical effects in healthy patients. In summary, our case demonstrates the relative stability and efficacy of remimazolam during an extended duration infusion in a patient with Fontan circulation. Remimazolam shows promise as an agent with a potentially desirable margin of safety even in patients with severe comorbidities.

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#FR 2 – Kiosk 3 – Poster 5

Holding on By a Thread: A Case of Mechanical Aortic Valve Dehiscence

Presenting Author: Elaine Prendiville, MD, Resident Physician – Anesthesia, OHSU

Introduction

A 64-year-old male with a history significant for HTN, RA on DMARDs, pneumonia 1 month prior with 2-week antibiotic course, recent dental procedures, craniotomy for meningioma resection 3 weeks prior, and a bicuspid aortic valve status-post 23 mm On-X aortic valve replacement 15 years prior presented with acute heart failure exacerbation. TTE demonstrated valve dehiscence with severe paravalvular leak (PVL) requiring emergent replacement.

Case Presentation

The patient presented with acute-on-chronic dyspnea, presyncope, and angina. Exam revealed a systolic ejection murmur, an early subtle diastolic murmur, absent LE edema, and a water-hammer pulse. Vitals demonstrated a pulse pressure greater than 100 mmHg. EKG found a new left bundle branch block and TTE showed abnormal rocking of the mechanical valve with severe PVL. Labs were notable for a troponin trend of 57 to 9,493. The patient was taken emergently for a re-do AVR. After intubation, a pre-surgical TEE showed the mechanical valve tethered to the posterior aspect of the LVOT and completely dehisced from the aortic position; moving into the LVOT during diastole and ejecting into the aortic root during systole. Before induction, radial pressure was 146/37 mmHg. The team agreed to lower SBP enough to maintain DBP greater than 30 mmHg until CPB initiation. On pump, the valve was found in the left ventricle with every suture torn, except one loosely anchored below the annulus. A bioprosthetic valve was exchanged successfully. The patient separated from CPB uneventfully and taken to the ICU, where his recovery resulted in discharge home on POD 8. The patient completed a course of antibiotics post-op for suspected infective endocarditis of oral origin given a history of recent dental procedures and negative blood/tissue cultures.

Discussion

This patient presented four months earlier to an OSH with similar acute-on-chronic heart failure symptoms. At the time, coronary angiogram was performed showing normal coronaries without comment on his known, long-standing PVL. Three years of yearly TTE's showed progressive LVEDVi increasing from 33 mL/m2 to 127 mL/m2, with the PVL each time being evaluated as "mild". Widening of pulse pressure and lowering of DBP were progressive over this time. It appears no further investigation into the site and quantity of PVL was performed, and the patient was cleared for neurosurgery 3 months later. At the time of his craniotomy radial DBP was 50 mmHg, indicating that the patient's valve likely acutely dehisced in the interval of presentation. This history serves as a cautionary tale that echo findings should be compared to prior studies and incongruencies warrant further investigation. This patient was high-risk for developing endocarditis with PVL and RA on DMARD's which should have prompted further investigation. Intraoperatively, this severe dehiscence presented a challenge. Careful maintenance of SVR, and therefore aortic diastolic pressure, was necessary to provide adequate coronary perfusion pressure in the setting of presumed elevated LVEDP

from long-standing PVL and acute valve dehiscence. The team tolerated higher-than-average SBP during dissection and cannulation prior to CPB to protect CPP to an at-risk LV.

Images



Figure 1. Mechanical aortic valve after removal.

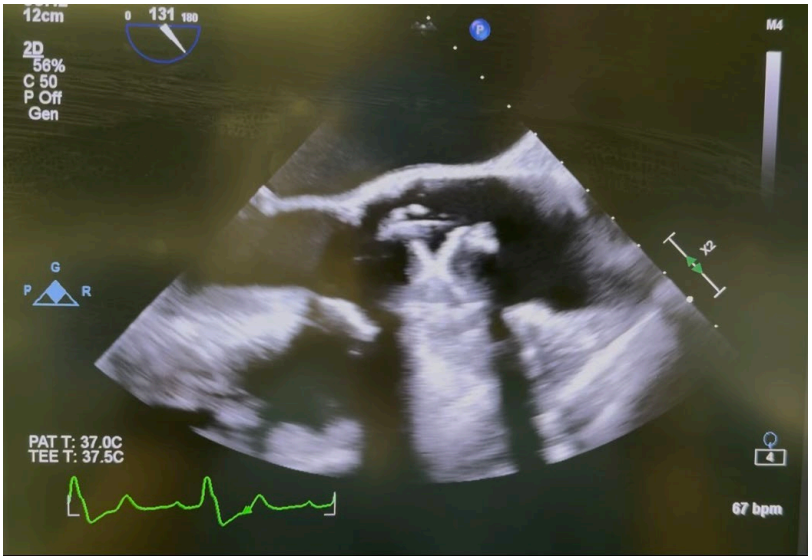


Figure 2. Transgastric long axis view in systole with mAoV in the aortic root.

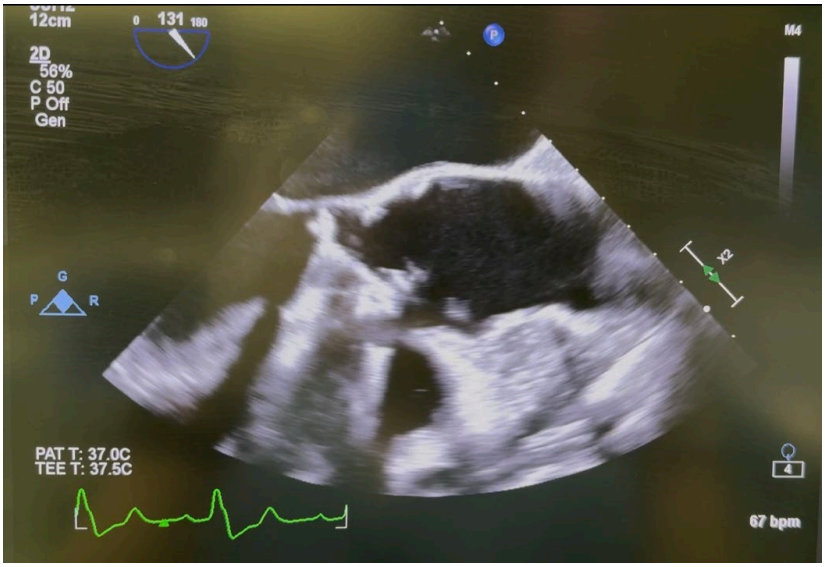


Figure 3. Transgastric long axis view in diastole with mAoV in the LV outflow tract.

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#FR 2 – Kiosk 3 – Poster 6

Case Report: Perioperative Management of Adult Fontan Patient Undergoing Non-Cardiac Surgery

Presenting Author: Miles Charles, Ponce Health Sciences University

Introduction

Fontan palliation is a surgical procedure for patients with congenital heart disease with a single functional ventricle. The procedure is performed during infancy in a stepwise process. First, the superior vena cava is connected to the right pulmonary artery (Glenn). Then, the inferior vena cava is connected to the right pulmonary artery, forming a complete cavopulmonary connection (Fontan).[1] Patients with Fontan physiology have a common atrium, one ventricle for systemic circulation, and passive pulmonary circulation through the Fontan conduit. Fontan palliation has enabled patients with once-fatal heart conditions to survive into adulthood. This can pose a challenge for anesthesiologists caring for these patients undergoing non-cardiac surgery.

Case Presentation

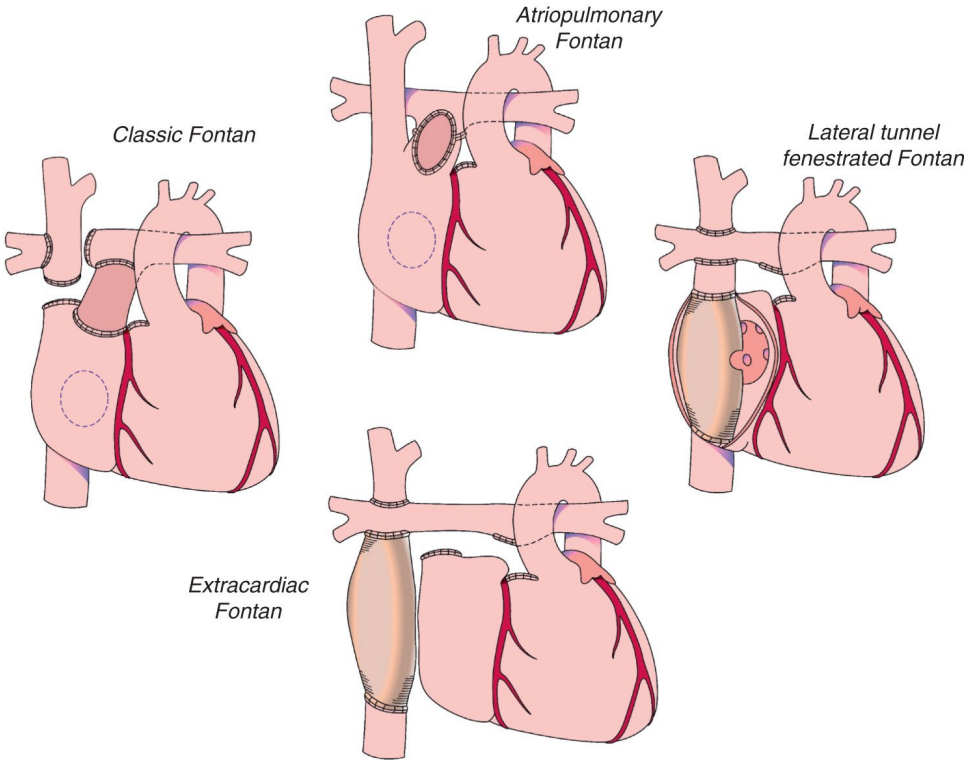
32-year-old female presenting for vaginal hysterectomy. She has a history of hypoplastic right heart and tricuspid atresia status post Glenn and extracardiac Fontan. She otherwise has a history of HTN, PONV and GERD. Prior to surgery, she was admitted to the ICU for volume and electrolyte optimization. The patient was brought to our cardiothoracic operating room. Cardiothoracic surgery and perfusion were on standby prior to induction. Standard ASA monitors were applied. Midazolam was administered and a pre-induction arterial line was placed. A standard IV induction was performed using fentanyl, lidocaine, propofol and succinylcholine. Propofol and remifentanil infusions were used for maintenance of anesthesia. The ventilator was set to PSV mode, and the pressure support was adjusted to deliver 6-8 ml/kg tidal volumes with PEEP set to 0 cm H_2O. FiO_2 was maintained at 1.0 throughout the case. Serial arterial blood gases were drawn to monitor acid-base status, oxygenation, and $PaCO_2$. The procedure was uneventful. Postoperative analgesia consisted of low-dose morphine, IV acetaminophen, ketorolac, and local anesthetic infiltration by the surgical team. The patient was extubated and taken directly to the ICU for recovery.

Discussion

Advances in treatment of congenital heart disease have enabled patients to survive into adulthood. These patients are at an increased risk of systolic dysfunction, arrythmias, and thromboembolic events, thus it is important to assess functional status prior to surgery.[1,2] The Fontan Conduit creates pulmonary blood flow down a pressure gradient. It is crucial to maintain forward flow by modulating PVR and venous return. To prevent elevations in PVR, it is important to avoid hypoxemia, hypercarbia, and acidemia.[3] Increases in intrathoracic pressure may also contribute to reduced pulmonary circulation. To minimize this, it is best to maintain low PEEP and avoid positive pressure ventilation by maintaining spontaneous respirations if feasible. Multimodal analgesia is recommended to minimize opioids and potential postoperative respiratory depression and hypercarbia. Finally, Fontan patients should receive postoperative care in the ICU for close cardiopulmonary monitoring.[3] Adult Fontan patients require specialized perioperative care given their

complex physiology. As survival outcomes improve for these patients, there will be an increased need for education and training for anesthesiologists to meet the needs of this growing population.

Images



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#FR 2 – Kiosk 3 – Poster 7

Percutaneous decommissioning of HeartMate 3 Left Ventricular Assist Device following recovery of cardiac function – An anesthesia case report

Presenting Author: Kwabena F. Lartey, MBChB MPH, Resident, University of Kansas Medical Center

Introduction

The HeartMate 3 Left Ventricular Assist Device (HM3 LVAD) provides a durable management option for end-stage heart failure. It acts as a bridge to myocardial recovery, heart transplantation, or as destination therapy. Consisting of a centrifugal pump-propelled conduit from the LV to the aorta, the HM3 LVAD is fueled by an external controller and batteries connected by a driveline. The cardiac anesthesiologist requires a good understanding of LVAD-induced hemodynamic alterations. Decommissioning of LVADs may be performed for device malfunction, palliative reasons, irreversible right ventricular (RV) failure, and recovery of native LV function[1,2]. It may provide a safer alternative for patients with high surgical risk.[3] We discuss the anesthetic management of a percutaneous decommissioning of the HM3 LVAD due to recurrent pump malfunction and recovery of LV function in a 73-year-old man.

Case Presentation

Our patient had a history of reduced ejection fraction (EF) after coronary artery bypass grafting, and a 3-year history of HM3 LVAD implantation complicated by pump failure from recurrent pulmonary thrombosis and pump ingestion. Antithrombotic management was complicated by multiple bleeding episodes in the setting of a distant history of intracranial and gastrointestinal bleeds. He was incidentally found to have good myocardial recovery with an estimated LV EF of 50%, and a decision was made to percutaneously decommission his LVAD. He received a general anesthetic due to his history of thrombosis and systemic bleeds, and possible intra-operative hemodynamic instability. His blood pressure was monitored using a post-induction femoral arterial line, inotropy maintained with dobutamine, and vascular tone with phenylephrine to a target mean arterial pressure (MAP) of 90 – 110 mmHg. Transesophageal echocardiography (TEE) and fluoroscopy were used to guide the proceduralist. Baseline hemodynamics were obtained, and the proceduralist performed a balloon occlusion of the LVAD outflow tract, followed by placement of three Amplatzer™ vascular plugs and decommissioning of the driveline. Post-occlusion hemodynamics were improved with a slight decline in his MAP (Figure 1, Table 1).

Discussion

Major intraoperative risks of percutaneous LVAD decommissioning include regurgitant flow through the LV outflow graft, LV/RV failure with cardiogenic shock, pulmonary or systemic thromboembolism, bleeding from access or graft sites, arrhythmias, and need for sternotomy. A balloon occlusion test is key to mimic the hemodynamic changes that will occur after permanent occlusion of the outflow graft. With reduced LV contractility, LV end-diastolic pressure can rise—temporarily or persistently—which may trigger RV failure, coronary ischemia, and cardiovascular collapse (Table 1). Management options include dobutamine or epinephrine to increase RV and LV stroke volume. TEE is useful to detect gross cardiac abnormalities and to

recharacterize RV/LV function or regurgitant lesions after permanent graft occlusion. The anesthesiologist must pay attention to possible life-threatening arrhythmias and treat those promptly. Finally, cardiac indices and filling pressures should be obtained pre- and post-occlusion to assess the overall heart function and need for continued inotropic or vasopressor support.

Images

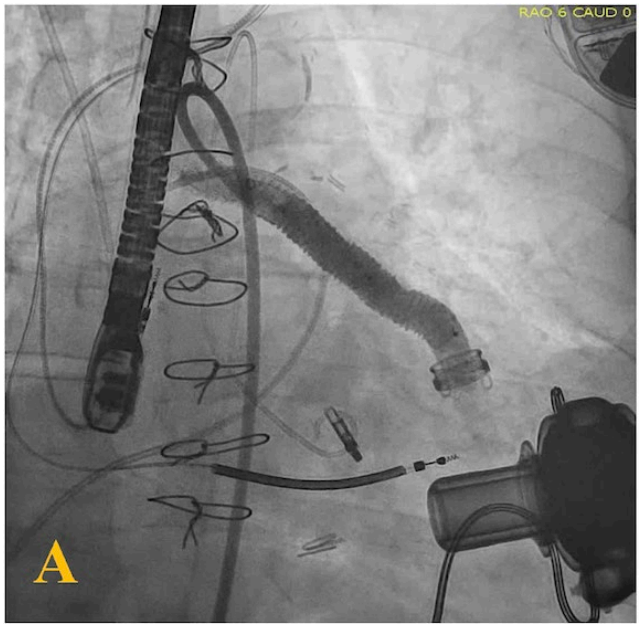


Figure 1A. Fluoroscopy image showing pre-occlusion of the LVAD outflow graft.

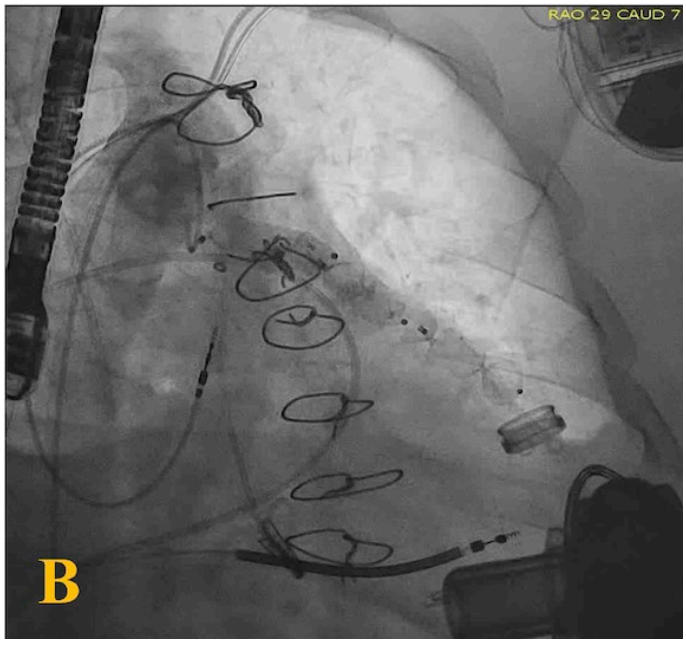


Figure 1B. Fluoroscopy image showing post-occlusion of the LVAD outflow graft.

	Pre-occlusion		Post-occlusion (successful recovery of LV Function)		Post-occlusion (failed recovery of LV function)
	Expected	Patient	Expected	Patient	
Possible Hemodynamic findings	MAP ↔ RAP ↑ RVP ↑ mPAP ↔/↑ PCWP ↔/↑ CO/CI ↔	MAP 108 RAP 9 RVP 37/10 mPAP 25 PCWP 14 CI 1.7	MAP ↔/↓ RAP ↔/↑ RVP ↔/↑ mPAP ↔/↑ PCWP ↔ CO/CI ↔	MAP 70 - 85 RAP Ø LVP* 101/17 mPAP 22 PCWP Ø CI 2.0	MAP ↓↓ RAP ↑↑↑ RVP ↑↑↑ mPAP ↑↑↑ PCWP ↑ CO/CI ↓↓
Determinants	Native LV systolic function RV systolic function LVAD speed and flow Volume status SVR		Native LV systolic function & compliance RV systolic function HR & rhythm; SVR Volume status Anesthetic agent; Vasopressors & Inotropes		

*Source: Authors; CI (cardiac index); CO (cardiac output); LVAD (Left Ventricular Assist Device); LVP (left ventricular pressure)MAP (mean arterial pressure); mPAP (mean pulmonary arterial pressure; PCWP (pulmonary capillary wedge pressure); RAP (right atrial pressure); RVP (right ventricular pressure); SVR (systemic vascular resistance); ↔ (normal); ↑ (increased); ↓ (decreased); Ø (not recorded); *Only recorded after pump occlusion.*

Table 1. Possible hemodynamic changes during percutaneous LVAD decommissioning

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#FR 2 – Kiosk 4 – Poster 1

Combined heart and liver transplant in a failing Fontan

Presenting Author: Danuka Ubayasena, MD, ACTA Fellow, Mount Sinai Hospital

Introduction

The Fontan procedure has increased the survival of children born with congenital heart disease. Adverse sequelae of Fontan circulation are thus becoming more prevalent. Specifically, Fontan-associated liver disease (FALD) due to increased central venous pressure and lack of pulsatility. This results in decreased hepatic perfusion and over time leads to cirrhosis. These patients will require a combined heart-liver transplant for definitive treatment. We present the case of a patient, who underwent a CHLT for FALD with a history of complex congenital heart disease.Our discussion focuses on the anesthetic considerations and perioperative challenges associated with such a case, particularly, in the context of underlying Fontan physiology.

Case Presentation

36 year old male, with a history of hypoplastic left heart syndrome with double outlet RV status post bidirectional Glenn and Fontan (2002) procedure. He presented with progressive shortness of breath, fatigue and decreased exercise tolerance. His symptoms were attributed to progressive RV failure complicated by chirrotic FALD. His symptoms improved with inotropic support, but was ultimately listed and underwent a combined heart-liver transplantation. The procedure was done in a sequential manner, and after 28 hours of total OR time, he was transported to the ICU on iNO, inotropic support (epinephrine, dobutamine) and peripheral VA ECMO.

Discussion

Due to the complex nature of these cases a methodical approach is key to predict, identify and address perioperative anesthetic challenges.

Preoperative:

- Multidisciplinary team to identify sequelae of heart failure/FALD, optimize patient and address comorbidities.

Induction:

- Discuss laterality/location of access sites with the surgical and perfusion teams.
- Upper and lower extremity arterial lines with large bore IV access above the diaphragm with PAC.
- Support the right ventricle on induction with every attempt to minimize PVR.
- Balance risk of aspiration, with cardio-depressant effects of anesthetics.

Pre-cariopulmonary bypass:

- With Fontan physiology, maintaining a transpulmonary gradient is key to ensure adequate cardiac output. This is predicated on adequate preload, minimizing PVR and maintaining sinus rhythm. A

CVP of 12–15 mmHg should be targeted to promote forward flow, although this will likely result in more bleeding due to higher venous pressures and development of venous collaterals.

- Prepare for re-sternotomy. Rapid transfuser primed with blood immediately available. Personnel available for immediate initiation of CPB.
- Consider the feasibility of TEE placement. Expect challenging and unfamiliar TEE imaging.

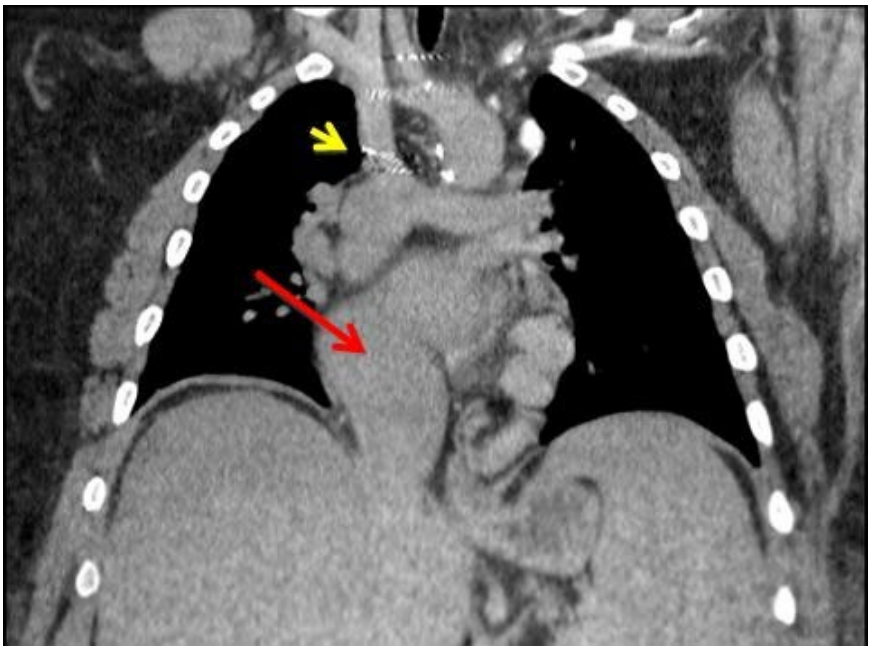
Heart phase:

- Chronotropic and inotropic support upon discontinuation of CPB. Satisfactory cardiac function is necessary to limit hepatic congestion.
- Vasoplegia is expected and treated with pressors.
- Inhaled nitric oxide pre-emptively to decrease RV afterload.
- VA-ECMO to assist cardiac allograft and help facilitate liver phase.

Liver phase:

- Aggressively correct metabolic derangement. Viscoelastic testing to guide hemostasis.
- Early treatment of hypotension, particularly during the reperfusion phase. Care should be taken to avoid prolonged decreases in coronary perfusion pressure.

Images



CT image of Fontan circulation with SVC connected to pulmonary artery

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#FR 2 – Kiosk 4 – Poster 2

When Intraoperative TEE Disagrees with Preoperative Testing in Subvalvular Mitral Stenosis

Presenting Author: Marcus Cluse, MD, MPH, The Ohio State University Wexner Medical Center

Introduction

Two-dimensional planimetry is the gold standard for evaluating native mitral valve (MV) stenosis (MS) but has limited value in prosthetic MS. Although mid-esophageal transesophageal echocardiography (TEE) is an excellent imaging modality for hemodynamic assessment of MS, it can be limited in its ability to evaluate the subvalvular apparatus (SVA)—and thus subvalvular stenosis—due to signal attenuation. Transgastric two-chamber (TG 2C) TEE imaging serves as a useful adjunct because it offers an alternate method to evaluate the SVA[1]. However, because hemodynamic assessment of MS is highly sensitive to flow dynamics, it is difficult to accurately estimate a patient's MS burden under general anesthesia (GA) [2]. Left-heart catheterization (LHC) directly and simultaneously measures left atrial (LA) and left ventricular (LV) pressures, providing an accurate measure of transmitral gradient, but it does not characterize the valve. To grade MS it is important to integrate all available studies and interpret each in the context of its respective strengths and limitations.

Case Presentation

A 53 y.o. woman with a history of mechanical MV replacement (MVR) in 2011 for MV regurgitation and HFpEF, presented with six months of worsening dyspnea. Preoperative transthoracic echocardiography (TTE) revealed severe aortic regurgitation (AR), severe MS (mean gradient (MG) 11 mmHg in the context of severe AR), trace tricuspid regurgitation (TR), and right ventricular systolic pressure of 97 mmHg. Poor TTE image quality limited further MV characterization. LHC demonstrated prosthetic MV MG of 13 mmHg, LV end-diastolic pressure of 18 mmHg, and no obstructive coronary disease. Based on the findings and the patient's symptoms, she was scheduled for a redo MVR, AV replacement, and possible TV repair. Intraoperative TEE demonstrated moderate MS (MG 6 mmHg), severe AR, and moderate TR. Assessment of the prosthetic MV showed appropriate leaflet opening; however shadowing from the prosthesis limited evaluation of the SVA. TG 2C imaging was inconclusive. Given the discordant findings on the echocardiograms, the preoperative LHC was reconsidered. Recognizing the limitations of TEE in this scenario and the strong support of the TTE by the LHC, our team elected to proceed with the MVR. On excision, a thick pannus on the ventricular side of the prosthesis was identified—a likely source of subvalvular stenosis.

Discussion

Though TTE is the imaging modality of choice for evaluating MS, image quality is a known limitation [1]. Intraoperative TEE can obtain superior images, but both modalities suffer attenuation in the presence of a prosthetic MV. The impact of GA on the hemodynamic evaluation of MS is heterogeneous and may lead to misclassification of MS severity due to altered loading conditions [2]. Severe AR may further confound measured gradients. LHC has been established as the most accurate method for calculating mean MV

diastolic pressure gradient [3]. While LHC findings do not replace echocardiography, they serve as an essential adjunct when TTE and TEE disagree—particularly in situations where a clear gold standard does not exist, such as subvalvular prosthetic valve MS. In these cases, LHCs provide a finite but reliable gauge that helps clarify an otherwise ambiguous clinical picture.

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#FR 2 – Kiosk 4 – Poster 3

Anesthetic Management of a Giant Anterior Mediastinal Teratoma

Presenting Author: Jesus Salcedo, Resident Physician, NYP - Columbia University Irving Medical Center

Introduction

Anterior mediastinal masses pose significant anesthetic risks due to potential airway compression and cardiovascular collapse (1,2). While pre-induction initiation of ECMO can provide hemodynamic stability, not all patients require it (1,3). We report a case of a giant cystic teratoma with cardiovascular mass effect (Fig. 1) that was successfully removed under general endotracheal anesthesia and thoracic epidural.

Case Presentation

A 23-year-old male presented with subacute chest pain, fevers, and weight loss. The chest CT revealed a 14×9×7cm anterior heterogenous mediastinal mass, spanning thoracic inlet to cardiac apex, displacing the SVC and aorta, and compressing the left lung (Fig. 2). There was no airway compression and the patient had no signs of SVC syndrome. Biopsy confirmed a mature teratoma, and he returned to the OR for resection. In the OR, a pre-induction arterial line, thoracic epidural, and lower-extremity IV were placed. Induction was performed incrementally with inhaled sevoflurane, ketamine, and small boluses of propofol and fentanyl. Gentle positive-pressure ventilation caused no hemodynamic changes, permitting cautious anesthetic deepening for intubation. Once the patient was under general anesthesia, a TEE was inserted and a right IJ central venous line and PAC were placed (Fig. 3). Paralytics were not administered until after the chest was opened. The teratoma was removed (Fig. 4) and hemodynamics remained stable throughout; no blood product transfusion or extracorporeal support was required. Postoperatively, he was extubated in the OR, transferred to the surgical ICU, and discharged home on POD 6 after an uncomplicated course.

Discussion

Anterior mediastinal masses require nuanced anesthetic planning due to unpredictable risks of airway collapse, venous collapse, or tamponade after induction (1,2). In our patient, tolerance of supine positioning, absence of airway compromise, and preserved hemodynamic stability supported proceeding without the need for awake intubation or ECMO. Given the tumor's proximity to major vessels, its mass effect on the heart and surrounding vasculatures, and the presence of pericardial effusion, the induction plan prioritized maintaining spontaneous ventilation and hemodynamic stability while securing the airway. This was achieved by combining inhaled induction with IV ketamine. Avoiding paralytics until the airway was secured and chest was opened remains standard for such high-risk cases (1). The arterial line, CVP, PA pressures, and TEE allowed for continuous hemodynamic monitoring. Pre-induction lower-extremity venous access ensured the ability to manage preload compromise from potential SVC obstruction, and a central venous catheter provided access for vasopressors and volume. Although thoracic epidural analgesia is sometimes deferred due to the potential for urgent heparinization if ECMO becomes necessary, multidisciplinary consensus deemed it appropriate in this scenario. Recent data suggest that ECMO use for mediastinal mass surgery should be individualized rather than automatic (3). This case reinforces that

comprehensive physiologic evaluation, rather than a default reliance on ECMO, can safely guide the anesthetic management of even large anterior mediastinal masses when risk is well-characterized (1-3).

Images

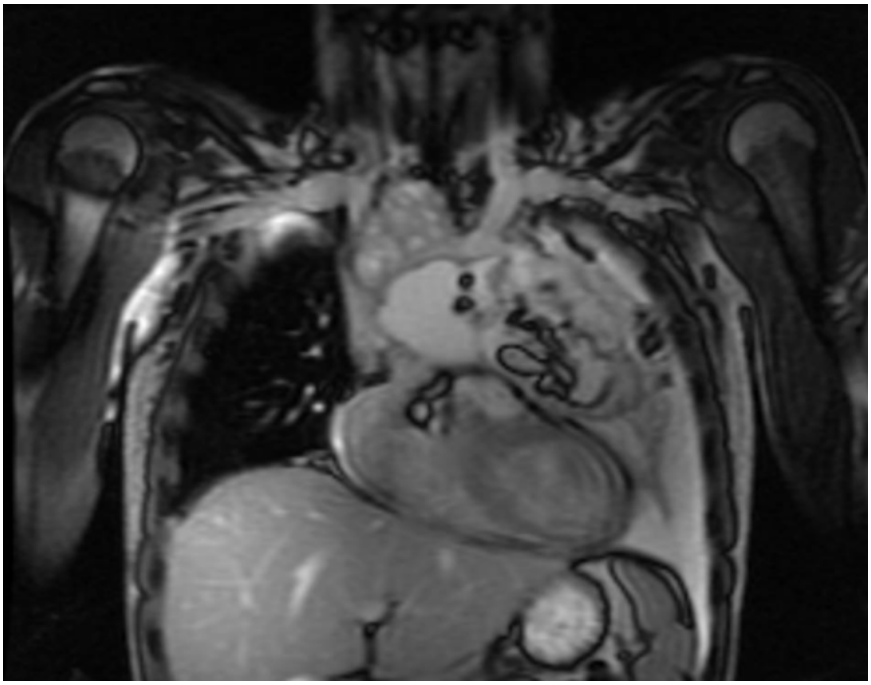


Figure 1. MRI of chest with IV contrast demonstrating 13 x 6.1 x 9.5 cm anteromediastinal mass containing fat, fluid, and calcifications, with significant mass effect.

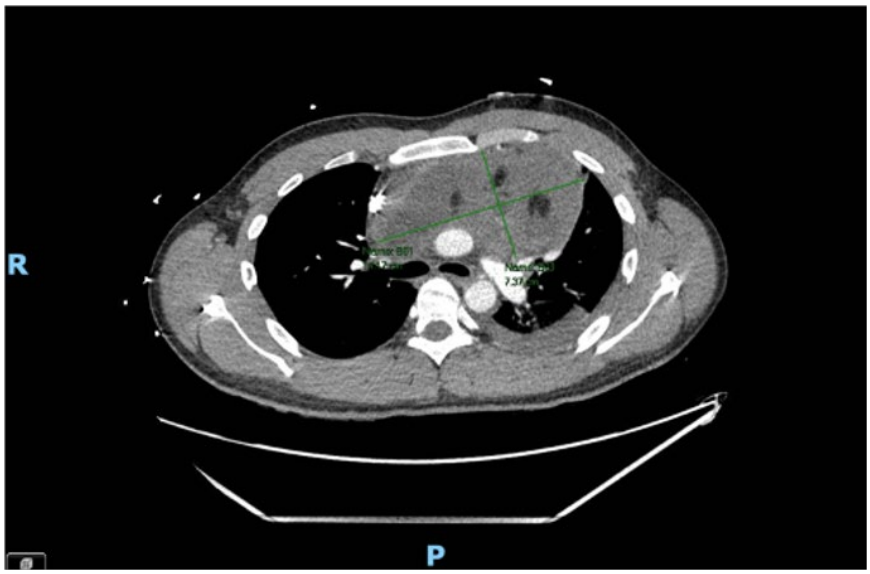


Figure 2. Axial view of large anterior mediastinal mass measuring 14.2 x 7.4 x 9.1 cm with heterogenous, predominantly internal fat and fluid density with some foci of increased density.

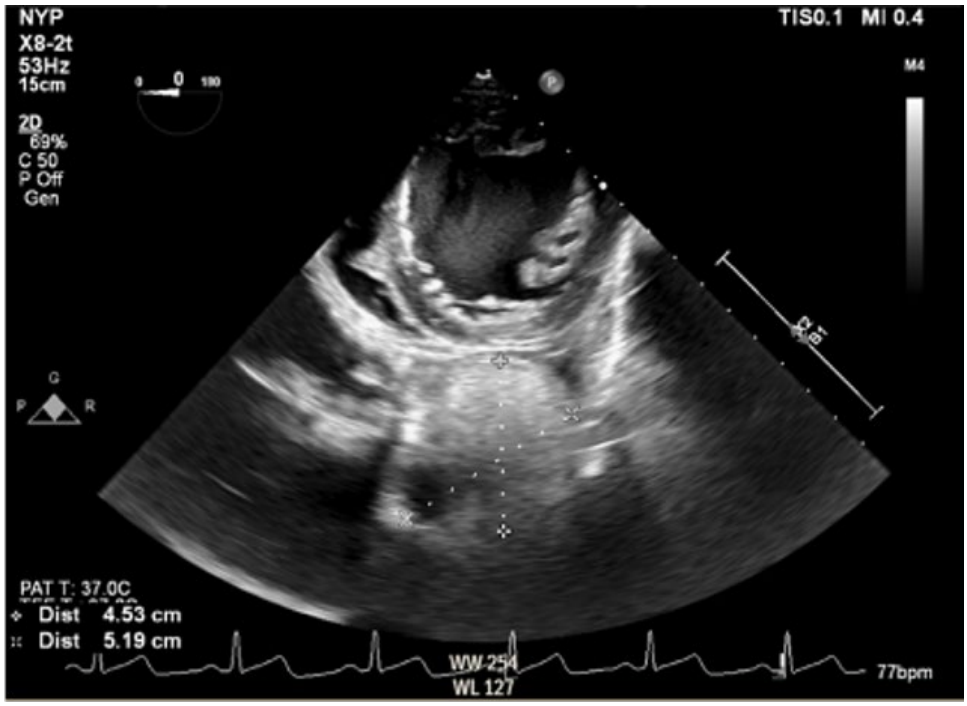


Figure 3. Intraoperative transgastric mid-papillary short axis view showing a large, incompletely circumscribed, heterogenous mass that is anterior to multiple cardiac structures.

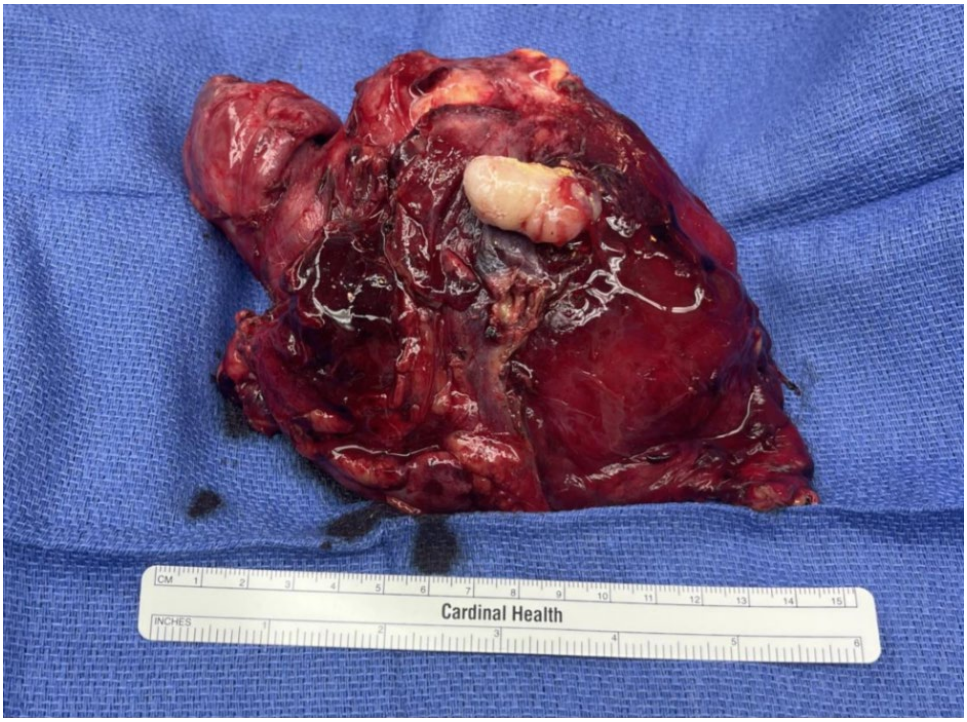


Figure 4. Excised mediastinal mass

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#FR 2 – Kiosk 4 – Poster 4

Thoracoabdominal Branch Endoprosthesis (TAMBE) Procedure Complicated by Spinal Cord Ischemia

Presenting Author: Cheng-Hao Jacky Chen, MD, Medical College of Georgia Department of Anesthesiology and Perioperative Medicine

Introduction

Thoracoabdominal branch endoprosthesis (TAMBE) is a next-generation off-the-shelf device that parallels thoracic endovascular aortic repair (TEVAR) for treatment of thoracoabdominal aortic aneurysms (TAAA). Due to its extensive coverage of the aorta, spinal cord ischemia (SCI) remains a feared complication. We present a case that follows the development and management of SCI following a TAMBE procedure.

Case Presentation

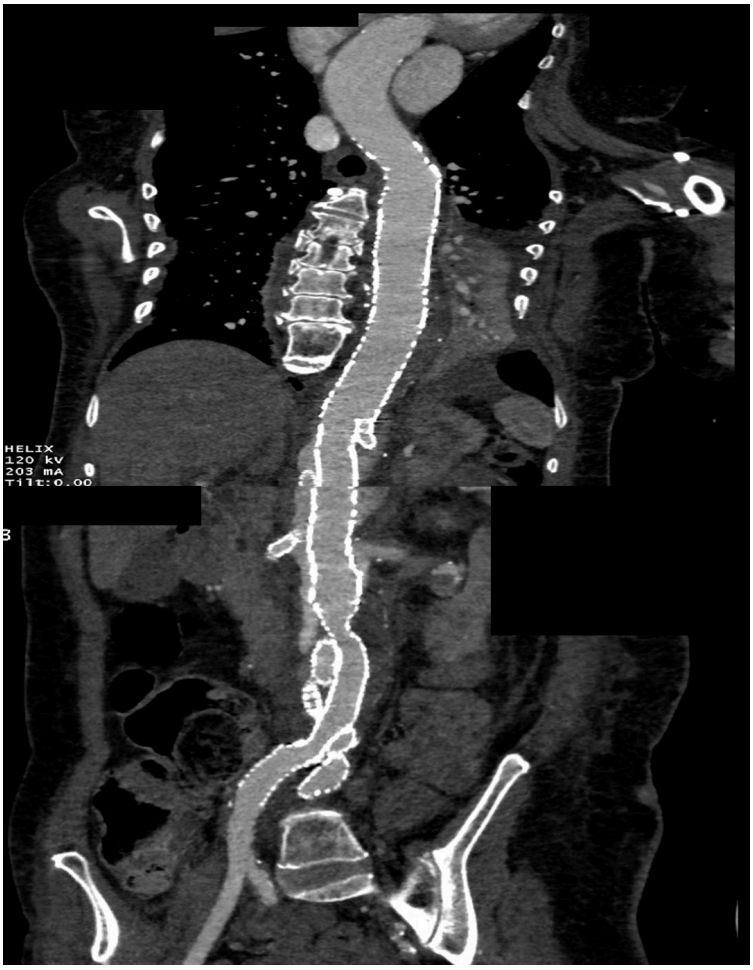
The patient is a 63-year-old female with a past medical history of TAAA status post TEVAR, hypertension (HTN), chronic obstructive pulmonary disease (COPD), hyperlipidemia (HLD), type 2 diabetes mellitus (DM), and tobacco use who presented to our institution for management of her TAAA. Computed tomography (CT) imaging showed aneurysmal dilatation of the aorta extending from the descending thoracic aorta to the iliac bifurcation. The patient was evaluated by vascular surgery and scheduled for a TAMBE. Following a pre-induction arterial line, the patient underwent standard controlled induction of anesthesia. A central venous catheter with pulmonary artery catheter were placed following induction. The procedure was complicated by 1 liter of blood loss, and the patient received 3 units of packed red blood cells. Urine output was adequate throughout the procedure, and the patient was noted to be neurovascularly intact following extubation. The patient was admitted to the cardiovascular intensive care unit (CVICU) postoperatively. On postoperative day 2, the patient acutely developed proximal right lower extremity weakness and paresthesia, concerning for spinal cord ischemia. Spinal perfusion pressure was optimized with a mean arterial pressure (MAP) goal of >95mmHg, hemoglobin (Hgb) >10g/dL, and a therapeutic spinal drain was placed emergently by the cardiothoracic anesthesiology team with an intraspinal pressure goal of 8-12mmHg. Subsequent magnetic resonance imaging (MRI) showed short segment signal abnormalities in the right T7-T8 hemicord, concerning for spinal cord infarction. Following spinal drain placement, right lower extremity strength and paresthesia improved, and the spinal drain was removed on postoperative day 6. The patient underwent extensive physical therapy and was discharged on postoperative day 13.

Discussion

TAMBE requires extensive coverage across the supraceliac descending aorta, which supplies major contributors to spinal cord perfusion, thereby predisposing patients to SCI[1]. In this patient, the presence of comorbidities such as HTN, DM, HLD, and COPD can impair collateral circulation and reduce spinal cord perfusion, ultimately increasing risk for SCI. Sickels et al. proposed an SCI prevention protocol (MAP >90mmHg, Hgb >10g/dL, selective spinal drain placement, and use of pharmacological adjuncts), which was shown to significantly decrease SCI rates and 30-day mortality[2]. The benefit of prophylactic versus

therapeutic cerebrospinal fluid drainage in endovascular aortic repairs remains controversial in the current literature and further research on its role in TAMBE is required.

Images



Thoracic, abdominal and pelvic computed tomography with intravenous contrast showing extensive coverage of the thoracoabdominal branch endoprosthesis from the descending thoracic aorta to the right common iliac artery

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#FR 2 – Kiosk 4 – Poster 5

Percutaneous mitral valve-in-valve replacement as a bridge to definitive open surgery

Presenting Author: Scott Jossart, MD, CT Anesthesiology Fellow, University of Alabama at Birmingham (UAB)

Introduction

Bioprosthetic valve deterioration is a well-known complication that occurs within 10 years in up to 35% of patients, with younger patients having the highest prevalence.[1] As a result, percutaneous methods of replacement are being explored as an alternative to redo sternotomy procedures. In the subset of patients with multiple valve lesions, the utility of percutaneous options is less certain. Patients undergoing TAVR with untreated mitral stenosis have a lower survival rate at 3 years than those without MS (76% vs 56%),[2] suggesting that multiple lesions should not be tackled piecemeal. Although there have been successful concurrent and staged TAVR and TMVR procedures described in the literature, open replacement of both valves remains the primary approach to handling multi-valve dysfunction, especially in redo procedures.

Case Presentation

A 48-year-old male with history of HLD, HTN, bicuspid aortic valve (s/p SAVR in 2014), and endocarditis (s/p redo sternotomy SAVR/MVR in 2019) presented following two months of dyspnea and hypoxia previously treated as pneumonia at an outside facility. Transthoracic echocardiography on admission demonstrated severe prosthetic aortic and mitral stenosis. Surgery was planned during admission, but the patient developed respiratory and hemodynamic decompensation requiring intubation and high dose inotrope and vasopressor support. Within an hour of intubation, he deteriorated further requiring emergent VA ECMO initiation. Following cannulation, he required support with epinephrine, norepinephrine, milrinone, vasopressin, angiotensin II, and iNO. A multi-disciplinary discussion determined he was a poor candidate for third time sternotomy redo double valve replacement unless he was better optimized. The decision was made to undergo transfemoral transcatheter mitral valve in valve replacement on VA ECMO support as a temporizing measure before definitive surgery. A 29-mm Edwards S3 valve was placed uneventfully 36 hours after ECMO initiation. Following placement, angiotensin II and vasopressin were weaned off before leaving the catheterization lab. On POD#1, the patient was transitioned to VV ECMO. By POD#7, he was decannulated, off all support, and ambulating. Interestingly, while waiting for blood cultures to clear for open surgery, he was found to have hemolytic anemia which hematology attributed to high gradients across

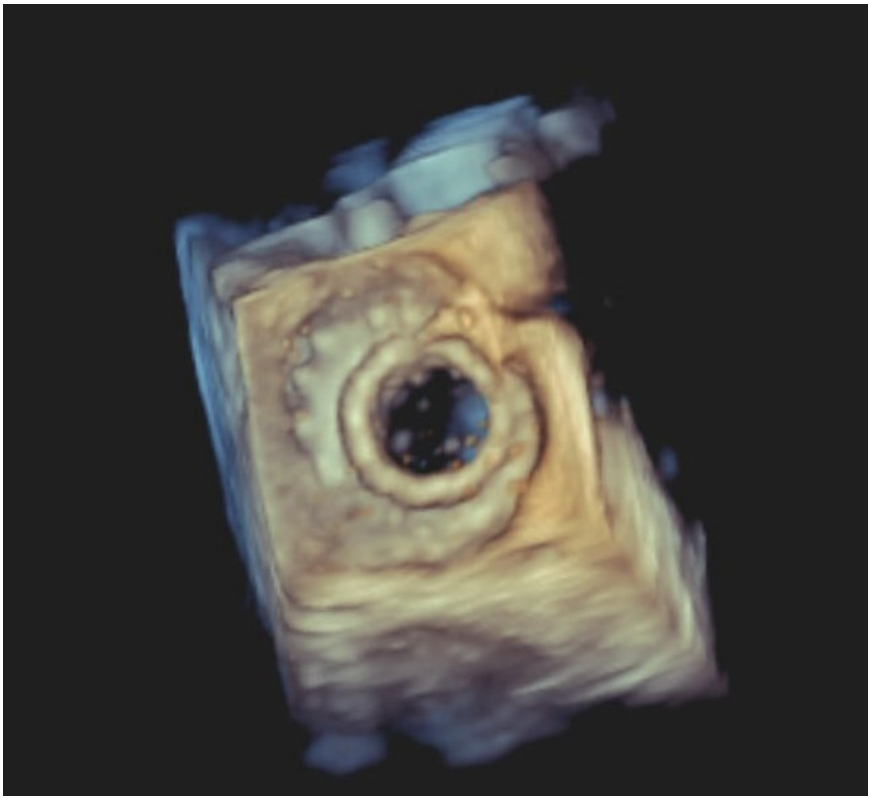
the two stenotic valves. Once blood cultures were clear, the patient underwent uneventful redo AVR and MVR, and he was transferred to the ICU on epinephrine, vasopressin, and IABP. He was extubated on POD#1, off pressors and IABP on POD#3, and transferred to floor on POD#8.

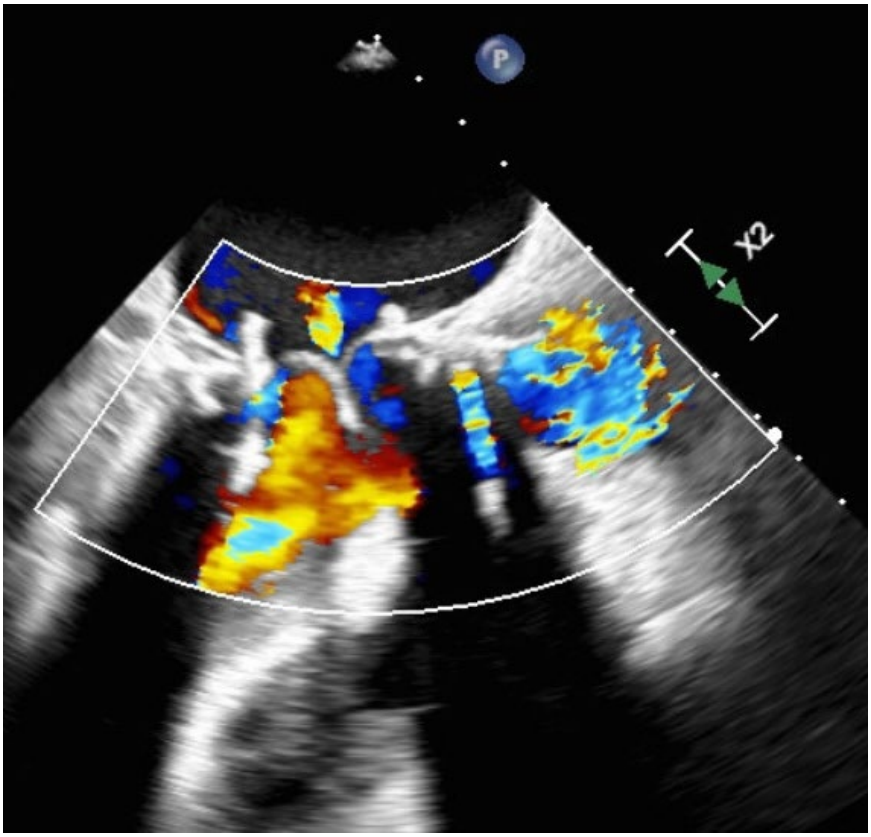
Discussion

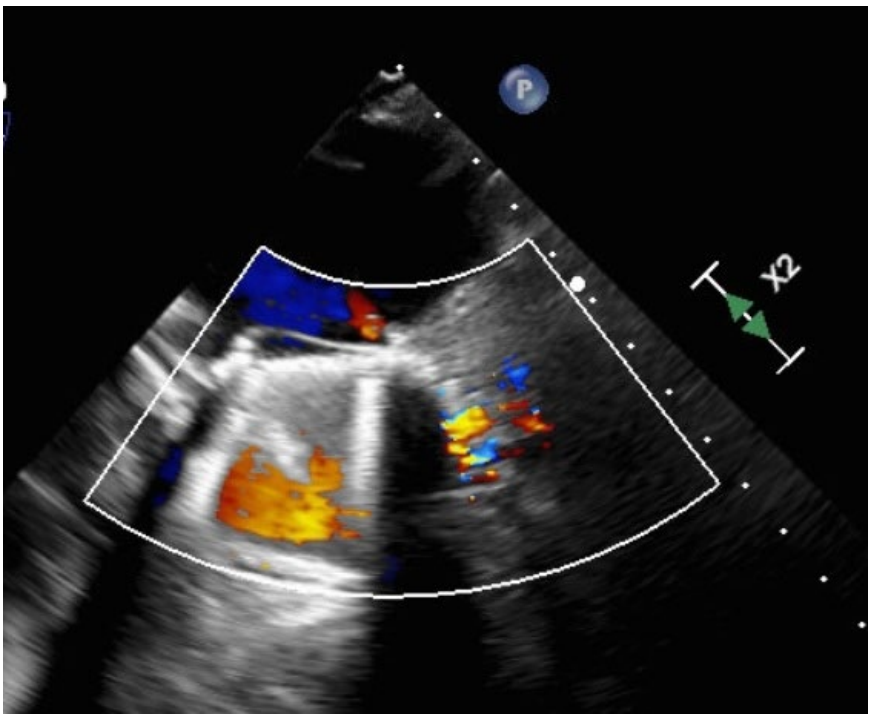
Percutaneous MVR has been found to be a safe and successful alternative to open redo MVR in certain populations.[1] While our patient eventually required definitive open redo replacement of both the AV and MV, the TMVR allowed for clinical improvement and optimization prior to a procedure that carries a 10% mortality.[1] Although hemolysis after percutaneous valves is not rare (15% in all TAVR patients at 6 months),[3] it is uncommon to have clinically significant anemia requiring transfusion. This case offers interesting future implications for use of percutaneous intervention as a bridge to definitive treatment.

Images






P
X2


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X2

References

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#FR 2 – Kiosk 4 – Poster 6

When Two Hearts Matter: Anesthetic Management of Aortic Dissection in Pregnancy

Presenting Author: Natalie J. Koons, DO, Anesthesiology Resident, Stanford Health Care

Introduction

Although acute aortic dissections in pregnancy are rare, with a prevalence of roughly 0.1% of patients[1], they are a life-threatening condition for both pregnant women and fetuses, with a mortality rate as high as 60%.[2] We present a case that demonstrates the unique multidisciplinary care that was crucial in managing an acute aortic dissection in a 30-week pregnant female with a viable fetus that was later diagnosed with Loeys-Dietz syndrome.

Case Presentation

A 30-year-old G1P0 female at 30-weeks' gestation with a history of mild Factor VII deficiency presented to the emergency department with chest pain radiating to her lower back. Computed tomography revealed a Stanford Type A dissection with a dilated aortic root and a dissection flap extending from the ascending aorta to below the renal arteries. The patient was transferred to our institution for immediate surgical intervention and following multidisciplinary discussion, the decision was made to place the patient under general anesthesia for impulse control, followed by cesarean delivery of the fetus, and then repair of the aortic dissection. She underwent rapid sequence induction of general anesthesia with placement of central venous access and transesophageal echocardiogram (TEE) probe. TEE demonstrated normal biventricular size and function, severe eccentric aortic insufficiency, and redemonstration of the aortic dissection flap. Propofol and remifentanil infusions were used as maintenance during the low-transverse cesarean section and until uterine tone was felt to be appropriate. Isoflurane and fentanyl were then utilized for the remainder of the case. After fetal delivery, the patient received a 3-unit bolus of oxytocin, followed by an oxytocin infusion for a total dose of 65 units, and 250 mg of Carboprost to achieve adequate uterine tone. The abdomen was deliberately left open to monitor for uterine atony and postpartum hemorrhage (PPH) while the patient underwent a supracoronary ascending aorta and hemiarch replacement on cardiopulmonary bypass (CPB) with circulatory arrest. The chest and abdomen were subsequently closed, and the patient was transferred to the cardiac intensive care unit in stable condition. The patient was discharged home on post-operative day 7 and the infant was discharged home following an 8-week stay in neonatal intensive care unit. Genetic testing in the patient was consistent with Loeys-Dietz syndrome.

Discussion

The physiologic changes that occur during pregnancy, including increased cardiac and vascular blood volume, fetoplacental circulation, hormonal adaptations and enlargement of the uterus, increase the risk of developing an aortic dissection in pregnancy.[1,2] Aortic dissections in pregnancy tend to occur during the third trimester or during the early postpartum period and within patients who have pre-existing connective-tissue disorders.[3] Intraoperatively, there is a careful balance between managing potential uterine atony and PPH with heparinization while on CPB after the cesarean section. Choice of anesthetic is important, as short-

acting agents and non-volatiles help preserve uterine and fetal tone. Our case illustrates the successful multidisciplinary intraoperative management of aortic dissection in pregnancy.

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#FR 2 – Kiosk 4 – Poster 7

Simultaneous LVAD Implantation and Ventricular Tachycardia Ablation in DSP-Related Cardiomyopathy

Presenting Author: Rachel Reindorf, MD, Fellow, Johns Hopkins Hospital

Introduction

Desmoplakin (DSP)-related cardiomyopathy involves fibrosis and high arrhythmic risk.[1] Patients with DSP variants face significant sustained arrhythmia risk (~ 3.9% per year), particularly in the setting of left ventricular (LV) dysfunction.[2]Management becomes complex when refractory ventricular tachycardia (VT) storm coexists with end-stage heart failure. Since VT complicates 20-25% of Left Ventricular Assist Device (LVAD) implantations,[3] simultaneous ablation is utilized to mitigate risk. We report the first known case of this combined approach in DSP-related cardiomyopathy, performed to manage arrhythmia burden while avoiding the risks of sympathectomy during acute decompensation.

Case Presentation

A 48-year-old woman with LV-predominant DSP familial cardiomyopathy and prior VT storm requiring implantable cardioverter-defibrillator (ICD) placement presented with chest pain and dyspnea preceded by wheezing, paroxysmal nocturnal dyspnea, and weight gain. Her current medication regimen included losartan, spironolactone, amiodarone, and mexiletine. Despite discharge one month prior on milrinone 0.375 mg/min, she presented in extremis with blood pressure 71/48 mmHg and heart rate 112 bpm. After initial stabilization and diuresis at an outside hospital, she was transferred to our institution for advanced heart failure evaluation. Upon arrival, N-terminal pro B-type natriuretic peptide was 587 pg/mL and creatinine 1.22 mg/dL. Electrocardiogram showed sinus tachycardia with anterior ST–T changes. Echocardiography revealed a dilated LV with ejection fraction 9%. Consistent with recent diuresis, Right Heart Catheterization (RHC) demonstrated low filling pressures (right atrial 1 mmHg; pulmonary capillary wedge 6 mmHg), but a persistently low cardiac index (CI) of 2.08 L/min/m^2 and pulmonary vascular resistance (PVR) of 3.02 Wood units. With persistent New York Heart Association (NYHA) class IV symptoms and inotrope dependence, a durable LVAD was planned. Electrophysiology interrogation found no spontaneous arrhythmia; however, Non-Invasive Programmed Stimulation (NIPS) induced two clinically significant VT episodes. Consequently, concurrent ablation was planned and sympathectomy was deferred to minimize hemodynamic risk. She underwent LV substrate ablation (ethanol and cryoablation) and HeartMate 3 LVAD implantation with Cardiopulmonary Bypass (CPB) without intraoperative complications. Her postoperative course was uncomplicated, and she was discharged on warfarin with a therapeutic INR.

Discussion

This case highlights the severe arrhythmic and hemodynamic consequences of DSP-related cardiomyopathy, emphasizing the need for individualized, multidisciplinary management. In progressive LV-predominant disease, recurrent VT storm and advanced heart failure necessitated durable mechanical circulatory support. Performing VT substrate ablation during LVAD implantation allowed for targeted

arrhythmia management while avoiding the heightened risks of sympathectomy in acute decompensation. This case suggests that early integration of electrophysiologic and surgical interventions offers a safe, effective strategy for DSP cardiomyopathy patients with refractory arrhythmia and advanced heart failure.

Images

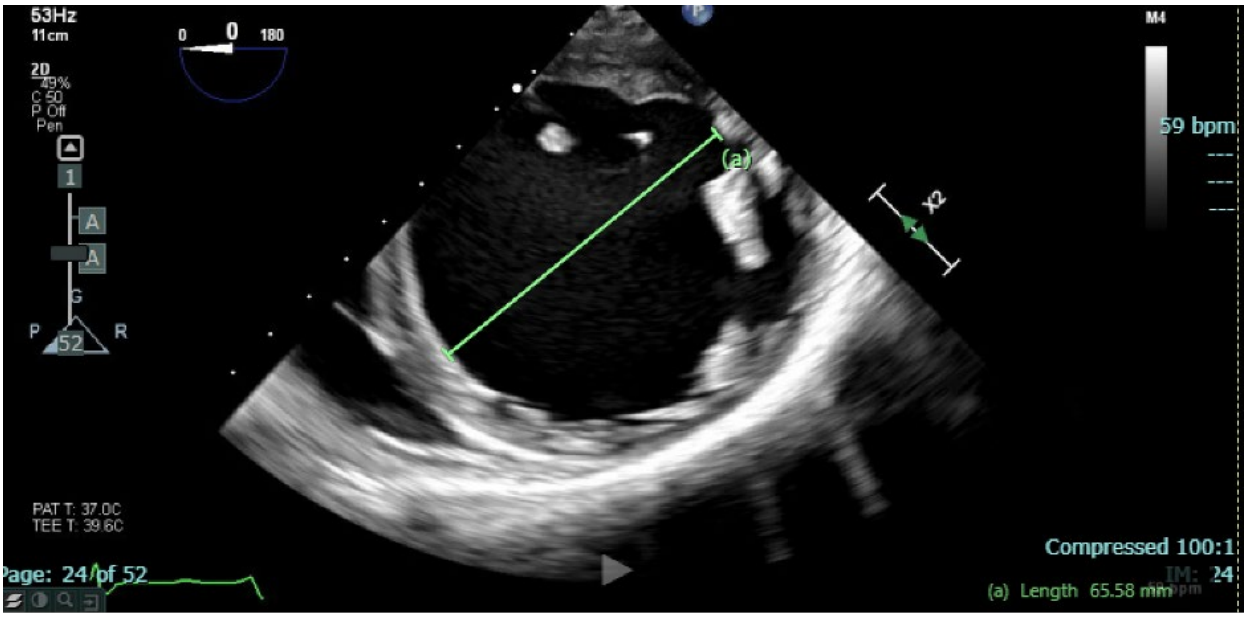


Fig 1. Severely dilated LV in DSP cardiomyopathy.

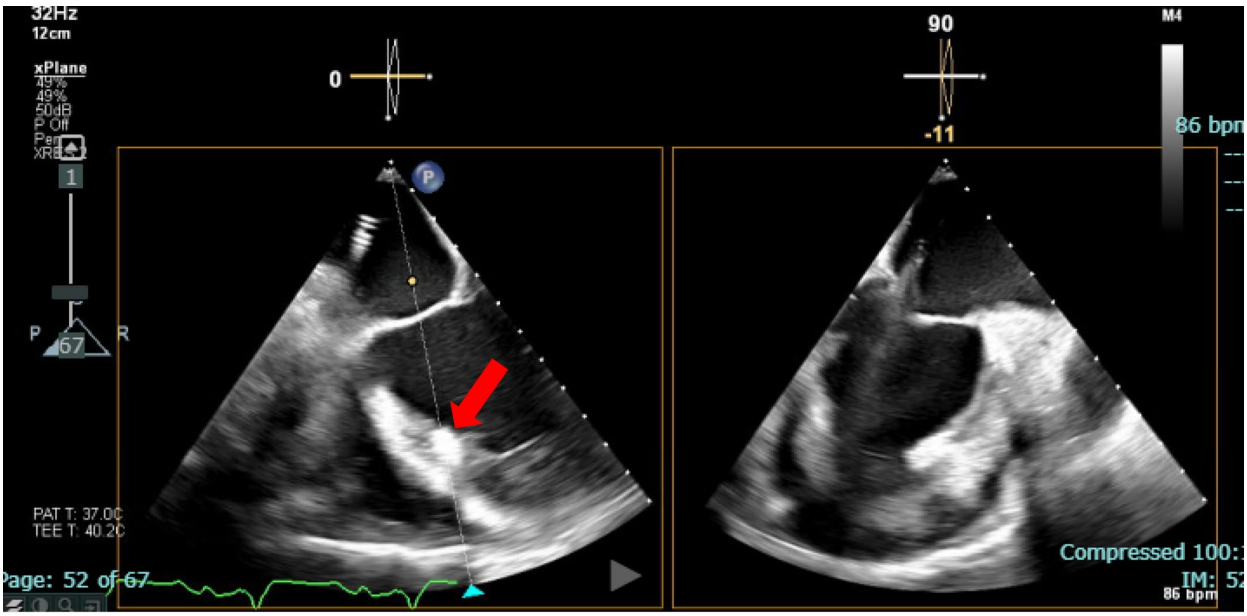


Fig 2. Lesion visualized after injection of ethanol at one of the foci of Ventricular arrythmias.

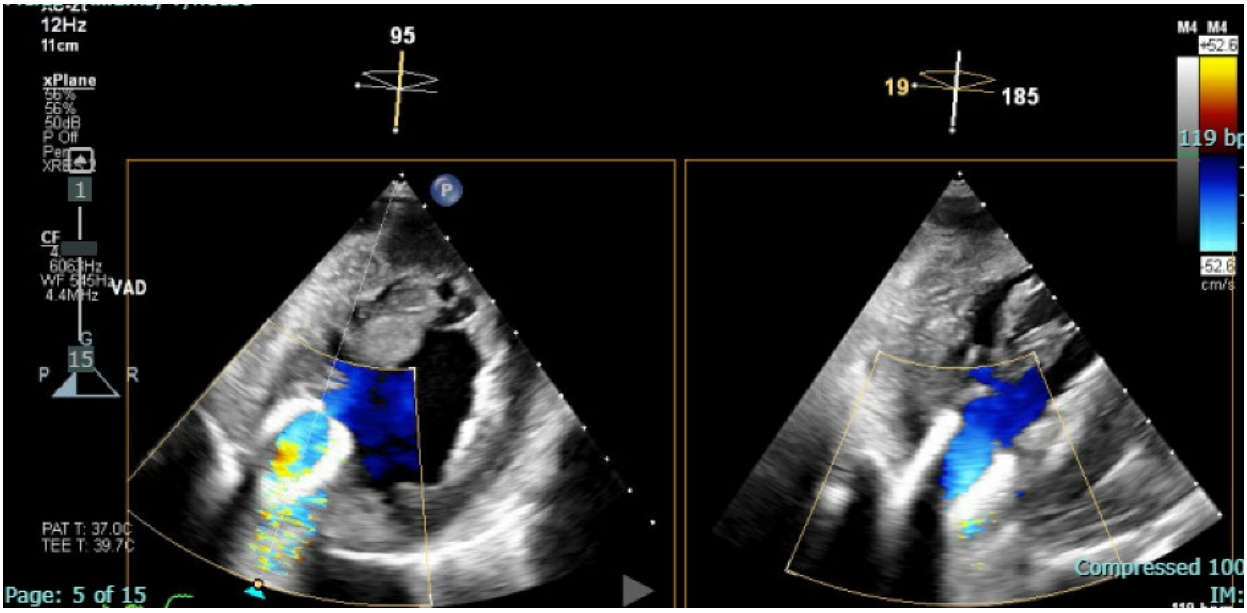


Fig 3. LVAD Implanted.

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#FR 2 – Kiosk 5 – Poster 1

Marantic endocarditis in a high-risk surgical patient

Presenting Author: Annie Yao, DO, Fellow, University of Nebraska Medical Center

Introduction

Nonbacterial thrombotic endocarditis (NBTE) is a rare condition (~1% autopsies) where sterile vegetations form on heart valves. This most commonly occurs in malignancies and autoimmune conditions. The pathogenesis is suspected to be caused by endothelial injury from circulating cytokines which trigger the accumulation of platelets, fibrin, and immune complexes. Treatment involves anticoagulation, treatment of the underlying condition, and surgery, though prognosis is poor as NBTE may be an advance stage of malignancy. This case explores the management of a complex and unique patient who presented with subacute endocarditis and a recent diagnosis of smoldering multiple myeloma on daratumumab.

Case Presentation

A 57-year-old female presented to her cardiology clinic for a 18month follow-up after percutaneous coronary intervention. She reported 2 weeks of worsening fatigue and lightheadedness, with a new murmur on physical exam. She underwent a TEE which showed aortic regurgitation, valvular perforation, and a vegetation. She was admitted and evaluated for endocarditis however the infectious workup was negative. Other pertinent medical history consists of poorly controlled diabetes, hypertension, smoldering multiple myeloma on weekly daratumumab, cirrhosis complicated by portal hypertension, BMI 41, and pancytopenia. Due to her negative infectious work-up, the diagnosis of NBTE was made. She was deemed a poor TAVR candidate and was transferred to another hospital for a second opinion as a surgical candidate. The patient underwent a successful surgical aortic valve replacement. Intra-operative cultures remain negative after five days and are still pending at the time of abstract submission.

Discussion

This case presents two considerations for care: diagnosis and management of NBTE and, to a lesser extent, immunotherapy affecting the type and cross process. Although rare, the differential of NBTE is important to consider. This patient's recent diagnosis of smoldering multiple myeloma with pancytopenia and immunotherapy with daratumumab puts her at increased risk of infectious endocarditis, which can be fatal if not treated appropriately. However, she was not started on empiric antibiotics due to her negative infectious work-up. While management of NBTE consists of anticoagulation, the patient was pancytopenic and suffered from a GI bleed, so anticoagulation was held. Due to the vegetation on her aortic valve, she was not a suitable TAVR candidate however her oncologic prognosis was good, so the decision was made to proceed with surgery despite a high surgical risk. Her immunotherapy, daratumumab, is a monoclonal antibody that targets CD38. CD38 is highly expressed on both cancer cells and red blood cells which yields false-positive antibody screens and results in significant delays from the blood bank. Given frequent shortages of blood products and the patient's high risk of coagulopathy and massive blood loss, a discussion was held with the surgeon, anesthesiologist, blood bank, and perfusionist prior to the procedure

to formulate a contingency plan. Management was well coordinated amongst different teams that led to a successful outcome in a complex and unique patient.

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#FR 2 – Kiosk 5 – Poster 2

Atrioventricular Groove Dissociation Following Mitral Valve Replacement

Presenting Author: David Cline, Resident Physician, University of Kentucky

Introduction

Atrioventricular (AV) groove dissociation is a rare complication occurring during cardiac surgery. The AV groove is a vulnerable anatomical region circling the heart separating the atria from the ventricles and containing the right coronary artery, left circumflex artery, small cardiac vein, and coronary sinus (2). AV groove dissociation is a disruption within the AV groove believed to be secondary to either surgical trauma or stretch injury (2). Incidence of AV groove dissociation is noted to be 0.5-2% with a mortality of 50-80% (1). This report presents the case of a patient who suffered an AV groove dissociation during mitral valve replacement ultimately resulting in her death seven days postoperatively.

Case Presentation

A sixty-year-old female with medical history significant for rheumatic fever initially presented to an outside hospital with tachycardia, dyspnea, and new onset atrial fibrillation. Her workup included an echocardiogram significant for mild tricuspid regurgitation, mild pulmonic valve regurgitation, and severe mitral valve stenosis with significant calcification. Follow-up involved outpatient cardiothoracic surgery evaluation resulting in scheduling for mechanical mitral valve replacement and left atrial appendage clipping. Intraoperatively, evidence of prior rheumatic disease of the mitral valve leading to foreshortening of the papillary muscles and chordae tendineae as well as friable tissue were noted. Following prosthesis valve placement, a large perivalvular leak was noted resulting in reseating and re-suturing of the valve. Unfortunately, a mild-to-moderate perivalvular leak remained, but secondary to friable tissue the decision was made to wean bypass. During removal of the venous cannula, a gush of bright red blood into the field occurred and the surgeon observed AV groove dissociation. The patient was placed back on cardiopulmonary bypass and the exterior atrioventricular canal was sutured, sealed with bio-glue, and covered with a pericardial patch. Postoperative complications included acute kidney injury and refractory atrial fibrillation as well as multiple vasopressor infusions to meet hemodynamic goals. Unfortunately, on post-operative day seven, family pursued hospice care and the patient passed as a result of renal failure and inability to wean from vasopressors.

Discussion

AV groove dissociation remains a rare but feared risk factor during cardiac surgery. Specific risk factors include calcified mitral annulus, rheumatic disease, female sex, reoperation with prosthesis in place, and hemodialysis patients (1). Operative prevention includes careful resection of mitral leaflets, minimal debridement of mitral annulus, accurate prosthesis sizing, careful suture placement and tying, avoidance of significant hypertension in immediate post-cardiopulmonary bypass period, and avoidance of ventricular over-dilation (1). Intraoperative examination after AV groove dissociation commonly shows a large hematoma on the inferior-posterior wall with transesophageal echocardiogram findings of air within the left

ventricular cavity and regional wall motion abnormalities. Few case reports exist of successful repair with reported mortality of 50-80% secondary to left ventricle failure and hemodynamic instability (3).

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#FR 2 – Kiosk 5 – Poster 3

Survival After Stone Heart Syndrome: A Case Report

Presenting Author: Bridgette Ho, MD, Resident, Los Angeles General Medical Center/University of Southern California

Introduction

Stone Heart Syndrome is a rare complication of postcardiotomy shock. This condition was first described in 1972 as a complication from cardiac surgery and cardiopulmonary bypass. It is characterized by a small, spastic heart arrested in systole.1 Unfortunately, there have been limited cases reporting Stone Heart Syndrome, and even fewer have favorable outcomes. This case describes a patient who developed Stone Heart Syndrome and recovered.

Case Presentation

A 24-year-old male with morbid obesity and a bicuspid aortic valve presented with a 6.5cm Type A aortic dissection. He underwent total aortic arch repair and aortic valve replacement. Intra-operatively, the patient could not be weaned from bypass and his heart remained persistently contracted. He was brought to the ICU on central ECMO. His immediate postoperative coronary angiogram was unremarkable. Given persistent thickened myocardium with near akinesis (< 5% LVEF on TEE) (Fig. 1), the leading diagnosis for his shock was Stone Heart Syndrome. Cardiac pulsatility initially was < 5 points but began showing improvement on post-operative day 3 with vasoactive support. His cardiac function continued to improve. ECMO was ultimately reconfigured with placement of an intraaortic balloon pump for left ventricular venting, both of which were eventually removed. By the third postoperative month, the patient had recovery of heart function and was weaned from vasoactive support. He was able to sit in a wheelchair outdoors and stand with physical therapy.

Discussion

This case highlights the possibility of meaningful recovery after Stone Heart Syndrome. Although its pathophysiology is not fully understood, Cooley et al. proposed that hypertrophic myocardium is particularly vulnerable due to its increased metabolic demand and relative coronary insufficiency.1 ATP depletion is critical to this disease development. Without ATP, the myosin-actin complex remains contracted, a process similar to rigor mortis.2 Because Stone Heart Syndrome is difficult to reverse, attention has focused on preventing its development by avoiding ATP depletion. Strategies include appropriate cardioplegia with

potassium, magnesium, and total body cooling as methods to limit myocardial metabolism. Coronary supplementation with ATP and glucose also delays the onset.2 Despite appropriate cardioplegia during the surgery, this patient's underlying hypertrophic myocardium may have caused increased susceptibility to inadequate cardiac metabolic protection during bypass. Mavacamten, a myosin inhibitor recently approved for hypertrophic cardiomyopathy, may represent a future therapeutic option for patients with stone heart syndrome. Current studies are underway to evaluate off-label use to treat prolonged contractility diseases, including Stone Heart Syndrome.3 Stone Heart Syndrome is a devastating complication of cardiotomy and bypass. This case demonstrates a rare example of a remarkable recovery following Stone Heart Syndrome with aggressive supportive therapy.

Images

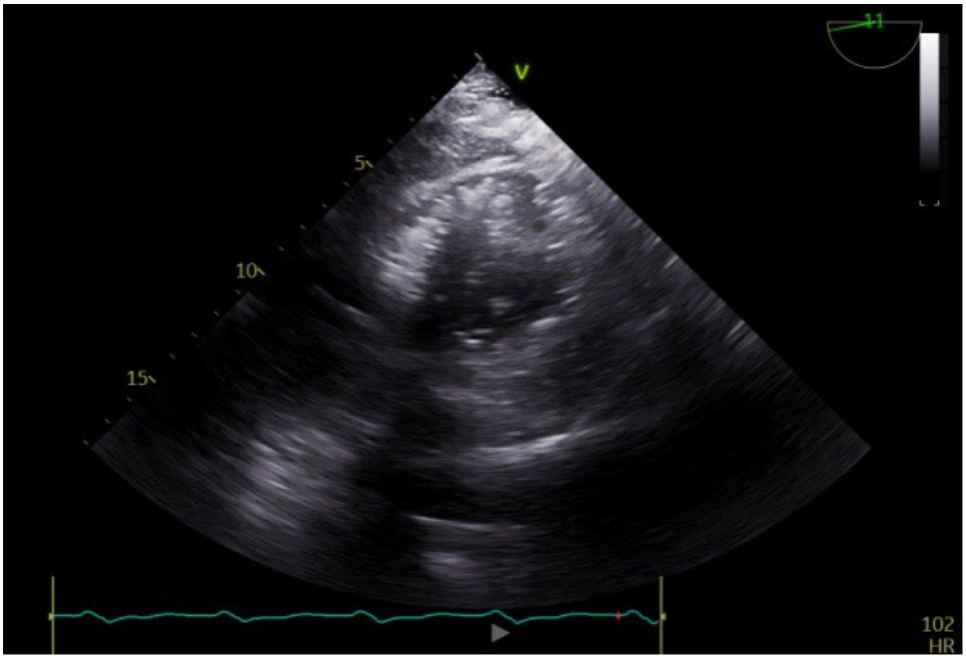


Figure 1: The transgastric mid-papillary short axis view during the patient’s index operation shows thickened LV walls and near obliteration of LV cavity.

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#FR 2 – Kiosk 5 – Poster 4

Harlequin Syndrome Following VA-ECMO: Complications and Management Strategies

Presenting Author: Caitlin H. Bakke, DO, CA1 Anesthesia Resident, University of Minnesota

Introduction

Harlequin Syndrome (North-South syndrome) is a complication of veno-arterial extracorporeal membrane oxygenation (VA-ECMO), particularly seen with femoral arterial cannulation, where there is an unfavorable mixing of poorly oxygenated pulmonary venous blood with retrograde ECMO flow. This can precipitate cerebral and myocardial hypoxia[1]. The following case highlights the management considerations for Harlequin syndrome in a postcardiac arrest patient with an intra-aortic balloon pump (IABP) and hemothorax requiring VA-ECMO.

Case Presentation

A 60-year-old male presented with out-of-hospital cardiac arrest secondary to acute left main coronary occlusion and cardiogenic shock. He was placed on femoral VA-ECMO with an IABP and received timely PCI. Dual antiplatelet therapy was initiated. On hospital day three, subsequent echocardiography demonstrated recovery of systolic function. However, the patient had rib fractures following compressions and developed significant left hemothorax from presumed intercostal artery injury. A chest tube was placed, but hemothorax was not adequately drained. The patient's ventilatory requirements increased and he developed Harlequin syndrome with refractory hypoxemia despite 100% FiO2 and PEEP 12. He presented for emergent thoracotomy and evacuation of hemothorax. During intraoperative management, supine positioning and single lung ventilation via EZ blocker led to worsened hypoxemia. Strategies to treat hypoxic admixture included esmolol and reversed I:E ratio of 2:1 which transiently improved O2 sats in the right upper extremity. IABP support was decreased from 1:1 to 1:3 to limit cardiac output and shift the mixing point. Lateral positioning during one lung ventilation was not attempted due to precarious femoral cannulation. Bronchoscopy showed good ETT position and minimal secretions. Inhaled prostacyclin and APRV were initiated in ICU. Persistent hemorrhage necessitated massive transfusion protocol and opening of the chest at the bedside. Ultimately, multiorgan dysfunction led to transition to comfort cares.

Discussion

In this case, recovery of native cardiac function unmasked impaired oxygenation. The requirement of supine one lung ventilation during hemothorax evacuation led to worsening hypoxemia. Strategies were performed to improve oxygenation during one lung ventilation as well as to temporize the hypoxemia in Harlequin syndrome via reducing cardiac output to shift the hypoxic admixture proximally. Ultimately, conversion of the ECMO configuration to VA-V with placement of an additional venous return cannula closer to the heart is a more definitive solution. In our case, Ying IV tubing from the ECMO return to an internal jugular vein MAC line was also considered as a temporizing measure. Other potential solutions with regards to cannula configurations include: conversion to a direct aortic return, more complete central venous drainage, and conversion to V-V ECMO should cardiac function allow[2]. This case highlights the complexity of balancing

mechanical ventilation, hemodynamics and ECMO flow in post-cardiac arrest VA-ECMO and emphasizes the need for early recognition and individualized management of Harlequin syndrome. Our interventions provided transient improvement but were limited by hemodynamic instability and bleeding.

Images

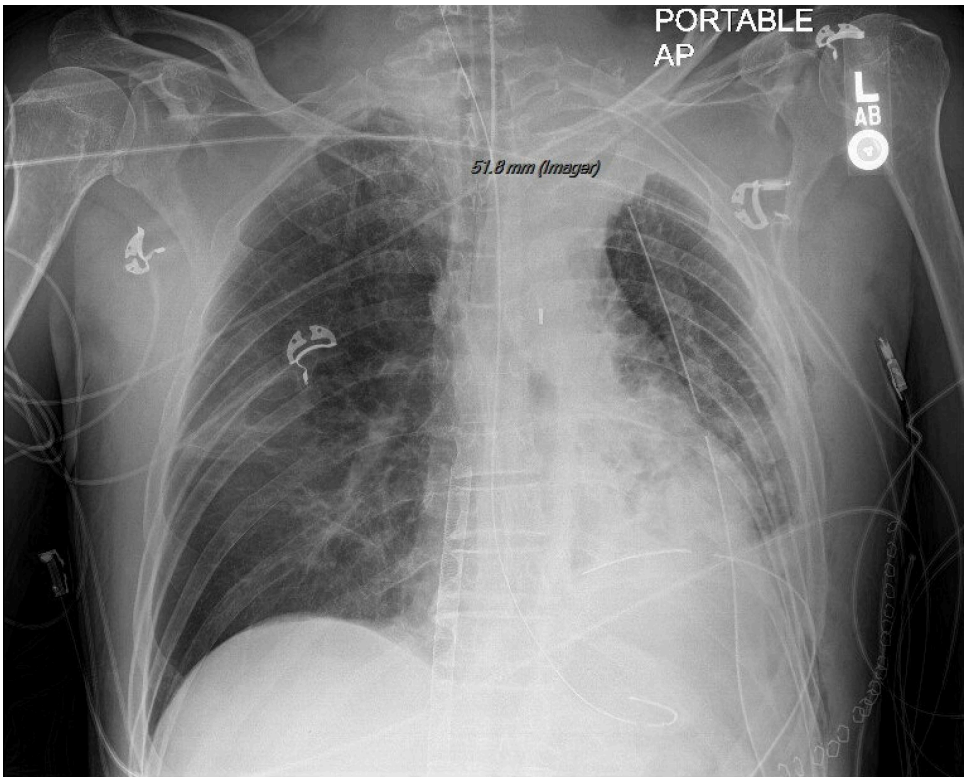


Image 1: Chest x-ray from hospital day four demonstrating left-sided hemothorax.

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#FR 2 – Kiosk 5 – Poster 5

Coronary to Pulmonary Artery Fistula-Induced Coronary Steal Syndrome: A Complex Case

Presenting Author: Sivan G. Marcus, MD, ACTA Fellow, Stanford University

Introduction

Coronary to PA fistulae is a rare anatomic variant. Most commonly congenital in origin, these lesions are often asymptomatic but can occasionally cause symptoms of heart failure and angina through a combination of shunting and coronary steal syndrome. They can be treated conservatively, interventionally, or surgically, depending on the specific anatomy and symptoms. We report a case of late-onset symptomatic Coronary-PA fistula requiring sternotomy, cardiopulmonary bypass, and surgical closure.

Case Presentation

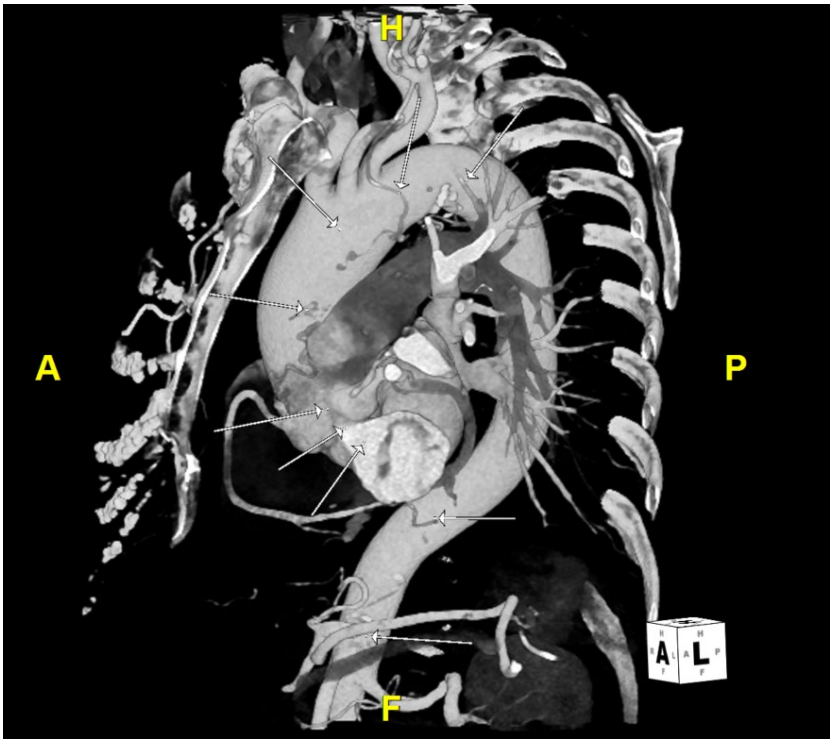
A 77-year-old woman with no significant past medical history developed one month of dyspnea with exertion and exertional angina. Workup, including echocardiography, CT-angiography, and stress testing, revealed no CAD but showed a large fistula between the LAD and main PA, with an additional smaller fistula between the RCA and PA. There was additionally evidence of left-to-right shunting, with symptoms attributable to coronary steal syndrome. The decision was made to proceed with an open fistula closure procedure. After induction, TEE confirmed the presence of the fistula, and PA catheterization revealed an increase in venous blood oxygenation in the PA compared to RA, consistent with a significant L- >R shunt. After sternotomy and TEE, it was revealed that the fistulae contained significant collateralization and were inserted close to the pulmonic valve. This would have made it difficult to sufficiently ligate externally and risked damaging the valve without sufficient visualization. Thus, the decision was made to initiate cardiopulmonary bypass and ligate the lesions from within the PA. The PA catheter was withdrawn, and the PA was opened, revealing a large fistula opening with continuous blood flow while on CPB. Bypass flows were briefly reduced to target a MAP of 30 to allow for ligation. After repair, the patient was weaned off CPB and taken to the ICU. TEE confirmed reduction of flow through the fistula, and mixed venous blood gas no longer showed evidence of L- >R shunt. She was extubated 4 hours later and left the ICU on postoperative day #2 without complication.

Discussion

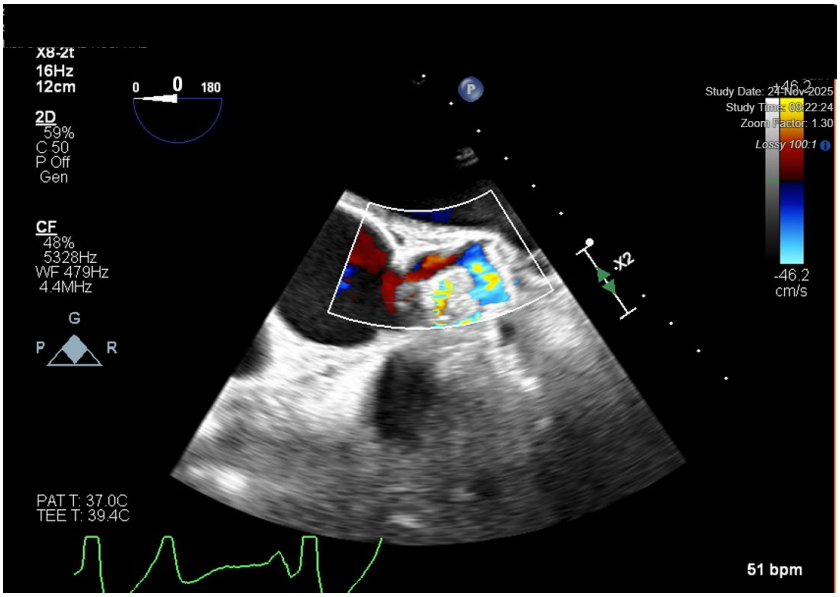
This case was performed using general anesthesia with a single-lumen endotracheal tube and cardiopulmonary bypass with central cannulation. Multimodality imaging was crucial, as it identified the fistula, revealed left-to-right shunting (thus explaining her symptoms), and highlighted that the fistula insertion was close to the pulmonary valve. This revealed increased the risk of damage to the valve if ligating the fistula externally, thus necessitating CPB and internal repair. Intraoperative Transesophageal Echocardiography, in combination with PA catheterization for mixed and central venous blood sampling, enabled the quantification of the shunt and confirmed successful closure. Coronary to PA fistula is a rare but significant defect that can cause symptoms including angina and heart failure. Definitive therapy includes closure of the fistula, either endovascularly or via an open surgical procedure. Intraoperative

guidance through a combination of echocardiography and PA catheterization allows precise targeting of the lesion and confirmation of adequate closure postoperatively.

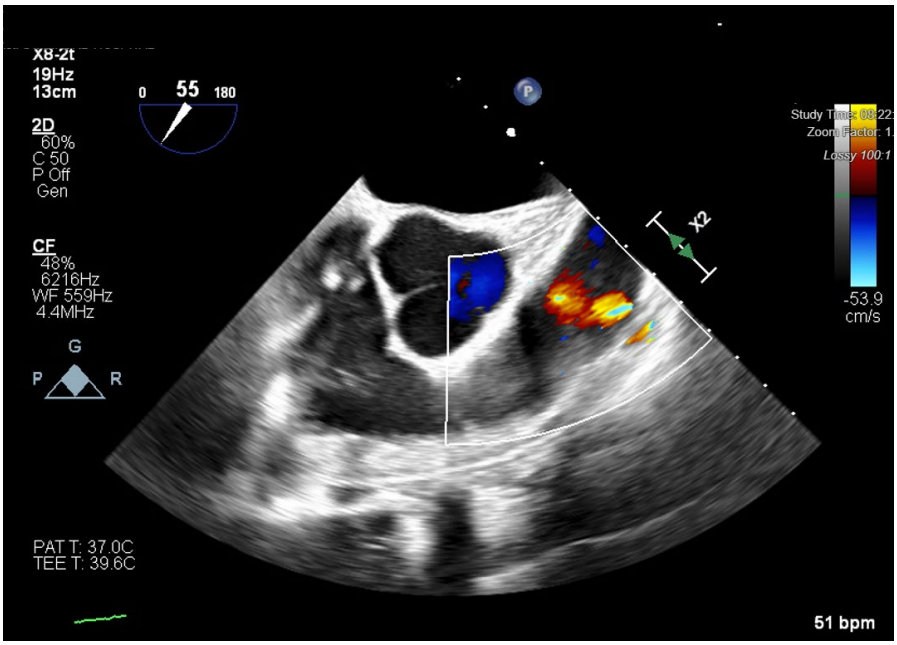
Images



3D reconstruction from CT-A showing Coronary to PA fistula with left to right shunt.



Pre-bypass mid-esophageal ascending aorta short-axis TEE image showing Coronary-PA fistula with significant left-to-right shunt seen via color flow doppler.



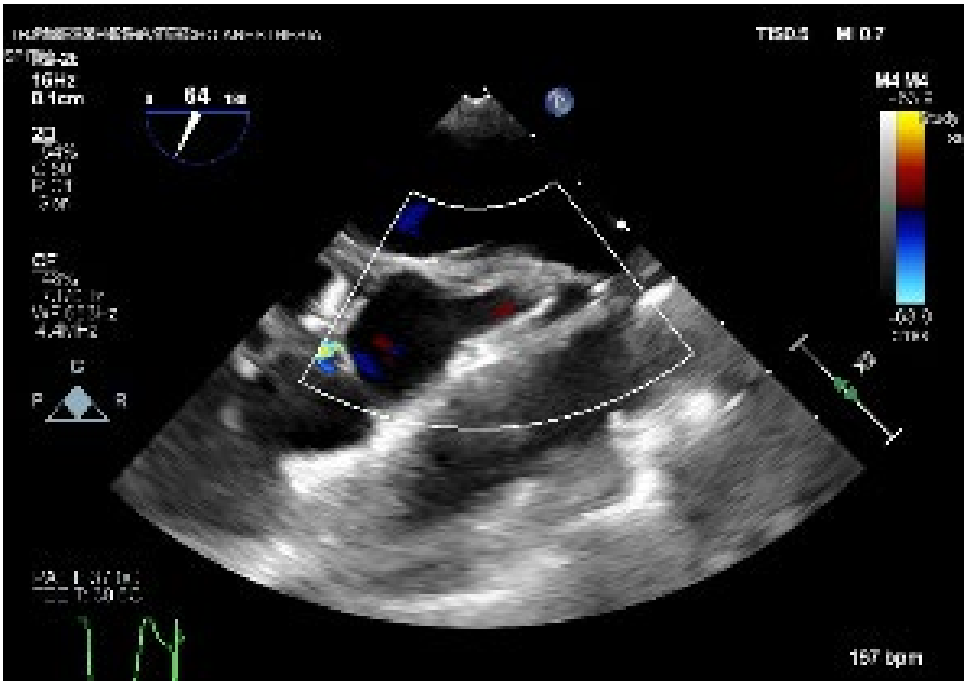
Pre-bypass mid-esophageal RV inflow-outflow TEE image showing Coronary-PA fistula with significant left-to-right shunt seen via color flow doppler.



Intraoperative photo showing blood flow from fistula into PA while on cardiopulmonary bypass.



Intraoperative photo showing ligation of the fistula with subsequent control of shunt.



Post-bypass mid-esophageal ascending aorta short-axis TEE image showing a significant decrease in flow through the Coronary-PA fistula.

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#FR 2 – Kiosk 5 – Poster 6

Left Atrial Appendage Occlusion Device Leak Intra-Op: Now What?

Presenting Author: Ragasnehith Maddula, MD, Anesthesiology Resident, Yale University

Introduction

In patients with nonvalvular atrial fibrillation, percutaneous left atrial appendage occlusion (LAAO) offers stroke prevention comparable to oral anticoagulation. PROTECT AF showed the WATCHMAN device to be noninferior to warfarin for cardiovascular death, stroke, or systemic embolism, with durable benefit at 5 years (1). The Amulet IDE trial demonstrated that, compared with WATCHMAN, the Amulet device more frequently achieved no/minimal residual leak at 12 months and was noninferior for safety at 12 months and efficacy at 3 years; at 5 years Amulet was associated with lower rates of device-related thrombus, peri-device leak, fatal/disabling stroke, and less long-term anticoagulation use (2). Despite these data, guidance remains limited regarding intra-operative and post-operative management of device-related leaks and anesthetic considerations, including risk stratification and antithrombotic strategies. Successful LAA occlusion with fixed-shape devices is challenged by wide variability in appendage size, shape, and orientation. PROTECT AF and Amulet IDE both reported peri-device leaks (PDLs) on follow-up imaging, with leak rates declining between early and 12-month assessments (3). Alkhouli et al. showed that PDLs are not benign, demonstrating associations with ischemic events and emphasizing the need for complete LAA seal while noting that leak incidence declines with operator experience and device refinement (3). Although PDLs are not typically classified as primary device failure, they often prompt discontinuation of anticoagulation, continuation of long-term antiplatelet therapy, and serial imaging surveillance.

Case Presentation

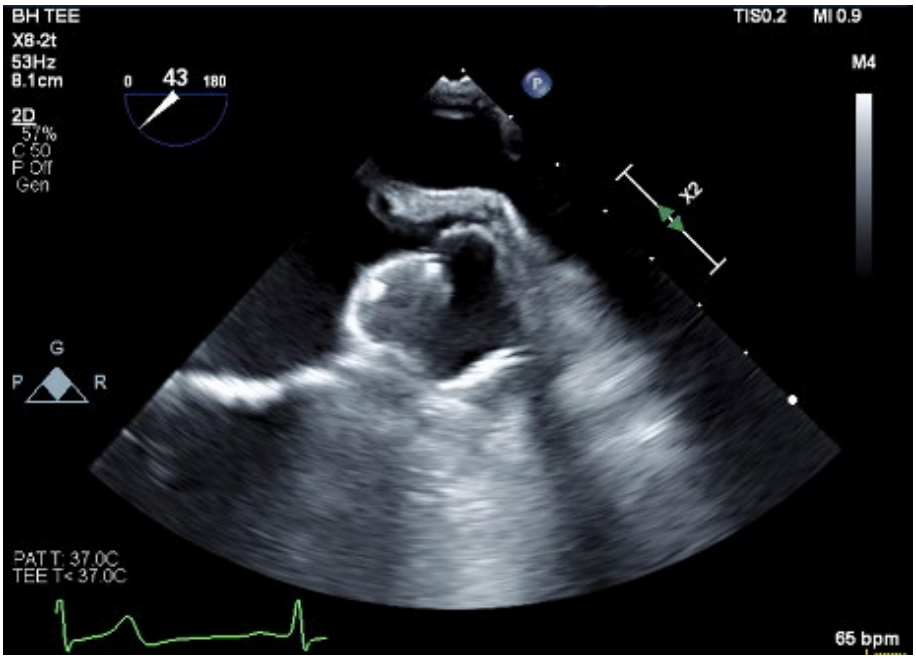
A 70-year-old woman with end-stage renal disease on hemodialysis, hypertension, paroxysmal atrial fibrillation, and obstructive sleep apnea underwent WATCHMAN LAAO. Her CHA2DS2-VASc score was 6 and HAS-BLED score 4. Intra-operatively, a 27-mm WATCHMAN device appeared well seated with satisfactory closure. Follow-up TEE demonstrated a new 2-mm trans-fabric intradevice leak. Given the small leak, warfarin was discontinued and dual antiplatelet therapy with aspirin and clopidogrel was initiated, with plans for repeat TEE or cardiac CTA in the ensuing months.

Discussion

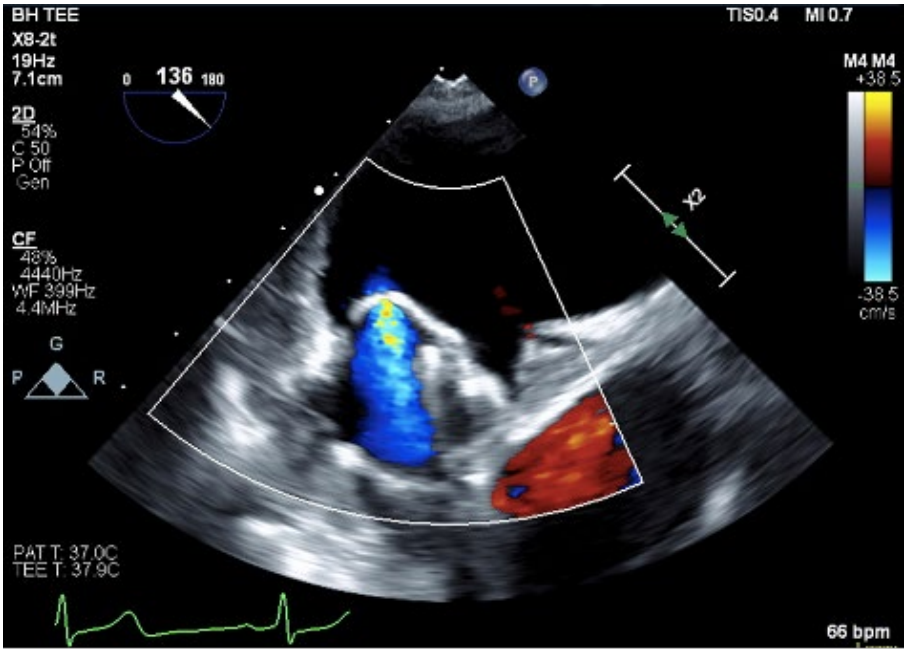
Saito et al. evaluated 1397 patients undergoing LAAO and identified PDLs in 84 patients (6%); 73 of these (86.9%) had leaks ≤3 mm, typically in patients with larger left atria and higher CHADS2 scores. Even small (≤3-mm) PDLs were significantly associated with increased risk of transient ischemic attack, ischemic stroke, or systemic embolism. Current literature provides limited guidance on anesthetic management of intra-procedural or incidentally detected leaks. Contemporary practice generally favors discontinuation of systemic anticoagulation once endothelialization is presumed and continuation of dual antiplatelet therapy with interval imaging to monitor the leak. Percutaneous or surgical repair strategies are not routinely pursued, as antiplatelet therapy remains the main approach to mitigate thromboembolic risk in the setting

of a small PDL. Ongoing device innovations, including more conformable devices and steerable delivery sheaths that improve coaxial alignment, aim to optimize seal and further reduce the incidence and clinical impact of peri-device leaks (3).

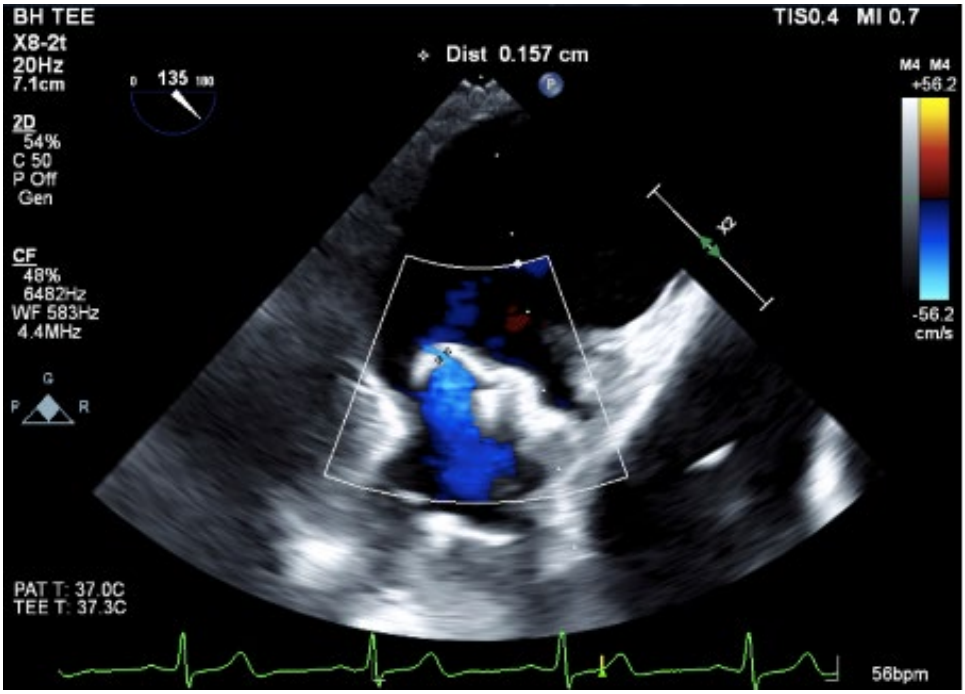
Images



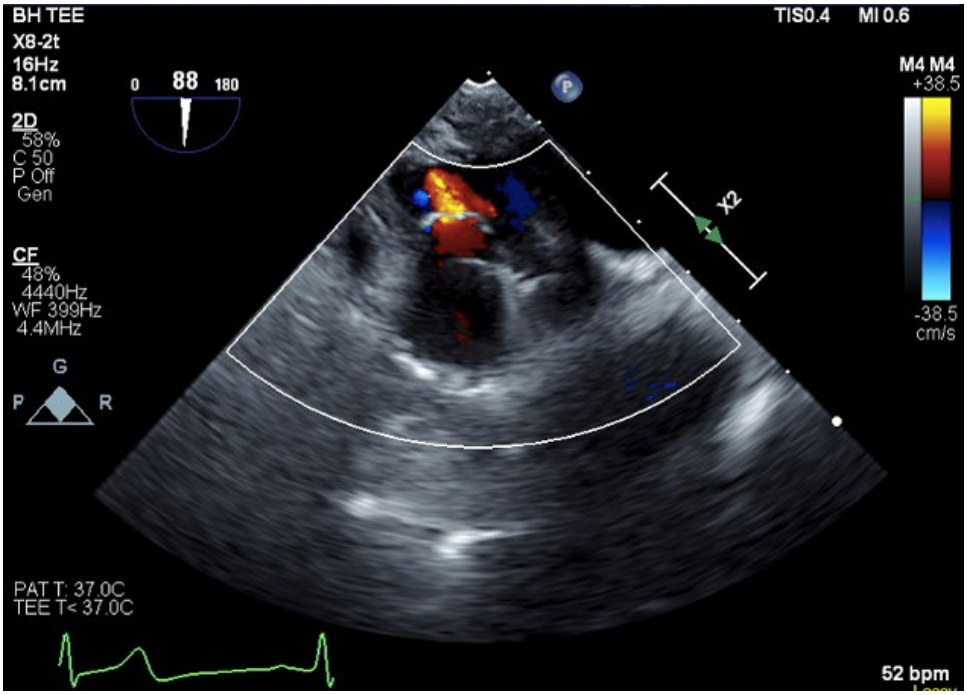
Device Well Seated in LAA



Intradevice Flow



Jet Width



Excursion Jet Out of Device

References

WATCHMAN Left Atrial Appendage System for Embolic Protection in Patients With Atrial Fibrillation - PROTECT-AF Trial (https://www.acc.org/latest-in-cardiology/clinical-trials/2014/03/24/23/43/protect-af)

Amplatzer Amulet Left Atrial Appendage Occluder IDE - Amulet IDE Trial (https://www.acc.org/latest-in-cardiology/clinical-trials/2021/08/26/01/36/amulet-ide)

Residual Leaks Post Left Atrial Appendage Occlusion (https://www.acc.org/Latest-in-Cardiology/Articles/2022/07/05/18/58/Residual-Leaks-Post-Left-Atrial-Appendage-Occlusion)

#FR 2 – Kiosk 5 – Poster 7

Aortic Insufficiency after Mechanical Mitral Valve Replacement due to Inadvertent Pinning of the Non-coronary Cusp: The Value of Deep Trans-gastric and Multiplanar TEE Imaging

Presenting Author: Rachel S. Hanke, MD, University of Florida

Introduction

Acute aortic insufficiency (AI) is an uncommon complication after mitral valve replacement (MVR). Many cases in the literature were found postoperatively, requiring reoperation. We present a case of AI identified intraoperatively on transesophageal echocardiography (TEE) after MVR for endocarditis.

Case Presentation

A 28-year-old male with asthma presented with 1 month of worsening dyspnea. TEE showed severe eccentric mitral regurgitation (MR) due to a large posterior mitral leaflet vegetation with perforation. He had normal LVEF, moderate RV dysfunction, trace AI and moderate pericardial effusion. A MVR was performed via an extended transseptal exposure using a 31/33 On-X mechanical valve and the left atrial appendage was ligated using a 50 Flex-V clip. Post cardiopulmonary bypass (CPB) TEE showed a well-seated mechanical prosthesis with no regurgitation. Midesophageal (ME) aortic valve (AV) evaluation was limited by acoustic shadowing. New severe RV dysfunction and moderate LV dysfunction required hemodynamic optimization with inotropes. After this, comprehensive TEE with deep transgastric (DTG), X-plane and 3D images revealed severe AI with restriction of the non-coronary cusp (NCC). The hemodynamically significant AI prompted reinitiation of CPB to inspect the valve. After cardioplegic re-arrest, low transverse aortotomy revealed the NCC was inadvertently pinned by two anterior mitral annular sutures. The mitral valve was re-exposed and the offending sutures were removed. A pair of new sutures were placed everting the left atrial wall onto the prosthetic cuff to prevent paravalvular leak. Post-revision TEE showed normal mechanical MV function (mean gradient 3mmHg) and mild AI with 2 trace regurgitant jets attributed to pinhole defects in the NCC from suture passage. LVEF was severely reduced to 15-20% with global hypokinesis. The RV was unchanged with moderate dysfunction. The patient was transported to ICU on inotropic support. The postoperative course was otherwise unremarkable. The patient was discharged home on postoperative day 15 on long-term IV antibiotics. Follow-up TTE showed trace AI with persistently low EF. He is continuing to follow up with the heart failure team.

Discussion

Anatomical proximity of mitral and aortic valves confers a low but serious risk of AV injury during mitral surgery. Mechanisms of AI after mitral surgery include aortic annular distortion, and leaflet or annular injury from mitral annular suture passage or anterior leaflet debridement. TEE after MVR is crucial to evaluate prosthetic valve function, paravalvular leaks and inadvertent injuries. In this case, ME views alone were insufficient to detect AI due to turbulent mitral inflow related to the mechanical prosthesis. DTG, X-plane, and 3D imaging were crucial in identifying severe AI and NCC restriction. Comprehensive imaging was delayed until after decannulation due to biventricular dysfunction needing hemodynamic stabilization. Prior

reports describe postoperative discovery of AI requiring reoperation. Our case emphasizes comprehensive intraoperative TEE after MVR in improving outcomes and highlights the utility of DTG and multiplanar imaging in the presence of mechanical prosthesis. Early acquisition of these views before decannulation may allow for rapid recognition and repair.

Images

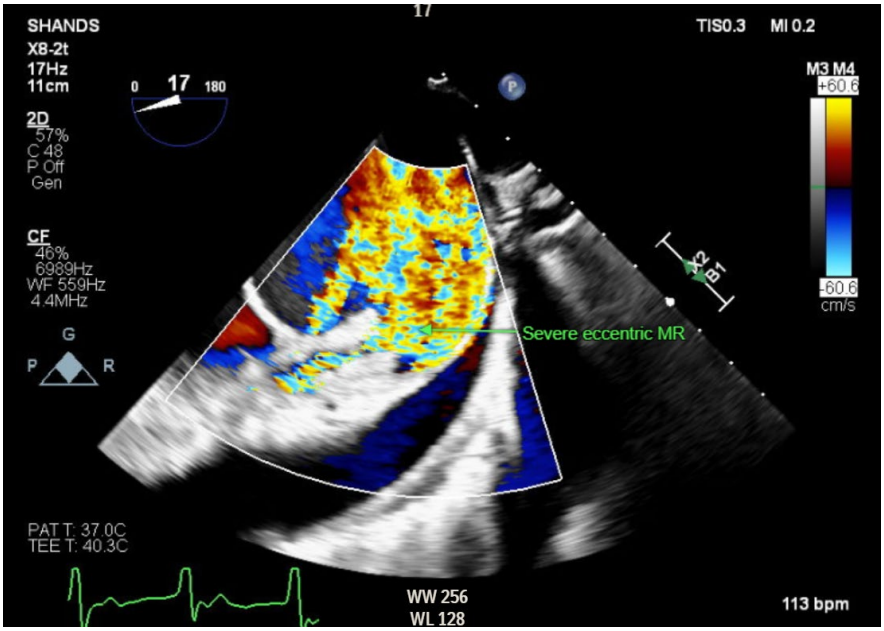


Figure 1. Preoperative posterior MVL perforation with severe eccentric MR.

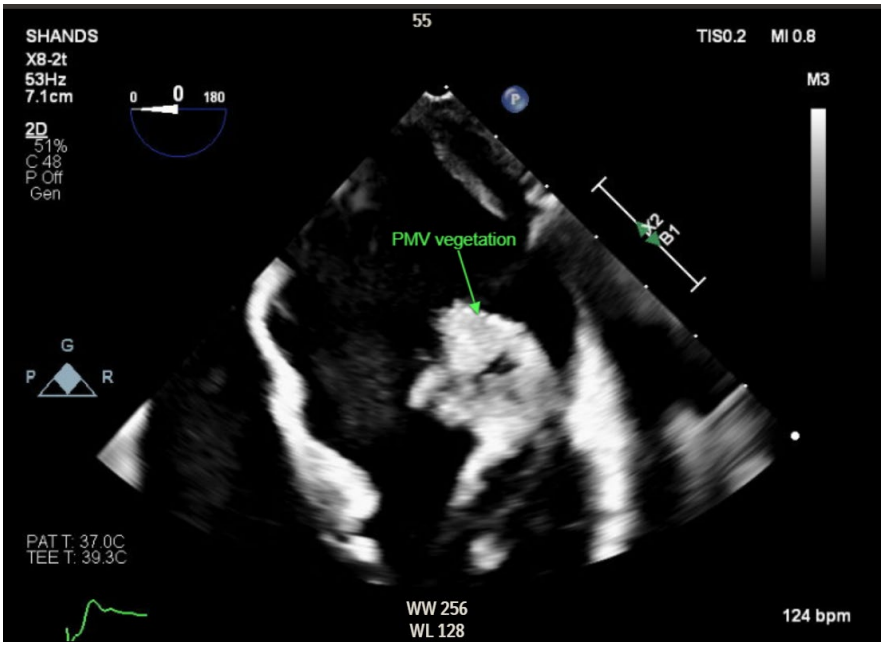


Figure 2. Preoperative posterior MVL vegetation.

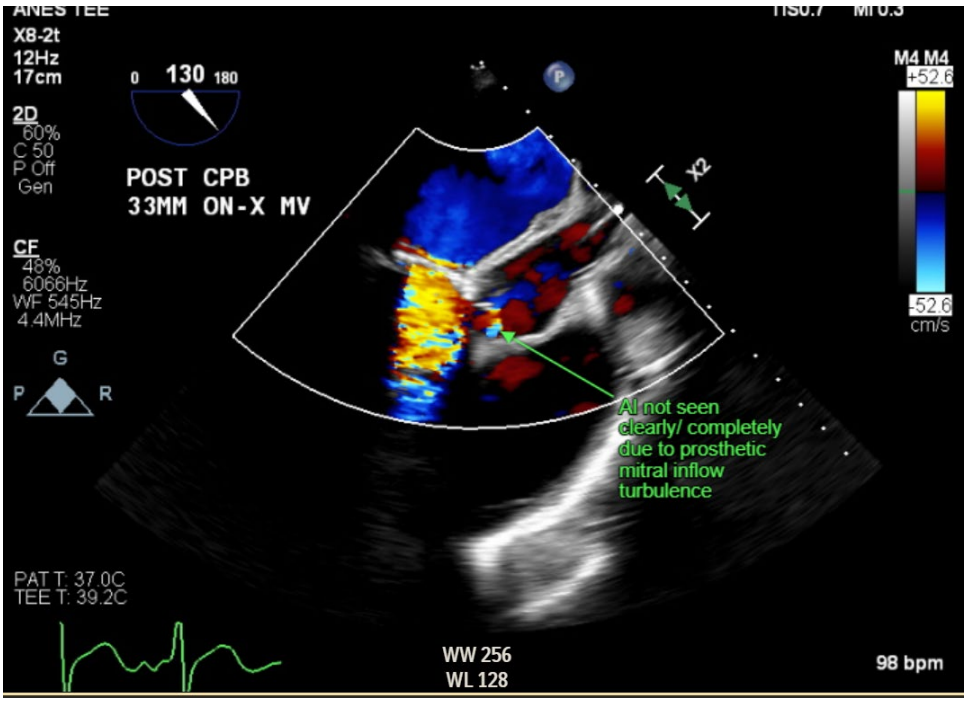


Figure 3. Postoperative AI incompletely seen due to turbulence from mitral inflow of the mechanical valve.

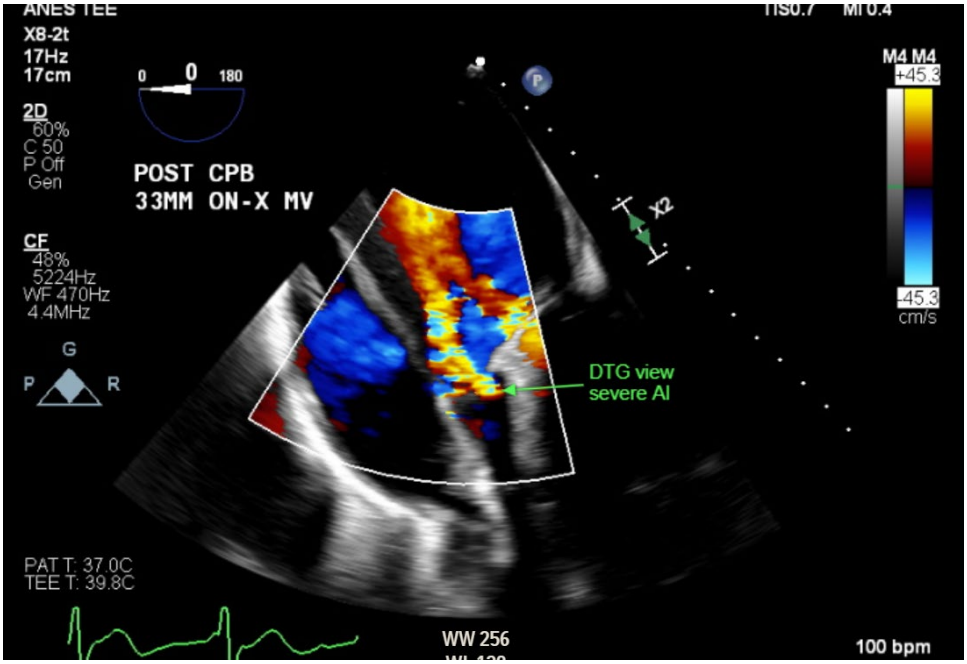


Figure 4. Postoperative severe AI in the Deep Trans-Gastric view.

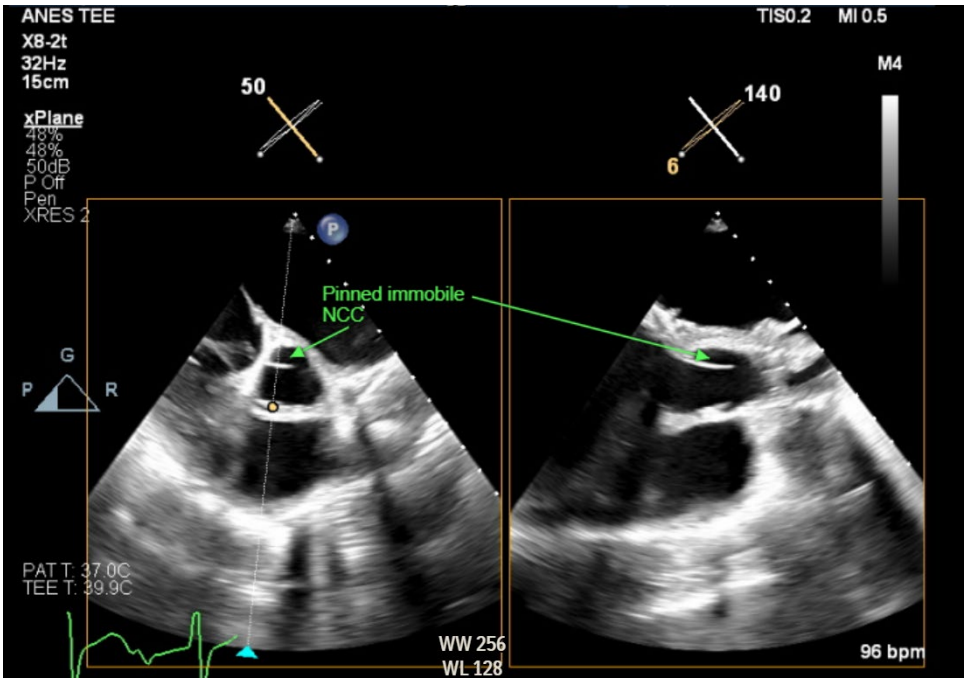


Figure 5. Postoperative AV in ME views with X-plane imaging showing the pinned NCC.

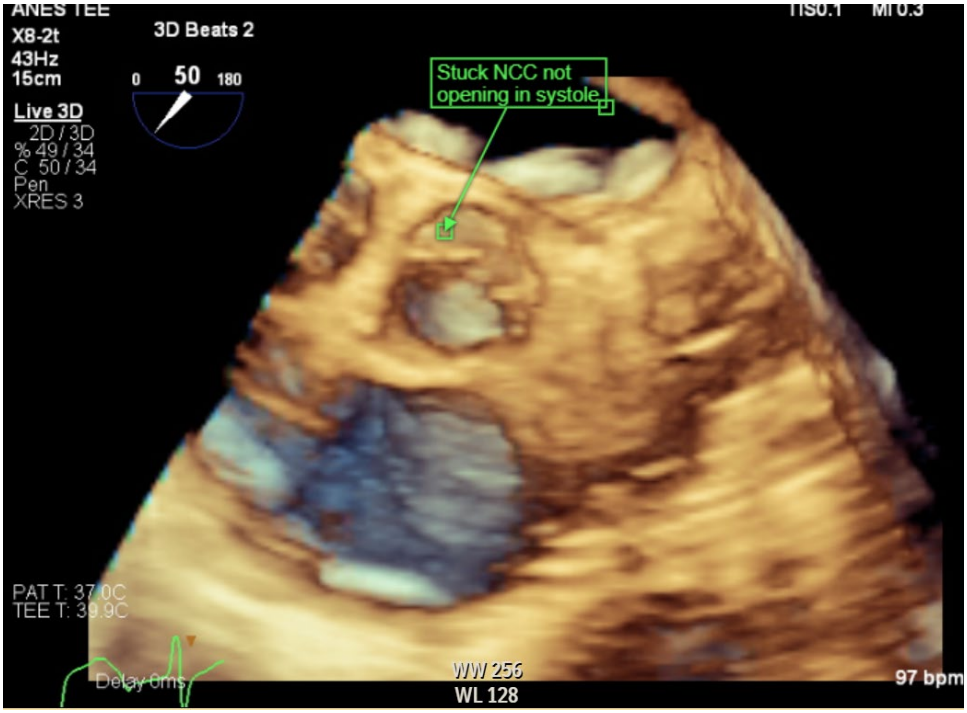


Figure 6. Postoperative 3D imaging showing the restricted NCC not opening in systole.

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#FR 2 – Kiosk 6 – Poster 1

Perioperative Management of a Left Atrial Myxoid Sarcoma: A Case of Postoperative Pulmonary Venous Stenosis

Presenting Author: Julian Conn Busch, MD, Resident Physician, Hospital of the University of Pennsylvania

Introduction

Primary cardiac sarcomas have an extremely poor prognosis, are exceptionally rare (< 0.03% of cardiac tumors) and may mimic benign myxomas at presentation. When feasible, complete surgical resection with adjuvant chemotherapy is preferred as it is associated with the longest survival. When masses are located near pulmonary venous inflow, they present distinct anesthetic and surgical challenges, including the risk of postoperative venous obstruction. Intraoperative transesophageal echocardiography (TEE) is critical for diagnosis, surgical guidance, and immediate assessment of complications. We describe the anesthetic and surgical management of a left atrial myxoid sarcoma complicated by postoperative pulmonary venous stenosis.

Case Presentation

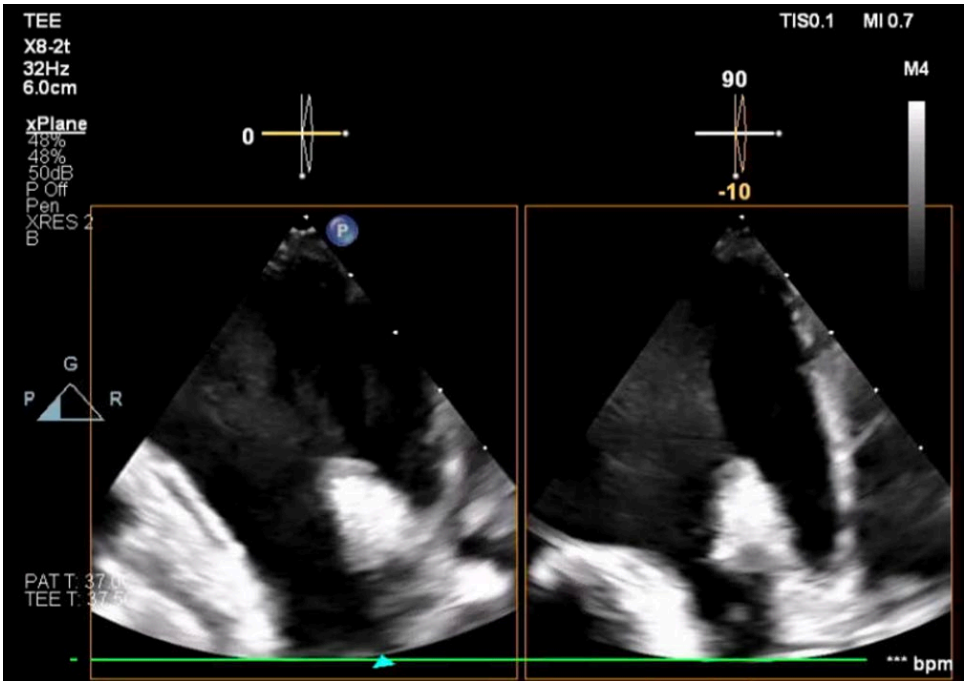
A 23-year-old man with autism and a remote seizure disorder presented after a new generalized seizure with multifocal intracerebral hemorrhages. Workup revealed a left atrial wall mass adjacent to the left lower pulmonary vein suggestive of a myxomatous or malignant lesion. The patient was subsequently taken to the operating room for mass excision. After uneventful induction of general anesthesia, pre-sternotomy TEE confirmed a 1.3 × 0.9 cm mobile mass attached to the lateral left atrial wall near but not invading the pulmonary veins. Following excision and bovine pericardial patch repair, post-bypass TEE demonstrated new flow acceleration (146 cm/s) in the left lower pulmonary vein consistent with mild stenosis and preserved RV function. LV function was moderately decreased (EF 35–40%). The patient was weaned from cardiopulmonary bypass without issue. The patient was extubated on postoperative day 0 and was discharged to acute rehab. Pathology confirmed myxoid-type intimal sarcoma (MDM2-positive). Transthoracic echocardiography one month post-operatively showed recovered LVEF (50-55%), normal RV function, and normal bi-atrial size. The patient subsequently underwent whole-brain radiation therapy for newly discovered brain metastases and started systemic chemotherapy.

Discussion

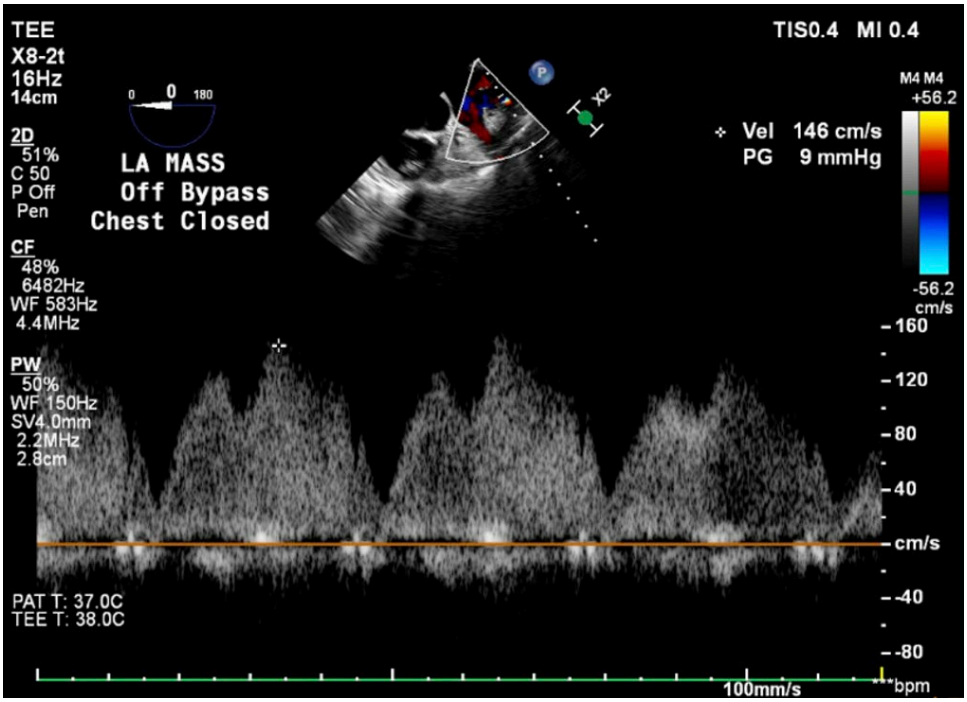
This case underscores key anesthetic considerations for cardiac tumors near pulmonary venous structures. In this case, venous flow abnormalities were inevitable given the location of the tumor. Preoperative imaging helps define the lesion's anatomic relationships and exclude inflow obstruction. Intraoperative TEE plays a pivotal role in guiding surgical resection, detecting residual flow abnormalities, and identifying early postoperative complications such as venous stenosis. Postoperative echocardiographic surveillance remains essential, as even subtle flow acceleration may signify evolving iatrogenic narrowing. Recognition of such hemodynamic changes can guide early intervention and optimize long-term outcomes in patients

undergoing resection of rare cardiac sarcomas.

Images







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#FR 2 – Kiosk 6 – Poster 2

Bringing the Pulmonic Valve into Focus: Epicardial Imaging as a Critical Intraoperative Guide

Presenting Author: Olivia Sonderman, MD MPH, Fellow, MGH

Introduction

Pulmonary valve disease is almost always congenital in origin, and pulmonary valve surgery is significantly less common than other valve surgery. As an anterior structure, the pulmonic valve is often challenging to interrogate with transesophageal echocardiography (TEE). We present a case of pulmonary valve replacement with primary pulmonary homograft failure where epicardial ultrasound augmented TEE imaging.

Case Presentation

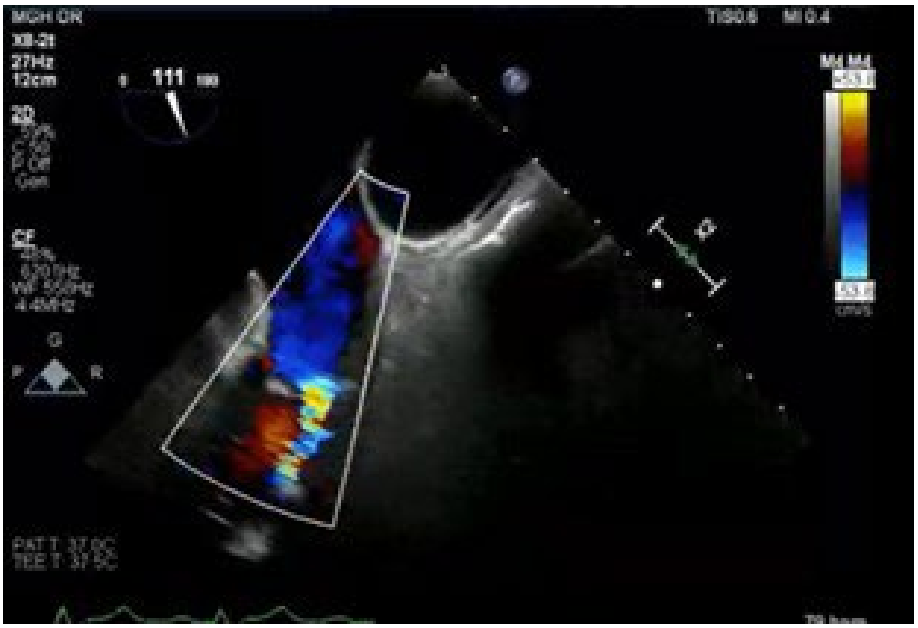
A 56-year-old female with a past medical history of congenital pulmonary stenosis status post surgical valvotomy and right ventricular outflow tract (RVOT) patch at 5 years of age presented with shortness of breath on exertion. Initial evaluation revealed recurrent pulmonary stenosis as well as pulmonary regurgitation, right ventricular dilation, pulmonary artery dilation, and right bundle branch block. A percutaneous intervention was attempted, but the procedure was aborted because the pulmonary annulus and pulmonary artery were too dilated to safely seat a HARMONY 25 mm transcatheter pulmonary valve. The patient was scheduled for pulmonary valve replacement with cadaveric pulmonary homograft. On the day of surgery, general anesthesia and cardiopulmonary bypass (CPB) were initiated without complication. Initial TEE assessment revealed mild pulmonary stenosis and moderate to severe pulmonary regurgitation (Image A & B). The native pulmonary valve was resected and the pulmonary homograft was implanted. On post-CPB TEE, visualization of the pulmonary valve leaflets was challenging. However, the exam was concerning for a hyperechoic 7 mm mass in the RVOT and moderate pulmonary regurgitation (Image C and D). An epicardial probe was placed in a sterile sheath with saline and applied directly to the neo-pulmonic valve. Epicardial evaluation (Image E,F,G) revealed the hyperechoic mass superior to and separate from the pulmonic valve leaflets. Moderate to severe pulmonic regurgitation was appreciated. The homograft was resected. Upon pressurizing the pulmonary artery of the homograft, eccentric leakage was noted. Exhaustive inspection revealed significant leaflet prolapse. A 29 mm KONECT bioprosthetic aortic valved conduit was placed in the pulmonic position. Post-CPB evaluation revealed trace pulmonary regurgitation. Peak and mean gradients were 8 and 4, respectively. The patient was discharged to home five days later.

Discussion

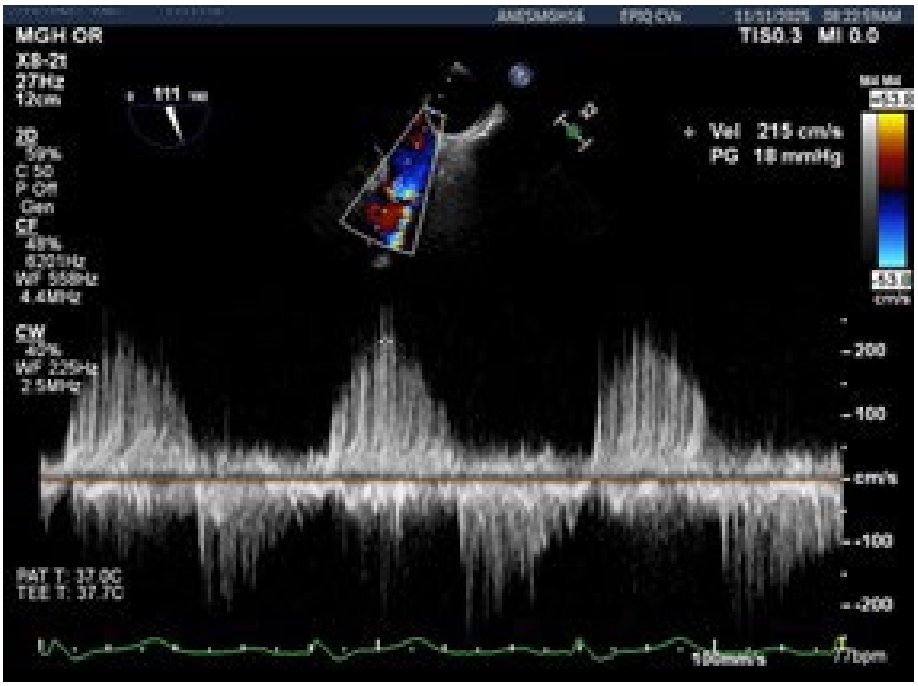
The pulmonic valve is infrequently intervened upon. Similar principles of interrogation applied to other valves may be applied for the pulmonic valve.1,2 Anterior structures, like the pulmonic valve, may be difficult to visualize with TEE due to intervening structures. In this case, the failure of the pulmonary homograft was unexpected. Epicardial ultrasound was critical in identifying the failure and mechanism of the pulmonary homograft and supported the decision to resect the homograft. It is important to comprehensively evaluate the pulmonary valve by TEE and if needed by epicardial ultrasound to guide management for patients. For

any anterior structures, epicardial ultrasound offers a tool to complete a more comprehensive evaluation when TEE is insufficient.

Images



Pre-cardiopulmonary bypass aortic arch short axis view revealing moderate pulmonary regurgitation.



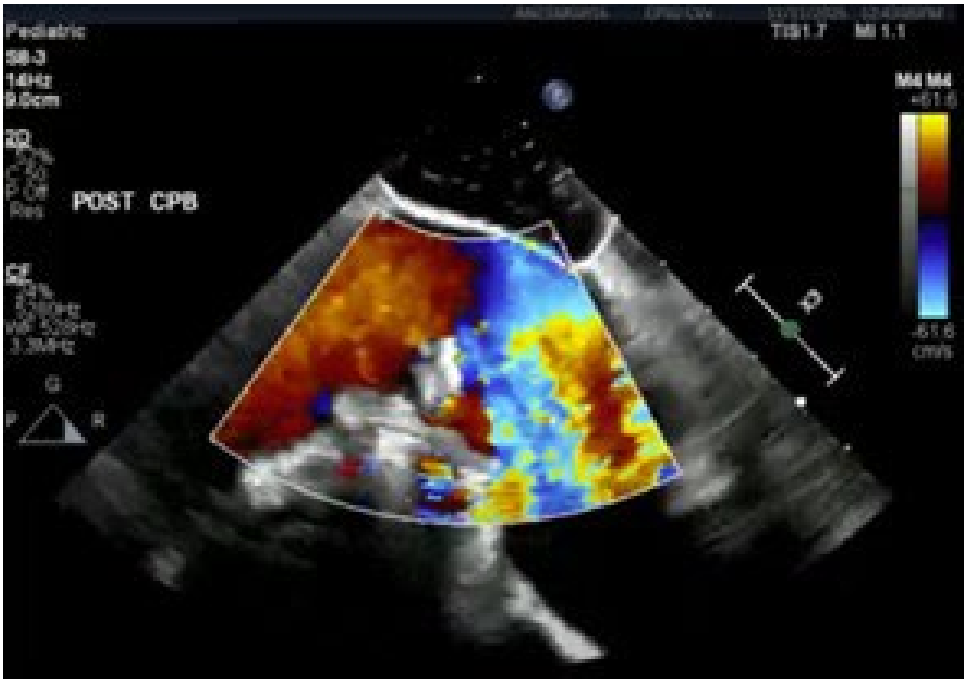
Pre-cardiopulmonary bypass aortic arch short axis view revealing mild pulmonic stenosis.



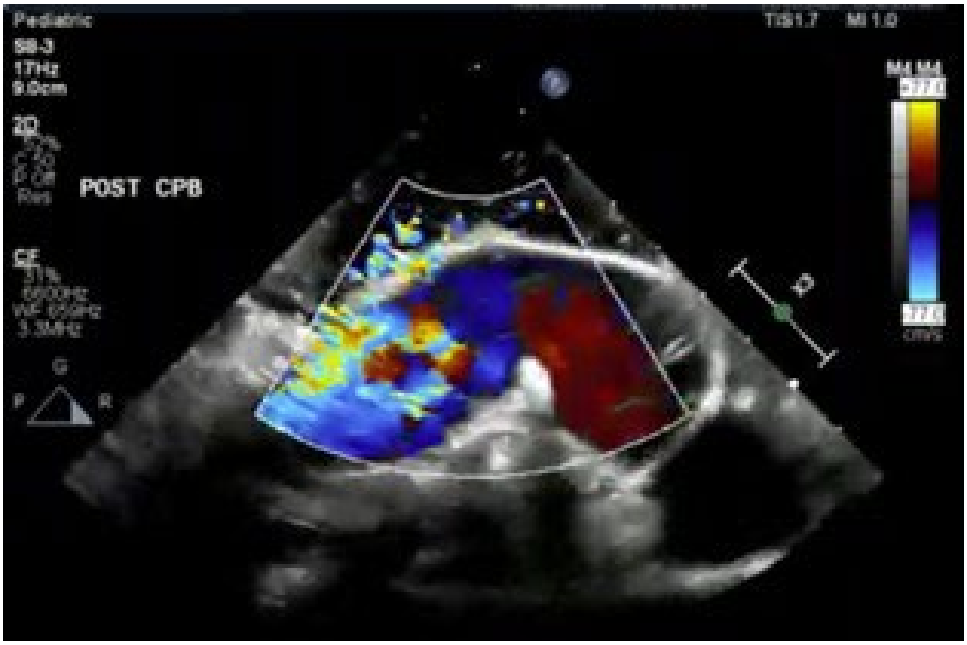
Post-cardiopulmonary bypass right ventricular inflow outflow view with hyperechoic mass and significant regurgitation of unknown etiology.



Epicardial ultrasound with fluid standoff at near field showing pulmonary valve leaflets and hyperechoic redundant tissue in main pulmonary artery.



Significant flow acceleration in the main pulmonary artery from hyperechoic mass.



Moderate to severe pulmonary regurgitation.

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Reeves ST, Glas KE, Eltzschig H, Mathew JP, Rubenson DS, Hartman GS, Shernan SK; American Society of Echocardiography; Society of Cardiovascular Anesthesiologists. Guidelines for performing a comprehensive epicardial echocardiography examination: recommendations of the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. J Am Soc Echocardiogr. 2007 Apr;20(4):427-37. doi: 10.1016/j.echo.2007.01.011. PMID: 17400124.

#FR 2 – Kiosk 6 – Poster 3

Treatment of LV Apical Pseudoaneurysm using an Amplatzer Device

Presenting Author: Andrew J. Egan, DO, Resident Physician, Maine Health

Introduction

Untreated left ventricular apical pseudoaneurysm (PSA) presents a high risk of rupture, up to 45% within one year[1]. Surgical repair is the traditional treatment of choice, but carries a high risk for mortality, ranging between 10-32%[2]. More recently, percutaneous closure has become a viable option for select high risk patients[3].

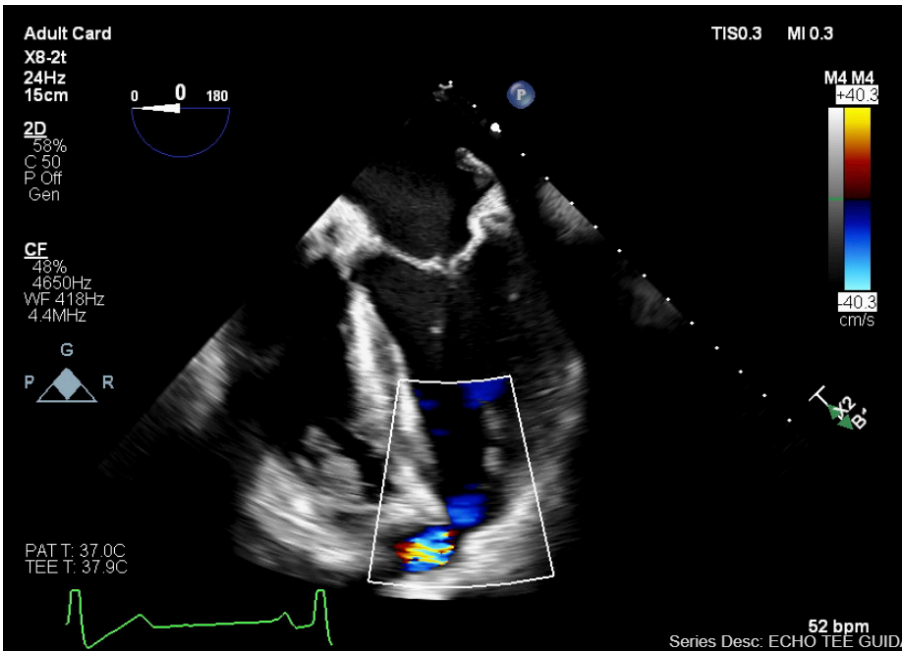
Case Presentation

We present the case of a 74 year old female with a complex cardiac surgical history involving hemiarch repair for a type A dissection, followed by redo sternotomy for treatment of an aortic root pseudoaneurysm. Approximately six months later, on routine surveillance imaging for newly diagnosed breast cancer, she was found to have an LV apical PSA measuring 3.0 x 2.8 x 4 cm. In light of patient frailty, her breast cancer diagnosis, and the perceived surgical risk of a third sternotomy, the decision was made to proceed with percutaneous closure of her LV PSA. The patient was taken to the hybrid operating room, where she was placed under general anesthesia. An arterial line was placed, and large-bore central venous access was secured. A transvenous pacing wire was placed from the right femoral vein, for the possibility of temporary pacing to prevent device distortion or dislodgement during deployment. Initial approach was via a retrograde fashion across the aortic valve. A stiff guidewire was able to be positioned within the PSA sac, but subsequent attempts to deliver the Amplatzer device beyond the neck of the PSA were unsuccessful. This was likely secondary to the acute angle between the aortic root and the vector of the neck of the PSA, preventing adequate length of the delivery sheath to reach beyond the neck into the body of the PSA. It was felt that an antegrade approach via transseptal puncture would reduce the distance and provide a more favorable angle required to reach the body of the PSA. Therefore, the femoral venous sheath was upsized and transseptal puncture was performed at the mid-mid aspect of the fossa ovalis. The antegrade approach allowed delivery of a catheter within the body of the PSA and subsequent deployment of a 10mm muscular VSD occluder. Post deployment ventriculography and TEE demonstrated cessation of flow into the LV PSA. Follow up CT scan 6 months later showed stable device placement and complete exclusion of the LV PSA.

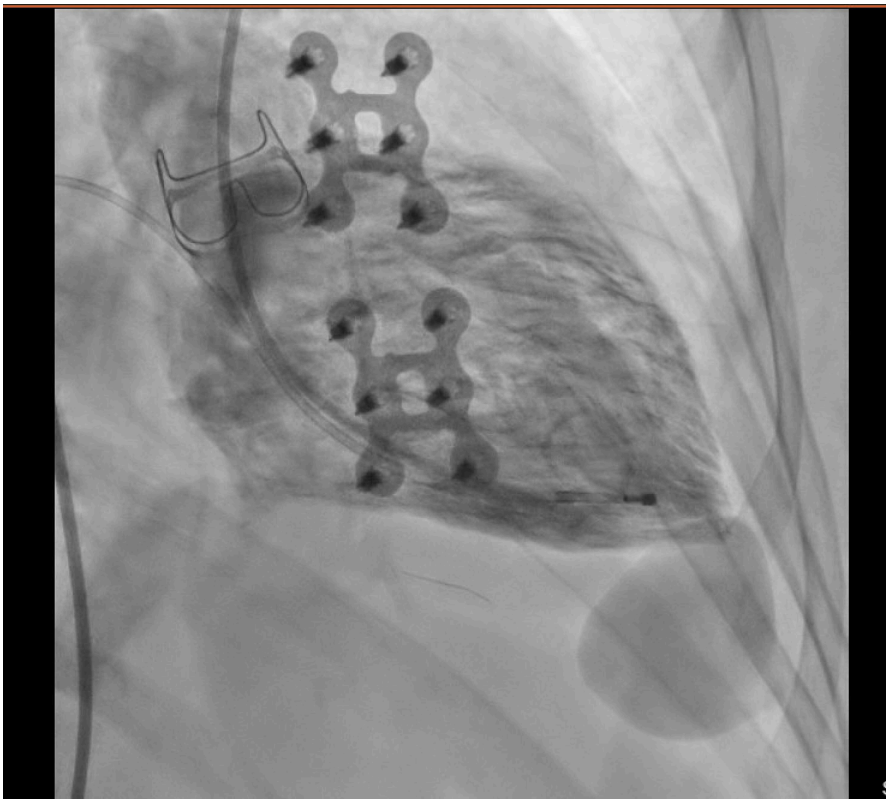
Discussion

This case highlights the need to take an individualized approach to complex endovascular interventions that account for variations in anatomy. As minimally invasive options for the treatment of LV apical PSA become more widespread, consideration of an antegrade versus retrograde approach depending on the angle of the PSA neck may prove helpful.

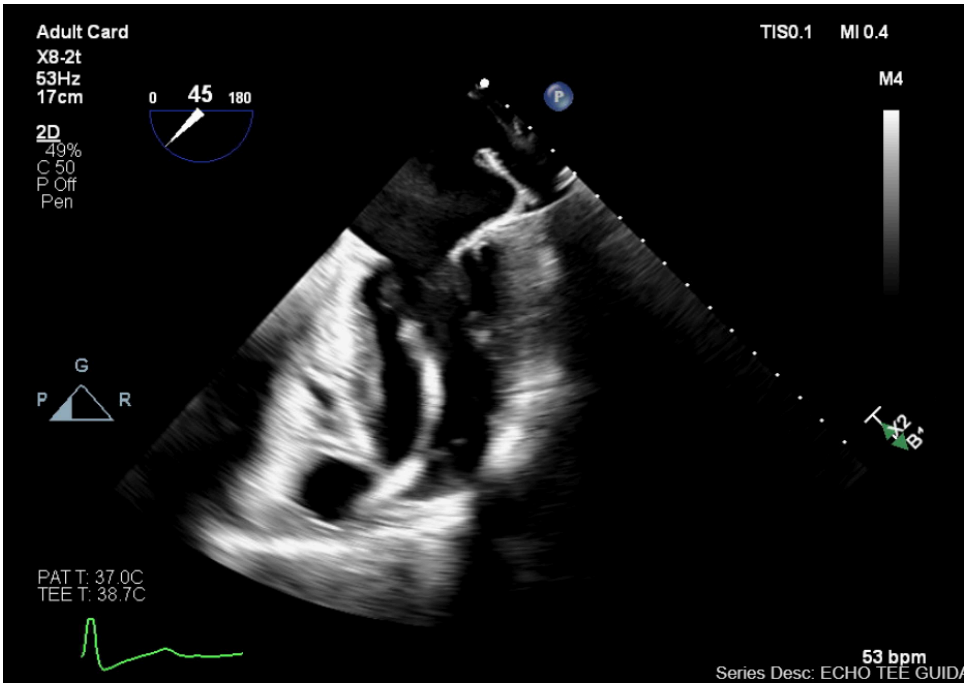
Images



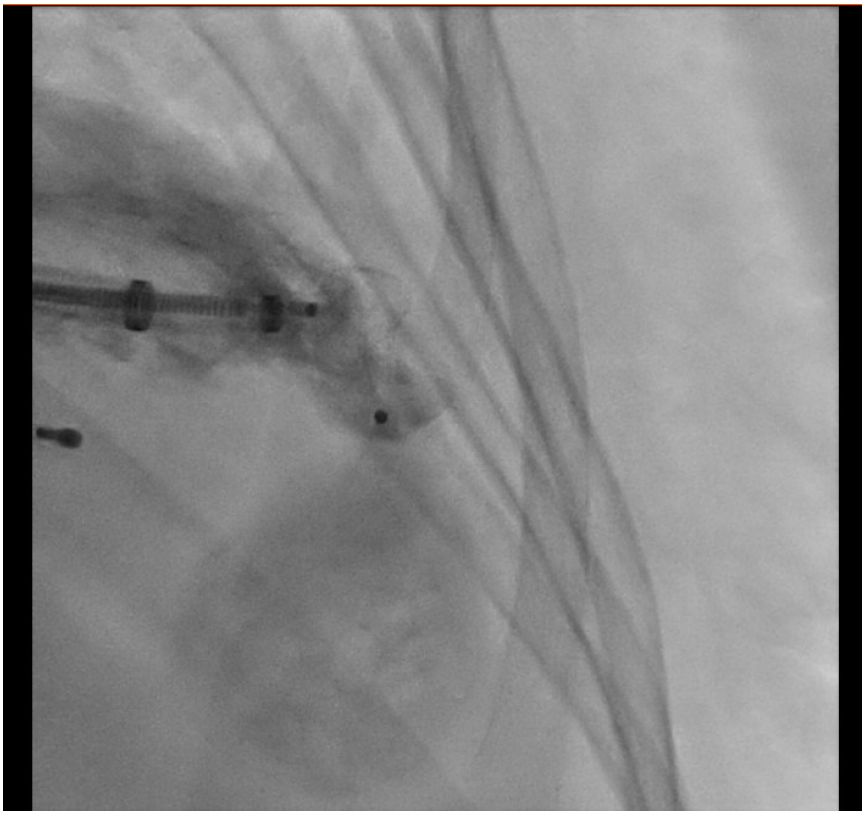
Mid-esophageal 4 chamber demonstrating flow into PSA



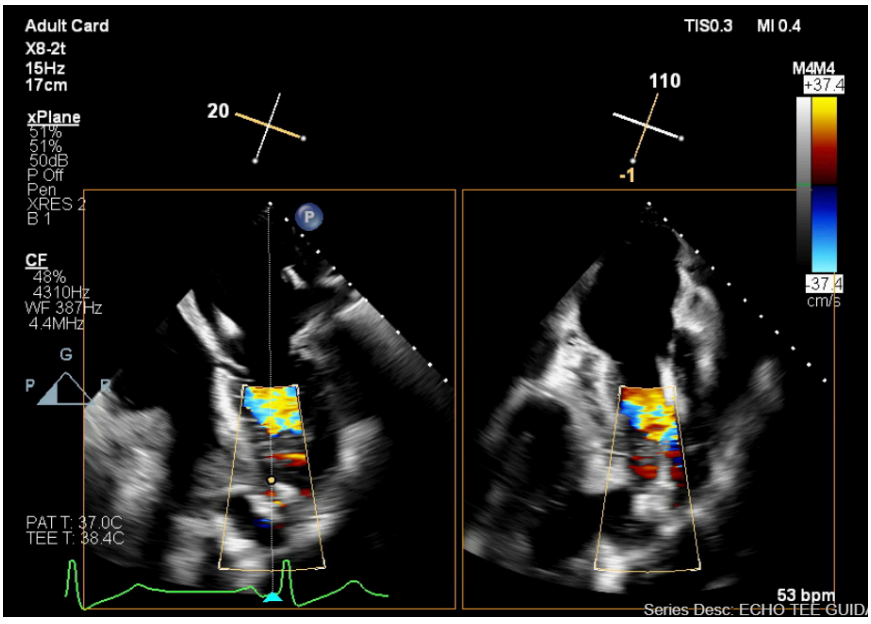
Antegrade approach demonstrating contrast filling PSA



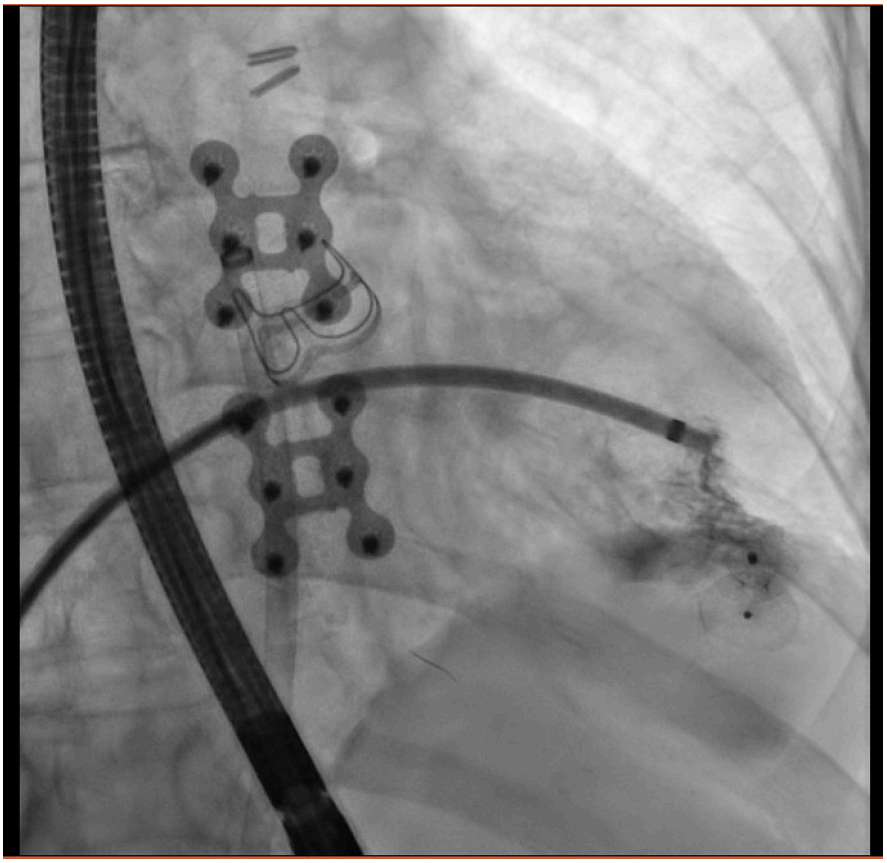
Antegrade approach, wire entering PSA



Antegrade approach, unable to adequately enter PSA with catheter to deploy Amplatzer



Retrograde approach demonstrating successful deployment of amplatzer into PSA



Fluoroscopy demonstrating successful occlusion of PSA with no contrast flowing into body via retrograde approach

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SCA ANNUAL MEETING & WORKSHOPS
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SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS

#FR 2 – Kiosk 6 – Poster 4

Anesthetic Considerations for UNICORN-assisted Leaflet Modification in Valve-in-Valve TAVR

Presenting Author: William H. Jones, DO, Resident Physician, Medical College of Georgia

Introduction

Valve-in-valve (ViV) transcatheter aortic valve replacement (TAVR) is critical for patients with failed bioprosthetic aortic valves who are poor candidates for redo surgery. Coronary obstruction, most often involving the left main, remains a serious complication. Measuring coronary ostial height, sinus of Valsalva width, and valve-to-coronary distance is critical to stratify risk. Traditionally, the BASILICA technique allows leaflet splaying from the coronary ostium after valve deployment., but is technically demanding, requiring dual-catheter manipulation and snaring. The UNICORN (Undermining Iatrogenic Coronary Obstruction with Radiofrequency Needle) technique offers a simpler alternative. Radiofrequency traversal followed by balloon dilation weakens the leaflet, enabling outward folding without snaring. While many ViV TAVRs are performed under sedation, cases requiring leaflet modification or precise valve positioning benefit from general anesthesia with transesophageal echocardiography (TEE). In this case, general anesthesia facilitated accurate positioning and visualization of both coronary ostia. We report anesthetic and perioperative considerations for a successful UNICORN-assisted ViV TAVR.

Case Presentation

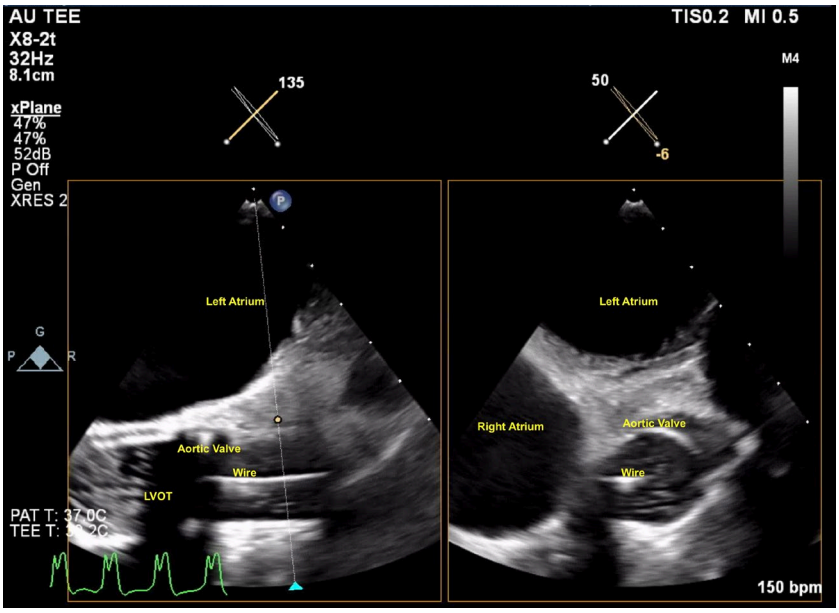
An 82-year-old female presented for ViV TAVR due to severe stenosis of a 2014 bioprosthetic aortic valve. She reported progressive dyspnea and lightheadedness over several months. Her history included HTN, HLD, GERD, COPD on home oxygen, OSA, and pulmonary hypertension. Cardiac catheterization showed nonobstructive coronary arteries and elevated pulmonary artery pressures. TTE showed preserved ventricular function and a mean AV gradient of 55mHg with an AVA of 0.67cm2. CT imaging demonstrated low coronary ostial height and a narrow sinus, indicating high risk for obstruction. General anesthesia was induced uneventfully with a left radial arterial line. Cerebral oximetry was used as a perfusion adjunct. Vascular access was obtained via the femoral vessels for valve deployment, angiography, and temporary pacing . A sentinel device was advanced through the right radial for cerebral protection. With TEE guidance, the UNICORN technique was performed using radiofrequency traversal of the left coronary cusp followed by serial balloon dilation with D5W as a conductive medium. A 20mm Sapien 3 Ultra Resilia valve was deployed within the prior 21mm surgical valve, restoring forward flow without coronary compromise. The patient was extubated in the operating room and transported to recovery in stable condition. Echocardiography showed a preserved ejection fraction, mean gradient of 17mmHg, and no perivalvular leak.

Discussion

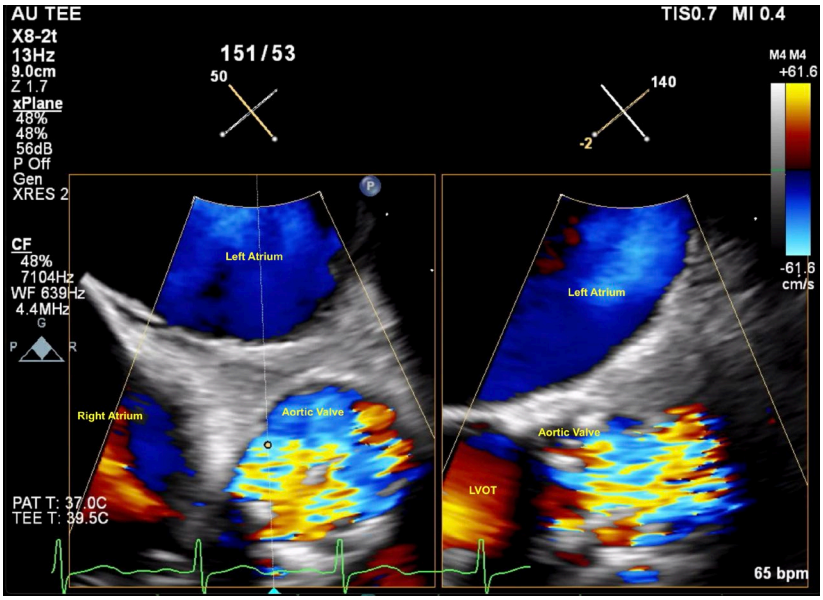
Coronary obstruction during ViV TAVR is rare but associated with high mortality, especially patients with small sinuses, low coronary height, or externally mounted leaflets. BASILICA remains effective, but complex. UNICORN offers a novel alternative by avoiding snaring and using radiofrequency traversal with balloon dilation, though long term data are still emerging. This case highlights the feasibility of the UNCORN

technique and emphasizes the role of tailored anesthetic management and TEE guidance in high-risk ViV TAVR.

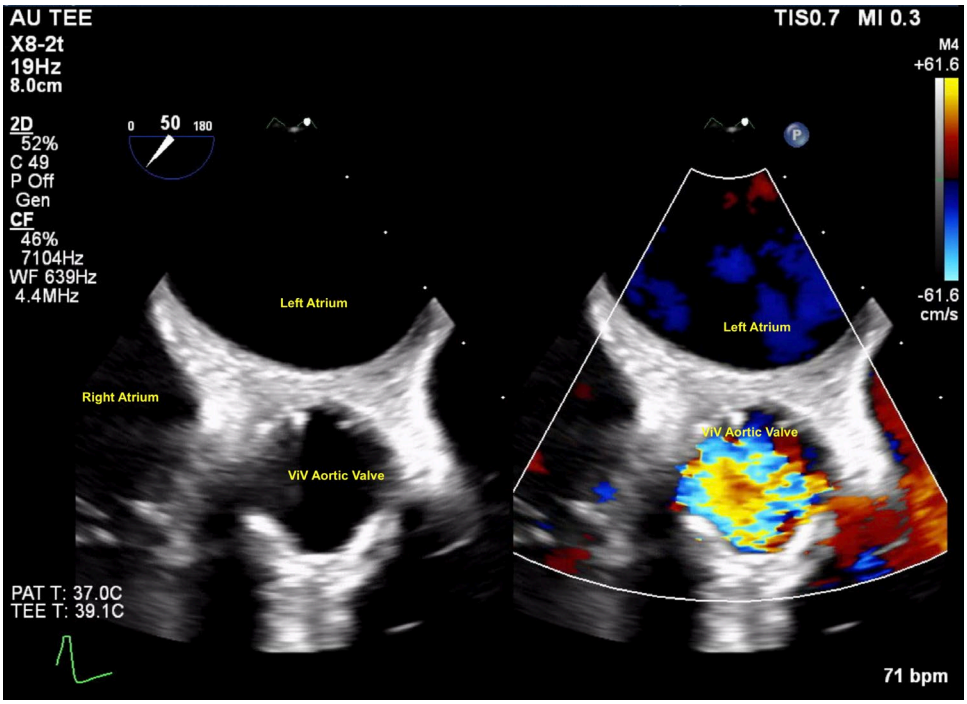
Images



xPlane TEE image showing correct, central guidewire crossing of a surgical bioprosthetic aortic valve during ViV TAVR, confirming safe transvalvular wire passage, with orthogonal confirmation in both long-axis and en-face views.



xPlane TEE image showing aortic insufficiency following the UNICORN leaflet-modification approach during ViV TAVR



xPlane TEE view showing a well-seated, symmetric valve-in-valve aortic prosthesis with normal central systolic forward flow and no paravalvular leak.

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#FR 2 – Kiosk 6 – Poster 5

Late Diagnosis of a Large Traumatic Ventricular Septal Defect Requiring Surgical Repair

Presenting Author: Sabrina Kunselman, MD, Resident Physician, New York Presbyterian Weill Cornell

Introduction

Post-mortem studies estimate the incidence of cardiac injury after blunt chest trauma to be between 10-20%, however the incidence may be higher than 50% in the setting of blunt chest trauma due to fall from a height.[1] Pericardial tearing is the most common cardiac injury seen in blunt chest trauma.[1] Traumatic ventricular septal defect (VSD) is an extremely rare complication of blunt chest trauma with incidence not reported in the literature.

Case Presentation

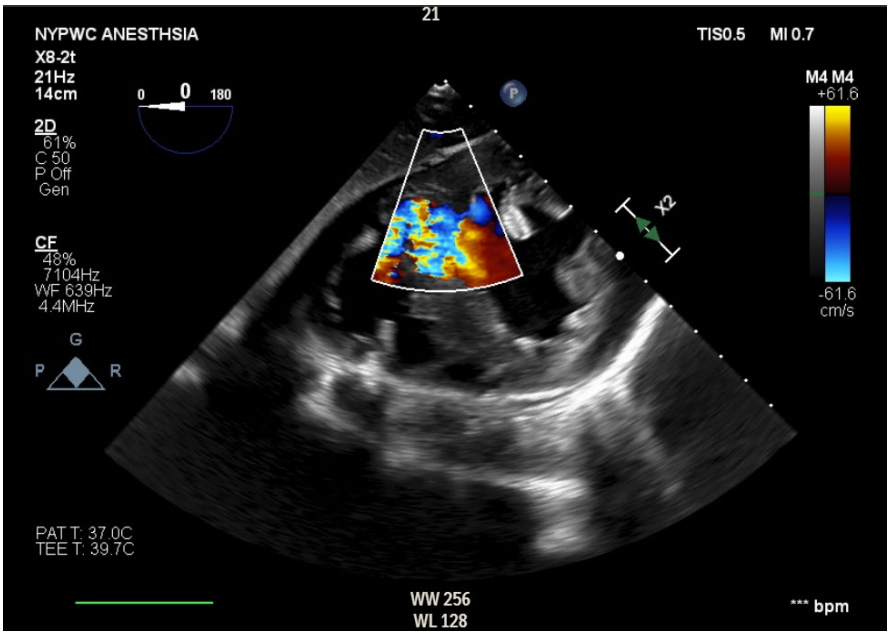
A previously healthy 18 year old male presented to the emergency room as a polytrauma after an accidental fall from approximately 60 feet. The patient sustained traumatic injuries to the pelvis, femur, left upper extremity, right brachial plexus, iliac arteries, and abdomen requiring multiple laparotomies, vascular repairs, and orthopedic surgeries over a course of several weeks. He required prolonged intubation and eventual tracheostomy placement due to acute hypoxic respiratory failure. Twenty six days after admission, transthoracic echocardiography demonstrated a 1.4cm x 1.8cm defect in the mid-ventricular septum with left to right shunting and the patient was transferred to a hospital with higher level of care for surgical repair. Cardiac catheterization demonstrated normal coronaries, mild to moderate pulmonary hypertension, a step-up of oxygen saturation from right atrium (53%) to right ventricle (95%), and a Qp/Qs ratio of 12:1. The patient arrived to the operating room sedated, with an existing right axillary arterial line, tracheostomy in situ, and peripheral IVs. During induction, the patient required phenylephrine boluses to maintain normotension. PaCO2, PaO2, and acid-base status was closely monitored. Intraoperative TEE showed a muscular VSD measuring 1.8 cm x 1.3 cm approximately 4 cm from the mitral valve annulus with left to right flow by color flow doppler (CFD) and otherwise normal biventricular size and function and no significant valvulopathies. Pulmonary artery pressures via Swan Catheter were not obtained due to concern of the catheter migrating across the VSD. Upon opening, the surgical team measured the defect to be 2.5cm by 2cm and repaired it with a Dacron patch. CPB was weaned without inotropic support, and post-CPB TEE showed a 3mm residual defect with left to right flow. The patient was successfully weaned from the ventilator to trach collar on POD2 and decannulated on POD9.

Discussion

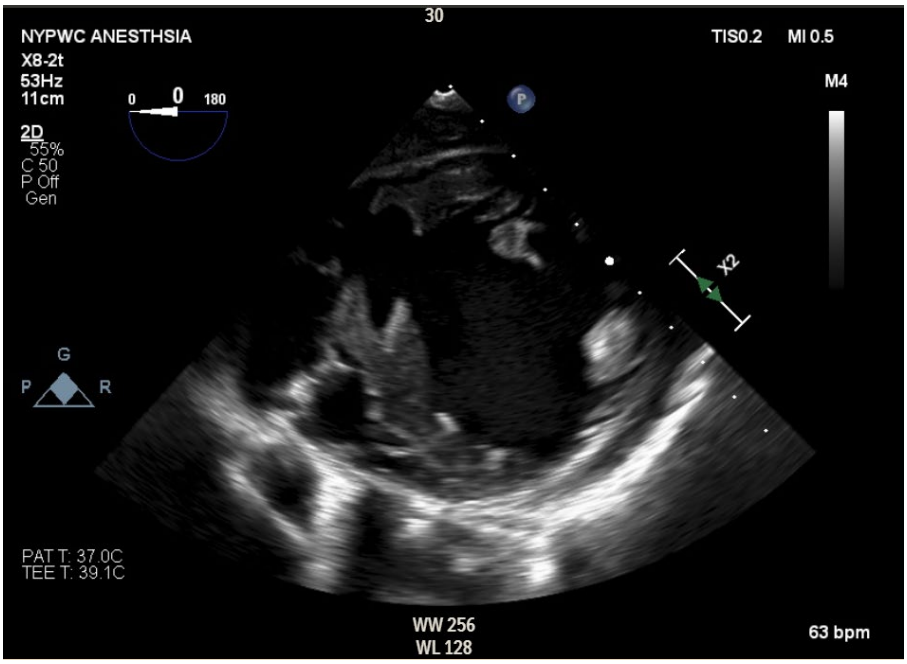
VSDs can be congenital or acquired. Congenital VSDs are more common and are more likely to occur in the perimembranous portion of the interventricular septum, while traumatic VSDs are rare and are more likely to occur in the muscular septum.[2] Traumatic VSDs can be categorized as early (< 48 hours) or late (>48 hours) presentation; early presentations tend to have a larger defect size and require emergency surgical repair.[3] This patient represents a rare case of late presentation of a large traumatic VSD leading to respiratory compromise and requiring urgent surgical repair. Intra-operative anesthetic management was focused on

minimizing left to right shunting by maintaining a balance between pulmonary vascular resistance (PVR) and systemic vascular resistance (SVR). Acute increases in PVR were avoided by avoiding hypoxia, hypercarbia, and acute sympathetic stimulation.

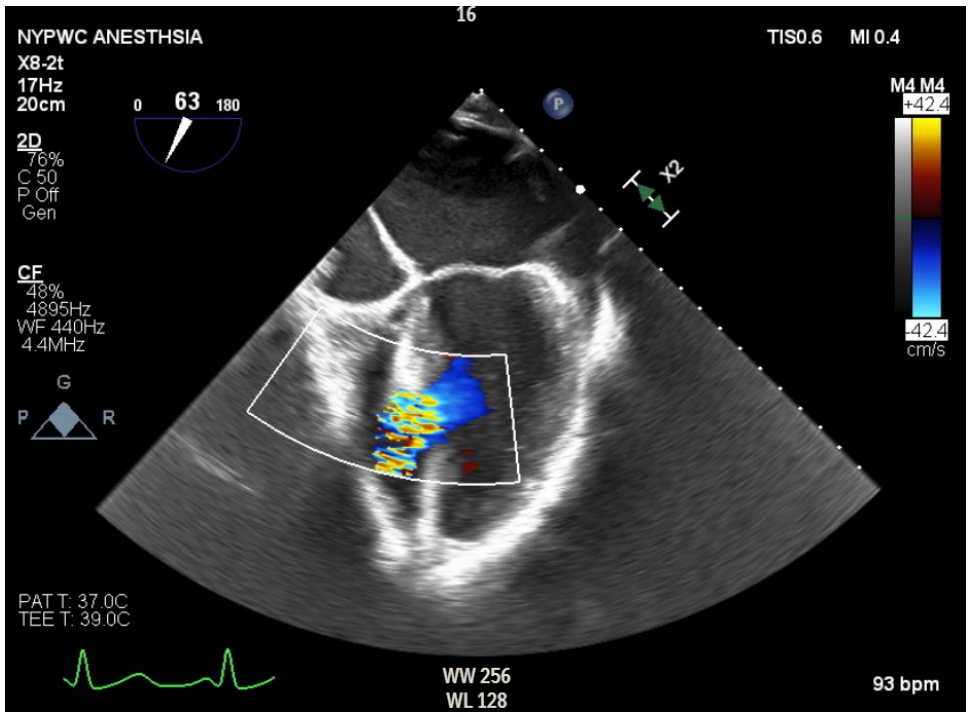
Images



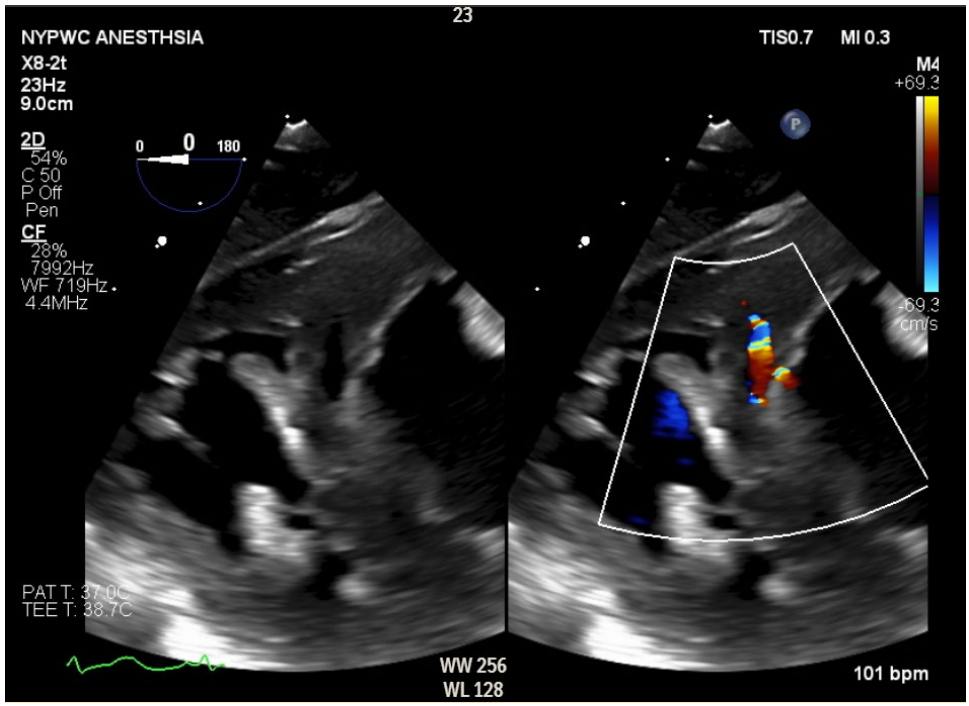
Pre-CPB muscular VSD with left to right flow on CFD, transgastric short axis view



Pre-CPB muscular VSD, transgastric short axis view



Pre-CPB VSD in inferoseptal wall with left to right flow on CFD, approximately midesophageal two chamber view



Post-CPB, residual VSD with left to right flow on CFD, transgastric short axis view

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#FR 2 – Kiosk 6 – Poster 6

Anticoagulation Management of a Hypercoagulable Patient Receiving Mechanical Aortic Valve Replacement

Presenting Author: Alec Seei, DO, Resident, University of Nebraska Medical Center

Introduction

For patients with severe aortic valve disease, replacement with a mechanical prosthesis can last for nearly 20 years, making them the favorable choice over bioprosthetic valves for younger patients. However, mechanical valves carry the risk of thromboembolic events necessitating lifelong anticoagulation, which itself carries the risk of life-threatening bleeding complications. These risks and benefits along with concomitant disease processes must be considered when determining a treatment plan. We present a case of mechanical aortic valve replacement in a hypercoagulable patient and the intraoperative anticoagulation strategy.

Case Presentation

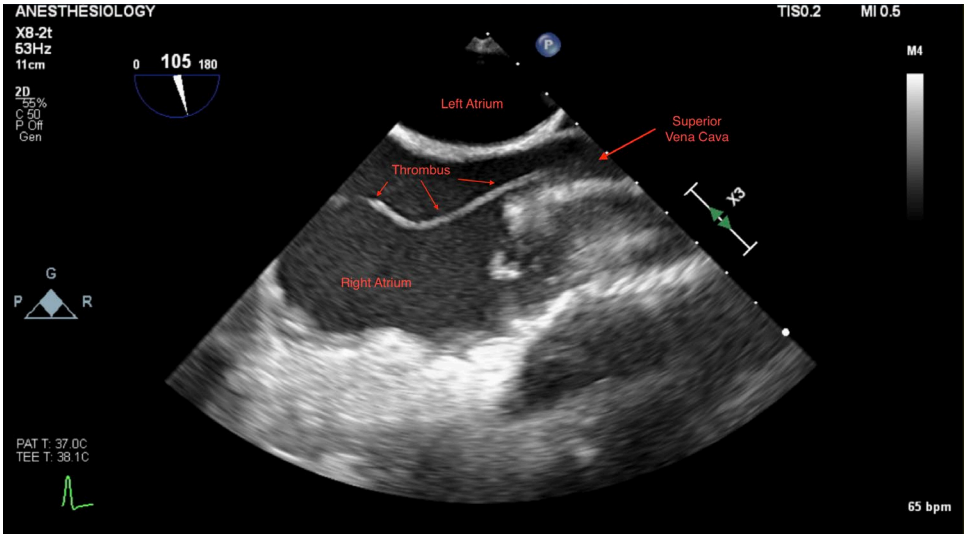
Patient is a 45-year-old with prolonged DRVVT ratio during workup for unprovoked deep vein thrombosis suggesting presence of a lupus anticoagulant. He subsequently presented with left upper extremity numbness. Further hypercoagulable workup demonstrated normal DRVVT values, negative beta-2 glycoprotein antibodies, and negative cardiolipin antibodies. He was found to have multiple mycotic aneurysms of the right middle cerebral artery as well as a large, mobile vegetation on the ventricular aspect of the aortic valve with associated severe aortic regurgitation. After embolization of the cerebral aneurysms, it was deemed safe that he receive systemic anticoagulation and he was taken for aortic valve replacement. Shortly after central line placement, thrombus was noted to have developed on the catheter. Stat coagulation labs were obtained including PT, PTT, INR, platelet count, and fibrinogen level, which were all normal. Thromboelastography demonstrated a shortened R and K time, steep alpha angle, and high maximum amplitude indicating a hypercoagulable state. Multidisciplinary discussion was had with cardiothoracic surgery and hematology, and the decision was made to proceed. The patient was initially anticoagulated with 400 units/kg of heparin with subsequent ACT testing below 400 seconds. An additional 10,000 units was given resulting in an ACT of 455, and cardiopulmonary bypass was initiated. Shortly after, ACT testing was below 400 seconds despite multiple repeat doses of heparin. Antithrombin III was then administered with maintenance of ACT above 400 seconds for the remainder of cardiopulmonary bypass. A fixed dose of 250mg of protamine was given for heparin reversal after termination of cardiopulmonary bypass. Warfarin was started on POD 1 with INR goal of 2-3 and the patient was discharged on POD 6.

Discussion

Heparin resistance is defined as the need for >500 U/kg of heparin to achieve an ACT of at least 400 seconds, with an incidence ranging from 4% to 26%. The most common cause of heparin resistance is antithrombin deficiency, which itself can be due to congenital or acquired etiologies. Acquired antithrombin deficiency is due to either reduced synthesis or increased consumption including liver disease, malnutrition,

nephrotic syndrome, heparin treatment, and sepsis. The first-line treatment for antithrombin deficiency is replacement with antithrombin concentrate. Alternative anticoagulation strategies are available including direct thrombin inhibitors such as bivalirudin or argatroban. It is imperative for us as cardiac anesthesiologists to be well versed in heparin resistance and alternative treatment strategies.

Images



TEE image demonstrating thrombus within the right atrium originating from the central venous catheter

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#FR 2 – Kiosk 6 – Poster 7

Aortic Root Thrombus After LVAD Implantation: TEE Diagnosis and Management

Presenting Author: Zafar Karimov, MD, Resident, Montefiore Einstein

Introduction

Left ventricular assist devices (LVAD) are increasingly utilized for destination therapy in advanced heart failure patients. There is a paucity of information in current literature on the complication of aortic root thrombus (ART) post-LVAD. A retrospective cohort study by Fried et al (2018) found the incidence to be 4.8%. While rare, the phenomena can carry significant morbidity and mortality.

Case Presentation

We present a 70-year-old male one month after LVAD placement as destination therapy whose postoperative course was complicated by recurrent episodes of ventricular tachycardia (VT), resulting in multiple shocks via his implanted cardioverter-defibrillator (ICD) and necessitated anti-arrhythmic pharmacotherapy. Investigation into the etiology of this arrhythmia by transesophageal echocardiography (TEE) revealed an ART. Recurrent VT was suspected to be ischemia-driven from thrombus compression on the left coronary ostium. A relatively high outflow graft anastomosis causing proximal stasis was considered the likely mechanism for clot formation. After a multidisciplinary discussion, the cardiothoracic surgery team elected for surgical intervention. The patient was brought to the operating room for aortic thrombectomy. The thrombus was visualized under TEE guidance, measuring 2.5 x 1.5 cm, located in the aortic root, predominantly within the left coronary cusp, with possible extension into the left coronary artery ostium. After initiation of cardiopulmonary bypass, aortic cross-clamp, and antegrade cardioplegia, an aortotomy was performed, and the thrombus was removed. Moving the LVAD outflow graft anastomosis to a more proximal location was deemed too complex. Instead, the surgeon placed a saphenous vein graft (SVG), originating from the midpoint of the existing outflow graft to the proximal ascending aorta. With this maneuver, blood flow through the LVAD now had two outflow paths to the aorta, existing in parallel circuits. The risk of further thrombus formation was now mitigated due to the elimination of stasis in the proximal aorta. Under TEE guidance, the anesthesiology team was able to visualize flow in the new SVG, thereby ensuring patency, alongside the existing outflow graft anastomosis. Restored flow into the left coronary ostium was also visualized.

Discussion

The LVAD outflow graft connects the mechanical pump to the ascending aorta, and the exact location and angle of the anastomosis can have significant hemodynamic consequences. Postoperative changes within the chest cavity can have further effects on the outflow graft position. In this case, an inadequate height and angle of the outflow graft anastomosis led to impaired blood flow to the proximal ascending aorta and aortic root, which created a favorable environment for clot formation. Progressive ART most likely led to the patient having recurrent ventricular arrhythmias. In this case, TEE was essential to verify and characterize ART and to assess the location and function of the outflow anastomosis. Post-revision, we were able to demonstrate complete thrombus removal and patency of both the existing outflow graft and the new SVG, which was critical to ensure the success of the operation.

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#FR 2 – Kiosk 7 – Poster 1

Stone-cold Killer: A Case of Stone Heart Syndrome

Presenting Author: Daniel D. Pulido, MD, Resident, University of Illinois Chicago at Peoria

Introduction

Stone heart syndrome (ischemic contracture) is a rare, irreversible complication in cardiac surgery patients on cardiopulmonary bypass (CPB) and arrest. This condition was first described in 1972 by Dr. Denton Cooley and, since then, has been poorly studied due to its rapid, often fatal course. The syndrome features myocardial band necrosis, causing a permanently contracted, untreatable heart. Animal studies suggest it begins with sudden myocardial ischemia, leading to contraction and calcium dysregulation—similar to rigor mortis.(1) We describe such a case during surgical aortic valve replacement.

Case Presentation

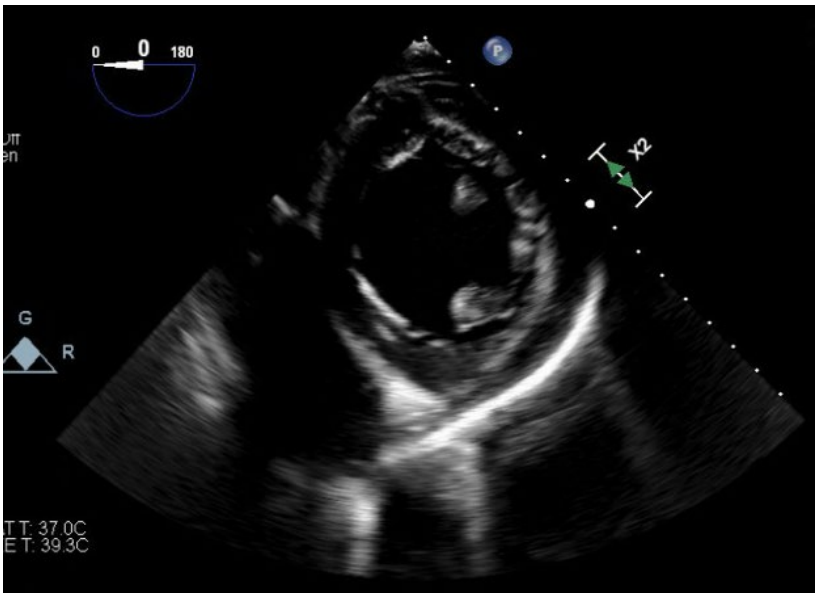
A 49-year-old male (6'7, BMI 28) with a history of a bicuspid aortic valve presented for aortic valve replacement. He had undergone routine surveillance for this disease throughout his life until severe aortic valve (AV) stenosis (mean gradient 52mmHg, peak velocity 4.2 m/sec2, dimensionless index 0.2, 106/46 mmHg) was found. Preoperative evaluation found no evidence of stenotic coronary arteries on coronary angiography and was found to have a left ventricular ejection fraction (LVEF) of 55-60% with mild-moderate concentric remodeling. Patient underwent general anesthesia with standard monitors in place. A partial sternotomy incision was performed with successful CPB cannulation. Approximately two liters of anterograde and retrograde cardioplegia were given, achieving isoelectric arrest. A mechanical valve was placed, and weaning from CPB was initiated. TEE showed new severe global hypokinesis with akinesis of the basal and midpapillary septal wall. CPB was reinitiated, with the suspicion that coronary air entrapment may be the culprit. A second wean from CPB was attempted, with no response to multiple inotropes or increased afterload, no ECG changes, an LVEF < 15% and a mechanical valve opening infrequently. With complete absence of relaxation, a fixed, small LV cavity, and rigid ventricle appearance, we now suspected stone heart syndrome. An intra-aortic balloon pump was placed once this event was recognized, but the patient then developed frothy secretions from the ETT and poor lung compliance. VA-ECMO and nitric oxide were initiated, and the patient was then transferred to the ICU with an open chest in hopes of an eventual improvement in cardiac function. Two days later, a bioprosthetic valve replaced the initial valve due to a lack of opening and a risk of thrombosis. Despite repeated cardioplegia, there was no myocardial relaxation. Serial echocardiography showed no improvement. The patient developed ARDS and renal failure, requiring CRRT. Given the decline and ineligibility for transplant, care was withdrawn seven days post-surgery.

Discussion

This case is a startling reminder of the risks associated with cardiac surgery despite modern advances. Initially, this patient seemed like an ideal surgical candidate; young, robust, and with no other comorbidities besides chronically progressing aortic stenosis. However, the degree of hypertrophy posed a significant risk for this syndrome despite appropriate preventative measures (anterograde and retrograde cardioplegia with

short arrest time). Despite the rarity of this event (< 0.2% of patients undergoing CPB), this condition must remain in the differential diagnosis for unsuccessful separation from CPB.

Images





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#FR 2 – Kiosk 7 – Poster 2

Modified BTT Shunt Thrombosis in an Adult Patient with Congenital Heart Disease

Presenting Author: Kelci R. Beardsley, DO, Resident Physician, University of Kentucky

Introduction

Initial repairs of cyanotic heart disease extensively made use of the Blalock-Taussig-Thomas (BTT) shunt. This shunt involved direct anastomosis of the subclavian artery to the pulmonary artery. This method later evolved to the modified BTT (mBTT) shunt, an implanted conduit between the subclavian and pulmonary arteries to maintain pulmonary blood flow. The mBTT is an important component of palliation of many types of congenital heart disease. However, the mBTT usually is temporary as pulmonary blood flow is usually later established by a Glenn, Fontan, or right ventricle to pulmonary artery (RV-PA) conduit. We present a unique case in which an adult patient had a thrombosis of their mBTT shunt.

Case Presentation

Our patient was a 54-year-old female with tricuspid atresia, atrial septal defect, patent ductus arteriosus, hypoplastic right heart s/p Glenn procedure, s/p mBTT shunt from left subclavian artery to left pulmonary artery, and occluded SVC. The patient presented to our facility with acute hypoxic respiratory failure. Initial workup at an outside hospital was significant for flu-like symptoms, hypotension, hypoxia with a peripheral oxygen saturation of 60% on room air, and elevated cardiac troponins. Chest x-ray was negative for acute pathology, but subsequent computer tomography angiography of the chest revealed an occlusion of her mBTT shunt. On exam, the patient was in respiratory distress requiring a non-rebreather mask. No murmur was appreciated on auscultation. The patient was placed on a heparin drip and transferred to our facility for further management. Upon arrival, the patient was taken emergently for right heart catheterization which confirmed complete mBTT shunt occlusion. Thrombolysis was initiated with local tPA via insertion of an EKOS ultrasound-assisted catheter-directed thrombolysis (USCDT) system. Initial interventions yielded an improvement in peripheral oxygen saturation and a decreased oxygen requirement. Targeted thrombolytic therapy was continued for 24 hours prior to repeat angiography and placement of stent within the mBTT shunt. Interventions were successful; the patient was weaned from supplemental oxygen, and she was discharged from the hospital ten days post-procedure. At time of discharge, a pansystolic and diastolic murmur was appreciated on cardiac auscultation further confirming the patency of her mBTT shunt.

Discussion

Palliation of congenital cyanotic heart disease via mBTT shunt is often completed in infants as a temporary procedure to provide pulmonary blood flow prior to more definitive corrective surgery. However, in rare cases, patients may keep their mBTT shunts into adulthood. Complications of mBTT shunts are estimated to occur in 5.3-13.8% of patients and are most common in the early postoperative period. However, catastrophic stent occlusion can occur in adult patients far removed from their initial surgery. Uniquely, in patients with mBTT shunts, the presence of a murmur is reassuring as it signals shunt patency. On presentation our patient lacked a murmur on exam; upon discharge, her shunt murmur had returned. This

case highlights an emergent intervention via USCDT and mBTT shunt stenting which allowed for restoration of pulmonary blood flow and improvement in peripheral oxygen saturation.

Images

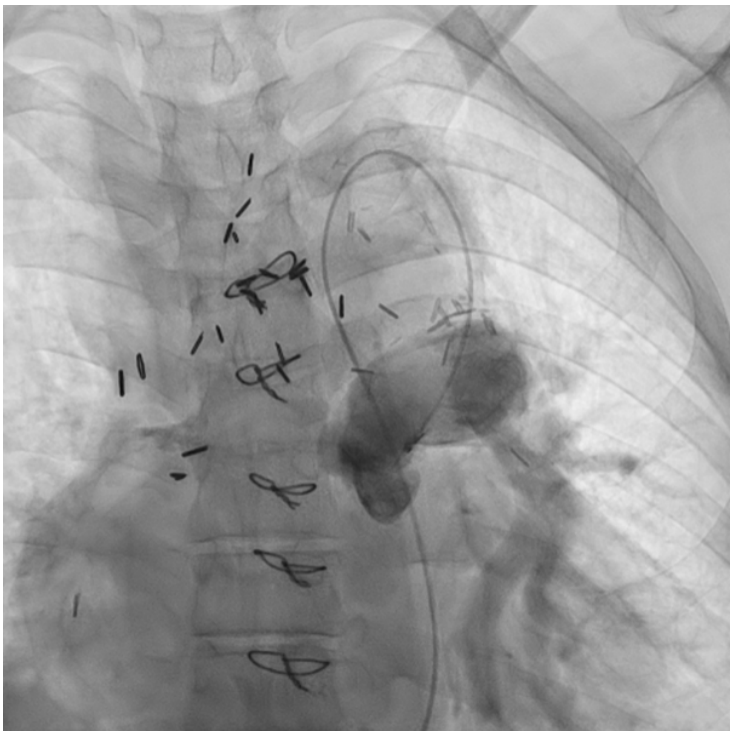


Image 1: Cardiac catheterization demonstrating occlusion of left mBTT shunt.



Image 2: Repeat cardiac catheterization status-post thrombolysis and stenting.

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#FR 2 – Kiosk 7 – Poster 4

Portable Dynamic Digital Radiography (pDDR) for Evaluation of Diaphragmatic Dysfunction and Recovery Following Sequential Single Lung Transplantation

Presenting Author: Grace S. Lee, MD, Brigham and Women's Hospital

Elana Folbe, MD, Brigham and Women's Hospital

Introduction

Diaphragmatic dysfunction (DD) is a recognized complication after lung transplantation that can adversely affect respiratory mechanics and recovery. Portable Dynamic Digital Radiography (pDDR) provides high-resolution dynamic imaging for quantitative bedside assessment of diaphragmatic excursion and function in real time.

Case Presentation

We present a notable case of a 67-year-old male with a history of chronic kidney disease, type 2 diabetes mellitus, gastroesophageal reflux disease, and interstitial lung disease who initially underwent a left single lung transplant (SOLT). His postoperative course was complicated by bronchial necrosis and stenosis, requiring multiple dilations and stenting. Due to persistent airway complications and declining performance status, he subsequently underwent a right SOLT. Eleven days after the right lung transplant, pDDR imaging was performed using the mKDR Xpress Mobile X-ray system paired with the AeroDR HD Carbon Flat Panel Detector (Konica Minolta Inc., Wayne, NJ). Images were acquired in a semi-recumbent position during both tidal breathing and 7-second breath-hold phases. Edge detection technology utilizing a Prewitt Filter was employed to delineate the diaphragms, with accuracy verified by a radiologist. Analytical software measured the vertical displacement of each hemi-diaphragm and plotted the excursion data for comparison between early post-transplant and 3-month follow-up studies. Initial pDDR imaging revealed relative left-sided diaphragmatic dysfunction (12.4 mm), while the right diaphragm demonstrated high-normal excursion (19.6 mm). Flexible bronchoscopy performed during this period revealed granulation tissue obstructing the left main bronchus, which was treated with cryotherapy and balloon dilation to 9 mm. The right airway remained widely patent, with a well-healed anastomosis, normal mucosa, and intact airway anatomy. Following successful bronchoscopic intervention and restoration of left mainstem airway patency, follow-up pDDR imaging at 3 months revealed marked improvement in diaphragmatic function on both sides, with left diaphragm excursion to 25.2 mm and right diaphragm excursion increasing to 32.4 mm.

Discussion

Severe DD soon after transplantation can resemble primary graft dysfunction or infection, leading to unnecessary interventions if unrecognized. In this case, pDDR provided a simple, reproducible way to quantify diaphragmatic motion and track recovery. Compared with ultrasound, it offers full-diaphragm visualization with less operator dependence, and unlike fluoroscopy, it can be performed at the bedside. This case demonstrates pDDR's value as a noninvasive, portable tool for monitoring diaphragmatic function in lung transplant recipients. The marked improvement in bilateral diaphragmatic excursion over three

months suggests reversible phrenic nerve injury (neurapraxia) rather than permanent damage, underscoring the potential for recovery with time and supportive care. Combined with bronchoscopic assessment, pDDR enables early DD detection, guide informed clinical decisions and optimize documentation of functional recovery. The study was supported by the research grant from Konica-Minolta, Inc.

Images

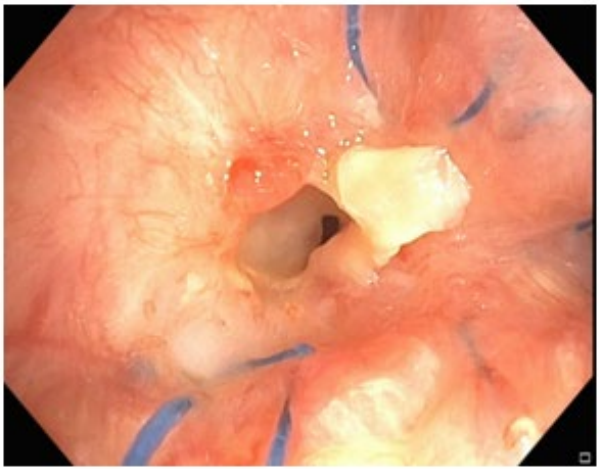
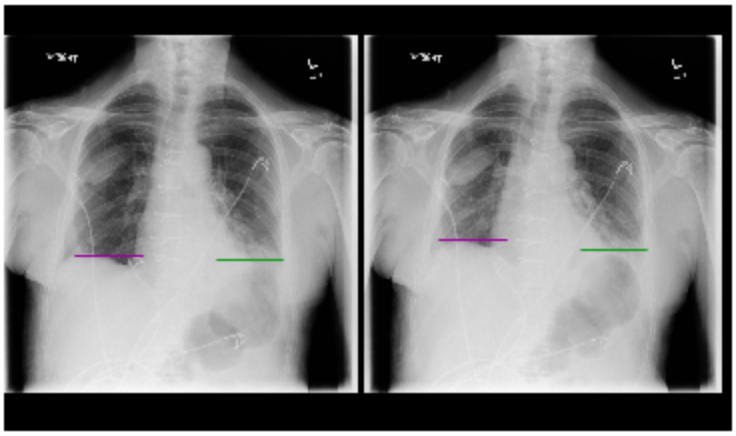


Figure 1. Granulation tissue in the left main bronchus removed by cryotherapy. Balloon dilation performed up to 9 mm.



Figure 2. Well-healed right main bronchus anastomosis. RUL, RML, and RLL bronchi show normal mucosa without lesions or secretions.

A B



C

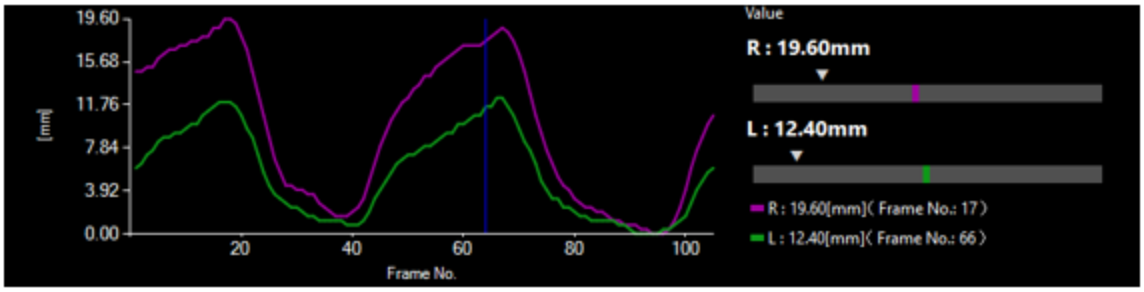


Figure 3. End-inspiratory (Fig 3A) and end-expiratory (Fig. 3B) pDDR chest image (with diaphragms marked) taken on postoperative day 11. Graphic representation and quantitative measurement of diaphragmatic excursion (Fig. 3C).

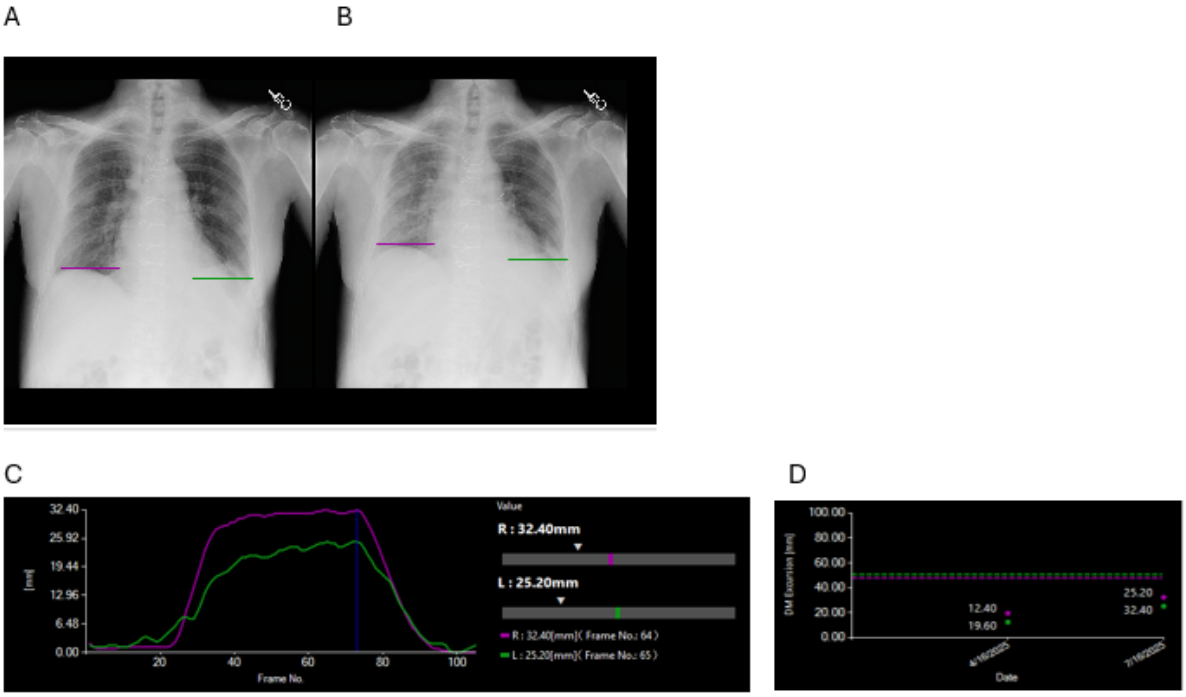


Figure 4. End-inspiratory (Fig 4A) and end-expiratory (Fig. 4B) pDDR chest image (with diaphragms marked) taken on postoperative day x. Graphic representation and quantitative measurement of diaphragmatic excursion (Fig. 4C) with tabulation of the findings (Fig. 4D).

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#FR 2 – Kiosk 7 – Poster 5

Mid-Cavity Membranous Septal Obstruction of the Right Ventricle in an Elderly Patient with Biventricular Failure

Presenting Author: Kenneth Schmitt, MD, University of Southern California/LA General

Introduction

Double-chambered right ventricle (DCRV) is an uncommon congenital anomaly in which anomalous muscular or membranous tissue divides the right ventricle (RV) into a high-pressure proximal chamber and a low-pressure distal chamber. [1] Although typically diagnosed in childhood, late presentation can result in progressive RV dilation, severe dysfunction, and biventricular failure. Intraoperative transesophageal echocardiography (TEE) is critical for identifying dynamic or atypical obstructive RV lesions. We report an elderly patient in whom TEE revealed a mid-cavity membranous septum with a 0.6-cm orifice causing severe RV obstruction and decompensated right-sided heart failure, prompting surgical correction. 2

Case Presentation

A sixty-three-year-old woman with atrial fibrillation status post Watchman, heart failure with reduced ejection fraction, and an ICD presented with worsening dyspnea, edema, and ascites. Transthoracic echocardiography demonstrated LVEF 35%, severe RV dilation, and estimated pulmonary artery systolic pressures in the 50s mmHg. Doppler interrogation identified a peak gradient of 37 mmHg across a membrane spanning the moderator band and septum in the RV mid-cavity. Right-heart catheterization confirmed elevated RV systolic pressure with subpulmonic obstruction. CT angiography showed increased trabeculations in the mid-to-apical RV. During cardiac surgery, intraoperative TEE revealed a dilated LV (EF about 30%) with global hypokinesis and a severely dilated, poorly contractile RV. A membranous mid-ventricular septum with a 0.6-cm orifice and turbulent flow was clearly visualized (Figure 1). The patient underwent median sternotomy, cardiopulmonary bypass, bi-atrial Maze, and resection of the obstructing RV membrane. Cardiopulmonary bypass time was 56 minutes; cross-clamp time was 16 minutes. Post-resection TEE confirmed unobstructed RV flow, persistent RV dilation/dysfunction, mild tricuspid regurgitation, and moderate mitral regurgitation. Postoperative management focused on RV-protective hemodynamics management including inotropic support, preload optimization, and pulmonary vascular resistance control.

Discussion

Congenital membranous RV mid-cavity obstruction is a rare etiology of adult RV failure and can mimic or coexist with other cardiomyopathies. Recognition is essential, as surgical resection can relieve obstruction even in the setting of biventricular dysfunction. Intraoperative TEE remains pivotal for defining anatomic mechanisms of RV failure, guiding surgical intervention, and confirming adequacy of repair. Anesthetic management should emphasize maintaining RV perfusion, optimizing preload, and minimizing pulmonary vascular resistance.[1], [2]

Images

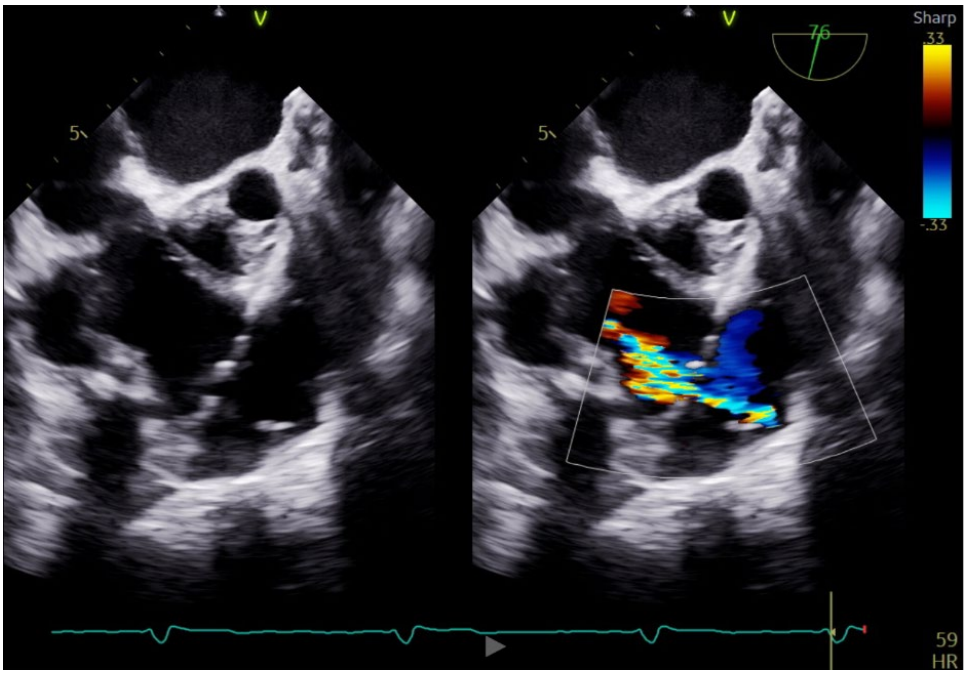


Figure 1. Preoperative TEE mid-esophageal RV inflow outflow view identifying a membranous septum with 0.6-cm orifice, color Doppler demonstrating high-velocity, turbulent flow.

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#FR 2 – Kiosk 7 – Poster 6

Anesthesiologist-Led Management of Severe HOCM and Extreme LVOT Obstruction

Presenting Author: Shriya Sharma, Boston Medical Center Brighton

Introduction

Hypertrophic cardiomyopathy (HCM) is the most common genetic cardiovascular disorder (prevalence 1:500), characterized by myocardial disarray leading to hypertrophy and uncoordinated contraction. HCM can present with or without dynamic left ventricular outflow tract (LVOT) obstruction; patients with obstructive HCM (HOCM) are more likely to be symptomatic and prone to perioperative complications. Severe HOCM with extreme LVOT obstruction (resting gradient >150 mmHg) poses unique anesthetic challenges, with heightened risk of hypotension, arrhythmias, and pulmonary edema during general anesthesia or hemodynamic stress. Perioperative triggers including induction agents and sympathetic surges can worsen obstruction, risking circulatory collapse or myocardial ischemia. The perioperative anesthesiologist functions as a perioperative physician, integrating cardiac, pulmonary, and functional status to tailor induction, maintenance, and postoperative monitoring.

Case Presentation

An 85-year-old female with HOCM with severe LVOT obstruction (SAM, gradient >150 mmHg), HFpEF (EF 80%), pulmonary hypertension, pacemaker-dependent complete heart block post-TAVR, COPD/asthma on home O_2, OSA, CAD, hypertension, insulin-dependent diabetes, and dementia sustained a right femoral neck fracture from a wheelchair fall. She was wheelchair-bound but able to pivot for transfers, ASA IV, with RCRI 5 (predicted MACE 10%), NSQIP cardiac risk 5.5%, and functional capacity ~3 METs. Labs showed Hb 10.3 g/dL. ECG revealed atrial-sensed ventricular-paced complexes with nonspecific conduction delay. Preoperative TTE confirmed hyperdynamic LV function (EF 80%), severe LVOT obstruction due to SAM, normal wall motion, RVSP 54 mmHg. The anesthesiology team prioritized hemodynamic optimization: maintaining preload, avoiding tachycardia, and preventing abrupt changes in afterload or contractility. Right radial arterial line and RIJ central venous catheter were placed. Induction used carefully titrated propofol (60 mg), fentanyl (150 mcg), and rocuronium (100 mg). Phenylephrine drip and esmolol were administered to maintain LVOT stability, and albumin 25 g supported preload. The patient underwent closed reduction and percutaneous screw fixation of the femoral neck with stable intraoperative hemodynamics. Post-extubation, she developed upper-airway obstruction with desaturation to 70–80%, requiring nasal trumpet insertion and high-flow oxygen. She was transferred to the SICU for monitoring, gradually weaned to nasal cannula, and discharged without cardiovascular complications.

Discussion

Severe LVOT obstruction in HOCM is highly dynamic, worsening with decreased preload, decreased afterload, or increased contractility, placing patients at high risk for hypotension, arrhythmias, and collapse. Anesthetic management focuses on maintaining sinus rhythm, preserving preload and afterload, and minimizing sympathetic stimulation. In this high-risk patient with resting LVOT gradient >150 mmHg,

multiple comorbidities, and pacemaker dependency, careful induction, titrated anesthetics, invasive monitoring, and avoidance of tachycardia or vasodilation allowed safe completion of surgery. Post-extubation airway compromise was promptly managed, preventing cardiovascular decompensation.

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#FR 2 – Kiosk 7 – Poster 7

VV ECMO for Severe Primary Graft Dysfunction After Lung Transplant in Anti-Synthetase Syndrome

Presenting Author: Marjo Beltoja, MD, Resident Physician, Anesthesiology, Virginia Commonwealth University Health System

Introduction

Anti-synthetase syndrome (ATS) is a rare cause of interstitial lung disease (ILD) characterized by specific antibodies.[1] Progressive ILD may lead to end-stage respiratory failure requiring lung transplantation, a procedure with significant morbidity and high risk of primary graft dysfunction (PGD).[2] This case describes ATS-related ILD treated with bilateral lung transplantation and highlights the role of transesophageal echocardiography (TEE), physiology-directed management, and veno-venous extracorporeal membrane oxygenation (VV ECMO) in severe PGD.

Case Presentation

A 68-year-old woman with anti-PL-12–positive ATS and end-stage ILD on home oxygen underwent bilateral lung transplantation for respiratory failure. Induction included thorough preoxygenation and rapid-sequence intubation. In addition to standard monitors, near-infrared spectroscopy (NIRS), arterial pressure, central venous pressure, and pulmonary arterial pressure were used. Cardiopulmonary bypass (CPB) was initiated due to minimal pulmonary reserve. Upon weaning from CPB, the patient developed severe hypoxemia and low P/F ratios consistent with early PGD. A right internal jugular dual-lumen (Avalon) cannula was placed for VV ECMO with prompt improvement. She required several early takebacks to the OR. TEE consistently demonstrated preserved biventricular function without pericardial effusion, excluding cardiogenic hypoxemia. The cannula tip was slightly deep (1–2 cm) with an off-axis return jet, supporting intrathoracic hypertension from graft edema impairing ventilation and ECMO drainage (“lung tamponade physiology”). The chest was left open and the patient transported to the ICU on VV ECMO. Chest closure occurred on postoperative day six. The patient then showed progressive improvement in hemodynamics and ventilatory mechanics. Rising donor-specific antibodies (DSA) were treated with anti-thymocyte globulin. Airway stent placement for anastomotic narrowing improved tidal volumes and plateau pressures. Inhaled nitric oxide was weaned as graft compliance improved. VV ECMO was discontinued two weeks post-transplant, consistent with recovery from PGD grade III.

Discussion

ATS-related ILD presents significant anesthetic challenges due to minimal oxygen reserve and susceptibility to PGD. This case underscores the importance of TEE in distinguishing PGD-related lung tamponade physiology from cardiogenic causes of hypoxemia and guiding the decision to initiate VV rather than VA ECMO. Early recognition of intrathoracic hypertension supported delayed chest closure, cannula optimization, and reduced ventilator-induced lung injury through early ECMO support. Airway stenting further improved mechanics by relieving anastomotic obstruction. Early TEE-guided identification of PGD and timely initiation of VV ECMO are critical for stabilizing graft function in high-risk autoimmune ILD. CPB

use is associated with increased PGD rates, and many centers now favor intraoperative ECMO to reduce ischemia–reperfusion injury.[2]

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#FR 2 – Kiosk 8 – Poster 2

Non-Operating Room Anesthetic Management of a Patient with Levo-Transposition of the Great Arteries

Presenting Author: Rishi R. Wagle, M.D., Anesthesiology Resident Physician, University of Maryland School of Medicine

Introduction

Transposition of the great arteries (TGA) occurs in ~0.02% of live births. While it commonly presents as dextro (D)-TGA, with ventriculo-arterial discordance, it can also present as levo (L)-TGA, also known as congenitally corrected (cc)-TGA, which is not inherently a cyanotic lesion. L-TGA patients have a normal sequence of blood flow but often have concomitant cardiac anomalies that require intervention. Advances in treatment allow most patients (~80%) to reach adulthood, yet the long-term burden of RV dysfunction and arrhythmia remains substantial. Anesthetic management in adults with L-TGA is challenged by limited ventricular compliance and function, valvular pathology, conduction disease, and sensitivity to preload and afterload shifts.

Case Presentation

Our patient was a 34-year-old woman with PMH of L-TGA status post VSD closure in infancy, recent onset A-fib with RVR on antiarrhythmics and heparin, cervical cancer on chemotherapy requiring blood transfusions, and bilateral hydronephrosis who presented to the IR suite for bilateral percutaneous nephrostomy tube exchange. TTE revealed a “moderately hypertrophied systemic ventricle” with “estimated LVEF 30-35%”, “normal right-sided ventricle”, no evidence of residual VSD, and “mild-moderate systemic ventricular AV valvular regurgitation”. After discussion with the IR team, our plan was for GETA due to the need for prone positioning, as well as foregoing invasive blood pressure monitoring and intraoperative TEE unless indicated. Before induction, defibrillator pads were placed on the patient who was noted to be in NSR. GA was induced with the addition of a small bolus of Epinephrine. The patient was intubated, and sedation was maintained with Sevoflurane and a Dexmedetomidine infusion. Hemodynamics were managed intraoperatively with Epinephrine and Norepinephrine infusions. At the end of the case, the patient was awakened, extubated, and transported back to the ICU without any hemodynamic support and free of complications from heart failure or arrhythmia.

Discussion

Adult patients with L-TGA present unique perioperative challenges, especially in non-operating room anesthesia locations where resources may be limited. The rare occurrence of L-TGA with a diversity of patient-specific physiology and clinical presentations have made research on the condition infrequent. Therefore, a thorough understanding of each patient’s anatomy, prior repairs, and residual lesions is essential for anesthetic planning. Echo results must be carefully read as the verbiage may not be representative of the patient’s unique anatomy, and ideally images should be reviewed. In this case, epinephrine and norepinephrine infusions were selected due to alpha and beta-adrenergic effects, which

support systemic vascular resistance, inotropy, chronotropy, and lusitropy, thus maintaining cardiac output, which is critical in patients with compromised ventricular function. Given the high risk of arrhythmia and adverse cardiac events, immediate availability of an external defibrillator is mandatory. Consideration should be given for appropriate lines and monitors while taking the intended procedure into account. These measures, along with tailored pharmacologic strategies, are essential to optimize outcomes in this population.

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#FR 2 – Kiosk 8 – Poster 3

Point of Care Transthoracic Echocardiography in Acute Type an Aortic Dissection: Guiding Interdisciplinary Discussion and Perioperative Decision-Making

Presenting Author: Olivia D. Hess, MD, Resident, Stanford University School of Medicine

Introduction

In acute type A aortic dissection, point-of-care transthoracic echocardiography (POC-TTE) offers a rapid, non-invasive assessment that can influence initial anesthetic and surgical decision making. Beyond assessing for effusion or tamponade prior to induction, POC-TTE provides early information on aortic valve pathology, ventricular systolic function, as well as any other valvular dysfunction which may affect surgical approach (1). We report a case of a patient presenting for emergent dissection repair where unexpected findings on pre-induction POC-TTE changed perioperative management and surgical decision making.

Case Presentation

An 81-year-old female with a past medical history of hypertension, atrial fibrillation on apixaban, congestive heart failure and chronic kidney disease presented to an outside hospital with left hemiplegia and right facial droop. Imaging revealed a type A aortic dissection involving the innominate, left common carotid and left subclavian arteries, a completely occluded right carotid artery, and extension to the distal descending aorta. She was transferred emergently to our center for operative intervention. POC-TTE prior to induction revealed severely reduced left ventricular ejection fraction, moderate aortic regurgitation, and moderate-severe mitral and tricuspid regurgitation (Figure 1). Given the significance of these findings, the surgical team was immediately notified and the plan modified to initial right carotid stent placement then reevaluation of hemodynamic status prior to aortic repair. The patient was hemodynamically stable during induction and aortogram, which showed lack of right carotid distal reperfusion and no stent was placed. She subsequently underwent supracoronary ascending aorta and hemiarch replacement with circulatory arrest and retrograde cerebral perfusion. Completion angiogram after separation from cardiopulmonary bypass showed patent arch vessels including the right carotid artery. She was discharged to a rehabilitation facility on post-op day 24 with residual but improving aphasia and full motor strength.

Discussion

This case highlights use of POC-TTE to identify significant, unexpected pathology prior to induction of anesthesia, prompting interdisciplinary discussion and a change in surgical plan, as well as increased risk and consent discussion with the patient and her family. In the emergent setting, a focused pre-induction TTE can provide important diagnostic insights to optimize patient safety and can be the source of additional data points in settings with limited preoperative workup which can lead to improved perioperative planning and decision making.

Images

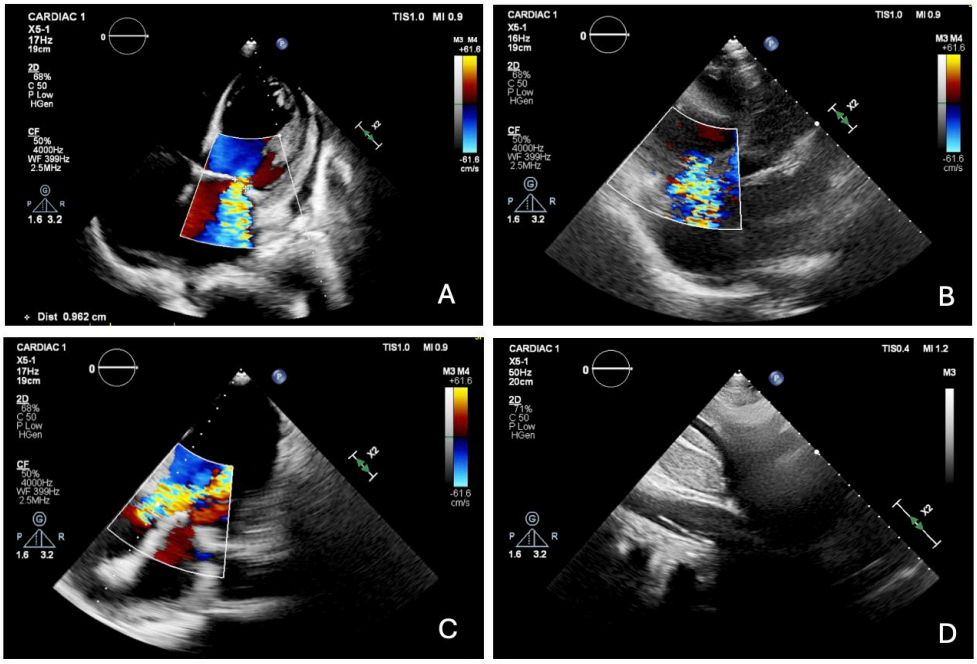


Figure 1: Pre-induction POC-TTE in patient with acute type A Aortic Dissection
A-Mitral Regurgitation. B-Tricuspid Regurgitation.
C-Aortic Regurgitation. D-Descending Thoracic Aorta

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#FR 2 – Kiosk 8 – Poster 4

Engineering Outflow: Managing ViV TAVR Failure in a Chronically Reconstructed LVOT

Presenting Author: Ahmed Yousef, MD, Fellow, Brigham and Women's Hospital

Introduction

Progressive stenosis of valve-in-valve (ViV) transcatheter aortic valve replacements (TAVR) poses management challenges in patients with multiple prior aortic operations. Likewise, revision sternotomy after repeated root and ascending aortic reconstructions can carry prohibitive morbidity. Alternative outflow strategies have been described for patients unsuitable for conventional reoperative aortic valve intervention.[1] Here, we report the use of a left ventricular (LV)–to–descending aorta valved conduit to treat severe ViV aortic stenosis in a patient with seven prior aortic valve and ascending aorta interventions.

Case Presentation

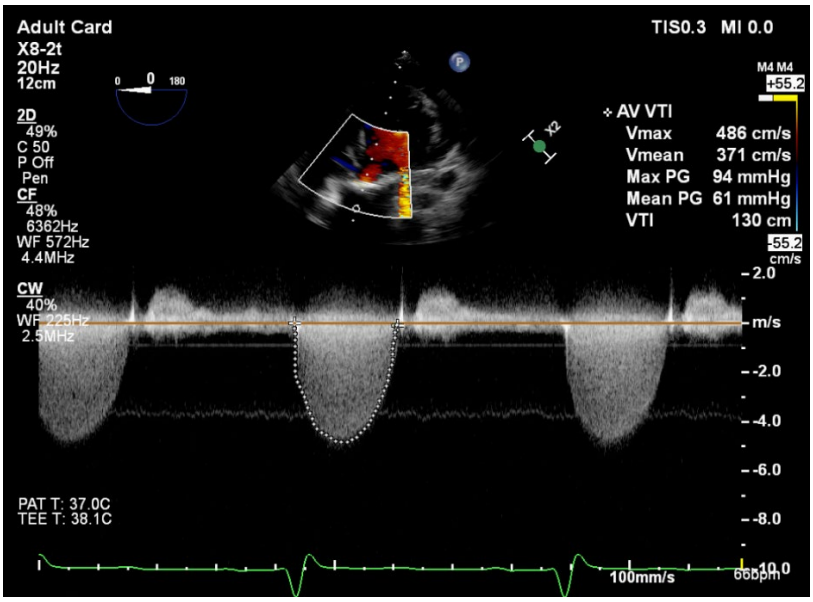
A 55-year-old male with a history of multiple aortic valve replacements presented with progressive severe stenosis of his ViV prosthesis. He had an extensive interventional history including thoracotomy for aortic coarctation repair (1980), two sternotomies for surgical aortic valve replacement (2005, 2009), endovascular ascending aorta pseudoaneurysm repair (2006), two sternotomies for ascending aorta pseudoaneurysm repair and hemiarch replacement (2007, 2010), and ViV TAVR (2016). Given extensive reoperative history and high sensitization precluding transplant, alternative outflow reconstruction was pursued. Pre-bypass TEE showed preserved left ventricular systolic function with restrictive diastology, mildly reduced right ventricular function, and severe ViV aortic stenosis (peak gradient 94 mmHg, mean gradient 61 mmHg) with mild aortic insufficiency. Via left thoracotomy, the LV apex and descending thoracic aorta were exposed. Cardiopulmonary bypass (CPB) was initiated after challenging femoral cannulation through dense adhesions. A 24 mm Dacron graft was sewn to the descending aorta under partial clamping. A hand-constructed 23 mm bioprosthetic valved conduit was anastomosed to a sewing cuff secured to the cored LV apex. Graft-to-graft anastomosis completed the conduit, and CPB was weaned following hemostasis. Post-bypass TEE showed preserved biventricular function on low-dose epinephrine, laminar conduit flow, and expected reduction in LVOT stroke volume. Since perfusion of aortic arch vessels would rely on partial retrograde flow post-CBP, bilateral arterial lines and cerebral oximetry were used to confirm adequate flow. Post-CPB, radial pressures were concordant and cerebral oximetry readings remained stable. The post-operative course was notable for slow wean of inotropes and volume overload requiring diuresis. He was discharged on post-operative day 10.

Discussion

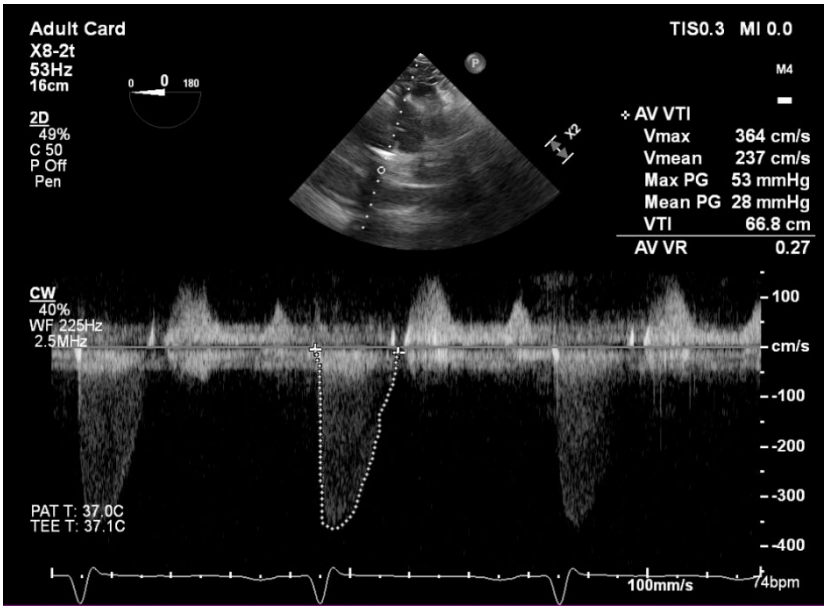
These findings underscore the value of alternative outflow procedures in patients facing prohibitive risk from conventional reoperative aortic surgery. As bioprosthetic valve longevity improves and ViV TAVR procedures increase, a subset of patients will present with complex structural deterioration in the setting of prior aortic reconstruction. For those who are neither transplant candidates nor suitable for redo sternotomy, LV apical–to–descending aorta bypass via left thoracotomy offers a durable pathway to restore forward flow and

reduce ventricular workload.[2] Careful preoperative imaging, hemodynamic assessment, and intraoperative TEE are essential to confirm suitability, optimize conduit geometry, and guide postoperative management in these selected patients.[3]

Images



Continuous wave spectral doppler through bioprosthetic aortic valve shows Vmax of 4.9 m/s, mean gradient 61 mmHg, and peak gradient 94 mmHg.



Continuous wave spectral doppler across the LVOT post-LV-aorta conduit shows a decrease in the velocity-time integral by about half.

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#FR 2 – Kiosk 8 – Poster 5

Double Trouble: A Rare Case of LA and LV Myxomas in Carney Complex

Presenting Author: Alejandro Vega, MD, Anesthesia Resident, SUNY Downstate Medical Center

Introduction

An 18-year-old male who presented with syncope was found to have a left atrial (LA) and a left ventricular (LV) myxoma. We aim to highlight the anesthetic management and diagnostic considerations of cardiac myxomas, including the Carney Complex (CC).

Case Presentation

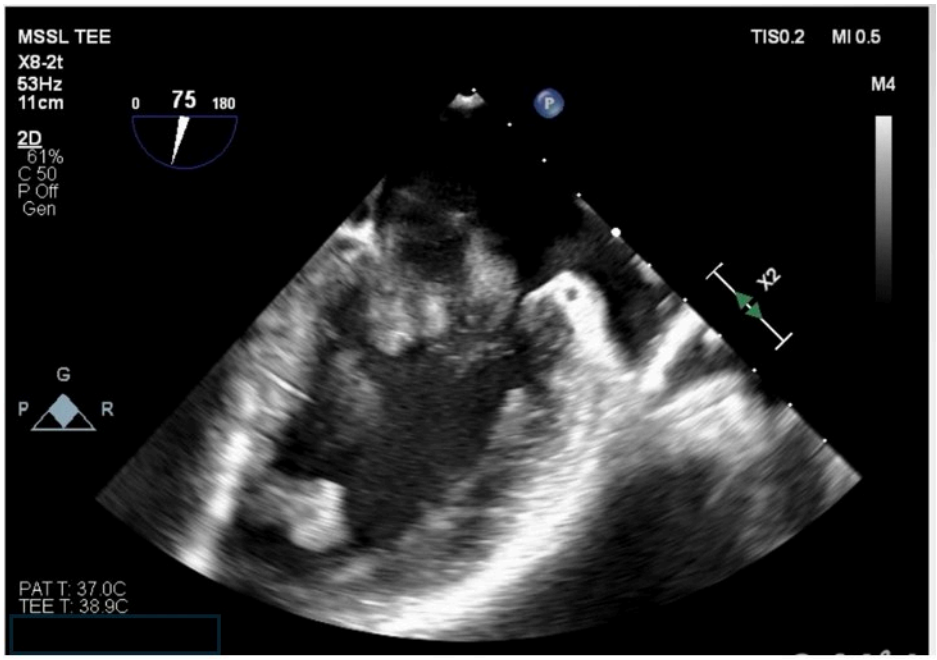
An 18-year-old male with a past medical history of asthma presented with chest pain and syncope on exertion. On arrival to the emergency department, he was hemodynamically stable but reported intermittent dyspnea and lightheadedness. ECG demonstrated sinus rhythm with a right bundle branch block (RBBB) and 1st degree atrioventricular (AV) block. Troponin was 8008. Transthoracic echocardiography revealed an LA mass prolapsing through the mitral valve (MV) during diastole with a mean transmitral gradient of 6 mmHg, and a normal ejection fraction (EF). Cardiac CT scan confirmed a 4 x 4 cm pedunculated mass attached to the interatrial septum (IAS) and also revealed a LV mass measuring 1 x 1 cm attached to the apical wall. Physical exam was notable for dark, cutaneous lesions on the patient's scrotum and umbilicus and a patient height of 5'1". In the operating room, a pre-induction arterial line was placed, a 500cc fluid bolus was given, and the patient was induced with 4 mg midazolam, 300 mcg fentanyl, 50 mg lidocaine, 10 mg etomidate, and 100 mg rocuronium, with epinephrine at 0.02 mcg/kg/min and norepinephrine at 0.02 mcg/kg/min. Blood pressure increased to 230/140 mmHg during intubation but normalized after epinephrine was discontinued. Intraoperative transesophageal echocardiography confirmed an LA mass that protruded through the MV during diastole (without direct MV involvement) and an LV mass on the apical wall. Both masses were excised on cardiopulmonary bypass with minimal intraoperative pressor requirements. The LA mass excision required reconstruction of the IAS with a pericardial patch. Histopathology confirmed both tumors were myxomas. Postoperatively, the patient recovered uneventfully and was discharged home on postoperative day five. Outpatient genetic testing was positive for the PRKAR1A mutation for CC. Endocrinology workup is pending.

Discussion

LA myxomas often originate from the fossa ovalis, the thinnest portion of the IAS.3 Despite this patient having no functional mitral stenosis at rest, there was concern that LV preload would be severely compromised with vasodilation during induction. Tumor proximity to the conduction system may explain the RBBB and 1st degree AV block. While most cases are sporadic[2], young age, tumor recurrence, and/or multiple lesions should prompt evaluation for associated conditions, including CC, a rare autosomal dominant syndrome caused by PRKAR1A mutations[1,3]. CC is characterized by multiple cardiac and extracardiac myxomas, cutaneous lentigines frequently near genitalia, and endocrine tumors such as primary nodular adrenocortical disease or pituitary adenomas[3]. This case underscores the importance of

prompt echocardiographic evaluation in young patients with unexplained syncope, recognition of ECG conduction abnormalities, and the need for genetic follow-up and close surveillance in patients with unusual presentations of cardiac myxomas.

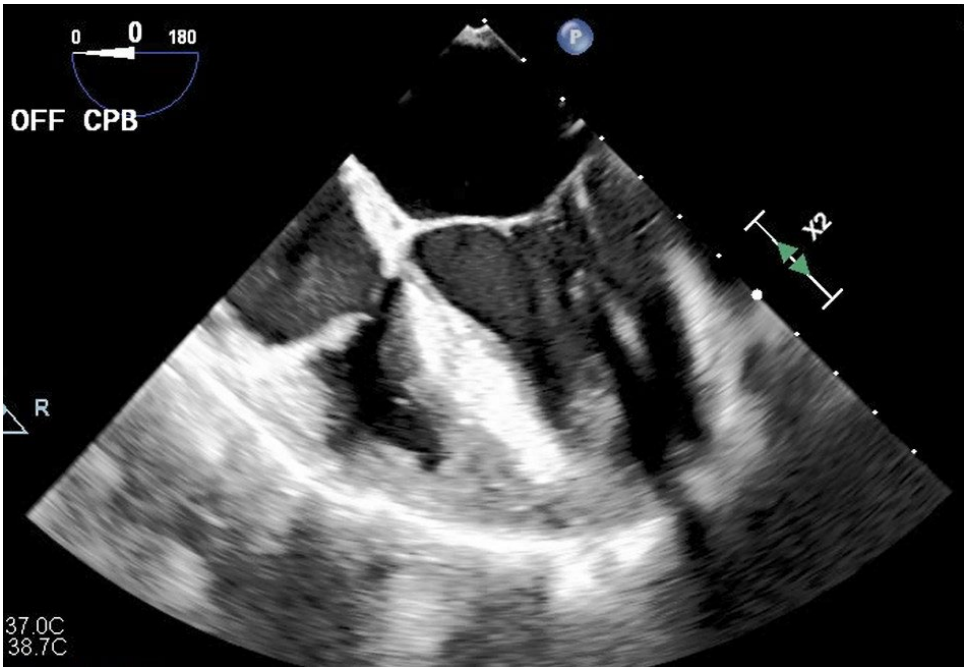
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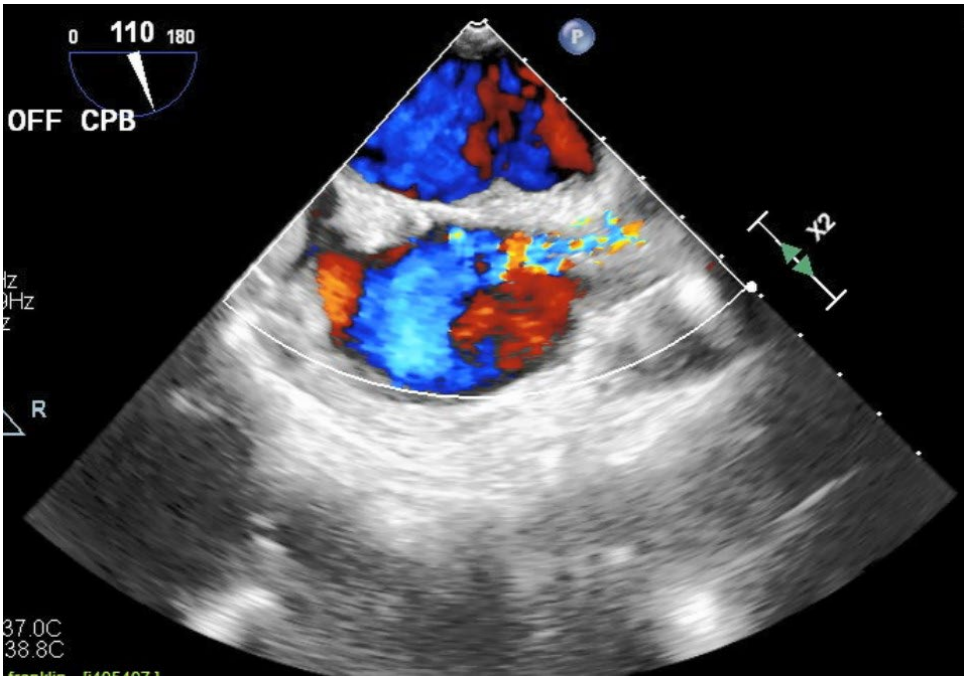
Midesophageal two chamber TEE view of LA mass protruding through mitral valve during diastole. Hyperechoic LV mass also seen on inferior wall near apex.



Transgastric short axis view. LV mass seen on inferior wall.



Post operative four chamber view. LA and LV mass removed.



Postoperative interatrial septum intact.

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#FR 2 – Kiosk 8 – Poster 6

Don't Discount Heparin Induced Thrombocytopenia: A Case of Piperacillin-Tazobactam Induced Thrombocytopenia Masking Concurrent Heparin Induced Thrombocytopenia

Presenting Author: Sean Gruen, MD MPH, Resident Anesthesiologist, Ochsner Clinic Foundation

Introduction

Thrombocytopenia occurs in 30–50% of postoperative cardiac surgery patients and may result from non-immune causes such as dilution, cardiopulmonary bypass–related consumption, or decreased production, as well as immune-mediated processes including heparin induced thrombocytopenia (HIT), drug induced thrombocytopenia (DITP), and post-transfusion purpura.1,2 Because HIT is a common cause in the post-surgical ICU, it must be considered even when initial criteria are incomplete or another etiology appears more likely. We present a case in which a patient developed severe piperacillin–tazobactam induced DITP and concurrent HIT during the same postoperative admission following cardiac surgery.

Case Presentation

A 73-year-old man with coronary artery disease, severe aortic stenosis, moderate mitral regurgitation, severe pulmonary hypertension, and obstructive sleep apnea underwent a three vessel coronary artery bypass graft, bio-prosthetic aortic valve replacement, and mitral valve repair. His postoperative course was prolonged by severe delirium, requiring extended stay in the ICU. He received a 3-day empiric course of vancomycin and piperacillin–tazobactam on postoperative days 1–3 for persistent fever, which was discontinued after negative blood cultures. On postoperative day 10, piperacillin–tazobactam was restarted for recurrent fever and leukocytosis, and within 24 hours his platelet count precipitously fell from 289,000 to 10,000 cells/microliter. Subcutaneous heparin was discontinued, a HIT panel was sent, and hematology was consulted. Evaluation for DIC and hemolysis was unremarkable. Hematology determined that the abrupt drop in platelets—reaching a nadir of 5,000 cells/microliter—was most consistent with DITP due to piperacillin–tazobactam rather than HIT. Intravenous dexamethasone 40 mg/day was started with intravenous immunoglobulin planned if needed. Piperacillin–tazobactam was switched to cefepime and discontinued after five days of negative blood cultures. Platelet count rose to 34,000 cells/microliter the next day and normalized over the following two days. Subcutaneous heparin was restarted on postoperative day 13. The initial HIT panel did not result due to a laboratory error, but was resubmitted despite the resolution of thrombocytopenia. Intravenous steroids were discontinued after four days because of worsening delirium, as platelet counts remained stable. The repeated HIT panel analysis returned weakly positive, and a confirmatory serotonin release assay was positive, establishing the diagnosis of HIT. Heparin was switched to fondaparinux. The patient eventually recovered and discharged to rehab.

Discussion

The patient's rapid platelet recovery after discontinuing piperacillin–tazobactam supports a diagnosis of DITP, driven by drug-dependent antibodies that accelerate platelet destruction. Although piperacillin–tazobactam related DITP has been documented, the syndrome remains under-recognized due to the

antibiotic's widespread use and the overlap of thrombocytopenia etiologies in hospitalized patients.3 Concurrent HIT further complicates the diagnosis, underscoring the need for a high index of suspicion and continued reevaluation. Early recognition and prompt withdrawal of offending agents are essential to prevent complications and facilitate recovery.

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#FR 2 – Kiosk 8 – Poster 7

ECMO as a Bridge to Lung Transplant in Mucinous Adenocarcinoma

Presenting Author: Nicholas Baltera, DO, Cardiothoracic Anesthesia Fellow, University of Miami/Jackson Health System

Introduction

The use of extracorporeal membrane oxygenation (ECMO) in patients with active malignancy has traditionally been limited due to concerns about poor prognosis, complications, and futility. However, selected patients with localized thoracic malignancies and potentially curative treatment options may benefit from ECMO as a bridge to definitive therapy. Mucinous adenocarcinoma of the lung poses additional challenges because of its rapidly progressive course, copious secretions, and high risk of severe respiratory failure. We present a case highlighting the role of ECMO as a bridge to bilateral lung transplantation (BOLT) in a young patient with mucinous adenocarcinoma.

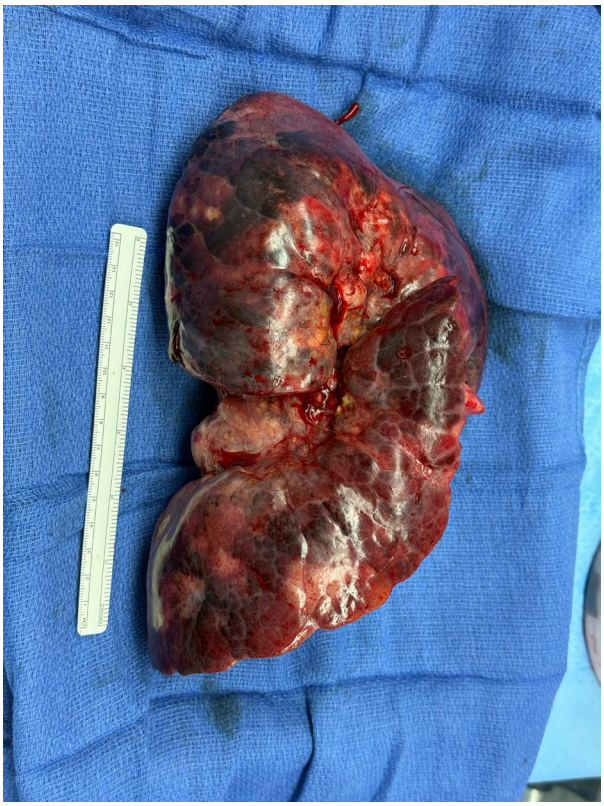
Case Presentation

A 30-year-old woman with mucinous adenocarcinoma of the lung, chronic respiratory failure on 4 L/min home oxygen, bronchiectasis, cavitary lung disease, asthma, and hypothyroidism was admitted to an outside hospital with hypoxic respiratory failure due to bilateral community-acquired pneumonia and progression of her underlying cancer. After clinical deterioration, she was intubated and placed on venovenous (VV) ECMO (right femoral–right internal jugular) for severe ARDS. She was transferred to our center for lung transplant evaluation. On arrival she remained on VV ECMO with a blood-flow rate of 4 L/min, sweep 4 L/min, and FiO_2 100%; ventilator settings were 10/10/10/40%. She required norepinephrine for a MAP of 65mmHg. Prior interventions included bronchoscopy, thoracentesis, and EBUS-guided biopsy confirming mucinous adenocarcinoma without distant metastasis. Imaging demonstrated bilateral consolidations, nodular opacities, hydropneumothorax, and stable left pulmonary artery obstruction. Despite maximal medical support, she developed progressive right-ventricular dysfunction requiring conversion to VV-A ECMO. She underwent successful bilateral orthotopic lung transplantation. Postoperatively, she required return to the operating room on postoperative day one for hemothorax evacuation and difficult ventilation, with washout and arterial decannulation on day two.

Discussion

This case illustrates that ECMO can serve as a bridge to lung transplantation in carefully selected oncology patients. Although malignancy has historically been considered a contraindication, mucinous adenocarcinoma confined to the thorax without metastatic disease may allow for curative transplantation. ECMO provided prolonged cardiopulmonary support, stabilized RV failure, permitted safe transport, and allowed time for multidisciplinary evaluation. The complexity of mucinous tumors (including abundant secretions, susceptibility to infection, and rapid progression) highlights the importance of aggressive multidisciplinary care.

Images



Pre: Right Lung



Post: BOLT

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#FR 2 – Kiosk 9 – Poster 1

Navigating High-Risk Fontan Transplantation: Challenges of Collaterals and Liver Disease

Presenting Author: Nicole A. Duster, MD, Anesthesiology Resident, Brigham and Women's Hospital

Introduction

The Fontan operation has markedly improved survival in single-ventricle patients since the 1970s. Over 40% of Fontan patients reach adulthood[1] but later develop Fontan failure from chronic venous hypertension and low cardiac output, leading to end-organ dysfunction such as Fontan-associated liver disease (FALD), collateral circulation, arrhythmias and more.[2] Up to 10% of Fontan patients require orthotopic heart transplantation (OHT) by the age of 21. We present a case of an adult with Fontan physiology who underwent OHT with a difficult perioperative course due to unique anatomy.

Case Presentation

The patient was a 32-year-old man with d-transposition of the great arteries, hypoplastic left heart syndrome, and prior staged palliation culminating in a lateral tunnel Fontan. He developed FALD, chronic heart failure, and extensive aortopulmonary collaterals (APCs). He presented with decompensated heart failure; after initial hemodynamic optimization, right-heart catheterization showed normal cardiac index and elevated Fontan pressures (22–24 mmHg). There were no significant venovenous collaterals (VVCs), but substantial APC burden from the left bronchial and bilateral internal mammary arteries, which were coil-occluded; small left phrenic APCs were not amenable to intervention. Combined heart–liver transplant (CHLT) evaluation revealed a low hepatic venous pressure gradient (< 2 mmHg), normal portal pressures and preserved synthetic function. Thirty-one days after admission, he underwent OHT complicated by massive hemorrhage from unrecognized APCs and severe cardiogenic and vasodilatory shock requiring VA-ECMO (central aorta, left femoral vein). On POD5, TEE showed improving LV function and support was transitioned to an RVAD (main pulmonary artery, left femoral vein). Worsening instability with an underfilled LV despite increased RVAD flow raised concern for shunting and prompted conversion to VAV-ECMO (central aorta, right internal jugular and left femoral veins). On POD15, during chest washout and ECMO reconfiguration, uncontrolled bleeding from collaterals occurred and the patient expired.

Discussion

Successful transplantation in Fontan patients requires multidisciplinary assessment of anatomy, hemodynamics, and end-organ function to determine candidacy for OHT versus CHLT. Without formal guidelines for FALD grading, biopsy-proven cirrhosis or signs of hepatic failure—varices, ascites, splenomegaly, or thrombocytopenia—should prompt CHLT consideration. Right-heart catheterization, endoscopy, and liver biopsy are essential.[3] Preoperative identification and treatment of APCs and VVCs are equally critical, as APC-related bleeding is a major source of perioperative mortality. Severe vasoplegia and coagulopathy are common, making viscoelastic testing, point-of-care coagulation monitoring, antifibrinolytics, and rapid access to blood products critical to safe operative management. These principles highlight key lessons in Fontan transplant management: selecting appropriate OHT versus CHLT candidates

and anticipating collateral-bleeding and hemodynamic instability that inform perioperative risk.

Images

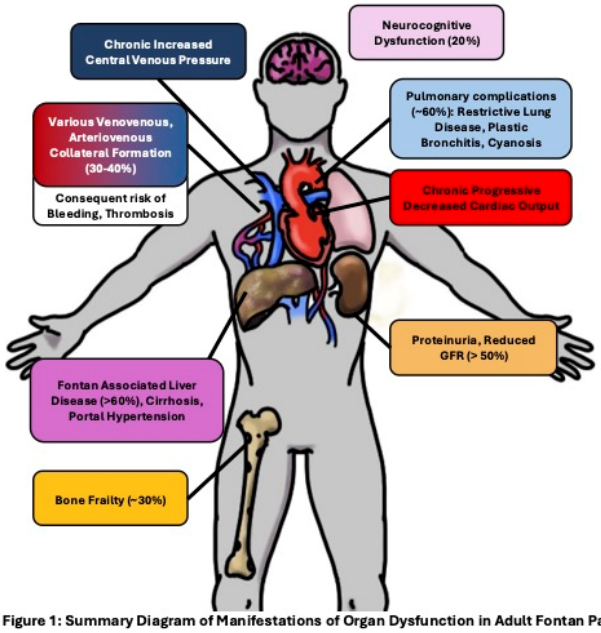


Figure 1: Summary Diagram of Manifestations of Organ Dysfunction in Adult Fontan Patients

Manifestations of Organ Dysfunction in Adult Fontan Patients

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#FR 2 – Kiosk 9 – Poster 2

Surgical Retrieval of Retained Delivery System After Attempted Transcatheter Pulmonic Valve Replacement in RV-PA Conduit Complicated by Balloon Rupture in an Adult Tetralogy of Fallot Patient

Presenting Author: Brendan Swain, MD, Fellow Physician, University of Washington

Introduction

Patients with congenital cardiac lesions often require repeated cardiac procedures and high-risk repeat sternotomies. Transcatheter pulmonic valve replacements (TPVR) are a desirable alternative to surgery with shorter length of stay, lower mortality, and lower recurrent valve regurgitation when compared to surgical valve replacement.1 However, these procedures are not without complications and morbidity of their own. In this case, we present a balloon rupture during an attempted transcatheter pulmonic valve replacement requiring surgical retrieval of the delivery system.

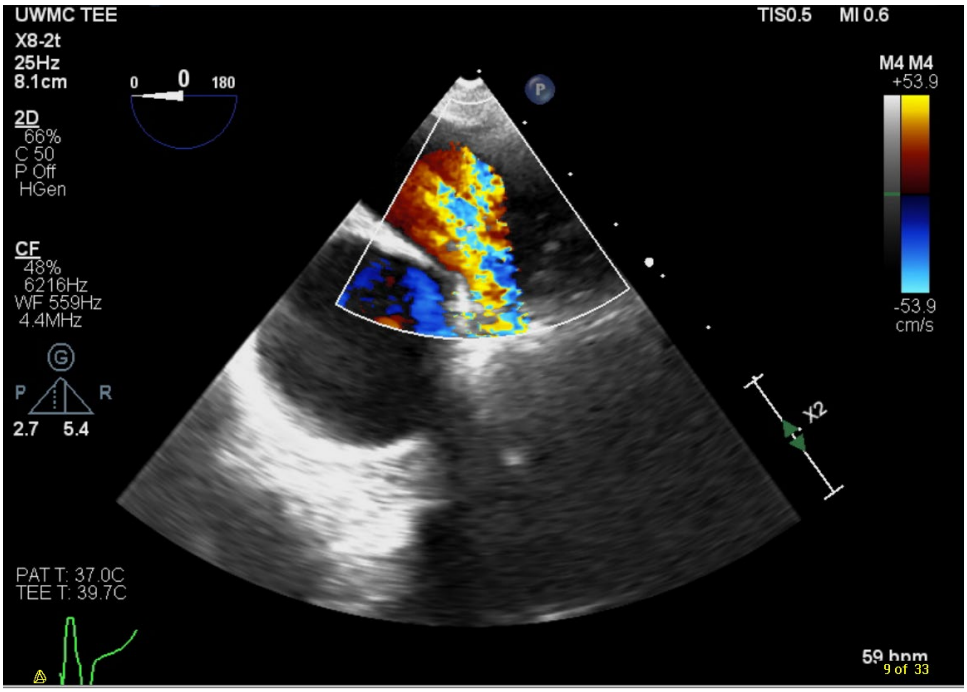
Case Presentation

Our patient is a 30-year-old woman with history of Tetrology of Fallot repaired with transannular patch at 6 months of age; surgical pulmonic valve replacement for severe regurgitation at the age of 6; and implantation of a right ventricle to pulmonary artery conduit (22mm Contegra) at the age of 15. She had recently been through her first pregnancy and was considering a second child. For this reason, her RV-PA conduit was evaluated and found to have moderate stenosis (mean gradient of 36 mmHg). She had recently undergone balloon dilation of the conduit without change to the invasive gradient. During the attempted TPVR (Sapien S3) and pulmonary artery stenting with interventional cardiology, the balloon of the valve deployment system ruptured and was unable to be removed. There was concern on fluoroscopy for balloon fragment entrapment by the PA stent, so the system was left in place, and cardiac surgery was consulted for potential retrieval. The patient arrived at the operating room intubated and under general anesthesia. Femoral venous and arterial sheaths were placed prior to sternotomy, and a difficult dissection followed. Prior to the system removal, it was noted that the balloon was tethered to the tines of the PA stent. The prior RV-PA conduit was subsequently replaced with a 26mm homograft.

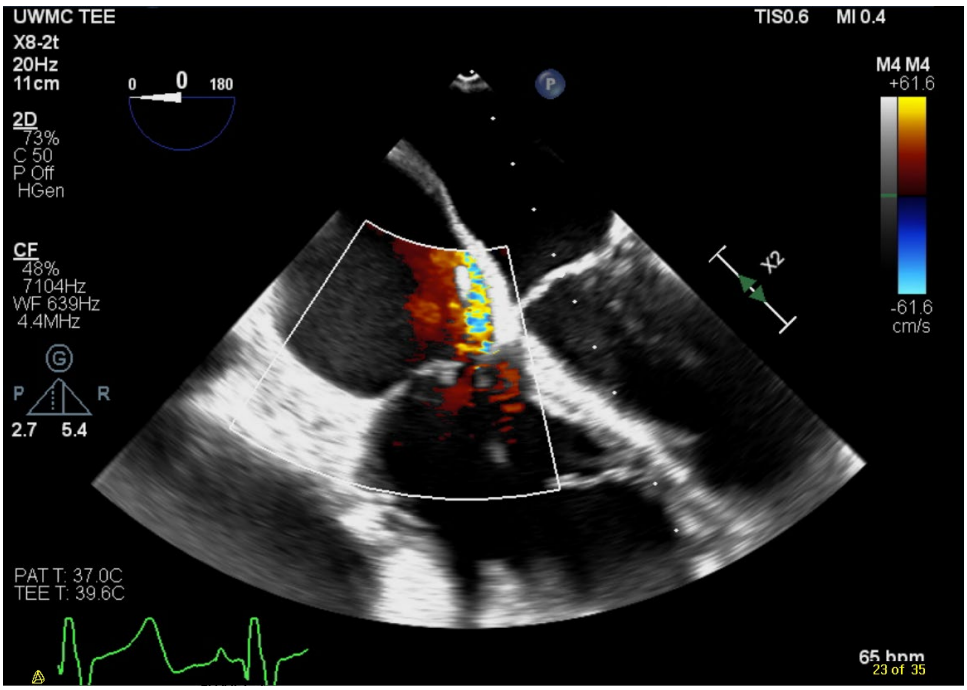
Discussion

Transcatheter pulmonic valve replacement has a very high success rate (~98%). While our patient did not have native right heart anatomy, studies have shown no difference in success rates among patients with native right ventricular outflow tract (RVOT) anatomy versus conduit anatomy.2 Balloon rupture is quite rare, best described in transcatheter aortic valve replacement with risk factors including annular and left ventricular outflow tract calcification.3 Risk factors for rupture during TPVR are not well described. In our patient, the previously deployed pulmonary artery stents likely served as an elastic structure against which the balloon sustained mechanical damage. The operative note supported this and noted that the balloon had become intertwined with the tines of the pulmonary artery stent.

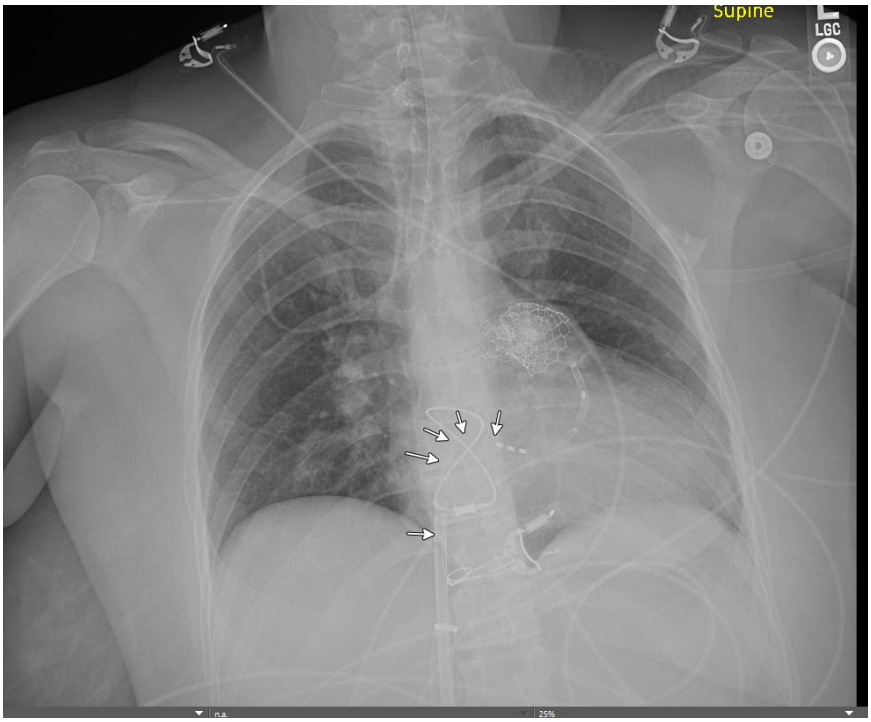
Images



This midesophageal ascending aorta short-axis view shows the hyperechoic delivery system in the right pulmonary artery causing some turbulent flow around it.



This midesophageal four-chamber view shows the delivery system along the septal leaflet of the tricuspid valve with associated regurgitation.



This x-ray demonstrates the delivery system near the recently deployed pulmonary artery stents and valve.

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#FR 2 – Kiosk 9 – Poster 3

Incidental VSD During Cardiac Surgery, to fix or not to fix?

Presenting Author: Jasmine Tuteja, MD, Northwell Health

Introduction

Ventricular septal defects (VSD) are the most common congenital cardiac anomaly in children, and also one of the most common heart defects in adults. They can either be congenital or acquired, with the acquired form usually occurring after ischemic events or inflammatory conditions. The decision of whether or not to repair a VSD in adulthood is made based on various factors, such as presence of hemodynamic compromise or signs of left ventricular dysfunction. Another congenital anomaly found in many adults is a patent foramen ovale (PFO). Although there exists literature on how to proceed with incidentally found intraoperative PFOs, no literature exists on how to proceed with incidentally found VSDs. We look at a case in which a patient presented for a coronary artery bypass grafting (CABG) and was found to have an incidental membranous VSD intraoperatively on transesophageal echocardiography (TEE).

Case Presentation

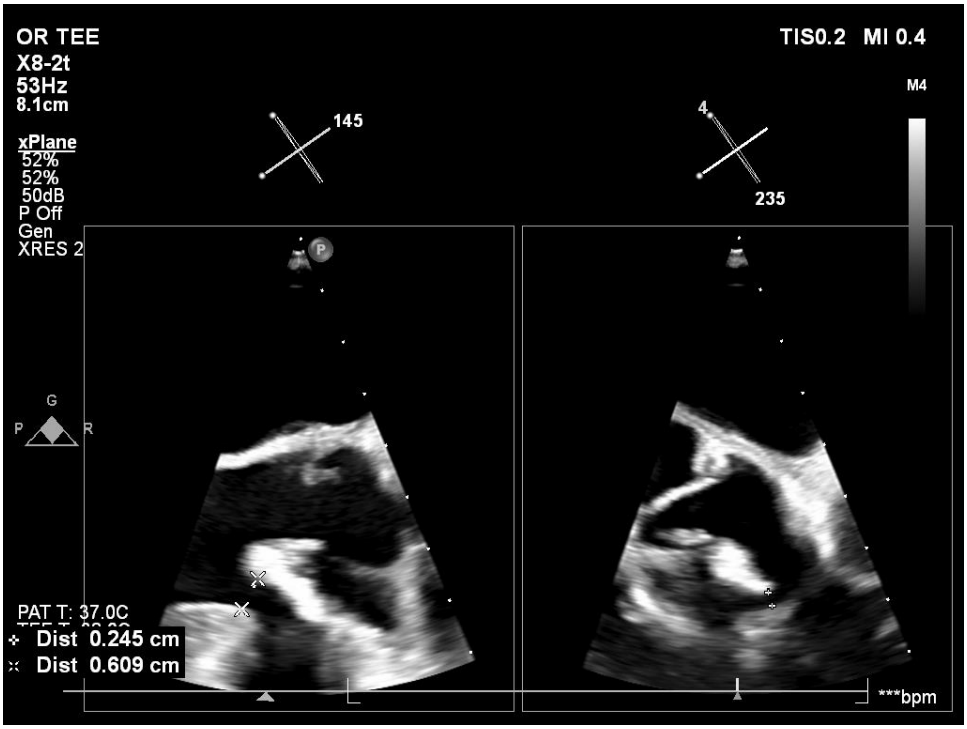
A 66-year-old male with history of hypertension and hyperlipidemia presented for CABG due to multivessel coronary artery disease. Intraoperative TEE unexpectedly revealed a 2.5mm x 6.1mm membranous VSD with left-to-right shunting, dilated RV, and normal RV function. RVSP was 23 mmHg with an RA pressure of 5 mmHg, resulting in a peak VSD gradient of 59 mmHg. The incidental VSD posed a significant intraoperative management dilemma. The absence of a preoperative PA catheter and unexpected finding necessitated real-time decision-making. Without preoperative PA catheter data, real-time hemodynamic assessment by the surgical and anesthesia teams was essential. TEE findings, combined with direct intraoperative hemodynamic measurements showing RV pressures of 20/5 mmHg and PA pressures of 19/7/12 mmHg, were crucial for assessing the VSD's hemodynamic significance. Ultimately, due to the patient's hemodynamic stability and the dimensions of the defect being amendable to percutaneous repair, the decision was made to not repair the VSD during the current CABG operation.

Discussion

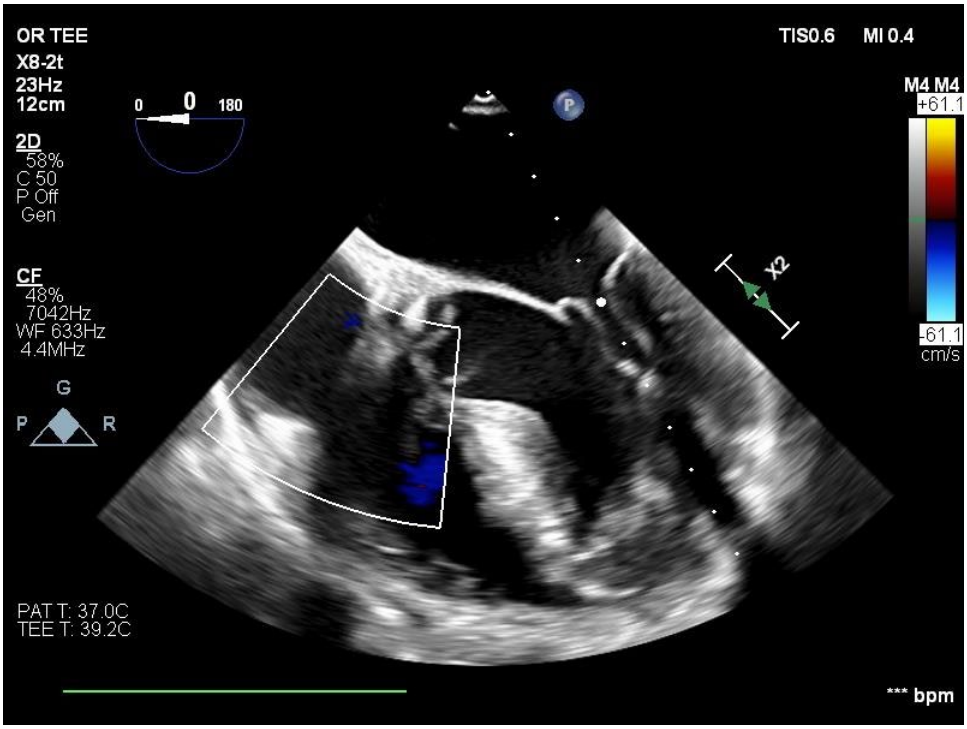
This case highlights the critical role of intraoperative TEE in uncovering unexpected findings and guiding surgical decisions. Intraoperative TEE can not only identify surgical complications such as aortic dissection, tamponade or valvular trauma, but can also reveal incidental findings that may necessitate intervention depending on severity. In this particular case, the decision to defer VSD repair was based on the patient's hemodynamic stability and the procedural risks involved with the repair. Aortic regurgitation, left ventricular dilation, and pulmonary hypertension are usually indications for closure, of which this patient had none. Procedural risks of surgical VSD repair can include valvular incompetence, arrhythmias, and progression of pulmonary hypertension. This case emphasizes the challenges of unexpected findings during surgery and the need for further research on managing incidentally discovered VSDs. Non-invasive options remain for

future management.

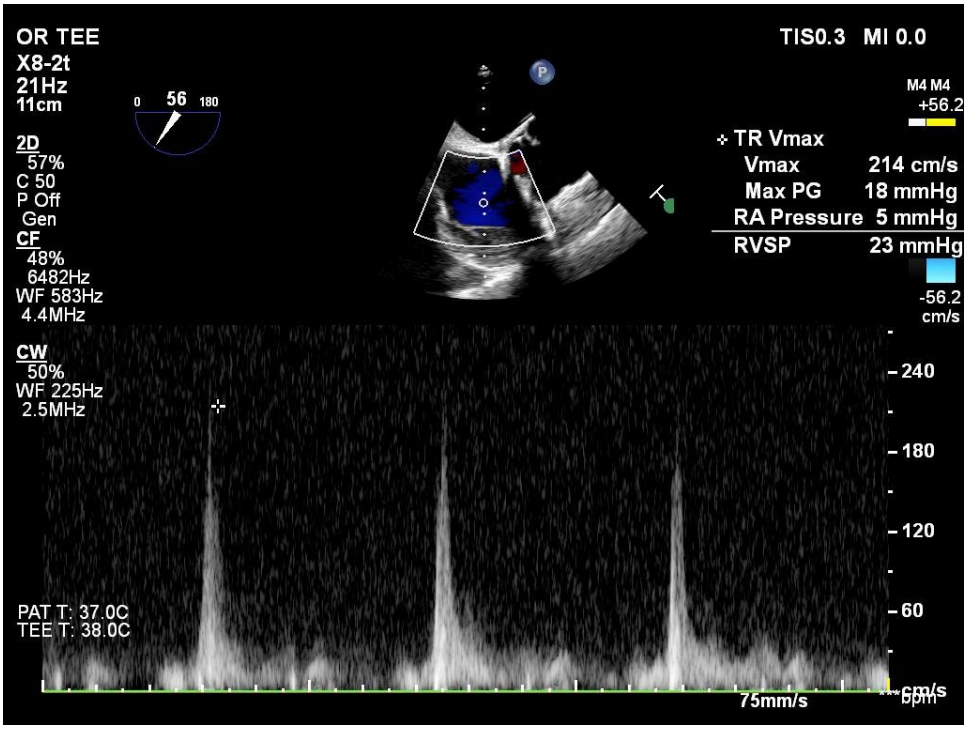
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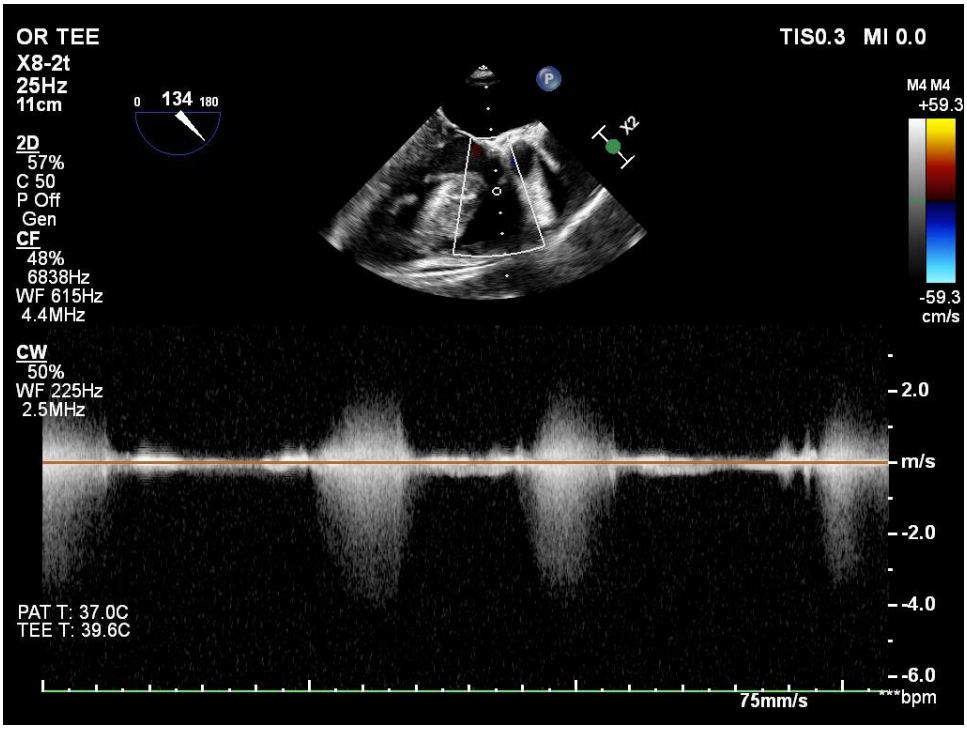
measurements of VSD



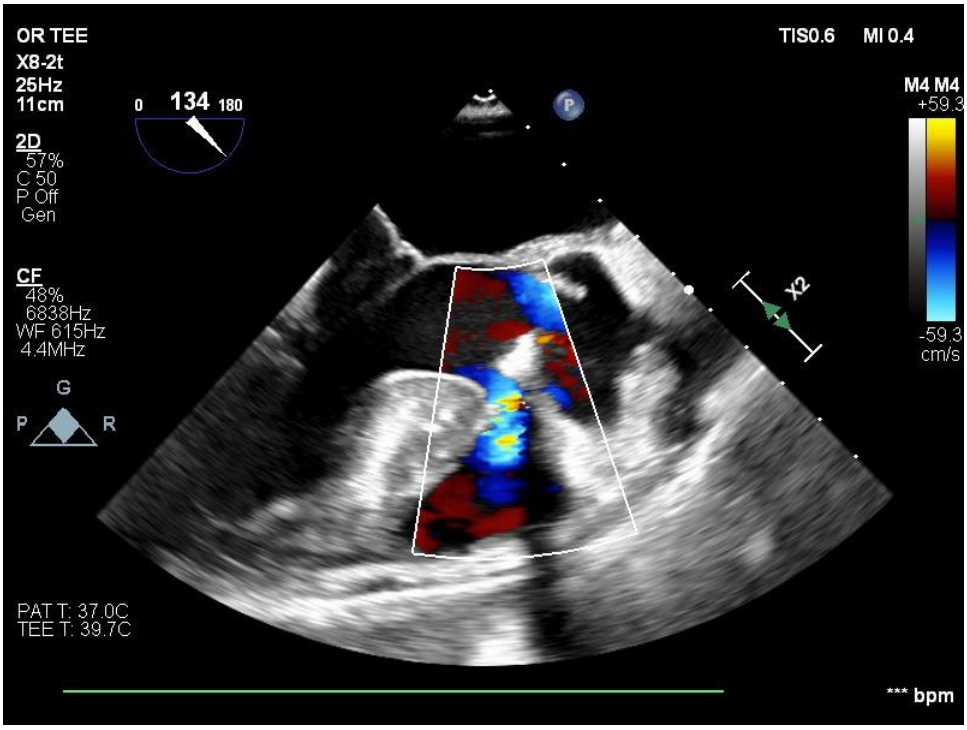
Dilated RV.



RVSP with RA Pressure



Flow VTI through VSD show left to right flow



Color flow doppler showing flow through membranous VSD

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#FR 2 – Kiosk 9 – Poster 4

Ventricular Septal Defect Associated with a Perimembranous Ventricular Septal Aneurysm

Presenting Author: Andrew Whang, MD, Cardiothoracic Anesthesia Fellow Physician, Duke University

Introduction

A perimembranous ventricular septal aneurysm (VSA) is a rare, often clinically silent condition, associated with other congenital heart defects such as a ventricular septal defect (VSD), and can be mistaken for a sinus of Valsalva aneurysm (SOVA). Its formation may be related to a spontaneous closure, either partial or complete, of a congenital VSD by approximation of the septal leaflet of the tricuspid valve across the defect.[1] In certain circumstances, a perimembranous VSA can cause arrythmias or hemodynamically significant complications such as obstruction of the right ventricular outflow tract, tricuspid insufficiency, aortic valve prolapse, and bacterial endocarditis. We present a case of an incidentally found VSD associated with a perimembranous VSA that was initially mistaken for a ruptured sinus of Valsalva aneurysm.

Case Presentation

A 75-year-old female with a history of hypertension presented with a complaint of palpitations, shortness of breath, and near syncope. Initial transthoracic echocardiography (TTE) findings were concerning for a severely calcified aortic valve, severe aortic stenosis, and a tricuspid valve with an echogenic mass possibly attached to the septal leaflet and septum, presumed to be a SOVA. There was trivial amount of tricuspid regurgitation. She was transferred to our institution for a higher level of care. Intraoperative transesophageal echo (TEE) revealed a VSA with a perimembranous VSD, without evidence of a SOVA. The patient subsequently underwent an aortic valve replacement and VSD closure with a patch. The presence of a perimembranous VSA and a perimembranous VSD were confirmed by the surgical team. Postoperatively, the VSD was repaired with a patch without residual flow by color Doppler.

Discussion

A VSA is a rare congenital abnormality that can be associated with other congenital heart defects such as a VSD. Although uncommon, they are possible to find in older patients.[2] Careful attention should be paid to exclude a SOVA on echocardiography because they occur anatomically adjacent to each other at the level of aortic annulus. A rupture of a SOVA typically has a more aggressive course depending on the fistula size and can lead to heart failure, arrhythmias, or sudden cardiac death. Hence surgical repair or transcatheter intervention is recommended. Key differences between a perimembranous VSA and SOVA are location, with a SOVA located above the aortic annulus and a perimembranous VSA located below (Figure 1, 2, and 3). However, due to the limited views with TTE, as in this case, it may be hard to differentiate the two pathologies based on location alone. Spectral Doppler can show diastolic reversal of flow in the descending aorta in the absence of severe aortic regurgitation, as well as continuous high-velocity flow across the aneurysm with accentuation of the waveform in diastole with a ruptured SOVA. A VSD will typically have high velocity turbulent flow during systole and low or absent velocity in diastole. Additionally, the shunt occurring with SOVA will be seen primarily in mid-diastole and with a VSD, in systole. These additional features can

help differentiate a SOVA from a VSD.[3] In our case, we did not see diastolic flow reversal in the descending aorta and observed the shunt primarily in systole (Figure 4 and 5).

Images

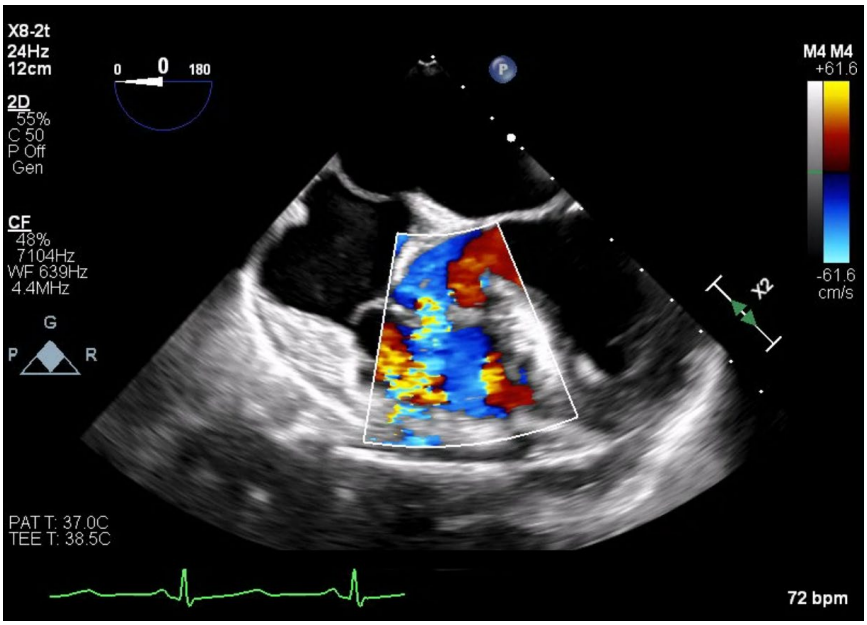


Figure 1: Color flow Doppler of the ventricular septal defect seen in the mid-esophageal four chamber view demonstrating left to right shunt

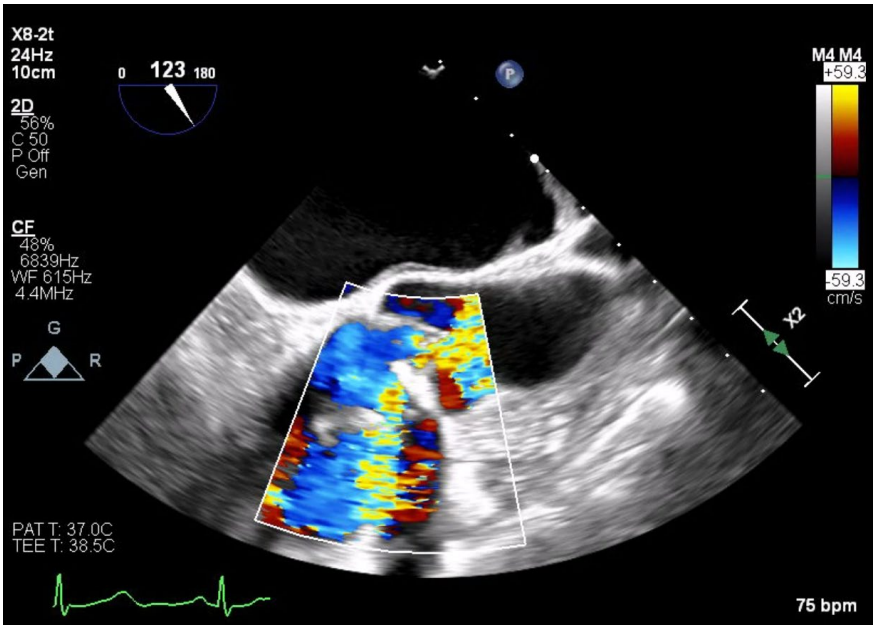


Figure 2: Color flow Doppler of the ventricular septal defect seen in the mid-esophageal long axis view demonstrating left to right shunt

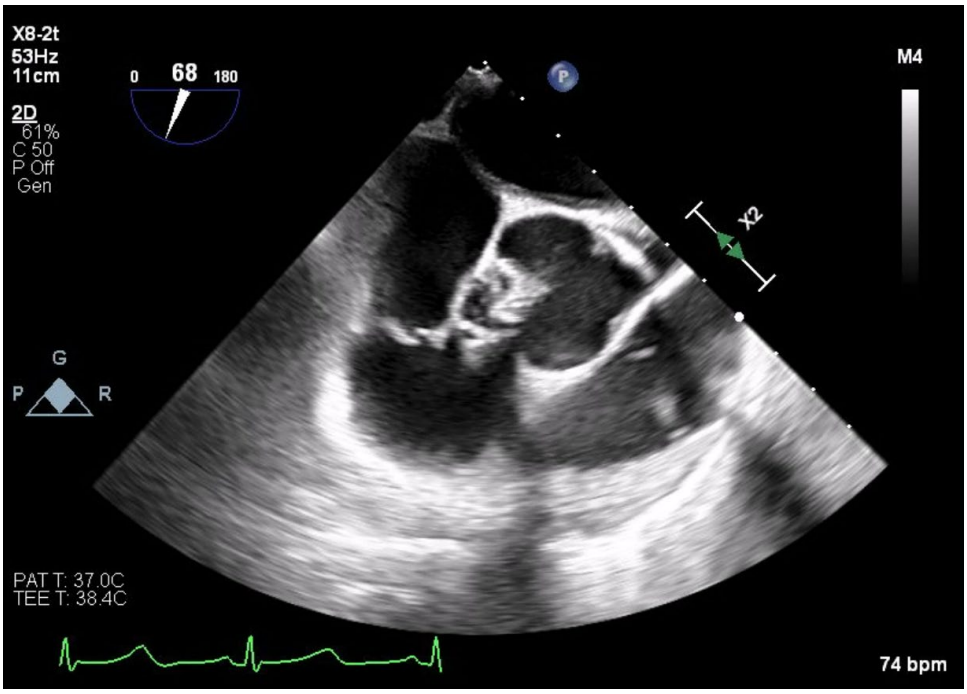


Figure 3: Ventricular septal aneurysm identified in the ME right ventricle inflow-outflow view

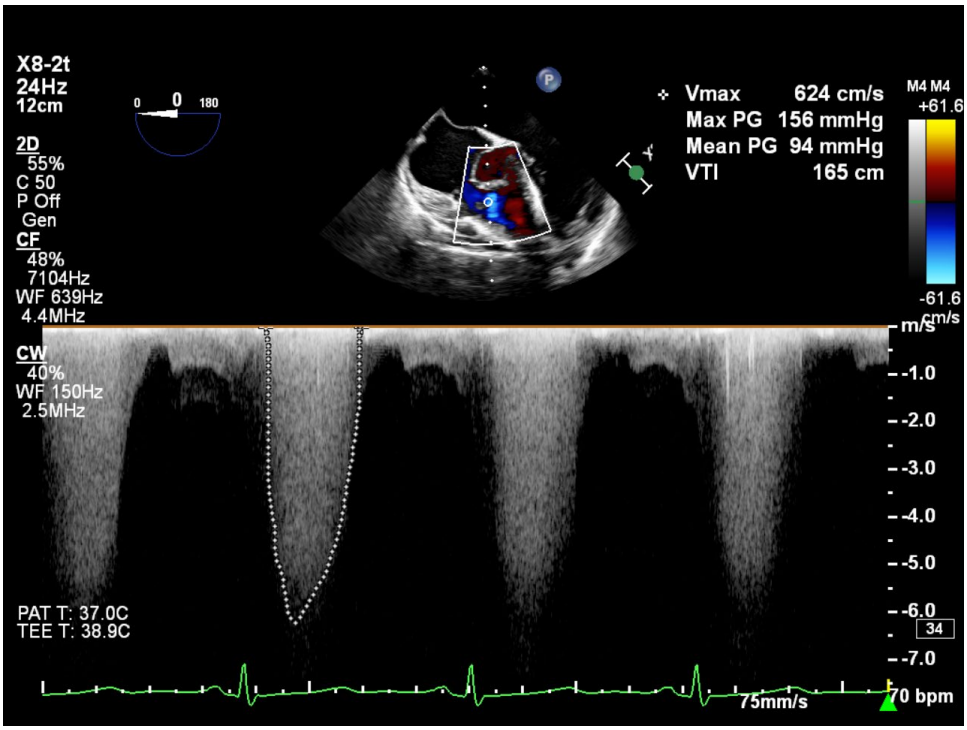


Figure 4: Spectral Doppler interrogation of the left to right shunt associated with the ventricular septal defect. The peak velocity was 6.24 m/s (peak gradient 156 mmHg).



Figure 5: Spectral Doppler for interrogation of diastolic flow reversal in the aorta

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#FR 2 – Kiosk 9 – Poster 5

Anesthetic Management of Fourth-time Reoperative Root Replacement with Multi-Structural Pseudoaneurysms, LVOT Fistulization, and Intracardiac Shunt

Presenting Author: Christina H. Le, MD, Resident Physician, UCLA Medical Center

Introduction

Reoperative aortic root surgery carries increasing risk, especially with prior homograft, coronary button reimplantation, and anatomy distorted by pseudoaneurysms or pacing hardware. We present a fourth-time aortic root replacement that highlights the anesthetic challenges of managing volume overload, conduction disease, and cerebral protection in high-risk reoperative cardiac surgery.

Case Presentation

A 51-year-old woman with a history of SLE, methamphetamine use, bicuspid aortic valve, and three prior aortic root replacements (2007/2013/2023) presented with worsening heart failure. History included heart block with pacemaker dependence, atrial flutter s/p ablation, hypertension, and recurrent endocarditis. Preoperative TEE revealed two aortic root pseudoaneurysms: a left main button pseudoaneurysm with LVOT fistula causing torrential paravalvular AR (Figure 1), and a second annular pseudoaneurysm (Figure 2). A perimembranous VSD with Gerbode physiology (Qp:Qs 1.8:1) and severe central TR were present. RHC showed biventricular volume overload with elevated LVEDP, RVEDP, and RPAWP. She was pacemaker-dependent with chronic lead dysfunction and intermittent loss of capture. A pre-induction arterial line was placed, and anesthesia was induced with midazolam, fentanyl, lidocaine, ketamine, propofol, rocuronium, and phenylephrine. After atraumatic intubation, a CVC and PA catheter were placed. Given high reentry risk, peripheral CPB was initiated before sternal entry. During cooling, an LV apical vent was placed via left thoracotomy to prevent distention in fibrillation. EEG/SSEP guided neuroprotection was employed during cooling to 19 °C and two circulatory arrest periods (total 21 min). Surgery included root replacement, Cabrol reimplantation of RCA/LM buttons, repair of both pseudoaneurysms, VSD closure, and tricuspid valve replacement. Post-repair TEE showed no residual shunt or regurgitation. New epicardial pacing leads were placed.

Discussion

This case exemplifies the layered planning required for high-risk reoperative cardiac surgery. Severe AR and Gerbode physiology created biventricular overload, influencing both induction strategy and bypass management. A gentle approach with midazolam and ketamine, while limiting propofol minimized cardiovascular depression. With chronic lead dysfunction, backup pacing was confirmed pre-induction. Anticipating reentry risk, we opted for peripheral CPB and pre-sternotomy cooling, consistent with published strategies for high-risk resternotomy.[1] During CPB, early LV venting was critical to mitigate distension from torrential AR and shunt physiology, which would have otherwise impaired venous return and myocardial protection. Shunt closure heightened RV afterload, necessitating vigilant weaning with TEE, PA catheter monitoring, and inotropes. This case highlights the importance of proactive planning, close interdisciplinary

collaboration, and an individualized approach to managing complex physiology in reoperative cardiac surgery.[2]

Images

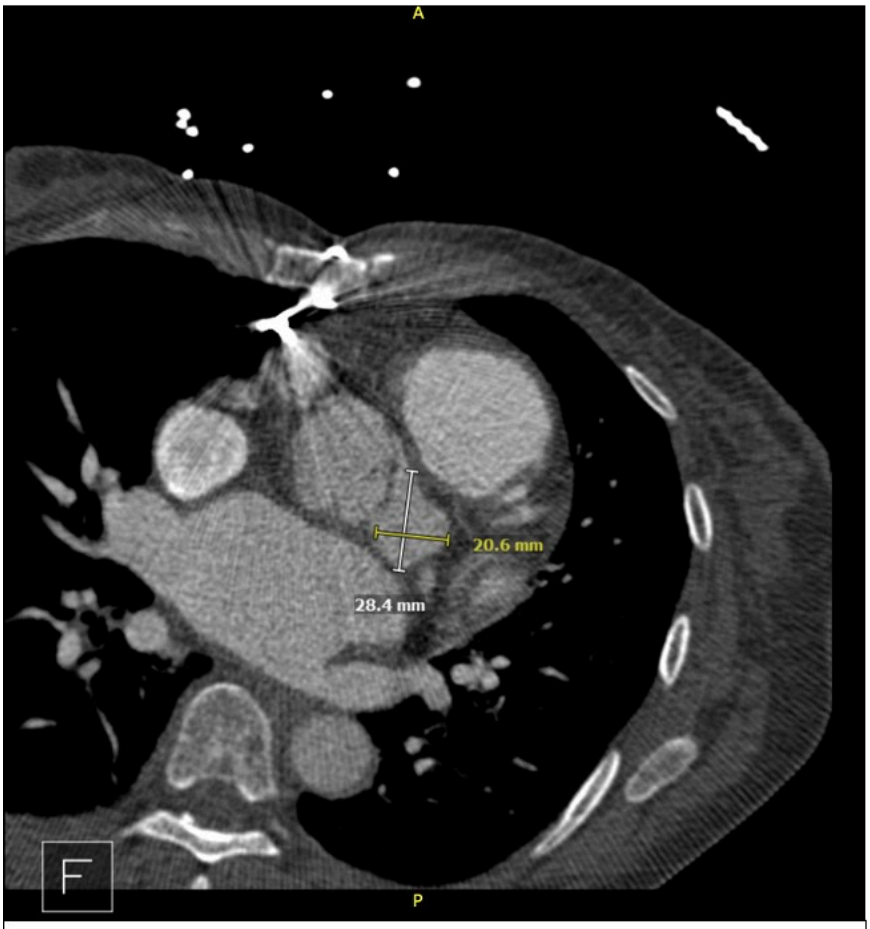


Figure 1. CTA demonstrating a 2.8 x 2.1 cm pseudoaneurysm arising from the posterior/left posteromedial aspect of the aortic root adjacent to the left coronary ostium, communicating with the LVOT and extending anteriorly between pulmonary artery and ascending thoracic aorta



Figure 2. CTA demonstrating second 1.8 x 1.6 cm pseudoaneurysm originating from posterior/right posterolateral aortic root and LVOT

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#FR 2 – Kiosk 9 – Poster 7

When Standard Practice Meets Rare Allergy: Anticoagulation Strategy for Cardiac Surgery in Alpha-Gal Syndrome

Presenting Author: Billy Nguyen, Resident, Brigham and Women's Hospital

Introduction

Alpha-gal syndrome is an acquired allergic condition characterized by an IgE-mediated reaction to the galactose-alpha-1,3-galactose sugar, otherwise known as alpha-gal. This condition can develop after a bite from the Lone Star tick, common in most parts of the eastern and south-central USA. Patients with alpha-gal syndrome are at risk for allergic reactions, including anaphylaxis, when exposed to alpha-gal, which is found in mammalian meat. When treating patients with this condition, anesthesiologists must carefully consider medications manufactured with animal-derived ingredients that may contain or be contaminated with alpha-gal.

Case Presentation

We present an 80-year-old man scheduled for laser-assisted pacemaker lead extraction due to RV lead dysfunction. His PMH included sick sinus syndrome with pacemaker placement 25 years ago and alpha-gal syndrome, diagnosed after multiple tick bites near his home on Cape Cod, an area known to harbor the Lone Star tick. He had repeated episodes of systemic anaphylaxis associated with ingestion of mammalian meats and RAST IgE was positive for alpha-gal. Because his lead was 25 years old, there was a concern for catastrophic cardiac and vascular complications during the procedure necessitating thoughtful preoperative planning. A particular question of interest was whether it would be safe to anticoagulate the patient with heparin if he experienced an intraoperative complication requiring cardiopulmonary bypass. Since heparin products are derived from porcine tissue that may contain alpha-gal, there is a theoretical risk of allergic reaction in patients with alpha-gal syndrome. After an allergist consultation, the plan was to proceed, with the understanding that if systemic anticoagulation became necessary, the risks and benefits of using heparin versus other agents (bivalirudin or argatroban) would be weighed. Fortunately, the patient's lead extraction under GA was straightforward. The dysfunctional RV lead was successfully extracted, a new lead was implanted, and he was discharged home the next day.

Discussion

Systemic anticoagulation is a key component of cardiac surgery requiring CPB. Heparin is preferred over

bivalirudin and argatroban because it can be reliably reversed with protamine, whereas the latter two agents lack reversal options if serious bleeding occurs. Several retrospective studies in recent years have suggested that the risk of allergic reactions to heparin in patients with alpha-gal syndrome is exceedingly low[1,2]. However, one retrospective study specific to cardiac surgery reported allergic reactions in up to 50% of alpha-gal patients after receiving a heparin loading dose, with risk correlating to elevated preoperative alpha-gal IgE levels[3]. These findings suggest that in patients with alpha-gal syndrome, preoperative evaluation should include a recent alpha-gal IgE titer. If levels are high, premedication should be considered prior to cardiac surgery. Despite its rarity, anesthesiologists should be familiar with alpha-gal syndrome and the implications it has on anticoagulation for CPB. While studies show that most patients with alpha-gal syndrome can safely receive heparin, careful consideration of the risks and benefits must be discussed with the entire care team prior to surgery to provide optimal care.

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#FR 2 – Kiosk 10 – Poster 1

Multidisciplinary Management of IgE-Mediated Heparin Hypersensitivity in a Patient Undergoing Re-Operative Mitral Valve Replacement

Presenting Author: Connie Nguyen, MD, Columbia University Medical Center / NewYork-Presbyterian Hospital

Introduction

Heparin remains the preferred anticoagulant for cardiopulmonary bypass (CPB) because it can be rapidly titrated, reliably monitored with activated clotting time (ACT), and fully reversed with protamine [1]. Although rare, both delayed and immediate IgE-mediated hypersensitivity reactions have been reported [2], and perioperative management guidelines are limited. Alternatives such as bivalirudin or argatroban lack reversal agents and pose a higher bleeding risk [1]. We describe the multidisciplinary management of heparin hypersensitivity in a patient undergoing a third re-operative mitral valve replacement.

Case Presentation

A 65-year-old woman with atrial fibrillation on warfarin and a history of endocarditis requiring aortic and mitral valve replacements in 2018—complicated by paravalvular leak (PVL) and repeat repair in 2024—presented for a third redo mitral valve replacement for symptomatic PVL not amenable to transcatheter therapy. The night before surgery, she administered enoxaparin and developed a pruritic, erythematous rash on her hands and feet. Diphenhydramine improved symptoms, although itching persisted on arrival to the perioperative area. After consultation with Allergy, a graded heparin challenge was performed in the operating room (OR). After placement of standard ASA monitors, two large-bore IVs, and an arterial line, she received 80 units of heparin. Thirty minutes later, she developed hives on the trunk, consistent with an IgE-mediated reaction (Images 1 and 2). The procedure was postponed, and the patient was admitted to the ICU for heparin desensitization. Following pre-medication, escalating heparin doses were administered per a graded desensitization protocol under close monitoring (Image 3). She was maintained on a heparin infusion until returning to the OR the next day, where she tolerated a test dose followed by therapeutic heparinization and subsequently underwent an uncomplicated CPB run for mitral valve replacement.

Discussion

This case illustrates key perioperative challenges when heparin hypersensitivity is identified before cardiac surgery. Although alternatives such as bivalirudin can provide effective anticoagulation during CPB, their use may increase transfusion requirements [1]. Given this patient's anticoagulation for atrial fibrillation, history of multiple sternotomies, and the complexity of the planned operation, bivalirudin was deemed to pose an excessive bleeding risk. A heparin challenge in the operating room allows for assessment of suspected IgE-mediated reactions under close monitoring, enabling early recognition and management. When a challenge confirms hypersensitivity, but CPB remains essential, rapid desensitization in the ICU provides a controlled environment for dose escalation, with immediate access to advanced support. For anesthesiologists, careful planning includes coordination with allergy and surgical teams, hemodynamic monitoring during

heparin administration, and readiness to treat anaphylaxis. Familiarity with desensitization protocols expands therapeutic options for patients who might otherwise be excluded from surgery and highlights the role of the cardiac anesthesiologist in coordinating the multidisciplinary management of rare but life-threatening medication allergies.

Images



Pruritic erythematous rash on the abdomen.



Pruritic erythematous rash with dermatographism on the hip.

Dose #	Bag #	Time (H:MM)	Rate ($ml.h^{-1}$)	Volume to be Infused (ml)	Dose given (IU)	Rate ($IU.h^{-1}$)	Cumulative dose (IU)	Infuse for
Start with bag #1, which is 5 units of unfractionated heparin in 250 ml 0.9% sodium chloride								
1	1	0:00	4 $ml.h^{-1}$	1 ml	0.02	0.08 $IU.h^{-1}$	0.02	15 min
2	1	0:15	8 $ml.h^{-1}$	2 ml	0.04	0.16 $IU.h^{-1}$	0.06	15 min
3	1	0:30	16 $ml.h^{-1}$	4 ml	0.08	0.32 $IU.h^{-1}$	0.14	15 min
4	1	0:45	32 $ml.h^{-1}$	8 ml	0.16	0.64 $IU.h^{-1}$	0.3	15 min
Change to bag #2, which is 50 units of unfractionated heparin in 250 ml 0.9% sodium chloride								
5	2	1:00	6.4 $ml.h^{-1}$	1.6 ml	0.32	1.28 $IU.h^{-1}$	0.62	15 min
6	2	1:15	12.8 $ml.h^{-1}$	3.2 ml	0.64	2.56 $IU.h^{-1}$	1.26	15 min
7	2	1:30	25.6 $ml.h^{-1}$	6.4 ml	1.28	5.12 $IU.h^{-1}$	2.54	15 min
8	2	1:45	51.2 $ml.h^{-1}$	12.8 ml	2.56	10.24 $IU.h^{-1}$	5.1	15 min
Change to bag #3, which is 500 units of unfractionated heparin in 250 ml 0.9% sodium chloride								
9	3	2:00	10.2 $ml.h^{-1}$	2.55 ml	5.1	20.4 $IU.h^{-1}$	10.2	15 min
10	3	2:15	20.5 $ml.h^{-1}$	5.125 ml	10.25	41 $IU.h^{-1}$	20.45	15 min
11	3	2:30	41 $ml.h^{-1}$	10.25 ml	20.5	82 $IU.h^{-1}$	40.95	15 min
12	3	2:45	81.9 $ml.h^{-1}$	20.475 ml	40.95	163.8 $IU.h^{-1}$	81.9	15 min
13	3	3:00	163.8 $ml.h^{-1}$	40.95 ml	81.9	327.6 $IU.h^{-1}$	163.8	15 min
Change to bag #4, which is 5000 units of unfractionated heparin in 250 ml 0.9% sodium chloride								
14	4	3:15	32.7 $ml.h^{-1}$	8.175 ml	163.5	654 $IU.h^{-1}$	327.3	15 min
15	4	3:30	50 $ml.h^{-1}$	50 ml	1000	1000 $IU.h^{-1}$	1327.3	60 min

Graded desensitization protocol with escalating doses of intravenous unfractionated heparin.

References

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#FR 2 – Kiosk 10 – Poster 2

Acute Aortic Intramural Hematoma with Refractory Right Ventricular Failure After Cardiopulmonary Bypass

Presenting Author: Jae Woong Lee, MD, Clinical Fellow, NewYork Presbyterian Hospital-Weill Cornell Medical Center

Introduction

Intraoperative aortic dissection and, less commonly, intramural hematoma (IMH) are rare, but life-threatening complications of cardiac surgery. We report a case of unusual appearing acute IMH involving the ascending, arch, and descending aorta without an obvious dissection flap discovered upon removal of the aortic cross clamp after aortic valve replacement and conduit graft replacement of the aortic root and ascending aorta (AVR conduit). This ultimately necessitated unplanned circulatory arrest and hemiarch replacement, which was complicated by severe right-ventricular failure necessitating VA-ECMO support.

Case Presentation

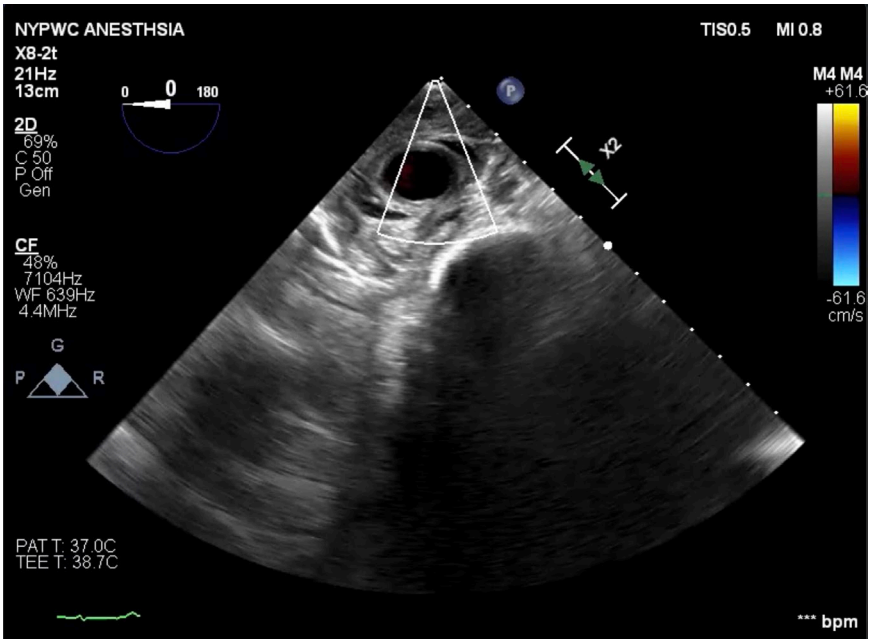
A 73-year-old man with hypertension, COPD, prior TIA, and anemia presented after a fall. Workup revealed severe bicuspid aortic stenosis (AVA 0.6 cm^2) and dilation of the aortic root and ascending aorta. The patient underwent AVR conduit and upon aortic unclamping, there was an acute loss of volume and inability to fully flow on cardiopulmonary bypass as well as a large hematoma noted at the distal ascending aorta on the surgical field. TEE showed an enlarging circumferential IMH extending from the distal graft to the arch and descending aorta without a clear dissection flap. Due to concern for evolving dissection, a hemiarch repair under deep hypothermic circulatory arrest was performed. Post-repair separation from bypass was limited by profound RV failure, refractory hypotension, and hypoxemia, requiring initiation of central VA-ECMO.

Discussion

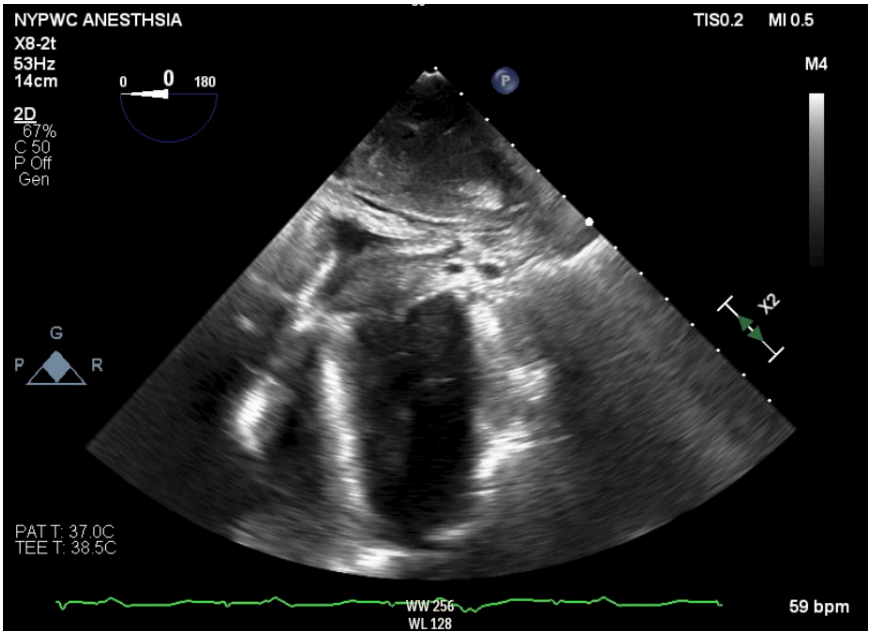
Rapid diagnosis and treatment is key in the rare case of acute intraoperative aortic IMH and dissection. In this case, the hematoma developed immediately following restoration of aortic flow and extended circumferentially from the distal graft anastomosis into the arch and descending aorta. The absence of an identifiable intimal flap created diagnostic uncertainty; however, the rate and volume of propagation and its proximity to the suture line raised concern for an evolving dissection. Several factors likely predisposed this patient to IMH formation, including longstanding hypertension, bicuspid aortopathy, and dilation of the ascending aorta. Mechanical stress at the distal anastomosis, disruption of the vasa vasorum, or occult intimal injury during graft manipulation are possible contributors. An additional notable feature of this case was the onset of severe right-ventricular (RV) failure during attempted separation from bypass after hemiarch replacement. In this patient, RV dysfunction was likely multifactorial, resulting from ischemia related to prolonged cardiopulmonary bypass and circulatory arrest, acute afterload elevation from hypoxemia-induced pulmonary hypertension, and impaired LV filling due to severe hypertrophy. The decision to initiate central VA-ECMO was crucial, providing both circulatory support and RV unloading. Despite the complexity of his intraoperative and postoperative course—including bleeding requiring re-

exploration, AKI requiring CRRT, and need for tracheostomy—the patient ultimately stabilized and was discharged. This case underscores the importance of rapid diagnostic assessment with TEE, early recognition of IMH progression, and flexibility in operative planning, including the timely initiation of mechanical circulatory support.

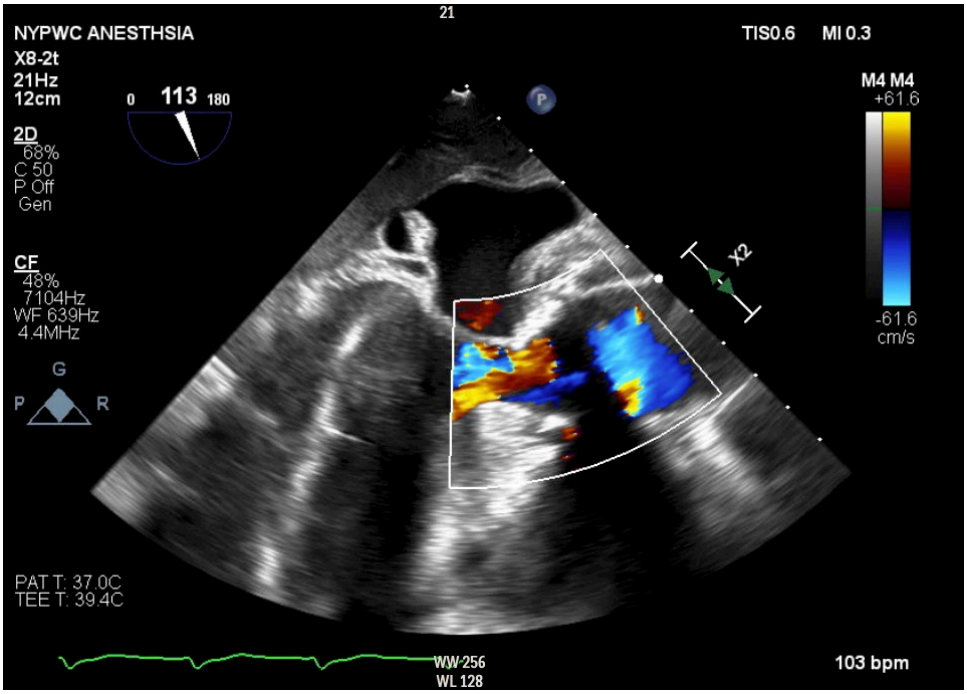
Images



Distal descending aorta SAX view



ME4C view



AV LAX view

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#FR 2 – Kiosk 10 – Poster 3

When the Shunt Takes a Detour: A Rare Case of Intact Ventriculoatrial Shunt Migration Causing Tamponade

Presenting Author: Joshua DeWalt, MD, Resident, University of Pittsburgh Medical Center

Introduction

Ventriculoatrial shunts (VAS) are a second-line treatment after ventriculoperitoneal shunts (VPS) for hydrocephalus when peritoneal drainage is not an option.1 However, VAS can have morbid complications to include: pneumothorax, tracheal injury, air embolism, catheter obstruction or migration, embolism, venous thrombosis, arrhythmia, and endocarditis.1 We present a unique case of remote right atrial perforation with cardiac tamponade from a VAS.

Case Presentation

A 50-year-old female with spina bifida presented with shortness of breath. As an infant, she had a VPS placed, which was converted to a VAS at age 37 due to ascites. Her VAS procedure was complicated by right ventricular perforation which required emergent sternotomy and repair. The patient presented to us with shortness of breath after multiple pericardiocentesis procedures for tamponade at an outside hospital. Her recent pericardiocentesis revealed 685 mL of clear aspirate which was positive for beta-2 transferrin. A CT chest showed the VAS catheter tip embedded in the right anterior inferior wall of the right atrium (Figure 1). A shuntogram revealed an intact VAS with contrast flowing directly into the pericardial space (Figure 2). Surgical planning began for redo sternotomy, shunt revision, and possible tricuspid valve repair vs replacement. In the operating room, TEE showed severe tricuspid valve (TV) regurgitation (Figure 3) and a small residual pericardial effusion (Figure 4). Intraoperatively, clear serous fluid was observed throughout the pericardium; the VAS tip was found perforating the right atrium near the diaphragm. The shunt was trimmed to the cavoatrial junction, and the perforation was sutured closed. The TV septal and posterior leaflets were severely restricted, and the subvalvular apparatus was very fibrotic. Because of this, valve replacement was chosen over repair, and a 29mm Edwards Mitris bioprosthetic valve was implanted. The remainder of her course was unremarkable, and her follow-up TTE showed no effusion or TV regurgitation.

Discussion

Myocardial perforation from VAS has been reported remotely in adults due to catheter disconnection and migration.2 To our knowledge, this is the only report in an adult of a remote myocardial perforation with an intact catheter. We hypothesize that accelerated age-related changes with spina bifida may have caused the VAS tip to move distally into the right atrium, and severe TV regurgitation added an anterolateral force leading to perforation over time. Proper positioning of the VAS tip is crucial to limit complications.1 Pericardial effusion leading to cardiac tamponade can cause severe hemodynamic instability and death. Prompt workup with echocardiography and pericardiocentesis can be helpful. Beta-2 transferrin assay can differentiate CSF from pericardial transudate or exudate. Ultimately, these cases require surgical correction where intraoperative TEE can aid in proper positioning of the VAS tip in the cavoatrial junction.3 VAS

complications require close coordination between cardiac surgery, neurosurgery, and anesthesiology for successful outcomes in these rare but potentially fatal presentations.

Images

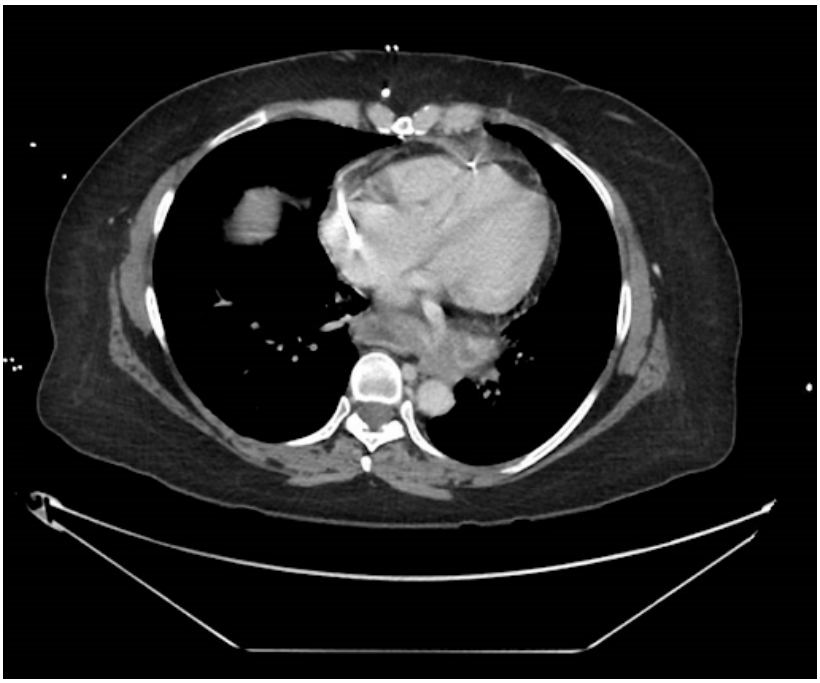


Figure 1: CT chest after pericardiocentesis showing the VAS traversing the anterior wall of the RA with the tip in the pericardial space.

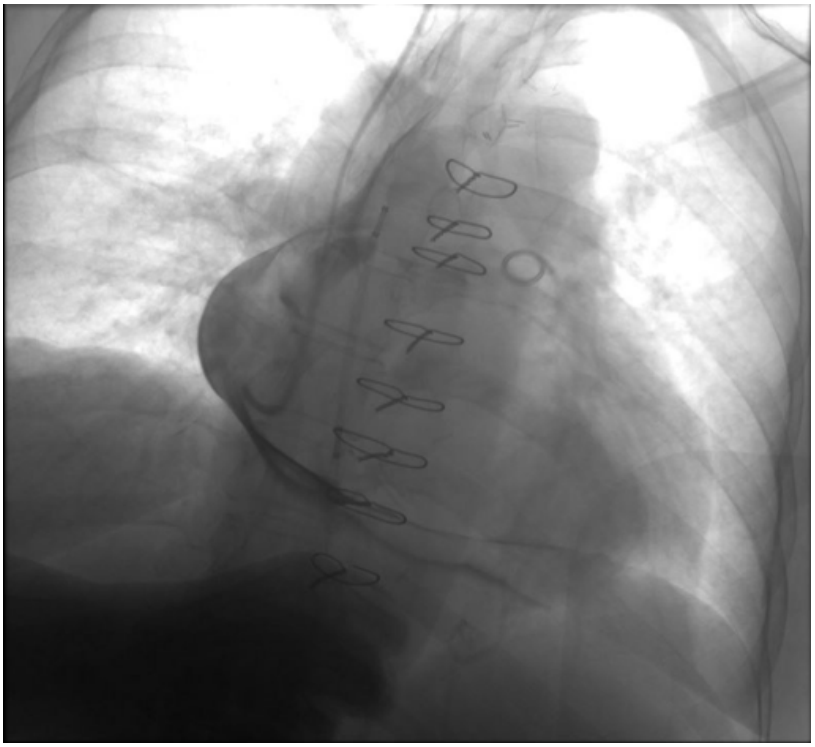


Figure 2: Contrast injection under fluoroscopy demonstrating contrast filling the pericardial space

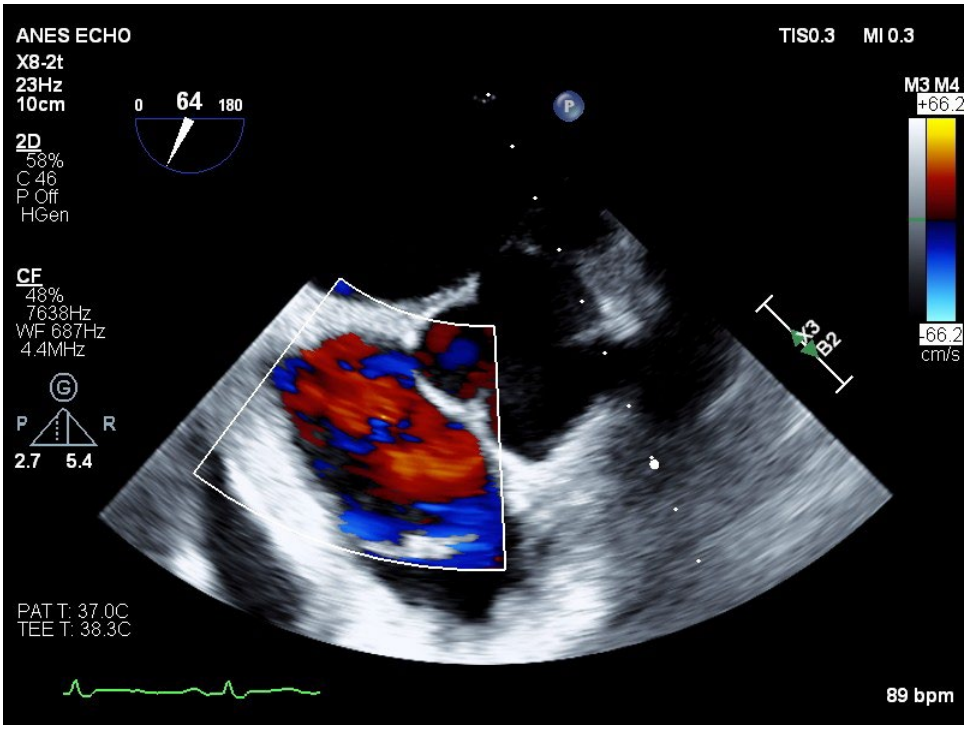


Figure 3: Modified bicaval view showing severe tricuspid regurgitation

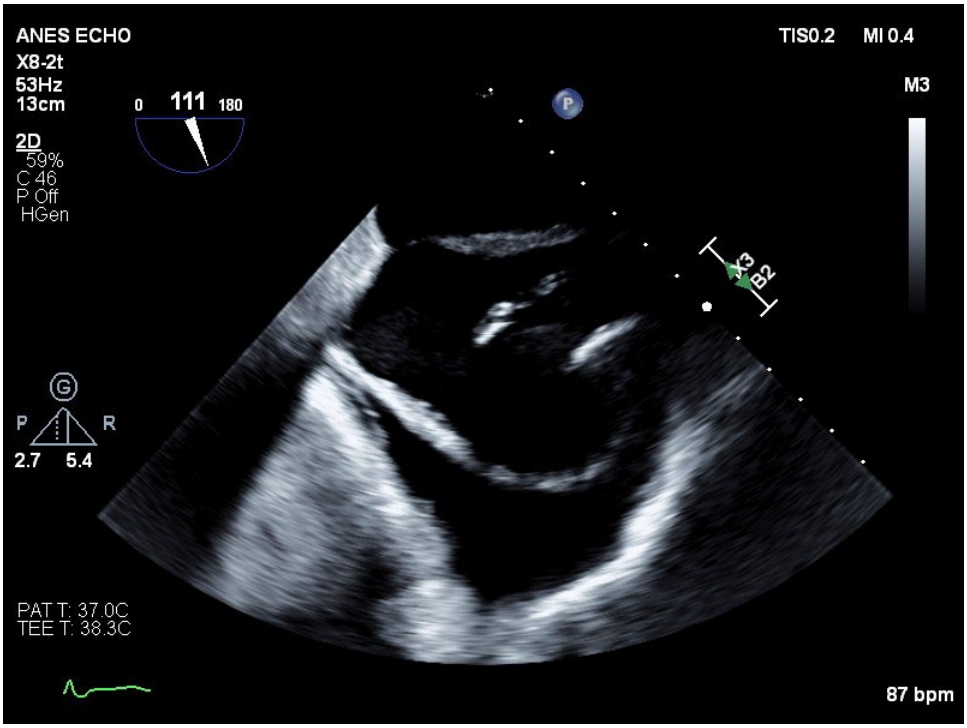


Figure 4: RA showing shunt and pericardial effusion

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#FR 2 – Kiosk 10 – Poster 5

Resection of a Norepinephrine-Secreting Paraganglioma in a Patient with Failing Fontan Physiology

Presenting Author: Benjamin Genovese, MD, Resident, University of Pittsburgh

Introduction

There is a growing population of adults with congenital heart disease (CHD) in the United States and many of these patients will need surgery and anesthesia throughout their lifetime. Cardiac anesthesiologists will continue to see a rise in the presentation of congenital heart disease patients to their operating rooms as the success of corrective procedures continues to improve. Herein we discuss a case of resection of a norepinephrine-secreting paraganglioma in a patient with failing Fontan physiology.

Case Presentation

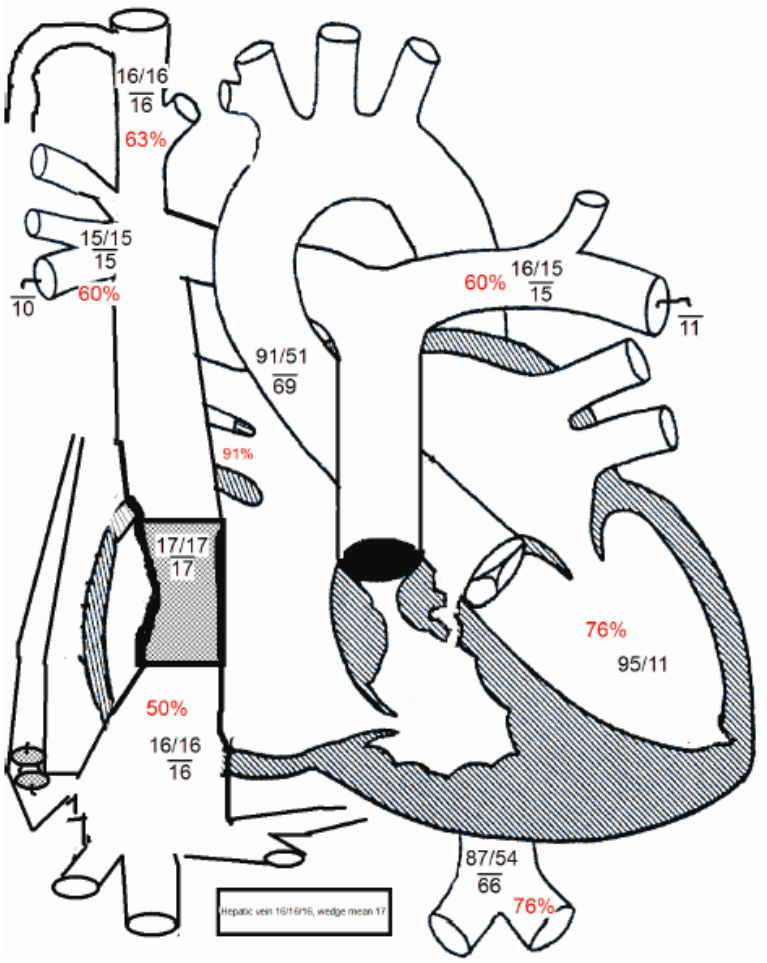
This was a 34-year-old male with a history of hypoplastic right heart syndrome (HRHS) and pulmonary atresia. He had undergone Fontan completion at 5 years of life. His chronically elevated Fontan pressures and venous hypertension led to liver disease and chronic hypoxia. Baseline oxygen saturation was 82-85% on room air and his Hgb 21g/dL. Doxazosin was started 14 days prior to surgery. The patient presented for open resection of an adrenal paraganglioma under general endotracheal anesthesia with neuromuscular relaxation and positive pressure ventilation. We preoxygenated with 100% O2 and a pre-induction arterial line was placed prior to an uneventful induction and intubation. A single lumen sheath was placed in the internal jugular to monitor Fontan pressures and a transesophageal echo (TEE) probe placed. Severe hypertension associated with manipulation of the mass was controlled with nitroprusside infusion. The mass was removed and approximately 700cc of blood loss had occurred and he was stabilized on epinephrine and vasopressin. After fascial closure, he developed refractory hypotension. Our differential diagnosis included hypovolemia, vasoplegia after NE tumor removal, LV dysfunction, and pulmonary hypertensive crisis. Fluid boluses were administered with minimal effect, and TEE was used to calculate stroke volume and cardiac index, which had not significantly changed from baseline. Mechanical ventilation was optimized and titrated near the patient's baseline according to arterial blood gas samples. The patient's Fontan pressure had begun to rise, ultimately tripling along with increases in peak airway pressure. A tense abdomen was noted, and we suspected bleeding had led to abdominal compartment syndrome. We began massive transfusion and the surgeon re-opened discovering 3 liters of blood in the abdomen and bleeding from large IVC collaterals. The patient ultimately underwent total nephrectomy and received large-volume product resuscitation prior to being taken to the ICU intubated with an open abdomen.

Discussion

Patients with cyanotic heart disease are at increased risk for development of pheochromocytoma/paraganglioma. Despite massive blood loss and the need for rapid, high-volume resuscitation our patient maintained his hemodynamics. We believe his single *left* ventricle provided significant physiologic advantage. This is supported by the CHD literature as HLHS is more likely to require transplantation and more likely to acquire failing Fontan physiology compared with HRHS. Refractory

hypotension after abdominal tumor resection in this patient presented a challenging differential diagnosis due to the combination of Fontan physiology in the setting of a hemodynamically significant neuroendocrine tumor with concealed intra-abdominal bleeding.

Images



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#FR 2 – Kiosk 10 – Poster 6

Intraoperative Management of Aortic Arch Graft Pseudoaneurysm Repair with Complex Vascular Anatomy

Presenting Author: Jonathan Hansen, D.O., Clinical Fellow - Adult Cardiothoracic Anesthesiology, Yale New Haven Hospital - Yale School of Medicine

Introduction

Ascending aortic dissection is a life-threatening condition with immediate and future surgical implications. Beyond expected issues relating to re-operation, our case presented challenges regarding cannulation strategies, IV access, and intraoperative findings which predict higher perioperative morbidity and mortality.

Case Presentation

Our patient is a 68 year old with a history of a prior DeBakey type 1 ascending aortic dissection, ulcerative colitis, hypothyroidism, and anemia who presented for repair of an expanding aortic arch graft pseudoaneurysm measuring 7.5 x 5.7 cm. Complicating their long-term course was a right subclavian graft infection with remaining subclavian artery supplied by a branch of the right vertebral artery and her right and left common carotid arteries connected to the proximal ascending aorta. She had a left carotid to subclavian bypass and zone 5 TEVAR with a right renal artery, superior mesenteric artery, and celiac artery supplied by true lumen while the left renal artery arose from false lumen. The inferior mesenteric artery was occluded, and the dissection flap extended to the proximal left common iliac artery with bilateral iliac artery aneurysms. The surgery team performed peripheral cannulation into the right femoral artery and vein through a cut down approach. Cardiopulmonary bypass was initiated, and we began cooling to 18 degrees Celsius prior to redo-sternotomy. Pre-bypass transesophageal echo showed moderate aortic valve regurgitation, due to limited movement of non-coronary and left coronary leaflets. The TEE also revealed the presence of graft material in ascending, though difficult to fully characterize the known pseudoaneurysm visualized on preoperative chest CT. Upon chest entry, we encountered the capsule of a large pseudoaneurysm on the aortic arch. The capsule was excised revealing a roughly quarter-size hole in the graft. It was repaired with sutures and a bovine pericardial patch with reinforcement. The left and non-coronary cusps were resected, and a 23 mm Inspiris Resilia bioprosthetic valve was implanted. Due to a tear in the LVOT at the level of the aortomitral junction, a bovine pericardial patch was placed over the area. Given the long bypass time and circulatory arrest, the patient developed mediastinal edema and coagulopathy requiring the chest to be closed several days later.

Discussion

Pseudoaneurysm and bleeding from a non-anastomotic site on a Dacron graft is a rare occurrence (Durmaz et el., 2023). While a pseudoaneurysm could be amenable to endovascular stent graft placement, our patient presents challenges with placement alone (Yamaguchi et al., 2013). The discovery of a hole in the graft encapsulated by a pseudoaneurysm may not be amenable to endovascular repair given the endovascular stent graft in the DTA and carotid artery branch points. As well, peripheral cannulation was

problematic due to persistent dissection extending to common iliac arteries, but axillary arterial cannulation was less than ideal given her anatomy. Peripheral cannulation and line placement required considerable thought, which proved successful.

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#FR 2 – Kiosk 10 – Poster 7

Diagnostic and Procedural Utility of Rescue Transesophageal Echocardiography in Cardiac Tamponade

Presenting Author: Samuel J. Fallon, MD, Resident Physician, Hospital of the University of Pennsylvania

Introduction

Transesophageal echocardiography (TEE) is a cornerstone of perioperative cardiac assessment but is less commonly employed as a real-time procedural adjunct during emergent hemodynamic collapse[1,2]. This case illustrates the pivotal role of rescue TEE in diagnosing pericardial tamponade and guiding image-directed drainage in a patient with acute circulatory failure.

Case Presentation

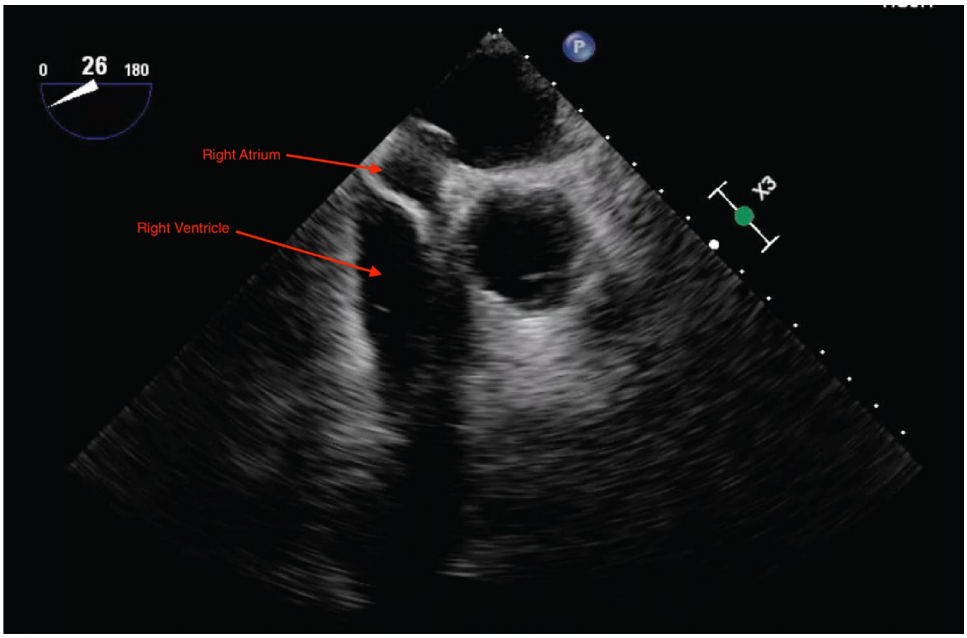
A 75-year-old man with severe multivessel coronary artery disease (occluded mid-LAD, 70–80% left main, 90% mid-LCx) presented with a suspected missed STEMI and managed medically after surgical revascularization was deferred. On hospital day three, he developed bradycardic pulseless electrical activity (PEA) arrest, requiring brief CPR. After return of spontaneous circulation, he remained hypotensive and hypoxemic with recurrent ST elevations and altered mental status, requiring emergent airway management. Transthoracic echocardiography demonstrated impaired ventricular function and a moderate pericardial effusion without tamponade. During cautious induction for intubation, the patient again experienced a near-arrest event. Rescue TEE demonstrated complete right atrial collapse throughout the cardiac cycle and a circumferential, homogeneously hypo-echoic pericardial effusion most prominent posteriorly, consistent with tamponade physiology. Due to limited transthoracic windows and poor visualization of a percutaneous wire, TEE with agitated saline contrast guided pericardial drain placement. Rather than advancing a wire, an angiocath was inserted into the suspected pericardial space to minimize myocardial injury and allow controlled confirmation of entry. Agitated saline injection demonstrated contrast confined to the pericardial sac without intracardiac passage, confirming accurate localization. Subsequent imaging visualized the drainage catheter within the pericardium. Aspiration led to rapid hemodynamic recovery, resolution of right atrial collapse, and elimination of vasopressor requirements.

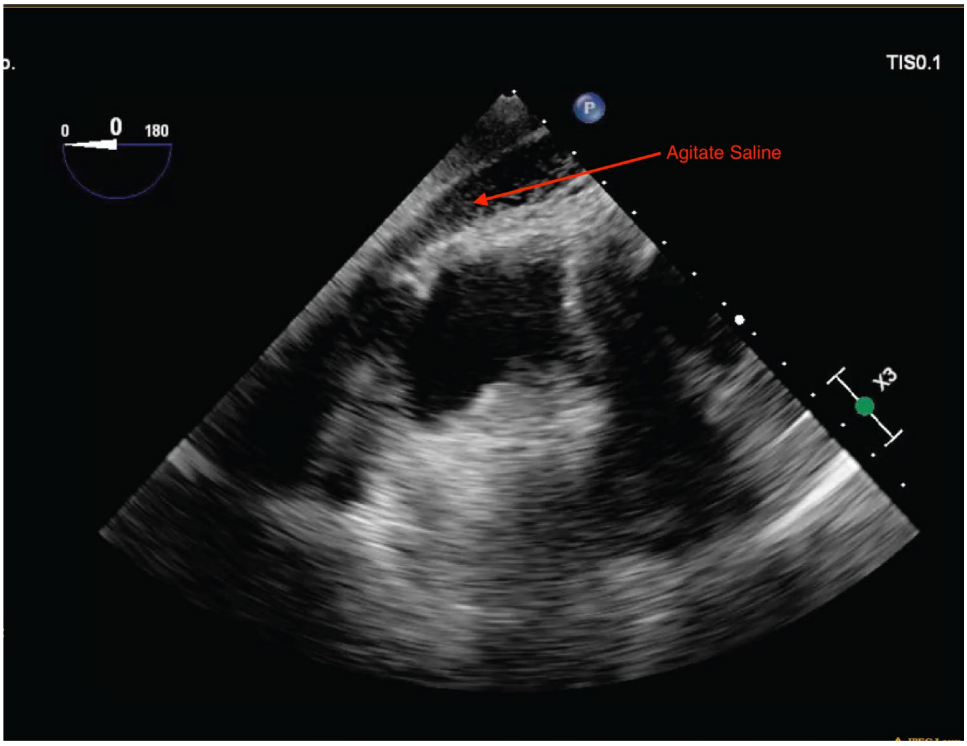
Discussion

The patient's recurrent instability was initially attributed to ischemic cardiogenic shock. In this context, TEE typically reveals regional wall motion abnormalities, depressed LV systolic function, and preserved right-sided filling. However, rescue TEE immediately demonstrated right-sided diastolic collapse and a large pericardial effusion—findings diagnostic of tamponade. This distinction fundamentally changed management, shifting from inotropic support to urgent decompression and volume expansion. TEE also provided procedural guidance. For this posterior effusion, an angiocath coupled with agitated saline contrast allowed safe pericardial access, confirming entry without fluoroscopy or wire visualization. TEE then verified catheter placement and monitored physiologic improvement following drainage. This case underscores the diagnostic and procedural value of TEE in acute hemodynamic collapse. By rapidly

distinguishing ischemic from obstructive shock and enabling real-time procedural guidance, TEE supports precise, physiology-driven interventions and improves outcomes in cardiac tamponade.

Images







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#FR 2 – Kiosk 11 – Poster 1

Unstable left-sided hemothorax during transcatheter edge-to-edge repair (TEER) with the MitraClip

Presenting Author: Melvin Mui, DO, Resident, Medstar Washington Hospital Center

Introduction

MitraClip procedures are typically performed on poor surgical candidates with severe symptomatic mitral regurgitation (MR). One of the possible complications is hemopericardium leading to tamponade physiology. While there has been a case report of a right-sided hemothorax causing tamponade physiology during a Mitraclip procedure, no record of left-sided bleeding has been published. In this case, we will discuss a patient on rivaroxaban who underwent a Mitraclip procedure complicated by a large left-sided unstable hemothorax requiring emergent chest tube placement.

Case Presentation

An 86-year-old man with atrial fibrillation on rivaroxaban, chronic HFrEF (EF 25–30%) status post biventricular ICD, and severe MR presented with worsening dyspnea and edema. Echocardiography confirmed severe MR (EROA 0.4 cm^2 with pulmonary venous flow reversal). Catheterization showed no significant CAD but was notable for markedly elevated filling pressures. Given his symptomatic decline, he was scheduled for a MitraClip procedure. The procedure was performed under general endotracheal anesthesia. Induction and intubation were uneventful. Transseptal access was obtained, heparin was administered, and two clips were deployed. 15 minutes after the second clip, he developed increasing vasopressor requirements. TEE revealed a small pericardial effusion and a new large left-sided pleural effusion. An emergent chest tube was placed with immediate drainage of 1 liter of blood. He received two units of packed red blood cells and fluid resuscitation with hemodynamic improvement. He was weaned off vasopressors and extubated later that evening in the CVICU. The chest tube was removed on POD 1 and ultimately being discharged on POD 3. At follow-up, he continues to do well.

Discussion

Transcatheter edge-to-edge repair (TEER) with the MitraClip is a safe and effective minimally invasive treatment for severe MR. The procedure is performed via femoral venous access with transseptal puncture, using real-time TEE guidance. Known complications include vascular injury, atrial septal defect, device detachment, residual or recurrent MR, cardiac perforation, and arrhythmias (3). Bleeding is one of the most common adverse events, in part because many TEER candidates require anticoagulation or antiplatelet therapy for comorbid conditions such as atrial fibrillation (1). Intraprocedural heparin further increases this risk. Severe bleeding requiring transfusion occurs in 0–17% of cases and independently predicts in-hospital mortality, though rates have declined with newer MitraClip generations and increased operator experience. Fewer than half of bleeding events are access-related; gastrointestinal bleeding associated with TEE is most common (2). Pericardial effusion or tamponade is uncommon, and hemothorax is even rarer. In this case, the cause of the patient's left-sided hemothorax was unclear but may have resulted from inadvertent trauma to the left pulmonary vein or left atrial appendage during transseptal access or device manipulation. Despite

this event, the patient recovered and was discharged. This case highlights hemothorax as a rare but important TEER complication and underscores the essential role of continuous TEE guidance for early detection and timely management of intraprocedural bleeding.

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#FR 2 – Kiosk 11 – Poster 2

Not Your Average Transplant: Concomitant Ascending Aortic Replacement and Bilateral Lung Transplantation

Presenting Author: Kevin Clark, MD, Fellow, University of Florida

Introduction

Patients who are listed for orthotopic lung transplantation often have significant cardiovascular comorbidities. While concomitant cardiac surgery at the time of lung transplantation is rare there is evidence to suggest similar rates of survival compared to those who exclusively undergo lung transplantation.[1,2] However, the combined surgery comes with its own unique set of complexities including management of mechanical circulatory support.

Case Presentation

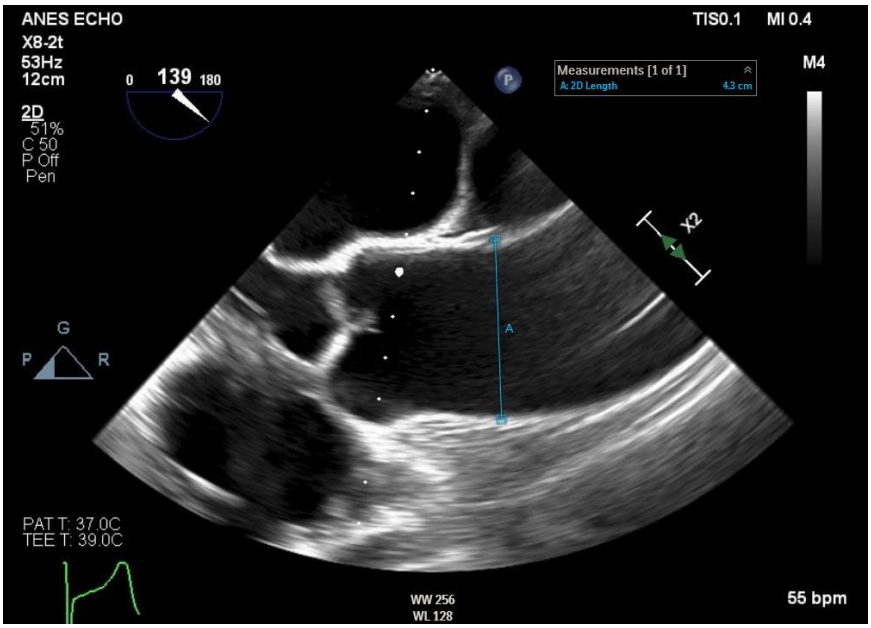
A 62-year-old-male with a history of end stage lung disease was admitted to the ICU for lung transplant evaluation. During the work-up he was found to have an ascending aortic aneurysm and moderate aortic insufficiency. Multispecialty discussion ensued and a combined procedure was determined to be the best course of action. Clamshell thoracotomy was performed by the transplant team with aortic and right atrial cannulation by the cardiac surgeons. Cardiopulmonary bypass (CPB) was initiated uneventfully and the patient was cooled to 32°C. Following cardioplegia and aortic cross-clamp, respectively, the ascending aorta was replaced without issue. Following inspection of the aortic root the annulus was dilated but the leaflets appeared normal. Decision was made to repair the sinotubular junction and forego valve replacement. The patient was then rewarmed to 36.5°C and bilateral lung transplantation ensued under CPB without issue. Following transplantation bypass was weaned uneventfully. Hemodynamically, the patient tolerated weaning from bypass without issue. The patient was transferred to the ICU on minimal vasopressor support.

Discussion

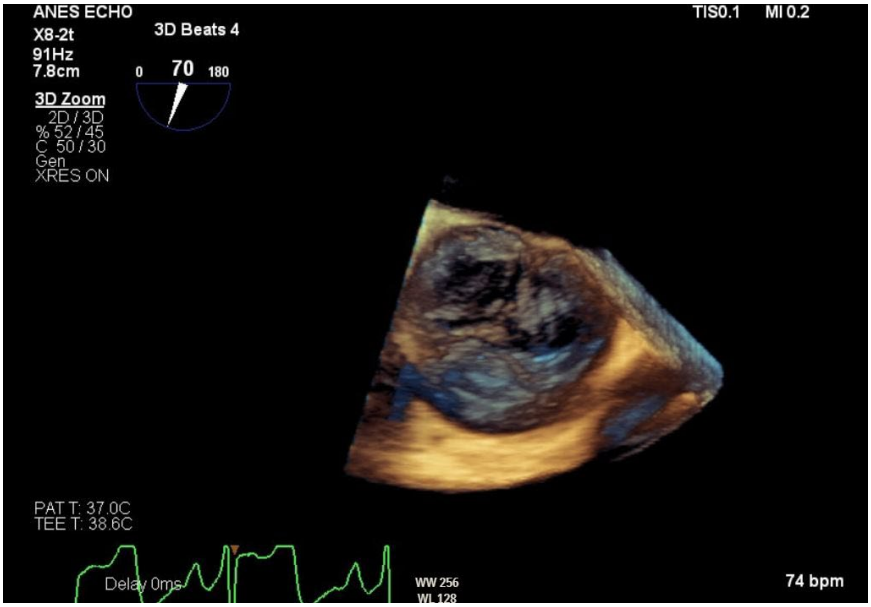
Cardiac surgery at the time of lung transplantation is a rarely performed operation. While sparse, current literature has shown similar survival outcomes for patients undergoing concomitant surgery.[1,2] However, these operations require careful planning and discussion, especially concerning the use of mechanical circulatory support. While aortic surgery requires the use of CPB, lung transplantation can be used with or without extracorporeal life support.[3] Lung transplantation can be performed with CPB, ECMO, or off-pump technique. While techniques often vary by institution and patient there lacks a consensus on which strategy is best.[3] The use of CPB allows for superior hemodynamic stability and oxygenation but exposes the patient to high doses of heparin, increased perioperative bleeding, and increased transfusion rates. In addition, there is evidence that CPB is associated with higher rates of primary graft dysfunction (PGD).[3] ECMO is widely considered a safer alternative and has emerged as the prominent mode of extracorporeal support, but remains associated with increased perioperative bleeding and PGD when compared to off-pump technique.[3] A multidisciplinary team decided to utilize CPB for the entirety of our patient's case. In this

instance, our patient was not considered to be at elevated risk for intraoperative bleeding given his baseline coagulation profile. In addition, our team felt use the of bypass would hasten the surgical procedure time while avoiding the technical challenges of transitioning from CPB to ECMO intraoperatively. Ultimately, our patient was weaned off bypass and resuscitated uneventfully and was transported to the ICU on minimal vasoactive support.

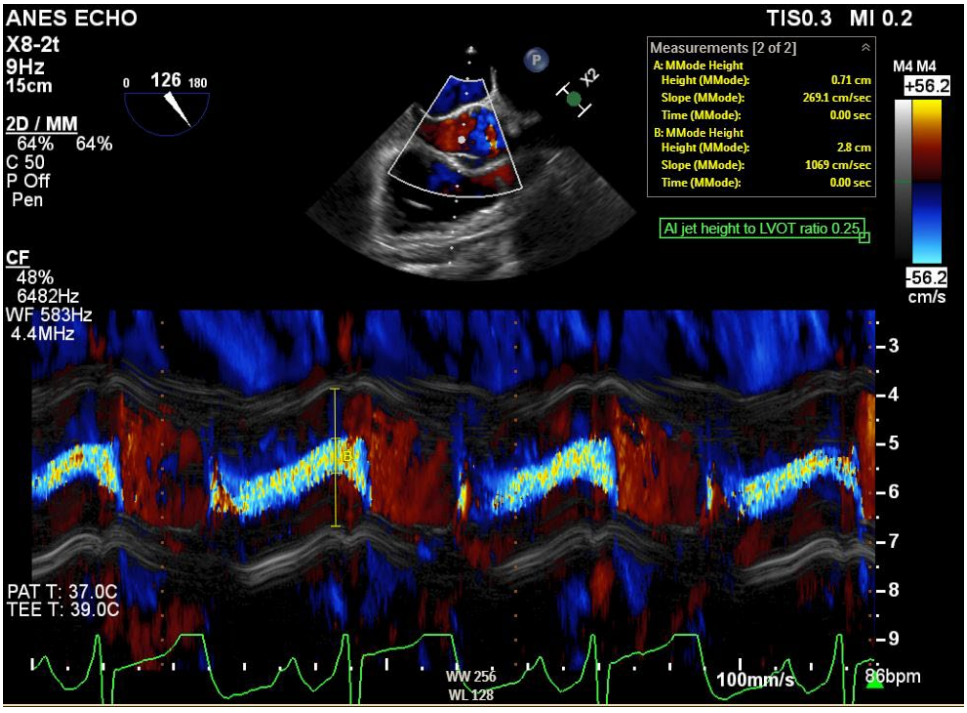
Images



Transesophageal echocardiography. Mid-esophageal long axis view. Ascending aortic aneurysm.



Transesophageal echocardiography. Mid-esophageal aortic valve short axis 3D image.



Transesophageal echocardiography. Color flow doppler and M-mode modality. Image depicting mild-moderate aortic insufficiency using AI jet height to LVOT ratio.

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#FR 2 – Kiosk 11 – Poster 3

Predicting persistent right heart dysfunction during redo Bentall procedure for complex congenital heart disease: potential of right ventricular longitudinal strain

Presenting Author: Sudhakar kinthala, Fellow, Fellow Adult Cardio Thoracic Anesthesiology, University of Rochester

Introduction

Right ventricular (RV) dysfunction following cardiac surgery is one of the determining factors for patients' outcome. RV function, assessment is challenging. This is partly because of traditional RV function assessment metrics may not be useful for cardiac surgery because of perioperative RV geometric changes. We are presenting a complex case with persistent RV dysfunction following cardiopulmonary bypass, for which RV global longitudinal strain (RV GLS) was helpful for preemptive treatment.

Case Presentation

70-year-old male with past history significant for tetralogy of Fallot status post repair (ventricular septal defect (VSD) patch closure, infundibular resection and pulmonary valvectomy) with residual small VSD and status post trans catheter pulmonary valve placement (at the age of 62). Significant medical issues include, morbid obesity , obstructive sleep apnea, hypertension, chronic kidney disease and peripheral vascular disease. Redo Bentall procedure with VSD closure was scheduled. Pre-operative echocardiography showed normal LV function (LVEF 63%) with low normal RV dysfunction (RVEF 41%, mildly enlarged RV), significantly depressed GLS -8.4%, mild tricuspid regurgitation, right ventricular systolic pressure 50.2 mmHg with right atrial pressure 8 mmHg. Mean Pulmonic valve gradient 4 mmHg. Mild aortic regurgitation with severely dilated aortic root (5.1 cm) and markedly dilated ascending aorta (4.0 cm). Surgical approach was determined with multidisciplinary discussion including pediatric cardiac surgery, adult cardiac surgery, and anesthesia team. General anesthesia with endotracheal tube, arterial line, central venous line, pulmonary artery (PA) catheter, and intra operative trans esophageal echocardiogram monitoring was performed. Separation from the bypass was challenging because of persistent RV dysfunction requiring maximum pharmacologic support with epinephrine, norepinephrine, and vasopressin. Epoprostenol started at the early stage to improve PA pressure. Patient was transported to ICU using a transport ventilator for the concern for RV function. By POD#2 Inotropes weaned off , and patient was extubated.

Discussion

Our successful patient care can be attributed to 2 factors: 1. Preoperative multidisciplinary approach and planing. 2. Comprehensive echocardiographic assessment including RV function assessment using 3D and GLS. In preoperative echocardiography, traditional RV indices were low normal, while GLS was significantly depressed (TABLE 1& FIGURE 1). As reported previously, GLS is more sensitive in detecting RV dysfunction compared with traditional echocardiography indices such as RVEF, RV fraction area change (RV FAC), or tricuspid annular plane systolic excursion (TAPSE).[2,3] RV geometry change after cardiopulmonary bypass might not be fully assessed using RVFAC or TAPSE. Also, as in LV, LS is more sensitive than EF for assessing

the function. In this presented case, we started epoprostenol relatively early because baseline depressed RV GLS might be the sign of RV low reserve, which might be improved with adjusting afterload. Although comprehensive assessment is critical for RV function assessment, LS indices have potential additive value to conventional echocardiographic parameters in the perioperative setting of cardiac surgery.[3]

Images

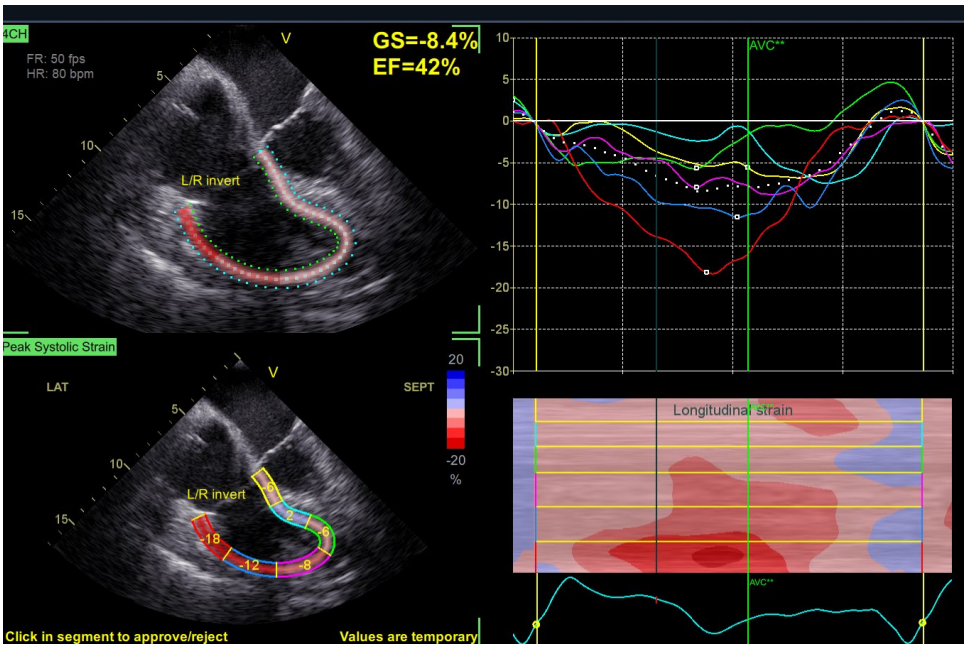


FIGURE: Pre- and post-procedure biventricular function

	LVEF (%)	LVEDV (ml)	LVESV (ml)	LVSV (ml)	CO (LPM)	LV GLS	RVEF (%)	RVEDV (ml)	RVESV (ml)	RVFAC (%)	TAPSE (mm)	RV GLS
Baseline	48	137	71	66	5.3	-9.3	42	205	121	36.4	17	-8.4
Post chest closure	46	91	50	42	3.3	-8.3	34	155	102	36.4	11	-9.2

Abbreviations:
LVEF: left ventricular ejection fraction, LVEDV: left ventricular end-diastolic volume, LVESV:
left ventricular end-systolic volume, LVSV: left ventricular stroke volume, CO: cardiac output, GLS: global longitudinal strain
RVEF: right ventricular ejection fraction, RVEDV: right ventricular end-diastolic volume, RVESV:
right ventricular end-systolic volume, RVFAC: right ventricular fraction area change, TAPSE: Tricuspid Annular Plane Systolic Excursion

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#FR 2 – Kiosk 11 – Poster 4

Catastrophic Complications of Percutaneous Left Ventricular Assist Device Placement

Presenting Author: Conner J. Elliott, M.D., Fellow, University of Louisville

Introduction

Cardiogenic shock is a devastating complication of acute MI that can occur in up to 10% of patients and is associated with a mortality rate as high as 50%. Management of this pathology is dependent on early reperfusion, but afterwards patients will often require aggressive correction of hypotension and volume status, ventilatory support, and inotropic support. In patients refractory to these interventions, mechanical circulatory support is an invasive option for supportive care that carries significant risk. We present a patient that suffered multiple complications from an Impella placement during the operative period.

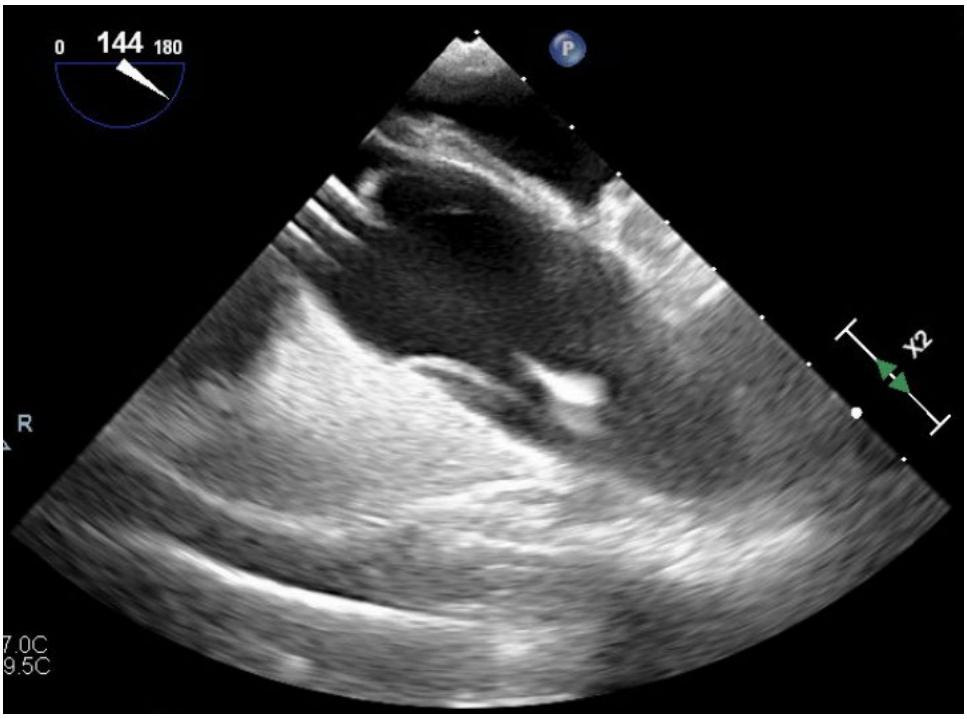
Case Presentation

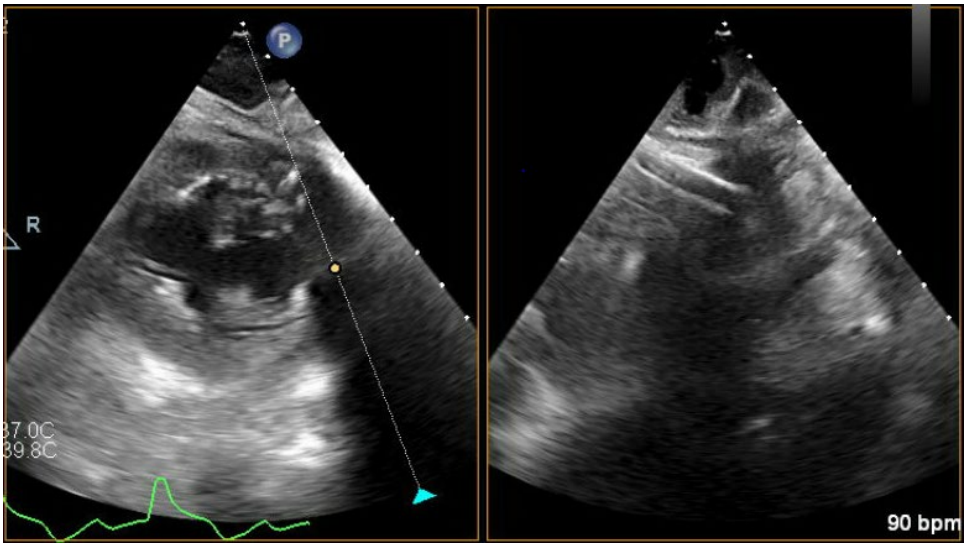
A 66-year-old man with no known cardiac history presented from an outside hospital after cardiac arrest secondary to acute MI. He underwent left heart catheterization with PCI and placement of an Impella CP for cardiogenic shock. Cardiac output remained insufficient with Impella CP and inotropic support and he was scheduled for exchange to Impella 5.5. The patient was transported to the OR and was maintained under general anesthesia with TEE for monitoring. After accessing the axillary artery, the femoral Impella CP was removed. The surgeon attempted to place a wire across the aortic valve into the left ventricle but there was significant difficulty in doing so and required the assistance of vascular surgery. During this process, an irregularity was noted in the ascending aorta on TEE that was determined to be a type A dissection. The wire was then attempted to be placed once more and was able to be placed in the true lumen with the tip in the left ventricle. The Impella 5.5 was advanced into the LV but was not well visualized with TEE despite multiple attempts to identify the inflow orifice and distance from the aortic valve annulus. There was also new moderate-to-severe eccentric aortic insufficiency around the device. The Impella 5.5 was turned on but was unable to flow at a level greater than P-2. The device placement was further examined via TEE and eventually it was determined that the tip of the Impella had penetrated into the inferolateral LV wall with concern for its tip being in the pericardium. Shortly after, the patient went into cardiac arrest and expired.

Discussion

Ventricular assist devices have a unique role in treatment of cardiogenic shock, but are not without significant risk. Impella devices carry a variety of potential risks, some of which include bleeding or hemolysis, thrombocytopenia, stroke, limb ischemia, and vascular or cardiac injury. Of these risks, some of the most serious are the potential injury of the aorta or the left ventricle, both of which were demonstrated in this case. TEE guidance is integral for placement of these devices to help ensure proper positioning, but it does not eliminate risk. In the event that such injuries do occur, TEE can be used to rapidly identify and diagnose the issue. However, these patients are invariably critically ill and it may be impossible to repair the issue promptly enough to prevent further morbidity or mortality. Our case serves as a reminder to respect the potential risks in these cases.

Images





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#FR 2 – Kiosk 11 – Poster 5

Stellate Ganglion Block for Refractory Ventricular Tachycardia

Presenting Author: Jared Olson, D.O., Anesthesiology Resident, University of Missouri

Introduction

Ventricular tachycardia (VT) is a deadly rhythm that can occur from a number of factors in the heart, with the most common being ischemia. If severe, the VT can be refractory to medical management and become recurrent. If unable to treat with medical management, then definitive treatment needs to be sought emergently. The gold standard in treating patients with refractory ventricular tachycardia is with catheter ablation in the electrophysiology lab. Due to the complexities of the foci of VT, mapping for the ablation requires mapping both the right ventricle and left ventricle to determine where the foci are being initiated. During this time, it is helpful for the patient to be in a narrow complex rhythm. The stellate ganglion block is a well-known treatment for refractory ventricular tachycardia. It can be utilized after failure of medical management to help stabilize a patient until definitive treatment can be obtained. This is a case of using a perioperative stellate ganglion block to stabilize a patient during a VT ablation to aid in mapping.

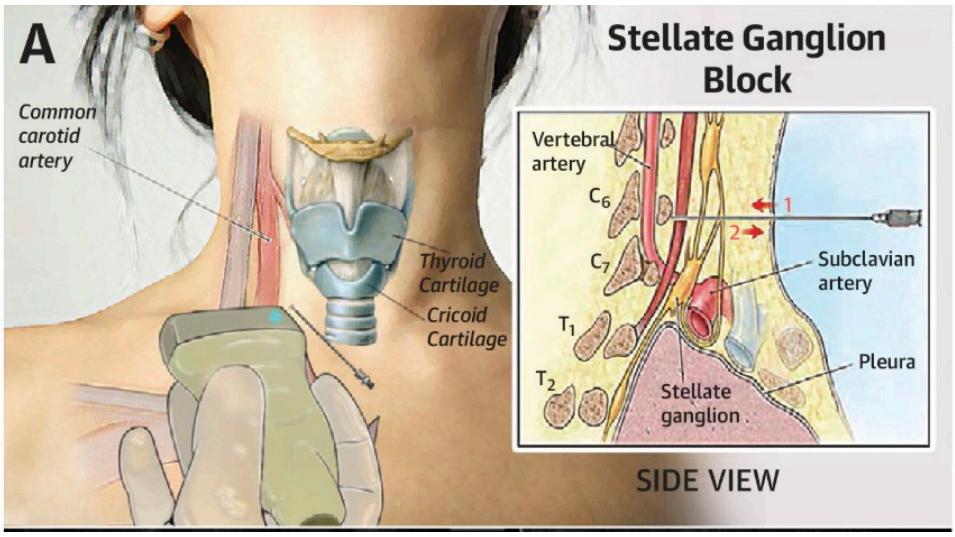
Case Presentation

The patient is a 70 year old male with a past medical history of coronary artery disease and hypertension. He presented initially with urinary retention and subsequently developed ventricular tachycardia while his bladder was being drained. He was taken for a left heart catheterization, which showed chronic occlusion of his RCA with collateral flow, and 95% ostial and mid stenosis of his LAD that had a stent placed. The patient developed VT again and was taken for an ablation, which was found to be PVC induced. The ablation was completed without any PVC signals. On the second day after the initial ablation, the patient developed VT again, requiring multiple shocks and initiation of lidocaine and esmolol without benefit. The patient was intubated and sedated and taken emergently to the electrophysiology lab for ablation. While under general anesthesia, mapping was not able to be performed due to frequent runs of ventricular tachycardia that were refractory to medications. Consent was obtained from the brother, who was next of kin, and an ultrasound guided left sided stellate ganglion block was performed using 0.25% bupivacaine. The patient was able to continue with the procedure without recurrence of his VT for the next 8 hours.

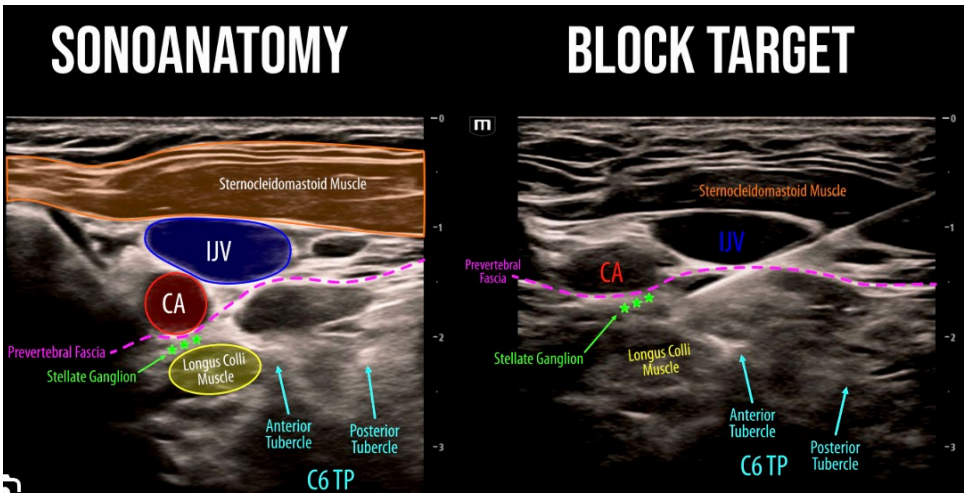
Discussion

While not widely used by cardiac anesthesiologists, the stellate ganglion block is an easy and effective block for refractory ventricular tachycardia. With the use of ultrasound, it can be safely placed and allows for stabilization of the patient. This case highlights the usefulness of an intraoperative stellate ganglion block for a VT ablation in patient management.

Images



Surface anatomy and representation of stellate ganglion block



Ultrasound imaging for stellate ganglion block

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#FR 2 – Kiosk 11 – Poster 6

Baroreflex-Mediated Hyperdynamic Response Leading to Hemodynamic Collapse on Cardiopulmonary Bypass Initiation

Presenting Author: Reginald Manuel, M.D., Resident, TidalHealth

Introduction

Before aortic cannulation for cardiopulmonary bypass (CPB), vasodilators are often used to reduce systemic vascular resistance (SVR) and systolic blood pressure (SBP) to theoretically minimize the risk of aortic injury. In patients with long-standing uncontrolled HTN, the baroreflex setpoint may shift upward. When SVR is lowered, these patients can respond with marked tachycardia and increased cardiac output (CO). If the native CO exceeds the maximal CPB flow, initiation of CPB can lead to abrupt hemodynamic collapse. This scenario, while not rare, carries high educational value for daily cardiac anesthesia practice.

Case Presentation

A 34-year-old man (185 cm, 173 kg, BMI 50) with a 7.3 cm aortic root aneurysm, 1.2 cm coarctation of the aorta (CoA) at the proximal descending aorta, life-long uncontrolled HTN, and CKD underwent staged repair: initial balloon angioplasty and TEVAR for CoA, followed two weeks later by valve-sparing aortic root and ascending aortic replacement. Preoperatively: HR 63, BP 163/91 on maximal antihypertensive therapy. Induction and line placement (radial, central, PA catheter) were uneventful. Baseline hemodynamics: CO 7.4, CI 2.7, RAP 18, PAP 37/21, SVR 1012. TEE showed concentric LV hypertrophy, preserved biventricular function with LVEF 60%, and a salvageable bicuspid aortic valve. Despite adequate anesthesia (sevoflurane 1.2 MAC, BIS 40s, fentanyl 750 µg), SBP remained in the 150s. As aortic cannulation approached, clevidipine was titrated to maximum, nitroglycerin added, and the patient placed in reverse Trendelenburg, lowering SBP to the 90s. During this period, HR increased gradually to the 100s, consistent with baroreflex activation. CO increased to 12.8, with a retrospectively calculated SVR of 308. At CPB initiation with its maximum flow at 7.5 L, MAP fell to the 20s. CPB was immediately stopped, volume returned to the patient, and native heart ejection resumed, restoring MAP >65. After 15 minutes of SVR optimization with norepinephrine and vasopressin, CPB was successfully reestablished (MAP 60-70s, flow 7.5 L, norepinephrine continued). The remainder of the case was uneventful. Post-CPB TEE findings were unchanged.

Discussion

In chronic HTN, the baroreflex setpoint shifts upwards but often becomes less sensitive, leading to a minimal HR response to BP changes, especially under general anesthesia. Some patients, however, retain baroreflex sensitivity with this upward shift. This case illustrates the danger of excessive vasodilation in such patients. The reduction in SVR triggered reflex tachycardia (HR 60s to 100s) and hyperdynamic physiology (CO 7.4 to 12.8). When the CPB could not match native CO, hemodynamics collapsed precipitously. In retrospect, the baroreflex should have been blunted using a short-acting β1 blocker during vasodilator titration. In patients with uncontrolled HTN and a shifted but sensitive baroreflex, vasodilators without β1 blockade can unnecessarily overly decrease SVR to achieve a target BP and provoke a hyperdynamic state

that overwhelms CPB capacity. Cardiac anesthesiologists should calculate and monitor hemodynamic parameters before CPB initiation as carefully as they do when coming off bypass to avoid collapse from an “overshoot” in native CO.

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#FR 2 – Kiosk 11 – Poster 7

Air Embolism After Transseptal Puncture: An Anesthetic Blind Spot

Presenting Author: Marc AbiKaram, MD MS, Resident, Saint Louis University

Introduction

The FDA's 2025 Early Alert regarding air embolism risks associated with left atrial appendage occlusion procedures highlights important concerns related to anesthetic technique. Despite advances in minimally invasive cardiac interventions and a shift toward conscious sedation, recent reports of catastrophic air embolism underscore physiologic vulnerabilities when negative intrathoracic pressures occur during spontaneous ventilation, particularly during transseptal procedures. Anesthesiologists play a key role in mitigating these risks and promote safer practice in the expanding field of structural heart interventions

Case Presentation

A 76-year-old male with long-standing nonvalvular atrial fibrillation, prior peptic ulcer bleeding, and a CHA_2DS_2-VASc score of 4 was referred for percutaneous appendage occlusion due to intolerance of oral anticoagulation. He underwent the procedure under moderate conscious sedation with intermittent dosing of midazolam and fentanyl. Baseline hemodynamics were stable, and intraprocedural anticoagulation achieved a target activated clotting time of >250 seconds. Following successful transseptal puncture, a 14-F delivery sheath was advanced into the left atrium. During device preparation, the delivery system was flushed per protocol; however, immediately after device insertion into the loader, the patient—previously awake and conversant—reported acute lightheadedness and chest pressure, followed by abrupt unresponsiveness, hypotension, and cardiac arrest. During CPR, TEE revealed bursts of echogenic air entering the LVOT. Unfortunately, after several days in the intensive care unit, the patient passed away.

Discussion

For percutaneous heart procedures involving transseptal puncture to access the left atrium, the use of conscious or deep sedation is associated with an approximately threefold increased risk of negative left atrial pressure and subsequent air entrainment. A recent study reported that over a third of patients undergoing transseptal puncture during sedation with spontaneous ventilation had inspiration-induced negative left atrial pressure (< 0 mmHg) measured upon entry to the left atrium, predisposing them to a high risk of air embolus. As minimally invasive heart interventions expand, anesthesiologists remain uniquely positioned to identify physiologic and procedural hazards before patient harm occurs. Mitigating the serious risks reported with LAAO or heart procedures requiring transseptal puncture requires both system level action plans along with close collaboration between anesthesiology, nursing, and cardiology teams to promote safety in this evolving area of cardiovascular care.

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#FR 2 – Kiosk 12 – Poster 1

The 6-Centimeter Surprise: Aneurysm, Alignment, and the Art of TEE

Presenting Author: Aditya Seshadri, University of Rochester

Introduction

Pulmonary artery aneurysms (PAAs) are exceptionally rare, with only 8 cases identified among more than 100,000 autopsies in early studies. Most PAAs involve the main pulmonary artery, where the normal upper limit of diameter on CT is 29 mm[1]. Pulmonic stenosis is one recognized congenital cause of PAA due to chronic post-stenotic dilation. However, the pulmonic valve can be difficult to image with TEE, which is an added challenge in the busy peribypass period.

Case Presentation

A 66-year-old woman with long-standing congenital pulmonic stenosis and a 6.3-cm main/left PAA on CT chest (Figure 1) was referred for repair. Despite the aneurysm size, her right heart catheterization showed normal PA pressures. Since no updated preoperative echocardiogram was available, the surgical team relied on intraoperative TEE to evaluate the pulmonic valve. After an uncomplicated induction and intubation, a radial arterial line, central venous catheter, and TEE probe were placed. Initial standard upper and mid esophageal views provided limited visualization of the pulmonic valve due to poor acoustic windows and doppler alignment. An off-axis transgastric (TG) RV inflow view gave excellent 2D imaging with reliable doppler alignment. The PV was thickened but opened well, with a peak gradient of 16.5 mmHg (Figure 2). These findings were confirmed by direct visualization in the operative field, and the valve was preserved. Surgical repair involved end-to-end anastomosis of the left and right PA with a straight Hemashield graft, followed by an end-to-side anastomosis of the main PA to the graft. On TEE, the main PA anastomosis appeared widely patent without obvious flow acceleration. In contrast, color doppler at the branch anastomoses showed areas of flow acceleration at the LPA and RPA takeoff, although doppler alignment was not adequate to obtain reliable velocity or gradient measurements (Figure 3). To clarify these findings, the surgeon directly transduced pressures in all three PA branches while temporarily off bypass. Mean pressures were in the low 20s, confirming no significant gradients across any of the anastomoses. The patient was extubated in the ICU several hours later and was discharged home on POD5.

Discussion

This case illustrates a rare combination of a large PAA with mild pulmonic stenosis, where determining the true severity of obstruction became central to surgical management. PS is considered severe when peak velocity is >4m/s or peak gradient is >64 mmHg, which helps distinguish clinically significant obstruction from mild disease present in our patient2. Although the PV can be seen in upper and mid esophageal views as well as TG views, the anterior location can make assessment difficult due to far-field positioning, acoustic shadowing, and doppler misalignment, making off-axis imaging particularly helpful to provide clinical answers. Pulmonic valve pathology is encountered infrequently in adult cardiac anesthesia, but familiarity with these imaging techniques becomes important when preoperative evaluation is limited or

incomplete. Lastly, this case highlights how close collaboration with our surgical colleagues is essential, as direct invasive pressure measurements complemented TEE findings to guide intraoperative decision-making and ensure patient safety.

Images



Figure 1
Contrast-enhanced sagittal CT demonstrating a giant main PAA measuring 6.3 cm in maximal diameter, with extension into the left pulmonary artery

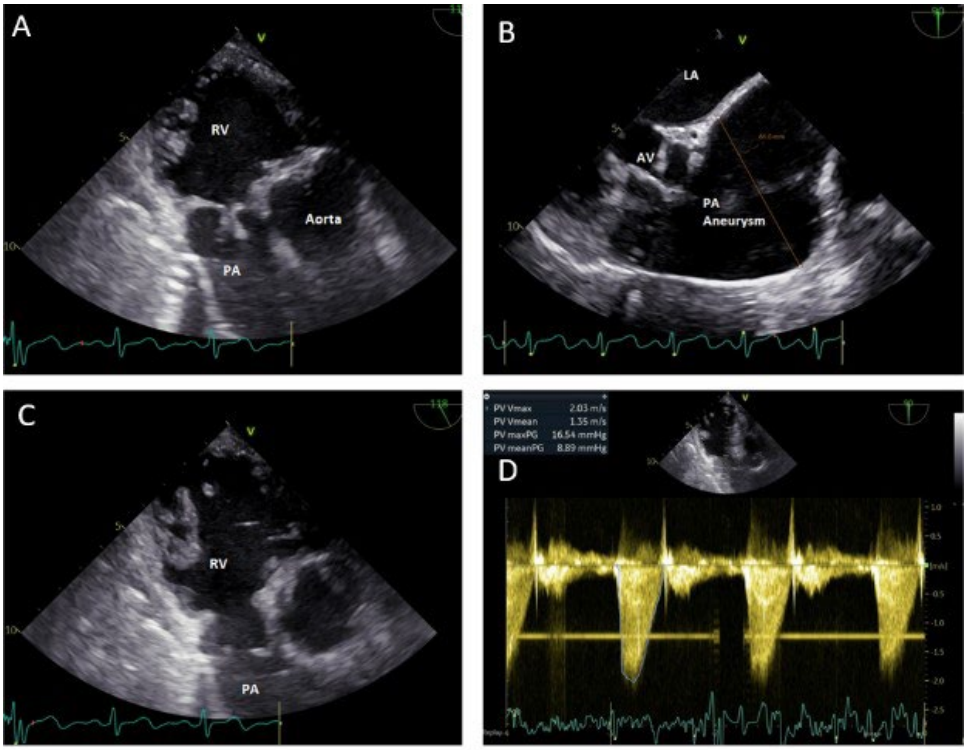


Figure 2
A: Off axis DG RV inflow view showing thickened pulmonic valve in diastole
B: Marked enlarged PAA measured at 61mm
C: Off axis DG RV inflow view showing pulmonic valve in systole that opens well, unlikely to have significant stenosis
D: Peak velocity and gradient across PV, demonstrating mild pulmonic stenosis

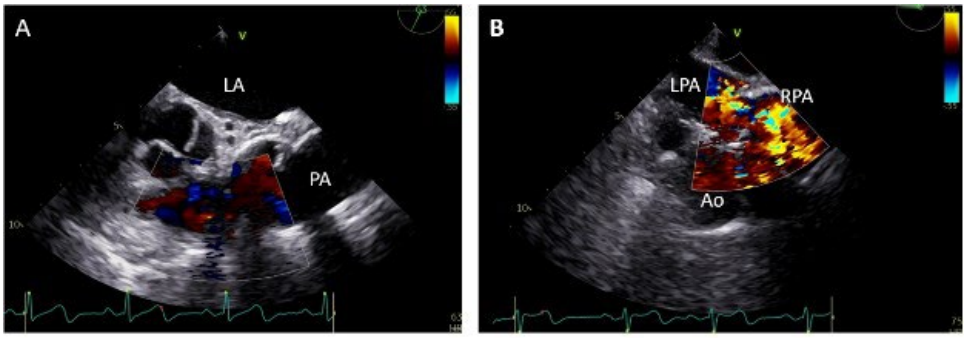


Figure 3
A: Off axis ME RV inflow outflow view showing no flow acceleration across the main PA anastomosis
B: ME ascending aorta SAX view showing flow acceleration across the LPA and RPA graft anastomosis site

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#FR 2 – Kiosk 12 – Poster 2

Open Right Atrial Thrombectomy for a Patient in Sickle Cell Crisis

Presenting Author: Rudresh H. Patel, DO, Resident Physician, Medical College of Georgia Department of Anesthesiology and Perioperative Medicine

Introduction

Sickle cell disease (SCD) presents a unique challenge in the setting of cardiac surgery with cardiopulmonary bypass (CPB). This patient population requires diligent preoperative optimization and intraoperative care to avoid stressors which could precipitate sickling. Here, we present a case of a young patient in sickle cell crisis requiring open excision of a right atrial mass with CPB.

Case Presentation

A 21-year-old African American female with a past medical history of SCD, seizure disorder, asthma, and depression initially presented to an outside hospital with complaints of generalized pain and shortness of breath. She was admitted for management of sickle cell crisis and acute chest syndrome. A transthoracic echocardiogram demonstrated a large, mobile mass measuring 2.4x3.4cm in the right atrium. She was started on a heparin drip due to concerns for a thrombus related to her right subclavian port-a-cath and transferred to our center. Cardiothoracic surgery and cardiology were consulted who had initially planned for an aspiration thrombectomy. However, the procedure was aborted after intraprocedural intracardiac echocardiography showed the mass to be too large and attached to the right atrial wall via a stalk. The patient was scheduled for an open right atrial mass excision with CPB. Preoperatively, the patient underwent aggressive blood transfusions to achieve a goal hemoglobin of 10 g/dL and hemoglobin S (HbS)< 30% following multidisciplinary discussions between the hematology, anesthesiology, cardiology, and cardiothoracic surgery teams. She was also diuresed gently to prevent volume overload from blood transfusions. The patient underwent standard controlled induction of general anesthesia and received a post-induction arterial line. A left internal jugular central venous catheter was placed carefully under transesophageal echocardiography guidance, given the location of the mass. The procedure was performed on CPB without myocardial arrest, although warm crystalloid cardioplegia was available should arrest be required. Venous cannulation was obtained through the right femoral vein and superior vena cava, as well as standard aortic cannulation. Intraoperatively, the patient received exchange transfusions to maintain HbS< 30%. The patient tolerated the procedure well without any immediate complications. Postoperatively, multimodal analgesia and patient-controlled analgesia were utilized to ensure adequate pain control. The postoperative course was uncomplicated, and she was discharged on postoperative day four.

Discussion

Patients with SCD have increased risk for surgical and anesthetic complications and require cautious management to avoid stressors which may precipitate vaso-occlusive episodes, including oxygenation, hydration, temperature, and analgesia (1). Further special considerations must be taken during cardiopulmonary bypass to decrease the risk of sickling, which includes the use of exchange transfusions,

warm crystalloid cardioplegia, and hyperoxygenation of priming fluid (2). There remains no clear consensus in the literature on whether conservative or aggressive preoperative blood transfusion should be pursued, with a 2016 Cochrane report finding insufficient evidence among published randomized trials (3).

Images

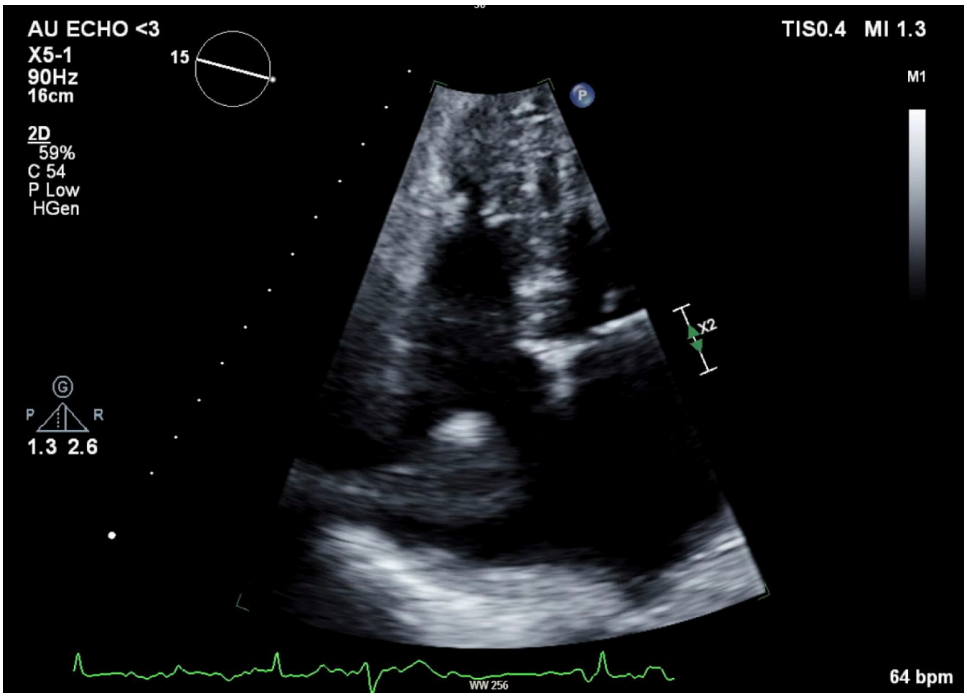


Figure Legend: 1) Transthoracic echocardiogram demonstrating a large, irregularly shaped, mobile mass in the right atrium

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#FR 2 – Kiosk 12 – Poster 3

Unmasking the Dominant Shock: Echo Guided Rescue in a Patient Bound for the Operating Room

Presenting Author: Alexander Zazaian, M.D., Resident, Wayne State University Department of Anesthesiology

Introduction

Early hemodynamic collapse in the sickest septic patients often conceals a mixed shock physiology, complicating efforts to determine the principal cause of their instability. Early bedside echocardiography can improve outcomes by revealing mechanical causes not initially evident on exam (1). Cardiac tamponade is a well-described mimic of distributive septic shock, sharing features including tachycardia, hypotension, and rising lactate (2). Systemic inflammatory illness can also produce serous effusions that become hemodynamically significant even without direct infection, further obscuring diagnosis (3). This case highlights a patient with presumed intra-abdominal sepsis whose refractory shock was ultimately due to tamponade physiology, underscoring the importance of physiology-guided and echocardiographically-informed decision in managing mixed shock states.

Case Presentation

A 36-year-old female with HFpEF, DM, HTN, and seizure disorder presented with abdominal pain, diarrhea, and a reported seizure. She was febrile, hypotensive, tachycardic, and had an elevated WBC count and lactate, prompting fluids, empiric antibiotics, and escalation of inotropic and vasopressor support. CT of abdomen showed colitis, a small pericardial effusion, a right pleural effusion, and two pelvic collections with one concerning for abscess. Surgical team planned emergent laparotomy for source control and presumed septic shock. Immediately prior to transport, significantly increasing vasopressor needs prompted bedside echocardiography, which revealed a large pericardial effusion with right-sided chamber collapse consistent with tamponade physiology. Surgery was deferred, and emergent pericardiocentesis yielded 350 mL of serous fluid with rapid improvement in lactate and reduction of vasopressor requirements. Pericardial fluid studies were negative, and repeat imaging later demonstrated no drainable abdominal source. She continued to improve clinically and was ultimately discharged home in stable condition.

Discussion

This case demonstrates the challenge of evaluating a patient in refractory shock, especially when both sepsis and obstructive physiology coexist. The patient appeared to have profound septic shock from a presumed pelvic abscess, yet her rapid decline despite fluids, antibiotics, and multiple vasopressors suggested a second process. Tamponade is a known mimic of distributive sepsis, particularly when another infectious source is present (2). Early bedside echocardiography is therefore an indispensable tool for cardiac anesthesiologists because it can identify mechanical causes that are not evident on examination (1). The patient's immediate improvement after pericardiocentesis confirmed that the obstructive physiology was driving her instability. The serous fluid and negative cultures aligned with sterile

inflammatory effusions that can nevertheless produce life threatening tamponade (3). Prioritizing relief of the reversible obstruction before proceeding with operative source control stabilized the patient, avoided a high-risk induction, and ultimately prevented an unnecessary laparotomy. This case reinforces the importance of physiology-based and echocardiographically-guided sequencing in complex mixed shock states.

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#FR 2 – Kiosk 12 – Poster 4

When the Donor Drives the Anesthetic Design: A Unique Cardiac Transplant Scenario

Presenting Author: Juan J. Morales Behaine, MD, Mayo Clinic Rochester, MN

Introduction

Malignant Hyperthermia (MH) is a rare pharmacogenetic disorder, with mortality rates as high as 70% when not treated appropriately. Most cases involve mutations in the ryanodine receptor isoform 1 (RyR1) in skeletal muscle, leading to excessive calcium release and a hypermetabolic state. Although the second isoform, RyR2, predominates in cardiomyocytes, cardiac mitochondria also contain RyR1, which participates in calcium handling, oxidative phosphorylation, and ATP production. Literature describing mitochondrial RyR1 and anesthetic implications in MH-susceptible donors is limited. We describe the anesthetic management for heart transplantation in a recipient of a donor heart carrying an RYR1 mutation.

Case Presentation

A 57-year-old male with hypertrophic obstructive cardiomyopathy status post septal ablation, transaortic and transapical septal myectomy, non-sustained VT, atrial fibrillation, severe tricuspid regurgitation, and NYHA III symptoms was scheduled for heart transplantation from a donor with a known pathologic RYR1 mutation. Given the potential MH risk, a MH-safe protocol was implemented during both organ procurement and during the cardiac transplant. This included uncontaminated anesthesia machine equipment, high flow flushing before surgery, and activated charcoal filters. For the recipient, anesthetic induction was achieved with midazolam, fentanyl, propofol, and rocuronium. Total intravenous anesthesia was employed using a propofol infusion. Analgesia included methadone and ketamine. A TTE probe as well as a radial and femoral arterial and central lines with a Swan Ganz catheter were placed. The patient's hemodynamics, including temperature, urine output, and acid base status, were monitored throughout the case. Upon separation from cardiopulmonary bypass (CPB), vasoactive support with epinephrine, norepinephrine, milrinone, and vasopressin was required. No intraoperative complications occurred. The patient was extubated and weaned off vasoactive medications on postoperative day two and discharged after three weeks with an ejection fraction of 70%. The patient was provided with a letter detailing the potential risk of triggering anesthetics to his transplanted heart.

Discussion

Although RyR1's expression is lower in cardiomyocytes, a heart transplanted from a MH-susceptible donor has a theoretical risk of developing isolated MH in the recipient. If knowledge of the donor's comorbidities is limited, this may be challenging to the anesthesia team. Such information is critical so that appropriate precautions can be employed, and a high index of suspicion maintained. While even traditional MH can be partially masked by hypothermia and carbon dioxide removal of CPB, identifying and treating MH isolated to the cardiac graft would be challenging. If MH is suspected, dantrolene remains effective for both cardiac and skeletal muscle RyR1 abnormal activity. Additionally, the risk extends beyond the initial transplant surgery, to all subsequent anesthetics, and such information should be disseminated to the patient in verbal and

written form. In summary, heart transplantation from MH-positive donors therefore warrants MH-safe precautions until the pathophysiology, mechanisms, and clinical implications of cardiac mitochondrial RyR1 are better understood.

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#FR 2 – Kiosk 12 – Poster 5

Third Trimester Percutaneous Balloon Mitral Commissurotomy in the Setting of Rheumatic Disease

Presenting Author: Gabriele M. D'Orsi, DO, Resident Physician, Allegheny Health Network

Introduction

Rheumatic heart disease (RHD) is uncommon in the United States but remains a leading cause of mitral valve pathology worldwide. Clinical presentations can be highly variable and often influenced by age at onset. For women of childbearing age with a history of RHD, multidisciplinary counseling and preconception evaluation are essential to mitigate risks to both mother and fetus. According to the 2020 AHA guidelines, symptomatic patients should undergo transthoracic echocardiography (TTE) to assess valve morphology as part of the evaluation for percutaneous mitral balloon commissurotomy (PBMC). Favorable anatomy for percutaneous intervention includes mobile, thin valve leaflets without significant calcification or fusion, less than moderate mitral regurgitation, and no left atrial thrombus. Surgical intervention is rare and generally reserved for patients with severe symptoms or anatomy not amenable to percutaneous procedures.

Case Presentation

We present the case of a 35-year-old female G1P0 at 31 weeks who underwent a PBMC secondary to severe mitral stenosis from RHD. She was diagnosed with RHD after an episode of syncope at the age of 20. She reported she was asymptomatic for years and had not followed up with a cardiologist since 2012. However, she began experiencing dyspnea on exertion, edema, and dizziness during her pregnancy. Finally, at 26 weeks, she re-presented to cardiology, who unearthed TTE findings of severe MS, mild MR, severe pulmonary hypertension, and a mean gradient of 15-20. Along with her symptoms, this placed her into NYHA class 3, with a baseline proBNP of 300. Furosemide was initiated at this time, followed by metoprolol soon after. Preoperative TTE showed a MVA of 1.7 cm^2, a mean gradient of 25 mmHg, moderate pulmonary hypertension, and a 1+ MR.

Discussion

This case required extensive multidisciplinary coordination across several departments to ensure optimal care. The urgency was heightened by the window to minimize risks for both mother and fetus. The patient's late presentation, advanced symptoms, and the absence of obstetric and NICU services at our primary cardiac center posed additional challenges. Preoperative coordination involved both NICU and high-risk OB teams in the OR, prepared to perform an emergency cesarean section. Continuous fetal monitoring was maintained throughout, and a helicopter was on standby for transfer to a facility with obstetric and NICU support if needed. Notably, PBMCs are most often performed during the second trimester for optimal maternal and fetal outcomes; however, our patient’s late presentation necessitated intervention in the third trimester, which is associated with greater risk. Anesthetic management in the third trimester introduces additional risks, including aspiration, airway complications, and maternal mortality. The potential for fetal neurodevelopmental effects from general anesthesia remains uncertain; current FDA guidance advises caution regarding possible impacts from exposure to inhaled anesthetics during the third trimester through

age three, though this is primarily based on animal data. This case report details our multidisciplinary approach and reviews the relevant literature for each clinical consideration.

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#FR 2 – Kiosk 12 – Poster 6

Heart-After-Liver Transplant with Domino Liver Transplant (HALT-D) in an Adult Patient with Non-Ischemic Cardiomyopathy status post LVAD and High Panel Reactive Antibodies

Presenting Author: Kevin Flood, MD, Resident, Emory University

Introduction

Heart-after-liver transplant (HALT) provides immunoprotection of the newly transplanted heart in highly allosensitized patients who failed desensitization efforts and for which a traditional orthotopic heart transplant (OHT) would be infeasible.1,2 Heart-after-liver transplant with domino liver transplant (HALT-D) entails significant anesthetic and logistic challenges requiring interdisciplinary planning and patient care.

Case Presentation

A 52 year-old woman with severe NICM s/p LVAD with chronic driveline infections, subsequent STEMI s/p DES to LAD, VT s/p ICD, epilepsy due to prior ICH, and elevated calculated panel reactive antibodies (>99% CPRA) was admitted with worsening RV failure and presented to the OR for HALT-D. In addition to standard preoperative workups for OHT and orthotopic liver transplant (OLT), preoperative apheresis was performed on day of surgery for acute desensitization. Redo median sternotomy was followed by extensive intrapericardial adhesiolysis leading to unstable ventricular arrythmias requiring defibrillation. Cannulation for and initiation of partial VA ECMO with bicaval drainage while continuing LVAD flow to allow LV venting facilitated the piggyback OLT in the presence of significant RV dysfunction. An OLT anesthesiologist was available for consultation during all parts of the case and co-managed liver reperfusion. Notable anesthetic challenges included profound hypotension after initiation of VA ECMO requiring rapid resuscitation which was continued through hepatic reperfusion. The OLT portion of the case was otherwise uneventful, as VA ECMO/LVAD flows allowed for stable hemodynamics and the patient had baseline normal liver function. VA ECMO was converted to full CPB under cross clamp for the OHT portion. Separation from CPB was successful with good biventricular function. Post-CPB was notable for high volume urine output (~5cc/kg/hr), significant transfusion requirements to achieve hemostasis, and modest vasopressor requirements. The biliary anastomosis was then completed, the chest and abdomen were closed, and the patient was transported to the ICU on low-dose inotropes after approximately 18 hours in the OR.

Discussion

HALT-D is a significant endeavor for all members of the perioperative team and requires a high degree of interdisciplinary communication and coordination across numerous services. Here we describe the successful inaugural implementation of a HALT-D protocol at our institution.

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#FR 2 – Kiosk 13 – Poster 1

Six Sternotomies to Transplant: Navigating the Impossible Glenn Heart

Presenting Author: Danielle Chism, DO, Anesthesiology Resident, Mayo Clinic

Introduction

Adults with congenital heart disease (ACHD) undergoing heart transplantation represent one of the highest-risk transplant groups due to prior surgeries, altered anatomy, and complex physiology.[1-3] Patients with pulmonary atresia with intact ventricular septum (PA/IVS) often undergo staged palliation, and those with Glenn/Fontan physiology have higher perioperative morbidity and mortality than biventricular ACHD patients. We present a patient with a hypoplastic right ventricle (RV), Glenn physiology, multiple pulmonary artery (PA) reconstructions, a transcatheter valve-in-valve pulmonary valve replacement (PVR), and six prior sternotomies who underwent orthotopic heart transplantation (OHT).

Case Presentation

A 35-year-old woman with PA/IVS and a hypoplastic RV had a long surgical history beginning with pulmonary valvotomy, a right Blalock–Taussig–Thomas shunt (BTT), and PDA ligation in 1989, followed by right PA patching, creation of a 6-mm atrial septal defect (ASD), placement of a 5-mm Gore-Tex central shunt to the right PA, and closure of the BTT shunt in 1991. She later underwent a classic Glenn to the right PA and received an epicardial pacemaker for sinus node dysfunction in 1994. In 2007, she had a bidirectional Glenn with unifocalization of the right PA and reconstruction using an 8-mm Gore-Tex graft. PRV with a 25-mm Magna Ease valve plus RV outflow tract and main PA augmentation was performed in 2017, followed by a valve-in-valve transcatheter PVR in 2022. Her heart failure progressed after two pregnancies. Echocardiography showed preserved LV function with a small but functional RV; catheterization demonstrated diastolic heart failure and a bidirectional ASD shunt. Given her complex anatomy, a multidisciplinary team developed a detailed operative plan. With difficult access expected, a right internal jugular central line was placed preoperatively by interventional radiology, and a Swan–Ganz catheter was avoided due to Glenn physiology. An awake brachial arterial line was placed before induction, which she tolerated with propofol, lidocaine, rocuronium, and fentanyl. Cardiopulmonary bypass was then established via the right femoral vein and left axillary artery. The redo sternotomy was difficult, with dense adhesions and systemic-to-pulmonary collaterals causing major bleeding. Her intrathoracic venous system required reconstruction. After graft implantation, severe biventricular dysfunction prevented weaning from bypass, necessitating central VA ECMO. She was transferred to the ICU with an open chest after massive transfusion (15 units PRBC, 4 units FFP, 13 units cell-saver blood, 8 units cryoprecipitate, 2,000 units Kcentra, and 6 liters of crystalloid). Her postoperative course remained extremely complex, and she died four months after transplantation.

Discussion

This case highlights the significant surgical and anesthetic challenges of OHT in adults with Glenn physiology and multiple prior operations. Redo sternotomy increases bleeding risk due to scarring, and

Glenn physiology complicates venous return, monitoring, and bypass strategy. Meticulous planning and multidisciplinary coordination were critical in caring for this exceptionally high-risk patient.

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#FR 2 – Kiosk 13 – Poster 2

On the Move and Out of Time: Managing Clot-in-Transit with Venoarterial Extracorporeal Membrane Oxygenation Rescue

Presenting Author: Kitae Chang, MD, Mass General Brigham

Introduction

Acute pulmonary embolism (PE) is a sudden obstruction of the pulmonary arteries, and it affects an estimated 121 per 100,000 people annually.[1] It remains a major cause of cardiovascular morbidity, with short-term mortality ranging from 0.2 to 0.6%, rising to 3 to 65% in patients presenting with hemodynamic instability or right ventricular (RV) failure.[2] Prompt recognition and treatment are essential to reduce the substantial risk of death and long-term complications.

Case Presentation

A 50-year-old male with no prior medical history presented with chest pain and shortness of breath and was found to have lower extremities DVTs complicated by a submassive PE as well as a clot in transit in the right atrium (RA). Given the intracardiac involvement, a decision was made to urgently retrieve the thrombi percutaneously. On TEE, the patient was found to have a 38 x 33 mm mobile thrombus in the RA-IVC junction (Figure 1) and a thrombus in the right pulmonary artery (PA) (Figure 2). The right ventricle was significantly dilated, and its function was severely reduced. Following suction of the atrial thrombus using the AlphaVac system, the patient went into obstructive and cardiogenic shock with persistent hypoxia. Epinephrine and milrinone were started for inotropic support and inhaled epoprostenol was initiated for RV afterload reduction. Due to persistent instability, the decision was made to emergently cannulate for peripheral VA ECMO. Following stabilization, the distal emboli were evacuated with the FlowTriever device (Figure 3). There was significant improvement of the RV function and gas exchange. The patient was transported to the ICU, intubated and with minimal pharmacological and mechanical support.

Discussion

Given the safety and efficacy of catheter-directed mechanical thrombectomy, it has become a widely adopted treatment option for patients with intermediate to high-risk PE. Despite its successes, the rate of intraprocedural arrests due to cardiovascular collapse still occur 1 to 6% of the time.3 In this case, the sudden instability following the initial thrombectomy was likely due to dislodgment and propagation of thrombi causing mixed obstructive and right-sided cardiogenic shock. The immediate benefit of ECMO initiation was reducing the RV preload and providing partial unloading of the failing RV. With a controlled physiologic environment, the procedural team was able to more safely perform the distal clot extractions. To mitigate the risk of acute right heart failure in acute PE, anesthetic interventions and pharmacotherapies should be directed toward increasing inotropic support (e.g., epinephrine, milrinone) and reducing RV afterload (e.g., avoiding hypercarbia and hypoxia, using inhaled pulmonary vasodilators). If these measures are inadequate, the use of peripheral VA ECMO as salvage support provides a critical bridge while the vascular obstruction is cleared. Although routine use is not fully adopted in treatment algorithms, especially

in the presence of floating intracardiac clots that portend risk for further embolization by catheter manipulation, it is prudent to have extracorporeal support readily available if possible.

Images

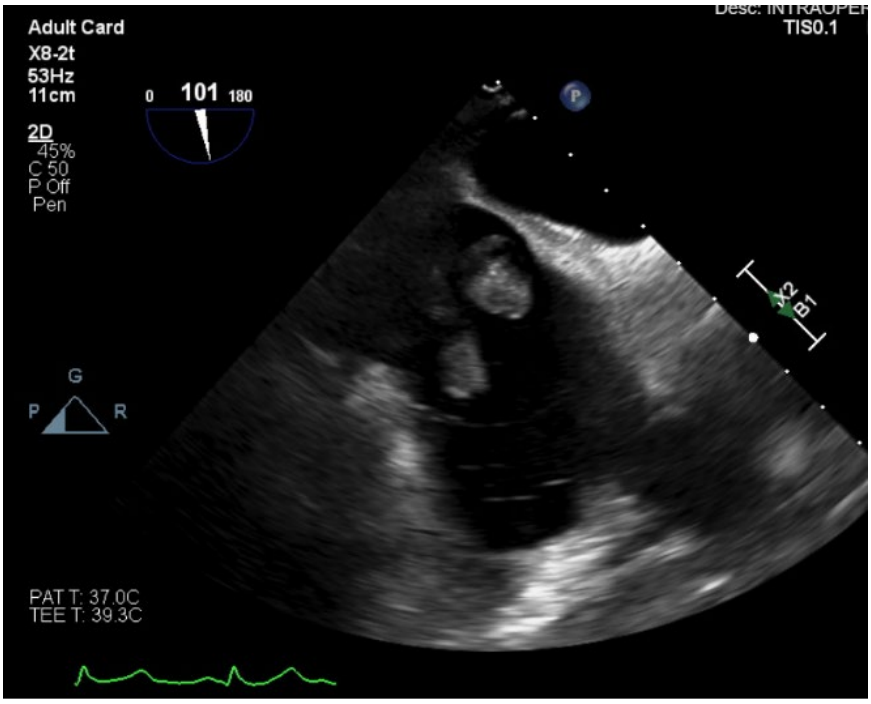


Figure 1. Intra-atrial clot viewed in the mid-esophageal bicaval view

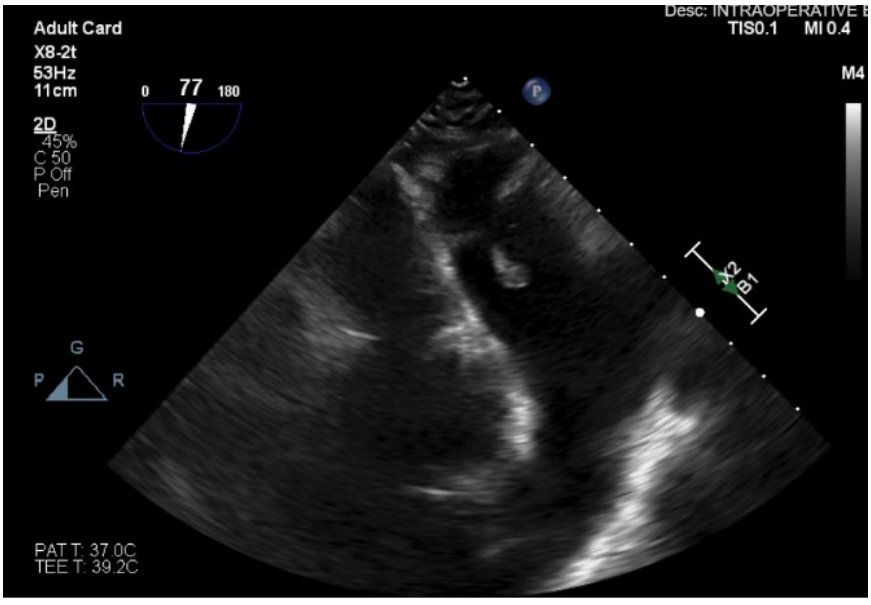


Figure 2. Intra-arterial clot visualized in the right pulmonary artery in the midesophageal ascending aorta short-axis view

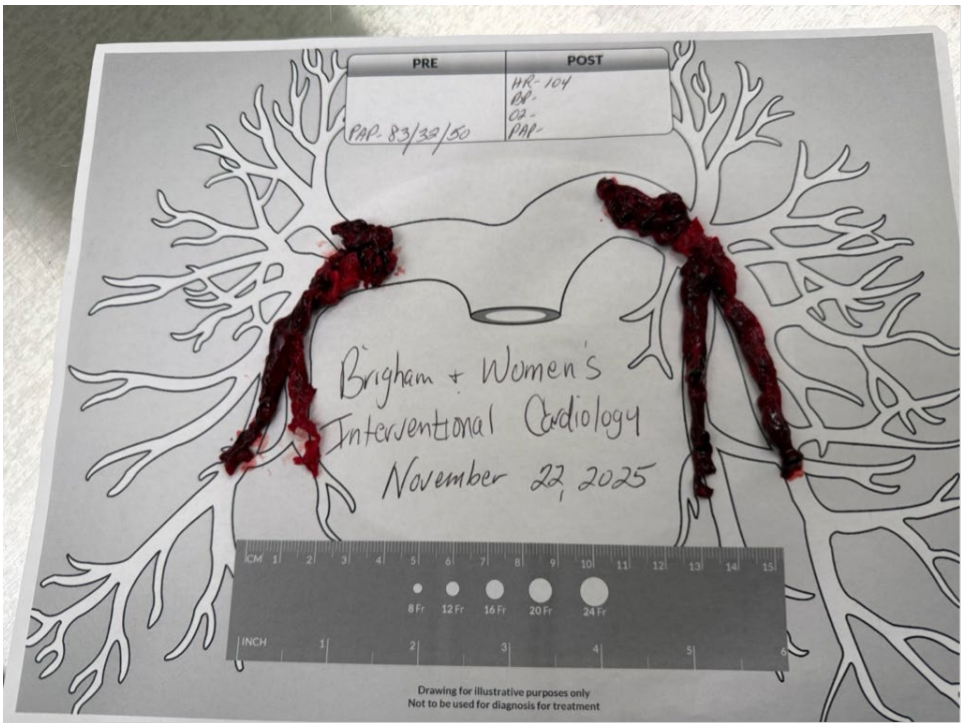


Figure 3. Distal arterial thrombi retrieved via the FlowTriever device

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#FR 2 – Kiosk 13 – Poster 3

Masked Mitral Prosthetic Valve Dysfunction During VA-ECMO Support: A Diagnostic Pitfall

Presenting Author: Samir Abdul Aziz, MD, Fellow, Henry Ford Hospital

Introduction

Prosthetic valve replacement during complex cardiac surgery becomes especially challenging when postoperative venoarterial extracorporeal membrane oxygenation (VA-ECMO) is required. ECMO alters preload, afterload, and transvalvular flow, creating a low-flow state that increases thrombotic risk on prosthetic valves and complicates transesophageal echocardiography (TEE) assessment. Low flow may make a prosthetic mitral valve (MV) appear to function normally despite early dysfunction.

Case Presentation

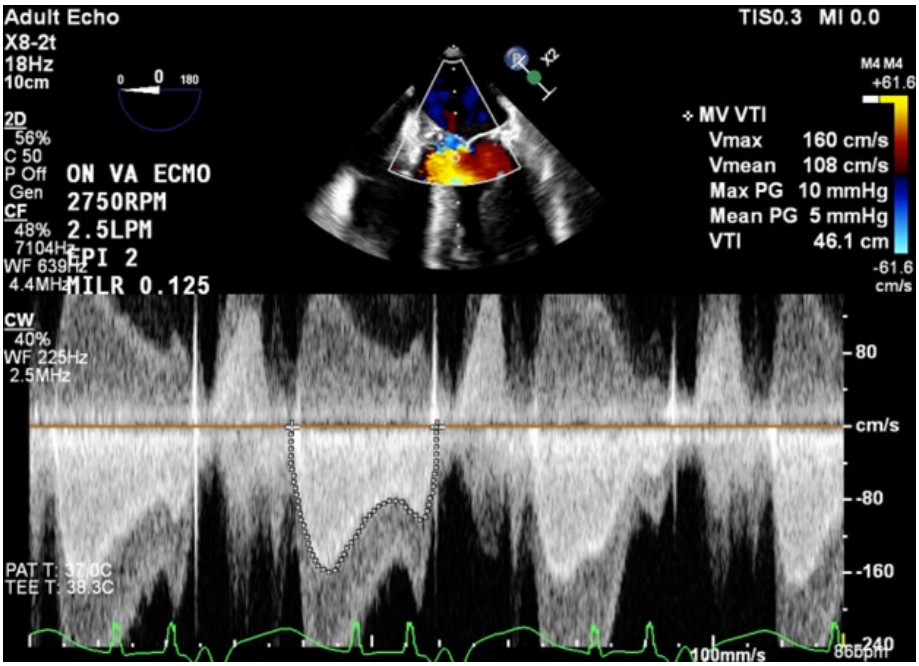
A 50-year-old woman with bicuspid aortic stenosis and an ascending aortic aneurysm underwent ascending aorta replacement, aortic root enlargement, and aortic valve replacement with a 19-mm valve. Biventricular dysfunction from suspected left coronary compromise during weaning from cardiopulmonary bypass (CPB) prompted a Bentall procedure with a 23-mm valve. During repeat weaning, severe mitral regurgitation was identified, requiring mitral valve (MV) replacement with a 27-mm valve. She was ultimately supported with vasopressors and central VA-ECMO and transferred to our center. Pre-decannulation TEE showed preserved ventricular function with what appeared to be normal MV leaflet motion. On ECMO, the prosthetic MV seemed to open and close normally and so she was taken for ECMO decannulation. Intraoperative TEE revealed a mean MV gradient of 5 mmHg on full ECMO support. This gradient increased to 10 mmHg with subtle, but definite leaflet restriction; findings unmasked as native cardiac output increased. After decannulation, gradients rose sharply with worsening oxygenation and lactate elevation, requiring immediate VA-ECMO reinstitution. Despite full support, she progressed to multiorgan failure and died following transition to comfort care.

Discussion

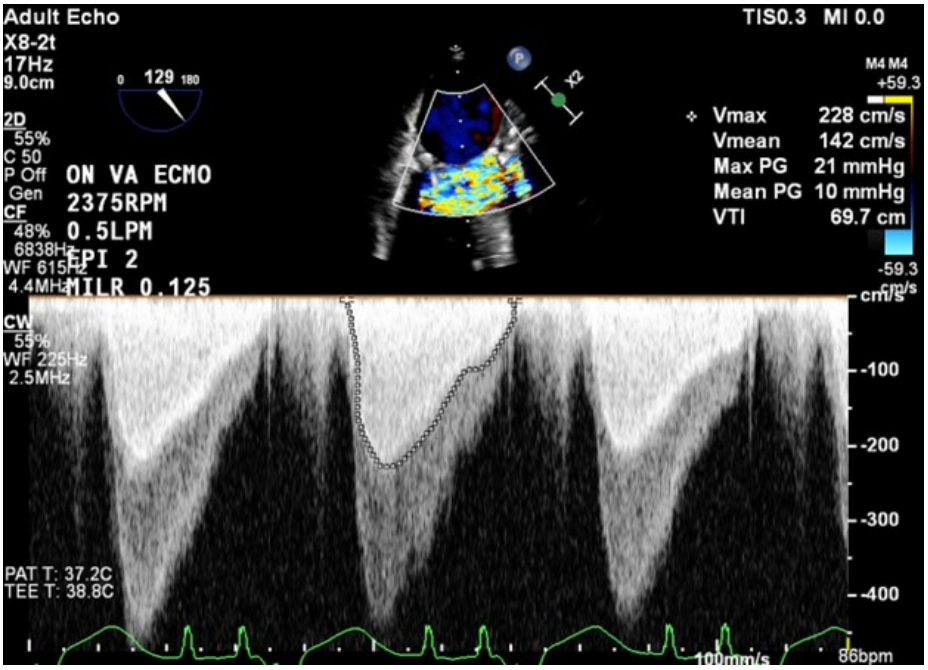
This case illustrates the difficulty of assessing prosthetic MV function on VA-ECMO. ECMO markedly reduces left ventricular stroke volume and transvalvular flow, causing Doppler gradients to appear deceptively low and potentially making leaflet motion seem normal. As ECMO flow decreases during weaning, previously masked obstruction may rapidly become apparent, as occurred here. Standard TEE parameters—peak velocity, mean gradient, and qualitative leaflet excursion—are highly flow dependent.

Low circuit flow can make even a significant obstruction appear normal. In addition, ECMO unloading promotes left-sided stasis and increases thrombotic risk, which may intermittently impair leaflet mobility before producing overt hemodynamic compromise. A comprehensive, multimodal approach is essential. Beyond Doppler data, clinicians should examine leaflet symmetry, hinge motion, and possible pannus or thrombus. Geometric analysis of opening and closing angles, shown in the literature to detect subtle dysfunction, is particularly valuable when flow is unreliable. Even mildly elevated gradients or subtle restriction on ECMO should raise concern, as obstruction may be masked until normal loading is restored. Careful, repeated assessment during ECMO weaning is critical to avoid delayed recognition of prosthetic dysfunction and its potentially catastrophic consequences.

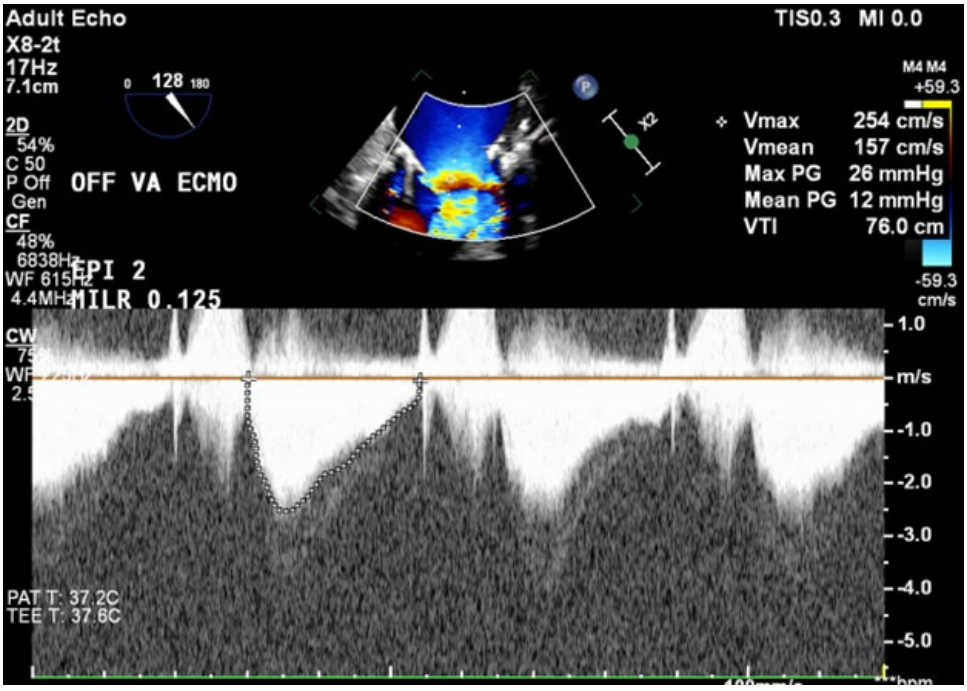
Images



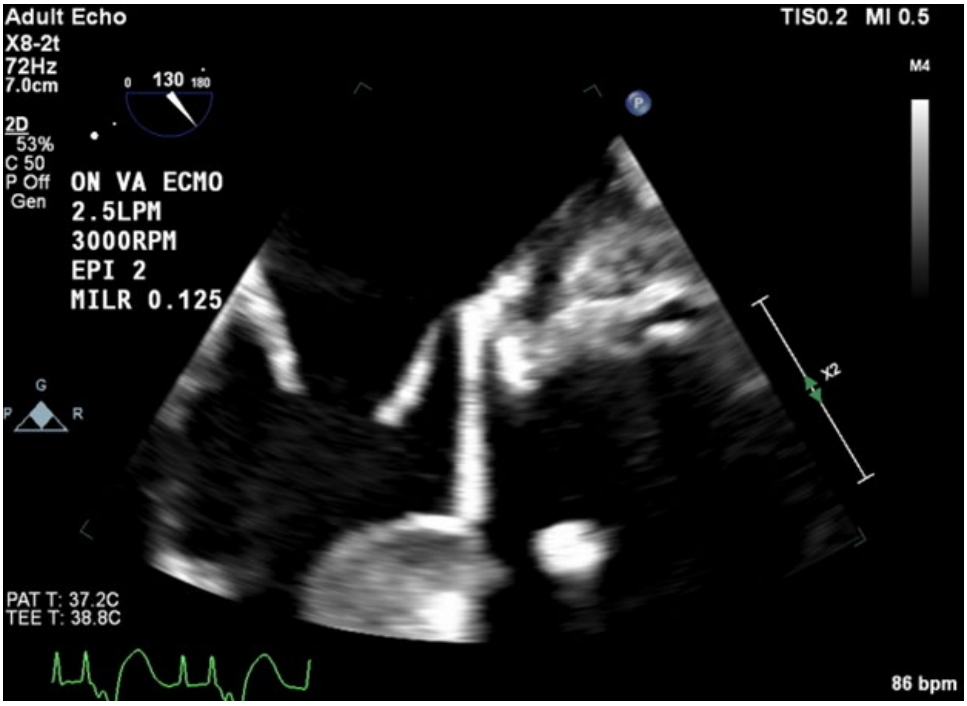
Transmitral gradient on full ECMO support



Transmitral gradient on minimal ECMO support



Transmitral gradient on off of ECMO support



Restricted leaflet motion



Normal leaflet motion in a different patient for comparison

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#FR 2 – Kiosk 13 – Poster 4

Airway Management for Robotic Assisted Enucleation of Esophageal Tumor

Presenting Author: James Severin, MD, CA-1, Cedars Sinai

Introduction

Esophageal schwannomas are rare benign neurogenic tumors that may require excision. Due to their location in the mediastinum and proximity to the airway, anesthetic management requires careful planning for airway patency and lung isolation.

Case Presentation

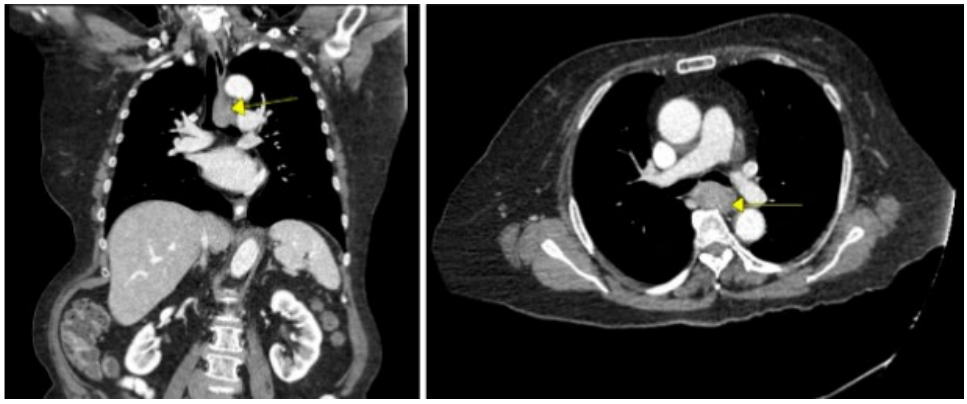
A 144cm 71yo female presented with progressively worsening dysphagia over the past 3 months. The patient was found to have a 4cmx3.4cmx2.6cm esophageal mass located in the mid to upper esophagus at the level

of carina (Picture 1). The patient was recommended to proceed with enucleation of the tumor via right robotic assisted lymphadenectomy and esophageal mobilization. CT scan showed compression of proximal left bronchus. The mass was noted to be inseparable from the esophagus with mass effect upon the distal trachea and proximal mainstem bronchi without significant proximal esophageal dilation. Two major considerations guided the anesthetic plan for this case. First, the tumor's close proximity to the carina raised the risk of airway distortion or injury during instrumentation and lung isolation. Our institution's standard of care is to place the double-lumen endotracheal tube (DLT) contralaterally to the planned surgical site. With this in mind, a left sided DLT was intended to be used during the procedure. However, because of a concern for significant mass effect on the proximal left bronchus possibly causing difficulty in advancing the DLT past the obstruction, a right sided DLT and bronchial blocker were also prepped and ready to be used if needed. Second, since the depth of invasion and underlying pathology were not yet fully defined, there was a possibility that the procedure could escalate to an esophagectomy, necessitating advanced planning for a significantly more complex and prolonged operation. In anticipation of this, two large bore IV's were placed for intraoperative access. Intraoperatively, standard ASA monitors were placed, followed by induction and placement of a left-sided 32Fr DLT. Initial fiberoptic bronchoscopy confirmed both the placement of the DLT and that the mass was not invading through the airway mucosa, allowing for passage to the open distal bronchus. After a left radial arterial catheter was placed, the patient was positioned in the left lateral decubitus position, and one-lung ventilation was initiated. A subsequent intraoperative esophagogastoduodenoscopy was used for identifying esophageal landmarks and testing of esophageal integrity. A myotomy was then performed with enucleation of the tumor and the esophageal mucosa remaining intact (Picture 2). After reinflation of the operative lung, the patient was extubated awake without difficulty. A follow up pathology report revealed schwannoma.

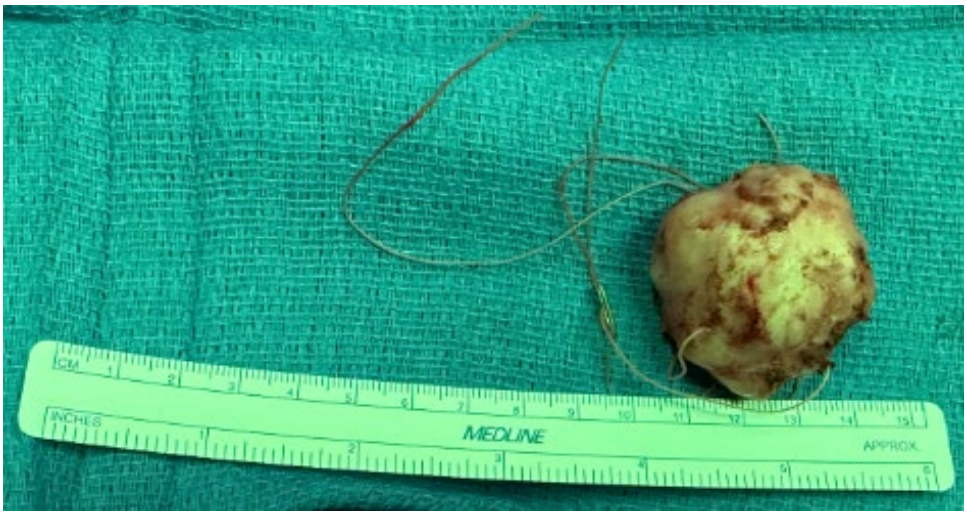
Discussion

Airway Management: Thorough image review and preoperative discussion regarding alternative options for airway management possibilities. Procedure Discussion: Preoperative planning with the surgical team regarding access of the mass and possible esophagectomy. Lung isolation: Difficult placement secondary to significant anatomical distortion related to mass effect and patient repositioning. Must reconfirm position with fiberoptic bronchoscopy.

Images



Picture 1 - Axial and coronal view of esophageal mass at the level of the carina compressing the left bronchus



Picture 2 - Enucleated esophageal schwannoma

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#FR 2 – Kiosk 13 – Poster 5

Intraoperative Intracardiac Echocardiography (ICE) For Aorto-Esophageal Fistula Repair

Presenting Author: Sumarth K. Mehta, MD, Department of Anesthesiology and Pain Management, The University of Texas Southwestern Medical Center

Introduction

Transesophageal echocardiography (TEE) allows anesthesiologists to manage complex hemodynamics during cardiac surgery. When TEE is absolutely contraindicated due to esophageal disease, unstable cervical spine, or perforated viscus [1], falling back to pulmonary artery catheters and other invasive hemodynamic monitors means losing the rich anatomic and physiologic information provided by echocardiography. One alternative is intracardiac echocardiography (ICE), more commonly used in catheterization lab structural heart procedures [2]. We present a case demonstrating the utility of ICE in the operating room for crucial hemodynamic management in the repair of a large aorto-esophageal fistula caused by erosion of an infected thoracic endovascular aortic stent.

Case Presentation

A 74-year-old female with extensive history including aortic and mitral valve replacements, aortic hemiarch replacement, and zone 3 TEVAR was found on imaging to have gas around the TEVAR graft suggesting infection. She was initially managed medically due to fragility, but presented with hematemesis and was noted to have an aorto-esophageal fistula (Fig 1A). A procedure was planned involving removal of the infected endograft, replacement of the thoracic aorta, and repair of the esophagus. Following uneventful induction, a 10 French introducer was placed in the right internal jugular vein and the ICE catheter (*Philips VeriSight*®) was advanced into the right atrium. Imaging with the ICE probe provided multiple acoustic windows corresponding to standard TEE views (Fig 2), as well as offering 3D imaging of valves (Fig 3B). One lung ventilation was initiated and the left lung was mobilized to expose the aorta. The femoral artery and vein were cannulated, and ICE was used to position a multistage venous cannula in the SVC (Fig 3A). Cardiopulmonary bypass with initial femoral arterial cannulation was used. Under deep hypothermic circulatory arrest, the infected TEVAR graft was removed and the descending aorta was replaced with a new graft. After resuming perfusion to the aorta, there was still significant bleeding through an aorto-esophageal fistula proximal to the graft as demonstrated on esophagoscopy. (Fig 1B). A second round of circulatory arrest was done and a more proximal region of the aortic arch was replaced with resolution of bleeding. Weaning from bypass was attempted, however ICE demonstrated severely depressed biventricular systolic function even on high dose inotropic support. The patient was placed on VA-ECMO and transferred to the ICU. Despite correction of coagulopathy, the patient continued to hemorrhage. After discussion with family, support was withdrawn and the patient passed away.

Discussion

The use of ICE in the cardiac operating room allows for more informed hemodynamic management than other alternatives to TEE [3]. During periods of partial bypass, monitoring ventricular filling allowed us to

direct the perfusionist to adjust drainage and flows. Real-time visual feedback was also critical when returning volume to the patient during attempts to wean bypass, and assessment of systolic function enabled early recognition of biventricular systolic failure that required restarting mechanical circulatory support.

Images

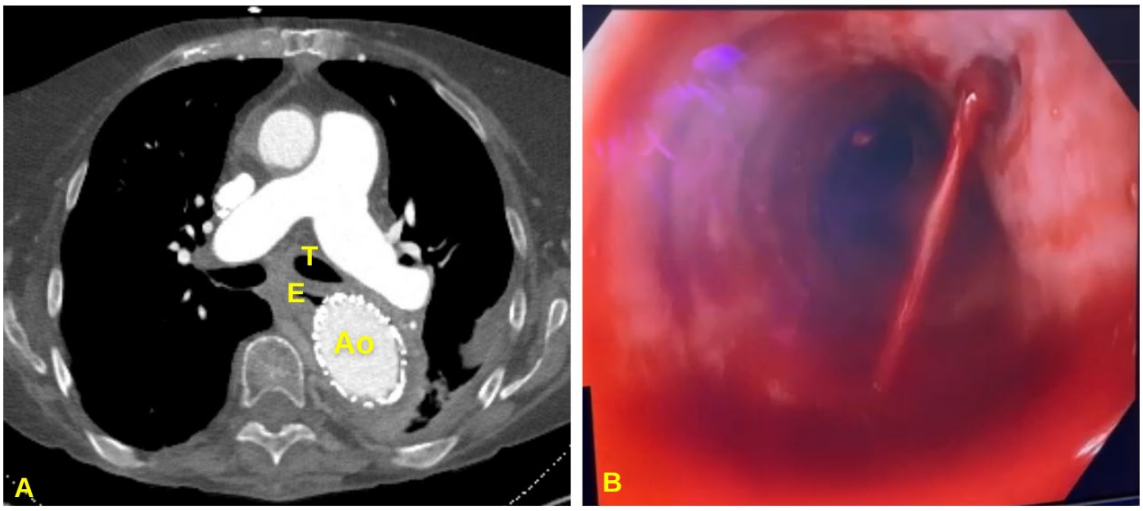


Fig 1: (A) CT with contrast showing erosion of stent towards esophagus. T: trachea; E: esophagus; Ao: aorta. (B) Bleeding seen via esophagoscopy after TEVAR stent graft was dissected out.

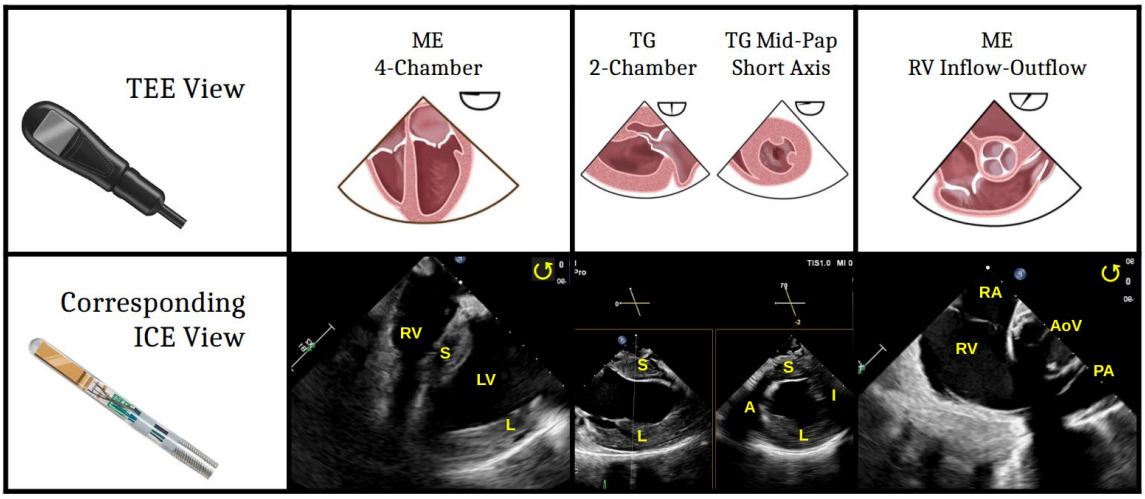


Fig 2: TEE Views and corresponding ICE views. RA: right atrium; RV: right ventricle; AoV: aortic valve; PA: pulmonary artery; A: anterior wall of LV; I: inferior wall of LV; S: septal wall of LV; L: lateral wall of LV. ↺indicates the ICE image has been mirrored horizontally to better correspond with TEE view. TEE drawings from the American Society of Echocardiography.

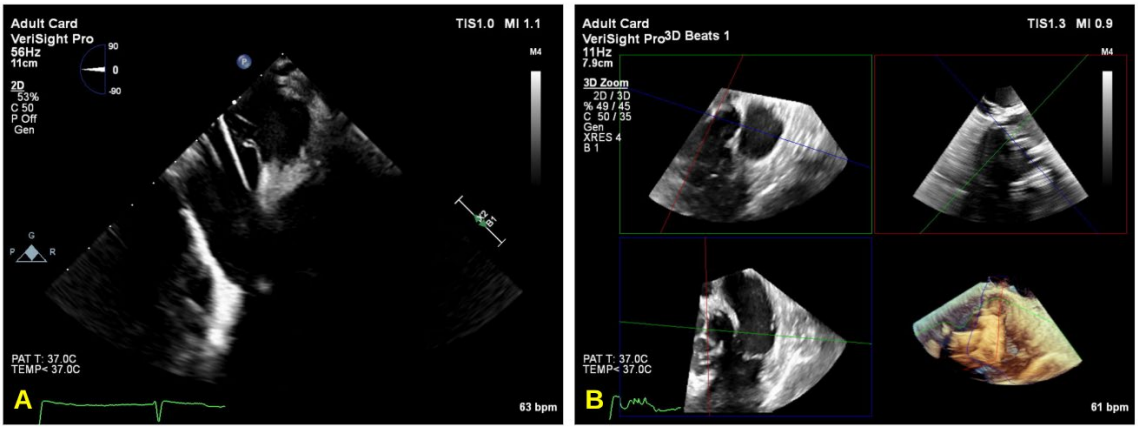


Fig 3: (A) ICE used to guide positioning of venous cannula. (B) 3D visualization of the aortic valve using ICE.

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#FR 2 – Kiosk 13 – Poster 6

Veno-Venous Extracorporeal Membrane Oxygenation in the Difficult Airway

Presenting Author: Josue D. Chirinos, MD, Resident Physician, UCLA Health

Introduction

VV-ECMO is a form of respiratory support used in severe respiratory failure when conventional therapies have failed. Emerging literature has demonstrated its role in the management of the difficult airway, particularly as a means of maintaining gas exchange during airway manipulation when awake or fiberoptic techniques carry prohibitive risk1,2. However, reports remain limited. Here, we describe the use of VV-ECMO in a patient with severe tracheal narrowing from compression by a massive vertebral artery pseudoaneurysm.

Case Presentation

A 55-year-old male with a history of traumatic left vertebral artery pseudoaneurysm status-post stenting presented in respiratory distress. On exam, he had audible stridor and dyspnea in supine positioning but normal work of breathing and oxygen saturation on room air while upright. CTA chest revealed an enlarging 11x4 cm superior mediastinal pseudoaneurysm with significant mass effect and slit-like narrowing of the distal trachea (Fig 1). The patient was therefore admitted to the CTICU, and after multidisciplinary discussion, a stepwise treatment plan was devised. The patient would require cerebral angiography to inform the surgical approach followed by vascular reconstruction and pseudoaneurysm excision. However, he could not tolerate lying flat for angiography, and his pin-point tracheal stenosis put him at extreme risk peri-intubation, even with an awake fiberoptic approach. Thus, the team elected to initiate VV-ECMO to facilitate further workup and eventual intubation. On hospital day 2, the patient was cannulated for VV-ECMO via bifemoral approach and underwent angiography under moderate sedation without issue. On hospital day 4, he was intubated in the CTICU in preparation for surgery. After preoxygenation via HFNC and optimization of ECMO sweep and FdO2, the patient was sedated and paralyzed. Video laryngoscopy provided a grade II view, and a 5.5 armored ETT was passed through the vocal cords. An attempt was made to bypass the area of tracheal narrowing under bronchoscopic guidance. However, the ETT was neither long nor stiff enough to negotiate the airway. Thus, the armored ETT was removed, and the patient was reintubated with a 5.5 MLT, which successfully passed the area of tracheal narrowing. Purulent secretions consistent with post-obstructive pneumonia were noted, and antibiotics were started. The patient tolerated the procedure well with no desaturations or airway complications. With his airway secured, the patient underwent TEVAR with left vertebral-to-carotid bypass followed by pseudoaneurysm excision and mediastinal hematoma evacuation (Fig 2). Postoperative bronchoscopy showed normal anatomy with no residual stenosis. He was therefore extubated on POD1 after pseudoaneurysm excision and decannulated from ECMO on POD2. He was discharged home on POD12.

Discussion

This case highlights VV-ECMO as a proactive intervention for the anatomically hostile airway. In our patient,

VV-ECMO avoided high-risk airway attempts, maintained oxygenation during airway manipulation, and supported postoperative respiratory recovery despite extreme preoperative airway distortion. While more evidence is needed to clarify indications and timing, this case supports VV-ECMO as a lifesaving modality in select patients with complex airway pathology.

Images

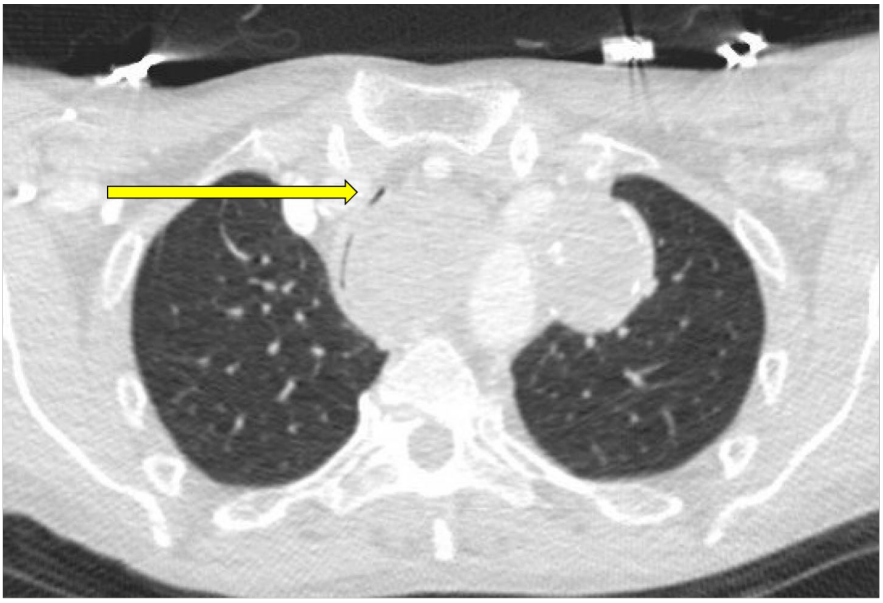


Fig 1. Slit-like narrowing of the distal trachea due to mass effect from left vertebral artery pseudoaneurysm.

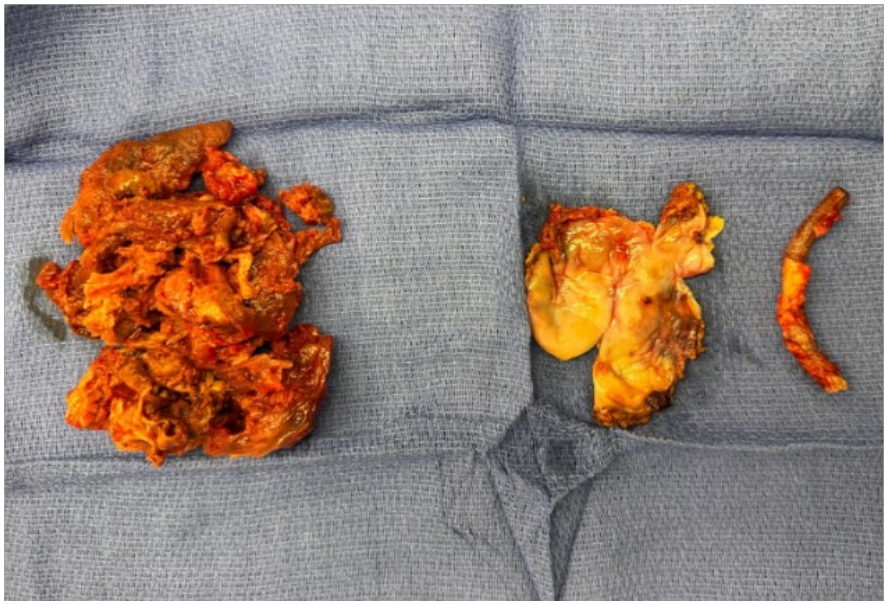


Fig 2. Contents of pseudoaneurysm post-excision, including previously placed stent (right).

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#FR 2 – Kiosk 13 – Poster 7

Difficult Airway Management in Severe Aortic Stenosis and Post Valve Replacement LVOT Obstruction: Navigating a Hemodynamic Minefield

Presenting Author: Ned Shashoua, MD, Anesthesiology Resident, University of Chicago

Introduction

The risks of induction of general anesthesia in patients with severe aortic stenosis (AS) are well documented. The added challenge of difficult airway management in this population presents a unique obstacle for anesthesiologists. Dynamic left ventricular outflow tract (LVOT) obstruction after aortic valve replacement (AVR) poses another perioperative pitfall. We present a case of surgical AVR in a patient with both a difficult airway and postoperative LVOT obstruction.

Case Presentation

A 63-year-old female with severe bicuspid AS (gradient 58 mmHg) and a 61% ejection fraction (EF) underwent surgical AVR. Two years prior, the patient had a difficult airway requiring fiberoptic intubation. A pre-induction arterial line was placed. The patient then received 2 mg of midazolam, 12 mcg of dexmedetomidine, nebulized 4% lidocaine, and 20 mg of esmolol. After further topicalization with an oral airway coated in 4% lidocaine, the patient was successfully intubated with a flexible fiberoptic scope. After 152 minutes of bypass time and placement of a functioning 19mm mechanical aortic valve, transesophageal echocardiography (TEE) demonstrated a hyperdynamic left ventricle (EF 70%) and a valvular gradient of 12 mmHg. Over the next hour, the patient began experiencing worsening hypotension with dynamic systolic LVOT collapse on TEE. She received 250 mL of albumin and a phenylephrine infusion, with gradual increase in left ventricular cavity filling and resolution of LVOT collapse. The patient was transferred to the ICU intubated and sedated on 0.2 mcg/kg/min of phenylephrine with AV epicardial wires, AAI paced at 80 beats per minute.

Discussion

During induction, patients with severe AS require maintenance of preload, afterload, and contractility, with preservation of normal heart rate and sinus rhythm 1. Anesthesiologists must sustain adequate coronary perfusion pressure, countering loss of vascular tone from hypnotics, and diminished preload from positive pressure ventilation. In patients with difficult airways, providers face the risk of hypoxemia in a population particularly reliant on myocardial oxygen supply. Safe airway management is predicated upon reliable topicalization and anxiolysis. In a calm, spontaneously breathing patient, significant hemodynamic variation is less likely during airway manipulation. In this case, valuable time was dedicated to topicalization and optimization before using the fiberoptic scope. LVOT obstruction after aortic valve replacement is uncommon; patients with concentric left ventricular hypertrophy, asymmetric septal thickening, and high preoperative transvalvular gradients are at increased risk2. Mechanistically, removal of a fixed valvular stenosis creates a subvalvular pressure gradient; high-velocity flow through a narrowed outflow tract causes dynamic collapse via Bernoulli's principle. Referenced interventions including volume repletion, afterload

augmentation, and RV pacing3 were utilized after recognition in this case. More aggressive measures in refractory cases such as septal myomectomy or ablation are also cited. Further research could assist in identification of at-risk patients preoperatively to avoid delayed recognition and treatment.

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#FR 2 – Kiosk 14 – Poster 1

Ruptured Sinus of Valsalva Aneurysm Presenting as Acute Liver Failure

Presenting Author: Bryce Johnson, MD, The Ohio State University Wexner Medical Center

Introduction

Sinus of Valsalva aneurysm is a rare cardiac anomaly characterized by dilation of the aortic sinus wall, typically above the aortic valve. It is most commonly congenital, often associated with ventricular septal defect (30–60% of cases) or bicuspid aortic valve. Although incidence ranges from 0.2–0.9% in cardiac patients, true prevalence is likely underestimated due to asymptomatic cases (Avati et al.). Rupture of the aneurysm, most commonly at the right coronary sinus, can lead to diverse symptoms, including heart failure, chest pain, or dyspnea, but unusual presentations are rarely documented (Arcario et al.).

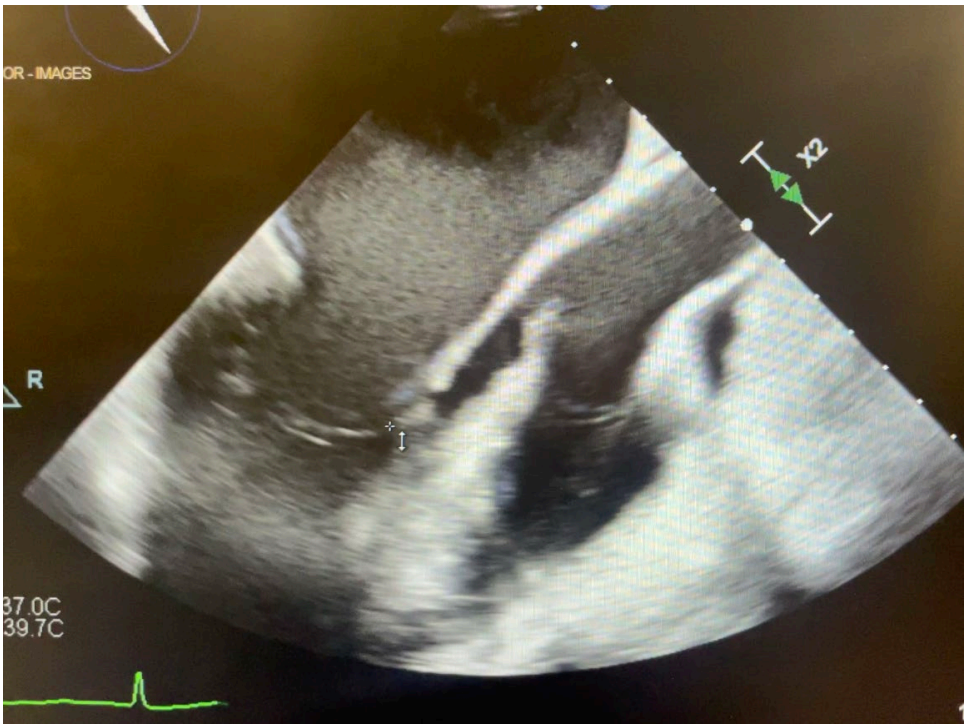
Case Presentation

This is a case of a 26-year-old male with a history of ventricular septal defect (VSD), rheumatoid arthritis, and substance use, who presented with nausea, vomiting, and altered mental status. Initial evaluation revealed acute liver failure (AST 5940, ALT 3890), hyperammonemia (ammonia 360 µmol/L), and acute kidney injury. As his condition deteriorated into multi-pressor shock, an extensive workup was initiated. Transthoracic echocardiography and CT angiography identified his known VSD with left-to-right shunting, mild biatrial dilation, and preserved ejection fraction (50–55%). Subsequent review by cardiothoracic surgery raised concern for a fistula between the sinus of Valsalva and the right ventricular outflow tract (RVOT). The patient was brought urgently to the operating room, where transesophageal echocardiogram (TEE) confirmed the presence of a 1.8 cm defect in the right sinus of Valsalva communicating with the RVOT. The patient underwent patch closure of the fistula and VSD repair. Postoperatively, his multi-organ dysfunction resolved, including normalization of liver enzymes, kidney function, and mental status. He was discharged home in stable condition on postoperative day twelve.

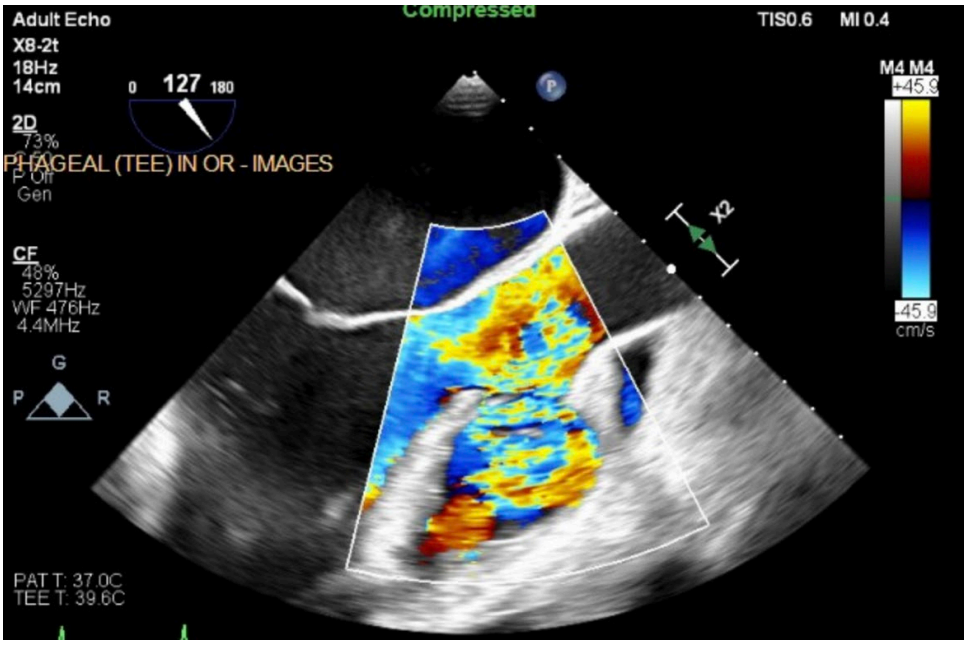
Discussion

This case highlights an unusual case of sinus of Valsalva aneurysm rupture; acute liver failure as the primary presentation secondary to right heart failure in the setting of rupture into the RVOT. Such pathophysiology has not previously been described in the literature. While sinus of Valsalva aneurysm rupture is rare, its varied presentations necessitate a broad differential diagnosis in cases of undifferentiated shock. This case underscores the importance of rapid identification of cardiac conditions in critically ill patients. Prompt interdisciplinary collaboration and diagnostic imaging can facilitate timely intervention, potentially averting fatal outcomes, as was successfully done in the case of this young patient.

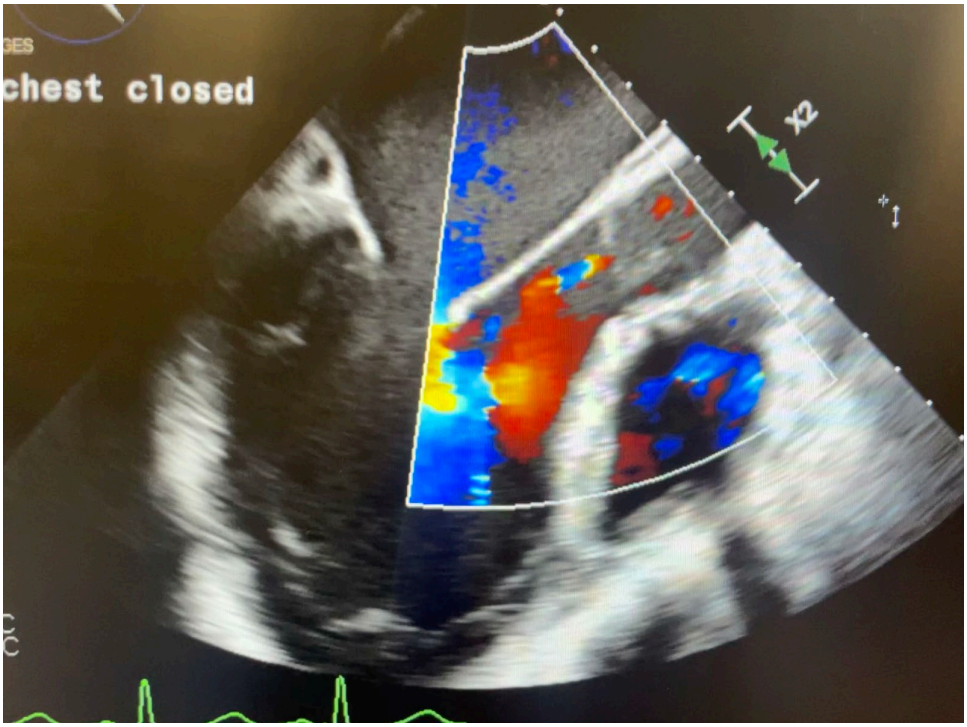
Images



Intraoperative TEE



Intraoperative TEE doppler



Post operative TEE

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#FR 2 – Kiosk 14 – Poster 2

Intraoperative TEE-Derived Qp:Qs Unmasks Significant Shunt Burden in Combined Atrial Septal Defect and Left Anterior Descending-Pulmonary Artery Fistula

Presenting Author: William Ho, MD, Anesthesiology Resident CA-2, University of Miami/Jackson Memorial Hospital

Introduction

Coronary artery fistulas (CAFs) are rare congenital anomalies, and those originating from the left anterior descending artery (LAD) to the pulmonary artery (PA) may present with subtle or nonspecific symptoms. When combined with atrial septal defects (ASDs), total left-to-right shunt burden may be underestimated by preoperative testing. Intraoperative transesophageal echocardiography (TEE) can provide essential real-time physiologic data that reshapes surgical management.

Case Presentation

A 42-year-old male with exertional dyspnea and palpitations underwent evaluation revealing a continuous murmur and mild right ventricular (RV) dilation. Cardiac CTA identified a tortuous LAD-to-PA fistula and two small secundum ASDs. Right-heart catheterization showed no significant oxygen step-up (RA 84.3%, RV 83.6%, PA 83%) with a calculated Qp:Qs of 0.93, suggesting minimal shunt. Despite this, symptoms persisted and the patient was scheduled for surgical evaluation. Intraoperatively, TEE confirmed both ASDs and the coronary–pulmonary fistula. Doppler-derived stroke volume measurements from the LVOT and RVOT, combined with pre-bypass oxygen saturations, were used to estimate flows. Real-time TEE demonstrated pulmonary blood flow exceeding systemic flow, yielding an intraoperative Qp:Qs of approximately 1.3—significantly higher than catheterization suggested. The additive effect of two iASDs plus continuous fistulous flow clarified the true physiologic burden. Given this discrepancy and the potential for long-term RV remodeling, the surgical team proceeded with bicaval cannulation and right atriotomy for closure of both ASDs, followed by direct ligation of the LAD–PA fistula via a pulmonary arteriotomy. Post-repair TEE demonstrated complete elimination of left-to-right shunting, normalized RV size, and preserved ventricular function. The patient recovered uneventfully and reported marked symptom resolution at follow-up.

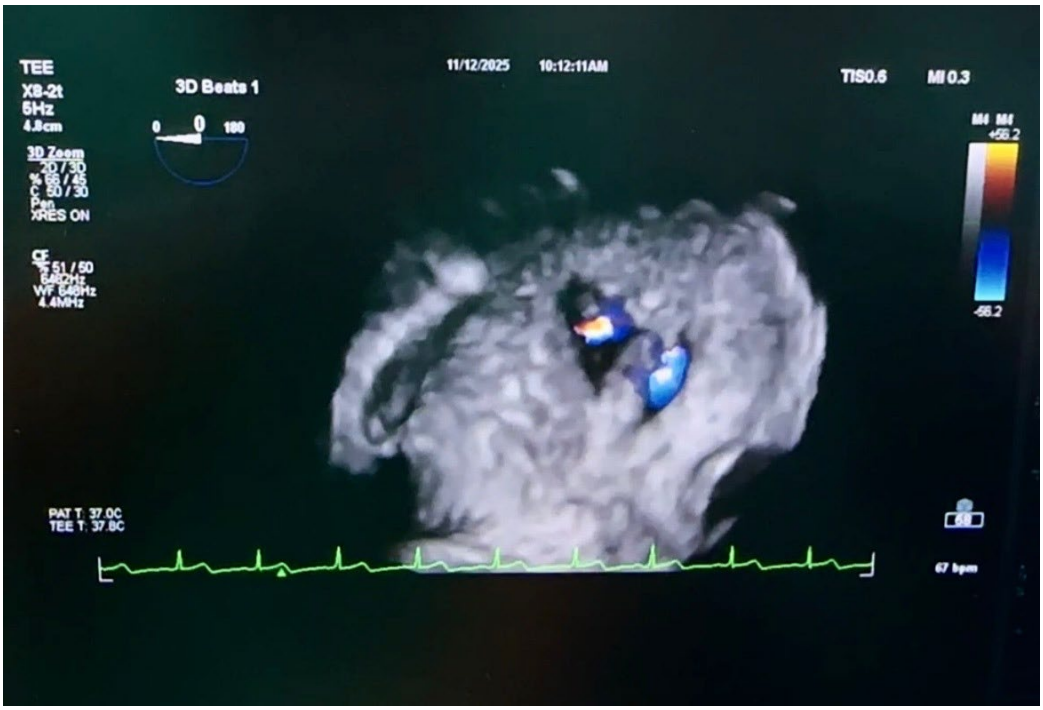
Discussion

This case highlights the critical role of intraoperative TEE in quantifying true shunt magnitude when multiple sources of left-to-right flow coexist. Preoperative catheterization underestimated Qp:Qs due to balanced saturations and low-flow physiology. TEE-based Doppler flow calculations provided decisive physiologic insight, altering surgical strategy and confirming the need for definitive repair. Intraoperative TEE should be considered essential in complex cases involving combined ASDs and coronary–pulmonary fistulas.

Images



LAD-pulmonary artery fistula first seen on TEE at commencement of case



2 iatrogenic persistent ASDs secondary to prior atrial fibrillation catheter ablations(2018, 2023, 2023) seen on TEE

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#FR 2 – Kiosk 14 – Poster 3

Raise the Roof: TEE-Guided Repair of an Isolated Coronary Sinus Defect

Presenting Author: Claire Luo, MD, Resident, Brigham and Women's Hospital

Introduction

Atrial septal defects (ASDs) comprise 5-10% of all congenital heart defects. A patent foramen ovale is found in 30% of patients with ASDs. Secundum ASD has a nearly 80% prevalence. Primum ASDs are typically associated with atrioventricular canal defects and account for 15% of ASDs. The least common defects include sinus venosus defects (both superior and inferior), and coronary sinus ASDs, otherwise known as unroofed coronary sinus (CS). Unroofed CS defects are often associated with a persistent left superior vena cava; they are rare in isolation and can be challenging to differentiate from an inferior sinus venosus defect even with advanced imaging. We present a patient who had a comprehensive preoperative workup with cross-sectional imaging and 3D reconstruction, who ultimately received a final diagnosis intraoperatively due to transesophageal imaging.

Case Presentation

An asymptomatic 63-year-old male underwent imaging for a cardiac murmur. Transthoracic echocardiogram showed right-sided enlargement with left-to-right interatrial shunt. Cardiac MRI showed an inferior sinus venosus ASD, anomalous right-sided pulmonary vein drainage, right ventricular volume overload, and normal biventricular function with a Qp:Qs of 2.8. Intraoperative TEE revealed an absence of tissue between the coronary sinus and the left atrium (Figure 1). A left upper extremity bubble study ruled out persistent LSVC. There was normal pulmonary venous return and normal tricuspid valve function with no annular dilation. Surgical exposure confirmed an unroofed coronary sinus. He underwent CS patch repair and TV commissuroplasty. Post-repair imaging showed no interatrial communication with laminar flow between the unroofed coronary sinus and left atrium (Figure 2). The patient was extubated in the ICU on post-op day one. Initial sinus bradycardia resolved, and he was discharged on post-op day five and was well on follow-up a month later.

Discussion

An unroofed coronary sinus constitutes less than 1% of ASDs. It can be challenging to differentiate from an inferior sinus venosus defect, as both require visualization of the posterior-inferior atrial septum. They both have left-to-right shunt with right-sided volume overload, and the coronary sinus can be dilated in both defects. There is a male predominance, and nearly two-thirds of CS defects are associated with a persistent LSVC. TEE provides superior resolution to thin posterior structures. However, if there is a complete deficiency of the roof of the CS with loss of its tubular structure, it is challenging to differentiate the CS from the left atrium. Cardiac MRI and cardiac CT can provide detailed structural visualization, and 3D models can enhance diagnostic accuracy. Despite these advanced imaging techniques, definitive diagnosis remains a challenge. Surgical repair remains the primary treatment approach; however, percutaneous techniques are being evaluated. In this case, the patient’s coronary sinus ostium was closed, leaving a communication

between the coronary sinus and left atrium, resulting in an inconsequential residual right-to-left shunt. Postoperative complications are low, with less than 3% incidence of atrial fibrillation, and less than 2% incidence of complete heart block requiring a permanent pacemaker.

Images

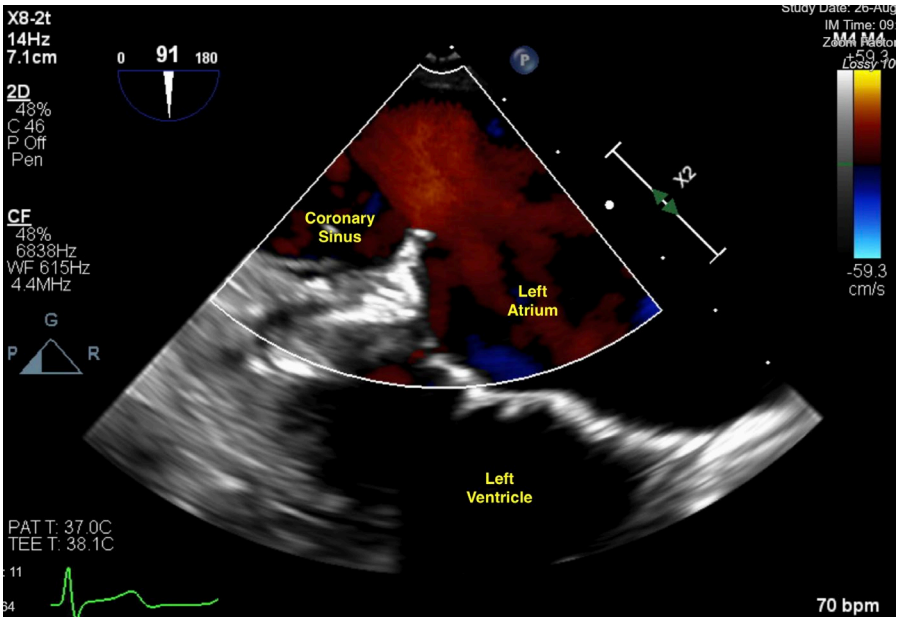


Figure 1: Pre-CPB TEE mid-esophageal 2C view showing partial unroofing of the coronary sinus

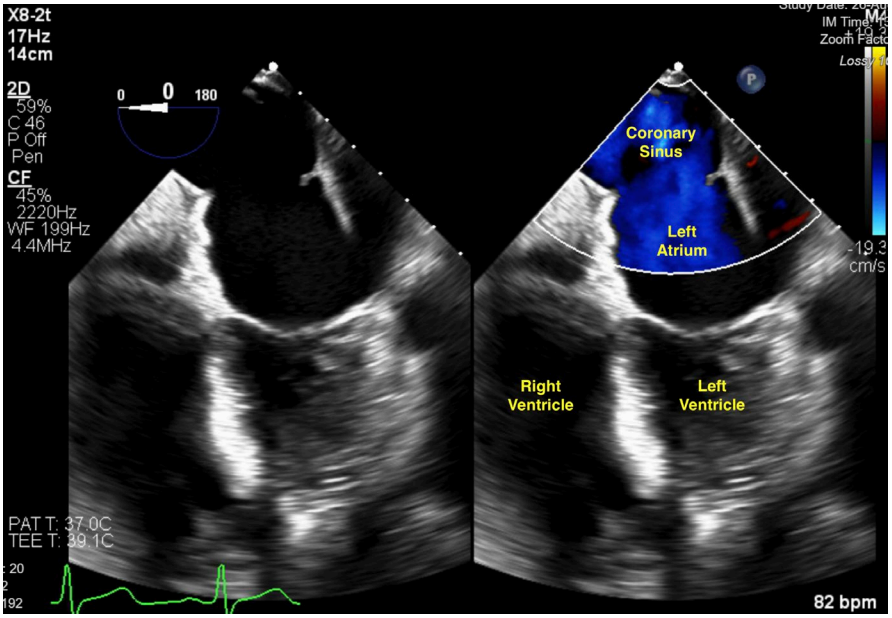


Figure 2: Post-CPB TEE mid-esophagal 4C view showing no interatrial communication with residual flow between the unroofed CS and the left atrium

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#FR 2 – Kiosk 14 – Poster 4

Electrical Storm during Primary Percutaneous Coronary Intervention A Case for Accessible Algorithms and Wider Provision of Extracorporeal Cardiopulmonary Resuscitation?

Presenting Author: Alexander Bell, MBChB, BSc, FRCA, St Bartolomew's Hospital London

Introduction

Electrical storm (ES) is defined as 3 or more episodes of sustained ventricular arrhythmias (VAs) separated by at least 5 minutes requiring interventions to terminate[1]. ES is often a cardiac emergency, most likely to occur in patients with structural or primary electrical heart disease, but can also be due to acute cardiac ischaemia.

Case Presentation

A male in his 60s underwent primary PCI to an occluded right coronary artery following VF out of hospital cardiac arrest. Revascularisation was successful despite periodic sustained ventricular fibrillation (VF) episodes. These were initially managed with defibrillation, 300mg amiodarone and adrenaline as per Advanced Life Support (ALS) guidelines. Electrical storm (ES) developed, with periodic and sometimes refractory VF episodes. Defibrillation with vector change and CPR were performed in VF. Escalating intravenous (IV) pharmacological therapy were given with optimised electrolytes: metoprolol 5mg, magnesium 5g and 1mg/kg of lidocaine. Electrophysiology specialists recommended a lidocaine infusion and a re-look angiogram, finding no ongoing cause for ischaemia. The depth of sevoflurane anaesthesia was increased and opiates given to blunt sympathetic tone and myocardial excitability. After 30-40 minutes sinus rhythm with good cardiac output was persistent. Following intensive care admission and a 24-hour amiodarone infusion, the patient made a good recovery and was discharged after 8 days neurologically intact.

Discussion

This case goes beyond current resuscitation guidelines but demonstrates how good quality ALS with specialist input can result in a favourable outcome. Recent published guidance does give some advice for the management of haemodynamically unstable ES. A 2024 European Heart Rhythm Association consensus statement recommends IV amiodarone or lidocaine following 3 defibrillation attempts alongside ALS in unstable ES, with consideration for extracorporeal cardiopulmonary resuscitation (ECPR) in selected patients[1]. This also mentions lidocaine may be useful in the context of acute ischaemia. 2022 European Society of Cardiology (ESC) guidance provides a flowchart recommending beta blockade and amiodarone for VAs in the context of acute ischaemia, also advising replenished electrolytes and that deeper sedation may reduce episodes. IV lidocaine should then be considered and ECPR for refractory cases[2]. Given myocardial ischaemia is a common reversible cause of cardiac arrest, alignment of this guidance and increasing accessibility by incorporation into resuscitation guidelines could be of benefit to patients. During the prolonged resuscitation of our case, extracorporeal cardiopulmonary resuscitation (ECPR) was felt to be appropriate in line with published guidance. However, as this occurred out of hours, the time to activate the

team had taken the patient out of the intervention window. The feasibility of a United Kingdom 24-hour ECPR service has been explored[3]. Sufficient numbers of refractory inpatient cardiac arrests occur to justify it, but to do so represents a significant financial and logistical challenge.

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#FR 2 – Kiosk 14 – Poster 6

Contained Atrioventricular Groove Disruption Presenting as Hemorrhagic Tamponade After Bioprosthetic Mitral Valve Replacement

Presenting Author: Naomie Ranatunge, MD, Fellow - Cardiac Anesthesia, University of Washington

Introduction

Atrioventricular (AV) groove disruption is a rare (1.2%) but catastrophic complication of mitral valve surgery, typically resulting from annular fragility, endocarditic destruction, or technical factors during valve implantation. Mortality exceeds 75%, and most cases require emergent repair on cardiopulmonary bypass (CPB). Select patients may present with a contained AV groove rupture that limits free rupture and may allow for a temporizing, conservative approach. Early recognition through transesophageal echocardiography (TEE) and meticulous intraoperative decision-making are critical for survival.

Case Presentation

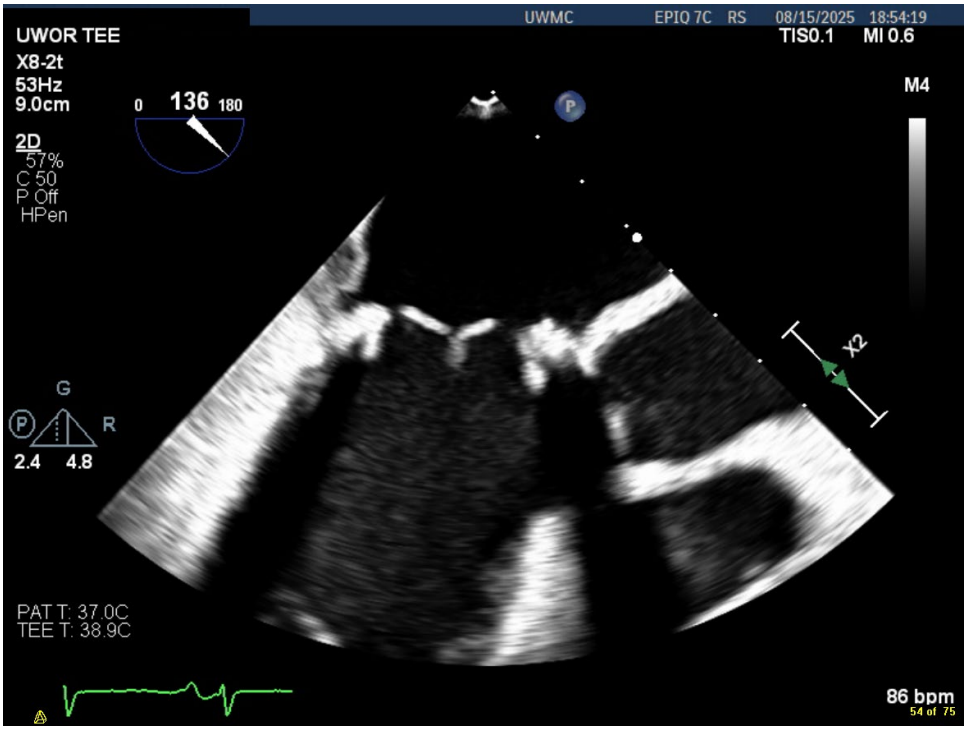
A 35-year-old woman with a history of IV drug use and MSSA endocarditis underwent trans-septal bioprosthetic mitral valve replacement and primary ASD repair. She was discharged to a nursing facility but returned three weeks later with coffee-ground emesis, shock, and profound coagulopathy (Hgb 5.1 g/dL, INR >12, lactate 23 mmol/L). Bedside echocardiography revealed a large pericardial effusion with tamponade physiology. Chest CT showed large hemopericardium with no active extravasation (Figure 4). Emergent pericardiocentesis was not performed due to the location of the effusion. After rapid resuscitation with blood products and prothrombin complex, she was transported to the OR for hematoma evacuation and chest exploration. Pre-CPB TEE demonstrated a circumferential pericardial effusion (Figure 3), biventricular failure, normal mitral bioprosthetic valve with no paravalvular leak (Figure 1), and continuous color flow from the left atrium into a loculated posterolateral thrombus adjacent to the mitral annulus, consistent with contained AV groove disruption (Figure 2). The patient was femorally cannulated in anticipation of emergent conversion to CPB. Upon re-entry of the chest, large volume of hematoma was evacuated from the left lateral pericardium, resulting in immediate hemodynamic improvement. Dense clot adherent to the AV groove was intentionally left in place due to the risk of unroofing a sealed rupture. No active bleeding was noted and definitive repair was deferred. Post-evacuation TEE confirmed decompression of the effusion with persistent localized thrombus but stable prosthetic valve function. The patient required brief inotropic and vasopressor support and was extubated on postoperative day 1.

Discussion

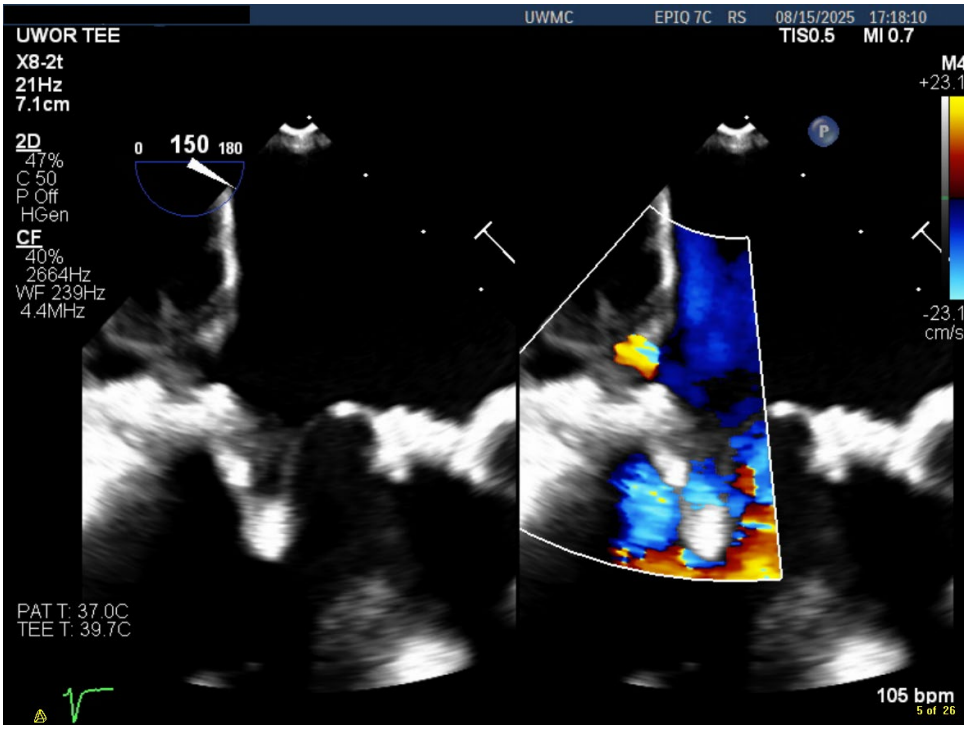
Contained AV groove disruption following mitral valve replacement is a devastating yet potentially survivable event when promptly recognized. The patient's delayed presentation and supratherapeutic anticoagulation likely exacerbated the rupture, which fortunately became self-contained by organized clot. Intraoperative TEE was crucial in diagnosing AV groove disruption by identifying color Doppler flow tracking from the left atrium into the pericardial space. Management required balancing hemodynamic rescue with avoidance of

catastrophic unroofing of the contained rupture, especially in tenuous patients.

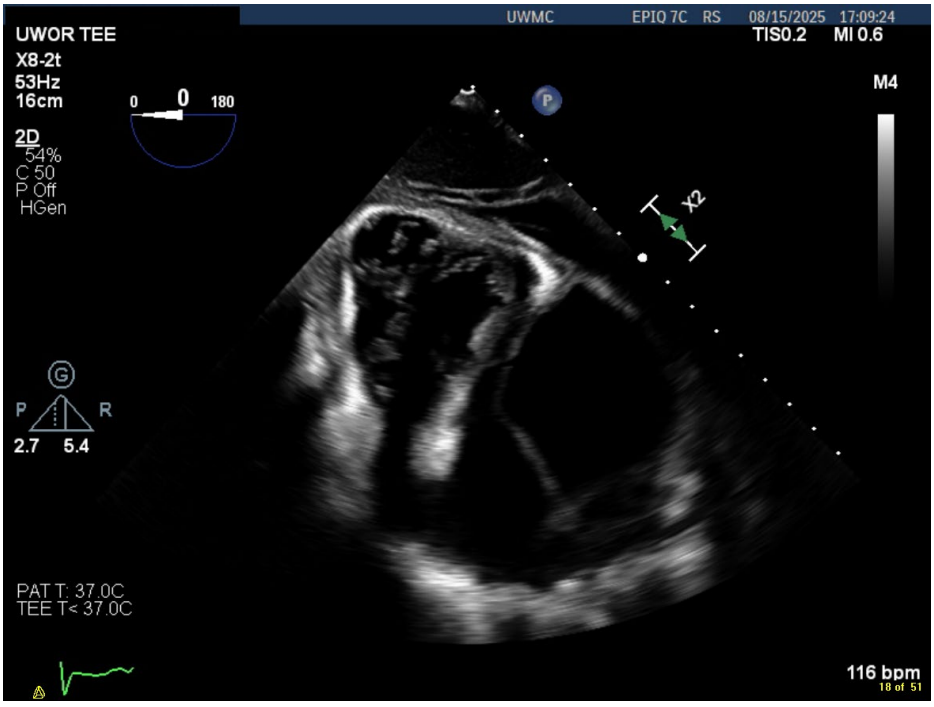
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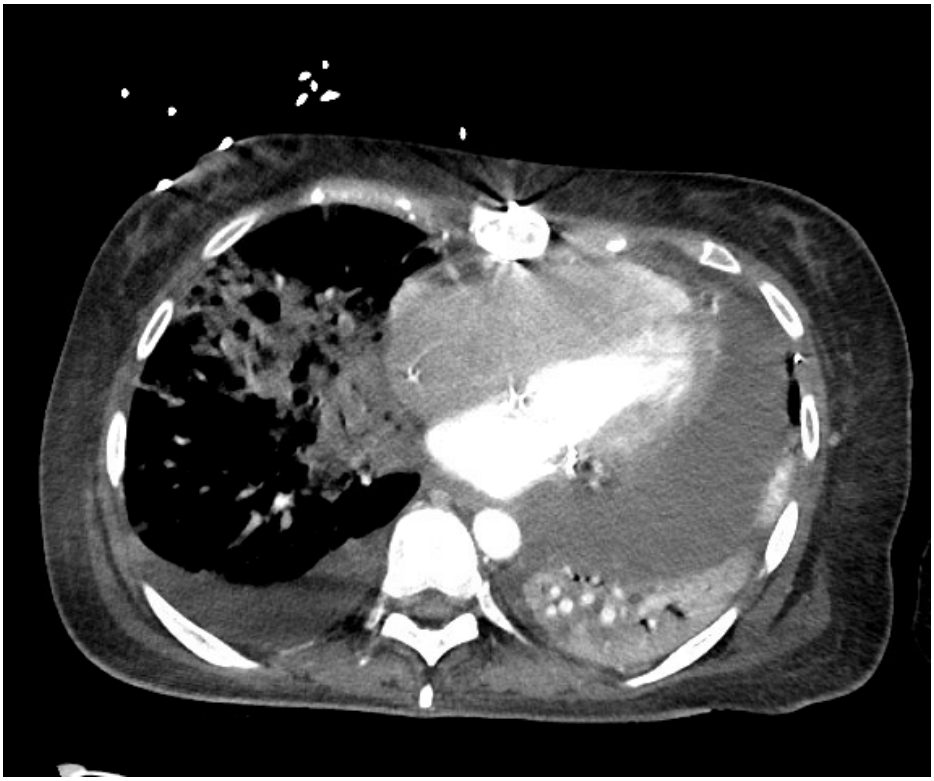
Midesophageal aortic valve long axis with focus on mechanical mitral valve



Color flow at AV groove



Pericardial effusion



CT Scan of large hemopericardium

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#FR 2 – Kiosk 14 – Poster 7

Cardiovascular Collapse in the Anhepatic Phase: A Case of Takotsubo Cardiomyopathy

Presenting Author: Nhat M. Pham, DO, Anesthesiology Resident, University of Cincinnati

Introduction

Takotsubo cardiomyopathy (TCM) is often defined as a reversible stress induced cardiomyopathy in the absence of CAD and is a rare complication of orthotopic liver transplantation (OLT) with a reported incidence of 0.3%. Risk factors include female gender, age greater than 65 years old, Caucasian, and cardiac arrythmias. In this case, TCM developed during the anhepatic phase of an OLT with significant hemodynamic and ethical ramifications.

Case Presentation

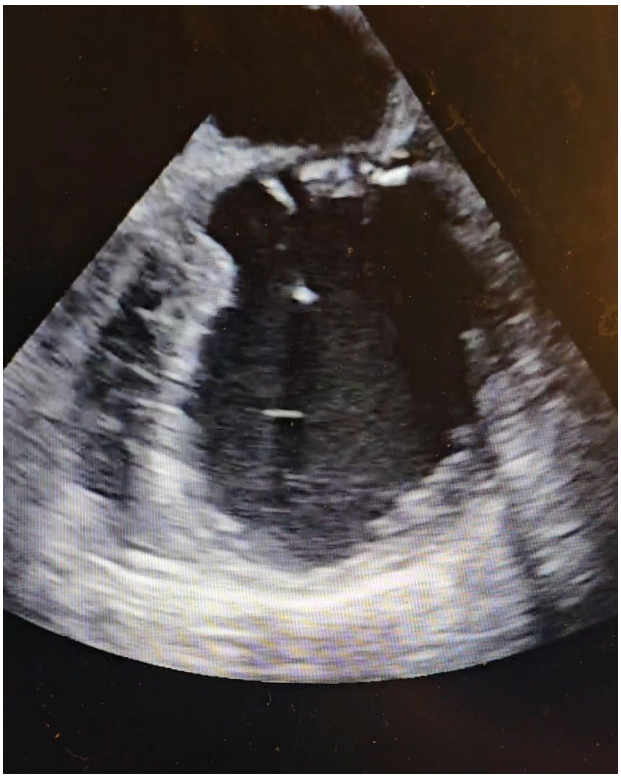
Our case was a 60-year-old female with a history ESLD secondary to Hepatitis C, s/p transjugular intrahepatic portosystemic shunt (TIPS) in 2022, with a MELD score of 13. Her pre-transplant echo showed normal bi-ventricular function, and LHC/RHC revealed non-obstructive CAD with normal filling pressures. The case began with uneventful induction, intubation, radial arterial line, introducer, and Swan-Ganz catheter placement, and she remained hemodynamically stable until the anhepatic phase. Upon clamping of the TIPS followed by the hepatic artery, the patient developed precipitous hypoxia, tachycardia, refractory hypotension, and ventricular tachycardia requiring four attempts at defibrillation before converting to normal sinus rhythm. Acute pulmonary emboli (PE) with RV dysfunction was suspected. A TEE probe, iNO, and VV ECMO were called for and TPA was administered. Hemodynamics improved after TPA, iNO, and with escalating vasopressors and inotropic support. Placement of a TEE probe, surprisingly, revealed normal RV function without obvious PE. The LV, however, showed an EF ~20% with signs of apical allooning concerning for TCM. Given severe LV failure, mechanical circulatory support (MCS) would be required to tolerate hepatic reperfusion. However, she would be ineligible for transplant if she received MCS, but she was already anhepatic with poor prognosis without completing the transplant. The difficult decision was made to temporarily close while still anhepatic in hopes of rapid cardiac recovery. However, in the ICU, she developed bleeding due to expected coagulopathy and further decompensated. After family discussions, she was transitioned to comfort care and passed away.

Discussion

The exact pathophysiology of TCM is unclear but is thought to be due to stress and elevated catecholamines resulting in direct myocardial dysfunction. Management is primarily supportive, and LV recovery can occur over hours up to weeks. In OLTs, the most stressful phase is often cited as reperfusion when the heart receives a bolus of cold, acidotic, and potassium-rich blood from the newly transplanted liver. However, our patient experienced cardiac dysfunction immediately after going anhepatic. Our leading theory is clot in the TIPS embolized when clamped, resulting in PE, hypoxia, and hypotension leading to TCM. While we did not see RV dysfunction or clot on TEE, this occurred after TPA administration, which could have potentially dissolved a clot and improved RV function. This case highlights not only the importance of close cardiac

monitoring during OLT, as sudden complications may occur at any phase, but also posed a unique ethical dilemma reinforcing the essential nature of multidisciplinary discussions to navigate complex clinical and ethical situations that may arise in these high-risk cases.

Images



LV Apical Ballooning on TEE during OLT concerning for Takotsubo Cardiomyopathy

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#FR 2 – Kiosk 15 – Poster 1

RV Failure when Inotropic Support is Contraindicated due to Ventricular Tachycardia in Emergent LVAD Implantation

Presenting Author: Zachary J. Theiler, DO, Anesthesiology Resident, University of Kansas Medical Center

Introduction

Refractory ventricular tachycardia (VT) in the setting of severe ischemic cardiomyopathy significantly complicates anesthetic management for patients requiring mechanical circulatory support (MCS). When inotropes exacerbate malignant arrhythmias, both perioperative stabilization and separation from cardiopulmonary bypass (CPB) become exceptionally challenging, especially when right ventricular (RV) support is needed. This case illustrates the interplay between refractory VT, acute RV failure without inotropic support, and the need for rapid escalation of MCS, including the critical use of TEE to confirm adequacy of support, during combined pulmonary embolectomy and emergent LVAD implantation.

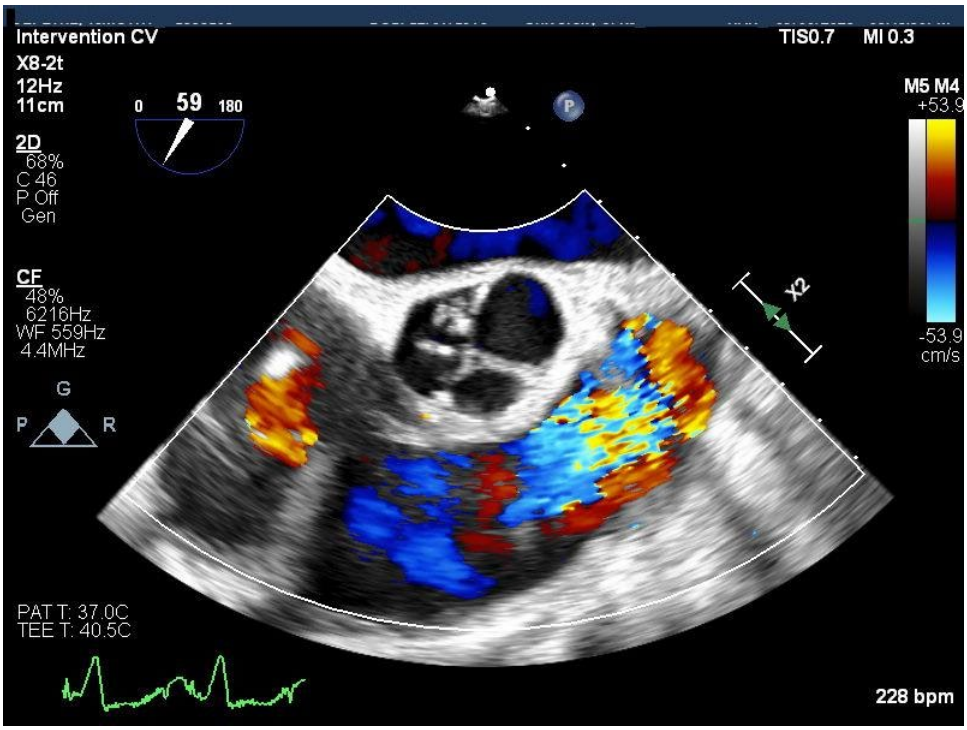
Case Presentation

A 54-year-old male with a history of a massive anterior myocardial infarction complicated by VT storm and cardiac arrest status post VA-ECMO initiation presented for emergent LVAD placement and pulmonary embolectomy for worsening hemodynamic instability in the setting of an acute saddle pulmonary embolism (PE). Anesthesia was induced, arterial and venous lines were replaced, and the patient proceeded onto CPB uneventfully. Attempts to wean from CPB were impeded by severe RV dysfunction following LVAD implantation. Persistent RV failure despite maximum vasoactive support and inhaled nitric oxide necessitated placement of an RVAD. Post-CPB, the patient continued to exhibit refractory VT requiring multiple defibrillations, so dobutamine and epinephrine were discontinued with improvement in VT. Hemodynamic stability was achieved only with avoidance of inotropes, implementation of biventricular mechanical support, and maximum vasopressor support. After initial chest closure, TEE assessment demonstrated new severe pulmonic regurgitation and inadequate RVAD flow, prompting chest reopening and RVAD cannula repositioning. The chest was reclosed, and the patient was transported to the ICU on biventricular MCS. On post-operative day one, CT imaging revealed multiple embolic strokes and ischemia in multiple limbs. Given minimal expectations for meaningful recovery, the family transitioned to comfort care.

Discussion

This case underscores the hemodynamic complexity created when inotropes required for RV support simultaneously exacerbate malignant arrhythmias. Anesthetic management required management of coagulopathy from prolonged CPB run, TEE assessment of MCS performance with re-positioning of the RVAD cannula, and transition from pharmacologic support to alternative methods to maintain appropriate hemodynamics. This complex case highlights the importance of early recognition of inotrope-induced arrhythmogenesis and the need for multidisciplinary intraoperative coordination and communication when escalating therapies.

Images



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#FR 2 – Kiosk 15 – Poster 2

"When Flow Fails": A Massive Aortic Thrombus Associated with Impella CP—A Surprising Echocardiographic Finding Leading to End-of-Life Decisions in a High-Risk, Low Cardiac Output Patient

Presenting Author: Frumentia M. Leon, MD, Fellow, McGaw Medical Center of Northwestern University

Introduction

Reconfiguration of a femoral Impella to an axillary Impella device is commonly performed to achieve early mobilization, reduce the risks of infection, reduce bleeding complications, and achieve higher available flows.[1] These advantages are attractive in patients requiring more extended hemodynamic support as a bridge. The benefits of these devices must, however, be weighed against potential complications.

Case Presentation

A 78-year-old man presented to the OR in cardiogenic shock due to ischemic cardiomyopathy. With an in-situ femoral artery Impella CP and VA-ECMO support, the operative plan was reconfiguration to an axillary Impella 5.0. Other significant past medical history included hypertension, hyperlipidemia, chronic kidney disease, GERD with dysphagia, and a history of liver transplant maintained on immunosuppressants, as well as dual regimen cangrelor and heparin infusion for anticoagulation. Induction and maintenance of general anesthesia were uneventful. Intraoperative LV EF was reported as 15% with severe mitral and tricuspid regurgitant valve disease. After removal of the femoral Impella and trans gastric assessment of left ventricular function, evidence of a large lesion on the posterior wall of the thoracic descending aorta concerning for thrombus vs. dissection flap with a thrombosed false lumen was apparent on TEE. This thrombus forced considerations of a few scenarios. Was it previously associated with the in situ Impella CP? Was this massive thrombus a consequence of an acute dissection associated with the removal of the device?

Further contemplation prompted:

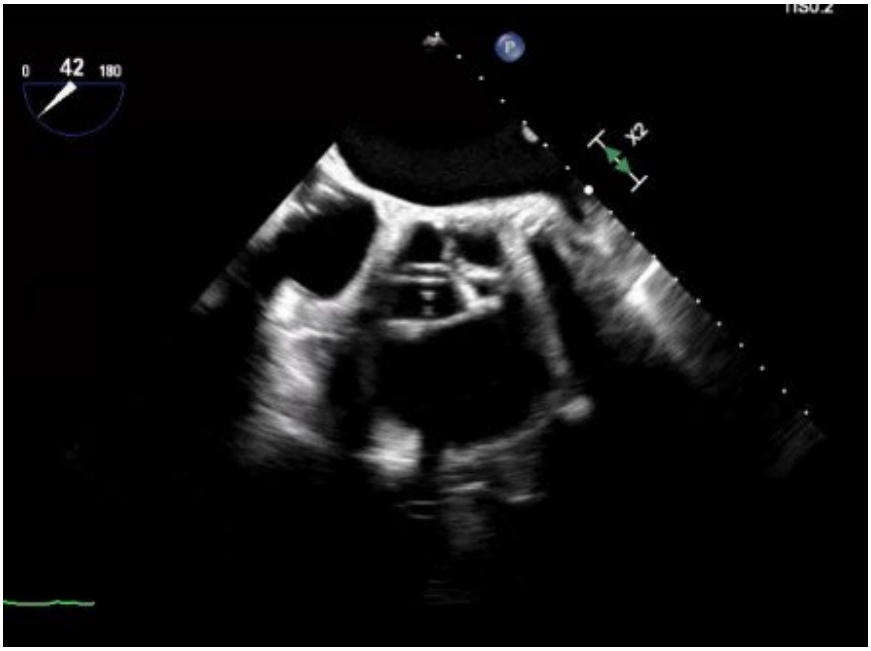
1. surgical decision making leading to aborting placement of the axillary Impella,
2. Further investigation with a gated CT Chest for delineation of a possible dissection
3. family discussions of perioperative and end-of-life decision making toward palliative care.

Discussion

Low cardiac output states and the presence of Impella devices are both associated with an increased risk of thrombosis. While an Impella is designed to improve cardiac output, it creates areas of turbulence and shear stress, which promote clot formation within the device and in the aorta. This, combined with increased positive hypercoagulability risk factors such as cardiogenic shock, other indwelling vascular devices, immobility, and critical illness, supports an environment for massive thrombosis. Although studied to decrease mortality in cardiogenic shock, these potentially devastating vascular complications require thoughtful consideration.[2] High-risk PCI for patients with significant coronary artery disease can require mechanical support to support hemodynamic stability. In the literature, there are only 3 case reports

describing patients with Impella CP who developed aortic thrombosis.[3] TEE has proven to be a valuable tool in the detection and management of thrombotic complications with Impella. Confoundingly, there are no standardized protocols to guide anticoagulation when Impella is used alongside VA-ECMO, and targets often vary by institution and are determined by clinicians. Decision-making after diagnosis of an aortic thrombosis is multidisciplinary, and strategies should be decided based on further complications such as total occlusion, fatal embolism, significant hemorrhage, and overall clinical picture.

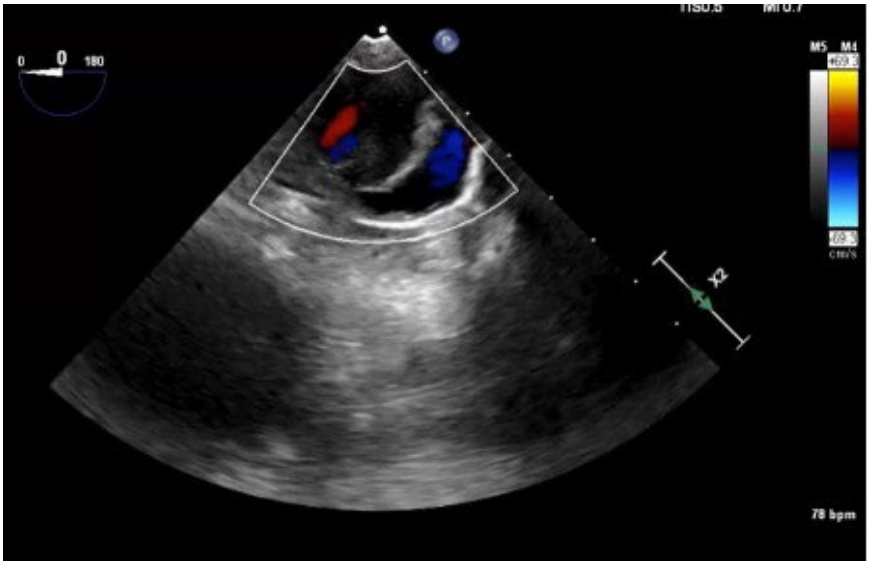
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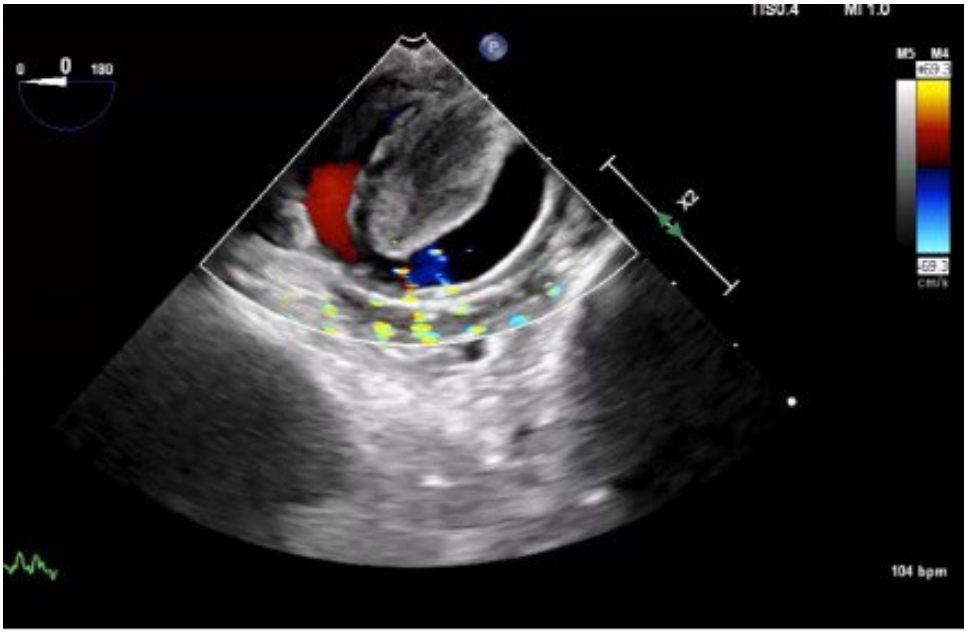
Femoral Impella CP in the Aortic Valve seen in the Midesophageal Aortic Valve Short Axis View



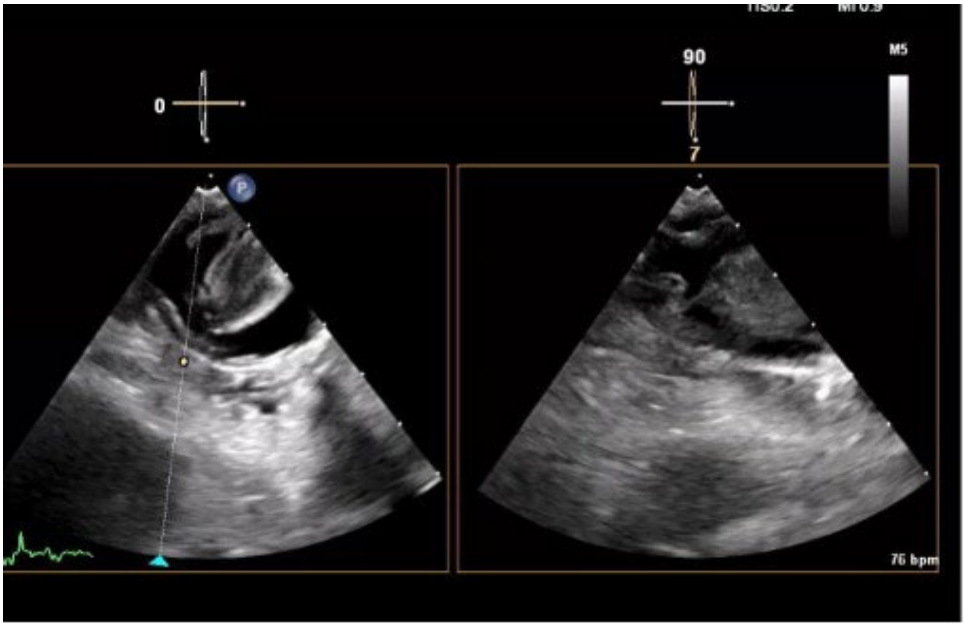
Femoral Impella CP Inlet within the Left Ventricle and Outlet Distal to the Aortic Valve in the Midesophageal Aortic Valve Long Axis View



Descending Thoracic Aortic Thrombus with Color Flow Doppler showing bidirectional flow surrounding the Thrombus



Massive Aortic Thrombosis in the Descending Thoracic Aorta



Orthogonal Views of A Massive Aortic Thrombosis in the Descending Aorta

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#FR 2 – Kiosk 15 – Poster 3

Implication of an Incidental Coronary Artery Anomaly in a Patient Undergoing Aortic Root Reconstruction

Presenting Author: Brianna N. Kilbane, DO, Resident Physician, University of Kentucky

Introduction

Advances in imaging have increased detection of anomalous aortic origins of coronary arteries (AAOCA), congenital variants found in ~1% of adults undergoing coronary CT angiography (Ref1, Ref2). These anomalies, most often involving a coronary arising from the opposite sinus of Valsalva, are associated with sudden cardiac death, ischemia, and arrhythmias. Their significance when incidentally found remains incompletely characterized. We report a case where AAOCA was detected intraoperatively during aortic root and ascending aortic aneurysm repair and outline its clinical relevance.

Case Presentation

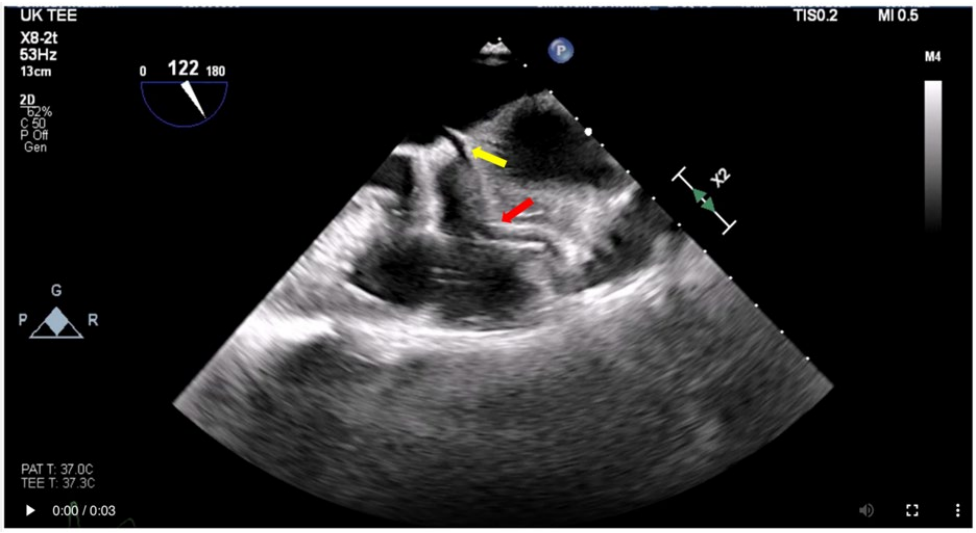
A 37-year-old male with an aortic root and ascending aortic aneurysm underwent root reconstruction, coronary reimplantation, and ascending aorta replacement via sternotomy with CPB. Intraoperative TEE confirmed aneurysm dimensions and revealed a bicuspid aortic valve with calcified leaflets and moderate eccentric regurgitation (Fig1). An unexpected coronary anomaly was also identified; both the right coronary and circumflex arteries arose from the right sinus of Valsalva. The RCA appeared typical, but the circumflex took a retroaortic course before assuming its expected trajectory (Fig1). These findings were communicated to the surgical team and later confirmed directly. During reconstruction, special attention was given to the retroaortic segment to prevent injury, and no high-risk features were found. Although this anomaly was absent from the initial CT report, retrospective review demonstrated it clearly (Fig2). The procedure and postoperative course were uneventful.

Discussion

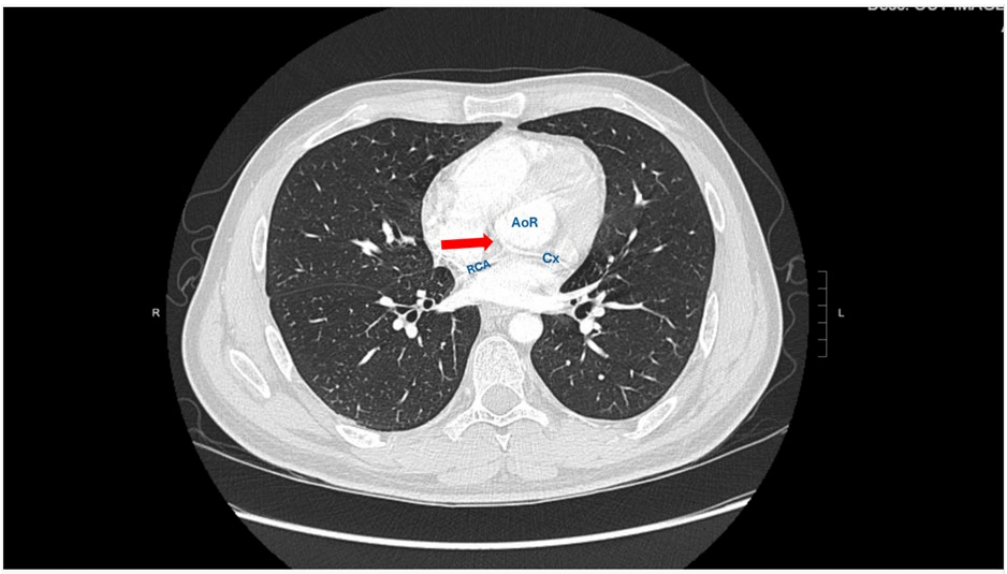
Our case emphasizes the importance of recognizing an anomalous circumflex originating from the right sinus; an uncommon, usually benign variant found incidentally in ~0.3% of patients undergoing CT angiography (Ref2). Because the patient's preoperative risk for CAD was low, angiography was not done. Although anomalies of the circumflex rarely cause ischemia, they may be overlooked on contrast CT and are better visualized on CT angiography. While ischemia is more often associated with left-sided anomalies, right-sided variants with high-risk features, such as an interarterial or intramural course, acute takeoff angle, or slit-like orifice, carry greater risk of ischemia and may require intervention (Ref3). Our patient exhibited none of these, though the retroaortic course placed the vessel at risk during root reconstruction. TEE can delineate the origin and course of anomalous coronaries and can suggest elevated risk, such as through a predominantly systolic Doppler pattern. However, because TEE sensitivity and specificity are limited, ACC/AHA guidelines recommend CT angiography or cardiac MRI for full assessment of AAOCA detected on TEE. Early intraoperative recognition in this case allowed the surgical team to avoid inadvertent injury to the anomalous circumflex. Management of AAOCA depends on symptoms and anatomic risk. Surgical repair is

reserved for symptomatic patients or those with high-risk features, ischemia, or arrhythmias, whereas conservative monitoring is appropriate for asymptomatic, low-risk patients (Ref3). The prognosis for isolated circumflex anomalies is generally favorable, while right-sided anomalies with high-risk features require closer monitoring and individualized management.

Images



Legend Figure 1 (Fig1): A modified mid-esophageal long axis view of the aortic valve, with the probe turned to the left, showing an anomalous origin of the circumflex coronary artery from the right sinus (yellow arrow) and coursing around the aortic root posteriorly (red arrow).



Legend Figure 2 (Fig2): Contrast-enhanced CT image of the chest showing the aortic root (AoR) in cross-section, with the right coronary ostium (red arrow) arising from the right sinus and dividing into the right coronary artery (RCA) and the circumflex artery (Cx), which takes a retro-aortic course.

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Suchodolski A, Głowacki J, Szulik M. Anomalous Origin of Left Circumflex Artery From the Right Sinus of Valsalva in Cardiac Computed Tomography in a Group of 16,680 Patients-Radiologic and Clinical Characteristics. Journal of Clinical Medicine. 2023;12(23):7240. doi:10.3390/jcm12237240.

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#FR 2 – Kiosk 15 – Poster 4

"A Zebra Among the Horses"-Rare Pseudoaneurysm Presentation Following Bentall Procedure with Preassembled Conduit

Presenting Author: Nickhil Patel, MD, Fellow, University of Maryland School of Medicine

Introduction

Treatment and management of patients with concomitant aortic valve and root disease can be challenging. The Bentall procedure is the gold standard for the treatment of patients requiring both aortic valve and root replacement. The advent of preassembled aortic valved conduits has streamlined the procedure, and the incidence of pseudoaneurysm formation in these patients was already rare, occurring in less than 1% of cases[1]. We present a case of a 43-year-old man with previous aortic valve and root replacement with a preassembled aortic valved conduit (Bentall procedure) for aortic valve endocarditis, who was found to have a pseudoaneurysm of the root, requiring reoperation.

Case Presentation

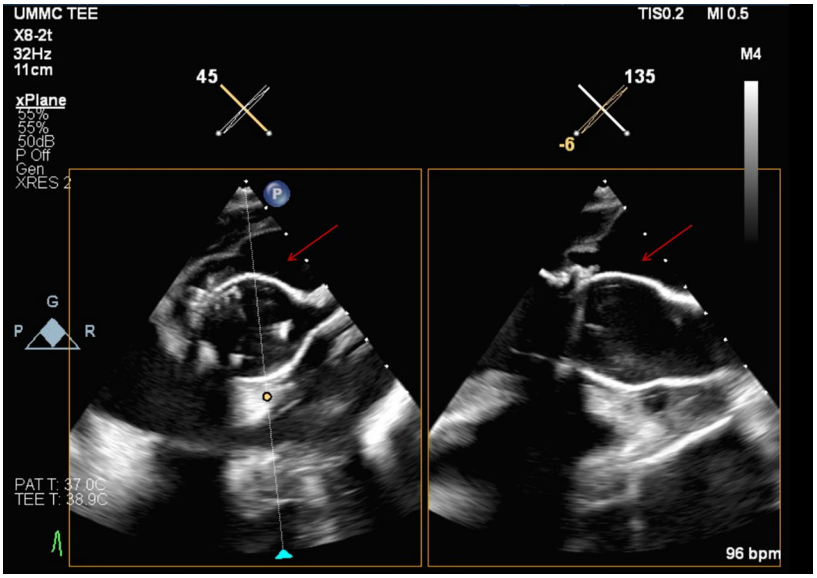
A 43-year-old male with a history of substance abuse and bicuspid aortic valve endocarditis with severe aortic insufficiency, who had undergone a Bentall procedure with a preassembled conduit 1 month prior, presented with chest pain and shortness of breath. Echocardiography revealed partial dehiscence of the aortic valve conduit with pseudoaneurysm formation and moderate aortic insufficiency (Images 1-2). Cardiac surgery was consulted and planned for reoperation. On surgical examination, there was large dehiscence of the valved conduit from the area of the mid left sinus to the mid right sinus with pseudoaneurysm formation. The patient successfully underwent repeat Bentall procedure. Postoperative echocardiogram showed no aortic insufficiency or leaks (Images 3-4). The patient was discharged one week later without complications.

Discussion

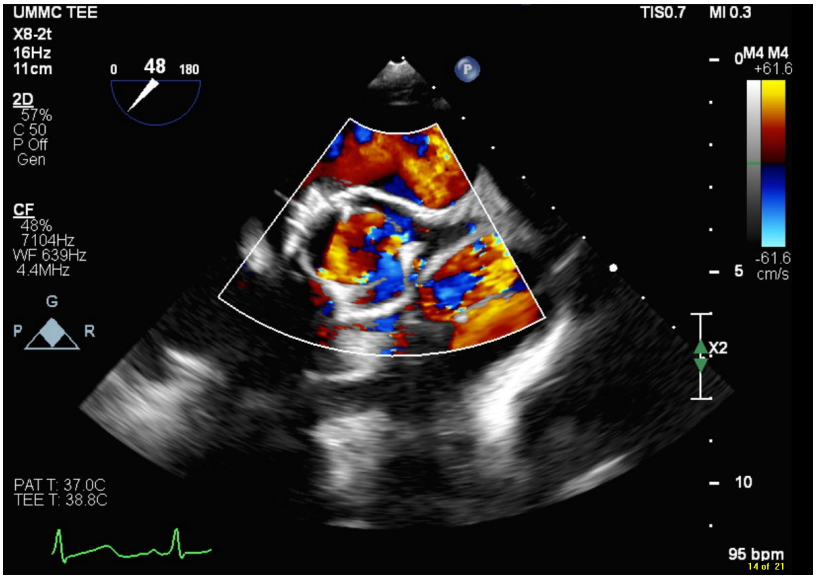
The Bentall procedure replaces the valve, root, and ascending aorta in one composite graft. These grafts were previously assembled during surgery, but preassembled grafts are also now available, reducing possible errors. Pseudoaneurysm is a rare complication of Bentall procedures, occurring in less than 1% of cases, but carries a high mortality rate of up to 17% during reoperation and a five-year survival rate of 77.3% following reoperation[2]. Unlike grafts assembled during surgery, preassembled grafts have seldom been reported to be complicated by pseudoaneurysms. The case is also unique in the location of the pseudoaneurysm. The most common locations are the distal anastomosis, proximal anastomosis, and the coronary buttons[3]. The patient had a rare presentation, with the involvement of the sinuses but not the coronary buttons. The case posed several challenges from an anesthetic perspective. Given the high risk of rupture and massive hemorrhage, there was emphasis on tightly monitoring and controlling hemodynamics during the case. Methadone was administered after induction to provide long-acting analgesia and avoid large hemodynamic changes that can result from surgical manipulation. The patient was also peripherally cannulated, and bypass was initiated before sternotomy given the high risk of bleeding from the

pseudoaneurysm. Longer cardiopulmonary bypass times also made it imperative to monitor coagulopathies and aggressively correct. Due to the variable nature of location and size of pseudoaneurysms, TEE for this case was challenging to completely describe the pseudoaneurysm and its impact on surrounding structures. The case highlights a unique presentation of a rare complication and the challenges it poses to anesthetic management.

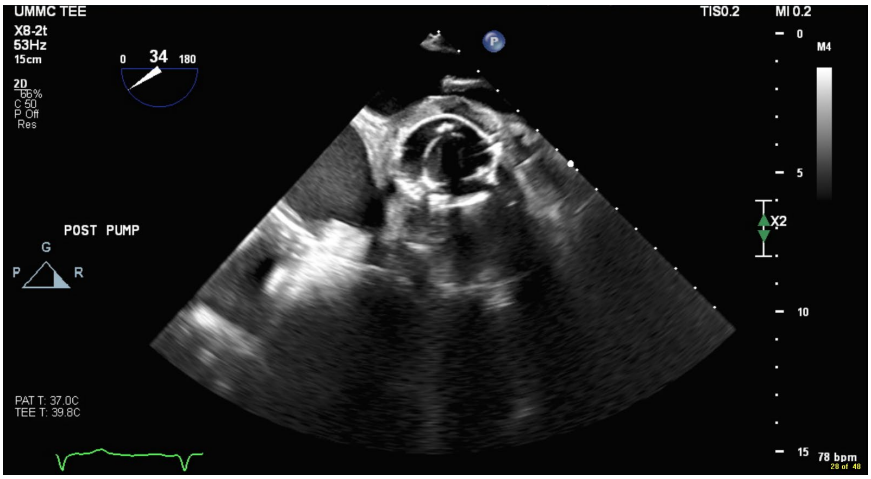
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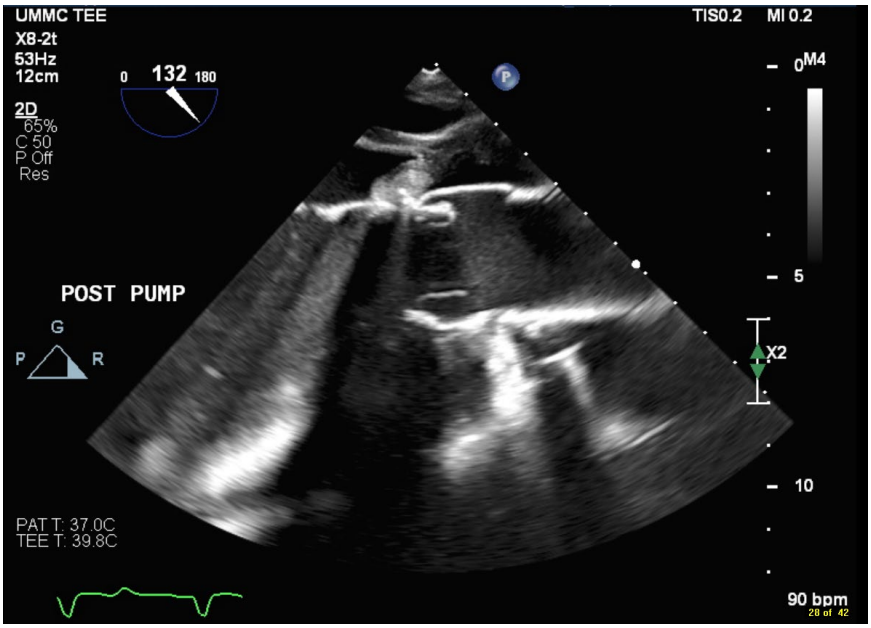
Short (left) and long (right) axis views of the prosthetic aortic valved conduit with partial dehiscence of the graft and pseudoaneurysm formation (red arrows).



Short axis view of the aortic valve and pseudoaneurysm with color doppler showing flow into the pseudoaneurysm space.



Post cardiopulmonary bypass short axis view of the replacement aortic valve. Small residual fluid seen in the peri-aortic space.



Post cardiopulmonary bypass long axis view of the replacement aortic valve and conduit. Small residual fluid seen in the peri-aortic space.

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#FR 2 – Kiosk 15 – Poster 5

RV Airlock Prior to Cardiopulmonary Bypass

Presenting Author: Sarah N. Saenz, MD, Cardiothoracic Anesthesia Fellow, Stanford University

Introduction

Right ventricular (RV) air lock is a rare but serious adverse event occurring prior to the initiation of cardiopulmonary bypass (CPB) that requires prompt recognition and intervention to prevent cardiovascular collapse. Typically, small volumes of air do not cause hemodynamic perturbations and are generally tolerated without consequence. The presence of a large volume of air within the heart, specifically the right heart, can cause acute hemodynamic collapse via reduction of pulmonary blood flow, severe RV dysfunction, and subsequent reduction of LV preload. Additional clinical signs supporting RV embolism are significant declines in blood pressure as a result of lost preload, loss of end-tidal CO2, and increased central venous pressure.

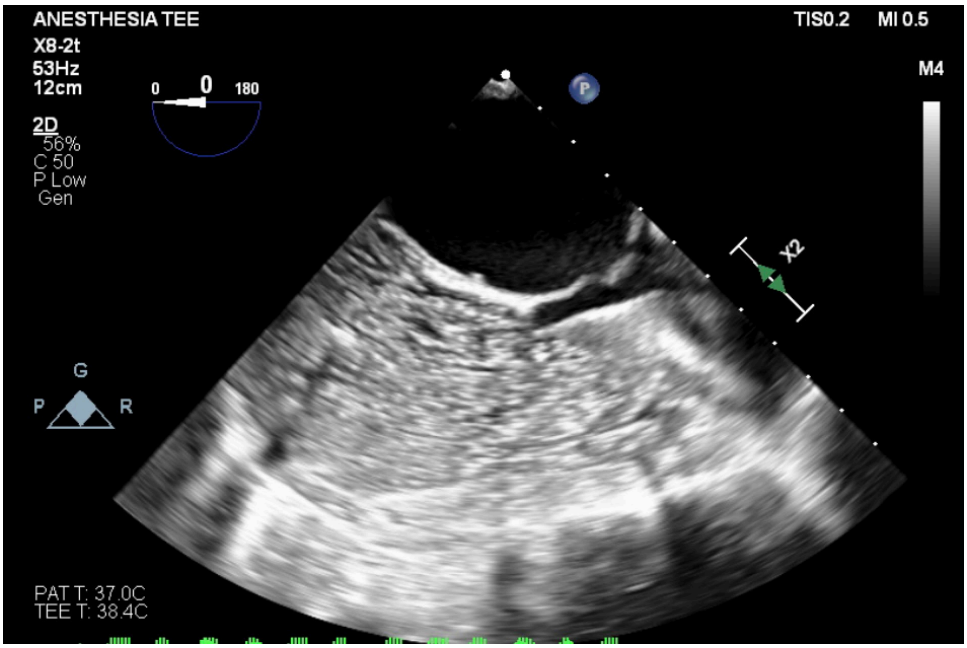
Case Presentation

65-year-old female presenting for minimally invasive mitral valve repair. Shortly after SVC and IVC cannula placement in reverse Trendelenburg positioning, the patient became hemodynamically unstable requiring high dose vasopressors and inotropes. Initial TEE evaluation demonstrated normal RV function and underfilled, hyperdynamic LV. Patient position was leveled. On re-evaluation of TEE, patient had significant air in right atrium (RA) and RV causing an air lock of the right sided chambers. Our perfusion colleagues aspirated from the SVC cannula, which improved hemodynamics and reduced the amount of air in the RA/RV.

Discussion

A large volume of air entrained in the RV can cause significant hemodynamic consequences. In this case, the air entrained in the RV resulted in an airlock that required significant inotropic and vasopressor support to avoid cardiac collapse. TEE is critical in early detection as well as monitoring resolution of the air lock and the effectiveness of treatment. Management included prompt discussion with perfusion colleagues and the surgical team allow diagnosis and restoration of forward flow by removing intracardiac air. Additional interventions included supporting hemodynamics with inotropes, head down positioning, and applying negative pressure to the venous cannulae to remove air. This case is an example of how communication and teamwork during cardiac surgery can protect patients against catastrophic complications and create quick, thoughtful solutions in high stress situations.

Images



Right Ventricle with air entrainment and an underfilled LV.

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#FR 2 – Kiosk 15 – Poster 6

Bridge to Nowhere: Navigating ECMO Decannulation in a patient with Congenitally Associated End-Stage Pulmonary Arterial Hypertension (PAH) and Right Ventricular (RV) Failure

Presenting Author: Premal Desai, MD, Resident, Loyola University Medical Center

Introduction

End-stage PAH can lead to supra-systemic pulmonary pressures, severe RV failure, and rarely, extrinsic compression of the left main coronary artery (LMCA) by a dilated pulmonary artery. This can cause super-imposed left-sided ischemia and cardiogenic shock[1]. PAH from congenital heart disease is under-diagnosed until the patient is in shock. Definitive treatment is transplantation, with VA-ECMO often used as a bridge. Patients with severe PAH on VA-ECMO are high-risk for any procedure, as even small hemodynamic shifts can precipitate abrupt RV collapse and circulatory arrest. We present a challenging case of a patient with PAH secondary to congenital heart disease and LMCA compression who was not a transplant candidate that underwent atrial septostomy followed by VA-ECMO decannulation.

Case Presentation

A 40-year-old woman with heart failure and undocumented childhood congenital heart surgery presented with dyspnea, hypotension and hypoxia. Right heart catheterization showed severe PAH (PA 155/68 mmHg) and low cardiac index (1.08 L/min/m^2). Coronary angiography revealed LMCA compression from a markedly enlarged pulmonary artery (Fig 1). Due to hemodynamic instability, VA-ECMO was initiated. Epoprostenol, sildenafil, macitentan, inotropes and vasopressors were started. ECMO was planned as a bridge to heart–lung transplant, but she was ineligible due to inadequate social support. Bleeding and worsening right leg ischemia prompted decannulation planning. Alongside medical management, an atrial septostomy and LMCA stenting were done to aid in weaning. With her pre-existing disease and RV dysfunction she maintained acceptable oxygenation during weaning trials. With PA pressures remaining suprasystemic, decannulation was planned in the OR under a femoral nerve block. With reduction of ECMO flows she developed hypoxia, hemodynamic collapse, and cardiac arrest. ACLS achieved return of spontaneous circulation. Rescue TEE showed a severely dilated, nearly akinetic RV with minimal output and a right-to-left shunt across the septostomy (Fig 2, Fig 3). Given her goals of care and lack of transplant options, no further escalation was pursued, and she expired in the ICU.

Discussion

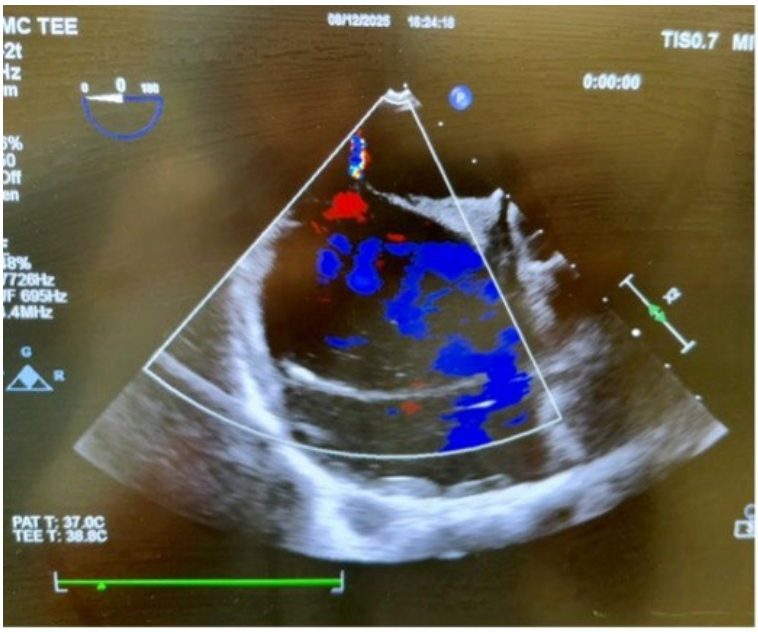
Treatment of longstanding PAH with RV failure is heart-lung transplantation. Persistent congenital left-to-right shunting can drive irreversible pulmonary vascular remodeling; so, late intervention may not reverse right heart structural changes or prevent future RV failure. This case showed the limits of medical therapy alone, as the patient had minimal response to three classes of PAH therapy (epoprostenol, sildenafil, macitentan). LMCA compression, a rare complication of severe PAH, added complexity by causing superimposed left-sided ischemia until stenting[1]. Atrial septostomy was used to decompress the right heart and enhance left-sided filling. A temporizing measure, though it can improve hemodynamics and

intermediate survival[2]. Decannulation posed additional challenges. Local and regional anesthesia were used to avoid loss of sympathetic tone, increased PVR and RV decompensation risks associated with general anesthesia[3]. Management of severe PAH and RV failure requires collaboration among cardiac surgery, critical care and anesthesia teams while aligning interventions with the patient's' goals of care.

Images



LMCA Compression Secondary to Enlarged PA



R to L Shunt Secondary to Atrial Septostomy and Severely Dilated RA



Severely Dilated and Nearly Akinetic RV

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#FR 2 – Kiosk 15 – Poster 7

Pulmonary Torsion After Open Thoracic Surgery

Presenting Author: Richard Bui, M.D., Resident, Baylor College of Medicine

Introduction

Pulmonary torsion is a rare but serious complication that can occur after thoracic or aortic surgery, including open thoracoabdominal aortic aneurysm (TAAA) repair. It involves rotation of a lung lobe around its bronchovascular pedicle, resulting in vascular compromise and potential infarction. The reported incidence of pulmonary torsion ranges from 0.089% to 0.4%, most commonly following lobectomy or major thoracic procedures.

Case Presentation

A 69-year-old patient with a history of ascending aortic aneurysm secondary to an underlying connective tissue disorder associated with a PRKG1 mutation who is status post repair is found to have an Extent-1 thoracoabdominal aneurysm and dissection now undergoes planned thoracoabdominal aortic repair. The re-operation was complicated by dense adhesions involving the left lung, which limited surgical exposure intraoperatively. Even so, the patient initially did well post-op and was extubated two days after surgery. On post-op day three, he developed acute respiratory failure necessitating reintubation. Oxygenation did not improve, and initiation of veno-venous extracorporeal membrane oxygenation (VV ECMO) was required. Plain film demonstrated diffuse left hemothorax as well as hemothorax in the right upper lung. Cardiac catheterization revealed an absence of blood flow to the LUL arteries. He was taken back to the operating room for video-assisted thoracoscopic surgery (VATS), but due to major swelling and obstructed views, a decision was made to convert to an open approach. Intraoperatively, a left upper lobe torsion was identified. While detorsion was successful, blood flow did not return to the affected portion of the lung. Due to nonviable lung tissue and the concern for necrosis, a left upper lobectomy was performed. The procedure proceeded without complication and the patient was returned to the ICU for further care.

Discussion

Left upper lobe torsion following open TAAA repair is exceedingly rare, likely related to extensive lung mobilization and disruption of pulmonary attachments during surgical exposure. Reported risk factors include prior thoracic surgery, altered thoracic anatomy, advanced age, and complex or urgent aortic repairs. Diagnosis can be challenging, as clinical findings such as dyspnea, hypoxemia, and radiographic consolidation are nonspecific. Characteristic CT findings—abrupt vascular cutoff or abnormal lobe orientation—should prompt suspicion. Early identification is critical, as delayed management often results in irreversible ischemia requiring resection. Treatment depends on lobe viability: detorsion may be attempted early, but lobectomy is indicated once necrosis is evident. In this case, detorsion failed due to nonviable tissue, and lobectomy was required. Mortality from lung torsion approaches 8%, emphasizing the importance of prompt recognition. Left upper lobe torsion after open TAAA repair is an uncommon but life-threatening event. Awareness of risk factors, vigilance for postoperative respiratory deterioration, and

expeditious workup are critical to patient survival. If index of suspicion is high, surgical exploration is considered first line treatment as well as definitive diagnosis.

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#FR 2 – Kiosk 16 – Poster 1

Anesthetic Management in Early-Staged Aortic Repair: Combined Coronary Artery Bypass Graft and Total Arch Replacement Followed by Extensive Thoracic Endovascular Aortic Repair

Presenting Author: Karla Adrielle N. Villasin, Fellow in Training, Division of Thoracic and Cardiovascular Anesthesia, Philippine General Hospital, Fellow in Training, Philippine General Hospital

Introduction

Aneurysmal disease of the ascending aorta, arch, and descending thoracic aorta poses major technical and anesthetic challenges. Total arch replacement (TAR) remains preferred for lower-risk patients, while management of distal thoracic disease often requires either a single-stage frozen elephant trunk repair or a staged approach. Staging allows physiologic recovery and collateral network development, lowering spinal cord ischemia risk. Most centers delay TEVAR for several weeks; performing it within the first postoperative week is rare. This report describes early extensive TEVAR on postoperative day (POD) 8 after combined CABG and TAR, emphasizing anesthetic strategies across two physiologically opposing procedures.

Case Presentation

A 60-year-old hypertensive male with a 6.4-cm descending thoracic aneurysm and significant LAD and LCx disease underwent CABG with TAR. Deep hypothermic circulatory arrest (24°C) with selective antegrade cerebral perfusion was used. BIS confirmed electrocerebral suppression and NIRS remained stable. CPB time was 4 h 54 m; cross-clamp time 4 h 34 m. He was weaned on norepinephrine, nitroglycerin, and dobutamine. Postoperative goals focused on graft protection (MAP 65–75 mmHg), coronary graft perfusion, and close neurologic monitoring. He was extubated on POD 2 without deficits and showed improving renal and hemodynamic stability. On POD 8, he proceeded to second-stage TEVAR. Before TEVAR, a spinal drain was placed under light sedation. Under general anesthesia, a long descending segment was stented; a minimal type II endoleak was noted. He was extubated immediately and remained neurologically intact. CSF pressure was kept 10–12 mmHg with MAP ≥90 mmHg to support spinal cord perfusion. He recovered uneventfully and was discharged on POD 7 post-TEVAR.

Discussion

The anesthetic strategy required navigating opposing perfusion goals within a short interval. During TAR, neuroprotection combined systemic hypothermia, selective cerebral perfusion, topical head cooling, and pharmacologic metabolic suppression using a propofol–remifentanil technique. BIS and NIRS guided adjustments throughout cooling and circulatory arrest, ensuring symmetric cerebral oxygenation and adequate metabolic suppression. During rewarming, nicardipine prevented hyperemic surges that could threaten the brain or fresh grafts. Hemodynamic management after combined CABG and arch replacement required maintaining a narrow MAP window that protected the arch anastomoses while preserving coronary graft flow. Continuous arterial waveform monitoring and responsive vasoactive titration were crucial. For extensive TEVAR, anesthetic priorities shifted toward spinal cord preservation. Prophylactic CSF drainage and permissive hypertension were central, targeting spinal cord perfusion pressure ≥70 mmHg. MAP

augmentation was achieved with vasopressors despite recent arch surgery, supported by stable NIRS trends. Streamlined general anesthesia allowed immediate extubation for continuous neurologic assessment. The favorable neurologic and cardiovascular outcomes demonstrate that early staged TEVAR can be safely performed when anesthetic management is tightly aligned with evolving perfusion priorities and neuroprotective goals.

Images

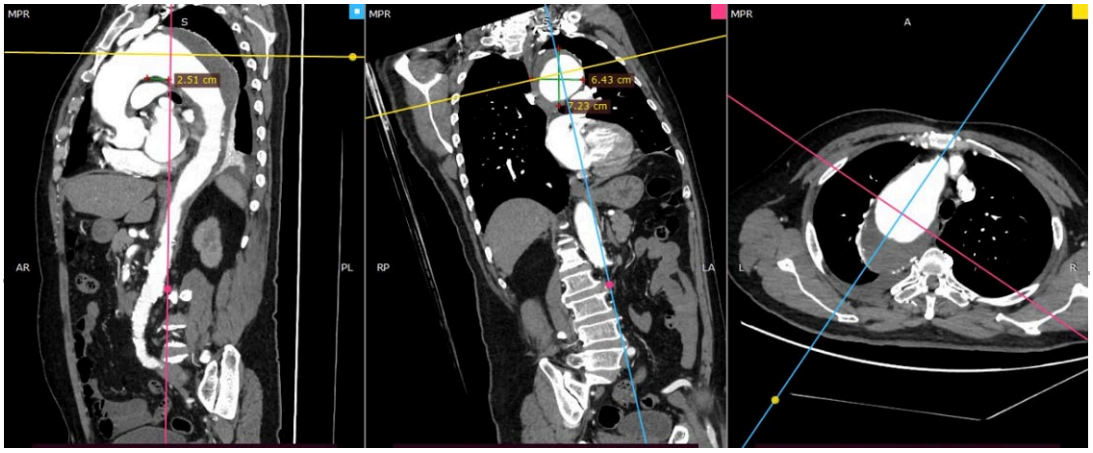


Figure 1. Preoperative CTA demonstrating a 6.4-cm aneurysmal dilation of the descending thoracic aorta, 3 cm distal to the left subclavian artery (sagittal and axial reformats).



Figure 2. Post-deployment digital subtraction angiography showing the thoracic endograft in situ with minimal proximal contrast blush consistent with a type II endoleak

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#FR 2 – Kiosk 16 – Poster 2

Sandwiched between the atria – an unusual pseudoaneurysm and a fourth-time sternotomy for triple valve surgery

Presenting Author: Daniel P. Weigle, DO, Fellow Physician, University of Florida

Introduction

Aortic root pseudoaneurysms (PSA) are rare complications of infectious endocarditis (IE) or prior cardiac surgeries. Redo cardiac surgery confers elevated risk and presents anesthetic, imaging, and surgical challenges. This case depicts complexities in perioperative management of an unusual PSA in a fourth re-sternotomy.

Case Presentation

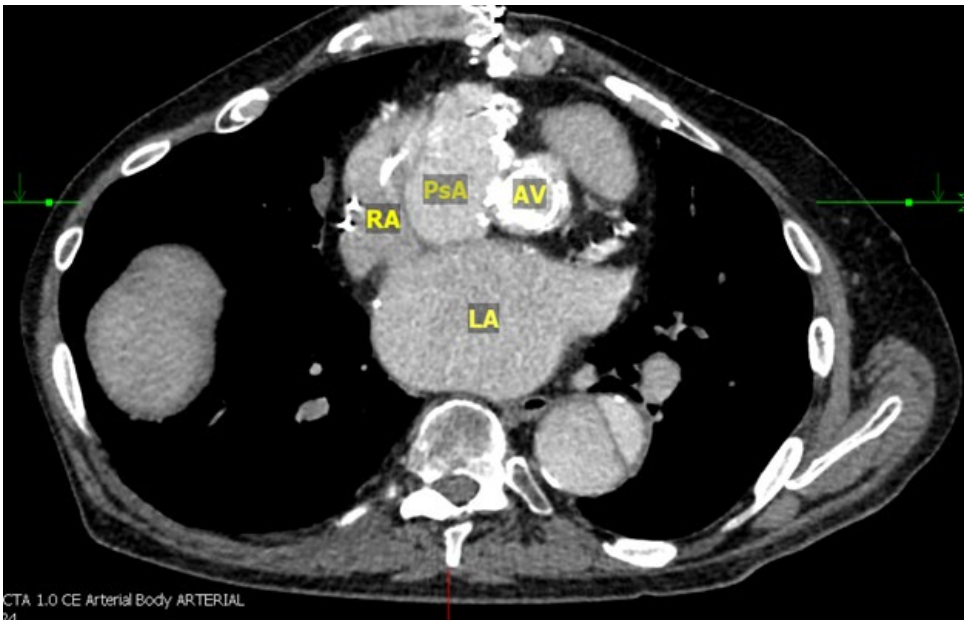
A 69-year-old male with complex cardiac history inclusive of remote IE status-post mechanical AVR and MVR, prior type A dissection repair, HFrEF with EF 25%, and sinus node dysfunction with pacer/ICD underwent TEE for progressive dyspnea. Exam noted a spherical enclosed abscess attached to right atrium and a mitral paravalvular leak (PVL). CT angiogram revealed a 6.9 x 3.7cm subaortic valvular PSA between the right AV annulus and RA. Subacute IE was suspected, yet blood cultures were negative and no other clinical signs of IE were present. Patient initially chose long-term antibiotics due to high surgical risk but later agreed to surgery for worsening dyspnea. Anesthesia was induced with inotropic support to maintain hemodynamics. A technically difficult pre-bypass TEE confirmed the aortic root PSA, sandwiched between right and left atria, without atrial communication. Exact origin was unclear due to artifacts. Other findings included stable mechanical aortic and mitral valves, moderate anterior mitral PVL, dilated tricuspid annulus and no vegetations. Graft proximity to sternum necessitated peripheral femoral cannulation before sternotomy. Apical LV vent was placed via left thoracotomy under TEE guidance. Cardiopulmonary bypass was initiated with hypothermia. Intraoperative findings were atypical for IE. The PSA origin was a small opening at the native annulus level, which appeared long-standing. Interventions consisted of a bioprosthetic mitral valve, Freestyle aortic root with Cabrol grafts, zone 1 debranching with transverse arch replacement, PSA exclusion with bovine pericardial patch, tricuspid ring, ICD removal, and Micra implant. Patient separated from bypass with vasoactive and inotropic support. Coagulopathy was corrected and patient was transferred to ICU. Operative cultures/pathology were negative for infection. Retrospective review of patient's external medical imaging redemonstrated the PSA since his aortic repair 12 years prior, casting doubt on infectious origin. Postoperative course was complicated by protracted instability, respiratory and renal failure, and ultimately patient elected to pursue hospice care after 5 weeks.

Discussion

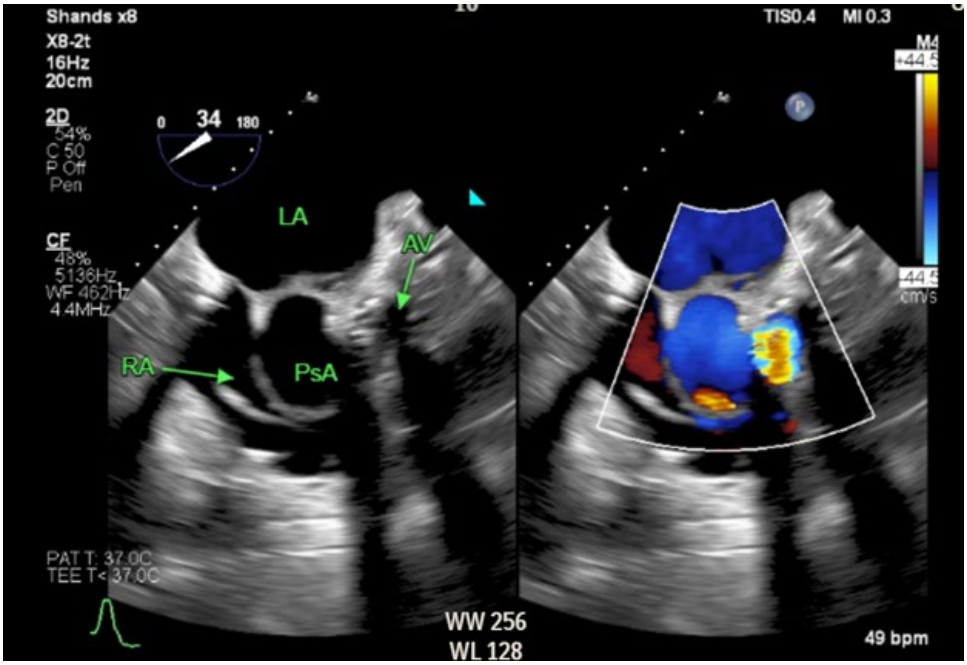
Aortic root PSA can be a life-threatening complication. While data is limited on natural history, evidence suggests differential courses of infectious vs non-infectious PSA. Those secondary to IE often warrant aggressive surgical measures, while stable or non-infectious PSA can be managed conservatively. Though the remote endocarditis history and paravalvular leak suggested an infectious origin in our patient, other

findings were contradictory. Delineating the etiology by multimodal imaging and careful history can drive risk-benefit discussions and informed decision making. Percutaneous closure of both PSA and PVL have been done successfully and may be worth consideration in patients with high surgical risk.

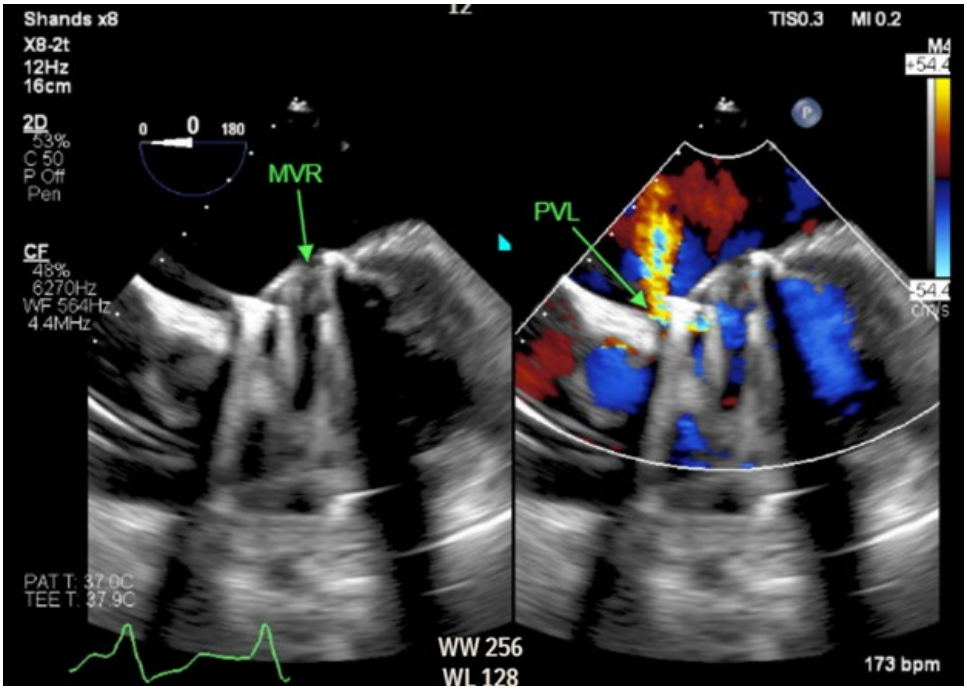
Images



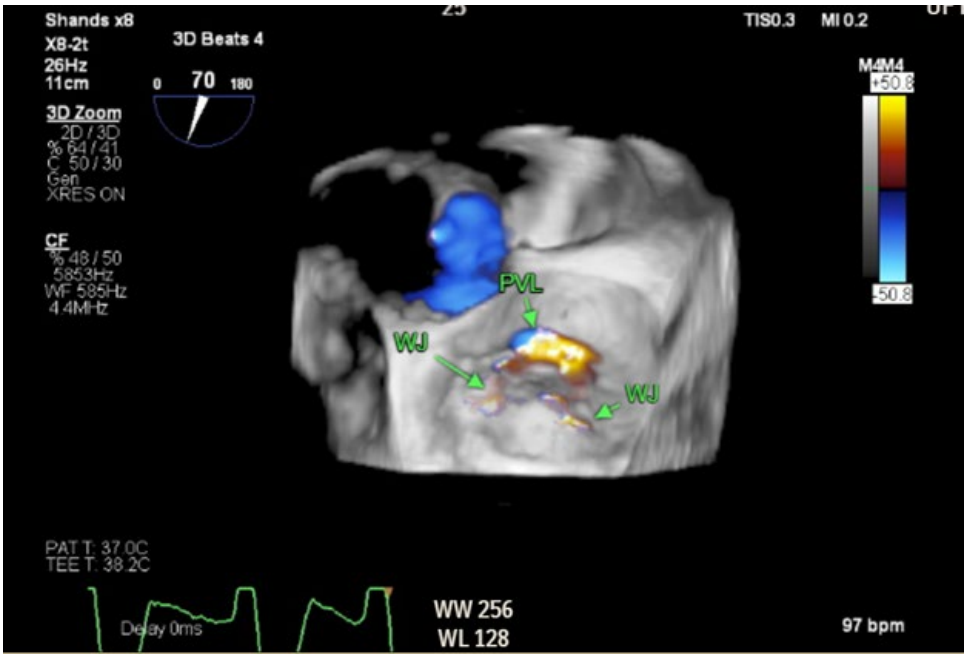
CT Angiogram demonstrating subaortic valvular pseudoaneurysm



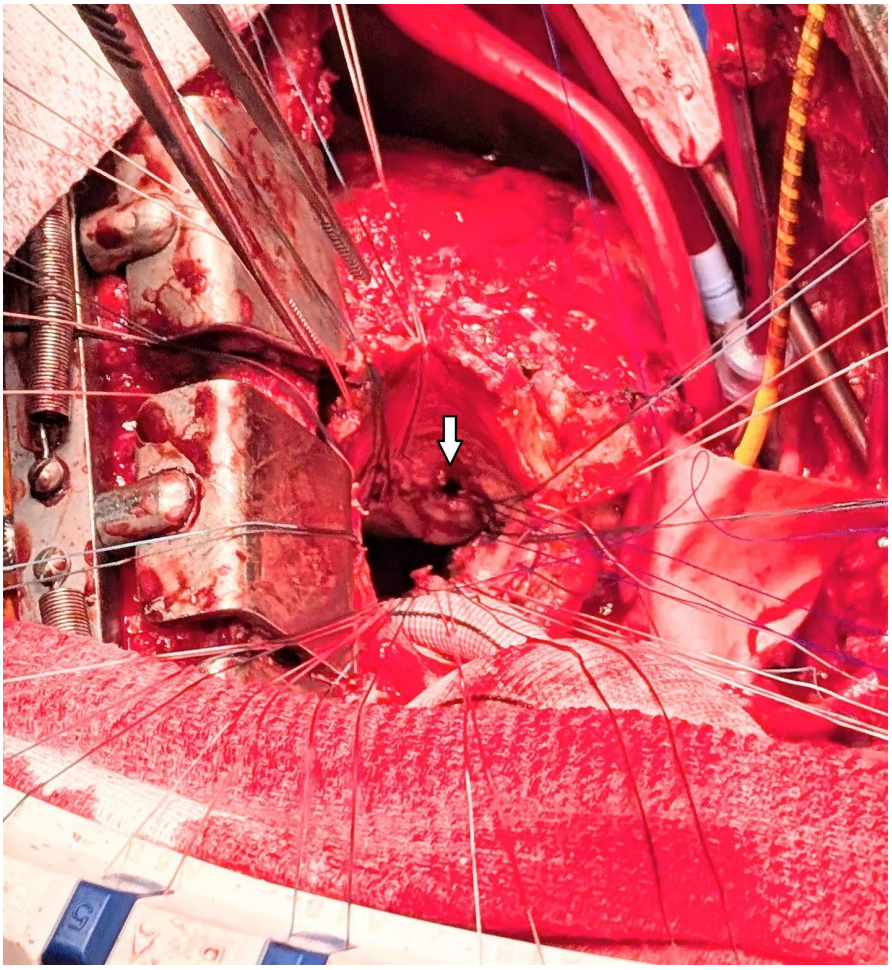
Intraoperative TEE, 2D biplane view of aortic root pseudoaneurysm



Intraoperative TEE, 2D biplane view of mechanical mitral valve paravalvular leak



Intraoperative TEE, 3D view of mechanical mitral valve paravalvular leak



Surgical view of pseudoaneurysm origin

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#FR 2 – Kiosk 16 – Poster 3

Facilitating Transplant Eligibility: A Case of Structured Optimization for Transcatheter Aortic Valve Replacement in Advanced Hepatorenal Disease

Presenting Author: Franco F. Incitti, MD, Resident, University of Rochester Medical Center

Introduction

Patients with concurrent end-stage liver disease (ESLD) and advanced chronic kidney disease (CKD) represent one of the highest-risk groups undergoing structural heart interventions. Multiorgan dysfunction - including coagulopathy, hypervolemia, portal hypertension, and impaired drug metabolism - significantly increases anesthetic complexity. For transplant candidates, timely optimization and valve intervention may be required to meet eligibility criteria. This case highlights multidisciplinary, organ-targeted strategies enabling successful transcatheter aortic valve replacement (TAVR) in a patient with severe hepatorenal dysfunction.

Case Presentation

A 67-year-old male with HTN, CAD (s/p PCI with BMS to LAD 1999), Afib, T2DM, MASLD cirrhosis (c/b encephalopathy, refractory ascites, hepatorenal syndrome, portal hypertensive gastropathy, and esophageal varices s/p banding), and CKD Stage IV presented with acute on chronic anemia and worsening renal dysfunction during outpatient liver transplant evaluation. His anemia was attributed to occult gastrointestinal losses. He underwent further inpatient liver transplant evaluation/optimization and was ultimately initiated on hemodialysis. A dobutamine stress echocardiogram revealed moderate aortic stenosis (PV 3.5, MG 27, AVA 1.02) without evidence of ischemia. Anesthesiology recommended valve intervention prior to transplant listing. For TAVR optimization, he received dialysis the night before the procedure. Severe dyspnea while supine improved after a 4L paracentesis the morning of the procedure. Preoperative labs were notable for K 4.1, H/H 8.1/25, Plt 34K, and INR 1.6. He received two units of FFP, one unit of platelets, and DDAVP preoperatively. TAVR was performed under moderate sedation with low-dose propofol, remifentanil, and phenylephrine infusions. He had stable hemodynamics throughout the case, recovered uneventfully, and was listed for transplant.

Discussion

Management of patients with ESLD and CKD requires individualized optimization. Hepatic considerations include addressing ascites to improve respiratory mechanics, goal-directed coagulopathy correction, and judicious sedative use to prevent encephalopathy.1 Cirrhosis-associated coagulopathy from thrombocytopenia, platelet dysfunction, and reduced synthetic capacity necessitates targeted transfusion strategies and DDAVP for uremic dysfunction, though blood products only partially correct liver-related coagulopathy and formal INR/platelet thresholds for TAVR do not exist. Renal considerations include pre-procedural dialysis for euvolemia and electrolyte stability, and careful drug selection to avoid accumulation and hypotension.2 Physiologic goals for ESLD, CKD, and AS often conflict - AS requires preserved preload, while cirrhosis causes third spacing and effective hypovolemia, limiting safe ascites removal. Avoiding

general anesthesia reduces vasodilation, preserves preload, and minimizes airway manipulation, all of which help mitigate risk in this population with limited physiologic reserve. This case demonstrates how with disease-targeted optimization - dialysis for volume control, vigilant paracentesis, targeted coagulopathy correction, and careful anesthetic titration - TAVR can be safely performed despite advanced multiorgan dysfunction.

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#FR 2 – Kiosk 16 – Poster 4

Successful anaesthetic and surgical management of Cardiogenic Shock with Heart Transplantation and Aortic Repair in a Single Operative Session

Presenting Author: Daniel Ruiz-Pardo, Fellowship Cardiovascular and Thoracic Anesthesia, Fellowship Cardiovascular and Thoracic Anesthesia, Universidad El Bosque / Fundación Clínica Shaio

Introduction

Simultaneous heart transplantation and repair of Stanford Type A aortic dissection is extremely rare and complex, requiring precise timing, surgical planning, and perioperative management. We report a 44-year-old male in cardiogenic shock with severe aortic and mitral regurgitation, coronary artery disease, and chronic Stanford Type A dissection who underwent combined orthotopic heart transplantation and hemiarch repair in one session, demonstrating feasibility.

Case Presentation

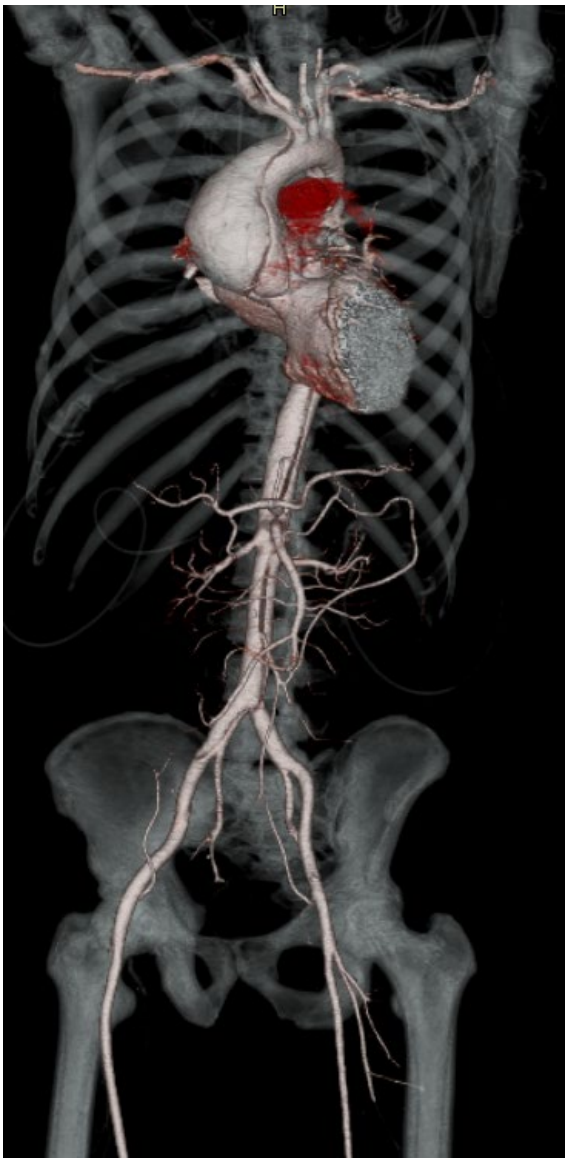
We present a patient with advanced heart failure due to severe valve regurgitation and coronary disease, complicated by chronic Stanford Type A dissection progressing to stage D (SCAI). TTE showed severe aortic and mitral regurgitation with LVEF 16%. CT confirmed dissection from the aortic root to the left femoral artery, involving carotids (Image1,2). Given critical status, he was listed for urgent transplant; a donor became available within 48 hours. At first, surgical plan was combined transplantation and ascending aorta reconstruction. Recipient repair began first, but donor instability required immediate retrieval before completion of aortic work. Advanced anaesthetic monitoring plan included ECG, PAC, NIRS, BIS, and TEE. Total intravenous anaesthesia was administered using dexmedetomidine, fentanyl, and propofol, titrated to maintain BIS between 45–55. Hemodynamic goals (MAP 60–70 mmHg, HR 60–80 bpm, SpO_2 94–96%) were maintained with dobutamine, vasopressin, and norepinephrine. TEE revealed a dissection flap at the left coronary cusp (Image 3) extending into the descending aorta, severe aortic regurgitation, (Image 4) LVEF 12%, severe mitral and tricuspid regurgitation, and re-entry communication in the descending aorta (Image 5). Median sternotomy and femoral cannulation were performed with previous TEE confirmation of the true aortic lumen; cardiopulmonary bypass was initiated. Under DHCA (20°C), the native heart was excised, and ascending aorta plus proximal arch were replaced with a 28-mm Dacron graft distal to the innominate artery. Neuroprotection included lidocaine, propofol, methylprednisolone, and phenytoin. No NIRS drop >16% occurred. CPB lasted 315 min, DHCA 22min, donor heart ischemic time 192min; two CPB weaning attempts were performed but persistent RV dysfunction was documented requiring central VA-ECMO, discontinued on day 2 after recovery. A cardiac tamponade on day 8 was drained surgically (Image 6). Immunosuppression began day 5. The patient was discharged with preserved graft function (LVEF 68%).

Discussion

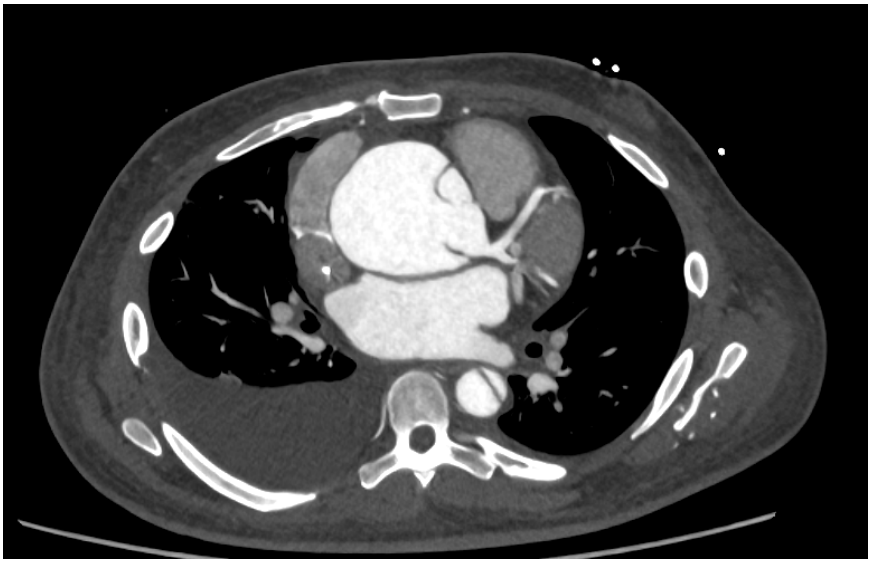
Combined heart transplantation and Stanford Type A dissection repair, though individually high-risk, can be successfully performed in one session with meticulous coordination. Key factors include donor timing, shock management, and ischemia minimization. Composite graft use enabled simultaneous correction

without valve replacement. This approach may benefit select patients with end-stage heart failure and complex aortic pathology, though further evidence is needed to optimize outcomes. The main anesthetic challenge was maintaining physiological stability during transplantation and aortic repair under deep hypothermia, while addressing risks of prolonged ischemia, reperfusion syndrome, and a stunned donor heart.

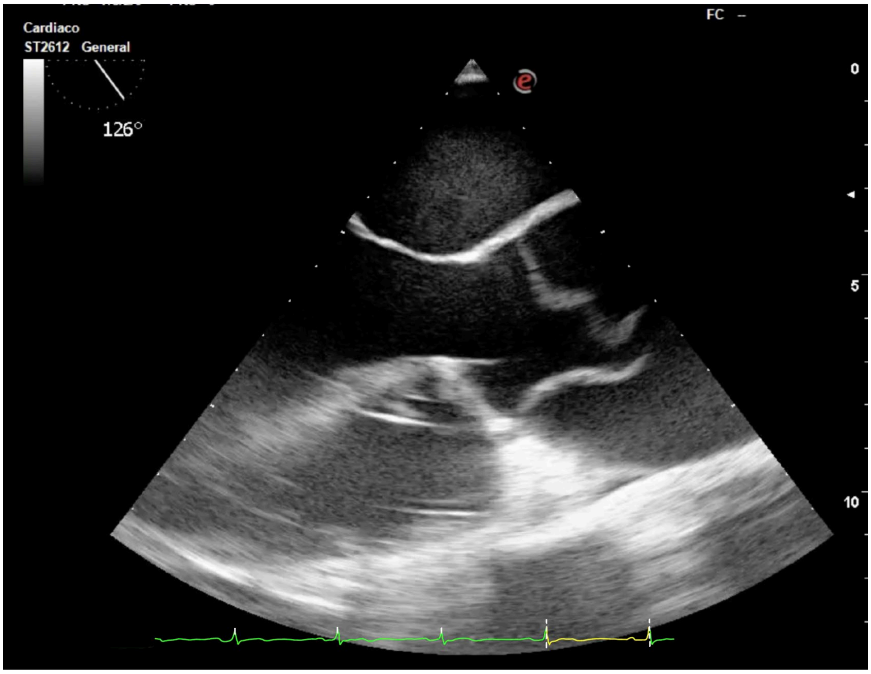
Images



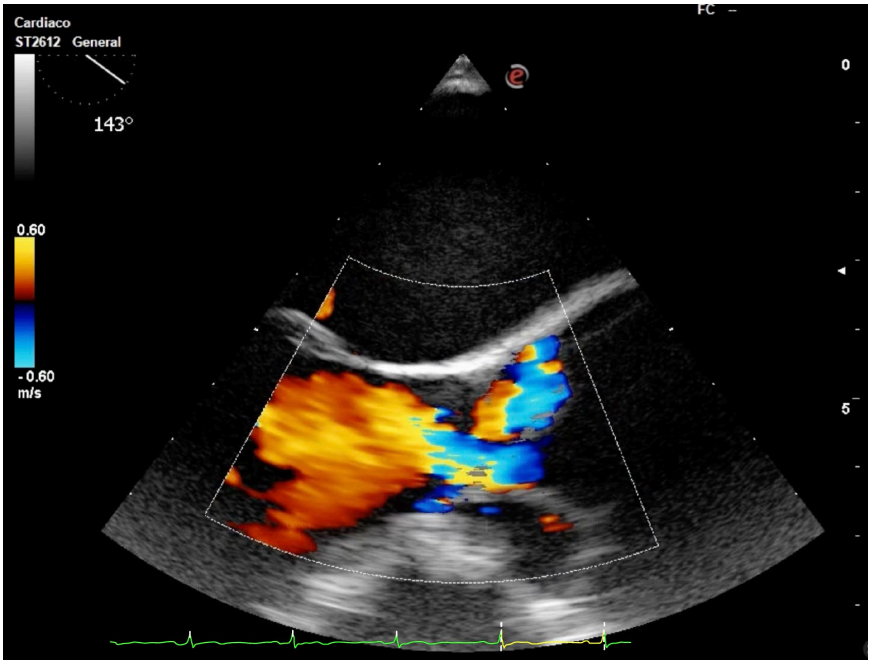
CTA 3D Reconstruction. Stanford Type A aortic dissection is observed, extending from the aortic root, involving the carotid arteries down to the right femoral artery.



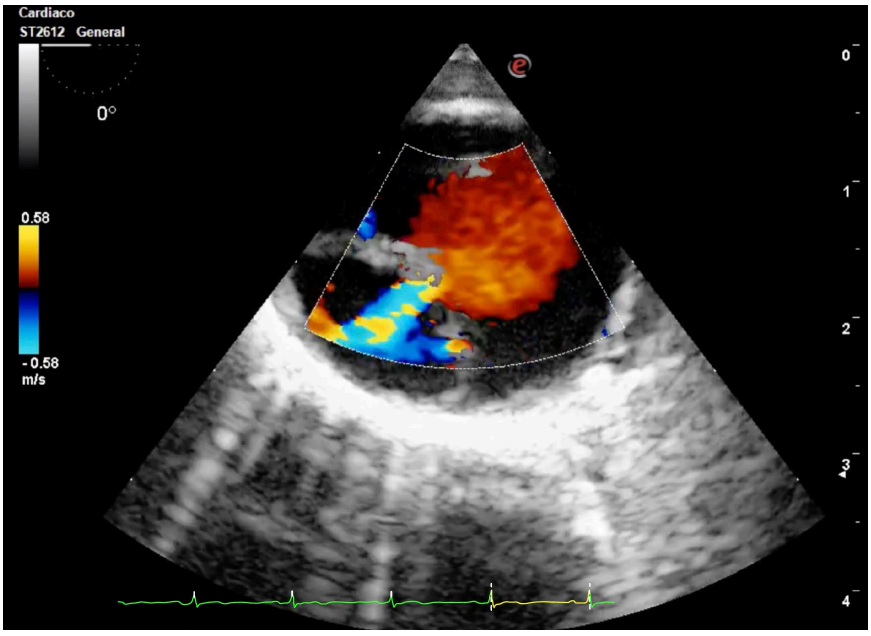
CTA. Axial contrast-enhanced CT image showing Stanford Type A aortic dissection involving the ascending aorta and extending to the left coronary sinus. The true and false lumens are visible, confirming proximal extension toward the coronary ostium.



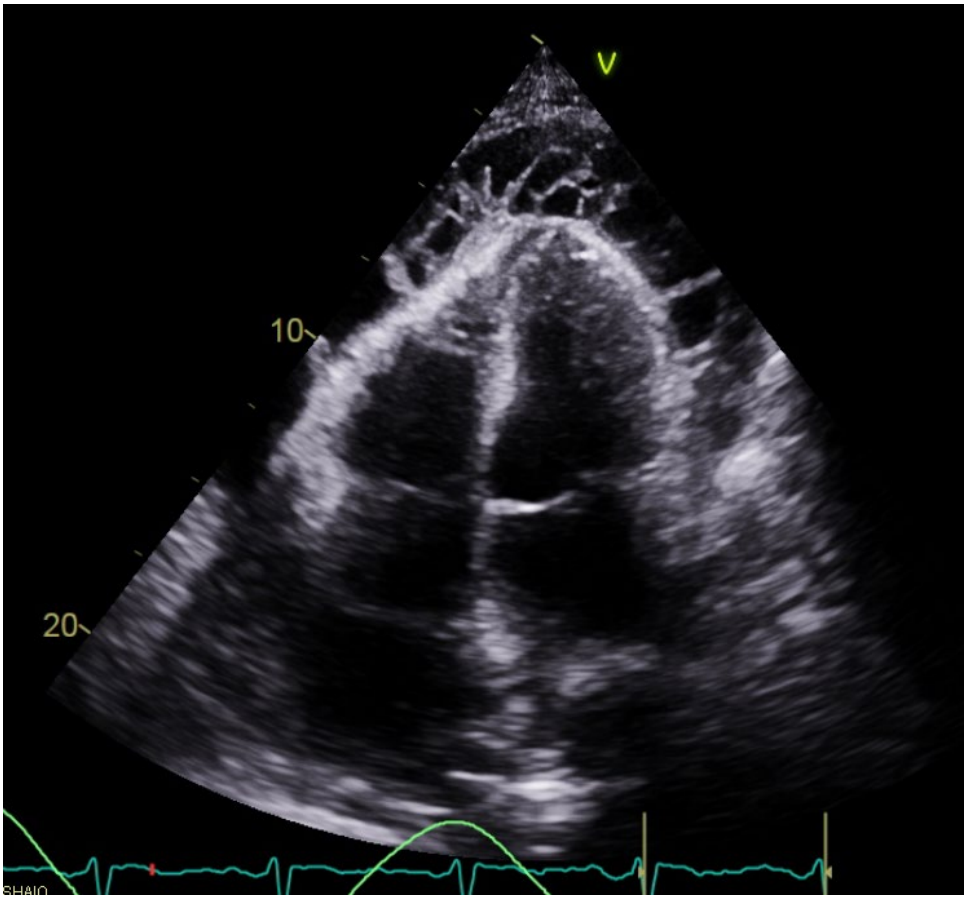
Transesophageal echocardiography (mid-esophageal long-axis view) showing an intimal flap consistent with Stanford Type A aortic dissection, originating near the left coronary cusp and extending into the ascending aorta.



Transesophageal echocardiography (mid-esophageal long-axis view) with color Doppler demonstrating severe aortic regurgitation associated with Stanford Type A aortic dissection. Turbulent flow is observed across the aortic valve into the left ventricle.



Transesophageal echocardiography (mid-esophageal descending aorta short-axis view) demonstrating a re-entry tear of the Stanford Type A aortic dissection within the descending thoracic aorta. Color Doppler highlights flow communication between true and false lumens.



Transthoracic echocardiography (apical four-chamber view) showing a large pericardial effusion causing cardiac tamponade, observed on postoperative day 8.

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#FR 2 – Kiosk 16 – Poster 5

An Unusual Presentation of Aortic Dissection Causing Pulmonary Artery Obstruction: A Case Report

Presenting Author: Scott Zheng, MD, Resident Physician, Hospital of the University of Pennsylvania

Introduction

Aortic dissection is an uncommon, but life-threatening condition, described as a tearing of the intimal layer of the aorta, resulting in blood flowing between the intima and media, which can result in inadequate blood flow to vital organs. There are multiple classifications of the distinct types of aortic dissection, one of which being the Stanford classification. Under the Stanford classification, a Type A dissection includes all dissections involving any part of the ascending aorta and a Type B dissection involves only the descending aorta. We present a case of aortic dissection with a contained rupture leading to compression and complete occlusion of the Right PA and subtotal occlusion of the left PA visualized on CT and TEE imaging.

Case Presentation

An 84-year-old female with past medical history of hypertension, asthma presented to the emergency department with several days of chest pain and shortness of breath. Her O2 saturation was 85% on room air, with a pre-op PaO2 of 52. TTE was suggestive of aortic dissection which was further characterized on CTA as a contained rupture with severe narrowing of left PA and complete occlusion of proximal right PA due to mass effect from a hematoma. The patient was induced with a narcotic heavy induction and via RSI leading to intermittent hemodynamic instability resolving with high-dose pressor boluses and infusions. Notably her O2 saturation remained 100% throughout. Care was taken to ensure adequate oxygenation and ventilation of the patient throughout induction. Intra-op transesophageal echocardiography prior to intervention revealed a type A dissection with origin at the non-coronary sinus, with complete occlusion of the right pulmonary artery. The left main pulmonary artery was over 50% occluded with turbulent flow. RV function was mildly to moderately decreased and the RV was dilated. The patient underwent a type A dissection repair with a bioroot ascending/hemiarch replacement, as well as a pulmonary arterioplasty. Following bypass, TEE demonstrated reconstitution of flow in the Right and Left PA's. The post bypass ABG demonstrated a PaO2 of 365 mm Hg. The patient was extubated on post-op day 1, vasopressor support weaned off by post-op day 5, and she was discharged from the hospital about two weeks after surgery.

Discussion

Aortic dissections have the ability to present in a variety of ways. Often, dissections lead to end-organ hypoperfusion due to blood flowing into the false lumen. This case was unusual in that the aortic dissection involving the aortic root created a contained rupture, which led to PA occlusion, causing pulmonary blood flow to be compromised. There have been various case reports of similar pathophysiology described; however, in many of these case reports, the patient expired. This patient presented similarly to how a patient with acute myocardial ischemia and pulmonary embolism would. Ultimately, CTA and TEE imaging led to definitive emergent surgical management, which ensured patient survival despite the high-risk nature of this

patient’s condition.

Images



Figure 1. CTA image of aortic dissection with hematoma mass effect causing compression of the right pulmonary artery

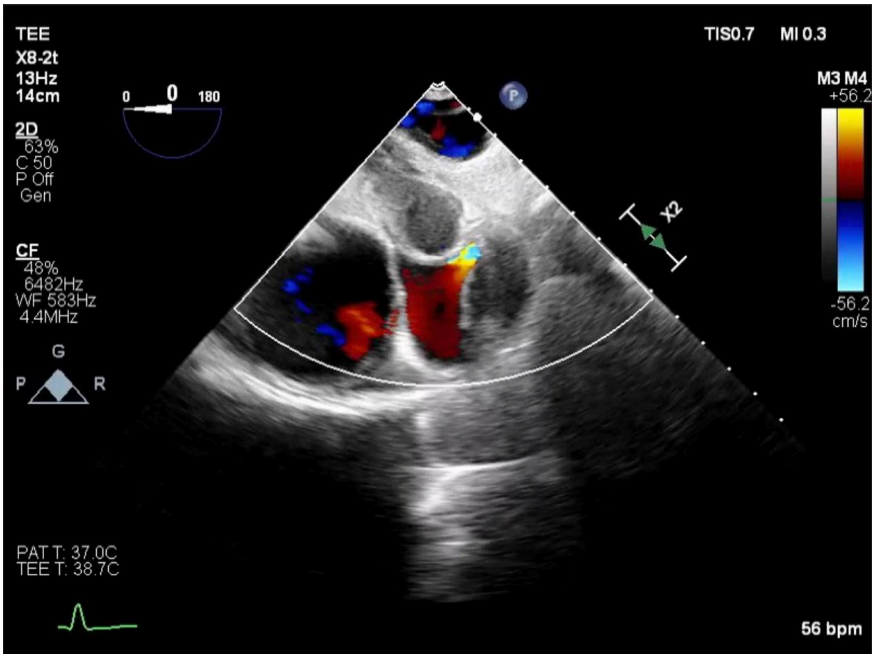


Figure 2. Midesophageal ascending aorta short axis view with color flow Doppler demonstrating absence of flow in the right pulmonary artery and flow acceleration in the main pulmonary artery

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#FR 2 – Kiosk 16 – Poster 6

Evaluation and Treatment of Left Circumflex Artery Injury Following Mitral Valve Repair

Presenting Author: Benjamin A. Jonik, MD, MPH, Resident Physician, University of Pennsylvania Hospital System

Introduction

Mitral valve disease is the most frequent valvular heart disorder, which ultimately may require either surgical valve replacement (MVR) or repair (MVr). Left circumflex (LCx) artery injury following mitral valve repair or replacement is a rare complication that has variable clinical presentations, ranging from clinical silence to overt cardiogenic shock.[1] This case report discusses a patient who underwent minimally invasive MVr who developed signs of regional wall motion abnormalities, and cardiogenic shock in the immediate post bypass period. Herein, we will explore the evaluation and treatment of myocardial ischemia following mitral valve intervention, and the patient and procedural risk factors which may increase the risk of LCx injury in this context.

Case Presentation

A 65-year-old female with a history of severe mitral regurgitation secondary to P2 prolapse with severe posterior MAC, and a right dominant system underwent a minimally invasive mitral valve repair via a right lateral thoracotomy approach (Figure 1). For repair, the patient received a P2 triangular resection with banding, and debridement of severe posterior mitral annular calcification. Following two aortic cross clamps for adequacy of repair, the patient in the immediate post-bypass period began to exhibit signs of overt cardiogenic shock with transesophageal echocardiographic (TEE) evidence of regional wall motion abnormalities in the left circumflex artery perfusion territory. There was high suspicion of LCx artery damage considering the difficulty of the surgical repair as a result of the patient's extensive MAC burden, and the clinical and echocardiographic findings following bypass. The decision was made to place an intra-aortic balloon pump prior to leaving the operating room, and to perform coronary angiography to elucidate the coronary culprit and identify any intervenable lesions. Angiography was notable for severe stenosis at the ostium of OM1, likely constrained by the annuloplasty ring or suture. Percutaneous intervention of the occluded LCx was complicated by a spiral dissection of the artery and was ultimately unsuccessful in restoring significant coronary flow. There were no appropriate targets for coronary artery bypass grafting (CABG) of the occluded LCx, and the decision was made to place the patient on VA ECMO and medically optimize them.

Discussion

Left circumflex artery injury is a reported complication of MVr or MVR. The risk of this injury is increased by patient risk factors which include extensive MAC and changing of the mitral annular architecture with annuloplasty. Despite having a right dominant circulation, the patient was clearly exhibiting signs of LCx artery injury following mitral valve repair. An algorithmic approach to postoperative myocardial ischemia includes: placement of mechanical circulatory support when hemodynamically unstable, followed by

coronary angiography and PCI vs. CABG intervention when feasible (Figure 2).2 In this case, decision making was complicated by the inadequacy of CABG target, even prior to LCx dissection following PCI, as well as the surgical approach being via right lateral thoracotomy incision. In this case, we demonstrated prompt evaluation and treatment of LCx injury in the perioperative period utilizing an algorithmic framework.

Images

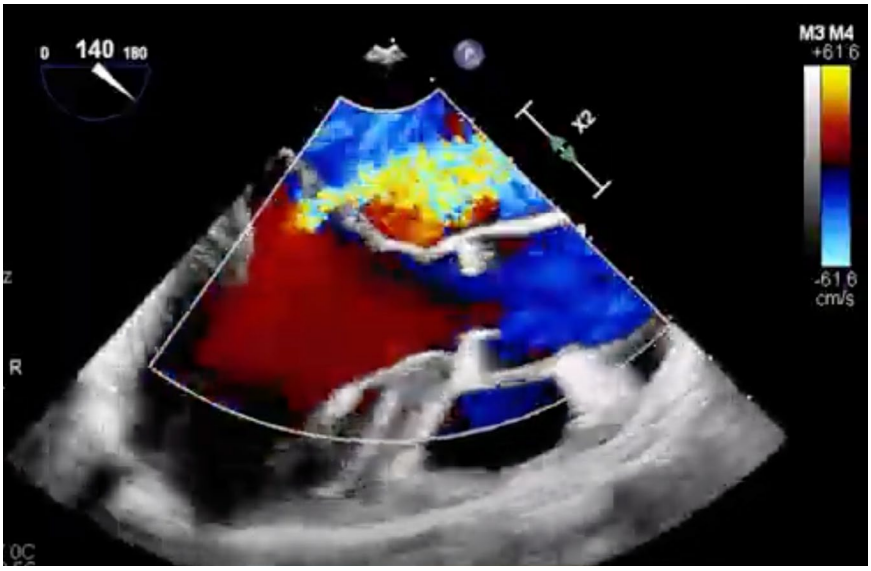


Figure 1. TEE in the mid-esophageal long axis view of severe mitral regurgitation secondary to P2 leaflet prolapse.

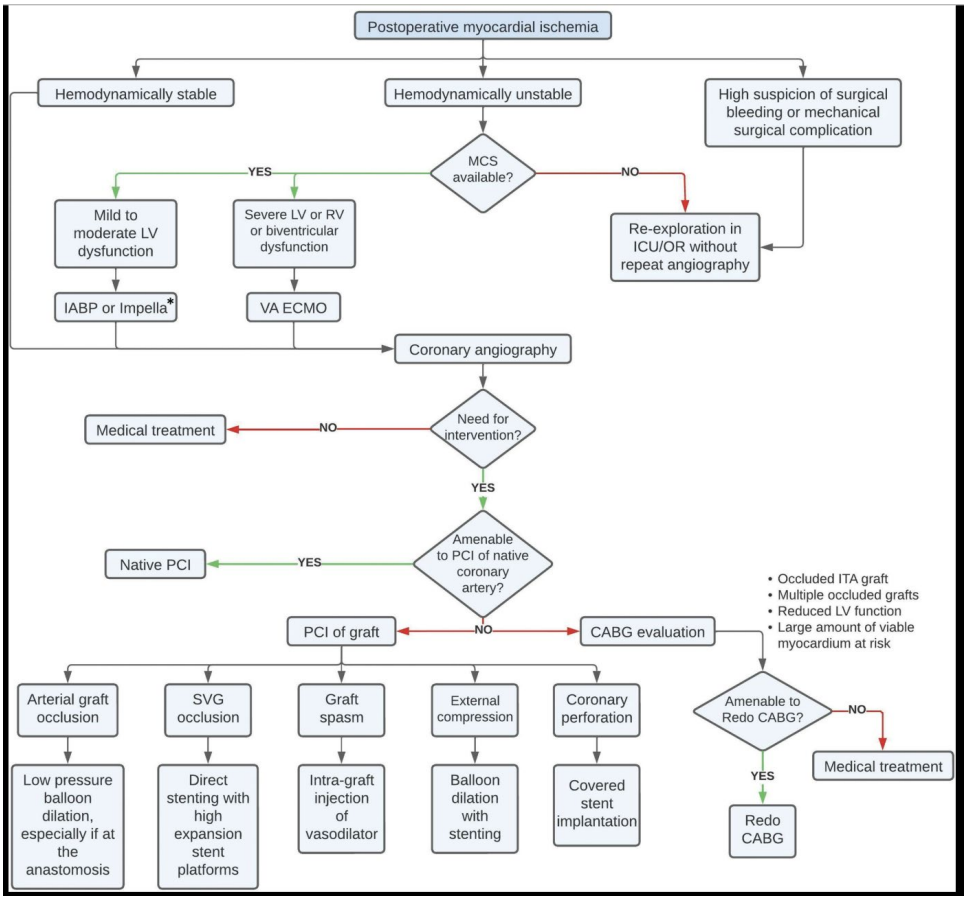


Figure 2. Algorithm for treatment and evaluation of postoperative myocardial ischemia from Guadino et al. 2023

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Left Circumflex Artery Injury After Mitral Valve Surgery: An Algorithm Management Proposal.Bargagna M, Trumello C, Sala A, et al.The Annals of Thoracic Surgery. 2021;111(3):899-904. doi:10.1016/j.athoracsur.2020.05.160.

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#FR 2 – Kiosk 17 – Poster 1

Anterolateral Papillary Muscle Rupture Following Acute Myocardial Infarction

Presenting Author: Sarah Fribance, MD, Anesthesiology Resident, University of Pittsburgh Medical Center

Introduction

Papillary muscle rupture (PMR) is a rare complication of acute myocardial infarction (AMI) taking place approximately 3-5 days after the ischemic event. Patients often present with acute heart failure since mitral valve (MV) function relies heavily on anterolateral and posteromedial papillary muscles. The posteromedial muscle is more likely to rupture due to its single blood supply by the posterior descending coronary artery. It is rare for anterolateral muscle rupture to occur due to its dual blood supply from the left anterior descending coronary artery and the left circumflex artery.

Case Presentation

A 61-year-old man presented with dyspnea and was found to be in acute hypoxemic respiratory failure. He was intubated and point-of-care TTE demonstrated decreased left ventricular ejection fraction (LVEF) and severe MV leaflet prolapse. Due to concern for PMR, he was transferred to a tertiary care center where an Impella 5.5 was placed. Intraoperative TEE demonstrated severe central mitral regurgitation (MR) with bileaflet flail, anterolateral PMR, and systolic reversal in pulmonary veins. The postoperative plan was to optimize the patient in the cardiac ICU prior to MV repair. Initially, temporary mechanical circulatory support (MCS) was able to decrease his vasopressor requirements. However, his ICU course was complicated by severe multiorgan failure, and he was transitioned to comfort care prior to return to OR.

Discussion

Anterolateral PMR frequently leads to severe MR and cardiogenic shock, as seen in this patient, for which temporary MCS is often used as a bridge to definitive surgical intervention. Observational data indicate that over 70% of patients with PMR require MCS prior to MV surgery. Intra-aortic balloon pump (IABP) is a guideline-recommended bridge to surgery due to its ability to reduce afterload and decrease regurgitant volume. Although the IABP-Shock II Trial demonstrated that IABP did not improve survival in patients with AMI complicated by cardiogenic shock, this study notably excluded patients with mechanical complications. The IABP has a favorable safety profile for this indication but limited randomized control trial (RCT) data on efficacy. Other percutaneous MCS devices, such as the Impella, can also be a useful bridge to surgery due to their ability to unload the LV and improve cardiac output. However, studies have shown increased bleeding and vascular complications when compared to IABP without a consistent mortality benefit. Their role in PMR specifically is not well defined, and evidence is limited to small studies and extrapolation from broader cardiogenic shock populations. VA-ECMO provides full cardiopulmonary support and can be used following PMR complicated by severe or refractory cardiogenic shock. One multicenter observational study demonstrated that VA-ECMO was used in 11% of PMR patients and ECMO-associated complications were present in over half. Case reports and observational studies suggest VA-ECMO can be lifesaving as a bridge to surgery, but robust RCT data is lacking. Overall, most evidence regarding

appropriate MCS as a bridge to cardiac surgery following PMR is observational or based on small case series. At this point, device selection should be individualized based on patient profile and institutional expertise.

Images

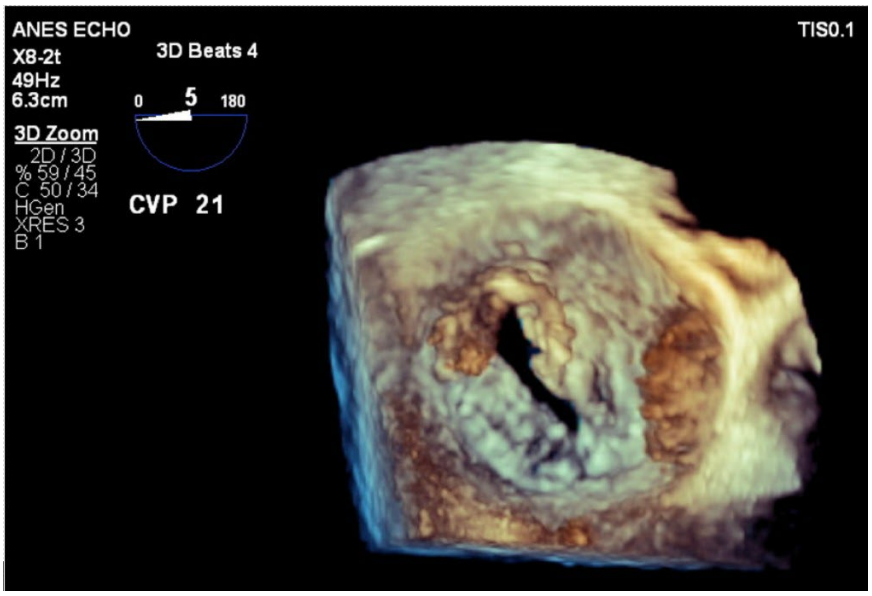


Figure 1. 3D reconstruction mitral valve showing A1, A2 flail from anterolateral papillary muscle rupture

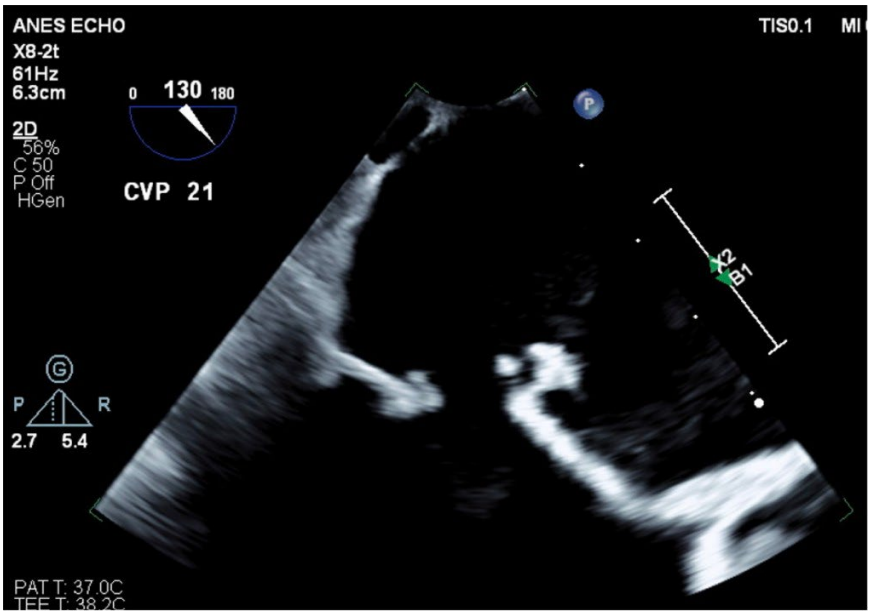


Figure 2. ME LAX view of mitral valve at A2, P2 showing A2 flail

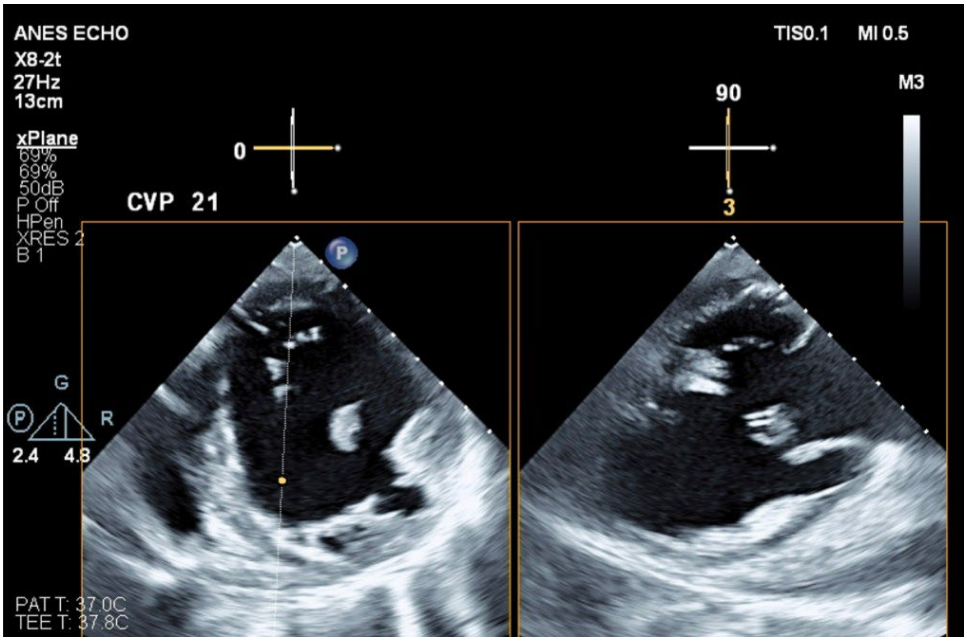


Figure 3. TG at midpapillary level with X-plane showing anterolateral papillary rupture

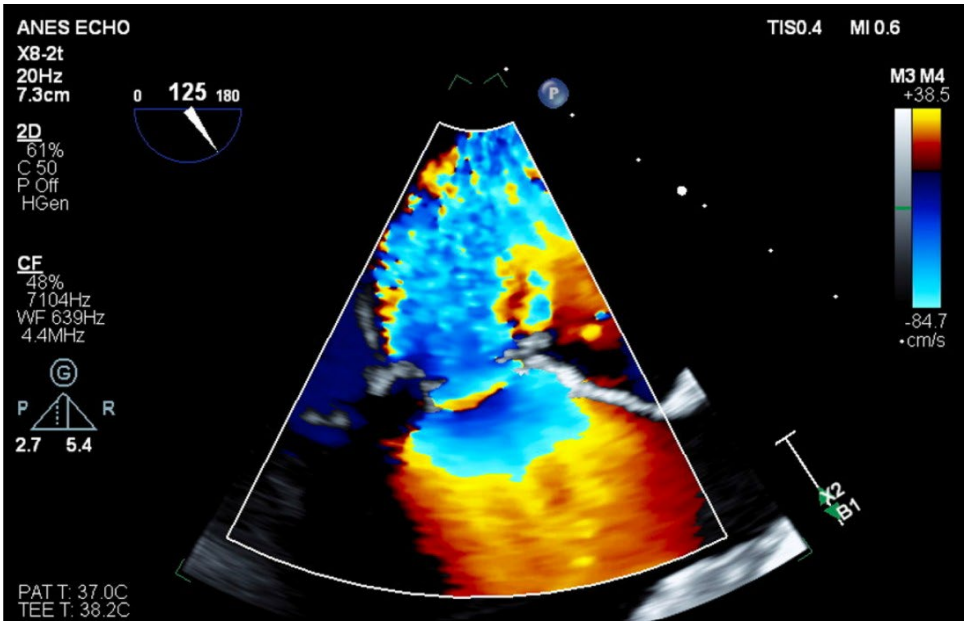


Figure 4. Color doppler showing severity of MR. Vmax of 6.2m/s. MR VTI of 1.09m. MR radius of 1.2cm. EOA by PISA is 0.56cm2. Regurgitant volume of 61mL.

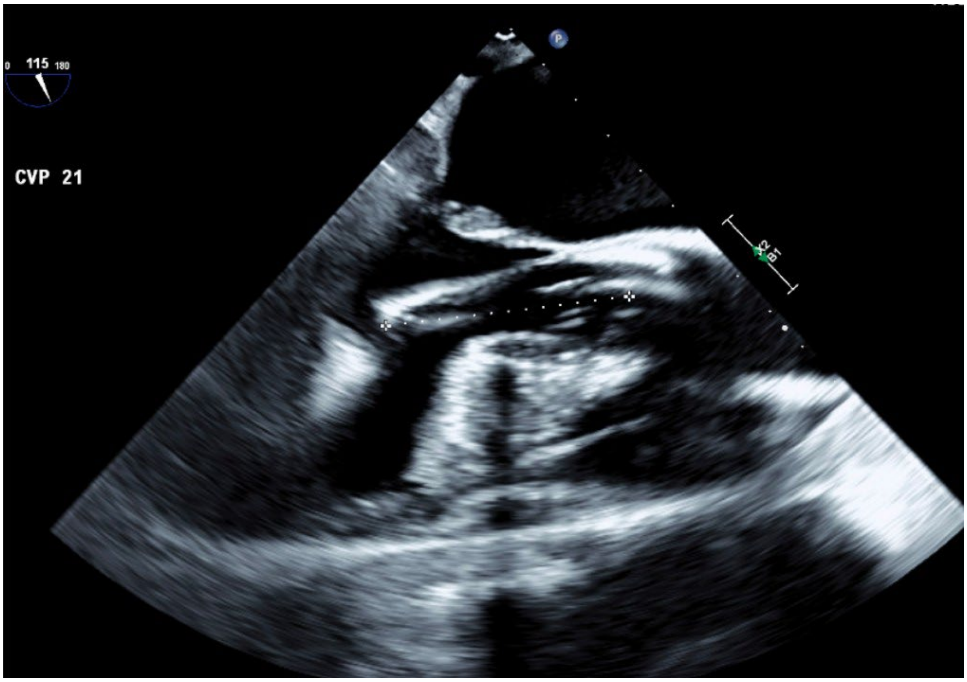


Figure 5. ME LAX with Impella 5.5, 5cm from aortic valve

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#FR 2 – Kiosk 17 – Poster 2

Management of Cardiac Surgery in a High-Risk Patient with Active Heparin-Induced Thrombocytopenia

Presenting Author: Joseph M. Joslin, MD, Resident Anesthesiologist, University of California, Irvine Medical Center

Introduction

Heparin-induced thrombocytopenia (HIT) is an immune disorder characterized by IgG antibodies that target the platelet factor 4–heparin complex, causing thrombocytopenia and creating a paradoxical prothrombotic state. Patients with HIT are a unique challenge to cardiac surgery as heparin is the preferred anticoagulant during cardiopulmonary bypass (CPB). This case demonstrates a patient with acute HIT who was transitioned from argatroban to a heparin-epoprostenol protocol during surgery, then back to argatroban post-operatively, an uncommon strategy in the setting of intra-aortic balloon pump support.

Case Presentation

A 72-year-old woman presented in cardiogenic shock secondary to an inferior myocardial infarction. An IABP was placed, and intravenous heparin was initiated. On hospital day 7, her platelet count fell precipitously, and she was transitioned to argatroban allowing for recovery of her platelet count. Platelet factor 4 antibodies were positive, and a serotonin release assay confirmed HIT. On hospital day 15, CPB was performed using a heparin–epoprostenol protocol. Epoprostenol infusion began pre-heparinization, titrated to 30 ng/kg/min, and tapered after protamine reversal. Intraoperatively, the patient developed hypotension during titration. Argatroban was restarted on postoperative day 1 as the IABP was weaned. Platelets decreased postoperatively but not to the same degree as before surgery.

Discussion

Management of HIT in cardiac surgery depends on the disease phase (acute, subacute A, or subacute B) and surgical urgency. When feasible, delaying surgery allows for heparin use after platelet recovery and a negative functional assay. In cases of active HIT, anticoagulation options include direct thrombin inhibitors, heparin with adjunctive antiplatelet therapy, or plasma exchange. Bivalirudin is currently recommended because of established efficacy and safety, but it lacks a reversal agent, can accumulate during hypothermia or renal dysfunction, and may promote circuit thrombosis in areas of blood stasis.3 Alternative strategies include combining heparin with platelet inhibitors such as epoprostenol or cangrelor. Epoprostenol inhibits platelet activation via cyclic adenosine monophosphate (cAMP)–mediated pathways,

while cangrelor blocks adenosine diphosphate (ADP)–induced platelet activation through P2Y12 receptor antagonism. However, in vitro data suggest cangrelor may be an unreliable inhibitor in the setting of acute HIT, requiring confirmatory platelet function testing before use.[1,3] Both agents have rapid onset and offset (< 6 minutes) and require continuous infusion, but the duration of post-infusion platelet inhibition is uncertain. Epoprostenol also causes potent vasodilation, leading to hypotension, as observed in this case.2 This case illustrates that for patients with active HIT requiring urgent cardiac surgery, a heparin–epoprostenol protocol can permit anticoagulation with rapid reversibility, though further data is needed to establish comparative safety and efficacy. It also underscores the importance of multidisciplinary planning when conventional heparinization is contraindicated.

Images

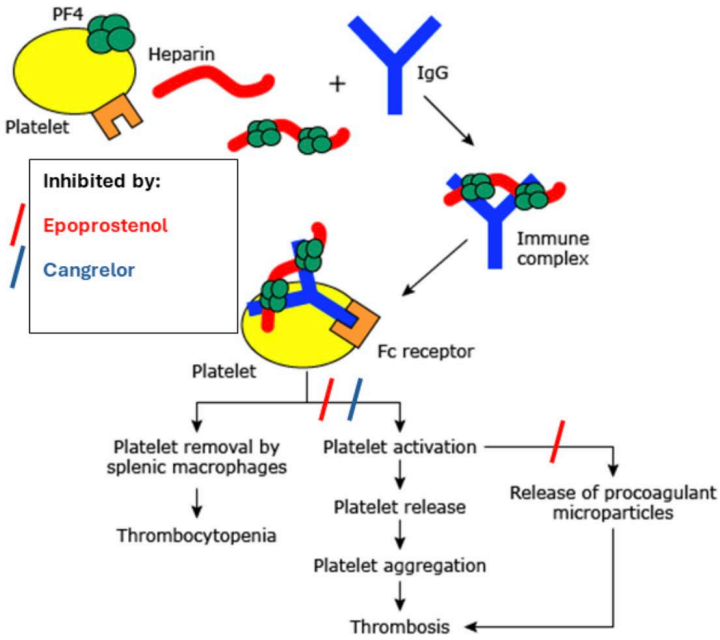


Figure 1. Mechanism of HIT platelet activation and inhibition. Adapted and modified from UpToDate, accessed October 2025.

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#FR 2 – Kiosk 17 – Poster 3

Case Report: Pulmonary Artery Catheter Placement in a Patient with Persistent Left SVC

Presenting Author: Riyadh Atrah, MD, Anesthesiology Resident, Sunrise Health GME

Introduction

Persistent left superior vena cava (PLSVC) syndrome is one of the most common congenital thoracic vascular anomalies with an incidence of about 0.3-0.5 in the general population. About 90% of patients with PLSVC have a left-sided SVC in addition to the normal right-sided vessel, whereas the remaining 10% have mainly PLSVC as the primary vessel draining the upper body and head. The pathophysiology of PLSVC is due to the failure of the left anterior and common cardinal veins to regress during vascular development. This condition is typically asymptomatic and is diagnosed incidentally during cardiac procedures.

Case Presentation

We present a case of PLSVC that was diagnosed intraoperatively in a 75-year-old male patient with past medical history of paroxysmal atrial fibrillation, hypertension, prior myocardial infarction, and mitral valve replacement (in 2018). The patient presented with intermittent fevers in association with subacute embolic and splenic infarcts. He was diagnosed with prosthetic mitral valve endocarditis requiring a redo-sternotomy with mitral valve repair. Intraoperatively, he was found to have mainly PLSVC without the typical right-sided SVC. A pulmonary artery (PA) catheter was inserted into the right internal jugular (IJ) vein and passed through the midline via the innominate vein into the PLSVC. It reached the right ventricle via the dilated coronary sinus and eventually placed into the main PA. During the cardiopulmonary stage, diastolic cardiac arrest was achieved via antegrade cardioplegia with success. Draining of the heart was done via a catheter at the aortic root.

Discussion

PLSVC poses various risks to patients undergoing cardiac surgery. These risks include coronary sinus (CS) injury, arrhythmias, embolic and/or thrombosis, and misleading hemodynamics parameters. Patients with PLSVC require mitigation of these risks. In our case, it was done by promptly removing the pulmonary artery catheter in the post-operative period. PLSVC also complicates the use of retrograde cardioplegia, which, in

our case, was avoided, and cardiac arrest was achieved mainly with antegrade flow of the cardioplegia solution. Finally, a coronary sinus catheter was placed to drain the heart and achieve a bloodless arrested heart for optimum operative conditions.

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#FR 2 – Kiosk 17 – Poster 4

Neonatal tricuspid valve thrombus causing cardiovascular collapse requiring extracorporeal membrane oxygenation and urgent surgical repair

Presenting Author: Stephen Aldous, MD, Anesthesiology Resident, Cedars-Sinai Medical Center

Introduction

Neonatal intracardiac thrombi are rare and carry high mortality due to small circulating volume, limited cardiopulmonary reserve, and complex anticoagulation management. We present the anesthetic management of a 13-day-old neonate with a tricuspid valve thrombus who developed sudden cardiovascular collapse requiring ECMO and urgent surgical repair.

Case Presentation

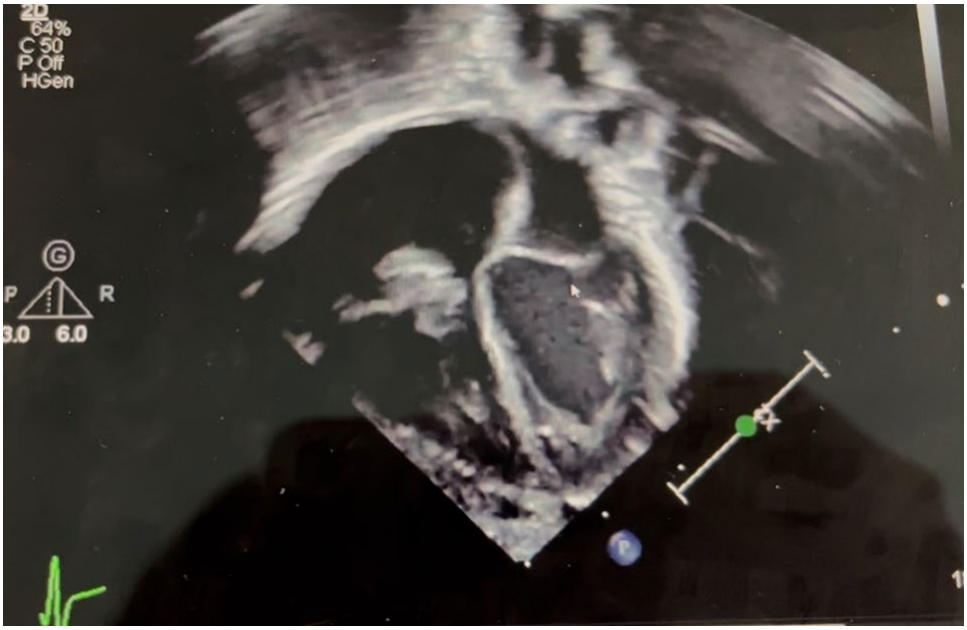
A previously healthy 13-day-old female presented with two tender, purple toes. She was hemodynamically stable but hypoxic. Laboratory evaluation revealed thrombocytopenia (10×10^9/L), and TTE identified a 5 × 9 mm mass on the septal leaflet of the tricuspid valve causing mild obstruction (mean gradient 4 mmHg), mild tricuspid regurgitation, and additional small thrombi in the PDA, SVC-innominate junction, and left pulmonary artery. A small PFO was also present. She was admitted to the PICU, transfused to a platelet count > 50×10^9/L, and started on heparin. CT angiography revealed extensive right-sided pulmonary emboli. She later developed new left ptosis, prompting MRI to rule out stroke. Under anesthesia in the MRI suite, she acutely desaturated and became hypotensive, resulting in an aborted study and emergent intubation. Despite escalation of support—including inhaled nitric oxide and high-dose epinephrine—she continued to deteriorate. After multidisciplinary discussion, VA ECMO was initiated, and she was urgently transported to the OR. Anesthesia was maintained with fentanyl and midazolam. Intra-operative TEE demonstrated a large,

partially obstructing tricuspid mass. She underwent mass excision, tricuspid valve repair, pulmonary artery embolectomy, PFO closure, and PDA ligation. Postoperative TEE showed only mild–moderate tricuspid regurgitation, and completion angiography demonstrated partial resolution of the pulmonary emboli. She came off bypass and was weaned from ECMO in the OR and returned to the PICU on epinephrine and milrinone infusions. Pathology confirmed a calcified thrombus. She made a full recovery and is meeting age-appropriate milestones.

Discussion

Tricuspid valve thrombi in neonates are rare and predispose to sudden cardiovascular collapse due to dynamic inflow obstruction, impaired right ventricular output, and high embolic potential. A mobile mass on the septal leaflet increases the risk of intermittent, unpredictable obstruction, and rapid hemodynamic shifts.[1] Key anesthetic principles include maintaining stable preload, avoiding increases in pulmonary vascular resistance, and minimizing sympathetic surges that could alter mass position. High-opioid anesthesia helps prevent myocardial depression and abrupt hemodynamic changes during induction and airway manipulation. Anesthetic agents must be titrated cautiously to minimize depressing cardiac function, increasing systemic vasodilation, and worsening right-to-left shunting through a PFO.[2] Given the potential for abrupt deterioration, continuous communication among anesthesia, critical care, cardiology, and cardiothoracic surgery teams is essential. Early planning for rapid initiation of mechanical circulatory support—including ECMO—is a critical component of safe anesthetic and peri-operative management in high-risk neonatal cardiac thrombi.

Images



Intra-operative TEE of the Tricuspid Valve Thrombus

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#FR 2 – Kiosk 17 – Poster 5

Double Trouble: Double Barrel Impella Exchange for Mechanical Circulatory Support in Cardiogenic Shock

Presenting Author: Richard Beckett-Ansa, Anesthesiology Resident, University of Virginia

Introduction

Impella devices are mechanical circulatory support systems placed across the aortic valve into the left ventricle, functioning as miniaturized ventricular assist devices. Based on the Archimedes' screw principle, they are inserted via an arterial approach and can augment cardiac output up to 5.0 L/min while reducing LV workload, myocardial oxygen demand, and facilitating other hemodynamic parameters. During standard Impella exchange, the original device is withdrawn into the thoracic aorta before the new one is advanced, creating brief circulatory stasis. In patients fully dependent on mechanical support, even short interruptions can trigger hemodynamic collapse. The uninterrupted "double barrel" exchange avoids this gap but introduces risks such as transient aortic insufficiency, which can be rapidly identified with TEE. This case highlights the physiologic implications of double barrel Impella support and the utility of TEE in guiding placement and detecting subtle real time changes.

Case Presentation

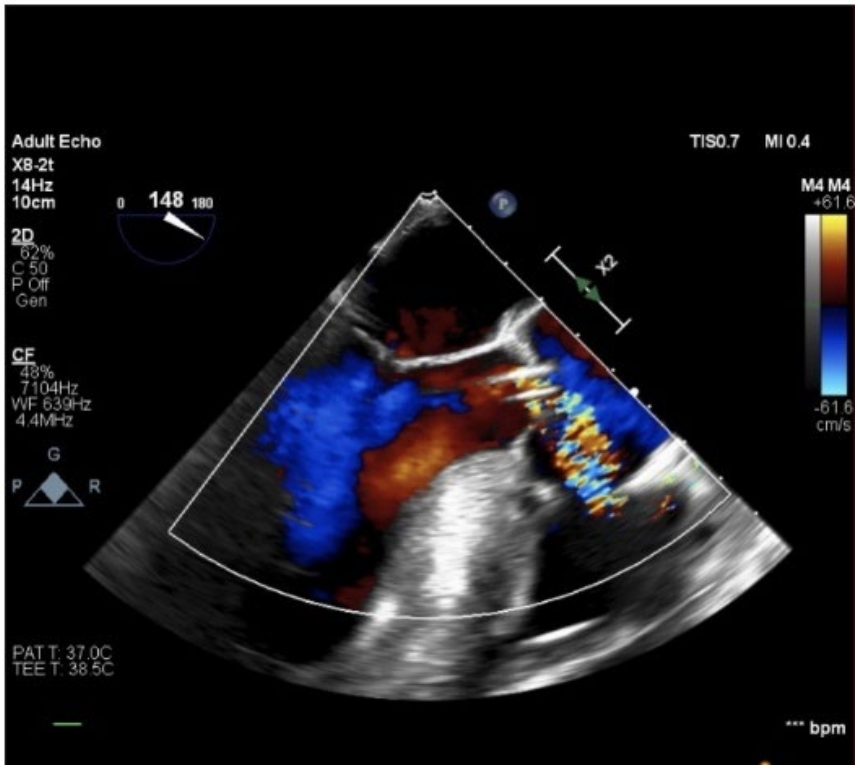
A 73-year-old male presented with a STEMI and required Impella CP support during initial catheterization. He was admitted to the CCU in cardiogenic shock, and his condition declined as the device produced only 2.7 L/min of flow. Given inadequate support, he was taken to the OR for exchange to an Impella 5.5. During induction with intravenous lidocaine and fentanyl, he developed profound hypotension and a brief PEA arrest, which was resolved with inotropic support before general anesthesia was established. After a coronary angiography confirmed stent patency, the Impella CP was exchanged for an Impella 5.5 under continuous TEE guidance. Even with the temporary presence of two devices across the aortic valve, the patient maintained stable cardiac output without arrhythmias or hemodynamic instability. The Impella CP was removed without complication, hemostasis was achieved, and he was transported back to the CCU stable.

Discussion

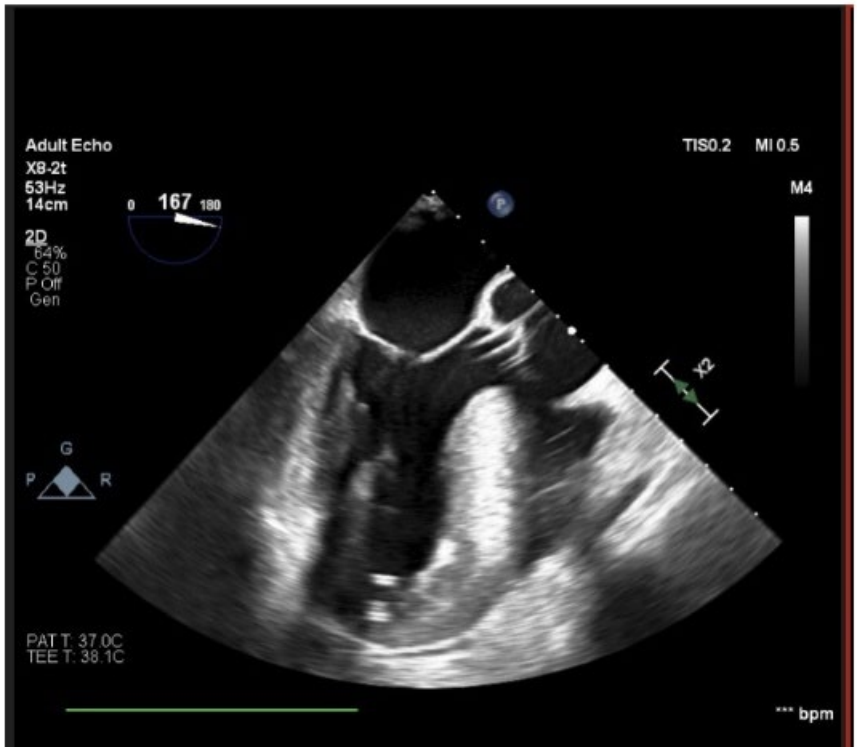
This case demonstrates the complexities of caring for patients fully dependent on mechanical circulatory support, where brief pauses in blood flow can lead to rapid hemodynamic collapse. As MCS technology evolves, innovative approaches to device management have become essential. The double barrel Impella exchange technique illuminates this adaptability: instead of withdrawing the original device and accepting a period without support, a second Impella is advanced alongside the first to maintain continuous unloading and perfusion of the LV during the exchange. Emerging evidence supports this strategy. Clothier et al. reported its safety in 31 consecutive exchanges, noting no intraoperative cardiac arrests and no new aortic insufficiency in 96.8% of patients despite both pumps temporarily traversing the aortic valve. Their subgroup analysis also showed that patients supported with Impella alone achieved an 83.3% survival rate,

significantly better than those requiring combined ECMO and Impella support. Our experience bolsters these findings and underscores the value of creative problem solving in complex mechanical support cases. Techniques such as the double barrel exchange under TEE guidance expand the clinical toolkit for managing severe hemodynamic instability and provide safer pathways to bridge patients toward recovery or definitive therapies.

Images



Mid Esophageal Long Axis View w/ Doppler Flow



Mid Esophageal Long Axis View w/ Impella CP and 5.5 inside of the Aortic Valve

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#FR 2 – Kiosk 17 – Poster 6

Use of Transesophageal Echocardiography to Diagnose Acute Aortic Dissection in a Patient with Negative Preoperative Imaging

Presenting Author: Riley Hogan, MS4, Medical Student, Indiana University School of Medicine

Introduction

Accurate and timely imaging is crucial in patients with acute aortic syndrome both to make the diagnosis and to guide management. Contrast computed tomography (CT) has become the preferred modality in the emergency department and for hospitalized patients. It is non-invasive, allows for quick scanning and interpretation, is less operator-dependent than other techniques, and is widely available. CT is also highly accurate, with reported sensitivity and specificity of >95% for acute aortic dissection; and it is helpful to the surgeon and anesthesiologist for invasive line placement, induction and maintenance of general anesthesia, cannulation, and the planned surgical repair. Here, we describe a case of CT-negative acute type A aortic dissection that was diagnosed intraoperatively with transesophageal echocardiography (TEE).

Case Presentation

A 53-year-old male with a history of hypertension presented with chest pain radiating to his left arm. His electrocardiogram showed ST changes concerning for acute coronary syndrome. He underwent an emergent coronary angiogram which was negative for coronary pathology, but the proceduralist noted severe aortic insufficiency (AI) and a possible ascending aortic dissection. The patient then had a CT, which demonstrated aortic dilatation but was negative for acute aortic syndrome. The decision was made to medically manage the patient's AI, but his condition quickly deteriorated, requiring high-dose vasopressors and inotropes and intubation for worsening pulmonary edema. The patient was taken to the operating room emergently with plans to replace the aortic valve. Induction of general anesthesia was uneventful, but TEE showed a localized dissection flap in the aortic root close to the left coronary ostium. There was no pericardial effusion or wall motion abnormality. The surgical plan was then altered to include an additional aortic root and proximal ascending aorta replacement. The case proceeded uneventfully, and the patient was discharged three weeks later.

Discussion

This case demonstrates that even when we are presented with normally reliable negative imaging, maintaining a healthy clinical suspicion of potentially lethal pathology is essential to taking the best care of our patients. CT has been shown to have a sensitivity as high as 100% and specificity of 98% for thoracic aortic dissection. In this case, the dissection flap was not seen, possibly because it was too small and localized to be detected by the scan, or because it was not present at the time the CT was done. The former seems more likely given that the dissection was first suggested during the patient's coronary angiogram. This case also highlights the utility of TEE in acute aortic syndrome. Although it was not utilized until the operating room, TEE may have been useful in the intensive care unit while the patient was decompensating. TEE has been shown to have sensitivity as high as 99% and specificity of 89%. Additionally, TEE is a dynamic monitor

that aids in hemodynamic management and surgical planning of aortic valve intervention and cannulation. Certainly, knowing preoperatively that this patient had severe AI secondary to an aortic dissection would have influenced the anesthetic plan.

Images

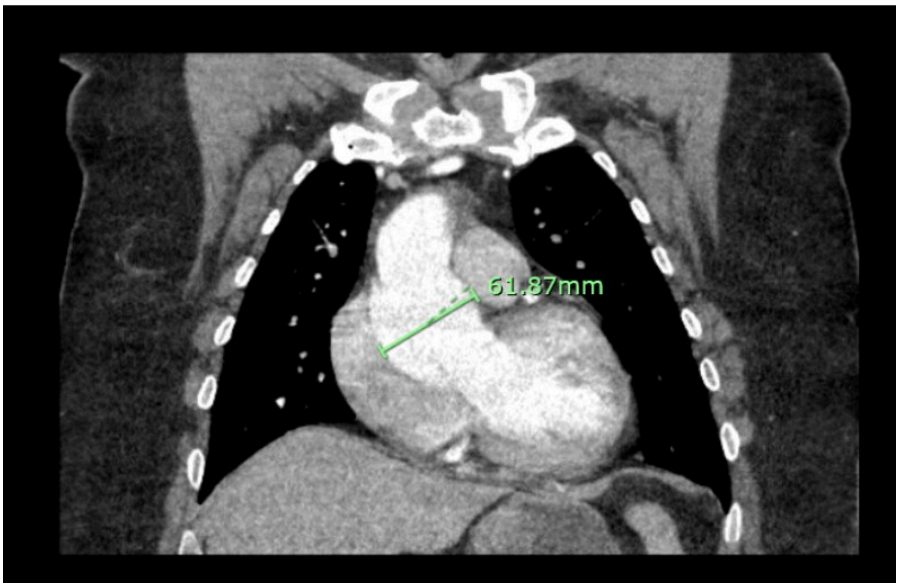


Figure 1: Contrast CT chest showing ascending aorta dilatation but no dissection.

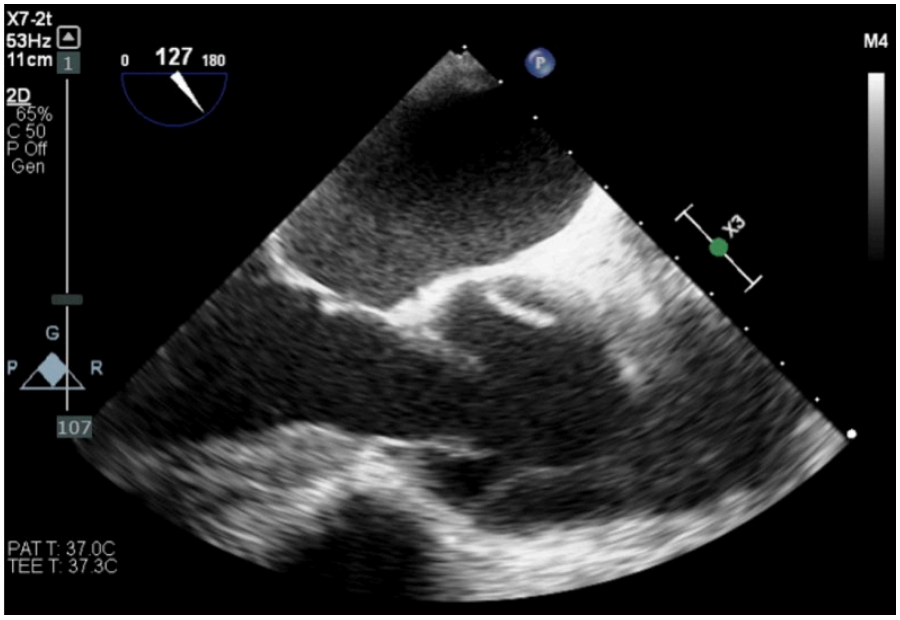


Figure 2: Mid-esophageal long-axis showing aortic dissection at the aortic root.

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#FR 2 – Kiosk 17 – Poster 7

Breaking the Biofilm: LVAD Explantation for Candida parapsilosis in a High-Risk Patient

Presenting Author: Harrison Burgess, DO, Physician, Maine Medical Center

Introduction

Infection of left ventricular assist devices (LVADs) with fungal organisms carries high morbidity and mortality, often necessitating device removal for source control. Candida parapsilosis, known for its affinity for prosthetic material and biofilm formation, presents a particular management challenge. We describe the anesthetic and perioperative considerations in a patient with C. parapsilosis fungemia undergoing LVAD explantation in the setting of heparin-induced thrombocytopenia (HIT).

Case Presentation

A 39-year-old man with nonischemic cardiomyopathy (LVEF 10–15%) status post HeartMate 3 LVAD implantation (May 2023), paroxysmal atrial fibrillation, and prior HIT presented with nausea, vomiting, and acute kidney injury (creatinine 7.5 mg/dL; baseline 1.7 mg/dL). Blood cultures revealed Candida parapsilosis. With evidence of recovered ventricular function (LVEF 50–55% at 5600 rpm), a multidisciplinary team recommended LVAD explantation for definitive infection management. The patient underwent explantation on August 18, 2025. Anticoagulation was achieved with bivalirudin given his HIT history. Intraoperative findings were consistent with device-associated infection. Postoperatively, he required vasopressor support with epinephrine, norepinephrine, vasopressin, and angiotensin II. Factor VII was administered for refractory bleeding. Serum lactate peaked at 11.6 mmol/L before clearing with hemodynamic optimization. On postoperative day 1 (POD 1), he returned to the OR for mediastinal washout and chest closure and was extubated that evening. His postoperative course was notable for atrial fibrillation with rapid ventricular response on POD 3, successfully cardioverted to sinus rhythm after amiodarone therapy. Antifungal therapy transitioned to oral fluconazole following micafungin completion, avoiding amphotericin-related nephrotoxicity. Transthoracic echocardiography on POD 10 demonstrated preserved LV function (EF 55%). He was gradually weaned from pressors, mobilized, and discharged on fluconazole, digoxin, and midodrine with a plan to resume guideline-directed medical therapy as renal function recovered.

Discussion

This case underscores the complexity of managing LVAD-associated fungal infection in a patient with HIT. Fungal colonization of prosthetic material often necessitates device explantation to achieve durable infection clearance. The combination of severe infection, prior HIT, and perioperative coagulopathy demanded close coordination among anesthesiology, surgery, and infectious disease teams. Non-heparin anticoagulation with bivalirudin allowed safe cardiopulmonary bypass, while aggressive antifungal therapy and early source control were pivotal for survival. Successful LVAD explantation with complete myocardial recovery remains rare, particularly in the context of systemic fungal infection. This case highlights the

importance of individualized anticoagulation strategies and multidisciplinary planning in optimizing outcomes for patients with complex mechanical circulatory support infections.



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#FR 2 – Kiosk 18 – Poster 1

Alpha-Gal Syndrome in a Patient Undergoing Mitral Valve Repair

Presenting Author: Evida Mars-Holt, DO, Fellow, Mount Sinai Hospital

Introduction

Alpha-Gal syndrome presents unique challenges in cardiac anesthesia as many standard medications and biologic materials used contain mammalian-derived products which can trigger severe allergic reaction. Careful preparation and multidisciplinary coordination are crucial to safely managing patients with alpha-gal syndrome undergoing cardiothoracic surgery.

Case Presentation

A 68-year-old gentleman with a history of alpha-gal allergy presented for elective mitral valve and tricuspid valve repair requiring cardiopulmonary bypass. Prior to his procedure, he was seen by the allergy department. An Alpha-gal IgE panel was drawn, and alpha-gal IgE was 31 kU/L which indicated as Class V, very high. The patient was then directed to have a heparin skin test and challenge which was negative/normal. A discussion with pharmacy of perioperative drugs was performed to identify medications which contained mammalian components. Vasopressin, milrinone, oral hydromorphone as well as certain manufacturers of propofol were indicated by pharmacy. Induction was achieved using midazolam, fentanyl, etomidate and rocuronium. Heparin was administered with premedication of Benadryl 50 mg, Hydrocortisone 100 mg and Famotidine 20 mg as recommended by the allergy department. The patient tolerated heparin without hypersensitivity, achieved therapeutic ACT, and underwent CPB uneventfully. No hypersensitivity reactions occurred. At case end, dexmedetomidine was used to provide sedation during transport to the cardiac surgery intensive care unit. The perioperative course concluded without any allergic manifestations.

Discussion

Alpha-gal syndrome poses unique anesthetic challenges largely because its triggers are not limited to food products but extend to numerous perioperative medications and surgical materials. Therefore, it is essential to perform a comprehensive review of all medications, equipment, and surgical adjuncts. The use of unfractionated heparin represents one of the most significant decision points as it is derived from porcine and/or bovine tissues. Anaphylaxis has been reported, however the risk is thought to correlate with alpha-gal IgE titers. Current literature suggests that patients with moderate-to-high IgE levels or prior reactions to biologics may be at increased risk. Strategies described in case reports include preoperative skin testing, graded challenge, premedication, or in extreme and emergent cases, the use of bivalirudin as an alternative anticoagulant. Other common medications used in cardiac anesthesia that are less commonly known to have mammalian components are vasopressin and milrinone. These medications should be avoided when possible or used with caution. This case demonstrates the need for advancement in management of alpha-gal syndrome. Many centers lack any formalized recommendations for managing alpha-gal syndrome,

specifically in cardiac surgery. Future research can help guide development of alpha-gal syndrome-specific perioperative guidelines which will improve safety and prevent unintended exposure.

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#FR 2 – Kiosk 18 – Poster 2

Left ventricular outflow tract to left atrium fistula following aortic valve replacement for infective endocarditis

Presenting Author: Stephen Soohey, MD, Resident, Lahey Hospital and Medical Center

Introduction

A left ventricular outflow tract (LVOT) to left atrium (LA) is a rare finding and can be caused by infective endocarditis (IE) or as a complication of aortic valve replacement (AVR) (1). In one study, aorto-cavitary fistulas were found in 1.8% of cases of native valve IE and 3.5% of cases of prosthetic valve IE (2). This same study found that heart failure developed in 62% of cases, with surgery performed for 66% of patients with a mortality of 41% (2). The decision to intervene mainly rests on whether the patient is symptomatic and can be surgical or percutaneous depending on operative risk.

Case Presentation

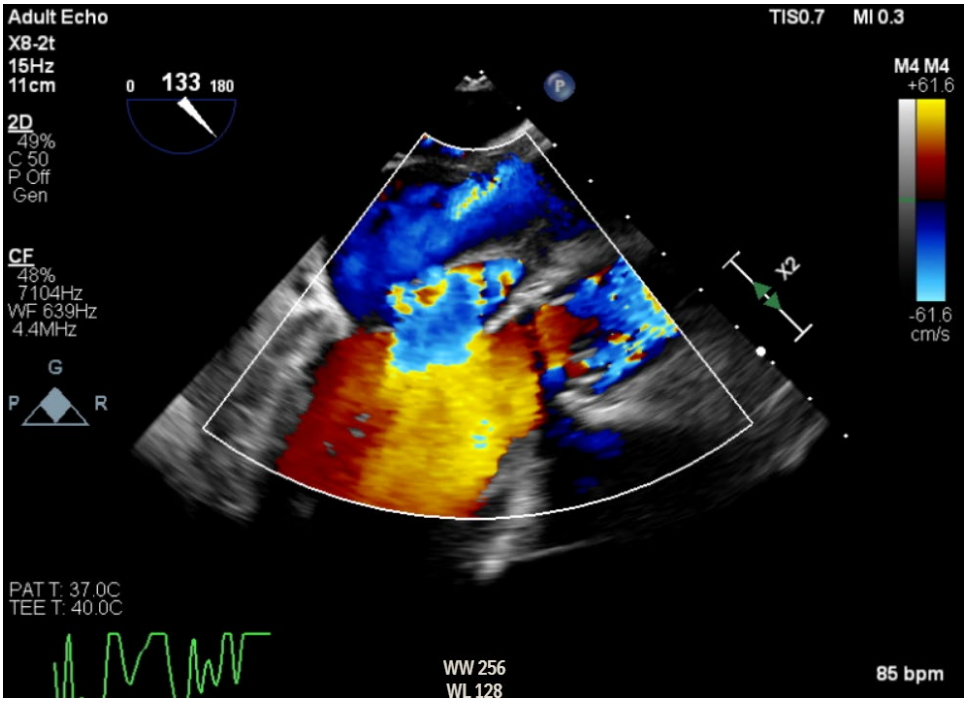
Our patient is a 49-year-old female with a history of morbid obesity, OSA, Group B Strep bacteremia, who originally presented with altered mental status. She subsequently became hypoxic and hypotensive, requiring intubation and transfer to the ICU. She was found to have Group B Strep IE with an aortic valve vegetation and aortic root abscess on transesophageal echocardiography (TEE) and underwent surgical AVR. Post-operative course complicated by renal failure, hemorrhagic shock, and pneumonia. One day developed severe pulmonary edema and acute hypoxic respiratory failure (AHRF) requiring intubation, with new torrential LVOT to LA fistula due to a ruptured pseudoaneurysm noted on echo. She was brought to the OR for patch closure of the fistula. The patient was given 2 units (U) of packed red blood cells (pRBCs) at the start of the case. Right axillary artery was cannulated with a SideArm graft for the arterial limb, and the right femoral vein was cannulated for the venous limb of the cardiopulmonary bypass (CPB) circuit. Pre-CPB TEE showed a grossly intact mitral valve (MV) with evidence of perforation of LVOT into LA causing torrential regurgitant flow. She was placed on CPB and given another 2U pRBCs. The LVOT to LA fistula was found to involve the annulus of the MV along the A1 and P1 segments. Debridement of the fistula destabilized these segments requiring MV reconstruction. Post-repair, the patient required Veletri and an epinephrine infusion once weaned from CPB. Post-CPB TEE showed severe mitral regurgitation (MR) originating below the patched area. CPB restarted for mitral valve replacement. A 27 mm MITRIS bovine pericardial valve was placed. The patient required ionotropic support to wean from CPB. TEE now showed a well-seated bioprosthetic MV and mildly dilated RV with reduced function. She was coagulopathic and given 2U platelets, 2U FFP and PCC. After achieving hemodynamic stability, she was decannulated, chest closed and transported to the ICU with minimal inotropic support.

Discussion

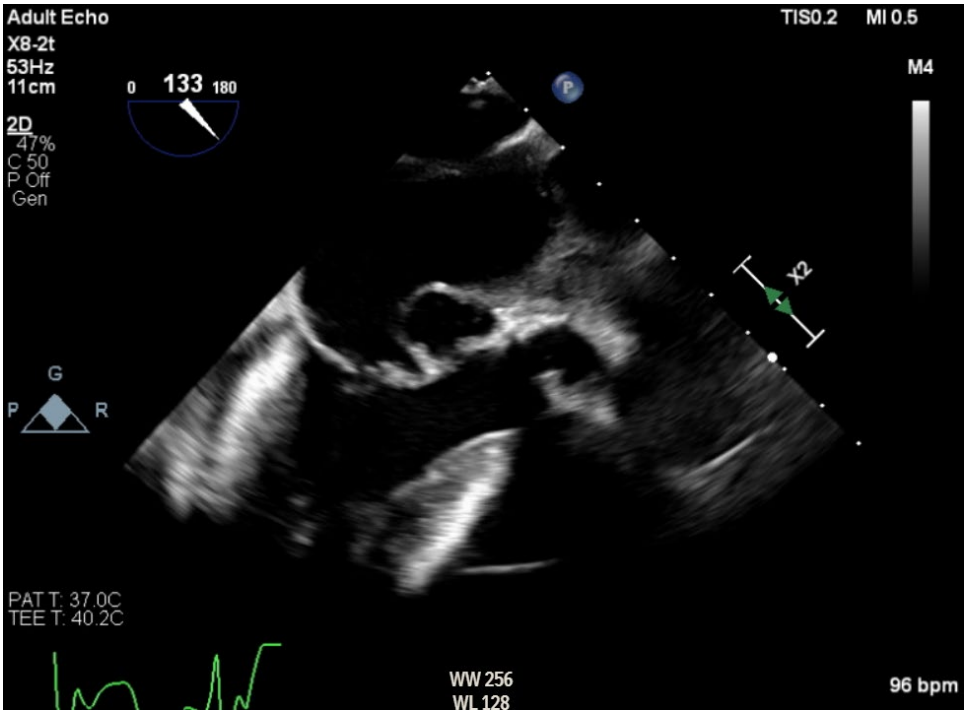
Our patient developed a LVOT to LA fistula following AVR for IE. No fistulus connection was noted during the original operation, and the patient was eventually stable for weeks following the initial operation. 4 weeks after, what appeared to be torrential MR was noted on echo but initially elected to manage conservatively

until she developed AHRF due to the fistula. As the patient was extremely symptomatic, intervention was required. This case was medically complex, requiring multiple CPB runs, and the patient was profoundly coagulopathic. TEE was imperative in deciding to return to CPB as there was MR present despite the patch closure of the fistula.

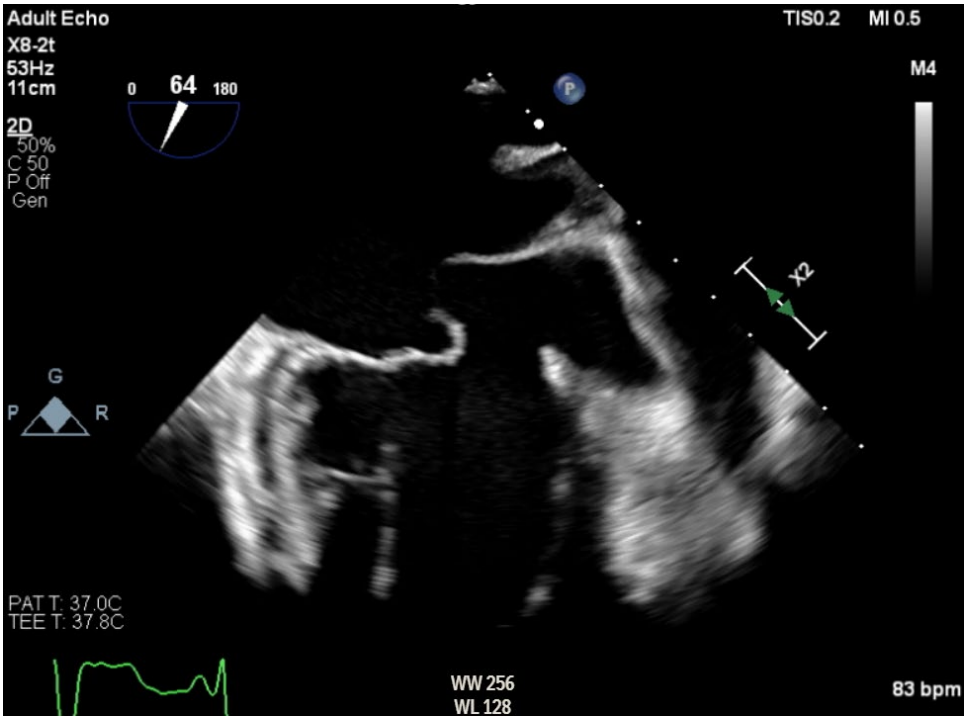
Images



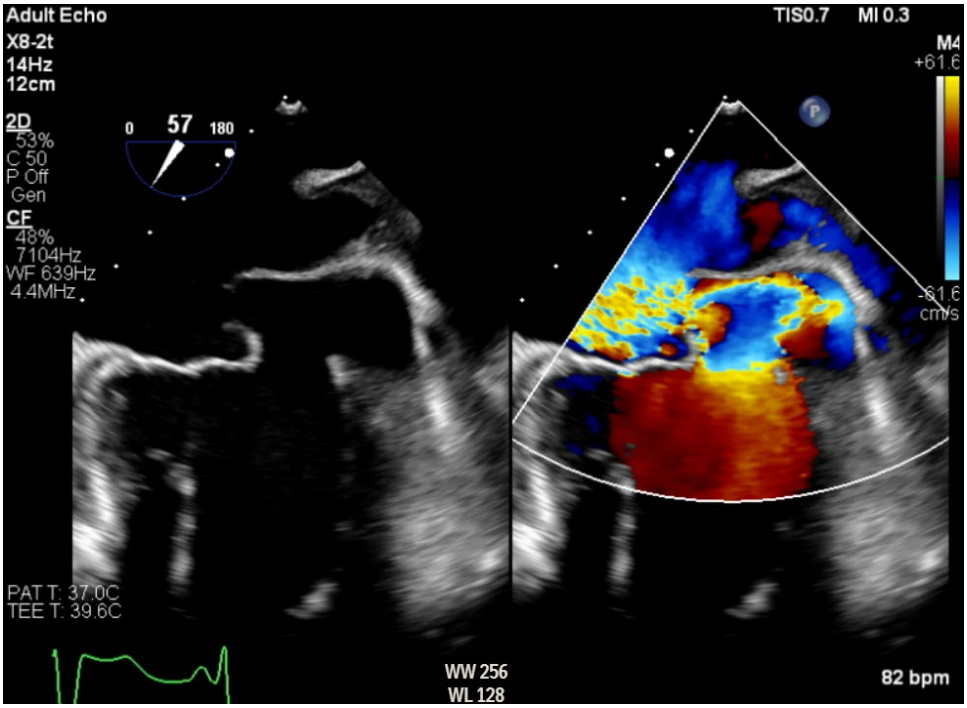
Perforation of LVOT into LA with color doppler



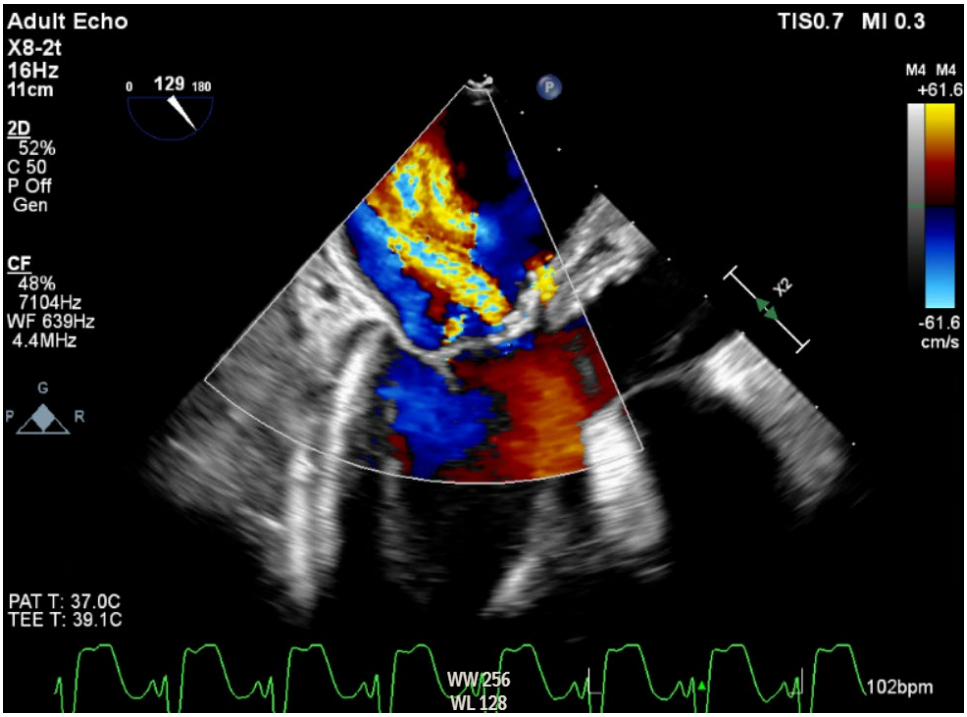
Perforation of LVOT into LA



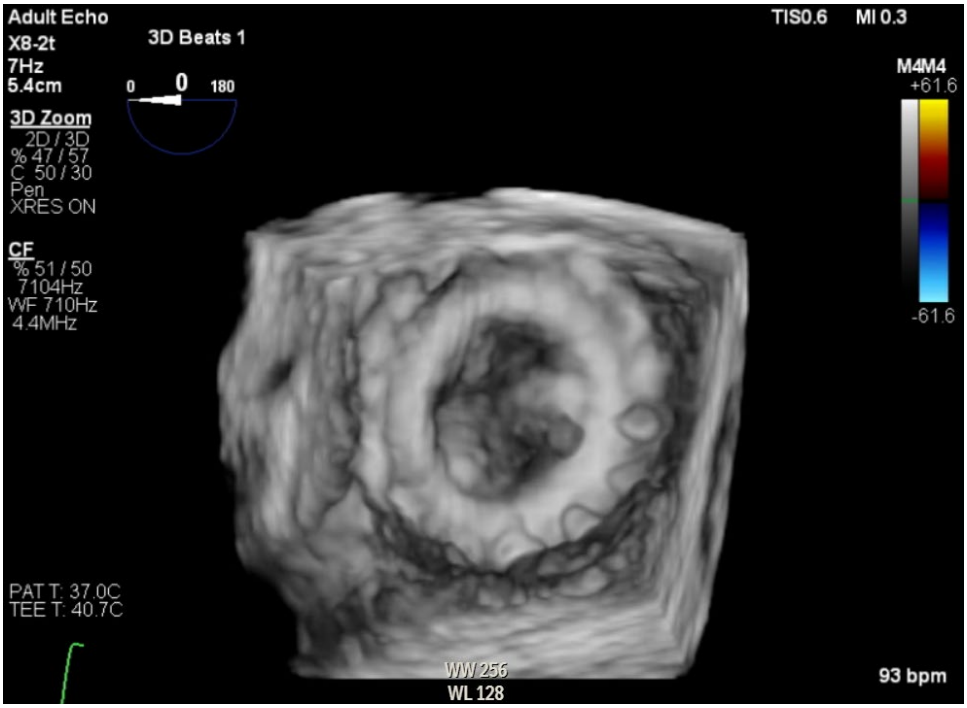
Perforation of LVOT into LA



Perforation of LVOT into LA with color comparison showing regurgitant flow



Post-bypass #1 showing severe mitral regurgitation originating from lateral aspect of valve below patched area



Post-bypass #2 showing a well-seated bioprosthetic valve in mitral position with no paravalvular leak

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#FR 2 – Kiosk 18 – Poster 3

Coronary Plaque Rupture and Cardiogenic Shock After Endoscopic Coronary Artery Bypass and Robotic Mitral Valve Repair/Aortic Valve Replacement

Presenting Author: Erik A. Ovrom, MD, Anesthesiology Resident, University of Chicago

Introduction

Post-cardiotomy cardiogenic shock (PCS) is a low cardiac output syndrome that occurs in 1-10% of all adult patients after cardiac surgery [1]. Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) is a rescue strategy used for support while the underlying cause is investigated. Proposed mechanisms of PCS include myocardial stunning, systemic inflammatory response to cardiopulmonary bypass (CPB), ischemic-reperfusion injury, or coronary occlusion due to direct tissue injury or acute plaque rupture.

Case Presentation

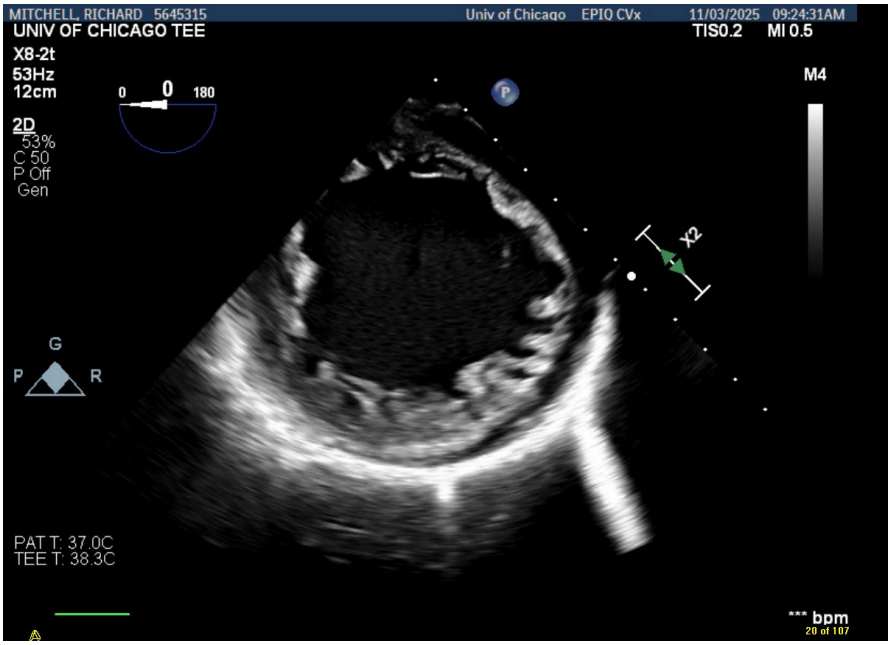
A 75-year-old male underwent minimally invasive cardiac surgery for severe mitral valve regurgitation (MR), severe aortic valve insufficiency (AI), and multi-vessel coronary artery disease with 90% occlusion of the mid left anterior descending (LAD) artery. A pre-bypass transesophageal echocardiogram (TEE) showed bicuspid aortic valve with severe AI, anterior leaflet mitral valve prolapse leading to an eccentric MR jet, and normal biventricular function. Following successful LIMA-LAD anastomosis, mitral valve repair (MVr) with partial ring annuloplasty, and aortic valve replacement, there was difficulty weaning from CPB. TEE now showed moderate global left ventricular hypokinesis and severe akinesis of the lateral wall. Despite inotropic support with dobutamine and epinephrine, the patient remained in cardiogenic shock and was ultimately placed on VA-ECMO and transferred to the Cath lab for Impella CP placement. Angiography done at that time showed complete occlusion of the left circumflex artery (LCX) due to plaque rupture, which was treated with balloon angioplasty.

Discussion

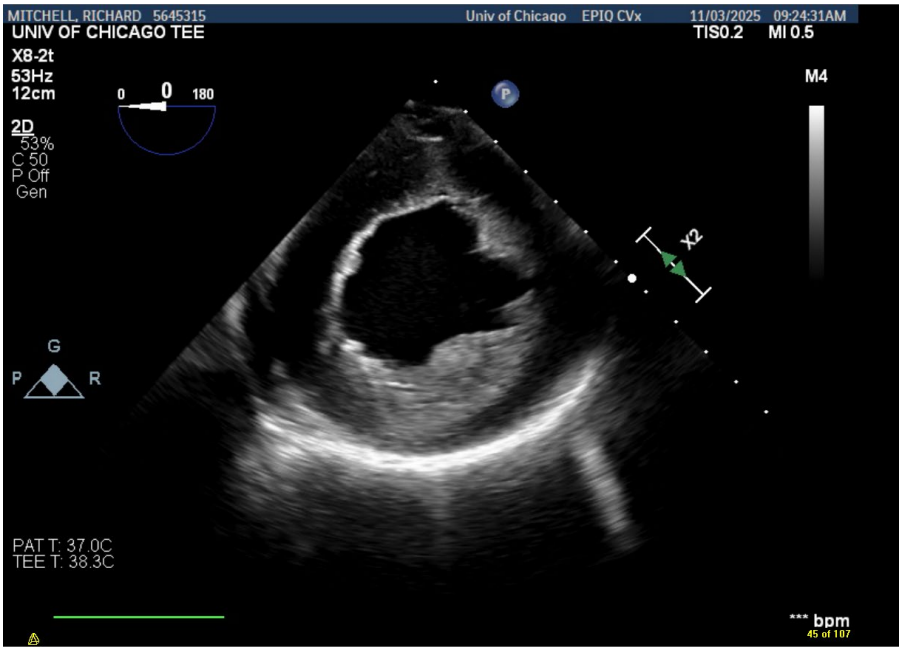
Post-cardiotomy cardiogenic shock may present early as an inability to wean the patient from CPB, and the use of rescue VA-ECMO, as in this patient's case, has been shown to improve survival to hospital discharge [2]. The cause of PCS varies based on procedure. Acute coronary occlusion is important to consider after MVr due to the proximity of the posterior mitral annulus to the LCX and the risk of placing a suture that dissects the artery. Hiltrop et al. suggested that minimally invasive surgery may pose an increased risk of LCX injury due to greater surgical complexity [3]. Despite the risk of injury to the LCX in mitral valve repair, acute plaque rupture is important to consider in any patient with untreated coronary artery disease. This patient had only mild luminal irregularities in the LCX on pre-op angiography. Though there is not consensus in the literature, the mechanism of LCX occlusion–mechanical injury vs coronary plaque rupture–may dictate whether creating a new bypass or PCI is the best course of action. In this case, the decision to proceed to the cath lab vs re-operation was driven by a low index of suspicion for mechanical LCX injury by the surgeon and severe cardiogenic shock that would benefit from Impella CP placement and diagnostic angiography. Angiography uncovered a lesion amenable to PCI. This case illustrates the importance of

employing clinical judgment to inform a broad diagnostic work-up when the cause of PCS remains unclear.

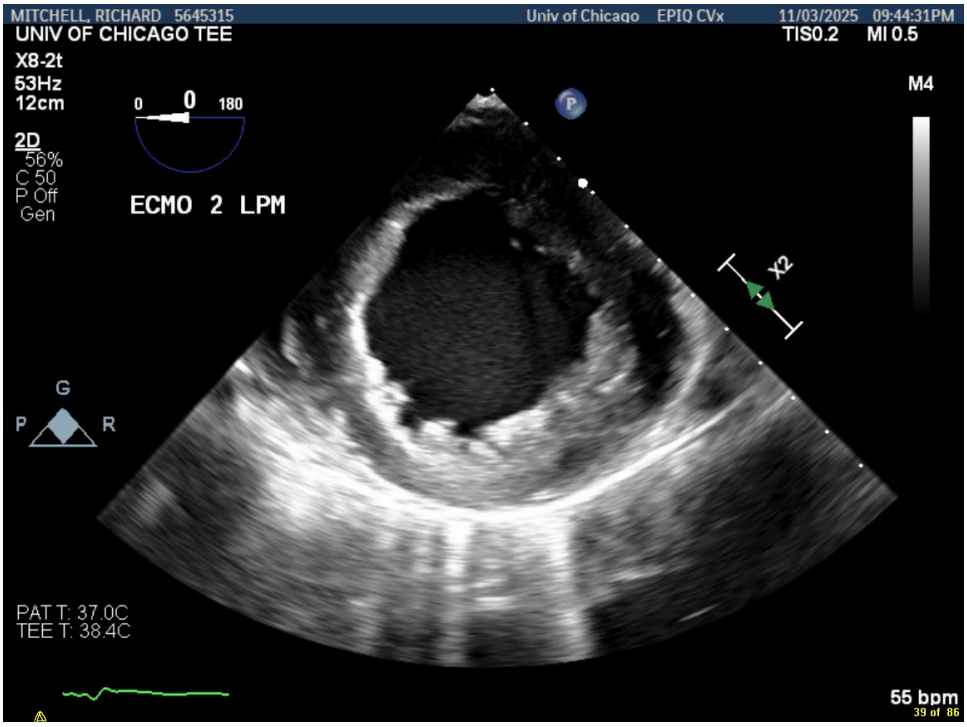
Images



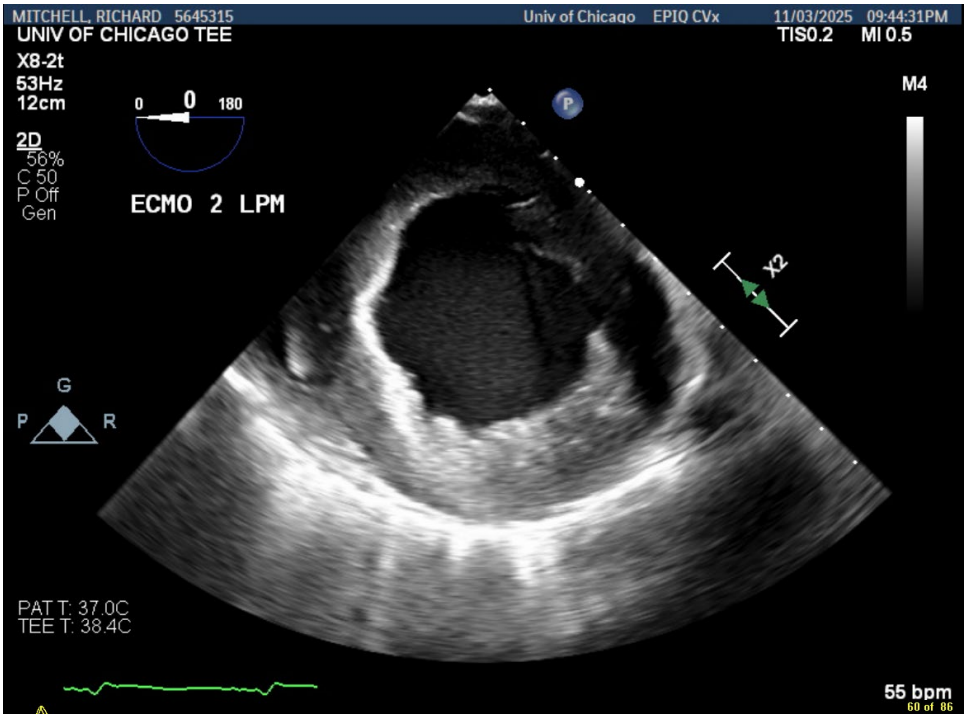
Pre-bypass Transgastric Diastole



Pre-bypass Transgastric Systole



Post TECAB/MVr/AVR Transgastric Diastole on ECMO



Post TECAB/MVr/AVR Transgastric Systole on ECMO

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#FR 2 – Kiosk 18 – Poster 4

Elective Cardiopulmonary Bypass for Non-Cardiac Surgery: Exploring the Applications of Extracorporeal Support

Presenting Author: Laura Lockard, MD, Resident Physician, The University of Rochester

Introduction

Extracorporeal support is widely used in cardiac surgery via cardiopulmonary bypass (CPB), but its application in non-cardiac procedures is far less common. Various modalities exist, including CPB and extracorporeal membrane oxygenation (ECMO), which can be configured as veno-venous (VV), veno-arterial (VA), or veno-veno-arterial (VVA). This case describes a patient with a large renal tumor and inferior vena cava (IVC) thrombus requiring elective use of extracorporeal circulation to facilitate resection.

Case Presentation

A 77-year-old woman presented with flank pain and was found to have a right renal mass. MRI revealed a 9.2 × 8.3 × 12.3 cm tumor with tumor thrombus extending from the right renal vein into the IVC and cephalad to the diaphragm. She was scheduled for open nephrectomy with radical lymphadenectomy and IVC thrombectomy. Multidisciplinary discussions involving urology, anesthesiology, cardiac surgery, perfusion, vascular and transplant surgery concluded that mechanical circulatory support would optimize surgical access and patient safety. Although VV ECMO was initially considered, the plan shifted to VVA ECMO with CPB backup. This would minimize the dose of heparin required and could decrease the risk of coagulopathy. Preoperative evaluation revealed mild left atrial enlargement on transthoracic echocardiography, normal cardiac stress testing and electrocardiography, and unremarkable labs. Physical exam was notable for obesity and Mallampati IV airway. After induction and intubation, central and peripheral venous access were obtained. Transesophageal echocardiography (TEE) demonstrated right ventricular dilation. Following laparotomy, concerns for massive hemorrhage and the need for a venous reservoir prompted reassessment, and the team elected to proceed with CPB rather than ECMO. Venous cannulas were placed in the distal IVC and left subclavian vein, and an arterial cannula in the left femoral artery. The patient was placed on CPB, enabling complete resection of the renal mass and IVC thrombus. She was successfully weaned from CPB with unchanged TEE findings. The abdomen was left open due to coagulopathic concerns. Postoperatively, her course was complicated by hemorrhage requiring re-exploration, hematoma necessitating drainage, portal vein thrombus treated with heparin, failed extubation, and pleural effusion requiring a chest tube. Despite these events, she was eventually extubated and discharged home.

Discussion

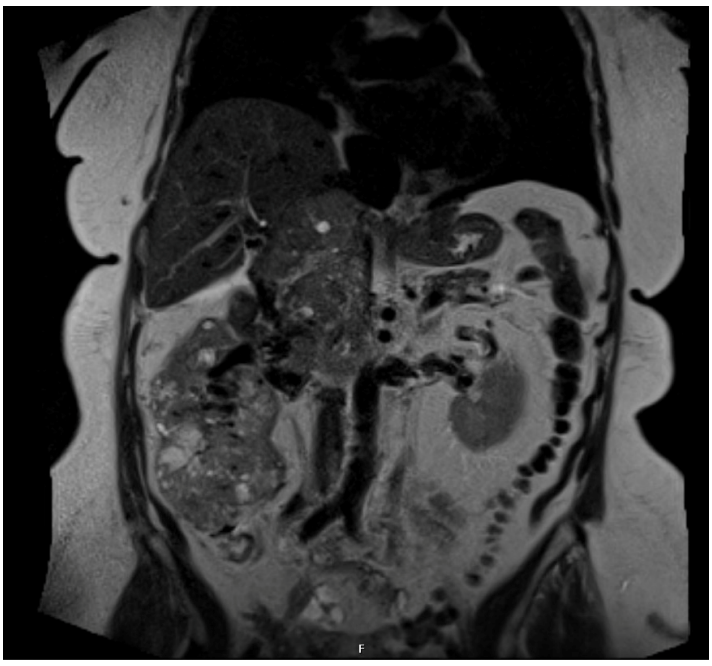
Preoperative planning focused on selecting the safest form of extracorporeal support. VV ECMO was initially considered to bypass the tumor, but the strategy shifted to VVA ECMO with low flow and CPB backup to preserve cardiac filling, minimize stasis, and allow controlled IVC clamping. However, the potential need for emergent conversion to CPB in the event of uncontrolled bleeding was a major concern. The team elected to initiate CPB electively rather than risk an urgent transition from ECMO. This approach enabled controlled

initiation and termination of bypass and complete tumor removal. This case highlights the value of proactive multidisciplinary planning and controlled implementation of CPB in complex oncologic surgeries involving major venous structures.

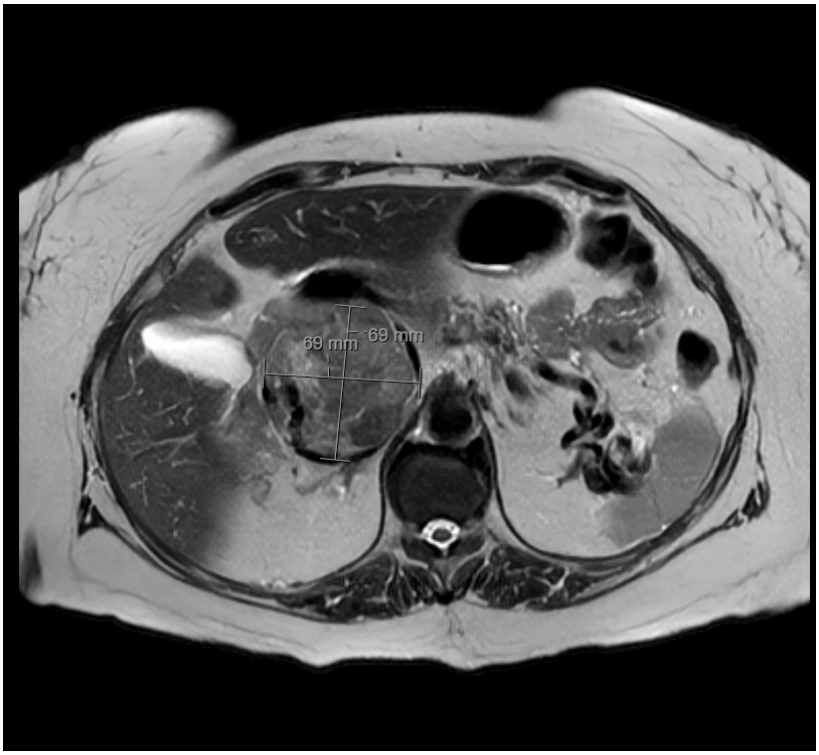
Images



Photograph of the solid renal tumor mass post-resection intraoperatively with ruler for scale.



Coronal T2 MRI imaging of the abdomen demonstrating the extensive heterogeneous right renal mass.



Axial T2 MRI imaging of the abdomen demonstrating IVC tumor thrombus.

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#FR 2 – Kiosk 18 – Poster 5

Intraoperative Annuloplasty Ring Dehiscence in a Minimally Invasive Mitral Valve Repair, Atrial Fibrillation Cryoablation and Left Atrial Appendage Ligation

Presenting Author: Shankha Chakravorty, MD, Resident Physician, Northwell - North Shore University Hospital

Introduction

Minimally invasive mitral valve repairs (MI-MVr's) have been an increasingly accepted surgical option to address mitral valvulopathies. Valve repair surgeries are also more commonly being combined with procedures for other comorbidities, such as left atrial ablations for atrial fibrillation. The data on outcomes of these relatively newer modalities of treatment for cardiac pathologies is limited. We present a case of a patient undergoing a MI-MVr, cryoablation, and left atrial appendage ligation complicated by posterior annuloplasty ring dehiscence.

Case Presentation

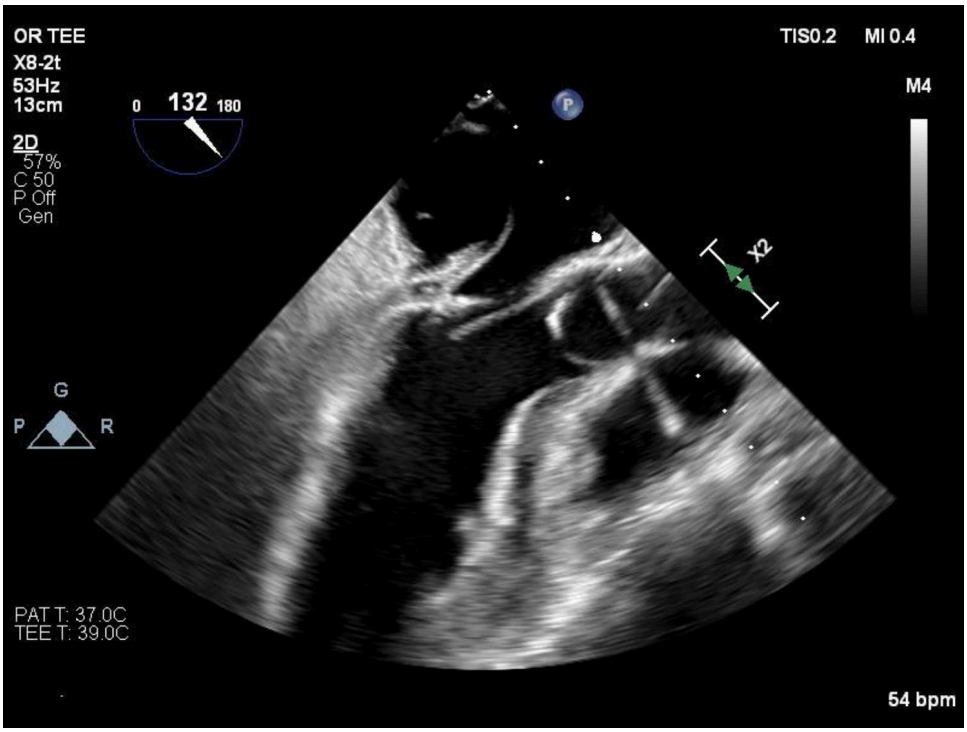
68-year-old male PMHx of HTN, HLD, A-Fib no longer on anticoagulation presented with exertional dyspnea with workup revealing severe MR. Patient was scheduled for a minimally invasive MVR, Cox-Maze A-Fib ablation, and LAA ligation. The patient was uneventfully induced and intubated with a double lumen tube. After initiation of cardiopulmonary bypass (CPB), a mitral valve annuloplasty was performed with a 30mm flexible ring. Prior to separating from CPB, a left atrial appendage ligation with a 40 mm atrial clip and a COX-MAZE cryoablation were performed. The aortic cross-clamp was removed, the heart resumed in sinus rhythm, and prior to weaning off CPB, the TEE exam now showed a dehiscence of the posterior aspect of the annuloplasty ring extending to P3. There was a return to CPB and the valvular ring was repaired. Subsequently, the patient was successfully weaned off bypass with no residual paravalvular leak. The patient's postoperative course was grossly uncomplicated and was discharged home within one week.

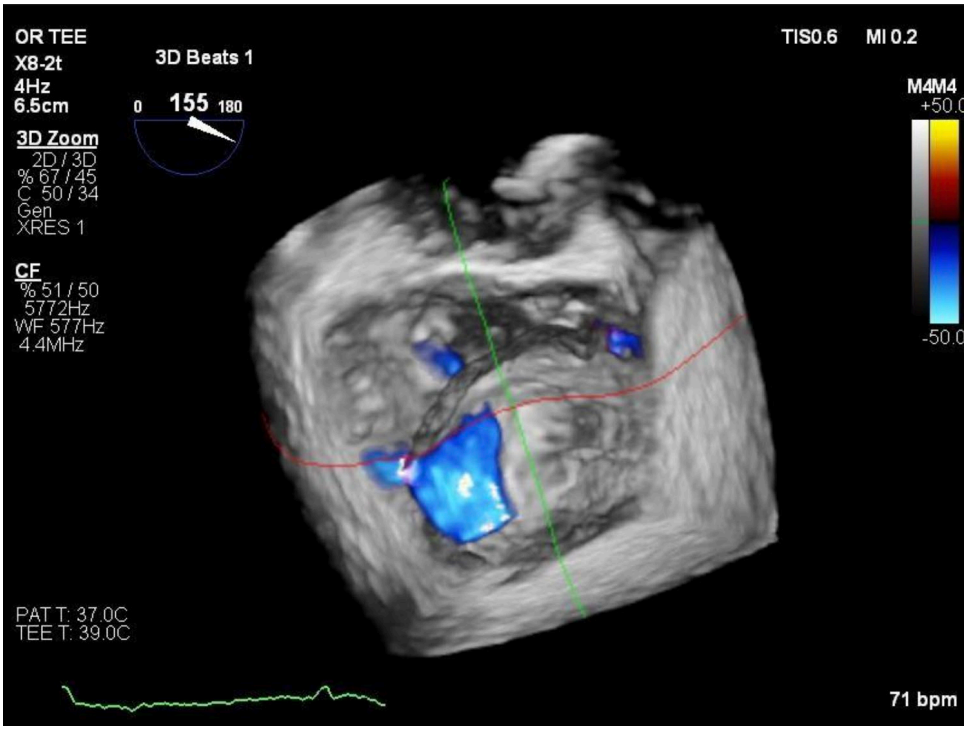
Discussion

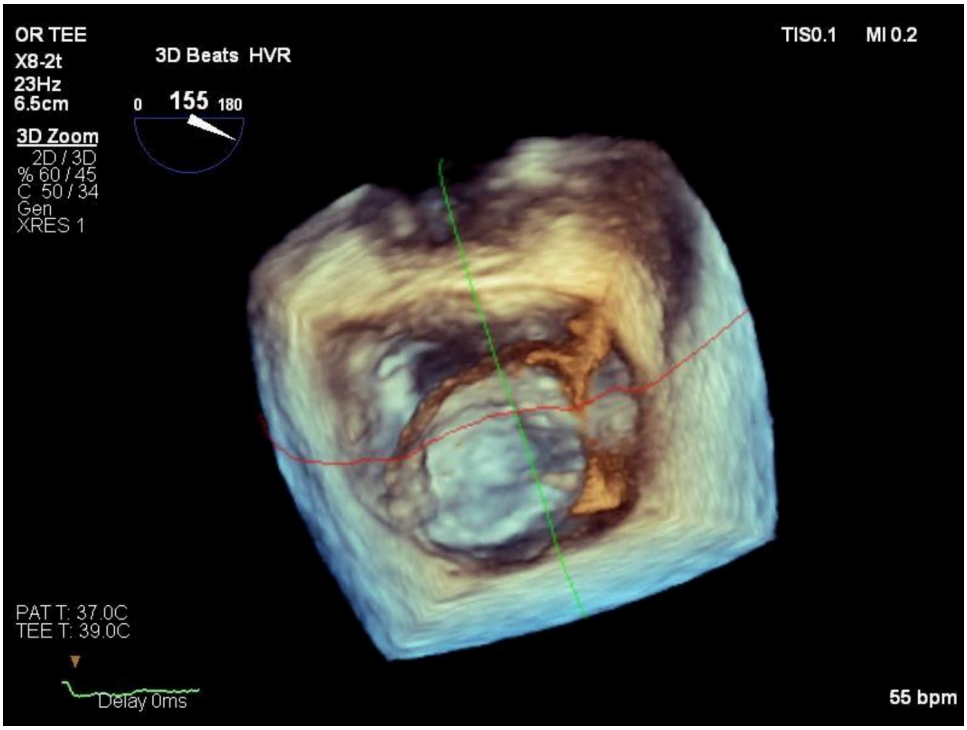
Studies have examined the incidence of annuloplasty ring dehiscence after mitral valve repair, which show that ring dehiscence is more likely to occur at the posterior annulus and is more commonly an acute/early complication (<30 days from operation). These studies, however, mostly look at isolated valve repair operations rather than also analyzing valve operations that were combined with procedures such as left atrial cryoablation for atrial fibrillation. A study investigating isolated valve surgeries compared to valve surgeries with cryoablation showed no significant difference in adverse outcomes between the two groups, but did not specifically analyze mitral valve operations nor instances of mitral valve annuloplasty ring dehiscence. Although one possible cause of the posterior ring dehiscence could be related to surgical technique, another potential cause could be related to the COX-Maze procedure. There was speculation that the maze cryoablation may have caused an insult to the integrity of the repair. Regardless of the cause of this intraoperative event, it remains an important component to evaluate with TEE prior to separating from

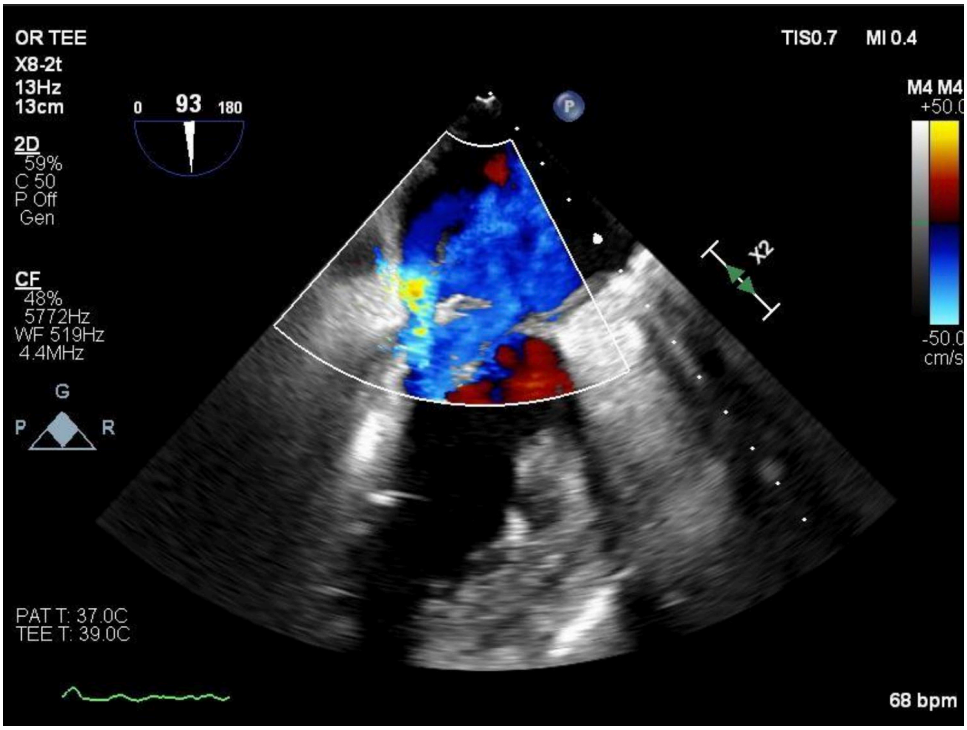
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OR TEE
X8-2t
23Hz
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C 50 / 34
Gen
XRES 1
TIS0.1 MI 0.2
PAT T: 37.0C
TEE T: 39.0C
Delay 0ms
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#FR 2 – Kiosk 18 – Poster 6

A Three-Decade-Old Patent Ductus Arteriosus: Easy Ligation or Something More?

Presenting Author: Luying Yan, MD, Resident Physician, Brigham and Women's Hospital

Introduction

Patent ductus arteriosus (PDA) occurs in about 0.02% of births, with most cases treated in childhood [1]. In adults, definitive closure can be complicated by cardiac and vascular remodeling, pulmonary hypertension (PH), and heart failure (HF). Detailed imaging and catheterization are essential in defining anatomy and hemodynamics to guide surgical and anesthetic planning. Understanding physiologic changes before and after PDA occlusion is critical for optimizing perioperative management.

Case Presentation

A 34-year-old male, previously residing at high altitudes, was found to have a large PDA (Figure 1) with significant left-to-right shunting, PH, and severe left ventricular (LV) dilation during evaluation of an asymptomatic murmur. Catheterization revealed a cardiac index of 2.7 L/min/m^2, Qp:Qs of 5.2:1, and near-normal pulmonary vascular resistance (PVR) of 3.2 iWU. Balloon test occlusion decreased pulmonary artery (PA) pressure by ~30mmHg but caused marked systemic hypotension, prompting a surgical approach to allow for controlled test occlusion and to prevent long-term HF. A right radial arterial line was placed prior to induction given the possibility of arch work. General anesthesia was induced with fentanyl, midazolam, dexmedetomidine, and rocuronium without hemodynamic alterations. Following intubation, FiO2 was reduced to 30% and EtCO2 was maintained at 35-40 mmHg to avoid shunting exacerbation. FloTrac was used for hemodynamic monitoring instead of a PA catheter given anticipated PA manipulation. Intraoperative transesophageal echocardiography (TEE) demonstrated a large PDA with torrential shunt flow (Figures 2 and 3) and severe LV dilation with preserved systolic function. Surgical inspection showed fragile ductal tissue, leading to initiation of cardiopulmonary bypass (CPB) for safe manipulation. Subtotal occlusion was first achieved with a temporary Silastic band and clips, followed by trial separation from CPB with milrinone, low-dose epinephrine, and inhaled epoprostenol to mitigate post-ligation cardiac syndrome (PLCS). With a 65 mmHg gradient across the band, LV systolic function was only mildly reduced and systemic pressures remained stable. A permanent Gore-Tex band was then placed for complete occlusion. Final TEE showed mild LV systolic dysfunction, normal right ventricular function, and no residual ductal flow (Figure 3). PA pressures improved from 70s/30s to 40s/20s mmHg, and Qp:Qs normalized to 1.1:1. The patient remained hemodynamically stable post-ligation, was extubated on postoperative day (POD) 1, and weaned off pharmacological support by POD 4.

Discussion

This case highlights important considerations in adult patients with large PDAs, where ductal friability, calcification, aneurysmal change, or unfavorable anatomy may require CPB and potential arch intervention. Pre-CPB management should balance SVR and PVR to avoid worsening shunting and systemic hypotension. Vigilance for PLCS is crucial, as abrupt increases in afterload after PDA occlusion can precipitate acute LV

dysfunction, systemic hypotension, and pulmonary edema [2]. In this case, proactive titration of milrinone, epinephrine, and inhaled epoprostenol supported ventricular function with afterload reduction, contributing to an uncomplicated recovery.

Images

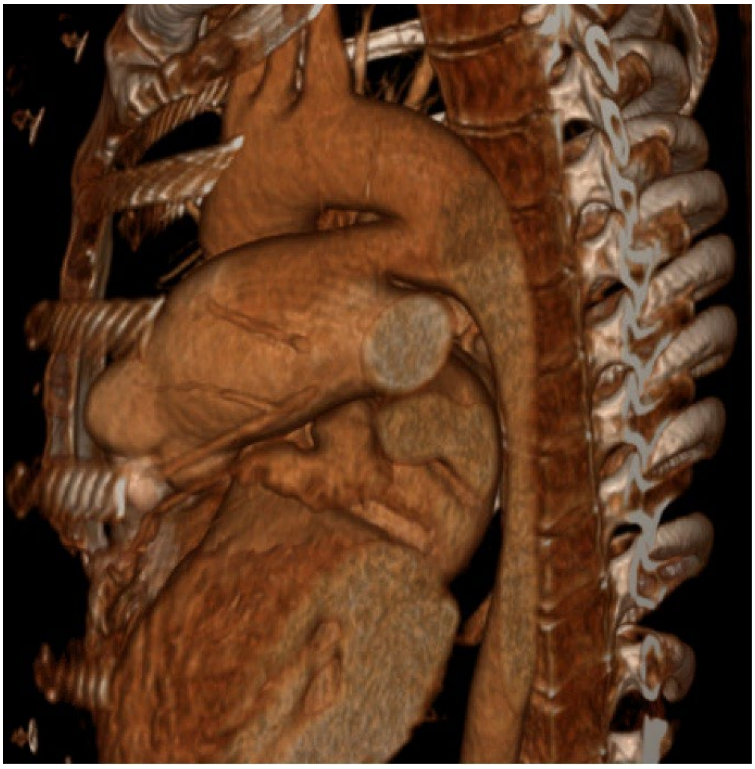


Figure 1. 3-Dimensional (3D) CT angiography reconstruction of the PDA, measuring 2.6 cm in length, 1.6 cm x 1.2 cm at the main pulmonary artery (PA), and 1.8 cm x 2.4 cm at the aorta.

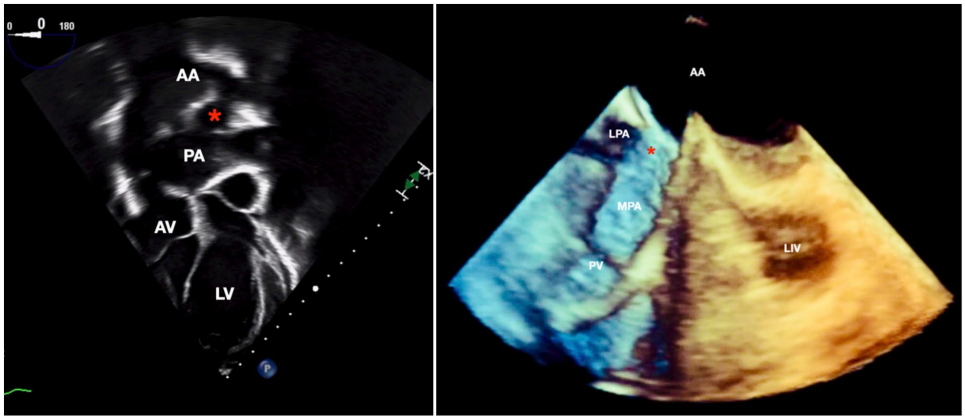


Figure 2. Intraoperative TEE visualizing the large PDA. Left: 2D; Right: 3D. AA: aortic arch, PA: pulmonary artery, AV: aortic valve, LV: left ventricle, LPA: left PA, MPA: main PA, PV: pulmonary valve, LIV: left innominate vein, Red asterisk: PDA

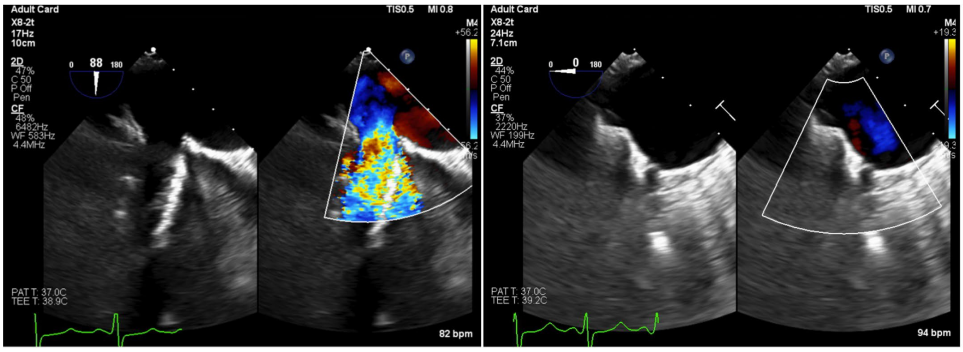


Figure 3. Color flow doppler comparison of the PDA pre- (left) and post-occlusion (right).

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#FR 2 – Kiosk 18 – Poster 7

Anesthetic Management of Valve Embolization During Transcatheter Aortic Valve Replacement

Presenting Author: Joseph Catania, MD, Resident, Northwell Health

Introduction

Valve embolization during transcatheter aortic valve replacement (TAVR) is a rare but serious complication with rates of approximately 1% (1). Post-deployment valve embolization can lead to life threatening complications including aortic injury or dissection, stroke, and myocardial infarction. The most common causes for valve embolization are initial malposition, poor anatomy, and pacing failure (2). In this case presentation, we discuss the perioperative anesthetic management of a TAVR procedure complicated by valve embolization.

Case Presentation

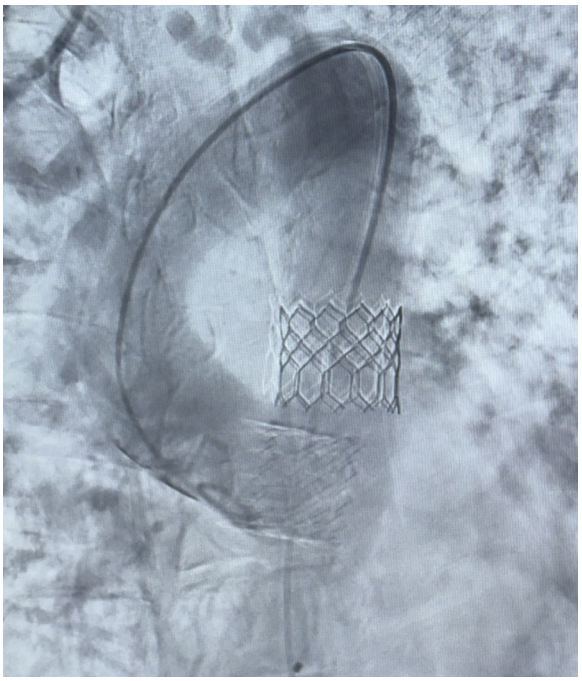
A 78-year-old male with a past medical history of hypertension, hyperlipidemia, and previously resected soft tissue malignancy presented for a TAVR. Intraoperative transthoracic echocardiography (TTE) showed a left ventricular ejection fraction of 53% and severe aortic stenosis. There were no other significant findings on TTE. The patient was sedated with propofol and dexmedetomidine. A low-dose remifentanil infusion was used for analgesia. The patient was rapidly paced prior to valve deployment at 180 beats per minute; however, this was complicated by a brief loss of pacing capture. The valve was deployed during this period and was immediately found to have embolized into the aortic root. The valve could not be repositioned to the native annulus and could not be recaptured. The patient became hypotensive, and frequent ectopic beats were observed, so a norepinephrine infusion was started to maintain hemodynamic stability. At this point, there was particular concern that the valve could further embolize and obstruct cardiac, systemic, or cerebral blood flow, and an attempt was made to mobilize the valve into the descending thoracic aortic using a balloon. A joint decision was made by the anesthesia and surgical teams to defer conversion to general anesthesia (GA) to allow for rapid intervention and a faster neurological evaluation postoperatively. A balloon was deployed, and the valve was successfully pulled into the descending thoracic aorta; the valve was then observed to ensure there would be no further migration. Following this, a second bioprosthetic valve was then successfully deployed in the aortic position. Pressors were weaned off, sedation was discontinued in the operating room, and the patient was transferred to the post-anesthesia care unit. The patient recovered uneventfully and was discharged home two days later. The embolized valve remains in place in the descending thoracic aorta and will be followed over time with serial imaging.

Discussion

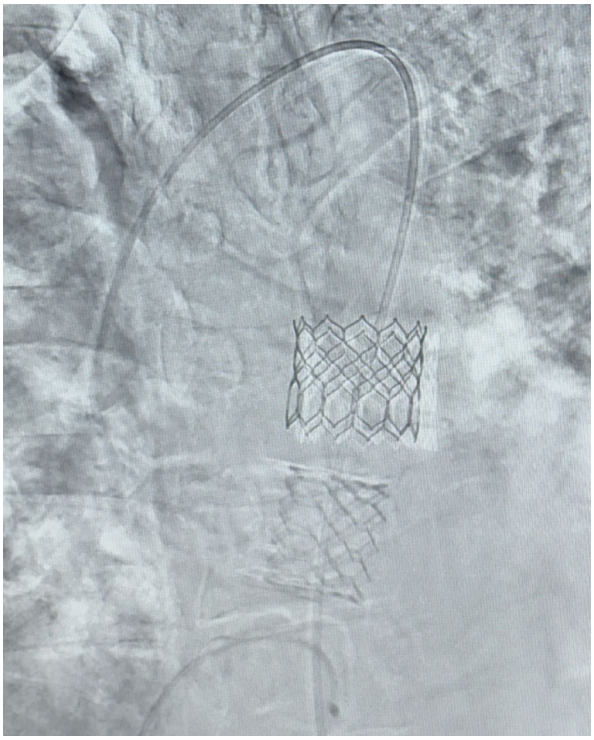
In this case, the loss of pacing capture directly led to valve embolization, which is consistent with other reports (2). The loss of pacing capture was immediately communicated to the surgical team, but the valve was already deployed and had embolized. This patient was under sedation, and TAVR procedures can be done with either sedation or GA. Sedation has been associated with improved outcomes (2). Although conversion to GA is often considered for major procedural complications, in this case conversion would

have further worsened hemodynamic instability during a time that required rapid intervention. This case report highlights the importance of prompt communication between the anesthesia and surgical teams and emphasizes adaptability during structural heart procedures.

Images



Two valves with contrast. One appropriately placed in aortic position and another in descending thoracic aorta.



Two valves without contrast. One appropriately placed in aortic position and another in descending thoracic aorta.

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#FR 2 – Kiosk 19 – Poster 1

Pheochromocytoma Resection in a Patient with Corrected Tetralogy of Fallot

Presenting Author: Martine Alison, M.D., Anesthesia Resident Physician, University of Virginia

Introduction

The anesthetic management during resection of pheochromocytomas (PCC) and paragangliomas in patients with cyanotic congenital heart disease (CCHD) has been discussed in several case reports/series and there is evidence of a link between patients with CCHD and development of these tumors1-2. We present a case of a patient with palliated CCHD undergoing resection of PCC.

Case Presentation

A 42 year-old female with history of Tetralogy of Fallot and aortic coarctation repaired via transannular patch and subclavian flap repair at 5 months of age initially presented to cardiac surgery clinic for evaluation of pulmonic valve replacement. Echocardiography noted free pulmonary insufficiency (PI) with significant right ventricular (RV) dilation and mild RV dysfunction. Past medical history included hypertension, hyperlipidemia, obesity, obstructive sleep apnea, and non-insulin dependent diabetes mellitus. During work up for her medically resistant hypertension (managed with metoprolol, chlorthalidone, candesartan and amlodipine), the patient was diagnosed with a PCC and scheduled for robotic assisted right adrenalectomy ahead of her cardiac procedure. She was started on doxazosin and the perioperative team decided she should hold her beta blocker for surgery as this could be detrimental to RV performance. On the day of surgery, the patient was hypertensive. Prior to induction an arterial line was placed in the right radial artery and she was started on nicardipine and nitroprusside infusions. Induction was performed with fentanyl, lidocaine, propofol and rocuronium. She was intubated with a 7.0 cuffed endotracheal tube. A triple lumen central venous catheter was subsequently placed in the right internal jugular vein. The vasodilators were stopped after induction and a norepinephrine infusion was started. During excision of the PCC, her blood pressure was extremely labile and norepinephrine was stopped and nicardipine and nitroprusside infusions restarted. She was also given 5mg of phentolamine. After tumor resection, the patient was profoundly hypotensive and supported by norepinephrine, epinephrine and vasopressin infusions. She tolerated the procedure well and was transported intubated and sedated to the surgical ICU stable on epinephrine, norepinephrine and vasopressin. She was extubated and weaned off pressors POD1. On POD3 she was discharged. The tumor pathology report was consistent with PCC.

Discussion

Although the patient's congenital heart disease was repaired, she had significant PI and a dilated RV with decreased systolic function. This required conservative fluid administration and intricate intraoperative blood pressure control and ionotropic support. Phentolamine was important in this effort as it can lower both pulmonary and systemic vascular resistance. Following the surgery, the patient had improved control of their blood pressure and diabetes. Her A1c reduction was likely from reduced beta-adrenergic activity resulting in less insulin secretion and decreased insulin resistance. Interestingly, the patient had worsening

of their respiratory symptoms with PFTs demonstrating responsiveness to bronchodilator therapy, a phenomenon described after PCC removal due to reduction in bronchodilation caused by beta-adrenergic activity from the tumor's catecholamine release3.

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#FR 2 – Kiosk 19 – Poster 2

Suspected Cryoprecipitate-Associated Allergic Reaction in a Patient with Alpha-Gal Syndrome Undergoing Aortic Valve Replacement

Presenting Author: Stephanie A. Michaels, MD, Anesthesiology Resident, MedStar Georgetown University Hospital

Introduction

A 42-year-old woman with alpha-gal syndrome and severe aortic stenosis underwent uncomplicated open aortic valve replacement with deployment of a bioprosthetic valve. Point of care testing during closure indicated a fibrinogen deficit, prompting administration of blood type B cryoprecipitate. Immediately post-transfusion the patient became hemodynamically unstable requiring vasopressor support and treatment for an allergic reaction.

Case Presentation

A 42-year-old woman with type O blood, mast cell disorder, alpha-gal syndrome, migraines, and severe aortic stenosis presented for surgical aortic valve replacement. She was diagnosed with alpha-gal syndrome nine months prior to surgical aortic valve replacement, and this was re-confirmed with allergen testing for alpha-gal, pork, beef and lamb IgE. General anesthesia was induced with fentanyl, etomidate, and rocuronium followed by pre-medication with diphenhydramine and hydrocortisone. Surgical exposure was uneventful, and the patient received porcine derived unfractionated heparin prior to cannulation without hemodynamic change. She was cannulated for CPB at the aorta and right atrial appendage, followed by administration of antegrade cardioplegia. A 21mm Inspiris Bioprosthetic tissue valve was deployed, and the patient was weaned from bypass. Post-protamine ROTEM showed a clotting deficit; 2 units of type O FFP and 10 pools of cryoprecipitate (5 type B, 5 type O) were administered. Immediately after administration of cryoprecipitate, the patient developed abrupt hypotension with MAPs dropping from the mid-80s to low-50s and a mild rise in airway pressures though oxygenation and ventilation were maintained. The patient was started on infusions of norepinephrine and vasopressin and given additional doses of diphenhydramine and hydrocortisone. MAPs stabilized, closure was completed, and she was transported to the (ICU) on epinephrine, norepinephrine and vasopressin infusions. In the ICU the patient did not show signs of alternative transfusion reactions. She was weaned from vasopressor support and extubated on POD 1. The remainder of her hospitalization was uncomplicated, and she was discharged on POD 4.

Discussion

Alpha-gal syndrome is an IgE-mediated hypersensitivity reaction to the oligosaccharide galactose-alpha-1,3-galactose which is commonly found in animal proteins and animal derived medical products. In cardiac surgery potential high-risk exposures include the use of porcine derived heparin and bioprosthetic valves (1). More recently transfusion-related alpha gal syndrome has recognized that blood products particularly from blood type B donors can trigger allergic reactions due to similarities between blood group B antigen and the alpha-gal oligosaccharide (2). There are recent reports of alpha-gal patients with blood type O receiving blood type B plasma and platelets resulting in anaphylactic and allergic reactions (3). This case reinforces and expands the known reaction association by demonstrating that in products with low dose ABO-incompatible plasma like in cryoprecipitate can result in allergic reaction in alpha-gal syndrome patients. Although a tryptase level could have confirmed the diagnosis, the timing and response to diphenhydramine, hydrocortisone, and low-dose epinephrine suggest an allergic reaction.

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#FR 2 – Kiosk 19 – Poster 3

Early Postpartum Type A Dissection in a Preeclamptic Patient: Implications of Pregnancy-Related Vascular Remodeling for Cardiac Anesthesia

Presenting Author: Lee Manchio, DO, Anesthesiology Resident, Rutgers NJMS

Introduction

Acute Type A dissection in the early postpartum period remains an uncommon yet high-risk presentation. Pregnancy produces structural and functional vascular changes that persist beyond delivery, while preeclampsia adds significant hemodynamic strain and endothelial disruption. Magnesium therapy shapes vascular tone and attenuates adrenergic responses. The postpartum period creates a fragile hemodynamic state easily exacerbated by physiologic stress. Combined with abrupt postpartum volume shifts and physiologic changes, these factors create conditions that complicate anesthetic planning and increase vulnerability during induction and cardiopulmonary bypass.

Case Presentation

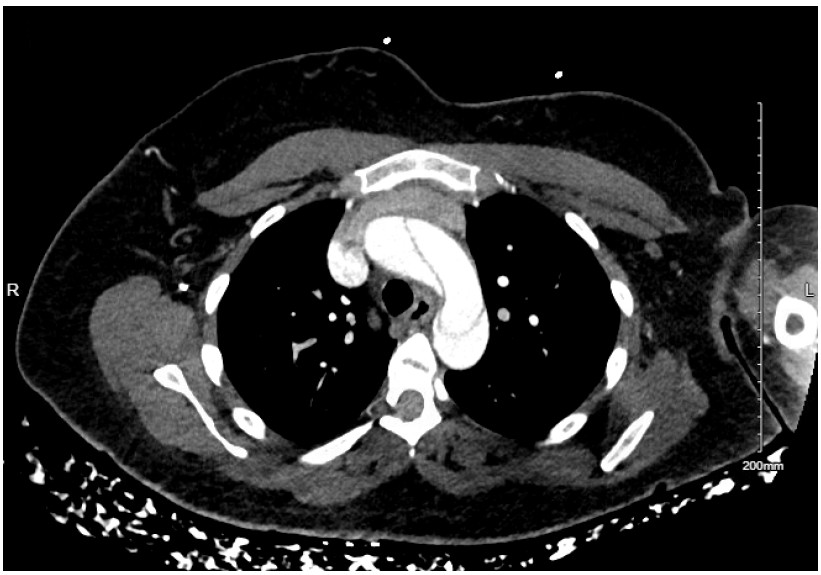
A 37-year-old African American woman, postoperative day 2 from a vaginal delivery, G4P3, with chronic hypertension and preeclampsia managed with a 24-hour magnesium infusion, developed sudden chest pain radiating to the back. Her systolic pressure exceeded 200 mmHg. CTA demonstrated a Type A dissection extending from the aortic root to the abdominal aorta as well as an ascending aortic aneurysm measuring 5cm. Intraoperative TEE identified a Type A dissection, originating just below the junction and extending into the distal thoracic aorta, moderate to severe LV hypertrophy, and preserved ventricular function. Induction with midazolam, propofol, fentanyl, lidocaine, and succinylcholine resulted in stable hemodynamics. Right brachial and left radial arterial catheters provided continuous monitoring. A central venous catheter, pulmonary artery catheter, and intraoperative TEE guided management. Surgical repair consisted of an ascending aortic replacement with a 32-mm graft and aortic valve resuspension. Cardiopulmonary bypass time was 198 minutes, cross-clamp 94 minutes, and circulatory arrest 29 minutes with retrograde cerebral perfusion. Intraoperative support included 2 units plasma, 2 units platelets, and 3 liters of crystalloids. The patient was weaned from bypass on milrinone 0.3 mcg/kg/min and was transported to the ICU. She was extubated POD#1 and discharged POD#6 without neurologic or renal sequelae.

Discussion

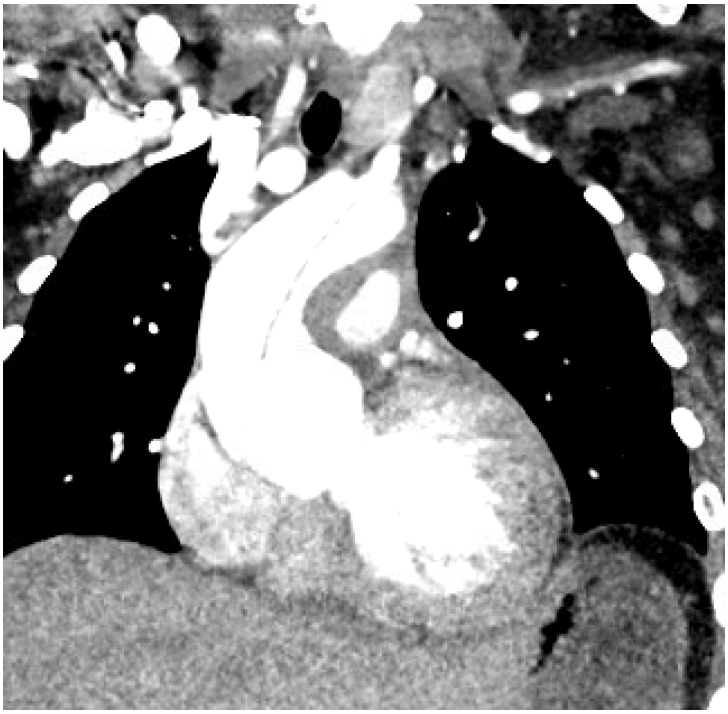
Postpartum Type A dissection introduces anesthetic challenges shaped by vascular remodeling from

pregnancy and the hypertensive impact of preeclampsia. These factors can blunt adrenergic responses and produce unpredictable hemodynamic reactions during induction. The excess shear forces during pregnancy can predispose to vascular aneurysms. The postpartum period adds increased cardiac output, abrupt shifts in intravascular volume, and inconsistent vasoreactivity. Magnesium exposure alters anesthetic effectiveness and increases the risk of uterine atony. Our patient had an aneurysmal aortic arch, which combined with her hypertensive disorders put her at risk for a vascular emergency. The coagulopathy associated with cardiopulmonary bypass can intensify bleeding concerns. This fragile physiologic state required tight blood pressure control and minimal changes in vascular tone to prevent growth of the dissection. Dual-site arterial monitoring enabled control of systolic and mean pressures to avoid propagation of the dissection and prevent malperfusion. Early imaging and coordinated multidisciplinary management supported a favorable perioperative outcome in this high-risk clinical scenario.

Images



Coronal view of Type A Dissection



Ascending aortic aneurysm measuring up to 5.0 cm in diameter

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#FR 2 – Kiosk 19 – Poster 4

Anesthetic management for endovascular retrieval of an iliac vein stent migrated to the right heart

Presenting Author: Jibril F. Kedir, MD, PhD, Resident, Massachusetts General Hospital

Introduction

Iliac vein stenting is a guideline supported intervention for symptomatic chronic venous stasis from common iliac vein obstruction in patients unresponsive to conservative therapy. While generally safe, stent migration is a rare (< 1%) but potentially devastating complication [1, 2]. Intracardiac stent migration can cause severe arrythmia, tricuspid regurgitation (TR), cardiac perforation, or pericardial tamponade with mortality rates up to 16% [3]. Here, we describe a patient whose iliac vein stent migrated into the heart, highlighting the challenging anesthetic management and the multidisciplinary coordination needed for its retrieval.

Case Presentation

A 38-year-old female with history of tobacco use and chronic venous insufficiency with bilateral lower extremity venous claudication was found to have complete obliteration of the left common iliac vein (CIV) on pelvic venography, which was then treated with the placement of an 18x80 mm Abre stent. Shortly after the procedure, the patient developed palpitations, dizziness, and dyspnea, prompting presentation to the emergency department. On examination, the patient was hemodynamically stable but had frequent premature ventricular contractions (PVCs) and runs of non-sustained ventricular tachycardia (NSVT) worsened with movement. A transthoracic echocardiogram (TTE) revealed a metallic stent extending from the right atrium (RA) to the right ventricle (RV), a normal RV function, and no pericardial effusion (Figure 1). The patient was admitted to the Cardiac Intensive Care Unit and placed on immediate bed rest. A multidisciplinary decision was made to attempt endovascular retrieval with cardiac surgery backup, as well as to develop specific plans for potential high-risk complications (Table 1). The patient was subsequently brought to the cardiac catheterization laboratory, induced under general anesthesia, and endotracheally intubated. Afterwards, invasive monitoring was established via an arterial line and a right internal jugular central venous catheter. A comprehensive intraoperative transesophageal echocardiogram (TEE) was then performed to guide the retrieval. The interventional team was able to snare the proximal end of the stent through the right femoral vein approach. The entire device was then successfully collapsed into a sheath and removed percutaneously. The patient remained hemodynamically stable throughout, and post-retrieval TEE confirmed complete removal of the foreign body, trace tricuspid regurgitation, and no pericardial effusion (Figure 2 & 3).

Discussion

Venous stent migration to the heart is rare, and its retrieval carries a high-risk of severe complications. This case highlights two key elements that enabled us to have a successful percutaneous retrieval of a large-caliber venous stent from the right heart: (1) a proactive, multidisciplinary team (including cardiac anesthesiology, interventional cardiology, and cardiothoracic surgery) for compressive planning and preparedness, and (2) the use of real-time TEE guidance to ensure precise snare navigation, thereby avoiding

valvular injury or cardiac perforation and eliminating the need for a sternotomy.

Images

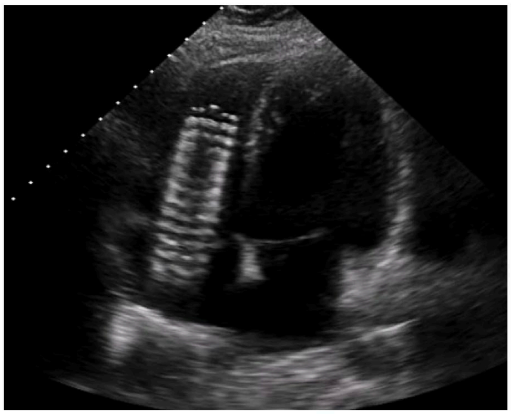


Figure 1: Apical 4-chamber view obtained from transthoracic echocardiogram (TTE). A long metallic stent measuring approximately 23 x 82 mm is visualized extending from the RA through the TV into the RV. The migrated stent is large and nearly occupies the entire tricuspid valve orifice.

Table 1: Case-Specific Concerns and Corresponding Anesthetic and Multidisciplinary Management Plan for Stent Retrieval

Potential Complication	Case-Specific Concern	Anesthetic & Multidisciplinary Preparedness Plan
Ventricular Arrhythmias	• Patient already presented with PVCs and NSVT, indicating myocardial irritability from the stent's position in the RV. • Manipulation during retrieval could provoke sustained VT or VF.	**Monitoring:** Continuous 12-lead ECG and arterial line. **Preparedness:** External defibrillator pads placed pre-induction, and antiarrhythmics (e.g., amiodarone, lidocaine) immediately available.
Tricuspid Valve (TV) Injury	• Stent crossed the TV and appeared to be lodged in the sub-valvular apparatus. • Retrieval could cause leaflet tear, chordal rupture, or flail leaflet, leading to acute severe tricuspid regurgitation and right heart failure.	**Monitoring:** Continuous direct TEE visualization to guide retrieval and immediately assess valve integrity. **Preparedness:** Norepinephrine in line with Epinephrine, Dobutamine, and Milrinone immediately available, and cardiac surgery team on standby for emergent sternotomy and valve repair/replacement.
Cardiac Perforation & Tamponade	• Abre stent is a self-expanding stent designed to exert a strong outward radial force. • The stent could perforate the RA or RV free wall during manipulation, leading to acute pericardial effusion and hemodynamic collapse.	**Monitoring:** Continuous TEE for new pericardial effusion, arterial line and central venous access for hemodynamic monitoring and administration of inotropes and vasopressors. **Preparedness:** Pericardiocentesis equipment immediately available, with emergent sternotomy backup.
Hemorrhage / Bleeding	• Significant bleeding risk from either the large-bore venous access site required for the retrieval sheath, from vascular injury during stent manipulation, or from a potential cardiac perforation.	**Monitoring:** Arterial line for beat-to-beat blood pressure monitoring and ability to obtain labs. **Preparedness:** Active T&S, large-bore IV access, blood products (pRBCs) are confirmed as available in room, with the ability to prepare FFP and PLT for balanced transfusion, and vasoactive infusions (e.g. norepinephrine).

Abbreviations: PVC, Premature Ventricular Contractions; NSVT, Non-sustained Ventricular Tachycardia; VT, Ventricular Tachycardia; VF, Ventricular Fibrillation; RV, Right Ventricle; TV, Tricuspid Valve; TEE, Transesophageal Echocardiogram; T&S, Type and Screen; IV, Intra-venous; pRBCs, Packed Red Blood Cells; FFP, Fresh Frozen Plasma; PLT, Platelets.

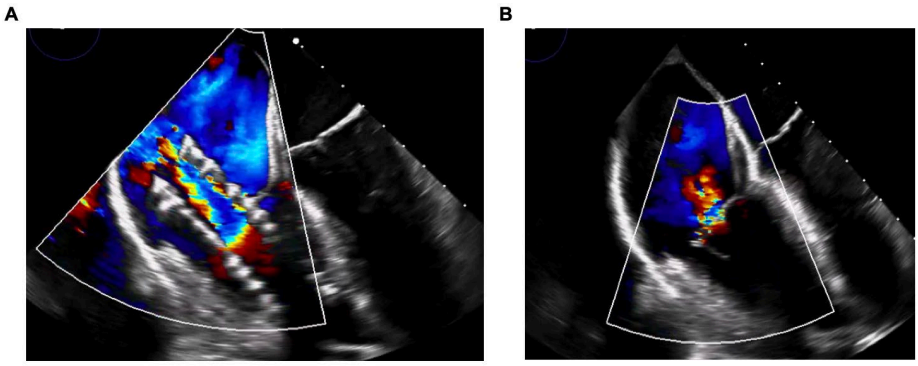


Figure 2: Mid-esophageal 4-chamber view from intra procedural transesophageal echocardiogram (TEE) pre and post stent extraction. (A) The embolized venous stent was visualized, extending from the RA into the RV, resulting in severe tricuspid regurgitation. (B) Post-extraction imaging demonstrated mild tricuspid regurgitation (TR), and mildly thickened TV leaflets, but the chordal apparatus was intact.



Figure 3: Explanted common iliac vein stent shown with a surgical scalpel for scale. The stent was removed intact.

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#FR 2 – Kiosk 19 – Poster 5

Congenital Pulmonary Valve Stenosis in an Adult Patient: Multimodality Imaging

Presenting Author: Jose Gallegos, MD, Fellow, Cleveland Clinic

Introduction

The current case showcases an uncommon presentation of the pulmonic valve (PV) stenosis and associated pulmonary artery (PA) aneurysm in an adult patient. A transesophageal echocardiography (TEE) was supplemented by epicardial echocardiography (EE) on the surgical field. This technique allowed evaluation of the PV morphology and the mechanisms of obstruction. We aim to highlight the potential benefits of epicardial ultrasound especially when evaluating complex anatomical structures of the anterior heart.

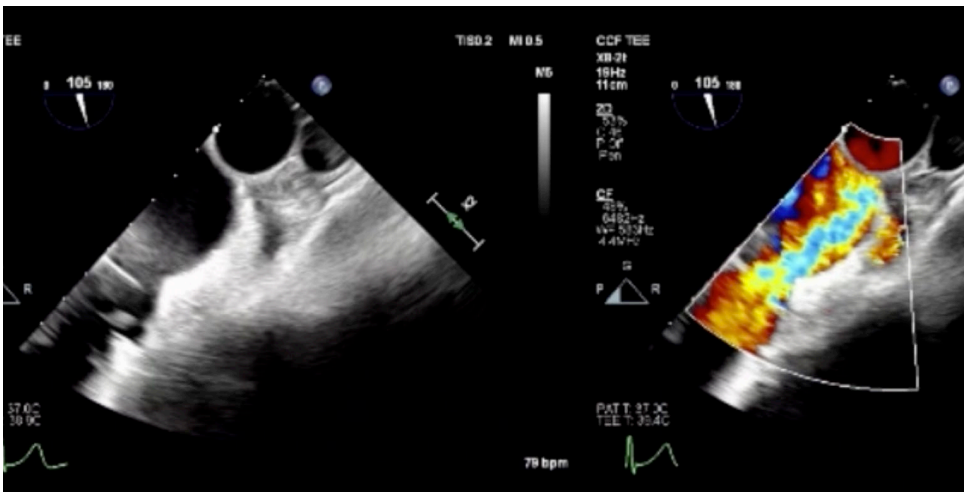
Case Presentation

This is a 63-year-old male presenting with supraventricular tachycardia, migraines, known left pulmonary artery aneurysm. Transthoracic echocardiography (TTE) showed mild PV insufficiency and stenosis (concordant with right heart catheterization), however not providing a clear mechanism. Intraoperative 2D and color flow Doppler TEE imaging suggested a lack of the obstruction at the level of the PV annulus but rather echogenic structure and flow acceleration distal to the PV suggestive of a potential supravalvular stenosis (Image 1). Similarly, pulsed wave/continuous wave Doppler showed no obstruction at the level on the PV annulus but rather indicating potential supravalvular lesion (Image 2). Poor spatial resolution of the 3D TEE was not conducive to discern the mechanism either. The EE, then, revealed a significantly thickened tips of the long PV leaflets producing a cone-shaped PV anatomy responsible for the stenosis (figure 3). Given that the discerned stenosis was directly related to the PV, a pulmonary homograft was used to replace the PV, which was distally anastomosed (T-shaped-anastomosis) to the avalvular aortic homograft used to connect aneurysmatic parts of the PA.

Discussion

Isolated PV stenosis is rare in adults accounting for only 10-12% of adult congenital cases. It is commonly associated with other congenital abnormalities, stenosis, and distal PA aneurysms occurring anywhere between the stenotic lesion and the peripheral branches. PV stenosis most commonly presents with a dome-shaped valvular apparatus with normal leaflet motion and a small central opening at the thickened tips which was observed in the current case. A decreased spatial resolution commonly poses a challenge when using TEE to evaluate the PV as it is an anterior structure. The implementation of EE is not commonly used for intraoperative evaluation. However, our case suggests its superiority when evaluating anterior structures such as the PV and PA. Previous studies have demonstrated better visualization of anatomical lesions using epicardial echocardiography when compared to TEE with lesions related to the pulmonary artery. As in our case employment of EE clarified pathologic findings which in turn influenced surgical planning.

Images



Upper esophageal view of the PA and PV in long-axis. Color Doppler reveals flow acceleration across valvular apparatus.

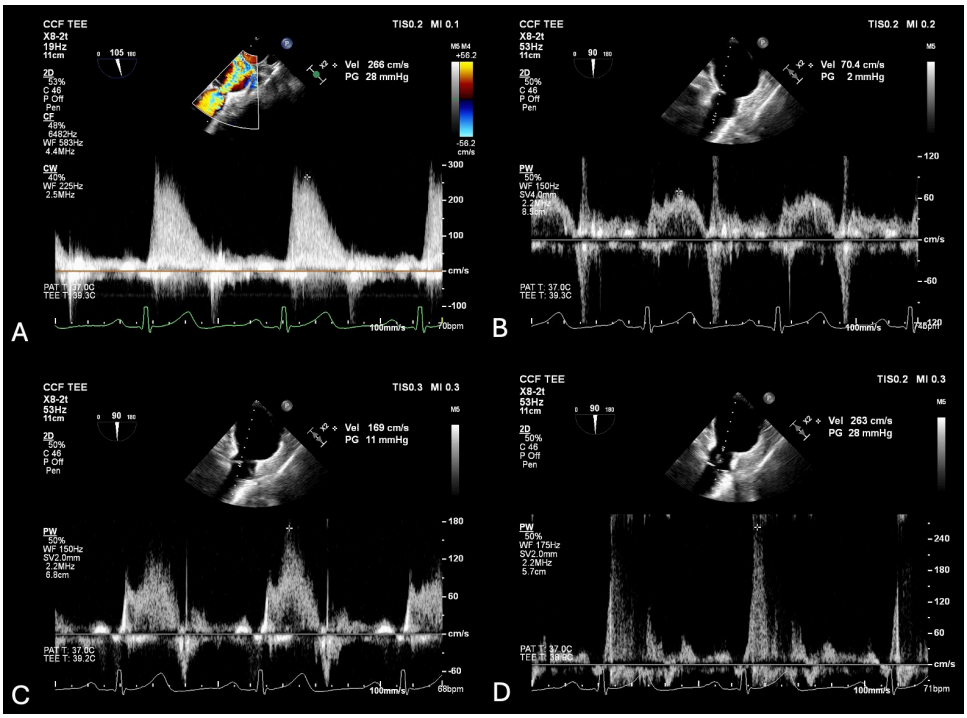
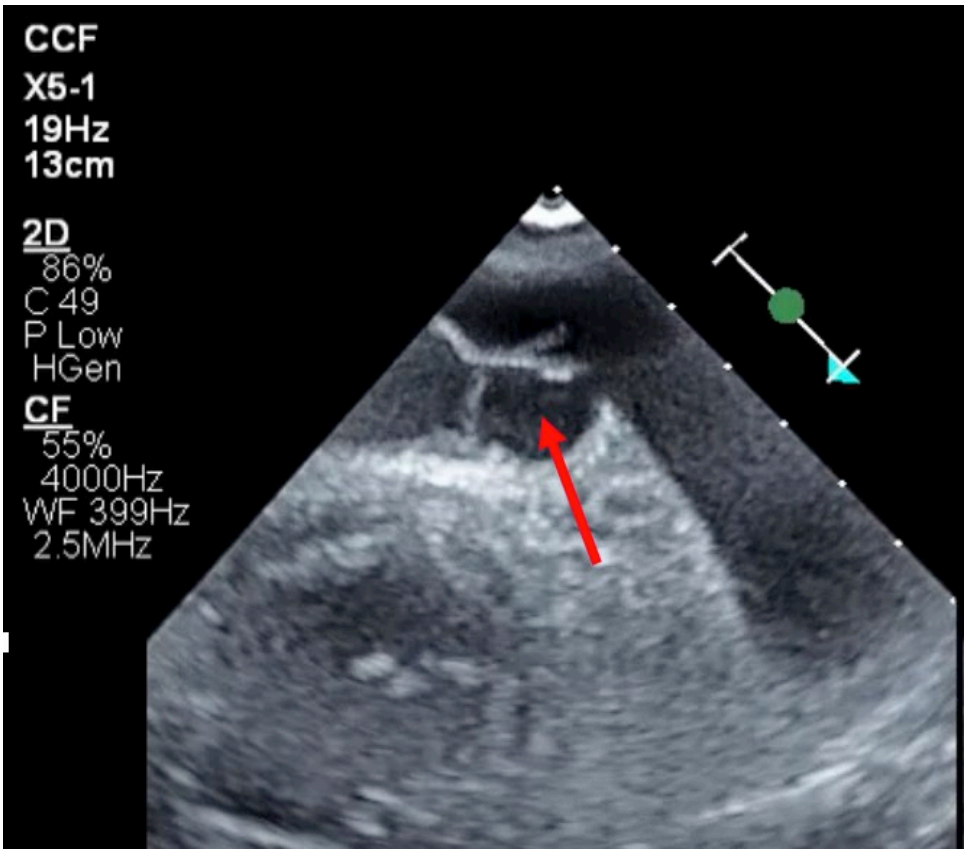


Figure 1. Spectral Doppler measurements across pulmonic valve in the upper esophageal view. (A) Continuous wave Doppler across valve with a peak pressure gradient (PG) of 28 mmHg. (B) Pulsed wave Doppler with sample volume collected proximal to valve annulus [PG 2 mmHg], (C) just distal to valve

annulus but proximal to stenotic region [PG 11 mmHg], and (D) just distal to stenotic region prior to aneurysm take off [PG 28].



Epicardial ultrasound image placed directly over the pulmonic valve. Structure closest to probe (red arrow) depicts the valve with long pliable leaflets forming a cone-shaped structure.

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#FR 2 – Kiosk 19 – Poster 6

Last-Minute Diagnosis of ACxAPA Masked by Progressive Mitral and Tricuspid Valve Disease in a 72-Year-Old Patient: Highlighting the Anesthesiologist's Role in Preoperative Clinics

Presenting Author: Mariusz Ligocki, MD, ACTA Fellow, Cleveland Clinic

Introduction

Congenital coronary anomalies arising from the pulmonary artery are rare and usually detected in infancy due to ischemia or ventricular dysfunction. Adult survival without prior diagnosis is uncommon and relies on extensive collateralization. Anomalous circumflex coronary artery from the pulmonary artery (ACxAPA) is particularly rare and may remain unnoticed when structural heart disease masks its physiologic effects. Chronic low-grade ischemia may therefore be misattributed to valvular pathology alone. This case illustrates how anesthesiologists in modern preoperative clinics, within the Perioperative Surgical Home, can identify subtle imaging discrepancies that meaningfully alter surgical planning and perioperative risk.

Case Presentation

A 72-year-old woman with exertional dyspnea, long-standing atrial fibrillation, and multivalvular disease was scheduled for mitral, tricuspid, and aortic valve surgery with Maze procedure. Serial echocardiograms over several years showed worsening MR and TR, bi-atrial enlargement, and evolving diastolic dysfunction. No regional wall-motion abnormalities were present, and prior coronary evaluation had excluded obstructive CAD, reinforcing the assumption that her symptoms arose from valvular pathology; however, coronary origin had not been assessed. During her routine preoperative anesthesia clinic visit—focused on airway assessment, medication review, and risk stratification—the fellow anesthesiologist reviewed her most recent echocardiogram. Subtle retrograde-appearing Doppler flow in the circumflex territory appeared discordant with the absence of wall-motion abnormalities and suggested chronic subendocardial ischemia not explained by CAD-negative testing (Figure 1a). Concern for an occult anomaly prompted urgent cardiology consultation and coronary CT angiography. Imaging revealed an anomalous circumflex arising from the pulmonary artery (Figure 1b–d). This ACxAPA physiology likely caused chronic coronary steal into the low-pressure pulmonary system, producing long-standing subendocardial ischemia, papillary muscle dysfunction, and progressive valvular regurgitation. Diagnosis only days before surgery required revising the operative plan to include coronary revascularization with the planned valve interventions.

Discussion

This case demonstrates how congenital coronary anomalies may remain undiagnosed into late adulthood, especially when ischemia is subtle and without wall-motion abnormalities that typically prompt further evaluation. Low-grade ischemia from ACxAPA can mimic or accelerate valvular progression, directing attention toward structural disease while the underlying cause remains concealed. Routine imaging may miss anomalous origins unless examined carefully. The anesthesiologist's recognition of imaging discordance during a standard preoperative clinic visit led to timely diagnosis and essential revision of surgical planning. This highlights the expanding diagnostic role of anesthesiologists in the Perioperative

Surgical Home, where integrating clinical findings and imaging review can uncover critical, previously unrecognized pathology. Diagnostic curiosity in preoperative clinics is vital, as last-minute discoveries such as ACxAPA can directly influence operative safety, myocardial protection, and long-term outcomes.

Images

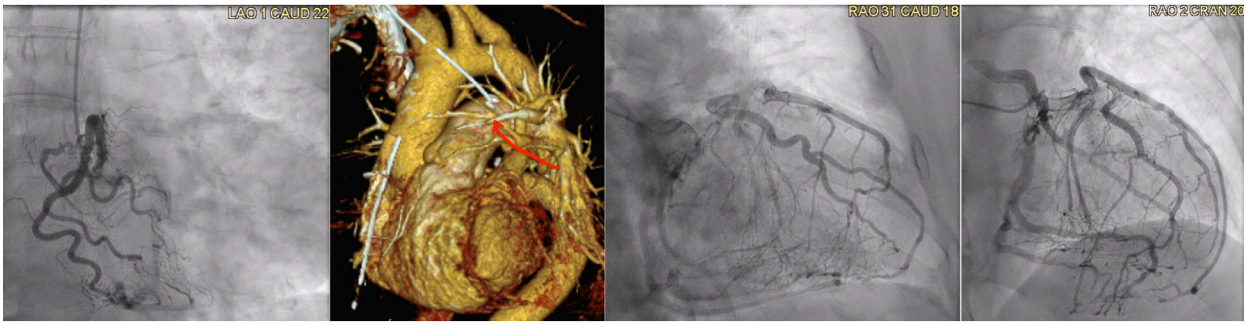


Figure 1. (a) Initial LHC showing LMT, LAD, RCA, with robust collaterals impersonating LCx. (b) CTA identifying potential origin of ACxAPA. (c) RHC confirming true origin of LCx (early phase). (d) RHC identifying collaterals of LCx with LAD and RCA (late phase).

Figure 1. (a) Initial LHC showing LMT, LAD, RCA, with robust collaterals impersonating LCx. (b) CTA identifying potential origin of ACxAPA. (c) RHC confirming true origin of LCx (early phase). (d) RHC identifying collaterals of LCx with LAD and RCA (late phase).

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#FR 2 – Kiosk 19 – Poster 7

When Chest Closure Opens the Door for Respiratory Failure: V-V ECMO in Morbidly Obese Patients

Presenting Author: Daniel Schiller, DO, Cardiothoracic Anesthesiology Fellow, Cleveland Clinic

Introduction

Pulmonary complications after cardiac surgery contribute to morbidity and mortality in the perioperative period. Here, we present a case of a patient with morbid obesity who underwent urgent cardiac surgery. His operative course was complicated by mixed hypoxemic and hypercarbic respiratory failure requiring venovenous ECMO (VV-ECMO) support.

Case Presentation

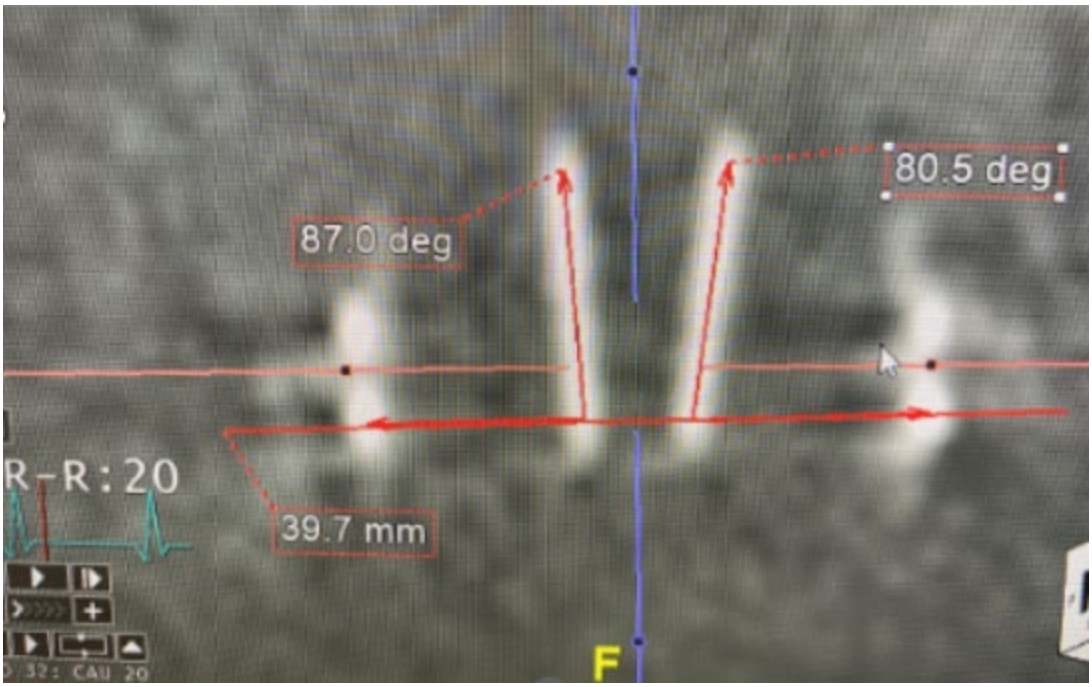
A 24-year-old, 196kg (BMI: 61) male with a history of a unicuspid aortic valve status-post Bentall procedure who presented with new onset chest pain. His workup was concerning for acute thrombosis of his aortic valve prostheses in the setting of anticoagulation nonadherence. Surgical intervention was initially deemed too high risk due to morbid obesity, recent NSTEMI, and redo sternotomy (STS risk score 15.9%). Surgery was initially deferred to thrombolytic therapy which ultimately resulted in persistent aortic stenosis. The patient underwent redo sternotomy and Ross procedure. The patient was successfully weaned from a 4.5-hour cardiopulmonary bypass (CPB) time with mild biventricular dysfunction and trivial aortic and pulmonary insufficiency. However, upon chest closure, several cardiopulmonary parameters declined. Pulmonary artery and peak pressures increased, oxygen saturation decreased, and hypercarbia ensued despite corrective measures. These measures included increasing FiO2 and PEEP, recruitment maneuvers, and Epoprostenol therapy. Despite efforts to improve oxygenation and ventilation, the patient remained hypercarbic and hypoxic until the chest was re-opened. A decision was made to leave the chest open and cannulate for peripheral VV-ECMO support.

Discussion

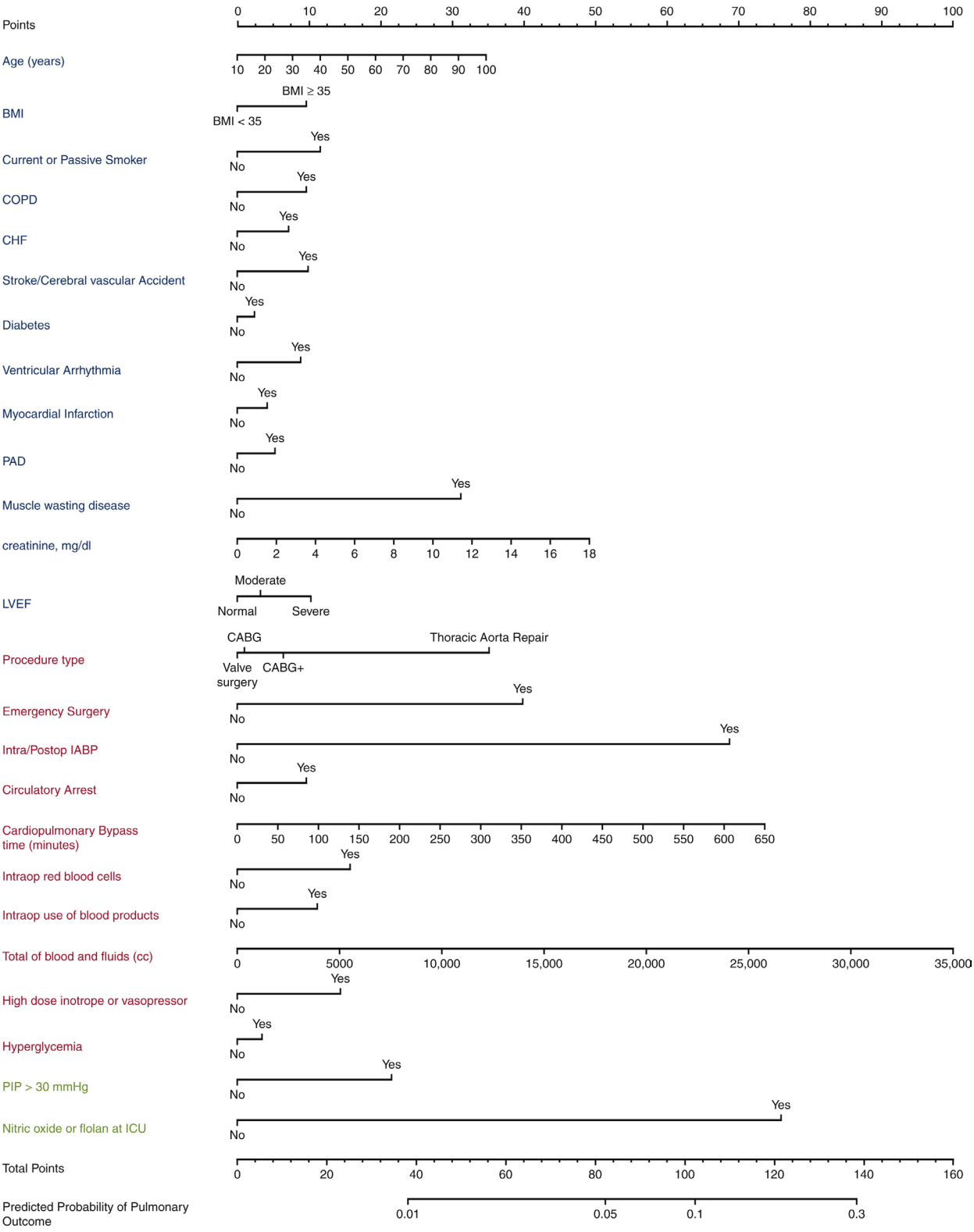
There are many risk factors that contributed to this patient's adverse outcomes. CPB places the lungs in a state of relative ischemia. As a result, the lungs are vulnerable to reperfusion injury leading to a proinflammatory state with subsequent increases in arteriolar resistance and edema, impairing gas exchange. The physiologic effects of sternal closure include increases in airway resistance and dead space with decreased lung compliance that are exacerbated in obese patients. Cardiothoracic anesthesiologists should be acutely aware of the potential for respiratory failure during chest closure in morbidly obese

patients. Intraoperative events such as blood product transfusion, prolonged CPB time, high dose inotropes/vasopressors, the need for pulmonary vasodilators, and peak inspiratory pressures of >30 mmHg may be indicators that a patient may need mechanical circulator support (MCS) after CPB. Perioperative lung protective ventilation strategies should be optimized in morbidly obese patients prior to chest closure. To further optimize ventilation, providers may consider switching from an anesthesia machine to a more efficient ICU mechanical ventilator with a total intravenous anesthetic. If efforts to ventilate the patient fail, anesthesiologists need to alert the cardiothoracic team about the potential need for MCS. ECMO support has many challenges in obese patients including difficult cannulation, potential need for higher ECMO flow rates, and exit strategies. This case highlights the role anesthesiologists should play in early risk stratification and anticipation for MCS after CPB.

Images



Normal opening and closing angles for the On-X mechanical valve in the aortic position are 90 degrees and 40 degrees respectively. Any change in opening or closing angle > 4 degrees is concerning for leaflet restriction. The patient's anterior leaflet appeared to be restricted on the CT with an opening angle of 80 degrees


Points
0 10 20 30 40 50 60 70 80 90 100
Age (years)
10 20 30 40 50 60 70 80 90 100
BMI
BMI ≥ 35
BMI < 35
Current or Passive Smoker
Yes
No
COPD
Yes
No
CHF
Yes
No
Stroke/Cerebral vascular Accident
Yes
No
Diabetes
Yes
No
Ventricular Arrhythmia
Yes
No
Myocardial Infarction
Yes
No
PAD
Yes
No
Muscle wasting disease
Yes
No
creatinine, mg/dl
0 2 4 6 8 10 12 14 16 18
LVEF
Moderate
Normal
Severe
Procedure type
CABG
Thoracic Aorta Repair
Valve surgery
CABG+
Emergency Surgery
Yes
No
Intra/Postop IABP
Yes
No
Circulatory Arrest
Yes
No
Cardiopulmonary Bypass time (minutes)
0 50 100 150 200 250 300 350 400 450 500 550 600 650
Intraop red blood cells
Yes
No
Intraop use of blood products
Yes
No
Total of blood and fluids (cc)
0 5000 10,000 15,000 20,000 25,000 30,000 35,000
High dose inotrope or vasopressor
Yes
No
Hyperglycemia
Yes
No
PIP > 30 mmHg
Yes
No
Nitric oxide or flolan at ICU
Yes
No
Total Points
0 20 40 60 80 100 120 140 160
Predicted Probability of Pulmonary Outcome
0.01 0.05 0.1 0.3

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#FR 2 – Kiosk 20 – Poster 1

Intraoperative Management of Elephant Trunk Kinking during Type A Dissection Repair

Presenting Author: Kush Gupta, MD, Resident Physician, Anesthesiology, Brigham and Women's Hospital, Mass General Brigham

Introduction

The conventional elephant trunk (cET) technique can be utilized for aortic dissections that span from the ascending aorta to the arch and into the descending aorta. While this provides a proximal graft-to-aorta anastomosis and thereby simplifies second-stage procedures, the floppy distal end of the cET is susceptible to kinking. We present a case of cET repair of an acute type A aortic dissection where transesophageal echocardiography (TEE) was used to identify kinking of the cET followed by real-time TEE guidance for balloon dilation and thoracic endovascular aortic repair (TEVAR) deployment across the cET.

Case Presentation

A 44-year-old female with a history of Marfan syndrome and hypertension presented with acute type A aortic dissection spanning from the aortic root through the arch down to the iliac arteries. After an unremarkable induction, the surgical team performed a mechanical Bentall with a total arch replacement and left an eight-centimeter Dacron cET floating within the distal arch. Shortly after weaning from cardiopulmonary bypass (CPB), significant bleeding was noted from the distal anastomosis along with an approximately 20mmHg drop in femoral arterial pressures relative to the bilateral radial line pressures. TEE of the proximal descending aorta identified kinking across the cET outflow tract (Figure 1) which was corrected with balloon dilation (Figure 2) followed by antegrade TEVAR across the cET in zone 3 to reinforce the graft-to-graft anastomosis (Figure 3). This resulted in equalization of femoral and radial arterial pressures and improvement of hemostasis at the anastomosis. The subsequent post-CPB period was notable for marked hypotension in the setting of ST depressions and regional wall motion abnormalities leading to cardiac arrest for which the patient was returned to CPB to perform a CABG x3. The patient was subsequently weaned off CPB without difficulty and was stable upon transfer to the ICU. She had no post-operative sequelae of bowel or lower extremity malperfusion and was discharged home on post-operative day 23.

Discussion

While graft stenosis or kinking is primarily described in the context of abdominal aortic aneurysms (AAA) repairs, it is a relevant complication in cET repair of acute aortic dissections. Risk factors for kinking include small aortic neck diameter, sharp aortic neck angulation, and female gender amongst other things. (1) Intractable bleeding at the site of distal anastomosis along with significant differences between upper and lower extremity arterial pressures are two key markers of cET graft kinking. Graft narrowing can also induce turbulent flow or elevated velocities within the aorta. (2). Intraoperative TEE can not only assess such phenomena and confirm cET graft kinking (as seen above) but also guide rescue efforts such as balloon angioplasty or TEVAR. In cases where kink recurs despite balloon angioplasty, it may be prudent to convert to a stented frozen elephant trunk (FET). In addition to resolving cET kinking, FET provides the key benefit of

improving the ‘sealing capacity’ (true and false lumen apposition), thereby depressurizing the false lumen to facilitate its obliteration and thromboexclusion. (3).

Images

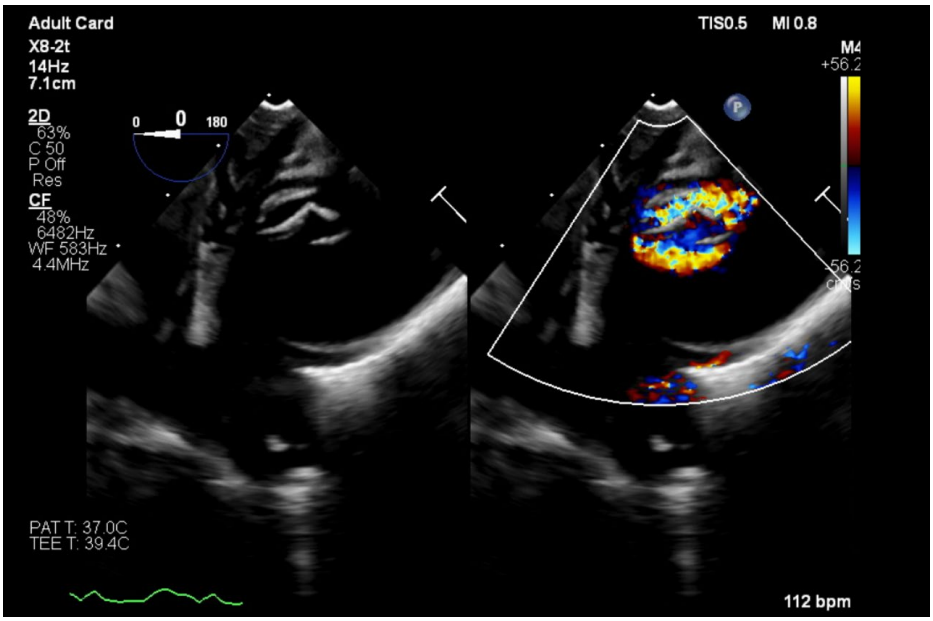


Figure 1: Kinking of cET (left) with color flow doppler (right) demonstrating turbulent flow in the proximal descending aorta

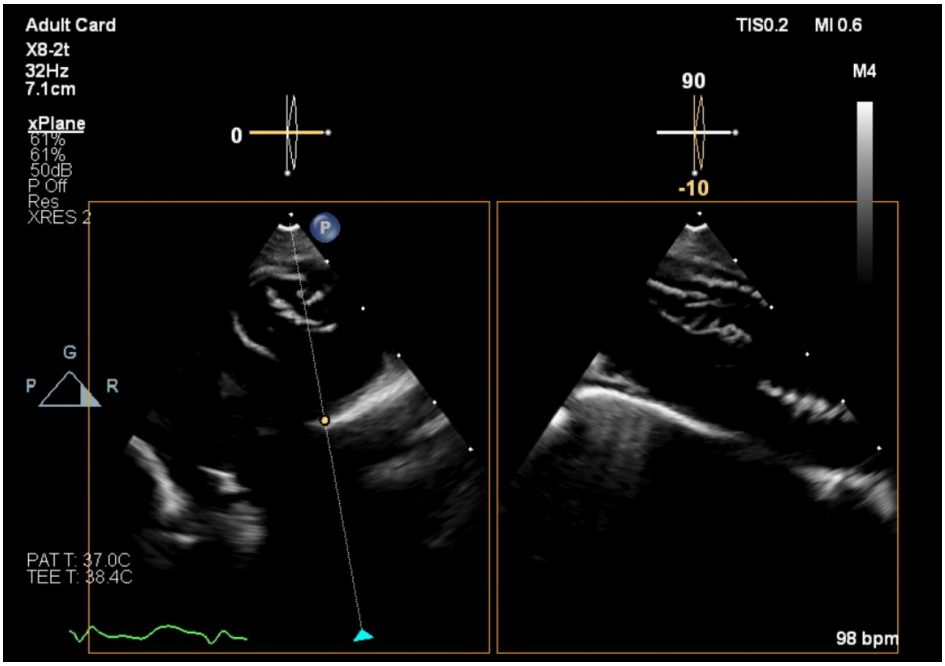


Figure 2: Balloon dilation of the kinked cET under real-time TEE guidance in short axis (left) and long axis (right)

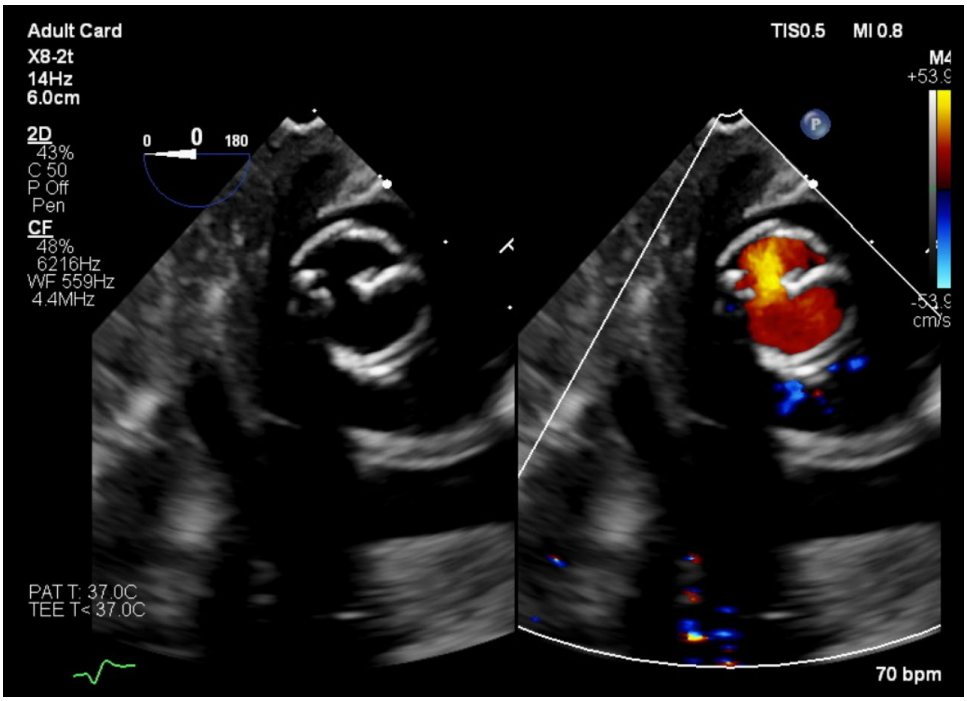


Figure 3: Resolution of graft kinking after balloon angioplasty/TEVAR (left) with doppler (right) demonstrating laminar flow

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#FR 2 – Kiosk 20 – Poster 2

Impella CP Catheter-Associated Thrombus Mimicking Aortic Dissection

Presenting Author: Monica Ha, MD, Resident Physician, UT Health

Introduction

The Impella Cardiac Power (CP) is a catheter-based temporary left ventricular assist device that provides life-saving mechanical circulatory support but carries meaningful complication risks. Differentiating thrombus from true aortic dissection on transesophageal echocardiography (TEE) poses significant diagnostic and management challenges. We present a case in which an Impella CP to Impella 5.5 upgrade was aborted intraoperatively after TEE revealed a flap concerning for aortic dissection. This imaging finding was later determined to represent thrombus adherent to the Impella CP catheter.

Case Presentation

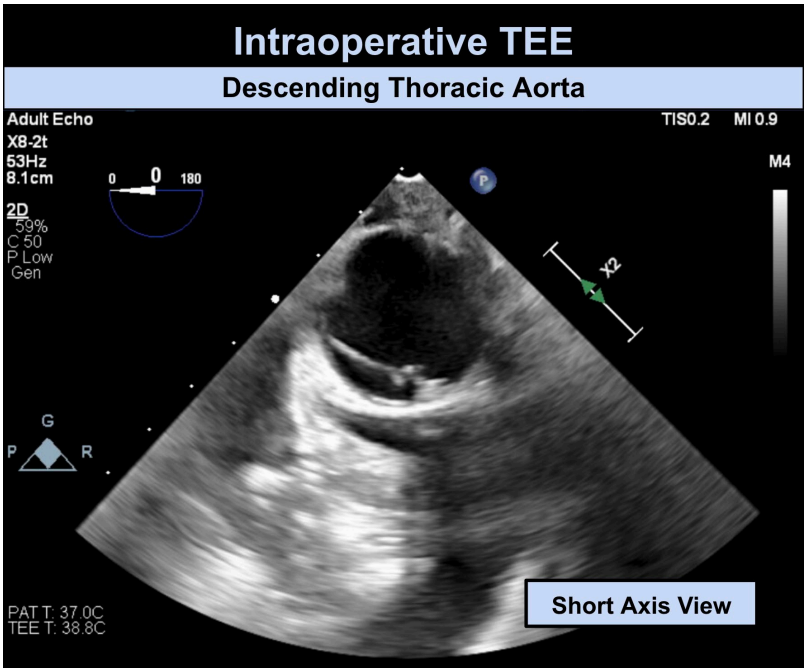
An 81-year-old male with nonischemic cardiomyopathy underwent ventricular tachycardia ablation which was complicated by cardiac vein injury and hemopericardium, requiring initiation of VA-ECMO and Impella CP placement for hemodynamic support. Thirty hours later, during a planned Impella 5.5 upgrade for left ventricular support, intraoperative TEE demonstrated a mobile linear flap in the proximal descending thoracic aorta. Concern for iatrogenic dissection from prior device placement prompted abortion of the procedure, and the patient's Impella CP support was maintained. Emergent CT angiography (CTA) subsequently revealed no aortic dissection. The intraoperative finding was then suspected to represent thrombus adherent to the Impella CP catheter rather than a true aortic intimal tear. The patient successfully underwent Impella 5.5 placement the following day with careful TEE guidance under general anesthesia and continued VA-ECMO support.

Discussion

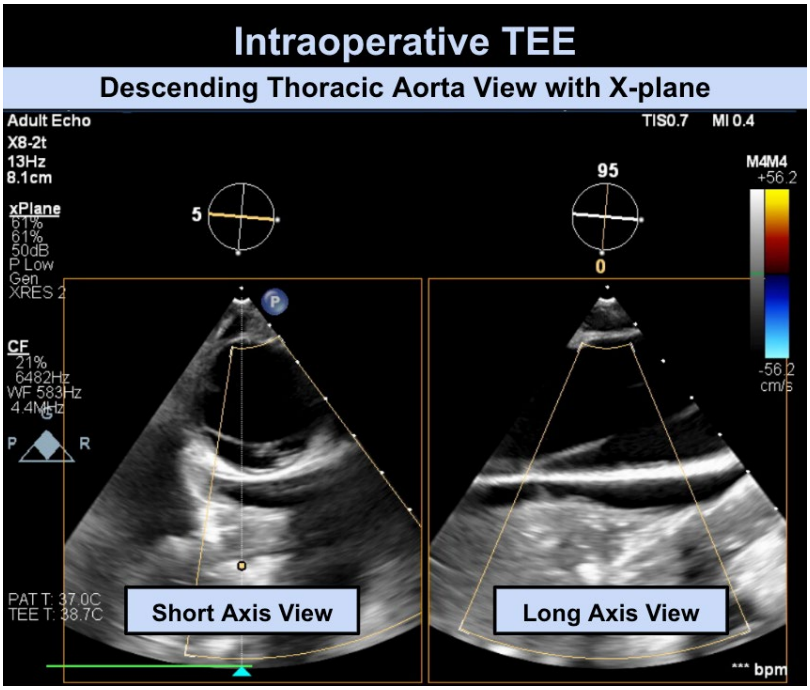
This case highlights a rare but important complication: an Impella CP-associated thrombus mimicking an aortic dissection. FDA MAUDE surveillance data report that approximately 15-19% of Impella adverse events involve vascular complications.[1] In a recent Impella 5.5 series of device-related mortality, aortic or graft complications (including dissection) occurred in 12% of cases, while device thrombosis or embolic stroke occurred in 14%.[2] Early thrombus formation may occur despite systemic anticoagulation; one MAUDE case described left ventricular thrombus formation within 24 hours of Impella CP insertion.[3] Both thrombus and

dissection appear as linear echodensities on TEE, though thrombus tends to move synchronously with the catheter and lacks the true luminal separation characteristic of an aortic dissection. In this case, vigilant intraoperative TEE evaluation allowed immediate recognition of an abnormal aortic finding, prompting procedural abortion and likely preventing a complication had a true dissection been present. The case underscores TEE's essential role in distinguishing a thrombus from an aortic dissection in real time, guiding surgical decision-making, and informing when confirmatory imaging such as CTA is warranted. For cardiac anesthesiologists managing patients on Impella support, maintaining a high index of suspicion for such complications and using multimodal imaging to verify findings is critical to patient safety.

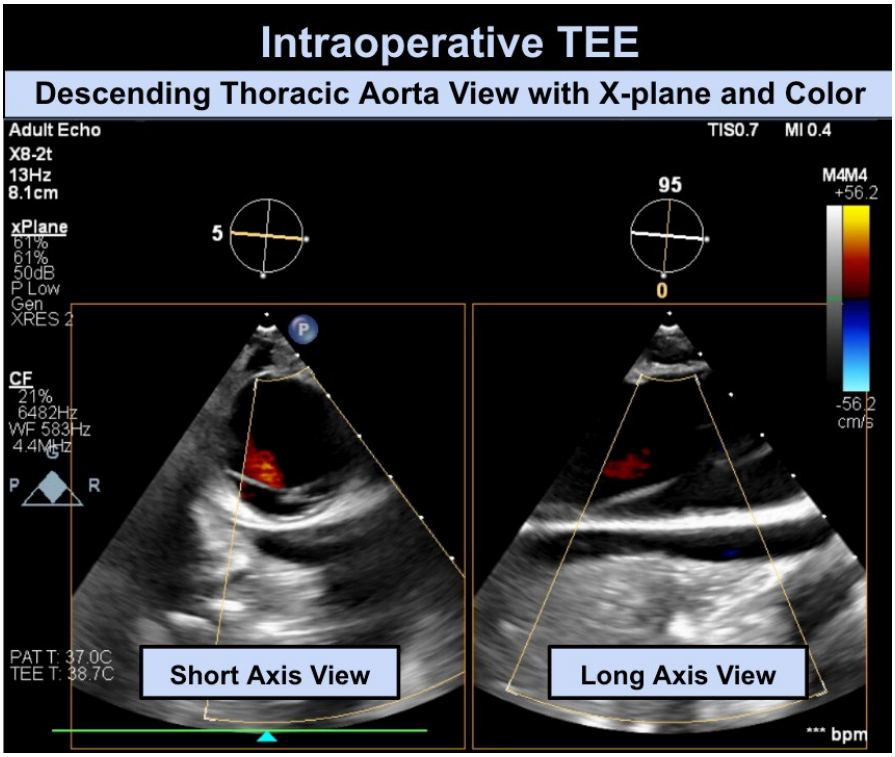
Images



Descending aorta in short axis view



Descending aorta in short axis view and long axis view



Descending aorta in short axis view and long axis view with color

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#FR 2 – Kiosk 20 – Poster 3

Acute Right Ventricular Failure Immediately Following CABG Requiring Emergent Cardiac Catheterization with PCI

Presenting Author: Dave A. Thiagaram, MD, Cardiothoracic Anesthesiology Fellow, Rutgers Robert Wood Johnson Medical School

Introduction

Coronary artery bypass grafting (CABG) is a well-established and safe treatment option for multivessel coronary artery disease (CAD). There is some evidence that some degree of right ventricular impairment does occur following CABG (Yadav 2010). However, right ventricular failure is a rare complication following CABG occurring just below 3% of the time in one study population (Levy 2021). There is some evidence that percutaneous coronary intervention (PCI) and stent placement in the right coronary artery (RCA) have been shown to improve right ventricular (RV) function (Nikdoust 2014).

Case Presentation

We present a case of a 79 year old female with a history of atrial fibrillation, CAD x2 Stents (1997/2004), HFrEF (LVEF 40%), CKD Stage 2, HTN, HLD, T2DM, Obesity who presented with NSTEMI 1 week prior and underwent a CABG surgery. Intraoperatively, the procedure was limited by porcelain aorta. Only the left internal mammary artery (LIMA) to left anterior descending (LAD) anastomosis was attempted off pump but the patient became hypotensive requiring increasing ionotropic support. Cardiopulmonary bypass was then initiated and the procedure was completed with a beating heart. Post-bypass, transesophageal echocardiography showed signs of severe RV dysfunction with severe tricuspid regurgitation. Despite milrinone infusion and inhaled nitric oxide, there was no change in RV function. The patient was then transported to the cardiac catheterization lab for emergent PCI with RCA stent placement after which RV function slightly improved. Additional stents were placed in the LAD as well as the left circumflex and an Impella CP was also placed.

Discussion

This was an interesting case due to the presence and management of RV failure following CABG, which required cardiac catheterization and stent placement due to the presence of a porcelain aorta. RV failure is a dreaded complication after CABG that can contribute to significant morbidity and mortality. There are some suggested independent risk factors for RV dysfunction after CABG such as preoperative atrial fibrillation, LVEF < 50%, systolic pulmonary artery pressure > 55mmHg, mitral valve surgery, and tricuspid valve surgery (Levy 2021). Protamine reactions can be a cause of RV dysfunction during CABG, although unlikely in this case because the symptoms were noted prior to administration. RCA occlusion is correlated with RV dysfunction, though pulmonary hypertension is a stronger determinant. We did not insert a pulmonary artery catheter, but it would have been useful to better assess the pulmonary artery pressures. It is well known that the most common cause of RV failure is typically left ventricular (LV) failure resulting in high pulmonary artery pressures. The LV function appeared to have been preserved throughout this case,

but this could have been masked by ionotropic support. The finding that the RV free wall appeared to have minor improvement in motion after the RCA stent was placed could suggest that right coronary ischemia may have contributed to RV dysfunction, but it is unclear which intervention contributed to the improved RV function if any. More studies are necessary to better evaluate the pathophysiology of RV failure and management after CABG.

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#FR 2 – Kiosk 20 – Poster 4

A Masquerading Myxoma

Presenting Author: Vivek Ramakrishnan, MD, Jackson Memorial Hospital

Introduction

Myxomas predominantly arise in the left atrium (LA) from the interatrial septum, but atypical locations complicate diagnosis, particularly in the setting of atrial fibrillation. Although intraoperative transesophageal echocardiography (TEE) is not always indicated, it significantly improves surgical management due to its superior resolution compared to transthoracic echocardiography (TTE). Real-time 3D TEE further enhances morphological detail, aiding surgical planning and potentially reducing postoperative risks.

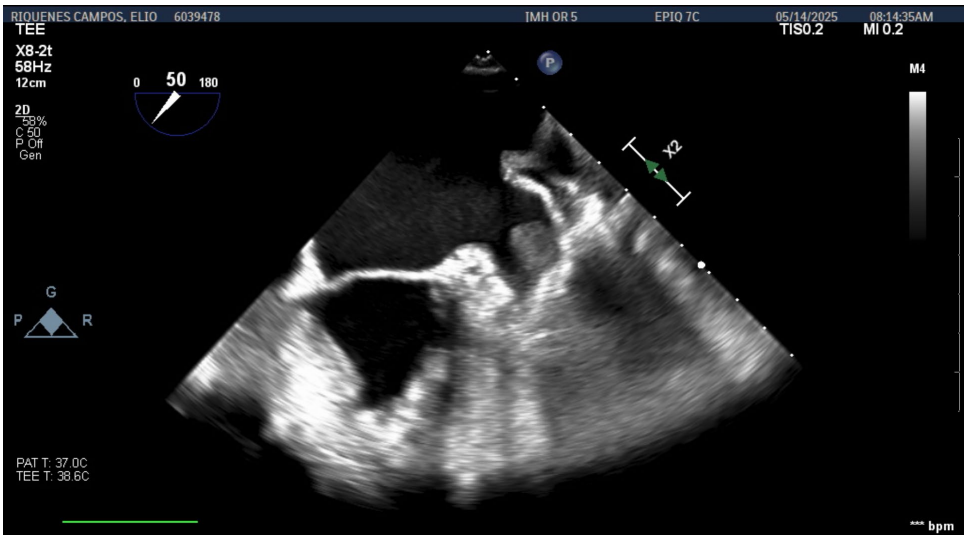
Case Presentation

A 71-year-old male with a history of coronary artery disease, hypertension, type II diabetes, and prostate cancer presented with chest pain, new-onset atrial fibrillation, and elevated troponin and pro-BNP levels. A TTE revealed regional wall motion abnormalities and a mobile mass in the LA connected to the interatrial septum. A cardiac catheterization showed single-vessel disease, prompting a multidisciplinary decision for CABG, left atrial mass resection, and left atrial appendage (LAA) exclusion. Following induction, TEE revealed a well-defined mass (approximately 2.2 × 0.9 cm) in the superior-anterior IAS, initially suspected as either an old thrombus or myxoma. The comprehensive TEE also discovered a second mass in the LAA. Surgical intervention involved median sternotomy, cardiopulmonary bypass (CPB), and a transseptal approach. The IAS mass was identified as a narrow-stalked pedunculated myxoma, while the LAA mass adhered to the endocardium and required excision. Pathology confirmed the IAS mass as an infarcted and hemorrhagic myxoma with inflammation, whereas the LAA mass exhibited myxomatous features suggestive of a true myxoma rather than thrombus.

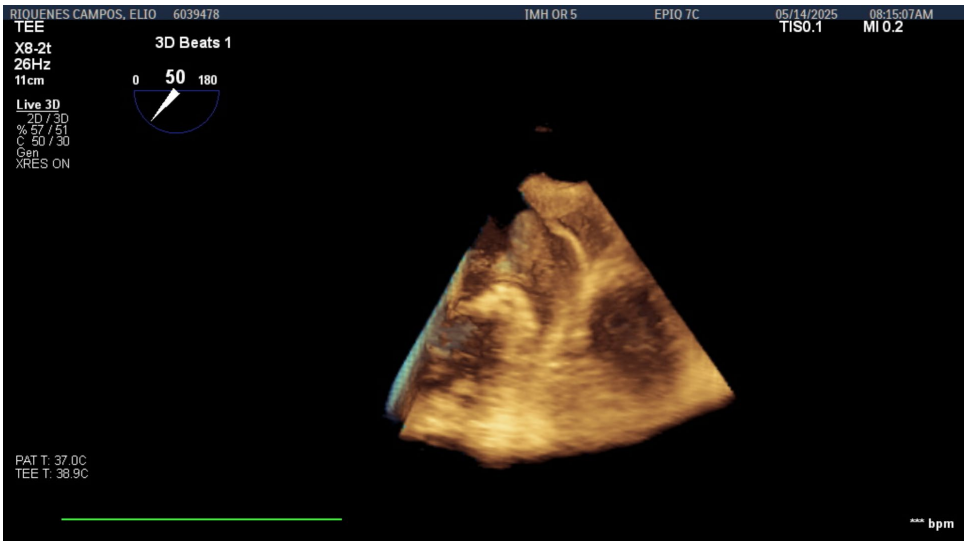
Discussion

This case underscores the critical role of intraoperative TEE in the management of intracardiac masses, particularly atypical myxomas in challenging locations such as the LAA. TEE was vital in revealing an additional mass that necessitated a surgical strategy change from simple ligation to excision, illustrating the value of thorough intraoperative imaging in risk assessment. Collaboration between anesthesiologists and surgical teams using TEE enhances patient outcomes through accurate surgical planning and risk mitigation. Mastery of comprehensive TEE is crucial for cardiothoracic anesthesiologists, serving as a potentially life-saving tool in clinical practice.

Images



LAA 50 Degrees



LAA 3D 50 degrees



LAA mass measurements

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#FR 2 – Kiosk 20 – Poster 5

Refractory Cardiogenic Shock in Recurrent Subaortic Stenosis: The Impact of an Acute Postoperative Ventricular Septal Defect

Presenting Author: Jamie Palmer, MD, Resident, University of Colorado - Anschutz Medical Campus

Introduction

Subvalvular aortic stenosis (SAS) is a fixed LVOT obstruction caused by narrowing below the aortic valve, often from a discrete membrane or congenital structural variants[1]. It is progressive and may recur after repair[2]. Significant LVOT obstruction presents complex hemodynamic challenges, particularly when compounded by pulmonary hypertension, multivalvular disease, or ventricular dysfunction. This case highlights the interplay of recurrent SAS, severe pulmonary hypertension, and postoperative VSD leading to biventricular failure.

Case Presentation

A 54-year-old Ethiopian-born woman with congenital SAS, status post resection in 2011, presented with dyspnea. She was found to have atrial flutter with RVR requiring cardioversion and ablation. Catheterization showed a mean LVOT gradient of 102 mmHg and severe combined pulmonary hypertension (PCWP 26 mmHg, mean PA 60 mmHg, PVR 14 WU). Echocardiography showed concentric LVH, severe LVOT obstruction (peak 133 mmHg at rest), moderate-severe MR with calcification, moderate AI, and moderate TR. She underwent redo sternotomy for AVR, MVR, tricuspid repair, and septal myectomy. Baseline pulmonary pressures were 90s/60s (≈80% systemic). Induction was tolerated, but she remained labile. A 25-mm Mitris mitral valve, 23-mm Inspiris aortic valve, and 30-mm tricuspid ring were placed and septal myectomy was performed. After bypass, a peak LVOT gradient of 83mmHg persisted; further myectomy was deferred. The RV was dilated with severely reduced function, requiring inotropes and pulmonary vasodilators to promote right-to-left flow and support LV preload. This improved the gradient slightly to 30mmHg due to improved LV filling. She arrived to the CTICU on multiple vasopressors, inotropes and Veletri. PA pressures fell to ~66% systemic, but rebounded due to persistent obstruction. Postoperative TEE showed reduced biventricular function and severe LVOT obstruction (Vmax >4 m/s), with PA pressures ~70 mmHg. On POD 5, she developed refractory cardiogenic shock with rising lactate. TEE revealed a new membranous VSD (Fig. 2), and VBGs demonstrated a step-up in O_2 saturation from the RA (67%) to the PA (83%) confirming presence of a VSD. She had an emergent reoperation for VSD repair and VA ECMO cannulation. She was decannulated with chest closure on POD 10.

Discussion

Hemodynamic goals in LVOT obstruction include maintaining preload and afterload, avoiding tachycardia, and preventing excessive contractility[3]. In this case, the postoperative VSD reduced LV afterload and end-systolic volumes, worsening the dynamic obstruction. Elevated LV systolic pressures then augmented flow across the VSD. Management was complicated by conflicting needs of the ventricles. Interventions that may reduce LVOT obstruction (afterload augmentation, preload optimization, HR reduction), can worsen RV

function, while RV-supportive measures (inotropes, higher HR) worsen obstruction. Although high PVR can limit left-to-right shunting with a VSD, her extreme gradients (~100mmHg) likely reflected LV pressures near 200mmHg, which were sufficient to drive shunting despite elevated PVR. The interaction of severe dynamic LVOT obstruction, VSD physiology, and competing biventricular needs led to rapid, refractory hemodynamic decline.

Images

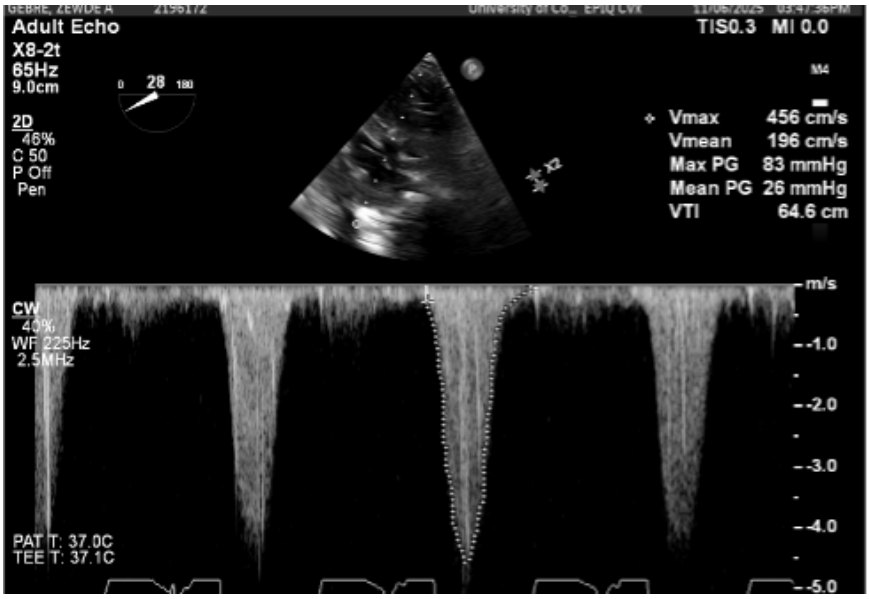


Figure 1. Post intervention demonstrating “late peaking dagger” shaped doppler flow demonstrating ongoing elevated gradients with dynamic obstruction.



Figure 2. 3D modeling representing ventricular septal defect.

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#FR 2 – Kiosk 20 – Poster 6

Right Ventricular Repair and Ventricular Septal Defect Closure for Penetrating Cardiac Injury After Prior Aortic Surgery

Presenting Author: Benjamin M. Todd, MD, Cardiac Anesthesia Fellow, Johns Hopkins Hospital

Introduction

Penetrating cardiac trauma with a traumatic ventricular septal defect (VSD) is rare and presents unique anesthetic challenges, particularly in patients with altered anatomy from prior aortic surgery. We present a case in which mediastinal adhesions likely prevented exsanguination, VSD physiology led to loss of pulsatility on cardiopulmonary bypass, and right ventricular (RV) repair was performed without cardiac arrest.

Case Presentation

A 51-year-old male with prior valve-sparing aortic root replacement and thoracic endovascular aortic repair (TEVAR) with carotid–subclavian bypass sustained a stab wound to the chest. He likely survived to presentation due to dense mediastinal adhesions from prior sternotomy that limited pericardial hemorrhage. Intraoperative transesophageal echocardiography (TEE) revealed a 2 × 1 cm RV free wall laceration and a mid-muscular VSD with left-to-right shunt (Qp/Qs 2.2), along with moderate-to-severe aortic regurgitation. Peripheral femoral artery cannulation established cardiopulmonary bypass (CPB) with confirmed access to the true lumen. Despite baseline aortic regurgitation, there was no arterial pulsatility on CPB because the VSD allowed regurgitant flow to pass directly into the RV and be aspirated into the venous cannula, effectively bypassing systemic circulation. Given the absence of ventricular distension and adequate surgical visibility, the heart was not arrested. Primary RV free wall repair was successfully performed without cross-clamping. The VSD was not surgically addressed due to favorable anatomy for later percutaneous closure. The patient received 3 units of packed red blood cells, 3 units of fresh frozen plasma, 3 units of platelets, 4 units of cryoprecipitate, and 1000 units of prothrombin complex concentrate for coagulopathy secondary to bleeding. On postoperative day 3, he underwent successful transcatheter VSD closure with an 18 mm Amplatzer occluder. TEE confirmed stable device position with minimal residual shunt. He was discharged home on postoperative day 11.

Discussion

This case highlights how traumatic VSD can unexpectedly influence CPB physiology by eliminating pulsatility in the presence of aortic regurgitation. It also demonstrates that, under select conditions, RV repair without arrest with delayed VSD closure can be feasible in traumatic settings. Critical factors for successful management included TEE guidance, adhesion-mediated tamponade, and a tailored cannulation strategy.

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#FR 2 – Kiosk 21 – Poster 1

Triad of Postural Orthostatic Tachycardia Syndrome, Ehlers-Danlos Syndrome, and Mast Cell Activation Syndrome Undergoing Rib Resection for Thoracic Outlet Syndrome

Presenting Author: Reuben D'Souza, MD, Anesthesia Resident, Rutgers Robert Wood Johnson Medical School

Introduction

Postural Orthostatic Tachycardia Syndrome (POTS) is a chronic, multifactorial condition characterized by orthostatic intolerance. It involves recurrent increases in heart rate upon standing without hypotension, accompanied by additional symptoms (headache, nausea, abdominal pain, lightheadedness, palpitations, etc.) that intensify in the upright position often due to autonomic dysregulation and impaired peripheral neurovascular function. Ehlers-Danlos syndrome (EDS) is a group of inherited connective tissue disorders marked by joint hypermobility, skin hyperextensibility, and tissue fragility (prevalence 1 in 5,000) with the hypermobility type (hEDS) being the most common subtype. Mast Cell Activation Syndrome (MCAS) is a recently recognized disorder caused by excessive mast cell degranulation and encompasses a broad range of conditions. Patients are categorized as primary/clonal, secondary, or idiopathic MCAS with serum tryptase considered the preferred diagnostic marker. Since no definitive treatment exists, management requires a multidisciplinary approach focused on trigger avoidance and preventing symptom recurrence (Table 1).

Case Presentation

A 45-y/o female with a history of asthma, MCAS, hypothyroidism, iron deficiency anemia, hEDS, and POTS underwent a robotic-assisted resection of the left first rib for Thoracic Outlet Syndrome (TOS). The patient experienced symptoms of neurogenic TOS for over a year and underwent MRA, which further suggested venous occlusion during abduction of the arms bilaterally. Medication and fluids were administered preoperatively to optimize for POTS and the avoidance of intraoperative triggers for MCAS (See Table 2). However, shortly after intubation, but before surgical intervention, the patient became hypotensive, requiring vasopressor infusions throughout the case with additional support necessary after initiation of single lung ventilation, repositioning, and insufflation. The patient did not develop tachycardia intraoperatively and was weaned off pressors prior to extubation (see Figures 1 and 2). The postoperative course was uneventful. The patient was discharged on post-operative day three and reported improvement of symptoms of orthostasis, range of motion, and pain on the operative side on one-month follow-up. Plans for right-sided intervention are underway.

Discussion

This case highlights the complexity of anesthetic management in patients with overlapping autonomic, connective tissue, and mast cell disorders. There is an absence of anesthesia literature on POTS; however, the need for prolonged hemodynamic support intraoperatively has been previously described.[2] A study has shown a higher prevalence of POTS who meet the criteria for EDS among participants. Although the exact

relationship between POTS and EDS is not clear, sympathetic cardiovascular responses are similar in each. Furthermore, connective tissue derangements in EDS could contribute to vascular laxity and orthostatic venous pooling. Newer evidence may suggest a link between EDS, POTS, and MCAS as a cluster of disease, but more research is needed to elucidate the relationship.[1]

Images

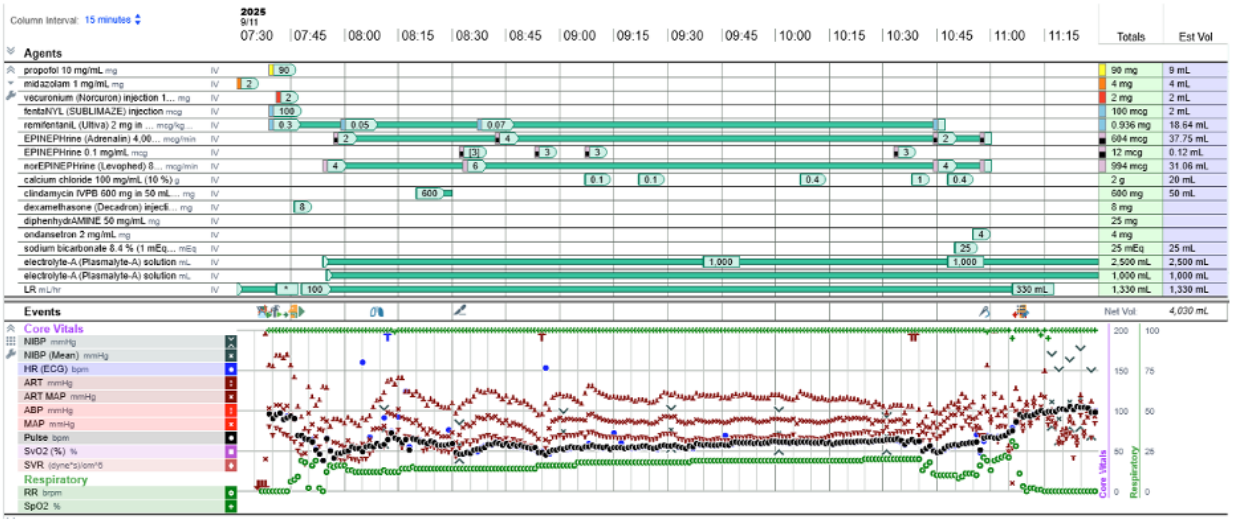


Figure 1. Intraoperative course. Boluses and infusions of epinephrine and norepinephrine infusions initiated shortly post intubation with additional support required after single-lung ventilation, patient repositioning, and insufflation.

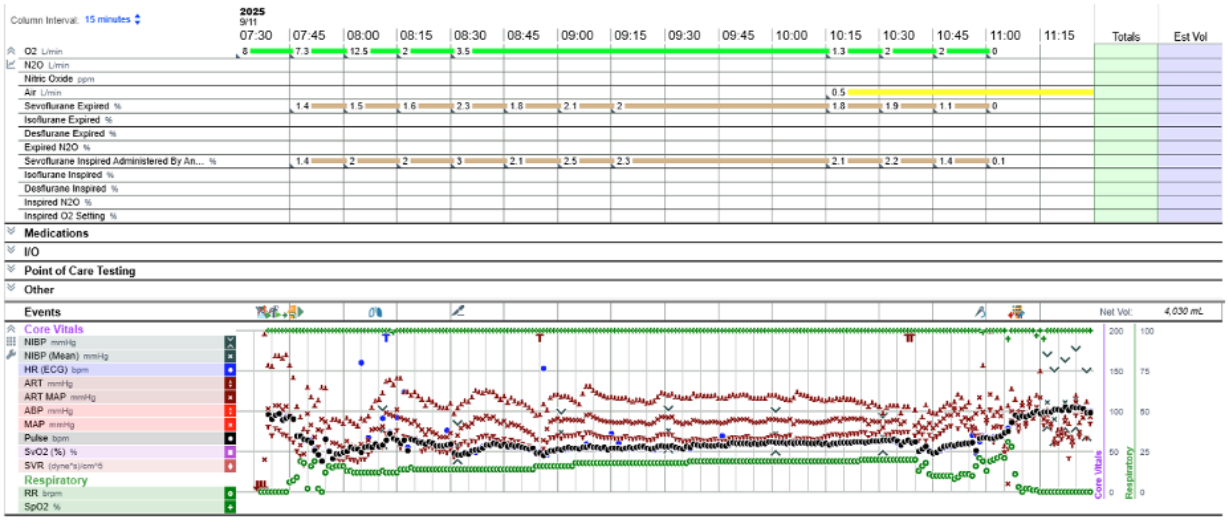


Figure 2. Intraoperative Volatile Anesthetic Administration.

Table 1.[3] Perioperative drugs and mast cell disease.

Class	Drug	Usage in mast cell disorders
Hypnotic/sedative agents	Propofol, dexmedetomidine, etomidate, ketamine	Acceptable
	Methohexital, thiopental	Thiopental causes histamine release
Inhalational anesthetics	Halogenated (isoflurane, sevoflurane, desflurane), nitrous oxide	Acceptable
Benzodiazepines	Midazolam, diazepam	Acceptable
Opioids	Morphine, meperidine, codeine	Causes histamine release
	Hydromorphone, fentanyl, sufentanil, alfentanil, remifentanil, buprenorphine	Acceptable
Nonopioid analgesics	Acetaminophen	Acceptable
	NSAIDs (ketorolac, nefopam)	Causes overproduction of leukotrienes (a mast cell mediator)
Neuromuscular blocking agents	Depolarizing NMBA (succinylcholine)	Acceptable
	Nondepolarizing aminosteroids[c] (vecuronium pancuronium)	Acceptable
	Nondepolarizing benzylisoquinolines (atracurium, mivacurium, cisatracurium)	Atracurium and mivacurium cause histamine release
Reversal of neuromuscular blockade	Neostigmine, Sugammadex	Acceptable
Local anesthetics	Amides and esters	Acceptable
Antiseptics	Alcohol, chlorhexidine, povidone-iodine	Acceptable
Intravenous fluids	Crystalloids, colloids, albumin, gelatin, hydroxyethyl starch	Acceptable
Common labor and delivery drugs	Oxytocin, prostaglandins, methylergonovine, tocolytic agent (terbutaline)	Acceptable, though role of prostaglandins in causing or worsening reactions is unclear
Antibiotics	Penicillins, cephalosporins, sulfonamides, vancomycin, polymyxin B, clindamycin, fluoroquinolones	Vancomycin and polymyxin B can cause histamine release
Miscellaneous	Adenosine, atropine, glycopyrrolate, ondansetron, beta-blockers, ACEI, protamine, aprotinin (fibrin glue), blood transfusion, dyes, contrast media, and latex	Acceptable; adenosine and protamine can cause histamine release; beta-blockers can attenuate the effect of epinephrine in anaphylaxis; ACEI can augment an anaphylactic reaction

(Adapted from Kumaraswami et al., 2018)

Table 2. Perioperative Pharmacologic Management.

POTS (intravascular expansion) MCAS (trigger avoidance and inflammatory mediators)	Fluids: 1L crystalloid fluid bolus and 3 L crystalloid fluid over course of surgery
	Preoperatively: Midazolam 2mg in early PACU and immediately before transport to OR. Benadryl 25mg IV, continued home medication (hydroxyzine, montelukast, cetirizine) Induction: 100 mcg fentanyl, 90mg propofol, 2 mg Vecuronium, Maintenance: remifentanil infusion (0.05 mcg/kg/Hr for most of the case). Sevoflurane. Antiemetics: 8mg dexamethasone, 4 mg Ondansetron Antibiotics: clindamycin Repletions: Calcium and Bicarbonate Pressors: norepinephrine (infusion up to 6 mcg/min) Epinephrine (infusion up to 4 mcg/min, max bolus dose 3 mcg

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#FR 2 – Kiosk 21 – Poster 2

Left Atrial Thrombus or Physiologic Variant?

Presenting Author: Rachna Dhanjal, MD, Lahey Hospital & Medical Center

Introduction

The left atrial appendage (LAA) is a common source of thrombus and has several recognized morphologies, including chicken wing, cactus, windsock, and cauliflower. The chicken wing type is the most common and carries a lower thromboembolic risk in atrial fibrillation.[1] However, it may create echogenic areas that mimic thrombus or masses on transesophageal echocardiography (TEE).[2] Here, we describe a case in which a chicken wing LAA was incidentally identified on intraoperative TEE during surgical excision of a left atrial myxoma, initially appearing as a suspected additional mass or thrombus, before being confirmed through multiplane imaging and surgical inspection.

Case Presentation

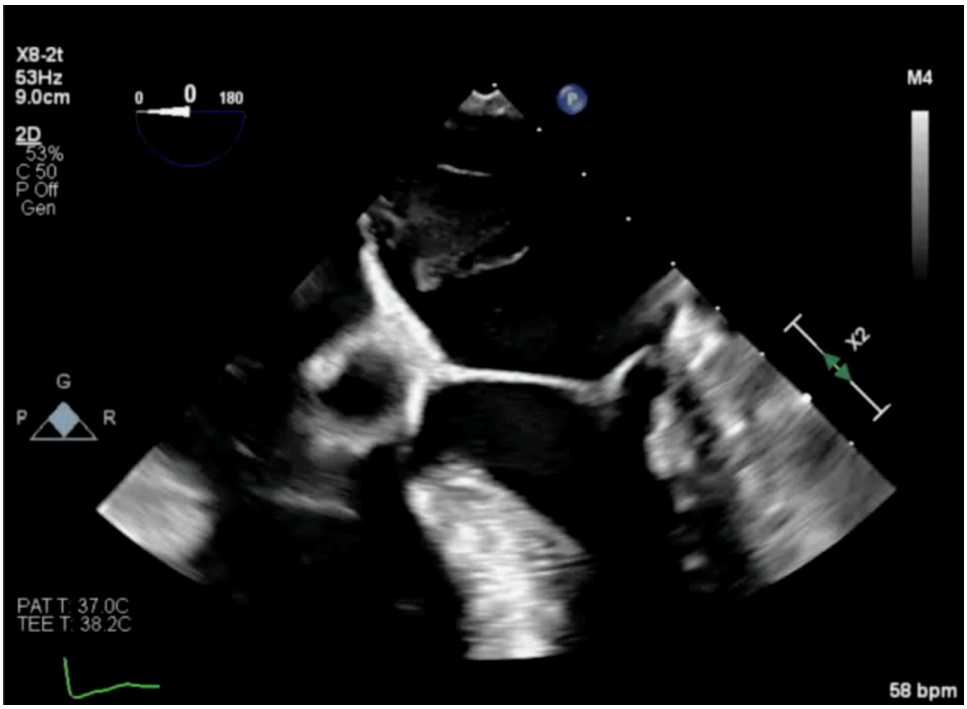
A 55-year-old male with a history of hypothyroidism presented for elective surgical resection of a left atrial mass. He initially presented 3 months prior after noticing palpitations and intermittent nonspecific chest pain. Transthoracic echo showed normal biventricular function, an ejection fraction 50-55%, and a mobile, isoechoic 0.9cm x 2.0 cm left atrial (LA) mass attached to the interatrial septum or the inferior LA free wall. The patient was started on Eliquis and referred for TEE, which confirmed a 2x2cm mobile mass adherent to the interatrial septum. No LAA or right atrial thrombus was seen. Peripheral enhancement of the mass favored myxoma over thrombus. Holter monitor showed normal sinus rhythm with rare preventricular contractions. On the day of the procedure, the patient's SpO2 was 100% on room air and he remained in normal sinus rhythm. A pre-induction arterial line was placed, and general anesthesia was induced with propofol, fentanyl, and rocuronium. A right IJ triple lumen central venous catheter was placed. TEE again confirmed a homogenous hypodense left atrial mass adherent to the septum. However, an extracardiac mobile element in the oblique sinus was also seen raising concern for possible LAA thrombus. After discussion with colleagues, this was deemed to represent a chicken wing LAA variant and surgery proceeded as planned with cardiopulmonary bypass support. Anesthesia was maintained with isoflurane. The remainder of the procedure was uneventful and surgical exploration confirmed that the mass was the tip of a chicken wing LAA variant. Postoperative TEE showed good biventricular function, complete removal of the left atrial myxoma, and the absence of the extracardiac mass. The patient was transferred to the ICU intubated and sedated with minimal inotropic support.

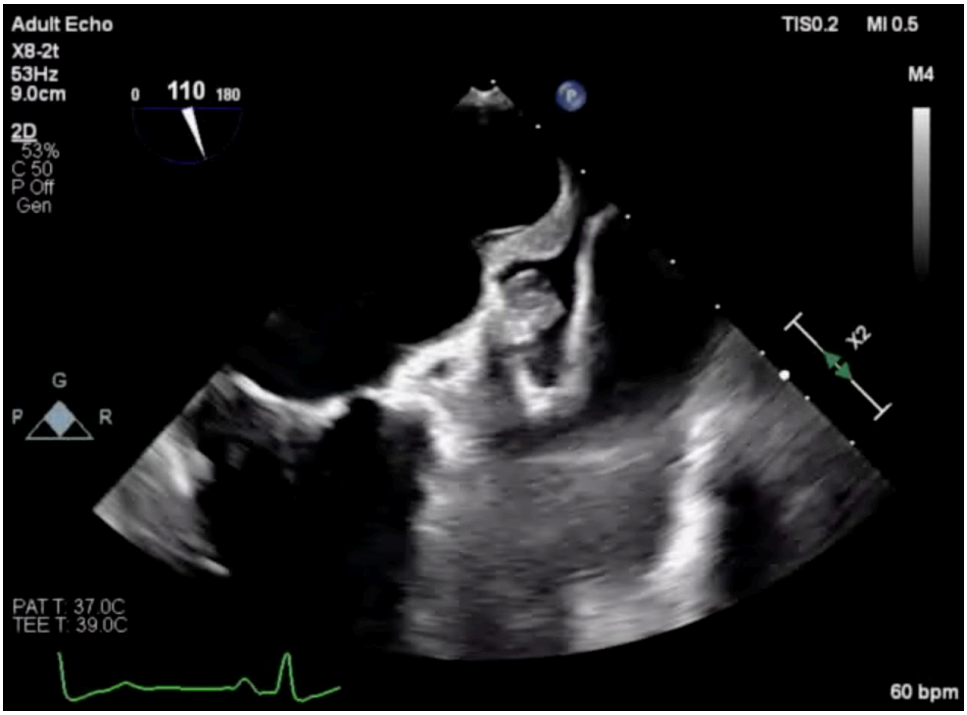
Discussion

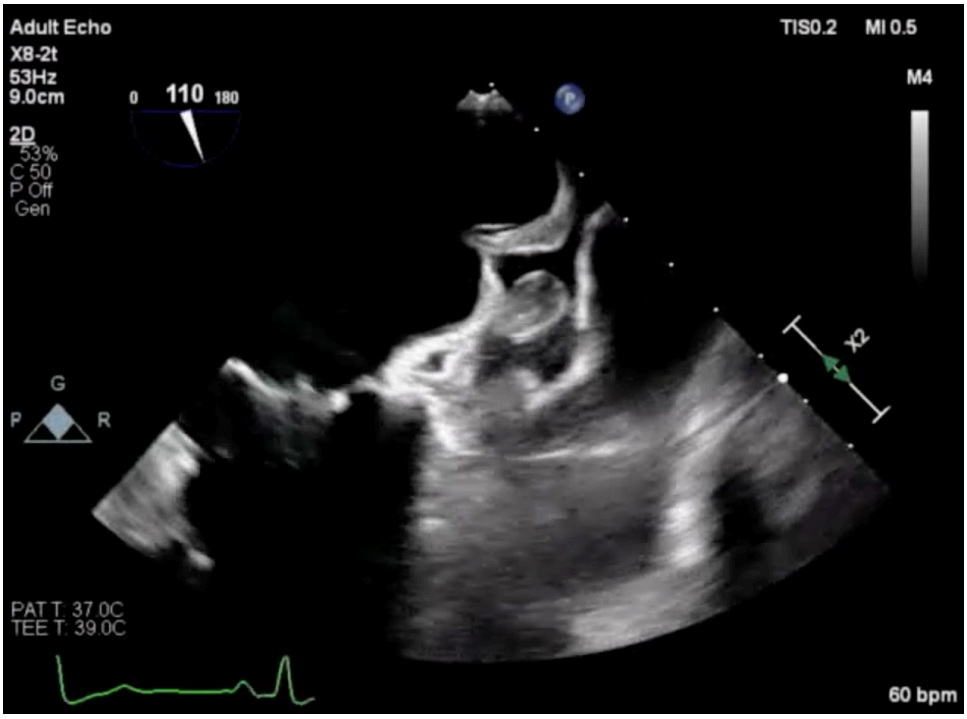
TEE remains the method of choice for evaluating LAA anatomy and function. The chicken wing morphology, seen in ~48% of patients, can mimic thrombus or masses, making multiplane imaging, observation, and Doppler assessment of LAA flow essential.[1] A myxoma may further complicate interpretation by distorting atrial anatomy and flow. This case highlights the utility of intraoperative TEE in real time characterization of LAA anatomy and shows how unusual variants can be mistaken for pathology. Such errors increase the risk

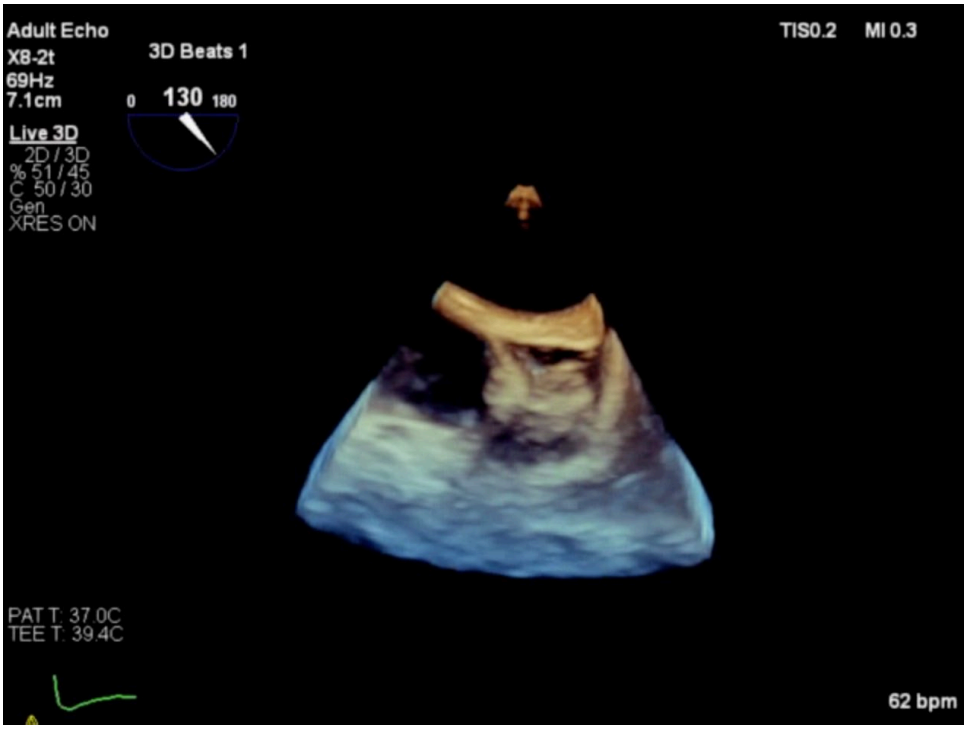
of overcalling findings and potentially misdirecting surgical management.[3] This supports the growing evidence that understanding normal LAA variants, and the limitations of each imaging modality is critical to avoid misdiagnosis and guide appropriate surgical decisions.

Images




Adult Echo
X8-2t
53Hz
9.0cm
0 110 180
2D
53%
C 50
P Off
Gen
TIS0.2 MI 0.5
M4
x2
G
P R
PAT T: 37.0C
TEE T: 39.0C
60 bpm


Adult Echo
X8-2t
53Hz
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PAT T: 37.0C
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60 bpm



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#FR 2 – Kiosk 21 – Poster 3

Intrathoracic Robotic Tracheal Resection Requiring Venovenous Extracorporeal Membrane Oxygenation and Collaborative Approach to Targeting

Presenting Author: Sean P. Moore, D.O., Anesthesiology Resident, University of Arizona

Introduction

Tracheal resection cases can be challenging for anesthesiologists due to a shared airway and limited ability to provide continuous or cross-field ventilation. Due to the ventilation limitations, veno-venous extracorporeal membrane oxygenation (VV-ECMO) has emerged as a valuable adjunct in complex airway surgery, offering a means of maintaining oxygenation and carbon dioxide clearance independent of the airway when other methods of ventilation are not possible due to factors such as robotic approach or location of lesion [1]. This case highlights the anesthesiologist's role in safely managing a patient with severe tracheal stenosis undergoing resection with planned intraoperative VV-ECMO support.

Case Presentation

A 54-year-old female with type 2 diabetes mellitus and severe (3 mm lumen, Figure 1) intrathoracic tracheal stenosis refractory to serial dilations presented for robotic tracheal resection. Given the location, robotic surgical approach, and severity of the lesion, cross-field ventilation was not possible and the decision was made to initiate VV-ECMO during the case. General anesthesia was induced with Cardiothoracic Surgeon at bedside, and the trachea was intubated with a 7.5 mm endotracheal tube (ETT) and position was confirmed with bronchoscopy. Ventilation was adequate with the ETT and the surgical team placed VV-ECMO cannulas under transesophageal echocardiography (TEE) guidance. After ECMO initiation, the ventilator was transitioned to a continuous pressure mode to limit atelectasis. The surgeons proceeded with a robotic thoracoscopic approach for tracheal exposure and resection, with flexible bronchoscopic guidance by anesthesiology to localize the lesion and guide the surgical approach (Figure 2). Serial arterial blood gases were obtained throughout the case to titrate ECMO support. At the conclusion of the procedure, the patient was successfully extubated in the operating room and was able to maintain airway patency and adequate ventilation. The ECMO circuit remained in place and ECMO was successfully weaned with cannula removal within three hours of ICU admission.

Discussion

Planned intraoperative VV-ECMO during complex intrathoracic procedures provides an additional margin of safety for the anesthesiology team and enhances the surgeon's ability to safely intervene. Key anesthetic considerations include optimal timing of ECMO initiation, management of oxygenation and carbon dioxide clearance, and anticoagulation strategy. Once ECMO is established, ventilator settings can be reduced to lung-protective parameters and to avoid interference with the surgical field [2]. Frequent arterial blood gas analysis guides adjustment of ECMO blood flow and sweep gas to maintain physiologic targets [3]. Close monitoring is especially important in cases requiring insufflation with CO_2 as clearance is less predictable than with a ventilated patient leading to rapid pH changes and hypercapnia. This case highlights the

importance of anesthesiologist expertise in ECMO physiology, coordination, and intraoperative management to optimize patient safety and facilitate successful surgical outcomes.

Images



Figure 1: CT Imaging of Tracheal Narrowing Demonstrating 3mm Intraluminal Stenosis

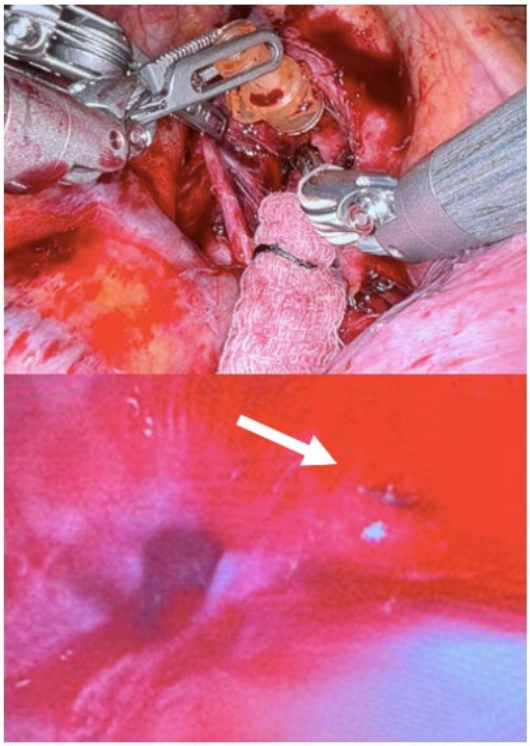


Figure 2: Robotic and Bronchoscopic Views of Surgical Localization

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#FR 2 – Kiosk 21 – Poster 4

ACHD, Cirrhosis and Coagulopathy Complicating Sternotomy Number Seven

Presenting Author: Catherine F. Jerman, M.D., Clinical Fellow in Adult Cardiac Anesthesia, Massachusetts General Hospital

Introduction

Patients with ACHD and congestive hepatopathy are a growing population in adult cardiac surgery. Successful management requires understanding the interplay between patient physiology and surgical conditions.

Case Presentation

A 38 y/o female with DiGeorge syndrome (type B interrupted aortic arch, posterior malalignment VSD) presents for 7th redo sternotomy and PVR. Prior procedures are in Table 1. She has recurrent, severe RVOT obstruction, RV failure and cirrhosis due to congestive hepatopathy (Table 2) with portal hypertension, varices, splenomegaly and thrombocytopenia. Preoperative data is in Table 3. Venous access included a right arm large bore PIV, right IJ introducer and a PAC. A right radial arterial line was placed. Lidocaine, etomidate and rocuronium were used for induction. Vasoplegia and RV dysfunction manifested post-induction requiring inopressor support. Peripheral CPB was initiated prior to sternotomy and the patient was cooled to 32 °C. Mediastinal dissection injured the RV free wall, innominate vein and RVOT. Cardiotomy suction catheters maintained euvolemia during dissection. On CPB, in the absence of an intracardiac shunt, vasoactives augmented perfusion despite massive hemorrhage. The ascending aorta and aortic root were not dissected - thus, unavailable for cross clamping or cardioplegia delivery. The RVOT, innominate vein and RV free wall were reconstructed on a beating heart using an acellular homograft and bovine pericardium. CPB time was 226 minutes. Vasoplegia, RV dysfunction and coagulopathy complicated separation. Interventions included pressors, inotropes, balanced transfusion, viscoelastic testing, cell salvage, antifibrinolytic, PCC, methylene blue and inhaled nitric oxide. The chest was left open and closed POD 1. She was extubated POD 2 and discharged home POD 10.

Discussion

We describe a 7th redo sternotomy for a patient with ACHD, cirrhosis and coagulopathy. Multiple sternotomies increase the risk of catastrophic re-entry injury. Pre-incision peripheral CPB decompresses cardiac structures, maintains circulating blood volume and allows better hemodynamic control with re-entry injury. Limitations include early full heparinization and prolonged hypothermic time on CPB [1,2]. Peripheral cannulation and operating on a beating heart avoid dissection through adhesions to expose the aorta and cardioplegia use. Avoiding deeper hypothermia prevented malignant arrhythmias and worsening coagulopathy. Anesthetic management required attention to both cirrhotic physiology and CPB-related derangements. Despite RV failure and RVOT obstruction, initial CO and CI reflected the high output state of cirrhosis. CPB flow should be optimized using large cannulas. Cirrhotic cardiomyopathy may be masked by low SVR [3]. These abnormalities are amplified with anesthesia and CPB manifesting as vasoplegia and

reduced CO. Cirrhosis impacts synthesis of pro- and anticoagulant factors. Thrombocytopenia occurs due to reduced thrombopoietin and splenic sequestration. CPB further disrupts functional coagulation. Viscoelastic testing may more accurately reflect real-time hemostasis and minimizes transfusion [3]. PCC should be considered as it reduces volume administration, which is especially important with a dysfunctional RV.

Images

Table 1. Prior Open and Percutaneous Cardiac Procedures

Sternotomy	Age	Procedure
1	1 month	Arch Repair, VSD Closure
2	2 years	Subvalvular AS Repair
3	6 years	Revision Subvalvular AS Repair
4	9 years	Ross/Konno Procedure
5	14 years	Mechanical AVR, TV annuloplasty
Multiple percutaneous procedures for RVOT homograft obstruction including percutaneous pulmonary valve replacement, right pulmonary artery stenting		
6	23 years	Pulmonic valve replacement (valve in valve), TV repair

Abbreviations: ventricular septal defect (VSD), aortic stenosis (AS), aortic valve replacement (AVR), tricuspid valve (TV) right ventricular outflow tract (RVOT)

Table 1: Prior Open and Percutaneous Cardiac Procedures

Table 2. Hepatic Risk Stratification

Risk Calculator	Result
VOCAL-Penn	30-day mortality: 16.4% 90-day mortality 19.9%, 180-day mortality 23.3%, 90-day decompensation 21.7%
MELD-Na	12
Child-Turcotte-Pugh	Class B (8)

Abbreviations: veterans' outcomes and costs associated with liver disease (VOCAL), model for end stage liver disease (MELD)

Table 2: Hepatic Risk Stratification

Table 3. Select Preoperative Testing

Test	Result
Right Heart Catheterization	Mean RAP 21 mmHg RV 91/9/16 mmHg Systolic transpulmonic gradient 52 mmHg Mean PAP 21 mmHg PAWP 18 mmHg CO 8.1 L/min, CI 3.1 L/min/m2 No intra-cardiac shunt
CT Angiography	Small (~1.5 cm) RV to PA homograft with bioprosthetic valve-in-valve. Third and fourth sternotomy wires in close proximity to the pulmonary artery and right ventricle.
EKG	NSR, RBBB, LAFB, PACs, PVC, RVH
Vascular Ultrasound	Femoral vessels suitable for peripheral cannulation
Laboratory Values	Hgb 10.8 g/dL, PLT 71 K/uL, Cr 0.9 mg/dL, Albumin 2.4 g/dL, Total Bilirubin 1.6 mg/dL, INR 1.2, Na 138 mEq/L
TEG	Normal

Abbreviations: right atrial pressure (RAP), pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP), cardiac output (CO), cardiac index (CI), pulmonary artery (PA), normal sinus rhythm (NSR), right bundle branch block (RBBB), left anterior fascicular block (LAFB), premature atrial complex (PAC), premature ventricular complex (PVC), right ventricular hypertrophy (RVH), hemoglobin (Hgb), platelets (PLT), creatinine (Cr)

Table 3: Select Preoperative Testing

References

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#FR 2 – Kiosk 21 – Poster 5

Leadless Pacemaker Malfunction During Complex Fourth Time Sternotomy for Mitral Valve Replacement and Tricuspid Valve Repair

Presenting Author: Alec Statler, MD, West Virginia University Hospitals

Introduction

The number of new medical devices has significantly expanded in recent years adding another hurdle to the anesthesia world. Leadless pacemakers (PMs) have been approved for use in the United States since 2016 with specific scenarios favoring their implantation [1]. Unsurprisingly, we are now encountering more patients with these devices. Leadless PMs lack a magnet response in an emergency and with no additional guidelines we often resort to recommendations for the traditional PMs [2]. We describe a case of complex anesthetic management involving a difficult airway, difficult echocardiographic imaging, and despite thoughtful management, loss of capture in a leadless device.

Case Presentation

A 30-year-old female presented for fourth-time sternotomy for tricuspid and mitral valve replacement. Her history consisted of remote intravenous drug use on lifetime suppressive antibiotics, mitral and tricuspid prostheses regurgitation, junctional bradycardia requiring a leadless PM, Factor V Leiden managed with warfarin, hepatitis C, and difficult intubation. Her preoperative imaging showed mitral prosthesis prolapse, and a tricuspid prosthesis flail leaflet. Upon patient evaluation, the leadless PM was noted to be an Abbott Aveir single-chamber device located in the right atrium. A day prior to surgery, due to PM dependence, cardiology was consulted. Given the complexity of the situation, they contacted the manufacturer representative who reprogrammed it to an asynchronous mode at a rate of 80 bpm prior to surgery. A brachial arterial line was placed per institutional practice. Induction of anesthesia and video laryngoscopy proceeded uneventfully. The TEE probe was placed without difficulty. The anesthesia team gained central access and additionally placed a percutaneous superior vena cava cannula via the right internal jugular vein. Surgery proceeded with peripheral cannulation. During dissection in the vicinity of the right atrium, the PM lost capture with resultant bradycardia to the low 20s. A right ventricular epicardial wire was urgently placed and pacing commenced at 80 bpm via an available temporary pacemaker. Following intraoperative TEE evaluation, a decision to proceed with tricuspid valve repair and mitral valve replacement was agreed upon. Imaging following cardiopulmonary bypass was significantly limited. Additional probes as well as gastric decompression and fluid-filling was unsuccessful at improving image quality. Epicardial imaging performed by the anesthesia team in the surgical field verified normal valvular function. A permanent epicardial pacing generator was placed with atrial wires at the conclusion of the case and the patient was transported to the ICU for postoperative care.

Discussion

Our case demonstrates a patient with a complex history that resulted in a challenging anesthetic. Following the guidelines set for traditional PMs, her device was placed in asynchronous mode due to the risk of

oversensing in a PM dependent patient. Unfortunately, the device failed to function during the case. On further discussion, it was determined to be unlikely that electrocautery to the device caused failure, and most likely that the energy transferred caused the impedance of the surrounding tissue to increase significantly, causing failure to capture.

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#FR 2 – Kiosk 21 – Poster 6

Undiagnosed Congenital Pulmonary Airway Malformation in a Preterm Newborn Undergoing Thoracotomy with Right Middle Lobectomy

Presenting Author: Evelyn Borchert Billik, MD, Anesthesia Resident, Rutgers Robert Wood Johnson Medical School

Introduction

Congenital pulmonary airway malformations (CPAMs) are rare developmental cystic lung lesions characterized with abnormal connection to the tracheobronchial tree and normal pulmonary vascularization.[1] CPAMs are the most common congenital lung anomalies, accounting for 30%, and are typically detected between 18 and 20 weeks of gestation.[2] Advancements in fetal ultrasound have improved detection rates, with an incidence of up to 1 in 2500 live births.[3] Stocker's classification divides CPAMs (Table 1) into five major types, with type1 the most common. Prenatal imaging helps assess associated complications such as mediastinal shift, fetal hydrops, polyhydramnios, and fetal demise. Management depends on the lesion's size, and secondary complications. Antenatal treatment includes maternal steroids for lesions with hydrops or fetal interventions in macrocystic CPAMs. Postnatally, symptomatic CPAMs typically require surgical resection. Symptoms include respiratory distress, infection, pneumothorax, hemorrhage, feeding difficulties and rare malignant transformation. Resection is also advised for asymptomatic lesions to avoid complications and to maximize the potential for compensatory lung growth.[1,2]

Case Presentation

Two-week-old ex-32-week-5-day female with no prenatal care was born at home and underwent CPR at birth before hospital transfer. CXR revealed a large right-sided thoracic mass (Fig.1) causing a massive mediastinal shift. She required high-frequency oscillatory ventilation due to respiratory failure. CT SCAN showed a 7.6-cm complex multiloculated cystic lesion in the right lung, collapsing the right mainstem bronchus and displacing the heart and mediastinum (Fig.2). Due to worsening respiratory failure and severe mediastinal deviation, decision was made to proceed with surgical intervention. A right thoracotomy with right middle lobectomy was performed under general anesthesia with vasoactive support. Although ECMO was standby, it was not required. Intraoperatively, 300 ml of pressurized fluid was drained from the cyst, and the mass was fully resected. Postoperatively, the patient was ventilated and managed supportively. Respiratory function improved steadily; she was extubated by POD#7 and transitioned to bubble CPAP for seven weeks. CXR showed improved lung aeration (Fig. 3). Initial echocardiogram revealed mildly depressed left ventricular systolic function, which gradually improved. Mild elevated pulmonary artery pressure persisted. Patient was able to advance progressively to full enteral feeds and no vasopressor support was required postoperatively. She was discharged on POD#86 to a specialized Children's Hospital for feeding therapy.

Discussion

Histopathology confirmed Stocker type I CPAM with atypical mucinous cells. Unresected type1 CPAMs have been associated with mucinous adenocarcinoma. Molecular testing including KRAS mutations and other genes (DICER1, SFTPB, ITGA3 and CCR2) was negative, suggesting no germline variants. Outcome in CPAMs depends on prenatal hydrops, symptom severity at birth, and lesion size.[3] This case highlights the potential severity of large lesions and mediastinal shift. The patient's positive outcome following surgical resection emphasizes the importance of early diagnosis, timely intervention, and coordinated multidisciplinary care.

Images

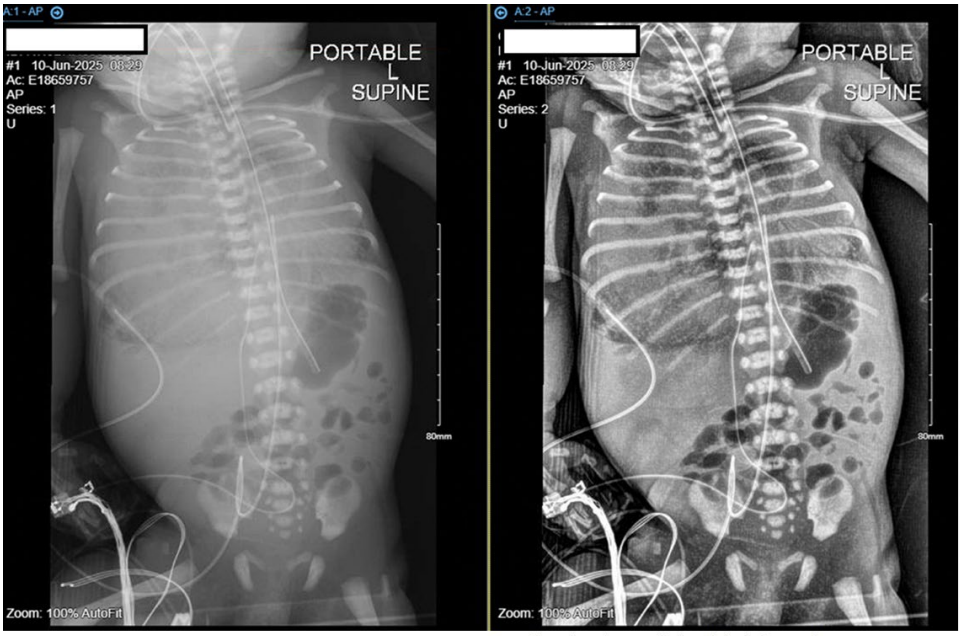


Figure 1: **Chest X-ray of the newborn on day 1 of life:** Showing large right-sided thoracic mass (diffuse soft tissue opacification of the right lung with some sparsely aerated regions). Shift of the heart and mediastinum into the left hemithorax.

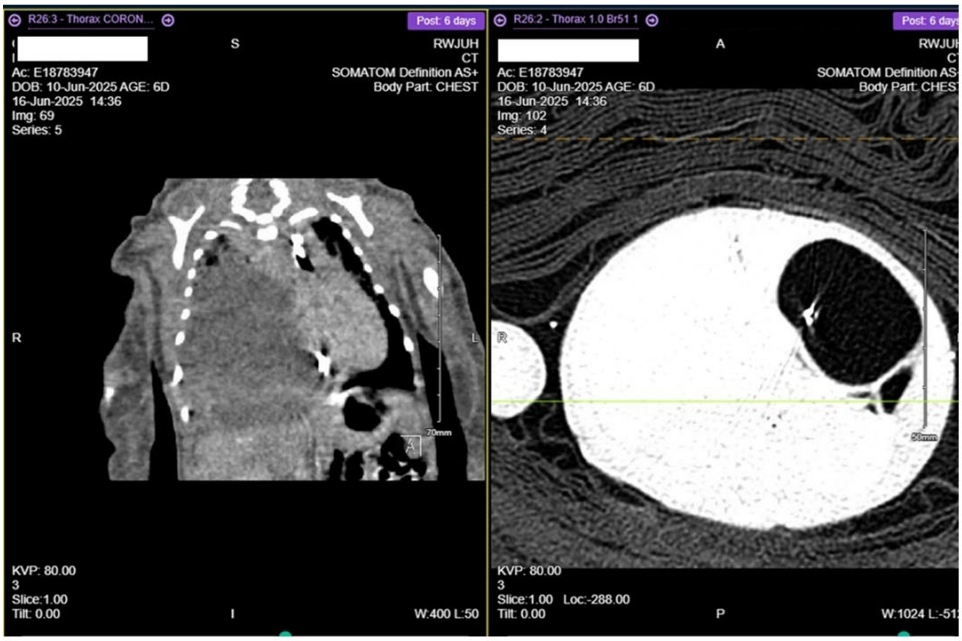


Figure 2: CT Chest Abdomen Pelvis of the newborn on day 1 of life: Large 7.6 cm complex lesion occupying the majority of the right hemithorax, with multiloculated air-filled components as well as a large hypodense component. This lesion exerts mass effect and shift the heart and mediastinum into the left hemithorax and the left lung is small.

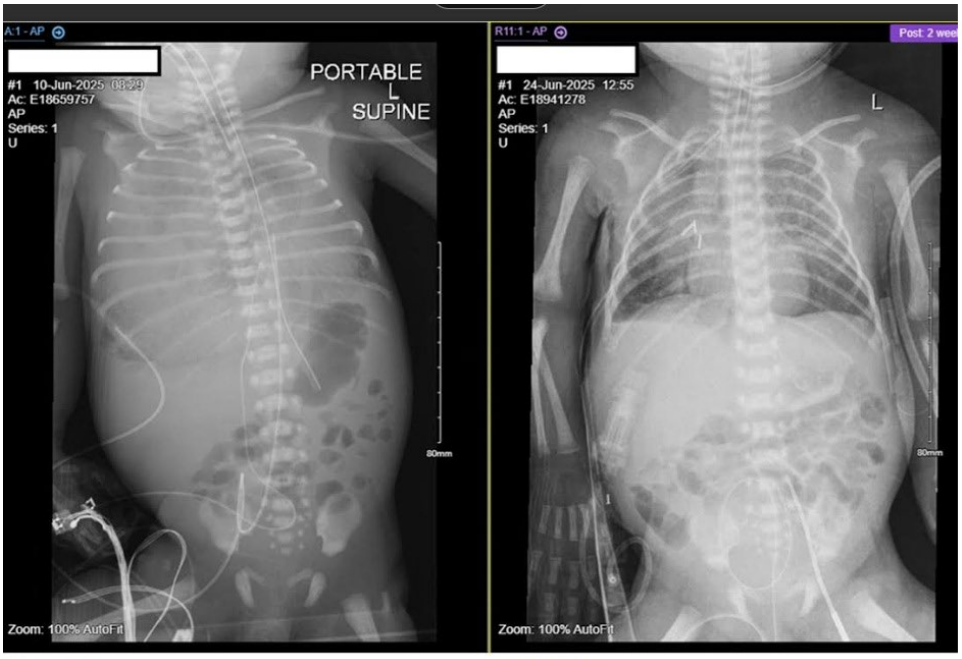


Figure 3A **Figure 3B**

Figure 3 pre and postoperative Chest X-ray comparison.

3.A Large right-sided thoracic mass (diffuse soft tissue opacification of the right lung with some sparsely aerated regions). Shift of the heart and mediastinum into the left hemithorax.

3.B Significant improvement in the aeration of the right lung. The large lucency which previously identified to overlie the mediastinum is no longer visualized. Right-sided chest tube terminating in the right lung apex.

Table 1 Stocker Classification Describing the Five Major Types of CPAM

Stocker Classification	Origin	Appearance	Prognosis
Type 0	Trachea or bronchi	Bronchial-type airways with cartilage, smooth muscles, and glands separated by mesenchyme	Incompatible with life
Type 1	Bronchi	Cysts are usually multiloculated, within one lobe and lined with pseudo-stratified columnar epithelium	Excellent prognosis following resection. Rarely undergoes malignant transformation
Type 2	Bronchiolar regions	Multiple small cysts versus no mass effect	Good prognosis, no malignant potential
Type 3	Bronchiolar regions	Typically solid versus cystic, usually devoid of pulmonary arteries within the lesion	Good prognosis, no malignant potential
Type 4	Acinar structures of the lung	Multiloculated, peripheral, thin-walled cysts lined with type 1 or type 2 alveolar cells	Strong association with pleuropulmonary blastomas

Source: Mehta PA, Sharma G. Congenital Pulmonary Airway Malformation. StatPearls. (2024).

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#FR 2 – Kiosk 22 – Poster 1

Sleeve Gastrectomy in a patient with an LVAD

Presenting Author: Raj Madhani, DO, Resident, University of Pittsburgh Medical Center

Introduction

Durable left ventricular assist devices (LVADs), such as the HeartMate 3 (HM3), improve quality of life and serve as destination therapy or a bridge to transplant. These patients often require anesthesia for cardiac and non-cardiac procedures. When a cardiac anesthesiologist is unavailable, the anesthesia team must understand LVAD physiology. LVADs are preload dependent and afterload sensitive; they use a centrifugal pump that moves blood from the left ventricle to the ascending aorta, with flow determined by pump speed and systemic vascular resistance.

Case Presentation

A 50-year-old male with ischemic cardiomyopathy, a HM3 implanted in 2023, and morbid obesity presented for sleeve gastrectomy to qualify for transplant listing. Right heart catheterization and preoperative optimization two days prior were unremarkable. HM3 interrogation showed a speed of 5000 rpm, flow 3.2 L/min, power 3.1 W, and pulsatility index 8.6. A right radial arterial line was placed preinduction. Baseline blood pressure was 115/75 mmHg. Induction with ketamine and etomidate caused hypotension (MAP 60 mmHg) that responded to norepinephrine and phenylephrine. A right internal jugular CVC was placed, and the patient remained stable during port placement and insufflation. During transition to steep reverse Trendelenburg, blood pressure decreased to 80/60 mmHg (MAP 64), and HM3 flows fell to 1.7 L/min with low-flow alarms. He had several brief runs of NSVT with MAP falling to 50 mmHg. Management included reducing pump speed to 4600 rpm, returning supine, giving fluid boluses, and starting epinephrine at 0.03 mcg/kg/min, after which flow and MAP normalized. After 20 minutes supine, the surgeon again required steep reverse Trendelenburg. Slow repositioning over five minutes led to a MAP decline to 63 mmHg, prompting vasopressin at 0.04 U/hr with recovery of pressures. He then tolerated maximal steep reverse Trendelenburg without further instability. After returning supine, blood pressure rose to 143/70 mmHg, allowing vasopressor discontinuation. He was extubated in the OR and recovered uneventfully.

Discussion

Bariatric surgery can reduce readmissions in LVAD patients and, in this case, offered a path toward transplant eligibility[1]. For selected elective procedures, benefits may outweigh risks. Anesthesiologists must understand LVAD physiology, anticipate surgical factors, and manage hemodynamic instability. Preoperative planning includes LVAD interrogation and confirming stability of heart-failure medications[2]. Right heart catheterization may help assess volume status. Intraoperatively, arterial lines are preferred because oscillometric readings may be unreliable with low pulsatility. Central venous access facilitates rapid administration of vasopressors, inotropes, and fluids[3]. Neuraxial anesthesia is generally avoided due to anticoagulation. Because LVADs are preload dependent, inadequate preload can cause suction events and arrhythmias, likely contributing to this patient's hypotension and NSVT. High afterload can impair pump

flow and lead to pulmonary congestion. MAP targets commonly range from 60–80 mmHg, with some centers favoring 70–90 mmHg. With thorough preparation and understanding of LVAD physiology, non-cardiac surgery can be performed safely in LVAD-supported patients.

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#FR 2 – Kiosk 22 – Poster 2

Complex Redo Thoracic Aortic Reconstruction for Infected Arch Graft with Aortobronchial Fistula

Presenting Author: Darshan Rola, DO, Resident, Henry Ford Hospital

Introduction

Aortobronchial fistula (ABF) is a rare but life-threatening complication of thoracic aortic graft infection. Clinical presentation ranges from hemoptysis to catastrophic hemorrhage, and survival requires rapid diagnosis and definitive surgical repair. Anesthetic management is challenging due to potential airway contamination, hemodynamic instability, and the need for circulatory arrest. We present a case of emergent redo thoracic aortic surgery requiring complex airway, perfusion, and postoperative critical care management.

Case Presentation

A 61-year-old man with a history of open repair for coarctation presented with abdominal pain and was found to have a mycotic superior mesenteric artery pseudoaneurysm requiring laparotomy. Subsequent CT imaging demonstrated a pseudoaneurysm of the ascending/arch graft, and blood cultures grew *Streptococcus*. He then developed massive hemoptysis requiring emergent intubation, raising concern for an aortobronchial fistula. Due to concern for impending rupture, the patient was taken emergently to the operating room. Intraoperative bronchoscopy revealed a pulsating lesion in the left mainstem bronchus. Given the location of the defect, a right-sided double-lumen tube was placed to minimize the risk of postoperative contamination. A left thoracotomy was performed, and cardiopulmonary bypass was initiated via percutaneous femoral cannulation. Deep hypothermic circulatory arrest was used to safely explant the infected arch and descending graft. A graft was anastomosed from zone 2 to zone 5, restoring continuity of the arch and descending thoracic aorta. A pericardial fat rotational flap was interposed between the graft and the left mainstem bronchus to buttress the fistula tract. Multiple wedge resections were performed for associated lung parenchymal injuries. On rewarming, the patient exhibited profound ventilatory dysfunction requiring inhaled nitric oxide. Persistent coagulopathy necessitated leaving the thoracotomy open. He was transferred to the ICU in critical but stable condition.

Discussion

This case highlights several anesthetic and surgical challenges in managing ABF secondary to infected aortic grafts. Emergency presentation with hemoptysis requires immediate airway control and preparedness for massive hemorrhage. Dense adhesions from prior thoracotomy, active infection, and fistulization necessitate complex dissection and prolonged bypass times. Deep hypothermic circulatory arrest provides a bloodless field for safe graft explantation but requires meticulous neuroprotection. Significant inflammatory response and pulmonary compromise are common following extensive aortic reconstruction and mandate a multidisciplinary postoperative strategy. Early recognition, coordinated airway management, and preparedness for physiologic collapse are critical determinants of survival in these high-risk cases.

Images



CT image showing the aorto-bronchial fistula in the left lung

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#FR 2 – Kiosk 22 – Poster 3

Anesthetic Management for Combined Heart-Lung Transplantation in a Patient with Prior Mustard Procedure, Biventricular Failure, and Severe Pulmonary Hypertension

Presenting Author: Anuradha Kanaparthi, MD, Resident Physician, Mayo Clinic

Introduction

D-transposition of the great arteries (D-TGA) results from ventriculoarterial discordance and atrioventricular concordance with complete transposition of the pulmonary and systemic arteries. Apart from atrioseptostomies and Blalog-Taussig shunts for palliation, Mustard/Senning atrial switch procedures have been historically performed for correction prior to now more common Rastelli/Jatene arterial switch procedures. While patients who undergo atrial switch procedures perform relatively well, they may present at later ages with complications and ventricular failure warranting heart and/or lung transplantations for definitive cure.[1]

Case Presentation

A 55-year-old male with D-TGA repaired via Mustard procedure complicated by moderate SVC baffle stenosis and IVC baffle leak with failing systemic right ventricle (sRV, RVEF 35-40%), severe pulmonary hypertension (PH), and failing subpulmonary left ventricle (spLV, LVEF 20-25%) presented for combined heart-lung transplant. Milrinone and dobutamine were started and continued in the intraoperative setting. Pre-induction radial arterial line was placed, and he underwent a hemodynamically stable induction with ketamine and propofol with subsequent endotracheal intubation, transesophageal echocardiogram (TEE) probe placement, and central vascular access with pulmonary artery catheter (PA-C). A left femoral arterial line was additionally placed to allow for peripheral cardiopulmonary bypass if needed due to redo sternotomy. Acute normovolemic hemodilution (ANH) was completed pre-CPB. Surgical procedure involved extensive adhesiolysis due to prior sternotomies. When initiating CPB, venous return was poor but improved with cannula repositioning. Following explantation of native and implantation of donor heart and lungs, vasopressin, norepinephrine, epinephrine, milrinone, isoproterenol, and calcium chloride boluses and/or infusions were utilized to separate from CPB. Inhaled nitric oxide was connected to the circuit but not required. He was weaned off vasoactive support over the ensuing days, extubated POD1, reintubated due to secretion burden, extubated POD3, and discharged POD18.

Discussion

To our knowledge, no published reports describe combined heart–lung transplantation in a patient post-Mustard repair, highlighting the rarity and complexity of this case. Apart from sRV failure, several anatomic and physiologic intricacies shaped our anesthetic management. First, the patient's severe PH, compounded by spLV failure, required careful control of pulmonary and systemic vascular resistance notably during induction and separation from CPB to avoid sRV collapse. Notably, his baffle anatomy required cautious PA-C placement due to risks of malposition or trauma to the channels. Second, his SVC baffle stenosis with extensive mediastinal collaterals altered venous return, rendering central venous pressure measurements

unreliable; thus, TEE served as the primary tool for assessing preload and guiding resuscitation. Third, his IVC baffle leak contributed to right-to-left shunting and created potential for paradoxical emboli, necessitating heightened vigilance with air management and de-airing. This case underscores the importance of understanding baffle anatomy, long-standing sRV physiology, and PH in patients following Mustard repair.

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#FR 2 – Kiosk 22 – Poster 4

Threading the Needle: Pulmonary Artery Catheter-Directed Thrombolysis for Massive Perioperative PE

Presenting Author: David R. Hollis, MD, Resident Physician, Northwestern University

Introduction

Management of massive pulmonary embolism (PE) in the perioperative setting requires rapid diagnosis and multidisciplinary coordination. Therapeutic strategies include systemic anticoagulation, catheter-directed thrombectomy, surgical embolectomy, and extracorporeal membrane oxygenation (ECMO) as rescue support. Transesophageal echocardiography (TEE) enables rapid diagnosis and guides lifesaving interventions. We report a case of profound hypoxemia during peripheral vascular surgery that revealed a right-to-left intracardiac shunt and extensive PE pathology requiring extracorporeal membrane oxygenation (ECMO).

Case Presentation

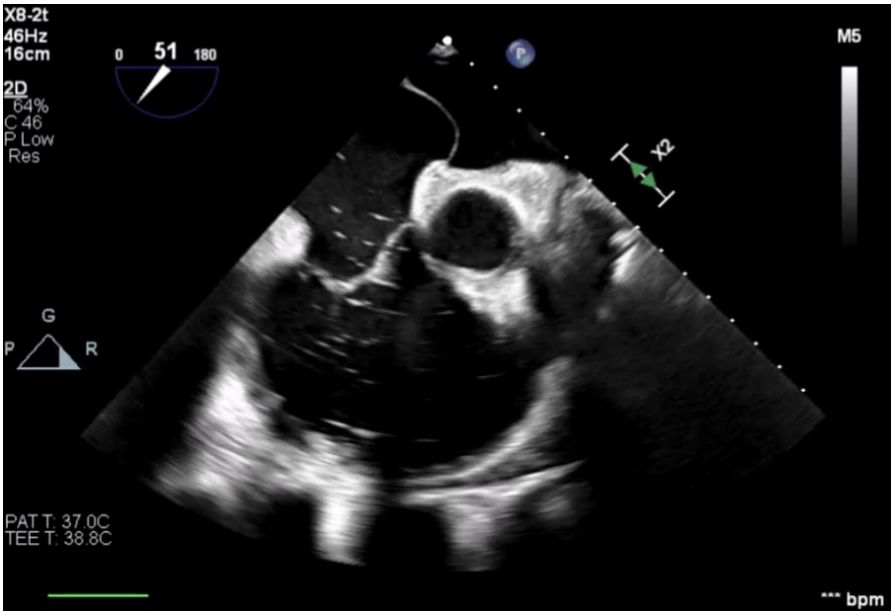
A 68-year-old male with hypertension, hyperlipidemia, diabetes, prior transient ischemic attack, and previous pulmonary embolism, not anticoagulated, presented with chronic limb-threatening ischemia for bilateral femoral-popliteal thrombectomies. During anesthetic induction, his oxygen saturation fell to 68–75% despite 100% FiO_2 and recruitment maneuvers. Transesophageal echocardiography revealed a dilated right ventricle with severely decreased function, a markedly enlarged right atrium, and right-to-left shunting through a patent foramen ovale (PFO). Concern for acute pulmonary embolism prompted activation of the Pulmonary Embolism Response Team and ECMO consults. The patient was cannulated for veno-arterial-venous (VAV) ECMO for cardiopulmonary stabilization. Subsequent CT pulmonary angiography revealed bilateral pulmonary emboli with severe anatomic distortion, including a large left-sided hernia causing rightward mediastinal shift and marked right-lung volume loss. Interventional cardiology and pulmonary hypertension specialists noted chronic complete obstruction of the right upper- and middle-lobe pulmonary arteries with extensive infarcted parenchyma, making catheter-directed thrombectomy unlikely to provide meaningful benefit. With rising lactate, suprasystemic pulmonary artery (PA) pressure of 120/42 mmHg, and cardiopulmonary collapse despite VAV ECMO, systemic thrombolysis was considered a last-resort, high-risk intervention. Tissue plasminogen activator (tPA) was infused via the pulmonary artery catheter, functioning effectively as targeted delivery. Following lytic administration, PA pressures improved substantially to 37/24 mmHg within hours, allowing ECMO conversion to VV support on postoperative day seven and eventual decannulation.

Discussion

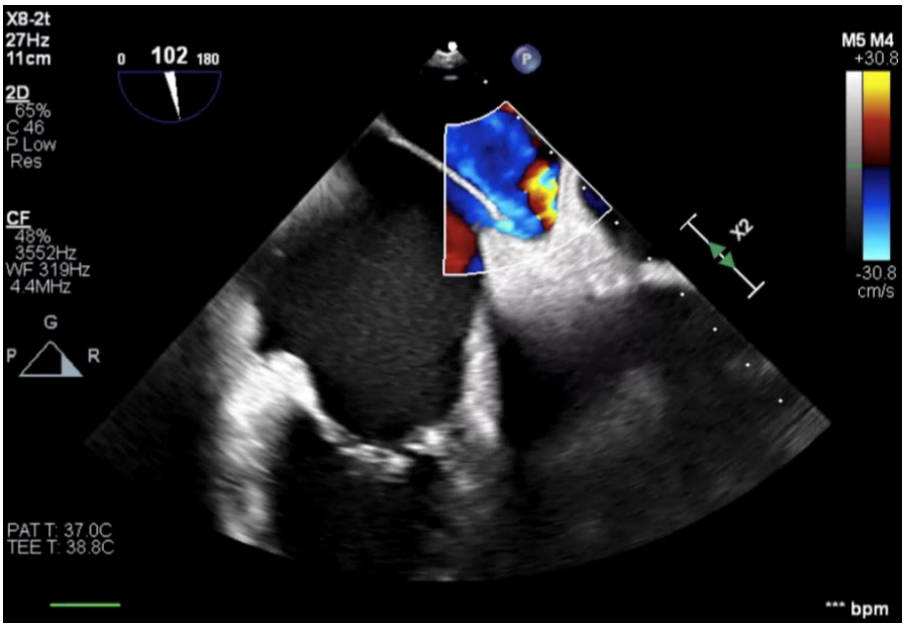
This case highlights the complexity of managing massive PE in the presence of distorted thoracic anatomy and chronic thromboembolic disease. Systemic thrombolytics remain an established therapy for massive PE with hemodynamic compromise, particularly when surgical or catheter-directed thrombectomy is unavailable or unlikely to be effective. Their use is limited by the risk of major bleeding and in this patient the decision to administer tPA represented a true last-resort intervention. ECMO served as a bridge to

reperfusion, maintaining oxygenation and perfusion while lytics restored pulmonary flow. This multidisciplinary approach underscores the anesthesiologist's expanding role in PERT activation, hemodynamic assessment, and coordination of advanced cardiopulmonary rescue in high-risk perioperative PE.

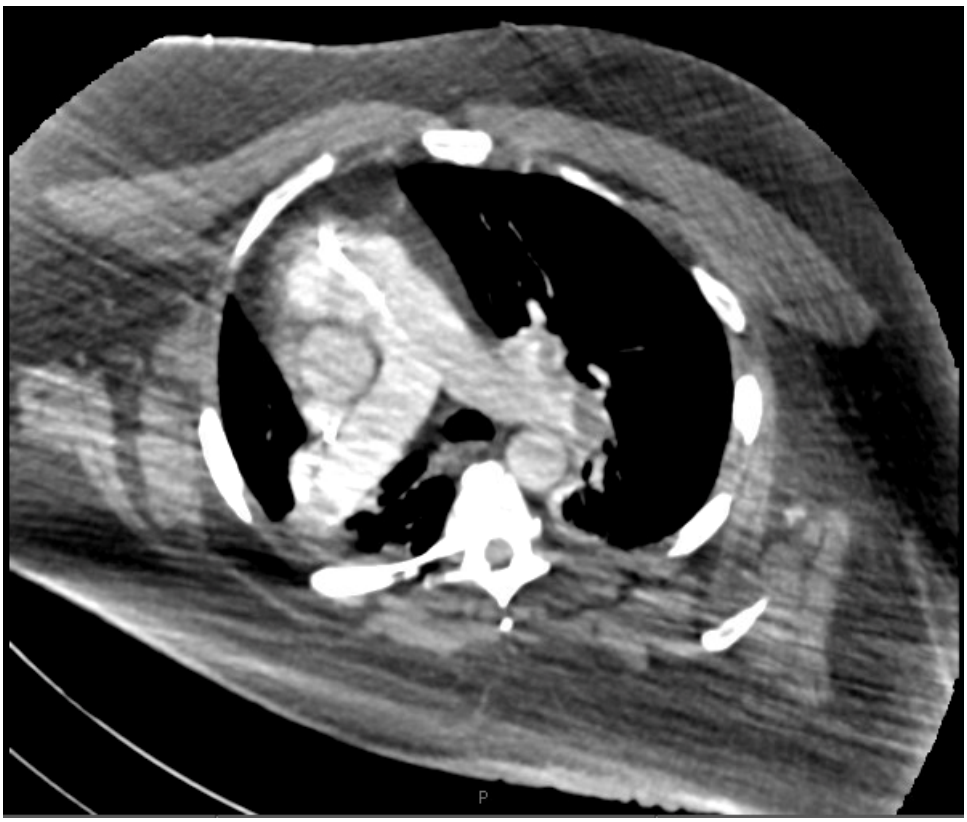
Images



Midesophageal RV inflow-outflow view showing dilated RV with bowing of the interatrial septum to the left.



Midesophageal bicaval view with PFO and evidence of right to left shunt



CT PE axial view displaying massive left pulmonary artery thrombus and concomitant proximal Swan-Ganz catheter.

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#FR 2 – Kiosk 22 – Poster 5

Early Detection of Postoperative Coronary Ischemia by Transthoracic Echocardiography

Presenting Author: Anthony Dao, MD, Fellow, Massachusetts General Hospital

Introduction

Valve sparing aortic root surgery (VSARR) is performed in situations where there is ascending aortic dilation without aortic insufficiency or functional aortic insufficiency with normal leaflet motion. Myocardial ischemia secondary to coronary ostial stenosis is an uncommon but potentially lethal complication following VSARR. POCUS has been increasingly used in critical care to help diagnose causes of shock. Here we present a case where POCUS was used to identify new post-operative wall motion abnormalities (WMA) leading to the identification of coronary ostial stenosis.

Case Presentation

We present a 35-year-old male with Marfan's syndrome and progressive aortic root dilation who presented for VSARR. Pre-op workup was notable for normal biventricular (BiV) function without WMA, non-obstructive coronary artery atherosclerosis without significant stenosis, and no significant valvulopathy. He underwent an uneventful surgery. He arrived to the ICU on 5 mcg/min of norepinephrine. His initial ECG in the ICU showed sinus rhythm with inferolateral ST elevations without reciprocal changes. Post-op TEE and bedside TTE showed normal BiV function without WMA. Given normal function and the lack of focal WMA, his initial ECG changes were attributed to air embolism to the right coronary artery. He was extubated on POD 0 and developed vasoplegic shock, requiring increasing doses of norepinephrine and vasopressin. His other hemodynamics at this time showed a CVP of 4-8 mmHg and a noninvasive CO of 11 L/min. His ECG continued to show sinus rhythm with improved nonspecific ST segment and T wave changes which were attributed to pericarditis. He was volume resuscitated and by the evening was able to be weaned off all vasoactive medications with resulting hemodynamics of MAP in 70s, CVP 7-13. His POD 1 TTE showed preserved LV function, but newly dilated RV with severely reduced function and diffuse hypokinesis. A stat ECG showed small inferior ST segment elevations, but with very low voltage and not meeting criteria for STEMI. On multidisciplinary discussion with the cardiac surgery team, it was noted that the right coronary artery was small and calcified. The interventional cardiology team was consulted and the patient proceeded to the cath lab for urgent left heart cath. The proximal RCA was found to have a focal 75 % stenosis in its ostium due to ostial stenosis or possible early postoperative thrombosis, and he ultimately received a stent to the ostial-proximal RCA. His repeat TTE on POD 3 showed normal LV function, and persistent RV dilation with reduced RV function and global RV hypokinesis, though improved from pre-PCI.

Discussion

This case highlights the importance of routine post-op TTE for any cardiac surgery involving the coronary ostia as well as interdisciplinary discussion for patient care. The incidence of ECG changes post VSARR is not well reported, but ischemic ECG changes post cardiopulmonary bypass is common and cited to be as high as 72.2%[2]. Etiology of ischemic ECG changes post cardiopulmonary bypass is broad with diverging

management pathways. Post-op TTE is a noninvasive procedure that can be implemented to help improve care for patients with new ischemic ECG changes post cardiac surgery.

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#FR 2 – Kiosk 22 – Poster 6

A Case of Severe Aortic Stenosis Post-TAVR and the SURPLUS Solution

Presenting Author: Jordan L. Horstman, DO, Anesthesiology Resident, Brooke Army Medical Center

Introduction

This case involves a patient presenting with heart failure 5 years after a TAVR found to have severe aortic stenosis of the implanted valve. The patient's comorbidities, clinical status, and anatomic considerations underscore several teaching points, including incidence and etiology of TAVR reintervention, contemporary approaches and decision making.

Case Presentation

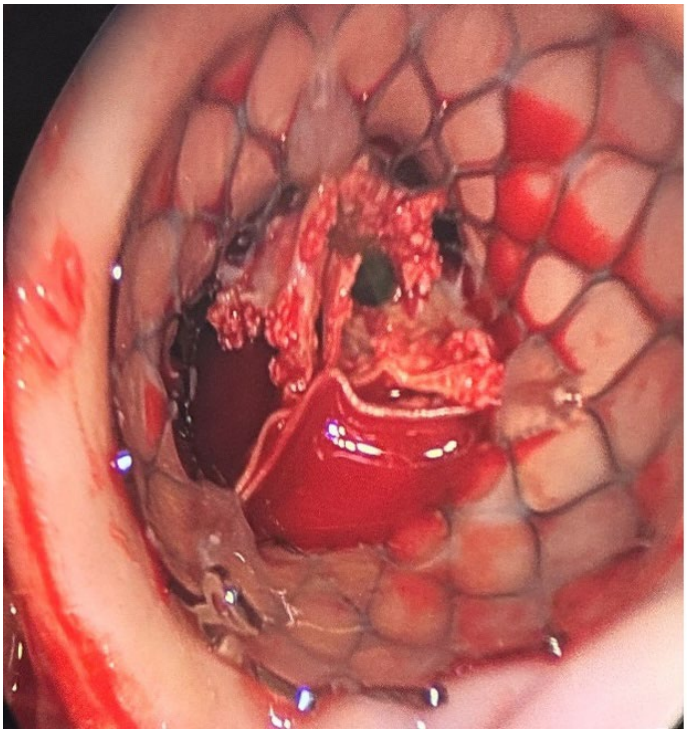
A 70-year-old man with a history of TAVR in 2020 presented with CHF symptoms. Initial workup revealed severe aortic stenosis and CHF, leading to diuresis. On hospital day 2, his condition had worsened, prompting an upgrade to the CCU due to signs of cardiogenic shock (EF 18%) and a TTE notable for AV mean gradient of 35 mmHg, with 2+ paravalvular leak. A CTA indicated commissural misalignment, increasing the risk for coronary obstruction post-TAVR, which led to consideration of either valve-in-valve TAVR versus open surgical management. Patients condition continued to decline, on hospital day 18 following RHC, an IABP was placed, with subsequent transition to an Impella. Ultimately after multidisciplinary discussion the consensus was that medical management alone would not suffice for survival prompting the decision to proceed with the SURPLUS procedure on hospital day 22. Following new valve implantation, the Impella was readvanced and the patient was separated from CPB on VA ECMO. The Impella was removed on POD 2 and that patient was decannulated from VA ECMO on POD 10.

Discussion

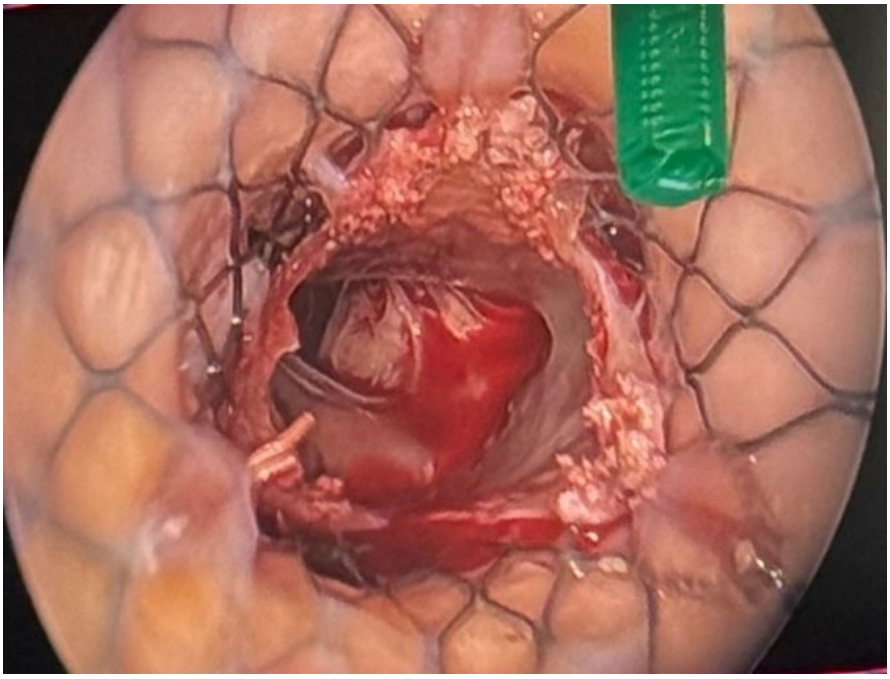
As TAVR use expands into younger and lower risk populations, the need for reintervention is expected to increase. Current data shows that overall reintervention rates remain low, approximately 1% with self-expanding valves demonstrating a slightly higher rate (1.19%). When reintervention is required, the underlying etiologies typically include structural valve deterioration, new or worsening aortic insufficiency, paravalvular leak, structural complications, or infective endocarditis. Despite the feasibility of redo valve in valve TAVR, not all patients are suitable candidates. Contraindications include unfavorable aortic root anatomy, the need for concomitant cardiac surgery, low coronary ostial height with risk of obstruction, and inadequate annular dimensions. When coronary height or potential obstruction is a concern, both endovascular and open surgical options must be carefully evaluated. In this patient's case, CTA demonstrated significant commissural malalignment, placing them at high risk for coronary obstruction even with endovascular protection techniques such as BASILICA or UNICORN. Given this unfavorable anatomy, it was determined that a valve-in-valve approach using the SURPLUS procedure offered a safer strategy. Other contraindications to endovascular techniques include active endocarditis, severe leaflet rigidity and unfavorable anatomy. A full TAVR explant was considered but carried significant risk in this patient. These procedures are typically prolonged, may require aortic root replacement with coronary

reimplantation, and are associated with significant perioperative risk with reported 30-day mortality ranging from 15-20%. SURPLUS, while not appropriate in cases of endocarditis or severe patient prosthesis mismatch, provided an optimal balance of safety and efficacy for this patient's complex condition.

Images



intra-op: pathologic TAVR leaflets pre-excision



intra-op: pathologic TAVR post-leaflet excision

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#FR 2 – Kiosk 23 – Poster 1

Descending Thoracic Aortic Aneurysm causing Left Main Bronchus Obstruction

Presenting Author: Sean B. Noormohamed Lee, MD, Cardiothoracic Anesthesiology Fellow, USC Keck School of Medicine

Introduction

While thoracic aortic aneurysms are known to cause compression of surrounding structures, compression of the upper airway is rare, presenting with dyspnea, hemoptysis, coughing, or wheezing (1). Such cases require a collaborative coordination of care between anesthesiologists, surgeons, and intensivists to execute effective treatment strategies. We present a case that required a multidisciplinary, staged surgical approach to treat a large, complex descending thoracic aortic aneurysm with Debakey Type IIIb aortic dissection, complicated by aberrant patient anatomy and upper airway compression.

Case Presentation

A 40 year old male with a past medical history of hypertension presented to a pulmonologist complaining of an intermittent cough lasting for 4 months, chest pain, lateral back pain, and shortness of breath. Chest x-ray illustrated complete opacification of the left hemithorax (Figure 1). Chest CT demonstrated a Type IIIb aortic dissection extending from the distal left subclavian artery to the right common iliac artery. The descending thoracic aortic aneurysm with intramural hematoma was noted to be obstructing the left main bronchus, measuring 12.1cm in diameter (Figure 2). Additionally, CT imaging revealed an anomalous origin of the right subclavian artery, arising directly from the aorta and distal to the left subclavian, coursing behind the esophagus. The right common carotid, left carotid, and left subclavian arteries were normal in course and caliber. The patient was subsequently intubated due to acute hypoxic respiratory failure secondary to aspiration while undergoing further diagnostic evaluation. He was taken to the OR for TEVAR of Zone 2-5 with concurrent right carotid and right subclavian artery transposition. The aberrant right carotid was anastomosed to the right subclavian, creating an “innominate” artery. Days later, he returned to the operating room for a total arch replacement with frozen elephant trunk, during which a left subclavian to left carotid transposition was performed. The surgical team elected to delay chest closure at the end of the procedure secondary to continued coagulopathy, as well as cardiac edema and concern for cardiac compression if chest closure was attempted. One week later, he returned to the OR for descending thoracic aortic aneurysm repair via thoracoabdominal incisional approach and femoral ECMO cannulation. The anesthesia team attempted to place a left 41F DLT but was unsuccessful due to the compression of the left main stem bronchus by the aneurysm. It was believed that the left lung and left mainstem bronchus were chronically collapsed due to external compression from the aneurysm. Placement of a right 39F DLT was subsequently attempted with successful placement. After aneurysm decompression and endoleak repair, the left lung was successfully reinflated under direct surgical visualization.

Discussion

Mass effect of a thoracic aortic aneurysm may cause upper airway obstruction, leading to respiratory

symptoms. Treatment options, such as endobronchial stenting and endovascular or open repair of the aneurysm, vary (2). Though our patient's case was complicated by aberrant anatomy, the aneurysm was decompressed and upper airway obstruction relieved through a multidisciplinary, staged procedure.

Images

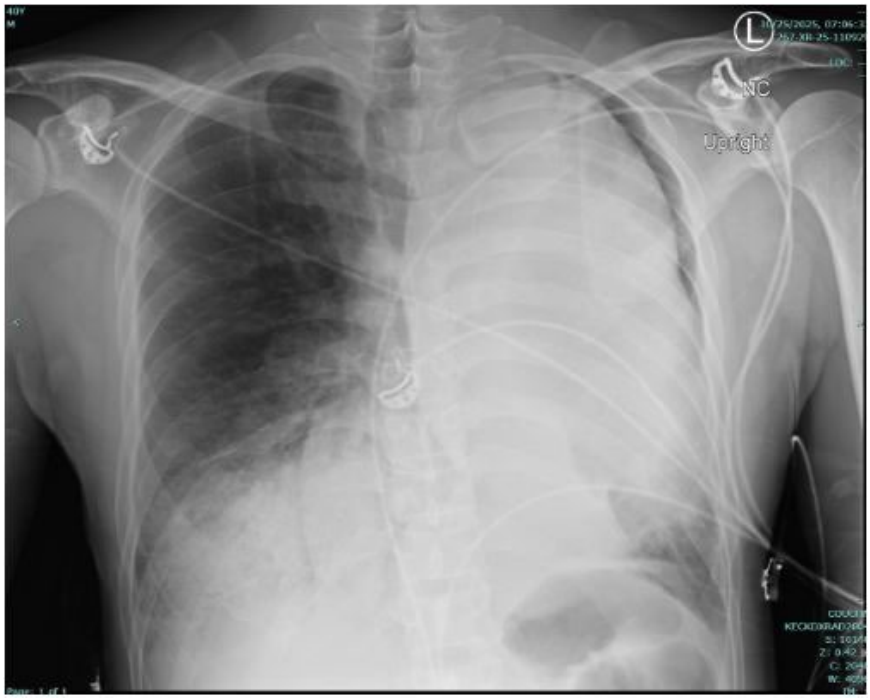


Figure 1. Chest X ray demonstrating complete opacification of left hemithorax.



Figure 2. CT Chest demonstrating 12.1cm descending thoracic aortic aneurysm causing left main bronchus obstruction.

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#FR 2 – Kiosk 23 – Poster 2

Transient Mitral Regurgitation After TAVR: A Case Report

Presenting Author: Natasha Singh, MD, Resident, University of Rochester

Introduction

Transcatheter aortic valve replacement (TAVR) has become a widely used alternative to surgical aortic valve replacement (SAVR), especially in older and at-risk surgical populations with severe aortic diseases. While TAVR and SAVR both provide significant reduction in mortality, each carries potential complications. For TAVR, the most common complication is the need for permanent pacemaker placement; other risks include bleeding, acute kidney injury, and stroke. We present a case of TAVR complicated by complete heart block, paravalvular leak, and acute severe mitral regurgitation (MR), the latter of which resolved spontaneously.

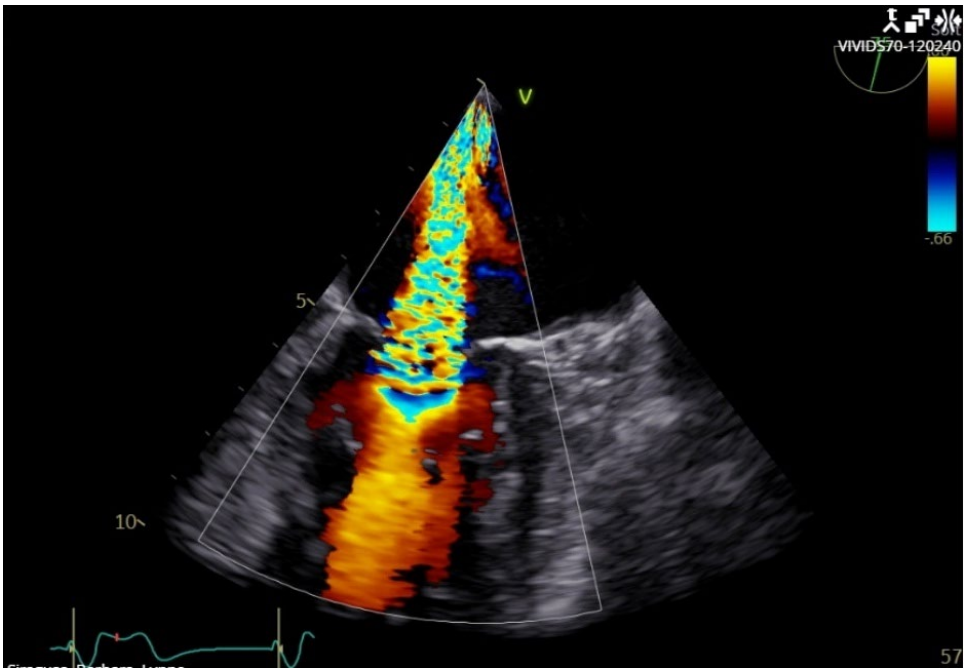
Case Presentation

81yo female with history of moderate to severe aortic stenosis and moderate to severe aortic valve insufficiency (AVA 0.7cm^2, AV mean gradient 31 mmHg, peak gradient 54 mmHg), HOCM s/p alcohol septal ablation 12/2024 with improved LVOT gradient but with persistent heart failure symptoms, COPD, and DM2 who presented for TAVI. Valve deployment of a Medtronic Evolut FX+ 23 via transfemoral approach was performed under moderate sedation with propofol and remifentanil. Immediately following the valve deployment transthoracic echocardiogram was performed that showed severe mitral valve regurgitation and paravalvular leak at twelve o'clock position and hyperdynamic LV. Patient had increased oxygen requirement and was subsequently intubated. 100mg of furosemide was administered for pulmonary edema. Despite the intubation and Intra-aortic balloon pump placement, she continued to struggle with oxygenation with low saturation, a decision was made to place the patient on VV ECMO prior to transferring the patient to CVICU with a temporary RV pacemaker for complete heart block. On repeat TEE 4 hours after, the patient was found to have spontaneous improvement in valvular function (AV mean gradient 12 mmHg, peak gradient 31 mmHg, MVR from severe to mild-moderate); with decreased paravalvular leak. The patient was extubated and IABP was removed on post-operative day one. Other than initial supportive measures described above, the patient did not require any additional intervention for the resolution of the valvular dysfunction.

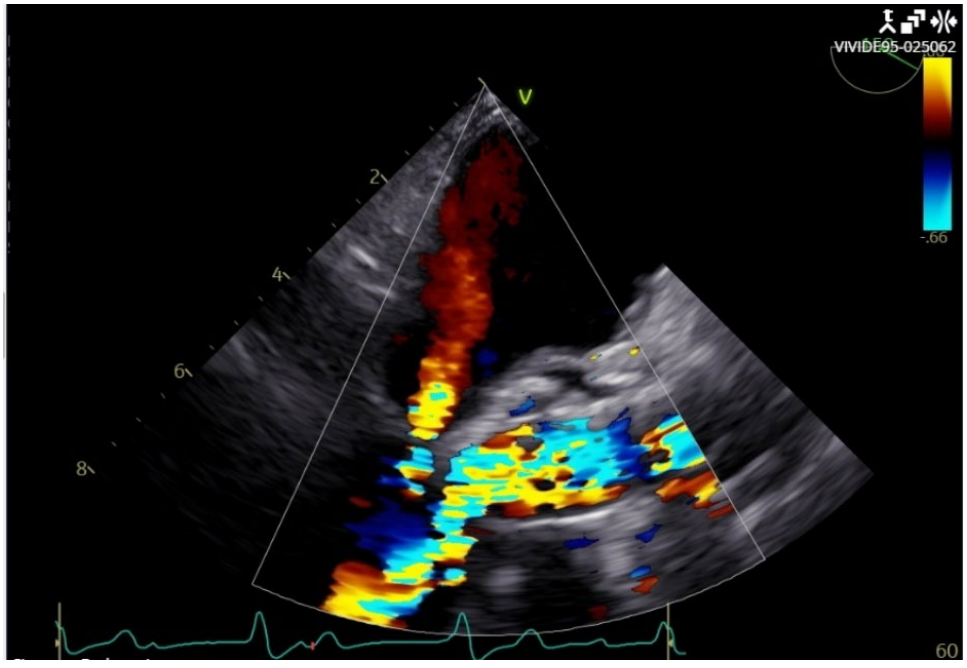
Discussion

Initial thoughts on the etiology of the mitral valve dysfunction included TAVR valve frame pressing on the calcified mitral annulus that resulted in an alteration of the anterior leaflet geometry; subsequently resulting in lack of coaptation leading to severe mitral valve regurgitation. Given the spontaneous resolution, however, the cause of dysfunction was attributed to transient papillary muscle ischemia after rapid ventricular pacing. This case highlights the importance of recognizing possible compilcations following TAVR, including transient acute mitral valve regurgitation and the role of supportive management while underlying mechanisms resolve.

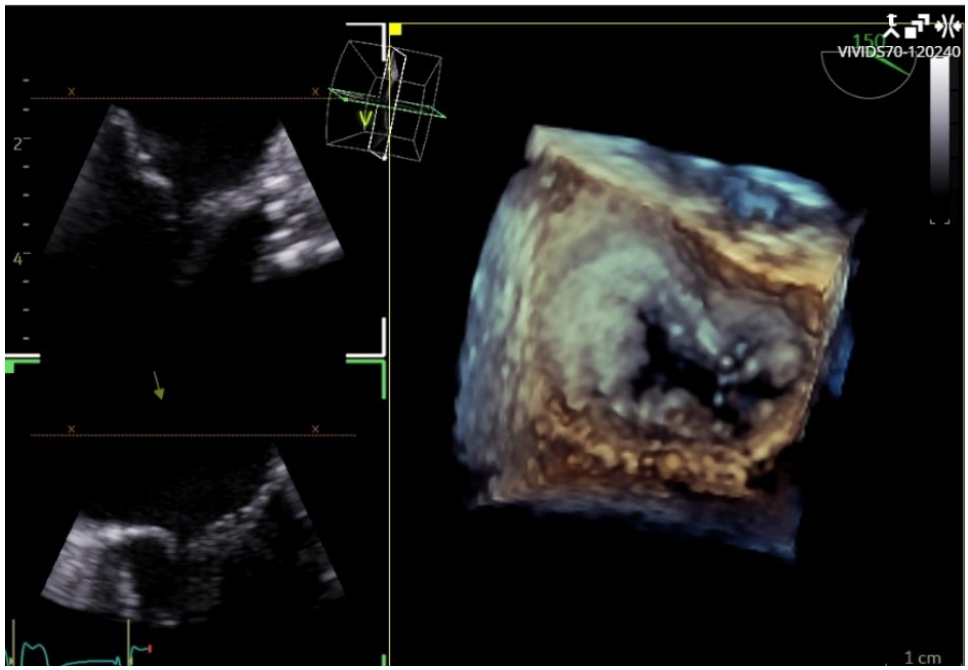
Images



Severe MR in systole



Paravalvular leak and MR captured in ME LAX view



3D TEE imaging of non-coaptation of mitral valve leaflets

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#FR 2 – Kiosk 23 – Poster 3

A Rare Case of Dual Outflow Stenosis Requiring Emergent Double-Valve Replacement

Presenting Author: Miles McNeir, D.O., Fellow, University of Nebraska Medical Center

Introduction

Combined severe aortic stenosis (AS) and pulmonic stenosis (PS) in adults is exceedingly rare and poses unique diagnostic and perioperative management challenges. Simultaneous left and right-sided fixed obstructions create preload-dependent physiology, limited cardiac reserve, and complex responses to anesthetic agents and cardiopulmonary bypass. Anesthetic management becomes more challenging when superimposed on severe biventricular failure and acute decompensation. We describe the perioperative course of a 25-year-old who presented critically ill with severe AS, PS, severe pulmonary hypertension and biventricular failure in cardiogenic shock requiring emergent dual-valve replacement.

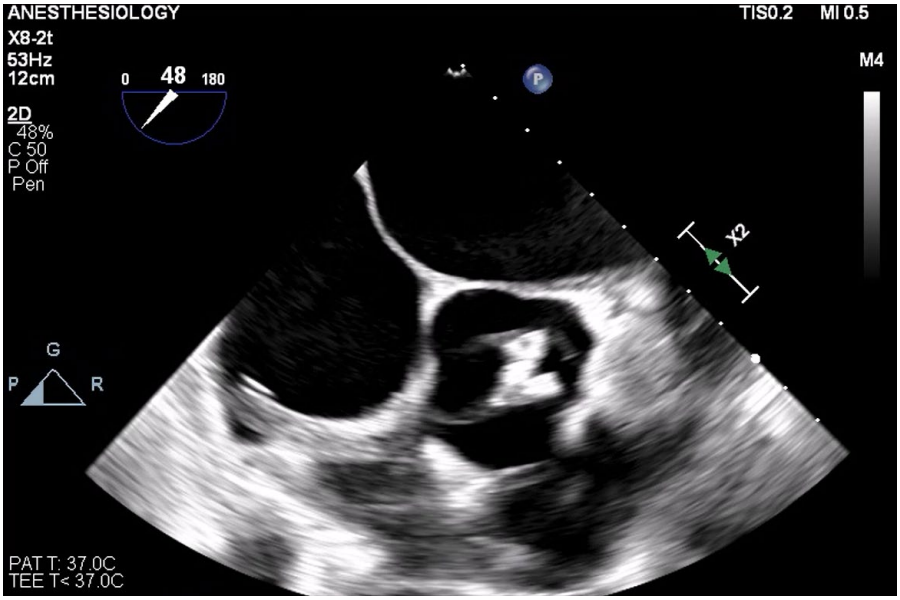
Case Presentation

A 25-year-old Spanish-speaking male from Guatemala with limited medical history presented with several days of upper respiratory symptoms. He became progressively hypoxic in the ED, requiring emergent intubation. CT chest revealed aortic valvular calcifications and a 4.4-cm ascending aortic ectasia. Transthoracic echocardiography demonstrated severe AS, severe PS, aortic root aneurysm, severe pulmonary hypertension, and severely reduced biventricular systolic function. Given rapid deterioration, he was taken emergently to the OR for combined aortic valve replacement with root enlargement and pulmonic valve replacement. With fixed biventricular outflow obstruction and minimal contractile reserve, anesthetic goals included maintaining sinus rhythm, avoiding reductions in systemic vascular resistance, and supporting biventricular filling pressures. Invasive monitoring included radial artery and central venous access. Induction used isoflurane and high-dose opioids to minimize hemodynamic instability. Perioperative TEE: Severely calcified unicuspid aortic valve; small, concentrically hypertrophied LV with EF 20–25%; dysplastic pulmonic valve with high-velocity jet and peak gradient consistent with moderate stenosis. Surgical intervention consisted of aortic valve replacement with root enlargement and pulmonic valve replacement under cardiopulmonary bypass. Despite his age, bioprosthetic valves were selected to avoid anticoagulation and maintain eligibility for future mechanical support in the context of systolic heart failure. The patient separated from bypass on epinephrine and inhaled pulmonary vasodilators and was transferred to the cardiovascular ICU. He improved rapidly, was extubated on postoperative day 1, and weaned off epinephrine within 24 hours.

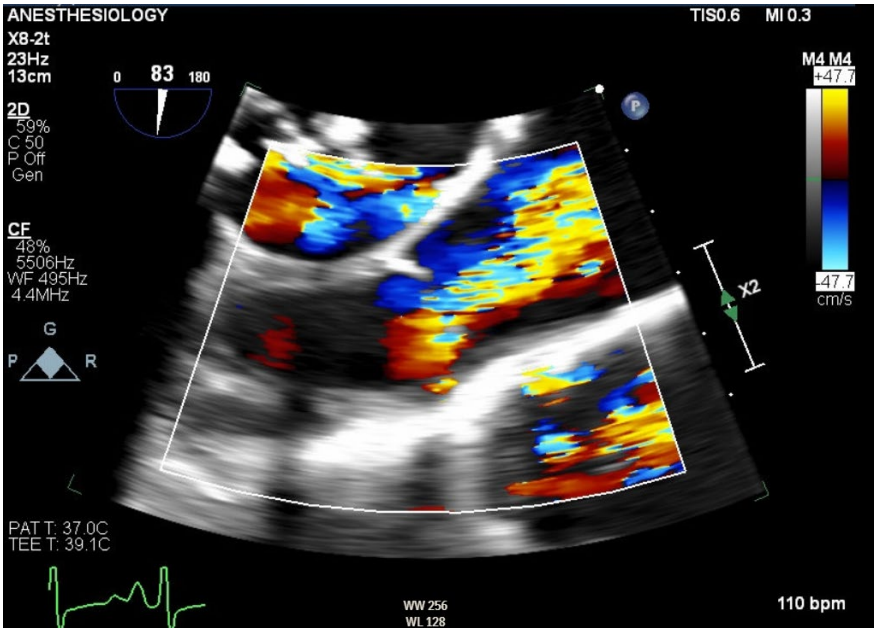
Discussion

Simultaneous AS and PS in a young adult is uncommon. Differential diagnoses include congenital syndromic disorders (Williams syndrome and Noonan syndrome), congenital non-syndromic disease (dysplastic aortic and pulmonic valves, bicuspid aortic valve with coexisting congenital PS), rheumatic disease (rarely involves pulmonic valve), carcinoid heart disease (typically right sided but can mimic multivalvular obstruction), or sequelae of prior endocarditis. The combined presence of dual stenotic lesions creates a unique physiology with simultaneous left and right-sided fixed obstruction, severely limiting the ability to augment cardiac output and increasing peri-induction risk. This case highlights the critical role of comprehensive TEE, meticulous hemodynamic management, and coordinated surgical decision-making.

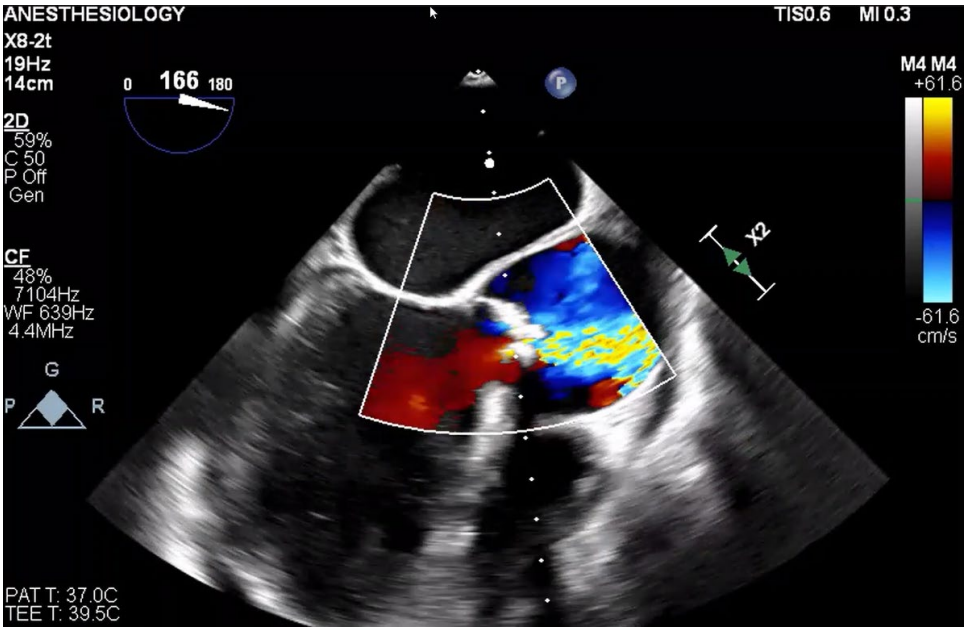
Images



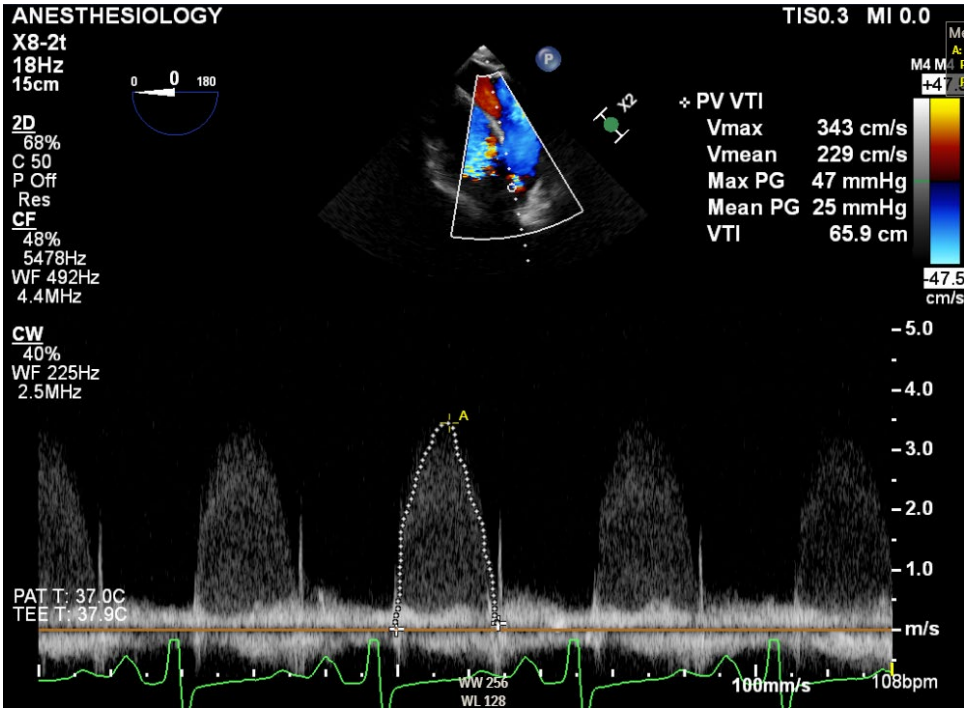
Mid-esophageal aortic valve short axis view demonstrating a unicuspid aortic valve.



Pulmonic valve focused mid-esophageal right ventricular inflow-outflow view with color flow doppler demonstrating significant stenosis of pulmonic valve.



Mid-esophageal aortic valve long axis view with color flow doppler demonstrating significant stenosis of aortic valve.



Continuous wave doppler tracing of pulmonic valve outflow with a max pressure gradient measuring 47 mmHg.

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#FR 2 – Kiosk 23 – Poster 5

Intraoperative Thrombus During Watchman Implantation

Presenting Author: Timothy Waite, DO, Fellow, Indiana University School of Medicine Department of Anesthesiology

Introduction

In patients with atrial fibrillation, the watchman device is commonly used to mitigate the risk of stroke when systemic anticoagulation is contraindicated. Before implantation, absence of thrombus is a requirement as the presence of pre-existing clot can drastically increase the risk of embolization during catheter manipulation and device deployment. For this reason, transesophageal echocardiography (TEE) is commonly used to evaluate for signs of exiting thrombus. Typically, the procedure will be aborted if clot is suspected. This case describes a patient with atrial fibrillation and an intolerance to anticoagulation whose initial echocardiogram showed no signs of thrombus. After transseptal puncture, a large mobile thrombus was discovered extending into the left ventricular outflow tract necessitating further intervention.

Case Presentation

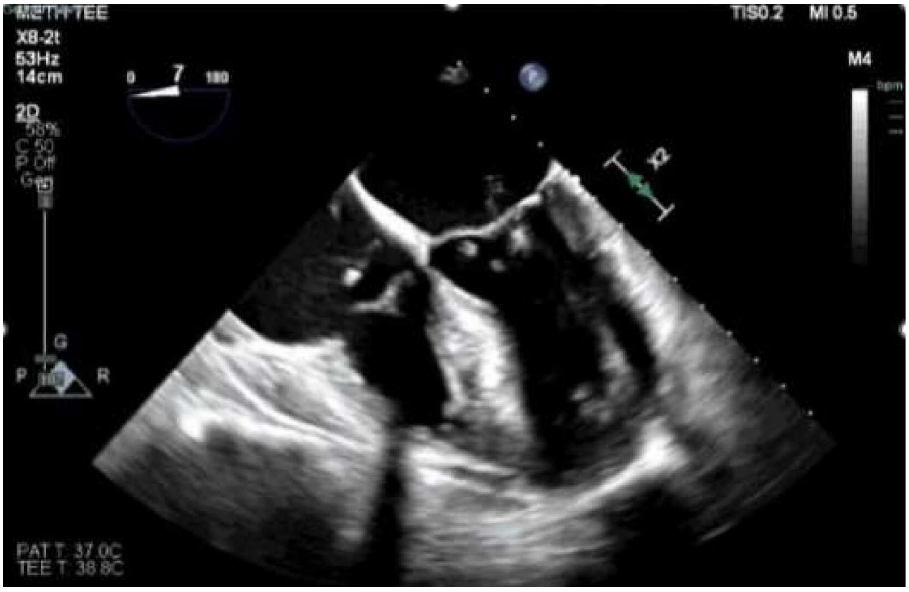
An 81 year old female presented for Watchman device placement after intolerance to oral anticoagulation due to genitourinary bleeding. The initial transesophageal echocardiogram showed a normal ejection fraction and was notable for an absence of clot in the left atrial appendage. After transseptal puncture was performed re-evaluation on TEE was significant for large mobile thrombus extending from the Inferior Vena Cava, through the transseptal puncture, and into the left ventricular outflow tract. At this time, the watchman was aborted and there was a multidisciplinary discussion on how to proceed with management of the new clot. The decision was made to utilize a Sentinel device to attempt to mitigate risk of clot embolization to the brain. After Sentinel deployment via radial artery, the team proceeded with AngioVac Thrombectomy for clot aspiration under TEE guidance. After successful removal of all left-sided clot, the patient was successfully extubated and transferred to the Intensive Care Unit on a heparin drip. There were no post-procedural neurological changes and a negative head CT. Patient subsequently underwent successful watchman device procedure 6 weeks later with no residual clot noted on TEE.

Discussion

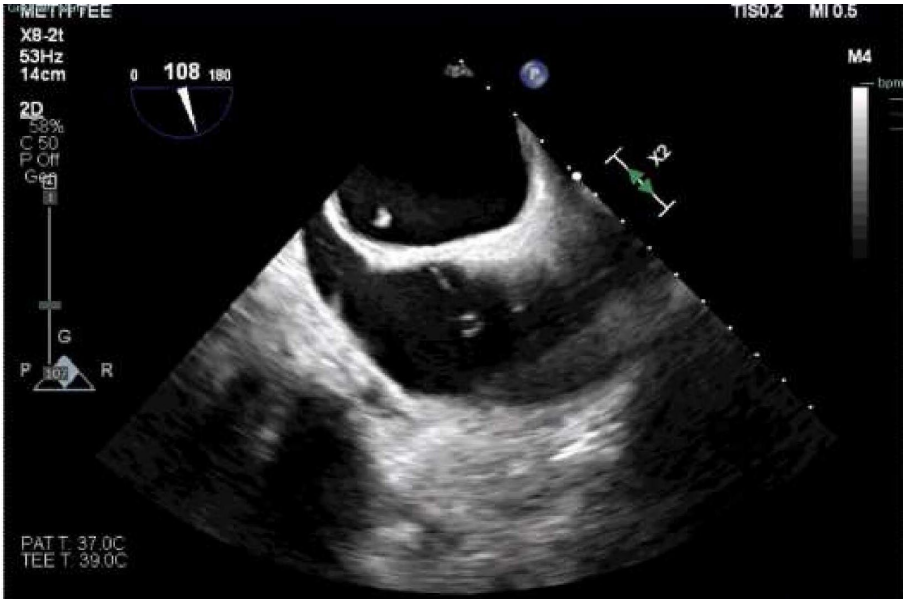
Device related thrombosis is associated with an increased risk of stroke or systemic embolism. In a registry study, nearly 80% of patients had thrombus resolution but patients' thrombi that did not resolve had higher rates of ischemic stroke and mortality over two years.1 Current guidelines address presence of thrombus on pre-procedural imaging but do not provide guidance on intraoperative thrombus discovery. A single center retrospective study examining watchman complications found 3 instances of intraoperative thrombus discovery, all of which were managed with targeted thrombolysis and cerebral protection device deployment. 2 Given the wide-spread clot burden and concern for embolization, AngioVac was thought to be a more viable alternative to targeted thrombolysis in this patient. This case provides a valuable example of the importance of intraoperative TEE for continued evaluation during watchman placement and the

necessity of multidisciplinary communication and management intraoperatively.

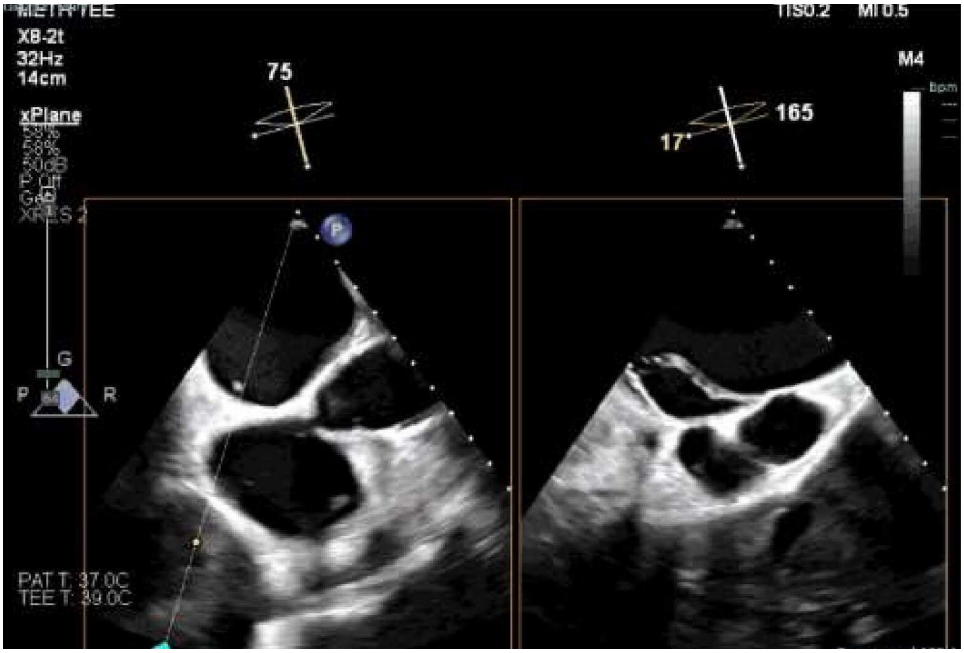
Images



Transesophageal Echocardiogram of Thrombus in RA, LA, LV



Transesophageal Echocardiogram of thrombus extending across the interatrial septum



Transesophageal Echocardiogram showing an x-plane view of the thrombus in the left atrium



Intraprocedural imaging of AngioVac performed in right atrium

References

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#FR 2 – Kiosk 23 – Poster 6

Anesthetic Management of Late Extrinsic Outflow Graft Obstruction in a HeartMate 3 Left Ventricular Assist Device Recipient

Presenting Author: Lawrence H. Dautel, IV, M.D., Cardiothoracic Anesthesiology Fellow, University of Nebraska Medical Center

Introduction

Extrinsic outflow graft obstruction at the bend relief (EOGO-BR) is a late complication of the HeartMate 3 (HM3) left ventricular assist device (LVAD), characterized by accumulation of acellular biodebris between the outflow graft (OG) and its polyethylene terephthalate bend relief (BR). This insidious process eventually leads to graft compression, reduced pump flows, and low-flow alarms, with an estimated incidence of 2.06% at 5 years post-implant. Clinical presentation includes heart failure symptoms, ventricular arrhythmias, or hemodynamic instability. Diagnosis involves echocardiography, CT angiography, and right heart catheterization. Management may require surgical revision or percutaneous intervention, with perioperative challenges including ongoing low-flow alarms and fluid management. We present a case of EOGO-BR revision 6 years post-HM3 implantation, highlighting anesthetic considerations.

Case Presentation

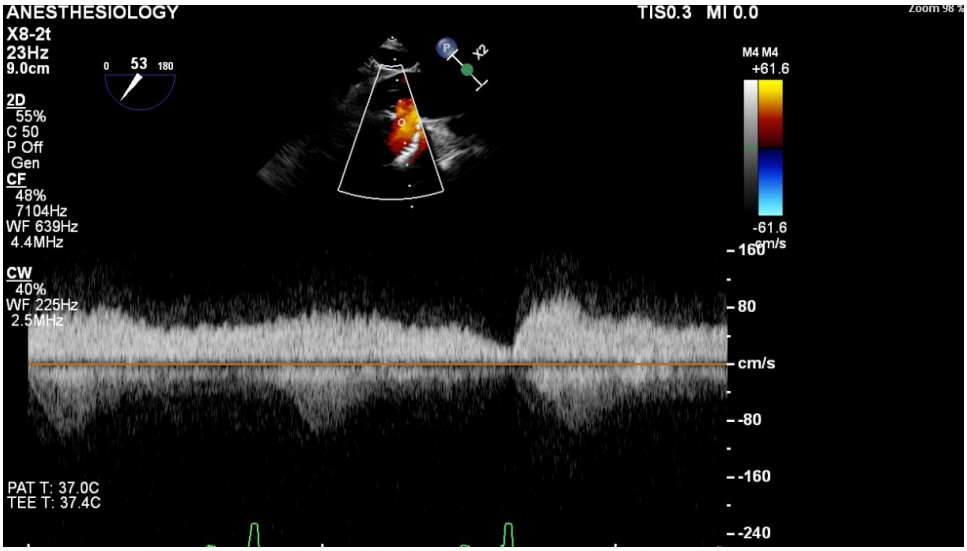
A 35-year-old female with peripartum dilated cardiomyopathy underwent HM3 LVAD implantation in November 2019 as destination therapy due to morbid obesity precluding transplant eligibility. She achieved significant weight loss following sleeve gastrectomy in 2022, becoming transplant-eligible. In May 2025, she presented with low-flow alarms and was admitted. Diagnostic evaluation confirmed EOGO-BR; LVAD speed was increased, and intravenous fluids (IVF) administered, resolving alarms. Recurrent asymptomatic low flows occurred in September 2025. In October 2025, continuous alarms prompted readmission. She underwent left thoracotomy on October 17, 2025, with OG revision and removal of compressive gelatinous biodebris. Anesthetic management included preoperative radial arterial line placement and a central venous catheter for potential escalation. Transesophageal echocardiography (TEE) was employed intraoperatively. Induction exacerbated low-flow alarms, prompting empirical IVF administration (1.3 L administered over 2.5 hours) to optimize preload. Hemodynamics remained stable with minimal vasopressor boluses (no continuous infusions required). For postoperative analgesia, no regional block was performed as the surgeon infiltrated the left thoracotomy incision with local anesthetic. The patient tolerated the procedure well without complications.

Discussion

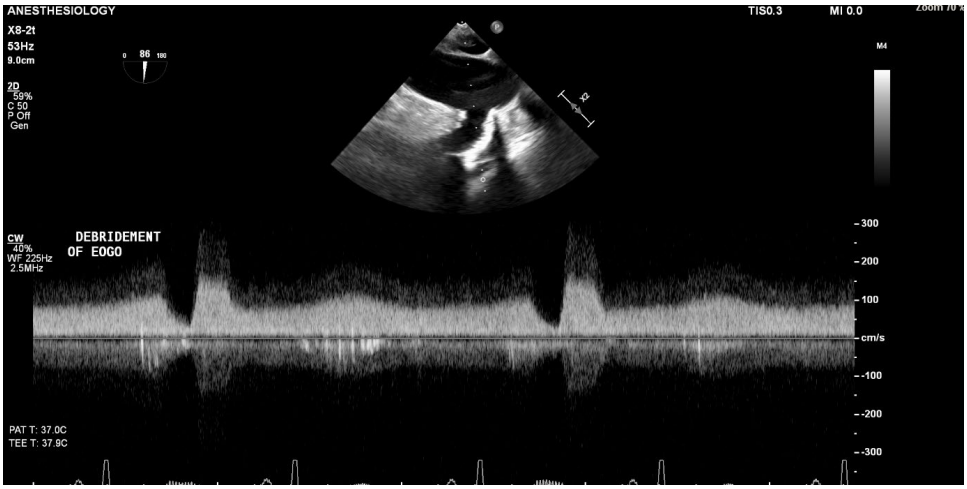
This case underscores perioperative challenges in managing EOGO-BR revision, including persistent low-flow alarms exacerbated by induction of general anesthesia due to graft compression and potential hypovolemia. Decision-making focused on aggressive preload optimization with IVFs to counteract reduced LVAD output, optimization of RV function guided by TEE, while minimizing vasopressors to achieve afterload reduction. TEE is also useful for real-time assessment of OG velocities. Novelty lies in the late presentation

(6 years post-implant, exceeding the typical 2.6-year mean onset) and the patient's transition from destination therapy to bridge-to-transplant following bariatric surgery-induced weight loss. This highlights the importance of long-term surveillance in HM3 recipients and adaptive anesthetic strategies for thoracotomy approaches, such as preemptive regional analgesia. Preventive measures, including OG wrapping modifications, may reduce future EOGO-BR incidence.

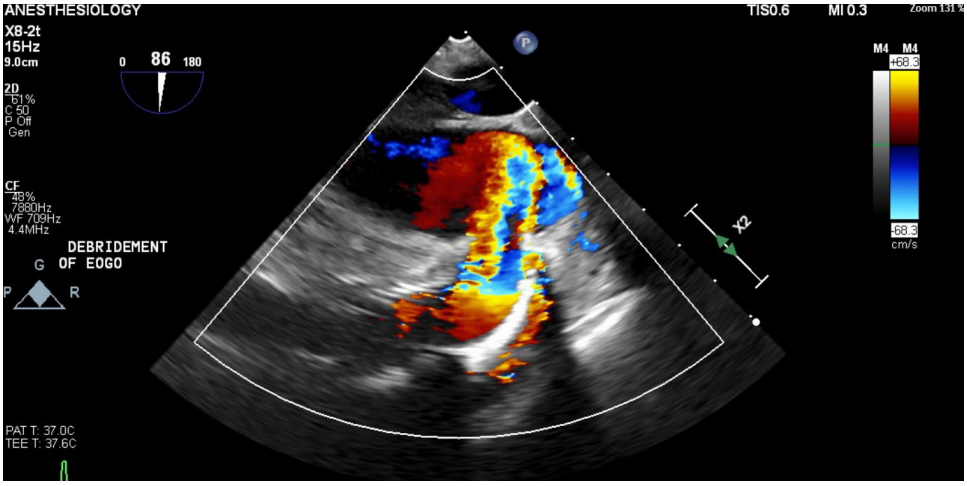
Images



TEE CWD of pre-debridement HM3 outflow graft.



TEE CWD of post-debridement HM3 outflow graft.



TEE CFD of post-debridement HM3 outflow graft.



Accumulation of acellular biodebris between the HM3 outflow graft which was debrided.

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#FR 2 – Kiosk 23 – Poster 7

Lucid Fibrillation: Fulminant Myocarditis Complicated by Persistent Ventricular Fibrillation Requiring Biventricular Assist Device

Presenting Author: Emily Eruysal, MD, NewYork-Presbyterian/Weill Cornell Medicine

Introduction

Myocarditis is the underlying cause of undifferentiated dilated cardiomyopathy in up to 10% of cases. Presenting as a constellation of nonspecific symptoms and cardiac complaints, severe cases of myocarditis may evolve into acute decompensated heart failure and cardiogenic shock, necessitating escalation in treatment with inotropes and even mechanical circulatory support. We present a case of fulminant myocarditis requiring cannulation onto venous-arterial extracorporeal membrane oxygenation (VA ECMO) and, ultimately, biventricular assist device (BiVAD).

Case Presentation

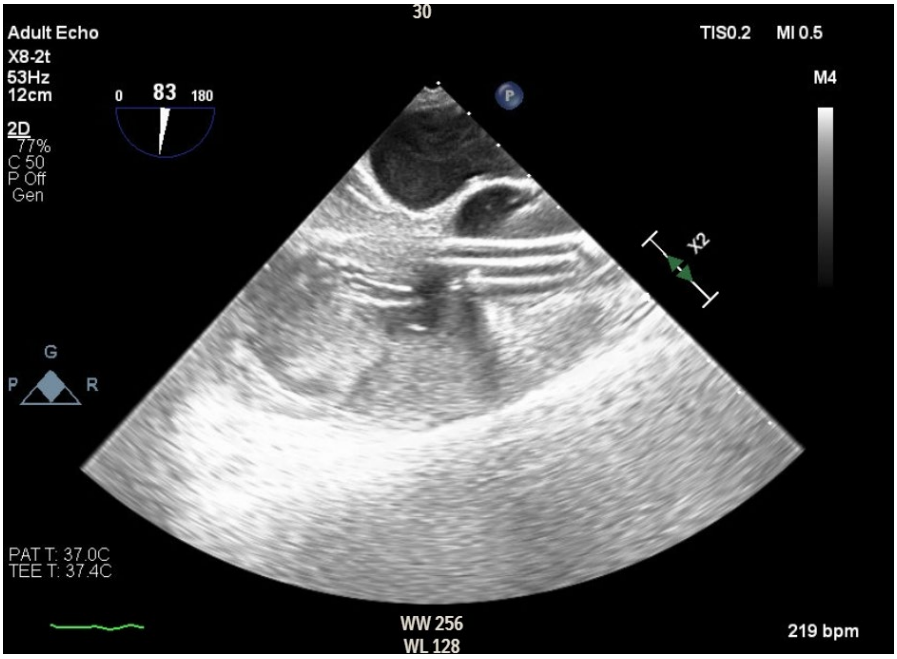
A 45-year-old male with no medical history presented with hypoxemia after three days of chills, fatigue, and chest pain. ECG showed ST changes concerning for acute coronary syndrome. He suddenly went into refractory ventricular tachycardia (VT) decompensating to ventricular fibrillation (VF) despite aggressive therapy. He was emergently intubated, cannulated for peripheral VA ECMO, and taken to the cardiac catheterization lab for coronary intervention. Catheterization demonstrated non-obstructive coronary arteries. An Impella device was placed to decompress the near static left ventricle (LV). The patient remained in persistent VF for 8 hours on ECMO flows of 3.5 L/min with Impella support, neurologically intact off sedation. The decision was made to proceed with CentriMag BiVAD to decompress the right ventricle (RV) and to bridge this patient to further treatment or myocardial recovery. Intraoperative TEE showed stasis of flow, with clot in formation in the RV and pulmonary artery (PA) and formed clot in the RV outflow tract (RVOT). Cardiopulmonary bypass was initiated, and shortly after the rhythm became organized. Prior to cannulation of the PA for RVAD, the PA and RVOT were inspected for clot. Prior devices were removed and BiVAD placed. Due to concern that the RV would not adequately decompress despite BiVAD placement, milrinone, dobutamine, and inhaled nitric oxide were added for weaning CPB to BiVAD support. An oxygenator was spliced into the RVAD outflow limb due to relative hypoxemia. Postoperatively, after diuresis on biventricular support, the oxygenator was removed on post-operative day (POD) 5, and the patient was extubated POD 6. Cardiac function with TEE demonstrating normal biventricular function on turndown of the

BiVAD, allowing for removal on POD10. The patient was discharged after 3.5 weeks with an EF of 50%.

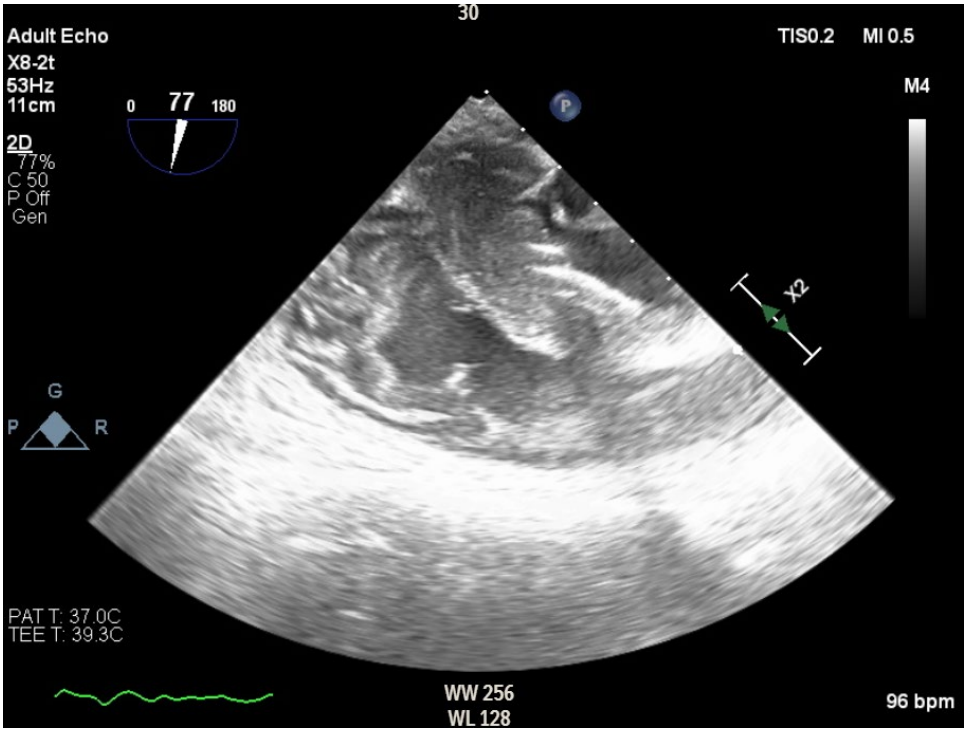
Discussion

Though outcomes remain poor for patients requiring BiVAD, BiVADs can be a bridge to heart transplant, destination therapy, or myocardial recovery. These patients tend to present with worse critical illness than those requiring LVAD alone and are at higher risk for morbidity and mortality. However, these concerns should not preclude timely institution of BiVAD support when appropriate. Our patient was an excellent candidate for BiVAD – he was previously young and healthy, and despite his acute presentation, he had evaded neurologic injury. Fortunately, he recovered his cardiac function quickly. Had he not, the BiVAD would have afforded him the ability to ambulate and otherwise rehabilitate before transplant or durable LVAD.

Images



Pre-BiVAD, RV/RVOT stasis/clot, RV inflow-outflow



Pre-BiVAD, RV stasis/clot

References

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#FR 2 – Kiosk 24 – Poster 1

Infundibular Pulmonary Stenosis Masking Primary Valvular Pulmonary Stenosis

Presenting Author: Shourik Dutta, MD, Fellow Physician, University of Alabama at Birmingham

Introduction

Pulmonary stenosis is a common congenital heart defect that involves a variable degree of narrowing of the right ventricular outflow tract (RVOT), pulmonary valve (PV), or pulmonary arteries. Stenosis can often occur proximal to the pulmonary valve, in the infundibular region of the right ventricle. This often occurs in patients with double chambered right ventricle or those with tetralogy of Fallot. Compensatory hypertrophy of the right ventricle (RV) over time can exacerbate the degree of stenosis[1]. This report details a case of infundibular stenosis masking primary pulmonary valve stenosis.

Case Presentation

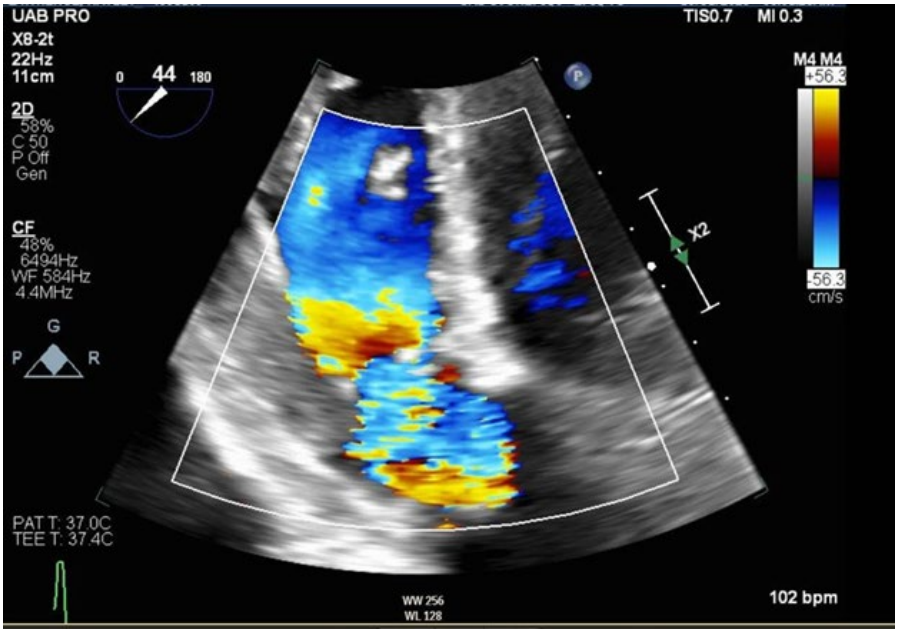
A 24 year-old female presented for elective infundibular resection for subvalvular pulmonary stenosis. Her past medical history included morbid obesity, severe pulmonary valve stenosis, moderate-severe RV hypertrophy and previous pulmonary valve valvuloplasty. Pre-operative transesophageal echocardiography (TEE) exam revealed several key findings, including: severely hypertrophied, double-chambered right ventricle, a well-opening pulmonary valve with thickened leaflets, and an obstructive infundibular muscle bridge resulting in severe RVOT obstruction. Mean and peak pressure gradient measured across the infundiblar muscle bridge were 51 and 96 mmHg, respectively. After resection and an uneventful wean off bypass, laminar flow was appreciated in the subvalvular region without any ventricular defects. While the valve opened well, turbulent flow remained on color flow doppler across the annulus and distal to the valve. Post-interventional mean pressure gradient ranged from 9-22 mmHg, indicating residual valvular stenosis. After further monitoring on TEE, a multi-disciplinary decision was made to defer valve replacement in favor of monitoring clinical symptom improvement while initiating beta-blocker therapy.

Discussion

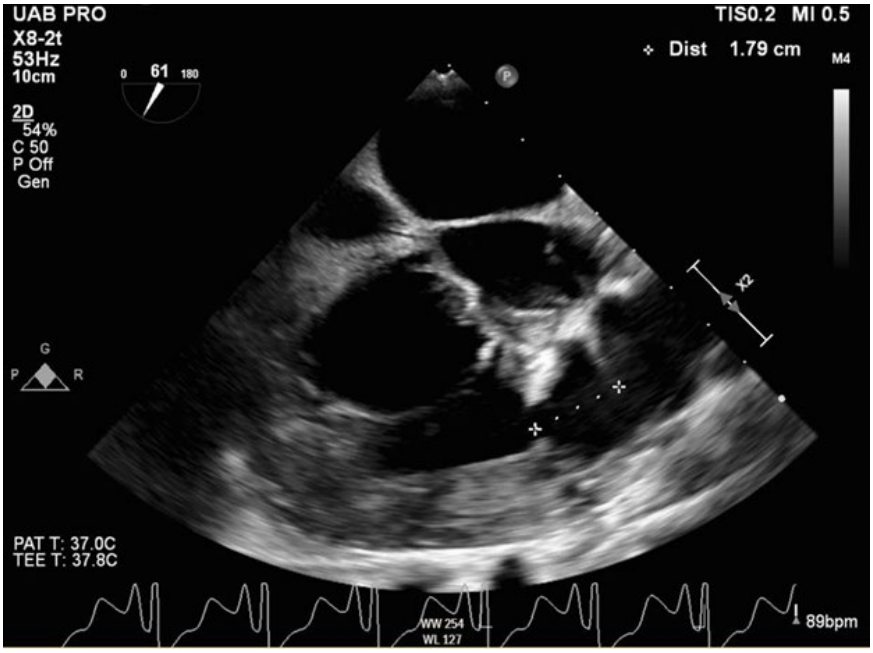
Infundibular pulmonary stenosis refers to obstruction in the RVOT proximal to the pulmonary valve, creating a minimum peak pressure gradient of at least 36 mmHg[2]. Key echocardiography considerations in these scenarios include an assessment of the degree of RV hypertrophy, thickness of infundibular muscular ridge, proximity of infundibular obstruction from PV annulus, PV structure and function, and degree of physiologic stenosis through the area. The present case highlights an additional, unique phenomena of subvalvular stenosis masking primary pulmonary valve stenosis. Although our patient's pulmonary valve opened well, had previously undergone valvuloplasty, and appeared adequately functional in the surgical field, post-interventional assessment revealed residual, mild PV stenosis. The decision to pursue further surgical intervention is made in a multidisciplinary fashion, guided by real-time echocardiography assessment of the PV and the patient's baseline clinical symptom presentation. This case underscores the importance of carefully assessing and identifying drivers of pulmonary stenosis. Moreover, it emphasizes the need for awareness that subvalvular stenosis may mask primary valve stenosis, which can complicate real-time

clinical decision-making.

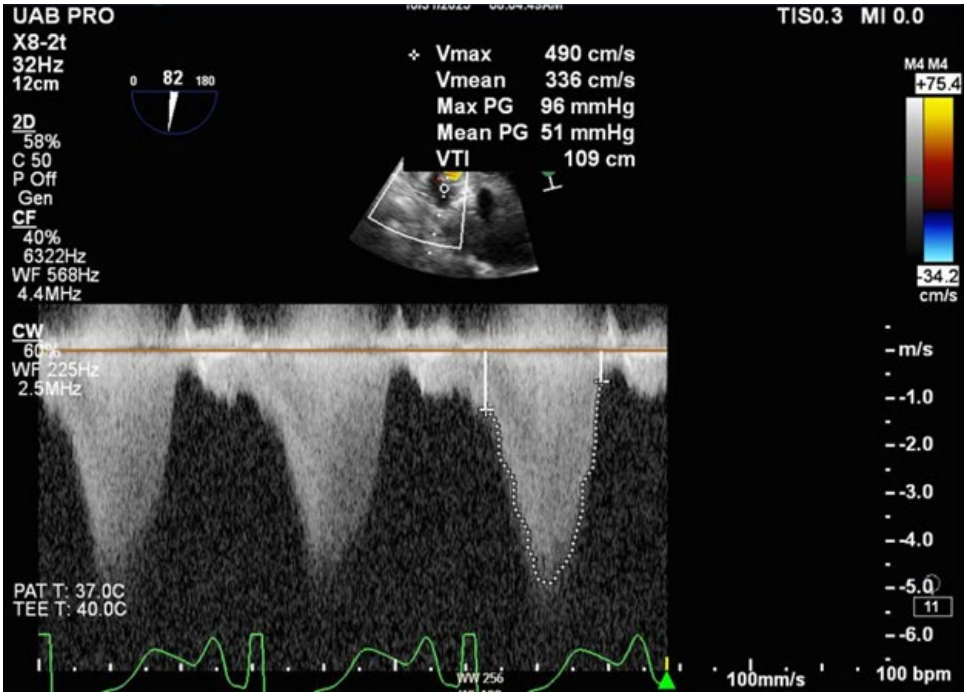
Images



Transgastric view of right ventricular outflow tract, showing Infundibular stenosis on color flow doppler proximal to pulmonary Valve.



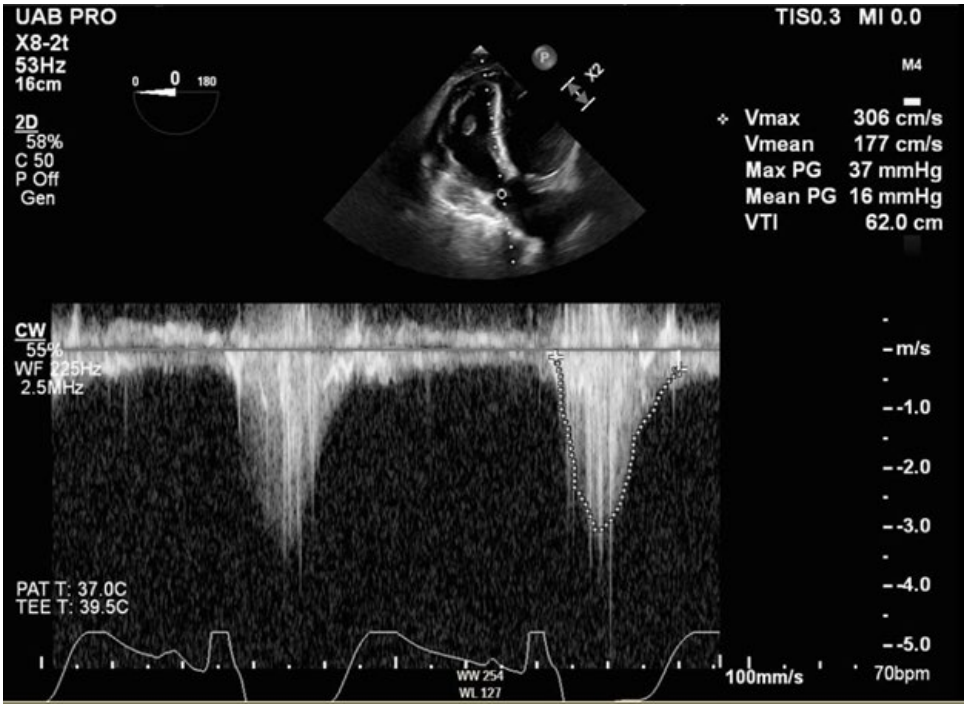
Midesophageal right ventricular outlfow tract view showing double-barrel right ventricle



Continuous wave doppler through right ventricular outflow tract, showing elevated peak and mean pressure gradient through subvalvular area.



Post-operative image of midesophageal right ventricular outflow tract, showing laminar flow proximal to pulmonary valve, but residual turbulent flow at pulmonary valve.



Post-operative image of continuous wave doppler through right ventricular outflow tract, showing residual elevated peak and mean pressures consistent with mild pulmonary stenosis.

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#FR 2 – Kiosk 24 – Poster 2

Gone in a Heartbeat: A Disappearing Cardiac Mass

Presenting Author: Rita S. Asuquo, MD, Adult Cardiothoracic Anesthesiology Fellow, Emory University School of Medicine

Introduction

We present a case of a patient with recurrent thrombotic events despite anticoagulation, who then underwent hematologic workup. As part of this workup, hematology oncology requested a transesophageal echocardiogram (TEE) to evaluate for a cardiac source of emboli. TEE showed a small, mobile mass on the aortic valve suggestive of a papillary fibroelastoma (PFE). Given his embolic history, surgical excision was recommended.

Case Presentation

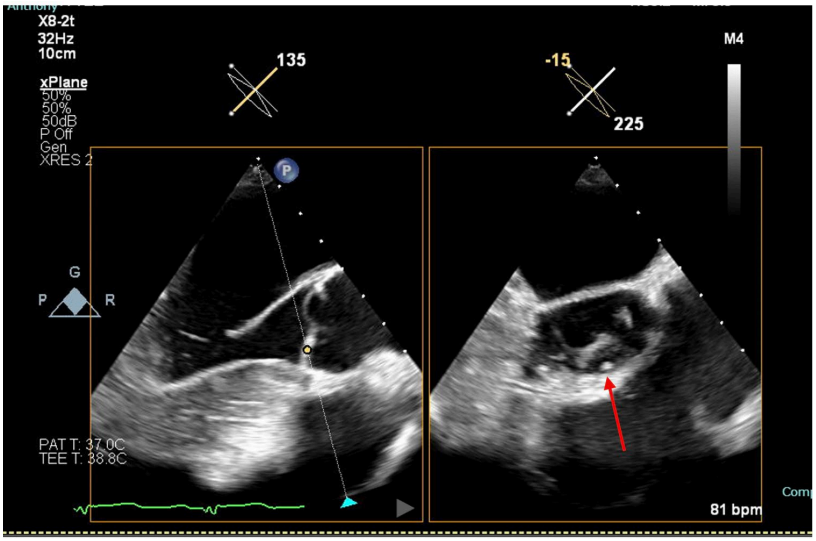
A 48-year-old male with coronary artery disease complicated by STEMI status post PCI ×4, peripheral arterial disease with multiple stents, type II diabetes, and GERD presented for cardiac tumor resection. He had undergone workup for recurrent thrombotic events over the past year, refractory to numerous anticoagulants. A year earlier he was found to have a right renal artery thrombosis with infarction. Over the following year, he continued to have thrombotic events — and later developed paralysis secondary to an aortic clot (Leriche syndrome). However, he had no strokes. Evaluation revealed JAK2 gene mutation, and TEE identified a 1.0 cm × 0.2 cm PFE on the ventricular side of the right coronary cusp of the aortic valve. Given that all thromboses were arterial, the decision was made to remove the PFE as a possible embolic source, though it might not be the definitive cause. On the day of surgery, the patient underwent general anesthesia with central line and Swan–Ganz catheter placement. Perioperative TEE, however, showed only a a 0.2 × 0.3 cm stump on the aortic valve. Multiple cardiac anesthesiologists attempted to locate the mass without success. The original echocardiographer was contacted intraoperatively and images were also reviewed in real time. With the assumption that the mass had embolized, the procedure was aborted.

Discussion

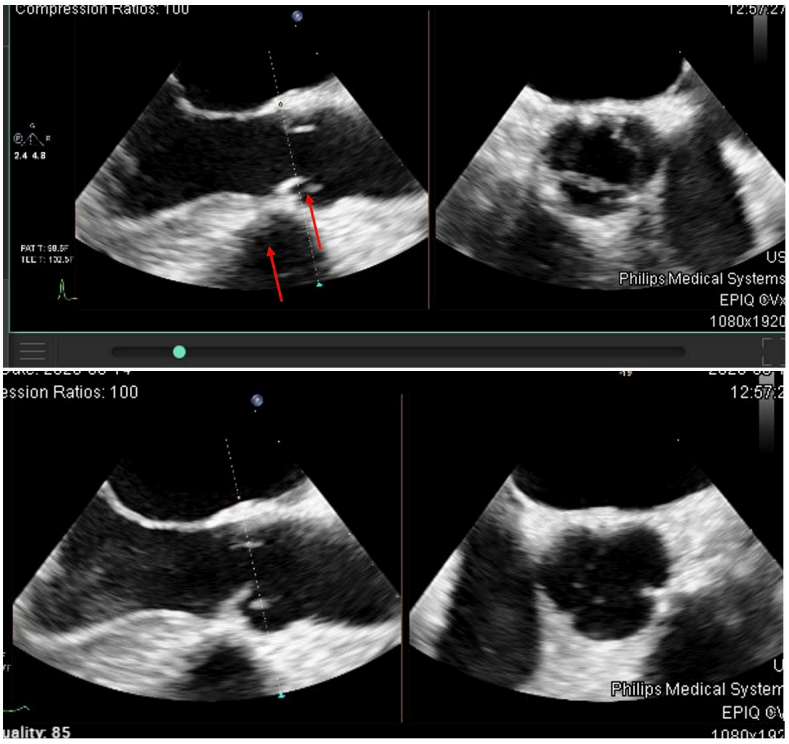
PFEs are cardiac tumors typically found on the underside of valves, most commonly the aortic valve. They consist of “collagen and elastic fibers with an endothelial covering and a short pedicle” (Tyebally, 2020). Their loose structure predisposes them to embolization, and stroke/TIA may even be the presenting symptom. Studies have shown patients who do not undergo surgical removal have increased risk of cerebrovascular events and mortality (Tamin, 2015). This case highlights the importance of echocardiography in guiding surgical decision making. The American Society of Echocardiography provides recommendations on imagingcardiac masses but does not issue guidelines on intervention. A 2020 Journal of the American College of Cardiology review notes that myxomas and symptomatic masses are those typically requiring surgery. The Society of Thoracic Surgeons recommends PFE resection only when larger than 1 cm or symptomatic. Patients undergoing surgery for a cardiac mass should have recent imaging, documentation of neurological or embolic events, and understanding of surgical risks. When embolization is

suspected, anesthesiologists must use multiple imaging modalities and views to be confident in the diagnosis in pursuit of determining whether a procedure is justified. As intraoperative imaging consultants, anesthesiologists played a key role in preventing an unnecessary sternotomy in this young patient.

Images



Day of Surgery TEE: Long Axis of Aortic Valve, with x-plane showing stump or previous PFE.



Baseline TEE: Long Axis of Aortic Valve Showing Fibroelastoma

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#FR 2 – Kiosk 24 – Poster 3

Craniofacial Edema with Otorrhagia from High Retrograde Cerebral Perfusion in a Patient with Left Innominate Vein Stenosis

Presenting Author: Jose Sanchez, MD, Resident – Anesthesiology, New York Presbyterian - Columbia University Irving Medical Center

Introduction

Venous congestion leading to craniofacial edema is a rare complication during cardiopulmonary bypass (CPB). Cases have been reported of super vena cava (SVC) syndrome in cardiac surgery due to malpositioned drainage cannulas[1]. Here we outline a case of craniofacial edema with otorrhagia in a patient with known left innominate vein stenosis who experienced elevated venous pressures during retrograde cerebral perfusion.

Case Presentation

A 75-year-old-woman with hypertension, hyperlipidemia, giant cell arteritis, polymyalgia rheumatica and a 5.5 cm ascending aortic aneurysm presented for ascending aortic hemiarch replacement and aortic valve repair. Preoperative imaging revealed marked narrowing of the left innominate vein. Induction of general anesthesia and placement of a right internal jugular maximum access catheter were uncomplicated. Following sternotomy and heparinization, cannulas were placed in the aorta, SVC, and inferior vena cava. Deep hypothermic circulatory arrest was used for cerebral protection after initiation of cardiopulmonary bypass. Retrograde cerebral perfusion (RCP) was started through the SVC cannula with goal central venous pressure (CVP) of 15 - 20 mmHg. Upon initiating RCP, elevated CVPs were noted, ranging from a baseline of 41 to a maximum of 239 mmHg upon increasing flow. No apparent issues were noted in the surgical field. Initial RCP during hemiarch replacement lasted 5 minutes and was followed by unilateral antegrade cerebral perfusion via the innominate artery. RCP was resumed for the final portion of the proximal transverse arch anastomosis for a total of 4 minutes. Again, CVPs were elevated and ranged from 45 to 95 mmHg. Throughout the case, cerebral oximetry saturations remained stable. At the end of the case, the patient's face was noted to be engorged with orbital swelling and chemosis. Bilateral ear bleeding was present. Given her facial edema, she was placed in reverse Trendelenburg position and received 40 mg IV furosemide. Postoperatively, she followed commands and responded appropriately with awakening trial shortly after surgery finished. The otolaryngology service was consulted and found oropharyngeal edema and hemotympanum. She was started on a dexamethasone taper and extubated on post-operative day 2. She was discharged on post-operative day 8 without apparent neurologic or auditory deficits.

Discussion

Though similar, we note that our case does not represent an instance of SVC syndrome. The SVC drainage cannula was not malpositioned nor was there any other noted point of SVC obstruction. Rather, increased RCP flow in the context of left innominate vein stenosis (and a likely patent Circle of Willis) pre-disposed this patient to vascular congestion. A similar mechanism has been described in dialysis patients who had

previously asymptomatic innominate vein stenosis and developed facial edema once faced with increased flow from hyperdynamic arteriovenous fistulae[2]. In our case, a marked but transient increase in venous pressures was enough to cause craniofacial edema in the presence of innominate vein stenosis.

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#FR 2 – Kiosk 24 – Poster 4

Embolization of an Amplatzer Septal Occluder Used for Peridevice Leak Closure After Watchman Implantation

Presenting Author: Isaac C. Gluesenkamp, MD, Resident, Indiana University School of Medicine

Introduction

Left atrial appendage (LAA) occlusion is used for stroke prophylaxis in patients who are poor candidates for long-term anticoagulation. Small peridevice leaks (PDL ≤5 mm) occur in approximately 25–40% of patients on early follow-up imaging, whereas large leaks (>5 mm) are uncommon, typically < 1%[1]. Although a definitive association between incomplete closure and thromboembolic events has not been established, interpretation is limited as patients with significant leaks are often maintained on anticoagulation. In an effort to eliminate residual embolic risk and avoid resuming anticoagulation, secondary percutaneous interventions may be pursued. Evidence for using Amplatzer septal occluders (ASO) or other septal/vascular occlusion devices to treat PDLs remains limited to case reports and small series.

Case Presentation

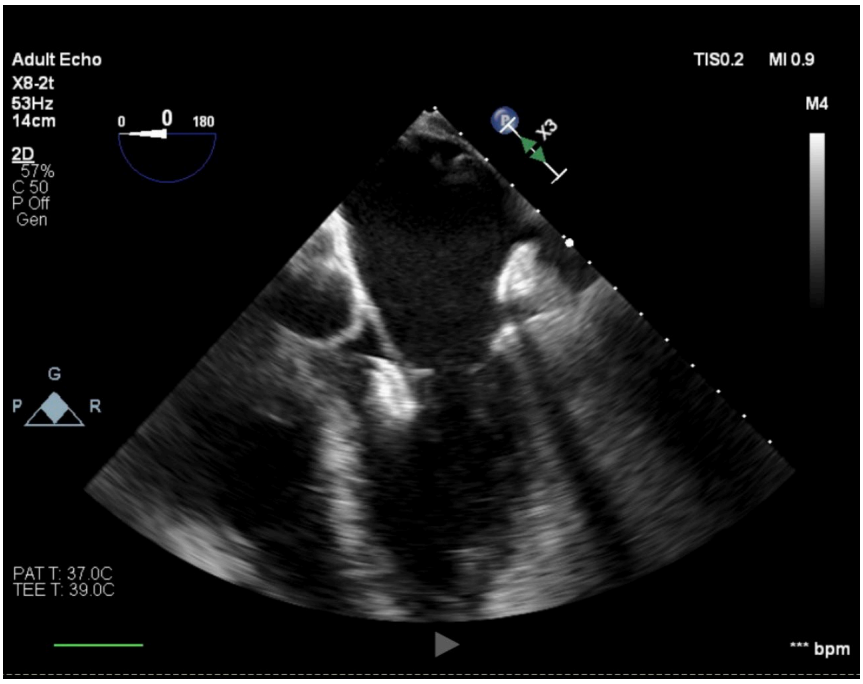
A 79-year-old man with paroxysmal atrial fibrillation refractory to ablation and prior Watchman implantation presented for closure of a 10-mm PDL. His CHA_2DS_2-VASc score was 4. He remained rhythm-controlled on amiodarone and continued full-dose apixaban following identification of the leak. A closure attempt was made using an 8-mm ASO device. Despite appropriate orientation and apparently stable positioning on intraprocedural imaging, the device embolized before case completion. Transesophageal echocardiography demonstrated sequential migration from the left atrium to the left ventricle, then into the aorta, ultimately lodging in the iliac circulation. The device was successfully retrieved percutaneously via the left femoral artery. The patient was discharged home the same day. Although a second revision procedure was initially planned, the patient elected to continue lifelong anticoagulation instead.

Discussion

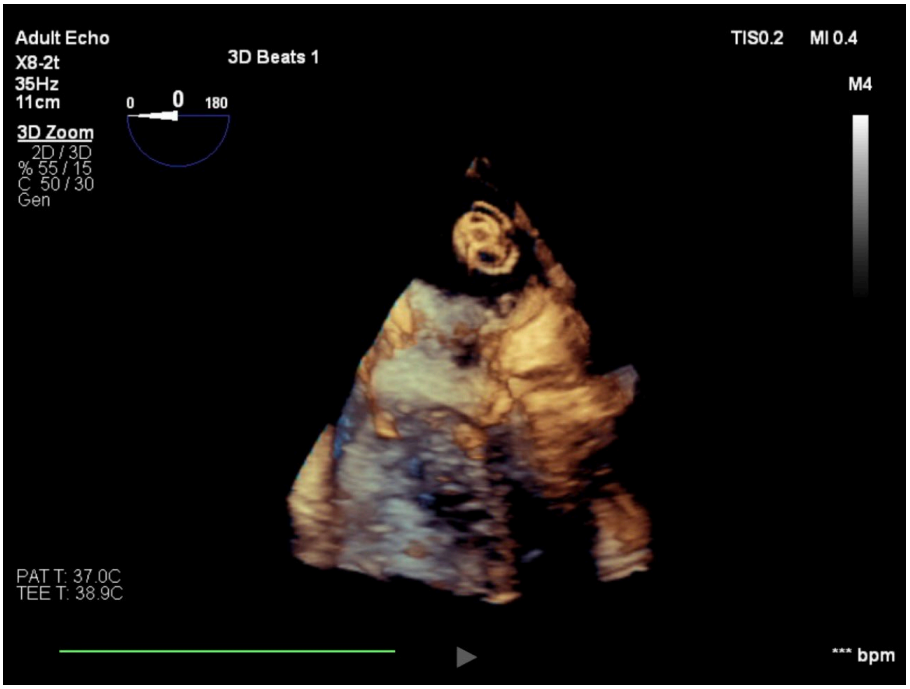
Residual PDLs remain a limitation of LAA closure and may result from undersizing, malposition, incomplete occlusion of multilobed appendages, device shift, or tissue remodeling. Secondary intervention is often considered for leaks >3 mm or for hemodynamically significant jets. In a small series by Hornung et al., 12 patients underwent secondary percutaneous closure (five with a second LAA device and seven with Amplatzer cardiac or vascular plugs), with no procedural complications or device embolization reported. However, broader experience is sparse, and evidence is confined to small case series and reports. While ASO embolization rates of 0.3–2.2% have been described in the context of atrial septal defect closure, the true embolization risk when ASO devices are used off-label for PDL management after LAA closure is unknown. In this case, prompt recognition and coordinated procedural response enabled successful endovascular retrieval without sequelae. Percutaneous closure using ASO or vascular plug devices can be effective, but the off-label nature of this strategy, combined with the mechanical challenges of device-on-device implantation, underscores the importance of careful patient selection, advanced imaging,

multidisciplinary planning, and readiness for emergent retrieval.

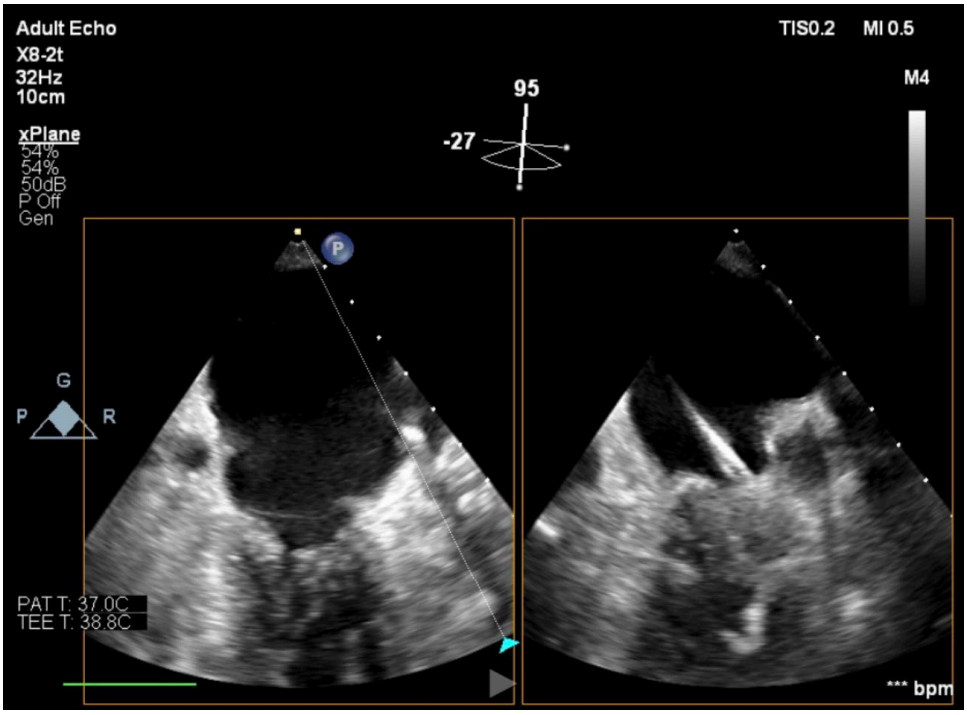
Images



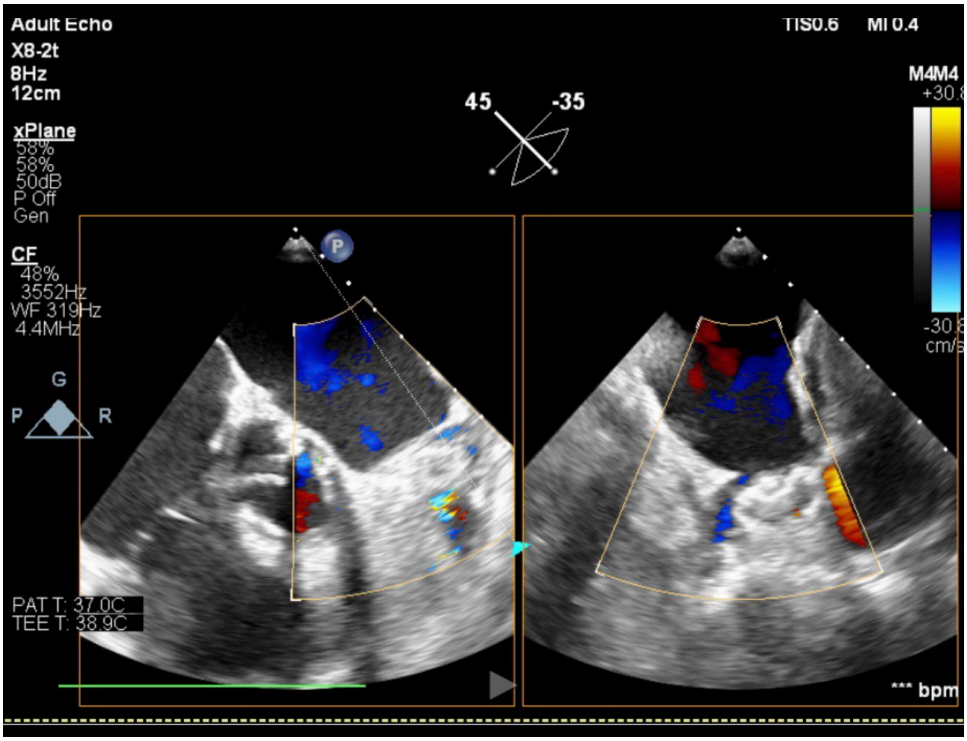
ASO floating in LV prior to migration through LVOT.



3D image of ASO floating in LA.



BiPlane ME 4 Chamber and 2 Chamber. ASO in place with no leak.



BiPlane Aortic valve short and long axis. Visible leak around Watchman.

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#FR 2 – Kiosk 24 – Poster 5

When the Valve Slips: Transcatheter Aortic Valve in Valve Rescue for Late Migration and Acute Paravalvular Leak

Presenting Author: Cindy Chu, DO, Wayne State University Department of Anesthesiology

Introduction

Transcatheter aortic valve in transcatheter aortic valve (TAV in TAV) is a minimally invasive strategy to treat a failing aortic valve prosthesis by implanting a new transcatheter valve within a prior device. Acute TAV in TAV procedures are typically performed for valve embolization, malposition, or severe paravalvular leak (PVL) recognized during the initial procedure, whereas delayed TAV in TAV is generally reserved for structural valve degeneration (SVD) or late PVL related to annular remodeling (1). In late PVL, balloon dilation is rarely effective because the mechanism is often prosthetic migration or loss of annular apposition rather than incomplete expansion, and dilation cannot re-establish frame alignment or seal a structural regurgitant channel. This case describes severe PVL and new severe MR associated with suspected late prosthesis dislodgement after TAVR, with subsequent clinical and echocardiographic improvement after TAV in TAV.

Case Presentation

A 77-year-old male presented on POD-12 following an initially uncomplicated TAVR (26 mm Edwards Sapien Resilia) with dyspnea, dark urine, and laboratory evidence of hemolysis. Immediate post-procedural TTE had shown trace PVL, trace MR, and a LVEF of 60%. On readmission, TEE demonstrated severe eccentric PVL directed toward the anterior mitral leaflet, left ventricular dilation with EF 40%, and severe MR. The prosthetic frame appeared subtly displaced relative to the annulus. After significant multidisciplinary discussion, the patient underwent TAV in TAV under general anesthesia with TEE guidance. A second 26 mm Resilia valve was deployed within the initial frame, and TEE demonstrated trace PVL and mild MR with improved mitral leaflet coaptation. The patient experienced rapid clinical improvement and resolution of hemolysis over the next 24 hours.

Discussion

Prosthetic migration after TAVR can create a narrow, high-velocity regurgitant orifice between the valve frame and a heavily calcified annulus. This paravalvular channel produces a substantial aortic regurgitant volume, abruptly increasing LV preload and causing acute ventricular dilation. The resulting chamber expansion causes mitral annular stretch, leaflet distortion, and apical papillary muscle tethering, all of which contribute to loss of mitral leaflet coaptation and functional MR. Clinically, this sequence can manifest as hypotension, pulmonary edema, and hemolysis from shear forces across the PVL jet. TEE is central to diagnosis; it can detect subtle prosthetic displacement, delineate PVL morphology and jet direction, and distinguish functional MR from regurgitation caused by direct prosthetic interference (2). When migration is partial and coronary flow remains preserved, valve-in-valve TAVR can stabilize the frame, seal the paravalvular channel, reduce shear forces, and restore mitral coaptation—often leading to rapid hemodynamic recovery, as in our patient. More extensive embolization may require alternative

percutaneous or surgical strategies. This case illustrates that prosthesis dislodgement or migration can result in severe PVL, acute MR, and hemolysis, and that perioperative TEE provides cardiac anesthesiologists with an effective tool to diagnose the mechanism and help guide targeted interventions.

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#FR 2 – Kiosk 24 – Poster 6

Off the Valve and Into the Ventricle: A Rare Case of Left Ventricular Papillary Fibroelastoma

Presenting Author: Jonathon R. Burns, DO, Fellow Physician, University of Kentucky

Introduction

Primary cardiac tumors are rare compared to secondary (metastatic) cardiac neoplasms.[1] Among primary cardiac tumors, papillary fibroelastomas (PFEs) represent the second most common benign subtype in adults.[2] PFEs typically originate from the valvular endocardium, most often involving the aortic or mitral valves; however, non-valvular locations such as arising from the left ventricular endocardium are exceedingly rare. Approximately 30% of PFEs are asymptomatic and discovered incidentally, while the remainder may present with stroke, transient ischemic attack, angina, myocardial infarction and syncope. Surgical excision is recommended in patients with prior embolic events, coronary ostial obstruction, or mobile or large tumors that pose an increased embolic risk.

Case Presentation

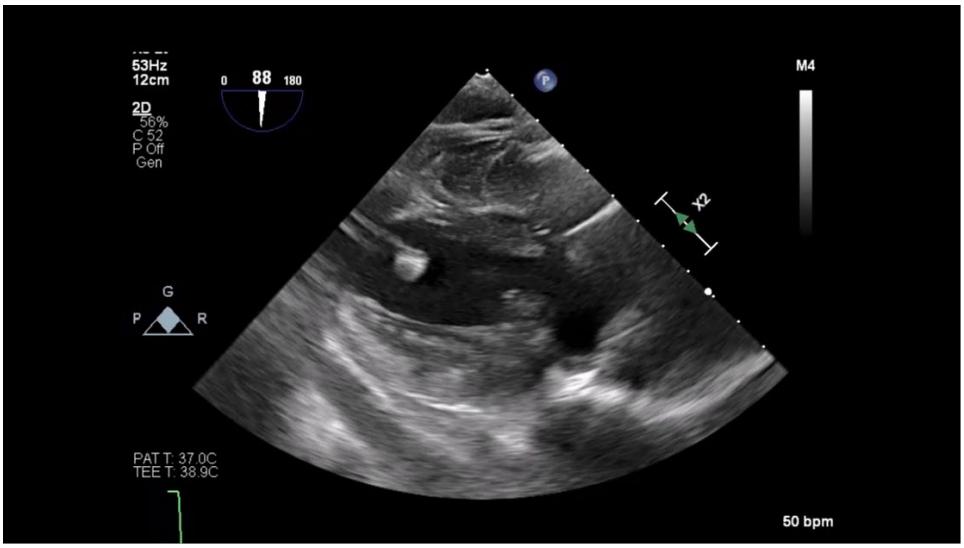
A 63-year-old woman with coronary artery disease, hypertension, type 2 diabetes mellitus, obstructive sleep apnea and frequent ventricular premature contractions presented for resection of a left ventricular mass. Transthoracic echocardiography was performed which revealed a mobile lesion attached to the inferior wall of the left ventricle and CT angiography demonstrated a 7 mm spherical mass tethered to the mid-septal wall by a thin stalk. She underwent median sternotomy and ventriculotomy with complete excision of the mass. Intraoperative transesophageal echocardiography (TEE) revealed a 1.02 × 0.87 cm mass attached to the inferior wall, along with mild-to-moderate tricuspid regurgitation and trace mitral regurgitation (see image 1 and 2). Histopathology confirmed the diagnosis of papillary fibroelastoma. Her postoperative recovery was uneventful, and she was discharged home in stable condition.

Discussion

Primary cardiac tumors are exceedingly uncommon, with PFEs accounting for approximately 8% of benign cardiac neoplasms. PFEs are typically small, avascular, and pedunculated lesions composed of fibroelastic connective tissue lined by endothelium.[2] Although most PFEs originate from valvular surfaces, involvement of non-valvular sites such as the left ventricular endocardium remains uncommon. Such locations are clinically significant because of their potential to cause systemic embolization or coronary ostial obstruction. The mobility of the tumor and the length of its pedunculated stalk are key determinants of embolization risk and often guide the decision for surgical excision.[3] Multimodal imaging plays a crucial diagnostic role.[1] Echocardiography remains the cornerstone for identification, while CT or MRI can help define lesion morphology and attachment site. From an anesthetic perspective, intraoperative TEE is invaluable for confirming preoperative findings but also for ensuring complete resection, detecting residual or multiple lesions, and evaluating valvular function and ventricular performance following excision. This case illustrates a rare presentation of a left ventricular papillary fibroelastoma. Additionally, this case emphasizes the importance of vigilant echocardiographic screening in patients presenting with unexplained

embolic or arrhythmic symptoms and highlights the anesthesiologist's integral role in perioperative imaging, diagnosis, and management of intracardiac masses.

Images





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#FR 2 – Kiosk 24 – Poster 7

Treatment of left-ventricular outflow tract obstruction following transcatheter mitral valve implantation

Presenting Author: Piyathida Obert, MD, Fellow, Cedars-Sinai

Introduction

Chronic mitral regurgitation (MR) is an insidious disease characterized by dyspnea due to increased left atrial pressure and pulmonary congestion. Transcatheter mitral valve implantation (TMVI) is a viable option for patients deemed as high or prohibitive surgical risk. However, the procedure has inherent risks, such as embolization, malpositioning, and structural damage. Hypertrophic cardiomyopathy (HCM) is one of the most common genetic cardiovascular disorders. HCM can lead to left ventricular outflow tract obstruction (LVOTO) and is associated with systolic anterior motion of the mitral valve (SAM). Symptoms of HCM include dyspnea on exertion, chest pain, syncope, or presyncope. [1] LVOTO is a leading cause of mortality after TMVI which excludes many patients from receiving this therapy. This potential complication can be predicted via a pre-procedural computed tomography simulating a TMVI individualized by the patient's anatomy. In addition, SAM can be predicted based on the length and laxity of the anterior mitral leaflet. [2,3]

Case Presentation

The patient is a 61-year-old female with HCM who was treated surgically with a septal myectomy in 2017, and subsequently underwent a reoperative sternotomy, redo myectomy, and a bioprosthetic MVR in 2019. She also has a history of pulmonary embolism, pulmonary hypertension, and chronic respiratory failure. She presented with dyspnea and palpitations, where a TEE revealed a bioprosthetic MV with no mitral valve stenosis, but severe MR from a flail leaflet (Figure 1). The peak LVOT gradient was 7 mmHg. Of note, there was possibly residual mitral chordal tissue contributing to flow acceleration, assessed by color Doppler across the LVOT. She was deemed too high risk for a redo surgical MV replacement. Therefore, she underwent TMVI with an Edwards Lifesciences (Irvine, California) SAPIEN 3 Ultra RESILIA transcatheter valve. Post-implantation TEE demonstrated appropriate valvular function without any central or paravalvular regurgitation. However, there was a new-onset significant LVOTO with pressure gradient of 32 mmHg caused by the direct impingement of the LVOT by the TMVI (Figure 2). An emergent LVOT-plasty was performed using a balloon catheter, effectively displacing the TMVI towards the left atrium in order to restore laminar LVOT flow and improve gradient to 25 mmHg. The patient's recovery was uncomplicated and her dyspnea improved. She was discharged home post-procedure day 1.

Discussion

This unusual case report emphasizes the importance of meticulous TEE examination and prompt recognition of LVOTO following TMVI. LVOTO can occur not only with a malpositioned prosthesis, but also high-risk patient anatomy such as SAM or a hypertrophied interventricular septum. This patient was categorized as high risk for a structural interventional procedure due to her history of HOCM. By promptly recognizing the LVOTO, she underwent LVOT-plasty and alleviated the obstruction in a timely manner. This

case is also unique in that the patient developed severe MR following mitral valve replacement within 6 years. She presented to the hospital multiple times with dyspnea before severe MR was diagnosed. This emphasizes the importance of recognizing MR symptoms in a patient with a bioprosthetic valve, even when placed relatively recently.

Images

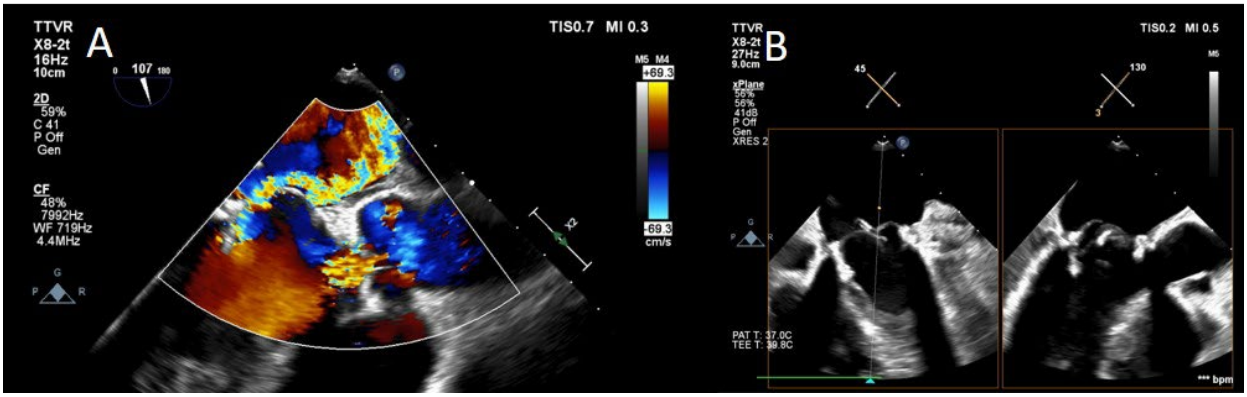


Figure 1. TEE showing bioprosthetic mitral valve regurgitation. A. Mid-esophageal AV LAX with color doppler showing eccentric jet of the mitral valve regurgitation. B. x-plane mid-esophageal view of bioprosthetic mitral valve prolapse

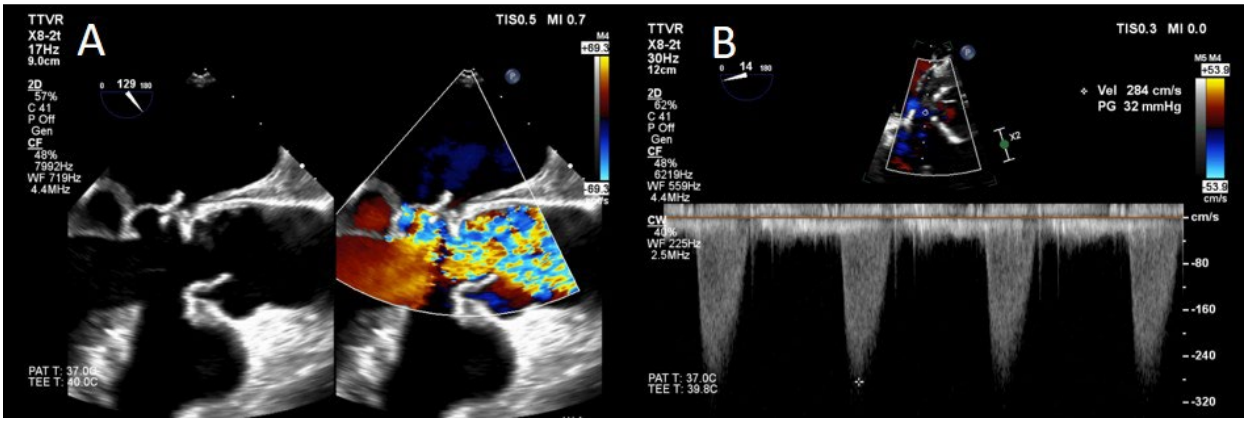


Figure 2. TEE demonstrating LVOT obstruction after deployment of TMVI A) mid esophageal AV LAX with color Doppler showing jet following across aortic valve. B) Continuous wave Doppler demonstrating a peak pressure gradient of 32 mm Hg and velocity of 284 cm/s

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#FR 2 – Kiosk 25 – Poster 1

From Heart to Hypophysis: Pituitary Apoplexy after Cardiac Surgery

Presenting Author: Anthony Dinkel, MD, Northwestern McGaw Medical Center

Introduction

Pituitary apoplexy is a rare clinical syndrome due to hemorrhage or infarction of the pituitary gland and is an uncommon perioperative complication of cardiac surgery potentially caused by systemic anticoagulation, changes in vasculature shear stress, and rapid blood pressure changes. We present a case and subsequent management of pituitary apoplexy post-cardiac surgery with cardiopulmonary bypass (CPB) in a patient with known residual pituitary macroadenoma.

Case Presentation

A 70 year old male with severe mitral valve regurgitation and moderate aortic insufficiency underwent sternotomy for mitral valve repair and bioprosthetic aortic valve replacement with CPB. He had a history of nonfunctioning pituitary macroadenoma resected 20 years prior with residual tumor on interval imaging and chronic hypothyroid and hypogonadism treated with medications. No neurologic symptoms were reported during his preoperative evaluation. The intraoperative course was uncomplicated. Heparin was adequately reversed following CBP, he was hemodyanically stable with minimal vasopressor support and no additional inotropy. Traditional and thromboelastic coagulation studies were normal as were cerebral oximetry values thorughout his operative course. Extubation occurred within hours of arrival to the ICU with a normal neurologic exam. On post operative day 1, the patient experienced an acute onset of a severe headache behind his eyes with diplopia. His exam revealed a fixed, dilated left pupil with cranial nerve 3 and 6 palsies with visual field deficits. CT brain revealed a sellar mass with hyperdense intralesional hemorrhage. MRI pituitary redemonstrated the 3cm sellar mass with compression of the optic chiasm and invasion of the left cavernous sinus. Neurosurgery was consulted and recommended urgent total transsphenoidal resection of the pituitary mass due to apoplexy, which he underwent on post operative day 2 revealing necrotic and acute hemorrhagic pituitary contents.

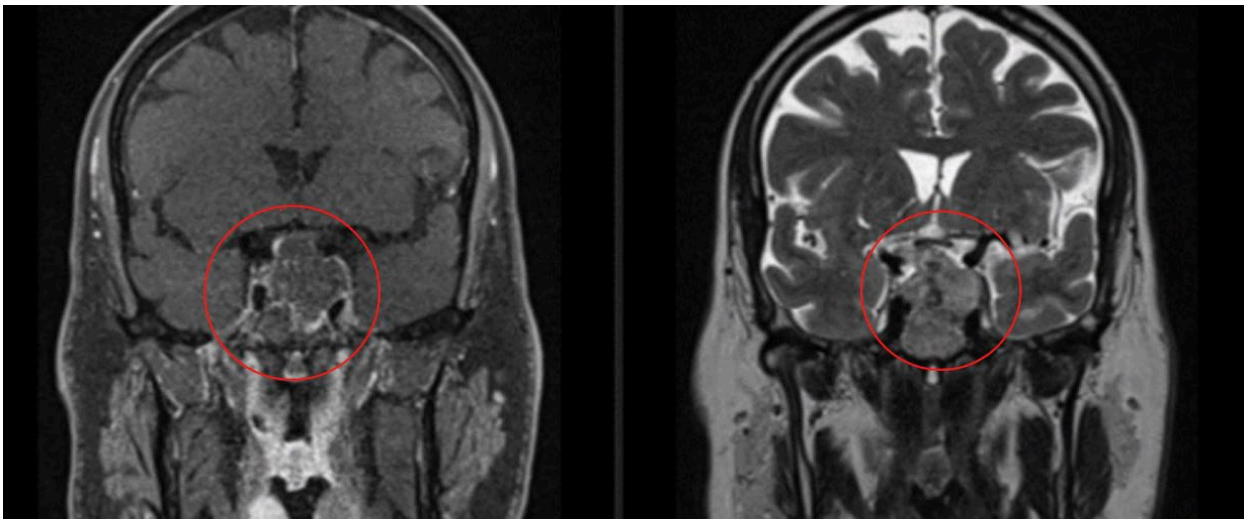
After resection, he had recovery of cranial nerve function over the course of days. He continues post-operative surveillance with progressive resolution of visual field deficits due to compressive optic neuropathy. He has continued his treatment for chronic thyroid and gonadal hormone deficiencies but no other endocrine abnormalities resulted during this course.

Discussion

Pituitary apoplexy after cardiac surgery is an uncommon but serious complication, often triggered by perioperative factors including anticoagulation, blood pressure fluctuations, and transient hypoperfusion during cardiopulmonary bypass. These stresses can precipitate hemorrhage or infarction in an unsuspected or known pituitary lesion. Prompt clinical recognition can be challenging and management complicated in settings with need for postoperative anticoagulation and potential for neurosurgical resection. There are no established guidelines for perioperative management or screening and most recommendations come from

restrospective case series. The decision to proceed with cardiac surgery requiring CPB is a patient with known pituitary lesions requires a multidisciplinary discussion to properly mitigate and limit risks where able.

Images



MRI Pituitary coronal sections, T1 (left) and T2 (right) with encircled pituitary lesion

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#FR 2 – Kiosk 25 – Poster 2

Trial and Error: Airway Management during Robotic Tracheobronchoplasty

Presenting Author: Holly Huber, MD, Resident Physician, UW Health

Introduction

Expiratory central airway collapse (ECAC) is characterized by exaggerated tracheobronchial narrowing. Severe cases are increasingly treated with surgical management such as tracheobronchoplasty, which fixes mesh to the cartilaginous airway edges and posterior membrane to prevent expiratory collapse. This technique involves isolated left lung ventilation in the left lateral position and periods of apnea while the surgeon works on the left main bronchus. The "Stop, Drop, and Roll" (DRS) maneuver, which retracts the endotracheal tube (ETT) into the trachea to expose the posterior membrane, presents unique anesthetic challenges[2]. We present one such case.

Case Presentation

A 68-year-old female presented with chronic cough and exertional dyspnea. On awake bronchoscopy, the patient had 80% collapse of the distal trachea with near 100% collapse of both mainstem bronchi. She improved following a trial with stents to her distal trachea and was consented for definitive treatment with robotic tracheobronchoplasty. Intra-operatively, we used a specialized wire-reinforced 40-cm endotracheal tube (Fuji Wire Reinforced Endotracheal Tube [WRETT]) to allow easier sealing of the left mainstem. During the left bronchoplasty portion of the procedure, the DRS maneuver was used to allow fixation of mesh. During the DRS maneuver, the ETT was retracted, sutures placed and ETT readvanced. The surgical team communicated that the ETT was inadvertently in the right bronchus. The ETT was retracted, there were no identifiable airway structures. Unable to mask or intubate in the lateral position, an LMA was placed and ETT exchanged via a wire exchange catheter kit. The lowest measured SpO2 was 33% during the incident. For subsequent DRS maneuvers, the fiberoptic scope was placed within the ETT prior to retraction. This allowed for more precise retraction given direct visualization of the lighted end of the fiberoptic scope on the surgical field as well as replacement into the left mainstem bronchus. The remainder of the operation was uneventful, and the patient had a reassuring neurological examination following extubation.

Discussion

Our patient had near 100% collapse of the left and right mainstem bronchi on forced exhalation. Her intra-operative course was complicated by accidental extubation with severe hypoxia following the DRS maneuver. There have been case reports of using a standard ETT in combination with an EZ blocker without the DRS method, but there is a risk of suturing the blocker into the airway[1]. Other airway techniques for this procedure mostly include utilizing the DRS method with some variation of specialized ETT which has risks of accidental extubation given frequency of airway manipulation – especially under the drapes and in a lateral position. Fiberoptic manipulation of this patient population's airways can be challenging due to positioning, distorted airway anatomy from their disease process, and worsening EDAC with intrathoracic insufflation. We believe using a fiberoptic scope like a lighted stylet to guide retraction and re-advancement of the ETT

during the left bronchoplasty portion of the operation provides an additional layer of safety to prevent intra-operative complications of frequent airway manipulation.

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#FR 2 – Kiosk 25 – Poster 3

Perioperative Management of a Unicuspid Aortic Valve in a Patient Undergoing Aortic Valve Replacement

Presenting Author: Lucas J. Anderson, M.D., Anesthesia Resident, AdventHealth Orlando

Introduction

Intraoperative TEE revealed unicuspid aortic valve anatomy, altering management during aortic root replacement and Bentall Procedure.

Case Presentation

A 47-year-old woman with hypertension and anxiety underwent evaluation of progressive fatigue, prompting cardiac imaging that revealed unexpected aortic pathology. CTA demonstrated a 5.4 × 5.1 cm ascending aortic aneurysm, while a transthoracic echocardiography (TTE) revealed a bicuspid aortic valve with moderate–severe aortic stenosis (AS) and severe eccentric insufficiency (AI). After discussion of surgical options, the patient elected a bioprosthetic valve to avoid lifelong anticoagulation, accepting the likelihood of future reintervention. Intraoperatively, following a pre-induction arterial line, a balanced anesthetic technique was used to induce anesthesia, intubation, and manage competing hemodynamic goals of AS and severe AI. Following CVC/PAC placement, intraoperative transesophageal echocardiography (TEE) unexpectedly demonstrated unicuspid AV morphology with a single commissure, restricted leaflet excursion, mild AV stenosis (peak velocity 2.3 m/s, mean gradient 12 mmHg), and moderate–severe eccentric AI. Due to the large aneurysm and anticipated hypothermic circulatory arrest, peripheral cannulation was performed to facilitate safe entry into the chest. The patient underwent a Bentall procedure with a 23 mm AVR, a 25 mm Konect RESILIA valved conduit, and hemi-arch reconstruction. Post-bypass TEE showed normal prosthetic AV function with preserved biventricular performance. She recovered uneventfully and was discharged on postoperative day eight.

Discussion

This case demonstrates: (1) the diagnostic limitations of TTE in abnormal valve morphology; (2) the impactful role of intraoperative TEE in accurately identifying unicuspid anatomy; (3) the stronger association of UAV with early age progressive aortopathy, supporting early combined root and ascending intervention; and (4) the importance of physiology-guided anesthetic management in mixed AS/AI.

Teaching Points:

1. Preoperative TTE may misidentify abnormal valve morphology; intraoperative TEE is essential for accurate identification of unicuspid or bicuspid aortic valve anatomy and case management.
2. Unicuspid aortic valves have a stronger association with progressive aortopathy at a younger age than bicuspid valves, supporting earlier combined root and ascending aortic intervention.
3. Mixed aortic stenosis and eccentric aortic insufficiency require physiology-guided anesthetic management to optimize hemodynamics and myocardial perfusion.

Images



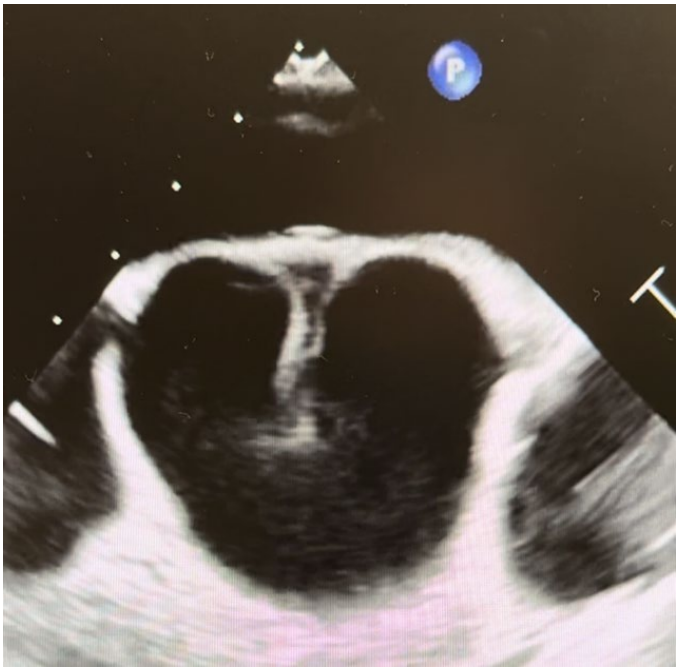
3D intraoperative TEE (systolic, en-face view) demonstrating a unicuspid aortic valve with a single commissure, showing the characteristic eccentric systolic orifice.



3D intraoperative TEE demonstrating complete coaptation of the solitary leaflet in this unicuspid aortic valve with a single commissure.



2D intraoperative TEE demonstrating a single functional leaflet opening in systole.



2D intraoperative TEE in diastole demonstrating restricted leaflet closure characteristic of a unicuspid aortic valve.



Excised aortic valve specimen demonstrating unicuspid morphology with a single commissure and dysplastic leaflet tissue.

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#FR 2 – Kiosk 25 – Poster 4

Rhabdomyolysis-Associated Hyponatremia in a VA-ECMO Heart Transplant Candidate: Vasopressin and Fluid Resuscitation Masquerade as SIADH

Presenting Author: Eric A. Krause, MD, Resident Physician, University of Kansas Medical Center

Introduction

Limb ischemia and rhabdomyolysis is a recognized complication of venoarterial extracorporeal membrane oxygenation (VA-ECMO)[1], however its downstream effects on sodium and fluid balance in the setting of aggressive volume resuscitation is less well described. We present a case of a 29-year-old female with complex congenital heart disease, requiring veno-arterial extracorporeal membrane oxygenation (VA-ECMO) for mechanical aortic valve thrombosis. The course was complicated by severe rhabdomyolysis and hyponatremia. The cause of hyponatremia was unclear. Laboratory data suggested syndrome of inappropriate antidiuretic hormone (SIADH) with a differential diagnosis of large volume fluid resuscitation sequelae and vasopressin induced water retention.

Case Presentation

A 29-year-old female with a history of repaired interrupted aortic arch, ventricular and atrial septal defects, and mechanical aortic valve replacement presented with cardiogenic shock due to acute aortic valve obstruction secondary to thrombus. She underwent peripheral VA-ECMO cannulation for stabilization. Within 48–72 hours of cannulation she developed rapidly rising creatine kinase levels peaking at 126,800 U/L, dark urine, and proximal lower-extremity pain and swelling around the venous cannulation site, consistent with severe rhabdomyolysis attributed to ECMO-related limb malperfusion. She received aggressive crystalloid resuscitation for renal protection. Concurrently, the patient required continuous vasopressin infusion for hemodynamic support. Despite isotonic fluids, she developed severe hyponatremia (sodium nadir 120 mmol/L). Following the discontinuation of vasopressin, the clinical picture rapidly shifted to frank polyuria (>19 L/day) and hypernatremia (sodium 148 mmol/L). Ultimately the cause of the hyponatremia was favored to be a mixed picture: vasopressin induced water retention superimposed on large-volume crystalloid administration. She was managed with desmopressin and free water replacement, eventually stabilizing to allow for successful orthotopic heart transplant.

Discussion

Rhabdomyolysis is a known complication of peripheral VA-ECMO, however, the concurrent development of profound hyponatremia presented a significant diagnostic challenge. This case illustrates the diagnostic difficulty of attributing a cause to hyponatremia when concurrent vasopressin infusion and massive resuscitation confound plasma and urine studies. In this case, the diagnosis of vasopressin-induced water retention was supported by the clinical course following drug cessation. Vasopressin exerts its antidiuretic effects via V2 receptors in the renal collecting duct. The abrupt withdrawal of the V2 receptor stimulation precipitated a rebound aquaresis, effectively flipping the patient from a state of SIADH to a state of diabetes insipidus, a phenomenon previously described[2]. In patients requiring both aggressive resuscitation for

rhabdomyolysis and vasopressin support, recognizing that vasopressin can mimic SIADH is critical to prevent iatrogenic sodium shifts during weaning.

Images

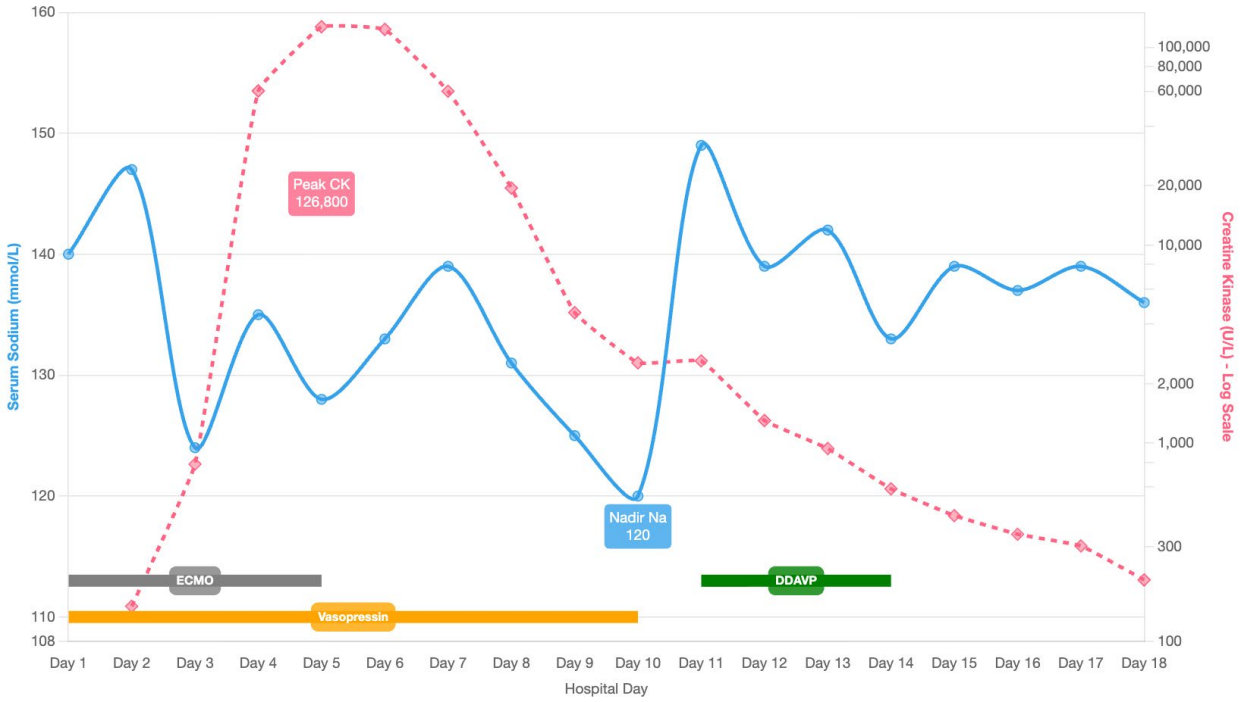


Figure 1. Timeline of VA-ECMO support, vasopressin and desmopressin therapy, serum sodium, and creatine kinase (CK) levels.

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#FR 2 – Kiosk 25 – Poster 6

Transcatheter Device Embolization into the Aorta

Presenting Author: Anika Tanwani, MD, Department of Anesthesiology and Pain Management, The University of Texas Southwestern Medical Center

Introduction

Transcatheter aortic valve replacement (TAVR) continues to grow as a therapeutic option for high-risk patients with severe aortic stenosis, yet it carries rare but serious complications. Device embolization is a rare but life-threatening event with low incidence in clinical trials (0.1% in PARTNER II, 0% in PARTNER 3, and 0% in SURTAVI) (1-3). Device embolization can occur despite careful planning and procedure execution due to anatomical, technical, or device-related factors and demands rapid multidisciplinary coordination and dynamic anesthetic management.

Case Presentation

A 79-year-old man with bicuspid aortic valve, severe low-flow, low-gradient aortic stenosis, nonischemic cardiomyopathy (EF 15–20%), ascending aortic aneurysm (5.2 cm), and paroxysmal atrial fibrillation presented for TAVR. The procedure began routinely under monitored anesthesia care using propofol, dexmedetomidine, and low-dose remifentanil infusions. A 26 mm Edwards Sapien S3 Ultra valve was implanted under rapid ventricular pacing (RVP) at 180 bpm. During full balloon inflation, a breakthrough PVC dislodged the valve into the ascending aorta. The patient was emergently intubated, transitioned to general anesthesia, and a TEE probe was inserted. Hemodynamics remained stable but were assisted with norepinephrine and epinephrine infusions. The valve was pulled into the descending aorta and secured with balloon inflation. A 29 mm Edwards Sapien S3 Ultra Resilia valve was then successfully implanted in the correct position under RVP. A P3110 balloon-expendable stent was positioned and deployed under RVP across the deployed 26 mm valve in the descending aorta. The stent migrated a bit distally but still captured the valve leaflets and was fully expanded and well apposed to the valve. Post-procedure TEE showed a well-seated prosthesis without paravalvular leak and a new small Type B aortic dissection with two flaps immediately distal to the takeoff of the left subclavian artery. The patient also developed new left bundle branch block and first-degree AV block. A right internal jugular central venous catheter was placed and used to position a right ventricular temporary pacing lead. The patient was then transferred uneventfully to the intensive care unit. He eventually had a leadless permanent pacemaker placed with outpatient imaging follow-up for his aortic dissection being managed with strict blood pressure control.

Discussion

TAVR valve embolization appears more frequently in anatomically complex patients, including patients with bicuspid aortic valve or reduced ejection fraction. Bicuspid aortic valves may have asymmetric, nontubular atrioventricular (AV) complexes, making the annulus difficult to define and increasing risks of underexpansion or annular injury. Strategies such as supra-annular deployment or mild oversizing in select patients may optimize valve seating. Low ejection fraction has also been linked to TAVR valve embolization,

potentially due to rapid valve deployment and limited ability to reposition the prosthesis. Having contingency strategies is essential, as deterioration can be sudden. Migrated valves may be retrieved endovascularly, as in this case, percutaneously with a snare, or surgically under cardiopulmonary bypass when necessary.

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#FR 2 – Kiosk 25 – Poster 7

Beyond the Right Side: Left-Sided Carcinoid Heart Disease in a Metastatic Patient

Presenting Author: Nilang N. Shah, Resident Physician, Vanderbilt University Department of Anesthesiology

Introduction

Metastatic cardiac carcinoid tumor involving the left ventricle represents a rare but clinically important manifestation of advanced neuroendocrine neoplasms. While carcinoid heart disease most commonly results from paraneoplastic fibrotic changes affecting right-sided cardiac valves due to circulating vasoactive substances, direct myocardial metastases can occur and have been documented in both ventricles or rarely isolated to the left ventricle [1-3]. Metastases are typically intramyocardial, well-circumscribed, and may be asymptomatic or present with arrhythmias, heart failure, or embolic phenomena. Diagnosis relies on imaging, with echocardiography and cMRI and somatostatin receptor-based PET/CT [1-2]. The presence of cardiac metastases in the left ventricle is associated with advanced systemic disease and may complicate management, necessitating a multidisciplinary approach. Here we present a case report describing the diagnosis and intraoperative management of a patient with metastatic carcinoid tumors involving the left ventricle, and the use of TEE to guide resection.

Case Presentation

A 53 year-old female with previously resected atypical left lower lung pulmonary carcinoid tumor (pT2N1) presented to her oncologist with several months of palpitations, wheezing, chest pain, rash, syncope and headache concerning for recurrent disease. Initial PET/CT revealed increased uptake at the apex of the left ventricle and follow up imaging with cMRI and TTE confirmed the presence of two small, well-circumscribed intramural masses in the mid-anterior and distal-inferior left ventricle (Figure 1) and mediastinal lymph node biopsy was non-diagnostic. She was referred for surgical evaluation and resection and underwent left ventricle metastatectomy with cardiopulmonary bypass. Intra-operative ABP, PAP and TEE were used to monitor hemodynamics. Induction of anesthesia was uncomplicated. After initiation of cardiac bypass both lesions were continuously visualized with TEE and excised. She was subsequently weaned from CPB without difficulty. Pre- and post-bypass echocardiogram noted no changes in cardiac function or wall motion abnormalities. She was transported to the ICU intubated without any vasoactive support and extubated the same day. The remainder of her hospitalization was uneventful and she was discharged on post-operative day 5. Final pathology for both masses demonstrated atypical carcinoid tumor with positive margins and she was started on lanreotide without need for further resection.

Discussion

The perioperative management of carcinoid heart disease is challenging with significant heterogeneity in both disease symptoms and presentation. Sequelae from cardiac carcinoid includes valvular degeneration and fibrosis, coronary vasospasm, intramyocardial involvement causing direct mass effect or outflow obstruction and arrythmias. Dissection can be challenging and the use of TEE to guide dissection near critical structures can be immensely beneficial. The use of somatostatin analogues (i.e. lanreotide or

octreotide) to mitigate carcinoid crises can cause significant hemodynamic instability under anesthesia. Lastly, patients with severe symptoms may be significantly deconditioned prior to surgery with poor functional status.

Images

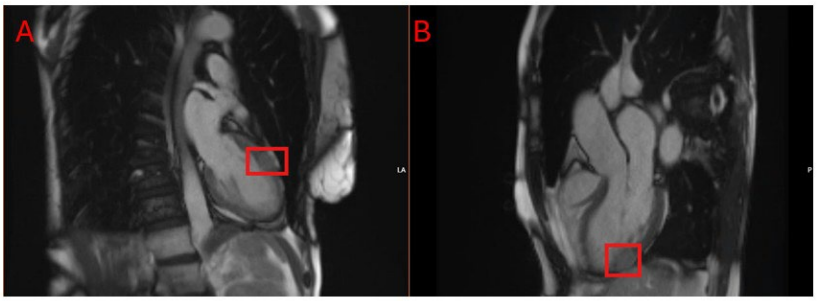


Figure 1. Cardiac MRI demonstrating mid-anterior (A) and distal inferior (B) well circumscribed, intramyocardial left ventricular masses.

Figure 1. Cardiac MRI demonstrating mid-anterior (A) and distal inferior (B) well circumscribed, intramyocardial left ventricular masses.

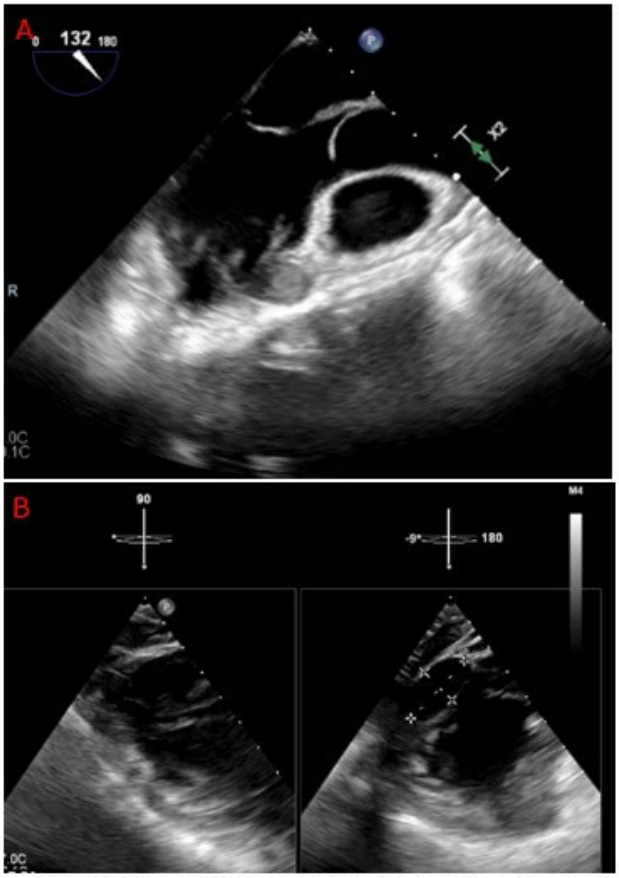


Figure 2. TEE images of intramural carcinoid masses. (A) LAX with apical mass on anteroseptal LV wall. (B) X-plane visualization of inferolateral LV mass.

Figure 2. TEE of intramural carcinoid masses. (A) LAX with apical mass on anteroseptal LV wall. (B) X-plane visualization of inferolateral LV mass.

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#FR 2 – Kiosk 26 – Poster 1

Severe Cardiac Complication During Routine Bronchoscopic Lung Biopsy

Presenting Author: Hunter C. Rose, Resident, Yale University School of Medicine

Introduction

Bronchoscopy is a vital tool in the diagnosis of various lung pathologies. Cardiac complications are rare, but can be associated with poor outcomes. The following case presents a severe complication and subsequent management during a robotic-assisted bronchoscopic lung biopsy.

Case Presentation

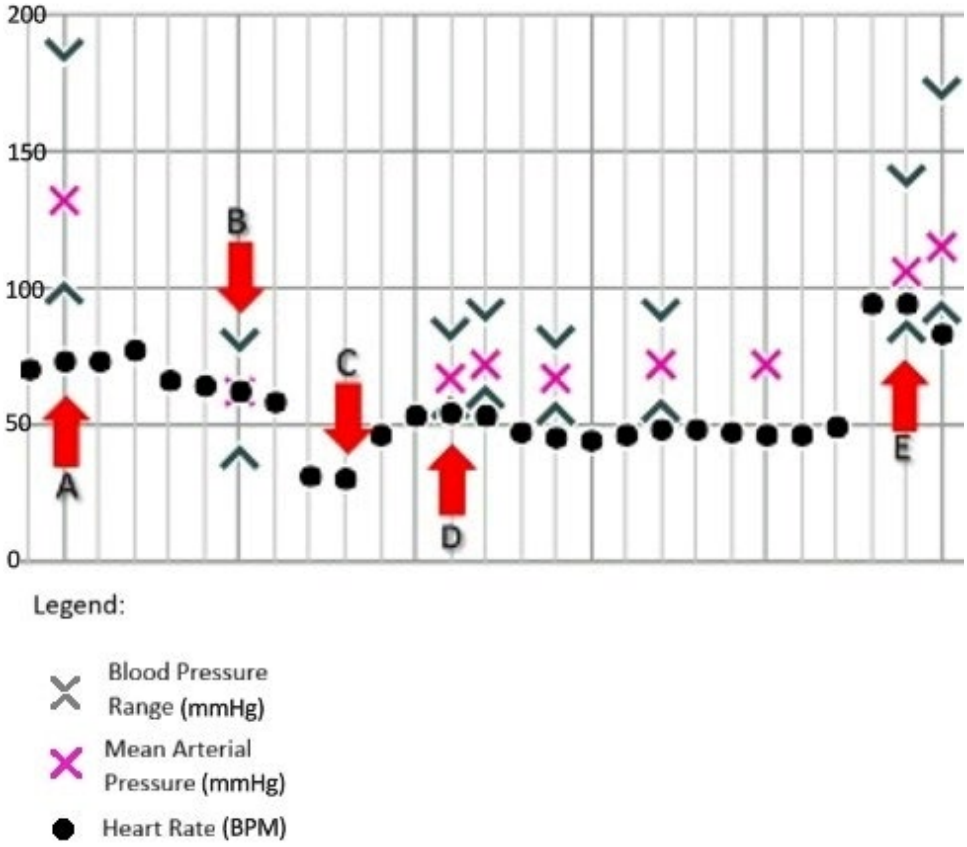
A 64-year-old male with a history of hypertension (HTN) and hyperlipidemia (HLD) was scheduled for biopsy of a 2.7 cm left upper lobe lung nodule. The case was performed under general anesthesia in line with institutional protocol for robotic-assisted bronchoscopies: tidal volume (TV) 8-10 mL/kg and positive end expiratory pressure (PEEP) 10-15 cmH2O. Localized bleeding was noted after biopsy. The patient became hypertensive during the airway instrumentation to locate and control the bleeding. Cold saline lavage was performed to reduce bleeding. The patient then developed inferior ST segment elevation, cyanosis, and transient bradycardia and hypotension (Fig. 1). Hemodynamic stability was promptly restored with gradual resolution of ST changes and cyanosis. A transesophageal echocardiogram revealed no visible air embolism or regional wall motion abnormalities (WMA). Immediate cardiac catheterization revealed a myocardial bridge in the mid LAD, but no significant coronary artery disease (CAD) or vasospasm (Fig. 2). The patient remained hemodynamically stable, but was unresponsive off sedation. CTA head/neck demonstrated no abnormalities. The next day, MRI brain was concerning for hypoxic-ischemic injury versus embolic infarcts (Fig. 3). After 12 days unresponsive in the ICU, the patient died shortly after supportive measures were withdrawn.

Discussion

Bronchoscopy is a routine and safe procedure with overall complication rates ranging from 0.1-11%[1]. Cardiac complications are rare[2], and include acute myocardial infarction (AMI)[3,4], coronary artery vasospasm (CAV)[5], and air embolism (AE)[4,5]. Increased myocardial oxygen demand due to sympathetic activation during bronchoscopy can lead to AMI in those with risk factors[3,6]. Myocardial bridges are congenital anomalies where a portion of the coronary artery traverses through the myocardium, and these are a known risk factor for CAV[7]. Relatedly, cold saline lavage has been used for decades to control bleeding during bronchoscopy[8] and has reportedly induced CAV leading to bradyarrhythmia[9]. In addition, global hypoperfusion was considered as a possible etiology. However, the patient had an isolated neurologic injury with transient troponin elevation. There were no other signs of end-organ damage from hypoperfusion, including no increase in creatinine, lactate, or serum aminotransferases. The absence of persistent WMA without appreciable CAD makes AMI unlikely, and isolated CAV would not explain the patient's neurologic injury. Altogether, these data suggest that the patient most likely experienced AE during the procedure. AE are a general risk during bronchoscopic biopsy due to increased airway pressures with a bronchoscope,

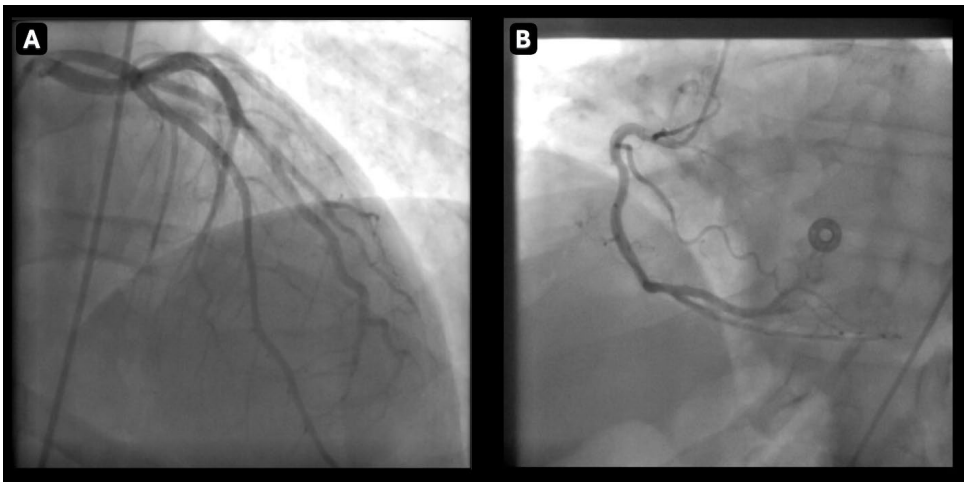
predisposing pressurized gas transfer into systemic circulation[5]. This patient may have been at particularly increased risk due to increased TV and PEEP contributing to increased mean airway pressure. The findings on brain MRI and the neurologic outcome are also in favor of AE.

Images

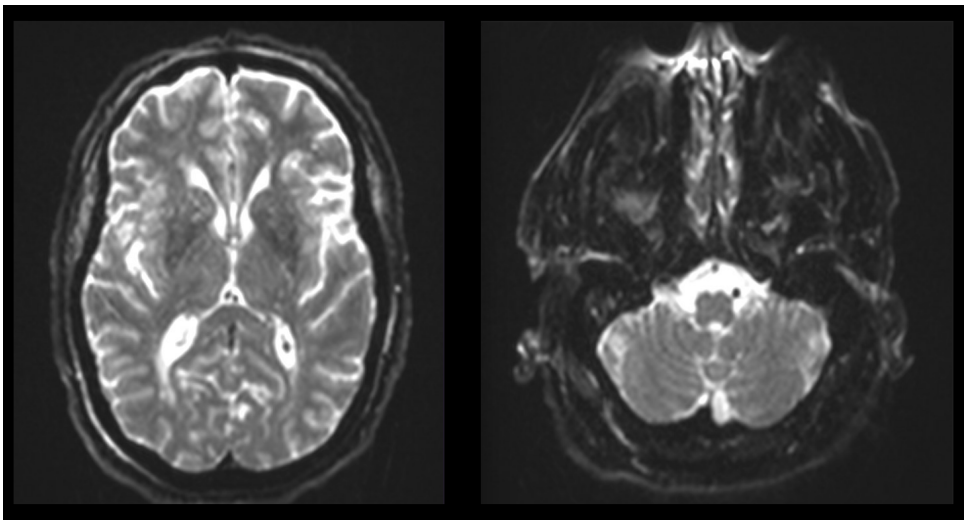


Minute to minute vitals showing hemodynamic changes during the procedure at the suspected time of complication. A: The time point of the initial hypertensive episode. The patient's blood pressure at this point was 184/101 (mean arterial pressure 132) with a heart rate of 73 beats per minute (BPM). B: The time point at which the patient became hypotensive. The patient's blood pressure at this point was 78/41 (mean arterial pressure 62), with a heart rate of 62 BPM, approximately 5 minutes after the hypertensive episode. After this point, the patient became increasingly bradycardic. C: The lowest heart rate recorded during the event at 30 BPM. Of note, the blood pressure was too low for the non-invasive monitor to read, and no blood pressure is recorded at this time point. D: With the aggressive administration of vasoactive medication, the patient's blood pressure was improved to 82/59 (mean arterial pressure 67), approximately 6 minutes after the initial hypotension was identified. The patient's heart rate also improved to 55 BPM. E: The point at which the patient's hypotension and bradycardia resolved, approximately 19 minutes after the initial hypotensive

episode. The patient's blood pressure was 138/87 with a mean arterial pressure of 106 and a heart rate of 94 bpm.



A. Left Main Coronary Artery. Minimal luminal irregularities were noted throughout. Note the inferior bowing in the mid LAD, which is the myocardial bridge that was identified. B. Right Main Coronary Artery. Minimal luminal irregularities were noted throughout.



Brain MRI obtained the day after the bronchoscopic biopsy and associated complication. Note the cortical restricted diffusion on the left, and punctate foci of restricted diffusion in the cerebellum on the right. These findings are concerning for hypoxischemic injury versus embolic infarcts.

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#FR 2 – Kiosk 26 – Poster 2

Undifferentiated Massive Hemoptysis During Zone 2 Pseudoaneurysm TEVAR with Carotid-Subclavian Bypass

Presenting Author: Drew Daniel, MD, Resident, Stanford University School of Medicine

Introduction

Hemoptysis as a herald sign of aortobronchial fistula (ABF) warrants immediate intervention. Mortality approaches 100% if untreated.[1-3] Careful review of imaging and symptoms is essential, as any pathology causing ABF may be associated with significant morbidity. This case highlights these considerations and utilization of differential lung ventilation (DLV).

Case Presentation

A 61-year-old male presented to an outside hospital with massive hemoptysis and respiratory distress. He was stabilized with inhaled tranexamic acid (TXA) and oxygen then transferred for emergent Zone 2 TEVAR with carotid-subclavian bypass for a 2.9 cm aortic arch pseudoaneurysm encased in chronic thrombus suspicious for mycotic aneurysm. Upon arrival, he was hemodynamically stable without hemoptysis for >24 hours, with an ongoing oxygen requirement and anemia (Hgb 7.2 g/dL). After transfer to the OR, radial arterial line placement, and intubation with a single lumen tube (SLT), hemodynamics and ventilator parameters were stable. Shortly after incision (>60 mins post-intubation), an abrupt drop in EtCO2 with high peak inspiratory pressures (PIP) occurred. The ventilator circuit filled with blood. Help was called and despite in-line suctioning, frank blood continuously filled the ventilator circuit. Massive transfusion was initiated. PEA arrest ensued, and the surgical team pivoted to stent graft deployment. With ongoing bleeding, fiberoptic bronchoscopy was futile, so the SLT was blindly advanced into the right mainstem bronchus. Return of spontaneous circulation and gas exchange were achieved with high PIP and low tidal volumes. With concern for imminent worsening of oxygenation and need to clear airways, the SLT was exchanged for a 37Fr left double lumen tube (DLT) using laryngoscopy and an airway exchange catheter. The surgical team obtained groin access in case of need for ECMO. After administration of nebulized TXA, a large clot was removed from the right lung, although worsening oxygenation necessitated veno-venous ECMO. DLV with two separate ventilators and individualized settings provided both lung-protective ventilation and lung isolation, with no further hemoptysis. TEVAR was completed without further incident. Postoperatively, the DLT was re-exchanged to a SLT with a bronchial blocker in the left mainstem. Before planned cryo-extraction of bilateral clots, recurrent massive bright red hemoptysis occurred, suggestive of arterial bleeding. Salvage rigid

bronchoscopy was unsuccessful. With no further interventional options, comfort measures were initiated, and the patient expired.

Discussion

This case illustrates the challenges of ABF and airway hemorrhage management. Airway hemorrhage remained a risk despite angiographically complete isolation of the pseudoaneurysm, possibly due to pulmonary vein or artery fistulae. Earlier ECMO initiation may have reduced risk of cardiac arrest, and postop DLV may have facilitated ongoing isolation between lungs though there was limited ability to clear clots. Given the magnitude of postop hemorrhage, we have been unable to identify any actions that may have prevented this outcome. Despite the fatal outcome, this case demonstrates the potential role of DLV in temporizing patients for life-saving intervention.

Images

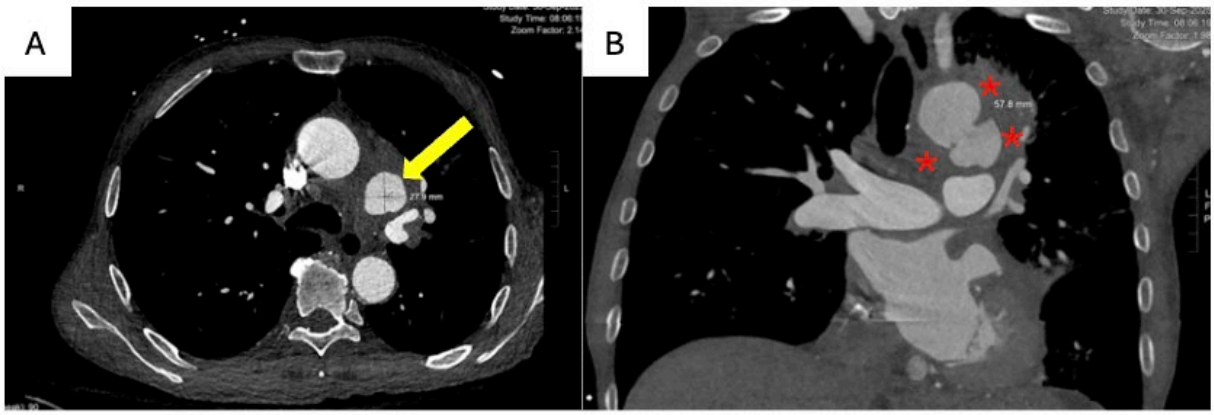


Figure 1: Preoperative CT imaging showing pseudoaneurysm (A, yellow arrow) on inferolateral aortic arch distal to subclavian takeoff, and thick rind of soft tissue and thrombus (B, red stars) which was noted to be also encasing left hilar pulmonary arteries and veins.

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#FR 2 – Kiosk 26 – Poster 3

Case Report: Anesthetic Considerations for a Patient with Prior Bleomycin Exposure Undergoing Coronary Artery Bypass Grafting (CABG)

Presenting Author: Michael Lugo, MD, Resident Physician, SLU Hospital Department of Anesthesiology and Critical Care

Introduction

Bleomycin, an antineoplastic agent, is associated with dose-related pulmonary toxicity, including interstitial pneumonitis and fibrosis. Bleomycin is a glycopeptide antibiotic that binds to DNA, produces free radicals, and results in DNA single-strand breaks in cancer cells.[1] Bleomycin preferentially damages lung tissue because of a deficiency of bleomycin hydrolase within the lungs. Without proper clearance, these free radicals trigger an inflammatory cascade which leads to alveolar damage, inflammation, and eventually fibrosis. This is further exacerbated by high oxygen tension in the lungs, which fuels free-radical reactions.[2] Patients with bleomycin exposure undergoing cardiac surgery are at increased risk for perioperative pulmonary complications, particularly when exposed to high inspired oxygen concentrations and large fluid shifts during cardiopulmonary bypass (CPB).

Case Presentation

60-year-old male with medical history of HFrEF, HTN, CAD s/p PCI, Hodgkin's Lymphoma s/p ABVD chemotherapy who presented with chest pain and found to have NSTEMI. He was started on a heparin drip and underwent cardiac catheterization which showed severe multi-vessel disease. Cardiothoracic surgery was consulted for CABG evaluation. Transthoracic echocardiography showed LVEF 41%, LV wall motion abnormality, and no significant valvular disease. Pulmonary function tests were not available. Laboratory values were unremarkable. Induction of anesthesia was achieved with midazolam, fentanyl, lidocaine, etomidate, and rocuronium. FiO2 was increased to 0.4 for 3 minutes allowing for pre-oxygenation then reduced to 0.3 after intubation. Ventilation goals included a lung-

protective strategy (tidal volumes 6–8 mL/kg). Perfusion targeted PaO2 80-100 mm Hg when coming off CPB. Post-CPB, FiO2 was limited to 0.3 or less. Restrictive fluid strategy and cell salvage were employed. There was no clinical evidence of acute lung injury post-CPB. The patient was transported to the ICU intubated and sedated in stable condition.

Discussion

Postoperative adult respiratory distress syndrome (ARDS) in patients with prior bleomycin exposure was first described in 1978 when researchers attributed 5 deaths to oxygen toxicity. They found even modest increases in intraoperative FiO2 in patients with bleomycin exposure resulted in significant pulmonary toxicity and even death.[3] This study also attributed liberal use of crystalloids as a potential cause of mortality in this population. The exact pathophysiology of bleomycin-induced pulmonary toxicity is not well understood. However, it is thought to be a combination of a deficiency of bleomycin hydrolase in lung tissue, oxidative damage via free radicals, and inflammatory cytokines. Even in patients with no recent exposure to bleomycin, it is possible that bleomycin-damaged lung tissue is unable to scavenge oxygen free radicals, which is further exacerbated by high-inspired oxygen concentrations during anesthesia.

Conclusion

Patients with previous bleomycin exposure undergoing CABG require meticulous anesthetic planning to reduce the risk of perioperative pulmonary complications. The key strategies include limiting FiO2, lung-protective ventilation, maintaining fluid balance, and close postoperative surveillance.

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#FR 2 – Kiosk 26 – Poster 5

From Arthroplasty to Ischemia: Emergent Revascularization Following Popliteal Artery Occlusion

Presenting Author: Hank Weinstock, MD, MPH, Anesthesia Resident, University of Illinois Peoria

Introduction

Popliteal artery occlusion following total knee arthroplasty (TKA) is a rare clinical entity which can portend catastrophic vascular complications if not addressed expeditiously.1 While randomized controlled trials have traditionally shown no differences in graft patency or short-term mortality between patients receiving local, neuraxial, or general anesthesia when undergoing lower extremity revascularization,2,3 evidence to guide anesthetic considerations in emergent cases does not exist. We present a case of acute popliteal artery thrombosis following routine TKA leading to acute limb ischemia and necessitating emergent femoral to below-knee popliteal artery bypass.

Case Presentation

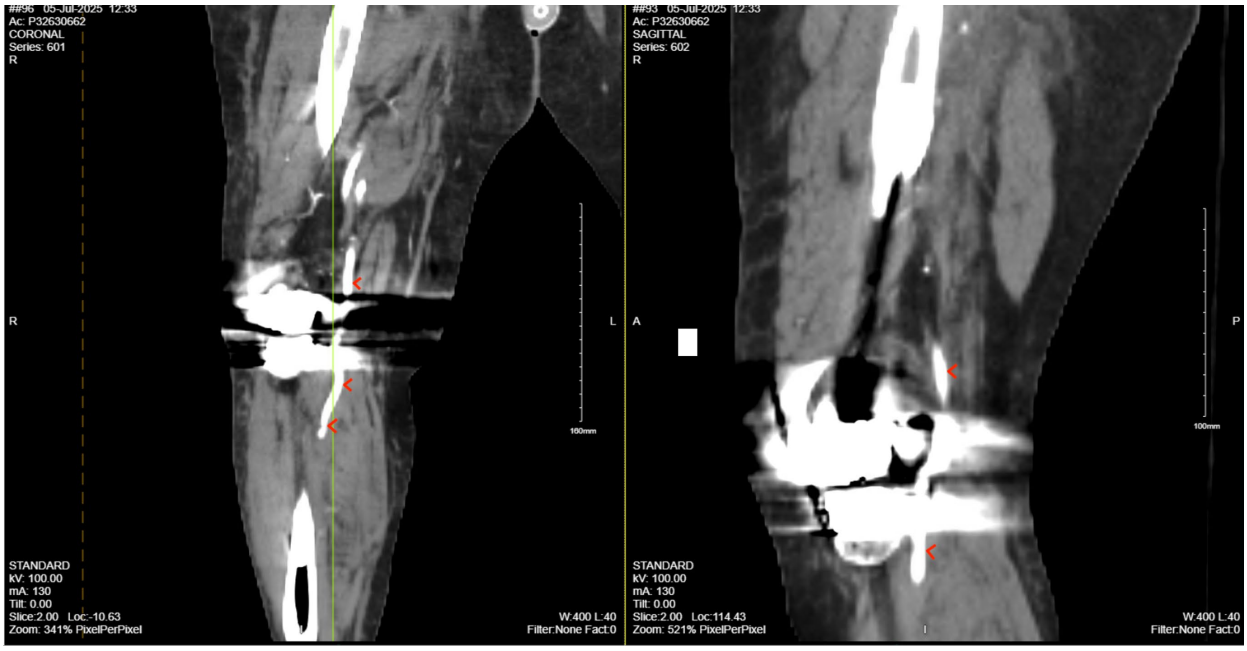
A 66-year-old female with past medical history significant for obesity, type 2 diabetes mellitus, hypertension, hyperlipidemia, and arthritis underwent routine right TKA at an outside hospital in our region. Review of the hospital record demonstrated that the procedure was completed uneventfully, and adequate surgical hemostasis was achieved with use of both bipolar electrocautery and tranexamic acid administration. In the post anesthesia care unit (PACU), nursing staff were unable to obtain right lower extremity (RLE) pulses or Doppler signals in either the popliteal fossa or distally in the right dorsalis pedis (DP) or posterior tibial (PT) distributions. In the first two hours postoperatively, the patient developed severe lower extremity pain, paresthesias radiating to the foot, and distal sensory loss. Urgent Doppler imaging was obtained in the PACU which revealed trickle flow in the proximal popliteal artery with total occlusion of the mid and distal popliteal artery, with 0 cm/s total flow in the proximal right popliteal artery. Given concern for acute postoperative popliteal artery thrombosis, the patient was subsequently transferred to our institution for emergent surgical intervention and the decision was made to proceed to the operating room for emergent right femoral to below-knee popliteal artery bypass grafting. Rapid sequence intubation (RSI) was performed, and general anesthesia was maintained using sevoflurane with intermittent boluses of fentanyl for analgesia. A phenylephrine infusion was instituted to maintain mean arteriolar pressures (MAP) of greater than 65, and was discontinued upon completion of the case prior to extubation. Computed tomography (CT) angiography obtained on postoperative day two revealed expected postoperative changes with adequate distal three-vessel runoff (Figure 1), and the patient was ultimately discharged home on postoperative day 13 in excellent condition with minimal postoperative pain. Doppler imaging of the graft obtained three months postoperatively in the outpatient setting demonstrated mildly elevated velocities in the femoral-popliteal artery bypass graft indicating graft patency, along with triphasic waveforms noted in distal graft outflow (Figure 2).

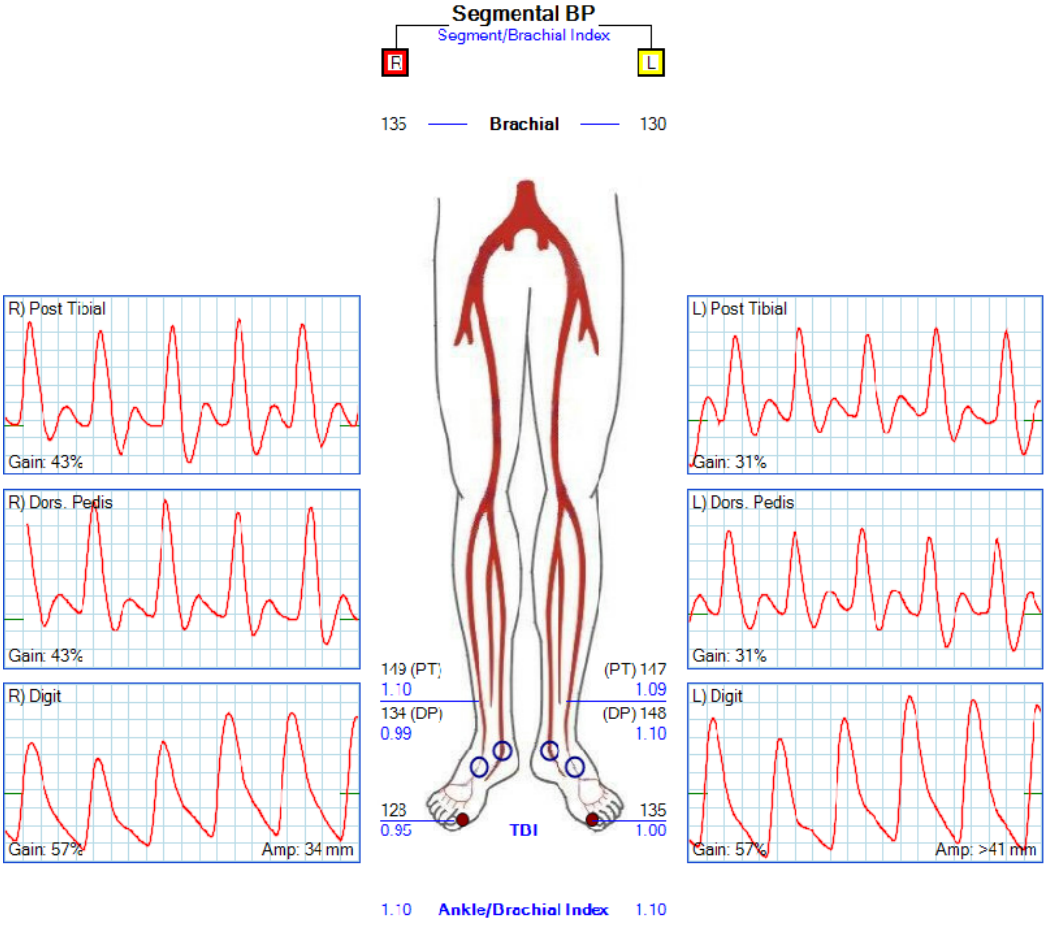
Discussion

In cases of acute popliteal artery thrombosis following TKA, urgency is paramount and prompt

revascularization of the affected extremity is critical. Vascular anesthesiologists must take particular care in selecting an anesthetic technique which both considers patient comorbidities and prioritizes hemodynamic stability to maximize graft survival.

Images





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#FR 2 – Kiosk 26 – Poster 6

Severe Systolic Anterior Motion of the Mitral Valve Following Resection of Hepatic, Inferior Vena Cava and Right Atrial Mass Under Circulatory Arrest

Presenting Author: Monica Liu, MD/MBA, Resident Physician, New York Presbyterian - Weill Cornell

Introduction

Very few case reports describe surgical resection of hepatocellular carcinoma (HCC) invading the inferior vena cava (IVC) and right atrium (RA)[1,2]. Here, we describe a left hepatectomy, RA and IVC mass excision and IVC reconstruction, performed under deep hypothermic circulatory arrest (DHCA), which resulted in new-onset, severe and treatment-refractory systolic anterior motion (SAM) of the mitral valve (MV).

Case Presentation

A 68-year-old female with hypertension, diabetes, and chronic hepatitis B presented after 1 month of epigastric pain and lower extremity edema. She was found to have a 4.7cm HCC mass, occluding the IVC and extending into the RA. Pre-CPB transesophogeal echo (TEE) showed normal biventricular (BiV) size and function, with IVC occlusion and a 5.2 x 2.8cm RA mass, abutting the inter-atrial septum. The tricuspid valve was unaffected. The MV was normal, with neither evidence of nor risk factors for SAM. Following the hepatectomy, the RA and IVC masses were excised and the IVC reconstructed. CPB time was 144 minutes, aortic cross-clamp time 40 minutes and DHCA time 32 minutes. Epinephrine was started empirically at 2mcg/min prior to separation from CPB. The patient was observed to have hyperdynamic BiV function and new severe SAM, as evidenced by holosystolic contact of the anterior MV leaflet and septum, LVOT flow acceleration and severe posteriorly-directed mitral regurgitation (MR). Epinephrine was discontinued and the patient was volume resuscitated. Post-CPB cardiac index (CI) by thermodilution and Fick was 1.3L/min/m2. In addition to SAM, the patient demonstrated severe coagulopathic bleeding, hypovolemia and vasoplegia. She received 11 units of red blood cells, 3 units of fresh frozen plasma, 3 units of cryoprecipitate, 1 unit of platelets, 4 liters of plasmalyte, and 1 liter of albumin 5%. Phenylephrine and vasopressin infusions were uptitrated, and norepinephrine reduced. SAM and MR improved from severe to moderate and CI increased to 1.7L/min/m2. Due to severely edematous bowels, the abdomen was left open with a “vac” dressing in place. In the ICU postoperatively, CI remained 1-1.5 L/min/m2. TEE on post-operative-day (POD) 1 showed persistent moderate SAM and MR, which did not improve in following days.

The patient developed liver- and multi-organ failure and expired on POD4.

Discussion

To our knowledge, this is among the first reported cases of severe SAM developing following this resection of HCC lesions from the liver, RA and IVC under DHCA. This case is remarkable for multiple challenges and competing priorities. SAM was exacerbated by vasoplegia and volume depletion from hemorrhage and third-spacing. Significant volume resuscitation was needed to help reduce SAM, but prevented abdominal closure and likely contributed to hepatic congestion and injury. Due to the treatment-refractory nature of SAM in this patient, we suspect that RA mass resection may have altered the patient's cardiac geometry and led to intractable, hemodynamically significant SAM. Future studies are needed to assess the cardiac risk factors and complications associated with this type of operation.

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#FR 2 – Kiosk 26 – Poster 7

Echocardiographic Characteristics of Massive Vegetations Mimicking Atrial Myxoma Demanding Valvular Replacement Complicated by Retention of Surgical Devices: A Case Report

Presenting Author: Xiangyu Zhao, MD, Resident Physician, Brown University Health

Introduction

Intra-atrial masses are commonly seen with atrial myxoma or infective endocarditis vegetations. Given the overlaps in clinical symptoms, such as fever, fatigue, myalgia, and systemic embolism, echocardiographic characteristics are worth discussing in the context of prompt valvular repair/replacement to guide intraoperative hemodynamic monitoring and postoperative management[1].

Case Presentation

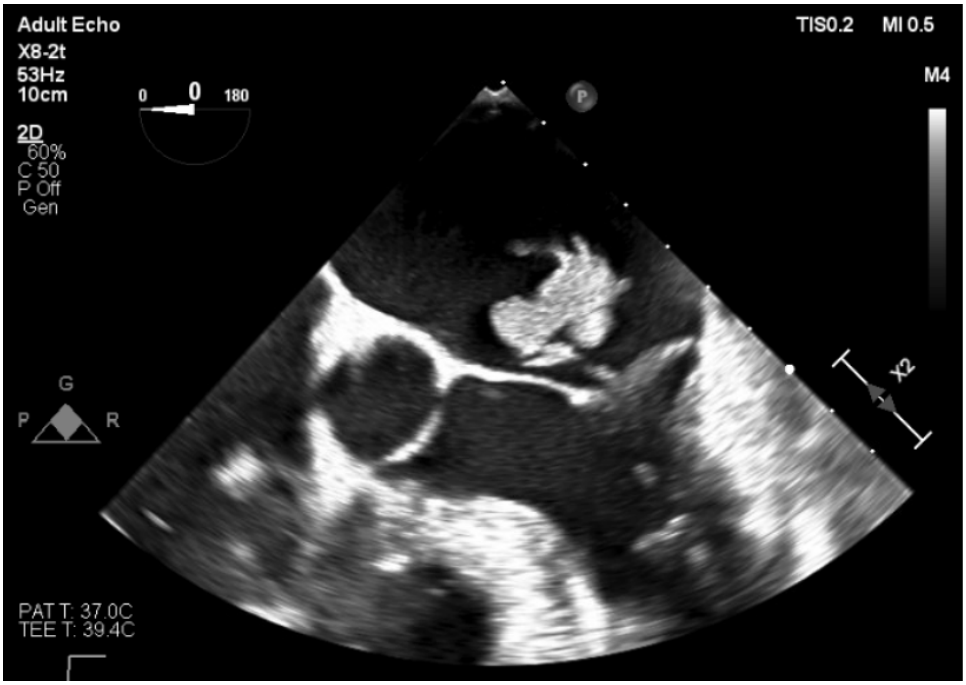
Patient is a 48-year-old male presenting to our facility for lethargy and dizziness. Initial workup completed by the critical care medicine team demonstrated splenomegaly with splenic infarcts, bibasilar cavitations in the right lower lobe, and distal left middle cerebral artery mycotic aneurysms in the setting of a heterogeneous, multilocular, pedunculated mass on the anterior leaflet of the mitral valve measuring 43 x 22mm and a mass of similar presentation on the tricuspid valve (Figure 1 & 2). Severe regurgitation was noted across the mitral and tricuspid valves. Blood cultures were positive for *group B streptococcus*, and Gram stain from valvular mass samples demonstrated Gram-positive cocci. However, no growth was identified from the vegetation cultures obtained intraoperatively. The mitral (MV) and tricuspid valve (TV) replacement surgery demonstrated a massive lobulated vegetation on P2-P3 leaflets with a loculated abscess between the papillary muscles and the MV annulus. The surgeon subsequently implanted the MITRIS RESILIS 27 mm mitral and 31 mm tricuspid valves. The surgical course was complicated by retention of the removable holding apparatus attached to the TV as identified on transesophageal echocardiography (TEE) (Figure 3-5). Patient's treatment course was further complicated by recurrent bacteremia after surgery with *Enterococcus faecalis* two months and four months after surgery, respectively, that required a prolonged course of ampicillin and ceftriaxone for synergy.

Discussion

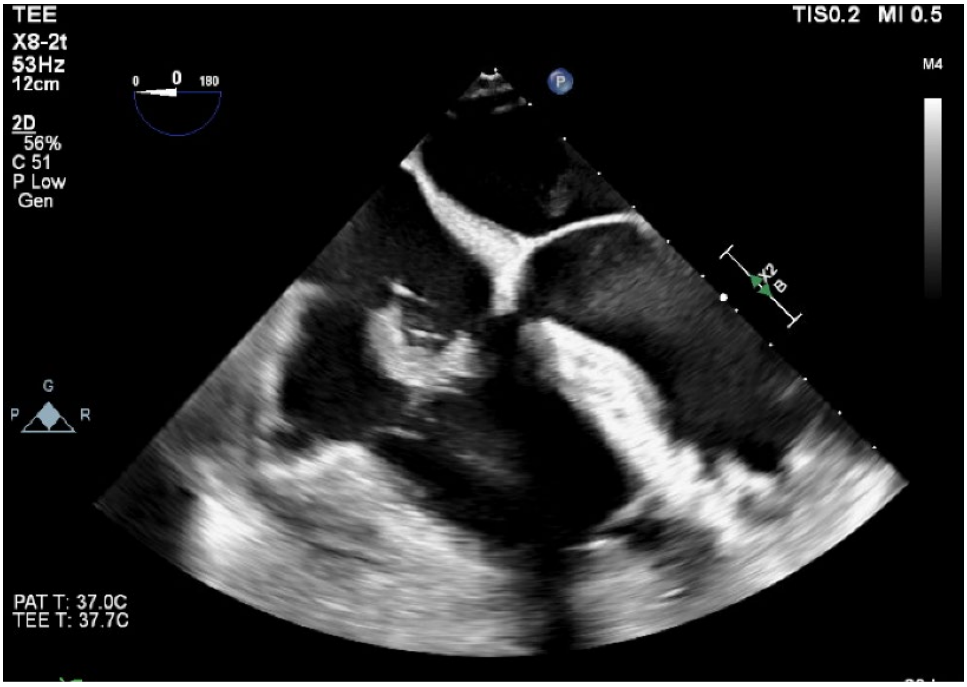
Without a definitive history of intravenous injections with recreational substances, the most likely etiology of his valvular vegetations in the setting of group B *Streptococcus* bacteremia remains invasion from oral microbial flora through periodontal infections. Atrial myxoma remained a likely diagnosis given the presentation of a pedunculated mass measuring over 30mm, despite myxoma favoring attachment by a pedicle to the interatrial septum, particularly at the fossa ovalis[1]. Echocardiographic findings consistent with vegetations include an amorphous, irregular morphology with ill-defined borders, while myxoma usually presents a globular, polypoid, or papillary mass[2]. Obstruction of the inflow of MV/TV from atrial myxoma could lead to functional mitral/tricuspid stenosis and present as chronic heart failure with a constellation of dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea, and acute pulmonary edema in extreme cases[3]. Alternatively, valvular regurgitation could be secondary to impedance of coaptation, flail leaflet from

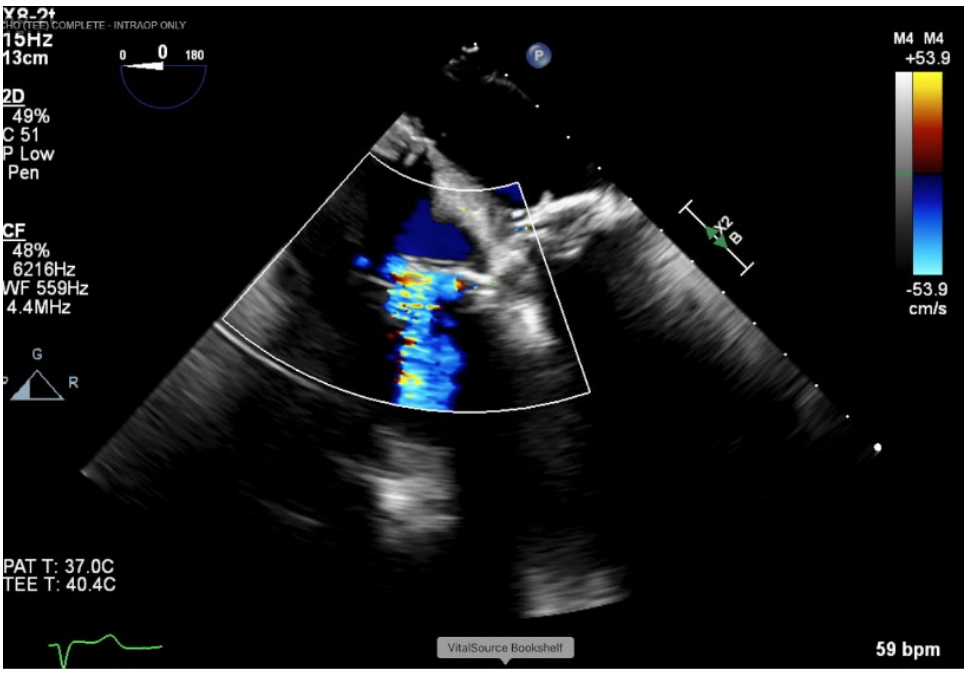
destruction of chordae tendineae, or valvular perforation in the setting of massive vegetations from infective endocarditis[3].

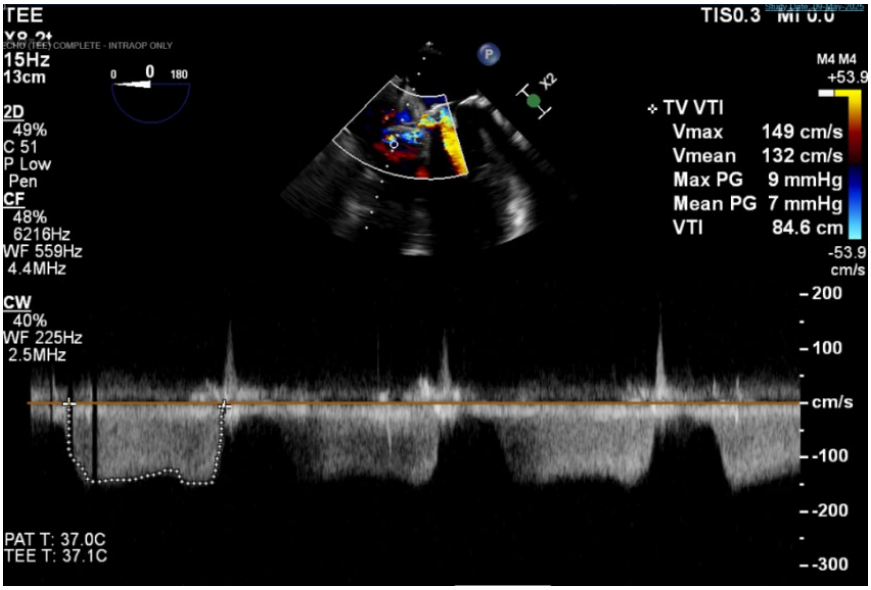
Images

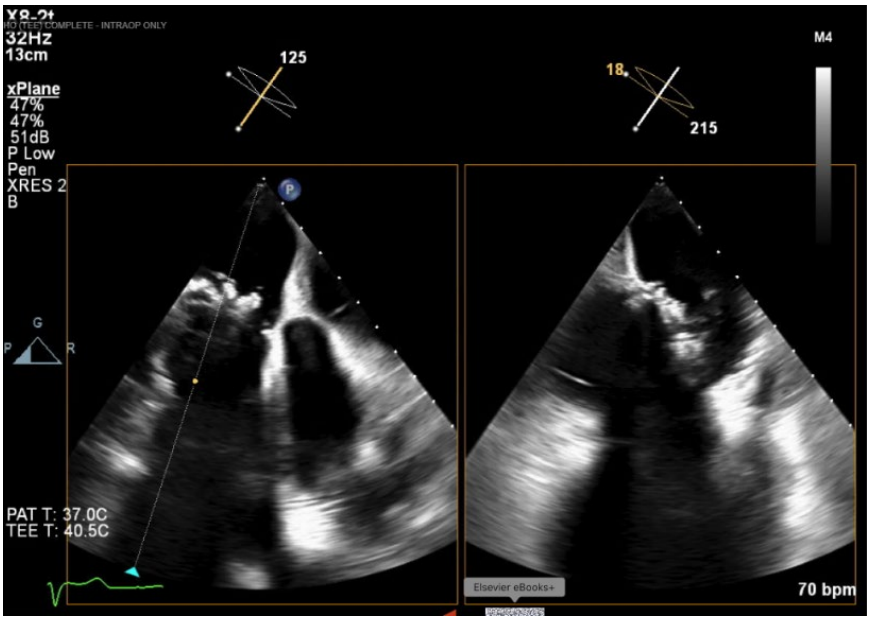





TEE
X8-2t
53Hz
12cm
2D
56%
C 51
P Low
Gen
TIS0.2 MI 0.5
M4
PAT T: 37.0C
TEE T: 37.7C


15Hz
13cm
2D
49%
C 51
P Low
Pen
CF
48%
6216Hz
WF 559Hz
4.4MHz
M4 M4
+53.9
-53.9
cm/s
PAT T: 37.0C
TEE T: 40.4C
59 bpm





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FELLOW AND RESIDENT ABSTRACTS

Poster Session 3

Saturday, April 25, 2026 | 3:30 PM – 4:30 PM

#FR 3 – Kiosk 1 – Poster 1

Azygous Vein Rupture Following Swan-Ganz Catheter Manipulation During Ventricular Assist Device Implantation and Tricuspid Valve Repair

Presenting Author: Michael R. Dougherty, DO, Fellow, Thomas Jefferson University Hospital

Introduction

Pulmonary artery (PA) catheterization remains a cornerstone of hemodynamic monitoring in patients undergoing ventricular support device placement. Although complications such as arrhythmias, catheter malposition, knotting, and pulmonary artery rupture[1] are well described, azygous vein injury is rare. Distortion of right-sided anatomy, may predispose patients to aberrant catheter trajectory. We present a case of azygous vein rupture following PA catheter manipulation during combined left and right ventricular assist device (LVAD/RVAD) implantation and TV repair.

Case Presentation

A 76-year-old man with ischemic cardiomyopathy (LVEF 10%), mitral regurgitation, and severe tricuspid regurgitation underwent LVAD implantation with concomitant TV repair. The PA catheter was withdrawn retrograde prior to TV repair to facilitate exposure. After annuloplasty, the catheter was directed to the right ventricle under surgeon visualization. Following LVAD placement, inability to wean from cardiopulmonary bypass (CPB) in the setting of RV failure prompted RVAD implantation. The PA catheter was abandoned for new catheter placement. Multiple attempts to place the new catheter using waveform and transesophageal echocardiography (TEE) guidance were unsuccessful, and the catheter was withdrawn to the start of the thermodilution coils. During chest closure, right-sided pleural output (>600 mL) accumulated. TEE revealed a large right pleural effusion. Emergent re-exploration via sternotomy demonstrated the PA catheter protruding from a longitudinal tear in the azygous vein adjacent to the superior vena cava. The injury was surgically repaired, achieving hemostasis. The patient received substantial blood product resuscitation and ultimately stabilized, was extubated on postoperative day (POD) 3, decannulated from RVAD support on POD 5, and discharged to rehabilitation on POD 48.

Discussion

This case highlights an uncommon complication of PA catheter insertion and manipulation. Anatomically, the azygous vein arches over the right main bronchus and drains into the superior vena cava 1–3 cm above the right atrial junction. Iatrogenic rupture likely results from misdirection, particularly when the catheter enters the azygous vein rather than the right atrium. Predisposing factors include any distortion of anatomy from prior cardiac surgery, elevated right-sided pressure, and excessive advancement of the catheter without continuous waveform confirmation.2 Secondary surgical factors, including re-operation or right-sided mechanical support, may alter the anatomy and loading conditions of right-sided heart structures. In this case, TV repair may have limited the ability to cross the valve. In addition, placement of the RVAD may have further complicated PA catheter placement by modifying right-sided preload. Continuous TEE monitoring may help identify aberrant catheter paths in high-risk patients. Mortality from venous rupture is

high if diagnosis is delayed.3 Institutional review following this event identified important differences in introducer-to-coil distances between continuous cardiac output PA catheters and traditional thermodilution catheters, prompting protocol modifications that limit advancement depth prior to balloon inflation.

Images

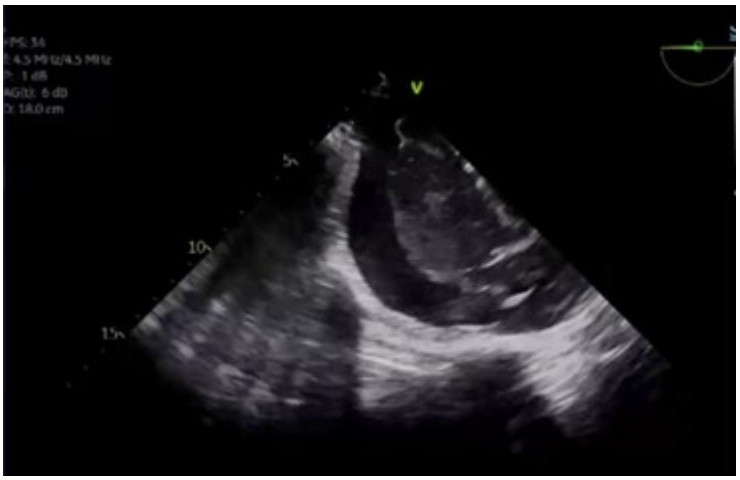


Figure 1: TEE demonstrating a large right pleural effusion

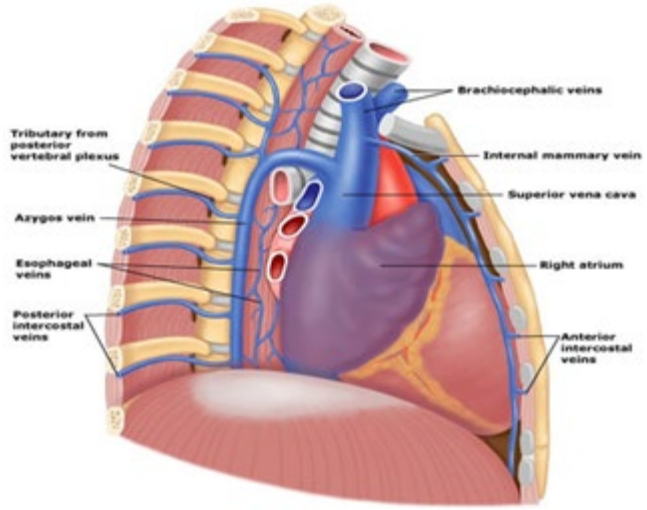


Figure 2: Azygous vein anatomy in relation to the superior vena cava

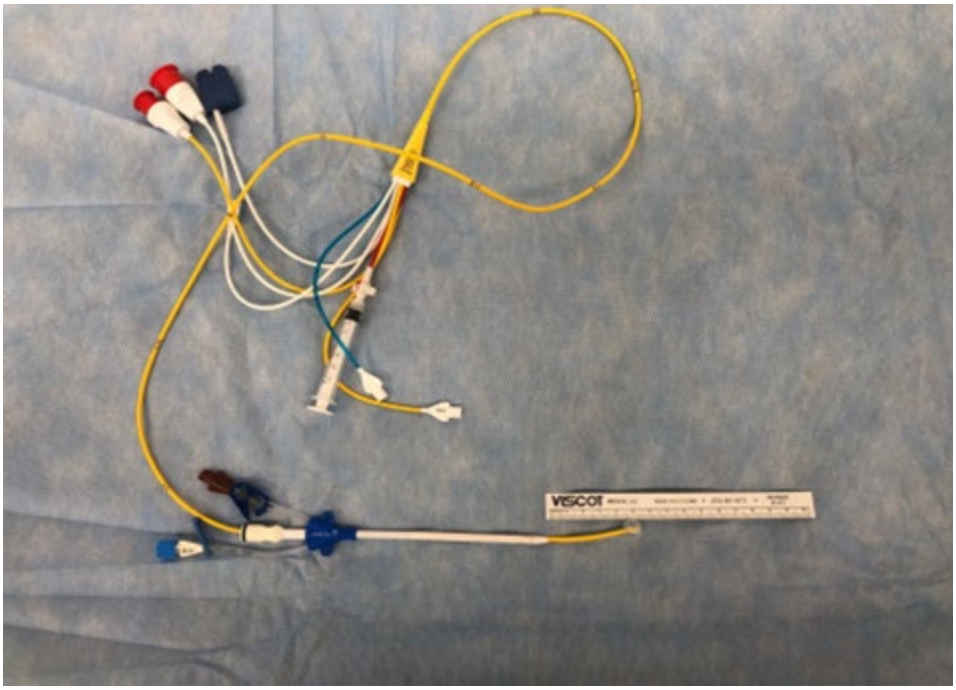


Figure 3: Approximate measure (cm) of CCO PA catheter from sheath introducer when loaded to end of thermodilution coil

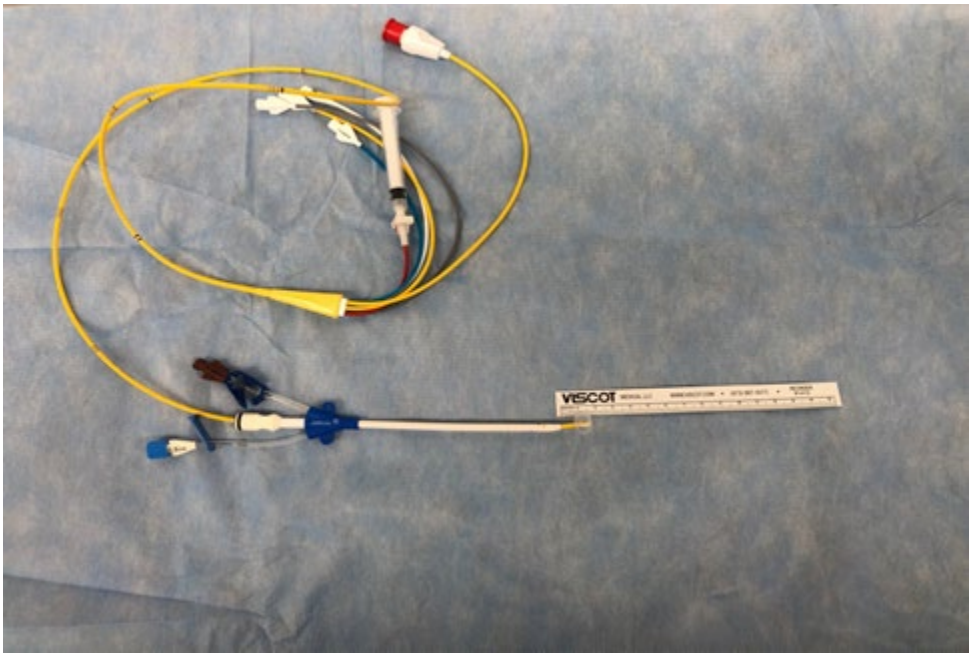


Figure 4: Approximate measure (cm) of thermodilution PA catheter from sheath introducer when loaded to 20 cm

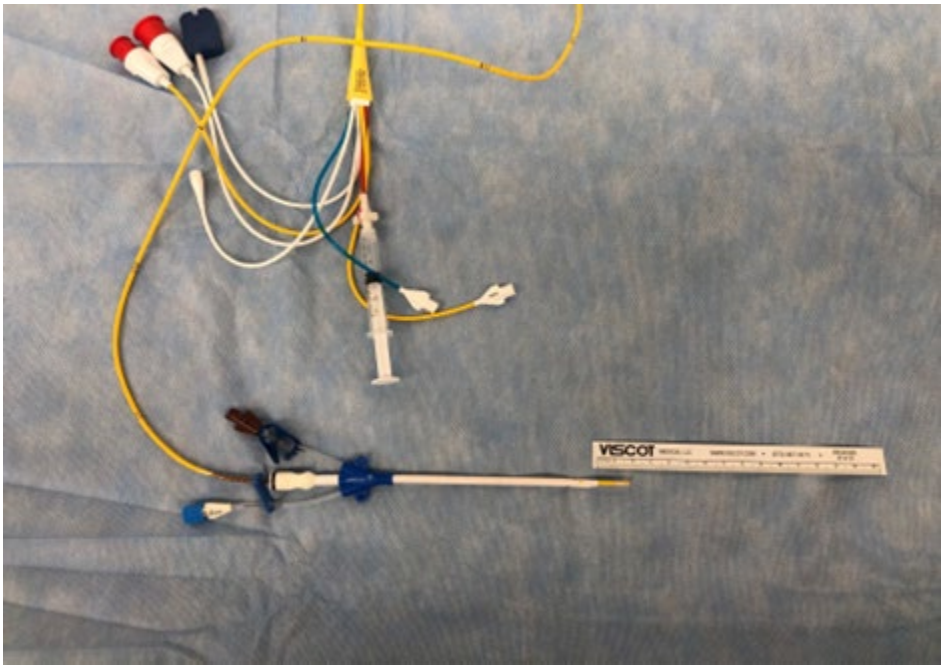


Figure 5: Approximate measure (cm) of CCO PA catheter from sheath introducer when loaded to 20 cm

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#FR 3 – Kiosk 1 – Poster 2

Migration of left atrial appendage occluder leading to severe mitral regurgitation and right heart failure

Presenting Author: Michael J. DePalma, MD, Resident, Hospital of the University of Pennsylvania

Introduction

Left atrial appendage occlusion (LAAO) devices including the Watchman and Amulet are used to decrease risk of stroke in patients with atrial fibrillation who have contraindications to oral anticoagulants. Migration of these devices is a potentially devastating complication that can pose intra-operative management challenges. This report highlights a case of Amulet device embolization which resulted in hemodynamically unstable acute mitral regurgitation and right heart failure.

Case Presentation

A 74 year old male with history of prior liver transplant, coronary artery disease status post multi-vessel coronary artery bypass grafting, complete heart block managed with a dual chamber permanent pacemaker, and persistent atrial fibrillation underwent Amulet implantation. Of note, Watchman implantation had been attempted unsuccessfully two months prior due to unfavorable left atrial appendage anatomy. On day 1 following Amulet implantation, the patient complained of mild dyspnea. Subsequent chest x-ray suggested that the Amulet had migrated, and point of care transthoracic echocardiography showed that the device was freely mobile in the left atrium. Given the possibility of further migration of the device, the patient was taken emergently to the electrophysiology lab for retrieval of the device. A pre-induction radial arterial line was placed to enable rapid detection of hemodynamic instability. Since the patient had recently eaten, he was induced via rapid sequence induction. Low dose esmolol was administered with induction to prevent an increase in cardiac output with laryngoscopy which could lead to further device migration. Following induction, the patient became bradycardic and suffered brief PEA arrest, with ROSC achieved within 20 seconds. Transesophageal echocardiography (TEE) was performed which showed the Amulet device was tethering the posterior mitral valve leaflet open and preventing closure of the anterior leaflet. This created torrential holosystolic mitral regurgitation, thereby leading to flash pulmonary edema and acute RV failure. Following ROSC, the patient was initially hypoxic, but this slowly improved over time with positive pressure ventilation and epinephrine infusion. The Amulet device was retrieved via transseptal approach, which immediately improved the patient's hemodynamics. The patient ultimately recovered well and was discharged.

Discussion

This case report demonstrates the rapid physiologic changes that can occur with intra-cardiac migration of LAAO devices, which highlights a few key lessons: 1) In patients who have had prior sternotomies, it may be prudent to have ECMO placeholders prior to induction, since, in this case, had ROSC not been achieved, initiation of mechanical circulatory support would have likely been delayed. 2) Decreasing cardiac output to prevent device migration may incur greater risks than it is worth in such a dynamic situation. 3) In retrospect, the patient's pacemaker proved to be beneficial, as without it, the bradycardia that developed with induction

would have likely progressed to an asystolic arrest rather than PEA, which likely protected the RV until epinephrine could be administered. Ultimately, device embolization is a dynamic complication that requires flexibility and the highest level of contingency planning.

Images



Figure 1. AP Chest X-ray suggesting migration of Amulet device into left ventricle



Figure 2. Transesophageal Echocardiography demonstrating tethering of the posterior mitral valve leaflet open with associated torrential holosystolic mitral regurgitation

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#FR 3 – Kiosk 1 – Poster 3

Pseudoaneurysm Following Primary Surgical Repair of Group B Strep Endocarditis Managed with Interventional Coiling

Presenting Author: Ryan T. Casey, Resident, Lahey Hospital and Medical Center

Introduction

49-year-old female who presented with a 1-month history of neck pain developing AMS, hypoxia, hypotension, and chest pain while in the ED. Four months prior, she delivered twins in Uganda with a remote history of GBS bacteremia. She was found to have gram+ bacteremia with AV vegetation and root abscess. She underwent AVR + aortic root replacement with cultures GBS+. In the CTICU, she developed worsening pulmonary edema and was found to have MV perforation related to recurrent bacteremia. She underwent replacement with patch closure of aortic root to LA fistula. Following her initial procedures, she had a prolonged recovery with steady improvement.

Case Presentation

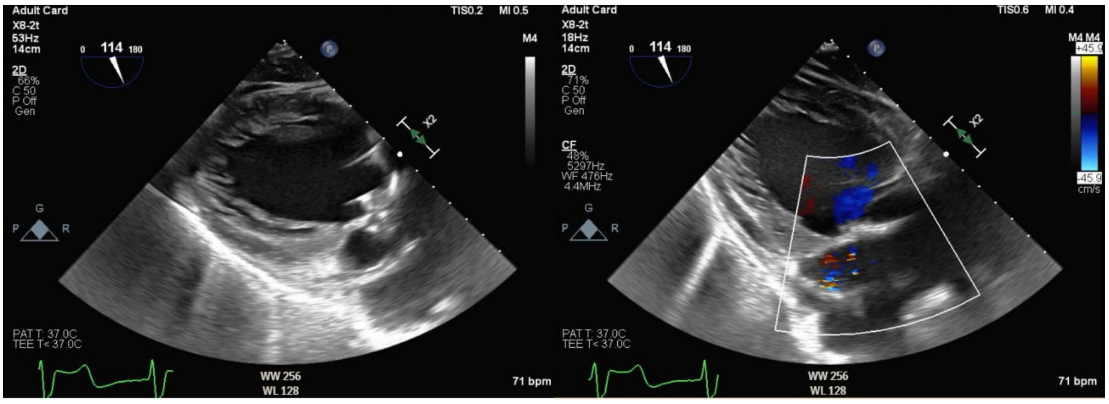
Several months after her initial presentation, she presented with anginal chest pain and underwent LHC. This showed dynamic, external compression of her LMCA confirmed to be caused by an aortic pseudoaneurysm. She was planned for endovascular coiling as the mortality/morbidity of third sternotomy was viewed to be prohibitively high. She was brought to the cath lab and general anesthesia was induced. Groin lines were placed for access and monitoring. The procedural team passed through the IAS under TEE guidance. Upon crossing the MV, severe MR was noted on TEE along with precipitous drop in BP. The procedural team was unable to enter the pseudoaneurysm cavity from this approach and all catheters were withdrawn. The team then utilized their arterial sheath to cross the aortic valve and attempted to enter the pseudoaneurysm from a retrograde approach. This allowed successful entrance and 21 penumbra coils were delivered with fluoroscopic and TEE obliteration of the pseudoaneurysm and communication point. LMCA angiography was difficult to visualize, but distal flow was deemed adequate. She was emerged from general anesthesia, extubated, and brought to recovery. She was discharged to acute rehab on post procedure day 2 and continues to do well to date.

Discussion

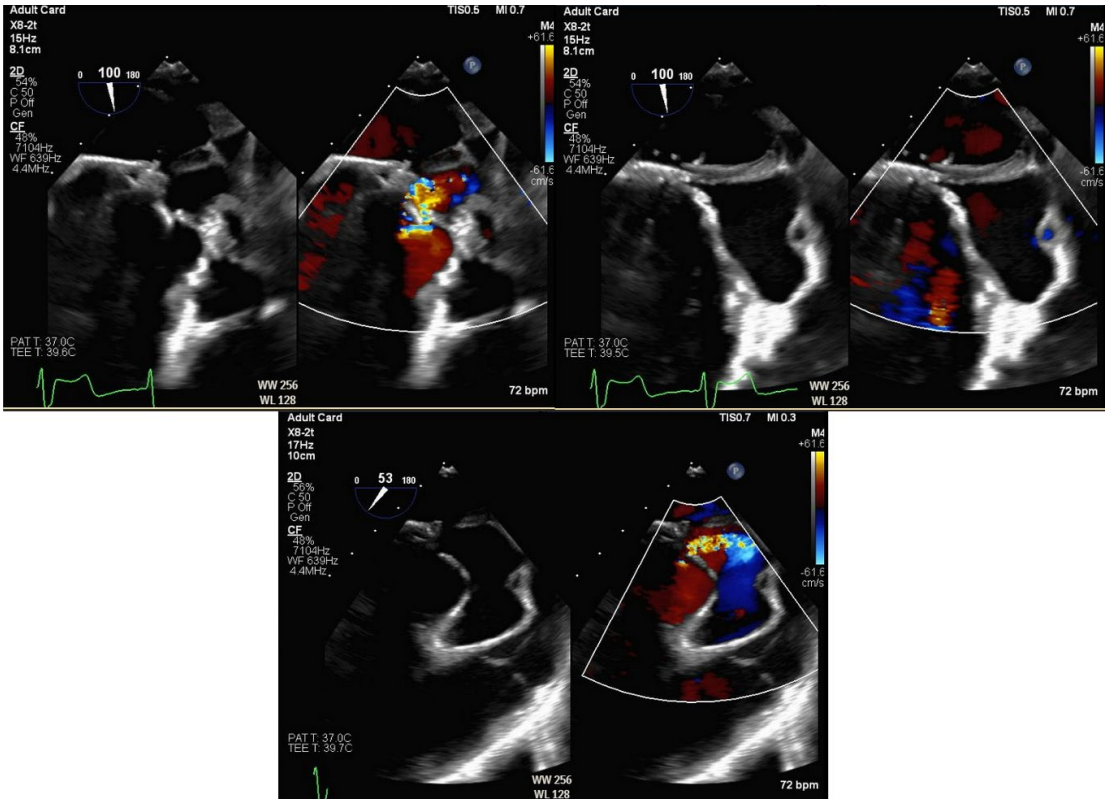
Endocarditis is a major cause of both morbidity and mortality warranting prompt management to mitigate the damage done by the causative organisms. Streptococcus agalactiae is a relatively common cause of endocarditis and is associated with the perinatal period. Endocarditis of the AV can lead to abscess from erosion of the perivalvular apparatus which can develop into pseudoaneurysm and fistulae. Pseudoaneurysm can arise from any location within the heart particularly related to graft material or prior surgical sites. Presenting symptoms of pseudoaneurysm can range from fulminant heart failure to insidious murmurs. Management of abscess, pseudoaneurysm, and fistulae can vary from a surgical approach with median sternotomy and bypass to minimally invasive percutaneous repairs. Many imaging modalities are beneficial for both the diagnosis and intraoperative management of these cases. TTE and TEE are viable early

options to identify endocarditis and the sequelae of endocarditis. CT and MRI can then aid with surgical planning. TEE proved invaluable in identifying the communication with the pseudoaneurysm as the flow dynamics under color doppler were easier to visualize than contrast flow under fluoroscopy. This case demonstrates the complex nature of infective endocarditis and the varied sequelae of this destructive disease state.

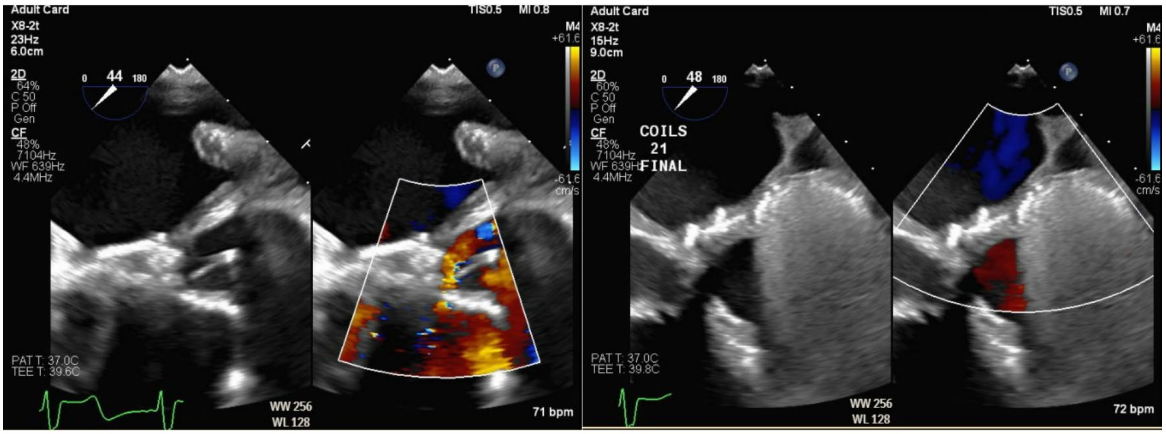
Images



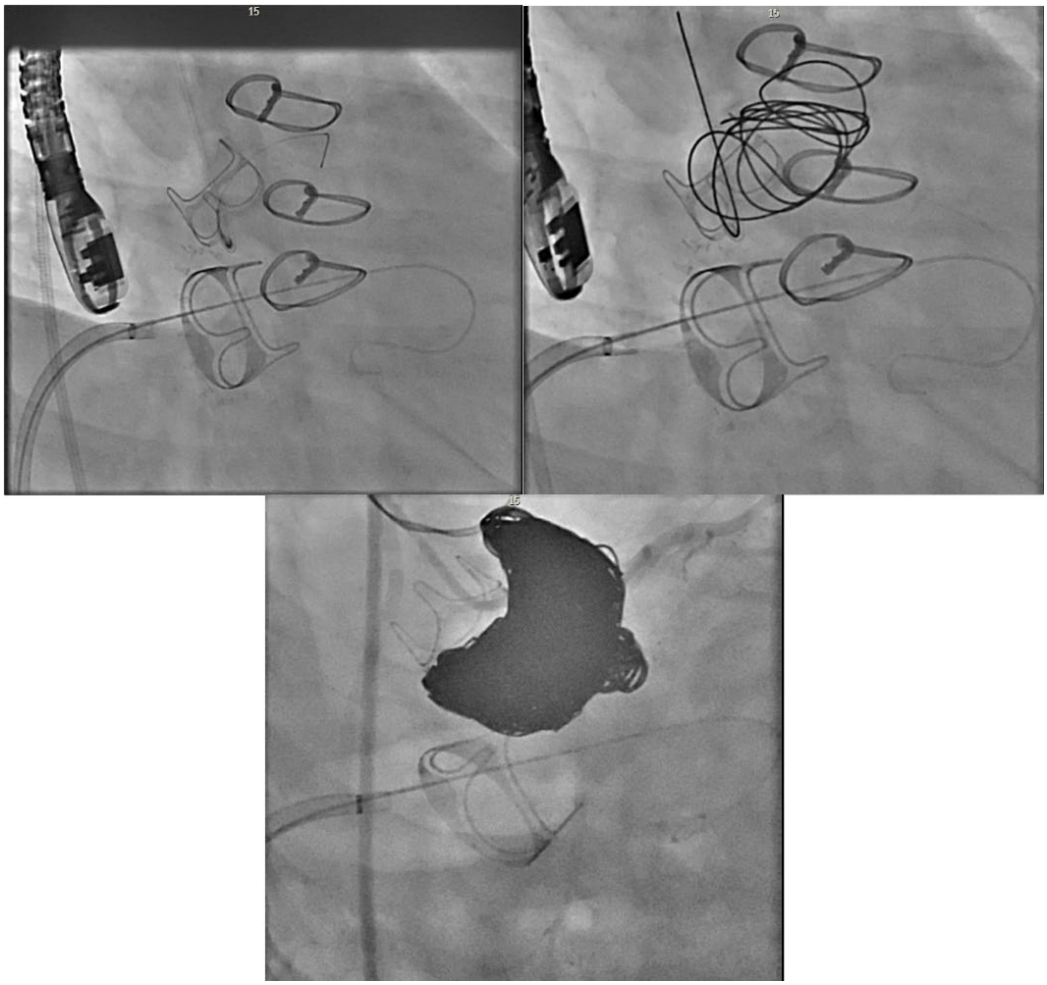
Pseudoaneurysm seen in transgastric TEE



Pseudoaneurysm surrounding LMCA with color flow demonstrating communication point



Pseudoaneurysm communication with color flow and obliteration following coiling



Fluoroscopic view of entry, coil deployment, and obliteration of pseudoaneruysm

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#FR 3 – Kiosk 1 – Poster 4

Sequential Bi-Coronary Malperfusion Unmasked on Post-Bypass TEE During Type A Dissection Repair

Presenting Author: Brock Lingle, MD, Tulane University

Introduction

Coronary malperfusion complicates 3-13% of Type A aortic dissections, but bi-coronary involvement is extremely rare (~1-2%) and associated with high mortality. These events may evolve dynamically during repair and are often first detected on post-bypass TEE. We present a rare case of sequential RCA and LMCA malperfusion following Type A dissection repair.

Case Presentation

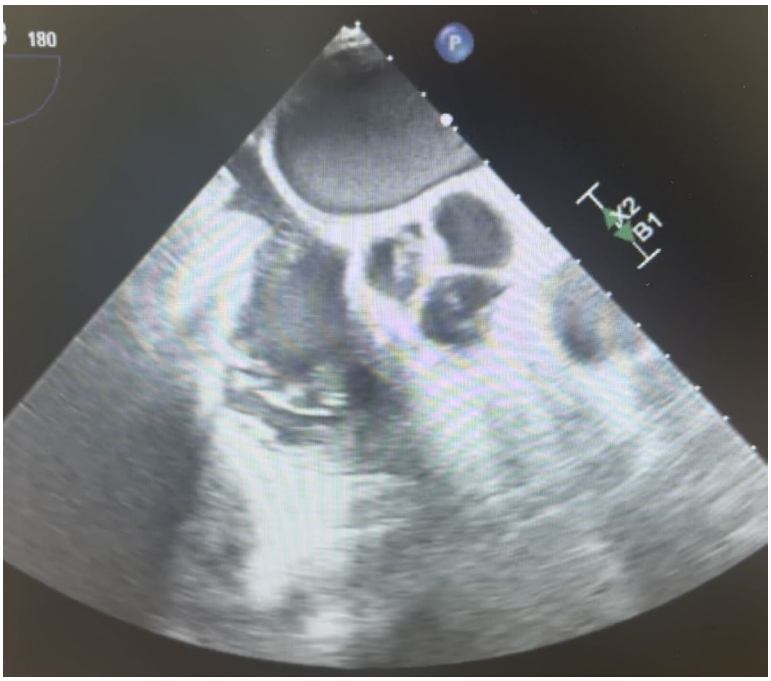
72yo female with PAF, LAD stent, HFrecEF, COPD and HTN presented with hemoptysis, dyspnea, and chest pain. Initial vitals were BP 91/40, HR 128, and SpO_2 82% on CPAP. Troponin was 138 ng/L and LHC/RHC showed 50% RCA stenosis, preserved RV function, and EF 50%. CTA showed a Type A dissection extending into the mid-arch and bilateral common carotid arteries, with an aberrant right subclavian artery. In the OR, pre-bypass TEE demonstrated new systolic dysfunction; EF 30–35%, normal RV function, and severe inferior/ inferoseptal hypokinesis, dissection flap extending into the right and non-coronary sinuses, and a 5.4cm dissection. After right axillary cannulation, sternotomy revealed hemopericardium. CPB was initiated and antegrade cardioplegia failed prompting direct ostial cardioplegia for arrest. The ascending aorta was resected to the sinotubular junction, the dissected R and non-coronary sinuses were reinforced, and the native valve was resuspended. DHCA was initiated for distal anastomosis. After cross-clamp removal, the heart developed a junctional rhythm for ~10 minutes. During rewarming on full CPB, the patient transitioned acutely from a junctional rhythm to a wide complex rhythm then asystole within one minute. Asystole was refractory to AV pacing (which produced electrical capture but no mechanical contraction), tactile stimulation, and multiple rounds of IV and intra-cardiac Epinephrine. Coronary Doppler flow appeared appropriate. ABG and electrolytes were normal. Post-bypass TEE revealed flap extension into the L coronary cusp and severe global biventricular dysfunction with new anterior and lateral akinesis despite pacing, consistent with left-sided coronary malperfusion in addition to pre-existing RCA dysfunction. After cross-clamp removal, the proximal anastomosis demonstrated bleeding, friability, and tearing from additional sutures. Given refractory myocardial dysfunction and persistent bleeding, root replacement or initiation of mechanical support was deemed non-viable.

Discussion

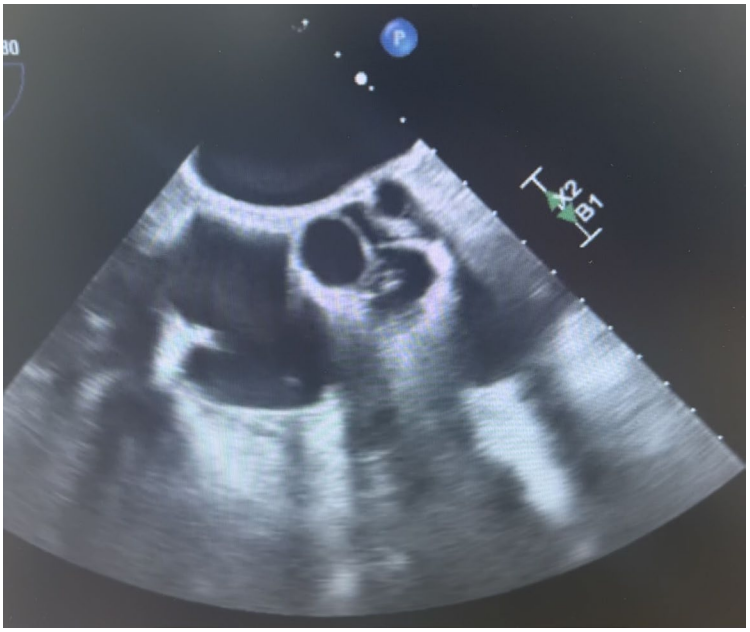
This case highlights the dynamic evolution of coronary malperfusion in Type A dissection repair and demonstrates how sequential RCA and LMCA involvement may only become evident after separation from CPB. LMCA compromise resulted from dissection progression into the left coronary sinus, supported by new anterior and lateral wall-motion abnormalities. Importantly, Doppler flow appeared normal, underscoring its limitations in this setting; false-lumen flow, intermittent flap motion, or partial ostial patency can all produce preserved Doppler signals despite true coronary malperfusion. This case emphasizes the need for

continuous reassessment of regional wall motion during rewarming, vigilance for abrupt rhythm instability as a surrogate for evolving coronary compromise, suspicion for malperfusion even when Doppler appears reassuring and the importance of coordinated communication during evolving pathology.

Images



TEE ME AV Short Axis



TEE ME AV Short Axis

References

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#FR 3 – Kiosk 1 – Poster 5

Identification of Complex Echodensity in Left Ventricular Outflow Tract Prior to Impella 5.5 Insertion

Presenting Author: Rosemary H. Kelley, MD, Resident Physician, CA3, Brigham and Women's Hospital

Introduction

Mechanical circulatory support (MCS) is being utilized with increasing frequency in the management of cardiogenic shock[1]. The Impella 5.5 is a surgically placed MCS device that may be used either in tandem with venoarterial extracorporeal membrane oxygenation (VA-ECMO) or as a stand-alone device to support the left heart [1-2]. Proper Impella positioning is critical to device functioning and can be achieved through fluoroscopy or echocardiography [1-2]. We present a case with a finding of an unusual left ventricular outflow tract (LVOT) echodensity, which highlights the importance of a thorough pre-procedural transesophageal echocardiogram (TEE).

Case Presentation

A 53-year-old male with a history of coronary artery disease status post multivessel coronary artery bypass grafting, end stage renal disease on hemodialysis, peripheral arterial disease, and poorly controlled diabetes complicated by recurrent gangrenous foot ulcers and osteomyelitis presented with fatigue, syncope, and hypotension, found to have acute biventricular failure. An intra-aortic balloon pump (IABP) was placed, and the patient was cannulated on peripheral VA-ECMO. On hospital day 5 the patient presented to the operating room for placement of axillary Impella 5.5 and evaluation for ECMO decannulation. After induction of general anesthesia, a pre-procedural TEE was performed while the patient was supported on VA-ECMO at 4 LPM, IABP 1:2, epinephrine and milrinone infusions. Findings were notable for reduced LV systolic function (EF 25%) with diffuse hypokinesis, diffusely thickened and calcified mitral valve leaflets with anterior leaflet flail at A2/A3 and associated moderate to severe eccentric mitral regurgitation. A calcified, echogenic, mobile echodensity was identified on the ventricular aspect of the base of the anterior mitral leaflet protruding into the LVOT (Figure 1). There was no color doppler flow acceleration in the LVOT to suggest flow obstruction. While the patient had no previously known diagnosis of endocarditis, he did have recurrent episodes of osteomyelitis and the echodensity appeared consistent with a healed vegetation. TEE findings were discussed in real time with patient's surgeon and intensivist. Due to the finding of a mobile echodensity in the LVOT along the planned trajectory of the Impella and high likelihood of either mass embolization or interference with Impella flow, the decision was made to abort planned placement of an Impella 5.5.

Discussion

This case underscores the importance of TEE for the safe placement of Impella devices. Absolute contraindications to the placement of an Impella 5.5 placement include: mechanical aortic valve, aortic valve endocarditis, free-floating/mobile thrombus in the LV, or stent in the ascending aorta/aortic dissection[1-2]. While a mobile echodensity in the LVOT is not included on the usual list of absolute contraindications, in this case it certainly posed significant risk and precluded Impella placement. A

thorough TEE exam prior to the placement of the Impella allowed the identification of the LVOT mobile echodensity and prompted multidisciplinary decision making with the surgical and critical care teams.

Images

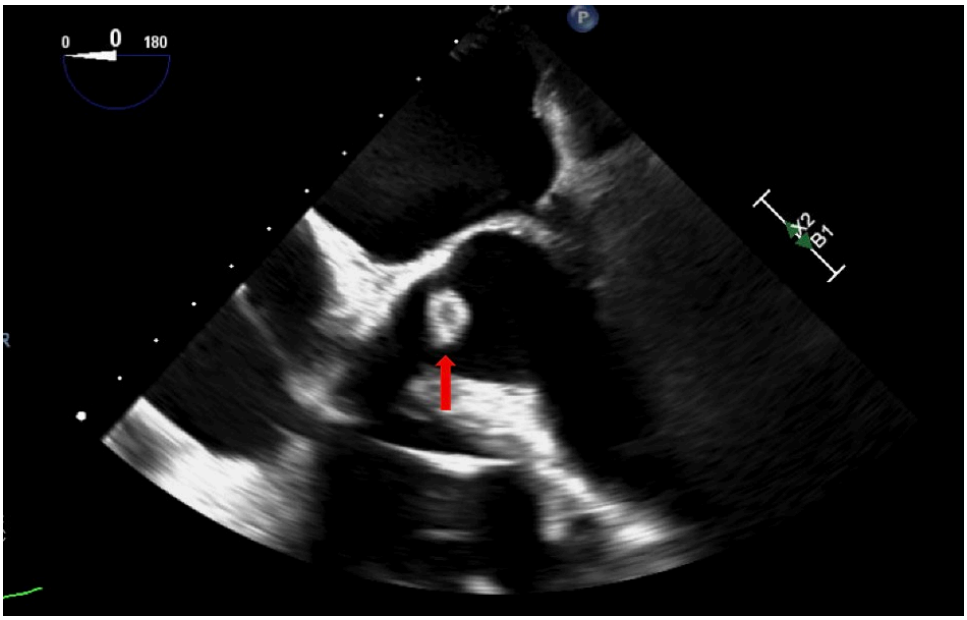


Figure 1. Mid Esophageal 4-Chamber View. Echo density in LVOT (red arrow)

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#FR 3 – Kiosk 1 – Poster 6

Complex case management of an adult with repaired TOF and recurrent severe pulmonic stenosis with unique bidirectional VSD

Presenting Author: Jay Saggu, MD, DMD, Assistant Professor, Stanford University

Introduction

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart defect resulting from an antero-cephalad deviation of the conal septum resulting in right ventricular outflow tract (RVOT) obstruction/pulmonary stenosis (PS), ventricular septal defect (VSD), overriding aorta, and right ventricular hypertrophy (RVH) (1). Definitive surgical repair is often performed in infancy; long-term cardiovascular sequela, like pulmonic valve and right heart dysfunction, is common, often necessitating additional interventions (2). Understanding hemodynamic consequences of repaired TOF is essential as these abnormalities influence perioperative physiology and anesthetic management.

Case Presentation

A 37yo male with prior TOF repair in infancy, repeat pulmonary valve replacement, Streptococcus mutans endocarditis and severe bioprosthetic PS presented for a 4th time redo-sternotomy and PVR. He underwent epinephrine and vasopressor-assisted, hemodynamically stable anesthetic induction and airway management. Pre-operative TEE was notable for supra-systemic RV pressures (113mmHg, fig 1), preserved left ventricular (LV) systolic function, moderately dilated RV with low-normal function (fig 2), severely stenotic bioprosthetic PV with a peak gradient 125mmHg (fig 3), peri-membranous VSD (fig 4 & 5) and inter-ventricular septal (IVS) flattening in systole (fig 6). He underwent a bioprosthetic PVR with RVOT patch augmentation and VSD closure. He was successfully weaned from CPB with ongoing vasopressor support. The patient was extubated within 6 hours upon arrival to the cardiovascular ICU. He continued to require vasopressor support and fluid resuscitation for the first 36 hours and was stable for downgrade to the floor on post op day 4.

Discussion

Adults with repaired TOF represent a growing population with unique challenges for the surgical and anesthesia teams, and increased perioperative morbidity and mortality related to cardiovascular events (3). A dilated RV generating supra-systemic pressures is exquisitely sensitive to changes in preload, afterload and arrhythmias, placing patients at high risk for acute RV failure. Traditional maneuvers to decrease RV afterload like hyperventilation or inhaled pulmonary vasodilators are ineffective given the fixed pulmonic stenosis. Instead, management hinges on maintaining systemic vascular resistance (SVR), optimizing RV perfusion and supporting contractility. Combination of RV inotropic augmentation and vasopressor guided SVR support in our patient underscores the importance of individualized hemodynamic manipulation. TEE provided a clear visualization of the VSD in this case as well. In early systole, LV pressures exceeded the RV, producing left to right shunting. In late systole, the hypertrophied RV generated pressures above the LV reversing the shunt direction (fig 5). TEE showed a moderately dilated/hypertrophied RV (fig 2), with low-

normal fractional area change (34.5%) and tricuspid lateral annulus S' (14.5cm/s) but with IVS flattening in systole consistent with RV pressure overload (fig 6). This rare presentation of supra-systemic RV pressures with cycle-dependent bidirectional VSD flow in repaired TOF underscores the unusually complex intracardiac physiology and need for precise, echocardiographically- guided anesthetic management.

Images

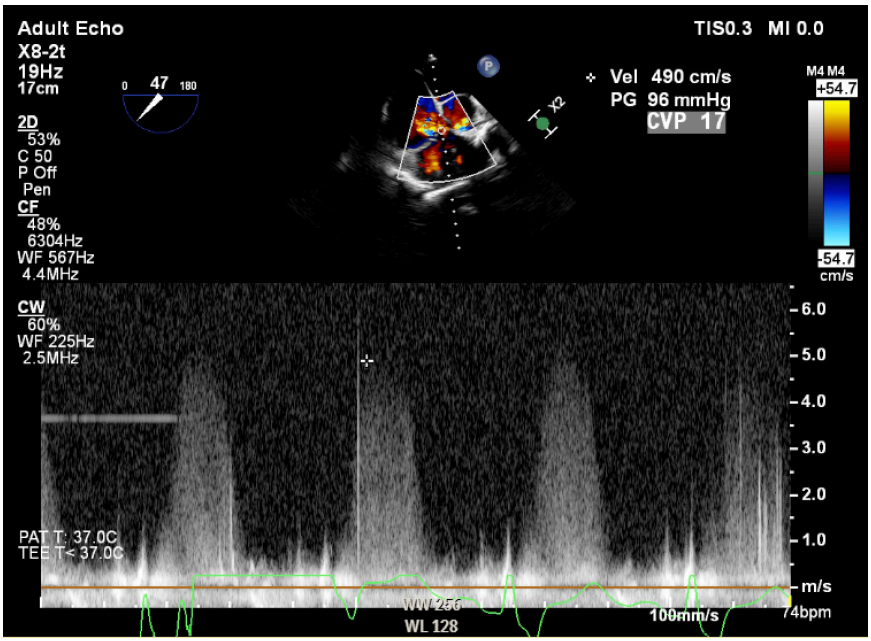


Figure 1 : RV focused mid-esophageal four chamber view with continuous wave doppler across the tricuspid regurgitation jet demonstrating a gradient of 96mmHg. RVSP is estimated to be 113mmHg (assuming right atrial pressure is equal to the central venous pressure of 17mmHg).

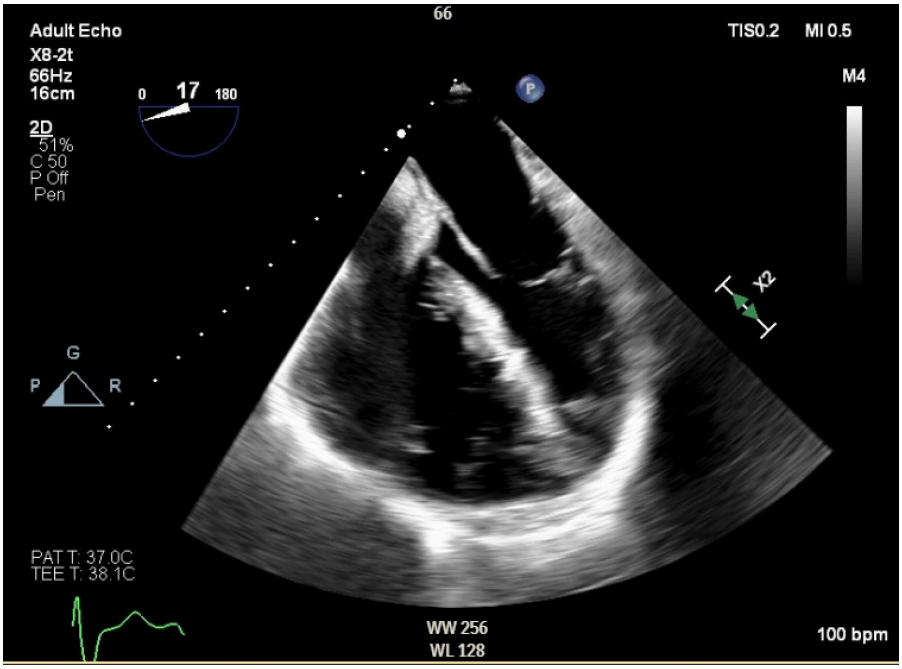


Figure 2 : Mid-esophageal four chamber view demonstrating mod RV dilation and RV hypertrophy.

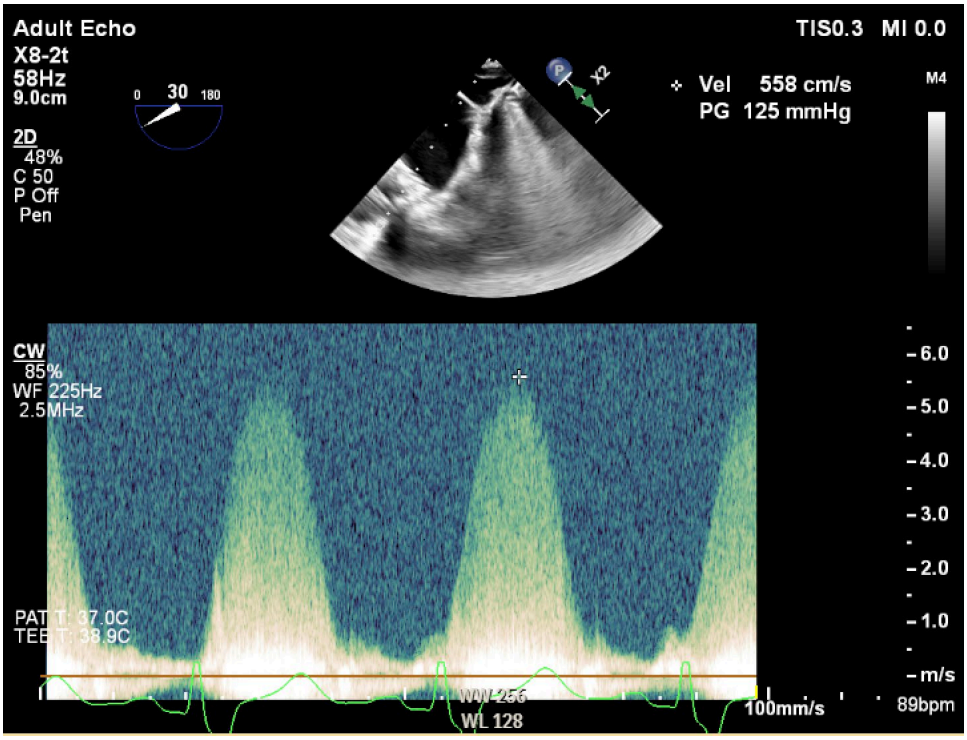


Figure 3 : Upper esophageal aortic arch short axis view demonstrating a peak gradient of 125mmHg across the bioprosthetic pulmonic valve.

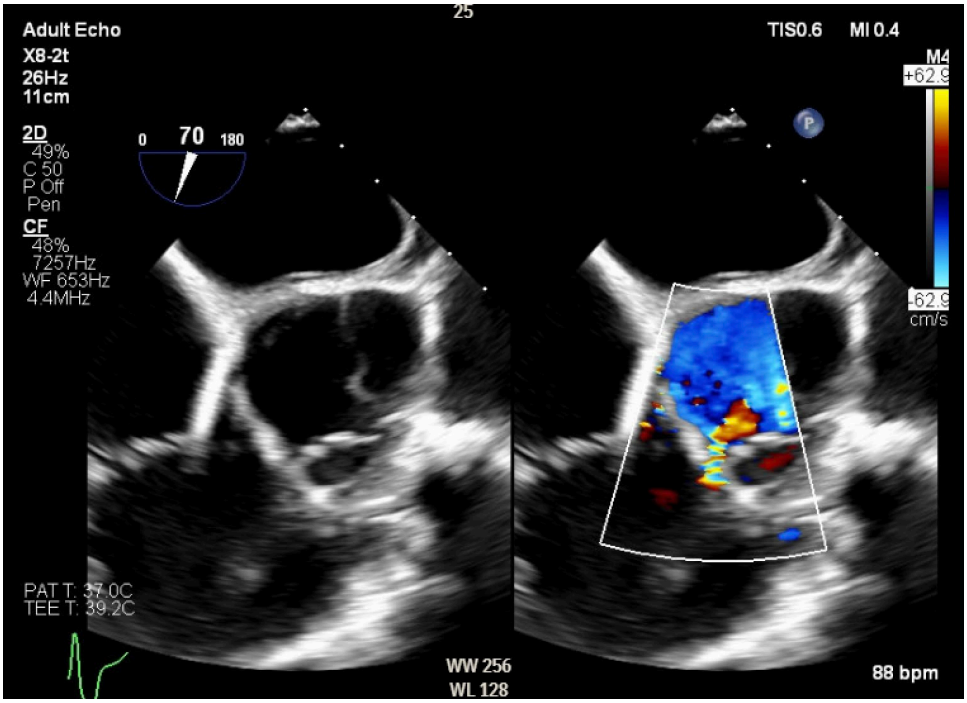


Figure 4 : Mid-esophageal aortic valve short-axis view demonstrating flow across perimembranous VSD.

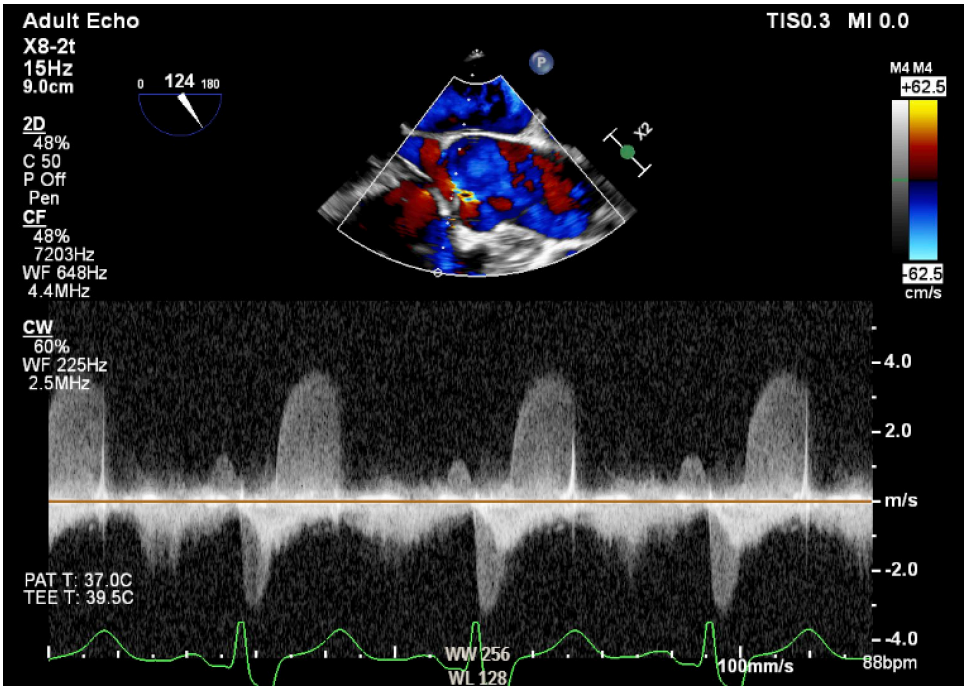


Figure 5: Mid-esophageal aortic valve long axis view demonstrating bidirectional flow across perimembranous VSD.

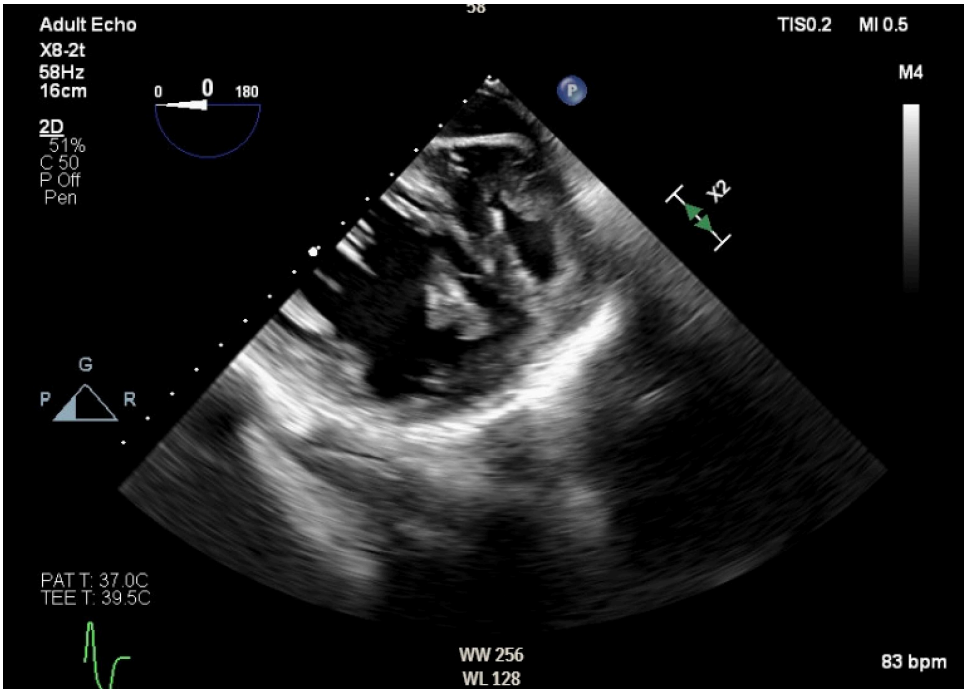


Figure 6: Transgastric mid-papillary short axis view demonstrating interventricular septal flattening in systole consistent with RV pressure overload.

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#FR 3 – Kiosk 1 – Poster 7

Hemodynamic Management of a Congenitally Corrected Transposition of the Great Arteries Patient Post-Palliation with a Biventricular Lateral Fenestrated Fontan Repair Undergoing Renal Transplantation

Presenting Author: Noble F. Lewallen, III, DO, Adult Cardiothoracic Anesthesia Fellow, Stanford University

Introduction

Congenitally Corrected Transposition of the Great Arteries (ccTGA) is a complex and rare congenital heart condition that composes less than 1% of all congenital cardiac anomalies. Historically, there have been two surgical approaches for corrective repair; Physiologic repairs address concomitant abnormalities, whereas Anatomic repairs restore concordant Atrioventricular and Ventriculoarterial connections. Choice of surgical approach is based upon the prevalence and repairability of co-existing cardiac anomalies. A third surgical option for ccTGA is a two-ventricular repair utilizing the Fontan procedure, traditionally reserved for univentricular heart repairs. Here, we present the case of a ccTGA patient who is post two-ventricular lateral tunnel fenestrated Fontan repair presenting for living donor renal transplantation.

Case Presentation

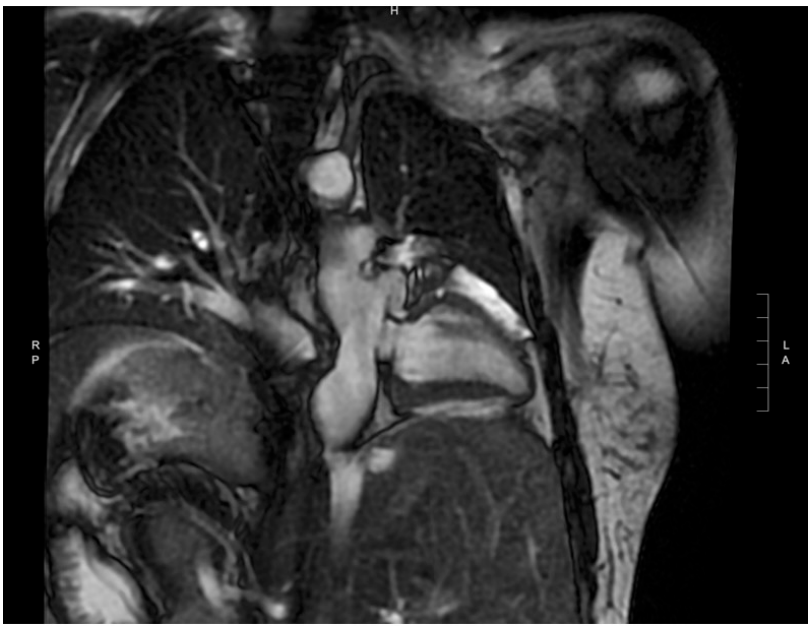
Our 24 year-old patient had a history of ccTGA with pulmonary atresia, a large perimembranous VSD, and viscero-atrial situs inversus with mesocardia. She ultimately underwent a modified (two ventricular) fenestrated lateral tunnel Fontan procedure. She developed post-infectious focal segmental glomerulonephritis w/ sclerosing features (FSGS) with rapid progression to CKD stage 5 in the absence of cardio-renal syndrome. Creatinine = 7.1 and GFR = 8. She was listed for isolated kidney transplantation after initially being rejected for combined heart-kidney due to adequate cardiopulmonary status. She presented to our institution for living-donor kidney transplantation. A pre-induction left brachial A-line was placed. General endotracheal anesthesia was uneventfully induced. Post intubation, additional monitoring was established including TEE and LIJ venous catheterization. The case proceeded without incident until graft reperfusion, at which point the patient experienced sudden, severe, refractory hypotension out of proportion to standard renal ischemic-reperfusion syndromes, with elevated Fontan pressures. After discussion with the surgical team, it was discovered that complete and prolonged common iliac artery cross-clamping had been required. Hemodynamic instability was temporized with aggressive acidemia neutralization, vasoactive support, and volume resuscitation. She was hemodynamically stabilized, successfully extubated at the conclusion of surgery, and delivered safely to the ICU.

Discussion

This case demonstrates the unique cardiovascular anesthesia strategies and intraoperative interpretation skills necessary to navigate a complex renal transplant in a ccTGA patient post Fontan where both the RV and LV are committed to the systemic circuit. Univentricular Fontan failure is a common occurrence in the second and third decade of life following a childhood Fontan palliation and often necessitates a heart transplantation at the time of, or prior to, other solid organ transplantation such as liver and kidney. In

contrast, long-term durability in the biventricular Fontan ccTGA patient is unknown. Our patient had relatively preserved cardiac function and a well-functioning Fontan. The physiological insult from ischemic-reperfusion of a new kidney graft in our patient's PVR-dependent circulation was significant, and excellent preparation and surgical communication was necessary to safely complete her anesthetic.

Images



Cardiac MR demonstrating Lateral Tunnel Fontan patency



Cardiac MR demonstrating large VSD and normal biventricular size

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#FR 3 – Kiosk 2 – Poster 1

Bedside Transesophageal Echocardiography for diagnosis of Frozen Elephant Trunk Graft Thrombosis

Presenting Author: Eric Lee, MD, Brigham and Women's Hospital

Introduction

Use of frozen elephant trunk (FET) grafts for thoracic and descending aorta pathology has shown to have similar mortality rates compared to the conventional elephant trunk technique. While the distal stented portion of FET grafts guards against kinking, FET intraluminal thrombosis (ILT) has emerged as a clinically relevant complication (1). We present a case of total arch replacement with the Thoraflex hybrid FET graft complicated by post-operative ILT and associated challenges of anticoagulation management.

Case Presentation

45F with ACTA2 mutation and prior type A dissection repair in her early 20s had progressive innominate artery aneurysm and presented for surgical management with revision sternotomy and total arch replacement with a 28 mm Thoraflex hybrid FET graft. Her intraoperative course was remarkable for difficulty isolating the left subclavian artery preventing a carotid-subclavian bypass and coagulopathy following bypass improving after transfusion of multiple blood products. On postoperative day 1, a bedside TEE was done for work-up of progressive renal failure that demonstrated multiple mobile echo densities in the visualized portions of the Thoraflex graft, including the proximal arch and proximal descending aorta, concerning for thrombi (Figure 1). The patient was started on a fixed dose heparin infusion for therapeutic anticoagulation against a backdrop of recurrent GI bleeding. The heparin infusion was slowly uptitrated as no increased bleeding from the surgical site or chest tubes was identified. CT angiography of the chest one-week postoperatively demonstrated areas of hypoattenuation within the graft, likely representing persistent mural thrombi (Figure 2). The patient did not develop any evidence of embolic complications to the lower extremities or gastrointestinal system.

Discussion

Thrombotic complications following use of FET grafts have been reported with an incidence of upwards of 17% and are associated with lower 6-month survival rates (2). Studies have observed ILT occurring more often in FET stent grafts with sizes ≤ 34 mm, such as in our patient with a 28 mm FET graft (1). Given the significant incidence, discussion of routine surveillance imaging and prophylactic initiation of anticoagulation have both been suggested (3). In this case, early use of bedside TEE in the ICU provided a quick and reliable diagnosis of the FET graft thrombosis while avoiding transportation for a patient requiring significant inotropic and ventilatory support. Balancing correction of post-bypass coagulopathy and post-operative initiation of anticoagulation was challenging in a patient that underwent a fourth redo sternotomy with significant bleeding risk but indications for anticoagulation initiation with a mechanical aortic valve and FET thrombosis. Some studies suggest various anticoagulation strategies including combinations of heparinization and antiplatelet therapy (3). Overall, use of routine diagnostic imaging modalities and anticoagulation initiation are important considerations particularly for patients undergoing complex or

revision aortic repair. Use of TEE for evaluation of FET graft thrombosis can allow for a quick and reliable diagnosis to provide early intervention in a critically ill patient.

Images

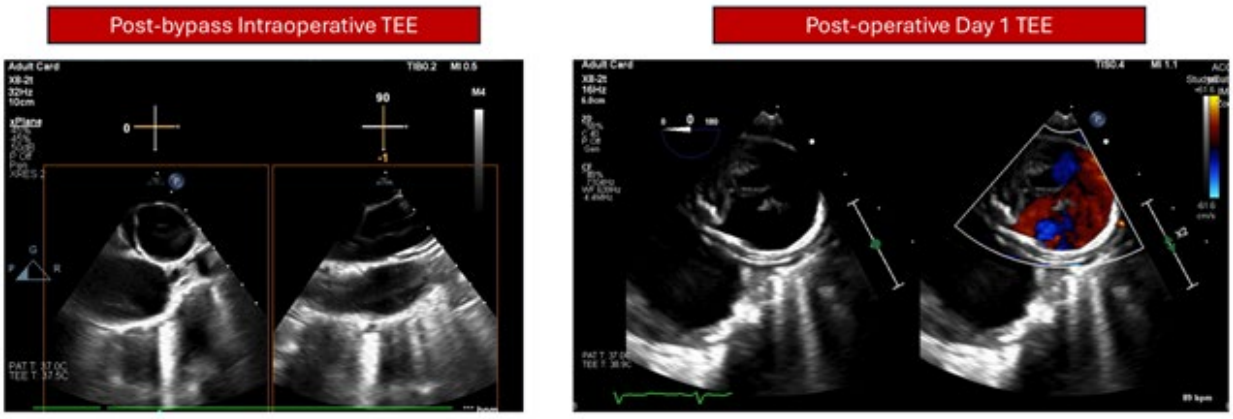


FIGURE 1: Intraoperative proximal descending aorta TEE on POD0 (left image) free of ILT versus Bedside TEE in the ICU on POD1 with ILT occupying >30% of the Thoraflex cross sectional area (right image).

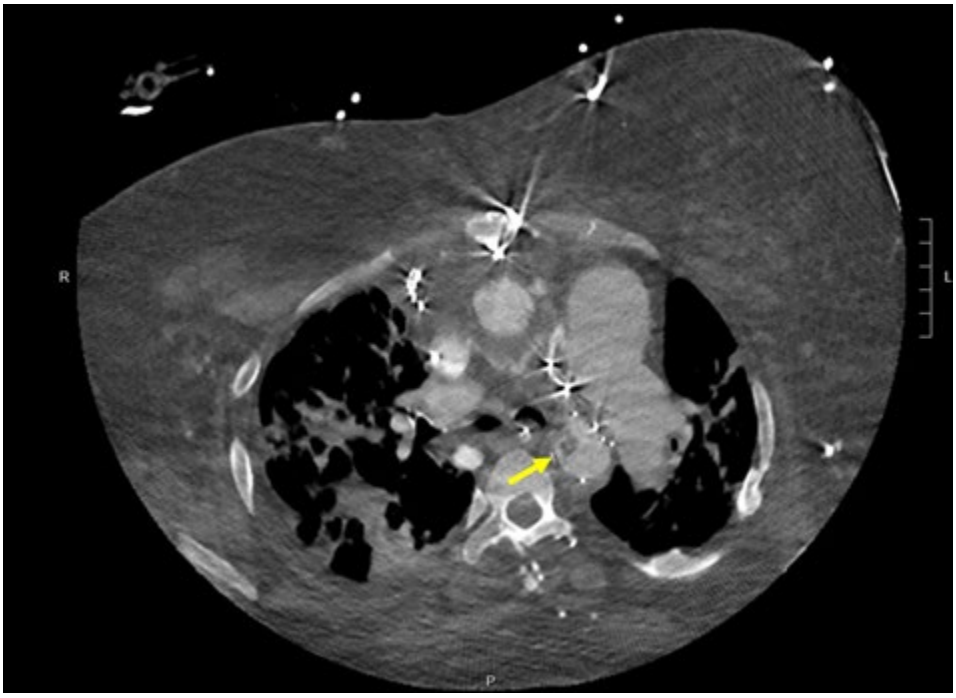


FIGURE 2: Follow-up CT angiogram (CTA) at 1-week postoperatively demonstrates persistent mural thrombi (yellow arrow) in the Thoraflex graft.

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#FR 3 – Kiosk 2 – Poster 2

Stellate Ganglion Block as a Treatment for Recurrent Ventricular Arrhythmias in a Patient with Cardiac Amyloidosis and Cardiogenic Shock

Presenting Author: Colt Moller, MD, Resident, University of Missouri - Columbia School of Medicine

Introduction

Ultrasound-guided peripheral nerve blocks have become integral to anesthesiology, primarily focusing on the management of perioperative and chronic pain. The stellate ganglion block (SGB) inhibits the sympathetic fibers of the head, neck, heart, and upper extremities. The transient sympathetic blockade makes this a versatile block with numerous indications, including the treatment of refractory ventricular arrhythmias.[1] Arrhythmias are common in cardiac amyloidosis (CA), developing from the disruption of the cardiac conduction system as misfolded proteins accumulate. They are often concomitant with heart failure, poorly tolerated, and refractory.[2] In this case, a unilateral SGB treated refractory ventricular arrhythmias in a patient with cardiac amyloidosis who required inotropic support for diuresis.

Case Presentation

A 62-year-old male with a medical history of atrial fibrillation, multiple myeloma, and heart failure with reduced ejection fraction of 20-25% secondary to AL-CA was admitted for management of a heart failure exacerbation. Hospital course was complicated by acute kidney injury from cardiorenal syndrome. Decompensation required transfer to the cardiovascular intensive care unit (CICU) for management of cardiogenic shock. Diuresis with bumetanide and anti-arrhythmic therapy with amiodarone were continued. Inotropic support for diuresis was initiated with dobutamine but quickly discontinued at the onset of ventricular tachycardia. Milrinone was trialed given its reduced arrhythmogenicity; however, still resulted in ventricular tachycardia. Cardiac anesthesiology was consulted for a SGB given the recurrent development of arrhythmias with inotropic support. A left-sided, single-shot, ultrasound-guided SGB was performed. 6 milliliters of 1.3% Exparel were administered. Milrinone was resumed and tolerated without recurrent ventricular arrhythmia for more than 72 hours, allowing for effective diuresis and clinical improvement. Ipsilateral ptosis was noted but resolved by post-block day 4. The patient was stable for transfer to the floor post-block day 5 and discharged home 15 days after CICU admission.

Discussion

The anti-arrhythmic effects of the SGB treats ventricular arrhythmias by limiting the sympathetic input to the heart.[1] Documented treatments for ventricular arrhythmias in CA include medical management with antiarrhythmics; ICD placement; mechanical circulatory support; and more definitive treatments such as catheter ablation, thoracic sympathectomy, and heart transplant.[2] Despite the evidence supporting the use of SGB for the treatment of ventricular arrhythmias, there is limited literature regarding its use in cardiac amyloidosis and as an adjunct for inotropic support in heart failure exacerbations. Most SGBs are performed as a single-shot injection of a long-acting, amide local anesthetic or continuous infusion via catheter. Liposomal bupivacaine was utilized in this case to maximize block duration and avoid the risks associated

with repeated injections and catheter placement. This case demonstrates the effective use of the SGB with liposomal bupivacaine in the treatment of refractory ventricular tachycardia in two patient populations that have otherwise not been extensively discussed in the literature: amyloid cardiomyopathy and cardiogenic shock.

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#FR 3 – Kiosk 2 – Poster 3

Intraoperative TEE-guided management of autograft hematoma during Ross procedure

Presenting Author: Barton Huang, MD, University of Southern California / Los Angeles General Medical Center

Introduction

The Ross procedure is a durable aortic valve (AV) replacement option for young and middle-aged adults. By replacing the native AV with a pulmonary autograft and the pulmonic valve with a homograft, the procedure offers excellent hemodynamics and avoids lifelong anticoagulation. However, its technical complexity predisposes patients to complications such as peri-graft hematoma, root distortion, and anatomic mismatch. Intraoperative transesophageal echocardiography (TEE) is therefore essential to assess graft integrity, diagnose complications, and guide timely surgical correction.

Case Presentation

A 35-year-old male with severe congenital bicuspid aortic stenosis (AS), status post balloon valvuloplasty at age four, presented for Ross procedure after an attempted AV replacement at an outside institution was aborted due to a small annulus (17–19 mm) relative to a body surface area of 2.08 m^2. Pre-bypass TEE demonstrated normal biventricular function and severe bicuspid AS (mean gradient 45 mmHg, DI 0.24), an AV area of 0.7 cm^2 (indexed 0.35 cm^2/m^2), and an annulus measuring 18–20 mm. A posterior root enlargement was performed, and the pulmonary autograft was incorporated within a 26-mm Hemashield graft to reduce the risk of neo-root dilatation. Coronary buttons were reimplanted, and a 26-mm homograft was placed in the pulmonic position. After the first cross-clamp removal, TEE revealed severe obstruction across the neo-AV (peak gradient 170 mmHg, peak velocity 6.53 m/s) and a potential space between the autograft and Hemashield graft that expanded in diastole. Re-clamping and reinforcement of the left and right sinuses with pericardial pledgets resulted in partial improvement (peak gradient 164 mmHg), though echodense hematoma persisted. Following multidisciplinary discussion, the Hemashield graft was incised externally to decompress the space, reducing the gradient to 127 mmHg. During final hemostasis, a tear in the left main coronary artery—likely the bleeding source—was identified and repaired with a bovine pericardial patch. Final post-CPB TEE showed marked improvement (peak gradient 89 mmHg at HR 87) with good leaflet motion and reduced turbulence. A postoperative day 4 transthoracic echocardiogram demonstrated further improvement (peak gradient 62 mmHg, mean 36 mmHg, DI 0.35 at HR 109).

Discussion

This case illustrates the complexity of the Ross procedure and underscores the central role of intraoperative TEE in detecting graft-related complications and guiding operative decision-making. As adoption of the Ross procedure expands due to its excellent long-term outcomes, cardiac anesthesiologists must be increasingly familiar with its technical steps, expected TEE findings, and potential anatomic pitfalls to optimize perioperative management and patient outcomes.


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Images

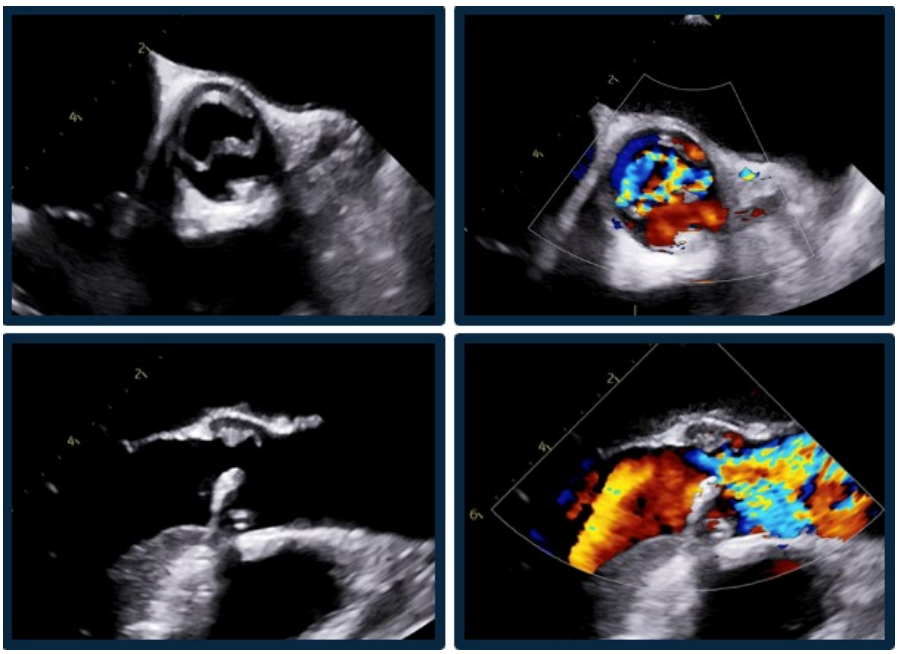


Figure 1. Initial TEE exam with bicuspid aortic valve (top left, AV SAX; top right, AV SAX with color Doppler; bottom left, AV LAX; bottom right, AV LAX color Doppler).

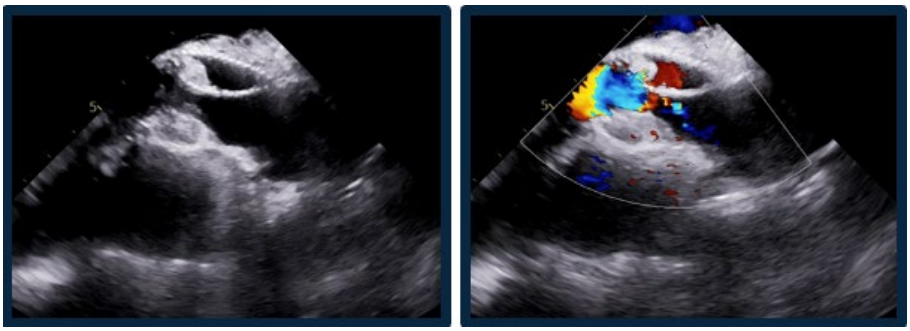


Figure 2. Large hematoma and turbulent flow noted after first cross-clamp removal (left, AV LAX; right, AV LAX with color Doppler).

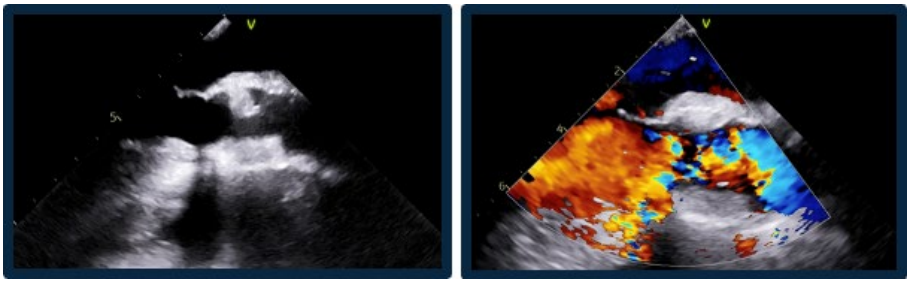


Figure 3. TEE exam after first attempted repair with pledget reinforcement. Continued turbulent flow noted (left, AV LAX; right, AV LAX with color Doppler).

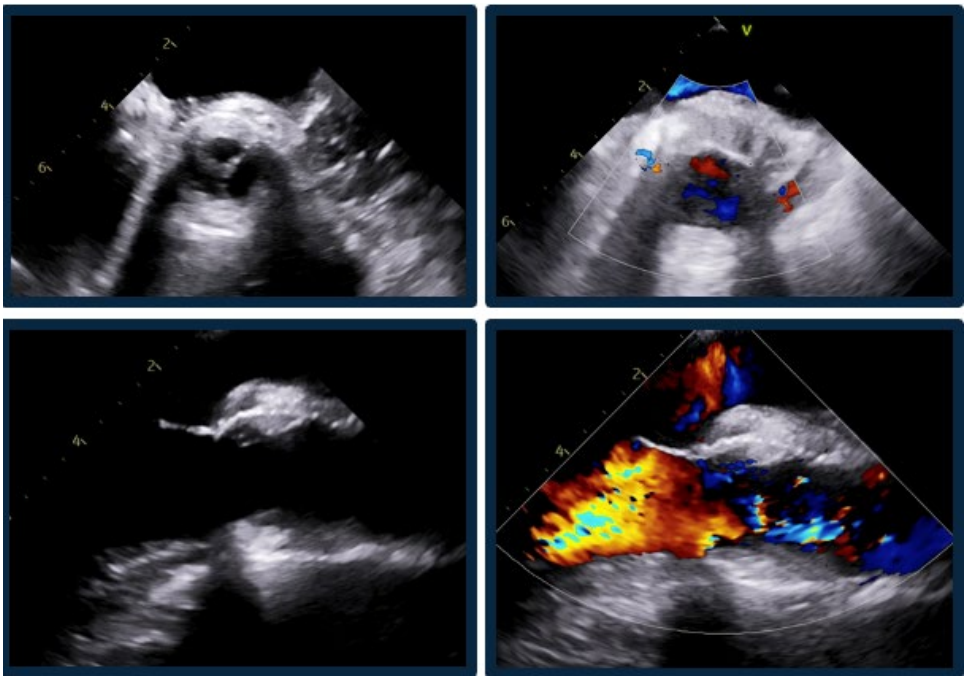


Figure 4. Final TEE exam of neo AV with adequate opening and patent flow. (top left, AV SAX; top right, AV SAX with color Doppler; bottom left, AV LAX; bottom right, AV LAX color Doppler).

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#FR 3 – Kiosk 2 – Poster 4

"A double whammy" – Cardiac tamponade and coronary injury secondary to annular rupture during transcatheter aortic valve deployment

Presenting Author: Brandon Eden, MD, Anesthesiology Resident, University of Maryland Medical Center

Introduction

Excessive aortic valve calcification increases the risk of adverse events during TAVR including valve malpositioning, paravalvular leak, coronary obstruction, and annular rupture.[1,2] We present a 62-year-old man with severe calcific AS who had annular rupture causing cardiac tamponade, coronary injury, and subsequent arrest during TAVR valve deployment requiring emergency sternotomy.

Case Presentation

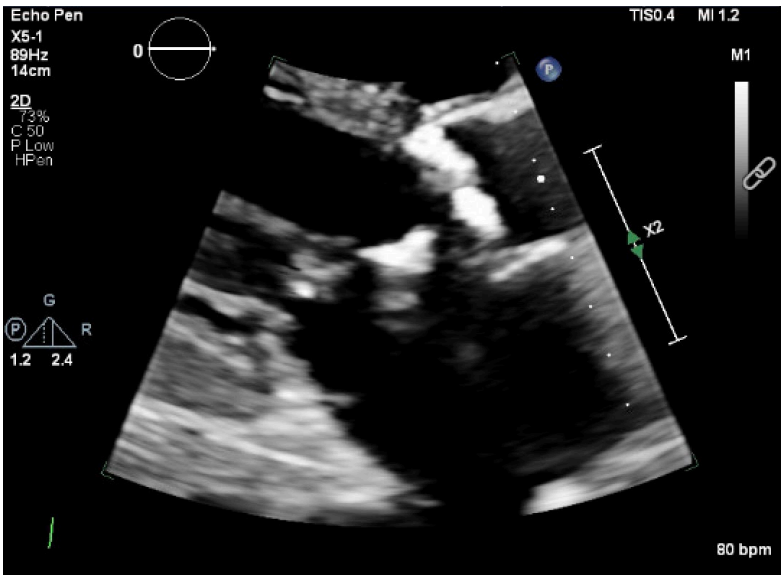
A 62-year-old man with metastatic rectal cancer and severe AS presented for TAVR under sedation. Preoperative TTE (image 1) revealed severe aortic stenosis with valve area 0.6cm^2 and mean gradient 70 mmHg. Excessive calcification was prominent on CT (image 2). Ejection fraction was 55%. Access included 2 PIV with femoral arterial access for blood pressure monitoring provided by the surgical team. A propofol infusion was initiated and the procedural team obtained femoral arterial and venous access. A 26 mm Edwards Sapien 3 balloon-expandable valve was deployed in the optimal position. Within one minute of valve deployment, the patient went into PEA arrest. Chest compressions were initiated, a total of 3mg epinephrine was administered, and the patient was placed on VA- ECMO using the current access. The patient was intubated and a right internal jugular multi-access catheter was placed. A large pericardial effusion was identified on TTE and pericardiocentesis was performed followed by pericardial drain placement. The effusion persisted and the patient was brought to the OR. A right radial arterial line was placed and VA-ECMO was converted to peripheral CPB. TEE (image 3) confirmed a large pericardial effusion with RA and RV collapse. Sternotomy was performed, an aortic cross clamp was applied, and cardioplegia was administered. Brisk bleeding was identified from a 1.5cm perforation near the base of the left atrial appendage inferior to the LCx and lateral to the LAD. The injury was due to annular/LVOT calcium protrusion during balloon expansion of the valve. It was repaired with a surgical adhesive patch. Due to the proximity of the injury to the LAD and LCx, both were bypassed with saphenous vein grafts. The patient was weaned off bypass, decannulated, and transported to the ICU on epinephrine and norepinephrine. A total of 7 units of PRBC, 2 units of FFP, and additional cell saver and pump blood were administered. Post bypass TEE revealed an EF of 45% and a well-seated TAVR valve with no paravalvular leak. The patient was extubated on

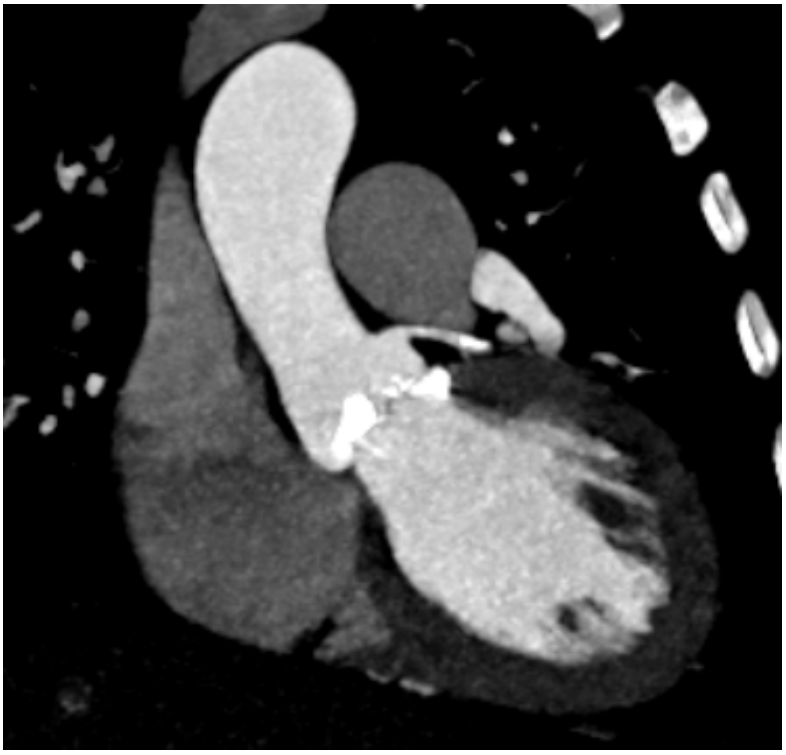
POD1, weaned off vasoactives by POD2, and discharged on POD5.

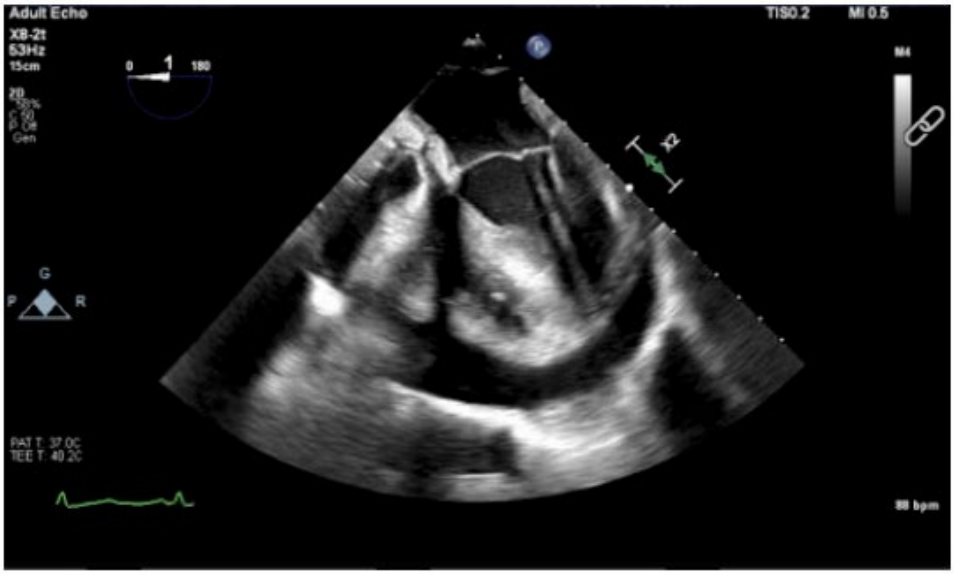
Discussion

Excessive AV calcification often extends from the cusps to the LVOT.[2] Annular rupture and cardiac tamponade are known complications of TAVR in the presence of extensive calcification and occurs more frequently with balloon-expandable valves compared to self-expandable valves.[1,2] This risk may be further exacerbated by aggressive balloon dilation or implantation of an oversized valve.[2] However, self-expandable valves are associated with higher rates of paravalvular regurgitation.[2] TAVR with balloon-expandable valves in patients with excessive calcification carries substantial procedural risk and warrants preparation for prompt rescue and bailout options such as a transcatheter valve-in-valve strategy to seal the annular rupture or emergency sternotomy if necessary.[2,3]

Images







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#FR 3 – Kiosk 2 – Poster 5

Leadless Pacemaker Implantation in a Mustard Repair Patient: Peri-operative Considerations for this Complex Post-Surgical Physiology

Presenting Author: Tyler K. Yoshida, MD, Anesthesia Resident, Northwestern University

Introduction

Dextro-transposition of the great arteries (D-TGA) is a severe congenital heart disease in which the aorta arises from the right ventricle (RV) and the pulmonary artery arises from the left ventricle (LV), resulting in two parallel circulations. This anomaly requires repair during infancy for survival. Atrial switch procedures (Mustard/Senning) involve the creation of an atrial baffle to redirect blood flow such that deoxygenated blood flows to a subpulmonic ventricle (LV) and oxygenated blood flows to a systemic ventricle (RV); this has largely been abandoned for arterial switch procedures due to late systolic dysfunction of the RV. Patients with atrial switch repairs remain at high risk of perioperative decompensation.

Case Presentation

A 55 year-old woman with a history of D-TGA repaired with a Mustard procedure presented for a leadless pacemaker (LLPM) after her existing transvenous pacemaker, implanted for sinus node dysfunction, started causing bothersome diaphragmatic pacing. Sequelae of her Mustard procedure included systemic RV failure with an ejection fraction of 30%, moderate tricuspid regurgitation, post-capillary severe pulmonary hypertension (PH) and mild LV dysfunction, suspected baffle leak, atrial flutter treated with ablation, and cardiac cirrhosis. Following placement of two peripheral intravenous catheters and invasive arterial blood pressure monitoring, the procedure was completed under local anesthesia with minimal sedation with dexmedetomidine; brief periods of a low-dose propofol infusion were permitted during dilation of the femoral veins to facilitate the large LLPM delivery catheters. Mean arterial pressures (MAPs) were carefully titrated with a low-dose phenylephrine infusion. LLPM implantation was aborted after two failed attempts; instead, a new transvenous atrial lead was implanted and the prior atrial lead was capped, extending the case to 7.5 hours. A total of 900 mL of crystalloid was administered. Two hours after case completion, the patient experienced sudden acute decompensated heart failure and was intubated for hypoxemic respiratory failure. Following diuresis, the patient was extubated and discharged to home on postoperative day 5.

Discussion

This case demonstrates the unique perioperative considerations of a patient with a Mustard repair. The most common complications are systemic RV dysfunction, tricuspid regurgitation, PH, sinus node dysfunction resulting in bradycardia, supraventricular and ventricular tachyarrhythmias, baffle stenosis, and baffle leakage. Local anesthesia with minimal sedation was chosen to minimize both myocardial depression and reduction of systemic vascular resistance secondary to anesthetic agents. This approach also avoided hypercarbia and hypoxemia in the setting of severe PH. MAP was titrated to maintain adequate coronary perfusion pressure while not excessively increasing afterload that could cause systemic RV failure. While

treatment was not required intraoperatively, management of undesired tachyarrhythmias was anticipated. Despite meticulous perioperative planning, the patient still experienced acute decompensation, highlighting both the limited physiologic reserve and the high risk of deterioration of this patient population.

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#FR 3 – Kiosk 2 – Poster 6

Pushing the Limits: Subaortic Stenosis in Liver Transplantation

Presenting Author: Brandon Cave, MD, Resident, University of Maryland

Introduction

Subaortic stenosis is a structural obstruction of the outflow tract that can manifest as a distinct ridge, fibromembranous obstruction, or muscular variant. It is commonly diagnosed in infancy due to identification of other cardiopulmonary complications, like aortic insufficiency, which presents as exercise intolerance. Significant impedance based on the extent of tissue involvement can lead to sudden cardiac arrest and death.[1] Subaortic stenosis is formally diagnosed and evaluated with transesophageal echocardiography(TEE). Surgical correction has shown mortality benefit.[2] Liver transplantation presents significant hemodynamic strain particularly in patients with acute liver failure. Severe aortic stenosis is a relative contraindication for liver transplantation, however, patients with mild or moderate aortic stenosis may still be appropriate for liver transplantation with thorough physical examination and appropriate exercise tolerance.[3]

Case Presentation

A 63 year old female presenting with alcohol related cirrhosis complicated by ascites, esophageal varices, gastrointestinal bleed was admitted to the intensive care unit for worsening acute kidney injury. TTE completed 2 weeks prior, noted subaortic webbing with aortic peak gradient of 42mmHg and mean gradient 24mmHg. Peak MELD 3.0 score of 48. A prior upper endoscopy noted Grade I varices. During this admission, the patient has been treated for Hepatorenal syndrome with octreotide and midodrine. She tolerated induction well. During reperfusion, she had a brief period of hypotension which was relieved with small boluses of epinephrine and later vasopressin infusion, through intraoperative TEE guidance. Throughout the surgery, she had an estimated 4.2L of blood loss, and received 25 units of blood products. She was transported to the surgical ICU on vasopressin and norepinephrine infusions as well as a propofol infusion. She returned to the operating room for surgical bleeding and hemostasis was achieved; she did not require further transfusions.

Discussion

In the setting of acute liver failure with a matched donor organ being prepared, the risk-benefit ratio favored proceeding with known risk that liver transplantation is a significantly morbid procedure. Subaortic ridge may increase the mortality risk but in the absence of further exercise intolerance, transplantation will provide the patient with the most benefit. In optimal conditions, surgical intervention through balloon valvuloplasty or open surgical correction may relieve the stenosis, however, the acuity of the particular patient and coagulopathy in the setting of end-stage liver disease, did not allow for further cardiac surgical optimization. Intraoperative TEE can provide useful real-time information to enhance decision-making during critical phases of a liver transplantation. During critical phases, like reperfusion, where hemodynamic variability is anticipated, having a live assessment of contractility and flow with a structural obstruction can

guide towards a more optimal resuscitation as well as identify complications more readily. While TEE is not frequently used in this patient population due to associated comorbidities with liver dysfunction, it should not be excluded when clinically indicated for intraoperative cardiac assessment when warranted.

Images



Fig 1- Midesophageal aortic valve long axis view showing sub-aortic ridge

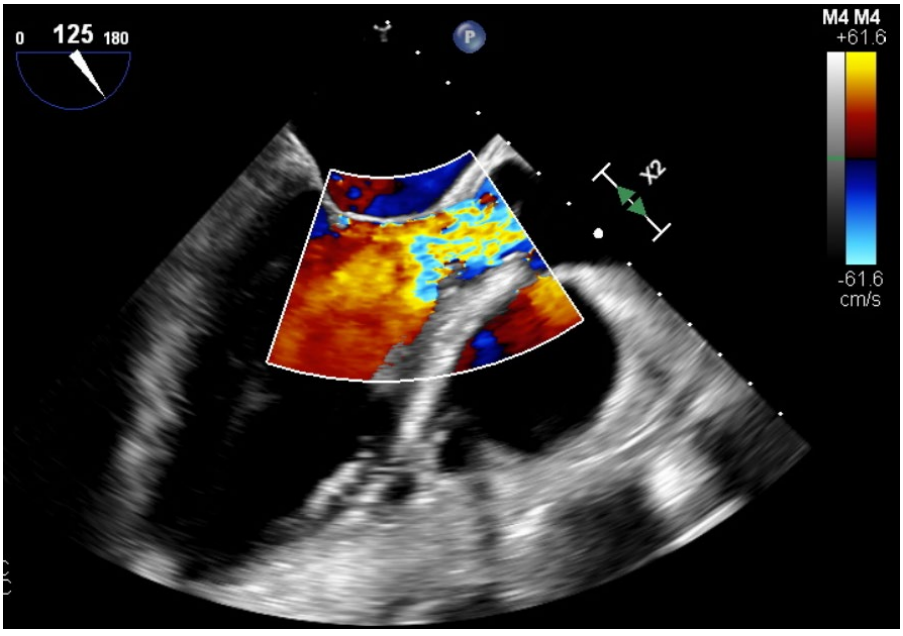
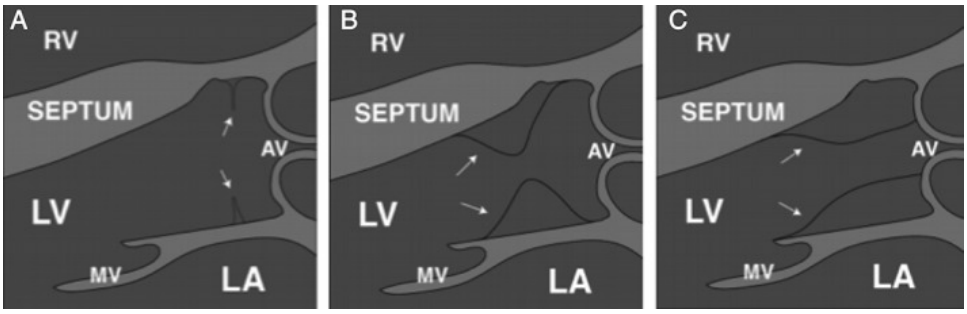


Fig 2- Midesophageal aortic valve long axis view with color flow Doppler showing flow turbulence suggestive of subaortic obstruction



(A) Discrete subaortic membrane (arrows). (B) Thick fibromuscular ridge (arrows). (C) Tunnel or tubular (arrows). Abbreviations: AV, aortic valve; LA, left atrium; LV, left ventricle; MV, mitral valve; RV, right ventricle

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#FR 3 – Kiosk 2 – Poster 7

Now You See It, Now You Don't: Hemodynamic Dependent Left Atrial Appendage Sludge During Anesthesia for Watchman Implantation

Presenting Author: Borys Shturman, MD, Anesthesia Resident, Maimonides Medical Center

Introduction

Left atrial appendage (LAA) sludge is typically considered to be a high-risk finding suggestive of thrombus; which is particularly contraindicated in device-based therapies like Watchman placement. However, its transient nature in conditions of low flow poses challenges in diagnosis, especially in elderly with comorbidities and those with arrhythmias. This case illustrates the dynamic nature of LAA sludge in low cardiac output states and highlights the importance of maintaining sinus rhythm in patients undergoing LAA occlusion procedures.

Case Presentation

A 77-year-old male with a history of paroxysmal atrial fibrillation, bioprosthetic aortic valve replacement, moderate pulmonary hypertension and recent gastrointestinal bleeding presented for LAA occlusion. Due to intolerance of full dose anticoagulation, he was placed on reduced dose apixaban (CHADS-VASc: 3). Pre-procedural TEE showed a severely dilated left atrium (volume index 57.8 mL/m2) and preserved LVEF (56–60%). Under general anesthesia, the patient developed junctional bradycardia with hypotension. TEE at this time revealed dense, smoke-like LAA sludge suggestive of thrombus, requiring termination of the procedure. During emergence, the patient returned to NSR with improved hemodynamics and the sludge resolved. After discussion with the team, the decision was made to proceed. Upon re-induction, junctional rhythm recurred and LAA sludge reappeared. Isoproterenol was initiated, restoring sinus tachycardia and improving cardiac output. The team interpreted the sludge as flow-related stasis rather than a fixed thrombus. The patient subsequently developed unstable AF intra-procedurally and was cardioverted to NSR. The Watchman device was successfully implanted, and the patient remained stable postoperatively.

Discussion

This case highlights the flow dependent nature of LAA sludge, which manifested during low-output states (junctional rhythm and hypotension) and resolved with restoration of NSR along with improved hemodynamics. These findings emphasize that sludge may not always represent a fixed thrombus and should be interpreted with mindfulness of the patient's rhythm and perfusion status. Anesthetic induced rhythm changes can unmask or exacerbate atrial stasis, potentially leading to misinterpretation and unnecessary procedural cancellation. Chronotropic support (e.g., isoproterenol) and prompt rhythm optimization played a pivotal role in allowing safe procedural completion.

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#FR 3 – Kiosk 3 – Poster 1

Thoracic Endovascular Aortic Repair Graft Embolization on Venoarterial Extracorporeal Membrane Oxygenation Following Descending Aortic Injury from An Intra-Aortic Balloon Pump

Presenting Author: Jordan E. Menczer, M.D., Resident, McGaw Medical Center at Northwestern University-Department of Anesthesiology

Introduction

Cardiogenic shock from STEMI remains a major cause of mortality and can be complicated by refractory ventricular arrhythmias. In addition to revascularization, management is often pharmacologic but may require escalation to mechanical circulatory support (MCS). Further complications from MCS are common, including bleeding, coagulopathy, and stroke. We report the case of a rare complication of aortic injury from an intra-aortic balloon pump in a patient with cardiogenic shock on peripheral VA-ECMO and IABP.

Case Presentation

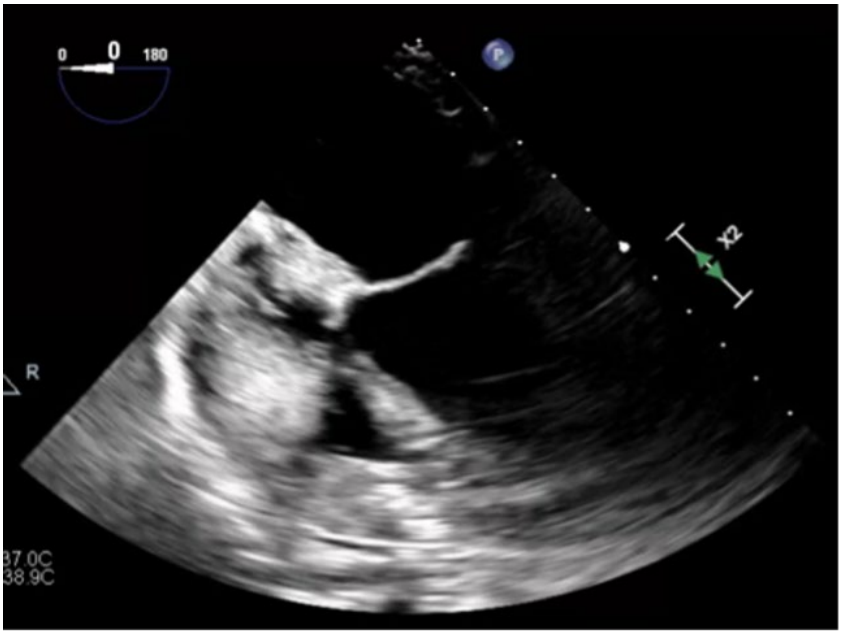
A 54-year-old man with hypertension presented with dyspnea, atrial fibrillation, and anterior ST elevations. Angiography showed 100% proximal LAD thrombotic occlusion without flow despite angioplasty and aspiration thrombectomy. EF was 15%, prompting femoral IABP placement. He developed ventricular fibrillation requiring CPR, defibrillation, and VA-ECMO. Recurrent VT/VF persisted despite amiodarone and lidocaine, leading to bilateral stellate ganglion blocks as a bridge to additional support. He was taken to the OR for Impella 5.5 placement and IABP removal. Before incision, ECMO flows acutely fell to 1 L/min with chugging. Volume resuscitation briefly improved flows, but was followed by severe hemodynamic instability, requiring ACLS, chest compressions, and external defibrillation. Circuit clot prompted emergent exchange with hemodynamic recovery, but VT persisted. TEE showed a right atrial thrombus with minimal flow across the tricuspid valve, RV dysfunction, and a large pleural effusion. Hemoglobin acutely dropped from 10 to 4 g/dL without obvious signs of hemorrhage. Massive transfusion was initiated and Impella placement was aborted. CT angiography demonstrated a descending aortic injury with contrast extravasation near the IABP marker and a large left tension hemopneumothorax with mediastinal shift. He underwent emergent TEVAR and an endograft was deployed from Zones 2–5, but it quickly embolized retrograde. Left subclavian flow remained, whereas left carotid flow was markedly reduced and cannulation for stenting was unsuccessful. Given refractory VF, complete ECMO dependence, and likely catastrophic stroke, further intervention was deemed futile and the family elected DNR status with withdrawal of support.

Discussion

This case highlights a rare aortic injury and hemorrhage from an IABP. Major complication rates range from 2%-4.6%.[1] In this patient, it is unclear what led to the aortic injury. Did the IABP migrate during patient transport and movement over to the surgical bed, which later led to cardiac arrest secondary to hemorrhagic shock? Was there another inciting event that caused the patient to arrest despite being on VA ECMO, and the subsequent chest compressions led to the IABP migration causing aortic injury? This case also describes TEVAR under peripheral VA-ECMO complicated by embolization of the graft leading to loss of left

common carotid flow. Limited literature describes TEVAR on VA-ECMO, primarily for distal malperfusion or cerebral protection in Zone 0 repairs.[2,3] ECMO flows were not reduced during deployment in these cases. Whether transient flow reduction to limit turbulence and retrograde flow would have altered the outcome in this patient is unknown.

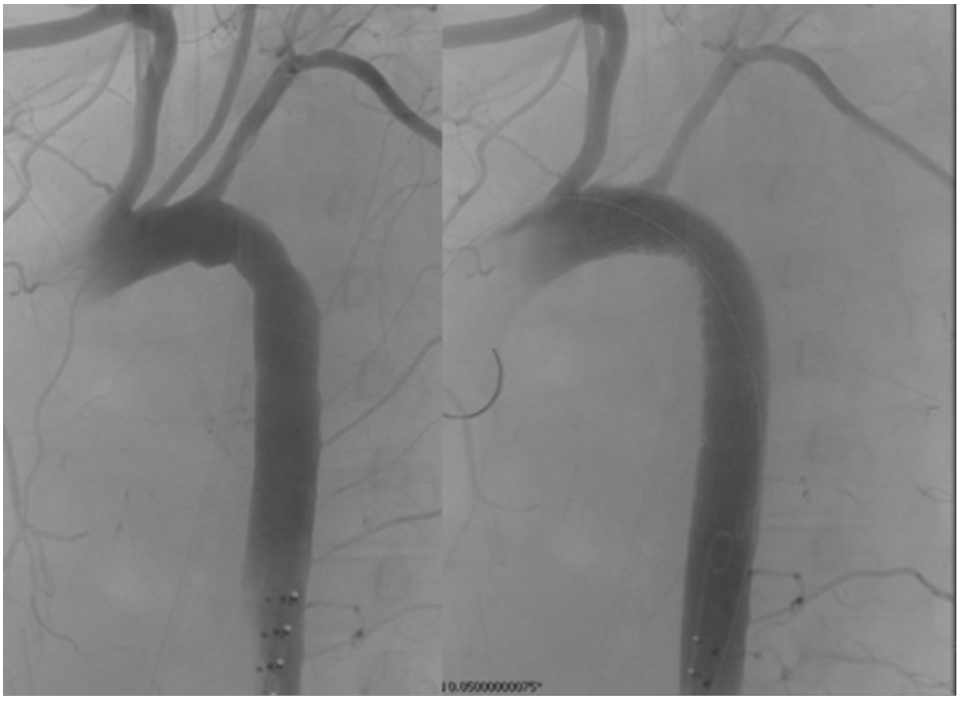
Images



Right Atrial Clot on TEE



Left Tension Hemopneumothorax With Mediastinal Shift



Loss of Left Common Carotid Artery Flow After Stent Deployment

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#FR 3 – Kiosk 3 – Poster 2

Incidental Intraoperative Superior Sinus Venosus Defect Finding without Partial Anomalous Pulmonary Venous Return: A Case Report

Presenting Author: Francine S. Moriguchi, MD, Resident, University of Minnesota

Introduction

Sinus venosus defect is a rare atrial septal defect subtype often associated with partial anomalous pulmonary venous return (PAPVR) with right ventricular overload and pulmonary hypertension1. We present a patient who was incidentally diagnosed with superior sinus venosus defect, without PAPVR, during intraoperative transesophageal echocardiogram (TEE) for a planned Bentall procedure with postoperative course notable for sinus node dysfunction.

Case Presentation

A 69 year old asymptomatic male presented for a planned Bentall procedure. Preoperative transthoracic echocardiogram (TTE) showed aortic root and ascending aorta dilation with moderate aortic regurgitation, mildly enlarged right atrium and ventricle, and severely dilated left atrium. EKG showed first degree atrioventricular (AV) block. Intraoperative TEE was notable for a large SVC type sinus venosus defect without

PAPVR. Spectral and color flow doppler revealed left to right shunt. Calculated shunt fraction (Qp:Qs) was ~3.8 as measured by TEE and blood gas analysis. Additional findings included a mixed venous oxygen saturation (SvO2) of 99% with discrepancy of cardiac output (CO) on FloTrac (5-7 L/min) and Swan-Ganz catheter continuous cardiac output (CCO) (12-14 L/min). The patient underwent the planned Bentall procedure with mechanical valved conduit, left atrial appendage clipping, and pericardial patch closure of the ASD with subsequent findings of equal CO on FloTrac and CCO, and a Qp/Qs ~1 as measured by TEE and blood gas analysis. Atrial and ventricular temporary pacemaker wires were placed for AV block. The patient was transferred to the ICU intubated and sedated. On postoperative day (POD) 1, the patient was found to have underlying sinus bradycardia suggestive of sinus node dysfunction. Interestingly, he was also found to have an intact AV node as he was able to be atrially paced. On POD3, bedside evaluation showed spontaneous recovery with sinus rhythm in 50-70 BPM.

Discussion

Superior sinus venosus defects represent an uncommon and often overlooked form of ASD. Diagnosis is frequently delayed due to anatomic variations (ie. with PAPVR or without PAPVR), superior location, and noninvasive diagnostic tool limitations (ie. far-field on TTE). Although TTE is a first line diagnostic tool for patients with suspected ASD, literature has suggested detection of sinus venosus defect in less than 50% (3/25 patients) of patients with TTE but 100% (25/25 patients) of patients with TEE2. In our case, aside from TEE findings, the discrepancy of CO measured by FloTrac (arterial line waveform contour analysis) and CCO (continuous cardiac output from Swan-Ganz) further supported increased blood flow in the right heart when compared to systemic circulation. In the post operative period, recent literature has suggested uncommon development of sinus node defect and need for permanent pacemaker placement3 as observed in our patient. Sinus venosus defects pose notable diagnostic challenges, yet their repair is marked by consistently low mortality and morbidity.

Images

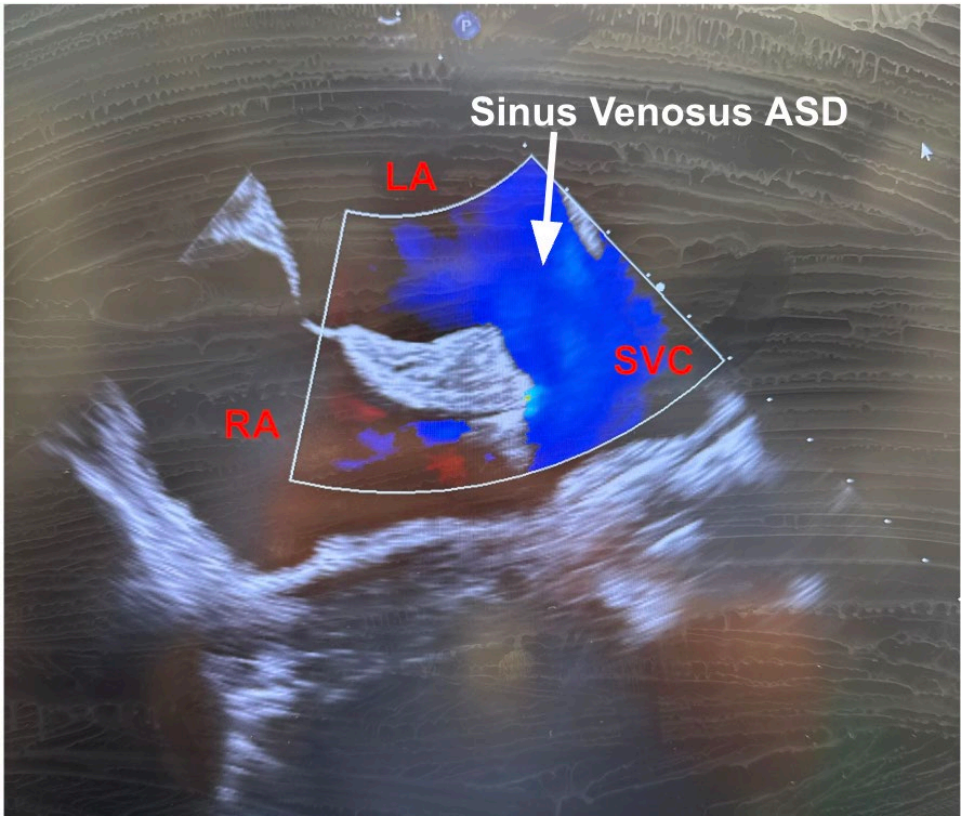


Image 1: Intraoperative TEE showing large SVC type sinus venosus atrial septal defect (LA: left atrium, RA: right atrium, SVC: superior vena cava).

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#FR 3 – Kiosk 3 – Poster 3

Iatrogenic Aorto-Right Ventricular Fistula Following TAVR: TEE Diagnosis and Management of a Rare Mechanical Erosion

Presenting Author: Yash B. Bisen, MD, Resident, Montefiore Einstein

Introduction

Cardiac fistulas are a rare but significant complication of transcatheter aortic valve replacement (TAVR). Injury to the aortic root or annulus during valve deployment can result in aortic fistulae. In this case, there was an aorto-right ventricular fistula, a pathologic connection between the aorta and the right ventricle (RV). Left-to-right shunting between the high-pressure aorta and lower-pressure right ventricle presents risks for RV overload, pulmonary hypertension, and ultimately heart failure. These risks highlight the importance of early recognition, careful anesthetic planning, and coordinated intervention.

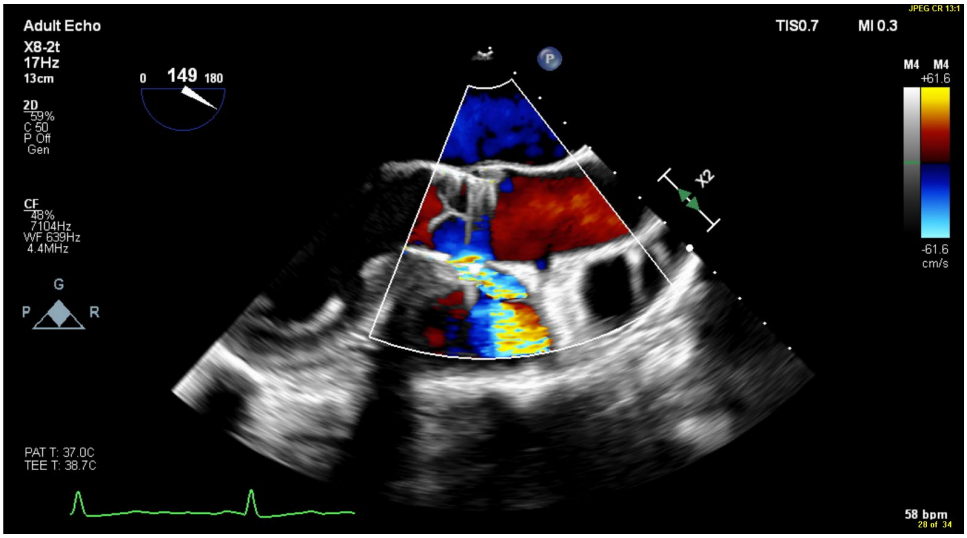
Case Presentation

An 81-year-old man with Type II bicuspid aortic valve and moderate-to-severe aortic stenosis (mean gradient 18.6 mmHg, aortic valve area 0.87 cm^2) presented for TAVR. The patient underwent transesophageal echocardiography (TEE) guided TAVR under general anesthesia. To optimize hemodynamics in the setting of aortic stenosis, arterial pressure was monitored invasively. A 26mm Sapien 3 Ultra valve was deployed. Following deployment, TEE interrogation revealed a moderate paravalvular leak (PVL). Balloon post-dilation was performed to address the PVL. Subsequent TEE visualization revealed a new, small aorto-right ventricular fistula. The patient remained hemodynamically stable with no signs of pericardial effusion or immediate RV failure. The Heart Team opted for a staged approach; the patient was extubated with a plan for cardiac computed tomography (CT) and surgical consultation. Cardiac CT confirmed the presence of a small fistulous communication between the aortic root and the RV outflow tract above the right-oriented prosthetic cusp; it was due to a perforation from the upper edge of the TAVR struts. The following day, the patient was taken back for aortic root repair and fistula closure. The pre-aortic root repair TEE visualized the fistula proximal to the coronary ostia with a left-to-right flow, which was also in communication with the PVL. Post-repair TEE no longer demonstrated the fistula, but still demonstrated the PVL.

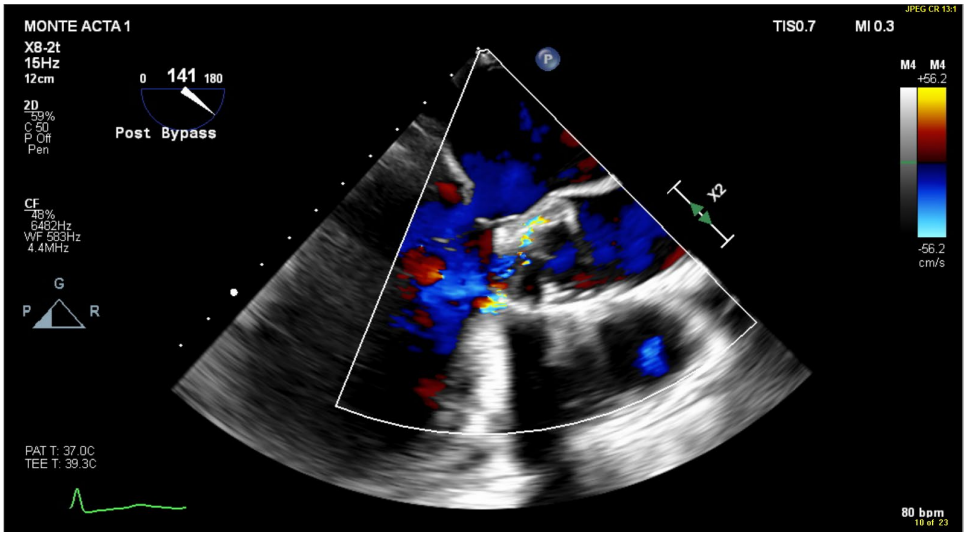
Discussion

Though rare, aorto-right ventricular fistulas may lead to a variety of complications. Large or expanding fistulas pose a risk of sudden cardiogenic shock due to left-to-right shunt and RV overload. Intraprocedural TEE allows for early diagnosis and characterization of size and severity of the fistula. In this case, TEE identified the connection between the aorta and right ventricle immediately following the post-dilation of the TAVR valve. The imaging reassuringly did not reveal any signs of significant RV dysfunction; however if left untreated, RV failure would likely develop. Additionally, it was plausible that the at-present contained aortic rupture could lead to hemorrhagic effusion and tamponade. In this case, intraoperative TEE helped guide effective surgical repair for this unusual TAVR complication.

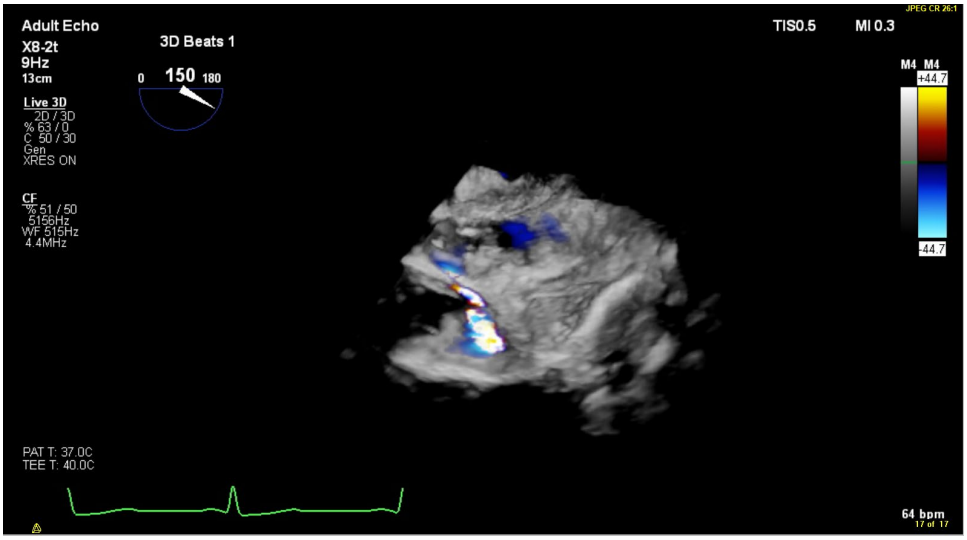
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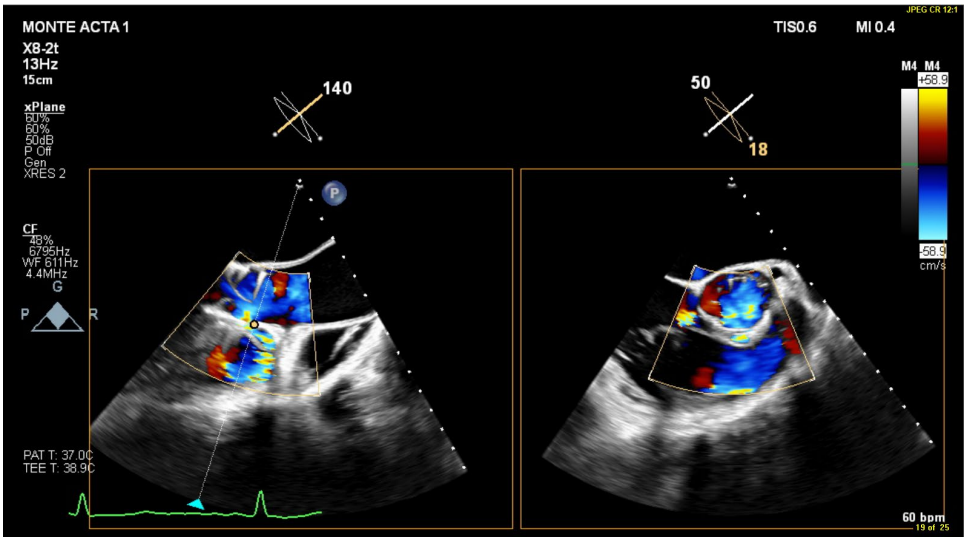
Mid-Esophageal Long Axis (ME LAX) View: Fistulous tract communicating between the high-pressure aortic root and the lower-pressure right ventricle (RV). The defect is located adjacent to the TAVR stent frame.



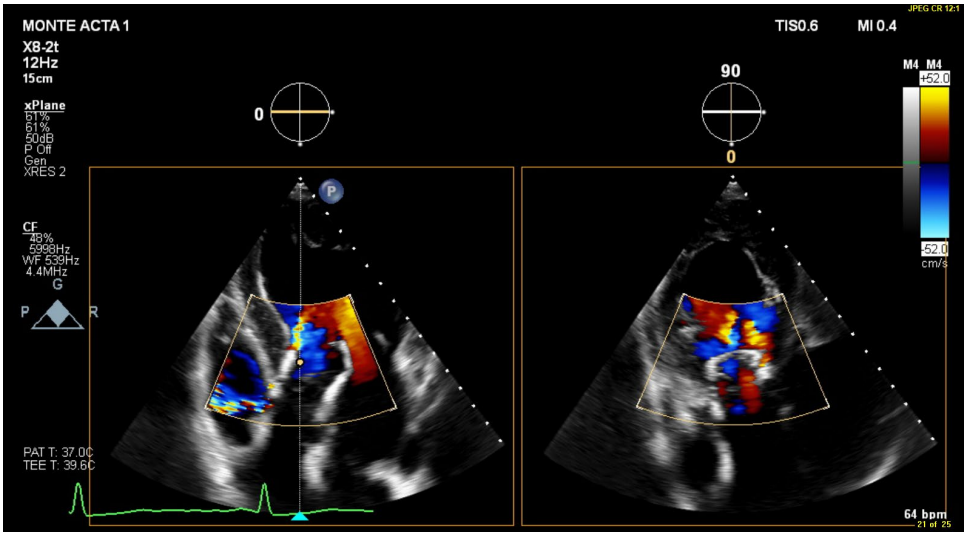
Mid-Esophageal Long Axis (ME LAX) View: After correction of fistulous tract.



3D Color Volume Rendering: Spatial extent of the fistula, demonstrating left-to-right shunt volume



Biplane (xPlane) Views: Short-axis view localizing the defect to the anteroseptal region. Orthogonal long-axis view confirming the trajectory of the shunt directly into the right ventricle outflow tract, diagnostic of an Aorto-RV fistula. Paravalvular leak visible.



Biplane (xPlane) Views: Short-axis view localizing the defect to the anteroseptal region. Orthogonal long-axis view confirming the trajectory of the shunt directly into the right ventricle outflow tract, diagnostic of an Aorto-RV fistula. Paravalvular leak visible.

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#FR 3 – Kiosk 3 – Poster 4

Bloody Cerebrospinal Fluid During Thoracoabdominal Aortic Aneurysm Repair

Presenting Author: Tyler Jackowski, MD, Fellow, Cardiothoracic Anesthesiology, Mayo Clinic

Introduction

Spinal cord ischemia is a known risk of thoracoabdominal aortic aneurysm repair. Ischemia can occur due to the disruption of blood supply with application of an aortic cross clamp, or due to exclusion of feeder arteries with graft material in both open and endovascular repairs.[1] Augmentation of spinal cord perfusion pressure (SCPP) can help to mitigate this risk. One strategy for increasing SCPP is placement of a spinal drain for intraoperative drainage of cerebrospinal fluid (CSF). Spinal drains can be associated with significant morbidity, however, and clinicians managing these devices must be prepared to troubleshoot complications as they arise.

Case Presentation

A 35-year-old female with history of Marfan syndrome and prior total aortic arch replacement presented for open repair of an extent II thoracoabdominal aortic aneurysm. Due to her anatomy, reimplantation of the intercostal arteries at the time of repair was not feasible, thus placing the patient at high risk for ischemia to the thoracic spinal cord. Preoperative placement of a spinal drain was therefore pursued. An intrathecal catheter was placed atraumatically prior to induction of general anesthesia. A maximum cerebrospinal fluid pressure (CSFP) of 10mmHg was maintained with 10cc or less of clear CSF removed each hour. The patient was placed on anterograde cardiopulmonary bypass (CPB) and cooled to an isoelectric EEG. At this point, anterograde flow was interrupted in exchange for retrograde total body perfusion to enable exposure of the proximal descending thoracic aorta. After a graft-to-graft anastomosis with her prior total arch replacement was completed, anterograde CPB was resumed while the remainder of the descending thoracic and abdominal aorta were replaced. Shortly before weaning of CPB, an abrupt increase in CSFP was observed. Per protocol, the spinal drain was opened and frank blood was noted. The surgical team was notified, and the remaining anastomoses were completed expeditiously. A mean arterial pressure of 90 mmHg was targeted and intermittent drainage of CSF was continued. Separation from CPB and reversal of heparinization occurred without issue. Coagulopathy was corrected with platelets and cryoprecipitate. Despite this, the CSF remained frankly bloody. An intraoperative neurosurgery consult was obtained. Awakening trial prior to leaving the operating room was pursued, and the patient was able to move her lower extremities to command. Emergent CT of the head and spine was without acute abnormality. The CSF eventually cleared and the drain was discontinued. The patient was discharged from the hospital two weeks later without any neurologic deficits.

Discussion

Bloody CSF during aortic surgery should raise concern for subdural/intracranial hemorrhage, venous congestion, or high velocity/high volume drainage.[2] Prompt correction of coagulopathy and augmentation of blood pressure to maximize SCPP should occur. Extreme care should be taken to avoid over drainage of

CSF, as this can lead to tearing of bridging veins and catastrophic subdural hemorrhage. Clinicians managing spinal drains intraoperatively should be aware of these potential complications, as prompt action should be undertaken to prevent devastating neurologic deficits.

Images



Figure 1: Computed tomography scan of lumbar spine showing intrathecal catheter inserting at L2-3 and extending cranially

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#FR 3 – Kiosk 3 – Poster 5

Under pressure: intra-operative abdominal compartment syndrome identified with transesophageal echocardiography

Presenting Author: Abigail MR. Bawden, MD, Resident Physician, University of Florida College of Medicine

Introduction

Abdominal compartment syndrome (ACS) is a highly lethal condition with mortality rates from 25-75% despite identification and treatment. As decompressive laparotomy reduces mortality rates by 16-37%, rapid identification is imperative[1]. Intra-abdominal pressure measurement remains the gold standard for diagnosis but may be impractical in the intraoperative setting. Transesophageal echocardiography (TEE) can detect evidence of ACS and is an evolving tool in intraoperative identification[1].

Case Presentation

A 69-year-old male with hypertension and hyperlipidemia was admitted to the hospital with a 6.8cm infrarenal aortic aneurysm and underwent open aneurysmal repair. The post-operative course was complicated by acute hypoxic respiratory failure requiring mechanical ventilation and veno-venous extracorporeal membrane oxygenation (ECMO). After persistent refractory hypotension developed, transthoracic echocardiogram demonstrated decreased biventricular systolic function. The decision was made to transition to veno-arterial ECMO with placement of percutaneous left ventricular assist device (LVAD). The patient was brought to the operating room supported on inhaled nitric oxide and epoprostenol with infusions of epinephrine, norepinephrine, vasopressin, amiodarone, and dobutamine. Initial TEE findings noted a dilated left ventricle with severely reduced ejection fraction (15-25%) and dilated right ventricle with moderately reduced systolic function. Percutaneous LVAD was placed with initial improvement in cardiac output and weaning of vasopressor requirement. Intraoperative blood loss was greater than expected, requiring resuscitation with blood products. Significant facial edema was noted, prompting reconfiguration to veno-arterio-venous ECMO. Subsequently, there was significant limitation in ECMO flows with concurrent refractory hypotension. TEE demonstrated reduced preload with near absence of biventricular filling, while the abdomen was markedly distended and tense. Cardiothoracic surgery performed initial decompressive laparotomy with evacuation of >2000mL of blood from the abdomen. There was immediate improvement in ECMO flow and TEE demonstrated markedly improved biventricular filling. Subsequently, vascular surgery identified the hemorrhagic source.

Discussion

This case demonstrates the utility of TEE in intraoperative ACS identification. Aortic hemorrhage is associated with ACS; one study noted 1.4% of patients developed ACS following aortic aneurysm repair[1,2]. This patient presented a complex clinical picture including cardiogenic shock, refractory hypotension, and significant intraoperative blood loss with large volume resuscitation. As such, the differential diagnosis was broad and multi-factorial. Intraoperative TEE findings of minimal biventricular filling aided diagnosis in this complex constellation of findings and guided anesthetic and surgical management. TEE provided

confirmation of presumed diagnosis when abdominal decompression resulted in prompt biventricular filling and improved hemodynamic stability. TEE proved to be a valuable and timely tool in ACS identification.

Images

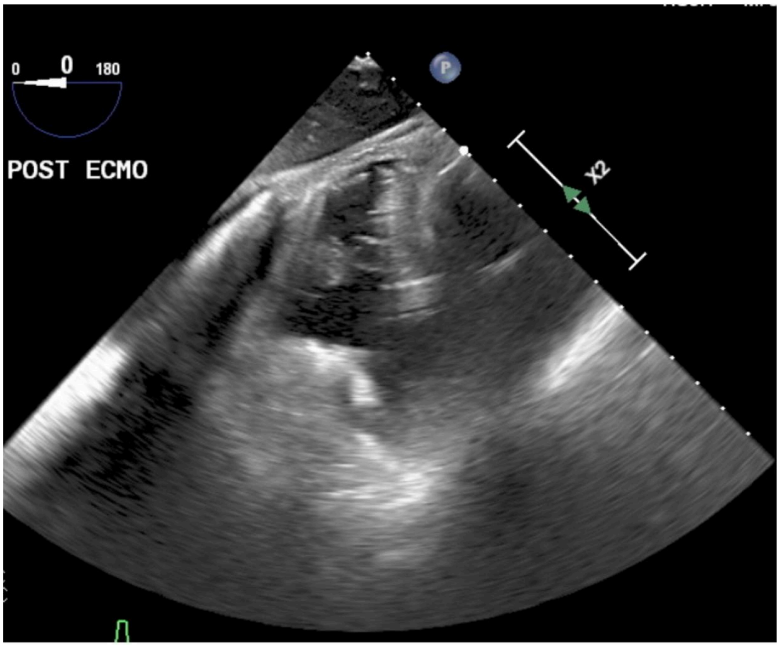


Figure 1: transgastric short-axis view of very empty right ventricle

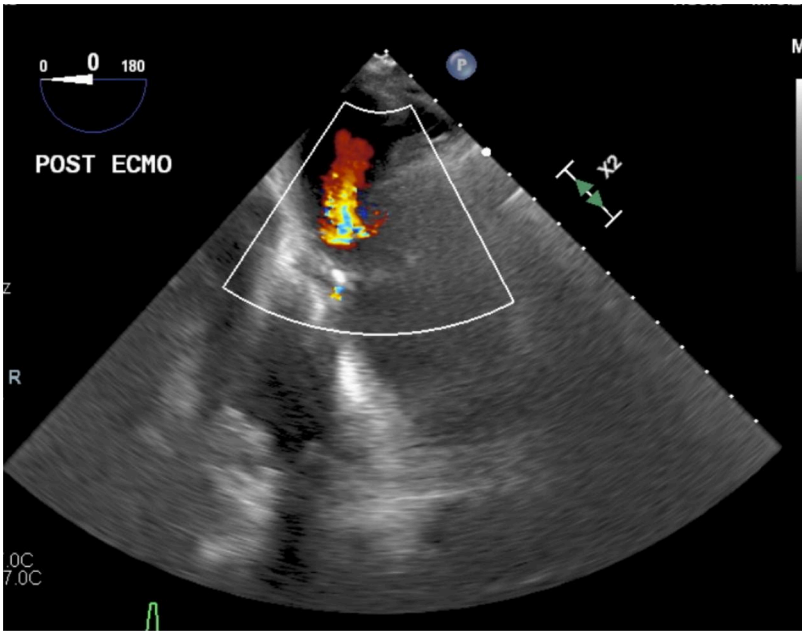


Figure 2: mid-esophageal four-chamber view with color Doppler flow over the mitral valve demonstrating very empty right and left ventricles

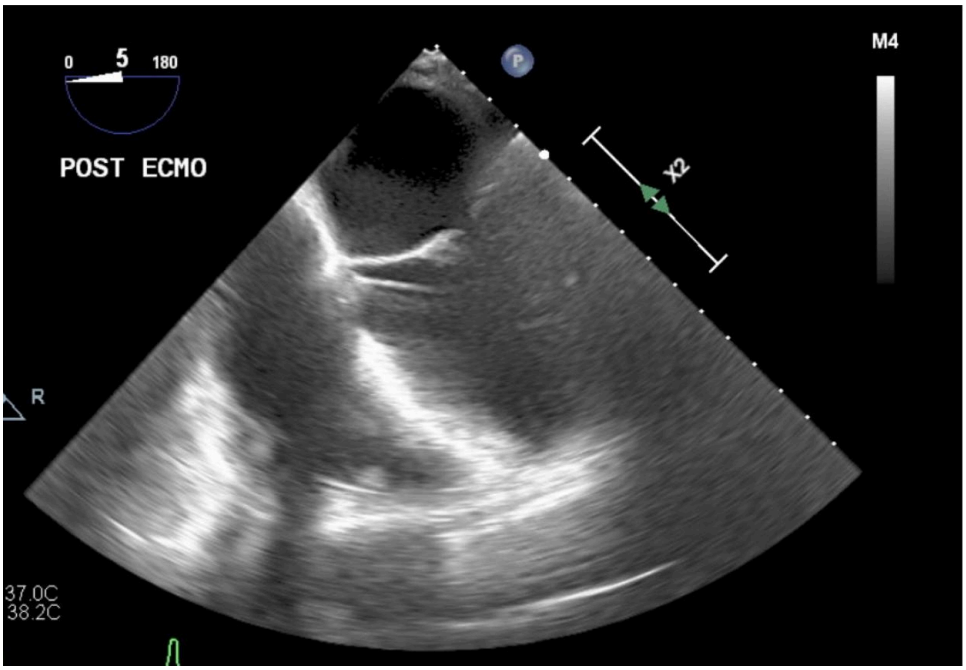


Figure 3: mid-esophageal four-chamber view demonstrating appropriate filling of right and left ventricles immediately following decompressive laparotomy

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#FR 3 – Kiosk 3 – Poster 6

When Tamponade Isn't Pericardial: CPB Readiness for Inflammatory Epicardial Compression

Presenting Author: Arvin Farhang, Resident, University of Connecticut

Introduction

Constrictive and inflammatory pericardial disease pose significant anesthetic complexity when imaging mimics infection or malignancy. We describe a multidisciplinary case highlighting an unexpected transition from a planned pericardial window to pericardectomy, intra-operative heparin resistance requiring targeted antithrombin repletion, CPB-enabled cardiac decortication, and dynamic ventricular function across peri-operative modalities.

Case Presentation

A 49-year-old male with hypertension was transferred for progressive dyspnea, pleural effusion, and a rim-enhancing pericardial collection causing severe RV compression with early tamponade physiology. Initial workup demonstrated sinus tachycardia with lateral T-wave inversions on ECG with an unremarkable CT angiography. cardiac MRI revealed a 6.6–8.3 cm multiseptated enhancing collection raising concern for an abscess or tumor, but the absence of systemic infection supported operative diagnosis. Limited TTE showed a hyperdynamic LV, a small externally compressed RV, elevated right-atrial pressures, ventricular interdependence, and paradoxical septal motion. Right thoracentesis drained 1 L of exudative fluid with marked symptomatic improvement. A pericardial window was attempted but abandoned due to lack of safe access. Median sternotomy revealed intact pericardium; instead, inflammatory epicardial tissue—not the pericardium, compressed the RV. The operation therefore pivoted from a planned pericardial window to pericardectomy due to the aggressive inflammatory epicardial rind directly causing RV outflow and chamber compression (as opposed to pericardial constraint)[1]. The patient exhibited heparin resistance, requiring antithrombin concentrate before CPB cannulation. An organized hematoma was evacuated, followed by extensive epicardial and peri-vascular decortication, after which RV tamponade physiology resolved. Diffuse coagulopathy from raw mediastinal surfaces mandated multimodal coagulation rescue with protamine reversal, 4-factor concentrate, blood products, and prolonged hemostasis. Ventricular function differed markedly: MRI LVEF 22% (likely LV underfilling), immediate post-CPB TEE LVEF 70%, and discharge-day limited TTE LVEF 33% with septal bounce but no clinical constriction. Cytology was negative and cultures remained sterile. Early guideline-directed heart-failure therapy was initiated with Entresto and Aldactone prior to discharge.

Discussion

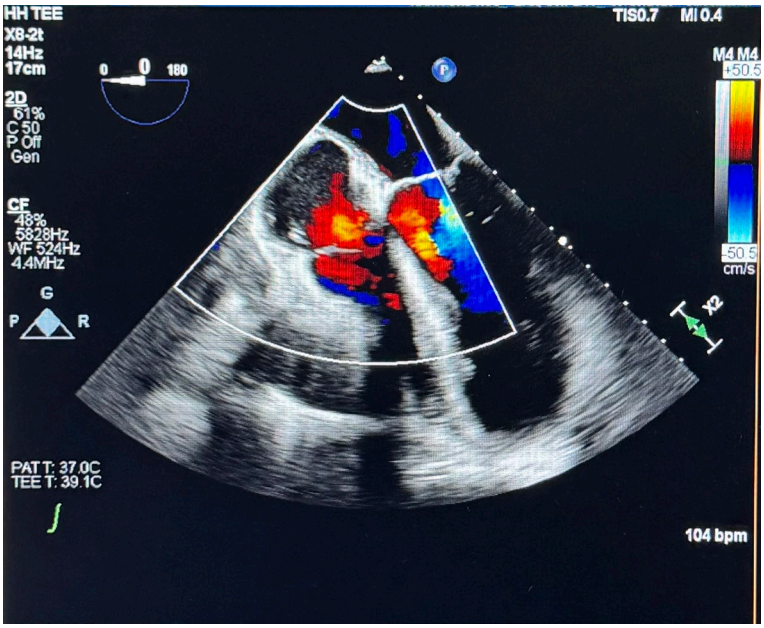
Key cardiac anesthesia lessons include: (1) preventing diagnostic anchoring by integrating imaging with clinical context, (2) recognizing that myocardial/epicardial inflammation, and not pericardial tissue, can be the true source of RV compression, demanding surgical and anesthetic flexibility, (3) managing acquired heparin resistance with targeted antithrombin repletion to achieve safe CPB anticoagulation, and (4) anticipating severe post-bypass coagulopathy from raw-surface bleeding after circumferential cardiac

decortication. Goal-directed TEE assessment guided real-time resuscitation and informed early post-operative cardiac medical optimization.

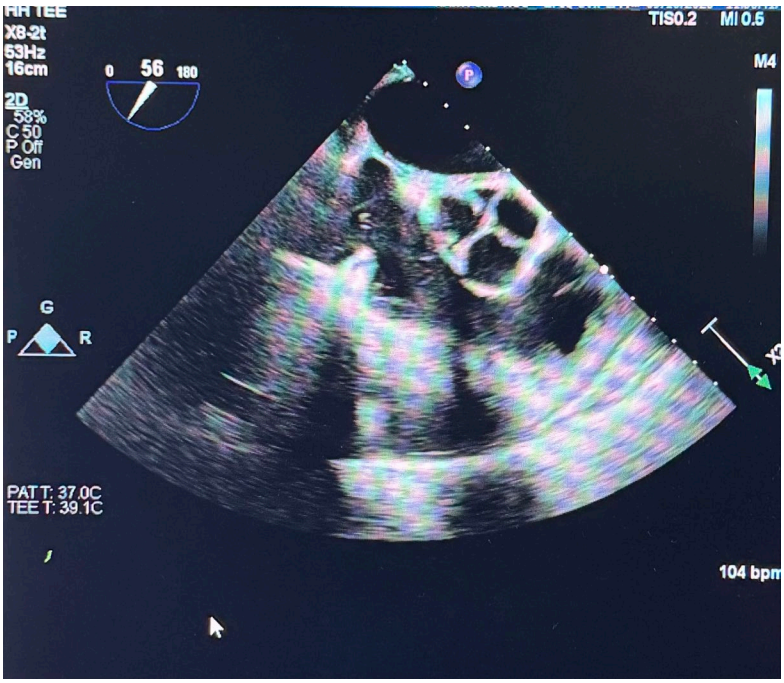
Images



Surgical Rind



4 Chamber View (with colour)



RV Inflow Outflow

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#FR 3 – Kiosk 3 – Poster 7

Acute intraoperative mitral valve disfunction

Presenting Author: Sijules Atang Abongwa, DO, Resident Physician, University of Alabama at Birmingham

Introduction

Prosthetic mitral valve obstruction immediately following cardiopulmonary bypass (CPB) is rare but potentially catastrophic. Early recognition relies heavily on post-bypass transesophageal echocardiography (TEE). We present a case of a Mitris Resilia valve holder detected on intraoperative TEE after mitral valve replacement (MVR), requiring an unplanned return to CPB. This case highlights the importance of meticulous post-implant imaging and proficient knowledge of bioprosthetic valve parts and implantation steps.

Case Presentation

A 73-year-old man with hypertension, CKD III, diabetes, and multivessel coronary artery disease presented with progressive dyspnea. Outside hospital evaluation revealed severe eccentric mitral regurgitation due to a flail anterior leaflet, and significant LAD and LCx lesions. He underwent CABG ×3 and MVR with a 33-mm Mitris Resilia bioprosthetic valve. Baseline TEE demonstrated LV EF 50-55%, moderately dilated RV with reduced function, and severe MR. During initial bypass separation, TEE evaluation revealed abnormal appearance of the bioprosthetic valve with a fixed central obstruction, anesthesia communicated with surgeon with hypothesis of valve holder as possible culprit once TEE 3D evaluation was completed. Bypass, cross clamps and cardiac arrest was re-established, and the retained holder was retrieved without further complications. Repeat TEE demonstrated a well-seated valve with normal leaflet motion and a mean gradient of 2 mmHg. Postoperatively, the patient experienced new-onset atrial fibrillation requiring DCCV. Remainder of hospital course was uneventful.

Discussion

This case highlights the critical importance of comprehensive post-bypass TEE using multiple imaging modalities (2D, Doppler, and 3D) to verify prosthetic valve function. Moreover, familiarity with the structural components of implanted devices and the sequence of surgical implantation steps enhances the clinician's ability to rapidly narrow the differential diagnosis for acute prosthetic dysfunction. Retention of the Mitris Resilia valve holder is uncommon but can occur due to its two-step removal process. After releasing the adapter–handle, the surgeon must tie and cut the securing sutures before subsequently removing the holder, creating a vulnerable interval in the workflow. In this case, prompt echocardiographic recognition and immediate communication with the surgical team enabled rapid correction and prevented significant prosthetic valve obstruction and hemodynamic compromise.

Key learning points include:

1. Thorough post-implant TEE evaluation is mandatory for all prosthetic valves.

2. Knowledge of surgical devices and parts expedites early recognition of prosthetic obstruction can prevent irreversible hemodynamic compromise.
3. Multidisciplinary coordination enables rapid re-initiation of CPB for foreign body retrieval and restoration of normal bioprosthetic function.

Images

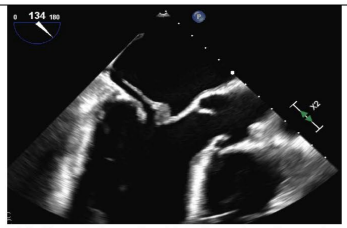


Fig 1: Flail anterior mitral leaflet of native valve



Fig 2: severe mitral regurgitation jet of native valve

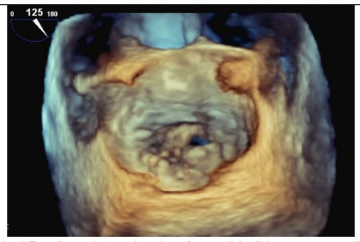


Fig 3: 3D of native mitral valve with A2 segment flail



Fig 4: 2D + Color doppler, post bypass 1, unable to identify leaflets and trans-mitral jet.

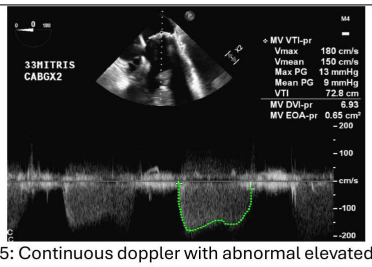


Fig 5: Continuous doppler with abnormal elevated Mean PG. Post bypass 1.

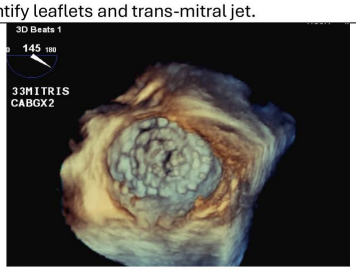


Fig 6: 3D reconstruction of mitral valve post bypass 1

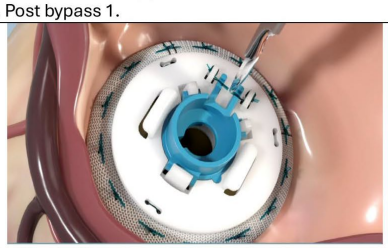


Fig 7: illustration of valve with holder



Fig 8. 2D showing mitral valve, post bypass 2 - valve leaflets identifiable, opening well.

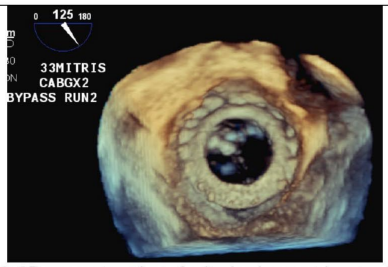


Fig 9: 3D reconstruction of mitral valve post bypass 2

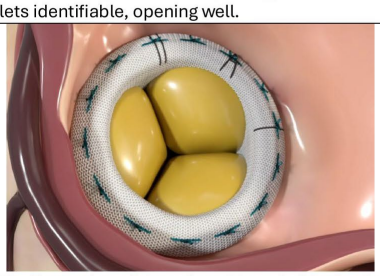


Fig 10. Illustration of the valve without holder in place.

series of images of native valve and bioprosthetic valve post bypass 1 and post bypass 2

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Edwards Lifesciences. Mitris Resilia Surgical Implantation Guide

#FR 3 – Kiosk 4 – Poster 1

Anesthetic Challenges during Simultaneous Anterior Mediastinal Mass Resection and Coronary Artery Bypass Grafting

Presenting Author: Grace Pierce, MD, Adult Cardiothoracic Anesthesiology Fellow, University of Colorado

Introduction

Invasive thymic carcinoma carries significant perioperative risk from airway and vascular compression. Symptomatic multivessel coronary disease with complete total occlusion of the proximal left anterior descending (LAD) artery adds additional challenges. Tumor encasement of the great vessels and left innominate vein mandated vein sacrifice, eliminating all left-sided central access and prohibited complete tumor resection. We report single-stage palliative debulking with off-pump single-vessel revascularization using left radial artery from innominate artery to LAD. To the authors' knowledge, this configuration in invasive tumors has not been published.

Case Presentation

A 75-year-old male with thymic carcinoma post-neoadjuvant chemotherapy and concomitant complete total occlusion of the proximal LAD and multivessel disease presented for anterior mediastinal mass (AMM) resection with 3-vessel coronary artery bypass grafting (CABG.) Preoperatively, he reported no difficulty with breathing while lying flat and imaging ruled out significant airway or vascular compression. It was discussed with the surgeon that if right sided veins also needed to be sacrificed, they would obtain femoral venous access for volume and drug administration. Following successful induction with a balanced anesthetic and double lumen tube intubation, central venous access was obtained via the right internal jugular vein and median sternotomy was performed. The tumor had invaded the manubrium, pericardium, bilateral internal mammary arteries (IMA), left innominate vein which was isolated and transected, ascending aorta, innominate/left carotid confluence, and pulmonary artery (PA). Limited resection was performed, but complete resection was abandoned as it required arch vessel debranching and arch replacement with circulatory arrest.

After a multidisciplinary discussion among senior surgical attendings, CPB with aortic cross-clamping and multivessel grafting were deemed prohibited by aortic involvement. Off-pump revascularization was limited to left radial artery from innominate artery to LAD for symptomatic relief. The aorta was not viable as a proximal graft site given tumor invasion. After successful bypass, the patient was reintubated with a single lumen tube and transported to the intensive care unit.

Discussion

This case presented several anesthetic challenges: induction considerations in an AMM, one-lung ventilation, limited venous access requiring preoperative planning, inability to utilize cell salvage, and increased susceptibility to hypotension-induced myocardial ischemia. Sacrifice of the left innominate vein significantly reduced access options for emergent scenarios, and sites for invasive monitors. Tumor involvement prevented CPB with cardioplegic arrest and the use of either IMA. Innominate artery use,

described almost exclusively for porcelain aorta or redo operations, has no reported precedent when mandated by tumor invasion. The single-vessel graft was successful but considered palliative. This case sites a unique intersection of mediastinal mass physiology, mandatory large vein sacrifice, prohibitive aortic disease, obligatory off-pump single-vessel CABG via an unreported conduit, and one-lung ventilation, demanding contingency planning and real-time multidisciplinary adaptation.

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#FR 3 – Kiosk 4 – Poster 2

Intraoperative Management of Systolic Anterior Motion /Left Ventricular Outflow Tract Obstruction Following Complex Mitral Valve Repair

Presenting Author: Amos Alcius, DO, Adult Cardiac Anesthesia Fellow, Tufts Medical Center

Introduction

Systolic anterior motion (SAM) resulting in left ventricular outflow tract obstruction (LVOTO) is a recognized complication following mitral valve repair (MVR). Risk assessment of the SAM predictors during pre-repair transesophageal echocardiography (TEE) and prompt diagnosis on the post repair TEE is crucial for a positive patient outcome. Once diagnosed, it is necessary to have a systematic approach to manage SAM and unmask any risk for future SAM.

Case Presentation

65-year-old male with hypertension and chronic mitral regurgitation that was medically managed and surveilled with annual transthoracic echocardiograms (TTE). The patient was asymptomatic, riding his bike daily. Most recent TTE demonstrated progressive left ventricular and left atrial dilation. Pre-incision TEE evaluation of SAM/LVOTO predictors revealed the patient has mild to moderate risk. The surgeon completed a complex MVR with a 34 mm Cosgrove annuloplasty band. Real time assessment by TEE following MVR revealed SAM with dynamic LVOTO with a peak gradient of 61 mm Hg. Ionotropic support was discontinued, norepinephrine was transitioned to phenylephrine, augmentation of preload was performed, and finally short acting beta blocker esmolol was administered which showed significant improvement of SAM/ LVOTO.

Discussion

The incidence of SAM post mitral valve repair is estimated to approximately 10% of cases[1]. The severity of SAM presents on a continuous spectrum with variable clinical presentations from a mild form without LVOTO to a more severe form with LVOTO resulting in profound hemodynamic instability[2]. Most cases resolve with medical management; however, persistent SAM has been reported in some patients requiring reoperation. Overall, the surgical management of SAM in the operating room can be controversial[3]. Taking into consideration the functional status of the patient, there were significant concerns of unmasked SAM during physical exercise after discharge. A successful mitral valve repair means durable correction of the mitral valve regurgitation with preservation of, or improvement in, ventricular function and exercise tolerance, without creating iatrogenic obstruction or stenosis. In attempt to ascertain SAM/ LOVTO, a 10 mcg/kg/min dobutamine stress was performed. This failed to induce SAM/LVOTO. The peak LVOT gradient was noted to be 20 mm Hg with trace mitral regurgitation. There was also improvement in the left pulmonary vein doppler profile. This is suggestive that SAM is less likely to be returned while the patient is exercising; however, there is a possibility that future remodeling of the ventricle could unmask SAM in the future. After discussion with the surgical team, the patient was taking to the cardiac surgical intensive care unit without additional intervention and extubated on postop 1 day and discharged on postop day 8. At 3 months postoperatively, the patient's condition was back to his regular routine, with no symptoms during exercise.

Follow up TTE showed neither SAM nor LVOT obstruction. It is important to assess the risk of SAM/ LVOTO prior to mitral valve repair and to follow a systematic approach to the management of post repair SAM/LVOTO. It is also important to consider that patient's baseline functional status when assessing for the success of any valvular repair.

Images

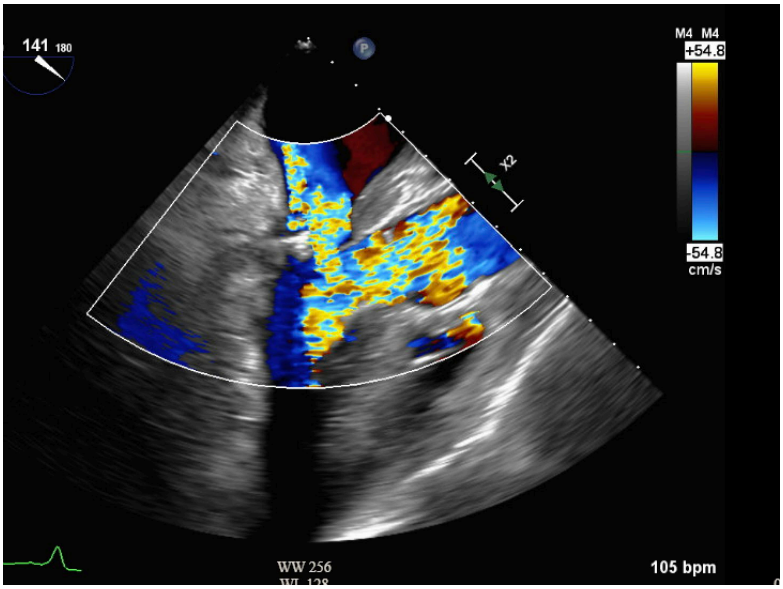


Figure1: Postoperative SAM following mitral valve repair with posteriorly directed flow representing concurrent LVOT obstruction

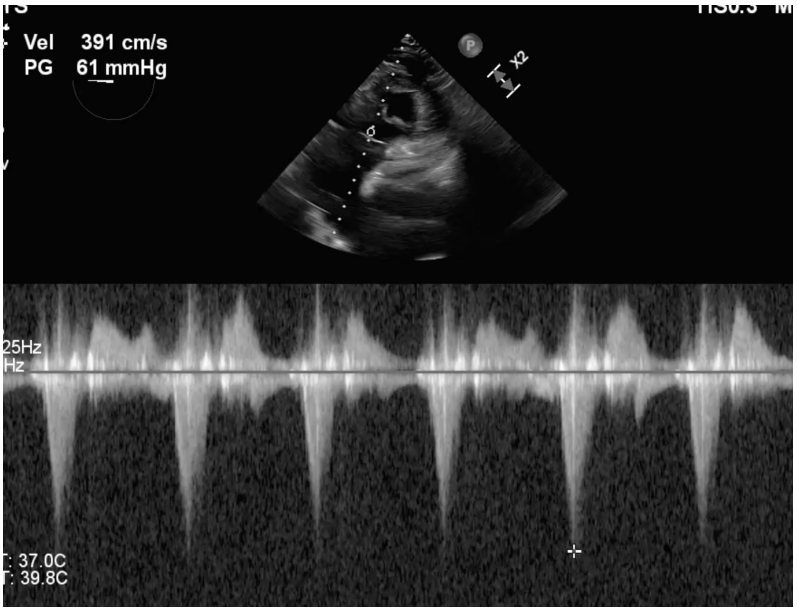


Figure 2: LVOT gradient > 50 mm Hg representing the severity of SAM

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#FR 3 – Kiosk 4 – Poster 3

A "Triple Trouble" Complication: Aorto-RV fistula versus Ventricular Septal Defect, Decompensated Heart Failure and Bioprosthetic Aortic Valve Stenosis after TAVR

Presenting Author: Gabriel Garcia, MD, University of Florida/Health Shands

Introduction

An aorto-right ventricular (RV) fistula or ventricular septal defect (VSD) is a rare but serious complication of transcatheter aortic valve replacement (TAVR), thought to arise from annular/root disruption during valve deployment—particularly in patients with heavy calcification, and asymmetric anatomy. These lesions may lead to right-sided heart failure. The optimal management strategy remains unclear but include conservative observation to surgical or percutaneous closure. [1,2] We report a case of a 65-year-old man with a PMH of TAVR who developed various complications in the setting of VSD versus aorto–RV fistula.

Case Presentation

A 65-year-old male with PMH of OSA, HLD, HTN, ICD, and progressive dyspnea on exertion was transferred from an outside hospital for an explant of a TAVR, ventricular septal defect (VSD) closure, aortic valve replacement, and a single vessel bypass. After induction on the initial operative day, TEE revealed severe right heart failure and depressed LV function (EF 10-15%) despite nitric oxide, prompting case cancellation and hemodynamic optimization with diuresis and inotropic support. Following stabilization, he returned to the OR for intended intervention. Intraoperative TEE demonstrated a moderately enlarged LV with severe global hypokinesis, a mildly dilated RV with reduced function, mild-moderate MR, moderate TR, and a bioprosthetic TAVR valve with stenosis (mean gradient 40 mm Hg), paravalvular leak (PVL) and a perimembranous VSD (images 1-3). The surgical team noted a Siever's type 1 bicuspid aortic valve (BAV) with fusion of the left and right cusps. The raphe was heavily calcified. The valve frame was bent (images 4 and 5), and the valve set at an angle directed towards the membranous septum. A communication between the LVOT and RV was found in the membranous septum (image 6) as seen in image 6. Post TAVR, the patient had encountered a clinical sequalae of eccentric valve deployment, bent valve frame, AV block requiring PPM, high RV pacing dependence, PVL, premature structural valve degeneration and a perimembranous VSD followed by TAVR explant, VSD closure, aortic valve replacement and single vessel CABG.

Discussion

TAVR is now a safe and feasible option for BAV-related aortic stenosis, no longer considered contraindicated. However, BAV patients experience higher rates of moderate to severe PVL, need for pacemaker implantation, and slightly higher rates of second valve implantation and conversion to surgery. BAV morphology such as calcified raphe and excess leaflet calcification is associated with worse outcomes after TAVR. [2,3] Despite these challenges, mortality and major complication rates are similar to those in tricuspid aortic stenosis patients after two years. [2]


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Images

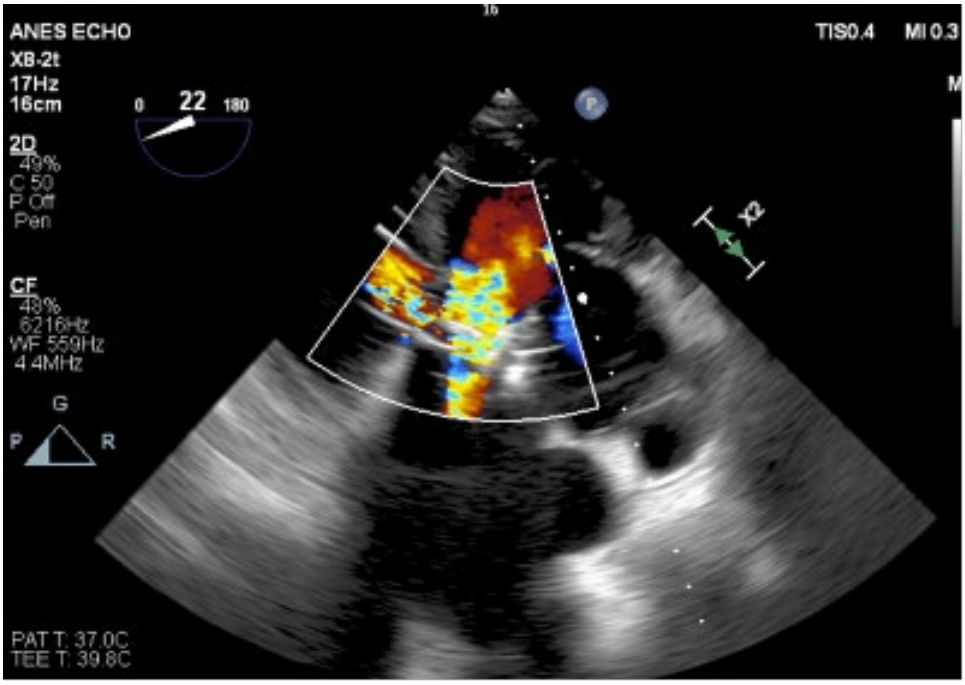


Image 1

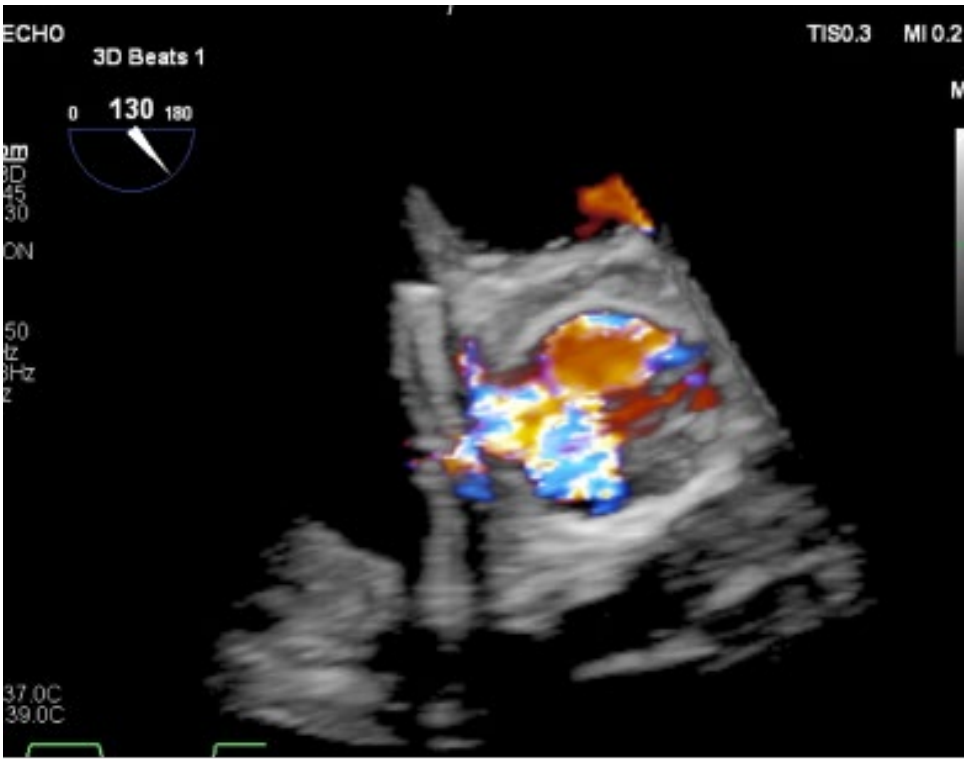


Image 2



Image 3



Image 4

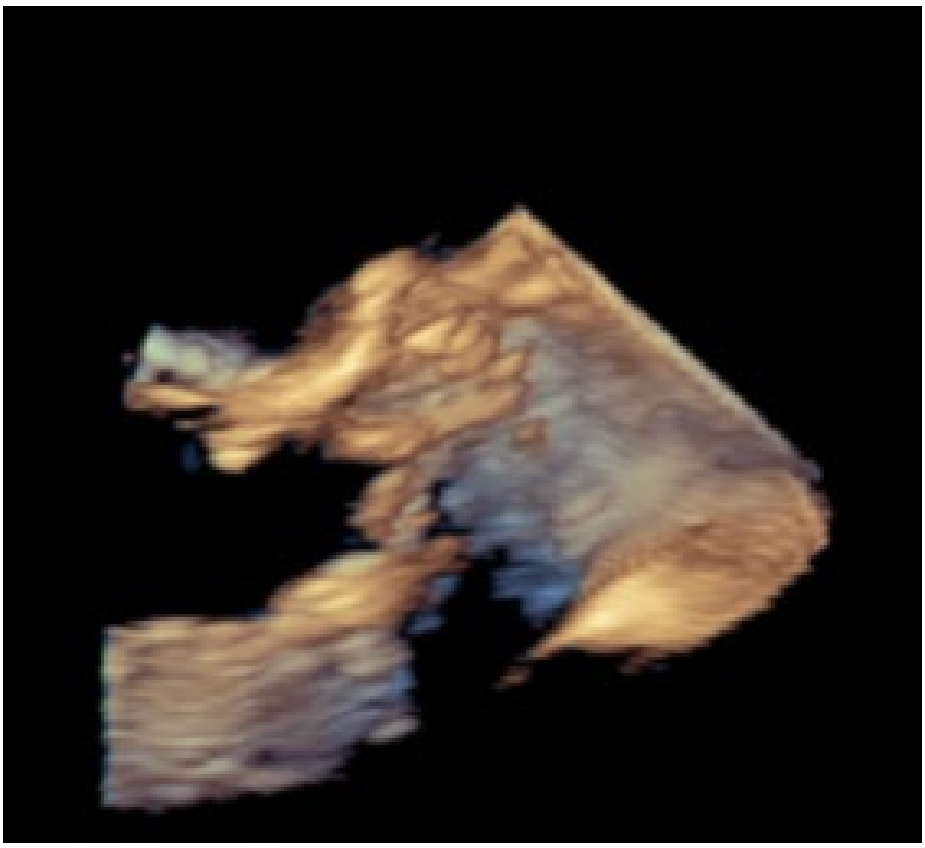


Image 5



Image 6

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#FR 3 – Kiosk 4 – Poster 4

Anomalous Inferior Vena Cava during Minimally Invasive Cardiac Surgery: The Diagnostic Role of Intraoperative TEE

Presenting Author: Joshua Durbach, MD FACP, Fellow, University of Miami/Jackson Memorial

Introduction

Inferior vena cava (IVC) anomalies are a rare occurrence, with an incidence of 0.2-3% in the general population. We report a previously undiagnosed absent intrahepatic IVC with azygous continuation, which complicated femoral venous cannulation in a patient undergoing minimally invasive cardiac surgery (MICS).

Case Presentation

A 68-year-old male with severe symptomatic aortic stenosis underwent minimally invasive aortic valve replacement under general anesthesia. 'Bicaval' femoral venous cannulation using a Seldinger technique was attempted prior to institution of cardiopulmonary bypass (CPB) under transesophageal echocardiography (TEE) guidance. However there was failure to visualize the guidewire in the right atrium (RA) on multiple TEE planes including the midesophageal bicaval view (figure 1A). Closer echocardiographic evaluation could not detect a distinct intrahepatic IVC, and the previously advanced guidewire appeared to be present in an extrahepatic vascular structure (Figure 1B). Considering the overall ease of passage of the guidewire, A 25 Fr venous cannula was advanced under fluoroscopic guidance, which initially appeared to be satisfactorily placed. However, there was continued inability to visualize the venous cannula in the RA on TEE, as well as failure to palpate it at the level of the RA- superior vena cava (SVC) junction, further raising concern for an anomalous IVC. A subsequent intraoperative venogram displayed drainage of contrast through the cannula into the SVC before transiting the RA, confirming our suspicion of a possible direct IVC communication with the SVC. CPB was resumed after placement of an additional sump suction in the RA, resulting in adequate venous emptying of the heart. The remainder of the surgery and perioperative period was uneventful. A post operative CT chest and abdomen demonstrated infrarenal IVC duplication with confluence above the kidneys. The IVC did not course intrahepatically. Instead, the IVC continued into a markedly dilated azygous vein before draining into the SVC, entirely bypassing the RA (figure 2). These findings were consistent with our intraoperative clinical impression of the anomalous anatomy.

Discussion

Despite perceived ease of advancement of a guidewire or venous cannula into the RA, failure of visualization on TEE should raise concerns for an IVC anomaly, and prompt further evaluation. These anomalies directly affect venous return, central venous access, cardiopulmonary bypass cannulation strategies, and overall hemodynamic management. Interrupted or absent infrahepatic IVC with azygos/hemiazygos continuation markedly alters normal venous drainage patterns and is frequently accompanied by other congenital cardiovascular defects. This case underscores important educational points of comprehensive understanding of IVC developmental variants and alternative venous cannulation strategies in these situations, ensuring adequate venous drainage, and may prevent potential vascular injury during MICS.

Lastly, thorough preoperative review of imaging (CT scan or MRI) to anticipate anatomical variations, plan alternative cannulation strategies and mitigate intraoperative complications.

Images

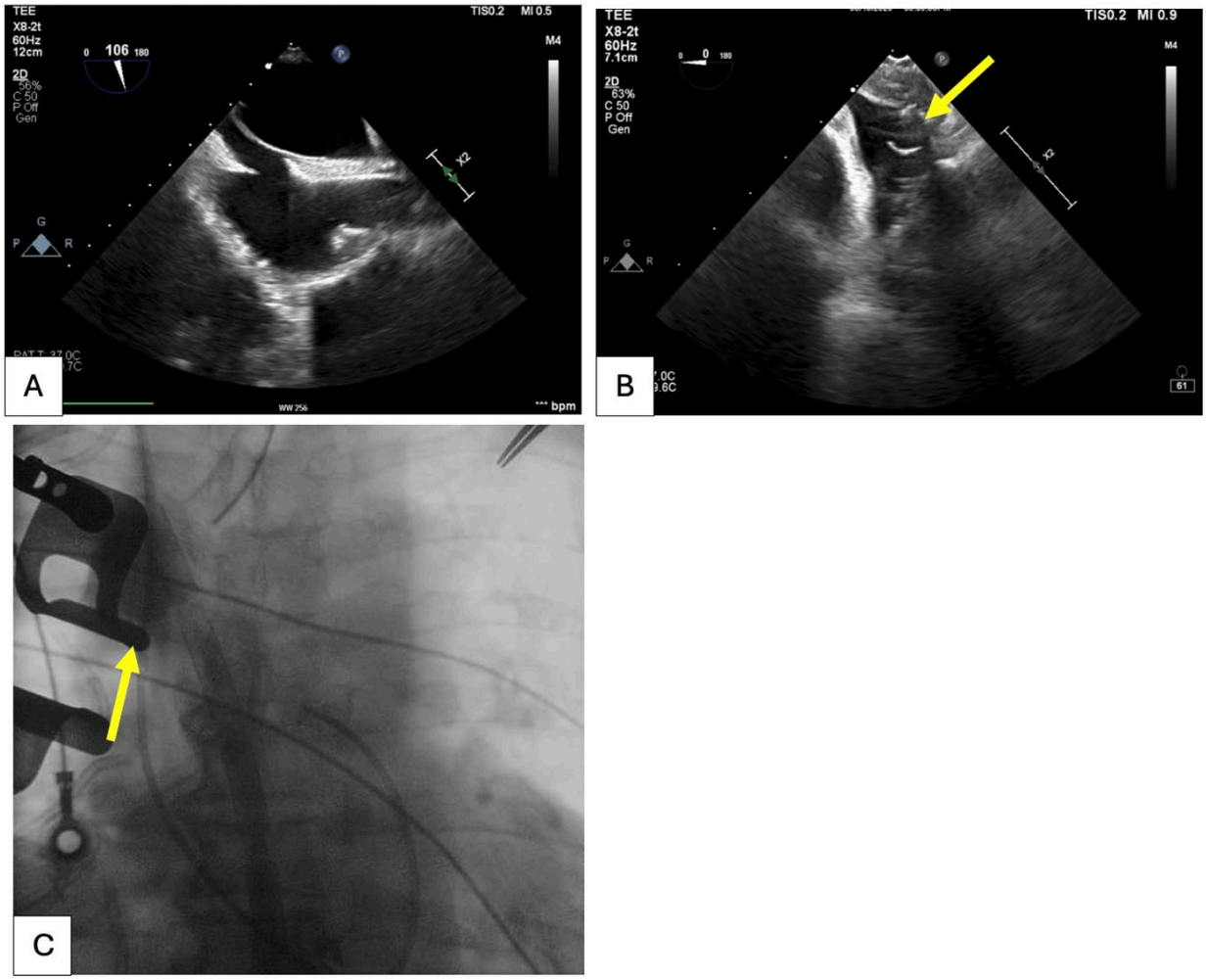


Figure 1 TEE ME bicaval view with the clockwise probe rotation unable to demonstrate guidewire despite easy advancement (A). Deep esophageal 0 degree with clockwise rotation with inability to visualize a distinct intrahepatic IVC. The guidewire appeared to be within an extrahepatic vessel adjacent to the liver (B). Venogram still showing the venous cannula with contrast appearing to enter the SVC without prior transit through the RA (C).

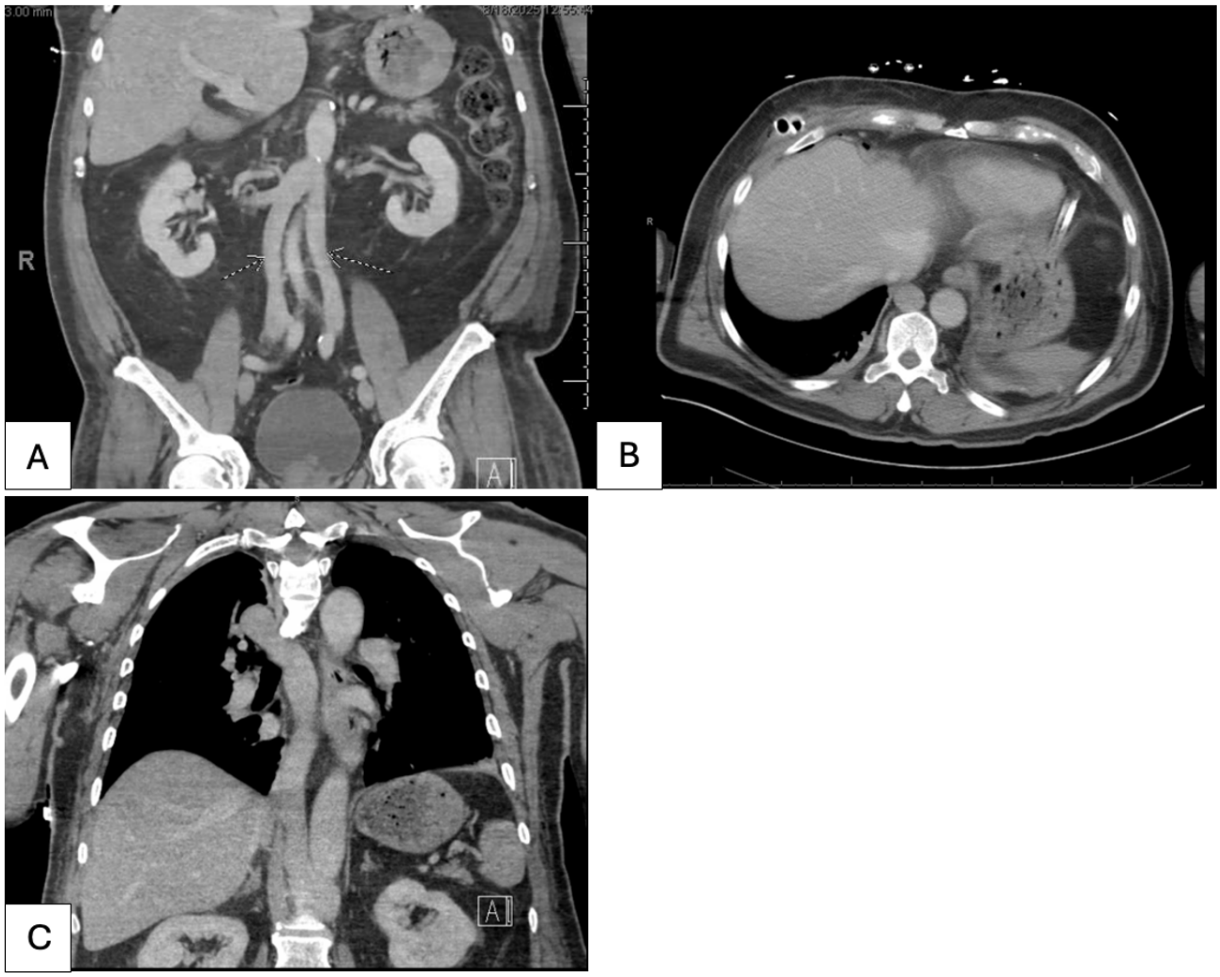


Figure 2: CT of the chest and abdomen showing infrarenal duplication of the IVC with subsequent confluence (A). The IVC maintains an extrahepatic course (B) before communicating with the SVC through a dilated azygous vein (C).

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#FR 3 – Kiosk 4 – Poster 5

Elective Coronary Artery Bypass Graft Complicated by Severe and Persistent Post-Operative Bleeding and Newly Diagnosed Factor XIII Deficiency

Presenting Author: Patrick J. Monette, MD, Anesthesiology Resident, University of Virginia

Introduction

Factor XIII (FXIII) deficiency is a rare coagulation disorder that can be inherited or acquired. Activated FXIII cross links fibrin and stabilizes clot. FXIII also has roles in wound healing, tissue repair, and immune response. Acquired forms of FXIII deficiency are more common and occur due to autoantibodies, liver dysfunction, or consumption. We present a case of a patient who underwent an elective CABG complicated by severe, recurrent postoperative bleeding. The patient was diagnosed with moderate FXIII deficiency which responded to treatment with FXIII concentrate.

Case Presentation

A seventy-year-old man with symptomatic CAD underwent elective CABG. His PMH was notable for HTN, T1DM, and CKD. Though he had no diagnosed bleeding disorder, family reported prior poor wound healing and easy bruising. The index operation was relatively uneventful, although he required multiple rounds of packing for hemostasis after CPB despite normal viscoelastic testing. His early post-operative course was complicated by saphenous vein harvest site bleeding requiring multiple transfusions and acute renal failure. He had cardiac arrest on POD6 requiring CALS with resternotomy, regaining ROSC but with massive bleeding and transfusion needs. After sternal re-closure, he continued to have slow bleeding from his harvest site and chest drains. Seven days later, he experienced another PEA arrest requiring CALS and resternotomy, again complicated by bleeding. On POD21 hematology was consulted given his unusual bleeding issues. VWF levels, FV, FVII, FVIII, FIX, FX, and FXI were normal. He developed mediastinitis and underwent sternectomy with pectoral flap closure on POD31. Three days later he was found to have a large mediastinal hematoma that required washout. Additional coagulation factor levels were checked and normal, though FXIII activity was 45%. The flap did not heal well and bled consistently, requiring numerous transfusions and two revisions on POD51 and POD87 due to persistent oozing. Five days after his second revision, bleeding continued and his FXIII antigen was 37%. At this time, he was treated with 40 IU/kg of FXIII concentrate. After treatment, his FXIII increased to 119% and bleeding stopped. Ultimately, the patient transitioned to comfort care and hospice given poor wound healing and persistent multiorgan failure.

Discussion

Herein, a patient with a history of poor wound healing and easy bruising experienced multiple bleeding complications after CABG as well as mediastinitis. The only abnormality found on extensive coagulation factor testing was moderate FXIII deficiency. Initially it was felt that his FXIII level was not low enough to be contributory to his bleeding; however after his treatment with FXIII concentrate his transfusion requirement ceased. FXIII levels were not low enough to be consistent with typical congenital FXIII deficiency, suggesting acquired deficiency. Initial efforts to replace FXIII with cryoprecipitate (roughly 60 IU per bag) were not

adequate, but treatment with FXIII concentrate and antifibrinolytics was effective. Acquired FXIII deficiency should remain in the differential diagnosis for patients with persistent coagulopathic bleeding after cardiac surgery, particularly those with poor wound healing.

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#FR 3 – Kiosk 4 – Poster 6

Anesthetic Management in a Patient with Intrathoracic Uterine Carcinosarcoma Metastasis

Presenting Author: Ryan M. Lynn, DO, Anesthesiology Resident, Loyola Medicine

Introduction

Large intrathoracic or mediastinal masses can cause catastrophic airway and cardiovascular compromise under general anesthesia due to compression of the tracheobronchial tree, great vessels, and cardiac chambers. Loss of muscle tone, decreased venous return, and positive pressure ventilation may precipitate sudden cardiovascular collapse. We present a case of a giant metastatic intrathoracic tumor causing severe right heart compression and near-total right lung collapse requiring high-risk anesthetic management.

Case Presentation

A 61-year-old woman with stage IV uterine carcinosarcoma, hypertension, and atrial fibrillation presented with worsening dyspnea and right-sided chest pain. CT revealed rapid tumor progression to a 16 cm mass causing near-total right lung collapse and marked compression of the right atrium and ventricle. Ventilation-perfusion (V/Q) scan showed absent ventilation and perfusion of the right lung. TTE showed right ventricular (RV) enlargement, reduced RV systolic function (TAPSE 13.5 mm), and interventricular septal flattening ("D-sign") consistent with severe RV pressure overload from extrinsic compression. Given worsening cardiopulmonary compromise, she underwent planned right hemi-clamshell thoracotomy with possible resection. Due to high risk of cardiovascular collapse, pre-induction arterial line and bilateral femoral venous access for potential ECMO cannulation were placed. Induction was performed with ketamine and dexmedetomidine, maintaining spontaneous ventilation. A single-lumen tube was placed and exchanged for a left double-lumen tube soon after induction. Intraoperatively, TEE demonstrated severe right atrial compression with right-to-left bowing of the interatrial septum and persistent RV dysfunction. Moderate vasopressor support was required. Central venous pressure (CVP) remained elevated (25–35 mmHg), reflecting ongoing right atrial obstruction. Surgical exploration revealed extensive invasion into the chest wall and diaphragm without a safe resection plane. Tumor resection was then aborted and the chest was partially closed to allow expansion and reduce cardiac compression. The patient was then transferred intubated to the ICU in stable condition.

Discussion

This case highlights the anesthetic challenges of massive intrathoracic tumors causing cardiac compression, with a high risk of peri-induction collapse. Despite outward hemodynamic stability on physical exam, risk stratification using advanced imaging demonstrated near-complete right lung exclusion, RA compression, and acute RV strain. Given this physiology, proactive planning for extracorporeal membrane oxygenation (ECMO) was essential, as recommended for giant mediastinal masses (Shen, 2025). Bilateral femoral venous access and invasive monitoring were established pre-induction to facilitate rapid ECMO if needed, with cannulation strategy guided by dominant physiology (Bertini, 2023). When the tumor was found

to be unresectable due to diaphragmatic and chest wall invasion, the operation was aborted to avoid catastrophic RV failure, consistent with prioritization of patient safety over a limited oncologic benefit.

Images

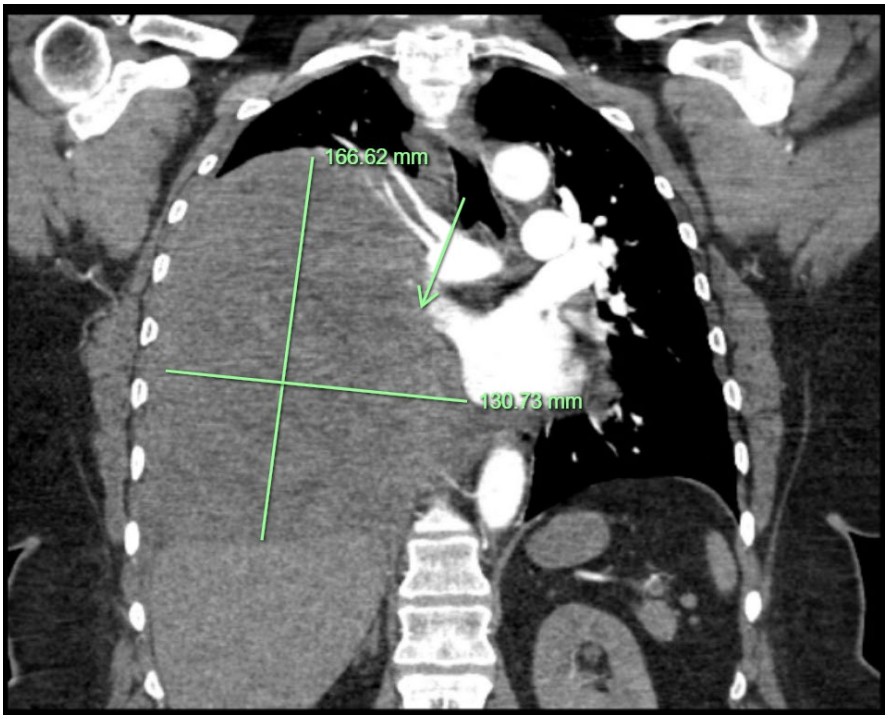


Figure 1: Coronal CT image of 16 cm × 13 cm × 14.9 cm intrathoracic mass demonstrating extrinsic compression of the right atrium and right pulmonary artery.

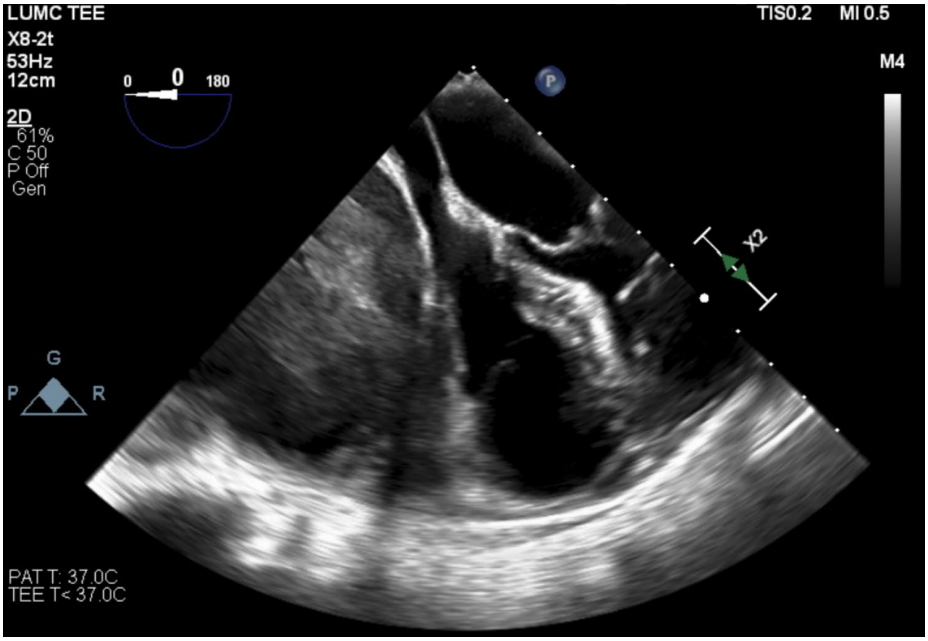


Figure 2: Mid-esophageal TEE view demonstrating severe extrinsic compression of the right atrium by a large intrathoracic mass, resulting in marked narrowing and distortion of the right atrium.

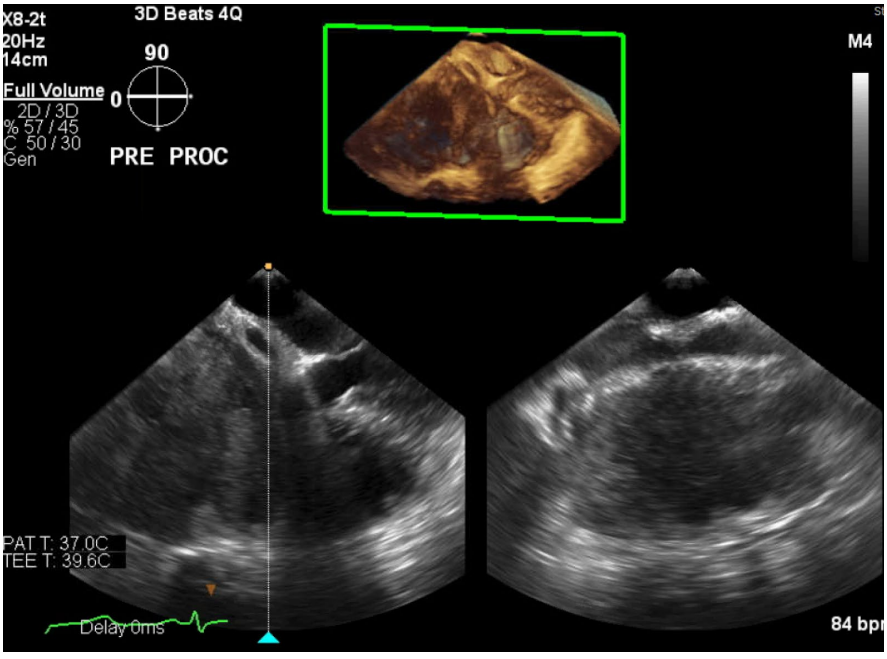


Figure 3: Three-dimensional transesophageal echocardiographic reconstruction demonstrating severe extrinsic compression and distortion of the right atrium by a large intrathoracic mass.

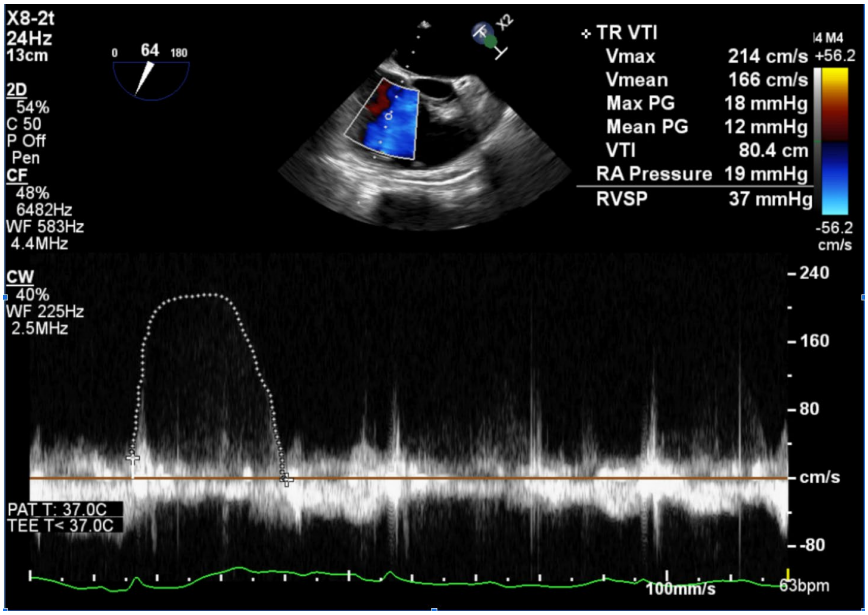


Figure 4: Mid-esophageal right ventricular inflow view with color and continuous-wave doppler demonstrating tricuspid regurgitation (TR). Continuous-wave doppler across the TR jet shows a peak velocity of 2.14 m/s, corresponding to an estimated right ventricular systolic pressure (RVSP) of 37 mmHg, consistent with elevated right-sided pressures in the setting of extrinsic right atrial and ventricular compression.

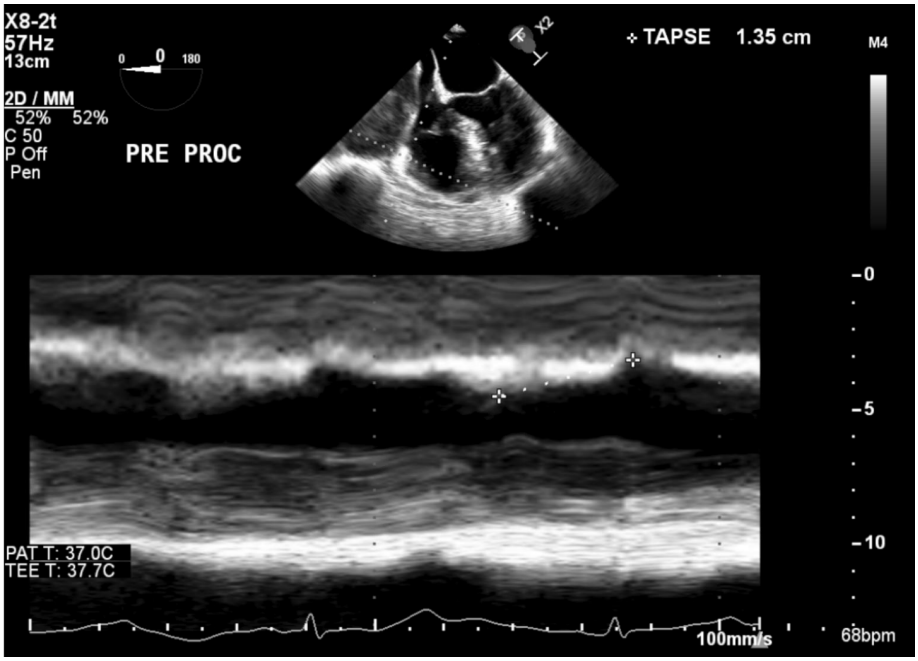


Figure 5: Mid-esophageal four-chamber transesophageal echocardiographic view demonstrating reduced right ventricular systolic function with tricuspid annular plane systolic excursion (TAPSE) of 1.35 cm, consistent with right ventricular dysfunction.

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#FR 3 – Kiosk 4 – Poster 7

Aortic valve bypass as an alternative to re-do aortic valve replacement in high-risk patient with severe bioprosthetic aortic stenosis: A case report

Presenting Author: Emily Larkin, MD, Johns Hopkins University

Introduction

Aortic valve bypass (AVB), also known as apicoaortic valve conduit, is an uncommon treatment of aortic stenosis with just over 100 reported cases (1). AVB involves creating a vascular graft with a bioprosthetic valve connecting the left ventricular apex to the descending thoracic aorta, bypassing the stenotic aortic valve (Figure 1A, B). It serves as an alternative for patients at high-risk for conventional surgical aortic valve replacement (AVR) when transcatheter aortic valve replacement (TAVR) is not feasible.

Case Presentation

An eighty-year-old female with a history of bioprosthetic AVR and two-vessel coronary artery bypass presented with progressive dyspnea on exertion. Evaluation revealed severe bioprosthetic aortic valve stenosis (mean gradient 41 mmHg) and severe two-vessel coronary disease (Table 1). Due to her coronary anatomy, she was not a candidate for valve in valve TAVR. Estimated risk for repeat sternotomy and AVR showed an 18% morbidity and mortality rate. A multidisciplinary team recommended AVB as an alternative. The patient was anesthetized and a left-sided double lumen endotracheal tube was placed. Central venous access was obtained, including pre-emptive femoral arterial and venous access by cardiac surgeon. She was positioned laterally for left thoracotomy with left lung deflated. After 45 minutes of adhesiolysis, the left ventricle was exposed for the apicoaortic conduit. A partial clamp was applied to the descending thoracic aorta, and outflow graft was attached. Following systemic heparinization, cardiopulmonary bypass was established using an 8 mm graft on the outflow graft in the descending aorta and the femoral venous line. The left ventricular apex was cored and the apicoaortic conduit secured. Total bypass time was 34 minutes with no cross-clamp time. After weaning from bypass, the patient had good biventricular function with low gradients through both the AVB graft and in situ valve. Post-operative echocardiography showed no aortic valve stenosis (mean gradient 4 mmHg; Table 1). She was discharged on post-op day 12.

Discussion

AVB offers significant advantages for high-risk patients with aortic stenosis, particularly the elderly and patients with prior sternotomy or bypass grafts, as it reduces risk for disruption of coronary grafts, conduction system injury, perivalvular leak, and surgical bleeding from redo sternotomy (1). The surgeon is not limited by valve size, so the chance of a patient-prosthesis mismatch is eliminated. AVB also requires less cardiopulmonary bypass time and eliminates the need for aortic cross clamping, potentially improving neurologic outcomes. The AVB configuration may further reduce thromboembolic stroke risk compared to AVR. Cerebral circulation remains supplied through the in situ valve, while systemic flow occurs via the conduit, decreasing embolic potential (1). Computational models show that AVB does not compromise and may in fact improve cerebral perfusion; interestingly this improved output comes from the in situ valve rather

than retrograde flow from the conduit (2). In summary, AVB remains a safe and valuable options to patients deemed high risk for surgical AVR.

Images

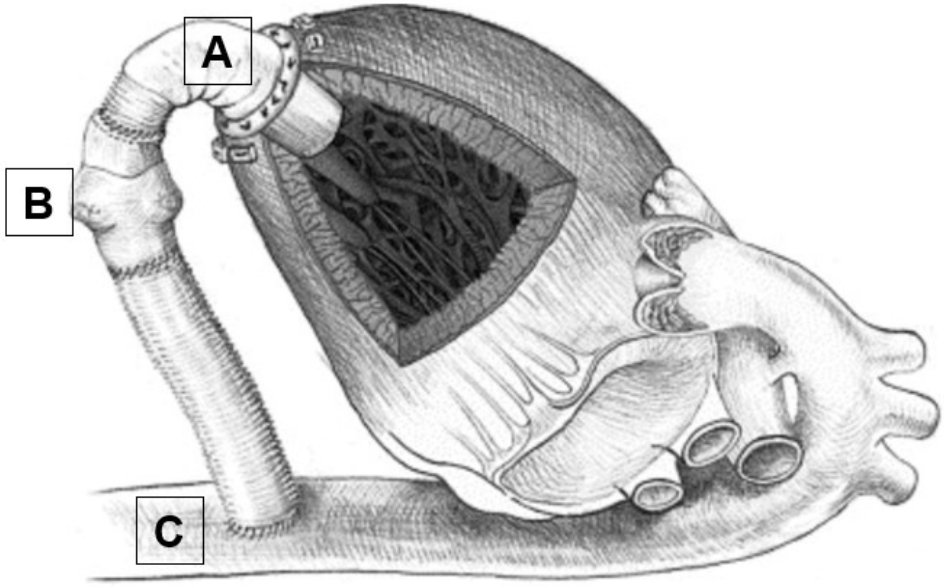


Figure 1A. Illustration of AVB (1): insertion of the conduit into the left ventricular apex (A), the bioprosthetic valve (B), and the insertion of the conduit into the descending thoracic aorta (C) (1).



Figure 1B. Postoperative image of computed tomography chest showing coronal slice showing AVB inserted into the apex of the left ventricle.

	Before Aortic Valve Bypass	After Aortic Valve Bypass
AV peak velocity	412 cm/s	134 cm/s
AV mean gradient	41 mmHg	4 mmHg
AV area	0.8 cm2	0.7 cm2
RVSP	45 mmHg	43 mmHg
TAPSE	1.7 cm	1.7 cm

Table 1. Various parameters collected via transthoracic echocardiography of the patient's in situ stenotic bioprosthetic aortic valve, before and after the patient's aortic valve bypass surgery. Acronyms: Aortic valve (AV), right ventricular systolic pressure (RVSP), tricuspid annular plane systolic excursion (TAPSE)

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#FR 3 – Kiosk 5 – Poster 1

Postoperative Cardiogenic Shock with Predominant Right Ventricular Failure Following Reinforced Ross Procedure: A Diagnostic and Hemodynamic Challenge

Presenting Author: Skylar Fuller, MD, Resident Physician, Oregon Health and Science University

Introduction

Cardiogenic shock following a Ross procedure is rare, particularly when left ventricular (LV) function is preserved and coronary angiography is normal. Technical modifications such as autograft reinforcement within a Dacron sleeve and the use of a synthetic right ventricle-to-pulmonary artery (RV-PA) conduit may create unique postoperative hemodynamic challenges. We present a case of multifactorial cardiogenic shock due to severe right ventricular (RV) failure after a reinforced Ross procedure.

Case Presentation

A 37-year-old female with a congenital bicuspid aortic valve and normal ventricular function underwent a reinforced Ross procedure using a pulmonary autograft within a 24-mm Dacron sleeve and a custom 24-mm polytetrafluoroethylene (PTFE) RV-PA valved conduit. Cardiopulmonary bypass time was 270 minutes (cross-clamp 221 minutes). At separation from bypass, she required maximal support with epinephrine, norepinephrine, milrinone, vasopressin, and inhaled nitric oxide in the setting of new inferior and inferolateral LV akinesis, moderate-severe RV dysfunction, and moderate tricuspid regurgitation, concerning for a right coronary artery (RCA) lesion. However, the RCA appeared patent by epicardial Doppler and on coronary angiography obtained immediately post-operatively. The patient continued to develop multifactorial cardiogenic shock with rising lactate and acute renal failure despite maximal inotropic support. Due to persistent RV failure, an Impella RP was placed before subsequently transitioning to VA-ECMO and later a Protek Duo. She also developed bacteremia requiring antibiotics. Over the following weeks, serial TEEs showed progressive recovery of RV function, allowing device removal and eventual discharge on home milrinone, later successfully weaned.

Discussion

The combination of inferior LV wall and persistent severe RV dysfunction with patent coronaries suggested multifactorial injury. Contributing mechanisms likely included pulmonary conduit dysfunction, as the stiff PTFE RV-PA graft increased RV afterload; reperfusion injury to the RV from prolonged cross-clamp and cardiopulmonary bypass; LV diastolic dysfunction due to the stiff Dacron-reinforced neo-aortic root causing

interventricular septal shift and impaired RV filling; and systemic inflammatory response from postoperative bacteremia, which may have depressed myocardial contractility. These factors likely acted in combination, producing mechanical, ischemic, and inflammatory insults that predominately affected the RV while preserving overall LV systolic function. In this case, early recognition of RV-specific dysfunction was critical to guide timely mechanical support and optimize recovery, highlighting the need for vigilant perioperative assessment of RV performance in complex autograft and RV-PA conduit reconstructions.

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#FR 3 – Kiosk 5 – Poster 2

Seeing Beyond the Scope: Iatrogenic Type A Aortic Dissection in Totally Endoscopic Cardiac Surgery

Presenting Author: Woong Y. Hwang, MD/PhD, Resident, Brigham and Women's Hospital

Introduction

Totally Endoscopic Cardiac Surgery (T-ECS) is an established alternative to conventional median sternotomy, offering reduced surgical trauma, faster recovery, and comparable long-term outcomes1. Despite these advantages, T-ECS is associated with a higher risk of iatrogenic acute aortic dissection (iAD) due to limited visibility and technical challenges inherent to T-ECS compared to conventional sternotomy (0.06% vs 0.75%)2. We present a successful management of type A iAD following T-ECS, emphasizing the importance of vigilant intraoperative monitoring for early diagnosis and preoperative risk assessment for improved outcome.

Case Presentation

A 68-year-old female with severe AI and moderate TR underwent endoscopic mechanical aortic valve replacement (AVR) and tricuspid valve repair (TVP). Cardiopulmonary bypass (CPB) was established via femoral cannulation under transesophageal echocardiography (TEE) guidance. Surgical access was established through a right peri-areolar incision (Fig 1). Mechanical AVR and TVP were performed through aortotomy and right atriotomy. Prior to CPB separation, TEE assessment revealed a Type A aortic dissection extending from the aortotomy site to the base of the innominate artery (Fig 2&3). After a multidisciplinary discussion, surgical access was converted to full median sternotomy for repair. CPB was maintained, deep hypothermic circulatory arrest (DHCA) was initiated, and additional right axillary artery cannulation provided antegrade cerebral perfusion. The dissected ascending aorta was resected and replaced with a dacron graft. Post-repair TEE showed resolution of the arch dissection with a small residual intramural hematoma. The patient was extubated on POD 1 and discharged home on POD 11.

Discussion

IAD is a rare but catastrophic complication of cardiac surgery, with a higher incidence in minimally invasive surgery compared with conventional sternotomy. Most cases arise from mechanical injury at sites of surgical manipulation and this case demonstrates the importance of early detection and rapid conversion to median sternotomy, which can result in favorable outcomes despite the high-risk nature of this complication. In our case, preoperative CT angiography demonstrated only mild ascending aortic dilation, without major high-risk features such as aneurysm, prior dissection, or severely diseased peripheral vasculature. Intraoperatively, TEE evaluation of the descending aorta following femoral cannulation showed no evidence of dissection, suggesting that CPB cannulation was not the precipitating cause. The dissection developed during repair of the root-vent aortotomy, highlighting that periods of direct aortic manipulation present the greatest risk for iatrogenic dissection. For the cardiac anesthesiologist, thorough preoperative risk assessment and routine intraoperative TEE surveillance until the end of the operation are essential for early recognition of iAD. Rapid conversion to full sternotomy and immediate aortic repair can significantly

improve outcomes, whereas delayed diagnosis or extension of the dissection beyond the aortic arch is associated with substantially higher mortality.

Images

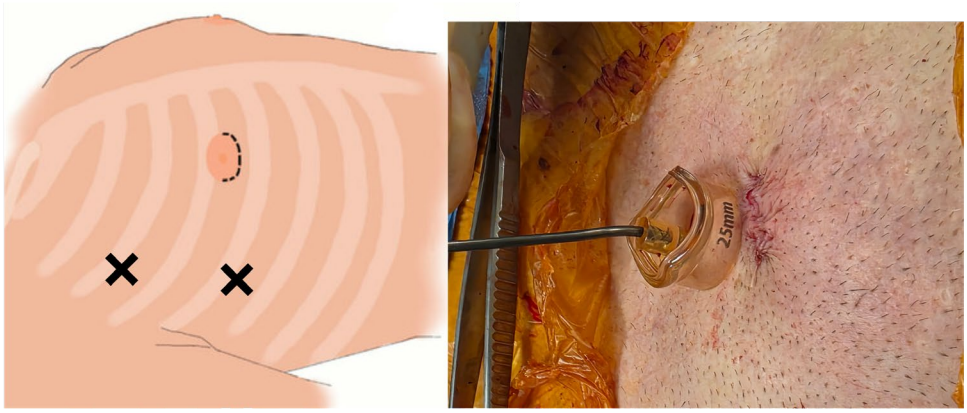


Figure 1. Left: Schematic illustration of the right peri-areolar incision approach. A small crescent-shaped incision is made around the right areola, with additional small port incisions (indicated by X) placed at the 2nd–4th intercostal spaces to allow instrument access and visualization[3]. Right: Picture of the aortic endoscopic incision.

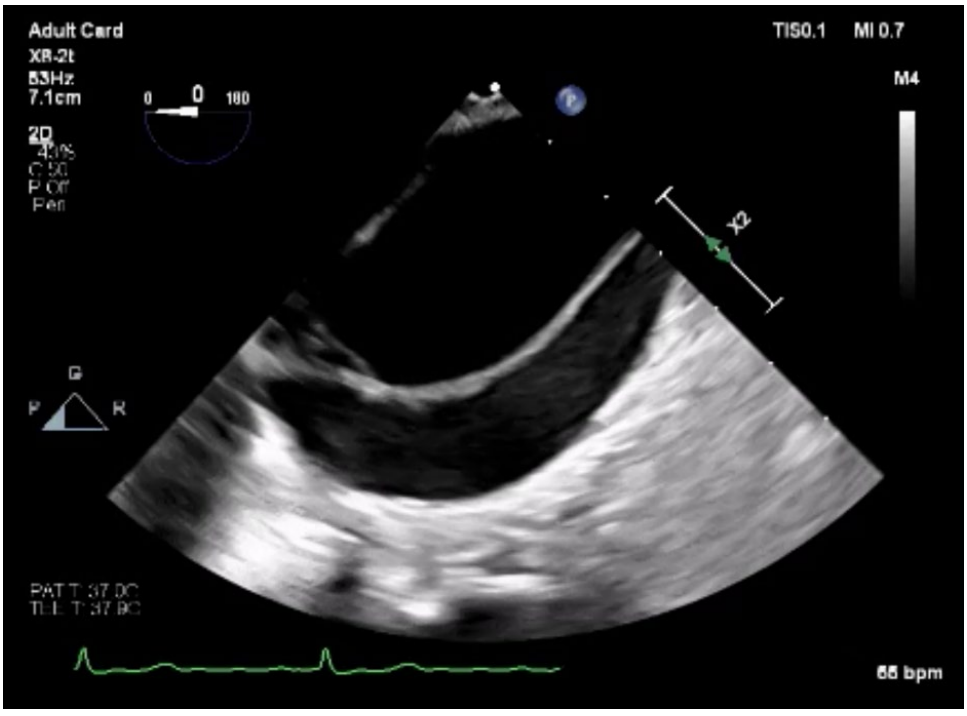


Figure 2. Intraoperative upper esophageal aortic arch long-axis TEE view demonstrating a Type A aortic dissection flap extending from the aortotomy site to the base of the innominate artery.

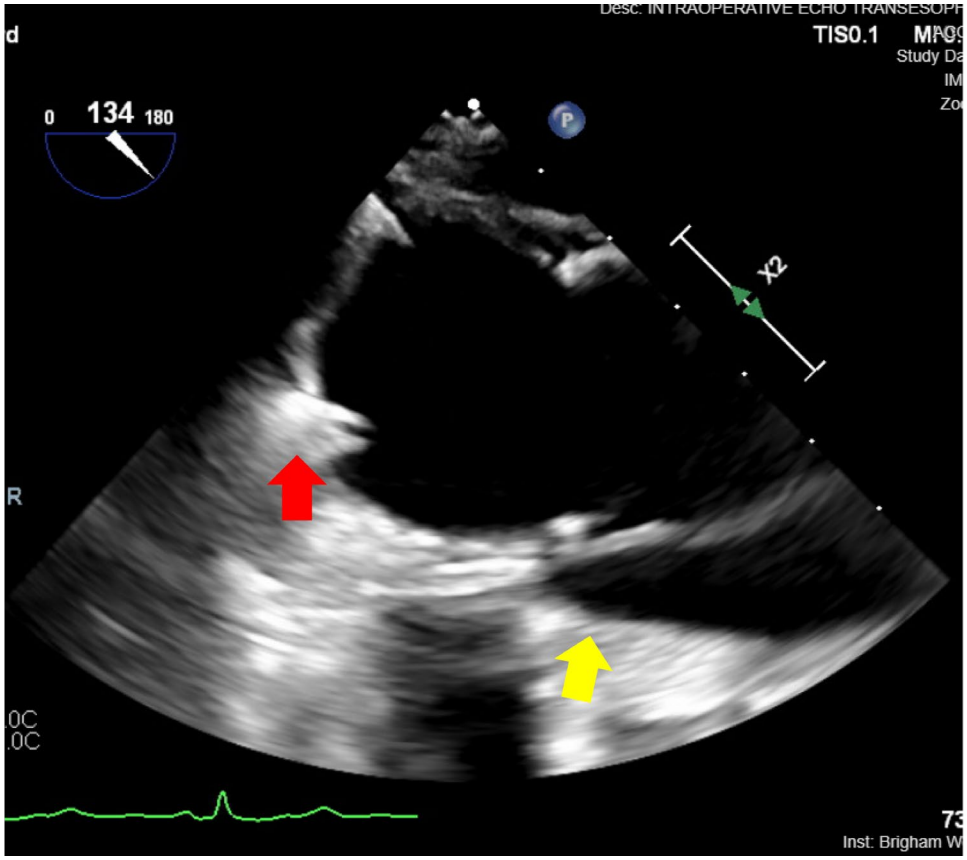


Figure 3. Modified mid-esophageal long axis TEE view demonstrating extension of the dissection from the aortotomy site (yellow arrow). Mechanical aortic valve denoted by red arrow.

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#FR 3 – Kiosk 5 – Poster 3

Fatal Type A Aortic Dissection in Alport Syndrome

Presenting Author: Alex Miller, Anesthesiology Resident, Loyola University Medical Center

Introduction

Alport Syndrome is a genetic disorder with defective type IV collagen, classically presenting with renal failure, hearing loss, and ocular abnormalities. Cardiovascular manifestations are increasingly recognized but under-appreciated, as this collagen deficiency affects the aortic media, predisposing patients to early-onset aortic disease1. We present a case of Type A aortic dissection in an adult with Alport syndrome, complicated by occult pulmonary disease, profound vasoplegia, and multi-organ failure.

Case Presentation

A 37-year-old male with Alport syndrome presented with progressive epigastric pain and severe hypertension. CTA revealed acute Type A aortic dissection with contained rupture limited to the aortic root, and diffuse ground-glass pulmonary opacities. Chart review revealed progressive aortic enlargement and chronic pulmonary hypertension. Upon initiation of cardiopulmonary bypass (CPB), profound vasoplegia occurred requiring high-dose vasopressors. Surgical exploration revealed extremely friable tissues and contained rupture with complete left main coronary ostium destruction, requiring pulmonary artery transection for graft reconstruction. Severe hypoxic respiratory failure with critically elevated airway pressures prevented weaning from CPB. Bronchoscopy cleared thick secretions without improvement. Pleural exploration revealed severely fibrotic, black lungs. VV-ECMO was initiated given adequate cardiac function by TEE and isolated respiratory pathology. Oxygenation improved, allowing separation from CPB. Post-bypass, acute liver failure developed as well as uncorrectable coagulopathy despite massive transfusion protocol. Ultimately, right ventricular failure led to cardiac arrest without return of spontaneous circulation.

Discussion

Defective type IV collagen in Alport syndrome disrupts the aortic media collagen network, predisposing to early thoracic aortic disease. This patient's aortic enlargement over four years demonstrates an aggressive natural history. Longitudinal imaging revealed missed opportunities for earlier intervention, underscoring the need for systematic cardiovascular screening in Alport patients. Beyond cardiovascular manifestations, this case highlights the importance of comprehensive pulmonary evaluation. Chronic ground-glass opacities and pulmonary hypertension were under-appreciated preoperatively as critical risk factors. Intraoperative discovery of severe fibrotic lung disease rendered CPB separation impossible without mechanical support. Interestingly, there are no recognized pulmonary manifestations of Alport syndrome - the severity encountered here may be related, or simply a confounding occult end-organ disease that contributed dramatically to morbidity. Intraoperatively, VV-ECMO was selected for isolated respiratory failure with preserved cardiac function, yet despite optimal interventions including high-dose vasopressors, mechanical support and massive transfusion protocol, cascading multi-organ failure (acute liver failure, uncorrectable

coagulopathy, and RV failure) proved fatal. This case demonstrates the extraordinary challenge of managing patients with advanced systemic collagen disorders.

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#FR 3 – Kiosk 5 – Poster 4

From Bridge to Breakdown: Challenges of ECMO Reconfiguration in a Transplant Candidate

Presenting Author: Dhanush Bathala, D.O., Resident Physician, Henry Ford Health

Introduction

Extracorporeal membrane oxygenation (ECMO) is an established bridge-to-transplant strategy for patients with interstitial lung disease (ILD), particularly when hypoxemia limits participation in pre-transplant rehabilitation. However, patients may deteriorate into right or left ventricular failure, requiring rapid ECMO reconfiguration. The choice of configuration, VV, VA, or hybrid RV-assist systems such as ProtekDuo, has major implications for survival, mobility, and transplant eligibility. This case highlights the complexity of escalating ECMO support in severe ILD and the impact of mechanical and anatomical complications on transplant candidacy.

Case Presentation

A 56-year-old man with recently diagnosed ILD presented with acute hypoxemic respiratory failure. Despite high-flow nasal cannula, he remained severely oxygen-dependent, prompting expedited lung-transplant evaluation. Progressive exertional desaturation and inability to maintain oxygenation led to VV-ECMO initiation via right femoral drainage and right internal jugular return as a bridge to transplant. He developed shock with acute RV then biventricular, failure. Given insufficient circulatory support on VV-ECMO, the team converted to peripheral VA-ECMO and reconfigured the previous circuit to a ProtekDuo cannula with oxygenator for RV support. Due to escalating instability, his transplant listing was changed to status 7. He developed recurrent hypotension and suction events secondary to kinking of the ProtekDuo at the RV apex. Despite emergent replacement, anatomical constraints caused repeated kinking. The ProtekDuo was removed, leaving the patient on VA-ECMO alone. Repeat echocardiography revealed severe biventricular dysfunction (LVEF 10–15%) and a large anterior pericardial echodensity concerning for hemopericardium, not amenable to drainage. Although VA-ECMO flows remained adequate, surgical intervention carried prohibitive risk, and he was no longer a transplant candidate. After multidisciplinary discussion and a family meeting, goals of care were transitioned to comfort, and ECMO and ventilation were withdrawn.

Discussion

This case illustrates the difficulty of managing cardiopulmonary collapse in ILD patients bridged with ECMO. VV-ECMO is preferred for isolated respiratory failure, but RV dysfunction, common in advanced ILD, often necessitates conversion to VA-ECMO or RVAD/veno-pulmonary support. The ProtekDuo offers minimally invasive RV unloading and allows mobility, which may improve transplant outcomes However, it is vulnerable to malposition, suction events, and kinking, particularly in patients with unfavorable RV anatomy. Survival in severe ILD without transplant is poor, but ECMO as bridge-to-transplant improves outcomes. Nevertheless, prolonged or unstable ECMO support increases waiting-list mortality. Ultimately, this case underscores the importance of selecting the optimal ECMO configuration, early recognition of device

complications, and multidisciplinary reassessment of transplant candidacy in patients with rapidly progressive ILD and evolving cardiopulmonary failure.

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#FR 3 – Kiosk 5 – Poster 5

Unexpected Challenges During Surgical VSD Repair

Presenting Author: Stephanie DeFord, MD, Cardiothoracic Anesthesia Fellow, Medical University of South Carolina

Introduction

Ventricular septal defect (VSD) is a known complication following acute myocardial infarction (MI). Presentation ranges from asymptomatic murmurs to circulatory collapse. Despite an overall operative mortality rate of 42.9%, surgical intervention is the treatment of choice as medical management alone has shown increased mortality rates at one year (46% and 83.3%, respectively) [1]. In patients who are hemodynamically stable, delayed surgery is preferred to optimize outcomes. The 2025 ACC/AHA guidelines for ACS management state that mortality rates are lower in patients where surgery was delayed by more than 7 days [3]. A 2012 study found that mortality rates for patients repaired within 7 days from MI were 54.1% compared to 18.4% for repairs delayed by 7 days. Delaying surgical intervention allows for scar formation in tissue surrounding the defect which results in a better anchor for sutures and decreased risk for patch dehiscence [2]. Unfortunately, not all patients remain hemodynamically stable, and surgery is indicated for patients in cardiogenic shock.

Case Presentation

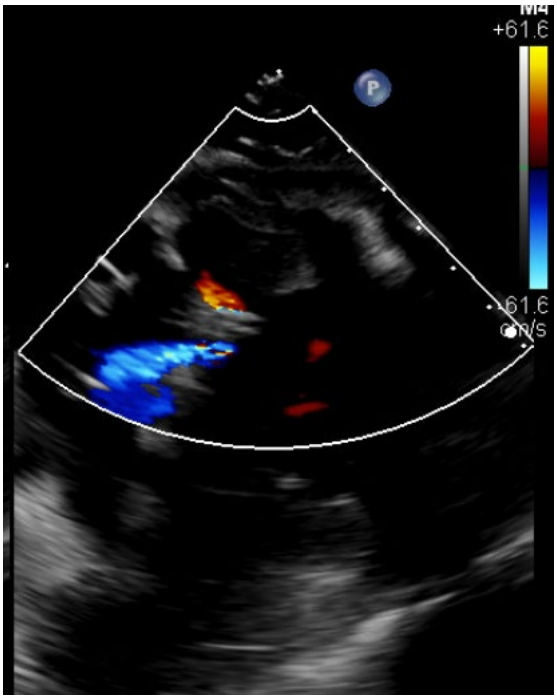
A 60-year-old man presented with inferior MI and received 3 stents to his RCA. The patient developed cardiogenic shock due to development of a VSD. The patient initially required an Impella 5.5 but continued to worsen with end organ dysfunction necessitating VA ECMO. Two weeks after his MI, the patient was brought to the OR for VSD repair. During surgical preparation, the arterial ECMO cannula was inadvertently dislodged. A vascular surgeon was called to repair the femoral artery while the cardiac surgery team proceeded with emergent sternotomy and aortic cannulation for initiation of CPB. During aortic cannulation, a large hole was inadvertently created on the posterior ascending aorta resulting in massive blood loss. The femoral artery was re-cannulated, and CPB was initiated. The aorta was repaired while on circulatory arrest without cerebral perfusion. Attention was then turned to the VSD which was uneventfully repaired. Attempts to wean from CPB were unsuccessful due to cardiogenic and hypovolemic shock secondary to blood loss and coagulopathy. The patient was transitioned to VA ECMO while undergoing massive transfusion. Surgical hemostasis and adequate resuscitation were eventually achieved, and the patient was taken to the ICU where he ultimately recovered and was discharged.

Discussion

Surgical VSD repair after MI should be delayed for at least 7 days if possible so that scar tissue can form and patients can stabilize. In this particular case, initial attempts were made to medically manage the VSD, but the patient continued to worsen while on mechanical support devices. Because the patient failed medical management, it was necessary that he undergo urgent surgical repair two weeks after his MI. Despite the complications the patient experienced, the actual VSD repair was uneventful due to the development of scar

tissue in this area. This case highlights the importance of delayed surgical VSD repair as well as the need for the anesthesia team to be adaptable and prepared for any complications that may arise. Because of the combined efforts of the anesthesia, surgery, and perfusion teams, this patient ultimately achieved a good outcome.

Images



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#FR 3 – Kiosk 5 – Poster 6

Hypoxemia Resulting from Iatrogenic Septal Puncture During Atrial Flutter Ablation

Presenting Author: Jacob Conroy, MD, Cardiovascular Anesthesia Fellow, Texas Heart Institute

Introduction

Atrial septal puncture for left atrial access during electrophysiology and structural heart procedures is increasingly common.[1] Iatrogenic atrial septal defects (iASD) created by these procedures rarely cause hemodynamically significant effects, but right heart failure (RHF), pulmonary hypertension (pHTN), and severe tricuspid regurgitation (TR) are all risk factors for right to left shunt development.[2] Left ventricular assist devices (LVAD) have the ability to promote myocardial recovery in select populations and leaving the decommissioned LVAD in-situ if this occurs has become an attractive option given the morbidity associated with an additional surgery to physically remove the device. There is a trend, however, with decommissioned LVADs toward higher rates of late RHF.[3]

Case Presentation

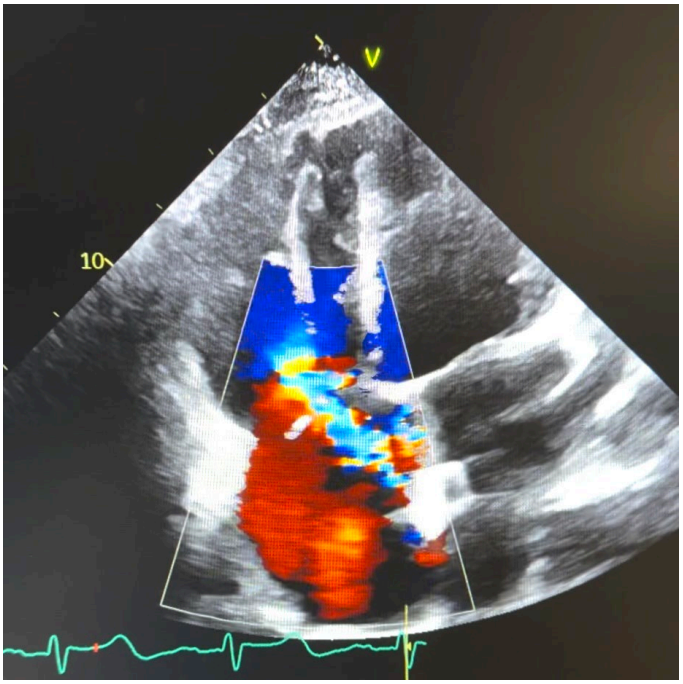
A 50-year-old female with a history of peripartum cardiomyopathy and atypical atrial flutter (AF) status post LVAD placement presented to the cath lab for bi-atrial cryoablation. Her LV function recovered and the LVAD was decommissioned years prior but she subsequently developed late RHF and severe TR leading to dyspnea and fluid retention. During her hospitalization, her heart rate maintained in the 120s to 130s in atypical flutter and she developed an AKI. On exam, she was tachycardic, short of breath, and had bilateral lower extremity edema. Her preop TTE showed LV EF of 35%, moderately to severely enlarged RV with severely reduced function, moderate MR, and severe TR. General anesthesia was induced with carefully titrated propofol, midazolam, and succinylcholine and maintained with sevoflurane. Systemic pressures maintained near baseline throughout the procedure. The left and right common femoral veins were cannulated and a transeptal puncture was performed, leaving a 15 Fr sheath in the interatrial septum. LAP was 36 mmHg and RAP was 40 mmHg. After multiple ablations were performed, the catheter and sheath were retracted. Quickly following, the patient desaturated on 100% FiO2 from 99% to 85%. After discussing possible causes with the EP team and considering the elevated RAP could lead to right to left shunt through the iASD, the sheath was guided back into the interatrial septum with resolution of the hypoxemia. Intracardiac echo (ICE) was performed and color doppler demonstrated the severe TR jet eccentrically directed at the iASD with significant right to left shunt. Given the elevated RAP, we had already attempted to

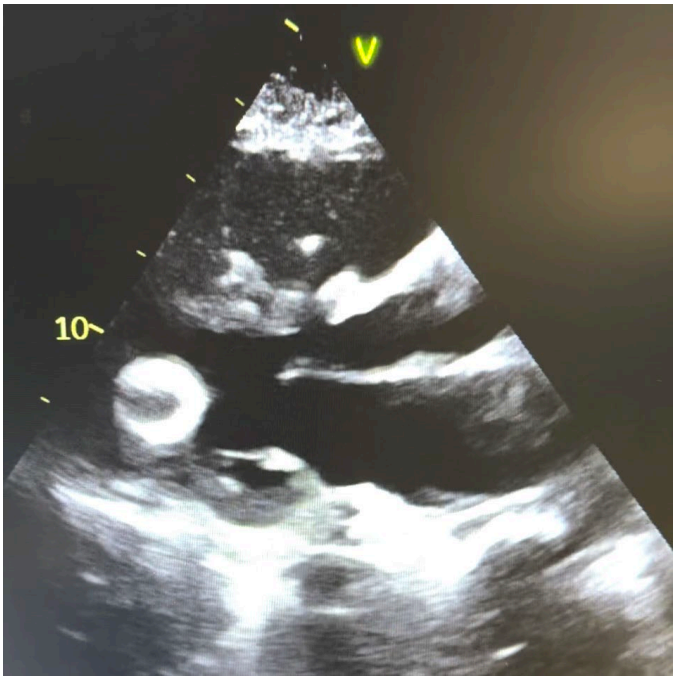
augment diuresis with two doses of furosemide to little effect. With medical therapy unlikely to improve the hypoxemia, interventional cardiology was consulted and they recommended occlusion of the ASD with a 30mm Amplatzer device. After device deployment, the SpO2 maintained in the high 90s even on FiO2 of 40%. We transported to the ICU for further care still intubated. Postoperative course was complicated by renal failure requiring dialysis and the patient was extubated on POD4 after volume optimization.

Discussion

Patients with decommissioned LVADs can present with a wide range of complications. We present a unique case of late onset RHF and AF requiring ablation in the cath lab. While iASD after septal puncture is not rare, right to left shunts causing hypoxemia are rare but should be considered on the differential diagnosis in appropriate situations.

Images





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#FR 3 – Kiosk 5 – Poster 7

Noncardiogenic Pulmonary Edema Requiring Venous-Pulmonary Arterial Extracorporeal Membrane Oxygenation

Presenting Author: Sabrina M. Karwowski, DO, Anesthesia Resident, Medical College of Wisconsin

Introduction

Noncardiogenic pulmonary edema after cardiopulmonary bypass (CPB) is a rare but life-threatening complication characterized by increased pulmonary capillary permeability1. Potential causes include transfusion-related acute lung injury (TRALI), protamine reaction, surgical stress, sepsis, and the inflammatory response to bypass2. Early recognition and distinction from cardiogenic pulmonary edema are essential. Management involves aggressive airway clearance with fluid replacement, appropriate mechanical ventilation, pulmonary vasodilators, and ECMO for refractory cases1.

Case Presentation

A 66-year-old man presented with bacteremia and severe valvulopathy from infective endocarditis. TTE showed normal biventricular function, an 18×9 mm aortic valve vegetation with severe insufficiency, a perforated anterior mitral leaflet with moderate regurgitation, and markedly elevated systolic pulmonary artery pressure (PAP, 81mmHg). Bioprosthetic replacement of aortic and mitral valves was planned. On CPB, the patient developed profound vasoplegia requiring high-dose vasopressors including vasopressin, norepinephrine, methylene blue, and hydroxocobalamin. During separation from CPB, PAPs rose to 60s/30s and inhaled nitric oxide (NO) improved PAPs to 50/20s; however, vasoplegia persisted. Significant post-bypass bleeding required 4 PRBC, 2 plasma, 2 cryoprecipitate, and 2 platelets. Following transfusion, PAP again increased and a liter of pulmonary edema fluid was suctioned from the endotracheal tube. Furosemide was given with improved oxygenation. After chest closure, the patient developed near-systemic PAP, severe hypotension, and hypoxia. TEE demonstrated severe RV dysfunction and preserved LV function. Suspecting TRALI with secondary RV failure, a Protek DuoTM (LivaNova) cannula was placed for V-PA ECMO with RV support, resulting in RV decompression and inappropriately normal LV function on high-dose inotropes. In the ICU, anticoagulation was withheld due to bleeding and fluid resuscitation was required for pulmonary losses, both resulting in low ECMO flows. Eight hours postoperatively, the patient decompensated, presumed due to Protek thrombosis, and returned to the OR for VA ECMO conversion where TEE revealed extensive intracardiac thrombus. Given this, the patient was transitioned to comfort measures.

Discussion

The etiology of this patient's decompensation and noncardiogenic pulmonary edema remains unclear. TRALI remains the leading diagnosis due to the timing of blood product administration and absence of left-sided heart failure on TEE. An alternative diagnosis was increased pulmonary vascular resistance due to methylene blue use and resultant reduced endogenous NO availability, although inhaled NO was used concurrently to mitigate this effect. The decision was made to initiate RV support with V-PA ECMO rather

than central VA-ECMO due to concern that the latter would produce lower flow states across the newly-implanted valves, increasing the risk for thrombosis; unfortunately, volume loss from the pulmonary edema led to persistently low ECMO flows that resulted in thrombosis. Despite aggressive medical management, the patient ultimately succumbed to complications from noncardiogenic pulmonary edema.

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#FR 3 – Kiosk 6 – Poster 1

Iatrogenic Mitral-Aorta Fibrosa Perforation Post-Prosthetic Valve Surgery

Presenting Author: Qasim Al Hassan, M.B.B.S, Adult Cardiothoracic Anesthesiology Fellow, Westchester Medical Center

Introduction

Mitral–aortic trigon perforation is a rare but serious complication of infective endocarditis or cardiac surgery, most often associated with mitral or aortic valve procedures. The trigon, or intervalvular fibrous body, is susceptible to injury because of its avascularity and its close continuity with the anterior mitral leaflet and the non-coronary and left coronary cusps of the aortic valve. Aortic valve endocarditis, particularly in prosthetic valves, is the most frequent cause of left ventricular outflow tract (LVOT)–left atrial (LA) communication (1). Although pseudoaneurysm of the mitral–aortic fibrosa (PMAF) is less common, it may lead to life-threatening complications including fistula formation, coronary artery compression, and pericardial rupture with tamponade (2). Echocardiographically, LVOT–LA communication typically appears as an eccentric systolic jet that may mimic anterior mitral leaflet perforation. We report a case of suspected iatrogenic rupture of the mitral–aortic fibrosa presenting four years after mechanical mitral and aortic valve replacement.

Case Presentation

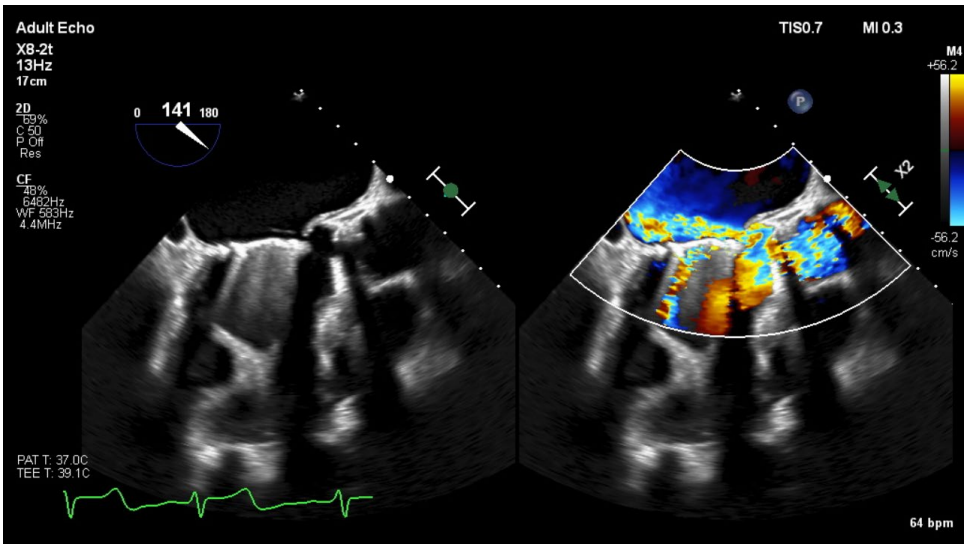
A 58-year-old man with chronic atrial fibrillation on anticoagulation, hypertension, type 2 diabetes, and rheumatic heart disease presented with progressive exertional dyspnea over three months. He had undergone redo mechanical mitral and aortic valve replacement four years earlier. He denied chest pain, orthopnea, syncope, fever, chills, or other systemic symptoms, and his international normalized ratio (INR) was therapeutic. Transthoracic echocardiography (TTE) demonstrated normal biventricular systolic function and well-seated prosthetic valves without stenosis or paravalvular leak, except for leaflet thickening. Because his symptoms remained unexplained, empiric broad-spectrum antibiotics were started for presumed endocarditis, and transesophageal echocardiography (TEE) was performed. TEE showed perforation of the mitral–aortic fibrosa with a systolic LVOT-to-LA jet, severe LA dilation, and possible mild aortic prosthesis dehiscence, without vegetation or left atrial appendage thrombus. Intraoperative TEE demonstrated two septate subaortic defects with eccentric systolic jets showing a Coandă effect and no evidence of abscess, fistula, vegetation, or dehiscence; surgical inspection confirmed these findings. The patient underwent redo aortic valve replacement, aortic root enlargement, and reconstruction of the mitral–aortic fibrosa via a hemi-Commando approach using a Cardiocel patch, followed by aortic reconstruction with a Hemashield graft. Blood and tissue cultures remained negative, leading to discontinuation of antibiotics postoperatively. His postoperative course was uneventful, and he was discharged on day seven.

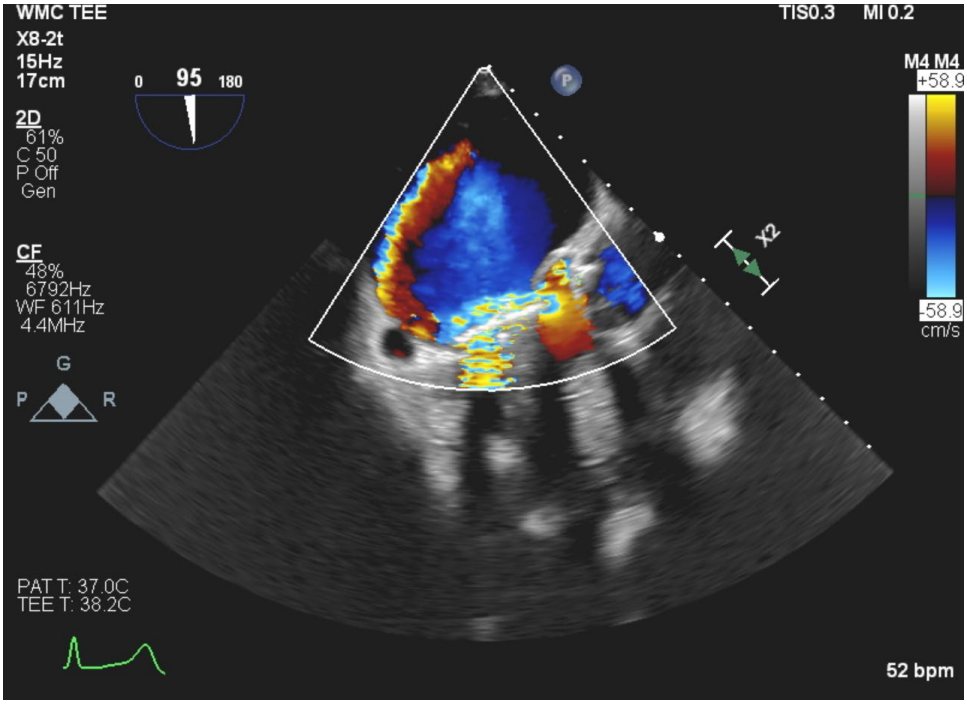
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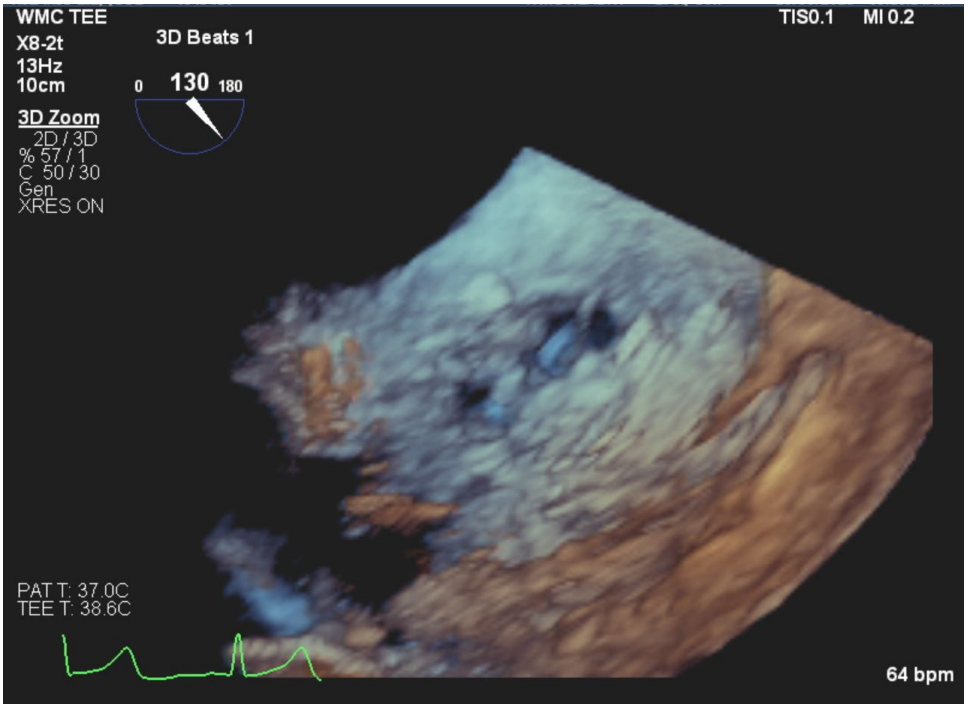
This case underscores the limitations of TTE in evaluating intervalvular fibrous body pathology in the setting of mechanical valves. TEE provides superior visualization of periannular extension, vegetation, abscesses,

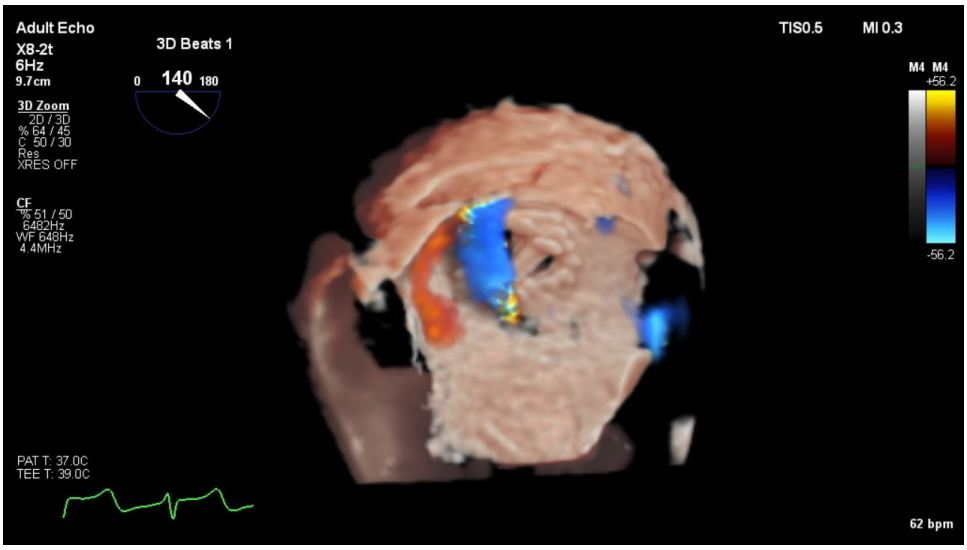
and structural disruption, making it essential for diagnosing mitral–aortic fibrosa perforation (1,3). The concordance between TEE and surgical findings in this case highlights its crucial role in guiding timely intervention.

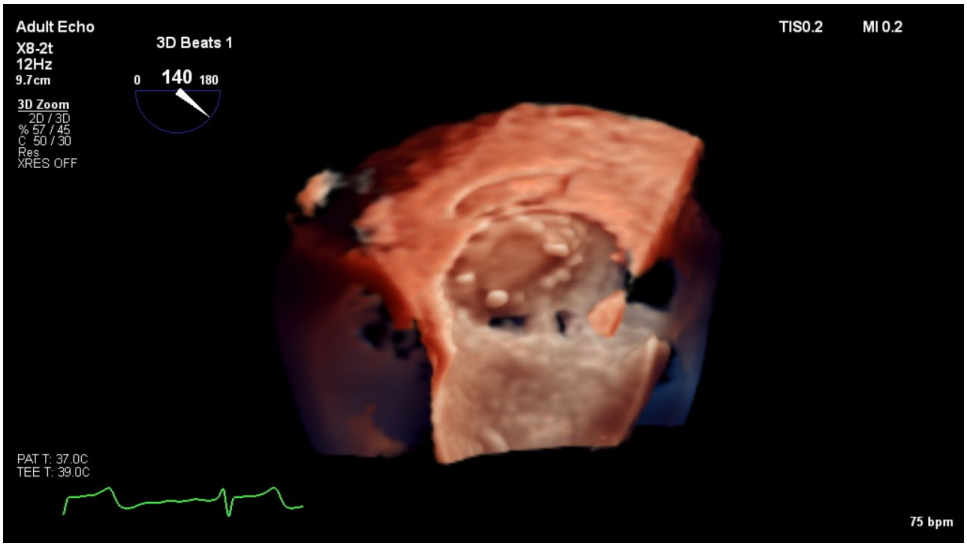
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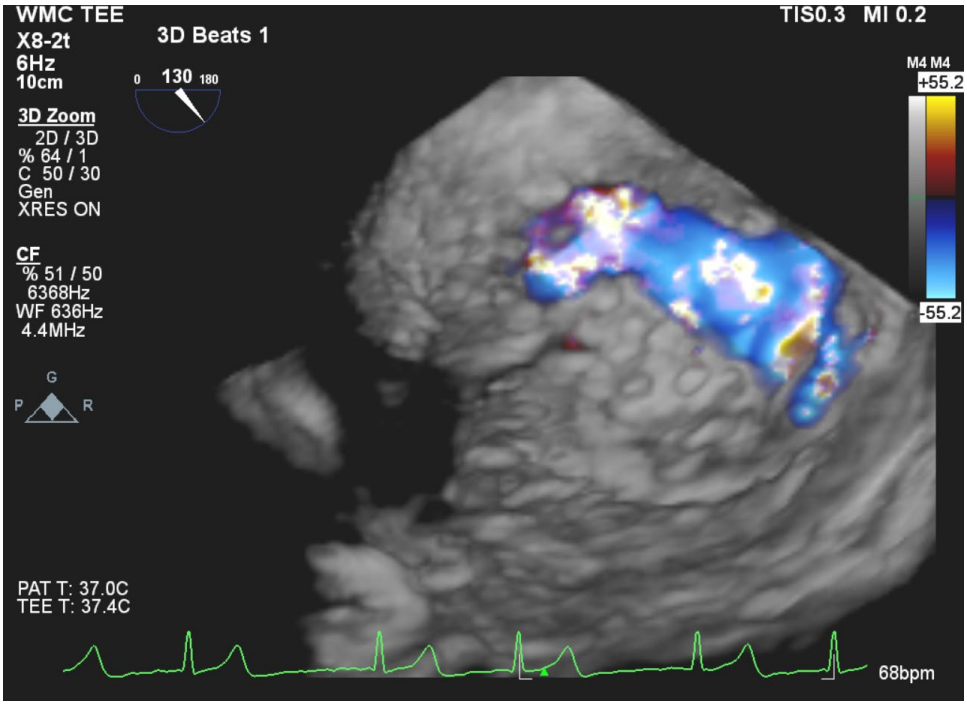





WMC TEE
X8-2t
13Hz
10cm
3D Zoom
2D / 3D
% 57 / 1
C 50 / 30
Gen
XRES ON
3D Beats 1
0 130 180
TIS0.1 MI 0.2
PAT T: 37.0C
TEE T: 38.6C
64 bpm


Adult Echo
X8-2t
6Hz
9.7cm
3D Zoom
2D / 3D
% 64 / 45
C 50 / 30
Res
XRES OFF
CF
% 51 / 50
6482Hz
WF 649Hz
4.4MHz
3D Beats 1
0 140 180
TIS0.5 MI 0.3
M4 M4
+56.2
-56.2
PAT T: 37.0C
TEE T: 39.0C
62 bpm





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#FR 3 – Kiosk 6 – Poster 2

Peripartum Sepsis Revealing Undiagnosed Rheumatic Valve Disease: Anesthetic Challenges and Perioperative Decision-Making

Presenting Author: Sadi Abukhalaf, MD, Resident, Advocate Illinois Masonic Medical Center

Introduction

Cardiovascular disease remains a leading cause of maternal morbidity and mortality (MMM), and Rheumatic Heart Disease (RHD) is an important consideration in patients from underdeveloped regions, where its prevalence is highest among women of childbearing age1. Physiological adaptations of pregnancy and acute hemodynamic shifts in the peripartum can unmask previously undiagnosed pathologies. We report a case of an immigrant mother who developed refractory shock after an otherwise uncomplicated cesarean delivery, ultimately revealing severe rheumatic mitral valve disease. This case highlights the need for broad differential diagnosis and awareness of epidemiologic risk factors when investigating cardiovascular postpartum complications.

Case Presentation

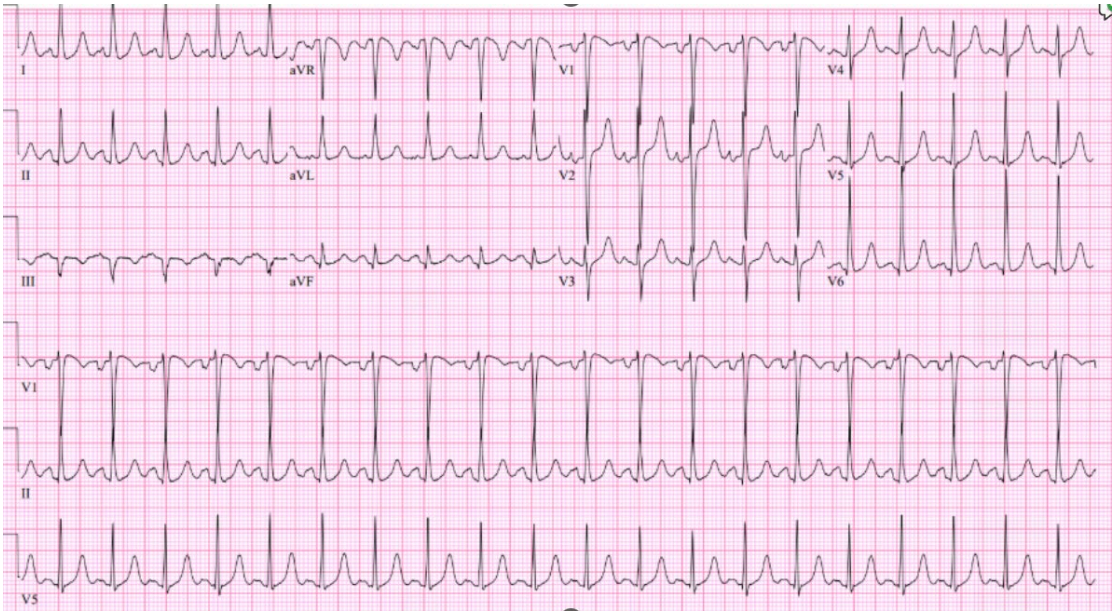
A 34-year-old Ethiopian primipara with no medical history presented at 31 weeks with PPROM and chorioamnionitis. As she progressed into sepsis with tachycardia and the fetus remained breech, an urgent cesarean delivery was performed. Spinal anesthesia was administered. Her intraoperative course was uncomplicated. She was hemodynamically stable, had a 500mL blood loss, and received 1400mL of crystalloids. Postoperatively, she developed chest pain, dyspnea and sinus tachycardia refractory to fluids, requiring vasopressors and high-flow oxygen. CT angiography showed pulmonary edema and bilateral pneumonia. TTE revealed normal biventricular function, a severely dilated left atrium, severe multijet mitral regurgitation (MR) with reduced leaflet mobility, consistent with rheumatic mitral valve disease. Findings were consistent with moderate mitral stenosis as well. She improved with antibiotics and diuresis and was discharged from the ICU on post-operative day 4.

Discussion

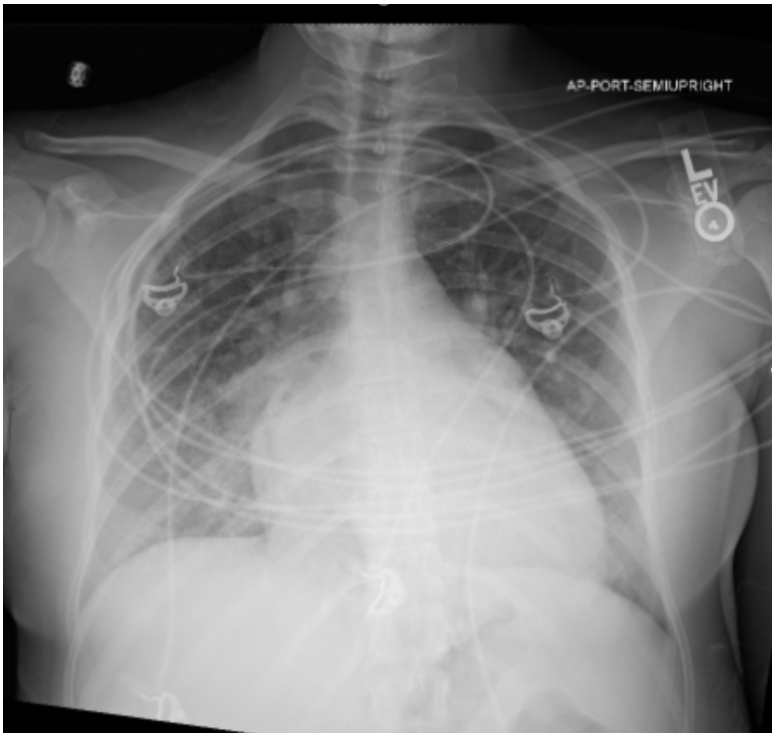
Cardiac etiologies should be considered early in women with post-partum hemodynamic compromise. In this case, bedside auscultation and POCUS might have expedited recognition of MR and pulmonary edema, prompting earlier initiation of diuresis. Hemodynamic changes in pregnancy and peripartum can precipitate

or reveal prior cardiac disease. MR can worsen, as intravascular volume can increase up to 45% 2, increasing the mitral annular diameter and worsening regurgitation. This is especially concerning after placental delivery and auto-transfusion of utero-placental blood. During this period, the tachycardia from the Bainbridge reflex can modestly alleviate MR, while bradycardia from the Bezold-Jarisch reflex after spinal anesthesia might worsen it. Mitral stenosis (MS) is also a frequent presentation of RHD. Pregnancy tends to increase the heart rate up to 25%, which may decrease LV preload and worsen symptoms. Utero-placental autotransfusion, along with expected tachycardia, can acutely decompensate MS and precipitate pulmonary edema. While routine advanced imaging is not required for all immigrant or high-risk patients, a careful cardiovascular physical exam can identify those in need of further evaluation. Patients with pre-existing heart disease carry a higher risk of MMM and should be thoroughly evaluated pre-operatively with multidisciplinary planning with cardiology and cardiothoracic anesthesiology. Selected cases might warrant cesarean delivery rather than vaginal and may benefit from arterial and central line placement pre-operatively.

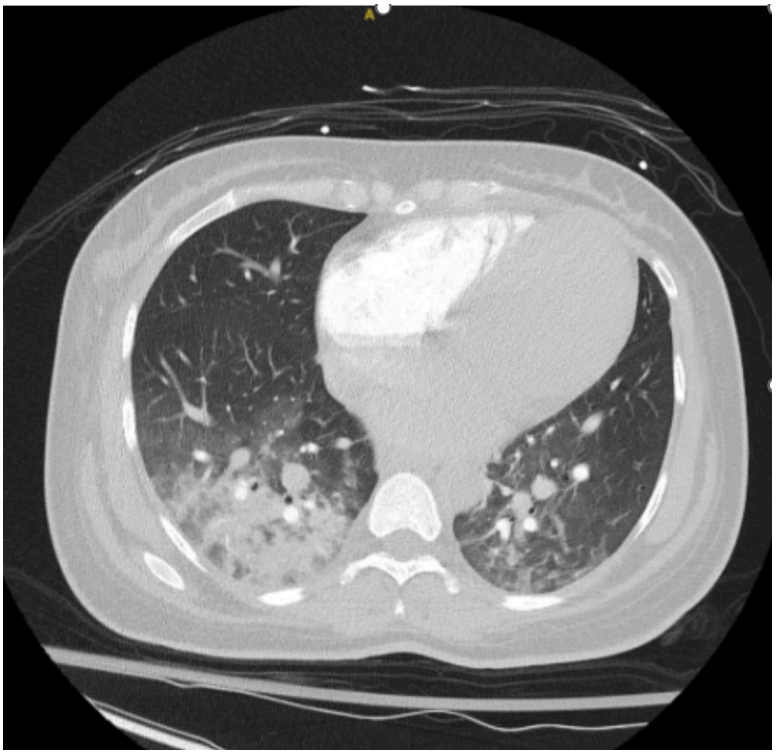
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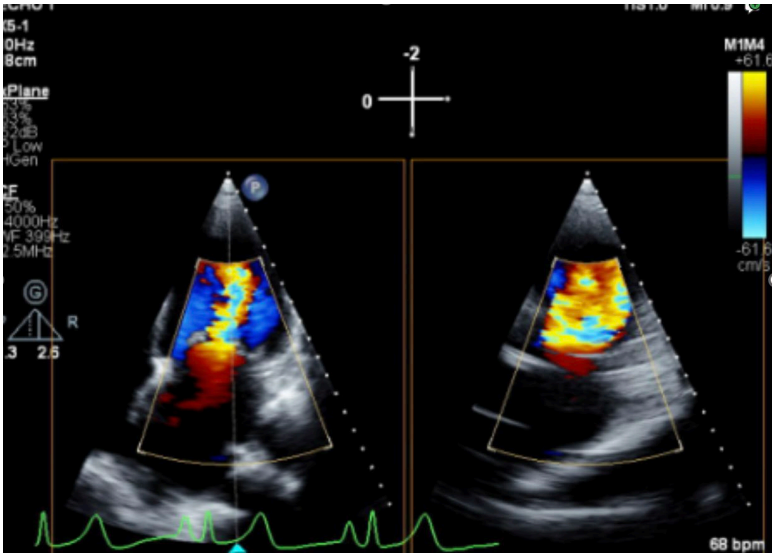
EKG Demonstrating Sinus Tachycardia With Possible Left Atrial Enlargement The 12-lead EKG shows sinus tachycardia, consistent with the patient’s acute hemodynamic stress during the peripartum period. Subtle P-wave abnormalities suggest possible left atrial enlargement, which correlates with echocardiographic findings of a severely dilated left atrium in the setting of rheumatic mitral valve disease.



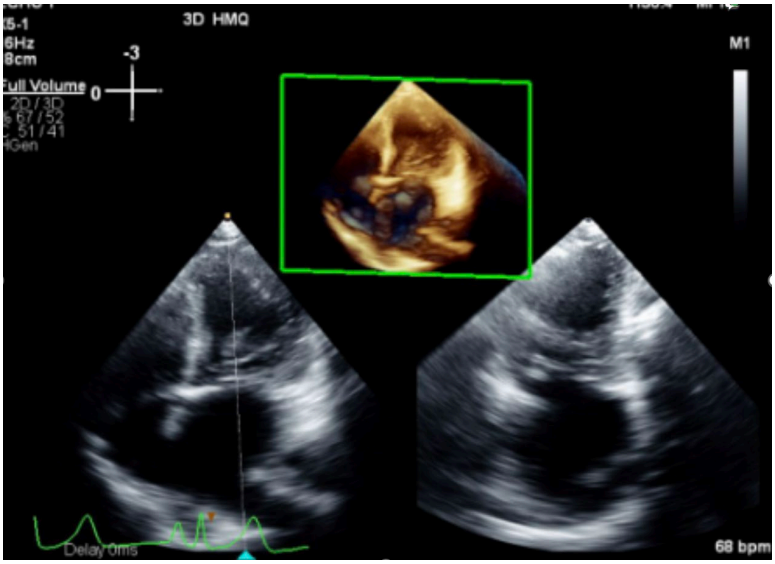
Chest X-ray Demonstrating Pulmonary Vascular Congestion and Pulmonary Edema The chest radiograph shows prominent pulmonary vascular markings with a diffuse interstitial and alveolar edema pattern, consistent with pulmonary congestion. These findings support the clinical picture of acute volume overload and cardiogenic contribution to postpartum shock



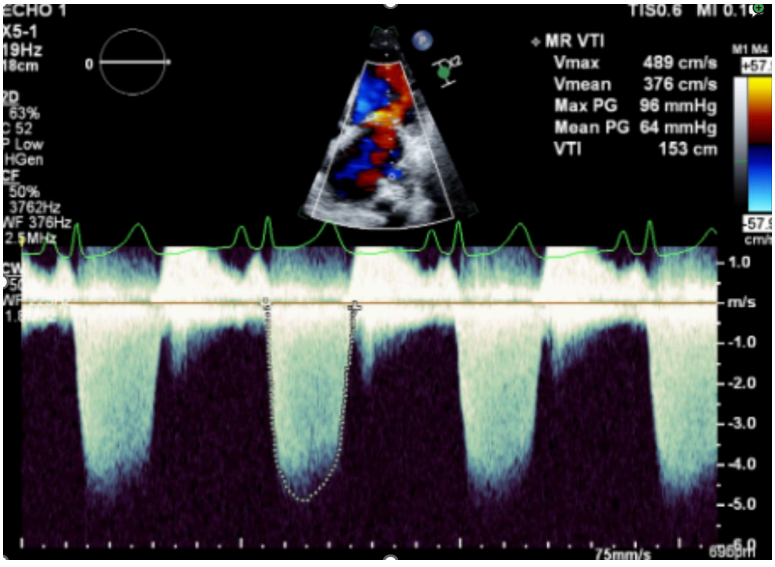
CT Pulmonary Angiography Showing No Pulmonary Embolism but Significant Pulmonary Edema and Bilateral Pneumonia The CT pulmonary angiogram demonstrates no evidence of pulmonary embolism. Instead, there is diffuse interstitial and alveolar pulmonary edema, along with bilateral lower-lobe airspace opacities consistent with multifocal pneumonia. These findings align with the patient's postpartum deterioration, supporting a combined picture of sepsis, volume overload, and severe underlying valvular disease.



Color Doppler Demonstrating Severe Mitral Regurgitation with Multiple Eccentric Jets Dual-plane transthoracic views show large, turbulent mitral regurgitant jets extending into the left atrium. The jets are eccentric and broad-based, consistent with severe MR. The appearance correlates with restricted mitral leaflet mobility and severe left atrial dilation, supporting a rheumatic etiology and explaining the patient's pulmonary edema and hemodynamic instability.



3D Echocardiography Showing Restricted Mitral Leaflet Motion Consistent With Rheumatic Pathology The 3D en face view of the mitral valve demonstrates restricted leaflet mobility and reduced commissural opening, characteristic of rheumatic involvement.



Continuous-Wave Doppler Across the Mitral Valve Demonstrating Severe Mitral Regurgitation The CW Doppler tracing shows a very high-velocity mitral regurgitant jet with a Vmax of 4.89 m/s and a maximum

pressure gradient of 96 mmHg, consistent with severe mitral regurgitation. The color Doppler inset demonstrates a large, turbulent MR jet. These findings correlate with restricted mitral leaflet motion and structural changes consistent with rheumatic heart disease

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#FR 3 – Kiosk 6 – Poster 3

Follow the Mitral Brick Road

Presenting Author: Sophie A. Leon, MD, Resident physician, Tufts Medical Center, Department of Anesthesiology and Perioperative Medicine

Introduction

Cardiac anesthesiologists play a pivotal role in transcatheter structural heart interventions through expert perioperative echocardiography, providing real-time risk assessment, procedural guidance, and complication management. Although alcohol septal ablation (ASA) is increasingly used to mitigate left ventricular outflow tract (LVOT) obstruction risk during valve-in-mitral annular calcification (ViMAC) procedures, unintended myocardial injury beyond the basal septum rarely occurs and can precipitate severe right-sided structural complications. This case underscores the cardiac anesthesiologist's critical role in identifying and differentiating mechanisms of tricuspid regurgitation (TR), tailoring transcatheter edge-to-edge repair (TEER) strategies, and guiding optimal management to facilitate successful ViMAC without unnecessary adjunctive procedures.

Case Presentation

An elderly female with worsening dyspnea was found to have severe mitral stenosis and regurgitation, extensive mitral annular calcification, basal septal hypertrophy, and a small LVOT. Pre-procedural modeling (3mensio software, Pie Medical Imaging, Utrecht, Netherlands) predicted high risk of LVOT obstruction following ViMAC with predicted valve-septal interaction. ASA was performed to enlarge the LVOT. Post-ablation, the patient developed acute cardiogenic shock. Transesophageal echocardiography by the cardiac anesthesiologist revealed a new flail anterior tricuspid leaflet due to rupture of the right ventricular anterior papillary muscle—distinct from the baseline CIED mediated moderate-to-severe TR. Tricuspid TEER with TriClip (Abbott Medical, Abbott Park, IL) was attempted but aborted due to excess interaction between the device frame and dense, flail chordal networks. Repeat attempt with PASCAL (Edwards Lifescience, Irvine, CA) was successful in eliminating the flail and restoring TR to baseline severity. Hemodynamics rapidly improved, with SvO_2 rising from 33% to 70%. Repeat CT and TEE demonstrated greater-than-anticipated LVOT enlargement, allowing for successful ViMAC without need for LAMPOON.

Discussion

This case demonstrates the indispensable role of cardiac anesthesiologists in structural heart interventions, particularly in complex anatomic and hemodynamic scenarios. ASA-induced myocardial infarction beyond the intended basal septum is rare, and extension into the right ventricular papillary muscle causing acute flail tricuspid leaflet is uncommon (1-3). Timely echocardiographic evaluation allowed differentiation between chronic CIED mediated TR and acute flail anterior leaflet pathology, directly influencing device selection and procedural strategy. By recognizing this complication and tailoring the TEER approach, the cardiac anesthesiologist ensured leaflet-capture feasibility and enabled hemodynamic recovery. Furthermore, precise imaging guidance prevented unnecessary LAMPOON and supported successful ViMAC

while mitigating LVOT obstruction risk. This case highlights the cardiac anesthesiologist's unique ability to integrate imaging, physiology, and procedural planning as part of the heart team, which optimizes outcomes in high-risk structural heart interventions.

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#FR 3 – Kiosk 6 – Poster 4

Down to the Wire: Percutaneous Paravalvular Leak Closure After Commando Procedure

Presenting Author: Emily Chung, DO, ACTA fellow, Montefiore Medical Center

Introduction

Paravalvular leaks (PVLs) are challenging complications of a prosthetic valve replacement. Surgical repair remains the gold standard but carries high operative risk and recurrence rates, prompting the development of percutaneous techniques. We present a case of urgent percutaneous mitral PVL closure performed 13 weeks after the Commando procedure.

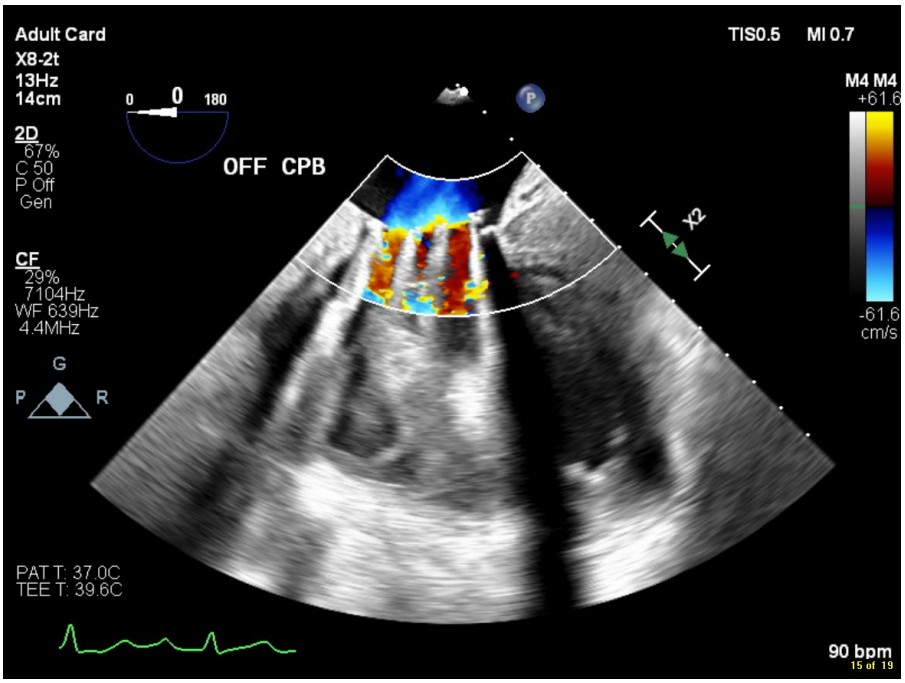
Case Presentation

A 58-year-old woman with a recent dental extraction presented to the hospital for chest pain and dyspnea. Transesophageal echocardiogram (TEE) revealed an aortic valve vegetation, severe aortic regurgitation, severe mitral regurgitation secondary to perforation of the A2 leaflet, and an ascending aortic aneurysm. She underwent urgent Commando procedure with mechanical aortic and mitral valve and hemiarch replacement for infective endocarditis. Intraoperative TEE and predischarge transthoracic echocardiogram (TTE) showed well-seated prostheses without PVL. Patient was discharged on POD 14 on warfarin and IV antibiotics. Two weeks later, she returned with chest pain. TTE showed mild mitral valve PVL. This finding prompted a brief admission for supratherapeutic INR and cardiology follow-up. Four weeks later, she was re-admitted for chest pain and new-onset atrial flutter. TEE demonstrated posteromedial partial valve dehiscence with severe PVL. Given the prohibitive redo surgical risk, the structural heart team was consulted and recommended deferred outpatient closure to minimize dehiscence progression. She returned 5 days later with worsening symptoms, prompting urgent in-hospital intervention. The procedure was performed in the cardiac catheterization lab under general anesthesia with invasive monitoring and TEE/fluoroscopic guidance. Heparin was titrated to ACT >300s before transseptal puncture. Three Amplatzer Vascular Plug II devices (12mm, 12mm, and 8mm) were sequentially deployed in the defect. Final TEE showed trace residual PVL. She was extubated after the intervention and later discharged on POD 17 without further complications.

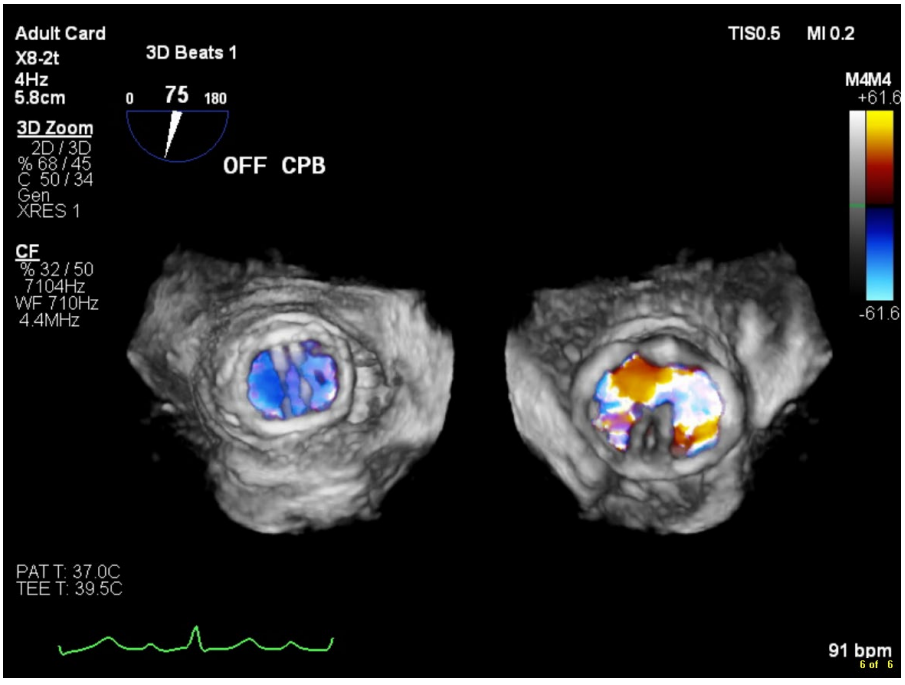
Discussion

This case highlights several anesthetic challenges to percutaneous PVL closure after a Commando procedure, including high-risk circulatory collapse during device manipulation. Perioperative decision-making is centered on the timing of the procedure and preparedness for rescue mechanical support and emergent surgical repair. The resolution of heart failure symptoms after successful closure highlights the efficacy of percutaneous techniques in this high-risk population and emphasizes the importance of vigilant follow-up echocardiography in endocarditis patients.

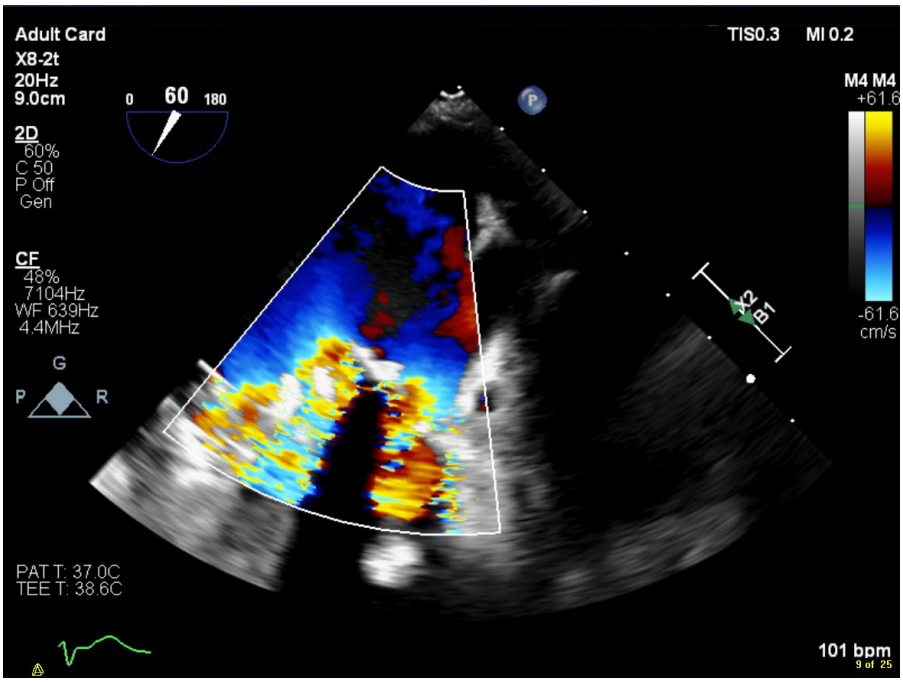
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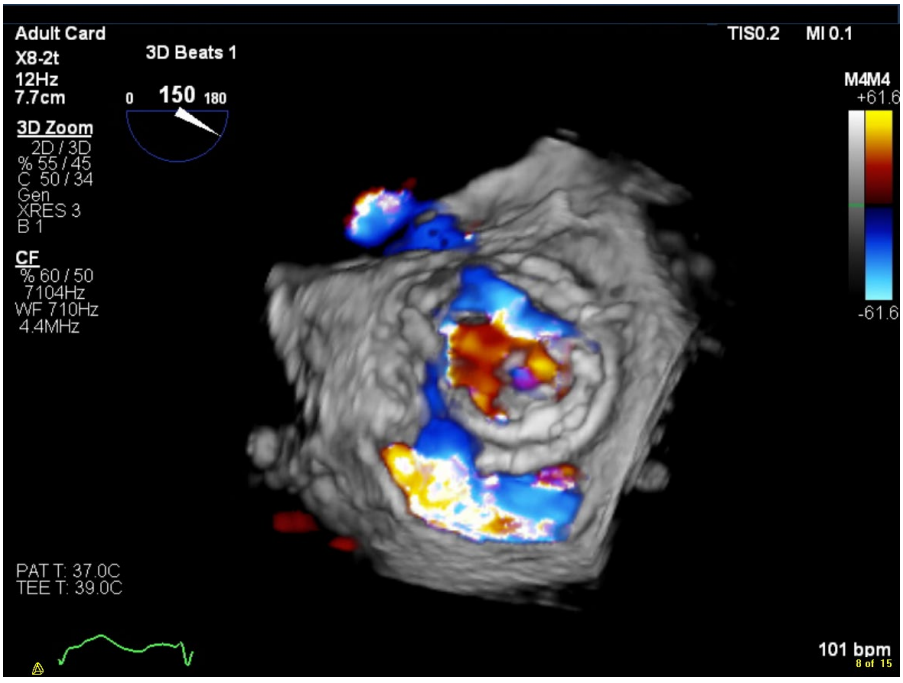
Intraoperative TEE image of the mitral valve after it was deployed. The mechanical valve appears to be well seated with no paravalvular leaks.



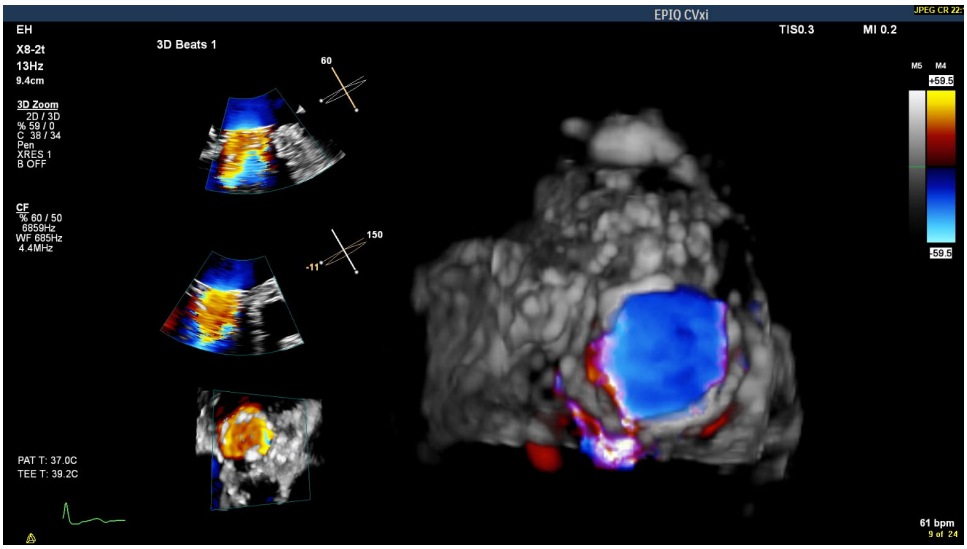
Intraoperative TEE image of the mitral valve after it was deployed. The mechanical valve appears to be well seated with no paravalvular leaks.



TEE performed by the cardiologist 10 weeks postoperatively. The official report reads partial dehiscence along the posteromedial aspect; severe paravalvular leak.



TEE performed by the cardiologist 10 weeks postoperatively. The official report reads partial dehiscence along the posteromedial aspect; severe paravalvular leak.



TEE was performed intraprocedurally by the cardiologist. Preintervention TEE showed partial dehiscence of anteromedial aspect with severe PVL. The leak was closed using 3 vascular plugs that were placed into the leak. The image shows trace residual PVL post-intervention.

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#FR 3 – Kiosk 6 – Poster 5

Suspected Pericardial Effusion on Echocardiography Following TAVR

Presenting Author: Tiffany J. Lim, MD, Resident Physician, Baylor College of Medicine

Introduction

Pericardial effusion is a possible consequence of transcatheter aortic valve replacement (TAVR). If left undiagnosed or untreated, it can progress to cardiac tamponade. During TAVR, myocardial injury is the most common cause of pericardial effusion. Echocardiography is the fastest and most sensitive diagnostic approach; however, other factors must be considered prior to intervention. We present a case with suspected pericardial effusion and the course of management for this patient.

Case Presentation

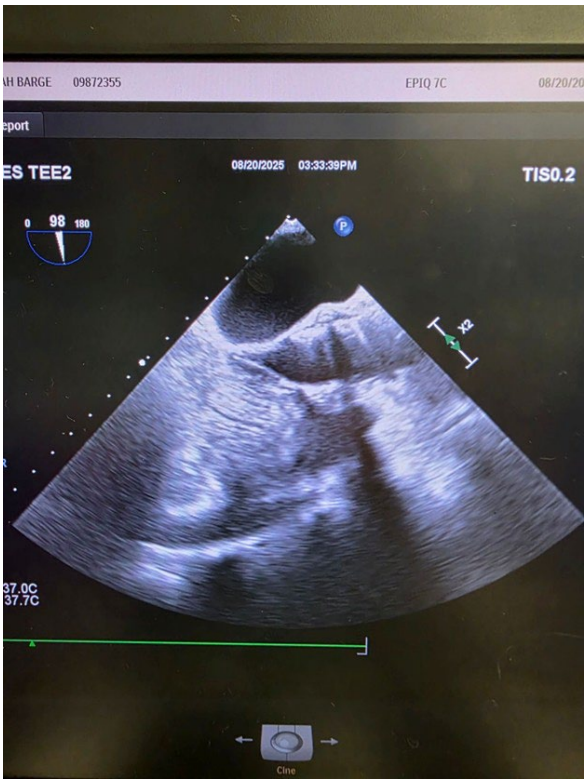
A 66-year-old patient with a history of severe aortic stenosis, asthma, hypothyroidism, and hyperlipidemia presented for TAVR. The anesthetic plan was general anesthesia due to patient preference. The procedure proceeded without complication, but post-op TTE showed a pericardial effusion. At this time, it was noted that the patient developed tachycardia without hemodynamically instability. A decision was made to do a TEE exam, which showed evidence of an enlarging pericardial effusion as confirmed by two cardiologists. After some discussion with the cardiothoracic surgeon, a decision was made to perform a pericardial window. Surprisingly, there was no significant blood or fluid within the pericardial sac upon exposure. Upon further examination, there was no indication of excessive fluid that would indicate a pericardial effusion on direct visualization. However, we were still seeing the hypodense area representative of a pericardial effusion on TEE. We repeated the TEE exam and noticed an enlarged left atrial appendage. We attempted to focus our exam in that area and after much discussion, determined it was likely that the left atrial appendage was large enough to wrap around the left ventricle, giving the false appearance of a pericardial effusion. The patient remained hemodynamically stable throughout the case and was transferred to the ICU post-operatively. She was able to be weaned off the ventilator and extubated within 6 hours and only reported mild chest pain. She was discharged 2 days later without further complications.

Discussion

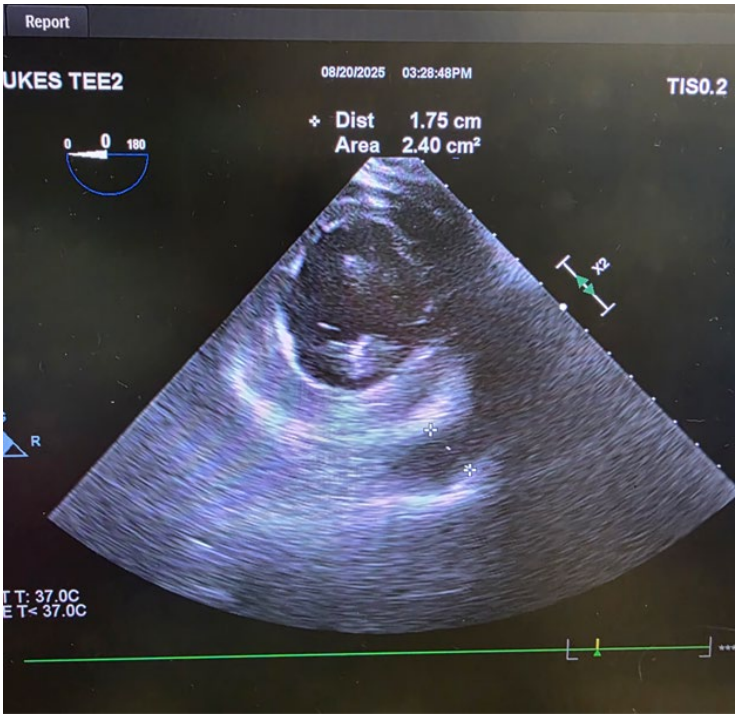
Pericardial effusion is a feared complication of TAVR, especially since there is a risk of progression to cardiac tamponade. In a recent study by the Cleveland Clinic, tamponade occurred in 0.9% of patients who underwent TAVR. They were most often diagnosed during the procedure by TTE or TEE. The source of bleeding can be either ventricles, the aortic root, or the ascending aorta. In approximately 79% of the time, injury occurs in the left ventricle, followed by 21% in the right ventricle. Injury from the left ventricle typically results from perforation by the stiff guide wire or the catheter after it retroactively passes the aortic valve. Right ventricular injury typically results from the transient pacing wires. There are different approaches to management of a pericardial effusion after TAVR. If the effusion is small and not enlarging, and the patient is hemodynamically stable, expectant management is appropriate. If the effusion is enlarging and/or patient displays signs of hemodynamic compromise, then a pericardiocentesis or surgical drainage may be

indicated.

Images







Transgastric short axis view



Post-sternotomy TEE

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#FR 3 – Kiosk 6 – Poster 6

BiVAD placement in the setting of severe dilated cardiomyopathy secondary to a TTN gene mutation

Presenting Author: Philip J. Cook, DO, Fellow, Maine Medical Center

Introduction

Left ventricular assist device (LVAD) placements can serve as a bridge to transplantation or a mechanism for improved quality and length of life in the setting of end-stage heart failure (ESHF) [1]. Patients requiring LVAD placement are often difficult to optimize hemodynamically, especially if biventricular heart failure is present, creating unique challenges for anesthetic management related to volume status and contractility. However, temporary RVAD placement can enhance optimization and improve outcomes in [3]. Variations in the TTN gene are a known risk factor for cardiomyopathy with truncating variants (TTNtv) present in ~25% of familial dilated cardiomyopathy cases [2]. These cardiomyopathies can result in ESHF that requires utilization of mechanical support devices.

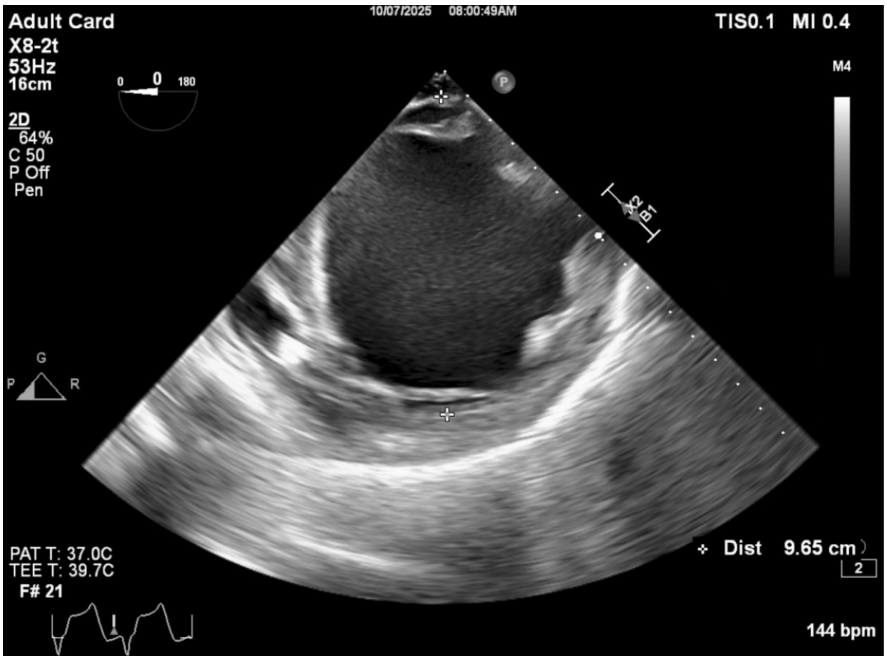
Case Presentation

A 32-year-old man with a known TTN gene mutation was transferred for management of decompensated biventricular heart failure. The patient had been lost to follow-up for several months with limited clinical care. His other medical history included a bicuspid aortic valve with moderate AI, a thoracic aortic aneurysm, and alcohol abuse. Planned LVAD placement was delayed for three weeks due to difficulty optimizing his volume status. Impella device placement was discussed, but an intra-aortic balloon pump (IABP) was inserted instead after the discovery of a left ventricular (LV) thrombus. The patient also developed hyponatremia and required initiation of continuous renal replacement therapy (CRRT) for further management. On the day of surgery, the patient presented for Heartmate 3 LVAD placement and LV thrombectomy, with consideration of RVAD placement in the setting of severe right-sided heart failure (Fig. 1A, B). After the induction of anesthesia, the patient became progressively bradycardic and mean arterial pressure (MAP) fell precipitously. A transesophageal echocardiography (TEE) probe was inserted and stagnant flow was noted. CPR was initiated and ROSC was achieved 2 minutes later with initiation of cardiopulmonary bypass 22 minutes after the initial arrest. The case involved multiple episodes of significant hypotension in the setting of LV “suck-down” related to the LVAD. Severe refractory vasoplegia developed post-bypass and required significant support. The patient’s initial post-operative course was complicated by accidental displacement of the RVAD outflow cannula on day 1 (Fig. 1C).

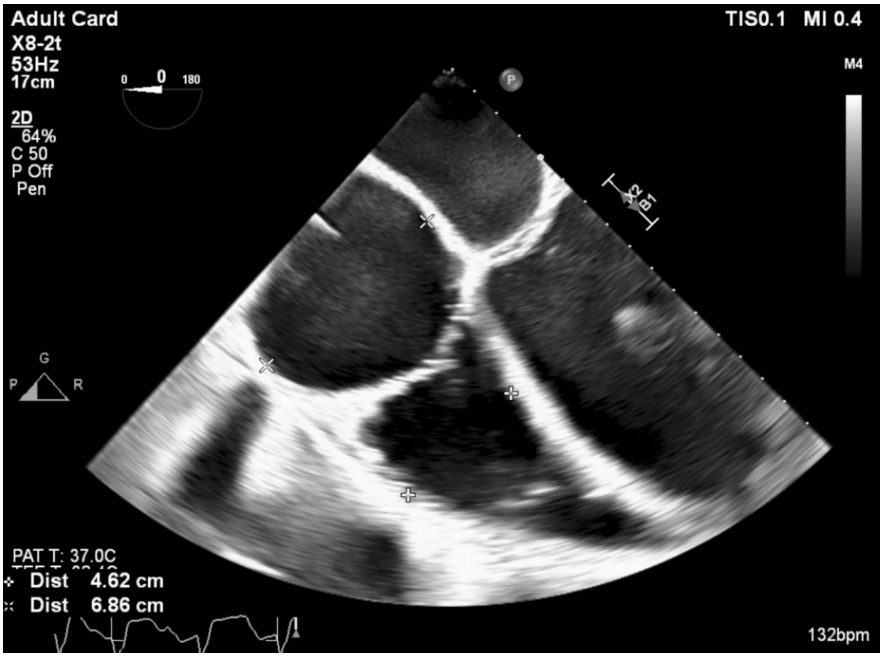
Discussion

Here, we present a case of complex anesthetic and hemodynamic management for BiVAD placement using a CentriMag RVAD and a Heartmate 3 LVAD. Post-operatively, the patient underwent successful RVAD removal one week later and was discharged home in three weeks. Despite numerous social issues, the patient remains in follow-up for potential transplant once he has reached 6 months of sobriety. This case illustrates the complexities of anesthetic management for device insertion in severe biventricular heart failure, as well as techniques for the management of intraoperative refractory vasoplegia.

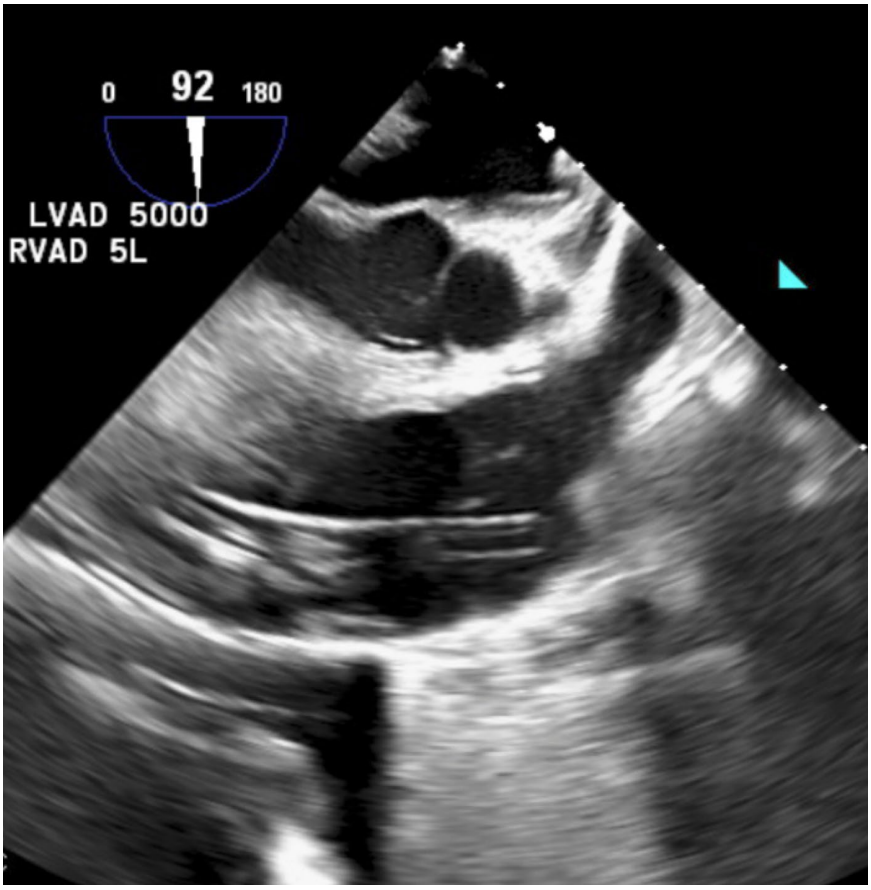
Images



Immediate pre-op left ventricle



Immediate pre-op right ventricule



POD #1 RVAD outflow at pulmonic valve annulus

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#FR 3 – Kiosk 6 – Poster 7

Resection of Pulmonary Artery Angiosarcoma Causing Right Ventricular Failure

Presenting Author: Daniel Casey, DO, MPH, Fellow in Adult Cardiothoracic Anesthesiology, University of Pennsylvania

Introduction

Pulmonary artery (PA) intimal sarcoma (PAIS) is a rare, aggressive malignancy arising from the vascular endothelium with incidence of 0.001 to 0.030%[1]. The symptoms associated with this tumor are often vague and ill-defined, delaying diagnosis until a patient has developed significant heart failure symptoms[2,3]. Moreover, there is no standard treatment protocol following diagnosis. In the appropriate patient, however, palliative surgical resection can lead to an improvement in quality of life and an unlikely chance of cure[2]. This case details a patient who underwent bilateral PAIS resection and pulmonic valve replacement (PVR).

Case Presentation

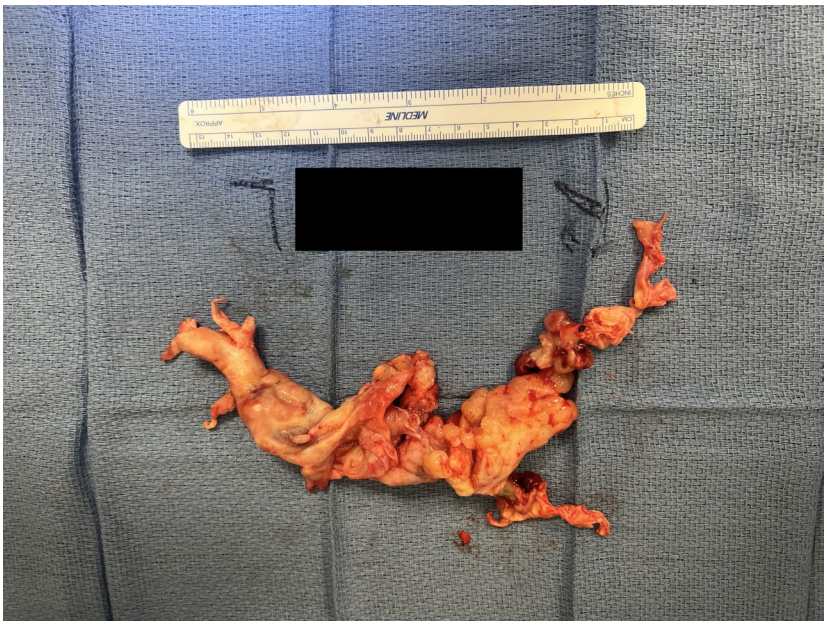
A 49-year-old man without prior medical history presented to the hospital with 6 months of progressive dyspnea. CT showed a mass extending from the pulmonic valve into the main and proximal left and right PAs. Biopsy confirmed PAIS and palliative surgical resection was planned due to his significant symptoms. Preoperative workup demonstrated right ventricular (RV) systolic dysfunction and RV systolic pressure of 110 mmHg. In the operating room, right brachial arterial line, right internal jugular vein introducer, and femoral arterial and venous access in preparation for possible emergency cardiopulmonary bypass (CPB) were obtained. After extensive preoxygenation with head elevated and initiation of epinephrine infusion for RV support, rapid sequence intubation with midazolam, ketamine, fentanyl, and rocuronium was performed using videolaryngoscopy to minimize apnea time and RV afterload from mask ventilation. The patient was hemodynamically stable throughout induction without need for additional inotropes or vasopressors. Intraoperative TEE found severely reduced RV function, moderate-severe pulmonic stenosis from tumor obstruction, and patent foramen ovale (PFO) with right-to-left shunt. CPB was initiated via ascending aortic and bicaval cannulation. The surgical team performed resection of a large tumor extending from the pulmonic valve into the bilateral PAs, bioprosthetic PVR, patch closure of the left PA, and PFO closure. Separation from CPB was uneventful on vasopressin infusion. Post-CPB management included avoiding inotropy, pulmonary vasodilators, and volume overload with diuresis to prevent pulmonary reperfusion injury. The patient was discharged on postoperative day 5 after uneventful recovery with significant improvement in symptoms and RV hemodynamics. He ultimately passed away from the malignancy 8 months post-procedure.

Discussion

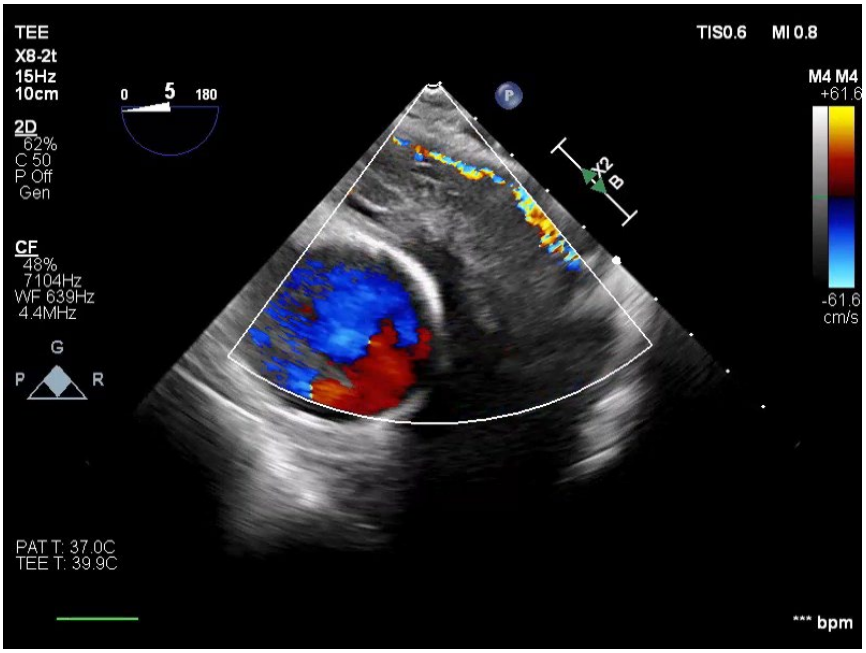
While a diagnosis of PAIS confers a poor prognosis, this case exemplifies the significant improvement in quality of life that surgical resection can provide to patients with operable disease[1,3]. It is important to recognize that this is a high-risk procedure given the associated RV dysfunction and significant increase in RV afterload[3]. Therefore, anesthetic management requires careful attention to hemodynamic control, RV

afterload reduction, inotropic support, and avoidance of pulmonary reperfusion injury. With these perioperative management principles, surgery affords a chance for improvement of heart failure symptomatology, future chemotherapy and radiation therapy, and the opportunity to return home from the hospital[2].

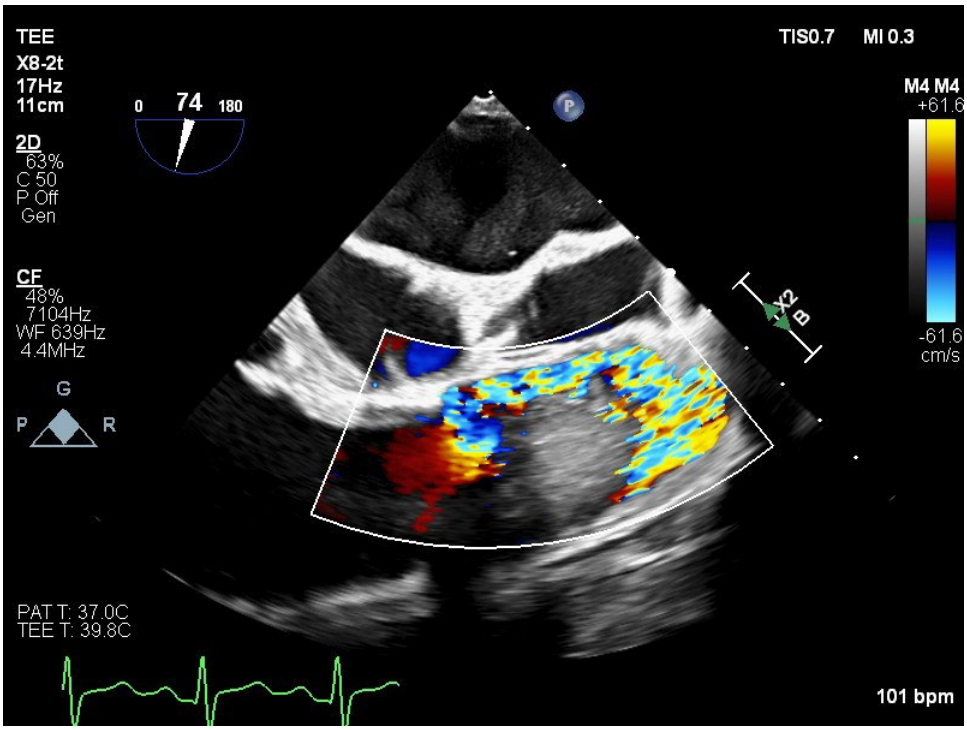
Images



Tumor specimen resected from pulmonary arteries.



Mid-esophageal ascending aorta short axis view with color flow doppler demonstrating tumor burden and flow obstruction in the pulmonary arteries.



Right ventricular inflow outflow view demonstrating tumor burden and obstruction in right ventricular outflow view.

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#FR 3 – Kiosk 7 – Poster 1

State-of-the-Art General Anesthesia Approach to a Patient with End-Stage Eisenmenger Syndrome Requiring Rigid Laryngoscopy for Expedited Heart-Lung Transplant Evaluation

Presenting Author: Akansha Tarun, MD, DPhil, Anesthesiology Resident, Stanford

Introduction

The natural course of uncorrected, unrestrictive, congenital left-to-right ventricular septal defects (VSDs) is the development of Eisenmenger Syndrome and ultimately the need for combined heart-lung transplantation (HLTx). Severe pulmonary arterial hypertension (PAH) with secondary combined systolic & diastolic right ventricular (RV) heart failure, reversal of VSD shunt directionality, and severe systemic hypoxemia are the end-results. The cascade is initiated by excessive pulmonary artery cardiac output relative to systemic cardiac output, culminating in pulmonary arterial remodeling with fixed PAH. Superimposed World Health Organization Group 4 chronic thromboembolic PAH worsens end-stage VSD pathophysiology. Malignancy screening is a core component of pre-HLTx patient eligibility given risks of lymphoproliferation with chronic immunosuppression.

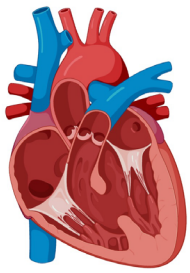
Case Presentation

A 54-year-old male with Eisenmenger Syndrome due to a large perimembranous VSD and chronic thromboembolic PAH (recurrent deep vein thromboses and inferior vena cava (IVC) filter history) presented to Stanford for HLTx evaluation and was found to have a left pharyngeal mass requiring biopsy. Preoperative assessment of the patient included exam, right heart catheterization, echocardiography, and labs (Table 1). Intraoperatively, spontaneous ventilation general anesthesia with no definitive airway was required to facilitate rigid laryngoscopy. Hemodynamic management centered around RV preservation: maintaining right coronary perfusion pressure with norepinephrine, RV inotropy with epinephrine, and preventing worsening of the fixed PVR thru ventilation/oxygenation control, prophylactic INO, and avoidance of light anesthesia. These goals were accomplished in a slow measured approach. Pre-induction was notable for placement of a 12cm brachial arterial line, oropharyngeal lidocaine, high-flow NC oxygen @ 40L/min with INO @ 20PPM, and background epinephrine and norepinephrine infusions. Cardiothoracic Surgeons in parallel placed femoral Arterial & Venous micropuncture catheters for VA ECMO standby, and Perfusionists set up a primed ECMO pump. The right internal jugular vein was not secured but anticipated for additional venous cannulation due to the IVC filter. Induction involved initiation and gradual escalation of a dexmedetomidine, ketamine, propofol, and remifentanil infusions and/or boluses. These anesthetics, chosen for their spontaneous breathing properties, were titrated to a continuous provocative jaw lift, aimed at emulating the impending rigid laryngoscope. This jaw lift was exchanged for laryngoscope, while preserving respiratory drive, airway patency, and necessary depth of anesthesia. Additional O2 was added to the laryngoscope side port. ENT successfully explored and obtained biopsies of the mass and left tongue base. The patient was re-animated and uneventfully weaned from hemodynamic support.

Discussion

Anesthetic management of end-stage right-sided cardiopulmonary failure due to Eisenmenger Syndrome from concomitant VSD and chronic thromboembolic disease requires well-defined hemodynamic goals and targeted hemodynamic support. The need for an unsecured, shared airway during rigid laryngoscopic surgery increases complexity. Immediate readiness for rescue VA ECMO provides an additional safety net.

Images



Right Heart Catheterization	**Room Air without inhaled nitric oxide**	**Room Air with inhaled nitric oxide (20 PPM)**
O_2 Saturations (%)	86	89
MvO_2 (%)	66	68
PAPs (mPAP, mm Hg)	142/50 (80)	134/51 (78)
PAWP (mm Hg)	5	7
PVR (WU)	22.5	19
Qp:Qs	0.77	0.91

Table 1: Patient's Preoperative Assessment. The patient's physical exam was remarkable for a normocephalic airway, unobstructed airway auscultation, 4L/M NC O2 Saturation of 86%, jugular venous distention, and poor peripheral capillary refill. Right heart catheterization was performed on room air with and without inhaled nitric oxide (INO) at 20 parts per million (PPM). Pre and post O2 Saturations were 86% & 89% respectively, Mixed venous oxygen saturation (MVO2) 66% & 68%, Pulmonary artery pressures (PAPs) 142/50 (80) & 134/51 (78), Pulmonary artery wedge pressure (PAWP) 5mmHg & 7mmHg, and Pulmonary vascular resistance (PVR) 22.5 Wood units (WU) & 19 WU. The pulmonary-to-systemic blood flow ratio (Qp:Qs) ratio rebalanced from 0.77 to 0.91 post INO. Echocardiography was confirmatory of a large bi-directional VSD as well as severe right ventricular (RV) hypertrophy, moderate RV dilatation, moderate RV systolic dysfunction, and mild left ventricular (LV) systolic dysfunction. Hematocrit was predictably polycythemic at 47%.

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#FR 3 – Kiosk 7 – Poster 2

Unmasking of Severe Mitral Regurgitation after Aortic Valve Replacement

Presenting Author: Anya K. Margalit, MD, Resident, University of California, San Diego

Introduction

The continuity of the aortomitral curtain has important implications for the pathophysiology of aortic and mitral valve disease; intervening on one valve can directly influence the function and dynamics of the other. We present a case of severe mitral regurgitation (MR) that developed after aortic valve replacement (AVR).

Case Presentation

A 75-year-old male with a medical history of hypertension, aortic stenosis (AS), and aortic aneurysm was admitted to our hospital for planned aortic aneurysm replacement and aortic valve replacement (AVR). Transthoracic echocardiography (TTE) revealed a dilated ascending aorta, severe AS, normal systolic function, and mild MR. Intraoperative transesophageal echocardiography (TEE) confirmed TTE findings. The patient underwent bioprosthetic AVR with ascending aorta replacement and was separated from cardiopulmonary bypass (CPB) without issue. However, post-separation TEE revealed new, severe MR with systolic anterior motion (SAM) of the mitral valve (Figures 1a and 1b). The remainder of the exam demonstrated the aortic graft replacement, normally functioning bioprosthetic AVR, and normal biventricular function without left ventricular outflow tract (LVOT) obstruction or basal septal hypertrophy. Conservative measures to treat SAM were employed but proved unsuccessful, and MR remained severe. Additionally, pulmonary artery systolic pressures rose from 35/20 mmHg to 60/30 mmHg. After discussion with the cardiac surgery team, the decision was made to reinitiate CPB for mitral valve replacement (MVR). The patient underwent successful bioprosthetic MVR and was transported to the ICU in stable condition. The patient was discharged home on post-operative day ten.

Discussion

Intraoperative TEE is essential for real-time assessment of cardiac function and valvular pathology during cardiothoracic surgery. In this case, the development of new SAM with severe MR following AVR was likely related to unmasking of previously subclinical pathology. Given the lack of significant MR on pre-operative imaging, the patient's severe AS was likely masking an underlying predisposition to SAM. Retrospective review of pre-bypass TEE revealed anatomical features predisposing to SAM: low C-sept distance (1.62 cm), low anterior/posterior leaflet ratio, anterior displacement of mitral coaptation, and acute aortomitral angle (110°) (Figure 2). Changes in left ventricular loading after AS relief increase drag forces on the anterior leaflet, further promoting SAM. While SAM after MVR is well documented,[1] SAM and MR after AVR can also occur due to these changes.[2,3] Conservative treatment proved unsuccessful, requiring MVR. Rapid interpretation of TEE after CPB guides resuscitation and surgical decision making. Understanding of the complex relationship between the mitral coaptation point, basal ventricular septum, and LVOT allows for diagnostic explanation of new onset mitral insufficiency. Preoperative appreciation of patients geometrically at risk for SAM and MR can obviate delays in recognition and management, and this case demonstrates this

potential value. This case underscores the need for vigilance in monitoring for new or worsening MR after AVR and highlights the complex anatomical and hemodynamic relationships that can lead to unexpected intraoperative findings.

Images

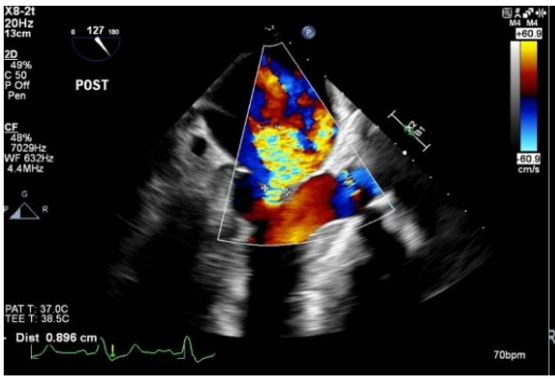


Figure 1a: New, severe mitral regurgitation after separation from cardiopulmonary bypass (CPB)

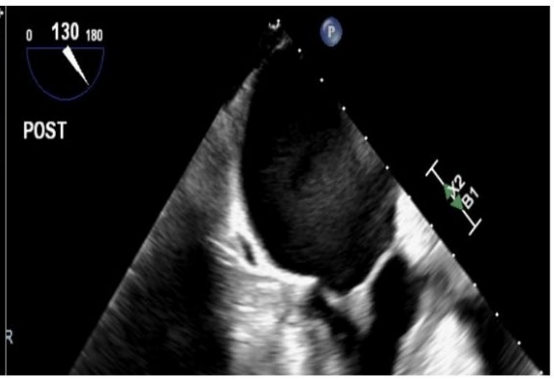


Figure 1b: New, systolic anterior motion (SAM) of the mitral valve after separation from cardiopulmonary bypass (CPB

Figure 1a: New, severe mitral regurgitation after separation from cardiopulmonary bypass (CPB). Figure 1b: New, systolic anterior motion (SAM) of the mitral valve after separation from cardiopulmonary bypass (CPB).

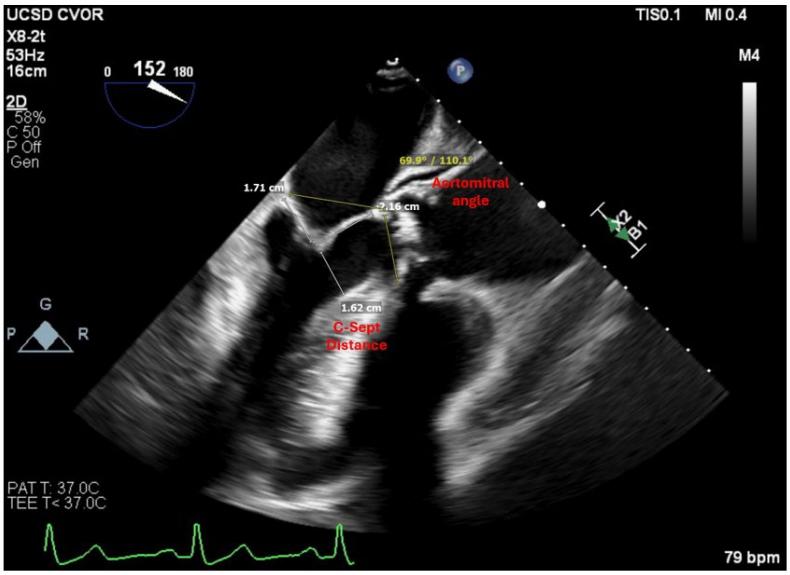


Figure 2: Imaging showing this patient's predisposition to systolic anterior motion (SAM) of the mitral valve with a c-sept distance of 1.62cm, aortomitral angle of 110°, and relative anterior displacement of mitral coaptation point

Figure 2: Imaging showing this patient's predisposition to systolic anterior motion (SAM) of the mitral valve with a c-sept distance of 1.62 cm, aortomitral angle of 110°, and relative anterior displacement of mitral coaptation point.

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#FR 3 – Kiosk 7 – Poster 3

Thrombus on the loose: Perioperative Management of Renal Cell Carcinoma Tumor-Thrombus Extending into the Right Atrium

Presenting Author: Justin Miller, MD, Anesthesiology Resident, University of Maryland School of Medicine

Introduction

Invasion of Renal Cell Carcinoma (RCC) into the renal vein and inferior vena cava (IVC) occurs in 5-15% of cases1-2. Tumor-thrombi below the diaphragm (type I and II) are managed with radical nephrectomy and excision3. However, supradiaphragmatic IVC and right cardiac chamber extension (type III and IV) occurs in only 1% of all cases, requiring cardiopulmonary bypass (CPB) in order to adequately visualize, and resect2. This case describes right-sided RCC extending to the cavoatrial junction, requiring CPB, hypothermic circulatory arrest (HCA), IVC thrombectomy with reconstruction, and right nephrectomy.

Case Presentation

A 69-year-old male was diagnosed with RCC after presenting to the emergency room with hematuria. Imaging revealed a large 12x13 cm mass in the right kidney with tumor thrombus extension into the IVC and RA. He underwent a complex procedure involving sternotomy with CPB and HCA to facilitate RA tumor thrombus removal, right nephrectomy, and IVC thrombectomy and reconstruction. On the day of surgery, after uneventful induction of anesthesia, arterial and central venous lines were placed. Transesophageal echo (TEE) showed thrombus extension from the intrahepatic IVC to the RA/IVC junction (Figure-1, 2), with otherwise normal biventricular function and valvular structures. Sternotomy and abdominal dissection were performed simultaneously to expose the heart and kidney tumor with associated vasculature. Cannulation sites were the distal ascending aorta, superior vena cava and RA appendage, with an additional return cannula in the infrarenal IVC. The innominate artery was used for anterograde perfusion. The patient was cooled to 26 degrees Celsius prior to circulatory arrest. A right atriotomy was performed to free partially adherent intracardiac tumor. The remainder of the tumor was pushed inferiorly and removed through the intra-abdominal vena cava. Angioscopy with a cystoscope confirmed complete removal. Nephrectomy with tumor resection was performed en bloc (Figure-3) and the IVC was repaired. Patient was weaned off bypass without complication and post-circulatory arrest coagulopathy was corrected. TEE confirmed resolution of right atrial thrombus. The postoperative course was notable for new atrial fibrillation with RVR but was otherwise unremarkable and he was discharged on post-operative day 11.

Discussion

Tumor extension of RCC into the right cardiac chambers is rare and requires multidisciplinary surgical approach with CPB often requiring hypothermic circulatory arrest to ensure a bloodless surgical field and enable complete tumor removal. Key anesthetic considerations include close communication with the surgical team about TEE findings prior to manipulation, as thrombus dislodgement can cause pulmonary embolism, RVOT obstruction, right heart failure, or circulatory collapse. Additional concerns include renal and hepatic congestion with IVC clamping and significant coagulopathy following hypothermic circulatory

arrest.

Images

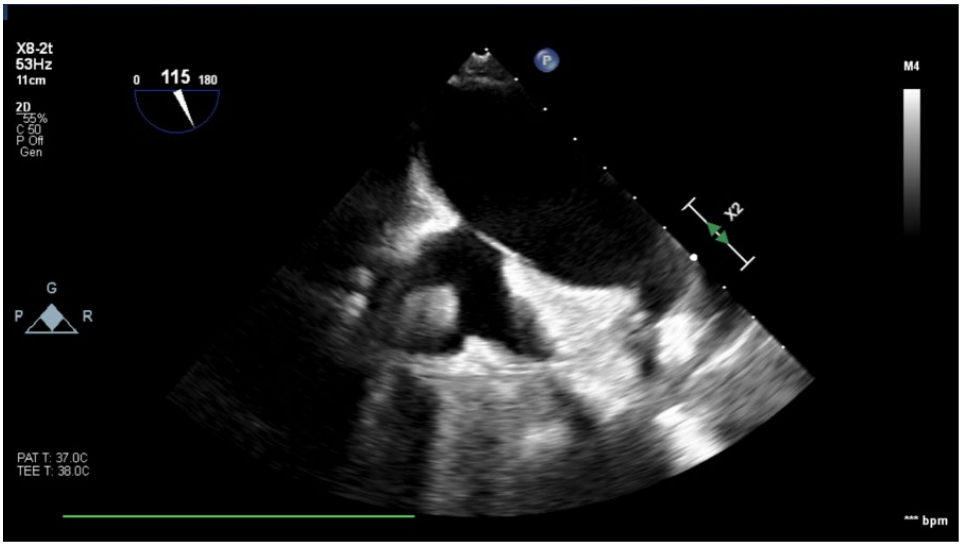


Figure-1: Thrombus in the RA

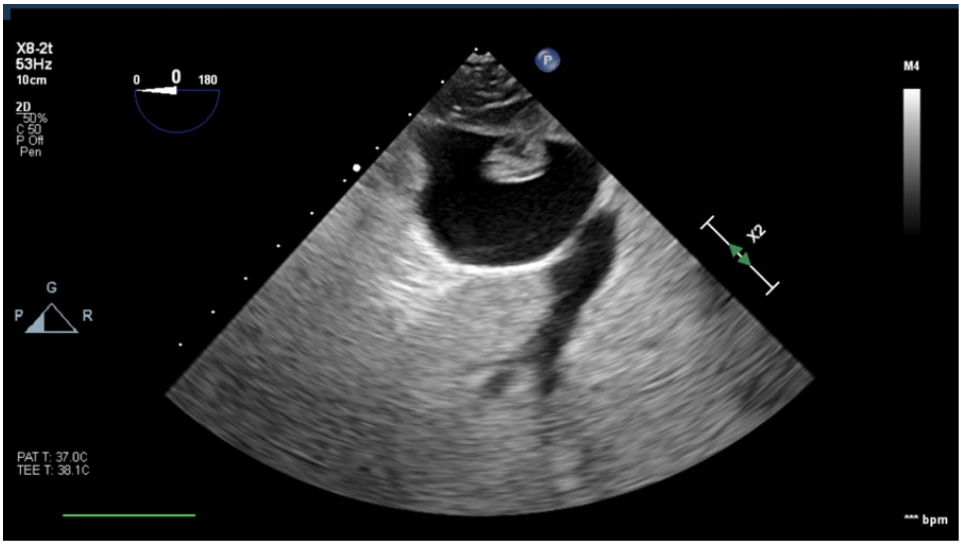


Figure-2: Thrombus in the intrahepatic IVC

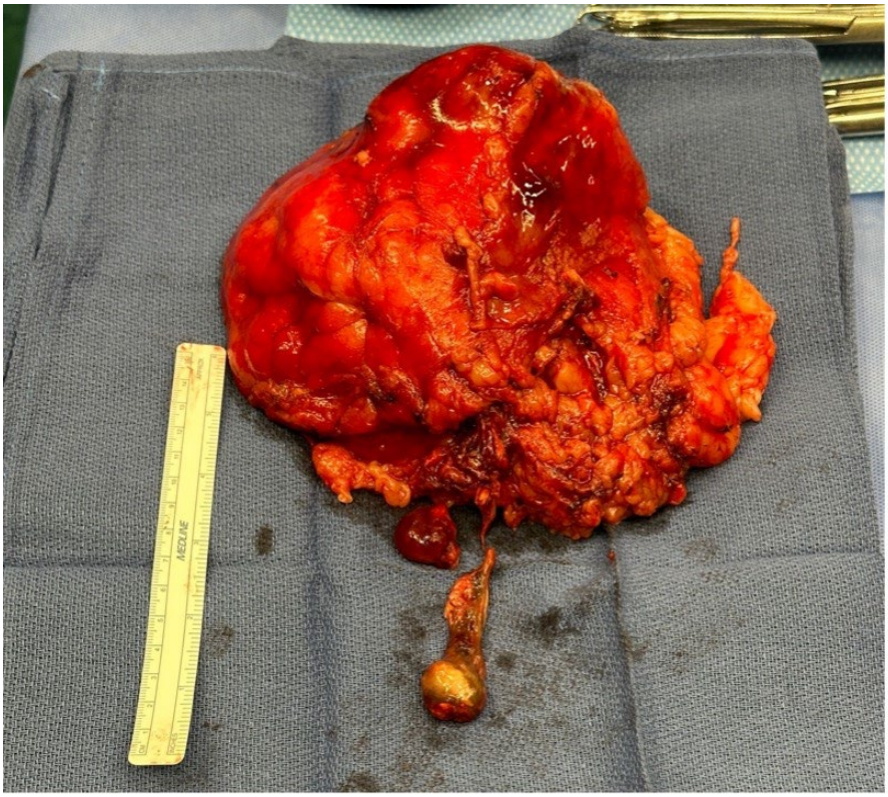


Figure-3: Kidney and thrombus after resection

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#FR 3 – Kiosk 7 – Poster 4

Threading the Wire: Complications from Superior Vena Cava Thrombus Recanalization

Presenting Author: Collin M. Goldstein, MD, Anesthesiology Resident, Emory University

Introduction

Venous thrombosis is a well-recognized complication of long-term central venous access. Management of catheter-associated thrombosis of the superior vena cava (SVC) typically requires a multidisciplinary approach. Here we present a patient with chronic thrombus of her SVC associated with a Hickman catheter causing SVC syndrome and its downstream complications.

Case Presentation

53 yo female with Crohn's, short gut syndrome with subsequent TPN dependence through a Hickman catheter, and known SVC occlusion presented for optimization of her central access. The distal portion of the Hickman was looped, with partial loss of functionality. During SVC recanalization, venous access was established and serial balloon dilation was used to plasty the tract in preparation for a stent. The patient became unstable with loss of pulses necessitating CPR. A Coda balloon was emergently inflated in the SVC to occlude blood loss and a pericardial drain was placed. After stent deployment, the patient once again lost pulses requiring emergent operative intervention. Given potential for exsanguination, peripheral cardiopulmonary bypass (CPB) was initiated to evaluate the extent of injuries. Upon entering the pericardium, a large volume of blood gushed into the mediastinum. The stent and balloon were extravascular with the wire traversing from the SVC-innominate junction into the right atrium. The defects were repaired and the patient was weaned off CPB without difficulty. Due to ongoing coagulopathy and edematous state, the chest was left open and then transported to the ICU for further care. Her chest was closed on POD 3. However, she developed a subdural hematoma with associated seizures and neurostorming. A brain MRI showed anoxic brain injury and the family elected to place her on comfort measures.

Discussion

Superior vena cava (SVC) syndrome encompasses a range of clinical presentations and its management depends heavily on the underlying etiology, location of obstruction, and acuity of symptoms. In patients with long-term intravascular access, catheter-associated thrombosis is common. Chronic thrombotic occlusions often require an endovascular strategy; however, these interventions can carry significant risk. A multidisciplinary approach is essential when complications arise. Immediate availability of cardiac surgery and cardiac anesthesiology teams with TEE expertise can significantly influence outcomes. In our case, the TEE team was mobilized to evaluate hemodynamic instability. Identification of a large pericardial effusion with concern for mediastinal hemorrhage prompted rapid transport to the operating room. The emergent nature of the situation required early planning for potential CPB cannulation strategies. Central cannulation would have been hazardous in the setting of suspected mediastinal bleeding and the risk of uncontrolled hemorrhage upon sternotomy. Peripheral cannulation was therefore selected, allowing the patient to be

placed on CPB quickly and safely. Ultimately, a thorough understanding of the patient's pathology and anticipation of procedure-specific complications were instrumental in completing emergent repair.

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#FR 3 – Kiosk 7 – Poster 5

Bilateral Parasternal Intercostal Fascial Plane Catheters for Postoperative Pain Control in an Opioid-Tolerant Cardiac Surgery Patient

Presenting Author: Aliena E. Lowell, MD, Anesthesiology Resident, University of Virginia

Introduction

Throughout recovery from cardiac surgery, adequate pain management is essential to facilitating an early neurological exam, supporting safe extubation, and reducing barriers to early mobilization and ambulation. Using a targeted approach, regional anesthesia for cardiac surgery supports patient recovery, especially in patients who are opioid tolerant.

Case Presentation

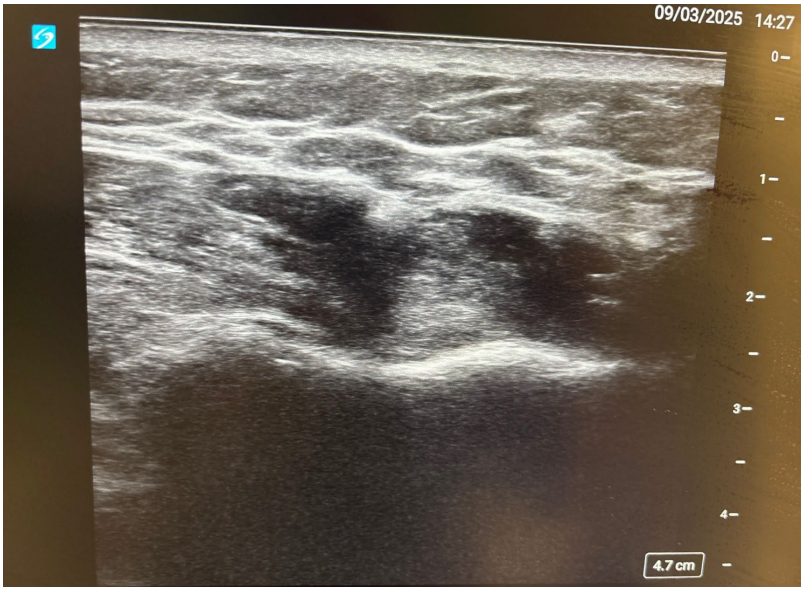
Patient SS is a 53-year-old gentleman with severe bicuspid aortic valve stenosis requiring surgical aortic valve replacement. He had also experienced a motor vehicle accident three years prior to presentation which involved bilateral lower extremity fractures. The protracted course of his lower extremity wounds caused him to develop opioid use disorder for which methadone therapy was initiated. At the time of his surgery, he was maintained on 75 mg of methadone daily. Due to concerns over the difficulty of his postoperative pain control, the decision was made to perform bilateral ultrasound-guided parasternal intercostal fascial plane (PIFP) blocks with placement of continuous infusion catheters. The single-shot blocks consisted of 15 mL of 0.2% ropivacaine administered to each side and the catheters delivered an infusion of 0.2% ropivacaine at 1 mL/hr with programmed intermittent boluses of 8 mL occurring every hour. The surgery went as planned without complication and the regional techniques were performed once closure of the skin was complete. SS was successfully extubated on post-operative day (POD) 0. His pain was well controlled with his baseline methadone dosing, a multimodal pain regimen, and the bilateral PIFP catheters. He was able to ambulate on POD 1 with assistance from physical therapy. His postoperative course was without complication, and he was discharged home on POD 6. Following removal of his Prevena dressing, his sternum appeared well-approximated without signs of infection.

Discussion

The use of regional anesthesia techniques in cardiac surgery is an emerging area of focus in cardiothoracic anesthesia. The PIFP block performed on SS involves the injection of local anesthetic between the pectoralis major muscle and the internal intercostal muscle and/or the external intercostal membrane adjacent to the sternum.[1] Multiple randomized controlled trials have been performed regarding the efficacy of single-shot PIFP blocks in cardiac surgery with midline sternotomies and have demonstrated a reduction in time to extubate, improved pain scores, and decreased postoperative opioid consumption.[2, 3] Improvements in these factors could lead to reduced risk of respiratory depression, decreased somnolence, and increased early participation in physical therapy. While multiple studies have been performed regarding the efficacy of single-shot PIFP blocks, less data is available regarding the efficacy of PIFP catheters. While a single-shot block may have proven helpful in the immediate postoperative period for SS, the pain relief would last only

as long as the local anesthetic. PIFP catheter placement, however, provided an option for continuous infusion of local anesthetic for days following surgery. This case suggests that PIFP catheters can be used to improve pain control for days following median sternotomy, especially in patients with preexisting opioid use.

Images



This is an ultrasound image taken from the placement of the left-sided parasternal intercostal plane catheter.



This is an ultrasound image taken from the placement of the right-sided parasternal intercostal plane catheter.

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#FR 3 – Kiosk 7 – Poster 6

A Life Dissected: End-Stage Renal Disease, Thrombosis, and Hemorrhage in a Type A Dissection

Presenting Author: August A. Allocco, MD, Resident Physician, Yale University School of Medicine

Introduction

Chronic kidney disease is a global health problem which affects approximately 14% of adults (Law et al., 2020). In end-stage renal disease (ESRD), there are significant alterations in fluid homeostasis, acid-base equilibrium, cardiovascular function, and blood coagulation (Kanda et al., 2017). Paradoxically, ESRD presents high risks for both thrombosis and hemorrhage which complicates anticoagulation use (Parul et al., 2025).

Case Presentation

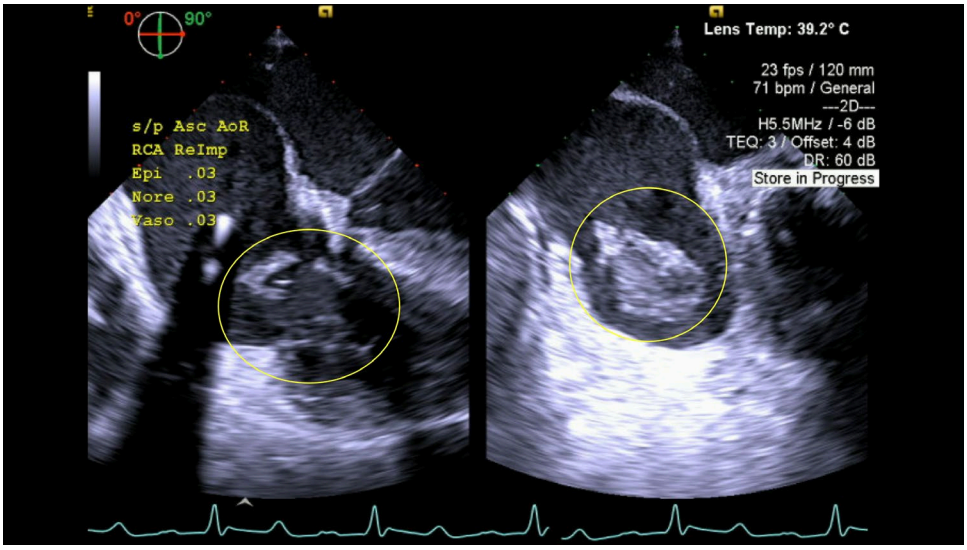
Herein, we present a Type A dissection in a 36-year-old woman with a history of ESRD secondary to hypertensive nephrosclerosis on hemodialysis who was last dialyzed the day prior to presentation. Her history was otherwise significant for hypertension, pulmonary hypertension, and marijuana use. Preoperative evaluation revealed a normal coagulation study and complete blood count with a potassium of 4.3 mmol/L. A pre-intervention transesophageal echocardiogram (TEE) showed a Type A dissection with the dissection flap at the sino-tubular junction, normal biventricular function, severe left ventricular (LV) and right ventricular (RV) hypertrophy, pulmonary artery dilation, a small left pleural effusion and small pericardial effusion. There was no evidence of thrombus or significant valvulopathy. Cardiopulmonary bypass (CPB) was initiated via the right subclavian artery and right atrium. Heparin was administered and the ACT was >1000 seconds after 3 minutes. TEE confirmed forward flow in the true lumen of the aortic arch. The aortic arch and root were resected and replaced with a Hemashield graft without major complications. The patient was weaned from CPB and post-repair TEE showed good biventriuclar function. At approximately 50% of protamine administration, the patient desaturated and became hypotensive refractory to multiple epinephrine and norepinephrine boluses. The patient was heparinized and placed back on CPB. TEE revealed a large thrombus in the RV (Image 1) and pulmonary artery. After removal of the thrombi, the patient was weaned from CPB for the second time, and platelets, cryoprecipitate, and fresh frozen plasma were slowly administered in lieu of protamine. Nevertheless, a new thrombus was detected in the superior vena cava (Image 2) and CPB was restarted to extract it. After weaning from CPB, the third time, the patient developed RV failure. CPB was re-initiated, and ECMO cannulation of the femoral veins was unsuccessful due to thrombi in the iliac veins. Central ECMO was initiated via the aortic graft and right atrial cannula but

rapidly failed due to impaired venous drainage refractory to circuit replacement. Further resuscitation was determined to be futile, and the patient expired shortly thereafter.

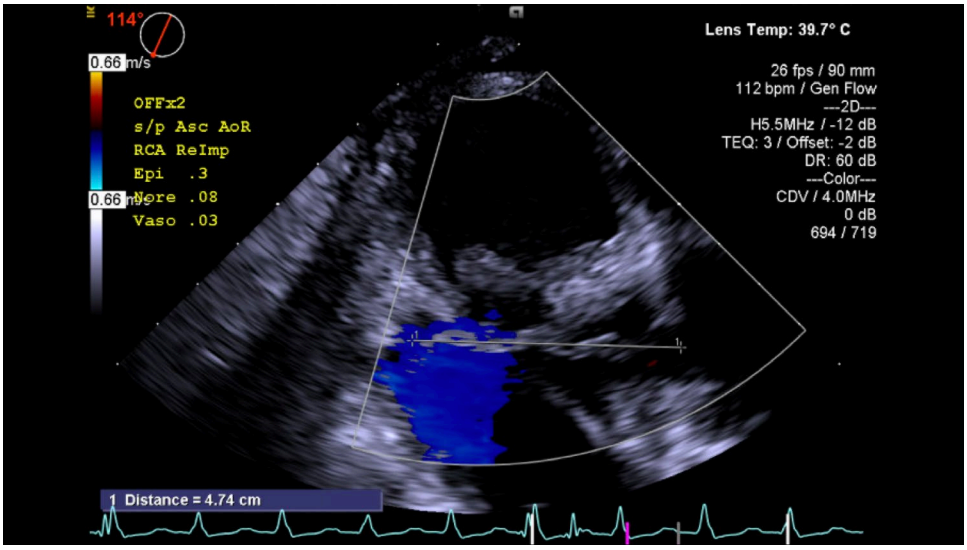
Discussion

Patients with ESRD face dual challenges of increased susceptibility to thrombotic events and bleeding complications. Venous thrombosis and pulmonary embolisms may arise due to a hypercoagulable state, while hemorrhage may occur due to impaired platelet aggregation, uremic toxins, and reduced production of coagulation factors. The use of anticoagulation in this population is challenging and warrants a multidisciplinary discussion, assessment, and plan particularly for the patient undergoing cardiac surgery.

Images



Thrombus in the right ventricle and pulmonary artery after administration of protamine.



Thrombus in the superior vena cava after second unsuccessful attempt to wean from cardiopulmonary bypass.

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#FR 3 – Kiosk 7 – Poster 7

When Vasospasm Mimics Infarction: The Role of ECLS in a Reversible Catastrophe

Presenting Author: Joshua E. Insler, Doctor of Medicine (MD), Resident Physician, Department of Anesthesiology, Yale School of Medicine

Introduction

The American Heart Association has yet to include recommendations for the initiation of extracorporeal life support (ECLS) and extracorporeal cardiopulmonary resuscitation (ECPR) modalities to its algorithm for the management of patients suffering from myocardial infarction in the absence of obstructive coronary artery disease (MINOCA).[1] Furthermore, while implementation of ECPR as part of timely management of out-of-hospital cardiac arrest to improve survival to hospital discharge has been encouraging,[2,3] randomized controlled trials comparing outcomes of ECPR to conventional CPR for such cases are limited in number and definitive guidelines for ECPR's implementation in such settings do not exist. We present a case of out-of-hospital MINOCA resulting from diffuse coronary vasospasm of unclear etiology, complicated by repeated in-hospital cardiac arrests necessitating multiple runs of ECLS for stabilization.

Case Presentation

A 35-year-old male with past medical history significant only for polysubstance use disorder—including known cocaine use— and no history of coronary artery disease was transferred to our institution after presenting to an outside facility following an out-of-hospital cardiac arrest. He received eight rounds of advanced cardiac life support (ACLS) for ventricular fibrillatory (VF) arrest with epinephrine and amiodarone en route, was intubated on arrival, and transferred to the medical intensive care unit (MICU) where ECG showed ST-segment elevations in V1–V6 and high-sensitivity troponins were >5,000 ng/L. Urine toxicology was positive only for fentanyl and benzodiazepines. Shortly after MICU admission, he suffered recurrent VF arrest requiring multiple rounds of ACLS, with telemetry preceding the arrest suggesting an R-on-T phenomenon. Post-arrest, a formal transthoracic echocardiogram (TTE) demonstrated global hypokinesis with a left ventricular ejection fraction (LVEF) of 10–15%. Urgent left heart catheterization revealed diffuse coronary vasospasm in all vessels (Figure 1), responsive to intracoronary nitroglycerine. Upon return to the MICU the patient suffered a third cardiac arrest refractory to ACLS and was emergently cannulated for femoral–femoral venoarterial extracorporeal membrane oxygenation (VA ECMO). He was decannulated four days later, yet required repeat cannulation for VA ECMO following a fourth VF arrest within 24 hours. TTE after repeat cannulation demonstrated completely recovered LVEF of 60–65% (Figure 2). Medical management with verapamil, isosorbide dinitrate, and mexiletine were advanced before final decannulation six days later for suspected vasospastic angina. He ultimately underwent single-chamber implantable cardioverter-defibrillator placement two days prior to hospital discharge. To date, he has not had recurrence of arrhythmia with this medical regimen.

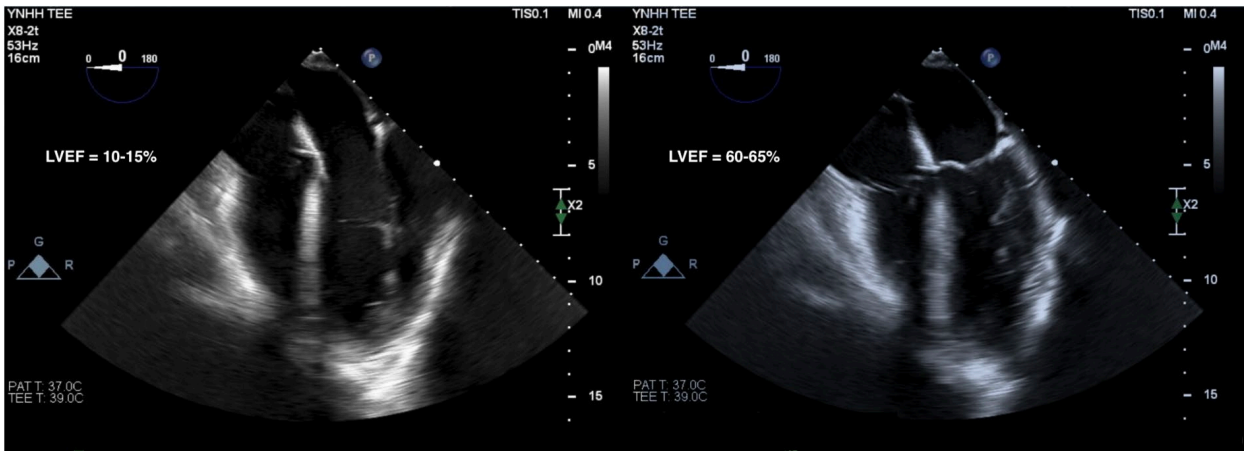
Discussion

In appropriate cases, ECLS modalities can and should be used in the management of patients suffering from

MINOCA with potentially reversible cardiac dysfunction when other therapies have proven insufficient. In cases of presumed vasospastic angina where conventional resuscitation and vasodilator therapy fail to restore adequate perfusion, VA ECMO may be considered as a viable bridge to survival while medical therapy is optimized.

Images





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#FR 3 – Kiosk 8 – Poster 1

Type A Dissection in a Patient Presenting for Orthotopic Heart Transplant

Presenting Author: Christopher Van Akin, MD, Fellow, University of Connecticut

Introduction

Implantable axial-flow rotary pump (Impella) devices are commonly used in patients with end-stage heart failure as a bridge to heart transplant. The reported incidence of aortic dissections in patients with Impella devices is very low; one study describes an incidence of 0.25%. Here we present the discovery of an aortic dissection in the OR prior to orthotopic heart transplant in a patient with an Impella in situ.

Case Presentation

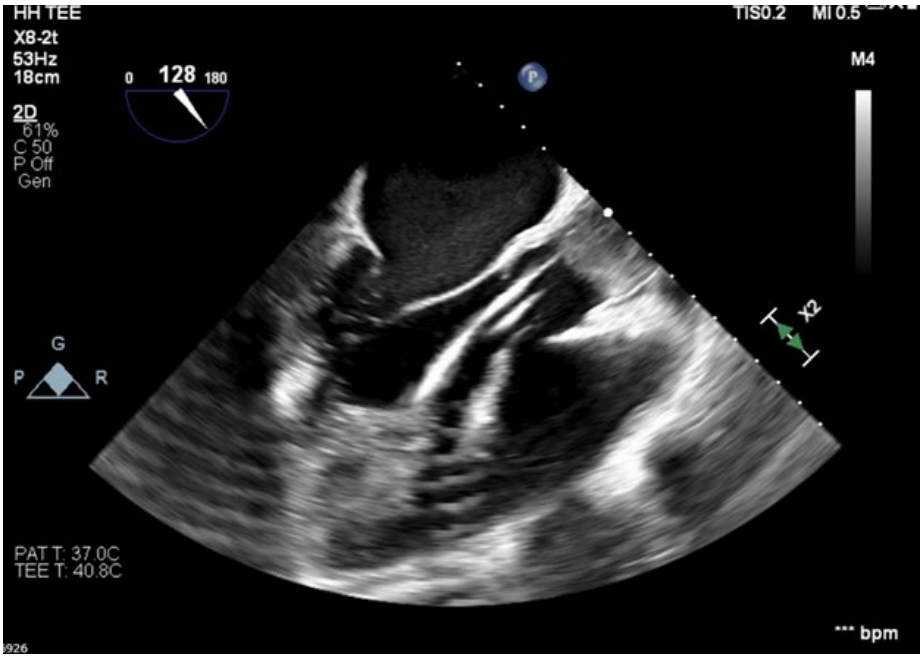
A 38-year-old male with a history of lymphoma, doxorubicin-induced nonischemic cardiomyopathy with ejection fraction < 20% on chronic home milrinone, implantable cardioverter-defibrillator (ICD) in place, and persistent atrial fibrillation was admitted for congestive heart failure exacerbation. His hospital course was complicated by cardiogenic shock requiring Impella 5.5 placement. The Impella was repositioned four times during his hospital course. His preoperative TEE demonstrated LV EF < 20% and moderately to severely reduced RV function. He then presented to the operating room for orthotopic heart transplant 62 days following Impella placement. General anesthesia was induced and a TEE probe was placed. Aortic and bicaval cannulation was performed and the patient was placed on cardiopulmonary bypass. The aortic cross clamp was applied. The ascending aorta appeared stiff. An aortotomy was made and the entire aortic root and ascending aorta were thrombosed. The preoperative TEE was then reviewed and intramural clot was present in the ascending aorta prior to aortic cannulation, but had not been initially recognized. No dissection flap was visible in the aorta on preoperative TEE. The heart transplant was completed, and during aortic decannulation there was an injury to the right pulmonary artery which required emergent return to bypass for pledgeted suture repair. He was then weaned from cardiopulmonary bypass and decannulated. His ICD and Impella were removed. After weaning from cardiopulmonary bypass, TEE demonstrated an ascending aortic dissection distal to the anastomosis that extended to the descending aorta. Because there was no communication between the recipient's dissected aorta and the donor aorta that would allow the dissection to propagate proximally into the donor aortic root and coronary arteries, the dissection was medically managed. CTA of the chest and abdomen showed a dissection to the level of the renal arteries. There was preserved flow to the great vessels and abdominal arteries. The patient was extubated on postoperative day 1. He had no neurological deficits. He required intermittent hemodialysis likely due to a dynamic obstruction of his renal arteries. On postoperative day 5 he underwent balloon fenestration of the descending aortic dissection flap. The urine output immediately increased in the operating room. He was discharged home on postoperative day 18.

Discussion

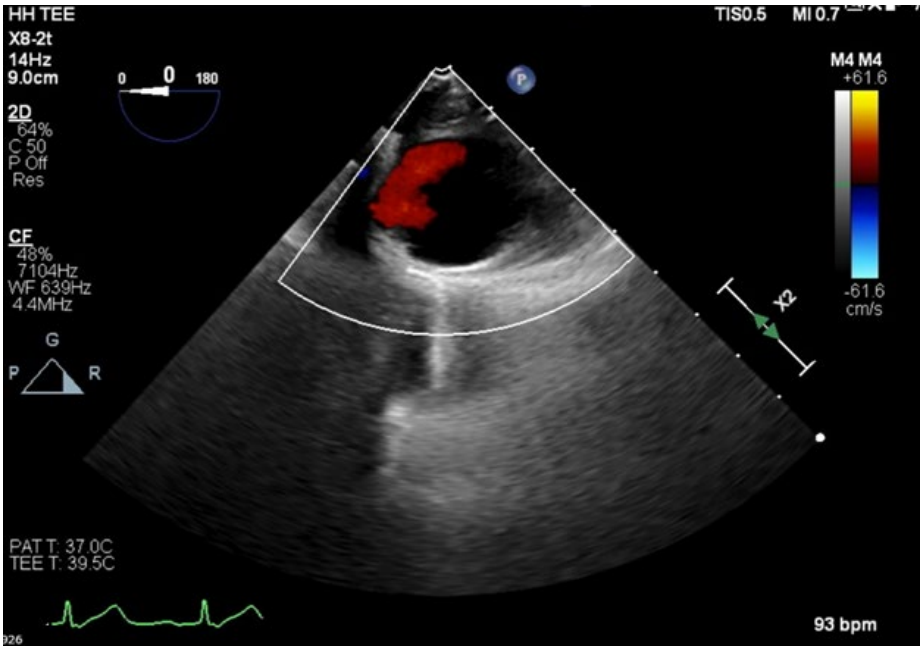
The Impella 5.5 is FDA approved "intended for short term (14 days) use". This time limit may be exceeded while patients await orthotopic heart transplant. While reported aortic dissection is rare, clinicians should

be vigilant about the possibility of aortic or vascular injury when a patient has an Impella device in place, especially if it has been repositioned multiple times or has been in place for a long duration.

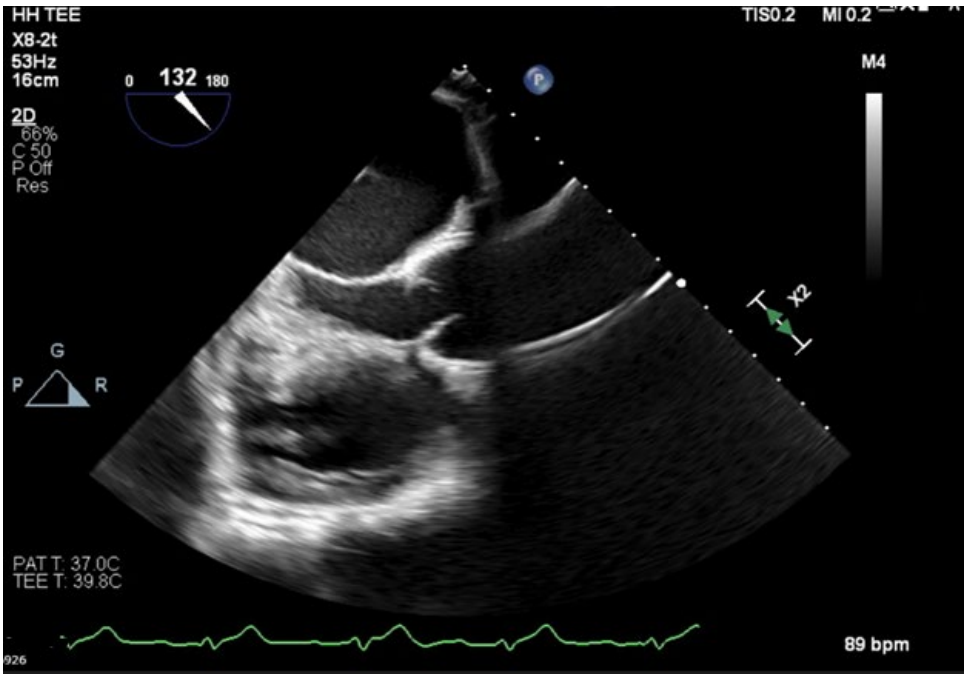
Images



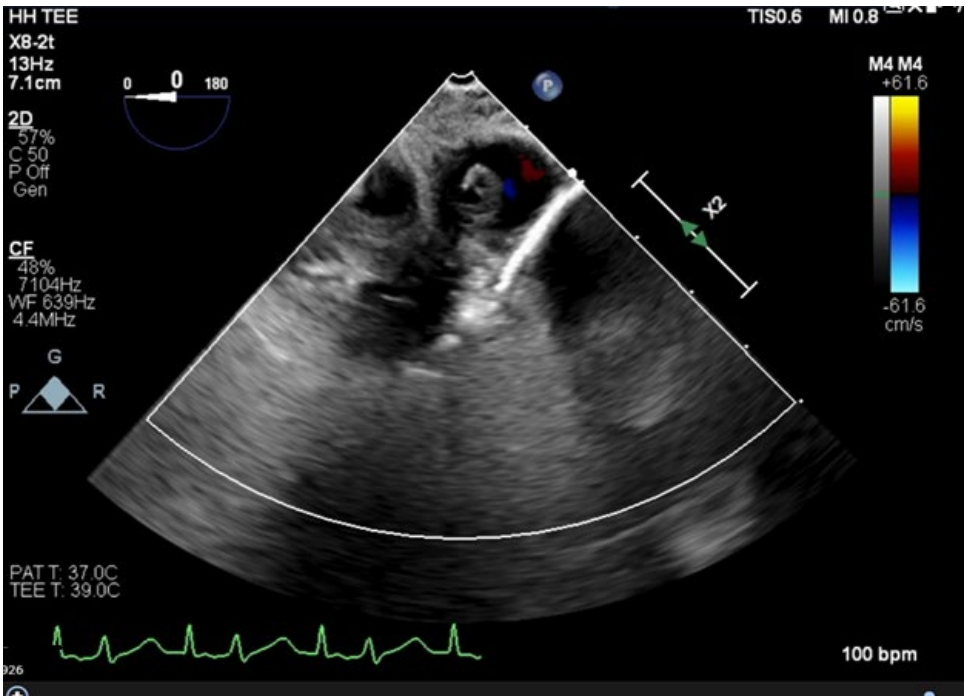
Ascending aorta, pre-operative



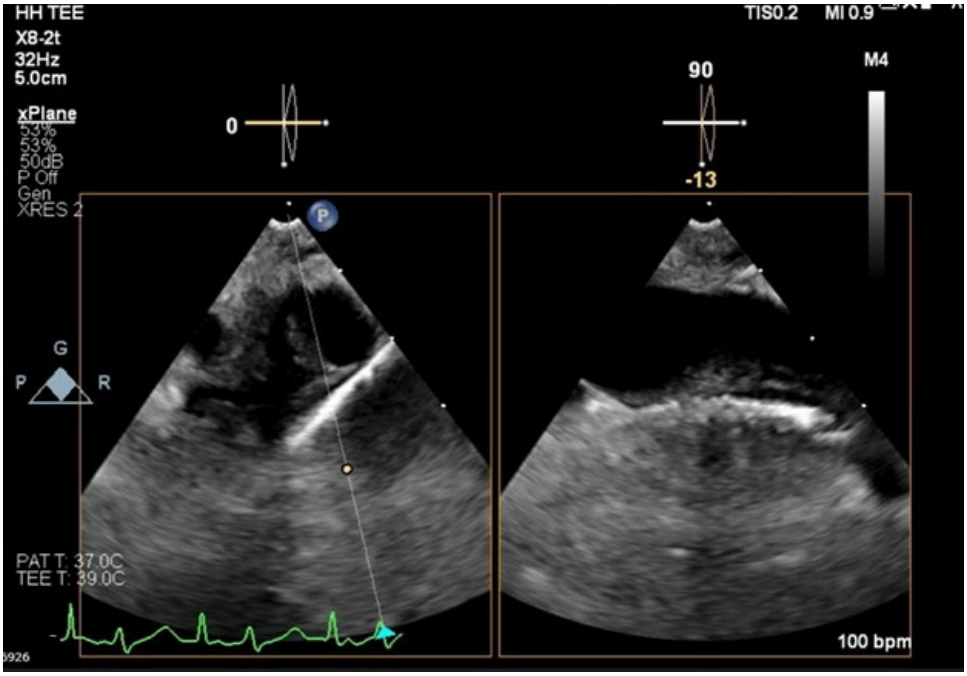
Descending aorta dissection, postoperative



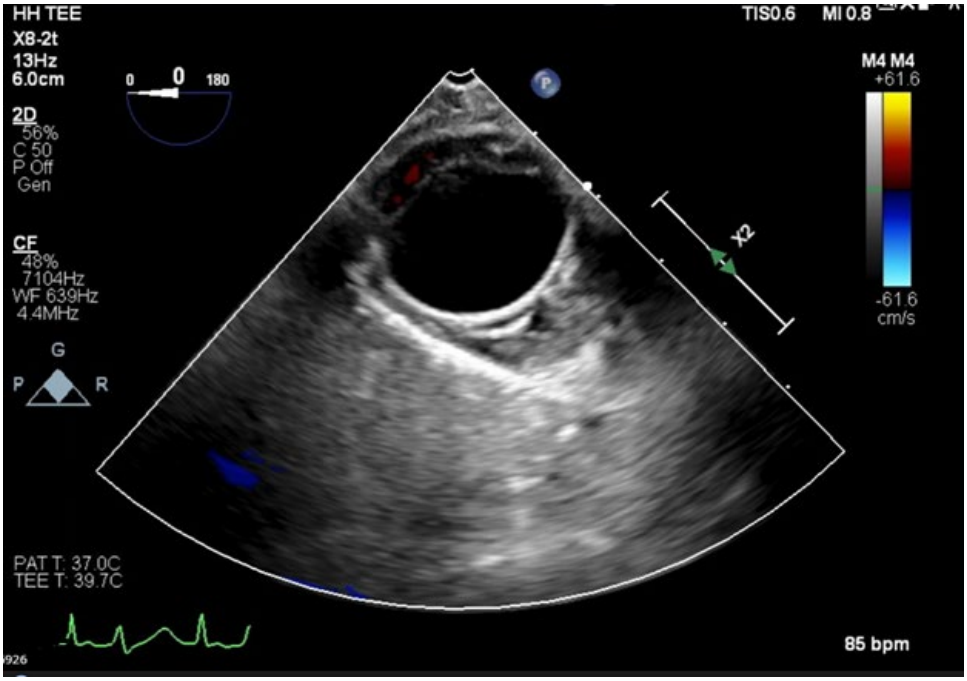
Ascending aorta, post-operative



Dissection, descending aorta



Dissection, distal arch



Dissection flap, descending aorta

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#FR 3 – Kiosk 8 – Poster 2

Hemopericardium and Hemothorax following Mitral Transcatheter Edge-to-Edge Repair

Presenting Author: Matthew A. Thimm, MD, Fellow, Columbia University Medical Center / NewYork-Presbyterian Hospital

Introduction

Mitral transcatheter edge-to-edge repair (mTEER) is a percutaneous procedure to treat mitral valve regurgitation, often utilized among patients at high surgical risk. The complication rate of mTEER is low (1). Here we describe a case complicated by a pericardial effusion requiring drainage and a hemothorax causing respiratory failure.

Case Presentation

The patient was a 56-year-old woman with a history of heart failure with reduced ejection fraction, high-degree atrioventricular block, and symptomatic severe MR who underwent mTEER. There was difficulty with transseptal puncture due to a short distance (septal height) between the optimal interatrial septal puncture site and mitral valve annulus. Following puncture, TEE identified a small pericardial effusion; she remained hemodynamically stable. The clip was deployed with improvement in her MR. Following, the effusion was enlarging with an associated increase in heart rate and pressor requirement (Fig 1). A pericardial drain was placed and 100 milliliters of blood was removed, resulting in improved hemodynamics. The effusion was attributed to a suspected right upper pulmonary vein puncture. Heparin was reversed and the procedure concluded uneventfully. Shortly after extubation, she developed tachypnea without hypoxia. Ultrasound demonstrated a large right-sided pleural effusion (Fig 2). She subsequently required non-invasive positive pressure ventilation. A chest tube was placed which immediately yielded 2 liters of blood. Fluoroscopy demonstrated re-expansion of the right lung, but she was intubated for hypercarbic respiratory failure. She received two units of packed red blood cells and was weaned off pressors. She remained stable and there was minimal further drain output. Given suspected low pressure venous injury and a history of anaphylaxis to contrast, no further intervention was performed. She was transferred to the ICU and was extubated on postoperative day 1. TTE showed a trace pericardial effusion with a small collection posterior to the left atrium and the pericardial drain was removed. The chest tube was removed on day 2. She was transferred to the floor on day 4.

Discussion

We report a case of hemopericardium and hemothorax during mTEER requiring pericardial and pleural drainage. Pericardial effusion is an uncommon complication of transseptal puncture that can quickly lead to tamponade (1). Hemothorax is a rare complication but can occur due to injury to the pericardium, pulmonary vein, or SVC/IVC (2). The suspected etiology of the effusions was injury to the right upper pulmonary vein following transseptal puncture. Patients with a short septal height are at higher risk of complications from transseptal puncture, as a more superior-posterior puncture is required to allow space to navigate the MitraClip in the LA (3). The right pulmonary vein ostia are superior and posterior to the fossa

ovalis, making inadvertent injury possible, especially when a more posterior puncture is required. Pulmonary vein injury in the pericardium, pleura, or lung parenchyma can lead to pericardial effusion, hemothorax, or pulmonary hemorrhage, respectively (2). To our knowledge, there are no other reported cases of both hemopericardium and hemothorax as a complication of mTEER.

Images

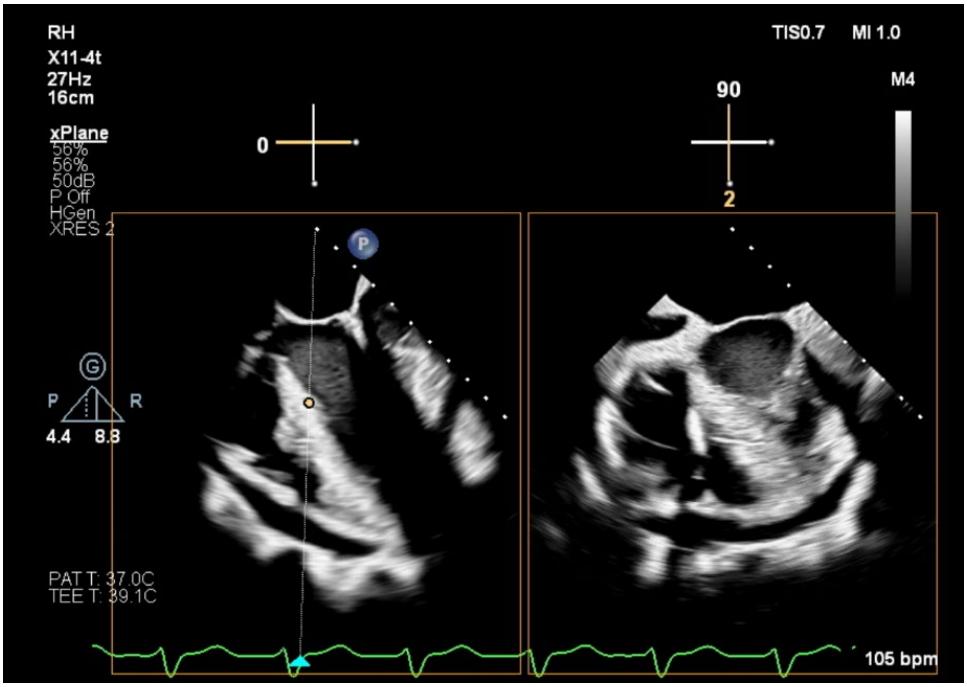


Figure 1: Circumferential pericardial effusion immediately prior to drainage.

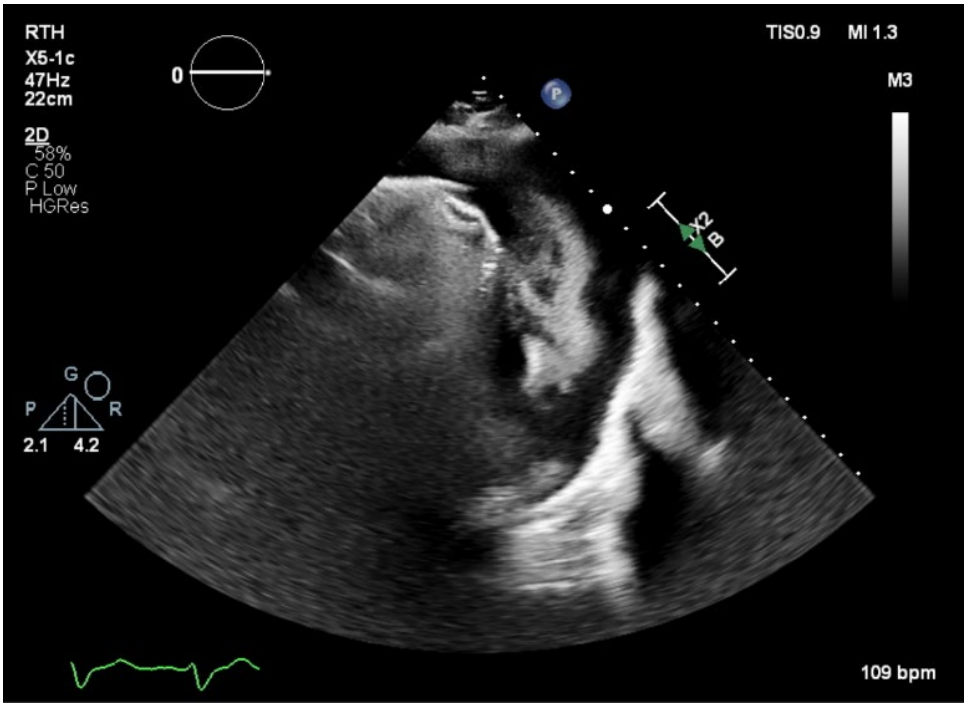


Figure 2: Point-of-care ultrasound image demonstrating a right pleural effusion with collapsed lung.

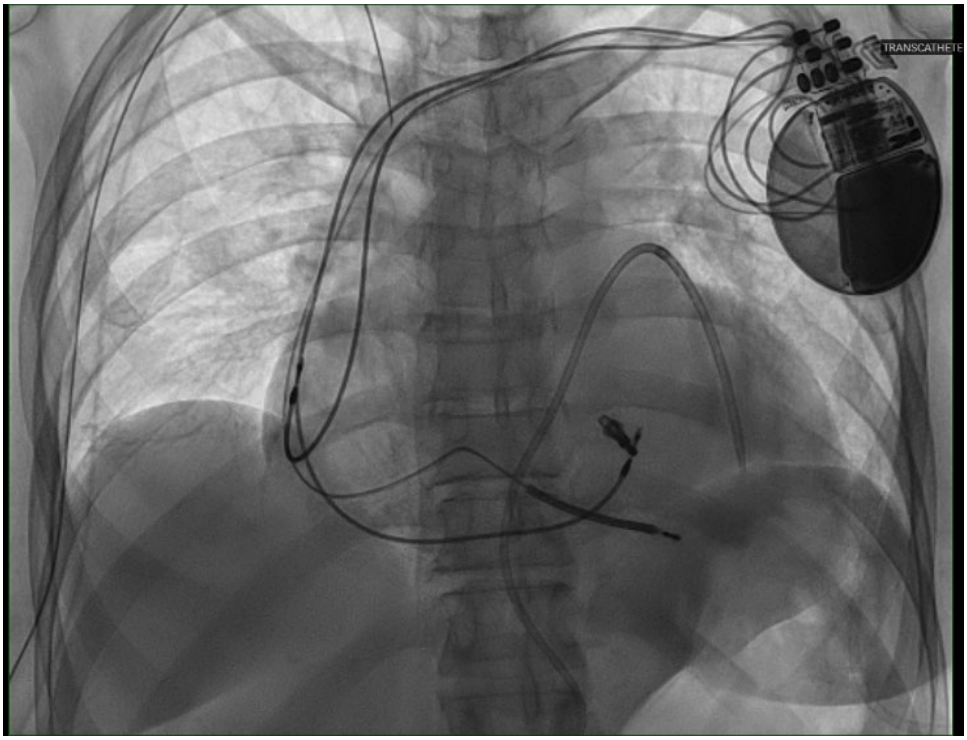


Figure 3: Fluoroscopic image demonstrating re-expansion of right lung after chest tube placement.

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#FR 3 – Kiosk 8 – Poster 3

Right heart failure after uncomplicated cesarean delivery in a patient with suspected hereditary pulmonary hypertension

Presenting Author: Ryan T. DeAngelis, MD, Adult Cardiothoracic Anesthesiology Fellow, Washington University in St. Louis

Introduction

When compared to idiopathic pulmonary hypertension, patients with hereditary pulmonary hypertension tend to present with manifestations of the disease at a younger age. Additionally, there is generally more significant hemodynamic compromise and a poorer response to treatment. This is a case of acute on chronic right heart failure in a patient with suspected hereditary pulmonary hypertension. Her mother and uncle both had the disease, with the latter testing positive for BMPR2. Patients with RV dysfunction tolerate a narrow range of optimal preload, and systemic hypotension may lead to reduced coronary perfusion and RV ischemia. Right heart failure may occur acutely with hemodynamic changes tolerable to a normal patient.

Case Presentation

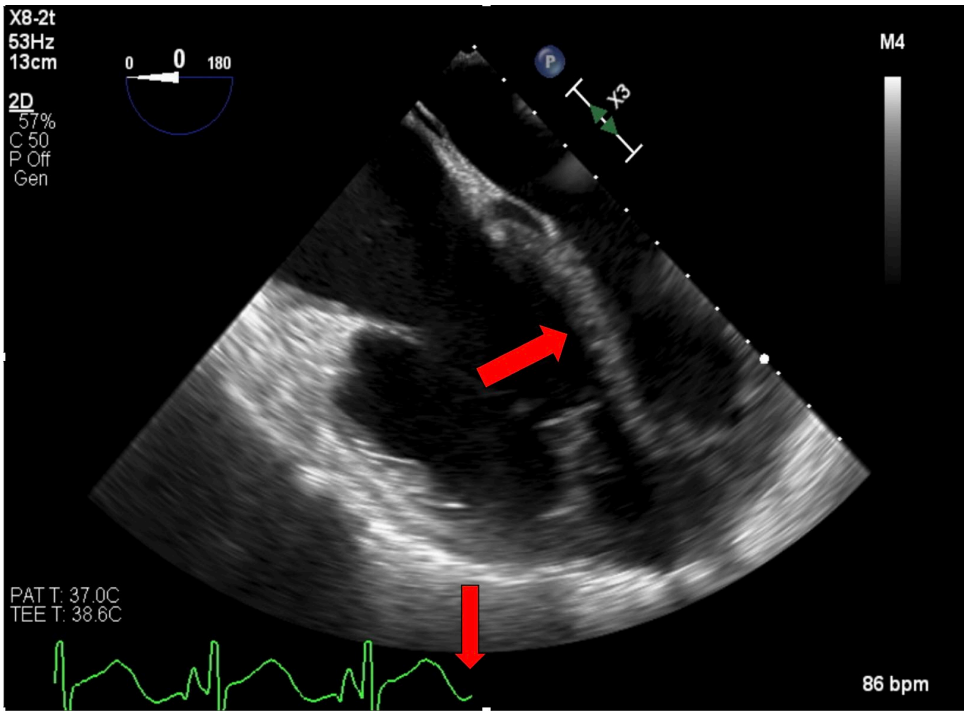
The patient is a 30 year-old G4P3003 female with a history of gestational hypertension and gestational diabetes mellitus who experienced progressively worsening dyspnea since 17 weeks gestation. At 33 weeks gestation she presented with severe dyspnea, orthopnea, and chest pain. She underwent TTE and RHC which showed severe right ventricular dilation, right ventricular hypokinesis, and a PAP of 115/55. She was admitted to the ICU, where she was given diuretics and systemic epoprostenol and sildenafil were initiated. Her PAP improved to 60s/40s. A cesarean section was planned for 37 weeks with cardiothoracic surgery on standby in the operating room. Both cardiothoracic and obstetric anesthesiologists were present. She underwent an uncomplicated cesarean delivery under a low dose combined spinal-epidural anesthetic. A pulmonary artery catheter was already in place and the ongoing intravenous epoprostenol infusion was continued throughout the procedure. Five minutes after initiation of oxytocin, the patient began to decompensate. Bradycardia and cardiac arrest soon followed for which CPR was initiated. The patient was promptly intubated and started on high dose infusions of epinephrine, vasopressin, and norepinephrine. A TEE probe was placed which showed severe dilation and akinesis of the right ventricle. ECMO was initiated 12 minutes after the onset of cardiac arrest. The patient was also started on inhaled nitric oxide. Subsequent TEE examination showed relative decompression of the right ventricle.

Discussion

There is added complexity in managing right heart failure in a postpartum patient with pulmonary hypertension. The patient had been medically optimized prior to the planned cesarean section. Her chronic right heart failure had been managed preoperatively with preload reduction and pulmonary circulation afterload reduction. Hypotension from the spinal anesthetic and oxytocin administration after delivery may have contributed to poor RV perfusion. Additionally, an autotransfusion of blood (300 - 500 mL) to the

maternal circulation from the contracting uterus as well as decreased IVC compression likely resulted in an acute increase in preload. In this patient population, planning for ECMO access is crucial. Furthermore, the literature suggests slow titration of a spinal anesthetic or using an epidural for more precise hemodynamic management. Lastly, patients with a diagnosis of pulmonary hypertension are often advised against becoming pregnant. A family history of pulmonary hypertension warrants further investigation.

Images



Bowing of the interventricular septum to the left during diastole

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#FR 3 – Kiosk 8 – Poster 4

Diffuse arterial and venous thromboses due to heparin-induced thrombocytopenia following cardiac surgery

Presenting Author: Carolyn Guan, MD, Brigham and Women's Hospital

Introduction

Heparin-induced thrombocytopenia (HIT) is a serious complication caused by antibody formation to the heparin-platelet factor 4 (PF4) complex, triggering platelet activation and a prothrombotic state. Up to 50% of affected patients develop thrombosis, most commonly deep venous thrombosis, pulmonary embolism, or skin necrosis, and the condition carries a mortality rate of up to 30% [1]. Patients undergoing cardiac surgery with cardiopulmonary bypass are exposed to high doses of heparin and are at especially high risk.

Case Presentation

A 70-year-old female with no history of heparin exposure or hypercoagulable disorders underwent endoscopic aortic valve replacement for severe aortic stenosis. She received 53,925 units of unfractionated heparin (UFH) intraoperatively followed by prophylactic low-molecular weight heparin (LMWH) daily. On postoperative day (POD) 6, she developed atrial fibrillation and was transitioned to therapeutic LMWH with initiation of warfarin. Her platelet count fell from 255 x 109 K/μL to 109 x 109 K/μL on POD 7, and she was discharged on POD 8. She re-presented on POD 9 with acute bilateral lower extremity pain, followed by dyspnea and complete loss of sensory and motor function below her groin. Labs revealed a platelet count of 26 x 109K/μL. CT angiography demonstrated extensive thrombus involving the distal abdominal aorta, bilateral common iliac arteries, both atria, and the right main, right lobar, and left lobar pulmonary arteries (Figures 1-3). She was started on a bivalirudin infusion. Follow-up labs showed hemoglobin 9.6 g/dL, platelets 20 x 109 K/μL, INR 6.3, PTT >150 seconds, D-dimer > 7650 ng/mL, fibrinogen 188 mg/dL, and a positive heparin-PF4 assay at 37.72 U/mL. Following a multidisciplinary goals-of-care discussion, the patient elected to pursue comfort measures and was discharged to hospice.

Discussion

HIT occurs in up to 3% of cardiac surgery patients and significantly increases the risk of limb ischemia, stroke, and mortality [1]. UFH carries up to a tenfold higher risk of HIT compared to LMWH, and this patient received both, prolonging exposure [2]. A platelet count drop of >50% occurred between POD 6 and 7, with

no labs checked on POD 8 prior to discharge. The timing, degree of thrombocytopenia, recent heparin exposure, absence of alternative explanations, and subsequent thromboses yielded a high 4T score [3]. In addition, warfarin was initiated during thrombocytopenia, which is contraindicated in acute HIT because it depletes protein C and can worsen thrombosis [3]. This case underscores that thrombosis may be the first sign of HIT, even with only moderate thrombocytopenia. Platelets should be monitored from POD 4 to 14, and a > 50% drop from peak should trigger early assessment using the 4T score and further testing. When HIT is suspected, all heparin products should be discontinued and non-heparin anticoagulation started. Warfarin should be deferred until platelet recovery and administered in conjunction with a non-heparin anticoagulant. In postoperative cardiac patients, new thromboembolism should prompt evaluation for HIT. Early recognition and intervention are essential to prevent irreversible thrombotic complications.

Images



Figure 1: Acute thrombus extending from the distal abdominal aorta into the bilateral common iliac arteries with near-total occlusion



Figure 2: Acute thrombus in right atrium and pulmonary embolism involving the right main, right lobar, and left lobar pulmonary arteries



Figure 3: Acute thrombus in left atrium

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#FR 3 – Kiosk 8 – Poster 5

Renal Cell Carcinoma in A Patient with A Solitary Kidney with Embolization of Inferior Vena Cava Tumor Thrombus to The Right Atrium

Presenting Author: Rachel Reindorf, MD, Fellow, Johns Hopkins Hospital

Introduction

Renal cell carcinoma (RCC) accounts for approximately 3% of all cancers. Between 4% and 10% of RCC cases are associated with tumor thrombus, which can extend from the renal vein into the inferior vena cava (IVC), and in rare instances, reach the heart. Current evidence only shows benefit from immunotherapy and tyrosine kinase inhibitors in managing tumor thrombus. Additionally, guidelines remain unclear regarding the role of anticoagulation in RCC patients (Williams & Myint, 2023). Tumor migration during radical nephrectomy has been reported, likely due to surgical manipulation such as clamping and unclamping of the IVC, which may increase the risk of embolization. Extension of tumor thrombus into the heart is extremely rare, occurring in approximately 1% of cases(Hatzakorzian et al., 2023).

Case Presentation

65-year-old female with a history of hyperparathyroidism, chronic kidney disease stage 4-5, right robotic nephrectomy (2015) and pulmonary nodules with associated dyspnea on exertion. Our patient was found to have a 7cm mass on her left kidney and a level 2 IVC thrombus. The patient was scheduled for a Left radical nephrectomy, IVC tumor thrombectomy, and reconstruction. Induction and intubation were uneventful. Additional peripheral intravenous access was obtained. IVC thrombectomy was started. During dissection, the surgeon noticed that the tumor burden decreased and the patient became hemodynamically unstable. Transesophageal echo probe was inserted and a 4.9cm x 2.1cm tumor was visualized in the right atrium abutting the tricuspid valve. Due to concern for embolization and hemodynamic instability, the decision was made to contact cardiac surgery to place the patient on VA ECMO. 30,000 units of heparin were administered for cardiopulmonary bypass. Right atriotomy was performed. The thrombus was identified at the cavo-atrial junction and able to be resected in one piece. Following closure of the right atrium, TEE confirmed the absence of residual tumor/thrombus in the IVC. Given complete removal of thrombus and hemodynamic stability, the patient was decannulated from ECMO and transported to ICU. She was extubated on POD 1 and discharged home on POD 7.

Discussion

Managing patients with renal cell carcinoma (RCC) presents significant challenges. While tumor thrombus associated with RCC is well-documented, there are currently no established guidelines for anesthetic management in these complex cases. Kostibas et al. reported that transesophageal echocardiography (TEE) provided new diagnostic insights in 58% of patients with level II thrombus undergoing nephrectomy, with its utility increasing as thrombus level advanced (Kostibas et al., 2017) . This case underscores the critical role of TEE in non-cardiac surgeries, offering valuable information that supports both anesthetic and surgical decision-making.

Images

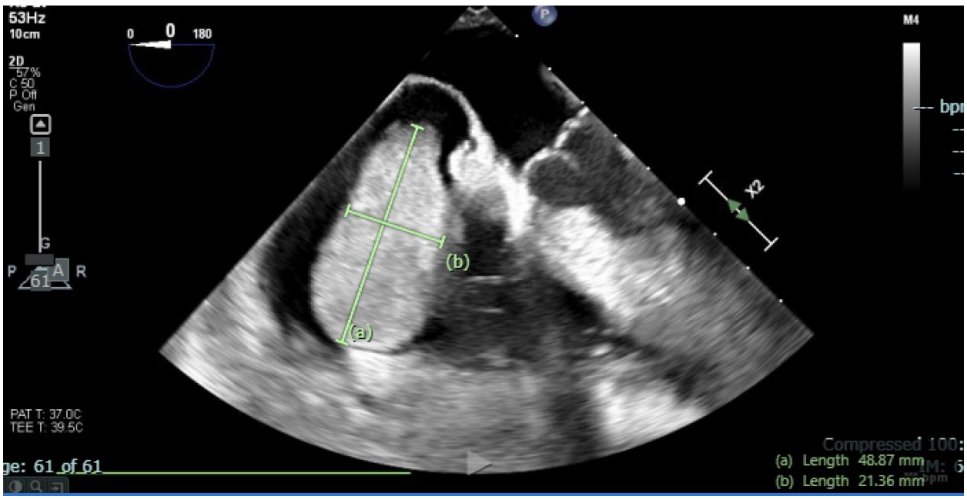


Fig 1. Tumor thrombus in right atrium

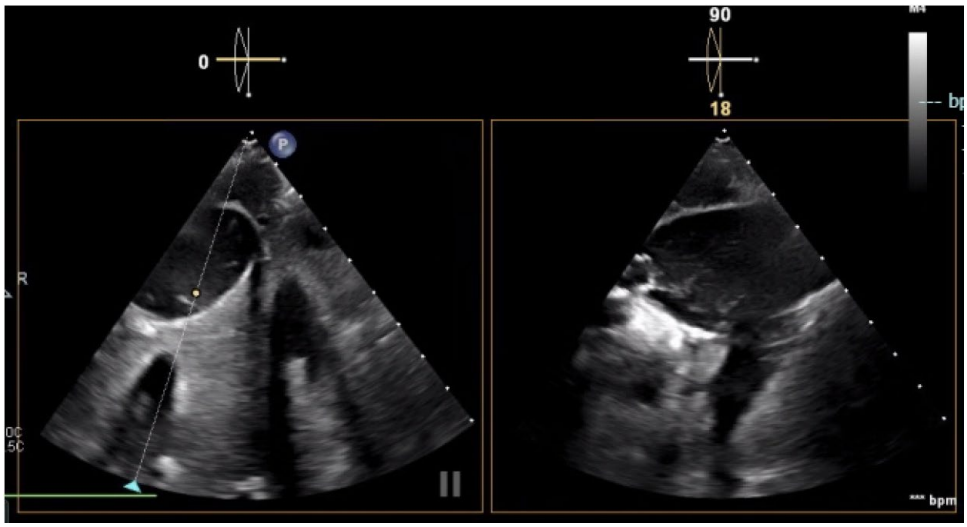


Fig 2. No residual tumor thrombus in IVC

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#FR 3 – Kiosk 8 – Poster 6

Urgent Alcohol Septal Ablation for Refractory Shock and Hypoxemic Failure in the Setting of Combined Takotsubo and Hypertrophic Cardiomyopathy

Presenting Author: William Ryan, MD, Anesthesia Resident, Hospital of University of Pennsylvania

Introduction

Takotsubo Cardiomyopathy (TCM) infrequently presents with refractory cardiogenic shock, and its concurrence with hypertrophic obstructive cardiomyopathy (HOCM) is rare. In such cases, basal hypercontractility in TCM may unmask or worsen a pre-existing, often silent, HOCM pathology, causing severe left ventricular outlet tract (LVOT) obstruction and acute mitral regurgitation (MR). We here detail the case of a hemodynamic collapse driven by this combined pathology, in which urgent alcohol septal ablation (ASA) was required to relieve obstruction and enabled weaning VV ECMO.

Case Presentation

A 61-year-old female with a history of hyperlipidemia presented after syncopizing while giving a eulogy. She was found to be in cardiogenic shock and acute hypoxemic respiratory failure and was transferred to our facility. Left heart catheterization at the referring hospital excluded significant coronary artery disease and placed an intra-aortic balloon bump (IABP). On arrival, the patient was in shock requiring maximal vasopressor support (Epinephrine 4µg/min, Norepinephrine 15–24µg/min, Phenylephrine 0–100µg/min, Vasopressin 0.04U/min) and an IABP. Given severe hypoxemia secondary to flash pulmonary edema, a decision was made to prioritize respiratory support by initiating VV ECMO and discontinuing the IABP, which was exacerbating her condition. Initial TEE established a dual pathology: LVEF was 20-25% with regional wall motion consistent with TCM, characterized by hyperkinesis of the basal walls and hypokinesis of the mid and apical walls. This acute dysfunction was superimposed on severe basal septal hypertrophy (1.8cm), which, in the setting of TCM hypercontractility, resulted in severe Systolic Anterior Motion (SAM). This resulted in LVOT obstruction with a peak gradient of 95mmHg and moderate-to-severe MR. Post-VV ECMO cannulation, the LVOT gradient dropped to ~30mmHg on adjusted pressor doses (Norepinephrine 10µg/min, Phenylephrine 100µg/min). On Hospital Day 4, follow-up TTE demonstrated worsened obstruction, with an LVOT peak gradient of 60mmHg at rest and moderate MR. Despite VV ECMO, persistent obstruction led to recurrent flash pulmonary edema and prohibited any attempts to wean sedation or ECMO. On hospital day 5, given the failure of medical and mechanical measures, an urgent ASA of the first septal perforator was performed. This intervention achieved immediate resolution of the obstruction: The LVOT gradient fell to <10 mm Hg on cardiac catheterization and 18 mm Hg on TEE. Post-ablation TTE the next day demonstrated no SAM or LVOT outflow obstruction (peak gradient 7mmHg at rest) and only mild MR. These structural improvements enabled rapid hemodynamic recovery, with LVEF improving to 35-40% and successful weaning of VV ECMO and vasopressors.

Discussion

This case highlights the hemodynamic interplay between TCM and pre-existing basal septal hypertrophy, in

which transient basal hypercontractility can transform a benign anatomical variant into a dynamic LVOT obstruction with catastrophic consequences. Though ASA is traditionally performed electively, it can serve as a definitive, life-saving intervention when performed emergently in select patients with refractory mechanical obstruction.

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#FR 3 – Kiosk 9 – Poster 1

Coronary Artery to Pulmonary Artery Fistula: Significant Incidental Finding or Echocardiographic Illusion?

Presenting Author: Sean Padungtin, MD, Fellow, Wake Forest Baptist University

Introduction

Coronary Artery Fistulas (CAFs) are rare cardiac anomalies, the vast majority of which are congenital. They account for 0.2%-0.4% of congenital cardiac anomalies with a prevalence estimated to be 0.002% of the general population. (2). We present a case of a patient presenting for off-pump CABG via thoracotomy with TEE concerning for a new diagnosis of a fistula between the LAD and PA. This case highlights the investigation of imaging artifacts as well as potential methods to clarify this diagnosis.

Case Presentation

A 68-year-old male with history of HFmrEF and COPD presented for single vessel MIDCAB via left thoracotomy for LIMA to LAD graft. Preoperative workup revealed NSTEMI and LHC demonstrating a 90% ostial and 50% mid LAD lesion without other anomalies. Intraoperatively, TEE revealed LVEF of 38% normal RV function, no significant valvular abnormalities, and a color Doppler jet originating below the left main and appearing to move toward the main PA (Figure 1). CW Doppler showed flow throughout the cardiac cycle, but primarily during diastole. The surgical team was alerted regarding a possible coronary fistula vs. artifact. A PA catheter was not originally planned but was floated to facilitate diagnosis, which demonstrated oxygen saturations and pressures of 70.5% (23mmHg), 79.4% (58/23mmHg), and 80.2% (60s/20s mmHg) in the RA, RV, and PA, respectively. Given the lack of a significant increase in oxygenation and pressure between the RV and PA, the jet was diagnosed as artifact and no further intervention was performed.

Discussion

Coronary to pulmonary fistulas are rare. On echocardiography, they manifest as continuous turbulent diastolic flow on Doppler interrogation, and can be associated with chamber dilation. They originate most commonly from the left main/LAD. The majority terminate in the main pulmonary trunk (89%). They are generally diagnosed on coronary angiography, but are increasingly diagnosed on CT angiography (2). Pulmonary artery oximetry is not a common method of diagnosis (3), but was used in this case given the lack of access to the other modalities since the surgery had already started. In this case, the color jet looked convincing and was reviewed by multiple echocardiographers. Given the surgical procedure, surgical inspection would have required a significant change to the operation. In order to validate the TEE findings, a PA catheter was used to obtain measurements as in a right heart catheterization. A LAD to PA fistula (left to right shunt) would be expected to manifest with higher PA oxygenation saturation as well as higher pressures compared to the RV. The lack of confirmation suggested the jet was artifact. Furthermore, the lack of flow acceleration of the blood immediately around the jet argued against a true shunt. The dilemma of a true finding vs artifact raises the risk of leaving a serious problem uncorrected during cardiac surgery versus unnecessarily performing a more complex procedure. Ultimately, the investigation carried out in the OR

ensured the patient received the proper care needed without subjecting the patient to unwarranted risk.

Images

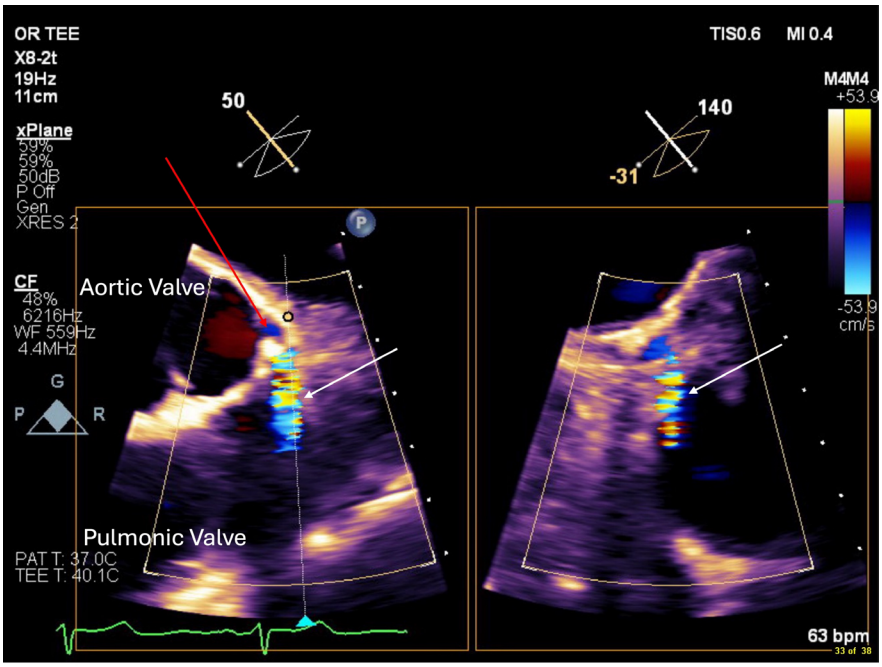


Figure 1: Zoomed TEE view obtained from RV inflow-outflow view with biplane imaging showing a high velocity jet (white arrows) on color flow Doppler originating below the left main coronary artery (red arrow) which appears to move into the main pulmonary artery

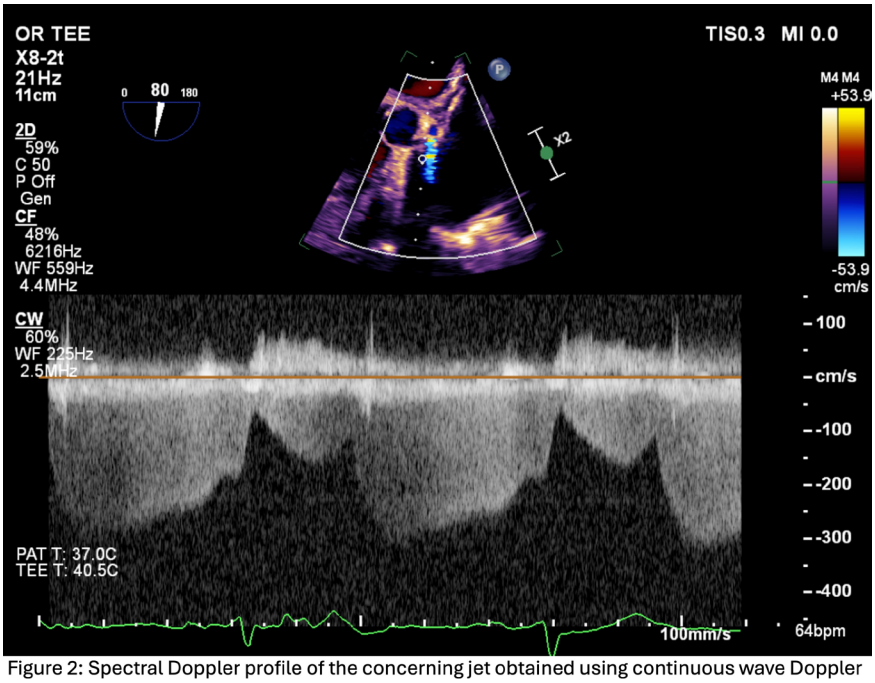


Figure 2: Spectral Doppler profile of the concerning jet obtained using continuous wave Doppler showing flow throughout the cardiac cycle but predominantly in diastole.

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#FR 3 – Kiosk 9 – Poster 2

Anesthetic Management of a Primary Pulmonary Artery Sarcoma Presenting as Chronic Thromboembolic Disease

Presenting Author: Rachel Rios, MD, Resident Physician, Vanderbilt University Medical Center

Introduction

Pulmonary endarterectomy is the surgical treatment for chronic thromboembolic pulmonary hypertension (CTEPH) with a mortality rate < 5% at experienced centers. Appropriate candidates include those with proximal disease (main, lobar, or segmental arteries). These patients may present with significant ventilation-perfusion mismatch causing hypoxemia and right heart failure, both of which are further worsened by anesthetics. Despite most CTEPH patients presenting with thromboembolic disease, very rare subsets may present with primary sarcomas that clinically present in a similar fashion.

Case Presentation

67-year-old female with chronic respiratory failure (2 L/min home oxygen), asthma, hypertension, and former tobacco use was referred for pulmonary thromboembolectomy (PTE) for suspected pulmonary artery sarcoma. Computed tomography (CT) scan showed bilateral chronic emboli with left mural thrombus narrowing the lumen (Figure 1); Ventilation-perfusion (V/Q) scan showed large left-sided mismatch. Positron emission tomography (PET) scan showed unusual uptake in the pulmonary arteries (Figure 2). Bilateral groins were wired pre-induction for emergent cardiopulmonary bypass access. Following arterial line placement, induction included inhaled epoprostenol (for pulmonary vasodilation), etomidate (to maintain systemic vascular resistance), fentanyl (to blunt sympathetic surge), and rocuronium. Hyperventilation with shallow tidal volumes was used for pulmonary hypertension precautions. Pulmonary arterial pressures were 106/20 (mean 50 mmHg) after pulmonary artery (PA) catheter placement. Three periods of deep hypothermic circulatory arrest (DHCA) were required to evacuate mixed clot and tumor (Figure 3). Serial bronchoscopy removed scant bloody exudate. Reperfusion was well tolerated with epoprostenol and dobutamine for right ventricular (RV) support. Pathology confirmed a primary PA spindle cell sarcoma. Transesophageal echocardiography showed improved RV function from moderate to mild dysfunction with reduced dilation. Tricuspid regurgitation remained insignificant. Extubated on post-operative day 3.

Discussion

PA sarcoma is a solid tumor that originates from the endothelial cells of the blood or lymphatic vessels in the lung with a 12-month survival. Primary disease has an incidence of 0.001 to 0.030% typically in middle-aged men. CT findings include filling defects in pulmonary arteries, pleural effusions, and a heterogenous appearance compared to more homogenous appearance of thrombotic lesions. These patients are often anticoagulated without interval resolution of symptoms or improvement in clot burden. PET scans are critical for preliminary diagnosis as high metabolic uptake would strongly favor malignancy over metabolically inert chronic thrombi. Anesthetic considerations should include all existing recommendations for PTEs such as pulmonary hypertension precautions, neuroprotective strategies during circulatory arrest,

inotropic support for right heart failure, and bronchoscopic evaluation for sequelae from reperfusion lung injury. Reperfusion bleeding and pulmonary edema may be profound and promptly evaluated with bronchoscopy and surgical intervention. PA catheter placement or manipulation may be ill-advised due to risk of embolization.

Images



Figure 1: Computed Tomography Scan Demonstrating Clot and Tumor Burden

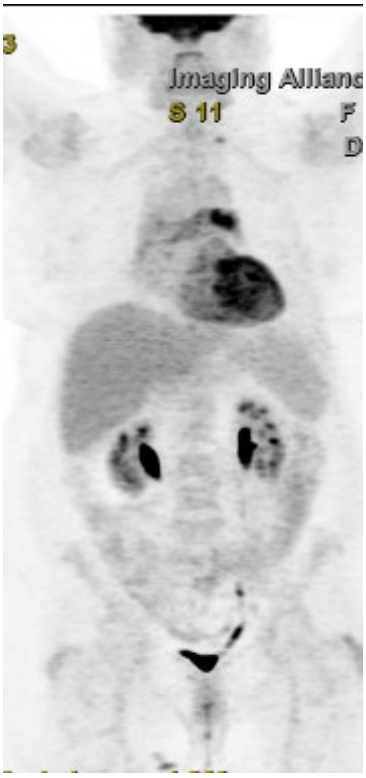


Figure 2: Whole Body Positron Emission Tomography Scan



Figure 3: Surgical Evacuation of Clot and Tumor Burden

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#FR 3 – Kiosk 9 – Poster 3

Percutaneous Mitral Valve Replacement Complicated By Severe LVOT Obstruction Due To Anterior Mitral Leaflet Displacement

Presenting Author: Vivian Hernandez Torres, MD, MS, Northwell Health

Introduction

Left ventricular outflow tract (LVOT) obstruction is a rare but serious complication of transcutaneous mitral valve replacement (TMVR). It typically results from systolic anterior motion or mechanical displacement of the anterior mitral leaflet (AML) toward the septum, creating a dynamic gradient that may lead to hemodynamic instability. Early identification of this complication with intraoperative echocardiography is crucial, as prompt management is required to prevent rapid deterioration. We present a case of severe LVOT obstruction following TMVR caused by AML displacement, ultimately requiring redo open-heart surgery for definitive treatment.

Case Presentation

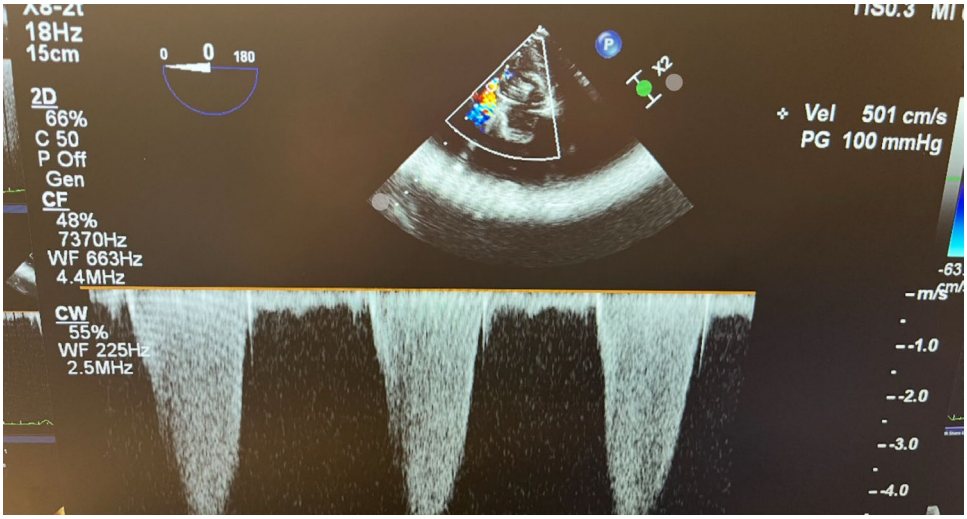
An 82 year old man with a past medical history significant for HFpEF, hypertension, hyperlipidemia, coronary artery disease status CABG ×2, prior mitral valve repair with a 30-mm annuloplasty ring, and lung cancer status post lobectomy presented with progressive fatigue and dyspnea on exertion. Transesophageal echocardiography demonstrated severe recurrent mitral regurgitation across the prior repair, and coronary angiography revealed no intervenable lesions. Given his symptoms and prior surgical history, he underwent trans-septal TMVR with implantation of a 29-mm Sapien valve. Immediately after valve deployment, the patient developed profound hemodynamic instability with evidence of severe LVOT obstruction. Intraoperative TEE demonstrated a peak gradient of 1000 mmHg caused by displacement of the remnant anterior mitral leaflet into the LVOT, producing a functional obstruction. Initial measures including volume resuscitation, afterload augmentation, and beta blockade resulted in only minimal improvement in the gradient. Postoperative TTE showed a newly reduced ejection fraction of 20–25% and persistent severe LVOT obstruction. Because the mechanism of obstruction was mechanical and related to the AML being pushed into the outflow tract by the newly implanted prosthetic valve, medical therapy alone was insufficient. The patient was therefore recommended for redo open-heart surgery to relieve the obstruction by addressing the obstructing leaflet and restoring unobstructed LV outflow.

Discussion

Although LVOT obstruction after TMVR is uncommon, it is one of the most feared complications due to its potential to cause rapid cardiovascular collapse. The pathophysiology often involves narrowing of the LVOT due to the interaction between the prosthetic valve and the native AML, particularly in patients with prior mitral repairs, smaller LV cavities, or unfavorable leaflet anatomy. Dynamic obstruction may initially respond to standard measures such as preload augmentation, increased afterload, and reduction of contractility. However, when the obstruction is fixed or mechanicalcaused by actual displacement of the AML into the LVOT medical optimization is rarely sufficient, and definitive surgical intervention becomes

necessary. This case highlights the importance of careful pre procedural imaging, detailed assessment of the predicted LVOT area, and immediate echocardiographic evaluation when hemodynamic instability arises after TMVR. Prompt recognition of the mechanism of obstruction allows early transition from medical stabilization to surgical management, which is essential for improving outcomes.

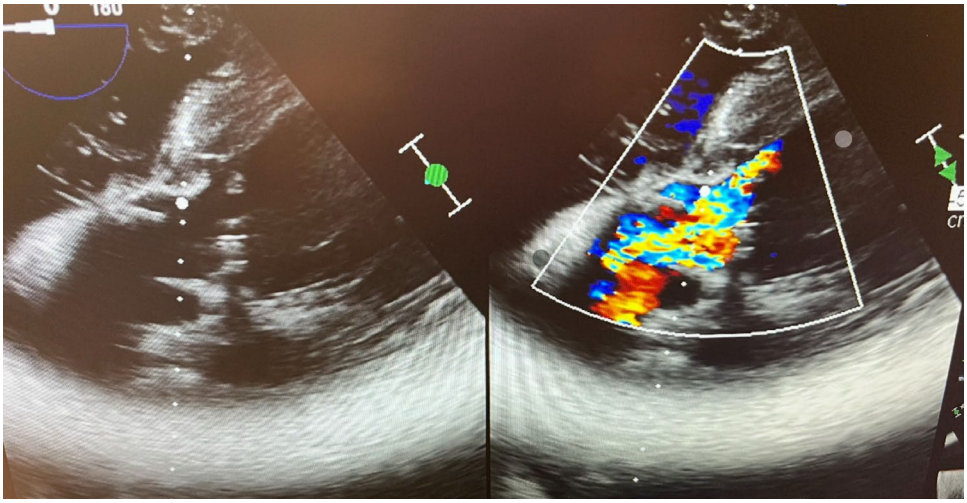
Images



Deep transgastric view with LVOT gradient of 100mmHg



Mid Esophageal long axis view demonstrating percutaneous MVR pushing native AML into LVOT creating mechanical obstruction



Deep transgastric view with very turbulent flow through LVOT creating mechanical obstruction

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#FR 3 – Kiosk 9 – Poster 4

Anomalous Right Coronary Artery Complicating Aortic Valve Replacement Bicuspid Aortic Valve Disease: The Critical Role of Coronary Inspection

Presenting Author: Joshua Ogogo, MD, South Shore University Hospital at Northwell Health

Introduction

Coronary artery anomalies occur in < 1% of the population and are usually benign, but certain variants can cause myocardial malperfusion and increase perioperative risk during aortic valve surgery. This is particularly relevant in patients with bicuspid aortic valve disease, who may have coexisting anomalous coronary anatomy. Although echocardiography can identify suspected anomalies, preoperative imaging may be incomplete, making careful intraoperative inspection essential. Given that anomalous aortic origin of the coronary arteries (AOCA) occurs in ~0.2% of angiographic studies, individualized preoperative planning with the surgical team is critical to prevent perioperative ischemia.

Case Presentation

A 50-year-old woman with insulin-dependent diabetes, hypertension, hyperlipidemia, and a known bicuspid aortic valve presented with intermittent chest discomfort and dizziness. Echocardiography demonstrated severe aortic stenosis (Vmax 6.03 m/s, mean gradient 100.6 mmHg, AVA 0.36 cm^2) with preserved ventricular function. Coronary CTA showed mild non-obstructive disease; catheterization revealed an anomalous RCA that could not be selectively engaged. She proceeded to surgical aortic valve replacement. Following initial separation from cardiopulmonary bypass, she developed abrupt hemodynamic collapse after chest closure, resolving upon reopening. A second closure attempt resulted in recurrent right ventricular failure with rising CVP and poor LV filling, prompting return to bypass. Concern for ischemia of the anomalous RCA led to saphenous vein bypass grafting, after which she weaned from bypass uneventfully. Final chest closure was successful. In the CTICU, she was extubated early, required brief inotropic support, and experienced short-lived atrial fibrillation treated with amiodarone. She recovered without further complications and was discharged home on postoperative day five.

Discussion

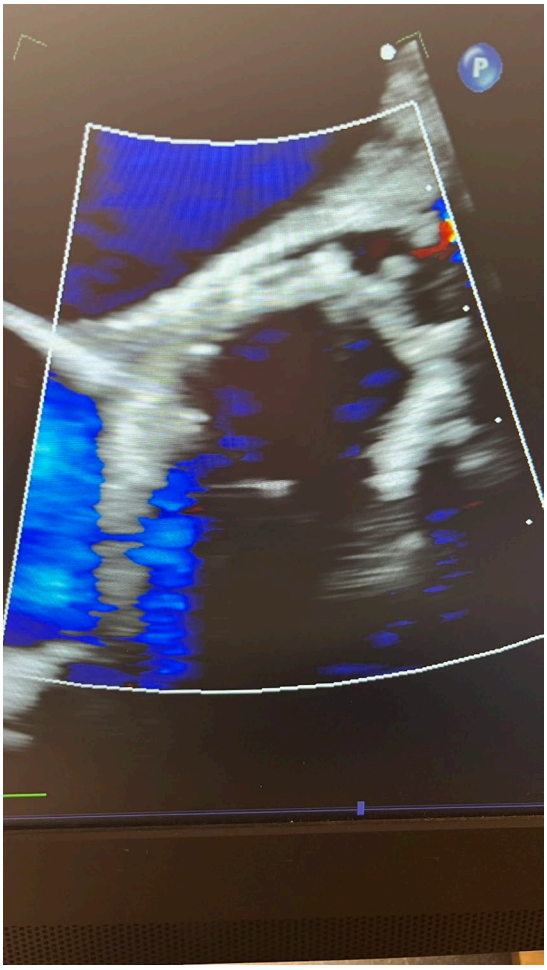
Advances in cardiovascular imaging have increased the detection of anomalous aortic origin of the coronary arteries (AAOCA) in adults. Although AAOCA can present with chest pain, syncope, arrhythmias, or even sudden cardiac death, many cases are found incidentally. Because anatomic variations are wide and symptoms vary, management must be individualized rather than strictly guideline-driven. Imaging is essential for diagnosis and risk assessment. Cardiac CTA offers excellent spatial resolution for defining coronary origin, course, intramural segments, and associated atherosclerosis. Cardiac MRI provides high-quality imaging without radiation or contrast exposure. Coronary angiography—via catheterization, CT, or MRI—is recommended by AHA/ACC and ESC guidelines when AAOCA is suspected or ischemia is present. Surgical options include unroofing of intramural segments, translocation and reimplantation of the anomalous coronary artery, and coronary artery bypass grafting (CABG). CABG may require proximal ligation

to prevent competitive native flow, while unroofing and reimplantation aim to restore normal coronary anatomy and physiology. Because each approach has distinct risks and benefits, surgery must be tailored to the patient's specific anatomy. Overall, optimal AAOCA management relies on detailed imaging and individualized surgical planning to reduce risk and improve outcomes.

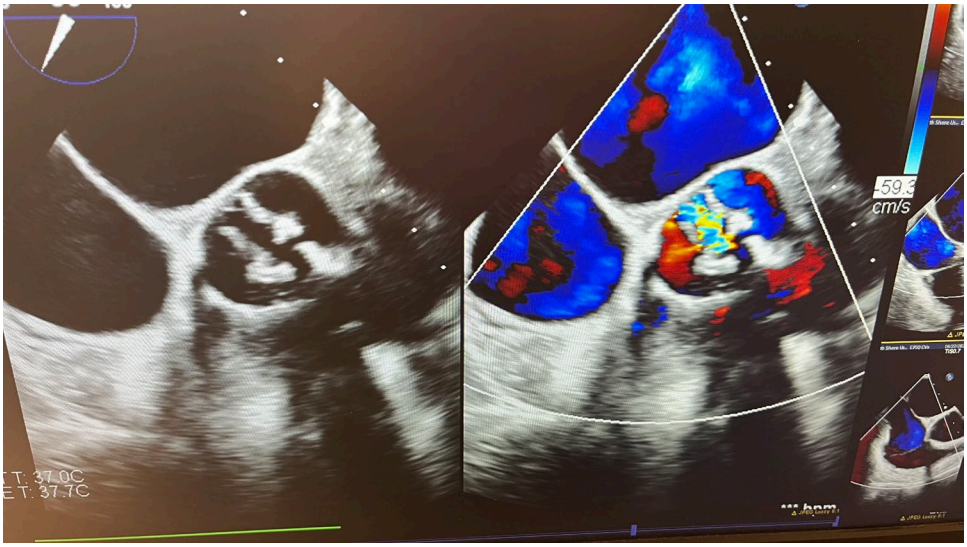
Images



Bicuspid AV Short Axis with Annomalous RCA Take Off



Bicuspid AV Short Axis with Annomalous RCA Take Off



Bicuspid AV Short Axis with Annomalous RCA Take Off

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#FR 3 – Kiosk 9 – Poster 5

Prevention of Neurological Complications from Rapid Sodium Correction During Massive Transfusion of a Hyponatremic Patient Undergoing Bilateral Lung Transplantation

Presenting Author: Siddak Kanwar, MD, Fellow, Northwestern Hospital

Introduction

Bilateral lung transplantation poses significant metabolic and coagulation challenges, especially in patients on ECMO. Hyponatremia increases the risk of osmotic demyelination if corrected rapidly[1]. Lung transplantation often requires substantial transfusion and hemodynamic resuscitation. Patients on ECMO are predisposed to coagulopathy and platelet dysfunction, driven by thrombocytopenia and impaired platelet aggregation during ECMO support[3]. We present an intraoperative strategy to prevent rapid sodium correction during massive transfusion in a hyponatremic patient undergoing lung transplantation.

Case Presentation

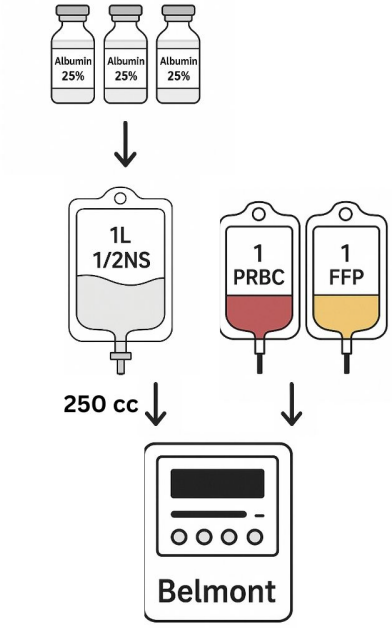
A 59-year-old male with bleomycin-induced ILD developed progressive respiratory failure, prompting VV ECMO cannulation. During cannulation, he became pulseless with VT, necessitating conversion to VAV ECMO. Preoperative serum sodium was 125 mmol/L, and urine studies were consistent with SIADH, likely triggered by severe lung pathology and critical illness, both of which promote non-osmotic ADH release. An additional contributor to hyponatremia included volume overload from heart failure, as evidenced by a dilated right ventricle with moderately reduced function. Preoperative laboratory values showed a hemoglobin of 7.1 g/dL and platelet count of 61. Risk of coagulopathy was high due to ECMO-related platelet consumption, chronic heparin exposure, critical illness, and high surgical blood loss. The goal was to mitigate rapid sodium correction from high-sodium products such as FFP, which contains an average sodium concentration of 172 mEq/L. We administered 250 mL of a low-sodium carrier solution consisting of 3 × 50 mL vials of 25% albumin diluted in 1 L of 0.45% NS for every unit of PRBC and FFP transfused in a 1:1 ratio. This was easily achieved by mixing the carrier with blood products in the Belmont rapid transfuser (Figure 1). The patient received 5 U PRBC, 3 U FFP, and 2 U platelets. Estimated blood loss was 754 mL. Despite transfusion of 10 units of blood products, serum sodium increased only from 125 to 126 mmol/L.

Discussion

Intraoperative management of hyponatremia during major surgery, including lung transplantation, relies on strategies that avoid rapid sodium shifts while maintaining intravascular volume and hemostasis[12]. Approaches described in the literature include staged transfusions, close electrolyte monitoring, and the use of CRRT in ECMO patients[1]. Given this patient's SIADH and ECMO-related coagulopathy, careful coordination of transfusion timing, fluid composition, and electrolyte management was essential. Using a low-sodium albumin–0.45% NS carrier enabled delivery of large-volume transfusion while minimizing sodium load and avoiding rapid correction. This approach maintained ECMO flows, supported oxygen delivery, stabilized hemodynamics, and effectively limited sodium rise during a period of high transfusion

demand.

Images



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#FR 3 – Kiosk 9 – Poster 6

HIT Happens, But Not This Time: Safe Heparin Re-Exposure in Open TAAA Repair

Presenting Author: Vinicius Moreira, MD, Fellow, Cleveland Clinic

Introduction

Anticoagulation for patients with a history of heparin-induced thrombocytopenia (HIT) undergoing cardiac surgery is challenging and requires careful preoperative planning. We present a case illustrating strategies to reduce HIT-related complications while ensuring effective anticoagulation for procedures with high risk of bleeding and coagulopathy.

Case Presentation

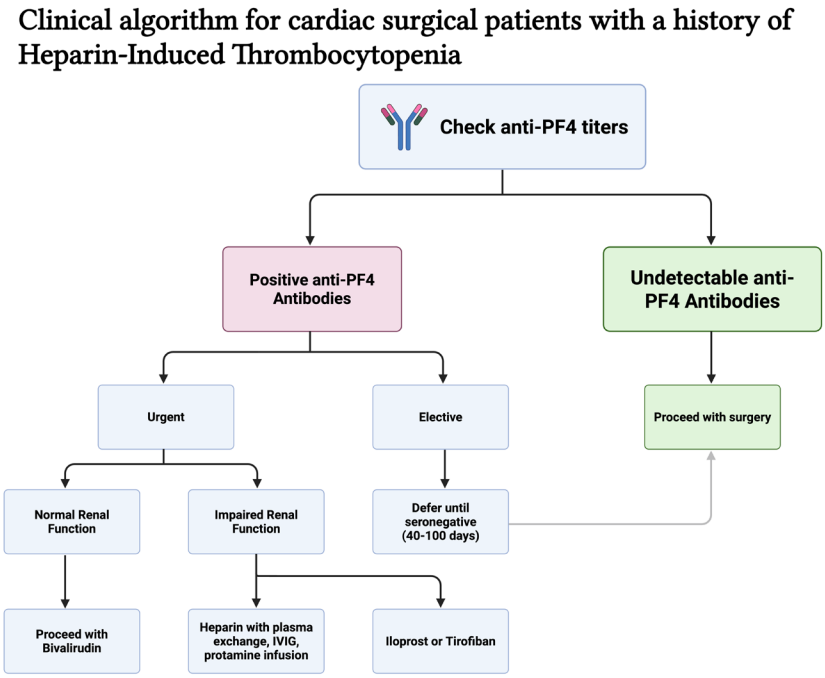
A 65 year-old male presented for reoperative thoracoabdominal aortic aneurysm (TAAA) repair. His history included antiphospholipid syndrome, CAD and Type A aortic dissection repair in 2005, complicated by postoperative HIT with arterial and venous thromboses and mesenteric ischemia requiring hemicolectomy. A progressive aneurysmal degeneration of his descending aorta prompted a staged repair comprising carotid–subclavian bypass with TEVAR followed by open TAAA repair. For the first stage, bivalirudin was chosen for anticoagulation due to concerns regarding his prior HIT complications. The procedure underwent well and he later returned for the second stage. The higher risk of bleeding and coagulopathy with TAAA repair prompted reassessment of the anticoagulation plan. Preoperative anti-PF4 antibodies titers were undetectable, allowing for intraoperative heparin. Heparin was titrated to an activated clotting time (ACT) goal and fully reversed with protamine. Postoperatively, fondaparinux prophylaxis was initiated and later transitioned to rivaroxaban. Platelet counts remained stable without evidence of thrombosis or HIT.

Discussion

Heparin remains the standard anticoagulant for cardiac surgery due to its rapid onset, reliable ACT monitoring, and complete protamine reversibility. HIT occurs in 1 to 3% of cardiac surgeries and significantly increases thromboembolic events, organ injury, infection, and mortality. Therefore, patients with a history of HIT require individualized anticoagulation planning. Evaluation should begin with anti-PF4 ELISA testing. If antibodies are undetectable, intraoperative heparin exposure is considered safe, as antibody regeneration requires several days, even in patients with prior HIT[1,2]. Elective cases should ideally be deferred until antibodies are undetectable, which may take up to 100 days. For urgent cases with subacute or active HIT and high antibody titers, re-exposure to heparin poses a risk of rapid platelet activation and thrombosis, and bivalirudin is the preferred alternative. It is a direct thrombin inhibitor with a short half-life and thrombin-mediated clearance. Limitations include a lack of reversal agent, unreliable monitoring at high doses, risk of clot in stagnant blood, increased early bleeding, and reduced clearance in renal dysfunction. When bivalirudin is contraindicated, therapeutic plasma exchange reduced anti–PF4 titers sufficiently to allow heparin administration, although it does not eliminate risk. Intravenous immunoglobulin may be added to reduce platelet activation by competitively occupying Fcγ receptors on platelets. Postoperative protamine infusions have also been used with these modalities to neutralize residual circulating heparin[3]. Finally,

inhibition of platelet activation using iloprost, a prostacyclin analogue, or tirofiban, a GP IIb/IIIa inhibitor, has been reported as an adjunctive strategy, though evidence is limited to small case series.

Images



Proposed decision-making flowchart for patients with HIT requiring anticoagulation.

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#FR 3 – Kiosk 9 – Poster 7

When BASILICA goes wrong: Left ventricular outflow tract injury causing Aortic root hematoma and tamponade diagnosed by TEE

Presenting Author: Ala Z. Jamal, MD, Clinical Fellow Physician - Adult Cardiothoracic & Vascular Anesthesiology, UC Davis Health

Introduction

The BASILICA (Bioprosthetic or Native Aortic Scallop Intentional Laceration to Prevent Iatrogenic Coronary Artery Obstruction) technique has expanded the safety of transcatheter aortic valve replacement (TAVR) in patients at high risk for coronary obstruction. However, BASILICA introduces unique procedural risks, including iatrogenic injury to the left ventricular outflow tract (LVOT) and aortic root. Intraoperative transesophageal echocardiography (TEE) is vital for the rapid diagnosis and management of these life-threatening complications.

Case Presentation

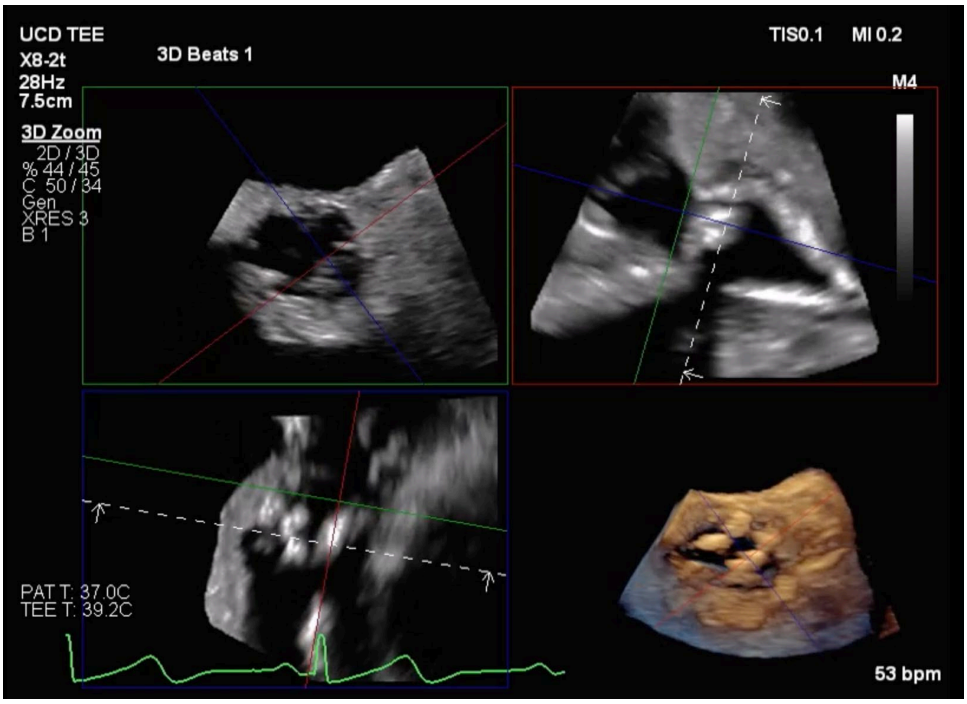
A 63-year-old woman with severe, heavily calcified bicuspid aortic valve stenosis and high surgical risk (4.2% STS risk of mortality) underwent TAVR with BASILICA due to low left coronary height (11 mm) as illustrated by pre-procedural gated-CT TAVR protocol. The procedure was performed under general anesthesia with continuous TEE guidance. Following successful traversal and laceration of the left coronary leaflet, TEE revealed a new aortic root hematoma, located near the non-coronary cusp on multiplanar and 3D TEE imaging. The patient remained initially stable, and a 23 mm balloon-expandable valve was deployed. Shortly after deployment, TEE detected a new pericardial effusion followed by rapid hemodynamic deterioration, consistent with acute tamponade. Emergent pericardial drainage was attempted, but the patient required urgent conversion to open surgery. Surgical exploration revealed a Type 0 Sievers bicuspid aortic valve and a 9 x 5 mm perforation extending from the annulus below the left coronary ostium to the LVOT. After explanting the TAVR valve, the LVOT and annular perforation were repaired with a bovine pericardial patch, and Nick's annular enlargement. A surgical bioprosthetic valve was implanted. Post-bypass TEE showed a small residual peri-aortic hematoma, mean aortic valve gradient of 5–6 mmHg, and no paravalvular leak. The postoperative course was complicated by bleeding requiring transfusion and thoracentesis, but the patient ultimately recovered and was discharged from the ICU.

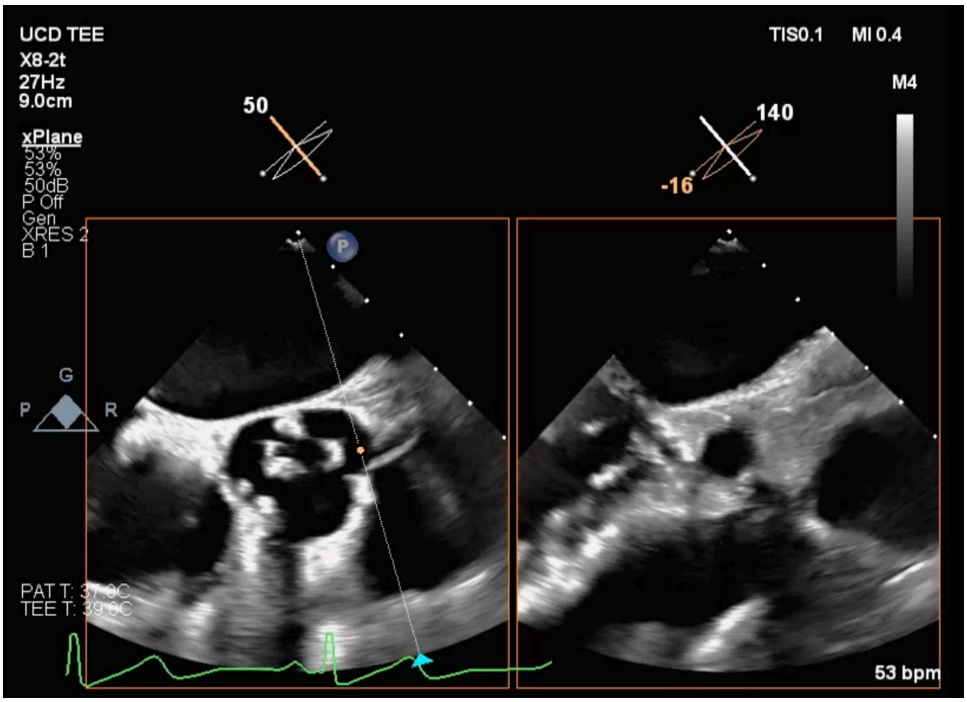
Discussion

This case highlights the essential role of TEE in perioperative management of complex TAVR procedures, especially when advanced leaflet modification techniques like BASILICA are used. TEE, utilizing advanced 2D and 3D imaging, enabled immediate recognition of aortic root hematoma, and pericardial effusion, directly influencing the decision to convert to open surgery and guiding intraoperative management. Prompt TEE diagnosis, multidisciplinary coordination, and surgical readiness were critical to patient survival. As novel transcatheter techniques continue to evolve, vigilant TEE monitoring and procedural planning are

essential to optimize outcomes and manage rare but life-threatening complications.

Images





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#FR 3 – Kiosk 10 – Poster 1

Refractory Coagulopathy after Cardiac Surgery and Cardiopulmonary Bypass

Presenting Author: Kyle G. Kaletka, M.D., Resident Physician, University of Missouri-Columbia

Introduction

Excessive bleeding after cardiac surgery occurs in up to 15% of patients, increasing length of stay, re-exploration rates, and overall morbidity and mortality. Post-bypass coagulopathy is well-documented and often contributes to bleeding.[1-3] Prompt identification and treatment can therefore minimize bleeding but lacks standardization and often relies on institutional guidelines. This case of post-bypass consumptive coagulopathy was refractory to product management and recurred postoperatively.

Case Presentation

A 76-year-old female with history of distant bilateral brachial DVTs, CAD, aortic insufficiency, and grade 3 diastolic dysfunction underwent surgical aortic valve replacement and two-vessel CABG. Total bypass time was 192 minutes. The post-bypass period was complicated by excessive bleeding, requiring administration of 5 units of FFP, 3 packs of platelets, 20 units of cryoprecipitate, and 2mg of recombinant factor 7a with minimal improvement in coagulation profile (*Figure 1*). The patient was normothermic, adequately reversed from heparin, and without evidence of surgical bleeding or consumptive clot in the chest or site of vein harvest. Additional product administered overnight in the CICU decreased the chest tube output and improved hemodynamic stability. The patient was extubated on POD1. Refractory hypoxemia developed on POD2 requiring re-intubation and initiation of V-A ECMO. TEE at this time revealed impaired RV systolic function, McConnell's sign, and a hyperdynamic LV. Severe hemoptysis and diffuse mesenteric ischemia developed on POD3, leading to the expiration of the patient. The autopsy report revealed multiple occlusive emboli resulting in mesenteric ischemia. Serotonin release assay returned negative.

Discussion

Predictive risk factors for bleeding after cardiac surgery include older age, female sex, certain preoperative medications, surgical complexity, duration of CPB, and hypothermia.[1-2] Not all bleeding is associated with coagulopathy, and most coagulopathy does not progress to DIC. One study reported an incidence of 11.9% for postoperative DIC, identifying female sex, higher preoperative DIC score, and increased anesthesia time as risk factors.[3] Multiple complex mechanisms contribute to coagulopathy after CPB including hemodilution; consumption of platelets, fibrinogen, and clotting factors; dysfunction of platelets; and hyperfibrinolysis.[1-3] Separation from the extracorporeal circuit and replacement of losses should result in clinical improvement. While no standardized treatment exists for coagulopathy, point of care tests such as viscoelastic testing and platelet function tests allow for targeted medical and product management with proven benefit.[1-2] In this case, coagulopathy worsened after initial product management suggesting ongoing consumption. No source of surgical bleeding or obvious thrombosis was identified intraoperatively. Given the patient's history of hypercoagulability, initial differentials included obscure microemboli, DVTs, rapid-onset HIT, and DIC. Continued transfusions resulted in a transient improvement on POD1 with subsequent

deterioration. The alternation between bleeding and hypercoagulable states, as evidenced by the massive hemoptysis on ECMO followed by abdominal compartment syndrome, was highly suggestive of DIC.

Images

Figure 1: Notable Laboratory Values and Product Administration

Notable Laboratory Values

	Preop	On-bypass	Post-bypass	End of Case	Postop	POD 1	POD 2	POD 3
Hemoglobin (g/dL)	11.9	6.5	7.4	8.0	11.1	10.1	8.5	7.8
Platelets (x10³/L)	243	126	88	79	134	176	96	54
Fibrinogen (mg/dL)	-----	141	150	204	306	345	-----	302
R time (minutes)	-----	9.0	10.1	11.7	-----	-----	-----	6.6
CRT-Max Amplitude (mm)	-----	56.4	51.7	47.5	-----	-----	-----	57.1
CFF-Max Amplitude (mm)	-----	18.2	16.3	14.5	-----	-----	-----	-----
LY-30 (%)	-----	0.3	0.2	0.0	-----	-----	-----	-----
ACT (seconds)	99	487	119	108	-----	-----	-----	-----

Product Administration

pRBC	-----	-----	2 units	5 units	2 units	3 units	-----
FFP	-----	-----	2 units	3 units	2 units	-----	-----
Platelets	-----	-----	-----	3 packs	2 packs	1 pack	-----
Cryoprecipitate	-----	-----	-----	20 units	10 units	-----	-----
Other	-----	-----	Protamine, TXA, Cell Saver	2mg Recombinant Factor 7a	16mcg DDAVP	-----	-----

Figure 1 presents the chronological order of notable laboratory values and product administration. The arrows (red) demonstrate the period during which the designated products were administered.

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#FR 3 – Kiosk 10 – Poster 2

A late presentation of a large ascending aorta pseudoaneurysm versus a negative culture bacterial endocarditis after aortic valve replacement

Presenting Author: Javier J. Polania Gutierrez, Md, Fellow Adult Cardiothoracic Anesthesiology, Temple University Hospital

Introduction

Aortic pseudoaneurysms are rare but potentially fatal post-surgical complications. Diagnosis is challenging, with presentations ranging from incidental findings to compression symptoms. Etiology often involves anastomotic failure or graft infection, although causality is not always confirmed.

Case Presentation

An 80-year-old AAF with HTN and a mechanical aortic valve replacement 39 years earlier, maintained on warfarin, presented after multiple falls with subjective new-onset LLE weakness. review of system was negative for prodromal, neuro, or cardiovascular symptoms, and vital signs and physical exam were unremarkable. Initial workup showed complete heart block (CHB) on EKG, mild troponin elevation, anemia, thrombocytopenia, normal renal function, and laboratory evidence of new-onset hypothyroidism. Vit. B12 and folate levels were normal, and both chest X-ray and head CT were unremarkable. EP confirmed CHB with a fascicular escape rhythm, while patient was hypothyroid her block was considered to be non-reversible and a permanent pacemaker was recommended. Shortly after admission, intermittent fever developed, prompting TTE (Image 1), which revealed a possible aneurysmal structure adjacent to the coronary sinus. CTA was subsequently obtained. CTA demonstrated a contour bulge along the inferomedial right atrium with mural thrombus and delayed contrast washout, suggestive of pseudoaneurysm (Image 2). Infectious disease workup revealed one in four blood cultures positive for pan sensitive Staph. Epi. Empiric antibiotics were initiated for suspected IE. Serologies for Coxsackie virus, Lyme disease, and syphilis were negative. CT Surgery recommended ascending aorta repair with epicardial PPM placement to manage the suspected pseudoaneurysm, possible infection, and CHB. Surgery was performed on hospital day four. After uneventful induction, intraoperative TEE was conducted (Image 3). A 6x6 cm pseudoaneurysm protruding into the right atrium with cm neck in ascending aorta was found. Interatrial septal defect was repaired using an autologous pericardial patch. No gross evidence of endocarditis was found. Postoperatively, the patient recovered uneventfully in the ICU, remained afebrile, and repeat blood cultures were negative. Pathologic analysis demonstrated findings consistent with a pseudoaneurysm wall. Although microbiologic data were inconclusive, Infectious Disease recommended a six-week course of antibiotics for presumed culture-negative infective endocarditis.

Discussion

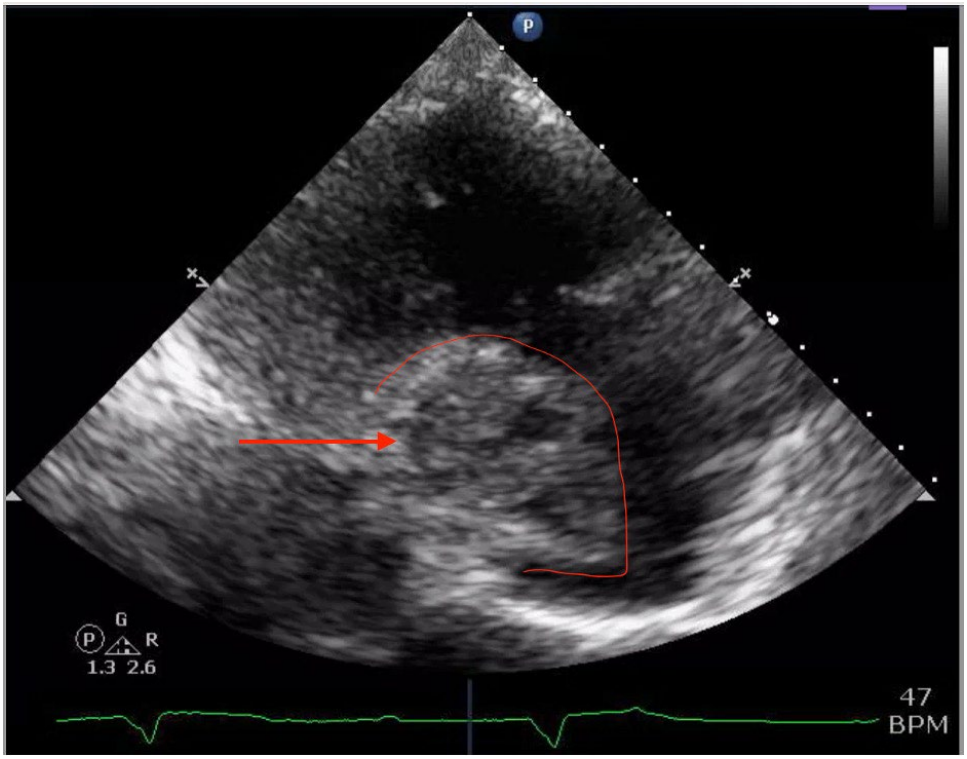
Antibiotic use before blood cultures and atypical pathogens are common causes of Culture Negative IE, accounting for up to 30% of cases, yet both were excluded in our patient. The absence of microbiological evidence, thromboembolism, and intraoperative findings—along with only “possible” IE by Duke criteria—

made clinical judgment crucial, supported by postoperative resolution of fever and thrombocytopenia. Although IE is a known cause of pseudoaneurysm, this case is unusual: diagnosed 39 years after surgery, asymptomatic, and located in an atypical posterior–medial Aorta. Pseudoaneurysm remains life-threatening and requires prompt intervention regardless of etiology.

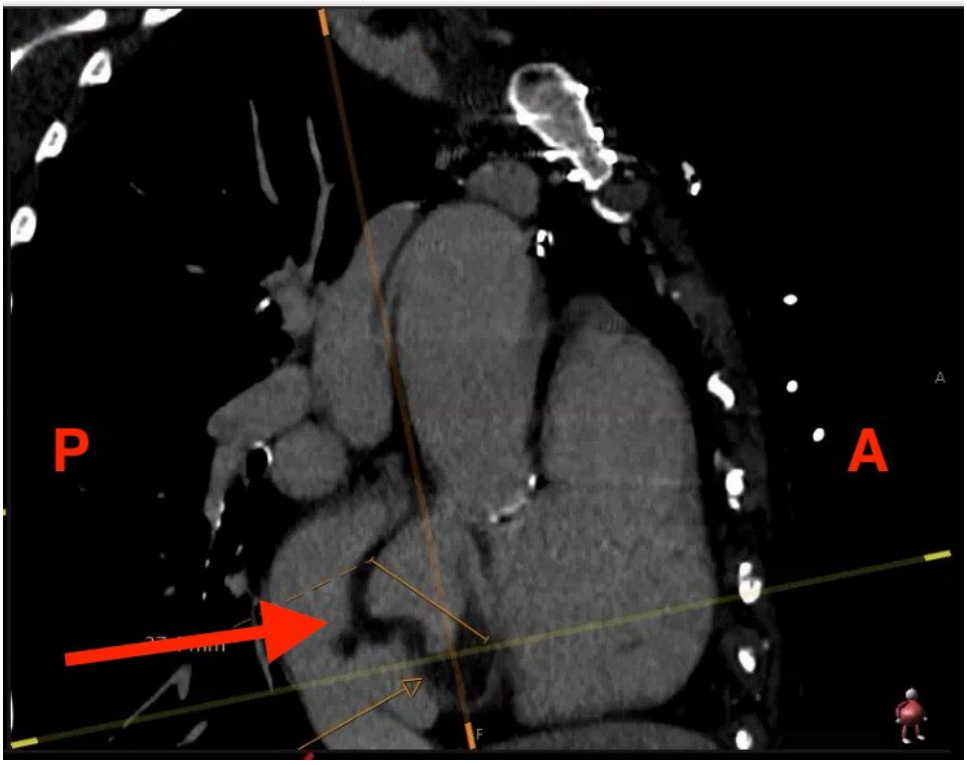
Images

Chamber	TTE	TEE
Right Atrium	Right atrium is severely dilated.	Dilated
Right ventricle	Right ventricle is mildly dilated. Mildly reduced systolic function.	Dilated with normal systolic function
Left Atrium	Normal Size.	Normal size, no masses
Left ventricle	normal LV size/function (EF 60–65%)	Preserved systolic function, EF ~50%, no RWMA
Tricuspid Valve	Severe annular dilation. Moderate to severe transvalvular regurgitation with an eccentrically directed jet toward the interatrial septum primarily due to malcoaptation of the anterior and septal leaflets	Annulus dilated to 4 cm with moderate central regurgitation displaced by pseudoaneurysm
Mitral valve	Mild thickening. No restricted motion. Mild transvalvular regurgitation with a centrally directed jet. No stenosis.	Normal, no MR or MS
Aortic valve	well-seated St. Jude Bi-leaflet mechanical aortic valve with elevated gradient (peak velocity 3.25 m/s; mean gradient 20.9 mmHg). Area continuity by VTI is 1.50 cm2. AV Doppler vel index VTI is 0.43	Well-seated mechanical prosthesis, mean gradient 7 mmHg, no paravalvular leak
Pulmonic valve	The pulmonic valve was not well visualized.	Leaflets poorly visualized
Pericardial space	No pericardial effusion.	No effusion or thickening
Aorta	Possible noncoronary sinus aneurysm abutting into right atrium.	Large pseudoaneurysm immediately distal to the valve, filling through a small defect near the non/right coronary cusps. Significant thrombus and areas of stagnant or low flow are present. No communication with the atria is observed, but the pseudoaneurysm compresses and displaces the septal right atrium and septal tricuspid annulus. Aortic arch: Normal size, no dissection.

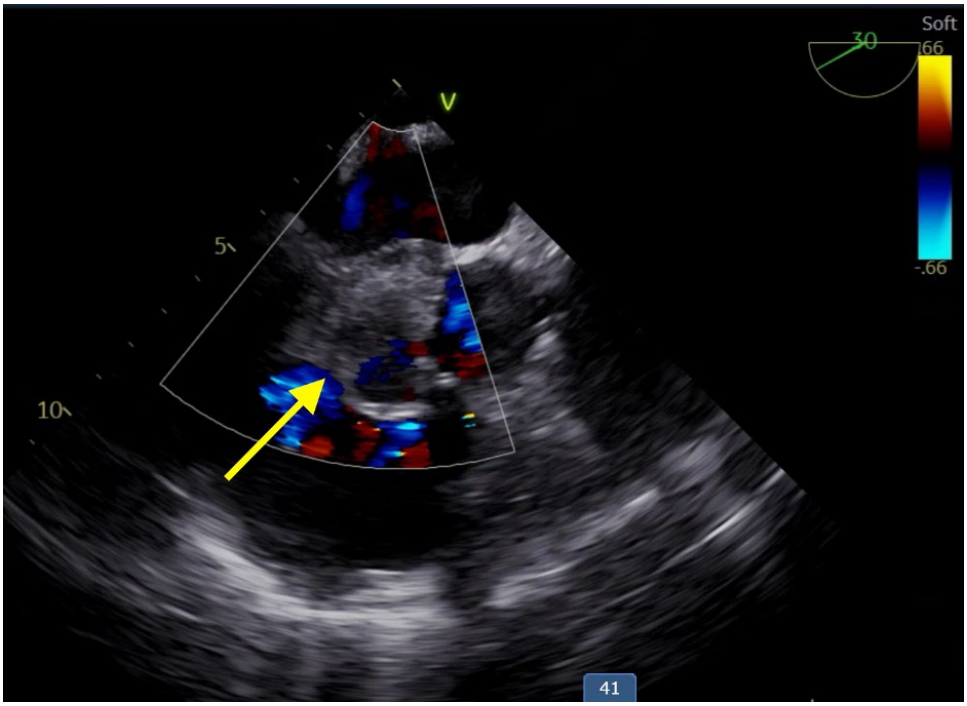
Transthoracic Echocardiogram results. Intraoperative Transesophageal Echocardiogram.



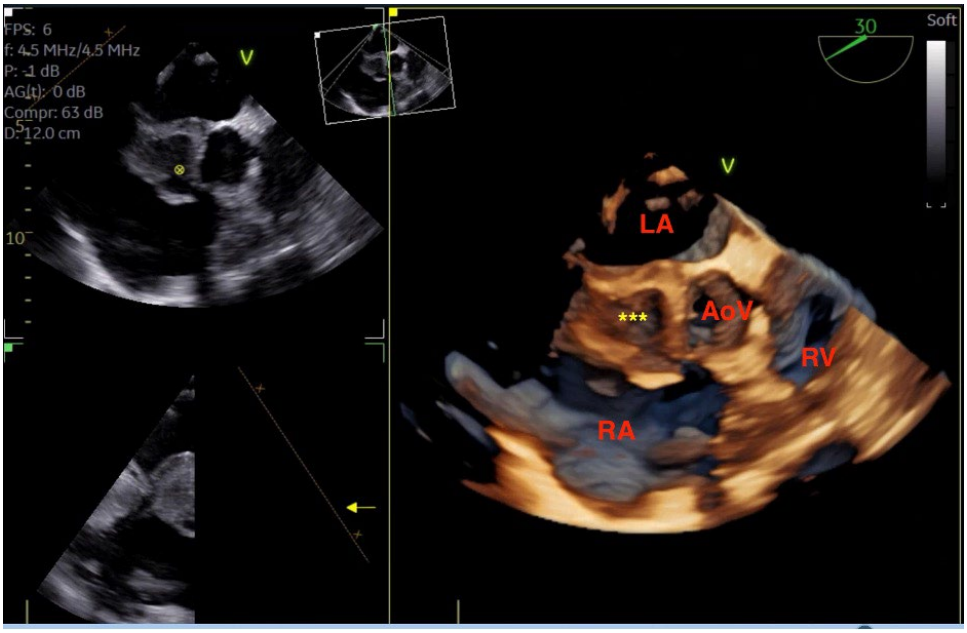
Transthoracic Echocardiogram right atrium and ventricle. Red arrow pointing Pseudoaneurysm.



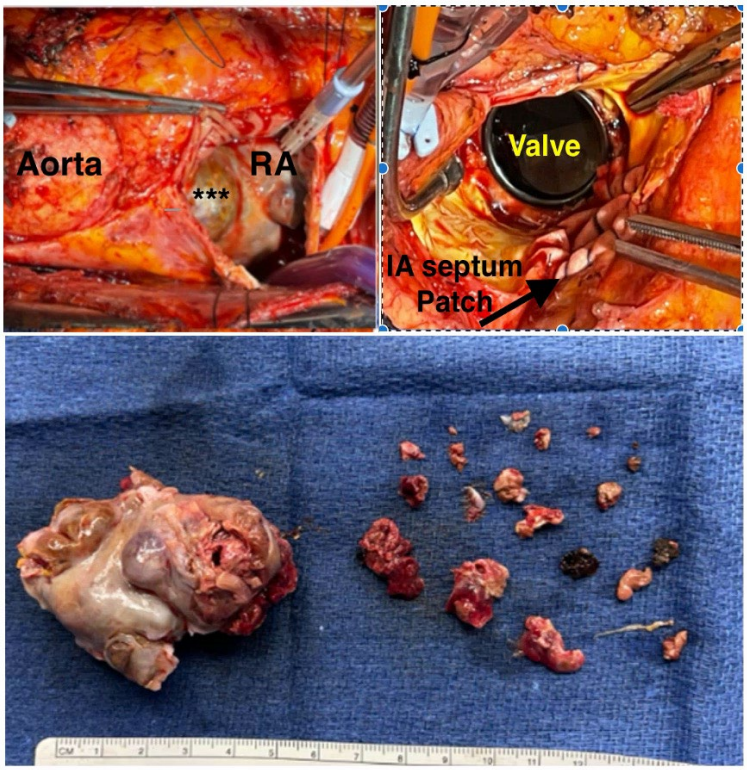
CT Angiogram. Red arrow pointing Pseudoaneurysm.



Intraoperative Transesophageal echocardiogram. Aortic valve short axis view. Yellow arrow pointing Pseudoaneurysm.



Transesophageal echocardiogram. 3D reconstruction Ao valve short Axis. *** Pseudoaneurysm.



Intraoperative surgical findings. Left upper: *** Pseudoaneurysm protruding on right atrium. Right upper: St.Jude mechanical valve well seated no evidence of infection.

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#FR 3 – Kiosk 10 – Poster 3

Not a Sign of Recovery: A Rare Case of Papillary Muscle–Induced LVAD Inflow Obstruction Revealed by Intraoperative TEE

Presenting Author: Krishnal Sail, MD, Fellow Physician, Loyola University Medical Center

Introduction

Left ventricular assist devices (LVADs) provide continuous flow, and an increased pulsatility index (PI) is typically interpreted as improved native LV function or reduced pump preload.[1] However, mechanical inflow obstruction can paradoxically produce both increased pulsatility and recurrent low-flow alarms despite stable systemic perfusion. Dynamic obstruction from intracardiac structures such as papillary muscles is rare and can be difficult to diagnose preoperatively. Echocardiography plays a key role in identifying these mechanisms of device dysfunction.[2] This case highlights the anesthetic challenges of induction in a patient with an LVAD under these very conditions.

Case Presentation

A 57 year old male with a HeartMate-3 LVAD as a bridge-to-transplant for one year presented for sternal incision and drainage due to pain from a fractured sternal wire. In the days prior, he experienced recurrent low-flow alarms and increased pulsatility despite preserved blood pressure, normal perfusion, and a transthoracic echocardiogram without evidence of suck-down, cannula malposition, or LV recovery. Upon arrival to the OR, pulse oximetry and NIBP readings were normal. Following induction, he became hemodynamically unstable. The arterial line demonstrated a pulsatile waveform, yet the LVAD continued to display persistent low-flow alarms and markedly elevated PI (Fig 1). Transesophageal echocardiogram (TEE) showed no aortic valve opening in 10/10 cycles, indicating continued LV dependence. It also revealed the posteromedial papillary muscle intermittently occluding the LVAD inflow cannula during diastole and moving away during systole, producing cyclical variations in preload and flow (Fig 2, 3). Active papillary muscle contraction during systole relieved the obstruction, explaining the pulsatility on the arterial waveform. Management focused on restoring preload and reducing suction forces. The patient improved with careful volume administration and temporary reduction of LVAD speed, which relieved inflow obstruction and normalized flow parameters.

Discussion

This case demonstrates a rare cause of increased LVAD pulsatility—dynamic inflow obstruction from the posteromedial papillary muscle—rather than improved LV systolic function. The combination of high PI, low-flow alarms, and absent aortic valve opening on TEE highlighted the mismatch between device parameters and actual physiology. A similar mechanism has been previously described in a case of intermittent suction of the posteromedial papillary muscle into the LVAD inflow cannula during diastole, resulting in cyclical obstruction of flow and low-flow alarms despite preserved systemic perfusion.[3] In this case, anesthetic induction exacerbated the obstruction by decreasing preload and increasing relative suction at the inflow

cannula.

Key learning points include:

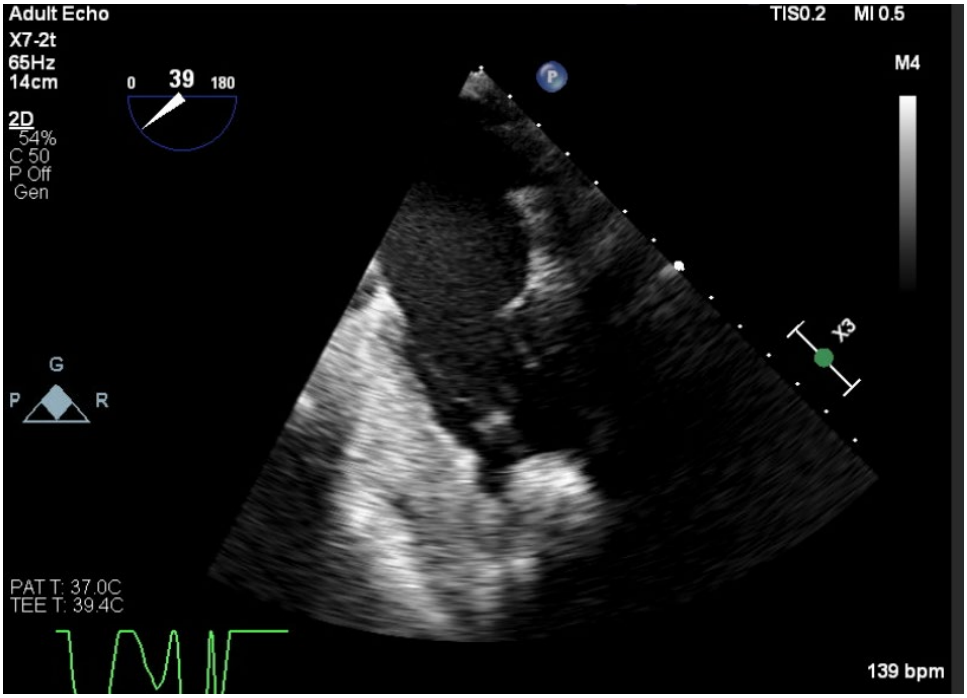
1. Increased pulsatility is not always a sign of LV recovery and may reflect intermittent inflow obstruction.
2. Dynamic intracardiac structures can cause preload-sensitive LVAD dysfunction detectable only by TEE.[2]
3. Induction may worsen occult inflow obstruction, making preload optimization and LVAD speed reduction essential management strategies.

Early intraoperative TEE was key in identifying the mechanism and guiding care, preventing further hemodynamic instability.

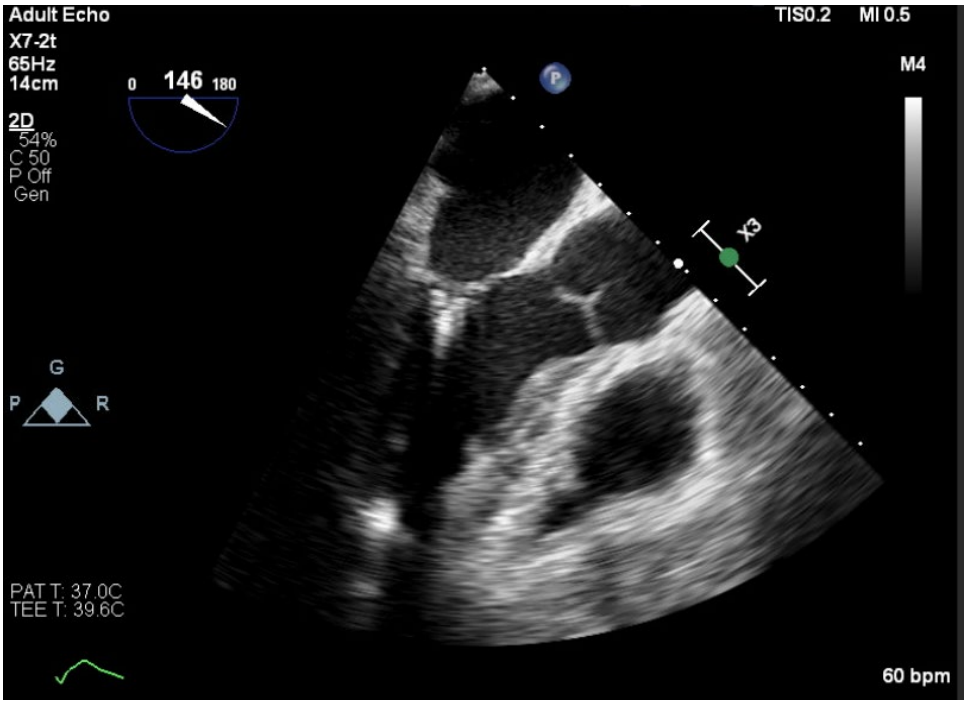
Images



Monitor Demonstrating Pulsatility



Papillary Muscle Occluding Inflow Cannula During Diastole



ME Long Axis View- Non-Obstructed Inflow Cannula & Closed Aortic Valve

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#FR 3 – Kiosk 10 – Poster 4

From Flutter to Failure: Unmasking of A Cor Triatriatum Sinister Saved by A Common Atrium In An Adult with New Onset Heart Failure

Presenting Author: Mira Bishawi, DO, Anesthesiology Resident, Riverside University Health System

Introduction

Congenital heart disease (CHD) affects 8-10 per 1,000 live births, and with modern advances, nearly 90% of patients now survive into adulthood. Consequently, previously undiagnosed congenital lesions are increasingly encountered in adult practice. Atrioventricular canal defects (AVCDs) comprise 4-5% of CHD and range from complete defects, typically symptomatic in infancy, to partial forms that may remain silent for decades. A common atrium can decompress the left atrium, delaying pulmonary venous hypertension and heart failure. Cor triatriatum sinistrum, an even rarer anomaly, occurs in only 0.1-0.4% of CHD and results from a fibromuscular membrane dividing the left atrium. Severe forms present in infancy with pulmonary venous obstruction, but nonrestrictive variants may remain concealed into adulthood, mimicking mitral stenosis or contributing to pulmonary hypertension, arrhythmias, and heart failure. In our patient, the coexistence of a large common atrium with partial AVCD mitigated the hemodynamic burden of cor triatriatum, delaying presentation until late adulthood.

Case Presentation

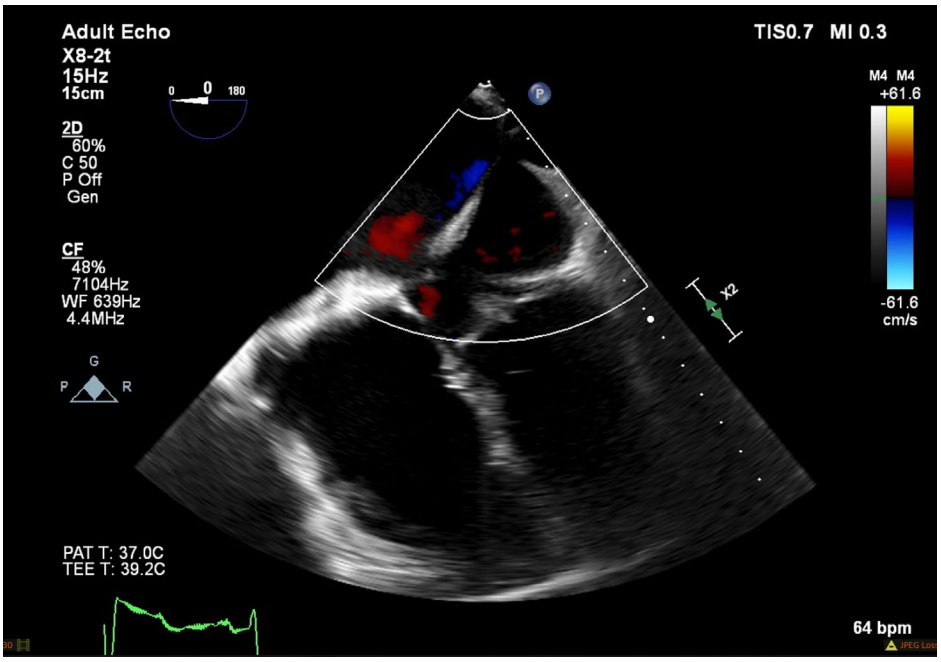
A 54-year-old male with no known cardiac history presented with dyspnea and chest pain. ECG showed atrial flutter with rapid ventricular response. Preoperative transthoracic echocardiography (TTE) revealed biventricular dysfunction but no structural abnormalities. During induction for a planned atrial fibrillation ablation, the patient developed hemodynamic instability, prompting intraoperative transesophageal echocardiography (TEE). TEE revealed a large common atrium, partial AV canal defect, cor triatriatum sinistrum, and severe atrioventricular valve regurgitation. Cardiac catheterization demonstrated severe pulmonary hypertension (mean PAP 56 mmHg, PCWP 28 mmHg) and a significant left-to-right shunt (Qp:Qs 3.2:1). Anesthetic management was tailored to maintain sinus rhythm, preserve biventricular function, and avoid increases in pulmonary vascular resistance through careful control of preload, afterload, and ventilation. Invasive monitoring included arterial, central venous, and pulmonary artery catheters. The patient underwent cor triatriatum resection, partial AV canal repair, and AV valve repairs. Intraoperative TEE was instrumental in confirming the diagnosis, guiding surgical correction, and assessing repair adequacy post-bypass. The postoperative course was uncomplicated, and he was discharged home in stable condition with improved functional status at follow-up.

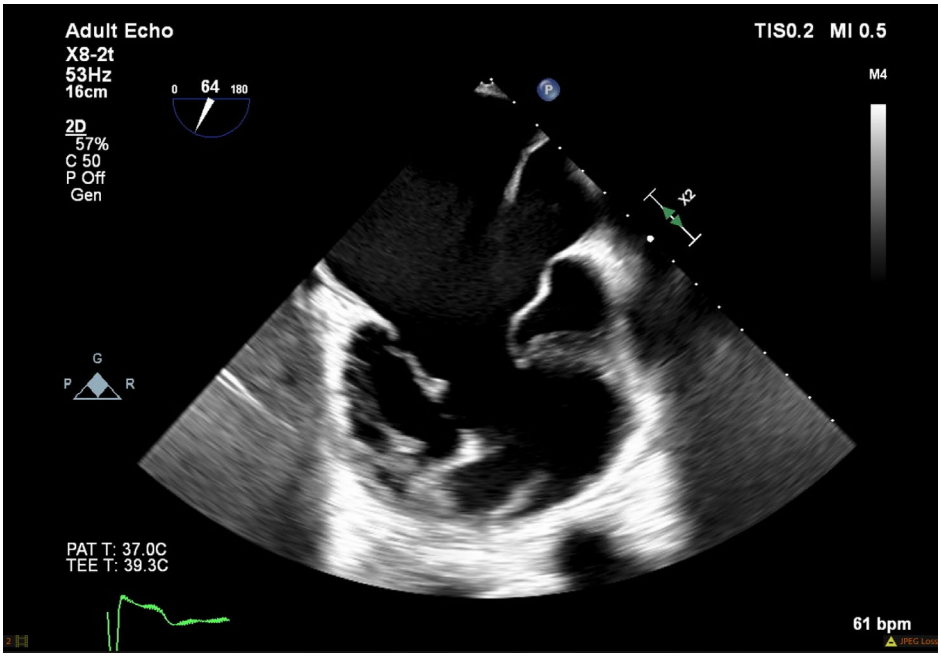
Discussion

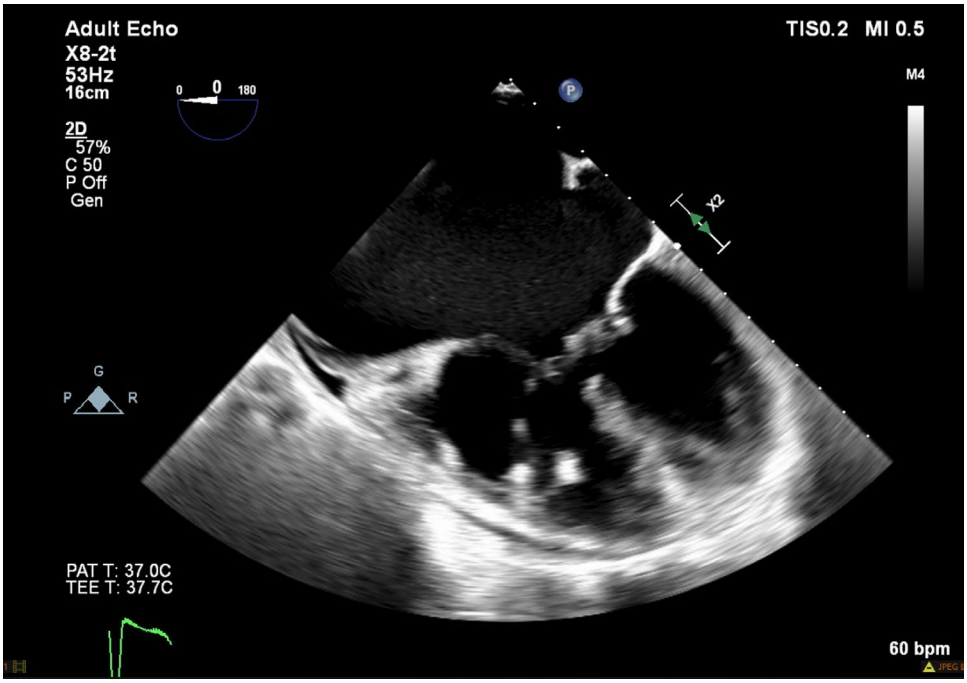
This case illustrates how rare congenital variants can modify hemodynamics to permit prolonged survival yet predispose to late decompensation. The patient's common atrium decompressed pulmonary venous return, masking the physiologic impact of cor triatriatum sinistrum until adulthood. For anesthesiologists, management requires vigilance in balancing preload and afterload, minimizing pulmonary vascular

resistance, and preventing arrhythmias and hemodynamic instability during induction and bypass. This case underscores the diagnostic and intraoperative value of TEE in identifying unexpected congenital anatomy and guiding surgical management.

Images







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#FR 3 – Kiosk 10 – Poster 5

Iatrogenic Ascending Aortic Injury After Coronary Artery Bypass Grafting: Delayed Recognition and Surgical Repair

Presenting Author: Jesse Roberson, MD, Anesthesiology Resident, University of California San Diego

Introduction

Transesophageal echocardiography (TEE) is fundamental for intraoperative assessment of the thoracic aorta during cardiac surgery. Technical maneuvers such as aortic cannulation, cross-clamp placement, and proximal bypass graft construction carry inherent risk of iatrogenic aortic wall injury. Communication with the surgical team is of paramount importance as blood pressure lowering techniques reduce risk of dissection during these critical moments. Although uncommon, these injuries may remain clinically silent and present months to years later as aortic injuries. We describe a patient who developed an ascending aortic dissection originating near a prior proximal saphenous vein graft (SVG) anastomosis.

Case Presentation

A 59-year-old woman with hypertension and coronary artery disease underwent CABG with a left internal mammary artery to left anterior descending artery and a SVG to the posterior descending artery. Her initial postoperative course was unremarkable, and she was discharged home. However, she was seen multiple times post-surgery due to chest pain and shortness of breath and eventually developed new-onset systolic heart failure. Transthoracic imaging revealed a focal ascending aortic abnormality adjacent to the origin of the SVG graft and she was referred for definitive repair. Intraoperative TEE demonstrated a localized ascending aortic dissection extending from the proximal SVG anastomosis toward the sinotubular junction (Figure 1). The aortic valve was competent, and biventricular function was preserved. Surgical inspection revealed a focal aortic lesion at the graft anastomosis with organized thrombus. The diseased segment was resected and replaced with an interposition graft, and the SVG was reimplanted. Post-repair TEE confirmed normal graft contour and no residual mural abnormality (Figure 2). The patient was weaned from bypass without issue and discharged home on postoperative day six.

Discussion

Iatrogenic injury of the ascending aorta is rare but clinically meaningful. These lesions often arise at focal sites of surgical manipulation, including cannulation, cross-clamping, and proximal graft anastomoses. Contemporary analyses of surgically induced ascending aortic injury describe a characteristic pattern in which disruption of the aortic wall may not be apparent at the time of surgery, later evolving into defects with substantial associated morbidity.[1] In patients undergoing CABG, the proximal attachment point of saphenous vein grafts has been repeatedly identified as an area of susceptibility, reflecting the mechanical strain and local trauma associated with establishing graft inflow.[2] TEE remains indispensable in recognizing these abnormalities. Baseline evaluation of the ascending aorta before cannulation establishes wall morphology, while post-bypass imaging provides the opportunity to identify new mural irregularity or disturbed flow that may signify injury. This case illustrates how a localized aortic lesion adjacent to a prior

graft site may be clinically silent at the time of surgery and only later manifest with symptoms, though index TEE was unavailable for review. Awareness of these subtle injuries, combined with deliberate imaging and follow-up, remains essential to prevent delayed progression and its potential consequences.

Images

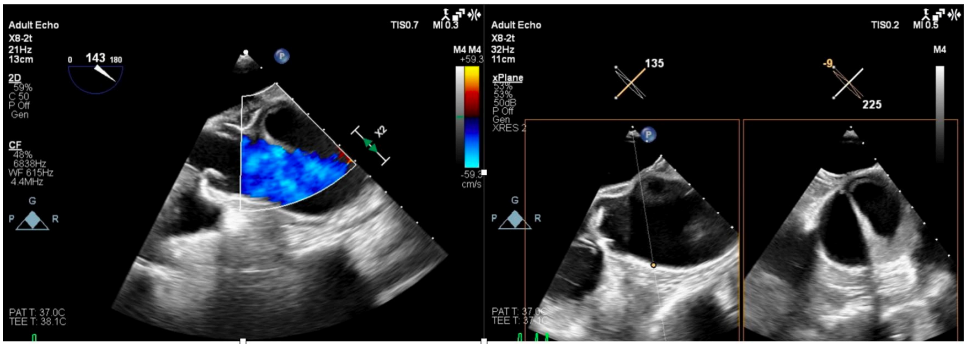


Figure 1: Mid-esophageal aortic valve long axis view with color flow (left) and x-plane (right) demonstrating a focal dissection extending to the sinotubular junction.

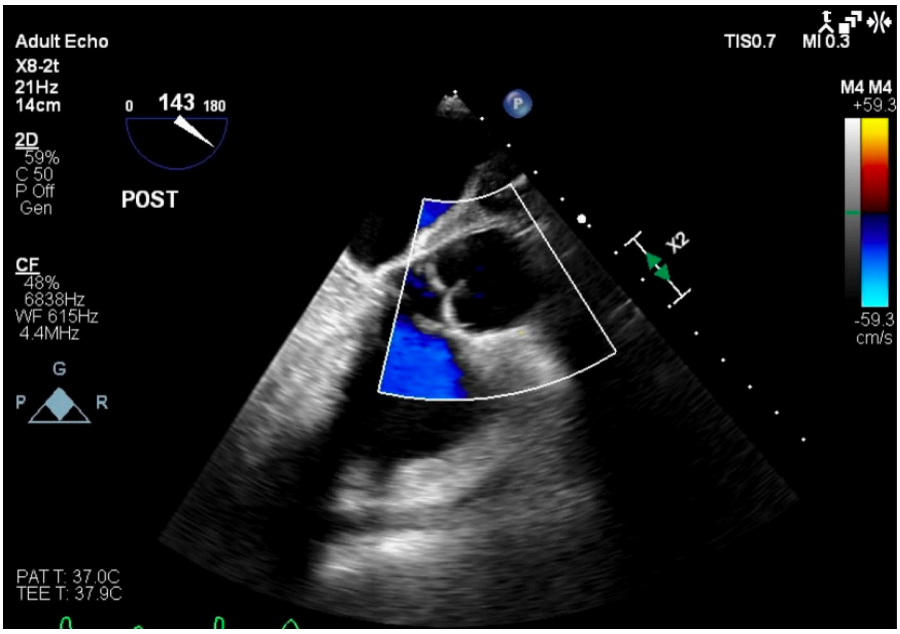


Figure 2: Mid-esophageal aortic valve long axis view after implantation of an interposition graft.

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#FR 3 – Kiosk 10 – Poster 6

Factor First: VWD and CABG in the DDAVP-Allergic Patient

Presenting Author: Harrison Burgess, DO, PharmD, MaineHealth - Maine Medical Center Portland

Introduction

Cardiac surgery presents substantial bleeding risk in patients with von Willebrand Disease (VWD). Desmopressin (DDAVP), the typical first-line therapy for enhancing endogenous von Willebrand factor (VWF) release, is contraindicated in patients with certain VWF subtypes or a confirmed DDAVP allergy. In such cases, perioperative hemostatic management must rely entirely on VWF-containing factor concentrates. We describe successful coronary artery bypass grafting (CABG) in a patient with VWD who required individualized hemostatic optimization using Humate-P.

Case Presentation

A middle-aged patient with severe multivessel coronary artery disease, Type 2 VWD, and significant comorbidities—including chronic heart failure, chronic kidney disease stage III, hypertension, prior cerebrovascular accident, and recurrent gastrointestinal bleeding—was evaluated for CABG. Preoperative transthoracic echocardiography demonstrated a normal-sized left ventricle with mild concentric hypertrophy, mildly reduced systolic function (EF 45–50%), grade I diastolic dysfunction, and regional wall motion abnormalities involving the basal inferior and inferoseptal segments. Right ventricular size and function were low-normal, and no significant valvular stenosis or regurgitation was present. Cath report from outside hospital demonstrated 80% left main coronary artery stenosis and 70 to 80% ostial LAD stenosis. Since the patient had a documented DDAVP allergy, hematology recommended perioperative VWF replacement using Humate-P on a scheduled regimen over several weeks. On the day of surgery, a single pre-incision dose of Humate-P was administered to optimize VWF activity prior to sternotomy and cardiopulmonary bypass. The operation proceeded without excessive bleeding and thromboelastography (TEG) values within a normal range. Hemostasis remained stable throughout bypass initiation, separation, and chest closure, with no requirement for intraoperative redosing of Humate-P or nonstandard blood products. Coagulation parameters and surgical field assessment remained within expected ranges. Postoperatively, the patient received scheduled low-dose Humate-P every 12 hours for seven days with hematology monitoring VWF:RCo levels. He remained hemodynamically stable without major bleeding

events and recovered appropriately.

Discussion

Patients with VWD undergoing cardiac surgery require meticulous perioperative planning due to the amplified bleeding risk associated with cardiopulmonary bypass. When DDAVP is contraindicated, VWF-containing factor concentrates such as Humate-P become essential. In this case, a single pre-incision loading dose provided adequate intraoperative hemostatic support, while scheduled postoperative dosing maintained stable coagulation during the highest-risk period. This case highlights the importance of hematology-guided dosing, multidisciplinary coordination, and individualized factor replacement strategies in safely performing major cardiac surgery in patients with VWD who cannot receive DDAVP.

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#FR 3 – Kiosk 10 – Poster 7

Cesarean Section in End-Stage Biventricular Cardiomyopathy: A Physiology-Driven Strategy Using Controlled Phlebotomy and Multimodal Hemodynamic Monitoring

Presenting Author: Yuya Takahashi, MD, Resident, Henry Ford Hospital

Introduction

Biventricular cardiomyopathy renders parturients highly vulnerable to delivery-related hemodynamic shifts. Right ventricular (RV) dysfunction is especially hazardous because the abrupt autotransfusion after uterine decompression can precipitate RV dilation, septal shift, and biventricular collapse. Anticipatory volume control and real-time physiologic monitoring are essential. We present a high-risk cesarean section (CS) managed with controlled pre-delivery phlebotomy and integrated hemodynamic monitoring.

Case Presentation

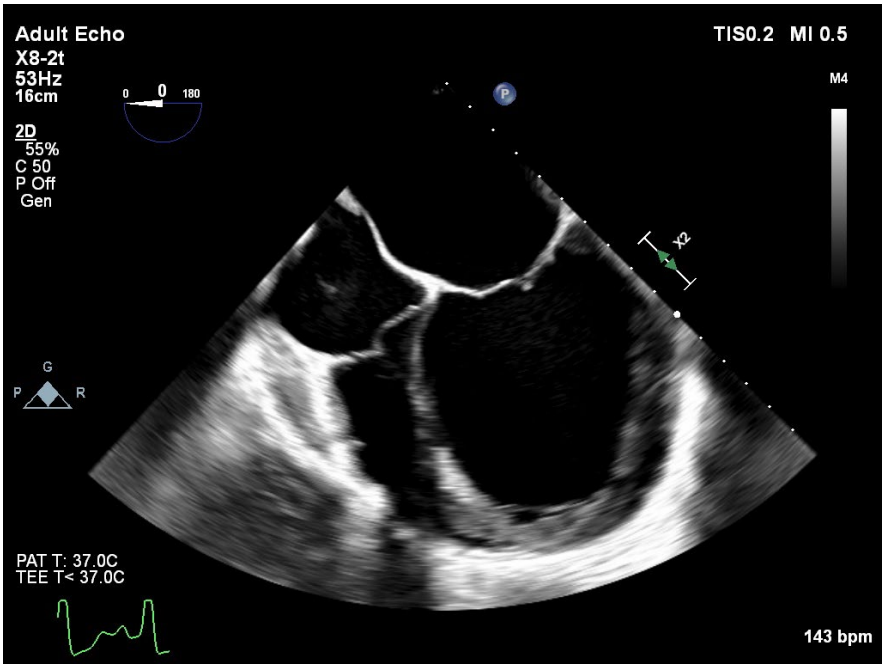
A 46-year-old Gravida 4 Para 1 with chronic heart failure with reduced ejection fraction (EF) of 15% due to mixed peripartum and ischemic cardiomyopathy, prior left anterior descending (LAD) coronary artery stenting, hypertension, and diabetes presented at 32+1 weeks for CS for breech presentation. Because of severe biventricular dysfunction, she was admitted to the cardiovascular intensive care unit (CVICU) for optimization. An arterial line, central venous catheter, and pulmonary artery catheter (PAC) were placed prior to induction, which she tolerated without major instability. As uterine decompression was expected to create a large autotransfusion that could overwhelm the RV, 1 L of controlled phlebotomy was performed immediately after induction into an autologous reservoir. Intraoperative transesophageal echocardiography (TEE) showed EF < 10% with severe RV systolic dysfunction, and PAC measurements confirmed markedly elevated filling pressures. CS proceeded uneventfully, with vasoactive support and autologous reinfusion titrated using integrated TEE and PAC data. After initial extubation, the patient developed acute hypoxemia requiring reintubation for suspected flash pulmonary edema. She stabilized in the CVICU, was extubated on postoperative day (POD) 1, and discharged on POD 10. Her heart failure progressed postpartum, leading to Impella 5.5 implantation via the axillary artery and ultimately orthotopic heart transplantation.

Discussion

This case underscores that in biventricular cardiomyopathy, the RV is the fulcrum of instability as abrupt post-delivery autotransfusion can sharply increase RV preload, provoke septal shift, impair left ventricular ffilling,and precipitate rapid collapse. Although controlled phlebotomy is not standard in obstetric anesthesia, selective reports describe its use in severe RV dysfunction. In this patient, removal of 1 L created a physiologic buffer against the immediate autotransfusion and facilitated staged reinfusion. Multimodal monitoring, including TEE for dynamic visualization of RV size, septal motion, and ventricular function, and PAC for quantitative trends in filling pressures and cardiac output, provided complementary real-time data that allowed precise vasoactive titration and safe timing of reinfusion. Lastly, severe RV dysfunction with EF < 30% portends a poor likelihood of recovery, consistent with this patient's progression to mechanical circulatory support and transplant. Individualized physiology-driven strategies, especially

targeted volume modulation and invasive multimodal monitoring, are recommended for similar high-risk cases.

Images



TEE showing the significant dilation of LV

NIBP mmHg							
NIBP MAP (mmHg)							
Arterial BP mmHg	139/81	138/75	139/80	133/83	135/83	133/82	132/81
Arterial MAP mmHg mmHg	101	102	102	99	100	100	98
ABP mmHg							
ABP MAP mmHg mmHg							
Fem BP mmHg							
Fem MAP mmHg							
Pulse	117	116	114	113	113	112	110
Spo2 Pulse Rate	116	117	115	114	115	112	111
PAP mmHg	64/15	64/12	86/42	89/46	89/37	57/15	57/14
PAP (Mean) mmHg	45	42	69	71	67	38	38

1L of blood aspirated from patient's central line.

phlebotomy acheived the drop in PA pressures.

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#FR 3 – Kiosk 11 – Poster 1

Anesthetic Management of a Large Anterior Mass with Severe Vascular Compression

Presenting Author: Jacob A. Braaten, MD, Resident Physician, Northwestern Memorial Hospital

Introduction

Large anterior mediastinal masses (AMM) pose anesthetic challenges due to the risk of airway obstruction, large vessel compression, impaired venous return, and right ventricular outflow obstruction—risks that are heightened with induction of general anesthesia (GA), supine positioning, and positive pressure ventilation (PPV). We present the case of a rapidly enlarging AMM with significant superior vena cava (SVC) and pulmonary artery (PA) compression that required complex anesthetic planning and intraoperative decision-making.

Case Presentation

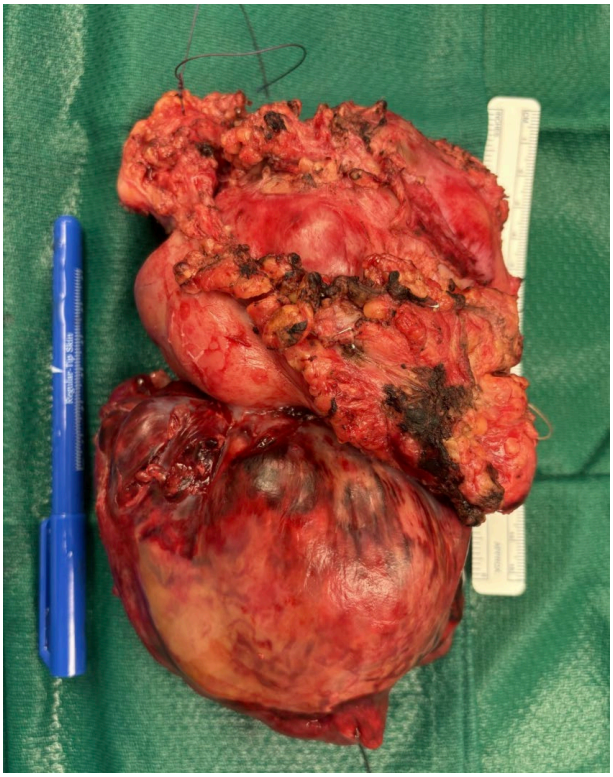
We report the case of a 39-year-old male who presented with a rapidly enlarging right paratracheal AMM. Initial CT demonstrated an 8.4 x 6.3 x 7.3 cm mass, which progressed to 12.9 x 11.6 x 8.2 cm within three months. Preoperative imaging demonstrated severe compression of the right mainstem bronchus, right PA, and SVC consistent with evolving SVC syndrome. Preoperative echocardiography demonstrated normal biventricular function, no significant valvular issues and a PFO. Airway assessment was reassuring (Mallampati I) without evidence of significant edema. He denied any postural symptoms. The patient underwent AMM resection with SVC reconstruction, right pulmonary artery patch, PFO closure, and right upper lobe wedge resection. A preinduction arterial line was placed and mask induction was performed with sevoflurane. Paralysis was administered after confirming mask ventilation, and he was intubated without difficulty with a left-sided double-lumen tube. A PA catheter was placed via the right IJ and additional femoral central venous access was established. Intraoperative TEE demonstrated the mass encasing the SVC with invasion noted at the SVC-RA junction. The tumor was mobilized from the lung, and the patient was placed on cardiopulmonary bypass without complication. Following complete resection and repair, the patient was easily weaned from bypass with low-dose inotropes and minimal vasopressor support. He was transported intubated to the cardiothoracic ICU and extubated on postoperative day one without complication. Final pathology revealed a grade 2 pleomorphic liposarcoma with negative margins.

Discussion

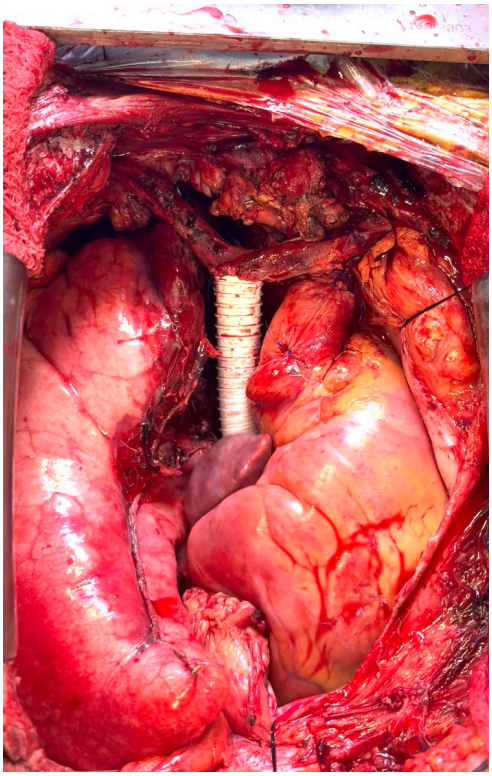
This case highlights the challenges of anesthetic management in AMM causing severe vascular compression. Thorough preoperative assessment is essential to stratify risk based on symptom severity, imaging findings, and the positional effects of the mass. Risk increases with more pronounced symptoms, rapid progression, and positional exacerbation. Airway management should prioritize relieving airway compression and, in severe cases, may necessitate rigid bronchoscope or airway stenting. Hemodynamic instability can result from compression of major cardiac and vascular structures, an effect that is exacerbated by supine positioning, induction of GA, PPV, and air trapping.[1] ECMO should be readily available as a contingency for hemodynamic or airway collapse.[2] Ultimately, the safest anesthetic plan

minimizes irreversible events and anticipates complications related to the underlying physiology.

Images



Excised anterior mediastinal tumor diagnosed as pleomorphic liposarcoma.



Intraoperative view following excision with right lung after wedge resection and SVC reconstruction with graft placement.

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#FR 3 – Kiosk 11 – Poster 2

Echocardiography to the Rescue: Life Saving Thrombectomy for Massive Intraoperative Pulmonary Embolism During Obstetric Procedure

Presenting Author: Tsian Caesar, D.O, Resident Physician, Jersey Shore University Medical Center

Introduction

Massive pulmonary embolism (PE) during pregnancy or abortion care is rare, but when it occurs, it can trigger sudden and catastrophic hemodynamic collapse, especially in patients with significant risk factors like morbid obesity. Pregnancy itself produces a prothrombotic state which compounds vulnerability to acute PE. Intraoperative transesophageal echocardiogram (TEE) allows rapid diagnosis of right ventricular obstruction and can quickly direct resuscitative strategies when PE is the suspected cause of cardiac arrest. When thrombolysis is not feasible or insufficient, catheter directed thrombectomy becomes a life-saving intervention. This case demonstrates the value of rapid diagnosis, multidisciplinary coordination, and decisive endovascular management given the patient's clinical presentation.

Case Presentation

37-year-old woman (G8P6016) with chronic hypertension and morbid obesity (BMI 59) presented for dilation and evacuation at 15 weeks for fetal demise in a hospital-attached ambulatory surgical center. She underwent induction with midazolam, fentanyl, and propofol with GETA. Approximately 20 minutes into the procedure, she developed profound hypotension that rapidly progressed to cardiac arrest. CPR was initiated, and cardiac anesthesia and cardiothoracic surgery were called emergently. Intraoperative TEE by cardiac anesthesiology revealed a massive PE with a large thrombus burden in the right ventricle. ECMO was considered but deferred due to inability to safely place femoral cannulas given the patient’s body habitus. Return of spontaneous circulation (ROSC) was achieved after 39 minutes with high-dose epinephrine infusion and high-quality CPR. A radial arterial line and right internal jugular 9 Fr Cordis were placed, and cardiology mobilized the patient to the catheterization lab for emergent pulmonary thrombectomy aspiration. The patient was transported on high dose Epi gtt and receiving cryoprecipitate infusion. Upon transfer to the cath lab table, the patient again became severely hypotensive. Repeat TEE demonstrated disappearance of the RV thrombus and worsening right-ventricular dysfunction, suggesting further embolization. Interventional cardiology accessed the pulmonary artery and performed aspiration thrombectomy with immediate hemodynamic improvement, allowing weaning to low-dose epinephrine. The patient was then admitted to the cardiac ICU, extubated the following morning with no neurologic sequelae, and discharged home on postoperative day four.

Discussion

Massive PE is a rare but catastrophic cause of intraoperative cardiac arrest, particularly in gravid patients with elevated thrombotic risk. In this case, rapid TEE evaluation enabled immediate identification of obstructive shock and guided targeted intervention when conventional options were limited by body habitus. Coordinated multidisciplinary action between anesthesiology, cardiology, and cardiothoracic surgery allowed timely pulmonary artery thrombectomy, resulting in swift hemodynamic improvement and full

neurologic recovery. This case illustrates how rapid diagnosis, multidisciplinary action, and adaptability to patient-specific constraints are critical determinants of survival in PE-related arrest.

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#FR 3 – Kiosk 11 – Poster 3

Coronary Confusion: A Rare Case of a Right Coronary Artery Aneurysm

Presenting Author: Jason E. Lieber, M.D., Resident Physician, University of Rochester Medical Center

Introduction

Coronary artery aneurysm (CAA) is a vascular malformation characterized by focal enlargement of a coronary segment with a diameter greater than 1.5 times the adjacent segment. It most often involves the right coronary artery (RCA) and can be saccular, in which the transverse measurement exceeds the longitudinal, or fusiform, wherein the opposite is true 1. CAAs can cause significant issues by compressing the pulmonary artery, worsening obstructive coronary artery disease (CAD), and rupturing which can lead to cardiac tamponade and cardiovascular collapse. The principal etiology in adults is atherosclerosis, although other systemic inflammatory diseases such as systemic lupus erythematosus have been implicated. Here we present the case of a 56-year-old male who was incidentally found to have an RCA aneurysm while undergoing a chest pain work up, resulting in a coronary artery bypass graft (CABG) and aneurysm ligation.

Case Presentation

A 56-year-old male with a past medical history significant for hypertension, diabetes mellitus, CAD, and MRSA bacteremia presented to an outside hospital complaining of chest pain and malaise. An echocardiogram revealed concentric left-ventricular hypertrophy with an ejection fraction (EF) of 53%, mild right ventricular hypokinesis, and a pericardial effusion without right ventricular (RV) diastolic collapse. CT demonstrated a 5.6 cm ovoid area of hypoattenuation inseparable from the right atrium concerning for a mycotic aneurysm. Repeat echocardiogram after the patient was found to be dyspneic with an elevated lactate demonstrated tamponade and cardiogenic shock, which was treated with a pericardial window. Once stabilized, he returned to the operating room for a triple-vessel CABG and RCA ligation. Intraoperatively, the RCA demonstrated severe calcification with complete obstruction and the aneurysm contained a small amount of pus and infected tissue. Pre-procedure transesophageal echocardiography (TEE) demonstrated mild-moderate right ventricular dysfunction which progressed to moderate dysfunction post-procedure. He tolerated the procedure well and was transferred to the ICU intubated and requiring hemodynamic support with vasopressors. He was extubated and weaned off pressors on post-operative day two.

Discussion

CAA is a rare clinical entity with significant perioperative considerations, including tamponade, right atrial/ventricular compression with nodal dysfunction, and coronary ischemia. Furthermore, RV compression can lead to extreme pre-load sensitivity wherein an inhalational induction to maintain spontaneous ventilation may be favorable from a hemodynamic perspective 2. Our patient developed tamponade and cardiogenic shock requiring an emergent pericardial window, demonstrating the importance of early recognition of CAAs and their potential to rupture/leak. He did well intraoperatively during his subsequent CABG and RCA ligation but developed worsening RV dysfunction and required

vasopressor support postoperatively. His clinical course highlights the importance of both educating anesthesia residents and cardiothoracic fellows on the presentation and management of CAA, and on the vital role of TEE in assessing the anatomy and resulting physiologic concerns of these malformations to best guide intraoperative decision-making.

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#FR 3 – Kiosk 11 – Poster 4

Persistent Left-sided SVC in a Donor Heart

Presenting Author: Hannah Thornburg, MD, Fellow - Adult Cardiothoracic Anesthesiology, University of Utah

Introduction

Persistent left superior vena cava is a rare congenital anomaly that can go undiagnosed in asymptomatic patients, but has considerable implications for surgical technique and route of myocardial protection in heart transplantation. We present a case of previously unidentified persistent left SVC in a donor heart for a patient undergoing orthotopic heart transplant.

Case Presentation

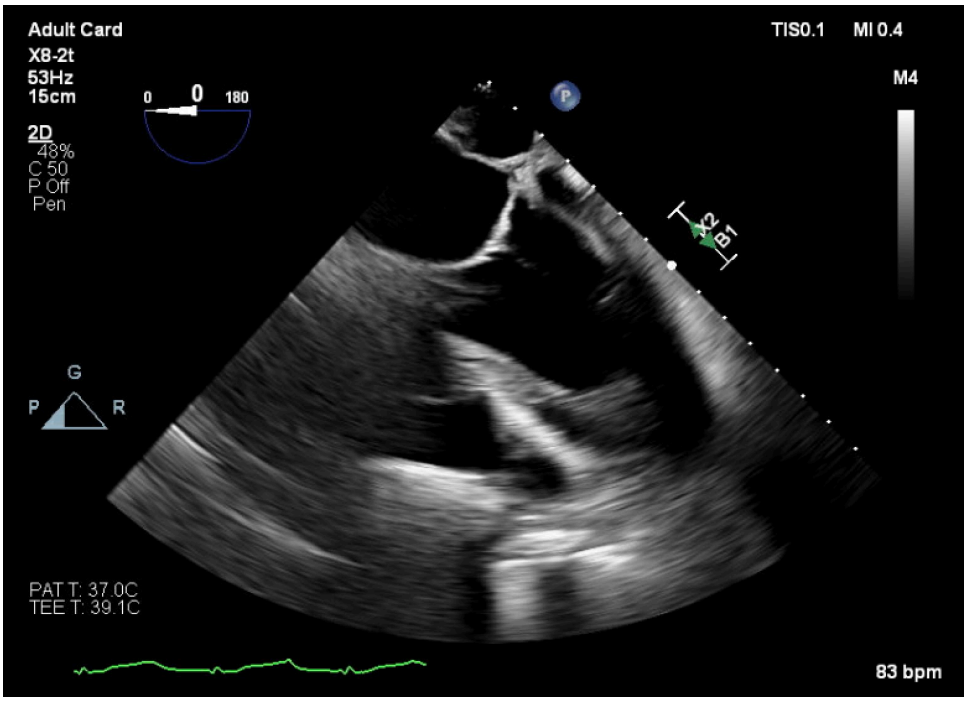
A 61 year old male with a history of ischemic cardiomyopathy and Factor V Leiden was brought to the operating room for orthotopic heart transplant. Induction of anesthesia and initiation of cardiopulmonary bypass were uneventful. On initial examination of the donor heart no anatomic abnormalities were noted. While weaning from cardiopulmonary bypass, the coronary sinus was noted to be very large and biventricular systolic function was extremely poor. In addition, there was significant bleeding around the innominate vein after removal of scarred ICD leads. Cardiopulmonary bypass was re-initiated. An intra-aortic balloon pump was placed and the SVC-innominate vein junction was repaired. Weaning from bypass was again attempted, again with very poor systolic function (EF 15%), and the patient was transitioned to central VA ECMO. After giving roughly 75% of the protamine dose, the heart was lifted to check for anastomotic competence, and a sudden large amount of dark blood filled the pericardium with an associated acute drop in blood pressure and ECMO flow. Protamine was stopped, the bypass circuit was noted to contain thrombus, and a new bypass circuit was brought to the room and bypass was re-initiated. During this process there were several minutes of severe hypotension. With further investigation, a persistent left SVC was noted on the donor heart which had not been recognized on donor echocardiography or during the procurement process. The persistent left SVC was oversewn, and the patient was again weaned from bypass and placed on central VA ECMO. Thrombus was noted in the left atrium on TEE. The left atrium was opened and thrombus was removed. The patient was taken to the ICU in critical condition where on post-op day 1, the left pupil was found to be fixed and dilated. CT head showed multiple large bilateral infarcts, diffuse cerebral edema, and 5 mm leftward midline shift. The patient was transitioned to comfort care and expired on post-op day 2.

Discussion

This was a complex case with multiple challenges including primary graft dysfunction and significant bleeding from both vascular injury while removing ICD leads as well as previously unrecognized persistent left SVC in the donor heart. Because persistent left SVC is commonly associated with other congenital abnormalities, surgical techniques for heart transplant in recipients with persistent left SVC are well described in the literature. Fewer examples of donor hearts from patients with persistent left SVC exist in the literature, and because isolated persistent left SVC is often asymptomatic, it is possible that it may not be

identified prior to procurement of a heart for transplantation. It is important to consider persistent left SVC when a dilated coronary sinus is identified as there are important implications for surgical technique.

Images



Dilated Coronary Sinus in Mid-Esophageal 4 Chamber View

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#FR 3 – Kiosk 11 – Poster 5

Right Ventricular Assist Device Reconfiguration resulting in Total Lung Bypass for the Management of Pulmonary Edema and Refractory Hypoxemia

Presenting Author: Tyler Austin, MD, Resident Anesthesiologist, University of Nebraska Medical Center

Introduction

As the prevalence of heart failure climbs, the need for advanced therapies, such as left ventricular assist device (LVAD), right ventricular assist device (RVAD), and heart transplant, will increase. RVAD placement is generally a beneficial, but temporary, measure for a failing right ventricle (RV). RVADs can be reconfigured in various ways to aid in the management of RV failure, including the use of an oxygenator for hypoxemia. Here, we describe the reconfiguration of an RVAD to achieve total lung bypass for the management of massive pulmonary edema and refractory hypoxemia.

Case Presentation

A 66-year-old male with ischemic cardiomyopathy and a left ventricular (LV) aneurysm presented to the University of Nebraska Medical Center (UNMC) from an outside facility in cardiogenic shock with severe mitral regurgitation after an unsuccessful transcatheter edge-to-edge repair and Impella CP placement. After transfer to UNMC, he underwent evaluation for advanced therapies and was deemed a candidate for durable LVAD. On hospital day 8, he underwent LVAD and central RVAD placement, LV aneurysm repair, and mitral valve replacement. The RVAD was placed in standard fashion with the inflow cannula in the right atrium (RA) and the outflow cannula in the main pulmonary artery (mPA). A second inflow cannula was placed into the RA via the left common femoral vein, allowing separation from cardiopulmonary bypass (CPB) after the initial attempt to wean from CPB resulted in decreased RVAD flows, pulmonary edema, and hypoxemia. On hospital day 9, he returned to the operating room (OR) for an RVAD reconfiguration for massive pulmonary edema. An additional cannula was inserted into the left atrium via the right superior pulmonary vein to serve as the new outflow cannula, while the previous outflow cannula in the mPA was converted into a third inflow cannula to achieve total lung bypass, thereby improving pulmonary edema and oxygenation. Thrombosis of the RVAD cannulas, resulting in critical low flows, led to an additional RVAD reconfiguration on hospital day 15. Though this improved RVAD flow, his coagulopathy, lung injury, and other complications led to the decision to withdraw care. The patient died on hospital day 16.

Discussion

RVADs are effective modes of support for patients with RV failure with or without hypoxia. While only a single venous drainage cannula is usually necessary to aid in bypassing the failing RV, additional drainage cannulas may be needed to manage hypoxia or pulmonary hypertension [1]. In certain patients, pulmonary edema can develop, resulting in significant lung injury. To allow for lung recovery, total lung bypass can be achieved via an RVAD through right atrial/pulmonary arterial drainage and pulmonary vein/left atrial return. RVADs are not without complications, though, such as thrombosis of the circuit. Managing anticoagulation in patients after LVAD/RVAD placement can be challenging, as upwards of 30-60% of patients are

coagulopathic [2]. As evidenced in this case, morbidity and mortality are high, often with 30-day survival less than 65% [3]. Nonetheless, this case demonstrates a unique RVAD reconfiguration that allows for total lung bypass to manage massive pulmonary edema with associated refractory hypoxemia.

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#FR 3 – Kiosk 11 – Poster 6

Management of A Patient with Severe Carcinoid Heart Disease Undergoing Triple Valve Replacement

Presenting Author: Truc Nguyen, Resident Physician, University of Iowa Health Care

Introduction

For 20% of patients with neuroendocrine tumors, carcinoid heart disease might be the first manifestation of carcinoid syndrome. It typically causes fibrotic endocardial changes to the right heart valves with rare involvement of the left (10-15%). Besides the common post-bypass challenges, valve replacement surgery in these patients is complicated with significant right heart dysfunction and the risk of carcinoid crisis. This case presents a patient with severe carcinoid heart disease who underwent triple valve replacement and developed significant post-bypass hemodynamic instability.

Case Presentation

A 64-year-old female with symptomatic carcinoid heart disease presented for tricuspid and aortic valve replacement prior to tumor resection. Pre-operative TEE showed severe tricuspid regurgitation, moderate-severe aortic insufficiency, enlarged right heart with preserved biventricular function, and a patent foramen ovale. Prior to induction, an octreotide drip was started at 50 mcg/h. Patient tolerated induction with midazolam, fentanyl, etomidate, and rocuronium and maintenance with isoflurane without major hemodynamic changes. However, during central line placement in Trendelenburg, she quickly decompensated due to the acute increase in preload. Octreotide was increased to 100mcg/h with the start of an epinephrine infusion. Intra-operative TEE also showed significant pulmonic regurgitation which necessitated pulmonic valve replacement. The triple valve replacement led to prolonged cross clamp and bypass times of 150 and 183 minutes. Patient separated from bypass on epinephrine, milrinone, and inhaled nitric oxide for moderately reduced right ventricular function. She also required vasopressin and progressive escalation of norepinephrine infusion. The persistent hypotension continued despite multiple vasopressors and calcium boluses. Hemodynamic stability was finally achieved with methylene blue. Patient was admitted to the ICU post-operatively. She was extubated on post of day (POD) 1. Epinephrine and milrinone were weaned off on POD 3 and POD 4, respectively. She was discharged home on POD 6.

Discussion

Management of patients with carcinoid tumor cautions against the use of exogenous catecholamines to avoid the release of kallikrein and, subsequently, bradykinin which worsens carcinoid symptoms.[1] However, inotropes and vasopressors are routine management of cardiac surgery patients after bypass. A case series of 11 patients with severe carcinoid heart disease who underwent multivalve surgery supports the administration of catecholamines to wean patients off bypass, particularly in the presence of cardiac dysfunction.[1] Similarly, recent studies have not shown paradoxical hypotension or prolonged carcinoid crisis in patients receiving β-adrenergic agonists.[2] The refractory hypotension in this patient, however, did raise concern for carcinoid crisis versus refractory vasoplegia. The absence of hallmark features such as bronchospasm, flushing, or facial edema lowered the suspicion for carcinoid crisis. Therefore, inotropes

were continued to support cardiac function while management effort was focused on treating vasoplegia with pressors and methylene blue, which has also been shown as an adjunct therapy in carcinoid crisis due to the indirect inhibition of bradykinin.[3]

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#FR 3 – Kiosk 11 – Poster 7

Perioperative Management of a Left Ventricular Papillary Fibroelastoma Attached to the base of the Posteromedial Papillary Muscle

Presenting Author: Brian O'Donnell, MD, Fellow, Hospital of the University of Pennsylvania

Introduction

Papillary fibroelastomas (PFE) are small (often < 1.5 cm) benign tumors which usually arise on valvular surfaces (> 80%) and can carry risk of embolization. Despite their valvular preponderance, PFEs can be found on nearly all endocardial surfaces, including ventricular walls, the aortic root, the atria, chordae and the papillary muscles. Sub-optimally characterized on other imaging modalities, PFEs can be identified on TEE with high certainty. PFE mobility is a high predictor of death or non-fatal embolization, and complete resection is recommended. We describe the anesthetic and surgical management of a hyper-mobile PFE attached to the base of the posterior papillary muscle.

Case Presentation

A 72-year-old woman with a PMH of hypertension presented to cardiac surgery after TTE (ordered in the setting of an elevated calcium score) incidentally showed a mobile 2.2 cm by 1.1 cm LV mass attached to the posterior papillary muscle. TTE differential diagnosis included LV thrombus, myxoma, sarcoma, and PFE. The patient was admitted for pre-surgical evaluation and started on systemic anticoagulation due to concern for thrombus and embolization. LHC was unremarkable. Cardiac MRI was notable for a 7 mm distinct spherical mass near the lateral wall of the LV likely representing a myxoma or fibroelastoma, though radiologists noted the location was atypical for these pathologies. After an interdisciplinary discussion, the patient was brought to the OR by cardiac surgery for TEE exam for better characterization of the mass, followed by open surgical excision. Patient met criteria for surgical excision due to the PFE being left-sided, larger than 1 cm, and mobile. After placement of a pre-induction arterial line, the patient was induced with lidocaine, propofol, fentanyl, and rocuronium and intubated. A central line and PA catheter were placed uneventfully. TEE showed normal bi-ventricular function, no significant valvular disease and a 1.4 cm by 0.8 cm hypermobile, hyperechoic mass attached to the base of the posterior papillary muscle. After discussion with the surgeon regarding feasibility of surgical approaches, the patient underwent sternotomy, initiation of cardiopulmonary bypass (CPB), and mass excision via a transverse aortic incision and retraction of the aortic valve. Following excision and closure, the patient was weaned from CPB without issue. Post-bypass TEE showed preserved bi-ventricular function and complete removal of the mass and unchanged valvular function. The patient's post-operative course was uneventful. Pathology confirmed PFE.

Discussion

This case underscores the utility and importance of TEE in identifying and characterizing cardiac masses. The discrepancy in size, thrombus involvement, and location of the mass on different modalities, is likely a consequence of one of the defining features of PFEs: their hypermobile, fluid structure which is well visualized on live TEE. TEE can also confirm the need for surgery, given hypermobility is our best predictor for

death or non-fatal embolization. Additionally, TEE can aid in decision-making regarding the approach of surgical excision. Based on its location, our surgical colleagues were confident the resection could be completed via a sightline into the left ventricle through the aortic valve.

Images

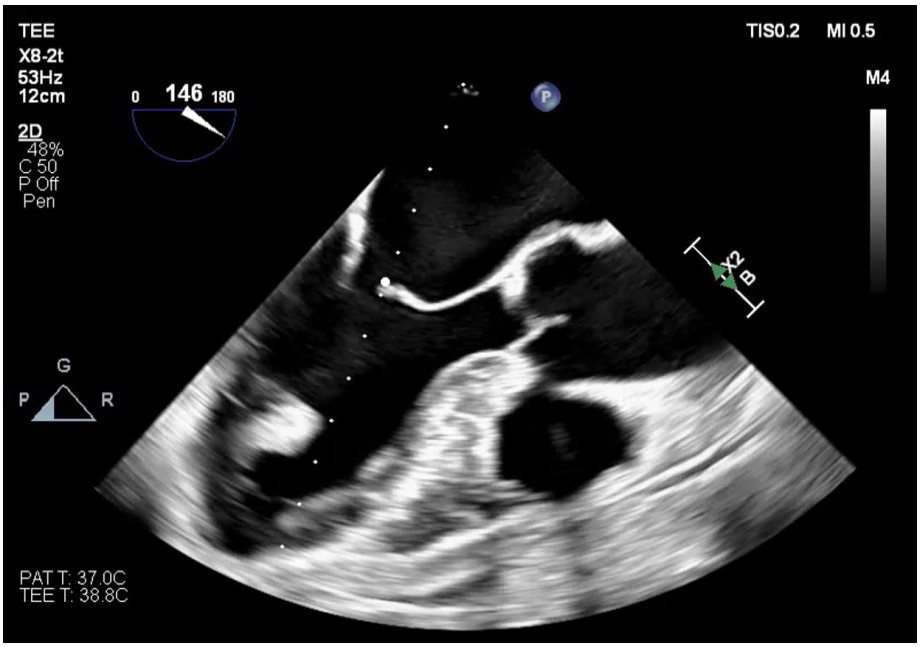


Figure 1: TEE Modified ME AV LAX view showing PFE location and viability of transaortic approach.

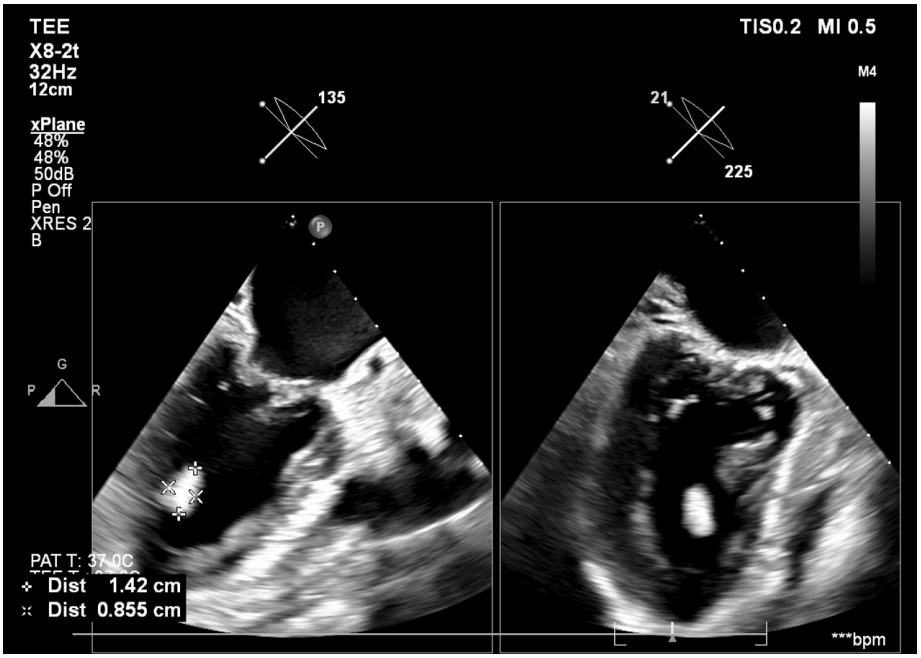


Figure 2: TEE x-plane of LV capturing sizing of hypermobile PFE.

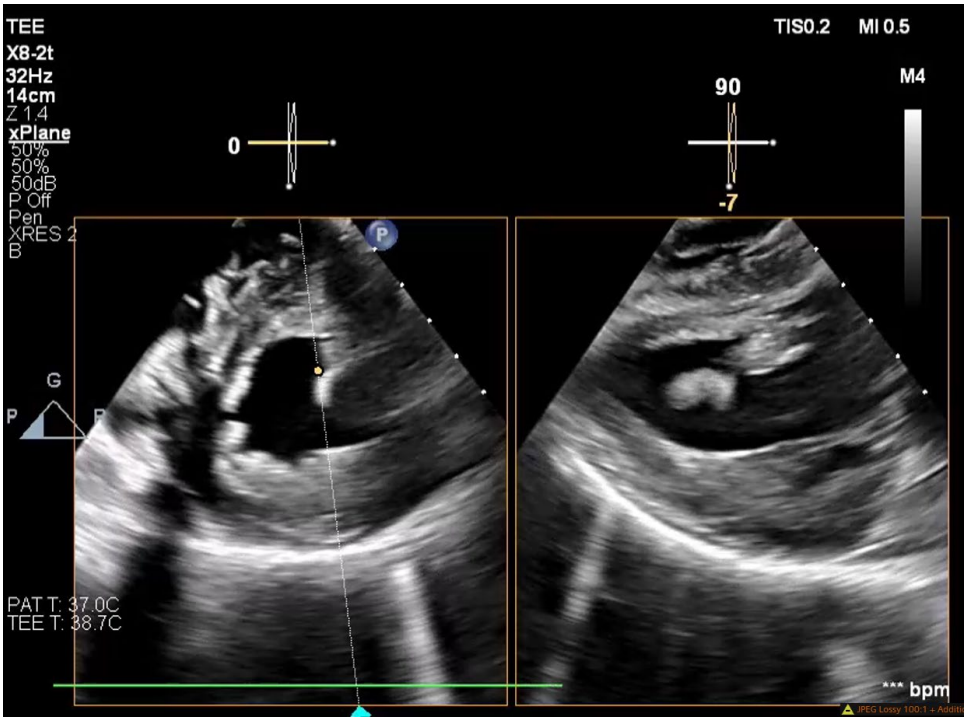


Figure 3: TEE Modified TG LV X-plane showing PFE association with the base of the posteromedial papillary muscle.

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#FR 3 – Kiosk 12 – Poster 1

Right Place, Wrong Time: Carcinoid Crisis After Transcatheter Tricuspid Valve Replacement

Presenting Author: Dimpie Shah, MD, Resident Physician, Montefiore Medical Center

Introduction

Carcinoid heart disease presents major perioperative challenges due to risks of hemodynamic instability, mediator release, and unpredictable hypercoagulability. Although octreotide is administered to blunt carcinoid crisis, atypical manifestations may still occur. We report a case of catastrophic right-sided intracardiac thrombosis immediately following transcatheter tricuspid valve replacement (TTVR), suspected to be triggered by carcinoid crisis despite standard prophylaxis.

Case Presentation

A 77-year-old man with chronic diastolic heart failure, mild-to-moderate pulmonary hypertension, prostate cancer, and metastatic carcinoid tumor to the liver and kidney was scheduled for TTVR for severe tricuspid regurgitation with annular dilation. Preoperative evaluation confirmed carcinoid valve pathology without active symptoms. A prophylactic octreotide infusion(150 mcg/hr) was initiated. The procedure proceeded uneventfully with stable hemodynamics and complete resolution of tricuspid regurgitation after device deployment and extubation in the hybrid operating suite. Minutes later, he developed progressive hypotension and loss of lower extremity pulses. Despite vasopressors and inotropes, he progressed to cardiac arrest. CPR was initiated with emergent reintubation and additional 1,250 mcg octreotide during resuscitation. Point-of-care transthoracic echocardiography showed no pericardial effusion. Transesophageal echocardiography revealed extensive thrombus filling the right atrium and right ventricle with near-complete obstruction of chamber volumes. No procedural factors or pre-extubation findings suggested thrombus formation. The presentation was most consistent with carcinoid crisis–induced hypercoagulability causing fulminant right-sided thrombosis.

Discussion

Carcinoid crisis is typically associated with severe vasodilation, bronchospasm, and cardiovascular collapse; however, abrupt serotonin and vasoactive mediator release can also trigger profound thrombosis through platelet activation and endothelial dysfunction. Patients with liver metastases are at increased risk for coagulopathy. The sudden, massive RA/RV clot burden in this case suggests a hypercoagulability-dominant crisis over classic vasoplegia. This case underscores several key points. Patients with metastatic

carcinoid disease may remain vulnerable to crisis despite standard-dose octreotide. Right-sided structural heart procedures may provoke mediator release even without tumor manipulation. Current prophylaxis may be inadequate for high-burden disease, raising consideration for intensified octreotide dosing or more aggressive perioperative anticoagulation. Additionally, patients undergoing TTVR may experience acute right ventricular stress due to sudden elimination of tricuspid regurgitation, which normally functions as a low-resistance “pop-off” valve. Selected patients may benefit from anticipatory inotropic or pulmonary vasodilator therapy, though supporting evidence is limited. This rare but fatal complication highlights the need for heightened vigilance and rapid recognition of atypical carcinoid crisis manifestations in post-TTVR instability. Further study is needed to refine prevention and management strategies for hypercoagulable carcinoid crises during advanced structural heart procedures.

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#FR 3 – Kiosk 12 – Poster 2

Intraoperative TEE Identification of Aortic Root Involvement in an Acute Type A Dissection

Presenting Author: Mihir Soni, DO, Cardiothoracic Anesthesiology Fellow, University of Rochester

Introduction

Acute Type A aortic dissection in young patients is uncommon, and accurate evaluation of aortic root involvement is crucial for selecting the appropriate surgical strategy. Although CT angiography (CTA) is the standard initial diagnostic modality, intraoperative transesophageal echocardiography (TEE) offers real-time, high-resolution assessment that may reveal proximal aortic pathology not fully appreciated on preoperative imaging. [1]

Case Presentation

A 26-year-old female with a history of methamphetamine use disorder, poor medical follow-up, and prior eclampsia complicated by seizure presented to an outside hospital with acute chest pain. CTA demonstrated an acute Type A aortic dissection involving the ascending aorta and arch, extending from the sinotubular (ST) junction into the distal thoracic aorta, initially appearing to spare the aortic root and valve. She was emergently taken to the operating room for ascending aortic repair. Following induction, comprehensive intraoperative TEE revealed significant aortic root dilation and a dissection flap originating near the ST junction with extension into the non-coronary sinus, associated with mild aortic insufficiency—findings that were not clearly appreciated on CTA. These discrepancies prompted immediate communication with the surgical team and revision of the operative plan to include aortic root repair in addition to ascending aortic replacement. Cardiopulmonary bypass was initiated, and the patient underwent ascending aortic and aortic root repair under deep hypothermic circulatory arrest (DHCA) without complication. Postoperative TEE demonstrated no residual intimal flaps or tears within the aortic root, a successfully graft-replaced proximal ascending aorta, and a persistent yet stable distal dissection flap in the descending thoracic aorta.

Discussion

CTA is highly sensitive for diagnosing acute aortic dissection; however, its ability to characterize aortic root involvement and to quantify associated aortic insufficiency is more limited, particularly when motion artifact or hemodynamic instability is present.[3] In contrast, TEE provides excellent diagnostic accuracy—with sensitivity and specificity often exceeding 95%—for identifying proximal aortic disease, defining intimal flap extension, and assessing valvular competence.[2][3] In this case, intraoperative TEE provided critical anatomic clarification that directly altered surgical management. This highlights the essential role of TEE in optimizing operative decision-making for acute Type A dissections, especially in young patients whose pathology may be more complex or atypical than initially appreciated. This case demonstrates how intraoperative TEE can reveal actionable aortic root pathology not fully detected on CTA, leading to timely modification of the surgical plan. TEE remains essential for intraoperative decision-making in acute aortic emergencies.

Images

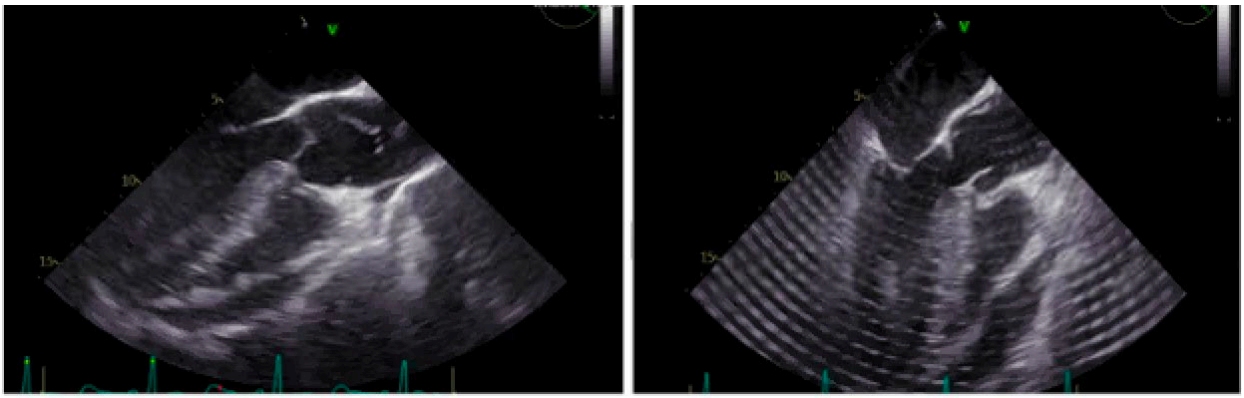


Figure 1 and 2: Mid-esophageal long-axis view demonstrating dissection flap above STJ

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#FR 3 – Kiosk 12 – Poster 3

Left Ventricular Free Wall Rupture

Presenting Author: Nola Darden, D.O, Fellow, Penn State Health

Introduction

Left ventricular free water rupture (LVFWR) is one of the most lethal complications following acute myocardial infarction (MI). Although reperfusion strategies have reduced its incidence to 0.01-0.5%, LVFWR still carries a high mortality rate. The clinical presentation varies depending on the type of rupture and can range from catastrophic blowout, characterized by active bleeding leading to massive hemopericardium, to the oozing type, which involves slower, progressive bleeding. Prompt diagnosis and emergent surgical repair remain the standard of care and definitive therapy. Echocardiography is critical for diagnosis of LVFWR in the emergent setting especially in hemodynamically unstable patients.

Case Presentation

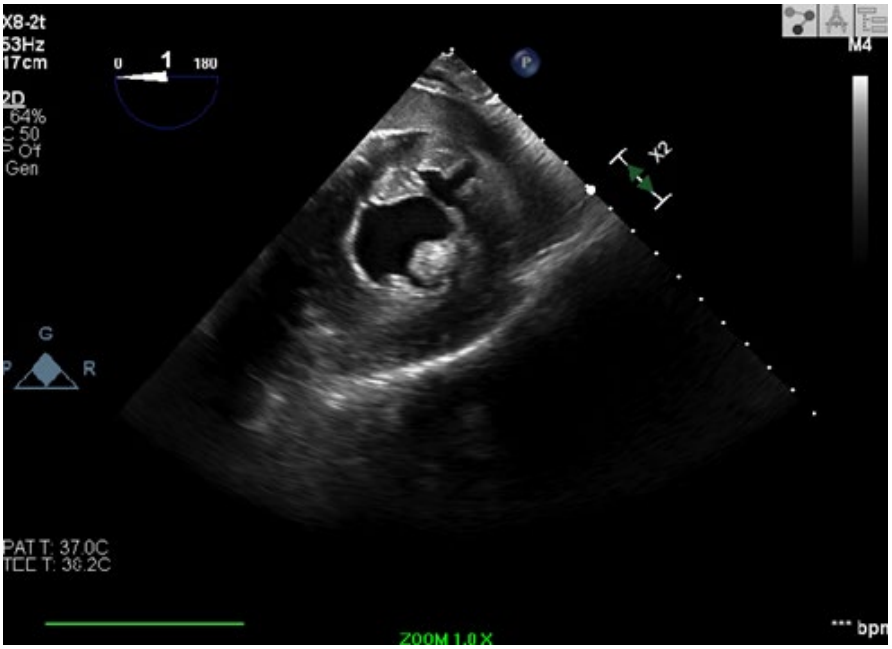
We present a 65 year old man with no known past medical history who was brought into the hospital with signs concerning for a stroke. Initial evaluation showed ST elevations in leads II, III, and aVF, concurrent with depression in V1-V2. POCUS was performed revealing a large pericardial effusion and raised concern for an aortic dissection and a CT scan was performed. Preliminary results from the CT scan indicated a Type B dissection alongside an acute MCA occlusion. As the patient continued to decompensate in the ED, a TEE was performed and showed findings concerning for aortic root dissection and a worsening pericardial effusion. A pericardial drain was placed resulting in an improvement in the patient's hemodynamics. The patient was then taken emergently to the operating room for repair of aortic dissection. A repeat intraoperative TEE was performed, which notably did not show a dissection flap, but demonstrated a LVFWR with approximately 1 cm of clot in the pericardial space. When the chest was opened the aorta appeared normal. Under echocardiographic guidance, the surgeon identified a transmural infarct with an epicardial hematoma located in the basal aspect of the posterior wall of the left ventricle. A patch repair of the free wall rupture was successfully performed. The patient made a full cardiac recovery after prolonged hospitalization due to MCA stroke and was discharged to a rehab facility.

Discussion

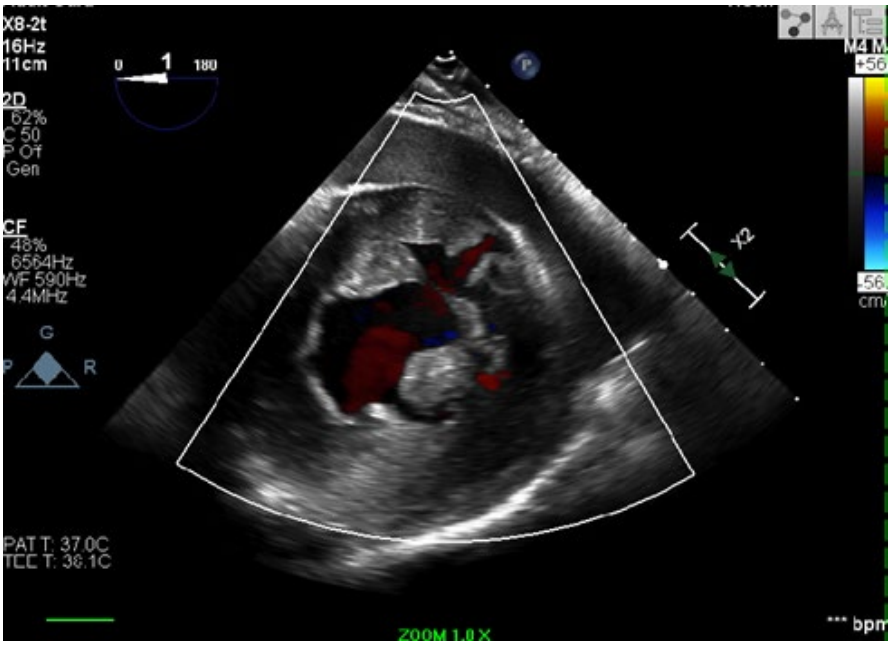
Despite significant advancements in reperfusion strategies for myocardial infarctions, LVFWR remains a rare but highly lethal complication. It typically occurs 3-5 days after MI, and even with surgical intervention, operative mortality can reach 32%. Early recognition is critical for diagnosing LVFWR in patients with acute hemodynamic instability following an MI. This case underscores the importance of having a high index of suspicion of LVFWR when a patient presents with a myocardial infarction and a pericardial effusion. It also highlights the dangers of having an anchoring bias when looking at imaging such as CT or prior echocardiography. In this case, what appeared on TEE was reverberation artifact – caused by ultrasound reflections between parallel structures – producing false echo images. Reverberation artifacts are common in the ascending aorta being observed in 44-55% of studies. To minimize diagnostic errors,

echocardiography should be performed using multiple planes and complementary imaging modalities to aid in confirming the diagnosis.

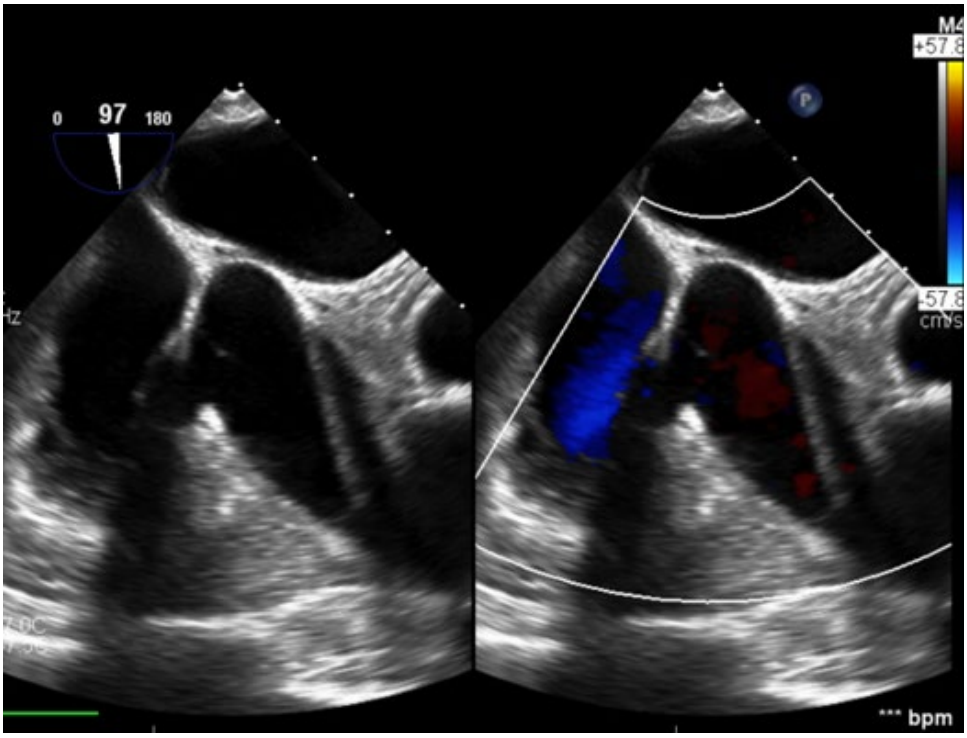
Images



LVFWR



LVFWR with Colorflow doppler



Ascending aorta with reverberation artifact

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#FR 3 – Kiosk 12 – Poster 4

Perioperative management of a patient on cardiac contractility modulation therapy undergoing cardiac surgery

Presenting Author: Paul P-H. Yen, MD, Fellow, Stanford Health Care

Introduction

Cardiac contractility modulation (CCM) is an implantable device therapy for heart failure patients with reduced ejection fraction, who are otherwise ineligible for cardiac resynchronization therapy (CRT). Only about 10000 patients worldwide have undergone CCM as of May 2024. We present the novel case of a CCM patient undergoing tricuspid valve replacement (TVR).

Case Presentation

This is a 60-year-old man with a history of dilated cardiomyopathy (LVEF 40%), dual-chamber implantable cardioverter-defibrillator (ICD), and remote mitral valve repair. Given his ineligibility for CRT, a CCM device was implanted, but his functional capacity continued to decline. Transthoracic echocardiography (TTE) revealed torrential eccentric tricuspid regurgitation (TR). He was not a candidate for transcatheter intervention and was ultimately booked for a TVR. Under general anesthesia, a pulmonary artery catheter (PAC) demonstrated equalization of the right atrial and ventricular pressures. A sterile magnet was placed over the ICD. Redo sternotomy was uneventful. On cardiopulmonary bypass (CPB), a 33 mm bioprosthetic valve was implanted. Intraoperative findings included an anterior leaflet tear and a septal leaflet perforation. The patient was weaned off CPB on a moderate dose of dobutamine. Post-CPB, transesophageal echocardiography (TEE) revealed mildly reduced biventricular function and a well-functioning valve. He was extubated later that day and his postoperative course was uneventful. Both CCM and ICD generators, along with their respective leads, were removed a week later.

Discussion

The primary mechanism of CCM is the regulation of intracellular calcium. It has been shown in several studies to improve functional capacity, reduce hospitalization, and even lower mortality. Biphasic, non-excitatory electrical signals are sent during the absolute refractory period of conduction; the resultant modulation of calcium channels helps to improve cardiac contractility. CCM is indicated in patients with New York Heart Association (NYHA) III/IV symptomatology and an LV ejection fraction of 25-45%, but who are not CRT candidates. Two leads are placed into the RV septal wall, with the generator implanted subcutaneously in the right chest to preserve left-sided venous access for defibrillators. CCM leads are standard pacemaker leads which are resistant to dislodgement during sternotomy and manipulation of the heart. CCM functions uniquely from all other cardiac implantable electronic devices. The CCM device is incapable of providing backup pacing for bradyarrhythmias. Continuous ECG monitoring will show multiple upward spikes in the RS segment, which generally have no effect on hemodynamics intraoperatively. Since CCM does not affect chronotropy or synchronization of the heart, hemodynamic parameters are unaffected by electromagnetic interference from surgical cautery. Magnet placement over the generator is therefore not

required. Moreover, CCM is MRI-safe. With further advancement of heart failure therapy, CCM will inevitably become more widespread and has recently been incorporated within a defibrillator system as well. Anesthesiologists should be familiar with the perioperative management of these devices during both cardiac and non-cardiac surgery.

Images

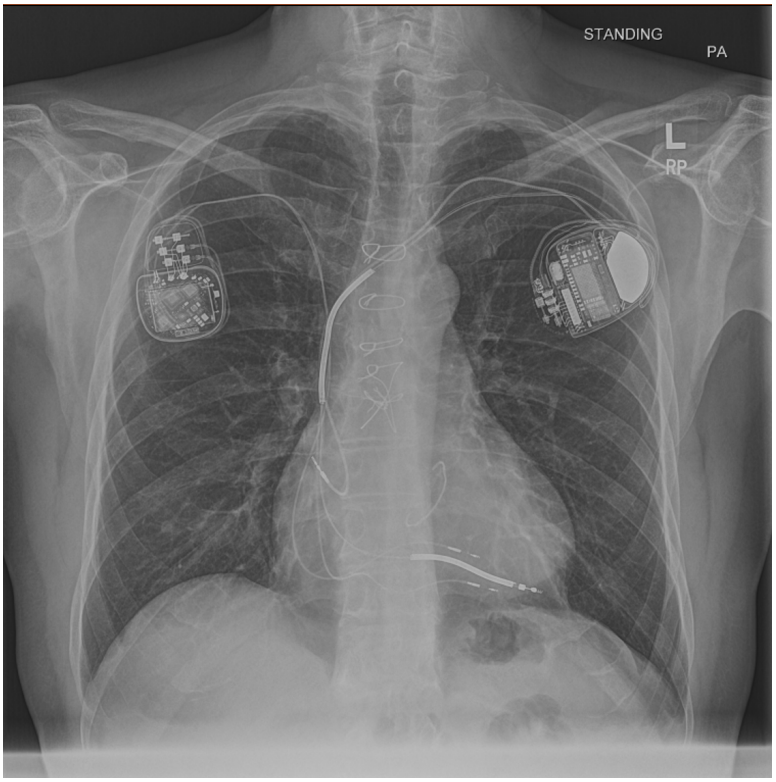


Figure 1. Preoperative chest x-ray demonstrating the ICD generator (left pectoris) and its two leads (right atrial and right ventricular), as well as the CCM generator (right pectoris) and its two leads (both right ventricular).

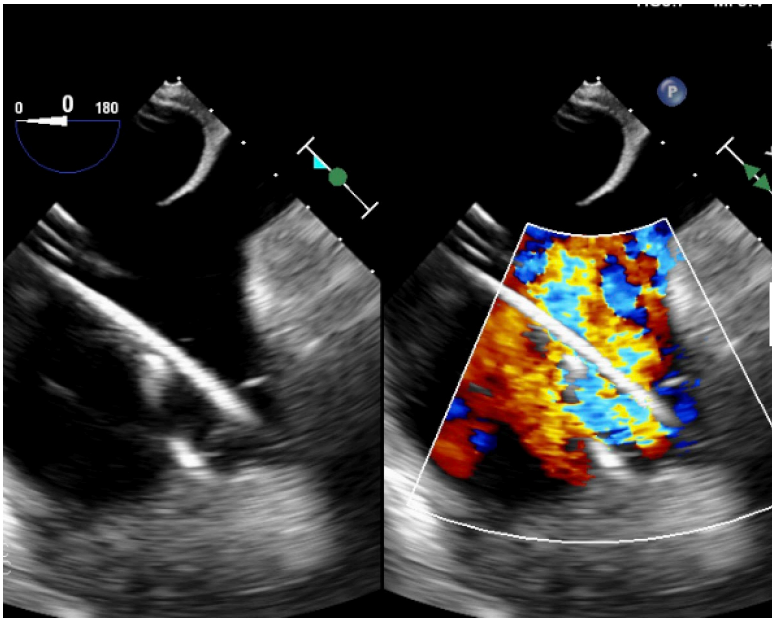


Figure 2. Mid-esophageal 4-chamber TEE view with color compare, demonstrating at least one device lead traversing the tricuspid valve in 2D (left), and at least severe TR by color flow Doppler (right).

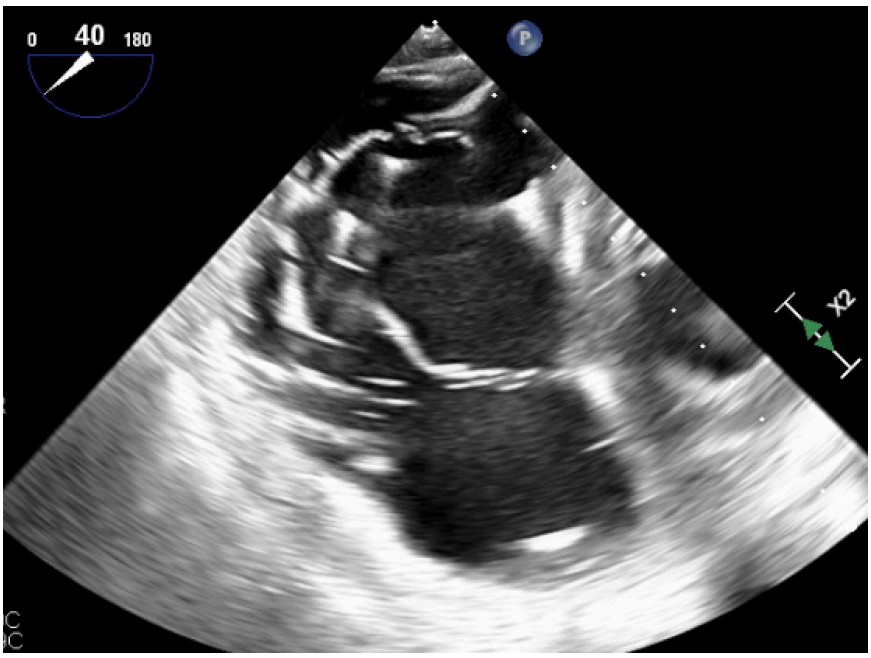


Figure 3. Modified transgastric short-axis TEE view of the RV, en face with the tricuspid valve, demonstrating the presence of three device leads in relation to the three valve leaflets.

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#FR 3 – Kiosk 12 – Poster 5

Inadequate Cardiopulmonary Bypass Flows During Intrapericardial Paraganglioma Resection Requiring Phentolamine

Presenting Author: Garrett M. Thomas, MD, Cardiac Anesthesia Fellow, Cleveland Clinic Foundation - Main Campus

Introduction

Paragangliomas (PGL's) are rare (incidence 0.04-0.95 per 100,000)[1], neural crest derived, vascular tumors which frequently arise from the sympathetic chain and secrete catecholamines. In extremely rare cases PGL's can originate within the pericardial sac[2]. We present a case in which a patient with a pericardial PGL experienced refractory hypertension on cardiopulmonary bypass (CPB) which significantly limited cannula flows. Following treatment with phentolamine adequate CPB flow was restored, allowing for successful cardiac surgery.

Case Presentation

A 67 year-old male with a medical history significant for hypertension and atrial fibrillation (AF) was discovered to have an extracardiac mass on CT. Imaging and elevated plasma normetanephrines >10x upper limit of normal confirmed the diagnosis of PGL and he was started on doxazosin. Three months after alpha-blockade treatment he underwent mitral and tricuspid valve repairs, MAZE, and resection of pericardial PGL with CPB. Despite excellent blood pressure (BP) control preoperatively, his high BP was extremely difficult to control while on cardiopulmonary bypass despite the use of nitroglycerin, nitroprusside, beta-blockers, and higher doses of isoflurane (Figure 1). This resulted in decreased CPB flow (maximum: 1.8L/min/m2) until phentolamine was administered on pump resulting in an improved cardiac index (CI) flow (>2.2L/min/m2) and adequate tissue perfusion. The patient was discharged on postoperative day #15 without any cardiac complications and without any prescribed antihypertensive medications.

Discussion

Careful BP management on CPB allows perfusionists to maintain appropriate CPB flow and tissue perfusion, preventing complications such as kidney injury, ventricular dysfunction, pulmonary edema, and neurological damage. Standard pharmacologic agents to combat hypertension on CPB include short acting vasodilators such as nitroglycerin, nitroprusside, and clevidipine as well as short acting beta-blockers like esmolol. This case demonstrates the importance of hypertension management on CPB and highlights the need to administer tailored medications to a patient's unique pathophysiology. In this case, our patient's preoperative alpha blockade was insufficient to control the response to catecholamines released with tumor manipulation during resection and he required phentolamine on CPB. Additionally, in the context of inadequate alpha-blockade the administration of beta-blockers may have exacerbated the hypertension. Cardiac anesthesiologists should be in close communication with perfusionists when taking care of patients with catecholamine secreting tumors to prevent complications arising from hypertension and tissue hypoperfusion during CPB. Common antihypertensive agents may not be effective in this population as

chronic exposure to catecholamines desensitizes the adrenergic receptors within the vascular smooth muscle, preventing clinically significant response to pure vasodilators3. Phentolamine is a short-acting, non-selective alpha-blocker (half-life 19 minutes) which provides a safe option for cardiac surgical teams when caring for patients with PGL's.

Images

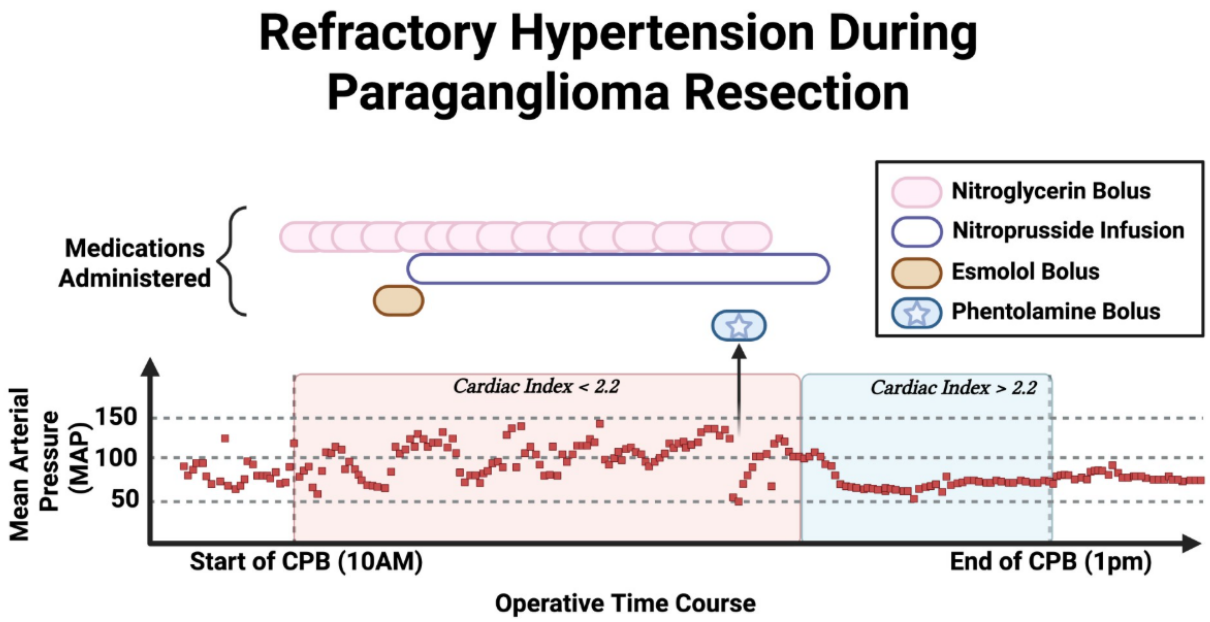


Figure 1. Depicts timing of anti-hypertensive medications, refractory hypertension, and response to phentolamine.

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#FR 3 – Kiosk 12 – Poster 6

An Unhappy Sequelae of Serotonin

Presenting Author: Yunjia Zhang, MD, Cleveland Clinic Foundation

Introduction

Carcinoid tumors are rare neuroendocrine neoplasms that may secrete vasoactive substances, including serotonin, histamine, and prostaglandins. Carcinoid heart disease results from the deposition of fibrous tissue on the endocardial surfaces, a rare sequelae affecting 1 in 300,000 individuals1. The tricuspid valve is most frequently affected, as serotonin is typically inactivated within the lungs. Left-sided involvement is far less common and generally occurs in the setting of right-to-left shunts, pulmonary metastases, or exceptionally high serotonin levels. Despite its rarity, the prognosis of carcinoid heart disease remains poor, with an estimated three-year survival of 31%2. Here, we present a case of extensive carcinoid heart disease involving all four cardiac valves, ultimately requiring replacement of each valve.

Case Presentation

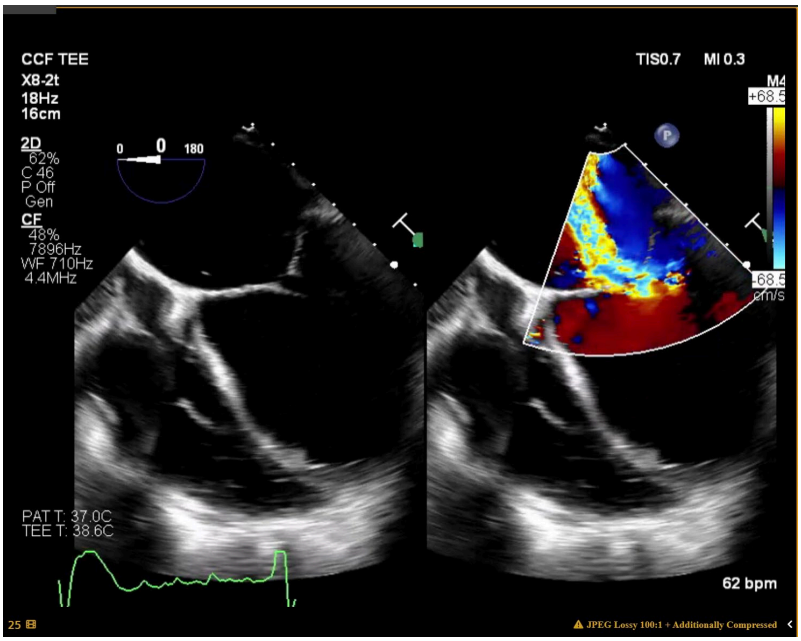
A 72-year-old woman with a history notable for HFrEF, NICM, CAD, and COPD was found to have severe regurgitation of the mitral, tricuspid, and aortic valves, as well as moderate stenosis of the mitral and pulmonic valves on TTE. The valves appeared thickened and restricted, raising suspicion for carcinoid heart disease. She underwent replacement of all four cardiac valves and a single-vessel coronary artery bypass. The patient's intraoperative course was complicated. Upon induction of anesthesia, the patient developed profound hypotension and ventricular tachycardia, requiring chest compressions, defibrillation, and emergent initiation of cardiopulmonary bypass. After several attempts to wean her from bypass, she exhibited severe biventricular systolic dysfunction, which prompted Impella support. Shortly after separation from bypass, she developed DIC and was found to have acute thrombi in the right atrium and ventricle with complete cessation of forward flow. Emergent recannulation to central ECMO was undertaken before transferring to the ICU. Her postoperative course was complicated by multiorgan failure, and she passed away four weeks later.

Discussion

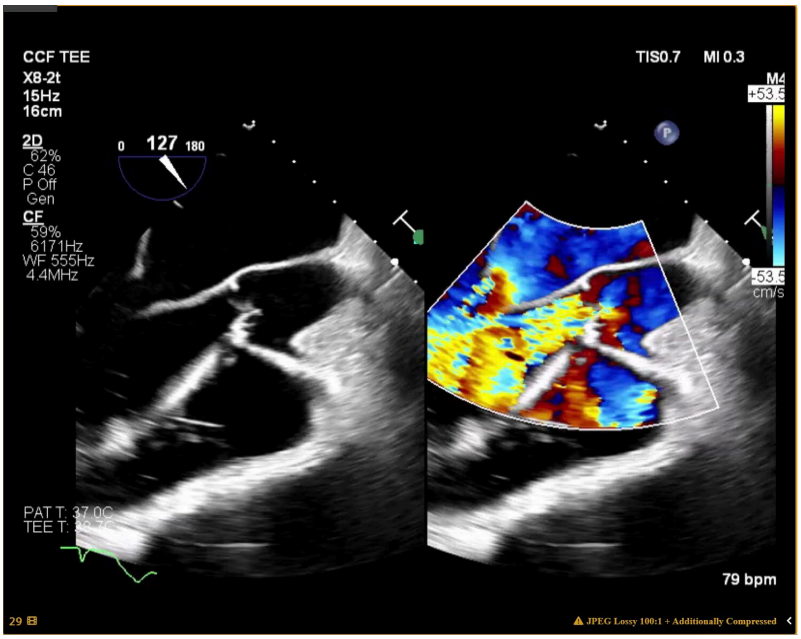
This patient's presentation demonstrates the profound hemodynamic consequences of severe valvular dysfunction secondary to carcinoid heart disease. The extent of her valvular pathology placed her at a significant risk for cardiovascular collapse with even small perturbations during anesthetic induction. Maintenance of preload, afterload, and contractility is essential in such patients. The simultaneous replacement of all four cardiac valves is rare and requires advanced surgical expertise. The physiological stress associated with cardiopulmonary bypass likely contributed to the patient's postoperative biventricular failure, necessitating mechanical circulatory support. Additionally, bypass-related coagulopathy precipitated DIC, placing the patient in a precarious state of both bleeding and thrombosis. The formation of right-sided intracardiac thrombi further complicated management by diminishing preload to the left side and compromising the Impella, contributing to her clinical decline. This case highlights the

complexity and fragility of patients with advanced carcinoid heart disease, particularly those undergoing high-risk cardiac surgery, demonstrating the importance of meticulous perioperative planning, and hemodynamic management.

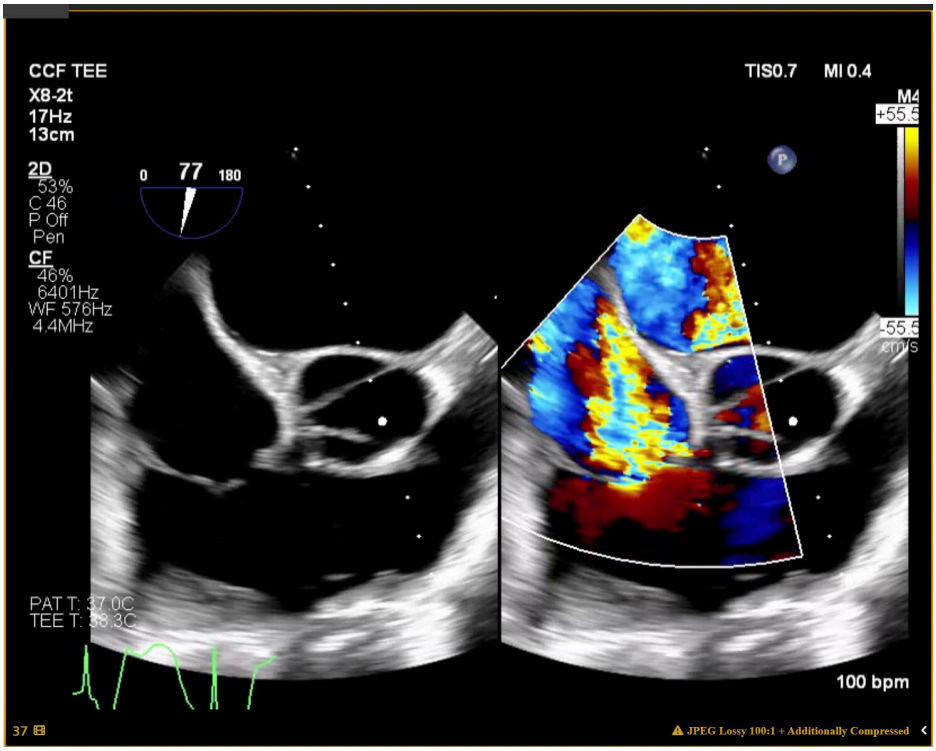
Images



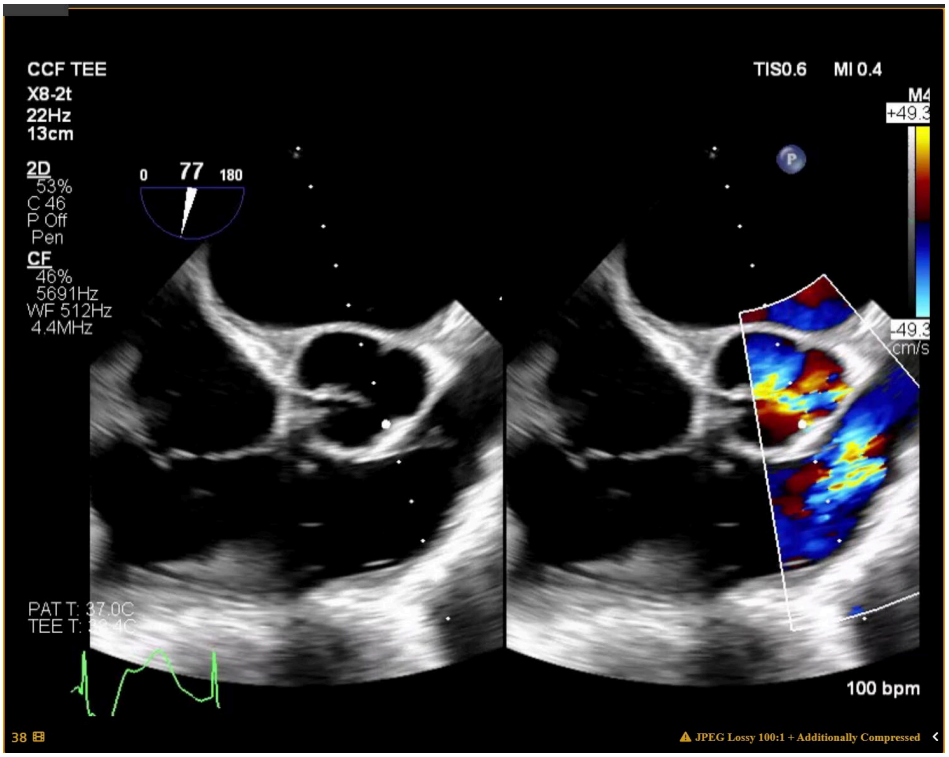
Severe mitral regurgitation



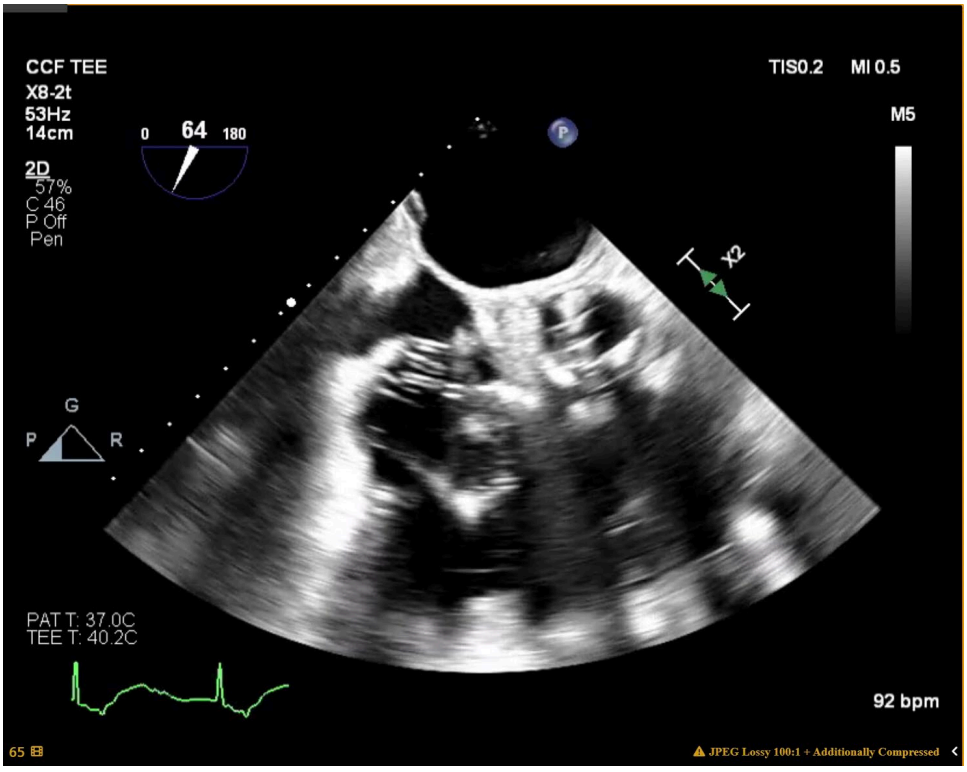
Severe aortic regurgitation



Sere tricuspid regurgitation



Severe pulmonic regurgitation



Blood clot on prosthetic tricuspid valve and venous cannula

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#FR 3 – Kiosk 13 – Poster 1

TAVR Explant: When One Aortic Valve isn't Enough

Presenting Author: Savannah Cofer, MD, University of Maryland Medical Center

Introduction

Transcatheter aortic valve replacement (TAVR) has allowed a broader population of patients with aortic stenosis (AS) a safe, less invasive treatment option. However, a small subset of this population will require further intervention. Patients requiring explantation of the TAVR valve followed by surgical aortic valve replacement (AVR) present a challenge, with higher risk of mortality and hemodynamic collapse.1 Here, we present a complicated patient who presented in acute decompensated heart failure in the setting of severe AS. After initial TAVR to temporize, he then underwent a TAVR explant and surgical AVR.

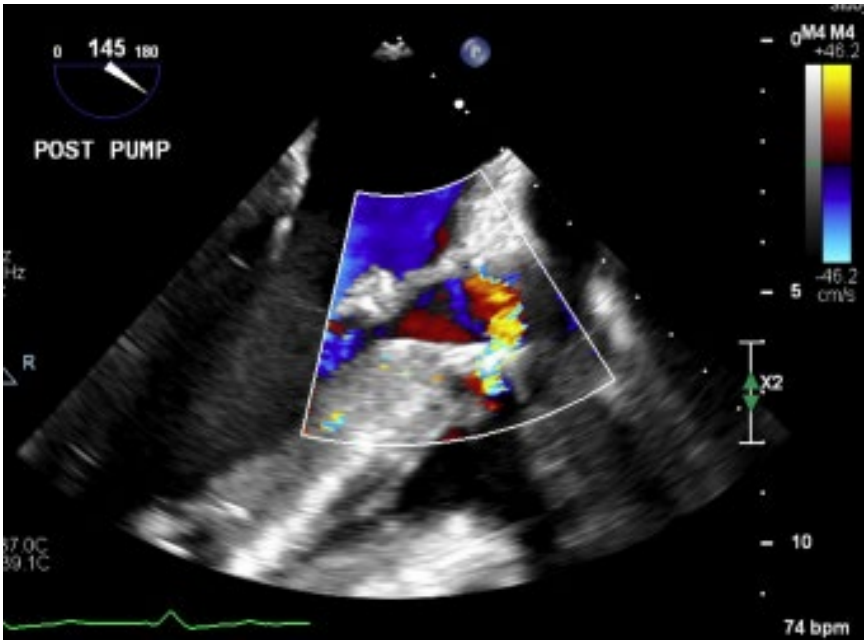
Case Presentation

A 61-year-old man presented in cardiogenic shock from acute decompensated heart failure with severe AS and an ejection fraction of 25%. He had no significant past medical history. Due to his very high-risk state at that time, he was not deemed a candidate for surgical intervention and underwent TAVR. This was complicated by left bundle branch block and perimembranous ventricular septal defect (VSD). He was discharged to cardiac rehabilitation on goal directed medical therapy. In the following months, his EF recovered to 55-60%. Repeat transthoracic echocardiogram showed moderate aortic regurgitation with perivalvular leak and 5.4cm ascending aortic aneurysm. He then underwent sternotomy, TAVR explant, ascending hemiarch replacement, AVR, VSD repair and CABG. Preinduction arterial line was placed. Induction, intubation and line placement was uneventful. Intraoperative transesophageal echocardiography (TEE) was challenging due to poor views. TAVR explant was also surgically difficult. When weaning off cardiopulmonary bypass, a color flow Doppler jet was noticed going from the right ventricle to the left ventricular outflow tract (image 1). On initial inspection it appeared to be a possible VSD. After thorough exam and re-evaluation, it was deemed to be a large paravalvular leak, requiring a second CPB run to repair.The patient was subsequently successfully weaned off CPB on epinephrine and norepinephrine.

Discussion

This case presents the unique scenario of a patient being temporized with a TAVR in order to become a candidate for surgical intervention. This highlights the challenges faced when undergoing a TAVR explant magnified in the setting of a technically difficult TEE where it is imperative that valve function and other associated abnormalities are evaluated. This demonstrates the importance of adaptability and evaluating new information within the clinical context. This applies to both the global context of the patient temporized in the setting of being too critically ill to be a surgical candidate, as well as during the TEE evaluation where the initial TEE read of a VSD had to be reevaluated, allowing for the true problem - of a large paravalvular leak - to be corrected.

Images



Post initial CBP run, mid esophageal long axis.

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#FR 3 – Kiosk 13 – Poster 2

All Four One: Anesthetic Considerations in Quadruple Valve Replacement for Carcinoid Heart Disease

Presenting Author: Soobin Kim, MD, Resident, Brigham and Women's Hospital

Introduction

Replacement of all four valves is exceedingly rare in cardiac surgery. We present a case of a successful quadruple valve replacement in a patient with carcinoid heart disease (CHD).

Case Presentation

A 52-year-old male with small bowel neuroendocrine tumor and prior ileocecal mass resection developed progressive dyspnea on exertion. Workup at the time revealed severe AI, MR, TR, and mod PI, and he was scheduled for quadruple valve replacement. He was admitted preoperatively for volume optimization. Labs were significant for an elevated urine 5-HIAA and an octreotide infusion was started preoperatively. Induction was stable with midazolam, sufentanil, and etomidate. Intraoperative TEE showed biventricular dilation with preserved systolic function, severe AS/AI (image 1), severe TR (image 2), mod MS/severe MR (image 3), and mod-severe PI (image 4). There was no evidence of PFO or shunts. He underwent mechanical AVR and MVR, and bioprosthetic PVR and TVR. Separation from CPB was successful with epinephrine, milrinone, vasopressin, and inhaled epoprostenol. Post-bypass TEE showed hyperdynamic LV, dilated RV with normal function, and well-seated valves without paravalvular leak. These infusions and inhaled medications were continued in the ICU. ICU course was complicated by persistent RV dilation with reduced function requiring gradual inotrope weaning and assisted-diuresis. Octreotide was continued at 100 mcg/hr, weaned to 50 mcg/hr on POD3, and discontinued on POD7. He was extubated on POD3 and ultimately discharged home on POD16. Pathology was consistent with CHD in all four valves.

Discussion

Mixed and multivalvular lesions, common in CHD, pose significant anesthetic and surgical challenges. Intraoperative management should prioritize predominant severe lesions, anticipating post-repair loading-condition changes. For example, reducing severe TR increases RV afterload and can precipitate acute RV failure if not carefully managed. CHD affects the tricuspid valve in 95% of patients and left-sided disease is rare (1). This is commonly associated with PFO or shunts but can occur without either (2). Observational data suggest valve surgery in CHD improves functional status, 2-year-survival, and reverses RV remodeling (2). Patients with carcinoid syndrome or elevated 5-HIAA should receive prophylactic octreotide to mitigate

risks of carcinoid crisis. Octreotide reduces the release of vasoactive peptides and serotonin, traditionally thought to surge in response to catecholamines. Although vasopressin and phenylephrine are commonly recommended first-line pressors, emerging data suggests beta-agonists may not worsen crisis severity or complications (3). Standard practice includes starting an octreotide infusion 12 hours before surgery at 50-100 mcg/hr (3). In this patient, the absence of classic carcinoid symptoms did not preclude prophylaxis: his elevated 5-HIAA, combined with the drug's excellent safety profile, supported initiation at 50 mcg/hr. It was increased to 100 mcg/hr at induction and continued perioperatively. Taken together, thoughtful anticipation of lesion-specific hemodynamics and proactive neurohormonal control with octreotide are central to preventing intraoperative decompensation and optimizing outcomes in patients with CHD.

Images

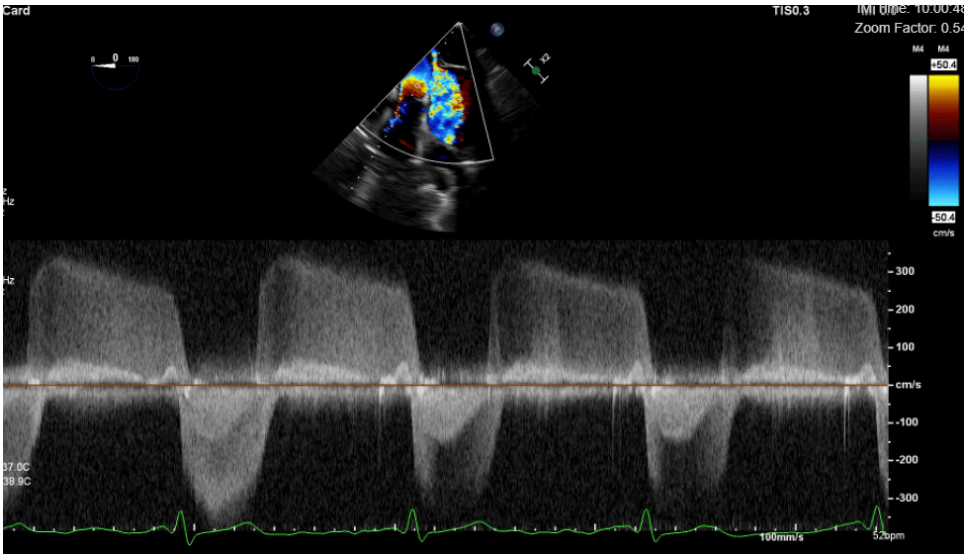


Image 1. Mixed AS/AI

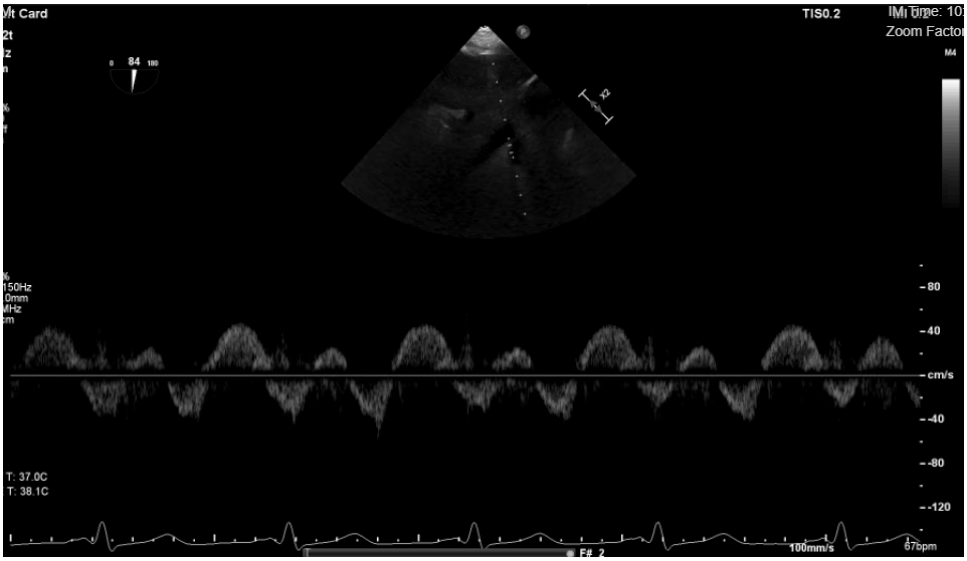


Image 2. TR with systolic flow reversal in hepatic veins

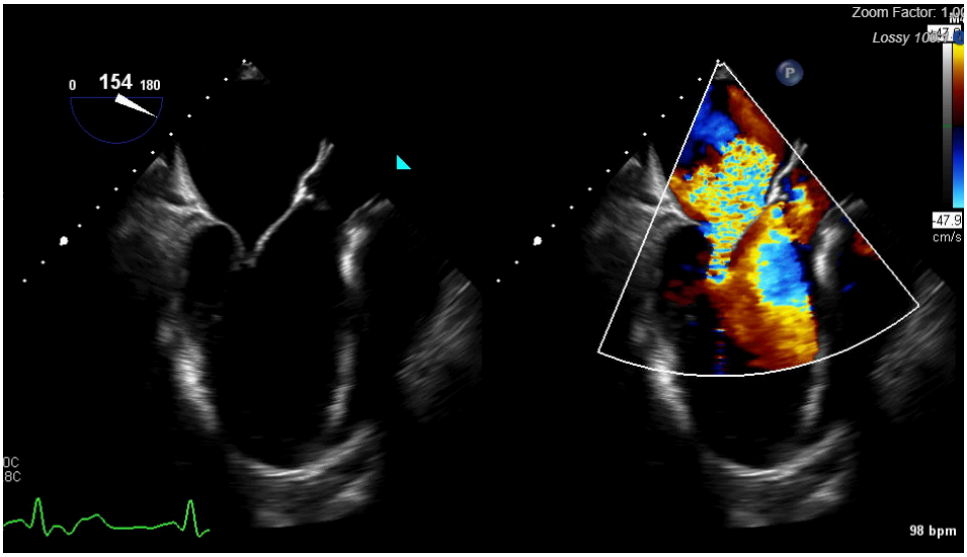


Image 3. Mixed MS/MR

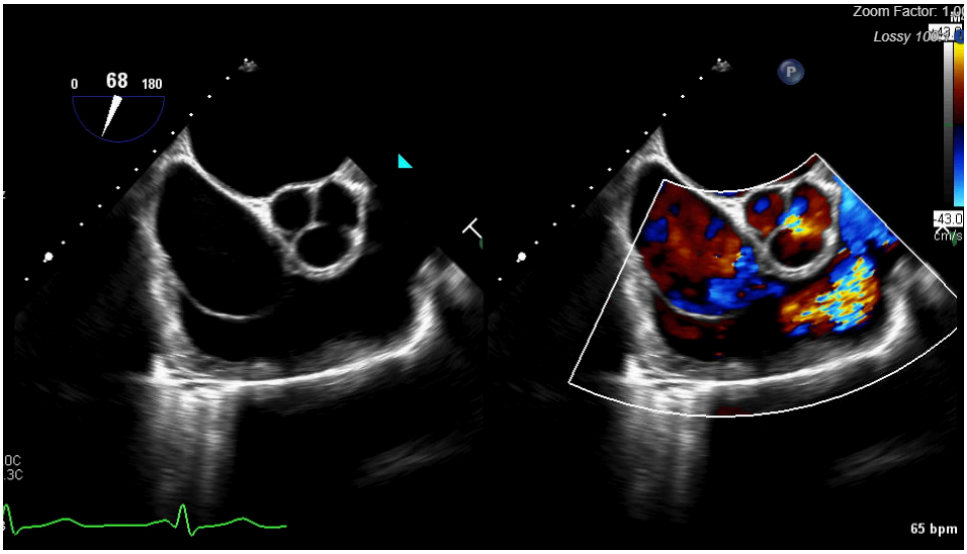


Image 4. PI

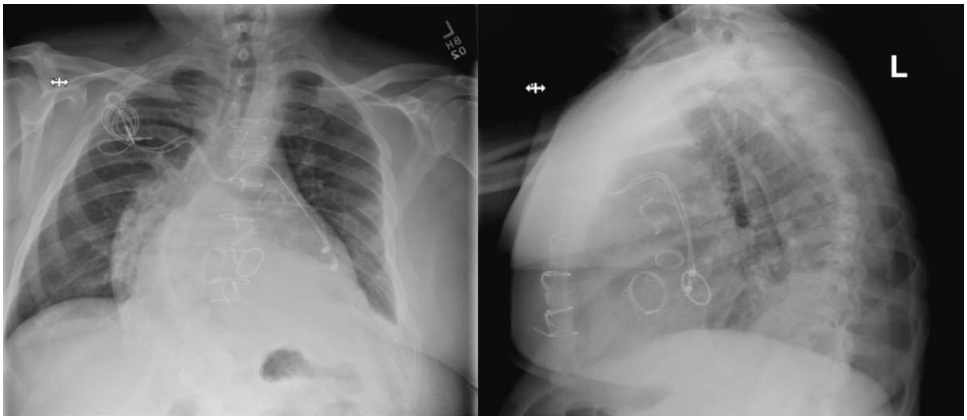


Image 5. Post-operative CXR

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#FR 3 – Kiosk 13 – Poster 3

The Unidentified Gerbode Defect in the Perioperative Period

Presenting Author: Rod Nault, MD, Cardiac Anesthesia Fellow, Cleveland Clinic Foundation

Introduction

- Gerbode defect is rare ventricular septal defect (VSD) that lead to communication between the left ventricle (LV) and the right atrium (RA) with left-to-right shunt.
- Account for 0.08% of all intracardiac shunts. [1]
- Direct type: the atrioventricular portion of the membranous septum
- Indirect type: the ventricular peri-membranous portion is involved and communication with the right atrium is secondary to a defect of the tricuspid valve itself
- Etiologies: endocarditis, iatrogenic injury, congenital, ischemia or unknown
- Hemodynamic consequences: right-sided volume overload and congestion, pulmonary hypertension, cardiac chamber dilation and cardiac dysfunction
- Sequelae of untreated defects include early heart failure and hemodynamic compromise. [2]

Case Presentation

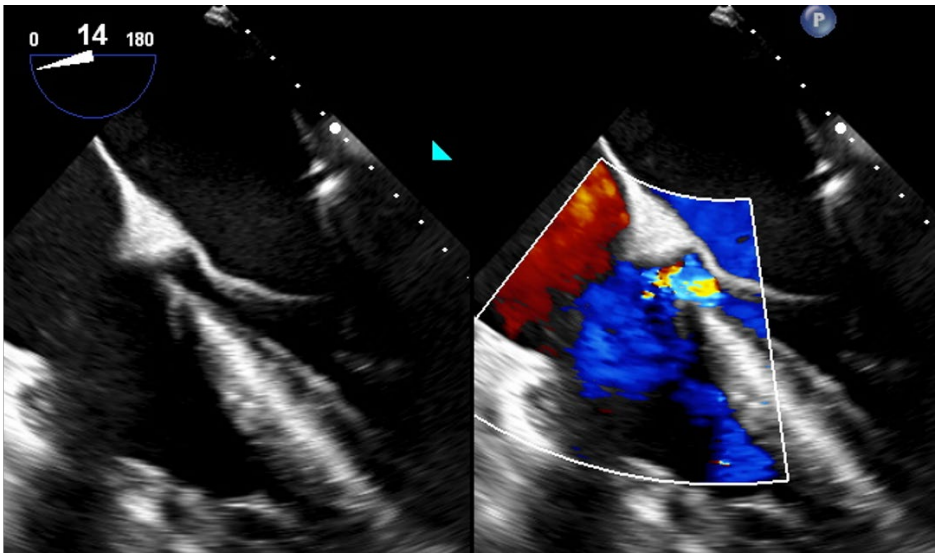
- 38 year-old male with previous bioprosthetic AVR and VSD (iatrogenic) repair presented for redo AVR and residual VSD repair.
- Asymptomatic except for minor orthopnea.
- Preoperative transthoracic echo (TTE) was notable for dilation of all cardiac chambers, severe bioprosthetic valve aortic stenosis, tricuspid regurgitation and residual VSD.
- Intraoperative transesophageal echo (TEE) was unable to identify the communication between ventricles or blood flow with color flow doppler (CFD). A systolic jet was noted in the RA across the tricuspid valve leaflets.
- A 0.5 cm defect was noted between left ventricular outflow tract (LVOT) and RA—consistent with an acquired, direct Gerbode defect.
- High-velocity turbulent jet directed from LVOT to RA was identified with CFD, causing left-to-right shunt with ratio of pulmonary blood flow to systemic blood flow (Qp/Qs) of 1.2, demonstrating a restricted defect.
- The patient underwent a Ross procedure, explant of his aortic valve, closure of the LVOT-RA defect, aortic root and ascending replacement, and pulmonary valve replacement
- Post-intervention TEE demonstrated closure of the Gerbode defect without residual flow, trivial tricuspid regurgitation, and a successful Ross procedure and ascending aorta replacement.

Discussion

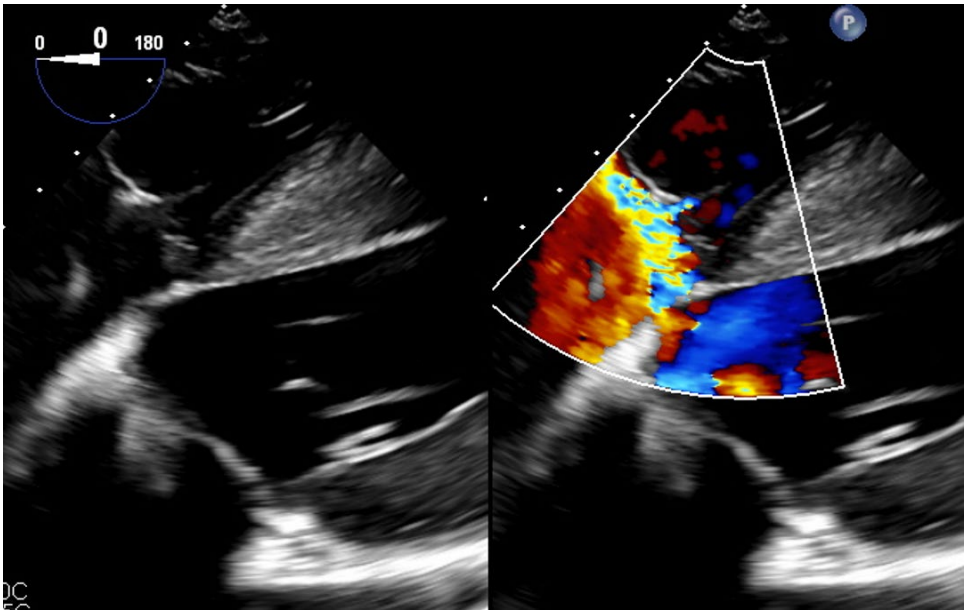
- TEE is the most sensitive modality for detecting the defect followed by cardiac magnetic resonance and TTE. [1]

- Echocardiographic clues to suggest Gerbode defect include atypical jet direction, persistent shunt flow into diastole, lack of ventricular septal flattening, absence of right ventricular hypertrophy and normal pulmonary artery pressures as estimated from the pulmonic regurgitant velocity. [3]
- Considerations for intervention: impaired functional capacity, right atrial with or without ventricular enlargement, and ratio of pulmonary to systemic flow (Qp/Qs) of greater than1.5 in the absence of cyanosis and significant pulmonary hypertension. [1]
- His postoperative course and follow-up visit were uneventful.

Images



An atypical jet direction noted in the mid-esophageal four chamber view (shown with and without color flow doppler)



Defect demonstrated in the deep transgastric view (shown with and without color flow doppler)

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#FR 3 – Kiosk 13 – Poster 4

Devastating Lead Extraction Complication: Tricuspid Valve Injury Leading to Cardiogenic Shock and Heart Transplantation

Presenting Author: Jennifer Saluck, MD, Cardiothoracic Anesthesia Fellow, The Mount Sinai Hospital

Introduction

The contemporary approach to lead extraction is via transcatheter endovascular intervention. These procedures require meticulous planning and execution due to the potential for significant complications. We present a case of one such complication, during which tricuspid valve injury from a lead extraction led to hemodynamic collapse that ultimately necessitated heart transplantation.

Case Presentation

A 64-year-old female with a history of rheumatic heart disease s/p mechanical mitral valve replacement, coronary artery disease s/p CABG, HFrEF (40%), A fib on warfarin, VT s/p ablation and single chamber ICD placement in 2012, presented due to multiple inappropriate shocks, found to have a lead fracture, and scheduled for ICD lead extraction. The patient underwent uneventful radial arterial line placement and intubation. Immediately after lead extraction, the patient became hemodynamically unstable. Rescue TEE revealed unchanged biventricular function but a new flail posterior leaflet of the tricuspid valve with ruptured chordae leading to severe tricuspid regurgitation, along with bowing of the intraatrial septum into the left atrium and systolic flow reversal in hepatic veins. Post op course was complicated by severe cardiogenic shock due to acute TR and right ventricular failure, requiring high-dose vasopressors and inotropes. The patient developed multiorgan failure, requiring VA ECMO on postop day 1. Despite maximal support, she ultimately underwent definitive heart transplantation on postop day 6.

Discussion

Complications of lead extraction include vessel injury, major bleeding, cardiac perforation, pericardial effusion/tamponade, thromboembolism, retained or embolized lead fragment, and, as seen in this case, tricuspid valve injury. The patient was also found to have developed a pseudoaneurysm of the innominate vein, a rare complication associated with lead extraction. Although overall complication rates for lead extractions are relatively low (2.3%), perioperative mortality is 0.9%. Among patients with major complications, there is a 34% mortality rate, and 16% require urgent cardiac surgery. Both patient and device-related factors must be carefully evaluated to ensure appropriate anesthetic and surgical planning. The most common indication for lead removal is infection; specific indications include positive blood cultures, endocarditis, and/or recurrent bacteremia or fungemia despite adequate antibiotic therapy. Noninfectious indications include vascular thrombosis, thromboembolism, chronic pain, recalled leads, lead perforation, persistent arrhythmias due to lead placement, and the need for device upgrade, MRI compatibility, or radiation therapy. This case illustrates the potential for catastrophic complications during lead extraction and underscores the importance of multidisciplinary planning and intraoperative vigilance. These events have heightened clinical interest in leadless pacemakers as safer alternatives for rhythm

management and have also prompted increased discussion regarding lead abandonment in high-risk patients.

Images

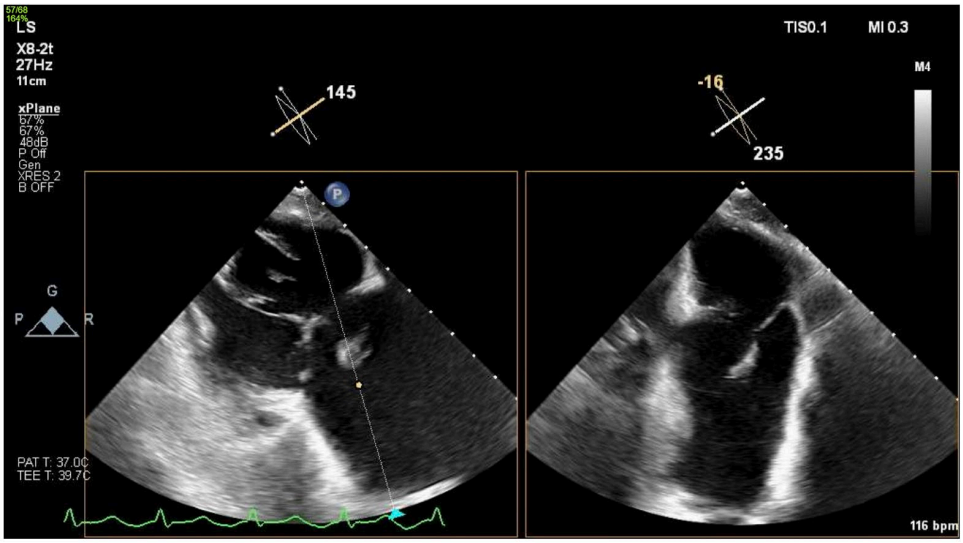


Figure 1a: Transgastric RV inflow view and x-plane showing the flail posterior leaflet of the tricuspid valve.

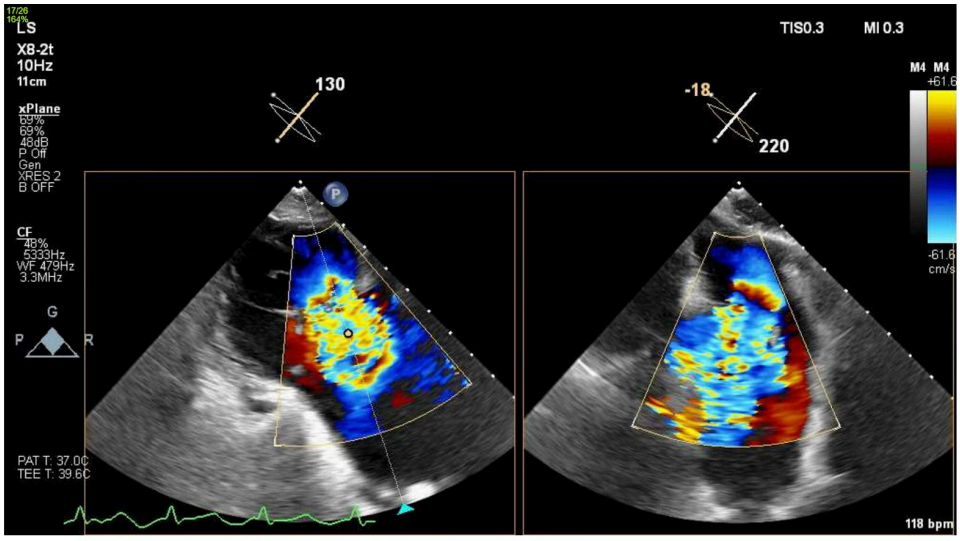


Figure 1b: Transgastric RV inflow view and x-plane with color, showing the flail posterior leaflet of the tricuspid valve.

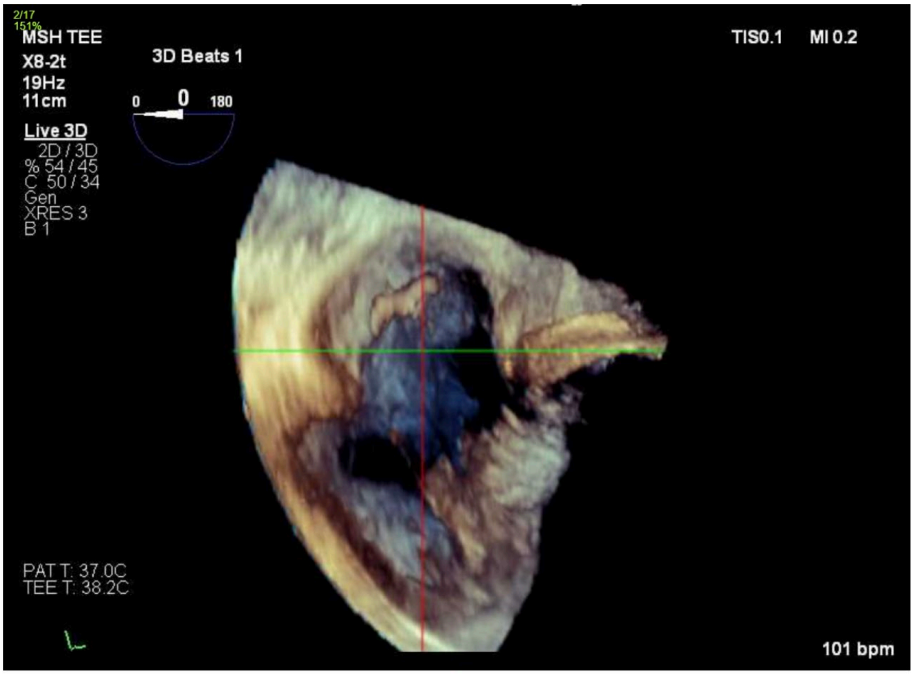


Figure 2: 3D image of the tricuspid valve en face.

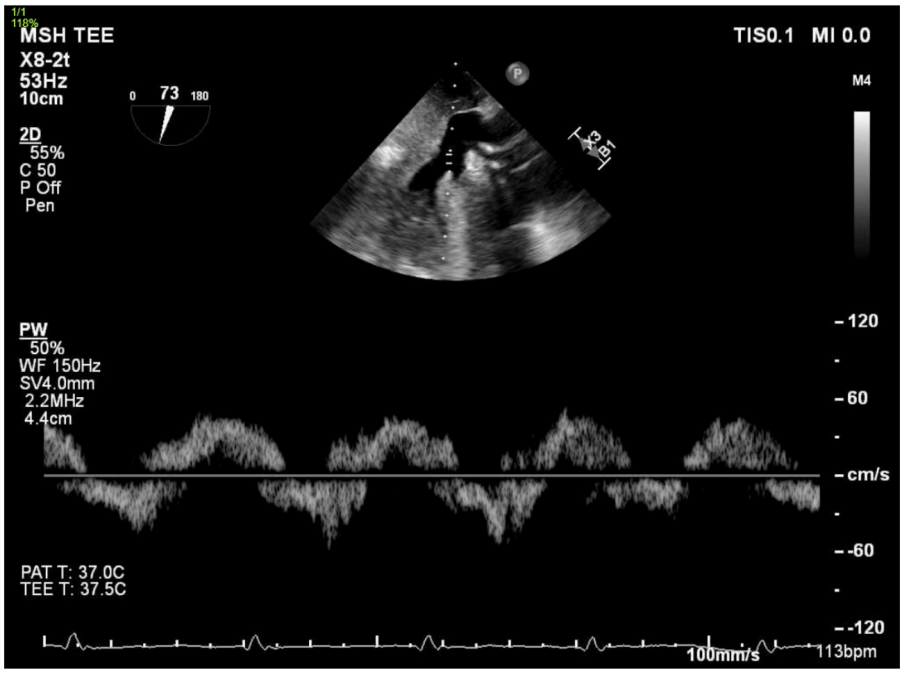


Figure 3: Pulse wave doppler through the hepatic vein showing systolic flow reversal.

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#FR 3 – Kiosk 13 – Poster 5

TEE-Guided Percutaneous Mechanical Thrombectomy of Persistent Left Atrial Appendage Thrombus

Presenting Author: Brian Giang, MD, Resident Physician, Cedars Sinai Medical Center

Introduction

Persistent left atrial appendage (LAA) thrombus in patients who are refractory to or intolerant of anticoagulation therapy presents limited treatment options. Although some cases resolve with prolonged or alternative pharmacologic therapy, the optimal approach for high-risk patients (e.g., increased bleeding risk, prior thromboembolism) remains unclear.[1,2] For this subset of patients, emerging case series suggest percutaneous aspiration thrombectomy with cerebral protection may offer a safe and feasible interventional rescue strategy.[3]

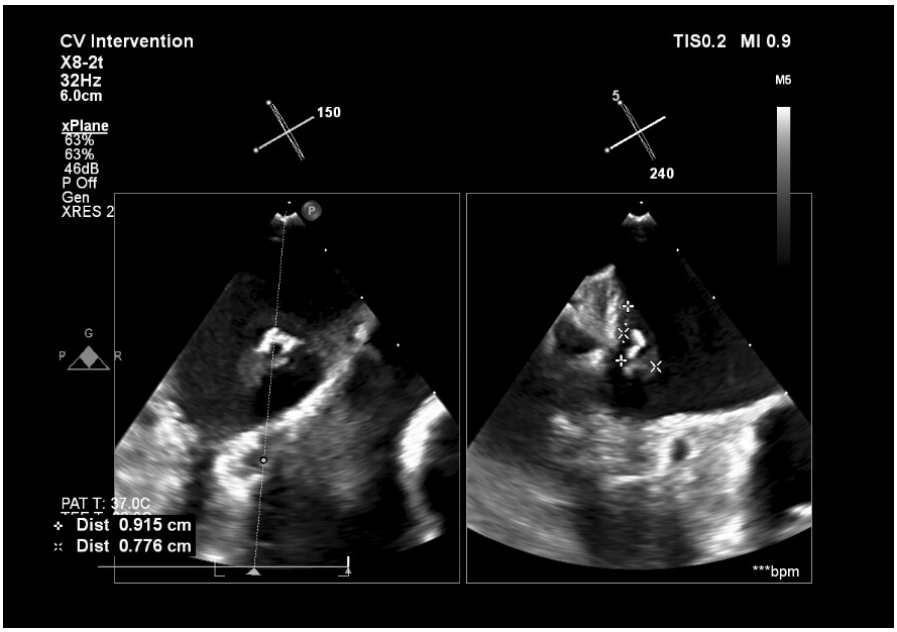
Case Presentation

A 69 year old female with chronic systolic and diastolic heart failure, severe rheumatic mitral stenosis status post balloon mitral valvuloplasty complicated by severe mitral regurgitation, chronic atrial fibrillation on anticoagulation, and an unspecified hypercoagulable disorder with multiple prior thromboembolic events (CVA, PE, lower extremity DVT requiring thrombectomy, and right atrial thrombus treated with aspiration thrombectomy) was referred for transcatheter mitral valve replacement through the ENCIRCLE trial. Notably, the hypercoagulable state and diagnosis of LAA thrombus despite adequate anticoagulation precluded her trial enrollment. A multidisciplinary team composed of cardiac anesthesiology, cardiac surgery, interventional cardiology, and perfusion medicine proceeded with percutaneous LAA aspiration thrombectomy. The patient underwent uncomplicated induction of general anesthesia with standard ASA monitoring, as well as continuous arterial blood pressure monitoring and central venous access. Due to her elevated baseline thromboembolic risk and inherent procedural embolization risk, bilateral cerebral protection devices were deployed through access points in bilateral radial arteries to safeguard all three aortic arch vessels. Partial right heart bypass was established using bilateral femoral vein cannulation under fluoroscopy. Under TEE guidance, an interatrial transseptal puncture was performed, and an AngioDynamics catheter was advanced into the left atrium for aspiration thrombectomy. The majority of the mass was successfully evacuated and the patient was weaned from partial bypass uneventfully. The patient emerged from general anesthesia in her baseline neurologic condition without procedural complications or new hemodynamic instability.

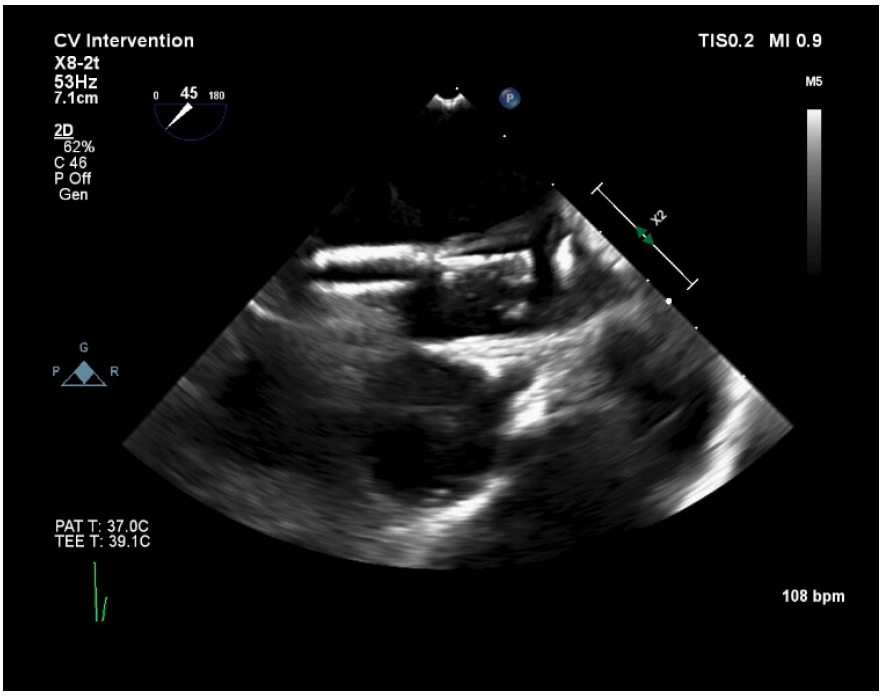
Discussion

This case supports percutaneous LAA aspiration with cerebral protection as a viable and promising option for refractory thrombus in high-risk patients, highlighting its potential to expand treatment options where pharmacologic therapy fails. Patient suitability requires careful consideration of thrombus size, location, mobility, and comorbidities to optimize outcomes. Existing evidence for percutaneous thrombectomy is primarily limited to case series, and larger prospective studies are warranted to establish its long-term safety and efficacy.

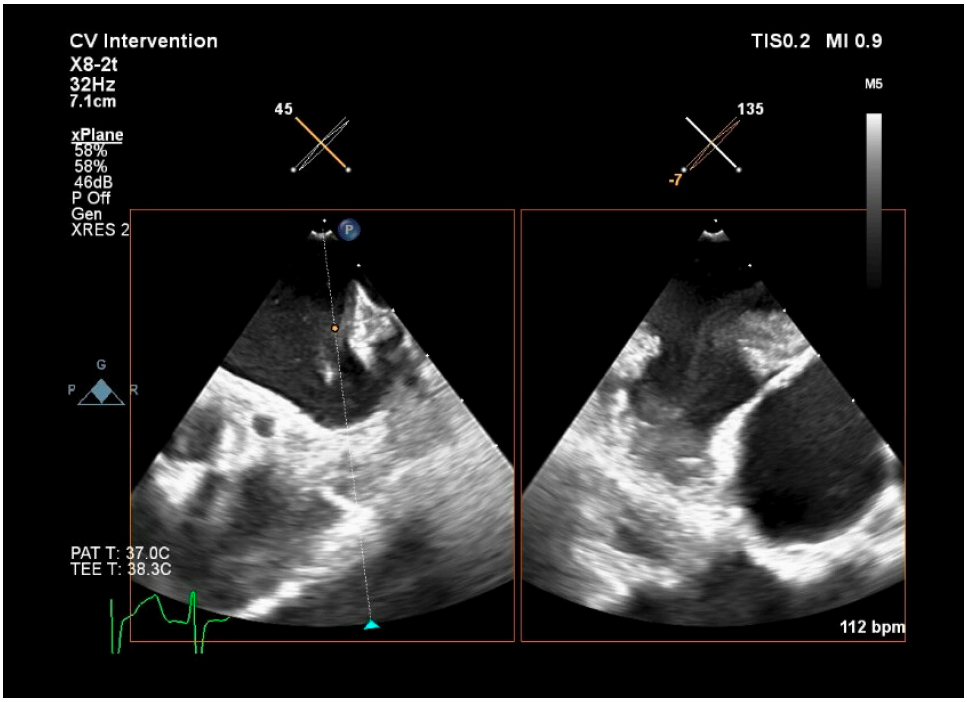
Images



X-plane transesophageal echocardiographic still of thrombus within the left atrial appendage, measuring 0.915 cm by 0.776 cm.



Mechanical aspiration thrombectomy of left atrial appendage thrombus using AngioDynamics catheter.



Left atrial appendage status post aspiration thrombectomy with majority of thrombus successfully evacuated

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#FR 3 – Kiosk 14 – Poster 1

Complex Case: Repeat Mitral Valve Repair After Mitral Valve Ring Dehiscence

Presenting Author: Jeffery P. DeGrauw, DO, Cardiothoracic Anesthesia Fellow, UT Health

Introduction

- Mitral valve annuloplasty ring dehiscence is a rare event with an incidence of around 1.4 – 1.6% in mitral valve repairs. Furthermore, there is a significant difference in incidence rate of dehiscence based on the ring type used in repair, time of occurrence, and the location of the dehiscence on the annuloplasty ring. Incidence was highest with rigid annuloplasty ring repairs at the P3 segment within 30 days of the original surgery.
- Management of mitral annuloplasty ring dehiscence was traditionally performed with mitral valve replacement as the standard of care. However, with recent advances in surgical technique, and a deeper understanding of mitral valve repair failures, re-repair has become an increasingly favored option for appropriately selected patients.
- Intraoperative transesophageal echo is critical in aiding proper re-repair in these patients.

Case Presentation

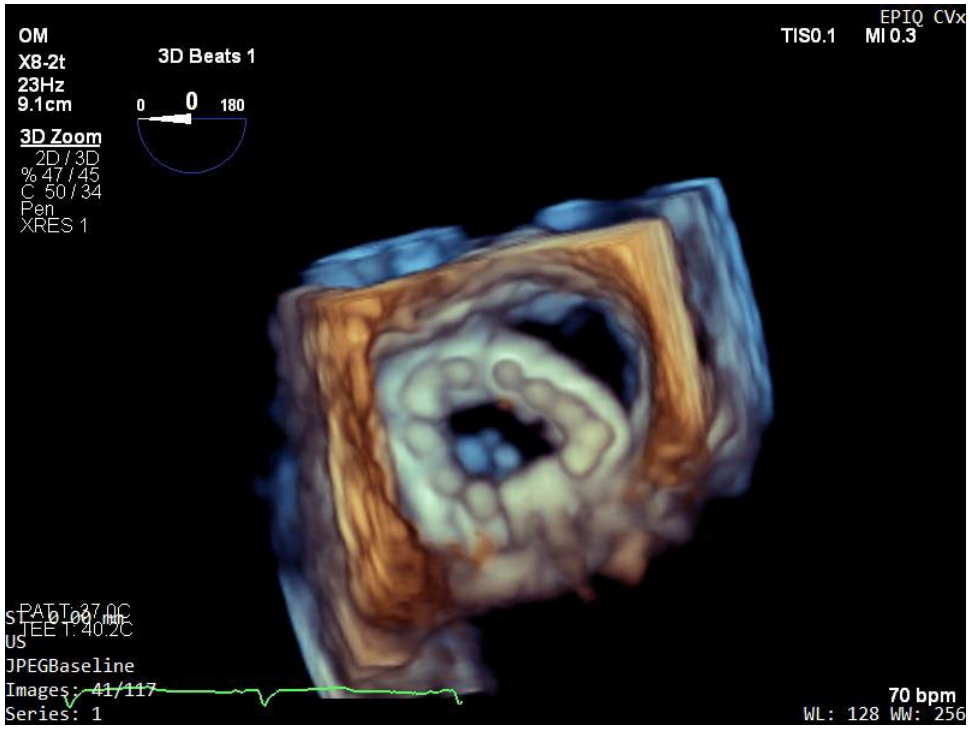
- Patient is a 78 year old male with past medical history of severe mitral regurgitation status post mitral valve repair April 2024, sleep apnea, atrial fibrillation status post atrial appendage closure device, and recurrent urinary tract infections who presented with shortness of breath.
- ECHO prior to surgery showed recurrent severe mitral regurgitation and moderate tricuspid regurgitation.
- Intraoperative transesophageal echo (TEE) showed severe mitral valve regurgitation with a free floating anterior portion of the annulus and prolapse of P2.
- Mitral valve was successfully repaired with a 33mm ring, and repair of the P2 leaflet. Post procedure TEE showed trace mitral regurgitation.
- Weaning from bypass was complicated by biventricular dysfunction requiring inotropic support and intra-aortic balloon pump (IABP).
- Postoperatively patient was extubated on postoperative day one. IABP was weaned and removed. Postoperative transthoracic echo showed a pericardial effusion which improved with colchicine. Once stable, the patient was discharged home from the hospital.

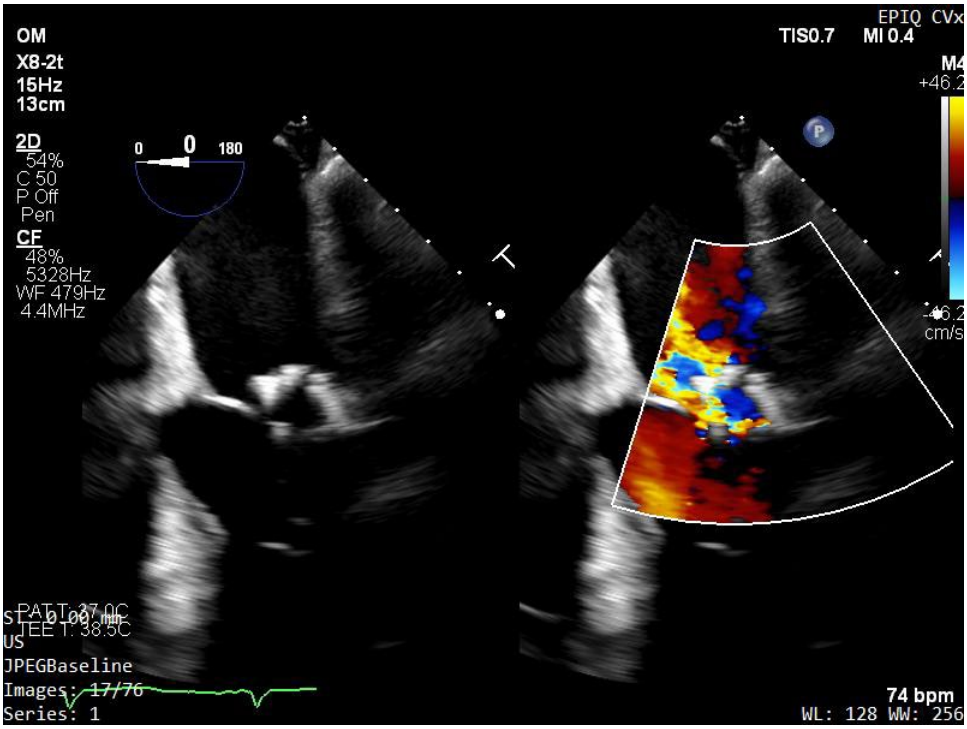
Discussion

- Mitral ring dehiscence is a rare complication of mitral valve repair and is diagnosed with echocardiography.

- Intraoperative transesophageal echo is essential to surgical repair for mitral regurgitation. In this case, transesophageal echocardiography aided in the diagnosis of the cause of severe mitral regurgitation.
- 3D echocardiography helped clearly identify the location of the dehiscence and the P2 prolapse causing the severe mitral regurgitation
- Post procedure echo showed durable repair with trace residual mitral regurgitation
- Prior studies have shown good results of re-repair being used for managing these patients with a 1 year survival rate of 85%.
- Of interest, previous studies list that ring dehiscence is primarily located posteriorly while in our case the dehiscence was anteriorly.

Images





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#FR 3 – Kiosk 14 – Poster 2

Management of Sickle Cell Trait and Cardiopulmonary Bypass

Presenting Author: Olivia Samoray, MD, Resident Physician, University of Wisconsin School of Medicine and Public Health

Introduction

Patients with sickle cell hemoglobinopathies presenting for surgery have a greater risk of perioperative complications, including sickle cell crisis, microvascular occlusion, and hemolysis. Particularly during cardiac surgeries that utilize cardiopulmonary bypass (CPB), patients are exposed to several predisposing factors that promote sickling, including low flow states, aortic cross-clamping, hypothermia, acidemia, and hemodilution.[1] This case represents a multidisciplinary approach to care for a patient with sickle cell trait undergoing cardiac surgery for mitral valve replacement and tricuspid valve repair with CPB.

Case Presentation

A 40-year-old female with a history of hidradenitis suppurativa, sickle cell trait, and newly diagnosed rheumatic heart disease and pulmonary hypertension presented for mechanical mitral valve replacement and tricuspid valve repair. Preoperative TTE demonstrated severe mitral valve stenosis and moderate mitral regurgitation, moderate tricuspid regurgitation, and reduced left ventricular ejection fraction of 45%. The patient denied prior complications related to her sickle cell trait. A multidisciplinary discussion involving hematology, perfusion, anesthesiology, and cardiac surgery was held prior to surgery. Exchange transfusion was not indicated as the patient's hemoglobin S percentage (HbS%) on electrophoresis was 29.2%, with our institutional goal being less than 33%. Institutional guidelines were followed on bypass including normothermia, warm cardioplegia with more frequent dosing, maintaining high flows to avoid stagnation in the bypass circuit, omitting cell saver, and avoiding hypoxemia and acidosis. Total CPB time was 150 minutes, and cross-clamp time was 98 minutes. The patient was uneventfully weaned from CPB and required no additional blood product transfusions. She was then admitted to the cardiothoracic ICU postoperatively, extubated four hours later, and discharged four days later. Throughout the peri- and postoperative course, the patient had no sickle cell-related complications.

Discussion

Although sickle cell trait is generally considered benign and associated with a normal life expectancy, affected individuals can still develop complications that resemble those seen in sickle cell disease. CPB can exacerbate sickling through physiologic stressors, contributing to significant morbidity and mortality. A patient with sickle cell trait who received a cardiac transplantation clotted a VA ECMO circuit and was found to have extensive myocardial infarction and widespread intravascular sickling on postmortem exam.[2] These risks are further amplified by prolonged bypass times, lower median temperatures, and utilization of deep hypothermic arrest.[3] Additionally, maintaining adequate hemoglobin levels appears more protective against sickling than HbS%, suggesting that non-anemic patients with sickle cell trait often do not require

preoperative transfusion.[1] Thus, patients with sickle cell trait still warrant careful preoperative evaluation and multidisciplinary planning when CPB is anticipated.

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#FR 3 – Kiosk 14 – Poster 3

Perioperative Management of an Acquired Gerbode Defect in Recurrent Prosthetic Valve Endocarditis: A Complex Redo Multivalve Case

Presenting Author: Juliana Veira, MD, Resident, Yale School of Medine

Introduction

A Gerbode defect is a ventricular septal condition in which blood abnormally shunts from the left ventricle (LV) to the right atrium (RA), creating a high-pressure left-to-right flow that can cause right-sided volume overload, pulmonary hypertension, and heart failure symptoms [1]. While originally thought to be a rare congenital malformation, it has more recently also been observed in association with endocarditis, prior cardiac surgery, or myocardial infarction, making diagnosis and management challenging [2-3]. This case highlights the hemodynamic impact, diagnostic evaluation, and perioperative considerations associated with this uncommon lesion.

Case Presentation

A 54-year-old man with hypertension, compensated alcohol-related cirrhosis, a pacemaker, and a history of recurrent native and prosthetic valve endocarditis - requiring multiple aortic valve and root surgeries - presented with worsening fatigue, dyspnea, edema, palpitations, and hematochezia shortly after discharge for anemia and aortic root infection. He was found to have acute hypoxemic respiratory failure from volume overload, pseudomonas pneumonia, and recurrent anemia, and was diagnosed with culture-negative prosthetic valve endocarditis complicated by a new aortic valve abscess, Gerbode defect, and left ventricular outflow tract (LVOT) peri-prosthetic infection. He underwent a complex third redo sternotomy with mitral and tricuspid valve replacement, bovine pericardial patch closure of the LVOT- right atrial fistula, and femoral cannulation for cardiopulmonary bypass. Prior to sternotomy, the patient underwent right groin cutdown with direct cannulation of the right common femoral artery and vein for cardiopulmonary bypass. Pre-bypass transesophageal echocardiogram (TEE) showed normal LV function, a severely dilated right ventricle (RV) with normal function, moderate mitral regurgitation and moderate-severe tricuspid regurgitation, and a persistent LVOT-to-RA shunt. Intraoperatively, he required vasopressor and inotropic support and received multiple blood products. Post-bypass TEE confirmed well-functioning prosthetic valves, successful closure of the Gerbode defect, preserved LV function, and mildly reduced RV function. He was transferred to the CTICU in stable condition and extubated the same evening.

Discussion

Redo sternotomy for combined mitral and tricuspid valve surgery carries substantial hemodynamic risk due to adhesions, altered anatomy, and prior prosthetic material, requiring precise control of preload, afterload, and contractility. In this patient, recurrent prosthetic valve endocarditis, an LVOT aneurysm, and a Gerbode defect created chronic hemodynamic instability, making preoperative optimization with milrinone essential to support forward flow and reduce right-sided pressures. After valve replacement, abrupt shifts in loading conditions - higher LV afterload and decreased right-sided regurgitation - caused strain on ventricles

accustomed to chronic regurgitation, necessitating inotropic support and careful management of pulmonary vascular resistance. Successful perioperative care in such patients is dependent on anticipating these physiologic shifts and maintaining stable perfusion throughout high-risk multivalve procedures.

Images

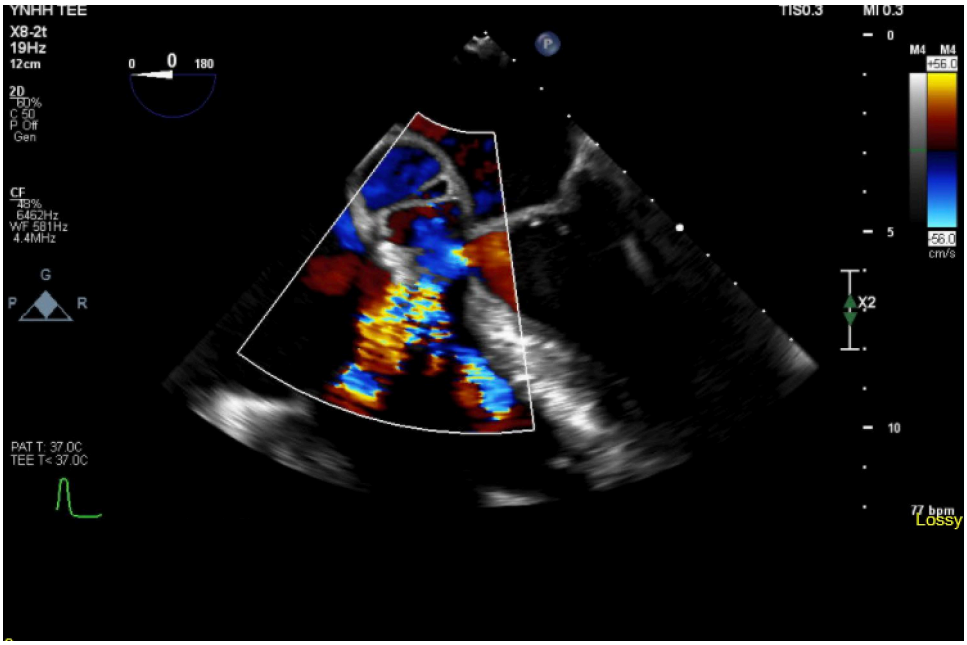


Figure 1. Mid-esophageal five-chamber transesophageal echocardiographic view with color Doppler demonstrating pre-repair flow patterns through the Gerbode defect as well as an aneurysm of the left ventricular outflow tract.

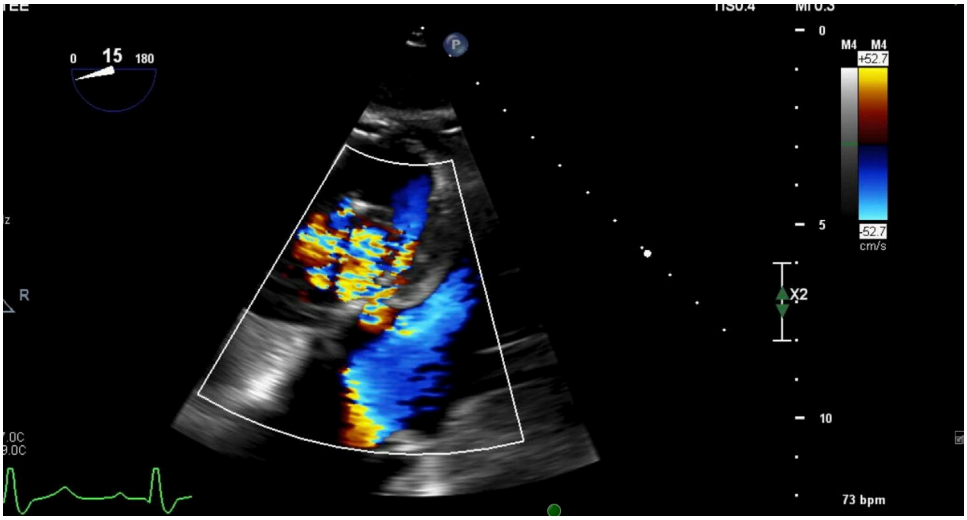


Figure 2. Deep transgastric five-chamber view with color Doppler demonstrating pre-repair flow patterns, highlighting the direction and velocity of regurgitant jets, shunting, and areas of turbulent flow.

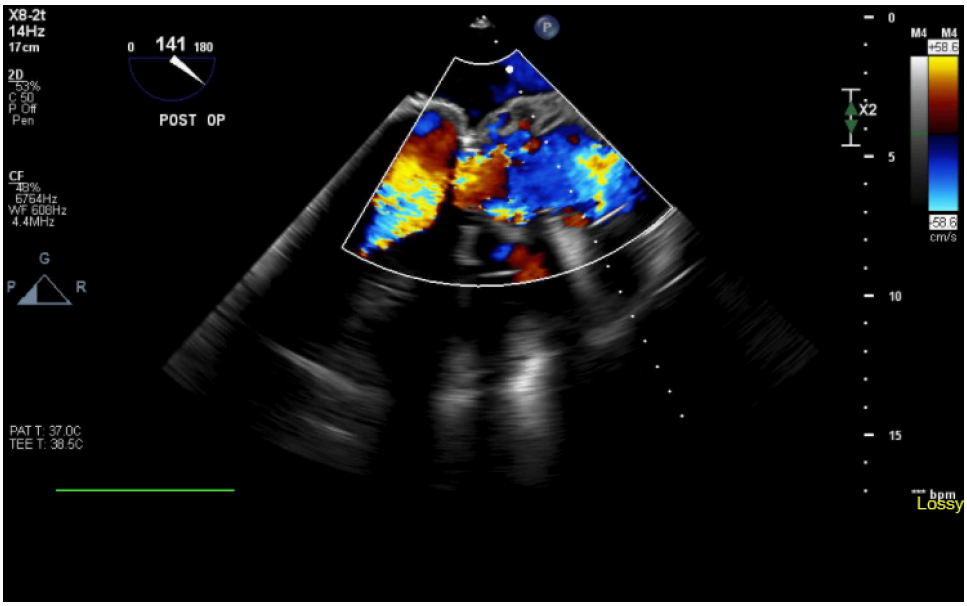


Figure 3. Postoperative mid-esophageal long-axis view showing the LVOT, aortic valve, and anterior mitral leaflet with absence of flow through closed defect.

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#FR 3 – Kiosk 14 – Poster 4

Bestill My Beating Heart: Reoperative Mitral Valve Repair using Deep Hypothermic Fibrillatory Arrest in a Patient with an Unclampable Neo-aorta

Presenting Author: Jennifer Shmukler, MD, Resident Physician, Mount Sinai West/Morningside

Introduction

Reoperations in patients who have previously undergone complex aortic reconstruction present many perioperative challenges. We describe a case of complex decision-making in the management of a patient with prior aortic surgery, now presenting with symptomatic severe mitral regurgitation (MR).

Case Presentation

A 47-year-old female with a history of Marfan syndrome presented for re-operative mitral valve repair. In 2019, she had a Type A dissection that was emergently repaired with a Bio-Bentall and hemiarch replacement. A mitral annuloplasty ring was also placed for severe MR. Her postoperative course was complicated by systolic anterior motion (SAM) of the anterior mitral valve leaflet. Annual TTEs continued to demonstrate SAM and moderate-severe MR; however, the patient remained asymptomatic. In July 2025, she was hospitalized for a heart failure exacerbation that improved with diuresis. In October, she re-presented with pre-syncope, and the decision was made to proceed with surgery. Preoperative imaging demonstrated a stable residual aortic dissection (Figure 1). The neoaorta and arch were noted to be densely adherent to the sternum, making a reoperative sternotomy extremely high risk and aortic cross-clamp difficult/impossible. Therefore, the decision was made to utilize a right anterior thoracotomy approach and fibrillatory arrest. After an uneventful induction, a left-sided double-lumen tube was placed, and the right lung was deflated. The right femoral artery and vein were cannulated. Cardiopulmonary bypass (CPB) was established, and the patient was cooled to 18° C to induce ventricular fibrillation (VF). VF was maintained using an alternating current (AC) fibrillator (Figure 2) (Medtronic, Minneapolis, MN). She then underwent successful mitral valve re-repair. The heart was defibrillated once. The patient was then rewarmed and successfully weaned from bypass. Post-CPB TEE showed resolution of the SAM and minimal residual MR. The remainder of her hospital stay was uneventful, and she was discharged on post-op day five. Her discharge TTE showed minimal MR and no SAM.

Discussion

This case highlights the importance of preserving older and now seldom-used techniques in cardiac surgery. Before the routine use of potassium-based cardioplegia, a common means of achieving myocardial protection was deep hypothermia and fibrillatory arrest. Oxygen consumption is reduced, and the potential for catastrophic ejection of air in the absence of a cross clamp is minimized. Using this technique, however, requires not only institutional knowledge but also the actual equipment to induce and maintain VF. In this instance, we used an AC fibrillator (Figure 2). To our knowledge, these devices are no longer manufactured. Other means of inducing and maintaining VF are to use a patient's own implantable cardiac defibrillator, which was not an option in this case. Avoiding a midline sternotomy reduced the risk of catastrophic aortic

injury, but an anterior thoracotomy approach required lung isolation. Hemodynamic vigilance was essential in navigating dissection-related perfusion considerations. This case highlights the importance of individualized, multidisciplinary planning in safely accomplishing mitral re-intervention in high-risk aortic patients.

Images

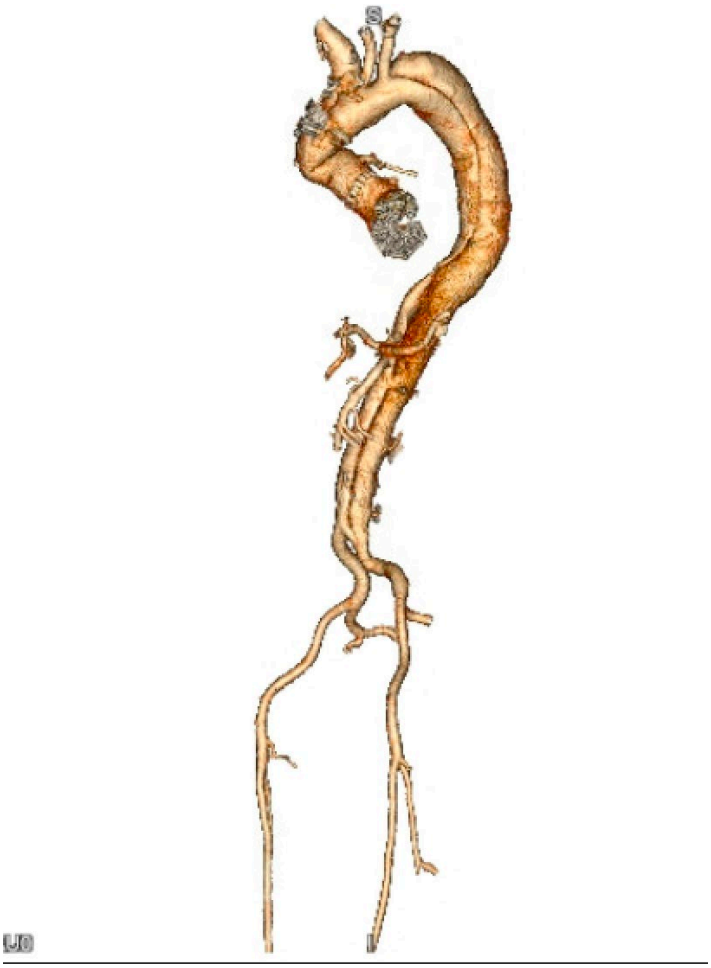


Figure 1: 3D reconstruction of the patient's ascending aorta, aortic arch, descending aorta, and abdominal aorta.



Figure 2: An AC fibrillator

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#FR 3 – Kiosk 14 – Poster 5

More Than a Benign Variant: Guidewire Entrapment and Other Clinical Considerations of a Persistent Chiari Network

Presenting Author: Curtis Duncan, MD, Resident, University of Arizona

Introduction

Embryologically, the right valve of the sinus venosus gives rise to the crista terminalis, Eustachian valve, Thebesian valve, and when it fails to regress completely, it forms the Chiari network. On echocardiography, the Chiari network appears as a mobile, curtain-like membrane within the right atrium, inferior vena cava, and coronary sinus. It is reported to be present in 3.75 to 13.6% of patients (1,2). Although typically considered benign, its presence is associated with patent foramen ovale in 83% of patients and atrial septal aneurism in 24%, and it increases the risk of complications during procedures involving right-sided venous access (3). During central line placement, these complications include guidewire entrapment, catheter malposition, and pulmonary artery catheter entanglement (4). During cardiac procedures, additional risks include pacemaker or ICD lead entanglement, interference with right atrial or bicaval venous cannulation for cardiopulmonary bypass or ECMO, and coronary sinus cannulation for retrograde cardioplegia. Here we present a case of guidewire entrapment under direct echocardiographic visualization during central venous catheter placement for elective cardiac surgery.

Case Presentation

A 59-year-old woman with aortic insufficiency was to undergo surgical aortic valve replacement. Following an uneventful induction and transesophageal echocardiogram (TEE), central venous catheterization with introducer sheath and pulmonary artery catheter placement was planned. Under ultrasound guidance, the right internal jugular vein was cannulated, and a flexible J-tip guidewire was introduced without issue. An introducer sheath with dilator was then advanced over the wire with a small skin incision and when the operator attempted to withdraw the guidewire and dilator, an unexpected amount of resistance was encountered. TEE visualization confirmed correct positioning of the catheter; however, the guidewire appeared bunched up adjacent to the inferior cavoatrial junction. Without any twisting motion, the wire was gently advanced and withdrawn multiple times until it was able to be removed without excessive resistance. The pulmonary artery catheter was then placed without issue and the surgery was able to proceed.

Discussion

Guidewire entrapment during central venous catheterization, or any catheter placement using the Seldinger technique, is a recognized complication. Although excessive resistance during guidewire withdrawal is most commonly caused by kinking or coiling of the wire, a persistent Chiari network is an under-recognized cause. In all cases, the proceduralist should avoid forceful withdrawal or advancement when resistance is encountered. Pulling against a kinked or coiled wire risks shearing and retained wire fragments; however, applying traction when the wire is entangled in a persistent Chiari network may result in significant

complications, including atrial arrhythmias, avulsion of the Chiari network, or even tearing of adjacent structures such as the coronary sinus, inferior vena cava, or right atrial wall.

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#FR 3 – Kiosk 14 – Poster 6

Transfusion-Free Type A Repair in a Jehovah's Witness: ANH/Cell Salvage, Factor-Guided Hemostasis, and TEE-Guided HOCM/SAM management

Presenting Author: Hirofumi Kanazawa, MD, Resident, University of Minnesota Medical School

Introduction

Jehovah's Witness (JW) patients undergoing emergent cardiac surgery pose unique anesthetic and surgical challenges due to their refusal of allogeneic blood products. These strategies include acute normovolemic hemodilution (ANH), cell salvage, and detailed conversation with the patient regarding specific products to optimize outcomes while respecting patient autonomy. We present a case of a Jehovah's Witness patient undergoing emergent ascending aortic repair for Type A aortic dissection with concomitant systolic anterior motion (SAM) and hypertrophic obstructive cardiomyopathy (HOCM) physiology, successfully managed without blood transfusion.

Case Presentation

A 76-year-old, 100-kg Jehovah's Witness man with cardiomyopathy (2015 Takotsubo), hypertension, hyperlipidemia, prostatectomy, mood disorder, and Parkinson's disease presented with abrupt chest–back pain. CTA showed an extensive Stanford Type A dissection to L4 with left-renal-artery involvement. He consented to emergent repair, refusing allogeneic blood but accepting cell salvage, fibrinogen concentrate, prothrombin complex concentrate (PCC), and acute ANH. The patient's starting hemoglobin was 14.5 g/dL. Ascending aortic repair proceeded on cardiopulmonary bypass with uneventful induction and intubation. Before incision, ANH was performed with 600 mL of whole blood for reinfusion. Intraoperative TEE revealed dynamic LVOT obstruction with SAM/HOCM physiology; esmolol 20 mg reduced contractility while afterload was maintained. After the repair and post bypass, hemostasis was achieved with fibrinogen concentrate 1,150 mg and factor concentrate 2,161 U with cell-saver autotransfusion and antifibrinolytic therapy. Vasoactive support targeted SVR and aortic pressure goals and included norepinephrine (0.03–0.08 µg/kg/min), vasopressin (2 U/hr, weaned successfully), and nicardipine for blood-pressure management. He remained stable, required no allogeneic transfusion, and was transferred to the CVICU with satisfactory hemoglobin and lactate levels.

Discussion

Managing aortic dissection in Jehovah's Witness patients requires meticulous preoperative optimization and intraoperative planning to minimize blood loss and maintain end-organ perfusion[1]. Acute normovolemic hemodilution allows preservation of red cell mass while maintaining normovolemia with crystalloids or colloids[2]. When combined with antifibrinolytics, factor concentrates, and cell salvage, it provides an effective blood conservation strategy. In this case, HOCM/SAM physiology added complexity by limiting the ability to augment contractility or reduce afterload—therapeutic maneuvers often employed in type A dissections[3]. The use of esmolol to blunt contractility while maintaining afterload proved essential in balancing myocardial oxygen demand and hemodynamic stability. This case demonstrates that even in

emergent, high-risk cardiac surgery, a multidisciplinary, protocol-driven approach emphasizing blood conservation, factor replacement, and physiologic management can achieve successful outcomes without violating the patient's beliefs.

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#FR 3 – Kiosk 14 – Poster 7

Post-Infarct Ventricular Septal Defect complicated by Coagulopathy and Vasoplegia

Presenting Author: Justin Le, DO, Resident, University of Florida, Jacksonville

Introduction

Ventricular Septal Defect due to myocardial infarction is a rare and serious complication that can occur after an ST segment elevation MI. The transmural ischemia and subsequent death and rupture of myocardium post MI can occur anywhere within the left ventricle. The case we present involves a large area of interventricular septum, where the patient's clinical presentation and events leading to their surgical repair presented additional challenges in the perioperative period.

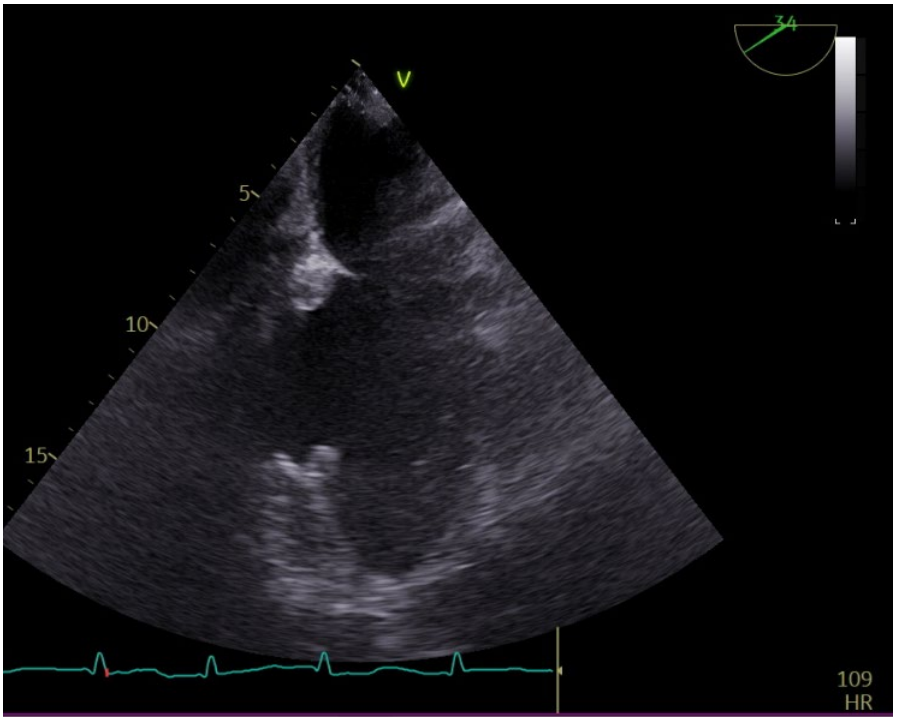
Case Presentation

A 64-year-old male smoker with no medical history presented to the ED with complaints of worsening shortness of breath with 2 weeks of "flu-like" symptoms. The patient was found to be in Afib with RVR, with elevated BNP and troponins. ECG showed inferior ST elevations with Q waves. TTE revealed normal LV function but severe RV enlargement and hypokinesis. PCI was performed with placement of a drug eluting stent of the proximal RCA and POBA of the RPDA. Left ventriculogram was not performed. The patient was given antiplatelet medications (Plavix 600 mg, tirofiban, aspirin). TTE post LHC demonstrated aneurysmal changes of the inferoseptal wall with a large basal-to-mid inferoseptal VSD measuring up to 3 cm with associated left-to-right shunting (Qp/Qs of 3.4). CT surgery was consulted, and due to ongoing hemodynamic decline, he was urgently taken to the operating room for VSD repair. The patient underwent uneventful induction of anesthesia, line placements, and course to CPB. An apical left ventriculotomy exposed a friable inferoseptal defect, 2.5 × 3.5 cm, that was repaired using a Dacron patch and felt-strip reinforcement. Post CPB, coagulopathy and hemodynamic instability ensued. After protamine administration, the patient received 4 units RBCs, 6 units Platelets, 3 units cryoprecipitate, and Factor 7 (90 mcg/kg). The patient continued with poor biventricular function, afib, and vasoplegia requiring epinephrine, norepinephrine, vasopressin, milrinone, angiotensin II, and methylene blue. His ICU course included continued coagulopathy, shock, initiation of CRRT , and eventual nosocomial pneumonia with COVID, upper GI bleed, and multiple reintubations. Two months after presentation, the family opted for comfort care.

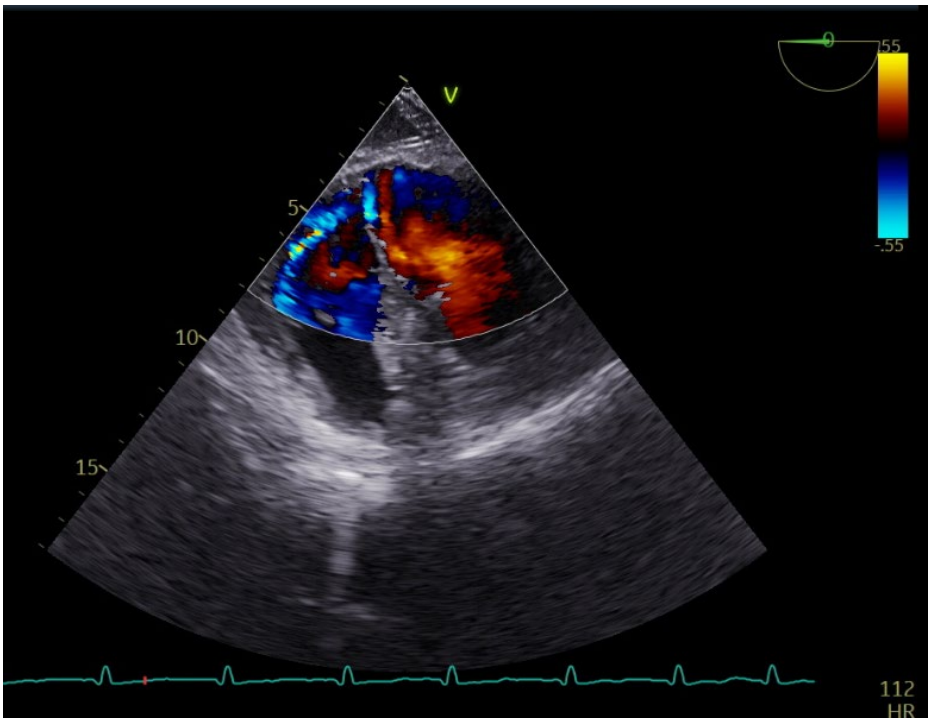
Discussion

Post MI VSD is a very serious condition that carries a high mortality rate ranging from 40 – 60% (1). There is debate in the literature as to the appropriate timing of surgery after diagnosis (2). This defect was too large to approach percutaneously, and the delay in diagnosis after LHC and PCI where the patient received antiplatelet therapy contributed to the challenging care to correct the ensuing coagulopathy and the cardiogenic shock post CPB.

Images



VSD as seen on echocardiogram



Shunting through VSD as seen on echocardiogram

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#FR 3 – Kiosk 15 – Poster 1

Adult Post-Rastelli Pulmonic Conduit Obstruction: Valve Sparing Surgical Management

Presenting Author: Robert N. Hawthorne, MD, PhD, Resident, Johns Hopkins University School of Medicine

Introduction

Surgical strategies such as the Rastelli procedure have markedly improved outcomes in congenital D-transposition of the great arteries. However, reintervention is common, with pulmonic conduit stenosis among the most common and morbid complications. We report a case of an adult post-Rastelli patient with severe obstruction of unclear etiology scheduled for bioprosthetic pulmonic valve and conduit replacement. Intraoperative transesophageal echocardiography (TEE) and surgical exploration revealed a discrete fibrovascular rind within the RVOT–pulmonary artery (PA) conduit. Excision of this rind relieved the obstruction, preserving the existing valve and avoiding a more invasive reoperation.

Case Presentation

A 31-year-old male born with complete D-transposition of the great vessels, ventricular septal defect, and PA discontinuity presents with pulmonary conduit hypertension. Surgical history is notable for Blalock-Thomas-Taussig shunt palliation followed by definitive repair with a Rastelli procedure in infancy. His anatomy on presentation consists of an RV–PA conduit with bioprosthetic valve and an LVOT baffle. His 30-year postoperative course was notable for recurrent subaortic stenosis and repeated RVOT obstruction involving both the conduit and branch PAs. His most recent conduit and PV replacement occurred at age 22. Routine transthoracic echocardiography demonstrated markedly elevated RV pressures with a peak RVOT gradient of 63 mmHg. CT imaging revealed thickened linear soft tissues within the conduit with a minimum diameter of 5 mm. Cardiac catheterization confirmed no valvular stenosis but suggested a fixed “kink” in the conduit not amenable to transcatheter intervention. Given his worsening gradient and limited percutaneous options, redo sternotomy with conduit and valve replacement was planned. Following uneventful induction of general anesthesia, intraoperative TEE demonstrated a prosthetic PV with normal function and leaflet mobility. There was tortuosity and flow acceleration in the conduit associated with echogenic tissue. On opening the conduit above the valve, the surgical team found a thick fibrovascular tissue layer. The tissue was consistent with a pannus but was highly unusual as it was confined to the conduit and did not involve the valve annulus. This tissue was excised and further evaluation revealed unobstructed PA and RV anastomoses as well as normal leaflet mobility and thickness. Final TEE demonstrated laminar flow through the reconstructed RVOT-PA conduit and unchanged biventricular function. Recovery was uncomplicated, and the patient was discharged on postoperative day seven. Pre-discharge TTE confirmed resolution of RVOT obstruction with peak gradient reduced to 20 mmHg.

Discussion

This case highlights an unusual form of RVOT obstruction in a post-Rastelli patient, caused by a fibrovascular rind within the conduit but sparing the valve. Although pannus is a recognized mechanism of prosthetic valve obstruction, isolated intraconduit formation is rarely described. TEE findings prompted

targeted exploration and excision of a discrete obstructive tissue rather than full conduit and valve replacement. This case underscores the value of intraoperative imaging in allowing for tailored decision making in reoperative congenital cardiac surgery.

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#FR 3 – Kiosk 15 – Poster 2

Mysterious Floating Object in the Heart Post Left Ventricular Assist Device Placement

Presenting Author: Taylor B. St Martin, MD, Anesthesiology Resident, University of Alabama at Birmingham

Introduction

Pledgeted sutures are commonly used in cardiac surgery to reduce the risk of suture-related myocardial damage by evenly distributing tension[1]. However, pledget dislodgement and embolization, though rare, can cause severe complications. We present a novel case of pledget migration following left ventricular assist device (LVAD) placement, resulting in complete flow obstruction of the device. This required cardiopulmonary bypass (CPB) and LVAD disassembly for pledget retrieval.

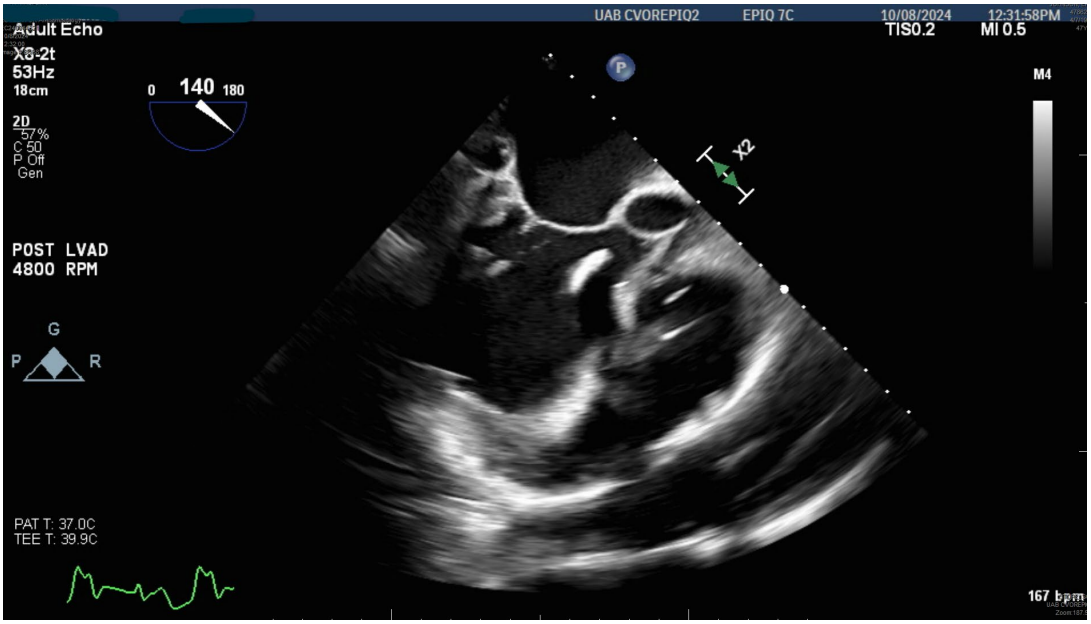
Case Presentation

A 47-year-old male with a history of nonischemic cardiomyopathy, severe biventricular failure, severe mitral regurgitation and tricuspid regurgitation, and ICD placement presented for urgent LVAD implantation following aborted ECMO cannulation. The patient arrived to the operating room on norepinephrine, epinephrine, vasopressin, dobutamine, and IABP. After an uneventful induction and line placement, the LVAD insertion at the apical site and initial separation from CPB was successful. However, intraoperative TEE revealed a mobile, echogenic object within the left ventricle (LV) (Figure 1). Initial suspicion was artifact given the presence of shadowing and multiple intracardiac, but closer inspection suggested either myocardial tissue or a dislodged pledget. During discussion with the anesthesia staff and surgical team, the object subsequently disappeared from view on TEE, and shortly afterward, LVAD flows precipitously declined (Figure 2). The patient was returned to CPB, and the LVAD was explanted. After disassembling, a small pledget was found obstructing the pump (Figure 3). Following reimplantation, normal LVAD flows were restored, and the remainder of the procedure was uneventful.

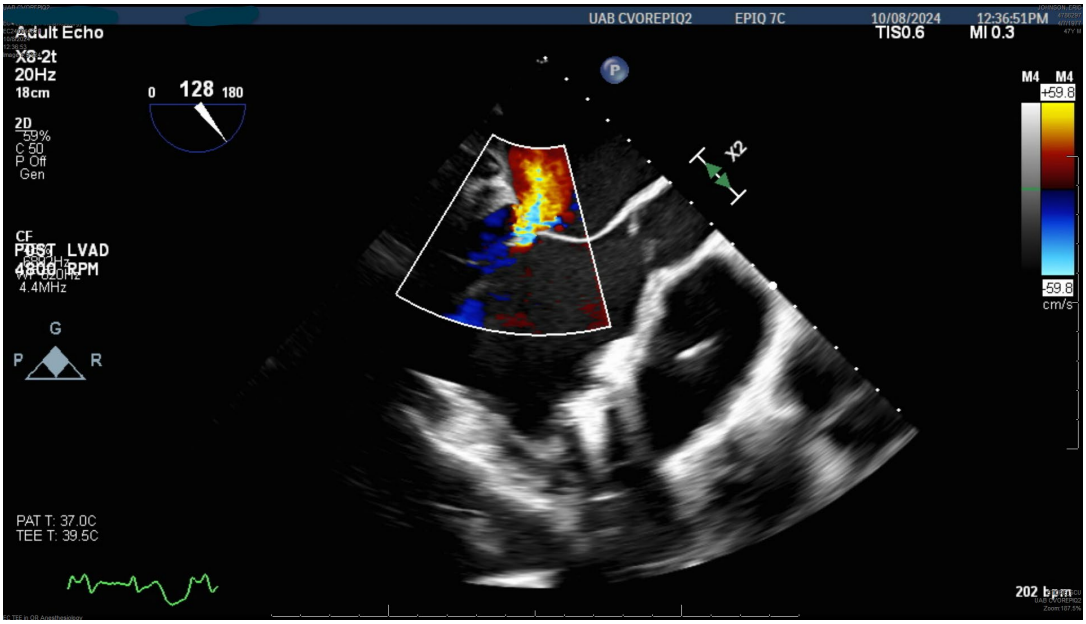
Discussion

This case highlights a rare but serious complication of pledget dislodgement post-LVAD implantation. In this instance, the aortic valve remained closed, preventing systemic embolization; however, migration into the LVAD caused complete pump obstruction. Most LVAD malfunctions (>60%) are due to components such as controllers, batteries, and patient cables, while only 13% stem from pump failure itself[2]. Early TEE identification of the pledget allowed for precise intervention, avoiding full device replacement, preventing a prolonged CPB run, and yielding significant cost savings. Previous reports describe pledget embolization into LAD resulting in LV failure. In one instance, the pledget was flushed out once retrograde cardioplegia was given for cardiopulmonary bypass[3]. Had the aortic valve been opening, systemic embolization with stroke, limb ischemia, or bowel infarction would have been likely. This case raises the question of whether similar events may go unrecognized intraoperatively and underscores the importance of vigilant echocardiographic assessment during LVAD placement.

Images



Mid esophageal long axis view depicting echogenic object within left ventricle following LVAD placement.



Mid esophageal long axis view depicting left ventricle without foreign body.

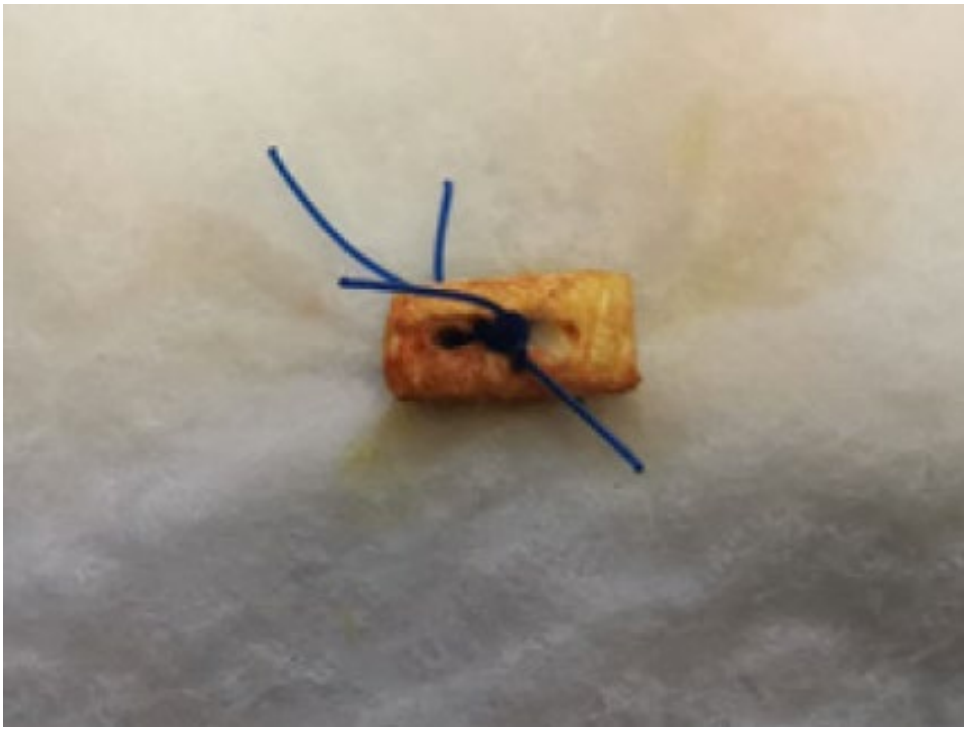


Image of pledget removed from LVAD pump following disassembly.

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#FR 3 – Kiosk 15 – Poster 3

Echocardiographic Considerations in a Calcified Right Atrial Mass Excision

Presenting Author: Matthew E. Mahoney, M.D., Resident Physician, McGaw - Northwestern Medicine

Introduction

Intracardiac masses (ICMs) pose unique challenges for anesthesiologists. The most frequent ICMs are thrombi and valvular vegetations secondary to infective endocarditis. Less commonly, ICMs represent primary cardiac tumors. Thrombi most commonly arise in patients with arrhythmias, but have been reported in patients with hypercoagulable states, such as autoimmune diseases.[1] Transesophageal echocardiography (TEE) is important to the characterization and excision of ICMs. In a review of 75 ICM removal surgeries, pre- and post-cardiopulmonary bypass (CPB) TEE identified new findings in 16% of patients. This resulted in a change of initial surgical plan in six patients, a return to CPB and further surgical exploration in three patients, and intraoperative management changes in three patients.[2] We present a case of a minimally-invasive right atrial thrombus excision guided by TEE in a patient with antiphospholipid syndrome (APS).

Case Presentation

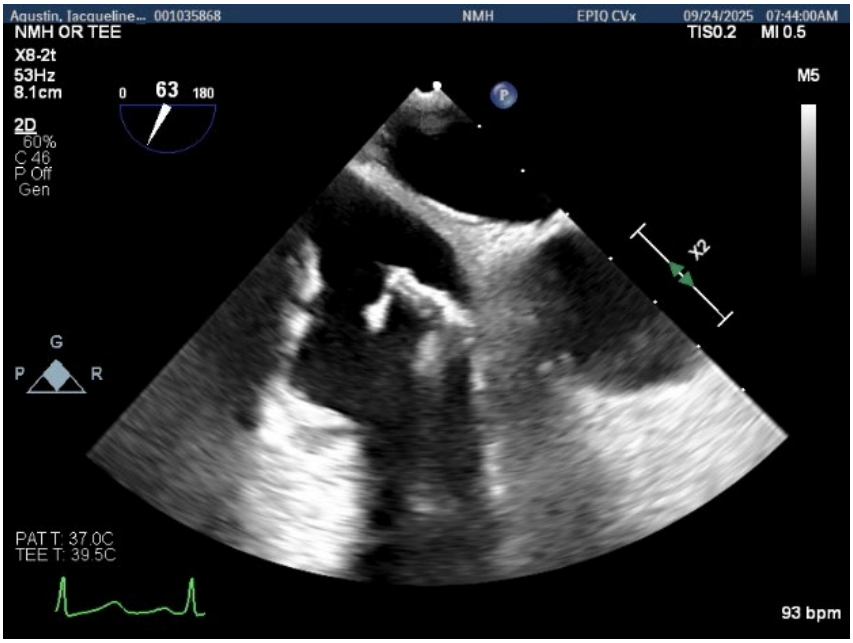
A 45 year old female (BMI 32) with APS complicated by pulmonary embolism (on Warfarin) underwent a coronary calcium score CT scan that incidentally revealed a right atrial mass. Subsequent transthoracic echocardiography and cardiac MRI characterized the mass as abutting the interatrial septum, with otherwise normal cardiac findings. The patient elected to proceed with minimally invasive resection via a right mini-thoracotomy. Intraoperative TEE confirmed a hyperechoic right atrial mass located along the posterior wall without involvement of the interatrial septum (Figure 1). The hyperechoic appearance of the mass caused shadowing in the field deep to the mass, precluding thorough imaging of this area. Following excision, post-procedure TEE demonstrated an echodensity deep to the former mass location, raising concern for a second mass (Figure 2). The surgeon manipulated the right atriotomy suture line, confirming the echodensity as an imbricated suture line (Figure 3). The patient was extubated, transferred to the intensive care unit, and discharged home on postoperative day five. The excised mass measured 2.9 x 2.1 x 1.4 cm. Pathology determined the mass to be a calcified amorphous tumor, and given the patient's history of APS, this was most consistent with a calcified thrombus.

Discussion

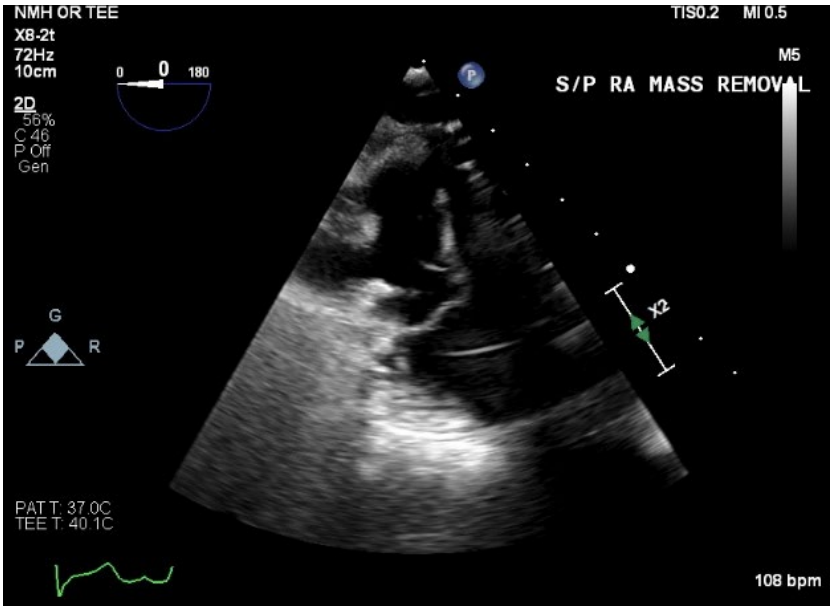
ICMs are rare, but familiarity with accurate imaging via TEE is an important skill for the cardiac anesthesiologist. ICMs can obscure portions of the heart being imaged, further supporting the importance of thorough TEE imaging following mass removal. It can be difficult for the surgeon to assess for complete mass removal, and TEE is a valuable adjunct to ensure completeness of ICM removal.[2] One case report by Golorgorsky *et al.* found that TEE imaging while repeatedly filling and draining the heart with blood after mass removal, but before separation from CPB, allowed for better assessment of residual ICMs, like thrombi. The authors also mentioned the potential for false positive findings due to anatomic variations, such as

thickened papillary muscles, chordae, aberrant tendons, and thickened wall projections, similar to the case we present.[3] TEE is the gold-standard in ICM imaging, but it is not without some limitations that the cardiac anesthesiologist must be aware of to best care for patients presenting for ICM excision surgery.

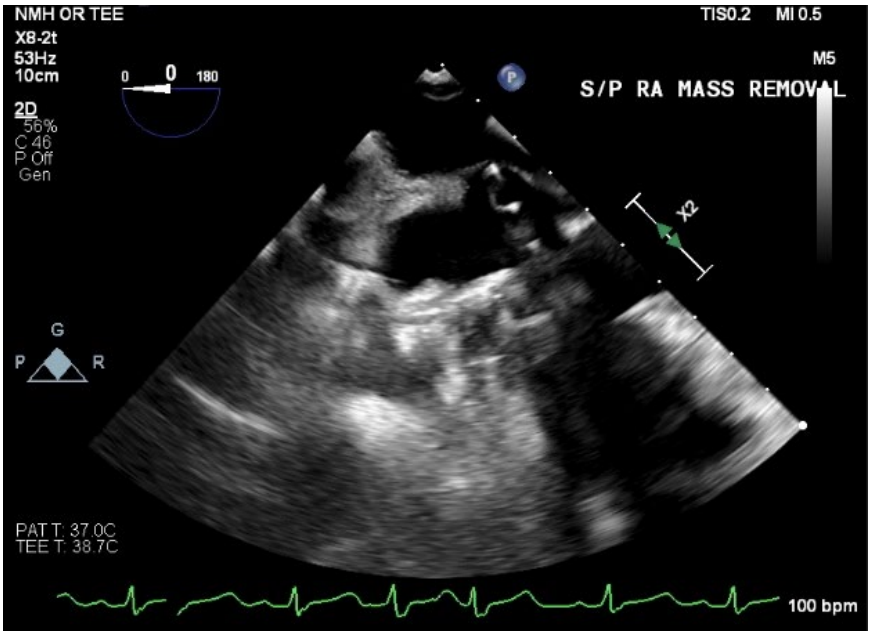
Images



Pre-CPB TEE image showing the right atrial mass and echodense calcifications resulting in acoustic shadowing.



Post-CPB TEE image showing a newly visualized echodense structure in the area previously shadowed by the right atrial mass.



Post-CPB TEE image while the surgeon manipulated the right atriotomy suture line, confirming the echodense structure visualized was an atypical appearance of an imbricated suture line.

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#FR 3 – Kiosk 15 – Poster 4

Anesthetic Considerations in Managing Atrioventricular Groove Rupture During Transatrial Mitral Valve Replacement

Presenting Author: Giovanna Patafio, DO, Adult Cardiothoracic Anesthesiology Fellow, Rutgers Robert Wood Johnson University Hospital

Introduction

Transatrial mitral valve replacement (TMVR) is a procedure for patients with high surgical risk and severe mitral annular calcification (MAC). Despite advances in structural heart techniques, TMVR in severe MAC carries notable perioperative risk. A rare but often fatal complication is an atrioventricular (AV) groove rupture. We present a case of catastrophic AV groove dehiscence following attempted transatrial SAPIEN valve implantation in a high surgical risk patient with severe symptomatic mitral regurgitation (MR).

Case Presentation

Patient is an 84-year-old female with prior CABG via sternotomy and bioprosthetic aortic valve replacement who presented with progressive dyspnea due to severe MR. She was deemed inoperable for conventional mitral surgery secondary to prior cardiac surgery and severe MAC. Given her declining quality of life, a multidisciplinary consensus advised an off-label transatrial TMVR via thoracotomy as her only therapeutic option. Anesthesia induction was uneventful. Intraoperative TEE confirmed severe MR and reduced left ventricular systolic function. A right thoracotomy was performed, followed by a left atriotomy. The anterior leaflet of the mitral valve was then resected. However, because of the approach, angled trajectory, and dense calcification, balloon dilation was necessary before valve expansion. Then a 29-mm valve was positioned and deployed uneventfully under direct visualization. TEE showed proper placement and function of the valve. When attempting to wean from bypass, an enlarging posterior pericardial collection was seen. TEE showed calcified material protruding through the myocardium, concerning for AV groove rupture. The anesthesia, CT surgery, and interventional cardiology teams agreed that the patient would not survive a redo sternotomy, AV groove reconstruction, prosthetic aortic valve excision, coronary bypass, and mitral valve re-replacement. The only chance of survival was if the rupture tamponaded. The atriotomy was closed, hemostasis was confirmed, and a transfemoral intra-aortic balloon pump was placed for circulatory support. The patient was separated from bypass, heparin was reversed, and massive transfusion was initiated. On TEE the posterior collection clotted, but her heart function deteriorated, progressing to right ventricular failure and heart stagnation. Despite maximal resuscitative efforts she expired intraoperatively.

Discussion

This case illustrates the fatal risk of AV groove disruption associated with transatrial TMVR with severe MAC. While TMVR may provide symptom relief for high-risk patients, procedural planning must recognize the limitations imposed by MAC, limited bailout options, and likely futility of emergent repair if rupture occurs. In this patient with prior CABG and AVR, the thoracotomy approach that was selected to avoid resternotomy limited operative exposure and hindered management of the posterior pericardial collection. Intraoperative

TEE was pivotal for injury identification, hemodynamic assessment, and real-time clinical guidance by detecting the effusion and monitoring heart function. This case underscores the importance of multidisciplinary planning, preoperative counseling regarding procedural risk, and heightened vigilance with continuous TEE during TMVR in high-risk patients.

Images





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#FR 3 – Kiosk 15 – Poster 5

Anesthetic Considerations for a Patient with Alpha-gal Syndrome undergoing Coronary Artery Bypass Graft

Presenting Author: Divya Jain, MD, University of Maryland Medical Center

Introduction

Alpha-gal syndrome is an allergic reaction in which IgE targets galactose-alpha-1,3-galactose (alpha-gal), a carbohydrate expressed in non-primate mammalian tissue.[1] It is acquired by certain tick bites and can cause anaphylaxis, angioedema, urticaria, and gastrointestinal symptoms after exposure to mammalian derived products, including medications and bioprosthetics.[2] This is particularly relevant in anesthesia, where many common medications contain mammalian proteins, and even more relevant in cardiac surgery because of the extensive use of heparin and bioprosthetic valves. Despite these risks, no widely accepted standards of care currently exist for managing these patients.[1]

Case Presentation

A 67-year-old male with alpha-gal syndrome (manifesting as rash) presented for quadruple coronary artery bypass graft (CABG). Pre-operative evaluation showed an alpha-gal IgE level of 19.6 kU/l (normal < 0.10 kU/l). He completed a heparin challenge in his allergist's office with 5,000 units subcutaneous heparin without reaction, thus his allergist recommended proceeding with intraoperative and postoperative heparin use for CABG. Based on the allergist and cardiologists' recommendations, the patient was pre-medicated with 10mg cetirizine for 2 nights before surgery, 50mg oral prednisone 13 and 7 hours prior to surgery, and oral methylprednisolone 32mg 1 hour prior to surgery. Furthermore, he received 1g intravenous methylprednisolone and 25mg diphenhydramine prior to induction. Propofol was avoided due to variable safety profile. He received 30,000U IV heparin prior to onset of cardiopulmonary bypass, without any reaction, and continued to receive heparin postoperatively. Other medications were cross-checked for mammalian derivatives, and potentially unsafe drugs including hydromorphone, oxycodone, oral acetaminophen, and oral ondansetron were avoided. Amicar, preparations of norepinephrine and epinephrine, sugammadex and nitroglycerin were found to be safe. The hospital pharmacy verified all medications. The patient did not have allergic reactions to heparin or other medications during his admission. The safety profiles of medications commonly used in cardiac surgery are shown in table-1.

Discussion

When caring for patients with alpha-gal syndrome, minimizing the risk of reaction is essential. This includes investigating which medications and formulations have potential for reactivity. For example, propofol's safety profile depends on the formulation. Additionally, alpha-gal IgE levels can help assess severity and, if necessary, allergy testing can be performed for medications such as heparin. This information may help guide anesthesiologists to make appropriate medication regimens. More research is required to develop universal guidelines for patients with alpha-gal syndrome. Anesthesia providers play a large role in keeping patients with alpha-gal safe in the perioperative period, thus understanding how to prevent reaction in

alpha-gal patients is critical.

Images

Antibiotics	Inactive Ingredients	Safe
Cefazolin	None	Yes
Vancomycin	Hydrochloric acid, sodium hydroxide	Yes
Dexmedetomidine IV	Sodium chloride	Yes
Propofol IV	Glycerin, egg phospholipids, soybean oil, benzoyl alcohol, sodium benzoate, sodium hydroxide Fesenius Kabi brand's glycerin is plant-derived so safe, (eggs are ok for alpha-gal), Pfizer cannot confirm how glycerin in their product is derived so not safe	Yes/No (Brand dependent)
Etomidate IV	Propylene glycol	Yes
Ketamine IV	Benzethonium chloride	Yes
Midazolam IV	Sodium chloride, hydrochloric acid, sodium hydroxide, edetate disodium, benzyl alcohol, sodium chloride, hydrochloric acid, sodium hydroxide	Yes
Lorazepam tablets	Magnesium stearate	No
Midazolam oral syrup	Glycerin	No
Rocuronium	Sodium acetate, sodium chloride, acetic acid, sodium hydroxide	Yes
Enoxaparin	Pork	No
Heparin	Pork	No
Epinephrine	Sodium chloride, sodium metabisulfate, sodium hydroxide, tartaric acid, edetate disodium, hydrochloric acid	Yes
Norepinephrine	Sodium chloride, citric acid, sodium hydroxide, hydrochloric acid	Yes
Clevidipine	Glycerin	No
Nitroglycerin	Dextrose monohydrate, alcohol, citric acid monohydrate, sodium hydroxide, water, hydrochloric acid, propylene glycol	Yes
Nicardipine	Sodium chloride, sodium hydroxide, 2,4 dihydroxybenzoic acid	Yes
Ondansetron tablets	Magnesium stearate	No
Acetaminophen tablet, oral solution	Stearic acid	No
Acetaminophen IV	Mannitol, cysteine hcl, sodium phosphate, dibasic, sodium hydroxide, hydrochloric acid, water, nitrogen	Yes
Ibuprofen tablet	Magnesium stearate	No
Hydromorphone IV, tablet	Magnesium stearate	No

Table-1: Safety profile of commonly used medications based on institutional pharmacy


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#FR 3 – Kiosk 15 – Poster 6

Combined Heart Transplant and Transverse Aortic Arch Replacement for End-stage Heart Failure with Chronic Residual Type A Dissection

Presenting Author: Alexander Roesler, MD, Fellow, Duke University

Introduction

Perioperative risk of transplant is increased in patients with aortopathies, which may complicate their candidacy for transplant [1]. However, the 2016 International Society for Heart and Lung Transplantation guidelines do not include aortopathy as a contraindication to transplant [2]. Perioperative morbidity and mortality for aortic dissection is significant and increases further with concomitant end-stage heart failure (ESHF) [3]. Here, we highlight the anesthetic management in a patient who underwent a combined heart transplantation with total aortic arch replacement for chronic type A dissection.

Case Presentation

Our patient presented with new-onset NYHA class IV heart failure with dyspnea and profound fatigue at rest from non-ischemic cardiomyopathy. He was managed outpatient with milrinone, sacubitril/valsartan and diuretics and evaluated for heart transplantation. During evaluation, he was found to have a residual aortic dissection extending from the proximal arch to distal abdominal aorta, status post Bentall procedure for acute Type A dissection. The surgical plan included combined orthotopic heart transplantation and total aortic arch replacement. The procedure was timed such that donor procurement and arch replacement happened concurrently, with the donor heart arriving on an ex-vivo heart perfusion (Transmedics™) system. General anesthesia was induced with propofol and vasopressor and inotropic support, and intravenous access with two 14-gauge IVs and two central lines was obtained. For cardiopulmonary bypass, bicaval venous and right axillary artery cannulations were performed. Deep hypothermic circulatory arrest with cooling to 16.9°C was done to facilitate a zone 2 aortic arch replacement with a 28 mm Dacron 4-branched graft after the native heart was explanted. Cardiac implantation was done during the rewarming period. The total cardiopulmonary bypass time was 275 minutes with 20 minutes of circulatory arrest with cerebral protection via selective anterograde cerebral perfusion. On inotropic support, post-bypass echocardiogram showed moderate RV and mild LV dysfunction (LVEF 45-50%) and patent anastomoses. The procedure was complicated by coagulopathy treated with 1 unit PRBCs, 5 units FFP, 3 units platelets, 2 cryoprecipitate pools, 2 mg factor VIIa and 2,200 units factor IX. Postoperatively, the patient was transferred to the ICU and was extubated in under 6 hours. The patient recovered well and discharged home 3 weeks later.

Discussion

Patients with both aortopathies and ESHF represent a small but high-risk population and are not candidates for mechanical circulatory support while awaiting transplant. At present, there are no clear consensus guidelines for their appropriate surgical management or transplant candidacy, and perioperative management is challenging (2). Aortic surgery with DHCA often induces significant inflammation and coagulopathies necessitating large volume resuscitation, which can worsen potential ischemic-reperfusion

injury and precipitate right heart failure. The success of this high-risk case was possible due to the availability of highly specialized anesthesiologists and multidisciplinary collaboration at all timepoints—from preoperative discussion of candidacy to recovery.

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#FR 3 – Kiosk 15 – Poster 7

Supporting the Systemic Ventricle in Congenitally Corrected Transposition of the Great Arteries During Cardiopulmonary Bypass Separation and Beyond

Presenting Author: Skylar Fuller, MD, Anesthesia Resident, OHSU

Introduction

Congenitally corrected transposition of the great arteries (L-TGA or cc-TGA) is a condition where the systemic ventricle is a morphologic right ventricle, and the subpulmonic ventricle is a morphologic left ventricle. Blood flows from the right atrium - > right atrioventricular valve [RAVV; morphologic Mitral Valve] - > subpulmonic ventricle - > pulmonary artery - > pulmonary veins - > left atrium - > left atrioventricular valve [LAVV; morphologic Tricuspid Valve] - > systemic ventricle - > aortic valve - > left-sided aorta (figure 1).[1] The natural course of uncorrected cc-TGA is LAVV regurgitation and systemic ventricular failure.[2]

Case Presentation

A 62 y.o. man with cc-TGA (diagnosed at age 40, no previous surgical interventions) presented for LAVV replacement due to severe LAAV regurgitation and progressing systemic ventricular failure. Given his high risk of systemic ventricular failure after cardiopulmonary bypass (CPB), pre-operative preparation was made for likely post-operative mechanical circulatory support (MCS). Heart transplantation was designated as a possible long-term contingency plan.

Pre bypass Course

Pre-intervention TEE showed a systemic ventricle that appeared moderately-severely dilated in size with severely reduced function; Fractional Area Change (FAC) was < 20% from a ME4Ch view. Systemic ventricular function was marginally improved to moderately reduced function with initiation of 0.02 mcg/kg/min of epinephrine. The LAVV appeared tricuspid by morphology with severe central regurgitation. TEE imaging of the systemic ventricle brought into question the surgical plan for pre-emptive Impella 5.5 placement given anatomical constraints: marked trabeculation and complex geometry.

Post bypass Course

Following LAVV replacement, preload, afterload, and contractility were optimized using epinephrine at 0.06 mcg/kg/min, milrinone at 0.375 mcg/kg/min, and vasopressin at 4 units/hr. Fortunately, the patient was successfully weaned from CPB without the need for MCS. The patient had significantly reduced systemic ventricular function but appreciable myocardial recruitment with inotropy. The patient recovered well; he was extubated on post-operative day (POD) 0, inotropy was slowly weaned over days, and he was discharged home on POD 7.

Discussion

There are case reports describing the use of ventricular assist devices (VADs) to provide long-term MCS in patients with cc-TGA and end-stage systemic ventricular dysfunction. However, limited data exists regarding

the optimal strategies for short-term MCS in patients like ours—those undergoing isolated LAVV replacement who require MCS to separate from CPB or as a bridge to transplant. In the event of difficulty weaning from CPB, we considered IABP, central VA-ECMO, and Impella CP for systemic ventricular support, though no option was clearly a superior approach. Two months after our case, a single report was published documenting the use of an Impella 5.5 as a bridge to a VAD in a cc-TGA patient.[3] Further investigation is needed to define best practices for MCS selection in this unique patient population.

Images

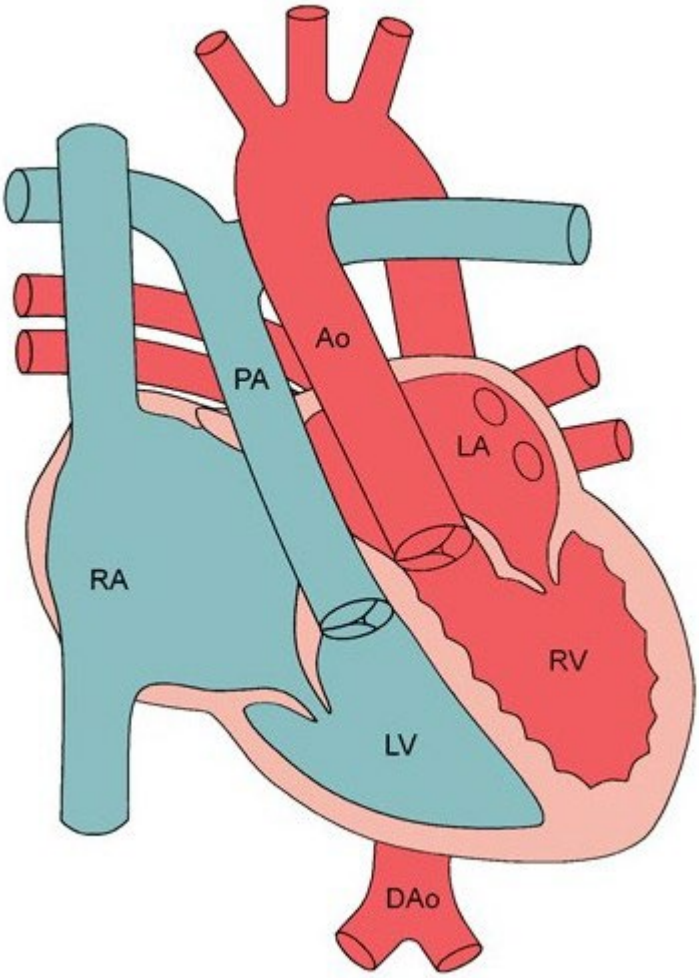


Figure 1: ccTGA(1)

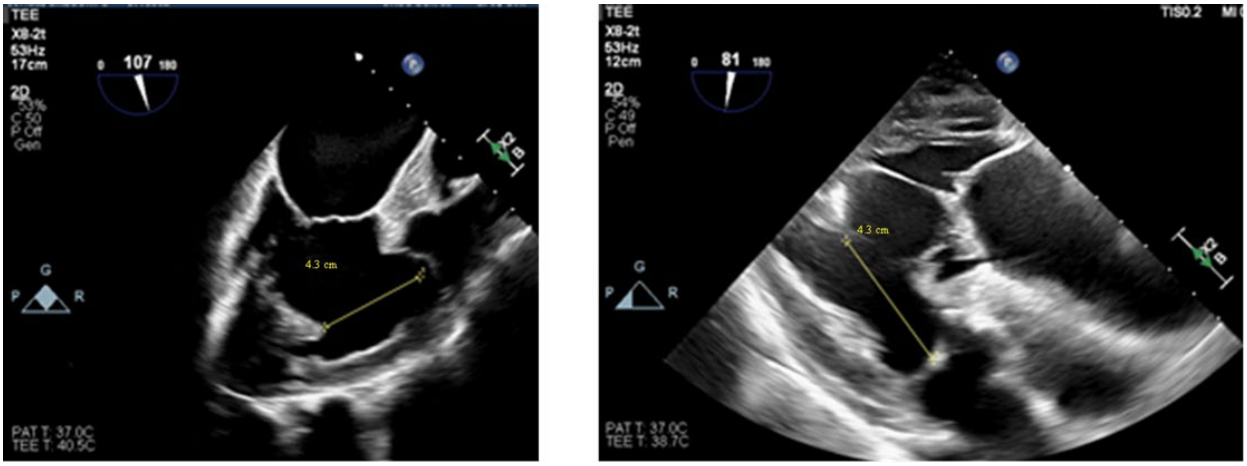


Figure 2: The distance from the aortic annulus to the lateral ventricular wall measured 4.3 cm.

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#FR 3 – Kiosk 16 – Poster 1

Redo Surgical Aortic Valve Replacement in a Young Woman with Previously Unrecognized Interrupted Inferior Vena Cava

Presenting Author: Austin Walker, MD, Resident Physician, University of Iowa Hospitals and Clinics

Introduction

Interrupted inferior vena cava (IVC) with azygos continuation is a rare congenital venous anomaly, with an incidence of 0.3% and up to 0.6% in patients with congenital heart disease.1 Although often asymptomatic, the anomaly can significantly affect venous drainage strategies during cardiopulmonary bypass (CPB) and affect transcatheter interventions.2 We present a case in which interrupted IVC was discovered intraoperatively and complicated planned peripheral cannulation for a redo aortic valve replacement (AVR).

Case Presentation

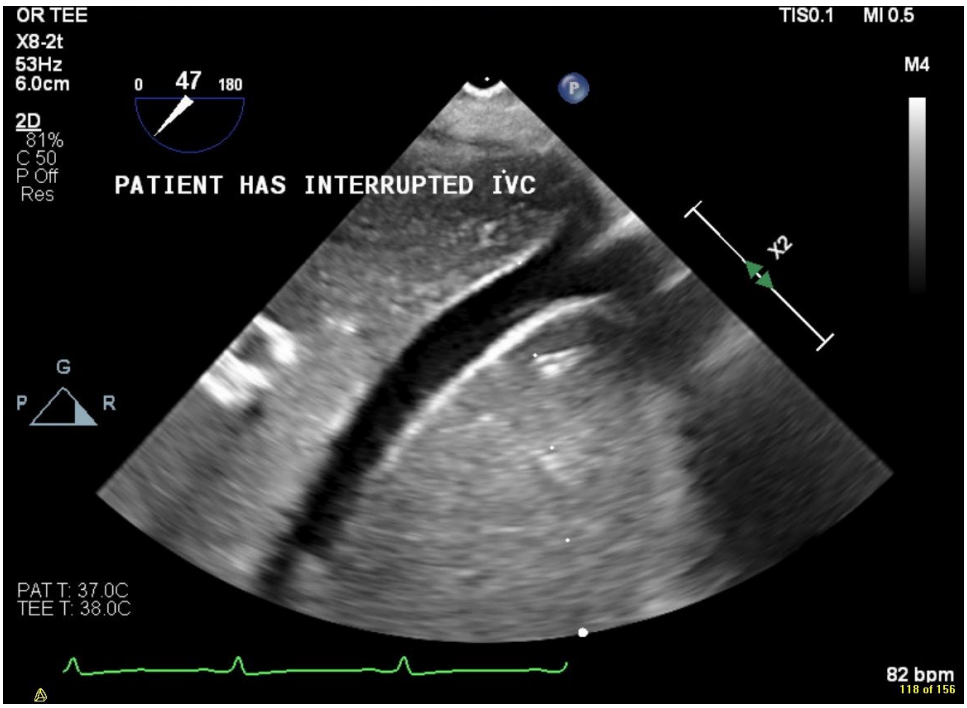
We present the case of a 25-year-old woman with bicuspid aortic valve who underwent balloon valvuloplasty at age 6, bioprosthetic AVR at age 16, and transcatheter valve-in-valve replacement at age 22. She presented to our center for redo surgical AVR with placement of a mechanical valve due to progressive symptomatic severe aortic stenosis with mean gradient 42mmHg. Given anticipated mediastinal adhesions, the surgical plan was to initiate CPB prior to sternotomy using bifemoral cannulation.

Discussion

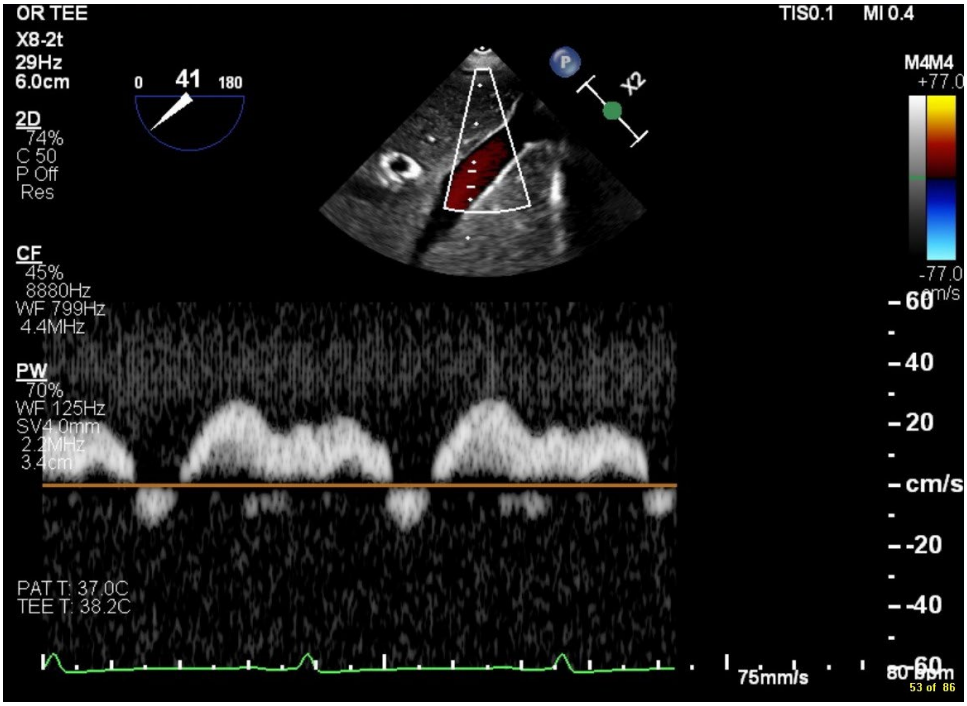
This case demonstrates the impact that interrupted IVC can have on the cannulation strategy for redo cardiac surgery. Because lower-body venous return travels through the azygos system, femoral cannulation will not provide sufficient drainage for full CPB. Intraoperative TEE was crucial in identifying the anomaly in this case. A potential opportunity for improvement would have been superior vena cava cannulation via the internal jugular vein prior to sternotomy to ensure adequate venous drainage for full CPB prior to sternotomy. Preoperative recognition of rare venous anomalies is important in patients with congenital cardiac disease, particularly when peripheral cannulation is planned for safe reentry.


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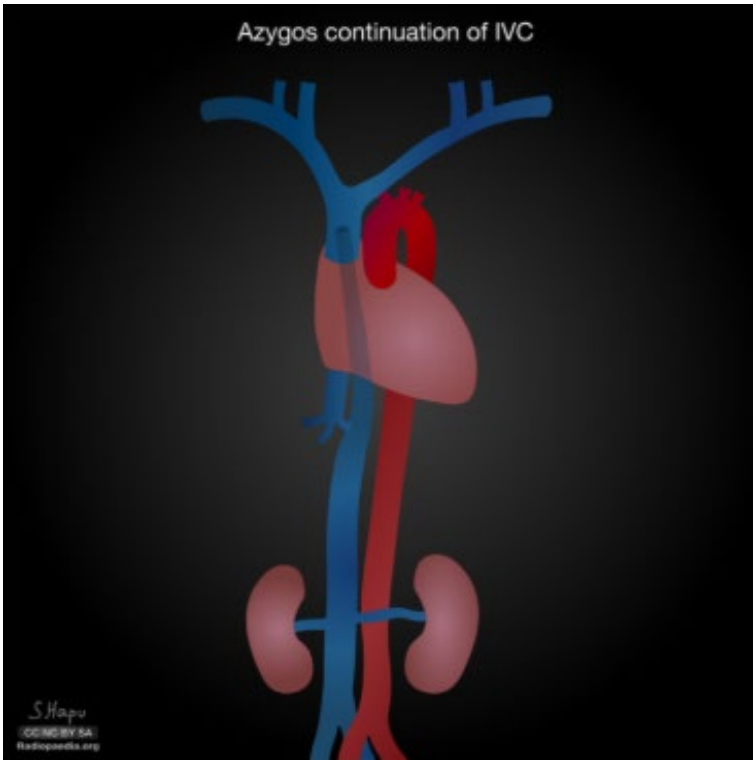
Images



TEE transgastric hepatic vein view demonstrating no intrahepatic IVC



Transgastric hepatic vein view with hepatic vein doppler



Schematic of the interrupted IVC with azygos continuation anomaly

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#FR 3 – Kiosk 16 – Poster 2

An Unforeseen Challenge: Severe Systolic Anterior Motion of the Mitral Valve After Aortic Valve Replacement

Presenting Author: Terrique Pinnock, MD, Mayo Clinic Florida

Introduction

Systolic anterior motion (SAM) of the mitral valve is a well-documented contributor to dynamic left ventricular outflow tract (LVOT) obstruction in hypertrophic obstructive cardiomyopathy (HOCM). However, SAM can also occur without classic HOCM features, where excessive anterior displacement of the mitral chordae tendineae is the primary obstructive mechanism. Specifically, isolated chordal SAM after aortic valve replacement (AVR) is uncommon and may become clinically apparent. In severe aortic stenosis, elevated end-systolic left ventricular (LV) pressure and volume help maintain LV cavity dimensions that may mask underlying LVOT obstruction. Restoration of aortic valve function with surgical intervention alters LV hemodynamic conditions and anatomy that may exacerbate SAM-mediated LVOT obstruction. We present a case of chordal SAM causing significant dynamic LVOT obstruction following AVR and septal myectomy.

Case Presentation

An 80-year-old male with history of severe aortic stenosis, HOCM, and hypertension, presented for a planned AVR and septal myectomy. Preoperative transthoracic echocardiogram (TTE) six months prior demonstrated LV ejection fraction of 74%, obstructive basal and mid-septal hypertrophy, severely restricted aortic valve with valve area of 0.9 cm2 and systolic mean doppler gradient of 78 mmHg, and mitral SAM with mild regurgitation. After an uneventful initiation of general anesthesia, pre-bypass transesophageal echocardiogram (TEE) verified his prior TTE findings. The patient underwent a median sternotomy, heparinization, and central cannulation for cardiopulmonary bypass (CPB). Elective left atrial appendage ligation and standard septal myectomy were performed without incident. The significantly calcified aortic valve was excised and replaced with a bioprosthetic valve. The patient was prepared for reperfusion and successfully weaned from CPB. Post-bypass TEE revealed new chordal SAM and notably worse mitral valve regurgitation due to dynamic left ventricular flow changes compared to pre-bypass TEE assessment. The decision was made to replace the mitral valve after a thorough evaluation by cardiothoracic anesthesiologists and cardiovascular surgeons. The patient returned to a second CPB run, and the previously placed aortic bioprosthetic valve was removed to facilitate visualization of the mitral annulus. The mitral and aortic bioprosthetic valves were inserted, and the patient was weaned from CPB uneventfully. TEE images showed well-functioning bioprosthetic valves, mild mitral valve regurgitation, and other unchanged findings. The rest of the intraoperative course progressed as expected, and the patient was transferred to the intensive care unit for recovery.

Discussion

This case illustrates how chordal SAM can become hemodynamically significant after AVR. Preoperatively, elevated LV pressures from severe aortic stenosis may have obscured dynamic LVOT obstruction. After valve replacement, altered loading conditions uncovered SAM, resulting in severe mitral regurgitation and

LVOT obstruction. Recognition of this phenomenon with intraoperative TEE allowed prompt mitral valve replacement, highlighting the importance of serial intraoperative TEE exams and anticipating SAM in patients with HOCM and valvular disease.

Images

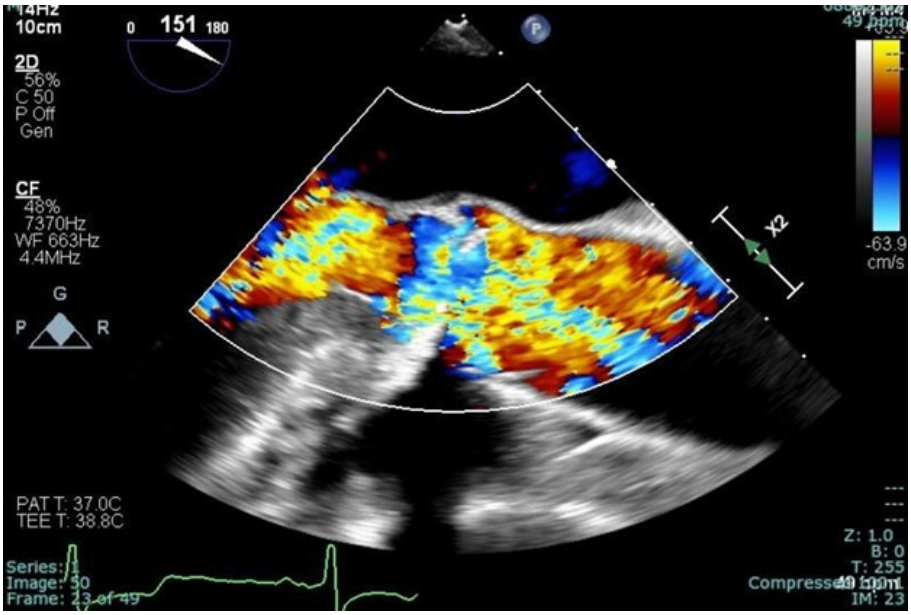


Image 1: Intraoperative transesophageal echocardiogram, midesophageal aortic valve long axis view with color flow doppler showing turbulent flow through the left ventricular outflow tract

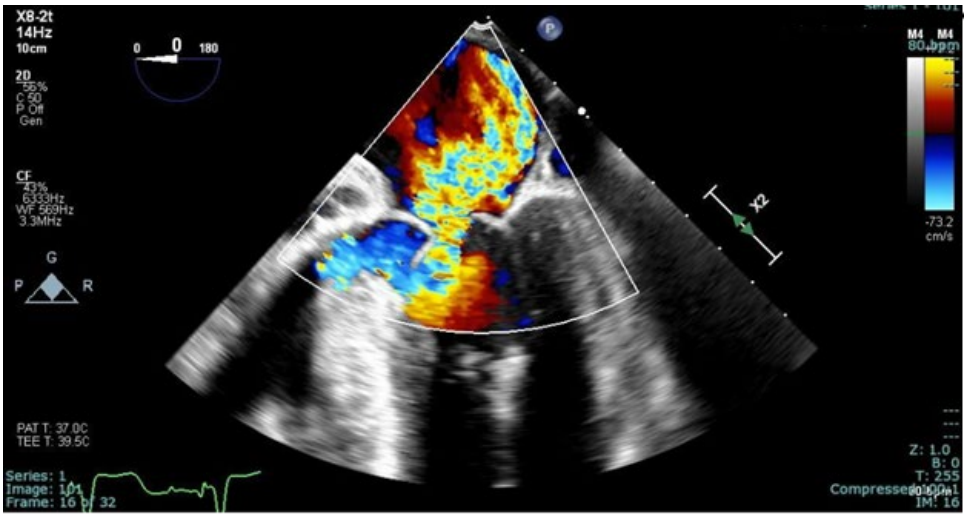


Image 2: Intraoperative transesophageal echocardiogram, deep transgastric long axis view with color flow doppler showing severe mitral regurgitation and significant systolic displacement of the anterior mitral leaflet after initial aortic valve replacement

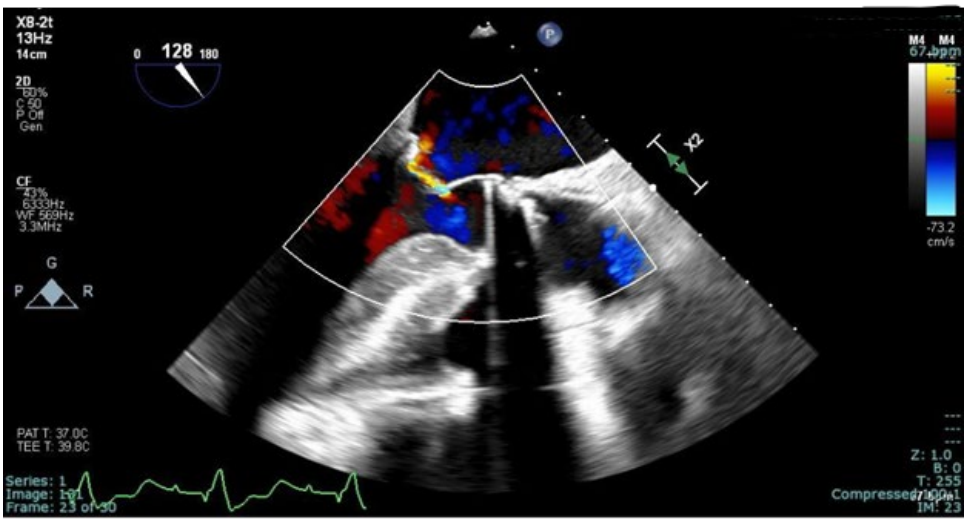


Image 3: Intraoperative transesophageal echocardiogram, midesophageal long axis view with color flow doppler showing mild residual mitral regurgitation after unplanned mitral valve replacement

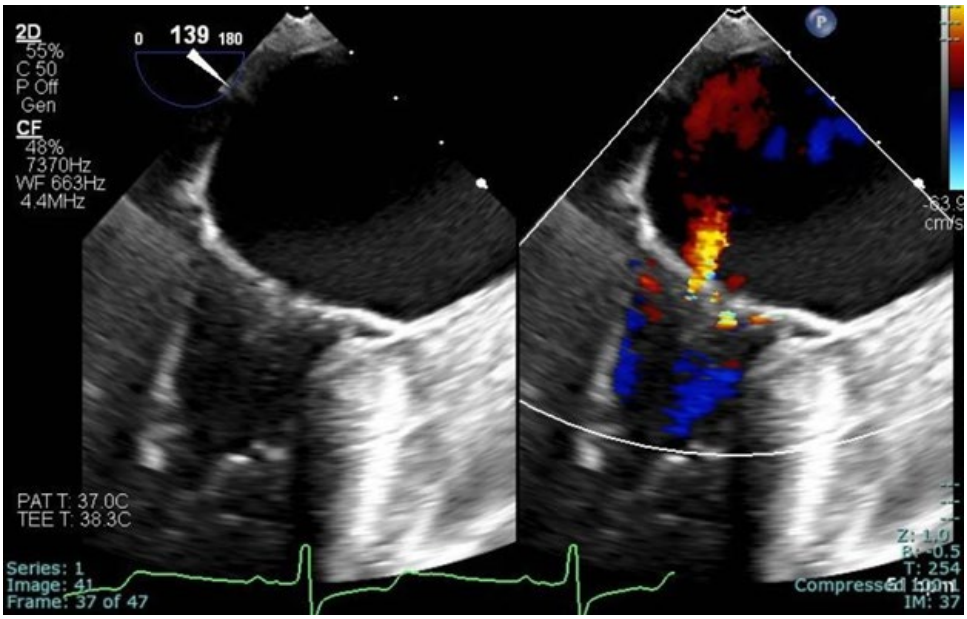


Image 4: Pre-cardiopulmonary bypass transesophageal echocardiogram demonstrating mild mitral regurgitation

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#FR 3 – Kiosk 16 – Poster 3

Intraoperative Detection of Right Pulmonary Artery Thrombus Altering Surgical Strategy in Ovarian Hyperstimulation Syndrome

Presenting Author: Alejandra Silva, M.D., Anesthesiology Resident, University of Miami/Jackson Memorial Hospital

Introduction

Ovarian hyperstimulation syndrome (OHSS) leads to marked third-spacing, hemoconcentration, and a hypercoagulable state capable of producing venous, intracardiac, and pulmonary artery thrombosis. These abnormalities create major anesthetic challenges, especially during airway management and positive pressure ventilation in the presence of right ventricular (RV) outflow obstruction.

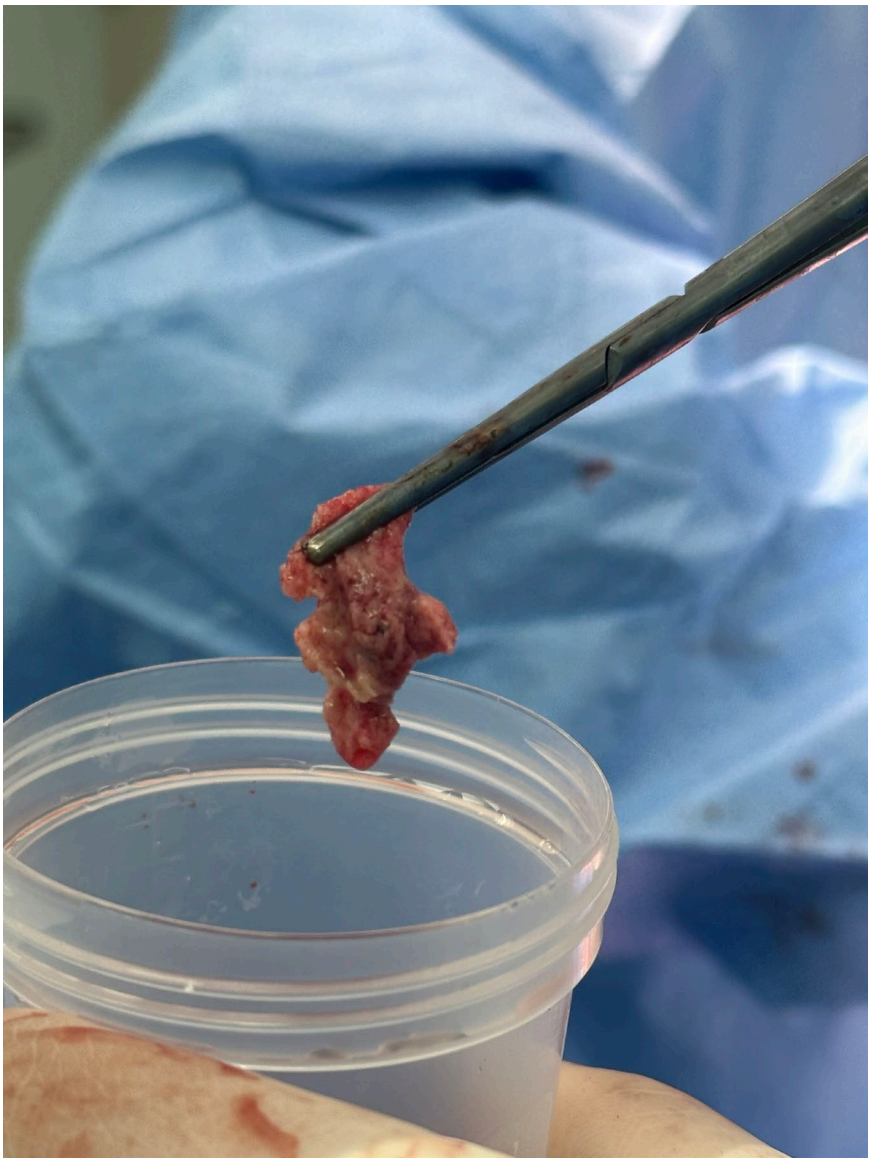
Case Presentation

A 28-year-old woman, 10 days after in vitro fertilization, presented with dyspnea, tense ascites, and bilateral pleural effusions. A transthoracic echocardiogram performed at an outside hospital suggested a possible RV thrombus, and she was transferred emergently for surgical intervention. Airway management and hemodynamic stability were primary concerns due to severe third-spacing and risk of cardiovascular collapse. Arterial and central venous access were obtained, and femoral sheaths were placed before induction for potential rapid initiation of mechanical support. Two awake fiberoptic intubation attempts were unsuccessful; video laryngoscopy resulted in successful intubation with preserved hemodynamics. Bilateral pleural effusions were drained prior to sternotomy. Transesophageal echocardiography (TEE) immediately after induction revealed two critical findings: a 1.8 × 1.83 cm mobile RV thrombus attached to the tricuspid septal leaflet and, unexpectedly, a thrombus lodged in the right pulmonary artery (RPA). This RPA clot had not been appreciated on the preoperative study and significantly altered the operative plan. Instead of isolated intracardiac thrombectomy, the team proceeded with combined RV thrombectomy and pulmonary artery embolectomy to relieve acute RV afterload obstruction. Cardiopulmonary bypass (CPB) was initiated with bicaval and aortic cannulation. A fibrinous clot was removed from the tricuspid valve. The RPA was then opened, and a long white thrombus was extracted. Because visualization of the left pulmonary artery (LPA) is limited by TEE due to anatomic constraints, the LPA was surgically explored and found free of thrombus. A diaphragmatic incision evacuated 2.5 L of ascitic fluid, improving ventilatory mechanics. Final TEE confirmed complete thrombus removal with preserved biventricular function.

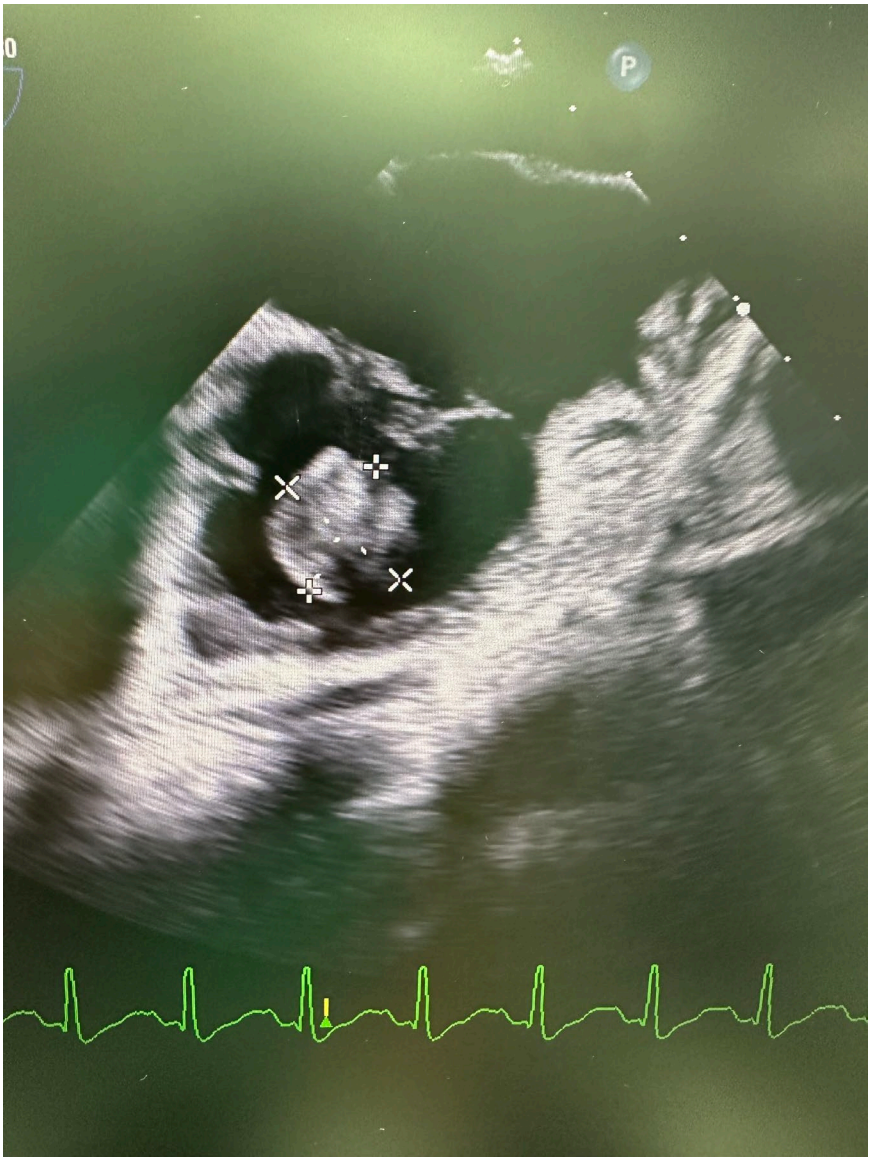
Discussion

This case highlights the essential role of intraoperative TEE in identifying new obstructive pathology that directly changes surgical strategy. Early recognition of the RPA thrombus prevented persistent RV outflow obstruction and potential perioperative collapse. OHSS-related hypovolemia and hypercoagulability further complicated induction and ventilation, emphasizing the need for meticulous hemodynamic planning and comprehensive echocardiographic assessment in patients with thromboembolic complications of OHSS.

Images



Extracted RV thrombus



TEE demonstrating a 1.8 × 1.83 cm echogenic mass attached to the tricuspid septal leaflet

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#FR 3 – Kiosk 16 – Poster 4

TEE-Guided Wire Correction of Intraoperative Aortic Graft Kinking

Presenting Author: Reema Shah, DO, The Zucker School of Medicine at Hofstra/Northwell Residency Program in Anesthesiology (Northshore/ LIJ)

Introduction

Kinking or malalignment of an aortic graft is an uncommon but significant complication that can lead to hemodynamic instability, malperfusion, and ischemia. Standard management often requires re-exploration of the anastomosis or graft repositioning, interventions that may necessitate returning to cardiopulmonary bypass and circulatory arrest. We present a case of a redo arch repair for chronic Type I dissection in which intraoperative graft kinking caused loss of distal arterial pulsatility. The complication was identified using transesophageal echocardiography (TEE) and corrected by advancing a wire through the graft under real-time echocardiographic guidance, immediately restoring systemic perfusion and avoiding a return to bypass.

Case Presentation

A 65-year-old male with prior repair of an acute DeBakey type I dissection presented with progressive back pain. Imaging showed aneurysmal degeneration of the aortic arch and a thoracoabdominal aneurysm concerning for impending rupture. He underwent redo sternotomy, aortic arch replacement, and placement of an elephant trunk into the descending thoracic aorta. Prior to weaning from bypass, the radial arterial line was observed to be pulsatile while the femoral arterial line was nonpulsatile. TEE of the aortic arch / descending aorta revealed kinking of the distal graft near a large fenestration between the true and false lumens. Under TEE guidance, a soft wire followed by a stiff Amplatz wire was advanced antegrade to re-expand the kinked segment. Blood flow was immediately restored, and a pulsatile waveform was seen on the femoral arterial line.

Discussion

Graft kinking is a rare but critical complication during aortic reconstruction. The direction of blood flow, length of the graft, positioning, and graft type can all play a role in the mechanism of this kinking, however none are known to be true risk factors. Vigilance is essential, as early detection can reduce patient morbidity and mortality. In this case, a distal kink occurred after systemic perfusion was reinstated, prior to separation from bypass, highlighting a critical timeframe during which this complication can present. A nonpulsatile femoral tracing provided the initial clue, and TEE confirmed the diagnosis. Traditional correction often requires reinitiating circulatory arrest and revising the distal anastomosis, which increases operative risk. In contrast, the TEE-guided wire technique allowed rapid, minimally invasive correction with real-time confirmation of restored flow. The success of this technique spared the patient a second cardiopulmonary bypass run and circulatory arrest, thereby reducing the risk of coagulopathy as well as perioperative neurologic, cardiac, and renal injury. This case highlights the value of TEE not only for diagnosis but also as a procedural tool in complex aortic surgery. It also underscores the importance of considering graft kinking in

unexplained distal malperfusion, particularly in reoperative dissections.

Images

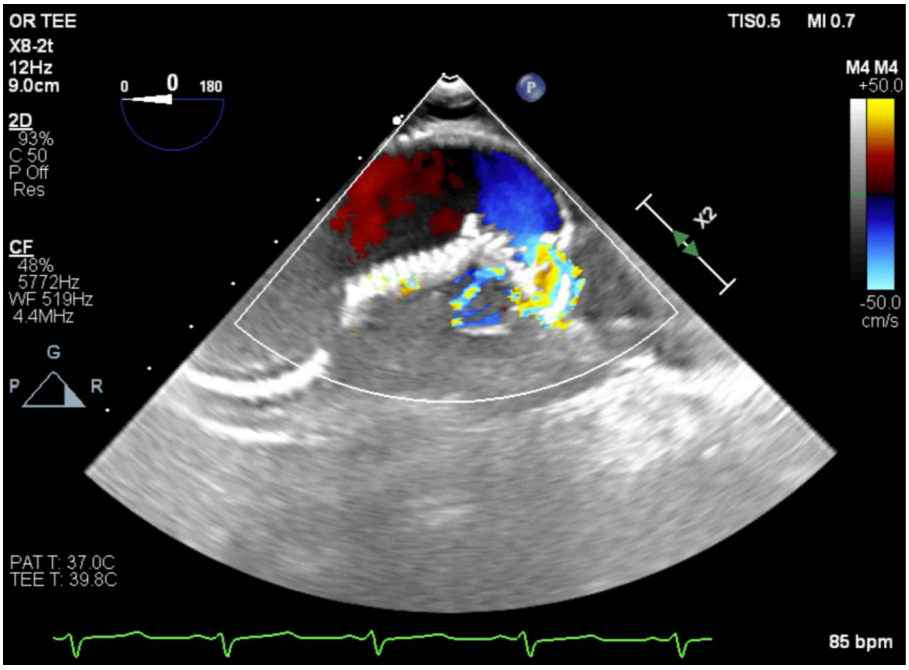


Figure 1: Upper esophageal long axis view of kinked distal aortic graft in distal aortic arch

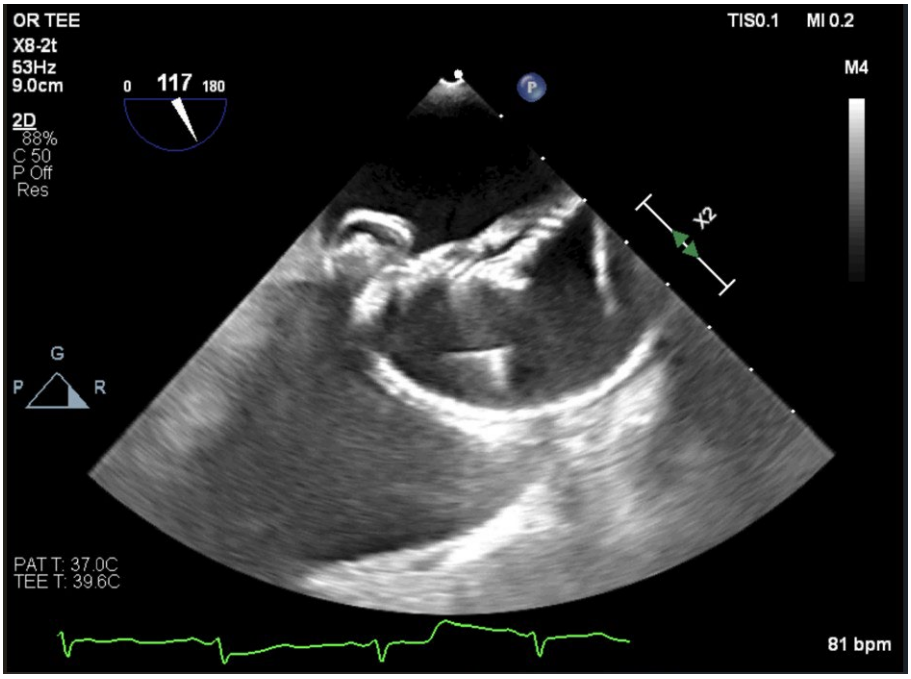


Figure 2: Descending aorta view of kinked distal aortic graft in a tortuous descending aorta.

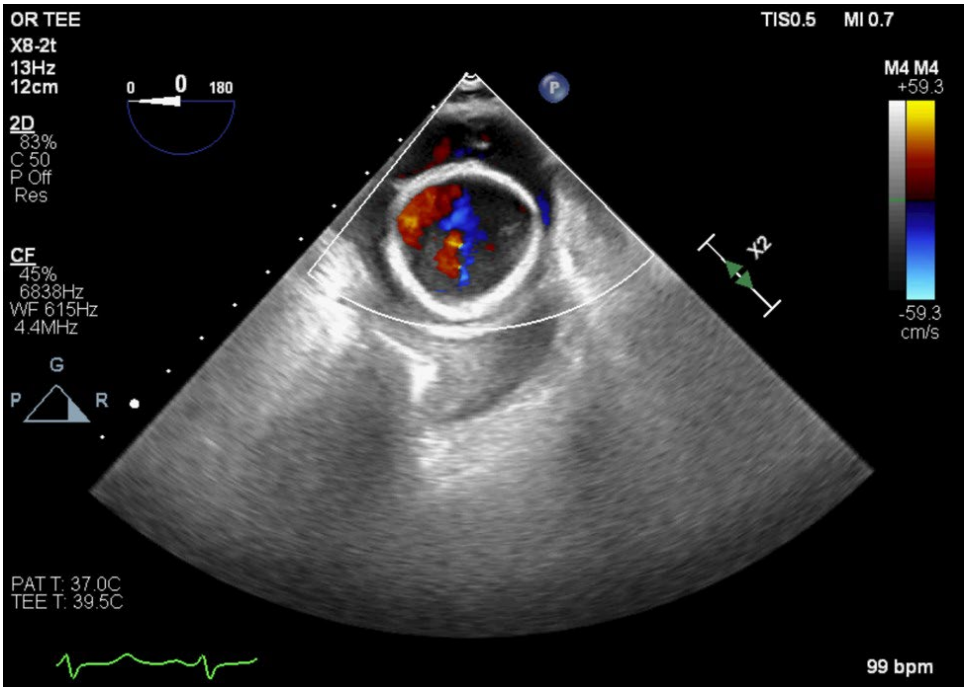


Figure 3: Descending aorta short axis color Doppler view of patent distal aortic graft after wire correction

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#FR 3 – Kiosk 16 – Poster 5

"It's a Bird, It's a Plane, It is a Ruptured Sinus of Valsalva Aneurysm!" -Rare Differential Diagnosis in a Patient with Left to Right Shunt

Presenting Author: Vincent L. Allen, MD, Resident Physician, Department of Anesthesiology, University of Maryland School of Medicine

Introduction

We present a case of diagnostic uncertainty in a patient with symptomatic left-to-right (L-to-R) shunt despite extensive preoperative workup. The shunt was initially thought to be secondary to a Gerbode defect, which is a shunt from the left ventricle through the atrioventricular membranous septum into the right atrium (RA). On intraoperative tranesophageal echocardiography (TEE), the etiology of the shunt was ultimately found to be a congenital Sinus of Valsalva Aneurysm (SOVA). A congenital SOVA results from weakness in the elastic lamina at the junction of the aortic media and annulus fibrosis. It is defined as a diameter greater than 4.0 cm in men and 3.6 cm in women. [1]

Case Presentation

A 37-year-old female with no significant past medical history first presented in 2017 with shortness of breath, exercise intolerance, and palpitations. She also reported multiple syncopal episodes, one notable for hypoxia (SPO2 77%) and tachycardia (147 bpm). Initial work up with an implantable loop recorder was without notable arrhythmias, but the patient was started on propranolol for presumed SVT. Due to continuing symptoms, the patient underwent cardiac catheterization (2017) that reported an ASD with L-to-R shunt. Cardiac MRI showed a restrictive membranous VSD, unable to rule out ventriculoatrial communication. TTE report (2023) noted possible small inlet restrictive VSD versus Gerbode defect versus possible sinus rupture. When symptoms worsened in 2025, the patient underwent repeat cardiac catheterization with intended closure of suspected Gerbode VSD. Intervention was deferred due to diagnostic uncertainty as angiography showed contrast flow from right Sinus of Valsalva into both RA and pulmonary artery. The patient was then referred to cardiac surgery for definitive management. Given the possibility of L-to-R shunt induced volume overload, pulmonary HTN, and right ventricular failure, a preinduction arterial line was placed. A pulmonary arterial catheter was placed under careful TEE guidance as altered anatomy and shunt pathway were likely present. TEE evaluation revealed the etiology of L-to-R shunt to be a large SOVA aneurysm with rupture into both the RA and right ventricle and a Qp:Qs of 1.3. Other potential co-existing cardiac congenital abnormalities were also ruled out on TEE. The aneurysm was surgically repaired. Post procedure TEE confirmed resolution of shunt through the original defect. Post repair Qp:Qs was within normal limits.

Discussion

SOVAs are either congenital from tissue weakness along aortic annulus or acquired after trauma or infections such as endocarditis. SOVAs are easily mistaken for more common pathologies since the estimated prevalence is only ~0.09%. Thus, a high degree of suspicion is required to make the diagnosis.[2] On

TEE, the best views for SOVA include ME AoV LAX and SAX views. Spectral doppler is used to differentiate between VSD and SOVA rupture. While VSD shows high velocity jet during systole, SOVAs will often show continuous flow through the shunt and increased velocity during diastole.[3] This case highlights the importance of a careful intraoperative TEE evaluation and the need to avoid anchoring bias, which together allow clinicians to detect pathologies that may otherwise be missed preoperatively.

Images

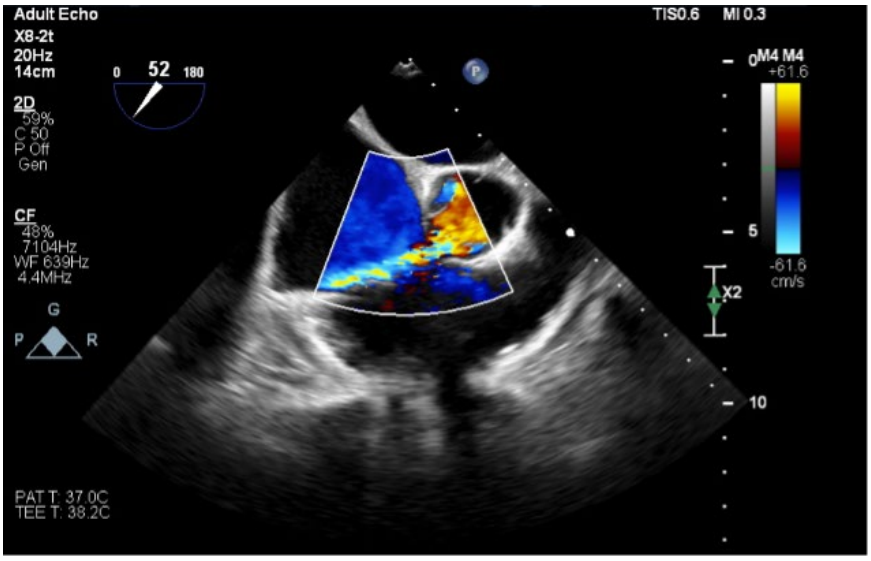


Figure 1: Mid esophageal short axis aortic valve view with color doppler showing flow from the right sinus of valsalva into the right atrium just above the tricuspid valve

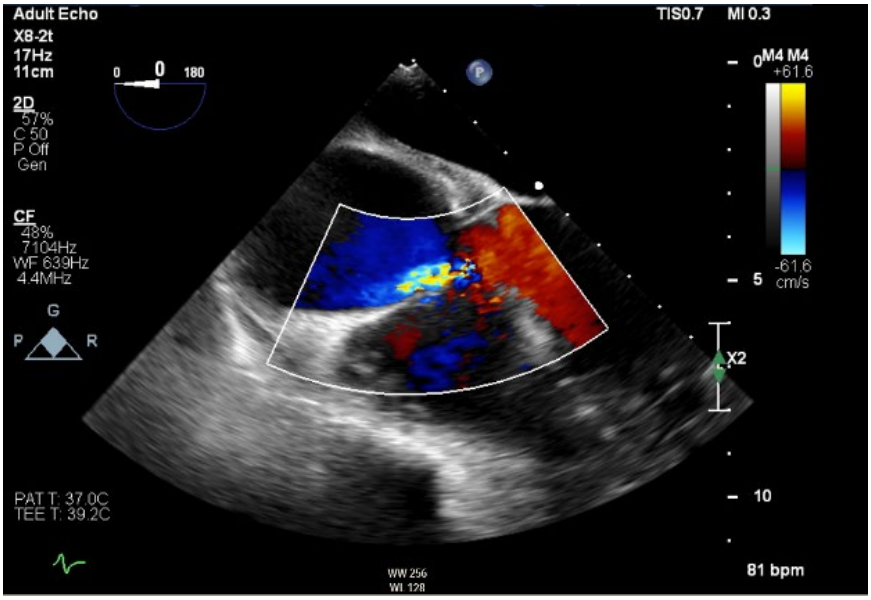


Figure 2: ME 5 chamber view showing flow from root to right atrium

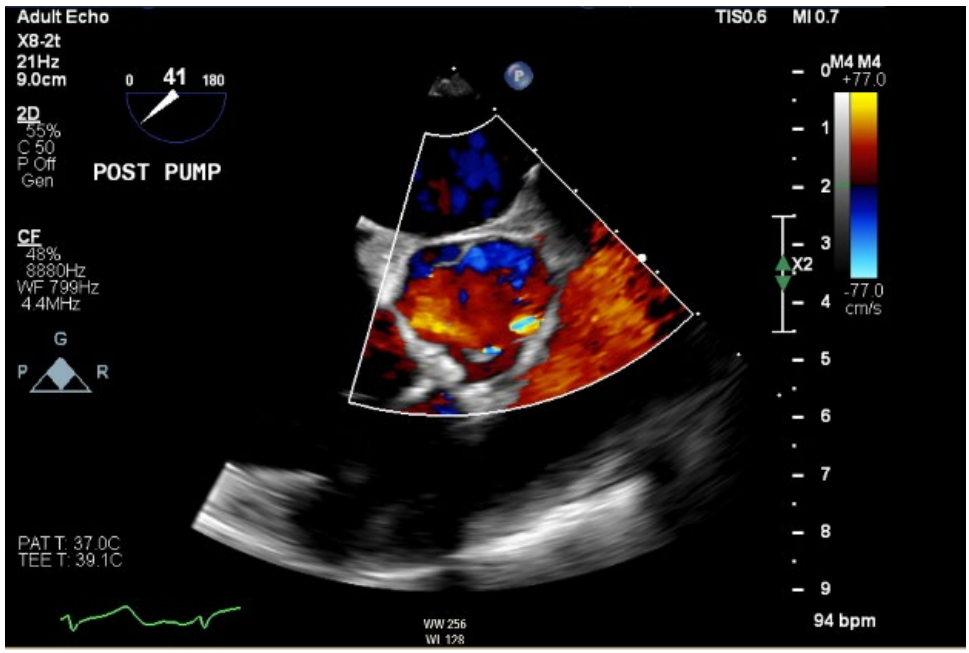


Figure 3: Post CPB ME AoV SAX view with doppler. No residual flow seen through defect

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#FR 3 – Kiosk 16 – Poster 6

Airway Compromise from Expanding Vertebral Artery Pseudoaneurysm in a Patient with Vascular Amyloidosis

Presenting Author: Mohammed Yassin, MD, Resident Physician, University of California Los Angeles

Introduction

We present a rare case of airway obstruction caused by an enlarging left vertebral artery pseudoaneurysm in a patient ultimately diagnosed with vascular amyloidosis. Management required staged surgical intervention and complex anesthetic management, including venovenous extracorporeal membrane oxygenation (VV ECMO) support. This case highlights the importance of early recognition, multidisciplinary coordination, and perioperative vigilance in managing vascular lesions with airway involvement.

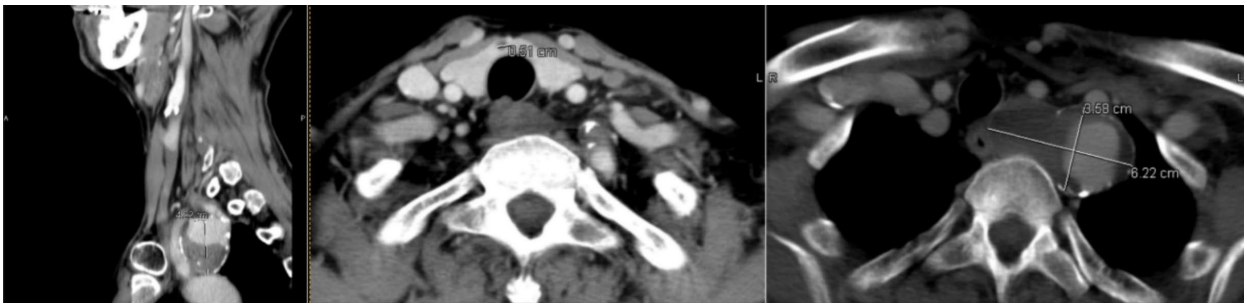
Case Presentation

A 55-year-old male with a history of traumatic left vertebral artery pseudoaneurysm presented in acute respiratory distress with stridor. Computed-tomography imaging revealed a 10.8 cm pseudoaneurysm compressing the trachea. He was preemptively placed on VV ECMO due to anticipated airway compromise. Initial intubation with a 6.0 mm armored endotracheal tube (ETT) failed to bypass the tracheal narrowing and was replaced with a 5.5 mm microlaryngeal ETT via fiberoptic bronchoscopy. A staged surgical plan was adopted: first, bypass of the aneurysm using a saphenous vein graft from the left carotid to the left vertebral artery; second, hematoma evacuation and pseudoaneurysm excision via median sternotomy. Postoperative bronchoscopy confirmed resolution of tracheal compression. The patient was successfully extubated and VV ECMO was discontinued postoperatively. Pathology of the pseudoaneurysm sac showed positive Congo red staining and immunohistochemical analysis was positive for amyloid A and transthyretin, consistent with the diagnosis of amyloidosis.

Discussion

Expanding cervical vascular lesions with airway involvement requires early recognition and coordinated intervention to prevent catastrophic airway obstruction [1]. In this case, ECMO was initiated preemptively to mitigate the risk of airway loss or aneurysm rupture during intubation. The anesthetic approach required fiberoptic intubation, careful tube selection, and hemodynamic control during aneurysm manipulation. The postoperative diagnosis of transthyretin amyloidosis adds complexity, as amyloid infiltration can compromise vascular integrity, cardiac conduction, and autonomic function. While typically associated with cardiac involvement, amyloid deposition in vascular structures may predispose to aneurysm formation and rupture [2]. This case underscores the value of multidisciplinary planning and ECMO as a bridge to airway control and awareness of systemic amyloidosis as a potential contributor to vascular pathology and perioperative risk. Further research is warranted to explore the relationship between amyloid deposition and pseudoaneurysm formation, and to refine anesthetic protocols for patients with dynamic airway obstruction secondary to vascular lesions.

Images



Sagittal and Transverse views of a large pseudoaneurysm arising from the proximal left vertebral artery with tracheal compression and rightward deviation

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#FR 3 – Kiosk 16 – Poster 7

Left Circumflex Injury during Mitral Valve Repair

Presenting Author: Joe Puthumana, Rush University Medical Center

Introduction

The left circumflex artery (LCX) is particularly vulnerable during mitral valve replacement (MVR) due to its proximity to the mitral annulus. The incidence of LCX injury after MVR is listed as between 0.5% and 1.8% (1). Injury can be attributed to a wide variety of causes, and the consequences range from asymptomatic to fatal.

Case Presentation

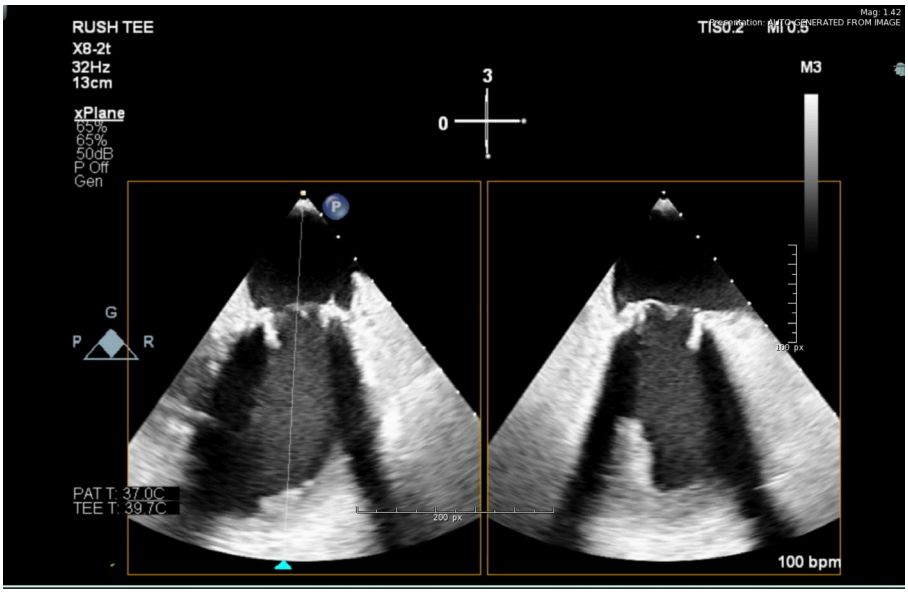
The patient was a 71-year-old female being evaluated for MVR due to severe symptomatic mitral valve stenosis. Preoperative studies revealed a preserved left ventricular ejection fraction of 70% but showed moderate-to-severe mitral stenosis with accompanying regurgitation and extensive annular calcification. Cardiac catheterization indicated minimal coronary disease with a left dominant circulation. Given her annular morphology, she was referred for open MVR. Induction of anesthesia was uncomplicated. Initial intraoperative transesophageal echocardiogram (TEE) redemonstrated preserved ejection fraction and no regional wall motion abnormalities. The patient was transitioned to cardiopulmonary bypass (CPB) without complication, and the mitral valve was successfully replaced with a bioprosthetic valve. However, while weaning off of cardiopulmonary bypass, the patient required atrioventricular pacing due to an underlying heart rate in the 30's. Her TEE demonstrated an akinetic inferior and lateral wall with a dilated right ventricle. This finding was conveyed to the surgical team due to the concern for LCX injury. The decision was made to provide inotropic support with dobutamine and norepinephrine and continue to the Cardiothoracic Intensive Care Unit (CTICU) for further resuscitation. Upon arrival to the CTICU, initial lab findings were notable for a mixed venous oxygen saturation of 63%, a lactate of 1.6 mmol/L, and a calculated cardiac index of 1.2. The patient was urgently taken to the catheterization lab and confirmed to have complete occlusion of the LCX. Although revascularization was achieved via stenting, the patient's lateral and inferior walls remained severely hypokinetic, and the patient required cannulation for Venoarterial Extracorporeal Membrane Oxygenation (VA ECMO). Her subsequent hospital course was complicated by recurrent gastrointestinal bleeds, dependency on mechanical ventilation and inotropic support, and she ultimately succumbed to mixed cardiogenic and septic shock three months postoperatively.

Discussion

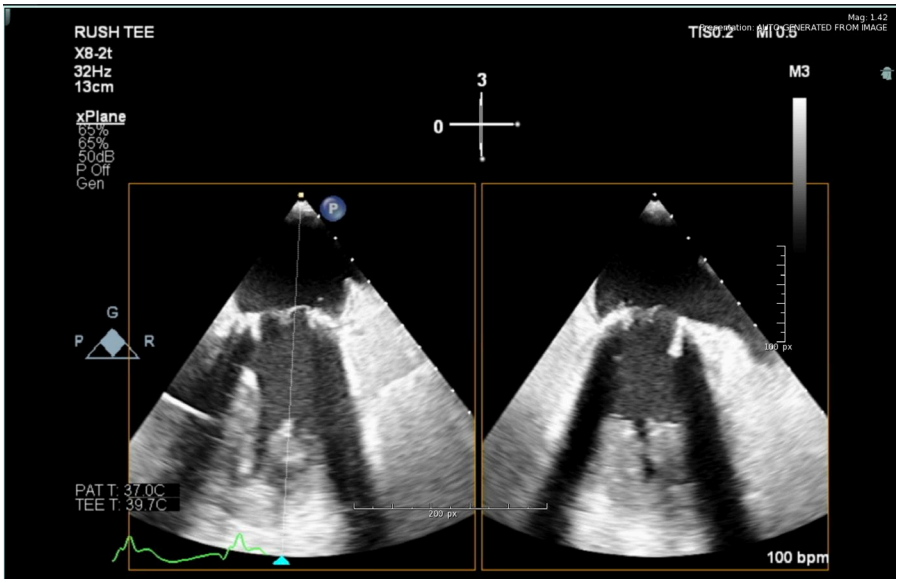
85% of the population has right dominant circulation where the right coronary artery supplies the posterior descending artery (PDA). In 8% of people with left dominance, the LCX supplies the PDA. In this population, the LCX provides blood flow to a much larger portion of the left ventricle including the lateral free wall, most of the inferior wall and the atrioventricular node (2). Complete LCX occlusion in a patient with left dominant circulation explains the intraoperative findings of inferior and lateral wall akinesis, and complete heart block. These characteristic findings should prompt further investigation by the operating room team to

diagnose LCX injury as delaying intervention can lead to irreversible myocardial injury. Methods of using TEE to measure coronary blood flow in patients with favorable anatomy have also been described and may aid in forming an intraoperative diagnosis (3).

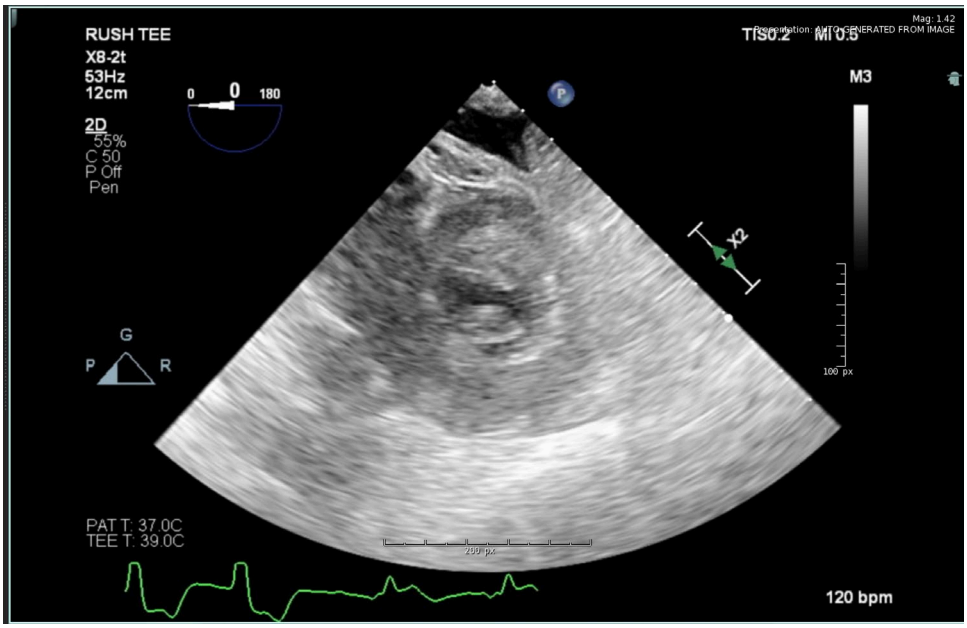
Images



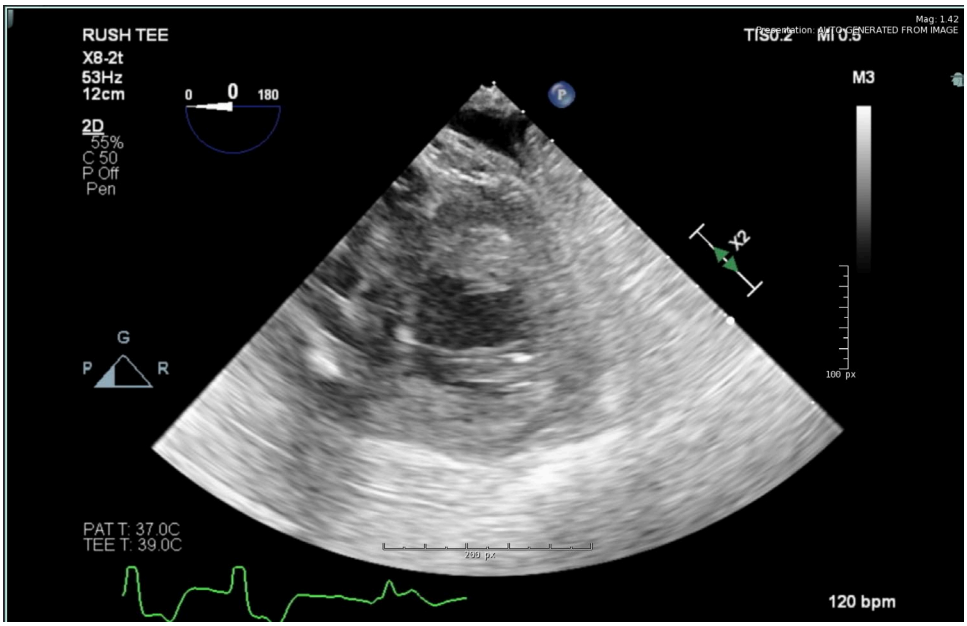
Transesophageal echocardiography on post-operative day 1 after recanalization of the Left Circumflex Artery demonstrating persistent inferior wall akinesis with xPlane of left ventricle during diastole. Shadowing notable from bioprosthetic mitral valve.



Transesophageal echocardiography on post-operative day 1 after recanalization of the Left Circumflex Artery demonstrating persistent inferior wall akinesis with xPlane of mid-esophageal 2 chamber view in systole.



Transesophageal Echocardiogram images of left ventricle in the transgastric short axis view demonstrating the left ventricle during systole



Transesophageal Echocardiogram images of left ventricle in the transgastric short axis view demonstrating the left ventricle during diastole



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#FR 3 – Kiosk 17 – Poster 1

The Use of Transesophageal Echocardiography for Intraoperative Diagnosis of Fungal Endocarditis During Suction Thrombectomy of a Right Atrial Mass

Presenting Author: Nathan B. Taber, MD, Resident Physician, Montefiore Medical Center: Department of Anesthesiology

Introduction

Transesophageal echocardiography (TEE) is a safe and effective tool not only for assisting proceduralists but also for intraoperative diagnostics. We present a patient with intraoperatively diagnosed fungal endocarditis using TEE while undergoing suction thrombectomy of a right atrial mass.

Case Presentation

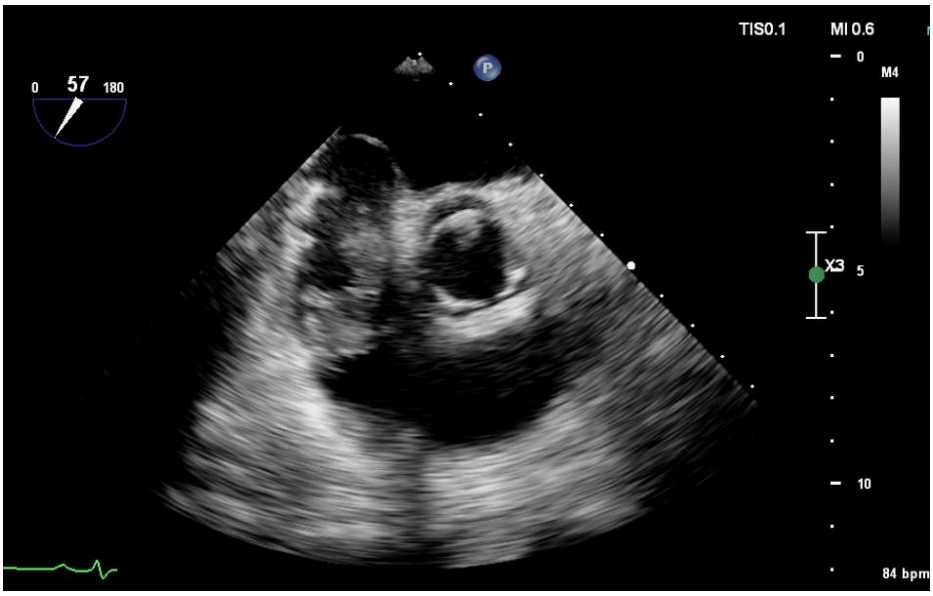
This is a 51-year-old female with morbid obesity, diabetes related end-stage renal disease on hemodialysis (via internal jugular catheter), heart failure with recovered ejection fraction, new bilateral upper extremity deep vein thrombi with superior vena cava (SVC) and right atrial (RA) thrombi, a left pulmonary artery calcified fibrin sheath, and candidemia who was scheduled for interventional radiology (IR)-guided suction thrombectomy with TEE assistance. The patient arrived to the IR suite intubated and sedated from the medical intensive care unit (MICU). General anesthesia was induced with sevoflurane and rocuronium, and a TEE probe was placed. Right common femoral and right internal jugular venous access was obtained by IR to facilitate the procedure. On TEE evaluation, the patient was noted to have a significant clot burden in the SVC and RA, which was debulked using suction thrombectomy. However, a considerable clot burden remained in the SVC after maximal thrombectomy. On interrogation of the patient's valves, she was noted to have tricuspid and mitral valve lesions, as well as thickening of the left coronary cusp of the aortic valve, which had not been seen on previous transthoracic echocardiography. These findings, coupled with Candida fungemia, revealed a diagnosis of endocarditis. The patient remained hemodynamically stable throughout the duration of the procedure. Post-procedure, the patient was transferred intubated and sedated to the MICU for further management and started on a prolonged antifungal course.

Discussion

TEE is a powerful tool for assisting proceduralists both in the operating room and non-operating room locations. In our case, TEE provided real-time evaluation guiding prodeculariss to the areas of heaviest clot burden and was integral to the diagnosis of endocarditis, a finding that was unknown to the primary medical team and altered clinical management post-procedurally. As shown here, TEE is not only used for procedural assistance but can also be a diagnostic tool of vital importance. When used in open cardiac surgery, TEE can alter the surgical plan by identifying pathology that was not previously known. In one study, the surgical plan was altered in 12.3% of CABG plus valvular surgery cases, highlighting its diagnostic utility. Additionally, its use in open cardiac surgery has been repeatedly associated with lower mortality, lower costs and greater quality adjusted life years per patient. Utilizing TEE for a thorough interrogation of all cardiac chambers and valves, we were able to impact both the intraoperative and postoperative course of

our patient. Anesthesiologists utilizing TEE must be diligent in assessing the entire heart, as this has the power to impact short-term decisions as well as long-term outcomes.

Images



Midesophageal Short Axis View showing significant Right Atrial Thrombus.



Clot Burden Removed from Right Atrium and Superior Vena Cava

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#FR 3 – Kiosk 17 – Poster 2

Pulmonary Arterial End-Diastolic Forward Flow in Carcinoid Heart Disease

Presenting Author: Andrew D. Stone, MD, Anesthesiology Fellow, Cleveland Clinic

Introduction

Pulmonary arterial end-diastolic forward flow (EDFF) occurs when the diastolic pressure in the right ventricle (RV) exceeds the diastolic pressure in the pulmonary artery (PA) during atrial contraction. EDFF has been associated with diastolic dysfunction of the right ventricle, especially after repair of tetralogy of Fallot (TOF). Carcinoid syndrome (CS) metastasized to the liver can cause carcinoid heart disease (CaHD) through serotonin (5-HT) production that induces endocardial fibrosis (1). This fibrosis primarily affects the tricuspid and pulmonic valves, resulting in tricuspid (TR) and pulmonic regurgitation and/or stenosis (PR/PS) due to thickened and restricted leaflets. EDFF may be found in the intraoperative transesophageal echocardiographic (TEE) exam of patients with CaHD as seen in the following case.

Case Presentation

A 62-year-old female with a history of CaHD, TR, and PR presented for tricuspid valve replacement (TVR) and pulmonic valve replacement (PVR). Preoperative transthoracic echocardiography (TTE) demonstrated a moderately dilated RV with normal function, severe TR, PS, and severe PR. A preoperative heart catheterization yielded elevated right-sided filling pressures, normal pulmonary artery pressures and normal left-sided filling pressure. In the operating room, invasive monitoring including an arterial line, central venous line (CVL) and pulmonary artery catheter (PAC) were placed prior to induction of general anesthesia while on octreotide, norepinephrine and vasopressin infusions. TEE showed torrential TR and severe PR due to restricted and thickened leaflets that looked “fixed in the open position”. The spectral Doppler envelope of the PA revealed EDFF during atrial contraction. The TV was replaced with a #31 Epic Plus, the PV with a #27 Epic Supra and the right ventricular outflow tract (RVOT) underwent patch reconstruction. Post bypass TEE did not demonstrate any significant abnormalities with the replaced valves. The patient was transferred to the ICU intubated on support with phenylephrine, vasopressin, and dobutamine.

Discussion

The echocardiographic finding of EDFF in the PA is infrequently described and etiology extrapolated mainly from the congenital literature on RV dysfunction in TOF patients (2). Historically, the cause of EDFF was thought to be due to restrictive RV physiology, where atrial contraction raised RV pressure enough to open the PV. Here, we propose that the EDFF is due to the combined effects of thickened and retracted PV leaflets that fail to close during diastole, increased right atrial pressure, decreased right ventricular compliance and normal PA pressures, allowing antegrade flow through the pulmonary valve during atrial contraction. Additionally, failure to recognize the EDFF using the electrocardiogram on spectral doppler envelope can lead to miscalculation of the PR index.

Images

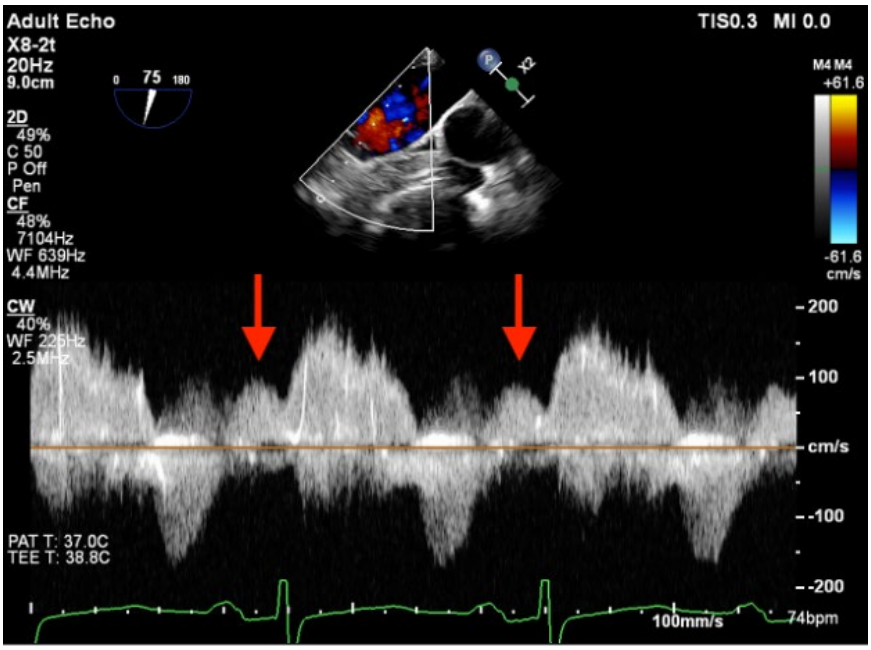


Image 1. Spectral doppler envelope demonstrating pulmonary arterial end-diastolic forward flow (EDFF), marked by red arrows.

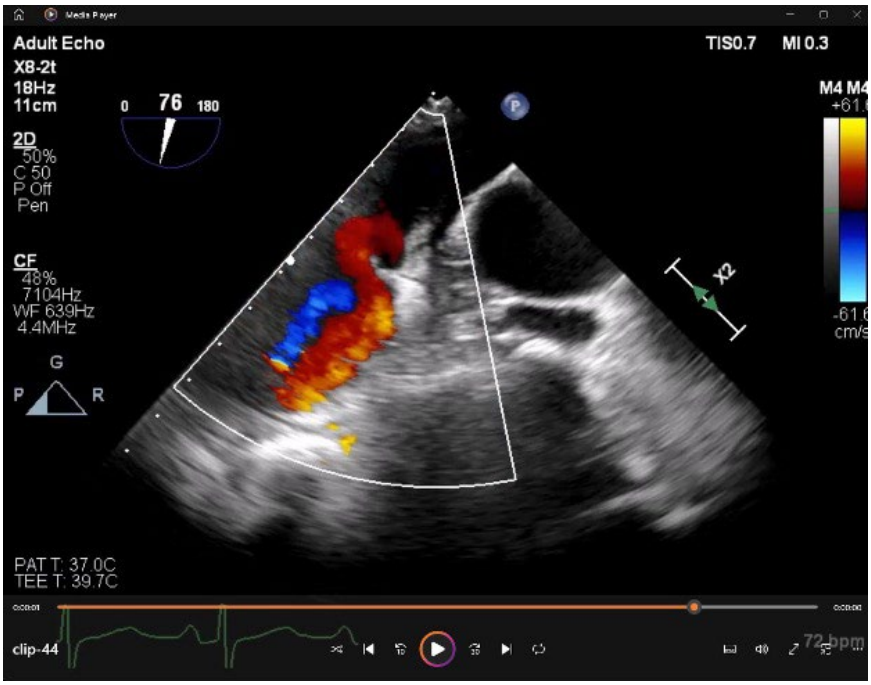


Image 2. Color flow doppler (CFD) demonstrating pulmonary arterial end-diastolic forward flow (EDFF) in the aortic arch short axis view.

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#FR 3 – Kiosk 17 – Poster 3

Development of Malignant Hyperthermia in a Patient Undergoing Cardiac Surgery

Presenting Author: Andrew M. Sexton, MD, Adult Cardiothoracic Anesthesiology Fellow, NewYork-Presbyterian/Columbia University Irving Medical Center

Introduction

Malignant hyperthermia (MH) is a rare, life-threatening complication from exposure to volatile anesthetics and succinylcholine in susceptible individuals. There are few case reports on development of MH during cardiothoracic surgery. Here, we present a case of a patient undergoing CABG and MVR that exhibited marked hypercarbia which resolved with administration of dantrolene.

Case Presentation

A 66-year-old man with a past medical history of HTN, HLD, moderate-to-severe MR, and 99% proximal RCA stenosis presented for MVR and single-vessel CABG. On pre-operative evaluation, the patient denied any personal or family history of adverse reactions to anesthetics. Shortly after the start of the procedure, he developed refractory hypercarbia with a markedly elevated pCO2 and resultant respiratory acidosis. Despite hyperventilating the patient to up to 18 liters per minute (L/min), his pH fell to 7.18 and pCO2 rose to 66. He remained normothermic and did not develop tachycardia, muscle rigidity, or lactic acidosis during this time. Malignant hyperthermia was suspected, and isoflurane was discontinued in favor of a propofol infusion. Upon transition to cardiopulmonary bypass, higher than expected sweep gas flow up to 6 L/min was required to decrease pCO2 levels. After consultation with our institution's MH representative, 2.5 mg/kg of dantrolene was administered for isolated hypercarbia. Shortly after dantrolene administration, pH began normalizing and sweep gas flow was able to be decreased incrementally. The patient maintained normocarbia with a minute ventilation of 9-10 L/min after cardiopulmonary bypass was weaned. After the procedure, the patient was transferred to the CTICU and dantrolene was given in 1 mg/kg doses every 6 hours for 24 hours. He was extubated on POD1, and lactate, potassium, and CPK were within normal limits throughout his ICU admission. He was referred for genetic testing and was found to have a missense mutation in the RYR1 gene, in which a proline in exon 28 was substituted with leucine. This was characterized as a variant of unknown significance (VUS).

Discussion

Malignant hyperthermia can be life-threatening if not properly identified and must be included in the differential of refractory hypercarbia. MH is associated with mutations in the RYR1, CACNA1S, and STAC3 genes, although up to 50% of patients with MH susceptibility do not carry pathologic variants of these genes.[1] Although this patient did not endorse anesthetic complications in the past, clinical symptoms of MH may develop in MH-susceptible individuals even after previous exposure of triggering agents without reaction.[2] Patients with VUS in the RYR1 gene have also been found to have positive caffeine-halothane contracture tests.[3] Endoscopic saphenous vein harvesting with CO2 insufflation may have also contributed to this patient's respiratory acidosis, although the degree of hypercarbia and its resolution after dantrolene

administration provides evidence toward a diagnosis of MH. Side effects of dantrolene such as muscle weakness and transaminitis are generally well-tolerated when the drug is administered as a short-term treatment, making it a reasonable therapy for profound, unexplained intraoperative hypercarbia and acidosis.

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#FR 3 – Kiosk 17 – Poster 4

Navigating Reentry: Anesthetic Management of Reoperative Sternotomy in a patient with Serial Aortic Valve Interventions

Presenting Author: Christopher P. Cariello, DO, Resident - CA-1, South Shore University Hospital / Northwell Health

Introduction

Introduction Reoperative cardiac surgery in patients with multiple prior aortic valve interventions and complex chronic wound pathology poses extreme anesthetic and perioperative challenges. Risks include catastrophic re-entry bleeding, thromboembolism, deep infection, impaired healing on immunosuppression, and irreconcilable conflicts between anticoagulation and surgical hemostasis. This case demonstrates multidisciplinary strategies for anesthetic planning, hemodynamic support, and wound management in high-risk redo sternotomy.

Case Presentation

Case Presentation Middle-aged male with prior surgical AVR, two TAVRs, Bentall procedure for type A dissection, recurrent embolic events, prosthetic valve thrombosis, chronic nonhealing sternal wound (pyoderma gangrenosum), and recurrent infections requiring pectoral flaps presented for redo surgery. Initial procedure was cancelled due to behavioral outbursts and illicit substances, necessitating psychiatric clearance.After optimization he underwent redo sternotomy, explant of degenerated prostheses, mechanical bileaflet AVR, CABG ×1, and IABP placement. Anesthetic plan included pre-induction large-bore access, cell salvage, immediate blood products, invasive monitoring, and TEE for valve seating, ventricular function, and vasoactive/IABP guidance. Fragile skin and prior flaps mandated protective positioning and dermatology-guided prep. Hemodynamics supported with vasopressors + IABP; anticoagulation carefully managed on CPB and bridged to warfarin.Postoperatively he developed skin-edge ischemia and pyoderma flare. After initial discharge he was readmitted for wound necrosis requiring debridement, omental flap, and skin grafting. Supratherapeutic INR required reversal and brief delay of wound care to balance bleeding vs thrombosis risk. Daily input from cardiac surgery, anesthesiology, ID, dermatology/plastics, hematology, and psychiatry led to stabilization and discharge with close monitoring.

Discussion

This case highlights unique considerations in redo aortic surgery with wound/anticoagulation complexity:

1. re-entry risk mitigated by pre-induction preparation (crossmatched blood, perfusionist readiness, cell salvage, TEE);
2. balancing mechanical valve thrombosis prevention with wound healing via protocolized INR targets and rapid reversal pathways;
3. skin/soft-tissue protection with early dermatology/plastics involvement in pyoderma gangrenosum and prior flaps;
4. tailored hemodynamic support using IABP and vasoactives;

5. integration of behavioral health factors affecting timing and adherence via psychiatric/social work engagement. Intensive multidisciplinary coordination and vigilant surveillance enabled successful management of wound complications while preserving valve function and stability. For similar patients, teams should predefine anticoagulation strategies, re-entry contingencies, leverage intraoperative TEE, and involve dermatology/plastics early when wound integrity is compromised.

References

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#FR 3 – Kiosk 17 – Poster 5

Persistent systolic anterior motion (SAM) of the mitral valve despite fluids, bradycardia, afterload, and even extracorporeal membrane oxygenation

Presenting Author: Spencer Nehls, DO, University of Wisconsin

Introduction

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant condition involving mutations in sarcomere proteins that impair myocardial contractility. Affecting roughly 1 in 500 adults, it is associated with complications such as left ventricular outflow obstruction (LVOT), diastolic dysfunction, mitral valve disease, arrhythmias, and sudden death.[1] Systolic anterior motion (SAM) of the mitral valve is a feared complication of hypertrophic cardiomyopathy and is defined as displacement of the distal portion of the anterior leaflet of the mitral valve toward the LVOT. SAM has been described in as many as 95% of HCM patients, with approximately 60-70 % of those with SAM exhibiting resting or provoked LVOT obstruction.[2,3] Using a perioperative case, we aim to provide recommendations for early diagnosis, echocardiogram findings, and clinical management for SAM.

Case Presentation

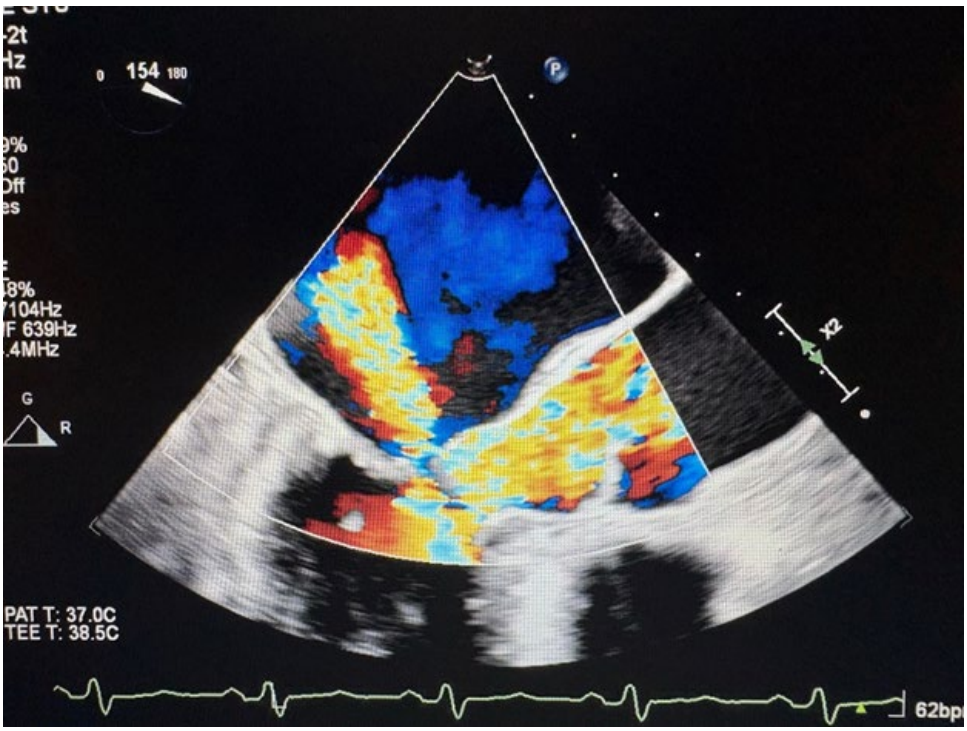
A 53-year-old female with a history of HCM, prior takotsubo cardiomyopathy, pancreatic cancer, diabetes, hypertension, and morbid obesity was scheduled for repeat right sided VATs and right lower lobe (RLL) wedge resection for an enlarging nodule. She was hospitalized in 2009 for takotsubo cardiomyopathy and TTE demonstrated apical ballooning of the left ventricle, EF of 25-35%, and normal coronaries on catheterization. TTE in August 2024 revealed an EF of 60%, moderate asymmetric LVH, and severe LVOT obstruction with valsalva. On 10/21/24, she underwent an uncomplicated VATs procedure and was extubated in the OR. One minute following extubation, she became bradycardic and hypotensive requiring external pacing. Hemodynamically stability was regained and pacing stopped. During transfer, she again became profoundly hypotensive, pulses were lost, and ACLS was initiated. Suspecting SAM with LVOT obstruction, the team gave phenylephrine, vasopressin, and fluid boluses without success. She was re-intubated and significant pulmonary edema was noted. TEE probe was inserted demonstrating significant LVOT obstruction secondary to SAM and significant mitral regurgitation (MR). Despite limiting sedation and escalating vasopressors, forward flow could not be established which prompted VA-ECMO cannulation. On ECMO, patient developed north-south syndrome due to intermittent LVOT obstruction and edematous lungs. VAV-ECMO was attempted without success. Nitric oxide was started and the patient was transferred to the ICU.

Discussion

This case demonstrates that despite rapid recognition and aggressive treatment of SAM-related LVOT obstruction, establishing forward flow is challenging. In patients with know HCM, refractory hypotension should raise concern for LVOT obstruction and empiric treatment should be initiated while TEE is inserted. A mid-esophageal long axis view typically demonstrates anterior mitral valve leaflet motion into the LVOT with

turbulence and posteriorly directed MR. Goals for management of SAM include maintaining preload, reducing heart rate and inotropy, preserving sinus rhythm, and sustaining systemic vascular resistance. Anesthesia providers caring for patients with HCM must anticipate SAM, recognize it, and have plans to optimize hemodynamic stability.

Images



Classic “Y” sign demonstrating severe posteriorly directed MR and turbulence in the LVOT.

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#FR 3 – Kiosk 17 – Poster 6

Complex Cardiac Surgery in a Patient with End Stage Renal Disease

Presenting Author: Megan E. Weis, MD, Resident, University of Florida Jacksonville

Introduction

The cardiovascular patient with significant coexisting disease presents a unique challenge when faced with the need to undergo cardiac surgery requiring Cardiopulmonary Bypass (CPB). We present a challenging case that involves reoperation for a patient with multiple valvular disorders as well as chronic hemodialysis (HD) and other medical comorbidities.

Case Presentation

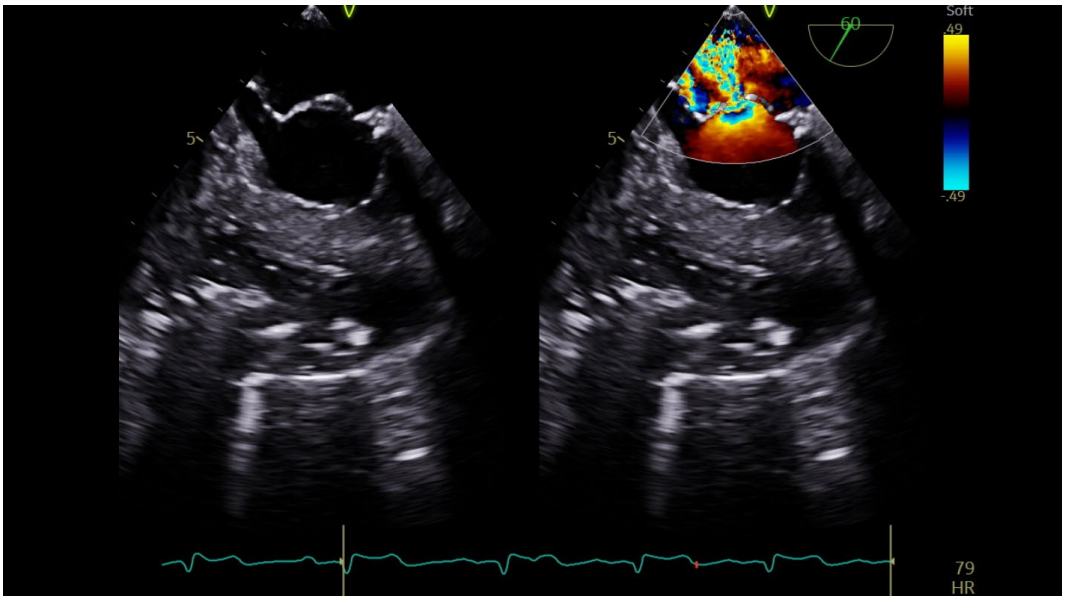
A 58-year-old male with NYHA III heart failure from mitral regurgitation (MR) secondary to recurrent endocarditis presented for surgical treatment. His history included mechanical aortic valve (AV) replacement 10 years prior, MR with prior attempted transcatheter correction, atrial septal defect (ASD), AV node dysfunction treated by Micra pacemaker, ESRD on HD via left internal jugular (IJ) permacatheter, multiple nonfunctional dialysis sites on bilateral upper extremities, prior renal transplant in rejection and on steroid therapy, and central venous stenosis. Pre-op echo showed LVEF 50-55%, enlarged right ventricle (RV), trace aortic regurgitation (AR), moderate mitral annular calcification, moderate-severe MR, and dilated 4.9 cm aortic root. There was concern that his mechanical AV was affected by prior endocarditis and plan for surgery was redo sternotomy, MVR, redo AVR, ASD repair, and ascending aorta repair. Prior to surgery, patient received erythropoietin, pRBC transfusion and HD 24 hours in advance. After induction of general anesthesia, radial arterial access was unsuccessful due to prior dialysis fistulas. Central line placement was attempted via the right IJ unsuccessfully with inability to pass a wire. Femoral venous and arterial access were achieved on non-transplanted kidney side. TEE prior to CPB showed LVEF 45%, moderate RV dysfunction, small ASD with continuous left to right flow, well seated mechanical AV with normal function and trace regurgitation, moderately calcified MV with thickened anterior and posterior leaflets, multiple MR jets (central and eccentric). The A2 leaflet portion showed a defect consistent with perforation. Surgical assessment of the AV did not reveal signs of dehiscence or effects of endocarditis. They replaced the ascending aorta and MV and closed the ASD. TEE post CPB showed bileaflet mechanical MV with small paravalvular leak, moderate to severe RV and LV dysfunction with LVEF ranging from 15 to 30%. Vasopressin, norepinephrine, epinephrine, and dobutamine were required to separate from CPB and support patient to the ICU. Postoperatively, notable edema of patient's head and neck due to SVC syndrome was managed by elevation of head of bed and dexamethasone for intracerebral edema. Continuous renal replacement therapy was initiated. Patient was extubated on post-op day 3 but had a tumultuous course that included sepsis, stress dosed steroids, atrial fibrillation, and emergent return to OR for delayed intrathoracic bleed and tamponade. On post-op day 20 he was discharged from the hospital.

Discussion

This case highlights multiple surgical and medical complexities, the most significant of which being ESRD

and its sequelae, where the odds of mortality for cardiac surgery are significantly higher when compared to general population (1). Increased surgical risk was also due to redo sternotomy, aortic surgery, and MV replacement adjacent to mechanical AV.

Images



Intraoperative transesophageal echo (TEE) image with mitral valve with perforation in anterior leaflet.

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#FR 3 – Kiosk 17 – Poster 7

Hemolysis and Acute Kidney Injury Following Off-Label Use of an Amplatzer Septal Occluder for Mitral Paravalvular Leak Closure: A case report

Presenting Author: Langely J. Chaparro- Rios, MD, Anesthesiology Resident, Boston Medical Center-Brighton

Introduction

Paravalvular leaks (PVLs) occur in up to 5–15% of mitral valve replacements and can lead to hemolytic anemia, heart failure, and end-organ injury. As the number of valve replacements rises, management of PVLs has become increasingly relevant. The Amplatzer Septal Occluder, originally designed for closure of atrial septal defects, has been used off-label for transcatheter repair of PVLs. While this minimally invasive approach can obviate the need for repeat sternotomy, altered flow dynamics across prosthetic valves may predispose to shear-mediated hemolysis and device-related complications. We present a case of severe hemolysis and AKI following off-label Amplatzer device deployment for mitral PVL closure, ultimately requiring surgical correction.

Case Presentation

A 54-year-old male with prior bioprosthetic aortic and mitral valve replacements presented with new-onset heart failure and syncope. Echocardiography revealed severe mitral regurgitation due to a significant paravalvular leak of the mitral bioprosthesis. A percutaneous PVL closure was performed using an Amplatzer Septal Occluder device, reducing regurgitation from severe to mild-to-moderate. Within several days, the patient's serum creatinine increased from baseline to 8–9 mg/dL, with dark urine and falling hemoglobin. Laboratory results demonstrated elevated LDH, indirect bilirubin, and hemoglobinuria, consistent with intravascular hemolysis. The residual high-velocity jet across the Amplatzer device likely caused mechanical destruction of erythrocytes from shear stress. The patient developed progressive volume overload and oliguria, requiring continuous venovenous hemodialysis (CVVH). He was scheduled for surgical repair; however, at induction of anesthesia, he developed profound hypotension and pulseless electrical activity, necessitating CPR and multiple vasopressors. An intra-aortic balloon pump was inserted, and the procedure was aborted. After hemodynamic stabilization, he returned to the operating room several days later. The Amplatzer device was removed, and the mitral PVL was repaired using Teflon felt. Post-repair TEE demonstrated no residual regurgitation, and both bioprosthetic valves functioned normally. The patient remained dialysis-dependent.

Discussion

This case demonstrates a rare but serious complication of off-label device used for PVL closure. The Amplatzer Septal Occluder, optimized for low-pressure atrial flow, may create high-shear turbulent jets when deployed across prosthetic mitral valves. The resulting mechanical hemolysis can precipitate AKI and hemodynamic instability. From an anesthetic perspective, patients undergoing re-operation for PVL repair may present with severe anemia, renal dysfunction, and fragile hemodynamics, requiring careful preload

management and avoidance of nephrotoxins. Early recognition of hemolysis after percutaneous interventions and multidisciplinary coordination among anesthesiology, cardiology, and cardiac surgery teams are essential.

Images

Figure 1: Amplatzer device

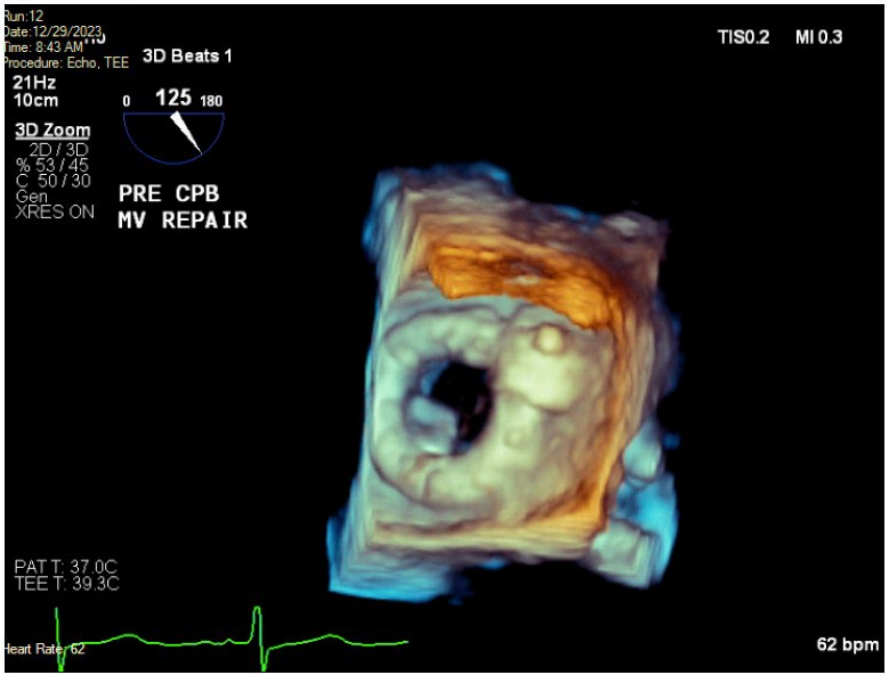


Fig 2. Mitral Valve Regurgitation

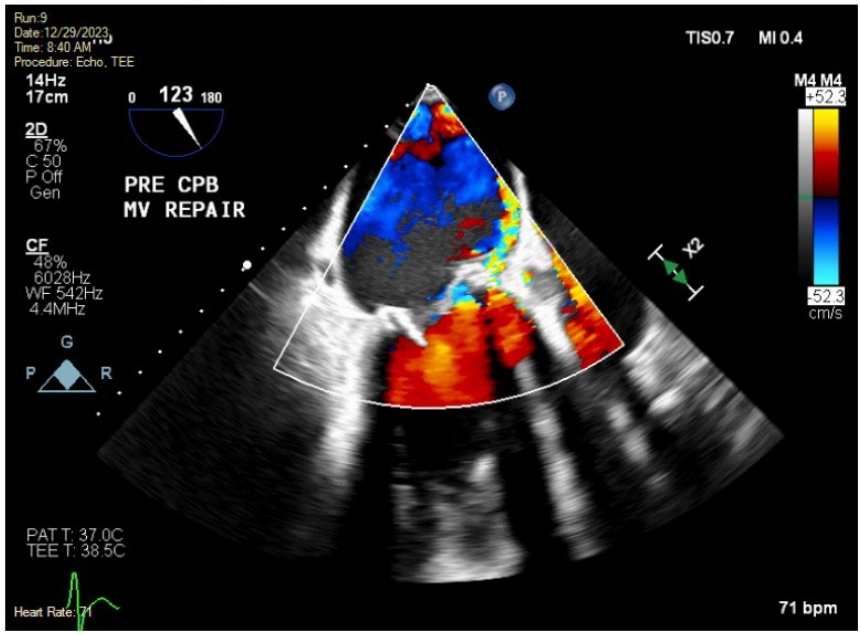
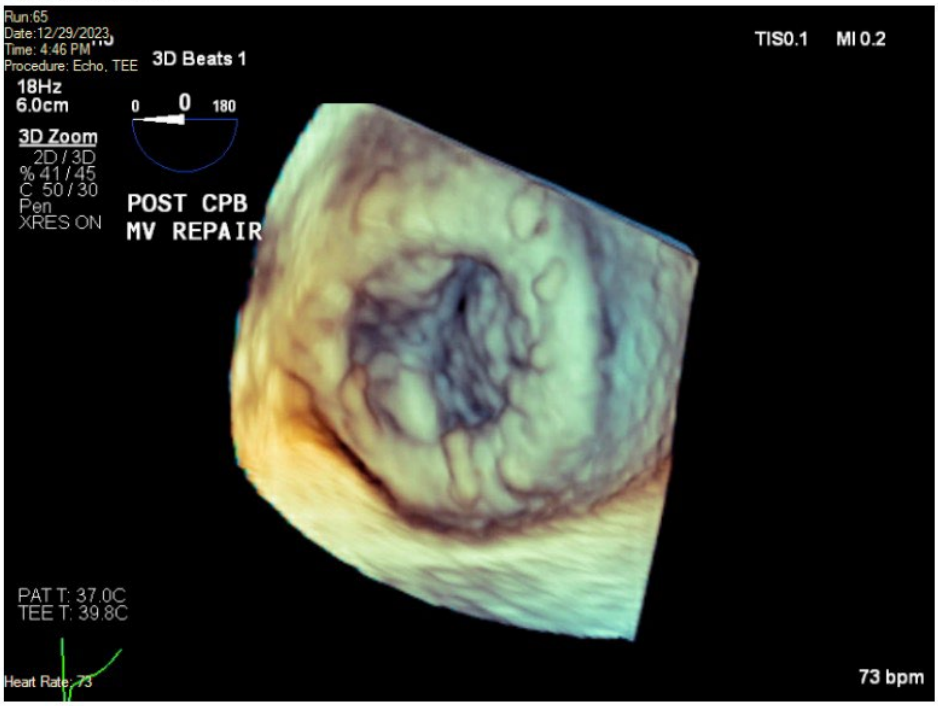


Fig.3: Post-repair



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#FR 3 – Kiosk 18 – Poster 1

BATMAN Technique to Prevent LVOT Obstruction During Valve in Ring TMVR

Presenting Author: Craig Mitchell, DO, Resident Anesthesiologist, SSM Health St. Louis University Hospital

Introduction

Transcatheter mitral valve replacement (TMVR) offers a less-invasive alternative for high-risk patients with mitral valve degeneration. However, TMVR carries many significant risks such as a risk of valvular leaks, irreparable regurgitation, strokes, and left ventricular outflow tract (LVOT) obstruction. LVOT occurs particularly in patients with long anterior leaflets, small predicted neo-LVOT area, or unfavorable aorto-mitral angulation. Preventive strategies including alcohol septal ablation and LAMPOON have been performed previously, but balloon-assisted translocation of the anterior mitral leaflet (“BATMAN”) technique holds another technique to selectively to mitigate this complication of neo-LVOT obstruction

Case Presentation

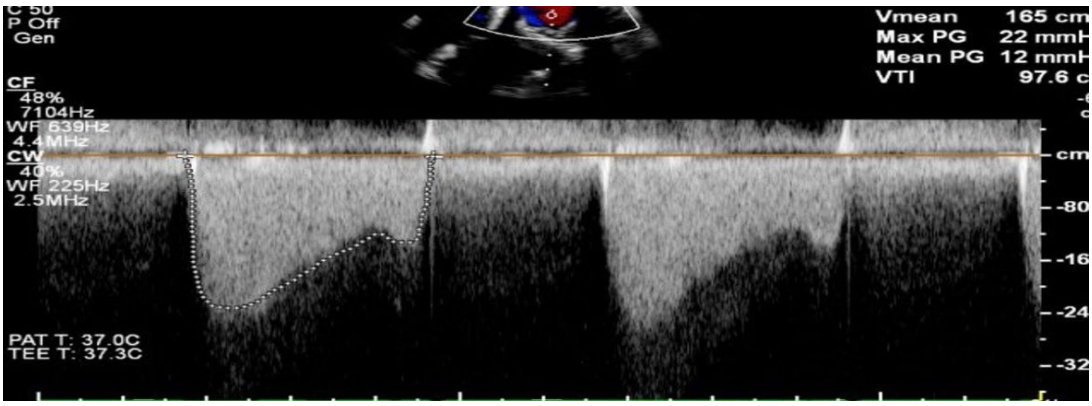
A 52-year-old man with prior bioprosthetic mitral valve replacements presented with severe dyspnea on

exertion and marked functional limitation. TEE demonstrated severe prosthetic mitral stenosis (mean gradient 12 mmHg) and moderate aortic stenosis. His many comorbidities included severe COPD, atrial fibrillation, CKD, and a remote history of metastatic non–small cell lung cancer. Given the prohibitive risk of redo sternotomy, percutaneous TMVR was pursued. Pre-procedural imaging revealed a critically small predicted neo-LVOT, placing the patient at high risk for obstruction. A transseptal BATMAN procedure was selected to increase the size of the Neo LVOT. Under TEE guidance provided by anesthesiology, the proceduralist punctured the anterior most leaflet of the bioprosthetic valve. The leaflet was then balloon dilated and then the new valve was deployed over the same guidewire thus obliterating the most anterior leaflet resulting and increasing the size of the NEO LVOT. The TMVR was completed successfully without hemodynamic compromise. Post-operative day 1 echocardiography showed stable prosthetic function and no evidence of LVOT obstruction or paravalvular leak.

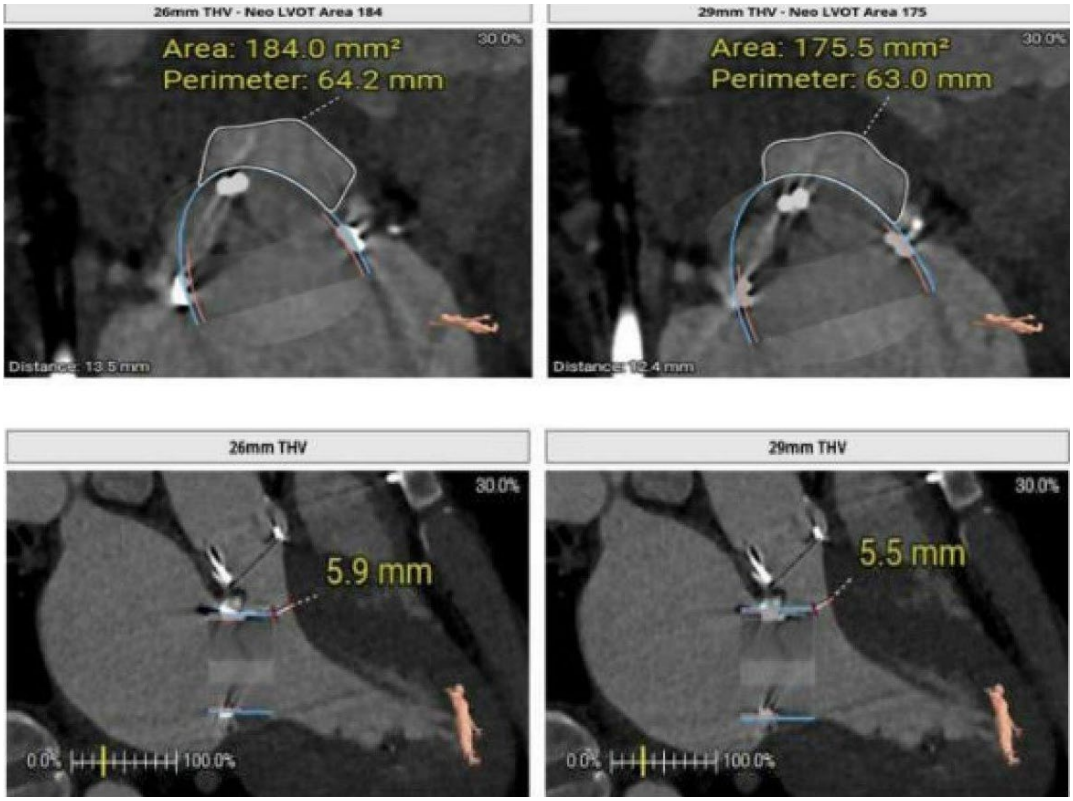
Discussion

This case highlights the role of the BATMAN technique in managing patients at extreme risk for LVOT obstruction during TMVR and an Anesthesiologist's role in these procedures. Compared with alternative strategies, transseptal BATMAN allows controlled anterior leaflet displacement while avoiding myocardial injury or complex transapical approaches. Appropriate patient selection and imaging-based prediction of neo-LVOT area remain essential.

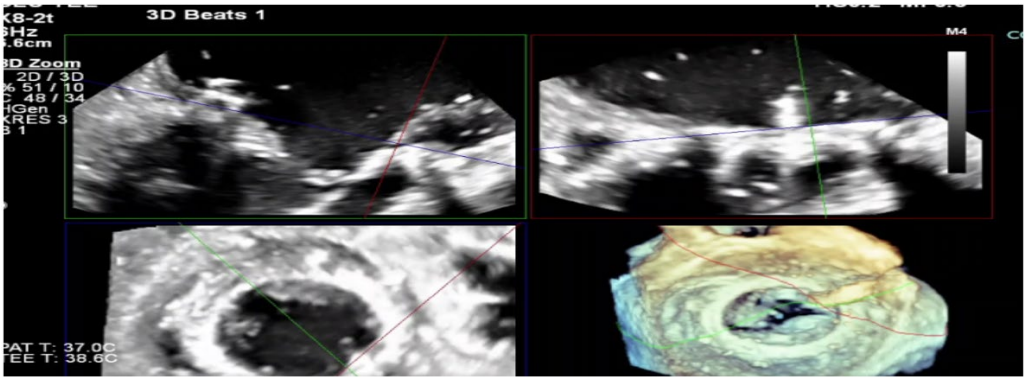
Images



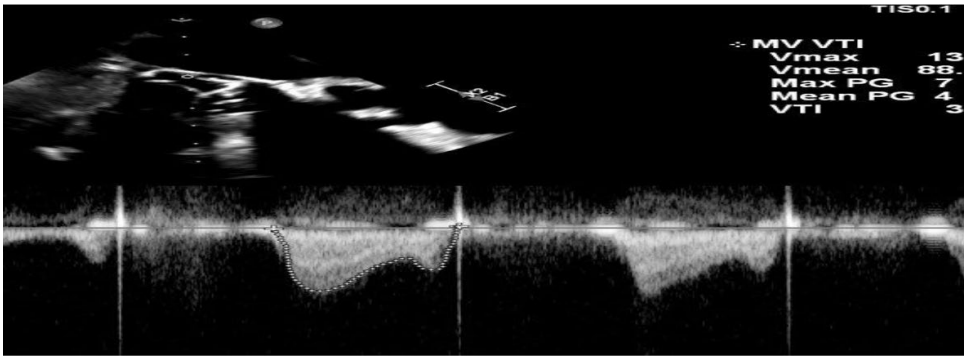
Continuous wave doppler across mitral valve with mean gradient 12 indicating severe MS



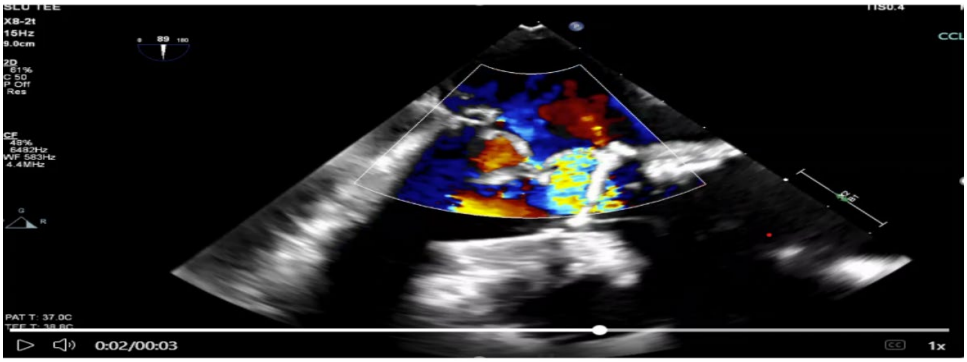
CT image of predicted Neo-LVOT with placement of the valve without BATMAN procedure.



Successful puncture of anterior leaflet of mitral bioprosthetic leaflet via TEE guidance



Post valve deployment with new Mean PG of 4.



Color doppler demonstrating showing reduced flow acceleration across new bioprosthetic valve

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#FR 3 – Kiosk 18 – Poster 2
Combined Open Heart Mitral Valve Repair and Left Pneumonectomy: Perioperative Management and Considerations



Presenting Author: Lucas M. Luong, DO, Adult Cardiothoracic Anesthesia Fellow, The University of Connecticut Health Center

Introduction

Combined cardiac surgery and major pulmonary resection is rare and associated with significant perioperative risk. Only 0.3–0.5% of patients with operable pulmonary malignancy present with concurrent cardiac disease requiring surgery. Both staged and single-session approaches are described and each carries unique physiologic challenges. We describe a rare case of combined left pneumonectomy and mitral valve repair (MVR) performed through a median sternotomy. Particular attention was paid to management considerations related to post-pneumonectomy syndrome (PPS); characterized by airway obstruction and circulatory collapse from cardiac herniation and mediastinal shift into the vacant hemithorax.

Case Presentation

A 60-year-old man with 20-pack-year smoking history presented with dyspnea, chest pain, and hemoptysis. Imaging revealed a left upper lobe mass, positive for stage IIIA lung adenocarcinoma, with mediastinal extension. After neoadjuvant chemotherapy, he was deemed a candidate for left pneumonectomy. Pre-operative evaluation revealed normal biventricular function but severe mitral regurgitation, prompting a combined surgical approach. The operation was performed via median sternotomy using a single-lumen endotracheal tube. Significant hemodynamic instability occurred during cardiac manipulation for lung exposure, requiring fluid resuscitation, high-dose vasopressors, and blood transfusions. After the pneumonectomy, avoidance of positive-pressure ventilation was considered due to possible stress on the new left bronchial stump; however, the surgical team deemed it unnecessary. Cardiopulmonary bypass (CPB) was initiated for MVR. Post-CPB separation, vasopressor and transfusion support was required. To prevent mediastinal shift and cardiac herniation into the pneumonectomy cavity, a bovine pericardial patch and breast tissue expander were placed for support and to occupy the left hemithorax. On postoperative day (POD) 0, he required re-exploration due to significant hypotension from possible bleeding given increased chest-tube output. Minor bleeding was identified and controlled; however, hemodynamics improved dramatically with pericardial patch revision relieving right ventricular outflow tract compression. He was extubated on POD 1, had an unremarkable hospital course, and discharged home on POD 6 without further complications.

Discussion

Combined cardiac and thoracic surgery is rare due to concerns of heparinization-related bleeding, limited pulmonary exposure, and CPB-associated inflammatory effects. Hemodynamic management is complicated by the tension between preload augmentation required during cardiac manipulation and excessive volume after pneumonectomy. PPS represents a significant postoperative concern due to mediastinal shift, rotation, and compression of central airways or cardiac structures. Left-sided pneumonectomy in particular carries a high risk given the heart's proximity to the vacant hemithorax. Placement of a pericardial patch and thoracic expander provides structural support to prevent PPS, but

careful implementation is required to avoid excessive cardiac compression. This case highlights the feasibility of a one-stage median sternotomy approach and the importance of anticipatory strategies to mitigate PPS in combined cardiac–thoracic procedures.

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#FR 3 – Kiosk 18 – Poster 3

The Brokenbrough-Braunwald-Marrow Phenomenon to Rule Out Left Ventricular Outflow Tract Obstruction in the Setting of Coronary Artery Disease

Presenting Author: Jeffrey Kim, MD, Cardiac Anesthesia Fellow, Albany Medical Center

Introduction

Determination of the presence of a LVOT obstruction often employs pharmacologic methods to increase heart rate and contractility. In the setting of a patient with significant coronary artery disease, this method is not ideal.

We present an intraoperative case in which we used the Brockenbrough-Braunwald-Morrow Phenomenon (BBM) to determine the presence of a LVOT obstruction in a patient with significant CAD.

Case Presentation

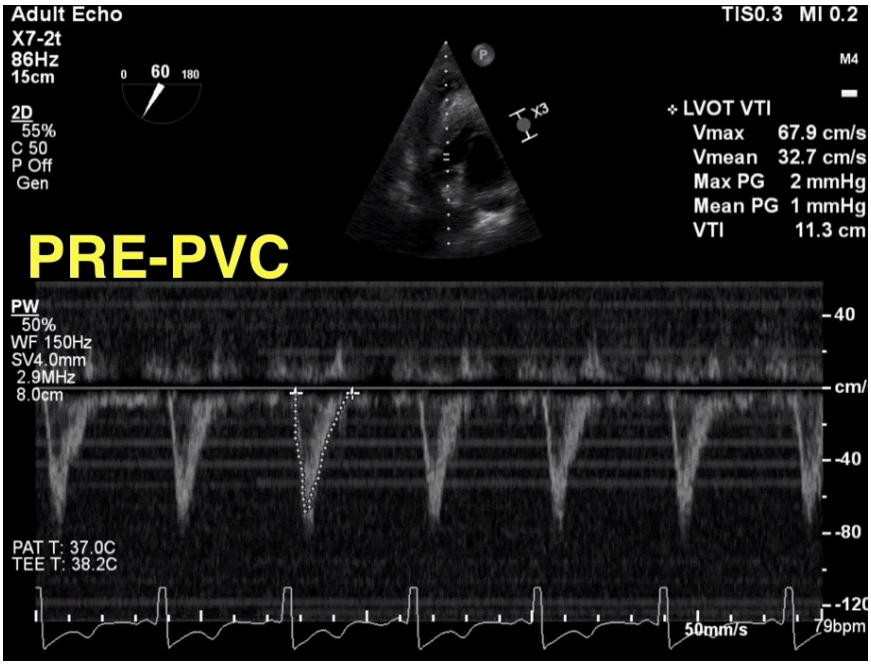
A 70-year-old F with a PMH of CAD, HTN, HLD, and DM2 presents with two weeks of exertional dyspnea and lower extremity edema. Cardiac catheterization showed a 99% mid-LAD stenosis. TEE demonstrated a normal EF, abnormal LV relaxation, concentric LVH, and hypertrophic CM suspicious for HOCM. The patient was scheduled for CABG and on the day of the operation, the surgeon requested guidance regarding the need to perform an additional septal myocardial resection. After inducing GA, invasive line and TEE probe placement, sternotomy and pericardial dissection, using our understanding of the BBM phenomenon, we had the surgeon induce a ventricular ectopic beat manually while analyzing any changes in the LVOT spectral Doppler profiles on TEE. Before the PVC, the LVOT VTI was 11.3cm, max velocity was 67.9 cm/s, and max gradient was 2mmHg. Immediately after the post ectopic beat, the LVOT VTI was 15.7cm, max velocity was 116cm/s and max gradient was 5mmHg. Because the post ectopic max gradient did not increase significantly remaining far below 30mmHg, we concluded that there was no BBM sign and thus no significant LVOT obstruction. The surgeon proceeded with the CABG without septal myocardial resection.

Discussion

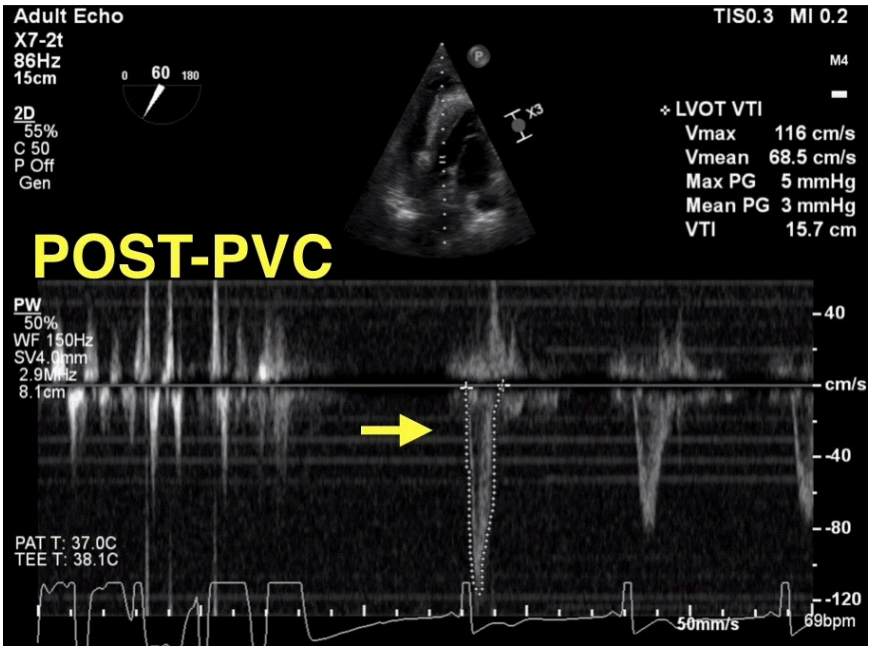
The BBM phenomenon was first described in 1961 as a paradoxical drop in aortic pulse pressure after a PVC indicating the presence of a LVOT obstruction.[1] Normally, after a PVC, there is a compensatory pause which results in increased LV diastolic filling. Following Frank Starling mechanisms, this would normally increase contractility, stroke volume and pulse-pressure. In HOCM however, increased preload and contractility exacerbates a LVOT obstruction causing a paradoxically decreased SV and pulse-pressure. During open heart surgery, the BBB phenomenon can be used to detect the presence of a LVOT obstruction. A PVC can be induced by manually tapping on the heart. The post PVC beat can then be analyzed using doppler echocardiography looking for increased max pressure gradients or other signs of LVOT obstruction. Alternatively, one could qualitatively analyze the LVOT after a post PVC beat and look SAM or increased turbulent flow. [2] This method avoids subjecting a heart compromised by CAD to a demanding pharmacologic stress test. The patient turned out not to have a significantly increased LVOT gradient and thus we concluded that there was no LVOT obstruction. We believe this method has widespread

applicability especially intra-operatively in the setting of CAD. Follow up research should be done to verify this method and to further define spectral doppler criteria that establish the diagnosis of HOCM.

Images



Pre-PVC Spectral Doppler Profile of LVOT



Post-PVC Spectral Doppler Profile of LVOT

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#FR 3 – Kiosk 18 – Poster 4

Complex Case of Durable Bi-Ventricular Assist Device Placement for Destination Therapy

Presenting Author: Cameron G. Berg, MD, Fellow, Cardiothoracic Anesthesiology, Mayo Clinic

Introduction

Durable biventricular mechanical circulatory support (BiVAD) is performed far less frequently than isolated left ventricular assist device implantation, particularly in patients considered high-risk due to psychosocial and psychiatric comorbidities. Compared with isolated left ventricular support, survival rates in these patients are significantly lower, and intraoperative placement presents unique technical challenges[1]. We describe a complex case involving both preoperative evaluation and intraoperative implantation of a durable BiVAD in a patient deemed ineligible for transplantation and managed with a durable BiVAD as destination therapy

Case Presentation

A 45-year-old male with ischemic and non-ischemic bi-ventricular cardiomyopathy (EF 15–20%), polysubstance abuse in remission, severe, untreated psychogenic polydipsia, and prior PCI complicated by in-stent thrombosis presented to our hospital's emergency department with acute decompensated heart failure and subsequent PEA arrest. ROSC was achieved, and an Impella 5.5 was placed for left ventricular support. Despite extubation, he remained fully dependent on MCS and developed acute on chronic bi-ventricular failure, receiving a temporary right ventricular assist device (Protek Duo) 28 days after presentation. Following RVAD migration and failure, he required VA-ECMO and was subsequently listed for durable BiVAD therapy using bilateral HeartMate 3 devices. In the operating room, standard induction and primary sternotomy were performed, and cardiopulmonary bypass was initiated without difficulty. Prior MCS devices were removed. The left HeartMate 3 was implanted with inflow at the LV apex and outflow to the ascending aorta. The right HeartMate 3 was placed with inflow from the IVC–right atrial junction and outflow to the main pulmonary artery. While initially with non-obstructive flows, the patient became very volume dependent with fluctuating right sided flows. With the cannula at the RA junction, the drainage cannula appeared prone to moderate changes in volume or positioning. After separation from by-pass the patient required multiple manipulations of his drainage cannula and significant resuscitation and pressor management before becoming hemodynamically stable. The patient was initially taken to the ICU with an open chest and required emergent return to the OR on POD#0 for obstructed right-sided flow caused by surgical packing. Chest closure occurred on postoperative day 4, with extubation the following day. The patient steadily improved, was transferred from the ICU on POD #15. On POD# 31 he was discharged to a skilled nursing facility and has had no major complications to date.

Discussion

This case highlights the novelty and technical complexity of bilateral HeartMate 3 devices in a patient previously considered a poor candidate for durable MCS. Careful multidisciplinary management, perioperative planning, and staged intervention allowed successful long-term destination therapy,

demonstrating that high-risk patients can achieve meaningful functional recovery with BiVAD support.

Images

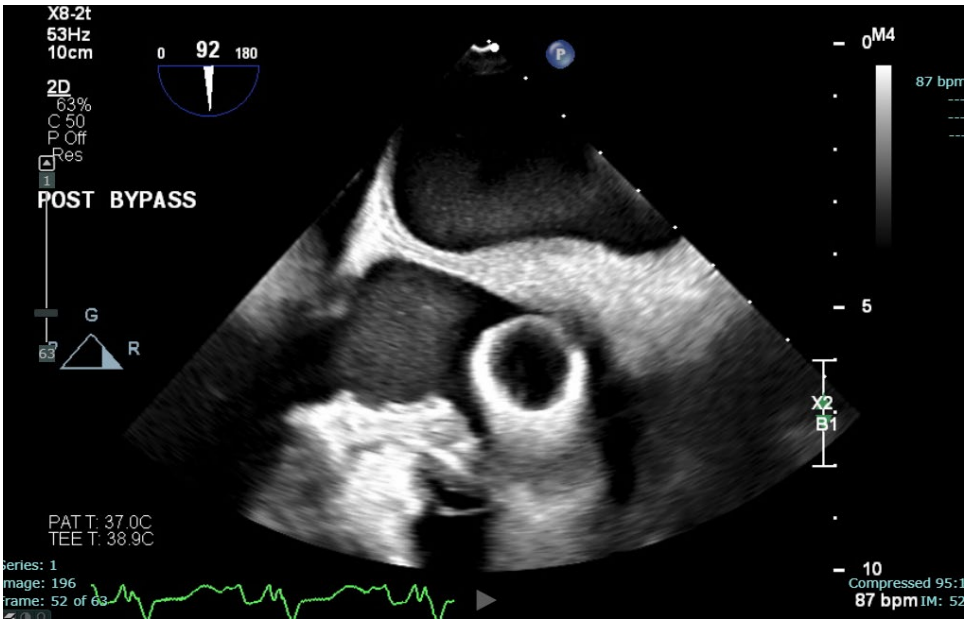


Figure 1. Right atrial drainage cannula with the cannula traveling posterior to the right atrium

References

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#FR 3 – Kiosk 18 – Poster 5

Anesthetic Management of Tracheoesophageal Fistula Repair Under Full Veno-Venous Extracorporeal Membrane Oxygenation Support

Presenting Author: Bradley Carlson, MD, Resident Physician, Northwestern Memorial Hospital

Introduction

Tracheobronchial injuries resulting from traumatic intubation are rare but associated with high morbidity. In elective cases, tracheobronchial injury occurs in approximately one per 20,000 to 75,000 intubations.[1] However, this rate increases in emergent intubations. In larger tracheal injuries, ventilation may become compromised and respiratory failure may occur. Veno-venous extracorporeal membrane oxygenation (VV ECMO) can provide life-saving oxygenation and ventilation in such cases.[2] Even so, its use as the sole intraoperative respiratory support during airway reconstruction is uncommon. We present a case of a patient with a tracheoesophageal fistula (TEF) secondary to traumatic intubation who underwent open surgical repair on full VV ECMO support without the ability to provide conventional ventilation.

Case Presentation

A 40-year-old female with end-stage renal disease presented to an outside hospital with septic shock and respiratory failure secondary to pneumonia and was intubated. The intubation was complicated by tracheal tear and resultant TEF which was recognized after accumulation of subcutaneous emphysema led to an endoscopic examination of the esophagus and airway (Images 1-2). She was transferred to Northwestern Memorial Hospital where VV ECMO was initiated. With an endotracheal tube (ETT) in place, the cuff was deflated, and the patient was placed on a T-piece for provision of supplemental oxygen without positive pressure. The patient was ultimately taken to the operating room to attempt surgical repair. Intra-operatively, general anesthesia was continued using propofol and fentanyl infusions. The 7.0 ETT was removed and easily replaced with an 8.0 ETT under video laryngoscope guidance. There was continued inability to ventilate the patient with tidal volumes only reaching a maximum of 17 mL with 20 cm H2O of driving pressure and without any detected end tidal CO2. Therefore, 100% FiO2 was administered through the ETT without positive pressure. Additionally, full VV ECMO support (flows 4.5-5 L/min, FdO2 100%, sweep 6 L/min) was continued throughout the case. Due to significant bleeding and coagulopathy, 12 units of packed red blood cells, 8 units of fresh frozen plasma, 8 units of platelets, and 5 pooled units of cryoprecipitate were administered. Intra-operative time was 12 hours and 22 minutes. After surgical repair, ventilation was attempted again via the ETT with minimal success, only briefly achieving a maximum end tidal CO2 reading of 8 mmHg.

Discussion

This case highlights the unique challenges of managing a TEF repair in a patient entirely dependent on VV ECMO for gas exchange. Conventional ventilation was impossible due to massive air leak through the TEF, profound atelectasis from prolonged periods without ventilation, and aspiration pneumonia (image 3). Pre-operatively, the patient was maintained on a T-piece rather than mechanical ventilation to minimize airway

pressures and avoid exacerbating the injury. This strategy of avoiding aggressive ventilatory settings was continued post-operatively out of concern for disrupting the newly constructed trachea. This case illustrates the use of this ventilatory strategy made possible by VV ECMO.

Images

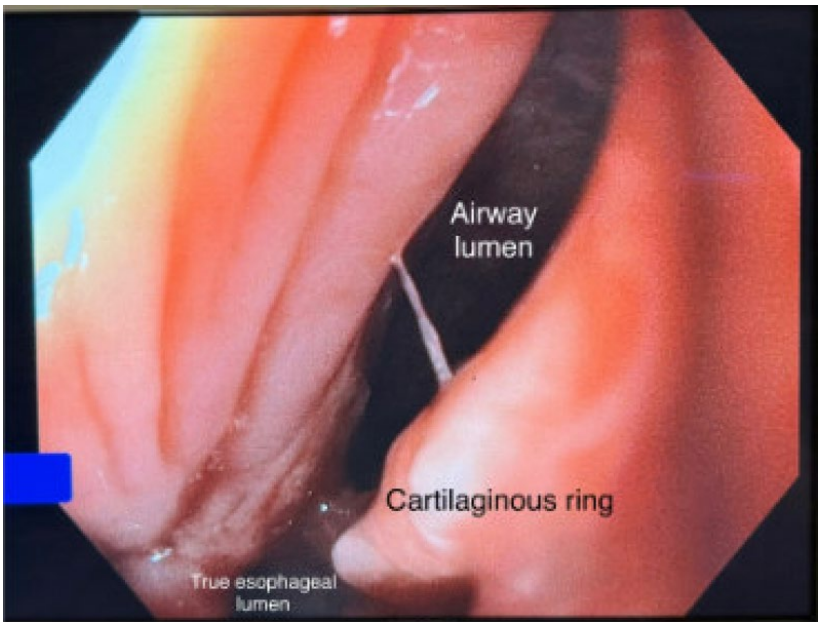
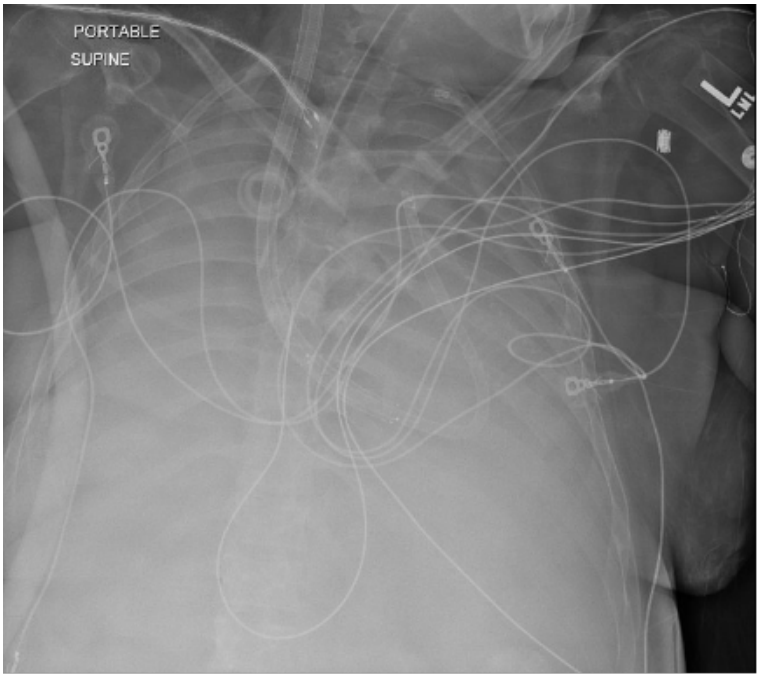


Image from EGD demonstrating full length tracheal defect.



View of carina using bronchoscope showing presence of posterior tracheal defect.



Chest X-ray demonstrating complete opacification of bilateral lung fields.

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#FR 3 – Kiosk 18 – Poster 6

Navigating Innovation and Risk: A Case of Pericardial Tamponade Following Leadless Pacemaker Placement

Presenting Author: AliReza A. Zaravar, DO, CA-1 Resident, HCA Oak Hill Florida Anesthesiology

Introduction

The integration of leadless pacemakers has revolutionized pacing by reducing complications associated with transvenous leads. Despite its minimally invasive profile, rare but severe complications including cardiac perforation and tamponade can occur, which pose critical challenges in perioperative care. Recognizing these events early and initiating prompt interventions are vital for positive patient outcomes, particularly in the post-operative setting where rapid deterioration can occur.

Case Presentation

An 81-year-old male underwent Medtronic Micra AV leadless pacemaker implantation for symptomatic bradycardia. In the post anesthesia care unit, he developed sudden severe hypotension and became unresponsive. Advanced cardiac life support was initiated and after one round of chest compressions, an IV fluid bolus, and a dose of IV ephedrine, ROSC was achieved. Point of care echocardiography was subsequently performed and revealed an acute 1 cm pericardial effusion with concern for tamponade physiology. Emergent pericardiocentesis was attempted but unsuccessful. A stat CT Surgery consult was placed, and the patient was taken to the OR for emergent subxiphoid pericardial window. After 300 mL of dark blood was evacuated from the pericardial space, there was immediate improvement in the patient's hemodynamics. A chest tube was placed, and the patient was taken to the CT ICU in stable condition. The patient was discharged four days later following an uneventful recovery in the ICU.

Discussion

Before the introduction of leadless pacemakers, about 1 in 8 patients with transvenous pacing systems experienced early, pocket- or lead-related complications.[1] Leadless pacemakers were designed to mitigate some of the complications and to provide additional benefits over traditional pacemakers.[2] This case highlights a rare but life-threatening complication of leadless pacemaker placement, emphasizing the importance of early recognition, prompt intervention, and the critical role of bedside echocardiography in the PACU setting to manage postoperative cardiac emergencies effectively. While the prevalence of pericardial effusion requiring intervention after a leadless pacemaker implantation is roughly 1.1%, patients who developed significant pericardial effusion experienced higher mortality rates, longer lengths of stay, and greater hospitalization costs.[3] Khan at al. also found that advanced age, female sex, chronic lung disease, chronic kidney disease, and connective tissue disorders were associated with a higher likelihood of a serious pericardial effusion.[3] Even though the prevalence is low, the clinical severity requires a high index of suspicion for rapid diagnosis. Utilization of point of care echocardiography proved to be an invaluable tool in the prompt recognition of tamponade physiology. The failure of bedside pericardiocentesis highlights the importance of surgical readiness for emergent surgical interventions and the necessity of a multidisciplinary

approach for management of such emergencies. Significant pericardial effusion remains the most serious and feared complication associated with leadless pacemaker implantation.[3]

Images

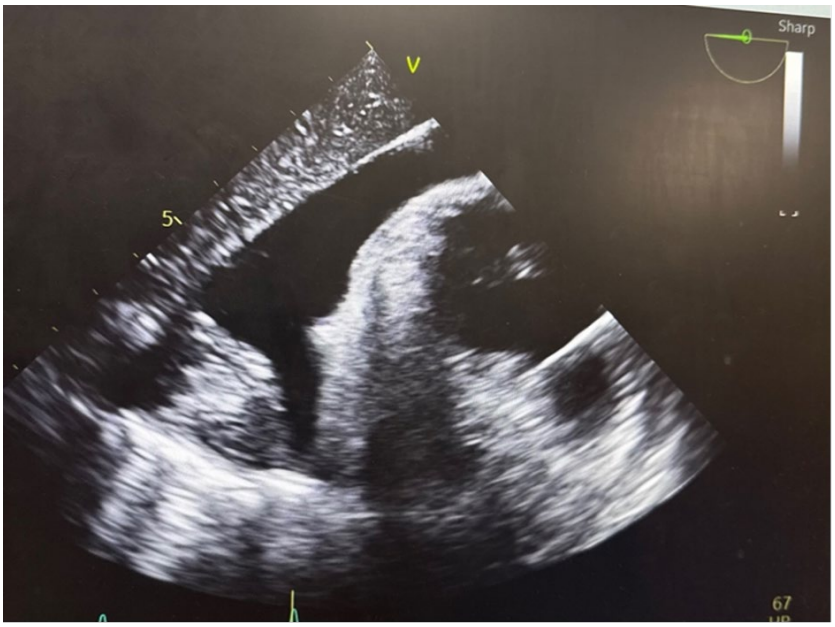


Figure 1: Intraoperative transesophageal echocardiogram showing an approximately 1cm pericardial effusion with compression of the left ventricle.

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#FR 3 – Kiosk 18 – Poster 7

Intrapartum Management of a Parturient with Severe, Symptomatic Mitral Stenosis

Presenting Author: Maia C. Young, MD, Resident Physician, University of Florida College of Medicine Jacksonville

Introduction

Rheumatic fever (RF) is underdiagnosed and undertreated in developing countries. When RF progresses to rheumatic heart disease (RHD), mitral valvulopathy occurs in 50-60% of cases.1 Mitral stenosis (MS), the most common sequelae of RHD, can cause elevated left atrial pressures, pulmonary edema, and heart failure, which may compromise maternal and fetal health.2,3

Case Presentation

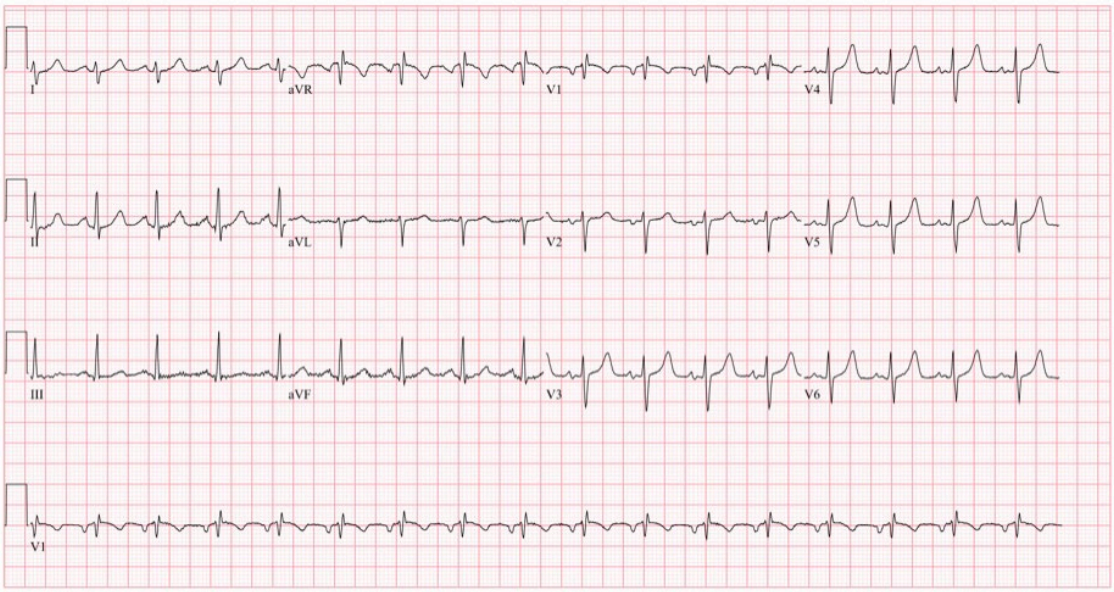
A 29-year-old G3P1011 patient from Honduras presented to labor & delivery at 36w3d GA with contractions and 1 week of worsening shortness of breath. Vital signs were within normal limits; physical exam revealed a diastolic heart murmur and lower extremity edema. Lab work was significant for a pro-BNP of 1086 pg/mL and troponin of 10 ng/L. EKG showed sinus tachycardia and left atrial enlargement (Figure 1). Chest x-ray revealed cardiomegaly and bilateral patchy opacities (Figure 2). Respiratory viral panel was negative. A transthoracic echocardiogram revealed an EF of 50-55%, thickened mitral valve leaflets with “hockey-stick” appearance of the anterior leaflet during diastole, fusion of the mitral commissures, and calcification of the sub valvular apparatus suggestive of rheumatic mitral valve pathology. The mean transmitral gradient was 13 mmHg and the estimated mitral valve area was 1.1 cm^2. Severe MS was diagnosed, metoprolol and furosemide administered, and the decision was made to deliver via cesarian urgently. Standard monitors plus an arterial line were used, and an L3-L4 epidural was placed. After negative aspiration, 3mL of 1.5% lidocaine with 1:200,000 epinephrine was given. The patient lost consciousness, and the HR rose to 148. She was laid supine, and pulsatility and respirations were confirmed. The catheter was re-aspirated and drew continuous heme. After return of consciousness and hemodynamic stability, the patient was sat upright and the epidural replaced. Aspiration and test dose were both negative. After supine positioning with left lateral displacement, 2% lidocaine with 1:200,000 epinephrine was given epidurally in 4mL increments spaced 5 minutes apart and with vigilant continuous blood pressure monitoring. Phenylephrine infusion was titrated to a blood pressure within 20% of baseline. Once a T4 surgical anesthetic level was achieved, the obstetric team delivered a viable female infant in a standard cesarean fashion. Uterotonics were administered including oxytocin, tranexamic acid, and Carbaprost. EBL was 500mL. The patient tolerated the procedure

well and was transferred to the cardiac intensive care unit. The postoperative course was unremarkable. After 48 hours of ICU monitoring, she was discharged on metoprolol and furosemide with cardiology follow up.

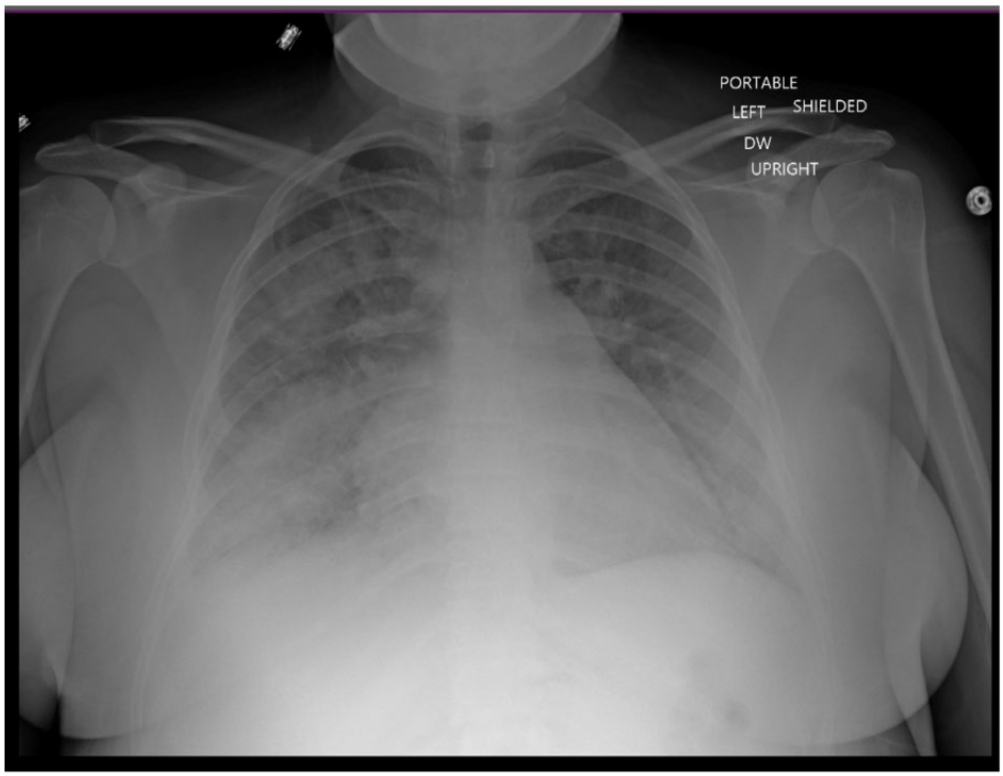
Discussion

Severe MS confers a high risk in pregnancy to the extent that pregnancy is not recommended. The physiologic changes in pregnancy can lead to decompensated heart failure, fetal growth restriction, and preterm labor. In parturients with severe, symptomatic MS with evidence of pulmonary edema, careful management of preload and systemic vascular resistance to preserve cardiac output and uteroplacental perfusion is critical. A slowly titrated epidural anesthetic with continuous blood pressure monitoring was chosen to avoid sudden, potentially deleterious hemodynamic changes.

Images



EKG on admission



Chest x-ray on admission

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#FR 3 – Kiosk 19 – Poster 1

Mechanical Cardiac Tamponade with Dynamic LVOT Obstruction Following CABG and Mitral Valve Replacement: Anesthetic Management of a Postoperative Hemodynamic Emergency

Presenting Author: Langely J. Chaparro- Rios, MD, Anesthesiology Resident, Boston Medical Center-Brighton

Introduction

Coronary artery bypass grafting (CABG) and mitral valve replacement (MVR) are commonly performed cardiac surgeries, but postoperative hemodynamic collapse may occur from a variety of mechanisms. Cardiac tamponade remains a critical concern, whereas dynamic left ventricular outflow tract (LVOT) obstruction, though rare, can mimic or exacerbate shock. Differentiating these pathologies is essential, as their management strategies are fundamentally opposite. We present a case of mechanical cardiac tamponade with concomitant dynamic LVOT obstruction after CABG and MVR, highlighting the role of transesophageal echocardiography (TEE) in diagnosis and mechanism-directed hemodynamic management.

Case Presentation

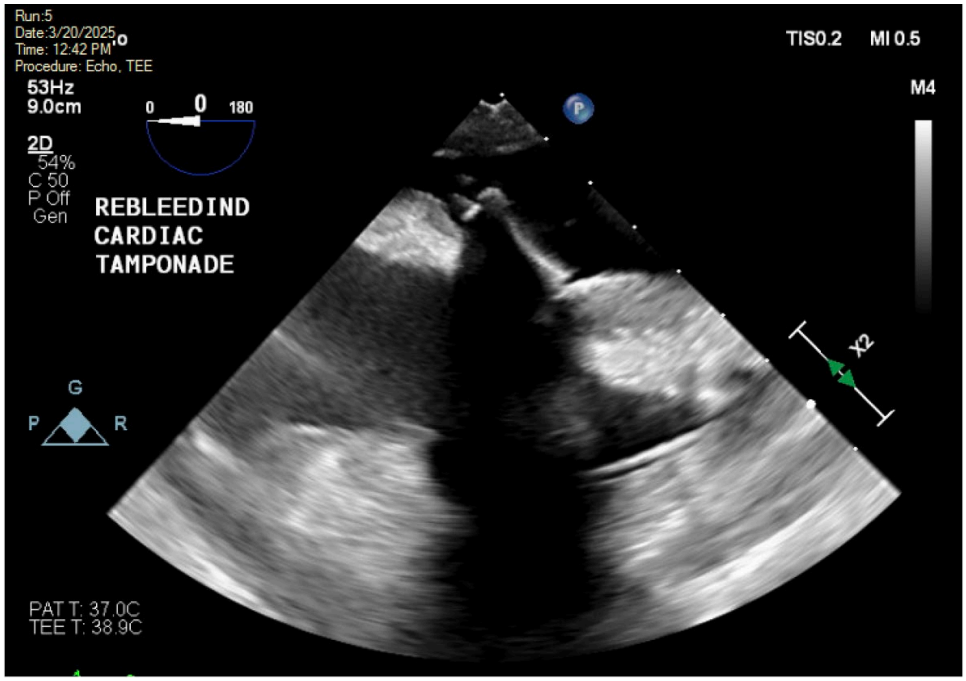
A 69-year-old man with multivessel coronary artery disease, severe mitral regurgitation, atrial fibrillation, and chronic kidney disease underwent CABG ×3 (LIMA→LAD, SVG→sequential OM/Diagonal) and MVR with a bioprosthetic valve. Intraoperative TEE revealed an ejection fraction (EF) of 65% after revascularization. Due to intraoperative coagulopathy, multiple transfusions were required, and the patient was transferred intubated to the ICU on norepinephrine. On postoperative day 1, his mixed venous oxygen saturation decreased from 47% to 37%, with rising vasopressor needs and equalization of pulmonary artery diastolic and central venous pressures (18 mm Hg). Chest radiograph showed mediastinal widening, and transthoracic echocardiography revealed a large mediastinal hematoma compressing the right ventricle with an underfilled, hyperdynamic left ventricle, consistent with mechanical tamponade. Emergency re-exploration confirmed the hematoma. During evacuation, the patient developed pulseless electrical activity but regained spontaneous circulation after 3 minutes of CPR and 1 mg epinephrine. Intraoperative TEE demonstrated severe dynamic LVOT obstruction (peak gradient 45 mm Hg, EF ≈ 75%) with systolic anterior motion of the mitral prosthesis. Norepinephrine and milrinone were discontinued, and phenylephrine (0.5 µg/kg/min) was initiated to increase afterload and LV cavity size. Hemodynamics stabilized (MAP 70 mm Hg, $EtCO_2$ 35 mm Hg), and the patient was extubated on postoperative day 3.

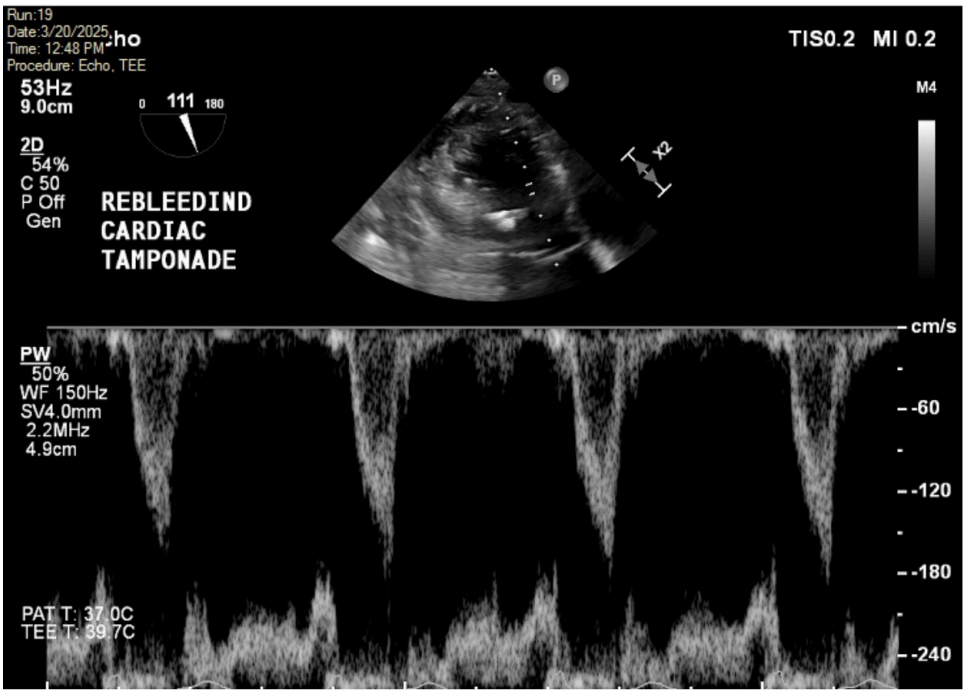
Discussion

This case demonstrates the coexistence of mechanical cardiac tamponade and dynamic LVOT obstruction, two conditions that both impair LV filling but require fundamentally opposite treatments. The mediastinal hematoma caused mechanical compression of the ventricles, whereas inotropic support and reduced preload triggered dynamic systolic narrowing of the LVOT. Recognizing this distinction was crucial: mechanical obstruction required surgical evacuation, while dynamic obstruction required physiologic

correction, cessation of inotropes, restoration of preload, and afterload augmentation with phenylephrine, a pure α-agonist. These interventions reversed the gradient and stabilized hemodynamics. This case underscores the pivotal role of TEE-guided diagnosis and mechanism-specific management in postoperative cardiac shock.

Images





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#FR 3 – Kiosk 19 – Poster 2

Planned VA ECMO-Supported Reverse Potts Shunt Deferred: Management of Right Ventricle-to-Pulmonary Artery Conduit Stenosis and Retrocardiac Pericardial Effusion

Presenting Author: Thomas Blankespoor, M.D., Anesthesia Resident, Mayo Clinic, Rochester

Introduction

Reverse Potts Shunt (RPS) creation is an emerging palliative strategy for patients with suprasystemic pulmonary hypertension and right ventricular failure. It decompresses the right heart via a conduit between the pulmonary artery and descending aorta. Anesthetic management is challenging, as induction of general anesthesia and positive-pressure ventilation can reduce preload, elevate pulmonary vascular resistance, and precipitate acute RV decompensation. We present a planned awake, VA ECMO-supported RPS, ultimately managed solely with RV-PA conduit stenting after intra-procedural reassessment revealed sub-systemic PA pressures complicated by a retrocardiac pericardial effusion.

Case Presentation

A 39-year-old male with repaired Tetralogy of Fallot and prior RV-PA conduit presented with severe conduit obstruction and pulmonary hypertension. Pre-procedural echocardiography revealed an RV-PA gradient of 62 mmHg, reduced RV systolic function with septal bowing and D-shaped LV, and RVSP 130 mmHg, concerning for suprasystemic PA pressures. He was planned for right heart catheterization, transcatheter pulmonary valve implantation, and percutaneous RPS under awake VA ECMO support, given risk of acute RV decompensation. A superficial cervical plexus block minimized pain, sympathetic surges, and intrathoracic pressure elevations during right IJ cannulation and procedural manipulation. Catheterization revealed RVSP 138 mmHg but sub-systemic PA pressures of 54 mmHg; the Potts shunt and ECMO were deferred. During conduit dilation and valve placement, the patient developed acute chest pain; TTE demonstrated a retrocardiac pericardial effusion causing hemodynamic compromise. The stenosed RV-PA conduit was stented to relieve obstruction, heparin was reversed with intravenous protamine, and remimazolam infusion facilitated echocardiography-guided pericardiocentesis, removing 175 cc of hemorrhagic fluid. Incremental fluid boluses and inotropic support maintained RV preload and systemic perfusion. Hemodynamics stabilized, oxygenation remained adequate, and the patient was transferred to the CVICU in stable condition.

Discussion

This case highlights anesthetic challenges in managing right-ventricular failure, pulmonary hypertension, and tamponade physiology. Reverse Potts shunts create a right-to-left connection between the left pulmonary artery and descending thoracic aorta, producing Eisenmenger physiology in suprasystemic PA pressures. The RPS shunts desaturated blood to the lower body while preserving oxygenated flow to the cerebral and coronary circulation, improves RV systolic function[1], and has been shown to improve 5-year survival[2]. In sub-systemic PA pressures, RPS can cause pulmonary over-circulation, RV volume overload, and worsening oxygenation. An awake approach requires careful patient selection but can avoid systemic

vasodilation from anesthetic agents and elevated intrathoracic pressures from positive-pressure ventilation. A superficial cervical plexus block minimizes sedation, blunts sympathetic responses during IJ cannulation, and reduces intrathoracic pressure from straining or coughing. The development of a pericardial effusion required prompt recognition; this case illustrates the importance of maintaining RV preload and systemic perfusion during acute tamponade.

Images

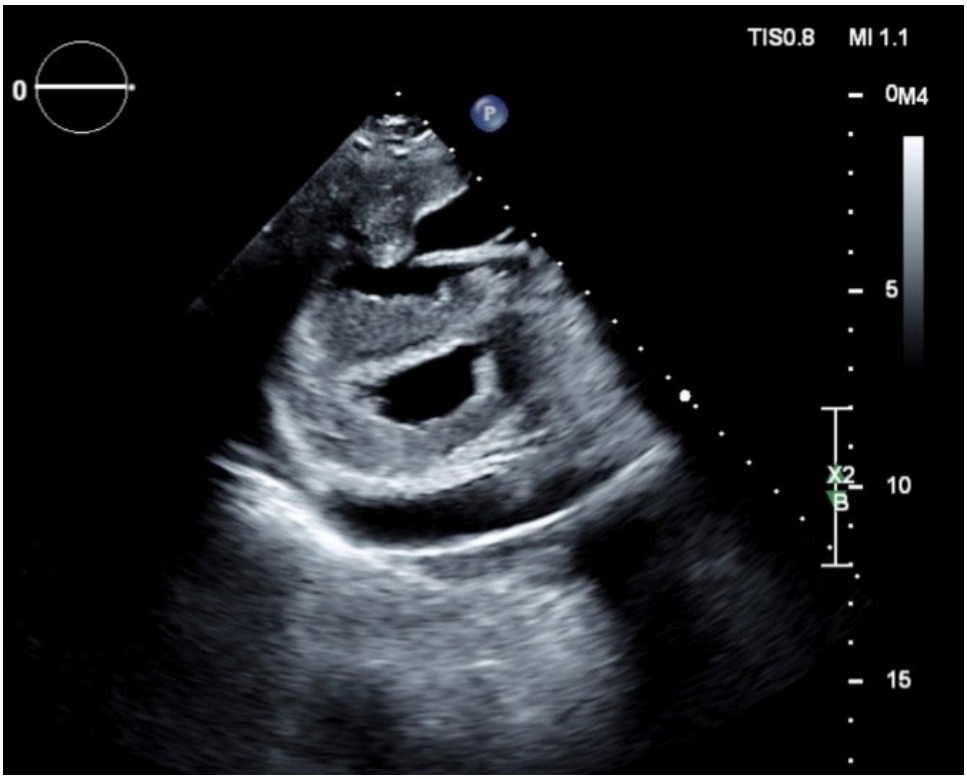


Figure 1: Transthoracic echocardiography demonstrating severe RV pressure elevation with D-shaped LV, septal bowing, and pericardial effusion impacting hemodynamics.

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#FR 3 – Kiosk 19 – Poster 3

A Case of Fourth Redo Sternotomy for Management of Left Ventricular Pseudoaneurysm in a Patient with Osteogenesis Imperfecta

Presenting Author: Brandy Sweeney, DO, MS, Cardiothoracic Anesthesiology Fellow, Cleveland Clinic Foundation

Introduction

Osteogenesis Imperfecta (OI) is a rare genetic disorder of Type I collagen caused by mutations in COL1A1 or COL1A2. Its clinical presentation is broad with fragile connective tissues and bone fragility being the hallmark features. These collagen mutations increase perioperative risk in cardiac surgery, including abnormal platelet aggregation, small vessel bleeding, tissue dehiscence, and even frank ventricular wall rupture. We describe a challenging case of an OI patient with severe left ventricular dysfunction and previous mechanical aortic and mitral valve replacements who developed a postoperative left ventricular pseudoaneurysm as a complication of their most recent surgery at the referring hospital, ultimately requiring, and successfully undergoing a high-risk fourth redo sternotomy for pseudoaneurysm repair at our center.

Case Presentation

A 42-year-old man with OI and a history of bicuspid aortic valve, previously treated with three sternotomies for endocarditis and multi-valve dysfunction, developed a LV pseudoaneurysm with contained rupture shortly after undergoing cardiac surgery at an outside hospital. He presented to our facility after several other institutions declined to offer high-risk fourth time sternotomy and pseudoaneurysm repair due to reduced heart function and limited rescue options. Preoperatively, the heart failure service had evaluated the patient and determined that heart transplantation would be the only viable rescue option if unable to separate from bypass. Intraoperatively, repair of the 7 cm x 4 cm LV pseudoaneurysm involved anticipated challenges, including massive balanced transfusion, severe coagulopathy, and increased vasopressor requirements. The chest was left open with packing and a wound vac to control bleeding. He returned to the OR the following day with stable hemodynamics and improved hemostasis allowing successful chest closure. His ICU course was complicated by pneumonia as well as three episodes of atrial fibrillation requiring cardioversion. He was discharged home 30 days after surgery.

Discussion

Beyond the inherent high risk of LV pseudoaneurysm rupture and tissue friability associated with OI, this patient also had a severely dilated LV with severely reduced ejection fraction. Preoperative planning focused on acknowledging high mortality risk associated with surgery, anticipating need for massive transfusion, difficult separation from cardiopulmonary bypass, and the limitations posed by mechanical valves, which precluded mechanical circulatory support. Close interdisciplinary communication allowed for a coordinated strategy, including the decision not to re-replace valves to bioprostheses, and planned delayed sternal closure to allow time for hemostasis and hemodynamic optimization. This collaborative approach was

central to the successful management of this complex case.

Images

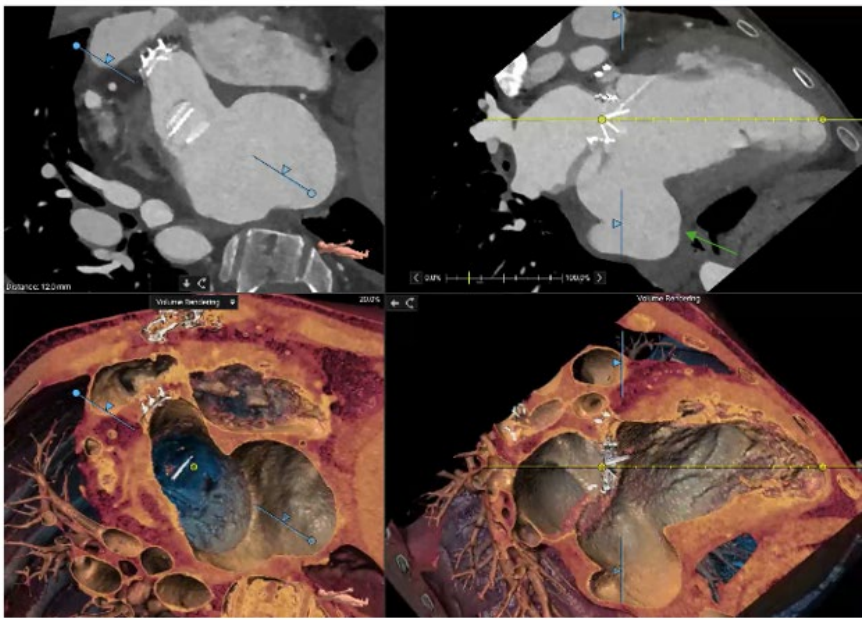


Image 1: Green arrow, LV pseudoaneurysm

LV Pseudoaneurysm

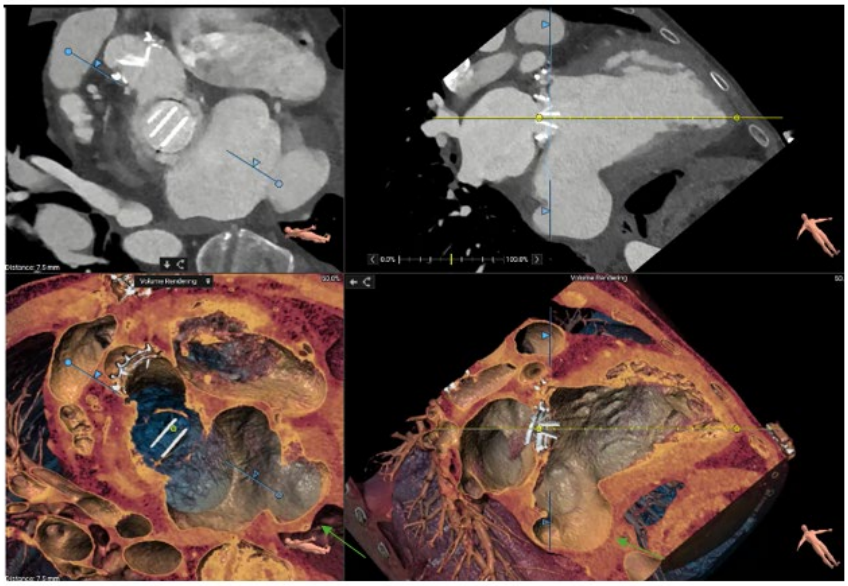


Image 2: Green arrow, LV pseudoaneurysm

LV Pseudoaneurysm

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#FR 3 – Kiosk 19 – Poster 4

Complex Sequelae of Circumflex Artery Myocardial Infarction: Ischemic Mitral Regurgitation and Left Ventricle Free Wall Rupture

Presenting Author: Amanda R. Tomlinson, MD, Adult Cardiac Anesthesia Fellow, The University of Pennsylvania

Introduction

Left circumflex artery (LCx) myocardial infarction (MI) can lead to a cascade of severe mechanical complications causing high levels of morbidity and mortality, most notably ischemic mitral regurgitation (MR) and left ventricle (LV) free wall rupture.

Case Presentation

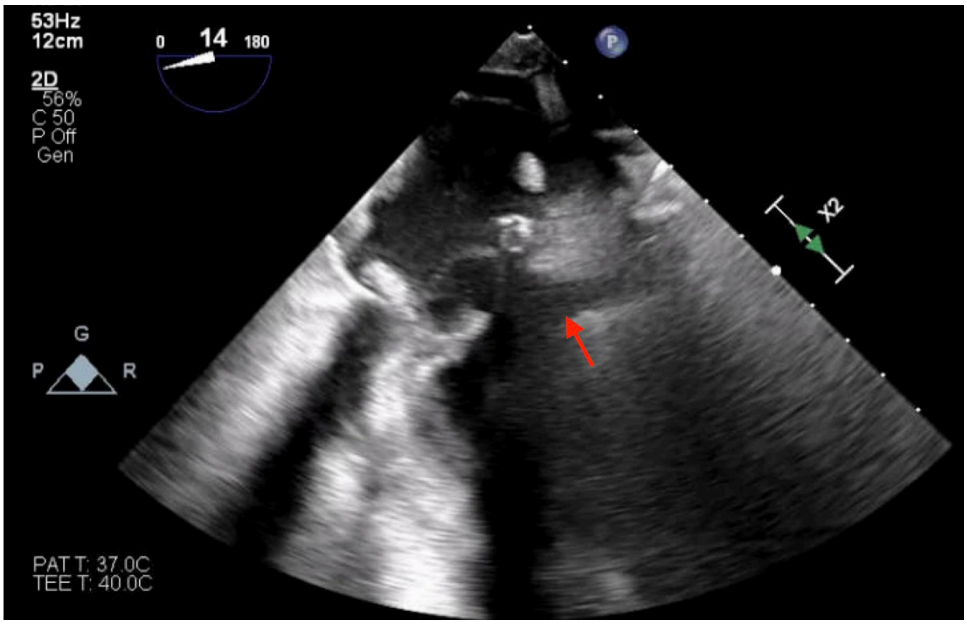
A 60-year-old female with a history of hypertension presented to an outside hospital with chest pain and shortness of breath. She precipitously decompensated into acute respiratory failure and cardiogenic shock. She was found to have a STEMI (LCx and right coronary artery (RCA)). She underwent revascularization of the RCA and LCx and Impella CP placement. Then she was transferred to our institution for further management. Due to severe oropharyngeal edema and bleeding a TEE was not performed during her initial presentation. TTE showed severe MR but did not pick up a papillary muscle rupture. Her cardiogenic shock was medically managed and her Impella was removed two days after transfer. A week later once her oropharyngeal edema and bleeding improved she was taken to the OR for a mitral valve replacement. The initial TEE exam showed severe anteriorly directed MR. There was a subtle rupture of the posterior medial papillary muscle and dyskinesis of the inferior and inferolateral walls consistent with known LCx and RCA STEMI. During the operation the posteromedial papillary muscle rupture was confirmed by the surgical team. The mitral valve was replaced with a Mitris prosthetic valve. When the cross clamp was removed after valve replacement there was severe bleeding in the surgical field. On transesophageal echo a defect in the lateral wall was noted consistent with an LV free wall rupture. The clamp was replaced and the surgical team made attempts to patch the LV free wall rupture but the left ventricular tissue was extremely friable and the repair would not hold despite multiple attempts to patch the defect. The patient was placed on central ECMO and taken to the ICU with an open chest. Shortly after arrival to the ICU the patient was transitioned to comfort care and expired.

Discussion

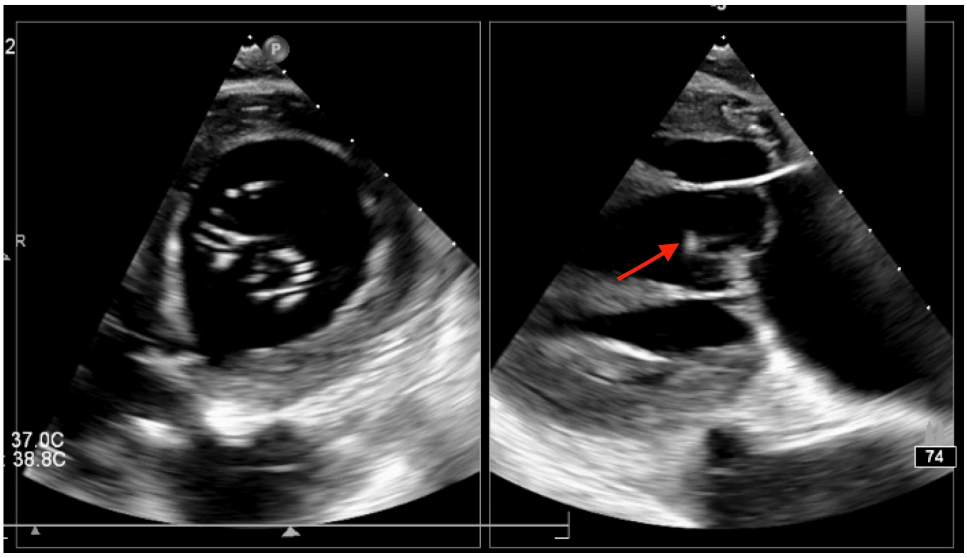
The occurrence of ischemic MR and LV free wall rupture following LCx MI are two of the most severe mechanical complications of acute MI. Ischemic MR most commonly results from papillary muscle infarction or rupture, particularly involving the posteromedial papillary muscle, which is supplied by the LCx in most patients (1). Approximately one third of patients with LCx MI will have some degree of ischemic MR which can lead to acute volume overload of the left heart and precipitate acute respiratory decompensation and cardiogenic shock. Left ventricular free wall rupture after MI is rare but risk increases when the culprit lesion is the LCx (2). In our case the classic presentation of complications from LCx infarction are present,

from the acute papillary muscle rupture causing MR leading to volume overload, acute decompensated respiratory failure and cardiogenic shock, then the left ventricular free wall rupture within a week of primary insult.

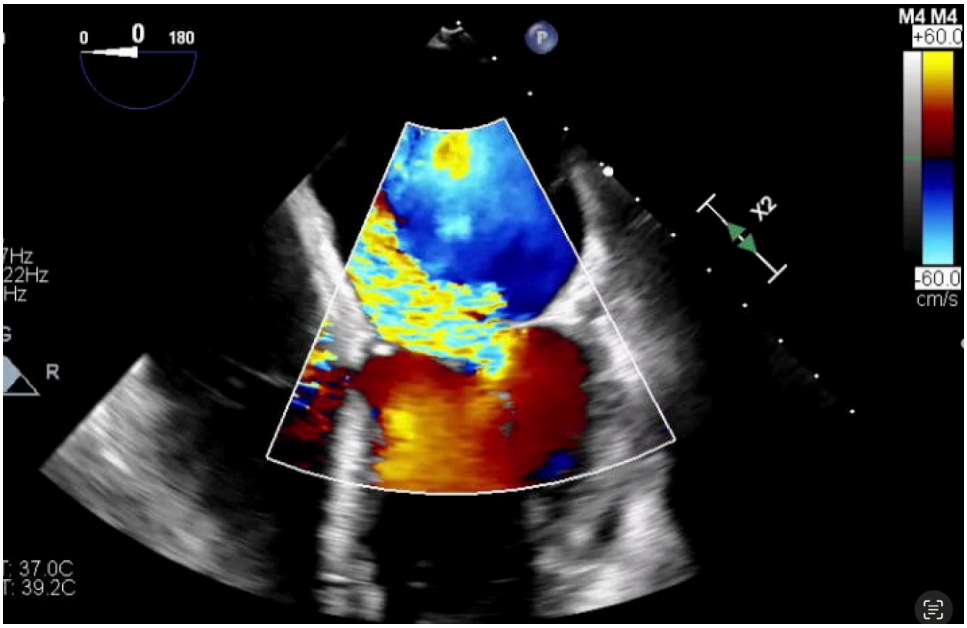
Images



Left ventricular free wall rupture after mitral valve replacement



Very subtle posterior papillary muscle rupture



Severe preoperative mitral regurgitation

References

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#FR 3 – Kiosk 19 – Poster 5

Durable Right Ventricular Assist Device Implantation as Destination Therapy in Irreversible RV Failure following LVAD Placement

Presenting Author: Charbel Barrak, DO, Henry Ford Health

Introduction

Left ventricular assist devices (LVADs) have transformed heart failure care, yet right ventricular failure (RVF) after LVAD implantation remains one of the deadliest complications. Although temporary RV support is well established in early RVF, the role of durable right ventricular assist devices (RVADs) has traditionally been limited to salvage therapy. Emerging experience suggests that durable RVADs may serve as destination therapy for select patients with irreversible RV dysfunction who are not transplant candidates. This strategy is rarely reported, especially among individuals with fixed pulmonary hypertension, chronic RV dysfunction, and morbid obesity, conditions that markedly reduce RV adaptive capability after LVAD unloading. This case highlights when durable RVAD therapy becomes not optional, but required for survival.

Case Presentation

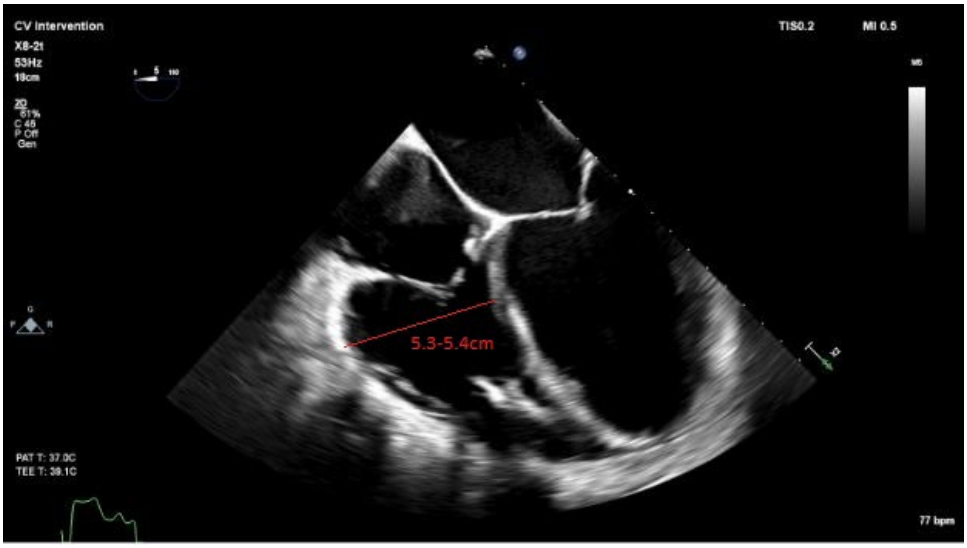
A 56-year-old woman with end-stage left heart failure, chronic kidney disease, group 2 pulmonary hypertension, and morbid obesity (BMI 46) presented in cardiogenic shock and was approved for LVAD implantation. During separation from cardiopulmonary bypass, she developed severe RVF despite maximal inotropes and pulmonary vasodilators. A temporary RVAD was placed with immediate improvement. One week later, attempts to wean support demonstrated persistent, profound RV dysfunction via an empty LV, septal shift, and minimal RV contractility, consistent with nonrecoverable RV failure. Because she was not a transplant candidate and was unlikely to survive without permanent mechanical support, the multidisciplinary team approved durable RVAD implantation as destination therapy. During reattempted weaning, she transiently tolerated reduced support but acutely decompensated during chest closure, requiring emergent CPB and urgent durable RVAD placement. Her postoperative course was complicated by pneumonia, sepsis, tracheostomy dependence, and ischemic stroke. She ultimately died in the ICU despite maximal therapy.

Discussion

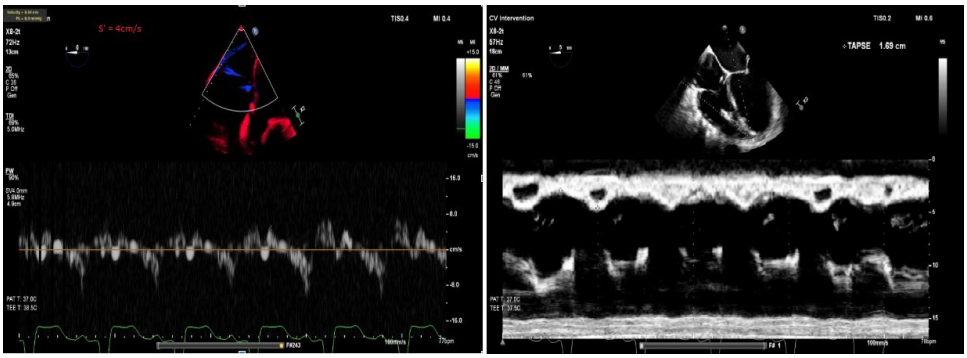
This case demonstrates a clinical scenario in which durable RVAD support serves as destination therapy rather than a bridge strategy. The patient's comorbidities created physiology in which RV recovery was not realistically achievable, even with early temporary support. Her course illustrates limitations of risk scores such as EUROMACS-RHF, CRITT, and the Michigan model, which cannot reliably identify patients whose RV dysfunction is fundamentally irreversible. In such cases, durable RVAD implantation may be the only viable long-term treatment, and delayed transition can worsen outcomes. Her case underscores key implications for contemporary MCS practice. First, durable RVADs should be considered earlier for patients showing clear markers of irreversible RV failure during or after LVAD implantation. Second, there is a need for standardized criteria defining candidacy for RVAD destination therapy, especially in patients with pulmonary

hypertension or chronic RV impairment. Lastly, improved models integrating hemodynamics, imaging, and comorbidities are needed to guide timely use of permanent right-sided support. As technology evolves, cases like this underscore the importance of defining durable RVAD therapy as a legitimate destination option for select patients with irreversible RVF after LVAD implantation.

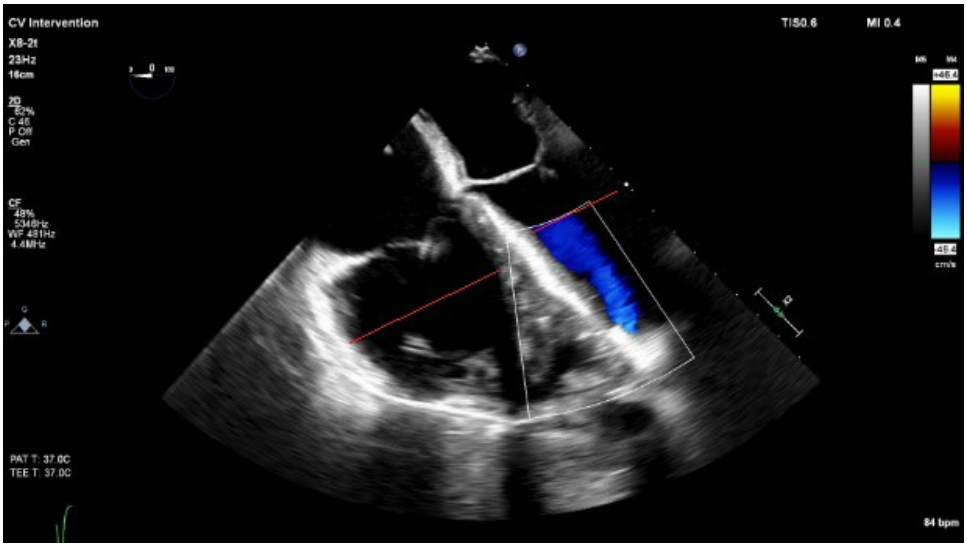
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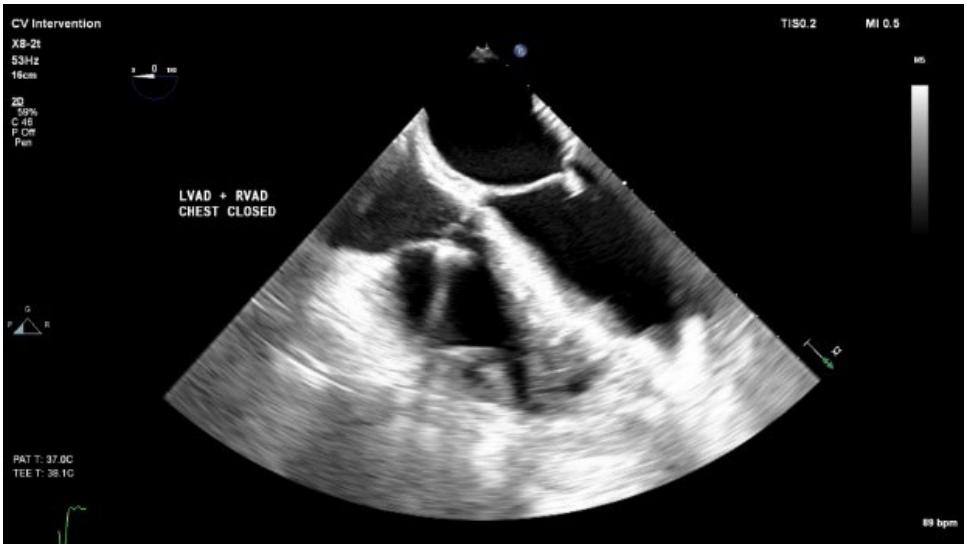
Pre-cardiopulmonary bypass transesophageal echocardiographic image of ME 4-Chamber view showing significant RV dilation.



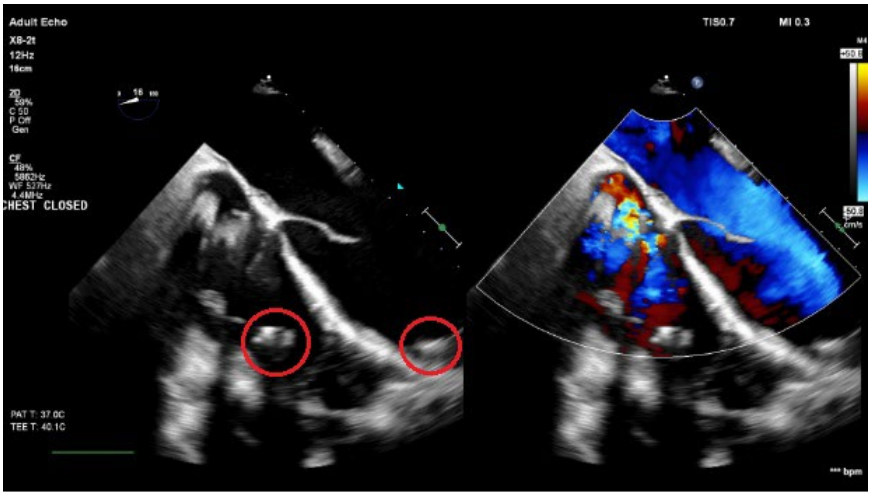
Pre-cardiopulmonary bypass transesophageal echocardiographic image significant for S' = 4cm/s with a TAPSE 16.9mm



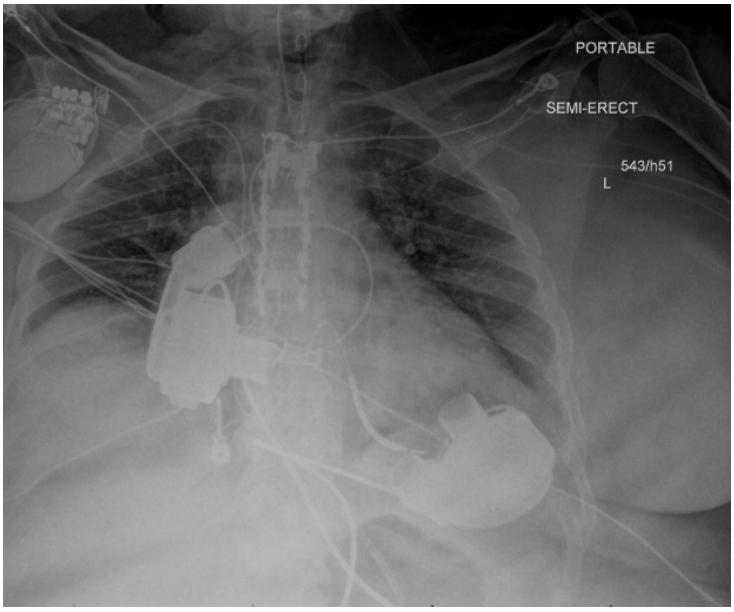
Post-cardiopulmonary bypass after LVAD placement showing a severely dilated RV with an underfilled LV, signifying RV failure.



Post-cardiopulmonary bypass after LVAD placement and temporary RVAD placement with resolution of RV dilation and LV emptiness.



Transesophageal image of ME 4-chamber view showing a durable LVAD and RVAD.



Chest x-ray showing the presence of both durable LVAD and durable RVAD.

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#FR 3 – Kiosk 19 – Poster 6

Anesthetic management of an infected cardiac implantable electronic device causing perforation of the tricuspid valve with severe tricuspid regurgitation

Presenting Author: Joshua Fridley, MD, Department of Anesthesiology, West Virginia University

Introduction

Cardiac implantable electronic device (CIED) infections are associated with substantial morbidity and mortality. Perioperative complications can arise in patients with infected CIED systems, and their anesthetic management presents significant challenges. We present a case of a patient with CIED-related infective endocarditis scheduled for percutaneous mechanical aspiration via a thrombus aspiration system, found to have new severe tricuspid regurgitation secondary to leaflet perforation on intraoperative trans-esophageal echocardiography (TEE).

Case Presentation

A 66-year-old female with a past medical history significant for coronary artery disease and heart failure with a reduced ejection fraction with a dual-chamber implantable cardioverter-defibrillator (ICD) implanted in 2011 presented to the emergency department with complaints of malaise. Inpatient workup revealed a positive blood culture for staphylococcus lugdunensis, concerning for endocarditis. The patient underwent trans-thoracic echocardiography (TTE) with concern for echo-densities on the ventricular ICD lead and tricuspid valve. A subsequent TEE the next day showed a large 2cm tricuspid valve echo-density, thought likely to be a vegetation fused with the ICD ventricular lead and mild tricuspid regurgitation. The electrophysiology team scheduled the patient for lead extraction and mechanical aspiration of the presumed vegetation under general anesthesia. Monitoring included standard equipment along with invasive arterial blood pressure measurement and intraoperative TEE. The TEE, performed 8 days after the first TEE, showed a mobile echo-density consistent with vegetation emanating from a flail anterior tricuspid valve leaflet with a perforation that further contributed to eccentric and posteriorly directed severe tricuspid regurgitation. Given these findings, the surgical team decided to cancel the procedure and plan for open surgical management. The patient underwent a tricuspid valve replacement via full sternotomy with 33 mm St. Jude bioprosthetic-heterograft valve and removal of infected ICD system. The patient was admitted to the cardiac intensive care unit postoperatively and progressed without complication before being discharged.

Discussion

According to the 2017 HRS consensus statement on CIED lead management, the incidence of CIED infection is 1.9 per 1000 device-years, with pocket infection being more common than blood stream infection (1). When lead infections are suspected, TEE is preferred for diagnosing these infections due to its increased sensitivity compared to other imaging modalities at detecting vegetations (2). In cases with significant lead infection and large vegetations (2cm), the decision between surgical and percutaneous extraction must be tailored to each patient's anatomy, comorbidities, and infection burden (3). This case highlights the critical role of the cardiac anesthesiologist in the perioperative management of patients with CIED-related endocarditis, along with the complex decision-making involved in high-risk CIED lead extraction. Even though the patient's clinical status had not changed, intraoperative TEE led to a significant alteration in procedural plan that may have otherwise gone unnoticed.

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#FR 3 – Kiosk 19 – Poster 7

Perioperative Management in a Patient with Severe Infective Endocarditis with Multivalvular Involvement and Aortic Root Abscess

Presenting Author: Rodney Chen, MD, Anesthesiology Fellow, Baylor College of Medicine

Introduction

Infective endocarditis (IE) is rare, with less than 7 cases in 100,000 people each year, and overall mortality rates approaching 30% despite advances in diagnosis and management (1,2). Delayed intervention can lead to severe complications such as multivalvular destruction, abscess formation and conduction abnormalities, all of which may rapidly escalate the complexity of surgical and anesthetic management (3). In patients with extensive valvular involvement, perioperative management is challenging due to hemodynamic instability, timely management of vasoplegia, coagulopathy, and the need for complex surgical reconstruction.

Case Presentation

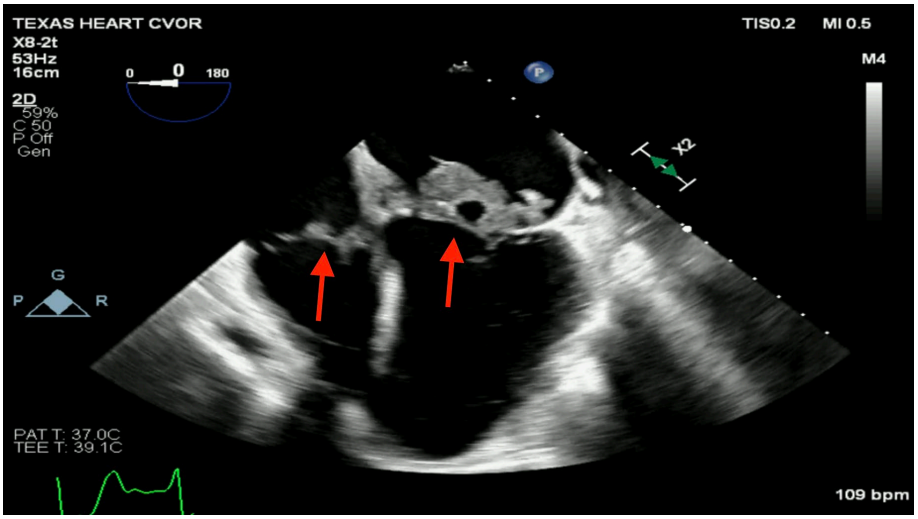
A 64-year-old male with multiple comorbidities including uncontrolled type 2 diabetes and severe aortic stenosis status post bioprosthetic surgical AVR, presents with severe infective endocarditis. Preoperative TEE shows a large anterior mitral leaflet vegetation, aortic root abscess with large aortic cusp vegetation, and smaller tricuspid septal leaflet vegetation. Given the severity of infection and structural complications, the patient was scheduled for mitral valve replacement and aortic root repair, and possible tricuspid valve intervention. Intraoperative TEE confirmed the above findings but demonstrated compromise of the aorto-mitral curtain with two major aortic root fistulae, one from the non coronary cusp to the left atrium and one from the right coronary cusp to the right atrium, along with severe prosthetic aortic stenosis. The patient ultimately underwent a commando procedure, involving aortic root repair using, reconstruction of the intervalvular fibrosa with pericardial patch, an MVR, and a hemi-Cabrol procedure of the left coronary artery. The intra-operative course was complicated by mixed shock, profound vasoplegia, and significant bleeding and coagulopathy around the aortic root requiring massive transfusion and a return to cardiopulmonary bypass. Multimodal vasopressor therapy with norepinephrine, vasopressin, and angiotensin II was required to maintain adequate perfusion. Ultimately, the decision to initiate VA ECMO was made for postcardiotomy shock and to keep the cardiac chambers decompressed to assist with hemostasis and ventricular recovery. Postoperatively, the patient underwent decannulation and chest closure a few days later. The patient required tracheostomy and CRRT but is otherwise recovering.

Discussion

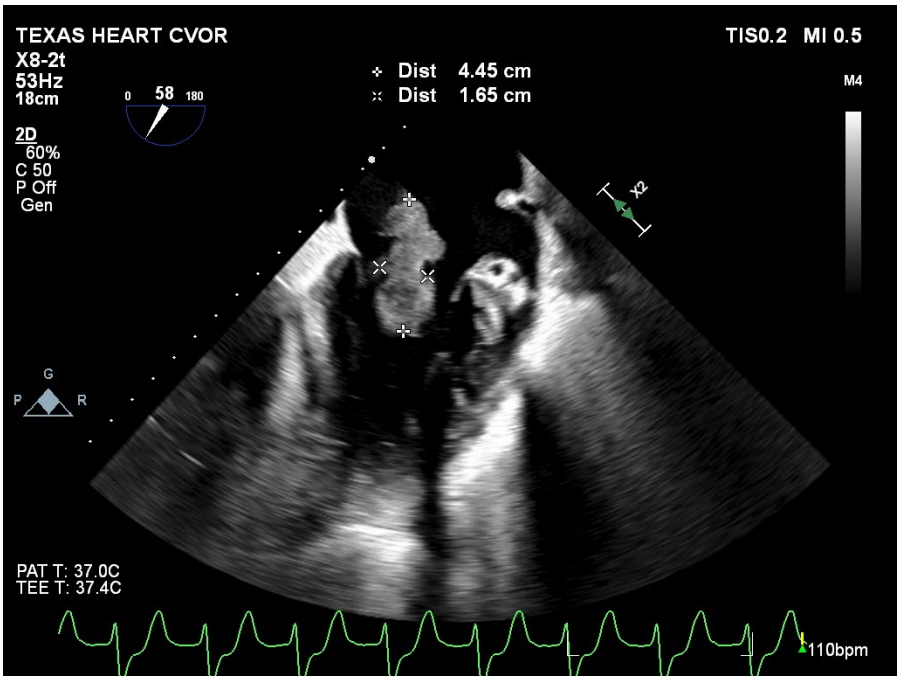
Infective endocarditis in patients with significant comorbidities require comprehensive echocardiographic evaluation as it can reveal additional valve involvement and structural complications. In this case, intraoperative TEE helped to guide decision making for a Commando procedure with an aortic root replacement and MVR. Careful multidisciplinary planning was essential to safely navigate perioperative management for this patient. Due to the severe root bleeding given the tissue quality, an additional return to

cardiopulmonary bypass was necessary for additional hemostasis. The decision to initiate and maintain VA ECMO allowed for cardiac decompression and clot formation. This case highlights the importance of comprehensive echocardiographic evaluation and proactive perioperative management strategies in patients with multivalvular destruction due to severe IE.

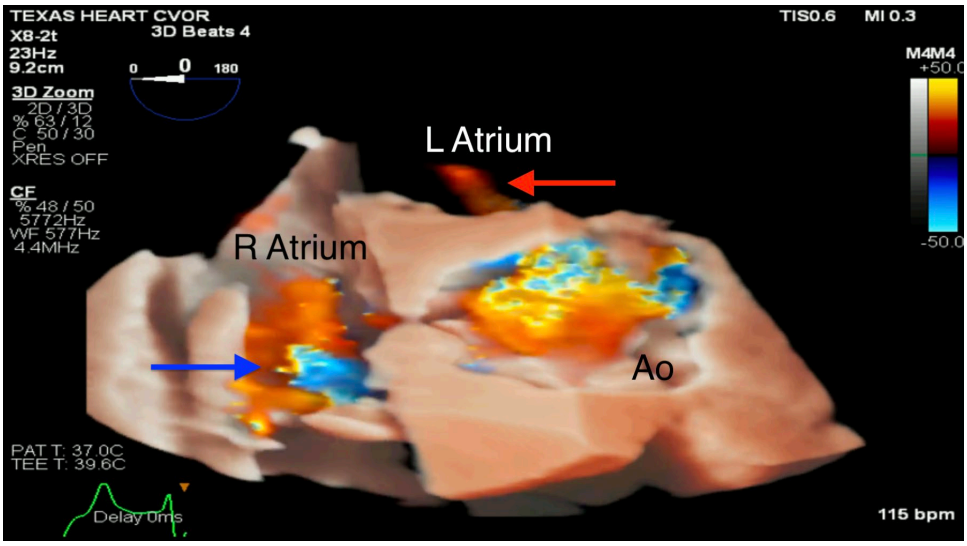
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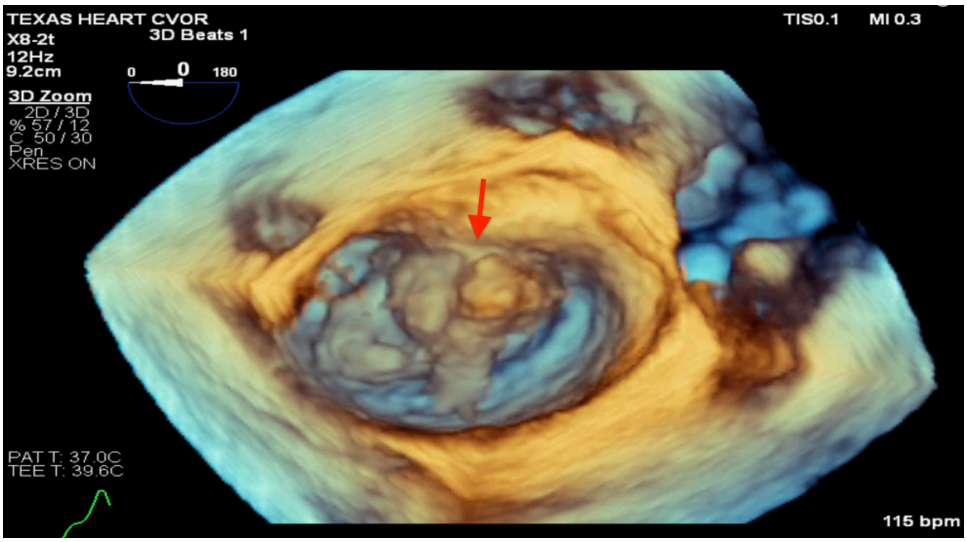
A mid-esophageal 4-chamber view of the mitral and tricuspid valves with noted vegetation growth along both valves (red arrows).



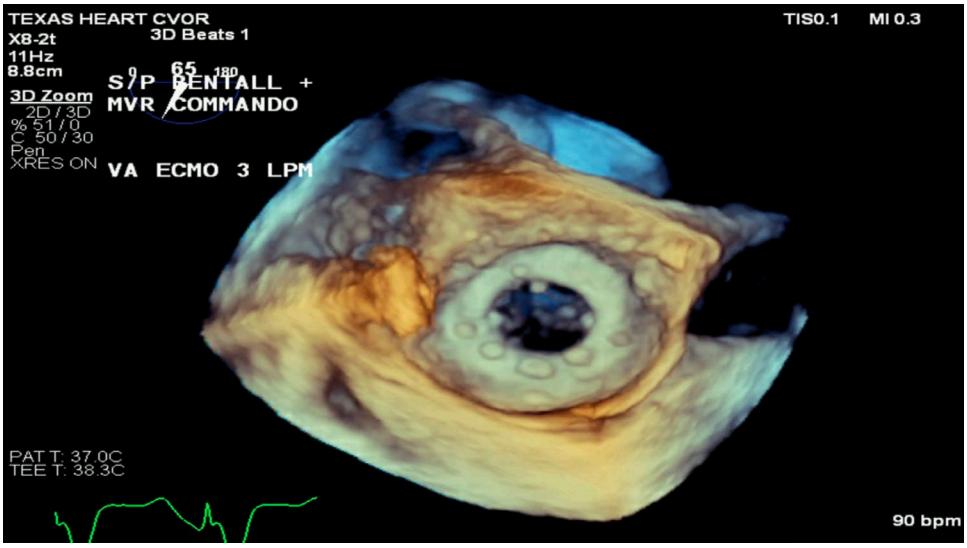
A mid-esophageal mid-commisural view of the mitral valve vegetation, measuring 4.5 cm x 1.5 cm in this view.



A true view 3D model of the aortic root with clear fistula jets traveling into both the left and right atria.



3-D mitral valve imaging demonstrating pre-procedural mitral valve vegetation predominantly along the anterior leaflet



A 3-D mitral valve image status post valve replacement, demonstrating a well-placed bioprosthetic mitral valve.

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#FR 3 – Kiosk 20 – Poster 1

Electrophysiologic Challenges During LVAD Placement: Management of CRT-D Noncapture Requiring Emergent Temporary Transvenous Pacer

Presenting Author: Courtney Vidovich, MD, Anesthesiology Resident, The Ohio State University Wexner Medical Center

Introduction

Many patients receiving left ventricular assist devices (LVADs) have pre-existing implantable cardioverter defibrillators (ICDs) or cardiac resynchronization therapy with defibrillator (CRT-D) devices. During LVAD placement, the proximity of a right ventricular (RV) lead predisposes it to dysfunction – most often from electromagnetic interference with the LVAD and, rarely but of more detriment, due to mechanical impairment[1] as presented here.

Case Presentation

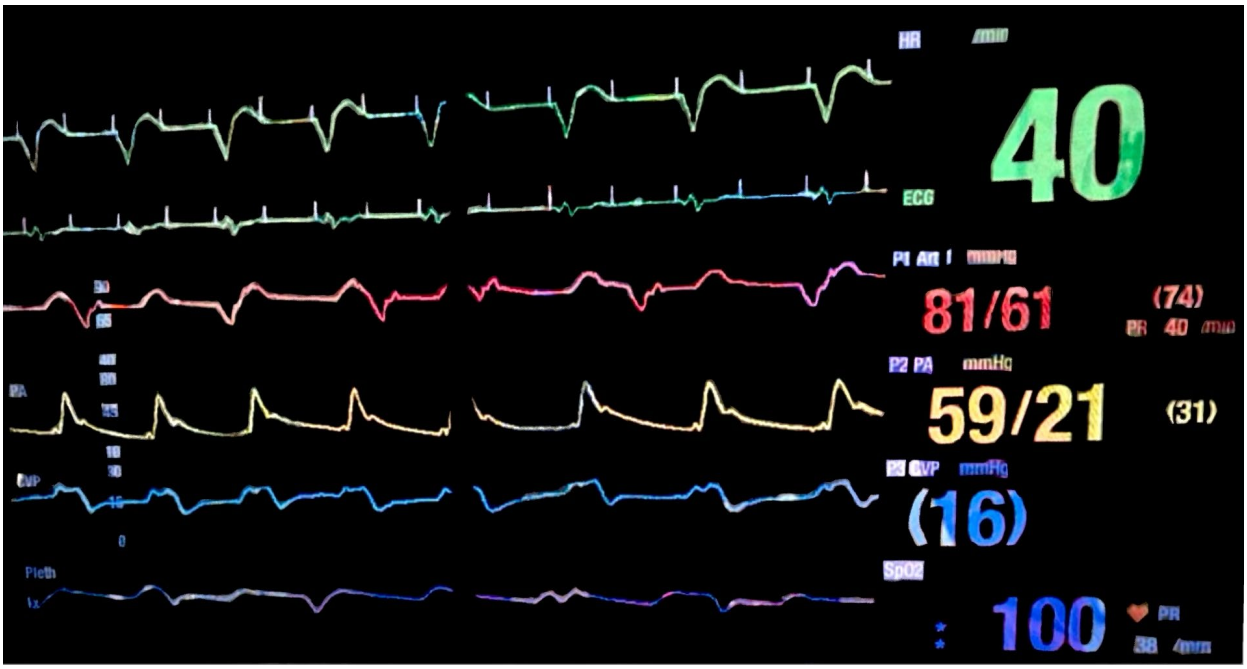
A 72-year-old woman with advanced heart failure presented for implantation of a HeartMate 3 (Abbot, Chicago, IL) LVAD. Her history included paroxysmal atrial flutter (AF) with complete heart block and ventricular tachycardia (VT) managed by CRT-D therapy, coronary artery disease status-post drug-eluding stent placement, prior mechanical mitral valve replacement, chronic kidney disease, and diabetes mellitus. Preoperative CRT-D interrogation confirmed pacemaker dependence with underlying AF, complete AV block, and a native ventricular rate of 40 bpm. The CRT-D was programed to VOO 80 bpm with defibrillation and anti-tachycardia pacing (ATP) disabled for surgery. Cardiopulmonary bypass (CPB) duration for LVAD implantation was 90 minutes, after which the LVAD was successfully activated. Upon weaning from CPB, telemetry revealed a ventricular rhythm at a rate of 40 bpm despite the CRT-D programed to VOO 80 bpm. The rhythm transitioned to VT which was medically managed with boluses of amiodarone, lidocaine, and magnesium. Given a concern for failed pacemaker capture, the electrophysiology (EP) team was consulted intraoperatively. Interrogation revealed underlying AF, but recurrent VT limited further assessment. The device was reprogrammed to VVIR at 80 bpm with shocks and ATP re-enabled. VT continued intermittently with diminished response to medications and no ATP/ICD therapies provided. Repeat interrogation revealed stable lead impedance but rising capture thresholds. The device was reprogrammed to DDDR 80 bpm, the LV lead was disabled, and the RV lead output was maximized. VT recurred, triggering ATP and a subsequent defibrillation, restoring pacing at 80 bpm. Discussions among surgery, anesthesiology, and EP teams raised concern for mechanical lead displacement during LV apical coring or suturing during the LVAD inflow cannula implantation. The patient was transferred to the EP suite for an emergent temporary transvenous pacer (TVP). Ultimately, she did not recover CRT-D function and required a new permanent pacemaker with prior RV lead left in situ given concern for suture entrapment.

Discussion

While rare, this case of CRT-D lead malfunction following LVAD implantation in a pacemaker-dependent patient likely resulted from intraoperative lead disruption. In such patients, separation from bypass presents

unique considerations, as new arrythmias may indicate device lead failure as evidenced here with poor capture, sensing failure, and R-on-T mediated VT refractory to first-line management.[2] Intraoperative device interrogation confirmed rising capture thresholds with stable impedance, consistent with lead micro-dislodgement requiring emergent TVP.[2,3] Early recognition, prompt interrogation, and multidisciplinary coordination were crucial for diagnosis and stabilization.

Images



Initial telemetry following separation from bypass.

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#FR 3 – Kiosk 20 – Poster 2

Atrioventricular Groove Disruption Identified as Left Atrial Dissection on Transesophageal Echocardiography

Presenting Author: Kayla Clodfelter, MD, Anesthesia Resident, Indiana University School of Medicine

Introduction

Atrioventricular groove disruption (AVGD) is an incredibly rare but potentially catastrophic complication of mitral valve surgery (MVS). Intraoperative transesophageal echocardiography (TEE) can be used to detect intracardiac pathologies such as left atrial (LA) dissection, a potential precursor to AVGD, in the post-cardiopulmonary bypass (CPB) period. Early detection of partial AVGD is critical to prevent further progression.

Case Presentation

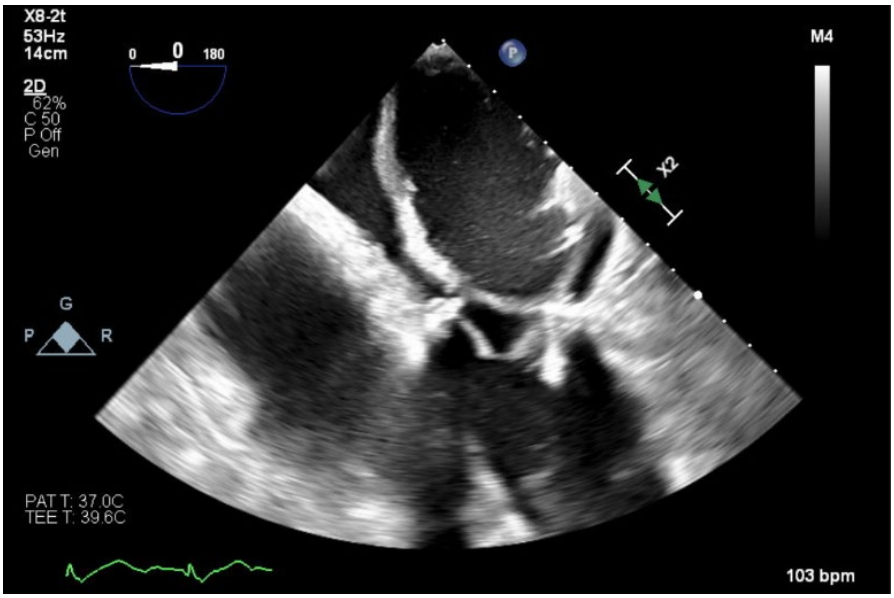
A 52-year-old male with history of STEMI complicated by cardiac arrest, heart failure with reduced ejection fraction (EF) of 40%, and severe mitral insufficiency (MI) presented for mitral valve replacement. The case started uneventfully, and a bioprosthetic mitral valve (BMV) was placed in the mitral position without any noticeable complications. During the first wean from CPB, TEE identified an expanding fluid-filled mass in the LA, concerning for LA dissection [Figures 1,2]. The heart was re-arrested for repair. Investigation revealed an LA intramural hematoma, and a patch was sewn into the atrial wall. A second attempt to wean from CPB revealed bleeding into the surgical field coming from the same area as the hematoma, raising concern for AVGD. The heart was arrested again, and the newly implanted BMV was removed to inspect the mitral valve annulus. A small hole was noted on the anterior aspect of the annulus, which was repaired with a patch. A new BMV was implanted, and the patient was weaned off bypass for the third time. Initially, the patient was stable, and there were no concerning findings on TEE. However, upon chest closure, the patient became acutely hypotensive, and TEE revealed redevelopment of the LA dissection with obstruction of mitral valve inflow [Figures 3,4,5,6]. The chest was reopened, and the patient was returned to CPB. The BMV was explanted, and a patch was sewn around the entire circumference of the annulus before a new BMV was sutured in place. After weaning from CPB for the fourth time, the patient was vasoplegic, but TEE showed no further recurrence of AVGD or LA dissection. The postoperative course was largely uneventful, and the patient was discharged from the hospital a few weeks later.

Discussion

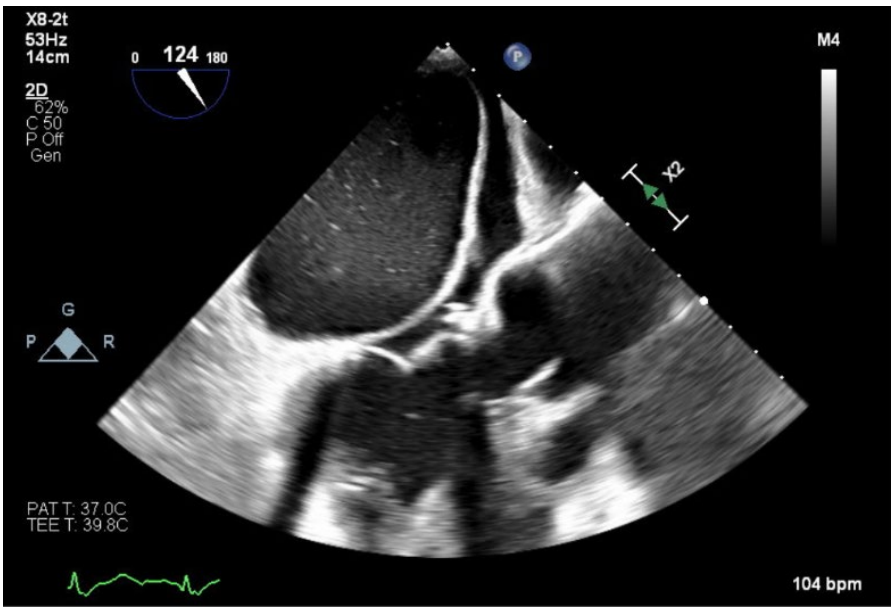
LA dissection is a rare complication of MVS and can be the result of a partial atrioventricular groove (AVG) injury, in which pressurized blood creates a dissection plane between the endocardium and myocardium. Therefore, LA dissection can be a sign of impending AVG rupture. In this case, the quick identification of LA dissection and immediate reinitiation of CPB may have prevented the development of full rupture by decompressing the AVG. This could explain why when the patient was taken off CPB the second time, an AVG injury was not immediately identified. Over time, the filling pressure gradually extended the AVG injury, bisecting the tissue until it traversed the entire groove resulting in frank rupture. This case highlights the

complex nature of AVGD along with the cardiac anesthesiologist's role in intraoperative diagnosis and management. Intraoperative TEE can detect intracardiac pathologies otherwise not visible to our surgical colleagues that require their intervention. If LA wall dissection occurs following MVS, consider AVGD as the source.

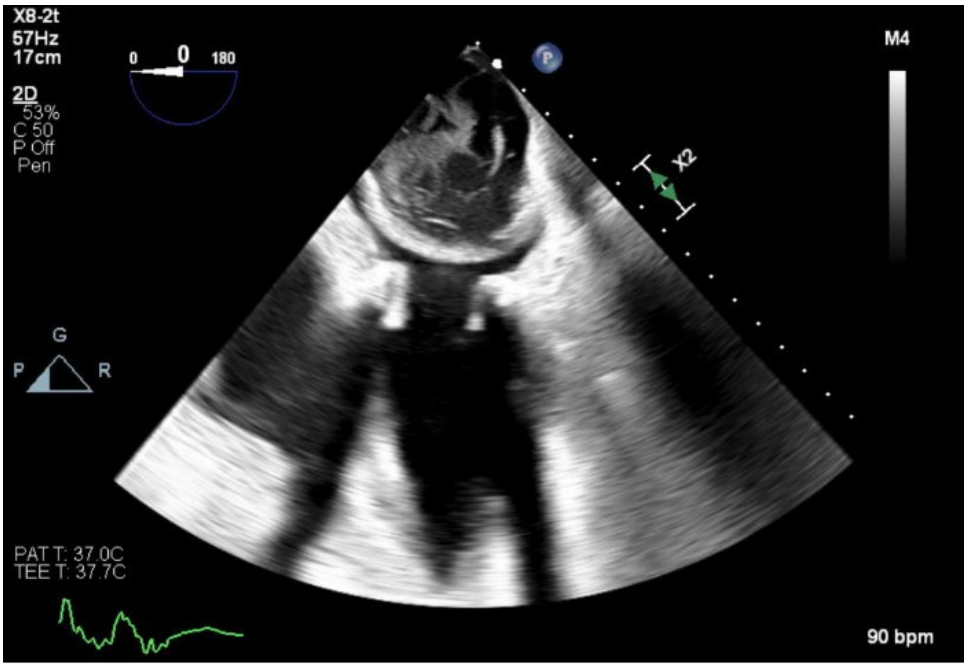
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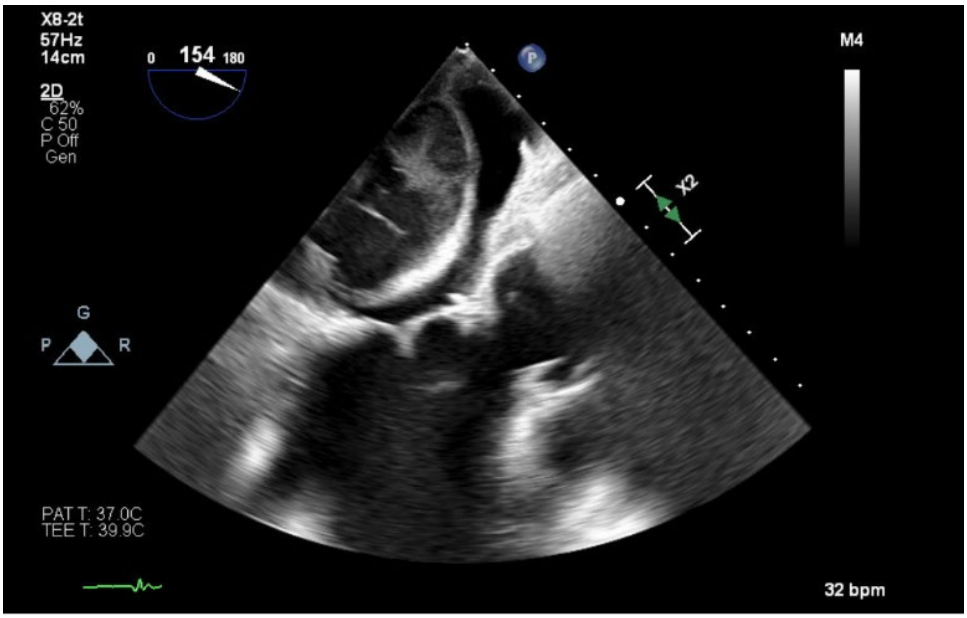
TEE midesophageal (ME) four chamber view showing LA dissection within the left atrium centered just above the newly placed bioprosthetic mitral valve.



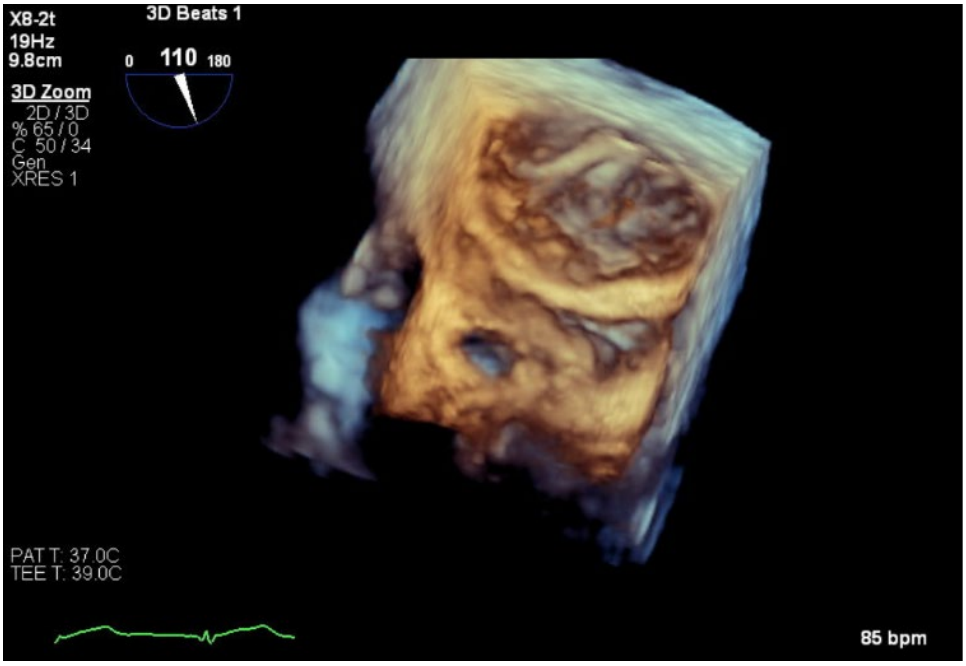
TEE ME long-axis (LAX) view showing LA dissection along the posterior wall protruding anteriorly within the left atrium.



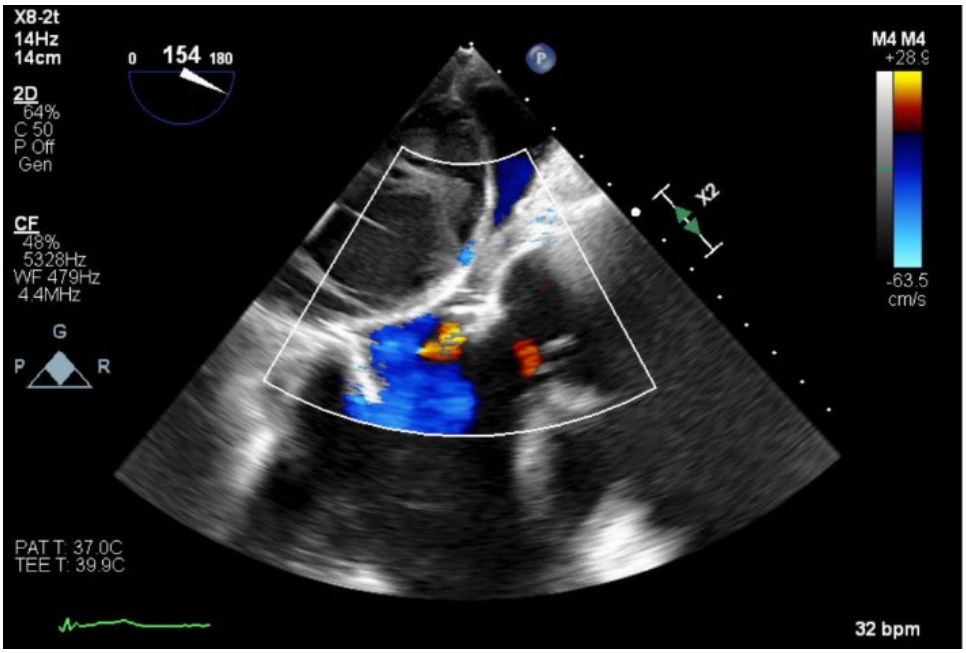
TEE ME four chamber view demonstrating redevelopment of LA dissection with new septation and heterogeneity of the false lumen contents after the first patch was applied to the AVGD.



TEE ME LAX view redemonstrating new characteristics of LA dissection seen in image 3.



Reconstructed 3D image from TEE showing the topographical characteristics of the false lumen and its proximity to the mitral valve.



TEE ME LAX with doppler demonstrating the flow obstruction across the mitral valve in diastole from the LA dissection.

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#FR 3 – Kiosk 20 – Poster 3

Recurrent Ventricular Tachycardia following Mask Induction for Transcatheter Mitral Valve-in-valve Replacement

Presenting Author: Daniel J. Courtney, M.D., Resident, Dartmouth Hitchcock Medical Center

Introduction

Volatile agents are considered a safe induction agent due to minimal effects on the conduction system, catecholamine release, and hemodynamic stability. When higher concentrations are used during a mask induction, arrhythmias reported include nodal rhythm with bradycardia, atrioventricular dissociation, and corrected QT prolongation (1). Populations in which arrhythmias are more likely to occur include patients with coronary artery disease, younger age, and use of 8% sevoflurane (2).

Case Presentation

A 69 year old male presented for a transcatheter mitral valve in valve replacement (ViV- TMVR) for severe bioprosthetic valve stenosis. His past medical history was significant for coronary artery bypass graft and bioprosthetic mitral valve replacement for severe mitral regurgitation 13 years prior. He presented after progressive dyspnea for 8 months, was found to have moderate to severe mitral stenosis (MS) with MG of 7 mmHg and valve area of 1.1 cm2. Further work up was notable for severe pulmonary hypertension (pHTN) with pulmonary artery pressures 90/40 mmHg with a PCWP of 28 mmHg. His echo showed preserved EF, severely dilated right ventricle (RV) with moderately reduced function and severe TR. Other history notable for CKD III, SSS s/p DC PPM. In preparation on the day of surgery, in addition to standard monitors, an invasive arterial pressure was obtained with opening systemic pressure of 87/39 which is equal to his pulmonary pressures. Given the severe pHTN and reduced RV function, a vasopressin infusion was started. With the goal of avoiding hemodynamic perturbations with induction, Sevoflurane inhalation at 4% was started to induce general anesthesia. Peri-induction EKG was notable for recurrent non sustained ventricular tachycardia (NSVT) associated with profound hypotension, these were new to the patient as he had no history of VT. VT kept recurring despite administering lidocaine and amiodarone boluses and infusions. Given the non sustained nature of the VT we elected to proceed with the percutaneous implantation of a #29 bioprosthetic mitral valve that appeared to be well seated and had normal trans-mitral gradients. Further work up showed no electrolyte abnormalities and no evidence of myocardial ischemia as an explanation of the recurrent VT. The post operative course was notable for arrhythmia quiescence and relative hemodynamic stability. He was extubated 6 hours postoperatively and weaned off antiarrhythmic infusions. His DC PPM was functioning normally upon interrogation.

Discussion

This report highlights the challenging nature of managing a patient with severe MS, severe pHTN and moderate RV failure presenting for a ViV-TMVR. The recurrent VT, despite the lack of VT history, in this patient is likely a combination of the patient's severe valvular cardiomyopathy, the initiation of arrhythmogenic inotropic infusion and the high concentration of Sevoflurane inhalation. There are reports of severe

bradycardia, supraventricular arrhythmias and QTc prolongation with high concentration sevoflurane inhalation, however, there were no reported cases of malignant ventricular arrhythmias.

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#FR 3 – Kiosk 20 – Poster 4

The "Clot" Thickens: An Unexpected Finding During LVAD Placement

Presenting Author: Ren Ariizumi, MD, Adult Cardiothoracic Anesthesia Fellow, Cleveland Clinic Foundation

Introduction

Placement of a durable left ventricular assist device (LVAD) can serve as a bridge to transplant or as destination therapy for patients with advanced heart failure. Anesthetic management during LVAD placement requires careful support and advanced transesophageal echocardiographic (TEE) imaging. To that end, we present a case for LVAD placement with unexpected findings on TEE requiring alterations in management.

Case Presentation

The patient is a 48-year-old morbidly obese male with advanced heart failure and sustained VT despite medical optimization scheduled for LVAD placement after inpatient workup. Preoperative TTE was significant for EF of 21%, severely dilated LV with severe systolic dysfunction, normal RV, no significant valvular abnormalities, no intracardiac shunts, and no thrombi. Chest CT obtained 2 weeks prior to surgical date similarly showed no acute pathology. Day of surgery the patient reported worsening dyspnea and was unable to move himself from the bed to the OR table. Peripheral IV access was secured as well as central venous access with sheath introducer in the right internal jugular vein. Invasive monitoring was placed with left brachial arterial line and pulmonary artery catheter through the sheath introducer. Epinephrine infusion was started prior to induction of anesthesia with IV lidocaine, etomidate, succinylcholine, and additional bolus doses of epinephrine. After intubation and TEE probe placement a large thrombus was identified in the right pulmonary artery, not previously seen on preoperative TTE or CT. After discussion of the findings with the surgical team, the decision was made to delay LVAD insertion and instead place a temporary Impella 5.5 device to support LV function in the acute setting. After placement, the patient was seen by the interdisciplinary Pulmonary Embolism Response Team for further workup and management.

Discussion

Patients presenting for LVAD implantation require thorough preoperative workup and identification of red flags. For this patient, worsening dyspnea and inability to move from the bed to the table were concerning for rapid decompensation and hemodynamic collapse upon induction of anesthesia. Management included placement of all lines and invasive monitoring prior to induction of anesthesia, preemptive inotropic support, and careful avoidance of hypoventilation. Preimplantation TEE evaluation involves a comprehensive exam with special focus on cardiac level shunts, thrombi, AV and TR regurgitation and baseline RV function.1 Updated guidelines also emphasize evaluation of LVAD cannulation sites (LV apex and aorta).2 In this patient, identification of a thrombus in the right pulmonary artery (Images 1-3) prompted discussion between surgery and anesthesia regarding management. While pulmonary thromboendarterectomy was considered, temporary mechanical circulatory support and comprehensive workup before intervention was deemed the safest option. Notably, a CT scan obtained immediately after

Impella placement was inconclusive, despite clear visualization of clot on TEE. Significant clot burden was removed by interventional radiology (Image 4) with a reduction in mean pulmonary artery pressure from 38 to 22. Thus, TEE evaluation can be an important tool for identifying thrombi despite negative pre-operative imaging.

Images

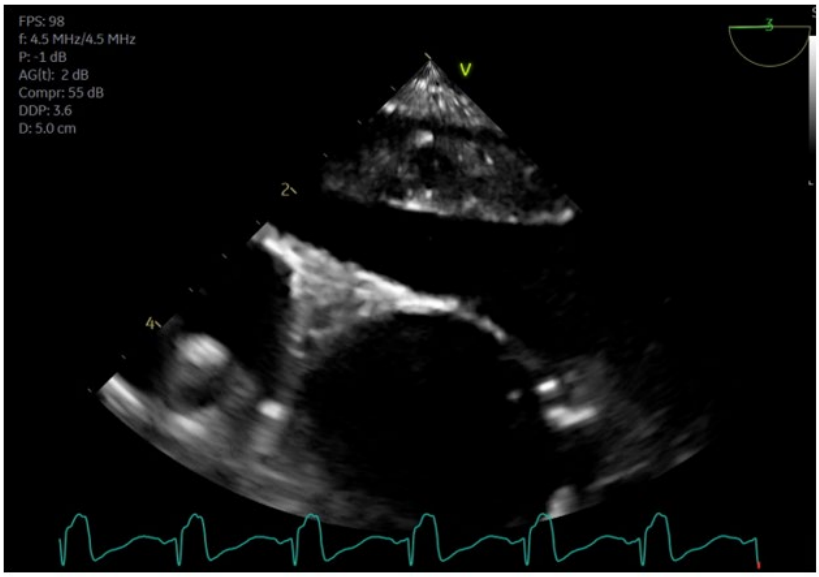


Image 1 – Right PA thrombus seen in upper esophageal ascending aorta short axis view

Image 1 - Right PA thrombus seen in upper esophageal ascending aorta short axis view

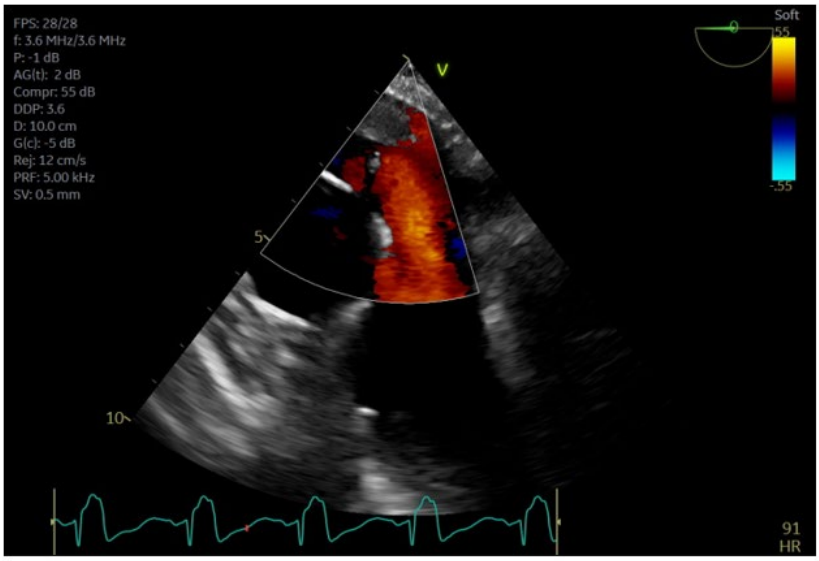


Image 2 – Color doppler flow in right PA around thrombus

Image 2 - Color doppler flow in right PA around thrombus

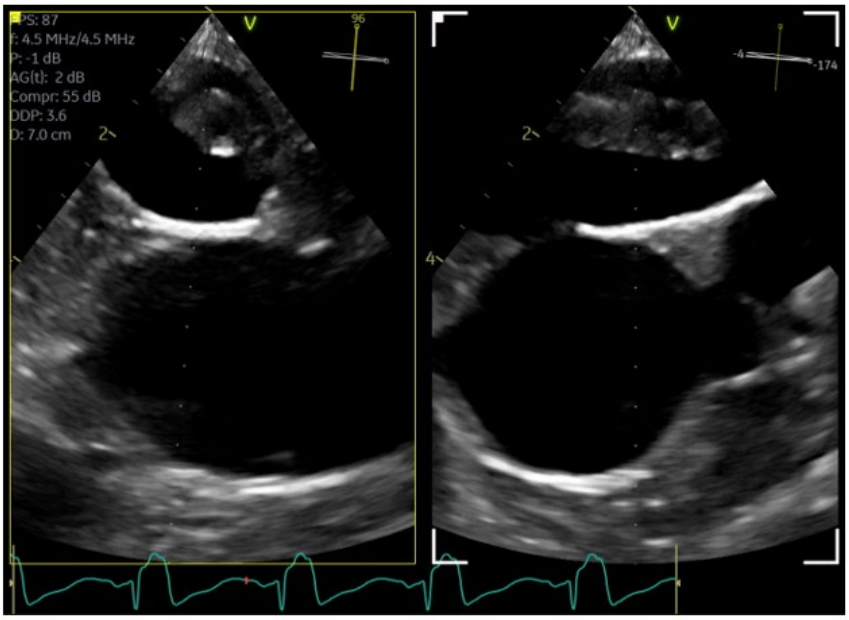


Image 3 – Right PA thrombus seen on multiplane imaging

Image 3 - Right PA thrombus seen on multiplane imaging

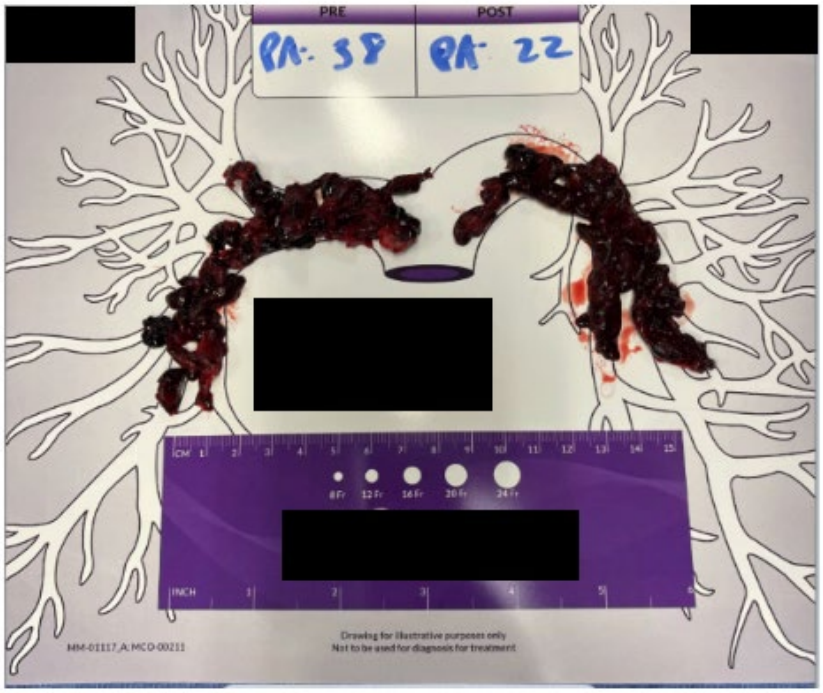


Image 4 – Bilateral PA thrombi removed by Interventional Radiology

Image 4 - Bilateral PA thrombi removed by Interventional Radiology

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#FR 3 – Kiosk 20 – Poster 5

A Case of Avoiding Unnecessary Aortic Surgery with Preincisional Transesophageal Echocardiography Assessment

Presenting Author: Andrew Clark, MD, Cardiothoracic Anesthesia Fellow, Massachusetts General Hospital

Introduction

The spectrum of acute aortic syndromes (AAS) are frequently misdiagnosed, life-threatening conditions, with some studies estimating 30-50% of patients with Type A classical dissection expire prior to reaching hospital care, and other studies estimate subsequent total in-hospital post-surgical mortality following Type A classical dissection at over 20%.[1,2] Within the AAS, an intramural hematoma (IMH) is a type of non-communicating aortic dissection that lacks an intimal tear and has been theorized occur from rupture of the vasa vasorum. It has the potential to rupture through the arterial intima to convert to a classical dissection. Ultimately in evaluation of AAS, rapid determination of the aortic involvement (Stanford Type A or Type B) along with assessment of clinical status are critical to appropriate management, whether primarily surgical or medical. This case highlights the use of echocardiography and how it can help uncover important information in this patient population in the peri-operative setting.

Case Presentation

Our case involves a 55-year-old female with prior history including thyroid carcinoma (status post thyroidectomy and lymph node excisions), chronic back pain (on home oxycontin), anxiety and ADHD who was helicoptered to Massachusetts General Hospital (MGH). After 4 days of persistent discomfort following an initial “tearing” chest/back pain, she presented to a rural emergency room where CT images were suggestive of Type A aortic intramural hematoma (IMH). She was started on esmolol infusion and soon arrived at MGH, briefly admitted to the ICU after repeat CT imaging. Following appropriate consent and discussions, she was promptly taken to the operating room given continued concern for IMH based on repeat imaging. Following anesthetic induction and line placement, a pre-incisional TEE was performed by the cardiac anesthetic team that found no convincing evidence of IMH or other aortic dissection, but did show evidence of fluid in the transverse sinus. Following a multi-disciplinary discussion, the decision was made to abort the surgery, and the patient was extubated and taken to the ICU. Later, more detailed review of the pre-operative CT imaging agreed with TEE assessment, suggesting that the aortic thickening was actually pericardial fluid within the superior pericardial recess. MRI Angiography imaging was subsequently done to again confirm these suspicions. The patient was discharged on a one-month course of colchicine with improvement in inflammatory markers on follow-up.

Discussion

While it does not replace the diagnostic role of CT imaging and has its other limitations, Transesophageal echocardiography (TEE) has unique advantages as a real-time imaging technique that can make diagnoses that inform surgical planning and impact peri-operative decision making. TEE has an important role in defining the extension of any dissection, assessing branch artery patency (e.g. coronary, carotid, etc),

assessing the presence and mechanism of aortic regurgitation, identifying any pericardial effusion or tamponade, and the evaluation of both the true and false lumen.[3] This case highlights the role that the TEE has in the peri-operative care of patients with AAS and its ability to potentially positively impact patient care.

Images

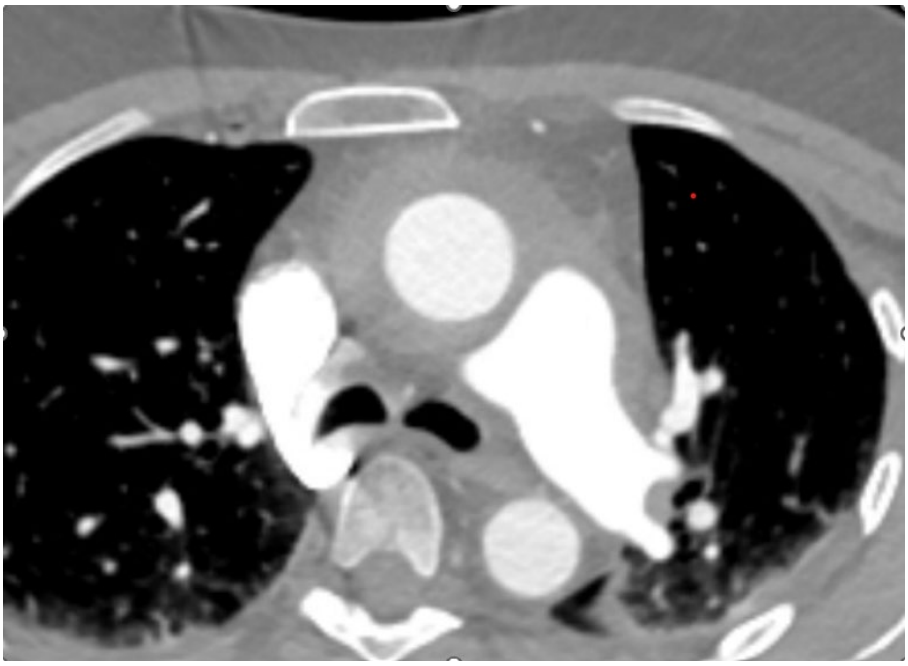


Figure 1. CT Angiography axial image pre-operatively with crescentic ascending aortic wall thickening.

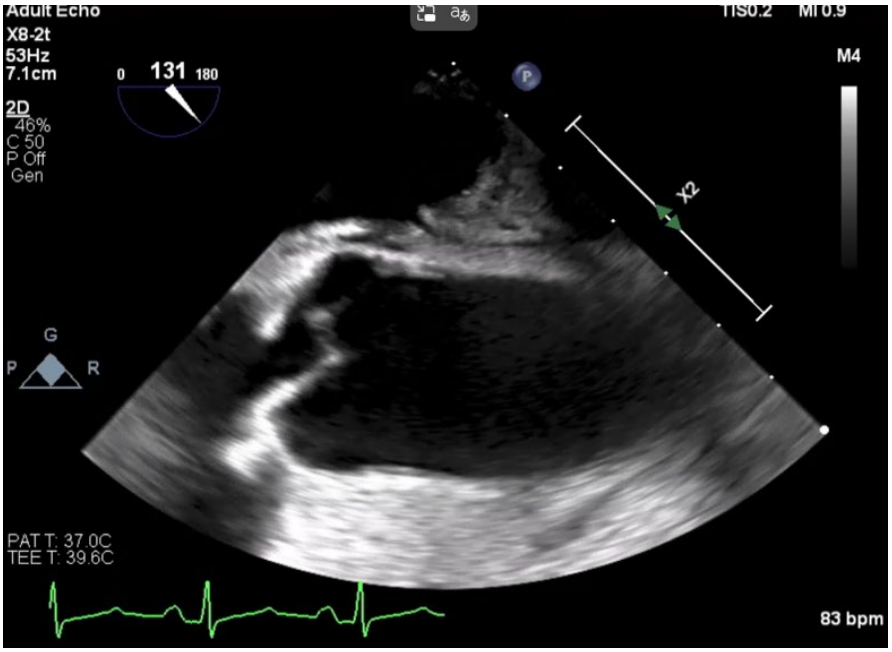


Figure 2. Transesophageal echocardiography (TEE) image performed intraoperatively pre-incision showing no obvious evidence of intramural hematoma (IMH) in the aortic root or proximal ascending aorta.

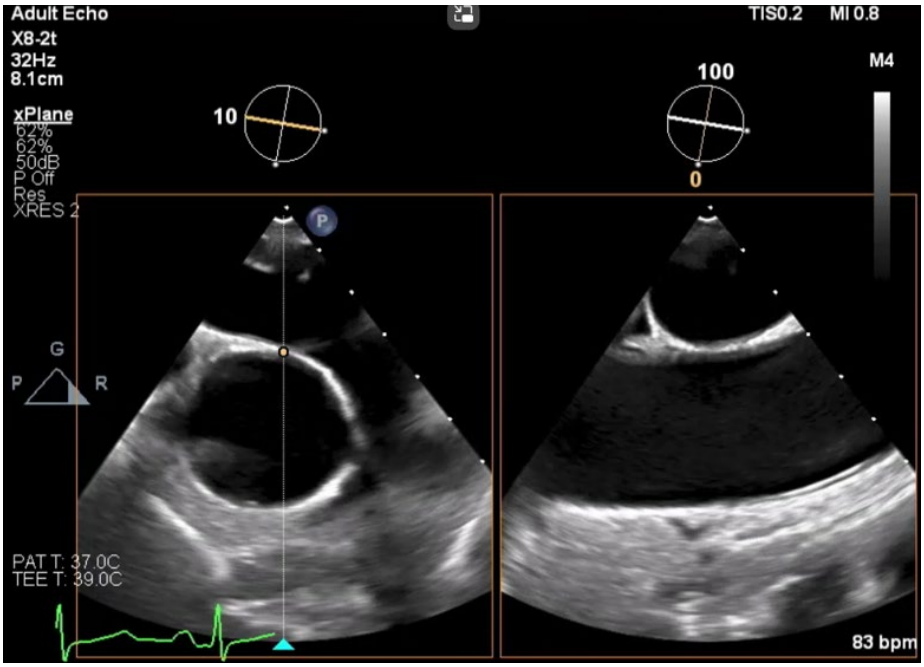


Figure 3. Transesophageal echocardiography (TEE) image performed intraoperatively pre-incision using X-plane showing no obvious evidence of intramural hematoma (IMH) in the ascending aorta.

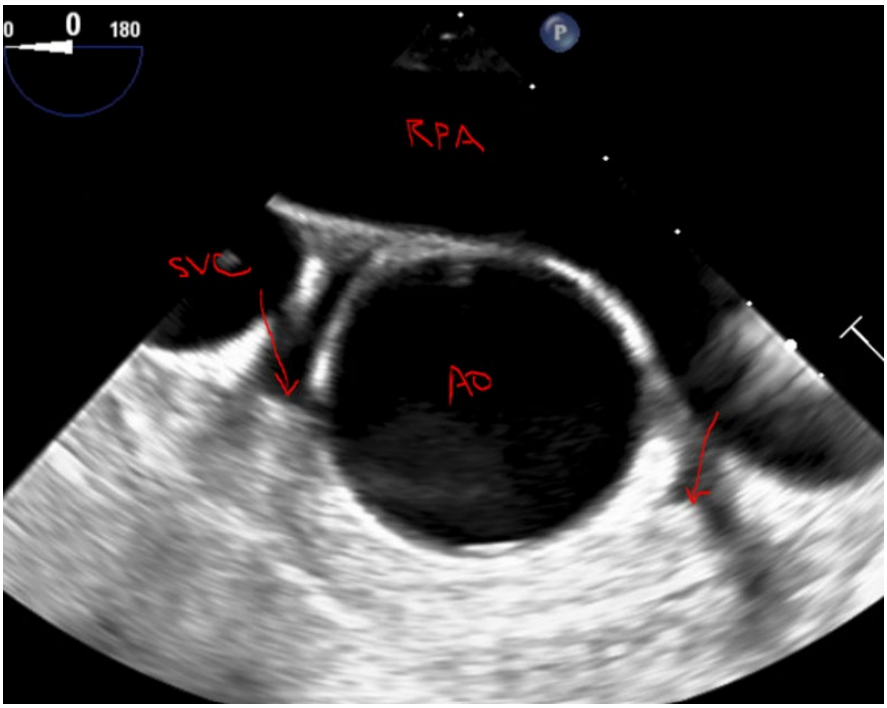


Figure 4. Transesophageal echocardiography (TEE) image with anatomic labels and arrows demonstrating the presence of the superior pericardial recess fluid.

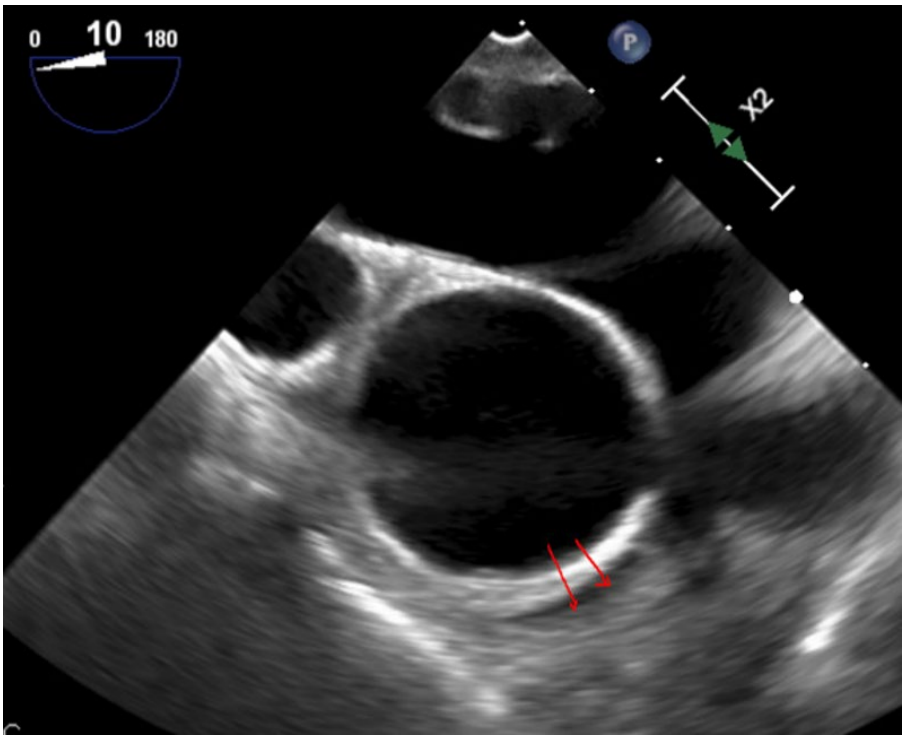


Figure 5. Transesophageal echocardiography (TEE) image in short sequence of prior showing the same superior pericardial recess fluid as distinct from the aortic wall.

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#FR 3 – Kiosk 20 – Poster 6

Baffle Procedure for Partial Anomalous Pulmonary Venous Return in 62-Year-Old Patient Undergoing LVAD Insertion for Acute Decompensated Heart Failure

Presenting Author: Colton Toy, DO, Allegheny General Hospital

Introduction

Partial anomalous pulmonary venous return (PAPVR) is a rare congenital heart defect involving oxygenated blood return from one or more pulmonary veins emptying into the right atrium or systemic venous circulation like the SVC. PAPVR is often associated with a sinus venosus atrial defect. We are presenting a case of a patient with acute decompensated heart failure who was incidentally found to have a PAPVR during preoperative planning for LVAD placement as bridge for future heart transplant.

Case Presentation

A 62 y/o male with history of NICM, chronic systolic HF (EF 15-19%, NYHA IV), moderate MR, CAD, CKD stage 3a, and Hx of Hodgkin's lymphoma s/p ABVD in 2022 now in remission. After being admitted for acute decompensated HF, the patient underwent a RHC that showed elevated RA, PA, and wedge pressures, but normal SvO2 and CI. The following day a TTE bubble study was performed for the discordance between CI and mixed venous saturation, however this showed no intracardiac shunting. Repeat RHC showed improved biventricular filling pressures and high cardiac output when using PA saturation but low CO when using IVC/SVC saturations. There was evidence of a significant left to right shunt at the level of the SVC suggestive of PAPVR, which later review of CT imaging showed emptying into the azygous vein. He was evaluated for advanced therapies given his end-stage NICM w/ NYHA class IV symptoms and an intolerance to medical therapy. Decision for LVAD placement was made as he wasn't a candidate for heart transplant at this time due to his recent hx of Hodgkin's lymphoma in the past 5 years. He underwent implantation of ventricular assist device, Baffle procedure, and L atrial appendage clip on 11/19/2025. Induction of general anesthesia, CBP, and Heartmate III insertion were all uncomplicated. The PAPVR was identified as originating from the RUL and connecting to the SVC posterolaterally, above the level of the cavoatrial junction and below the azygous vein that entered medially just beneath the SVC-innominate vein junction. There was no ASD present, so the foremen ovale was opened and the anomalous drainage was baffled down through the new ASD with a large bovine pericardial patch. The patient was then successfully weaned off of CBP with the aid

of modest inotropic and vasoactive infusions. Post-bypass TEE showed good positioning and function of the LVAD and intact repair of PAPVR with unobstructed flow into the LA across the surgically created ASD. The patient was extubated and remained neurologically intact in the ICU. Postoperative course was complicated by atrial flutter s/p DCCV and ongoing inotropic support for moderate RV dysfunction.

Discussion

There is sparse literature pertaining to concurrent LVAD placement and repair of partial anomalous pulmonary venous return. While it's uncommon to discover PAPVR in adults, it can pose significant difficulties when planning for other cardiac procedures in those patients. No current guidelines or recommendations exist for the management of these patients.

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#FR 3 – Kiosk 21 – Poster 1

Catastrophic Type-A Aortic Dissection During TAVR: Intraoperative Vigilance and Management of Complications—A Case Report

Presenting Author: Jia Wang, Thomas Jefferson University Hospital

Introduction

Transcatheter aortic valve replacement (TAVR) has emerged as an effective therapy for patients with severe aortic stenosis. Significant improvements in device design and imaging techniques have made this minimally invasive procedure relatively safe and effective. However, rare and serious complications can occur during or after valve deployment. Here we present a 76 yo male with severe bioaortic valve stenosis who underwent a TAVR procedure that was complicated by aortic dissection requiring a total aortic arch replacement and thoracic endovascular aortic repair.

Case Presentation

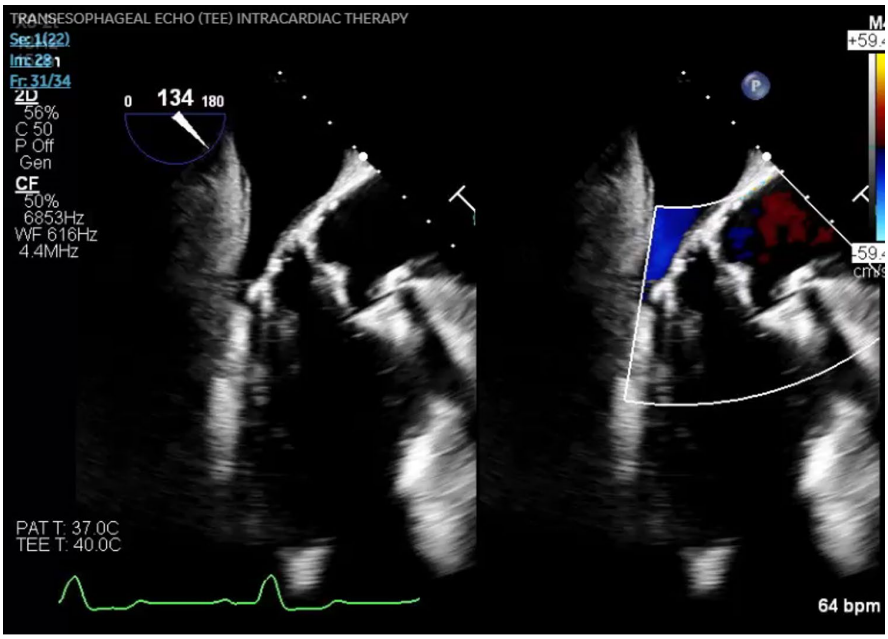
This is a case of a 76 yo with a history of coronary artery disease, hypertension, obstructive sleep apnea, chronic kidney disease and prior bioprosthetic AVR in 2010, complicated by severe aortic stenosis presented for an elective TAVR. Intraoperative TEE showed normal biventricular function with LVEF of 70%. The aortic valve area is 1.0 cm2, peak transaortic valve gradient is 38 mmHg, and a mean vale gradient of 23 mmHg. Under TEE and fluoroscopic guidance, a 26mm Evolut transcatheter aortic valve was positioned across the valve. After deployment, the valve appeared to be well seated, and appropriately positioned, but there was turbulent flow with elevated gradients. Upon further inspection, the valve appeared to be under-expanded. A balloon valvuloplasty was then performed, which was complicated by migration of the valve into the aortic root causing severe aortic insufficiency. The decision was made to utilize a snare to further retract the valve into the ascending aorta. During manipulation, however, a new type-A dissection was observed from the root down into the descending thoracic aorta. The cardiothoracic surgeon then emergently converted to a redo sternotomy with right axillary artery and right atrium cannulation followed by cardiopulmonary bypass. A Thoraflex was utilized to replace the aortic arch along with implantation of a new bioprosthetic aortic valve. After circulatory arrest, however, it was determined that the distal end of the frozen elephant trunk landed in the false lumen. Consequently, a TEVAR procedure ensued with transfemoral placement of graft from the true lumen. The patient was transferred to the cardiac ICU where he successfully followed commands, moved all extremities, and was extubated on POD2.

Discussion

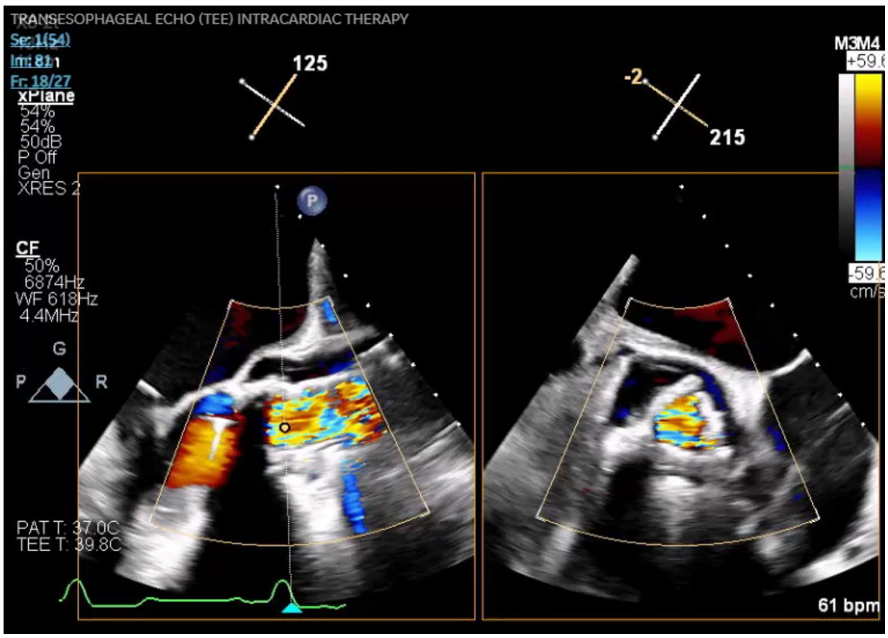
Several complications have been well documented with the TAVR procedure including aortic dissection and device migration [1]. Intraoperative management requires meticulous hemodynamic control to maintain coronary perfusion and avoid arrythmia, myocardial ischemia, or even cardiac arrest. In cases utilizing a Evolut transcatheter valve, it is crucial to confirm the reliability of rapid ventricular pacing as loss of capture during deployment can result in valve migration and catastrophic complications. It is critical that appropriate expansion occurs to avoid high intravalvular gradients, such as above. If an aortic dissection is

detected following valve implantation, as described, a strict Mean arterial pressure (MAP) should be maintained at or below 65 mmHg to prevent further dissection. Here we present the essential need for timely management and close communication for successful treatment of such a catastrophic sequela.

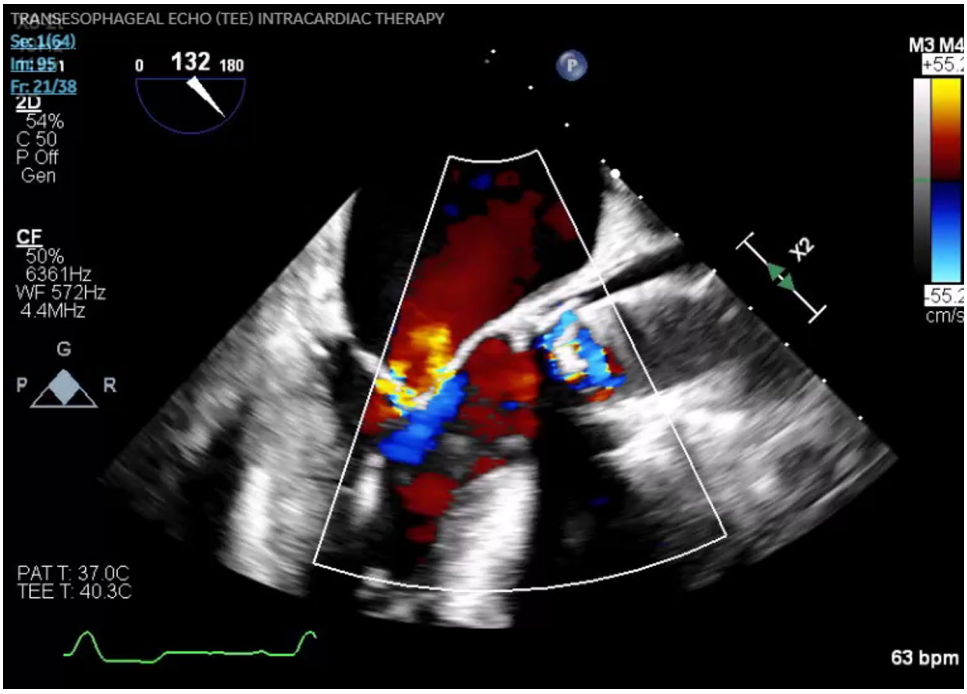
Images



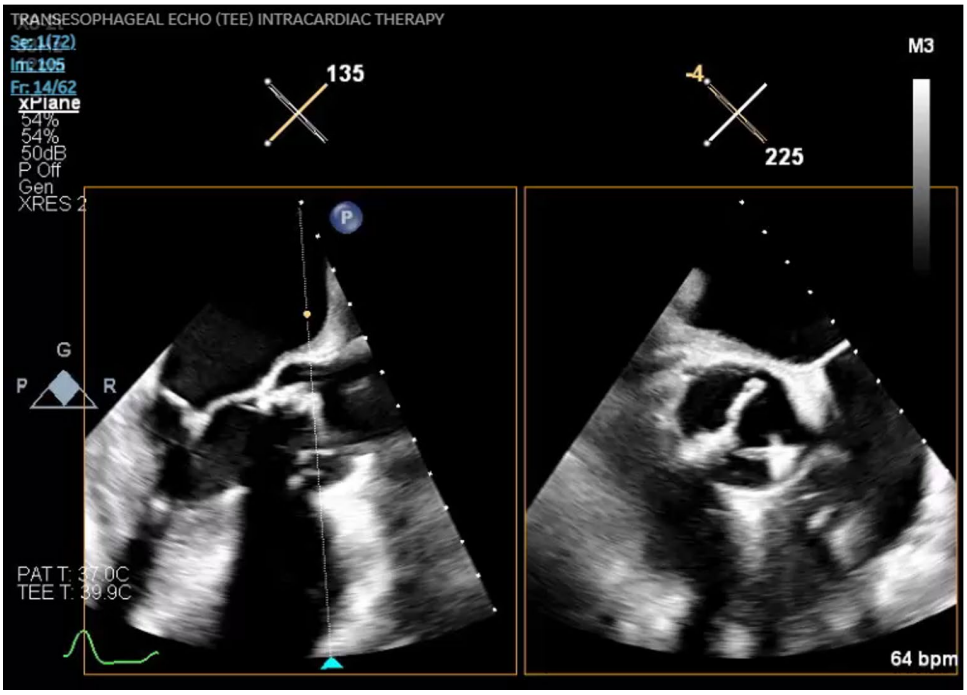
pre-intervention-severe bioprosthetic stenosis



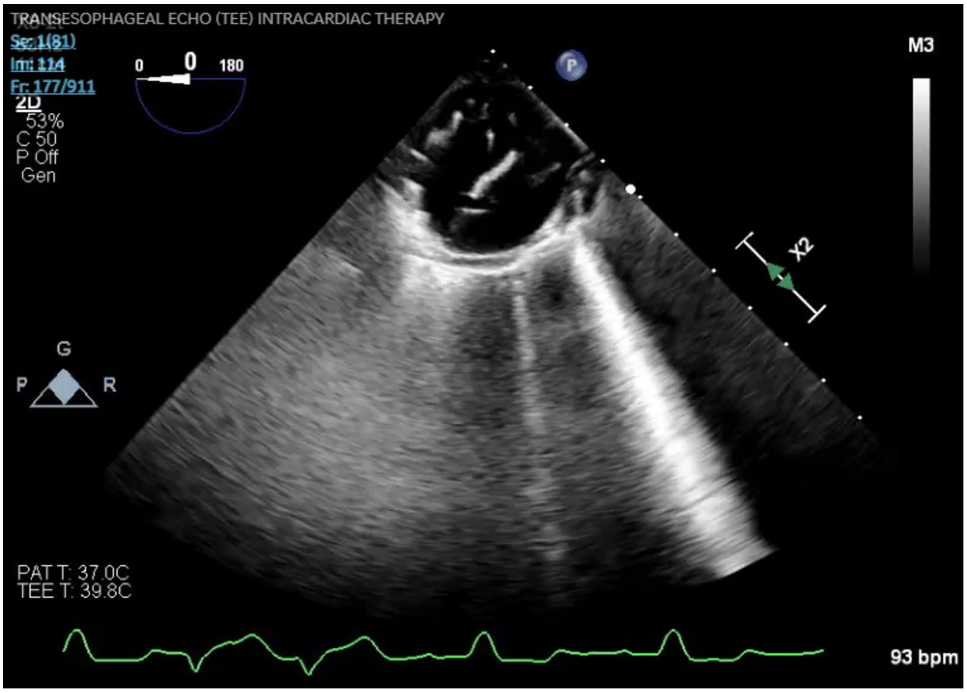
TAVR deployed



post balloon angioplasty severe aortic insufficiency



valve pulled into ascending aorta



aortic dissection

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#FR 3 – Kiosk 21 – Poster 2

The Hunt for a Shunt: A Case Report of a Non-Iatrogenic Acquired Gerbode Defect

Presenting Author: Deen Debryn, Fellow, Emory University

Introduction

The first reports of abnormal LV-RA connection, or Gerbode defect, date back to the 1830s (1). While initial studies focused on the congenital causes of Gerbode defects, we have recently seen an increase in reports of acquired causes, including iatrogenic (surgical trauma), and non-iatrogenic (blunt trauma, infection, etc) (3). This case report will highlight a non-iatrogenic Gerbode defect, and its insidious clinical course following blunt cardiac trauma.

Case Presentation

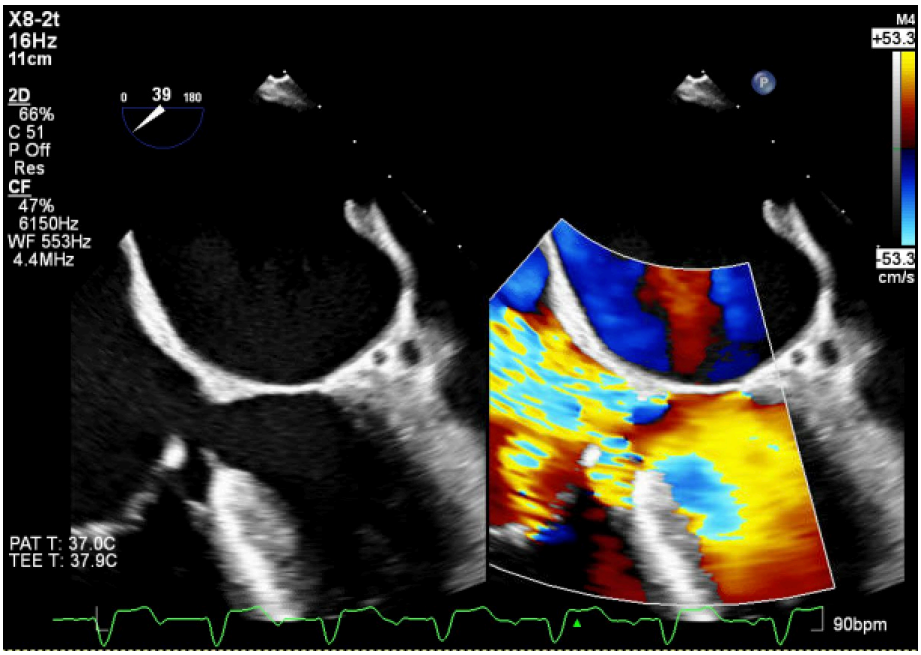
A 58 year old man with PMH of HTN, T2DM, and PSUD (ETOH and cocaine) presented to the ED after an MVC with blunt chest injuries. Initial workup was notable for elevated troponin and new high degree AV block. A TTE was obtained and revealed LVEF 60%, moderate TR, and elevated PA pressures. His heart block required pacemaker placement, however the remainder of his injuries were managed nonoperatively, and he was discharged home after 10 days. Over the next 6 days, he experienced progressive dyspnea, and returned to the ER where CT revealed worsening pleural effusions. Repeat TTE demonstrated worsening TR with continued signs of pHTN. He received a work up for PE, which returned negative. With no clear diagnosis, his cardiology team carefully reexamined prior TTEs and identified a high velocity jet by color flow doppler traveling from the LV to the RA, raising suspicion for a Gerbode defect. Cardiac MRI was obtained and demonstrated a 7mm defect involving the membranous septum at the level of the LVOT with systolic flow from LV into RA. The diagnosis was confirmed and he was scheduled for surgical repair. On the day of surgery, his induction and pre-bypass courses were uncomplicated. Baseline TEE exam confirmed the defect with both supravalvular and infravalvular components (images 1 and 2), suggesting intermediate type Gerbode defect. The right ventricle was noted to be moderately dysfunctional. He went on cardiopulmonary bypass for a total of 121 minutes, and the defect (image 3) was surgically repaired with 2 Gore-Tex patches. Inhaled epoprostinal and IV epinephrine were initiated prior to separation from CPB. TEE confirmed successful closure of the defect, with no residual blood flow across the ventricular septum, and much improved TR. He had an uncomplicated postoperative ICU course, and was medically stable for discharge to subacute rehab 8 days after his surgery.

Discussion

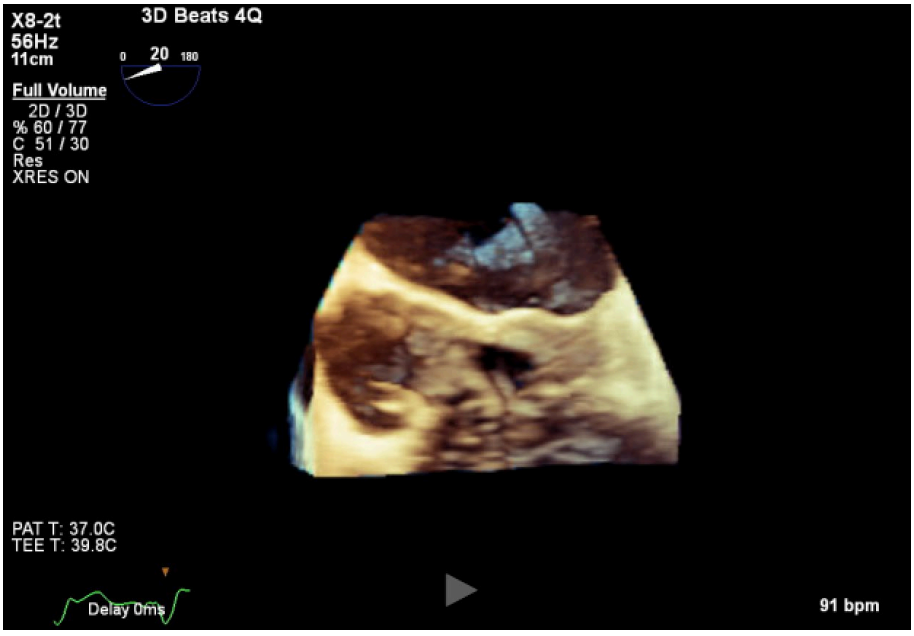
This case details a rare though increasingly reported cardiac pathology: the acquired Gerbode defect. Of note, is the subtle presentation of symptoms which when combined with imperfect imaging tools, resulted in delayed diagnoses and readmission for worsening heart failure. We can expect to see a rise in patients with acquired Gerbode defects, considering the growing cardiac surgical patient population. Notable populations at risk for acquiring this insult include those with infective endocarditis, recent valve operations, and blunt cardiac trauma (as in our case) (3). Transesophageal echocardiography is currently thought of as

the most sensitive modality in diagnosing this defect (3). Proficiency in 3-D TEE will become increasingly important as a diagnostic tool as well as for real time guidance in less invasive percutaneous closure options, as these become more available to repair this defect (2).

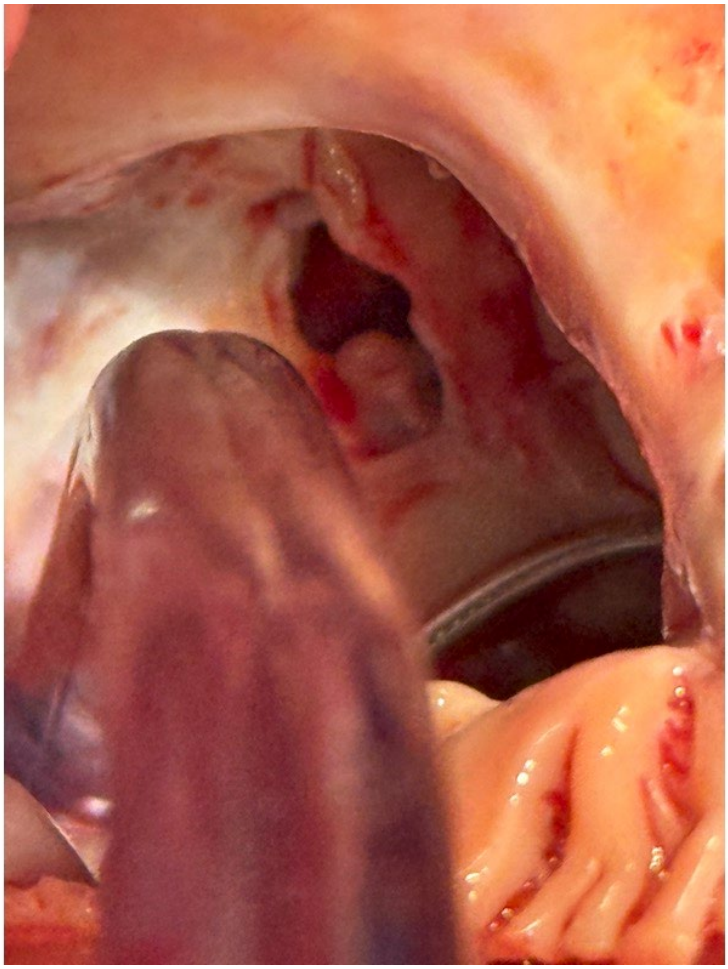
Images



Gerbode Defect 2D with CFD



Gerbode Defect 3D



Gerbode Defect Surgeon's View

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#FR 3 – Kiosk 21 – Poster 3

Transcatheter Mitral Valve-in-Valve Replacement in a Patient with Bioprosthetic Stenosis and Paravalvular Leak: Anesthetic and Intraoperative TEE Considerations

Presenting Author: Pulkit Vats, DO, Resident Physician, UT Health Houston

Introduction

Transcatheter mitral valve-in-valve replacement (TMVIV) has become an important option for high-risk patients with failed bioprosthetic valves[1]. These cases require thoughtful cardiac anesthetic management and comprehensive TEE to assess valve function, guide device trajectory, evaluate residual regurgitation, and detect complications in real time. We present a case of severe bioprosthetic mitral stenosis (MS) with concomitant paravalvular leak (PVL) in a patient who required a transapical approach following a failed transfemoral attempt.

Case Presentation

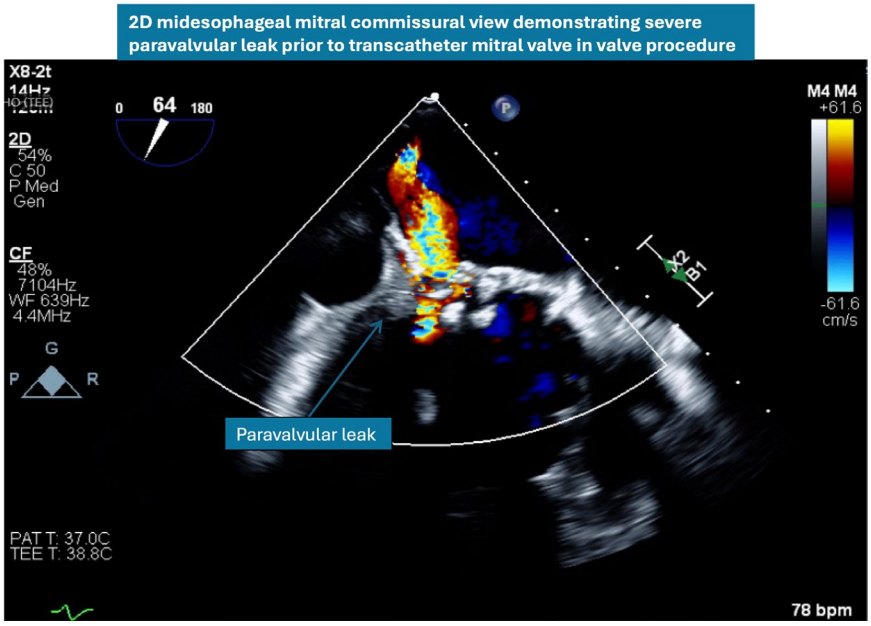
A 78-year-old man with prior mitral valve repair complicated by PVL requiring bioprosthetic valve replacement presented with worsening dyspnea and severe bioprosthetic MS and severe PVL. A transfemoral TMVIV procedure was attempted; however, the case was aborted due to an inability to cross the interatrial septum safely. The patient subsequently underwent a transapical procedure via left mini thoracotomy under general anesthesia. Given the concomitant MS and PVL, intraoperative management focused on maintaining preload and optimizing diastolic filling while avoiding tachycardia and pulmonary congestion. Phenylephrine was utilized as the vasopressor of choice, and fluid administration was guided by TEE. Under fluoroscopic and TEE guidance, a 29-mm pericardial tissue valve was delivered across the prior prosthesis and deployed during rapid ventricular pacing without issue. The pre-existing PVL improved from severe to moderate.

Discussion

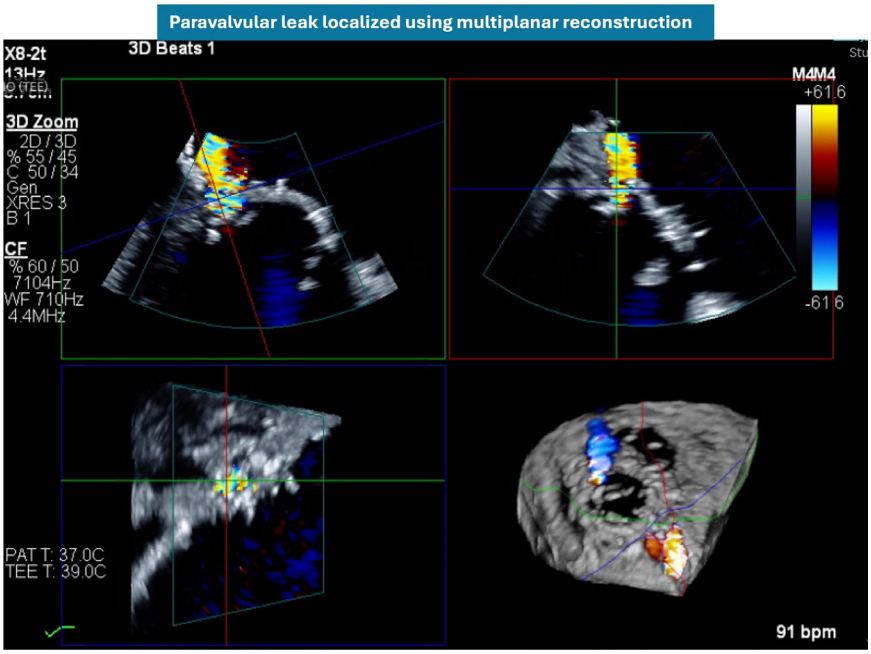
Transfemoral transseptal access remains the preferred route for TMVIV due to shorter recovery time and less myocardial trauma; however, it may fail in the presence of severe peripheral vascular disease, thickened interatrial septum, or when the delivery system cannot be safely aligned across the septum. In such cases, transapical access provides coaxial entry to the mitral annulus but requires a surgical incision and apical puncture, resulting in greater procedural morbidity, including increased risk of bleeding, myocardial injury, and postoperative pain compared to the transfemoral transseptal approach[2]. This patient's inability to traverse the interatrial septum necessitated the transapical approach despite higher procedural risk. The coexistence of severe MS and PVL also presents a complex physiologic burden, combining fixed inflow obstruction with regurgitant volume overload. Intraoperative management thus prioritized adequate ventricular filling and forward flow while minimizing tachycardia and volume overload. Post-deployment assessment confirmed gradient reduction, leaflet mobility, the absence of left ventricular outflow tract obstruction, and improvement in PVL severity. Evaluation of PVL relies on TEE to determine leak location, mechanism, and circumferential extent. 3D TEE further improves localization and

quantification. PVL severity is commonly graded by extent around the sewing ring, with < 10% classified as mild, 10–30% moderate, and >30% severe[3]. In this case, the PVL decreased in severity and improved to moderate after the TMVIV was deployed. This case highlights how procedural access selection, anesthetic management of complex stenotic and regurgitant physiology, and comprehensive TEE can together safely guide TMVIV intervention.

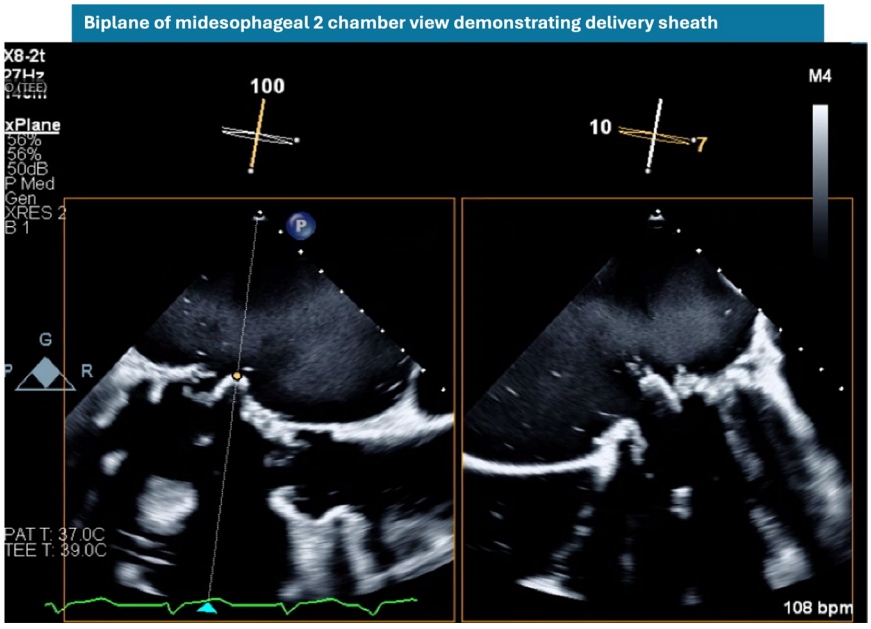
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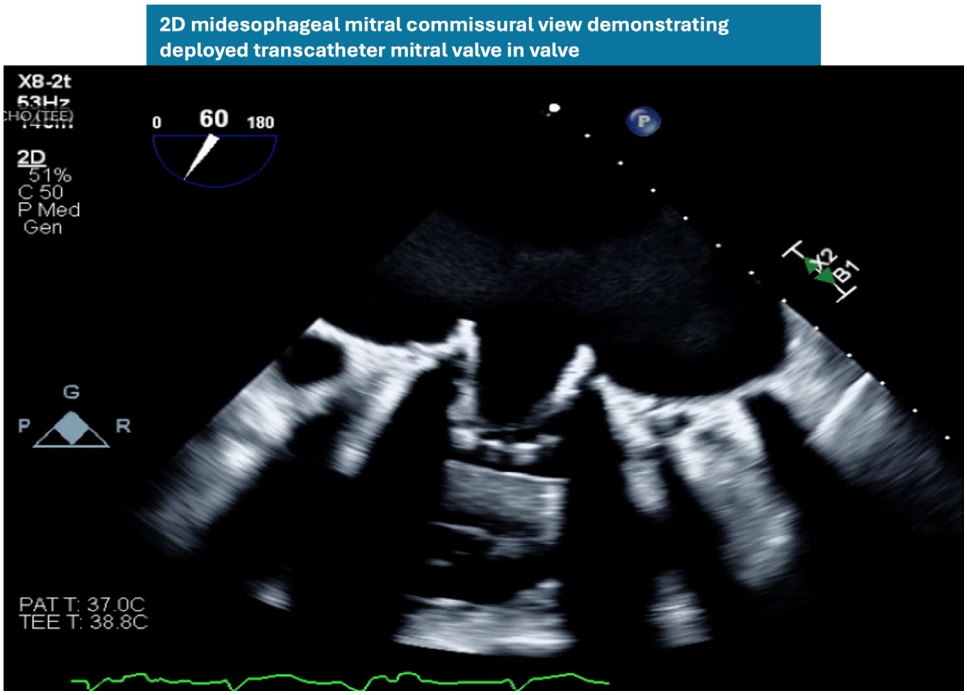
2D midesophageal mitral commissural view demonstrating severe paravalvular leak prior to transcatheter mitral valve in valve procedure



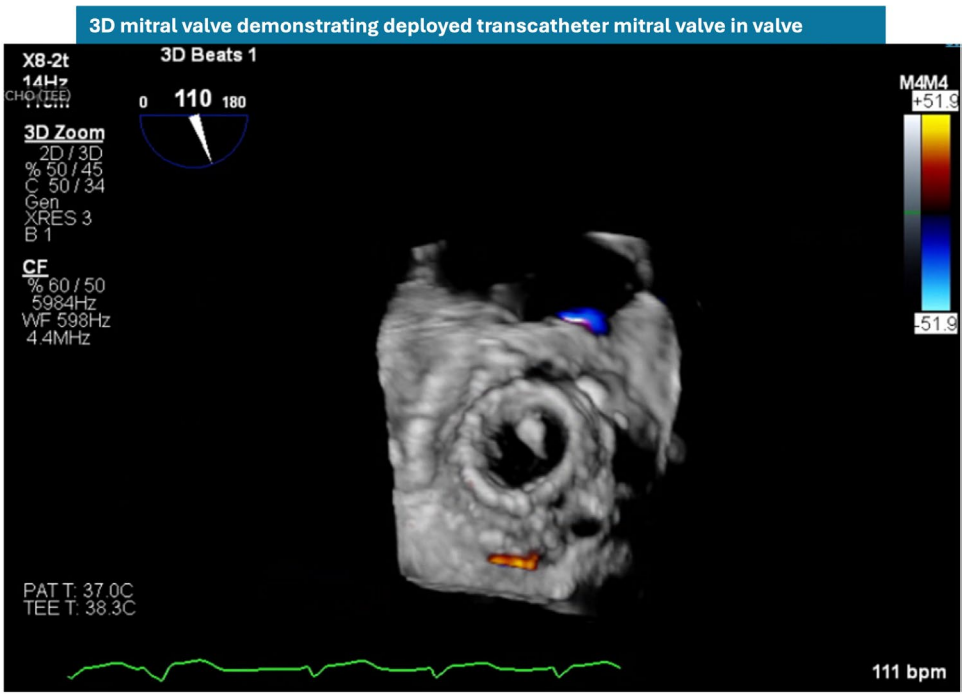
Paravalvular leak localized using multiplanar reconstruction



Biplane of midesophageal 2 chamber view demonstrating delivery sheath



2D midesophageal mitral commissural view demonstrating deployed transcatheter mitral valve in valve



3D mitral valve demonstrating deployed transcatheter mitral valve in valve

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#FR 3 – Kiosk 21 – Poster 4

A Tear-rific Discovery: Unexpected Aortic Valve Perforation After Rapid Deceleration Injury

Presenting Author: Jack A. Grossweiler, M.D., Resident, University of North Carolina Hospitals

Introduction

Rapid deceleration events are well known to cause aortopathy including aortic transection or dissection, but direct injury to the aortic valve from this mechanism is exceedingly rare, with only isolated cases described. The proposed mechanism involves abrupt displacement of the heart within the mediastinum and a sudden rise in intrathoracic pressure during deceleration, generating extreme transvalvular gradients that can tear or perforate valve cusps, particularly when the aortic root is stretched or distorted. Concomitant aortic transection with valve leaflet perforation is exceptionally uncommon. This case illustrates a unique manifestation of deceleration trauma causing direct aortic valve injury and emphasizes the pivotal diagnostic value of intraoperative three-dimensional transesophageal echocardiography (3D TEE).

Case Presentation

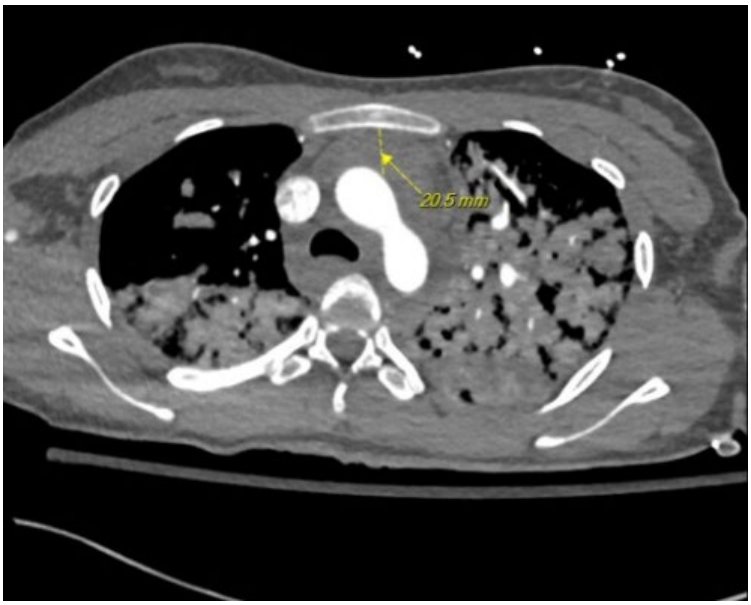
A previously healthy 28-year-old female presented to the emergency department after a high-speed motor vehicle collision. Computed tomography angiography (CTA) revealed a complex pericardial and superior mediastinal fluid collection concerning for vascular injury and possible aortic pseudoaneurysm. Repeat imaging a few hours later demonstrated an enlarging pseudoaneurysm with worsening hemodynamics and the patient was emergently brought to the operating room for an ascending aorta repair (Figure 1). Intraoperative TEE showed normal bi-ventricular function but severe eccentric aortic insufficiency. Further investigation with 3D TEE exhibited a perforation of the non-coronary cusp of the aortic valve; a finding not previously appreciated on preoperative imaging (Figure 2). Upon surgical inspection, the native valve demonstrated a clear perforation in the noncoronary cusp consistent with intraoperative echocardiographic findings (Figure 3). The patient underwent successful bioprosthetic aortic valve replacement and aortoplasty. Post-bypass TEE showed normal ventricular function and a well functioning bioprosthetic aortic valve with no paravalvular leak. She was weaned from cardiopulmonary bypass without difficulty and recovered well postoperatively.

Discussion

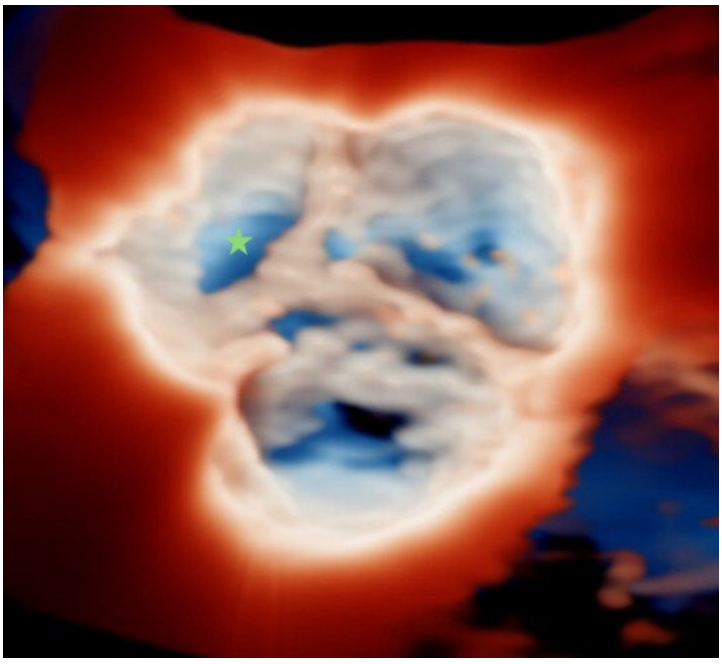
This case represents a rare example of aortic valve injury secondary to blunt deceleration trauma. While aortic transection is a known sequela of rapid deceleration, concurrent valve leaflet perforation has been described only rarely. The mechanism likely involves shear and traction forces transmitted through the aortic root and valve apparatus, resulting in tearing of the delicate valvular tissue. In cases of aortic injury requiring emergent surgery, intraoperative TEE can be crucial for identifying additional pathology and guiding surgical management. Three dimensional TEE is an excellent modality to better characterize causes of aortic regurgitation as it can demonstrate valve size, morphology, and function. In this case, it allowed for precise localization and characterization of the cusp perforation, distinguishing it from other causes of acute aortic insufficiency. Overall, this case underscores the need for a high index of suspicion for valvular injury in

deceleration-related thoracic trauma and demonstrates how advanced echocardiographic imaging can decisively alter intraoperative management.

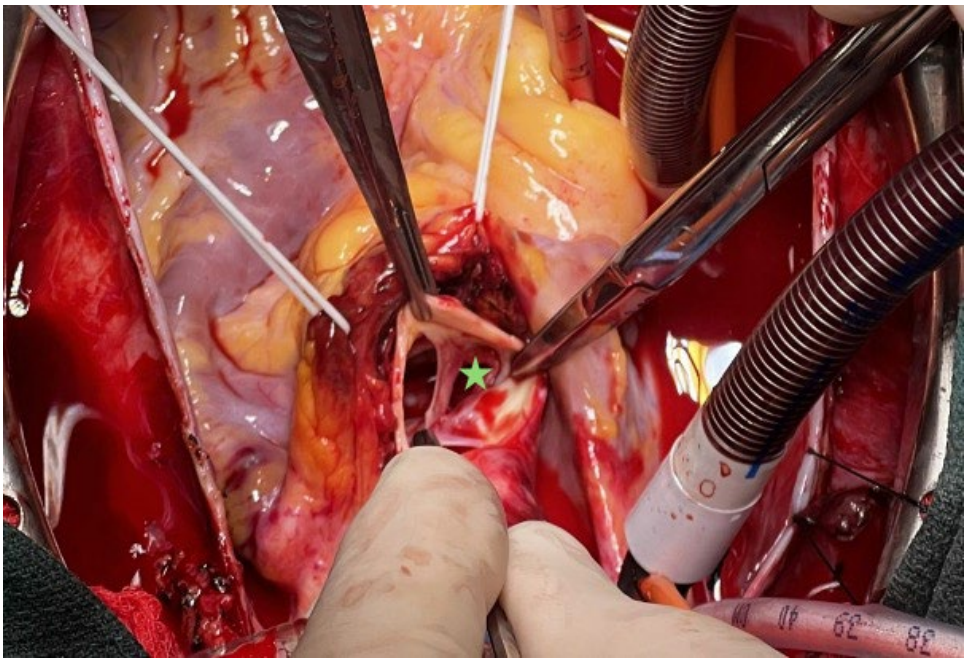
Images



Computed tomography angiography demonstrating an enlarging pseudoaneurysm and the patient was emergently brought to the operating room for an ascending aorta repair due to worsening hemodynamics.



Intraoperative three-dimensional transesophageal echocardiography demonstrating perforation of the non-coronary cusp of the aortic valve, indicated by the green star.



Native aortic valve demonstrating a distinct perforation in the noncoronary cusp, indicated by the green star.

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#FR 3 – Kiosk 21 – Poster 5

A Simple Pediatric Facemask/Tent Assembly Provided Nasal CPAP Oxygenation and Immediate Assisted Nasal Ventilation in a Morbidly Obese Patient with COVID-19 Pneumonia and A-Fib during TEE Attempt

Presenting Author: Pankti P. Acharya, DO, MPH, Robert Wood Johnson University Hospital

Introduction

Patients often receive IV sedation and oxygenation via a nasal cannula (NC) during MAC. Over-sedation and/or airway obstruction may cause severe desaturation. A pediatric face mask assembly has been shown to maintain spontaneous nasal continuous positive airway pressure (CPAP) ventilation and improve oxygen delivery in sedated OSA patients in the OR and NORA.[1-2] A combined nasal mask-face tent provided pre/apneic nasal oxygenation and reduced droplet spread during rapid sequence induction of general anesthesia, intubation and extubation in COVID-19 patients (Fig. 1).[1,3] We used this technique in a morbidly obese patient with OSA during TEE in the Echo Lab.

Case Presentation

A 35-y/o male, BMI 61.55 kg/m^2, with HTN, stage 5 CKD, anemia, bilateral pneumonia from COVID-19, acute hypoxic respiratory failure, and A-Fib, presented for TEE/possible cardioversion. An infant face mask (size #2) with fully-inflated air cushion was secured over his nose with elastic head-straps to obtain a tight seal. A bandage and silk tape was placed over his nasal bridge to reduce pressure on his nose. A face tent (clear plastic sheet) covered his mouth that was kept open by a bite block to reduce droplet spread. The nasal mask-face tent was connected to the anesthesia machine via a breathing circuit. The adjustable pressure-limiting valve was adjusted to deliver 8-10 cm H_2O CPAP with fresh O_2 flow of 4 L/min. A NC with air sampling tubing was taped on his lower lip to continuously monitor exhaled CO_2 from his mouth and evacuate oral droplets. Following nasal CPAP pre-oxygenation, his SpO_2 increased from 96% (with NC O_2 4 L/min) to 100%. After he was pre-treated with glycopyrrolate (0.2 mg) and oral viscous lidocaine and benzocaine spray, sedation was titrated with lidocaine (100mg), propofol boluses (90 mg total) and propofol infusion (75 mcg/kg/min). The TEE probe was inserted smoothly under the face tent and he maintained spontaneous ventilation and 100% SpO_2 (Fig. 2-3). However, after TEE probe was successfully advanced, he vomited clear fluid. Oral suctioning immediately initiated while nasal mask delivered CPAP oxygenation. Simultaneously, TEE probe was withdrawn, he was put into Trendelenburg position with head tilted. His mouth was suctioned clear of fluid (30 cc). (Fig. 4). However, his SpO_2 continued to decrease to 50% (Fig.3). Assisted nasal ventilation was performed with his mouth closed under the face tent and bilateral jaw thrust until he regained spontaneous ventilation. He maintained 98-100% SpO_2 with nasal CPAP mask–face tent (Fig. 3). Portable X-ray showed no evidence of aspiration. TEE was canceled and CT scan was requested by the cardiologist to rule out atrial thrombosis prior to cardioversion the next day. Patient was awake and alert and had no complaints. Next day, he spontaneously converted to NSR and maintained 97-98% SpO_2 on room air (Fig. 5).

Discussion

Besides providing spontaneous nasal CPAP ventilation, this nasal mask-face tent provided continuous oxygenation during oral suctioning and assisted nasal ventilation in a morbidly obese patient with OSA during TEE attempt. It also reduced aerosol/droplet spread during the aerosol generating procedure, which may improve patient safety and provider protection at no extra costs.

Images



Fig. 1. A nasal mask-face tent was secured over the nose with elastic head-straps and connected to a breathing circuit.



Fig. 2. The TEE probe was inserted smoothly under the face tent.

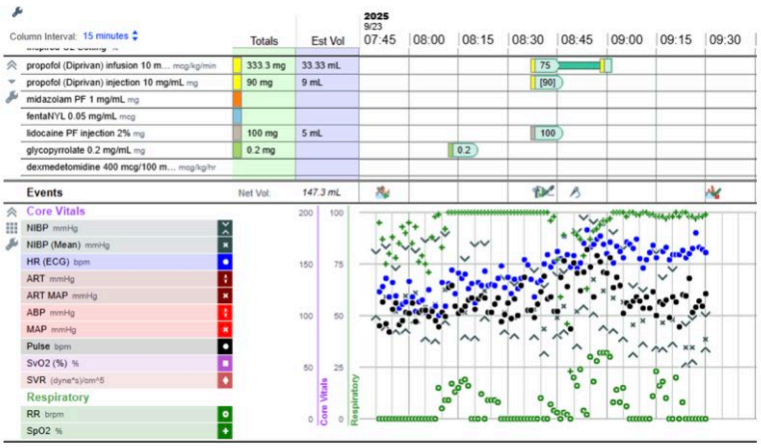


Fig. 3. The patient maintained spontaneous ventilation and 99-100% SpO_2 with the nasal mask-face tent after insertion of TEE probe.

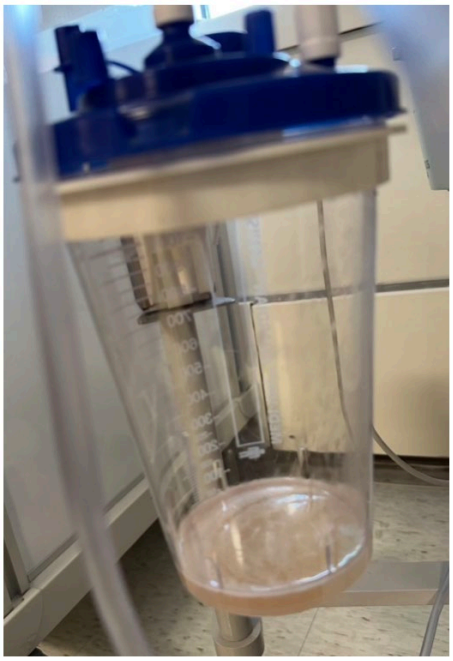


Fig. 4. Suctioned 30 cc clear fluid.

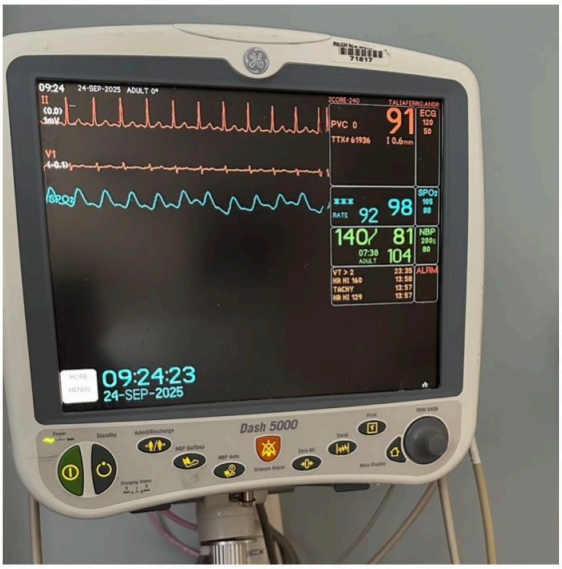


Fig. 5. Post process day One, the patient spontaneously converted to NSR and maintained 98% SpO_2 with room air.

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ASA virtual AM: (MC1280), 2020

#FR 3 – Kiosk 21 – Poster 6

Anesthetic management of novel endovascular therapy for ascending aorta pathology

Presenting Author: Lauren M. Nahouraii, MD, Emory University

Introduction

Ascending aorta and arch acute aortic syndromes have historically required open repair with cardiopulmonary bypass and is associated with significant morbidity and mortality. Novel aortic endovascular procedures have enabled minimally invasive treatment of proximal aortic disease for patients previously considered unsuitable for surgery. These novel endovascular techniques pose significant anesthetic challenges due to proximity to supra-aortic vessels, risk of cerebral malperfusion, and high hemodynamic vulnerability.

Case Presentation

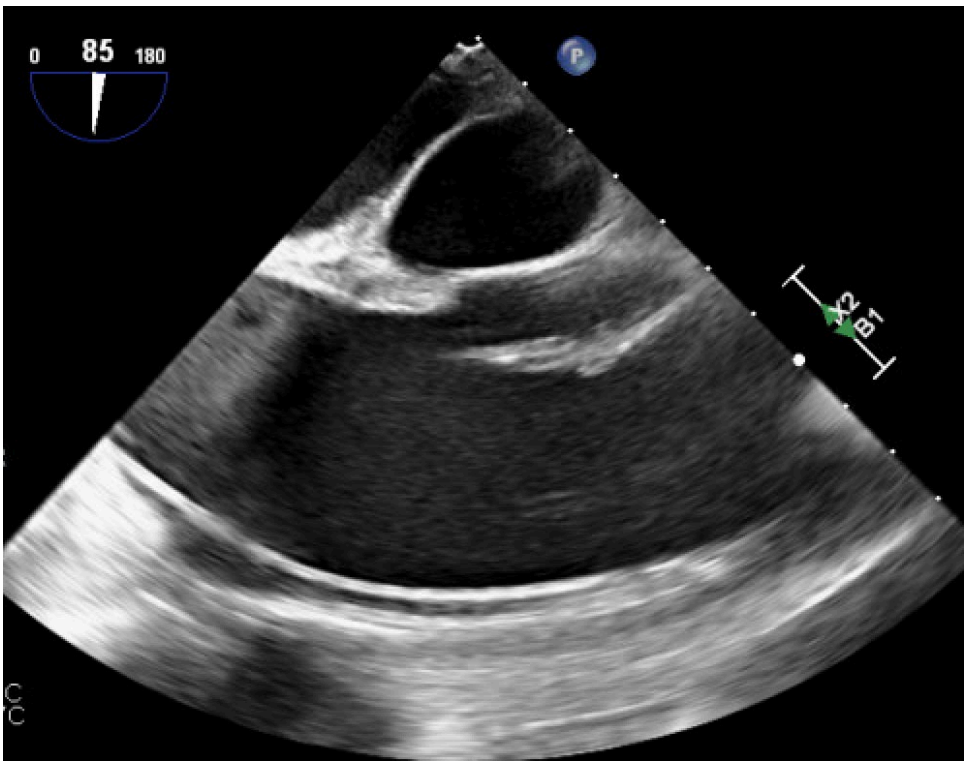
An elderly, frail patient presented with shortness of breath and was admitted for chronic obstructive pulmonary disease (COPD) exacerbation. Computed tomography angiography chest revealed an ascending aorta penetrating atherosclerotic ulcer and intramural hematoma. Because of age, comorbidities and goals of care, a staged, endovascular debranching approach was pursued. She first underwent left carotid to subclavian bypass and stenting with vascular surgery. Four days later, she returned for ascending and arch stent grafting with a single branched arch device placed into the brachiocephalic artery. Continuous blood pressure monitoring via an arterial catheter was essential given planned rapid ventricular pacing and controlled hypotension, frequent activated clotting times, arterial blood gases, and potential for catastrophic complications. A right radial arterial catheter was placed under ultrasound guidance for detection of cerebral ischemia or malposition affecting brachiocephalic artery flow. Given the need for patient immobility and the patient's COPD exacerbation, general endotracheal anesthesia with neuromuscular blockade was utilized. Due to the potential for acute aortic rupture or dissection during graft manipulation or vascular injury as well as baseline patient anemia, large bore central venous access was obtained and blood products were available. Having an additional site for blood pressure monitoring was important given bilateral brachial artery surgical access intermittently causing inaccurate readings. Therefore, our surgical colleagues placed a right femoral arterial line. Continuous electroencephalogram (EEG) and somatosensory evoked potentials were performed to monitor cerebral perfusion and monitor for embolic events. Temporary transvenous pacemaker was placed via the right common femoral vein and used

for rapid ventricular pacing during the deployment of the aortic stent graft. Transesophageal echocardiography (TEE) was utilized to guide positioning of the transvenous pacing wires into the right ventricular apex and aortic wire placement in the true lumen, and eventually across the aortic valve. Further, TEE was useful to evaluate the aortic valve, coronary ostial flow, and cardiac function after stent deployment. She tolerated the staged procedures well; however, her postoperative course was complicated by left brachial artery dissection and thrombosis, and necrotic bowel.

Discussion

Successful anesthetic management of novel ascending aorta and arch endovascular therapies relies on meticulous preoperative planning, advanced hemodynamic monitoring, and proactive management of cerebral and end-organ perfusion. A structured, team-based approach in the operating room is essential.

Images



Ascending aorta

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#FR 3 – Kiosk 21 – Poster 7

Too Good to Be True: The Normal TEE That Wasn't

Presenting Author: Monique J. Cheng, MD, ACTA Fellow, Johns Hopkins Hospital

Introduction

Left circumflex artery (LCx) injury is a serious, though uncommon, complication of mitral valve replacement (MVR) due to its close anatomic relationship to the posterior mitral annulus. Intraoperative transesophageal echocardiography (TEE) is central to valve surgery by further characterizing valve pathology and providing real-time guidance to hemodynamic management. However, heavy reliance on TEE can overshadow important information from other monitors. This case highlights the diagnostic value of the electrocardiogram (ECG) when its findings are discordant with reassuring TEE findings, emphasizing the anesthesiologist's responsibility to integrate multimodal data and appropriately escalate care.

Case Presentation

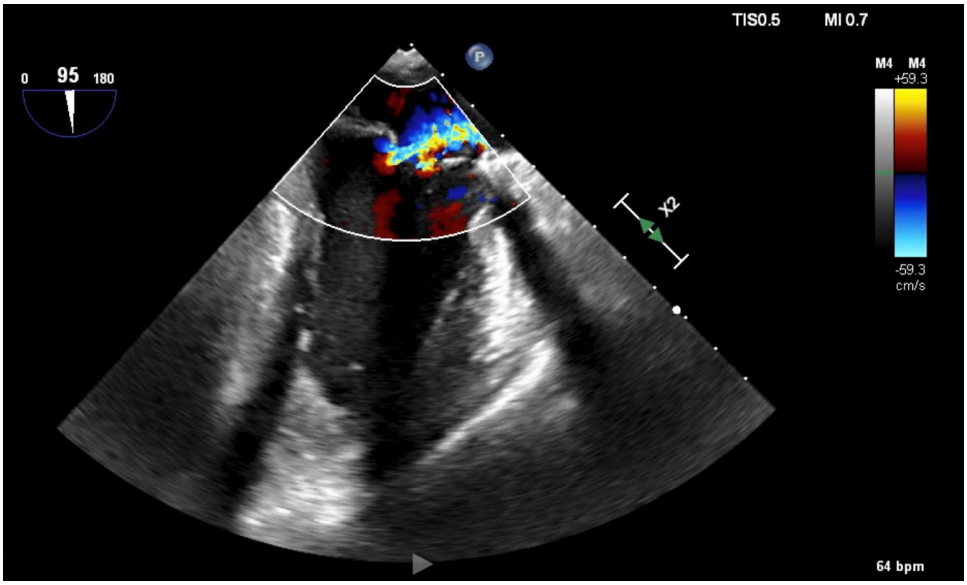
A 64-year-old man with atrial fibrillation and prior mitral valve repair presented for mitral valve replacement, maze procedure, and left atrial appendage occlusion in the setting of recurrent, symptomatic severe mitral valve regurgitation. The planned procedures were executed without issue, and he was weaned from cardiopulmonary bypass uneventfully. Hemodynamics remained stable on minimal vasopressor support, and TEE demonstrated a well-seated prosthesis with normal biventricular function and no new regional wall motion abnormalities. However, immediately after separation from bypass, the ECG showed diffuse ST-segment elevations across all leads, that persisted throughout closure. The anesthesia team communicated these incongruent findings to the surgeon and initiated a multidisciplinary conversation including interventional cardiology regarding the concern for possible myocardial ischemia. A formal 12-lead ECG was performed and continued to show abnormalities consistent with lateral ischemia, while TEE remained

grossly normal and hemodynamics were stable without escalating vasopressor support. Despite an otherwise reassuring intraoperative assessment, the team elected to transfer the patient emergently to the catheterization lab, where coronary angiography revealed acute LCx occlusion, likely from intra-operative suture technique. The patient returned to the operating room for coronary artery bypass grafting to bypass the LCx occlusion and was successfully discharged one week later.

Discussion

This case emphasizes the need for anesthesiologists to continually integrate multiple monitors with the evolving clinical picture, since reassurance from a single modality—such as TEE—cannot outweigh concerning changes elsewhere. TEE is often the dominant monitor in cardiac surgery, and it is known that wall motion abnormalities typically precede and highlight ECG changes1. However, in this case, reassuring TEE – even in the setting of complete occlusion of the LCx – may have delayed recognition of this patient's coronary compromise and ultimate decompensation. Detection of global ST elevations prompted urgent evaluation, underscoring the essential role of ECG in cardiac surgery2. Additionally, this case highlights a unique anatomic hazard during MVR: the proximity of the LCx to the posterior mitral annulus. Awareness of this risk, combined with vigilance for ischemic changes, should guide timely recognition and management of potentially life-threatening complications.

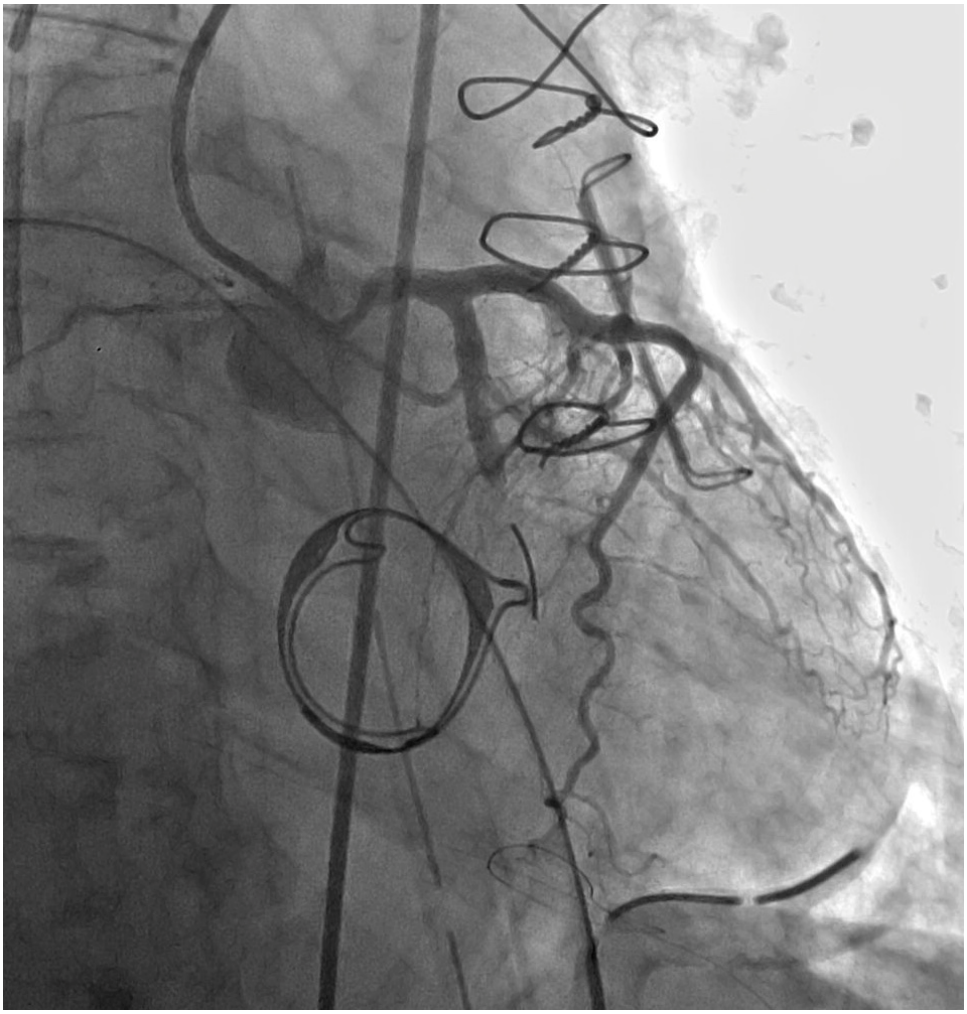
Images



Baseline intraoperative TEE demonstrating severe mitral regurgitation with an eccentric jet.



Immediate post-CPB EKG demonstrating new ST-segment elevations.



Coronary angiogram demonstrating complete occlusion of the left circumflex artery.

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#FR 3 – Kiosk 22 – Poster 1

Aortic Root–RV Fistula on V-A ECMO: A Mechanical Support Challenge

Presenting Author: Vishal Patel, MD, Anesthesiology Resident, University of Michigan

Introduction

Mechanical circulatory support (MCS) for refractory cardiogenic shock, such as venoarterial (V-A) ECMO, intra-aortic balloon pumps (IABP), and Impellas, rely on pressurization of the aorta (Ao) to improve visceral and myocardial perfusion. In the case of a right ventricle (RV) and Ao communication, MCS may increase left-to-right shunt flow into the RV, precipitating RV failure. We present a case of a patient on V-A ECMO with an Ao root-RV fistula with considerations for imaging and management.

Case Presentation

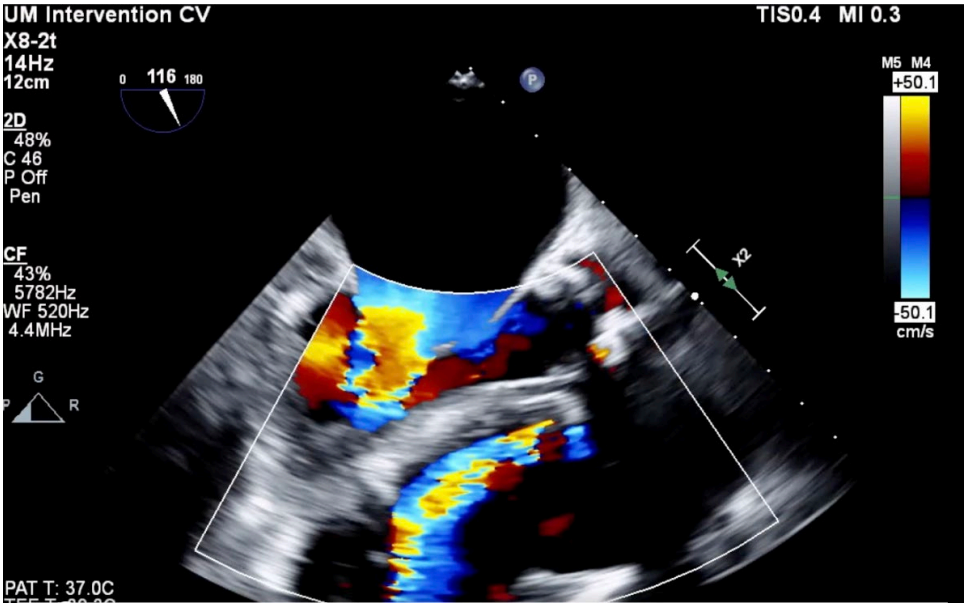
The patient is a 48 year-old male who presented with Staphylococcus aureus bacteremia and acute aortic and tricuspid insufficiency due to endocarditis. He developed shock and underwent emergent aortic valve (AV) and tricuspid valve (TV) replacement with ventricular septal defect (VSD) repair. Transesophageal echocardiography (TEE) described an Ao root abscess with perforation into the interventricular septum and biventricular dysfunction. Unable to separate from cardiopulmonary bypass, peripheral V-A ECMO cannulas and an intra-aortic balloon pump (IABP) were placed. His IABP was dislodged prior to transfer to our institution. On arrival, he was taken for a mediastinal washout. TEE demonstrated severe RV dysfunction, left ventricular (LV) hypokinesis, Ao root-RV fistula, and an immobile TV bioprosthesis. A pulmonary arterial (PA) vent was placed as a drainage cannula for RV unloading. The improved drainage increased V-A ECMO return, augmenting flow through the fistula. The next day, he was taken for AV explant, AV replacement, fistula repair with pericardial patch, and TV prosthesis debridement. Post-operatively, he remained on V-A ECMO with a PA vent and was then transitioned to a surgical RV assist device (RVAD) using the PA vent as a return. This was weaned and he was discharged with improved RV function.

Discussion

Ao-RV fistulas complicate MCS, as it is difficult to pressurize the Ao without precipitating RV failure. Conversely, MCS is highly beneficial in the setting of VSD. Differentiation of shunts into the RV using TEE

guidance is paramount. The pressure differential between the Ao and RV is greatest during diastole, creating diastolic predominance of flow on color flow and continuous wave Doppler with an Ao-RV fistula. Conversely, VSDs have systolic predominance as the pressure gradient between RV and LV is greatest in systole. For Ao-RV fistulas, MCS promotes left-to-right flow, causing RV failure. This is likely why the patient's TV prosthesis thrombosed, as ECMO flows raised RV pressures and restricted valve opening. Unloading strategies for ventricular distention in peripheral V-A ECMO confer little benefit in the setting of an Ao-RV fistula, as they reduce LV filling pressures by diverting blood into the Ao, increasing fistula flow into the RV. In this case, PA venting generated greater V-A ECMO flow and increased flow into the RV. RV dysfunction was unchanged until definitive repair, when the PA vent could be utilized to offload the RV while the patient remained on V-A ECMO. This case demonstrates the importance of recognizing the origin of shunts into the RV with TEE guidance, the physiologic consequences of MCS use in the setting of an Ao-RV fistula, and the need for quick, definitive repair to optimize outcomes.

Images



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#FR 3 – Kiosk 22 – Poster 2

Flash Pulmonary Edema after Caesarean Delivery in a Patient with Severe Mitral Stenosis

Presenting Author: Timothy Miller, MD, Resident (Anesthesiology PGY-1), University of Louisville

Introduction

Severe mitral stenosis poses a substantial perioperative risk during pregnancy due to limited ability to accommodate rapid hemodynamic changes. Following delivery, uterine autotransfusion can increase intravascular volume by up to 500mL, potentially precipitating acute decompensation in patients with fixed cardiac output[1]. We present a case of flash pulmonary edema triggered by uterine autotransfusion immediately following cesarean delivery in a patient with severe bioprosthetic mitral valve stenosis.

Case Presentation

A 33-year-old female with bioprosthetic mitral valve replacement seven years prior for endocarditis presented for scheduled cesarean delivery at 32 weeks gestation. The patient had been hospitalized since she was 29 weeks pregnant due to worsening chest pain and shortness of breath found to be secondary to her known mitral stenosis. Preoperative echocardiography demonstrated severe mitral stenosis with mean gradient 20.7mmHg (Figure 1). Preoperative heart rate was 100-120bpm with systolic blood pressure in the 90s. She received midazolam 1mg and esmolol 10mg for anxiolysis and rate control. An L4-5 epidural catheter was placed preoperatively. Invasive arterial and central venous monitoring were established. A cardiac anesthesiologist with transesophageal echocardiography capability was present. Epidural level was slowly titrated to T6 with 1% lidocaine, maintaining stable hemodynamics. Norepinephrine and vasopressin infusions supported blood pressure. The baby was delivered successfully. Within minutes of delivery, oxygen saturation declined to 75% despite 6L nasal cannula, and vasopressor requirements increased. Two units of packed red blood cells were transfused for presumed hypovolemic shock without improvement. The patient became increasingly distressed with persistent hypoxemia. Emergent endotracheal intubation was performed; arterial blood gas showed P_aO_2 52mmHg on 100% F_iO_2. The patient was placed in semi-upright positioning and transferred to intensive care. Thirty minutes later, oxygenation improved dramatically with P_aO_2 over 300mmHg, and she was successfully extubated the same day. She underwent mechanical mitral valve replacement one month postpartum and survived.

Discussion

This patient's sudden decompensation likely resulted from flash pulmonary edema secondary to uterine autotransfusion. Following delivery, uterine contraction autotransfuses approximately 300-500mL into the maternal circulation. In severe mitral stenosis, the fixed mitral valve area limits left ventricular filling, causing acute left atrial pressure elevation and rapid pulmonary capillary pressure increase. The dramatic improvement with upright positioning and lack of response to blood transfusion support this mechanism rather than hemorrhagic shock. Initial attribution to hypovolemia led to packed red blood cell transfusion, potentially exacerbating pulmonary edema. Multidisciplinary planning with invasive monitoring, controlled neuraxial anesthesia, and preparedness for acute volume-related decompensation are essential in

situations like this. Clinicians must recognize autotransfusion-mediated pulmonary edema early to avoid inappropriate transfusion and implement appropriate interventions including upright positioning and diuresis.

Images

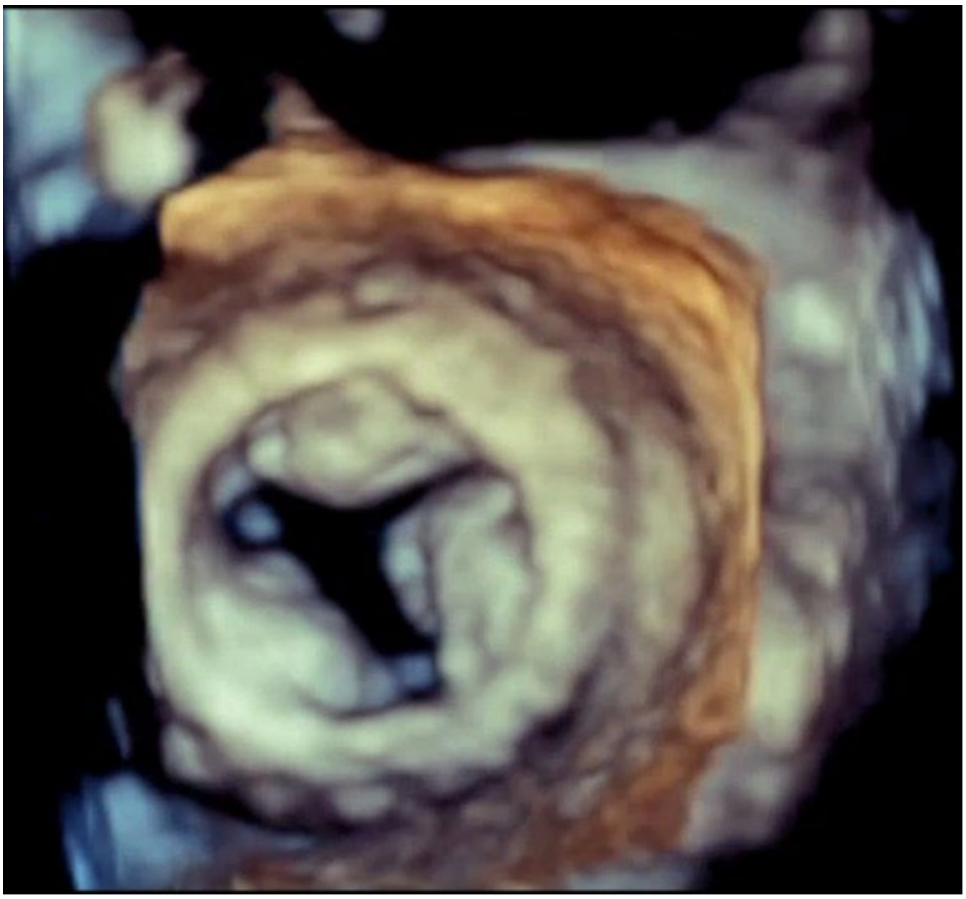


Figure 1. 3D transesophageal echocardiography demonstrated severe stenosis of a bioprosthetic mitral valve during diastole in this pregnant patient.

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#FR 3 – Kiosk 22 – Poster 3

An Unusual Cause of Immediate Post-Bypass Mechanical Aortic Valve Regurgitation

Presenting Author: Sruthi Rangu, MD, Fellow, University of Kentucky

Introduction

Management of prosthetic aortic valve regurgitation detected immediately after cardiopulmonary bypass (CPB) is challenging. In addition to assessing the severity of regurgitation in real time, identifying the precise mechanism is essential, as some causes require an immediate return to CPB. The patient's ability to tolerate another bypass run and the surgeon's experience must also be considered. We report an unusual cause of severe prosthetic aortic regurgitation immediately post-bypass and how it was successfully managed in a patient undergoing aortic root reconstruction with valve replacement (AVR) secondary to ascending aortic aneurysm.

Case Presentation

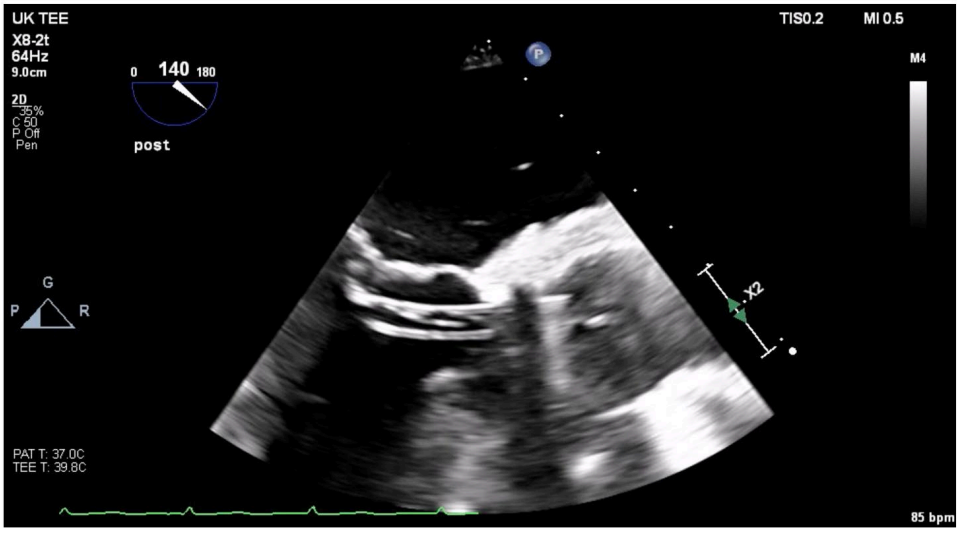
A 62-year-old man with severe aortic regurgitation (AR) and an ascending aortic aneurysm (4.9 cm at the sino-tubular junction and 5.1 cm at the mid-ascending aorta), along with coronary artery disease, hypertension, and diabetes, underwent aortic root reconstruction with a 23-mm St. Jude valved conduit and coronary reimplantation, plus coronary bypass grafting under CPB. For surgical exposure and LV decompression, a left ventricular (LV) vent was inserted through the right upper pulmonary vein. The first attempt to separate from CPB failed due to significant innominate artery bleeding, requiring a return to bypass. After the repair, a second attempt succeeded. However, immediately after separation, transesophageal echocardiography (TEE) demonstrated severe AR with an immobile posterior leaflet stuck in an open position. Further imaging revealed the LV vent positioned between the posterior leaflet and the sewing ring, mechanically preventing leaflet motion. After the vent was withdrawn, valve function normalised with complete resolution of AR. The procedure concluded uneventfully, and the patient recovered well, being discharged on postoperative day 8.

Discussion

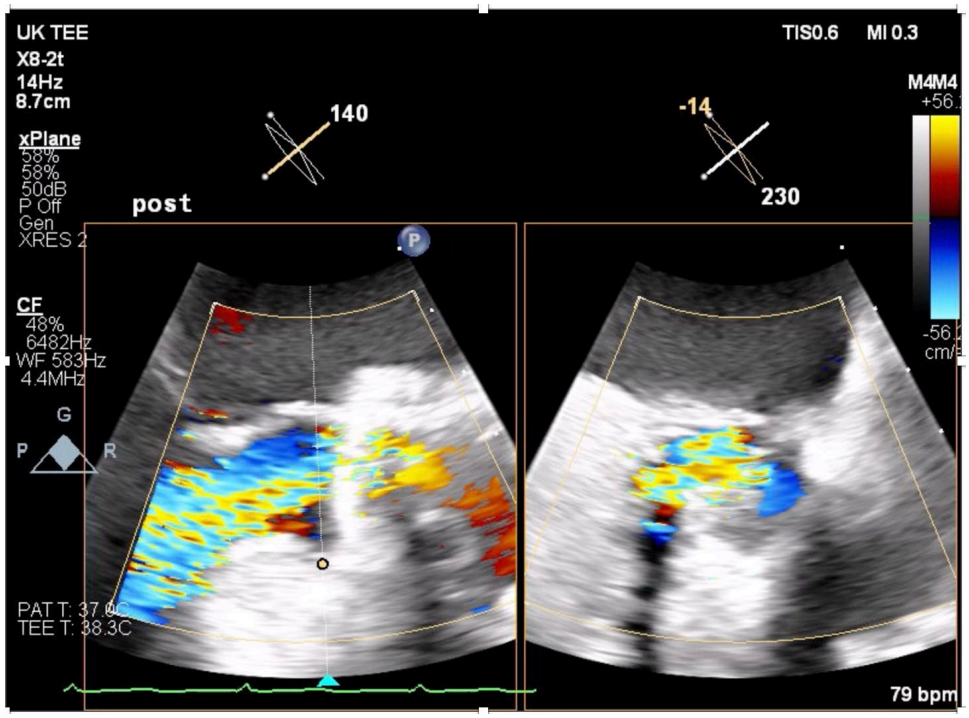
Prosthetic valve regurgitation may occur early after implantation—either in the immediate post-CPB period or later in the postoperative course. Doppler echocardiography is the primary tool for evaluating prosthetic function, distinguishing intravalvular from paravalvular regurgitation and differentiating pathological leaks from normal hinge-point washing jets. When regurgitation exceeds mild severity, determining the

mechanism is critical to guide management. Early prosthetic AR is most often caused by residual suture material or subvalvular structures impairing leaflet closure. Thrombus is unlikely immediately after CPB because patients remain anticoagulated until satisfactory TEE assessment, after which protamine is administered. Primary mechanical failure is rare with modern prostheses. Uncommon early causes include bioglue obstruction and catheter-related leaflet jamming. In this case, LV vent malposition produced severe regurgitation—an unusual but important mechanism—quickly corrected by repositioning, thus avoiding another bypass run. Late prosthetic regurgitation is more commonly caused by thrombosis, vegetation, or pannus interfering with leaflet mobility.

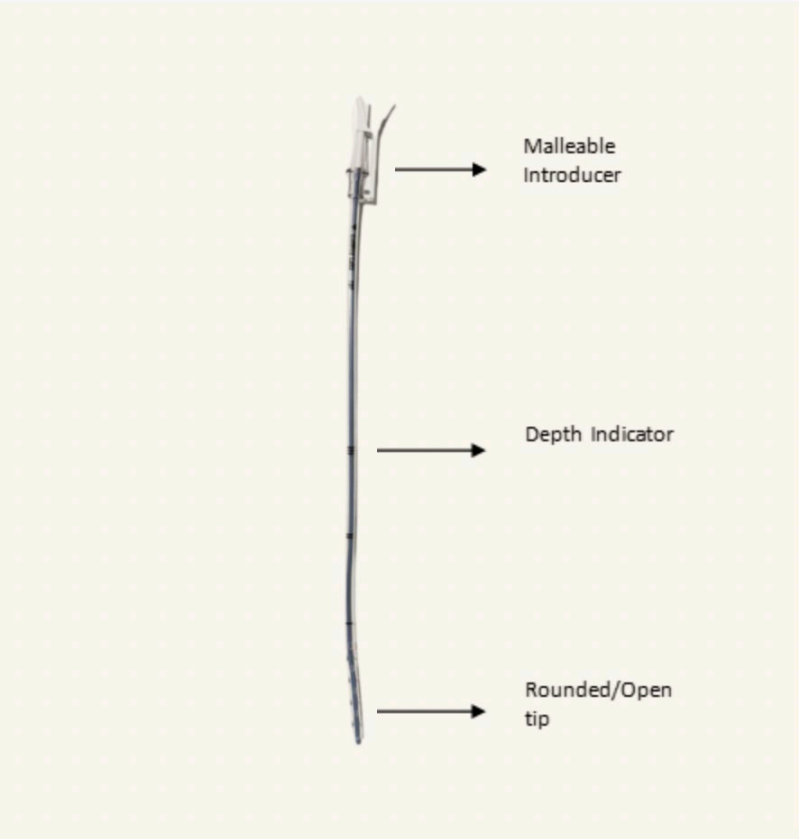
Images



Transesophageal echocardiographic (TEE) image of the aortic valve in long axis in mid-esophageal position showing the LV vent, positioned posteriorly, likely interfering with leaflet motion.



Left is a transesophageal echocardiographic (TEE) image of the aortic valve in long axis in mid-esophageal position showing severe aortic regurgitation (AR) due to an immobile posterior leaflet of a mechanical prosthesis. Right is the orthogonal view showing the jet occupying most of the posterior aspect of the outflow tract.



18 Fr Left Ventricle Vent

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#FR 3 – Kiosk 22 – Poster 4

Anticipating the Surge: Multidisciplinary Perioperative Management of a Hypervascular Cardiac Paraganglioma

Presenting Author: Tina Esfandiary, MD, Resident, Stanford HealthCare

Introduction

Cardiac paragangliomas are rare catecholamine-secreting tumors that present significant anesthetic challenges due to hemodynamic lability, tumor vascularity, and proximity to major coronary structures.1 We report the multidisciplinary perioperative management of a highly vascular right atrioventricular (AV) groove paraganglioma in a patient with hereditary paraganglioma-pheochromocytoma (PGL/PCC) syndrome.

Case Presentation

A 37-year-old female with familial succinate dehydrogenase B mutation (familial heterozygous), secondary hypertension, and anxiety was found to have uncontrolled hypertension and paroxysmal palpitations. Imaging revealed a 3.5-cm right AV groove mass with markedly elevated urine and plasma normetanephrines consistent with PGL/PCC syndrome. Preoperative evaluation included a coronary angiogram (Figure 1) demonstrating a hypervascular tumor with dual supply from the right coronary artery (RCA) and mid-left anterior descending artery (LAD), as well as a transesophageal echo (TEE) showing a right AV groove mass without compression. In coordination with endocrinology and the anesthesia preoperative clinic, the patient underwent alpha-blockade with phenoxybenzamine followed by metoprolol for persistent tachycardia. Given the high risk of vasoplegia during cardiopulmonary bypass (CPB), phenoxybenzamine was held for 24 hours before surgery to allow partial washout. Additional preoperative medication adjustments included avoiding agents such as metoclopramide, corticosteroids, droperidol, and meperidine due to their potential to precipitate a pheochromocytoma crisis. Intraoperatively, the patient underwent cardiopulmonary bypass with bicaval cannulation for tumor resection. While cardioplegia can be accompanied by catecholamine-mediated hypertension, this patient instead exhibited significant hypotension, necessitating high-dose norepinephrine and vasopressin. A 2.5 cm mass involving multiple structures was excised with preservation of coronary integrity, after branches of the RCA supplying the tumor were clipped and the feeder LAD branch was ligated. The patient separated from CPB uneventfully with minimal vasoactive support and did not require any blood transfusions. Post-bypass TEE showed normal biventricular function with mild RV dilation. The patient was then transferred to the ICU for close monitoring of refractory vasoplegia and hypoglycemia, the latter being more commonly associated with epinephrine secreting tumors. Postoperative course was uneventful, with floor downgrade on postoperative day 1and hospital discharge on postoperative day 4.

Discussion

This case highlights the anesthetic complexity of cardiac paragangliomas, where catecholamine secretion, extreme tumor vascularity, and coronary involvement create substantial hemodynamic challenges. Careful preoperative medication optimization, intraoperative readiness for hemodynamic lability, and continuous

postoperative vigilance for refractory vasoplegia and metabolic disturbances enabled the safe resection of surgically complex tumor. The patient's rapid recovery and early discharge highlight the effectiveness of a proactive, multidisciplinary perioperative strategy.

Images

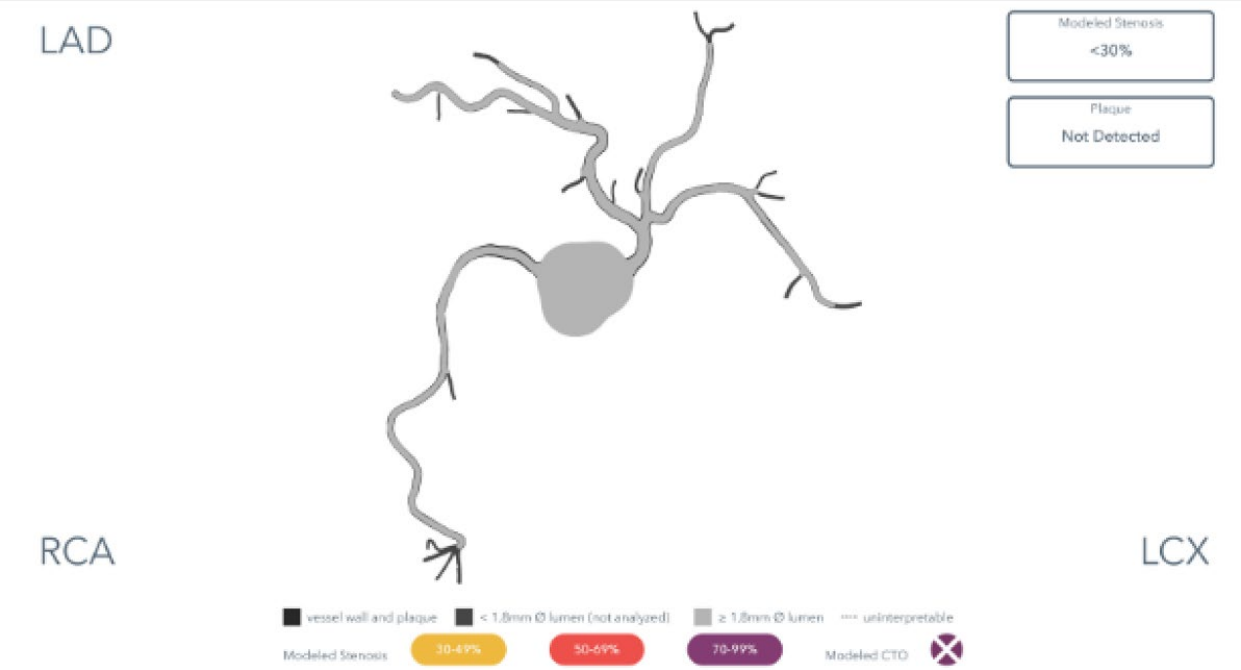


Figure 1: Coronary mapping depicting tumor vascular supply from both the RCA and the mid-LAD

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#FR 3 – Kiosk 22 – Poster 5

Unanticipated Aortic Insufficiency in a Patient Undergoing Septal Myectomy: A Clinical Dilemma

Presenting Author: Kaavya Mahajan, M.D., Cardiac Anesthesia Fellow Physician, Beth Israel Deaconess Medical Center

Introduction

We present a unique case of a patient undergoing septal myectomy, found to have severe aortic insufficiency (AI) on intraoperative TEE. This discovery positioned the cardiac anesthesia and surgical teams at a challenging crossroads to reconsider the most appropriate way to exercise therapeutic privilege while balancing patient safety.

Case Presentation

A 57-year-old male patient with a past medical history of HTN, HLD, OSA, morbid obesity (BMI 42) with worsening pre-syncope and dyspnea on exertion in the setting of hypertrophic obstructive cardiomyopathy presented for a septal myectomy with possible mitral valve repair. Intraoperative TEE exam showed normal biventricular systolic function, asymmetric basal septal hypertrophy, mild MR and no systolic motion of the anterior mitral valve leaflet (SAM) at baseline. Under pharmacological stress, there was evidence of SAM with LVOT obstruction, severe MR, and a peak LVOT gradient of 168 mmHg. At baseline, there was evidence of a trileaflet aortic valve, moderate-severe AI, with an eccentric jet originating between the noncoronary (NCC) and left coronary cusp (LCC), directed towards the interventricular septum, with prolapse of the LCC into the LVOT, holodiastolic flow reversal in the descending aorta and LVEDD 5.04 cm (Figures 1-3). Severity of AI was likely underestimated in the setting of general anesthesia. A second opinion was obtained to validate our TEE findings and after an interdisciplinary discussion, the decision was made for the surgical team to assess the aortic valve in the field to determine feasibility for intervention. Ultimately, the patient underwent a septal myectomy, mitral valve and aortic valve repair, with closure of torn fenestrations between the NCC and LCC. After separation from CPB, TEE exam demonstrated laminar flow through the LVOT, chordal SAM without LVOT obstruction and a peak gradient of 25 mmHg under pharmacologic stress, mild MR and trace AI (Figure 4).

Discussion

In this patient, a detailed 2D and 3D TEE exam with multiplanar reconstruction was vital in characterization of an unanticipated valvular lesion, which led to an alteration of the surgical plan. The anesthesia and surgical teams were perplexed by the following question: Do the benefits of aortic valve intervention outweigh the risks in this patient? Considerations favoring surveillance of the patient's AI included asymptomatic presentation, normal LV systolic function and LV size, and possibility of a non-FDA approved transcatheter solution in the future. Further, survival rates in asymptomatic patients with severe AI are almost like those of an age-and sex-matched population[1]. Despite a Class 1 indication for aortic valve intervention per the 2020 ACC/AHA guidelines[2], several concerns regarding intervention included the ethical considerations surrounding anticoagulation if the patient were to undergo an aortic valve replacement.

Further, explant and re-implantation of aortic valve would be required if the patient required an additional CPB run for septal myectomy revision, which would be associated with increased morbidity and mortality. Also, the patient was likely unsuitable for TAVR in the future given the lack of calcium deposition on aortic valve and would likely be a challenging redo sternotomy given elevated BMI.

Images

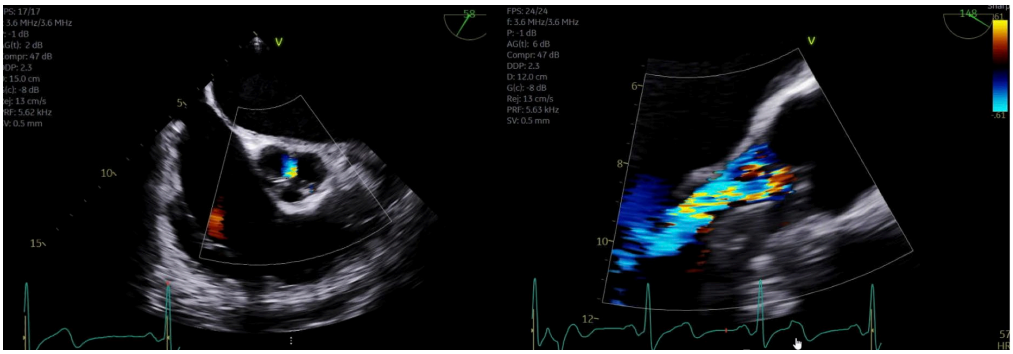


Figure 1. Aortic valve short axis and mid-esophageal long axis views depicting trileaflet with color flow doppler aortic valve, moderate-severe AI with eccentric jet directed towards the interventricular septum.

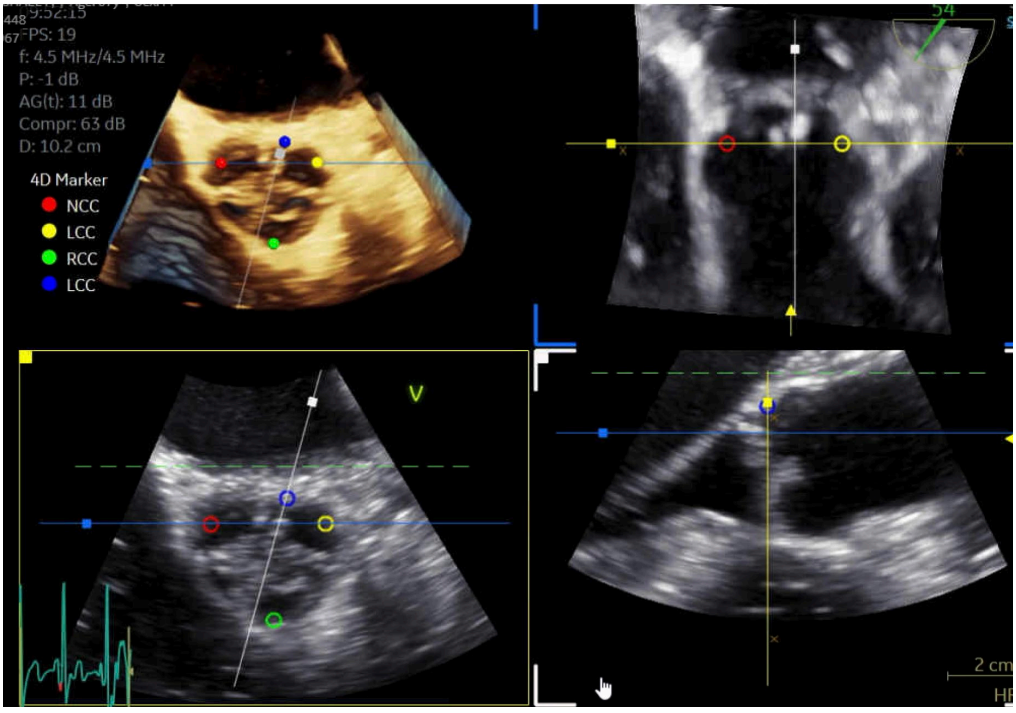


Figure 2. Three-Dimensional Multiplanar Reconstruction of the aortic valve demonstrating prolapse of the left coronary cusp.



Figure 3. Proximal descending aorta long axis view with pulse wave doppler showing persistent holodiastolic flow reversal and transgastric mid-papillary view showing LVEDD 5.04 cm

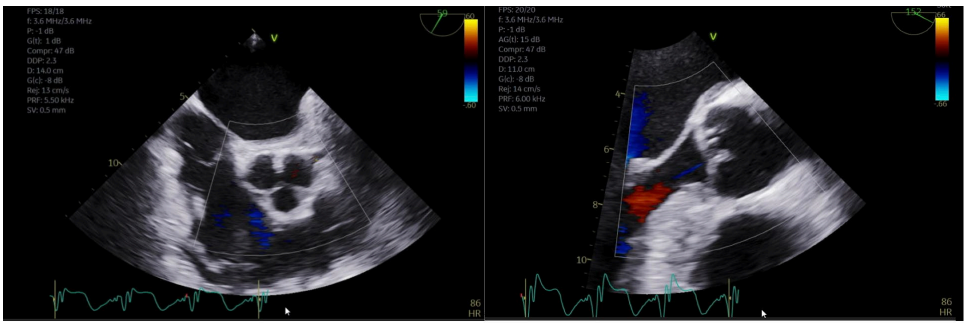


Figure 4. Aortic valve short axis and mid-esophageal long axis views with and without color flow doppler showing trace AI with evidence of sutures closing the torn fenestrations between the NCC and LCC.

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#FR 3 – Kiosk 22 – Poster 6

Superficial Temporal Artery Arterial Line Placement In a Patient With Severe Peripheral Artery Disease Undergoing Aortobifemoral Bypass

Presenting Author: Michael T. Arnold, M.D., Resident Physician, Harbor-UCLA

Introduction

Patients with severe peripheral artery disease (PAD) pose unique challenges for both arterial access and accurate invasive blood pressure monitoring.

Case Presentation

A 59 year old man with hypertension, hyperlipidemia, heavy alcohol use, 20 pack-year smoking history, and worsening claudication from severe aortoiliac occlusive disease presented for aortobifemoral bypass with right common femoral endarterectomy (RCFE). CT imaging of the abdominal aorta and lower extremities showed occlusion of the aorta proximal to the iliac bifurcation, bilateral common iliac occlusions, near-occlusion of the right common femoral artery, and occlusion of the left superficial femoral artery (SFA) (Figures 1 and 2). CT imaging of the thoracic aorta and upper extremities showed severe right subclavian stenosis, severe right vertebral artery stenosis, and total left subclavian artery occlusion with collateral reconstitution distally (Figure 3). Given bilateral iliac occlusion, left SFA occlusion, and planned RCFE, lower extremity access was not feasible. With complete left subclavian occlusion and severe right subclavian stenosis, a right radial arterial line was the only option, though its accuracy was doubted. The surgical plan included intraoperative placement of an aortic pressure line to assess central to peripheral pressure gradients. Bilateral femoral and infrarenal aortic dissection was performed, with cross-clamping of the infrarenal aorta. After graft tunneling, insertion of an 18-gauge angiocatheter into the graft limb for pressure monitoring showed a systolic pressure 110 mmHg higher than the radial line despite an optimal waveform, system checks, and square wave tests with appropriate damping. The radial pressure was deemed unreliable and aortic pressures guided management. Postoperatively, continuous accurate monitoring was needed with removal of the aortic line. Given unreliable peripheral sites, a superficial temporal artery (STA) line was placed under ultrasound guidance with the bullseye technique using a 20-gauge angiocatheter. The waveform was crisp and correlated with aortic readings.

Discussion

Peripheral arterial lines generally reflect central arterial pressures, but in severe PAD, significant gradients can occur due to reduced compliance, stenosis, collateral flow, and waveform damping. When radial pressures are unreliable, alternative sites include the brachial, axillary, femoral, or pedal arteries; however, in this patient, all were contraindicated due to diffuse disease and surgical exposure. The STA, though rarely used in adults, can serve as an alternative when conventional sites are unavailable. It arises from the external carotid artery and divides into frontal and parietal branches and the auricular artery, which provides collateral flow. Cannulation should target the proximal STA distal to the auricular branch to preserve perfusion. Potential complications include thrombosis, pseudoaneurysm, ischemia, skin necrosis, and

facial nerve injury [1]. These can be minimized with ultrasound guidance, avoidance of puncture near the zygomatic arch, and careful postoperative monitoring. This case highlights the STA as a viable alternative for invasive blood pressure monitoring in adults with severe systemic PAD when conventional sites are unavailable or unreliable.

Images

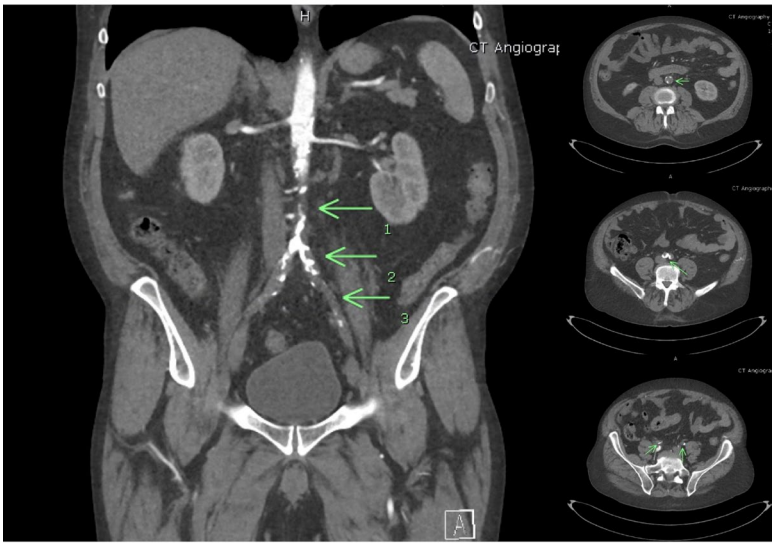


Figure 1: CT abdomen/pelvis with and without contrast showing complete occlusion of the infra-renal aorta (1) proximal to the bifurcation (2) with subsequent occlusion of the bilateral common iliac arteries (3).

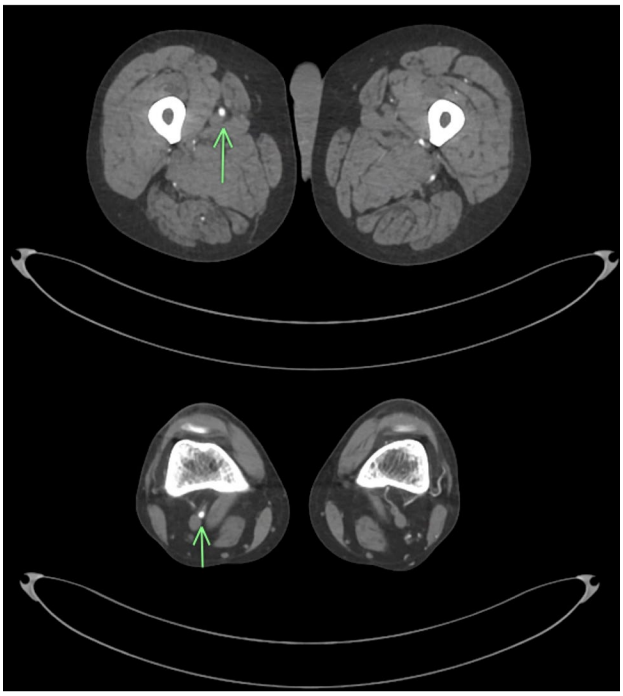


Figure 2: CT angiography of the lower extremities showing the SFA as it courses around the medial aspect of the femur on the right lower extremity and its absence on the left (top photo). The left lower extremity arterial supply remains occluded until the proximal popliteal artery but is then re-constituted by collaterals at the distal popliteal artery (bottom photo).



Figure 3: CT chest showing occlusion of the left subclavian artery with subsequent reconstitution via collaterals in a coronal and the corresponding axial view.

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#FR 3 – Kiosk 23 – Poster 1

Unexplained Aortic Insufficiency and Bi-Ventricular Failure in Previously Healthy Teen Athlete

Presenting Author: Nicholas A. Bohannon, MD, Resident Physician, University of Texas at Tyler Health Science Center

Introduction

Aortic valve insufficiency and bi-ventricular failure are a relatively rare combination for patients in the first two decades of life without known congenital cardiopulmonary anomalies. Additionally, papillary muscle rupture is a relatively rare, but known, complication of myocardial infarction and is also exceedingly rare in the first two decades of life.

Case Presentation

An 18-year-old male presented with known aortic valve insufficiency (Ai) for worsening symptoms with concern for infectious etiology. The patient was hemodynamically stable and ultimately discharged home on antimicrobials for planned interval surgical aortic valve replacement (SAVR) to improve perioperative outcomes relative to immediate surgical intervention. Approximately 10 days after the initial encounter, the patient presented via EMS for cardiac arrest with CPR in progress. An emergent consult was placed to the ECMO and Cardiothoracic Surgery teams with point-of-care-ultrasound (POCUS) echocardiography demonstrating severe Ai and overt left ventricular failure and overall clinical presentation consistent with INTERMACS Profile 1 leading to the ultimate decision to proceed with left ventricular assist device (LVAD) placement via subclavian artery access. Subsequently, the patient was admitted to the CVICU and remained critically ill and unstable for the duration of the hospital stay. The patient ultimately underwent a bioprosthetic aortic valve replacement (AVR) with replacement of LVAD through the prosthesis. Immediately coming off cardiopulmonary bypass the patient developed worsening hypoxia and notable, progressive pulmonary edema. Upon verifying LVAD position and evaluating for perivalvular leaks via intraoperative transesophageal echocardiography (TEE), we discovered new, severe mitral valve regurgitation and rupture of a papillary muscle.

Discussion

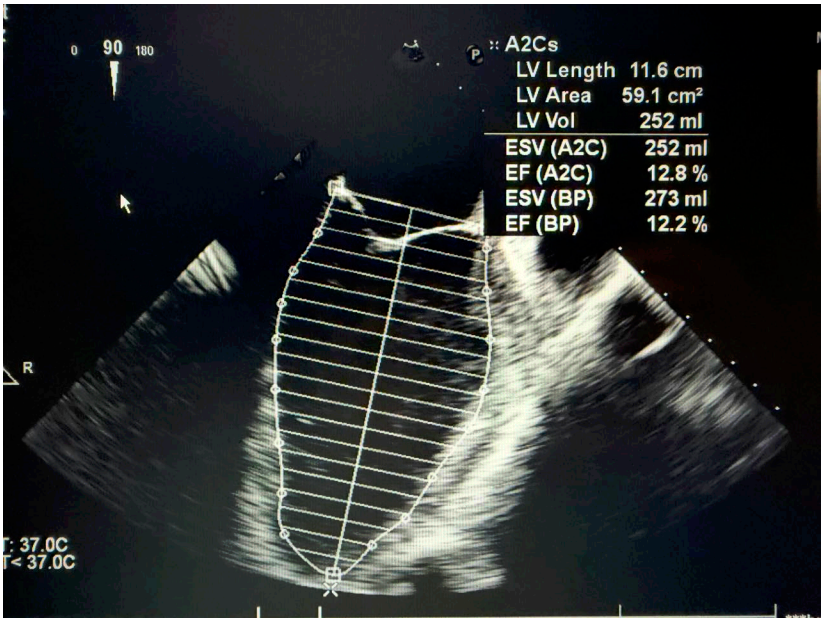
Cardiac catheterization was performed at admission with unremarkable coronary findings and impaired LV function. In the setting of bi-ventricular failure and need for ongoing mechanical circulatory support, the

decision to proceed with a bioprosthetic valve replacement was made in order to facilitate continued LVAD support. Unexplained, new-onset, severe mitral valve regurgitation complicated the clinical course and ultimately led to cannulation for VA-ECMO and transfer to a quaternary care center for further management. This case highlights a number of management dilemmas, including the challenging decision of valve type for aortic valve replacement and determination of etiology and appropriate intervention related to bi-ventricular failure in an otherwise previously healthy teen. This patient had a prolonged hospital stay at a tertiary care facility and was eventually transferred to a quaternary care facility for further management. This patient underwent SAVR, MVR, redo SAVR, and redo MVR during his prolonged hospitalizations all while on continued LVAD and VA-ECMO support. The etiology of this patient's bi-ventricular failure, aortic valve insufficiency, and subsequent mitral valve regurgitation remain unidentified.

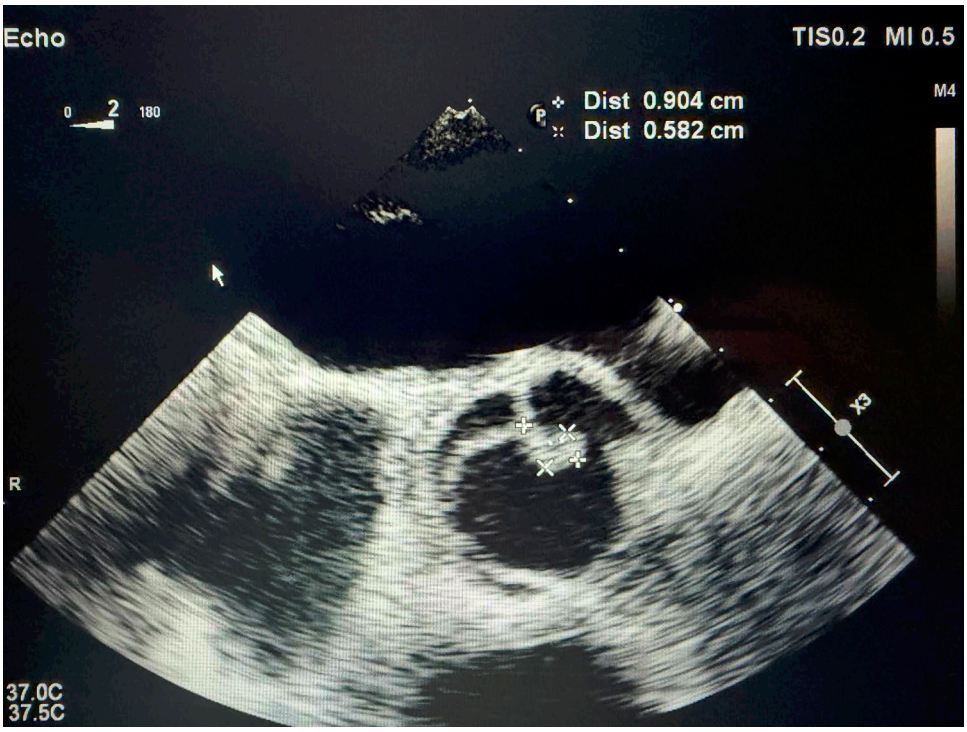
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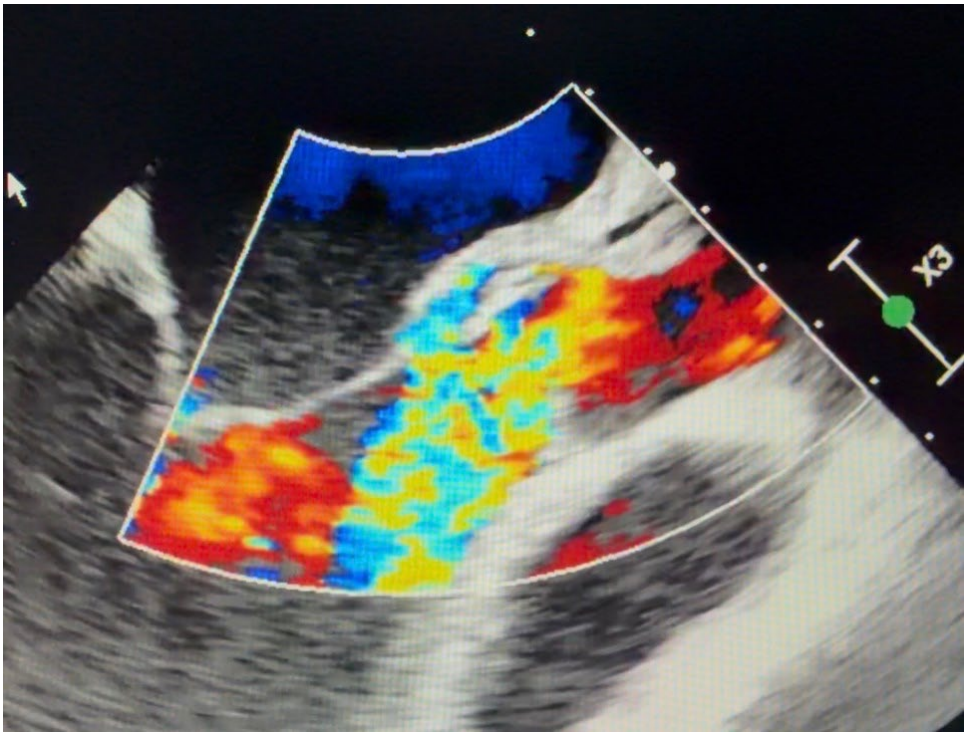
Ruptured papillary muscle on TEE



Biplane EF



Aortic Valve Short Axis with Vegetation



Severe AI on TEE

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#FR 3 – Kiosk 23 – Poster 2

From Shock to Transplant: Anesthetic Considerations in Danon Cardiomyopathy

Presenting Author: Sarah Dinegar, MD, Resident, University of Colorado

Introduction

Danon disease is a rare X-linked lysosomal storage disorder caused by *LAMP-2* deficiency, producing severe infiltrative cardiomyopathy, ventricular arrhythmias, skeletal myopathy, and neurocognitive impairment. Because cardiac involvement is profound, these patients frequently present to cardiothoracic anesthesiologists with advanced heart failure, electrical instability, and high sensitivity to anesthetic agents. We report the perioperative management of a Danon disease patient whose cardiogenic shock required V-A ECMO and subsequent orthotopic heart transplantation (OHT).

Case Presentation

A 42-year-old woman with Danon disease, epilepsy, recurrent ventricular arrhythmias, and HFrEF was hospitalized for worsening heart failure, necessitating cardioversion and CRT-D placement. Months later, she presented with sustained ventricular tachycardia and multiple ICD shocks. She progressed to cardiogenic shock refractory to inotropic therapy, requiring peripheral V-A ECMO cannulation under sedation. Anesthetic management emphasized hemodynamic stability and careful titration given her baseline conduction disease and myopathy; midazolam, fentanyl, ketamine, and limited propofol were used to balance sympathetic tone and avoid exaggerated depressant effects. She demonstrated no myocardial recovery on ECMO and was listed for transplantation. For OHT, standard cardiac monitoring was used, including an arterial line, pulmonary artery catheter, and intraoperative TEE. Induction was performed with fentanyl, lidocaine, etomidate, ketamine, and rocuronium in rapid-sequence fashion to maintain hemodynamic stability and reduce aspiration risk. Her ICD tachytherapy was disabled prior to induction, and external defibrillator pads were applied. Neuromuscular blocker redosing was minimized due to skeletal myopathy. Isoflurane was used without incident. Postoperatively, she developed acute kidney injury requiring CRRT and myoclonic status epilepticus. She remains hospitalized for rejection and nutritional support.

Discussion

Danon disease poses significant anesthetic challenges due to arrhythmogenic cardiomyopathy, conduction abnormalities, skeletal myopathy, and multisystem dysfunction. Many patients have WPW or other pre-excitation patterns on EKG, and some develop dynamic LVOT obstruction from hypertrophic/infiltrative physiology—both vital considerations in anesthetic planning, although these features were not seen in our patient. Induction should prioritize rhythm and hemodynamic stability; etomidate or ketamine is generally preferred over propofol, which may worsen bradyarrhythmias. Standard cardiac monitoring of TEE and PA catheterization supports timely recognition of hemodynamic changes, particularly in V-A ECMO patients. Skeletal myopathy warrants avoidance of succinylcholine and cautious dosing of nondepolarizing neuromuscular blockers with quantitative monitoring. While malignant hyperthermia has not been directly

associated with Danon disease, vigilance with volatile anesthetics remains reasonable. Broader neurologic and metabolic involvement calls for careful postoperative surveillance and conservative sedative and opioid dosing. This case underscores the need for tailored anesthetic strategies when caring for Danon disease patients requiring ECMO and cardiac transplantation.

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#FR 3 – Kiosk 23 – Poster 3

Novel placement of dual-chamber intracardiac pacemaker under direct visualization during open cardiac surgery

Presenting Author: Alex Will, MD, Fellow, Virginia Commonwealth University

Introduction

Intracardiac pacemakers, or leadless pacemakers (LCPs), are small, cylindrical pacemakers placed directly into the myocardium, traditionally by transfemoral approach. Recently, the development of dual-chamber (atrial and ventricular) leadless systems has expanded the capability of LCPs to those of dual-chamber pacemakers. LCPs have been increasingly utilized due to their smaller size and lack of surgical pocket, leading to a less invasive procedure. With no surgical pocket, complications such as infection and hematoma are reduced, and with no leads, complications such as lead fracture and dislodgement are eliminated. LCPs placement has expanded to placement in the right ventricle under direct visualization during cardiac surgery. To our knowledge, this case is the first instance of placement of communicating atrial and ventricular LCPs under direct visualization.

Case Presentation

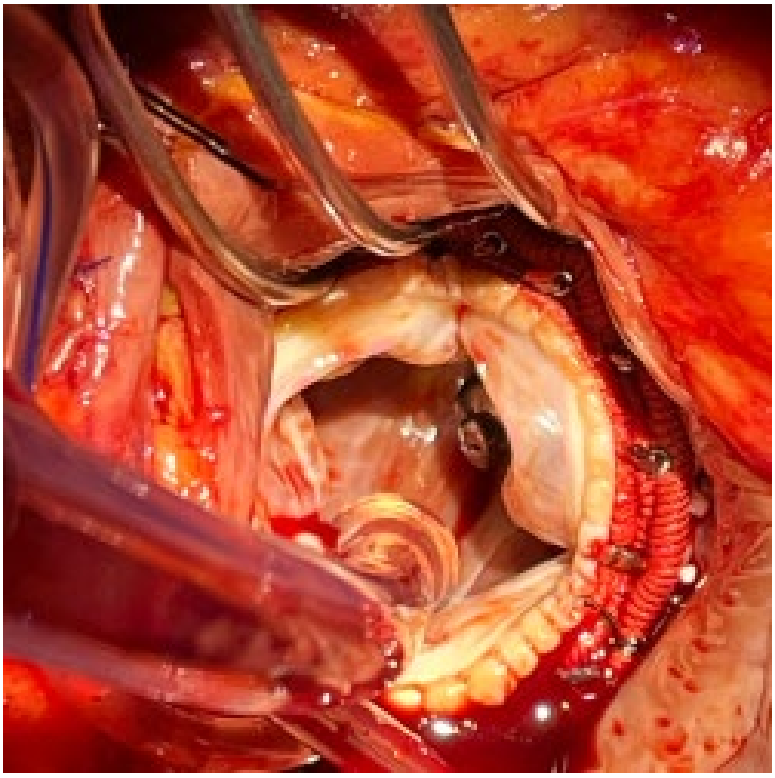
A 56-year-old with history of prior myectomy for hypertrophic obstructive cardiomyopathy complicated by complete heart block status post DDD pacemaker underwent VSD repair for Gerbode defect, mitral replacement, and tricuspid replacement. Her case proceeded without complication, and she underwent pacemaker removal followed by dual-chamber intracardiac pacemaker implantation prior to closure of the right atrium. The ventricular AVEIR lead was implanted in RV apical septum, and the atrial AVEIR lead in the anterior/lateral base of right atrial appendage. Both devices were placed under direct visualization with confirmation of stable placement by tension test. Subsequent interrogation of the device confirmed the appropriate atrial and ventricular pacing function.

Discussion

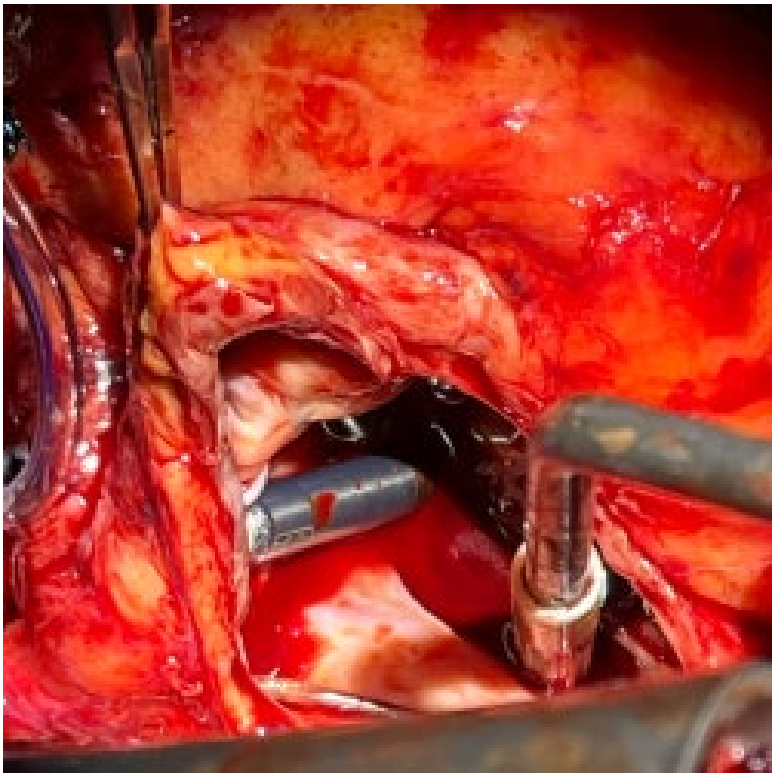
This is the first instance of a surgically implanted atrial and ventricular LCP system; prior, dual-chamber systems were implanted via transcatheter approach, and surgically implanted devices were limited to the right ventricle. The ability to surgically place a dual-chamber LCP confers multiple benefits to patients and clinicians. For anesthesiologists, intraoperative placement during sternotomy removes the need for an additional procedure and reduces the number of anesthetic incidences and associated risks. Temporary pacing wires were still placed intraoperatively, providing a backup for potential device failure until the device could be interrogated and verified to function appropriately. Multiple populations will benefit from the intraoperative approach, and our patient was selected based on her perceived benefit. Firstly, she was pacemaker-dependent in the setting of CHB and was undergoing a procedure that would require removal and subsequent replacement of her pacemaker. Secondly, she was undergoing cardiac surgery with a high risk of conduction abnormalities. With implantation of the device intraoperatively, she did not require additional intervention, and furthermore, was not exposed to usual risks of pocket infections, lead

dislodgement, and lead fracture. Lastly, patients undergoing dual-chamber LCP placement intraoperatively versus single ventricle benefit from physiologic sequential atrial-ventricular conduction. This is favorable in patients with reduced ejection fractions to avoid long-term complications such as pacing-induced cardiomyopathy.

Images



Leadless pacemaker in right ventricle as seen through the tricuspid.



Leadless pacemaker in right atrium.

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#FR 3 – Kiosk 23 – Poster 4

Multivessel Coronary Disease with a Large Circumflex–to–Coronary Sinus Fistula: A Rare Cause of Ischemic Chest Pain

Presenting Author: Elijah Weber, MD, Resident Physician, Virginia Commonwealth University-Department of Anesthesiology

Introduction

Coronary artery fistulas are rare congenital anomalies, accounting for only 0.1%-0.2% of cases (1). Their presentation ranges from incidental murmurs to ischemia and heart failure, depending on fistula size, connections and complications (1,2,3).

Case Presentation

59-year-old male with a past medical history of paroxysmal supraventricular tachycardia, smokeless tobacco use, alcohol use, obesity and gout who presented with acute onset chest pain and shortness of breath. Initial troponins were negative, but he was taken for urgent left heart catheterization given new lateral ST-segment depressions, which revealed severe LAD stenosis (80%), complete RCA occlusion with collateral flow, and a large calcified fistula from aortic root to right atrium. Preoperative right heart catheterization demonstrated elevated right and left filling pressures. CT coronary angiography confirmed coronary sinus fistula with a dilated and tortuous left circumflex terminating at the coronary sinus. He was transferred for planned CABG and repair of the coronary fistula. In addition to the above findings, intraoperative TEE showed severe mitral regurgitation due to a flail leaflet. Intraoperatively, the end of the circumflex artery appeared to join the coronary sinus, forming a single vessel draining into the right atrium. The circumflex was opened and the junction between the sinus was primarily closed along with a more proximal opening. After the LAD and PDA were anastomosed, a mixture of dark and bright red blood was visualized coming out of the coronary sinus ostium with warm blood cardioplegia flush. This suggested additional fistulous connections between the coronary sinus and circumflex artery. Given the significant tortuosity and anticipated risks associated with prolonged aortic cross-clamp time, the procedure was concluded after completing the coronary bypass. Plans were made for a staged intervention to address the coronary fistulas and severe mitral regurgitation at a later date.

Discussion

Coronary artery fistulas present unique challenges to anesthesiologists. The location of the fistulous connection can create a downstream hypoperfusion resulting in ischemic disease to the supplied region, and surgical correction may often be necessary (2). While direct surgical visualization is sometimes possible, other imaging modalities are often necessary. Since coronary angiography is not readily available during cardiac surgery, TEE can aid in the diagnosis and evaluation of surgical repair (3). In this case, transesophageal echocardiogram was able to identify fistulous flow into the coronary sinus prior to and after surgery; however, diagnosis of persistent, non-visible fistulas arose from the unique modality of visualizing antegrade warm blood from the coronary ostia out of the coronary sinus. This case demonstrates the

shortcomings of cardiac surgery alone for coronary artery fistulas and the need for multiple modalities for treatment.

Images

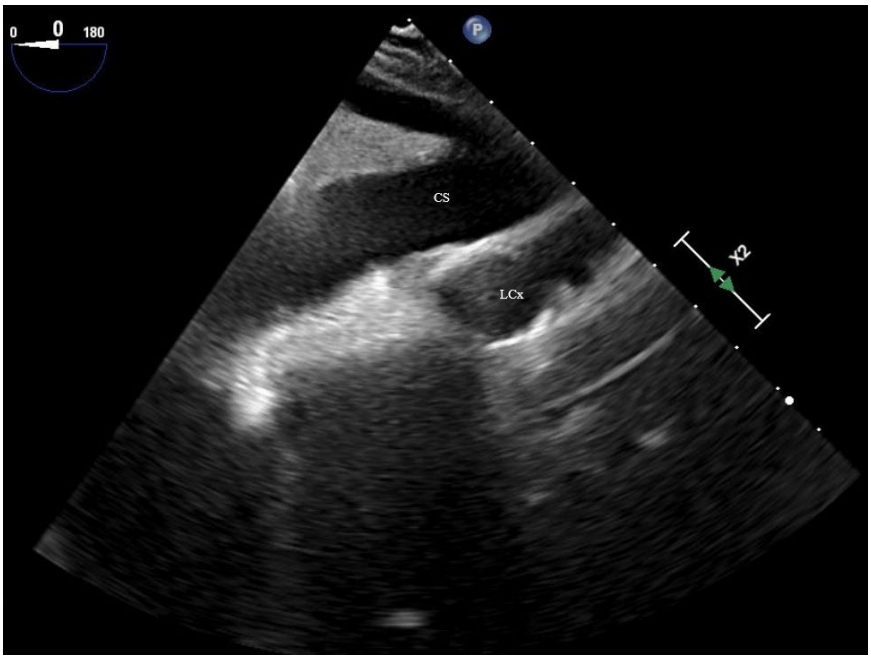


Figure 1. Dilated coronary sinus (CS) as it transverses adjacent to the left circumflex artery (LCx).

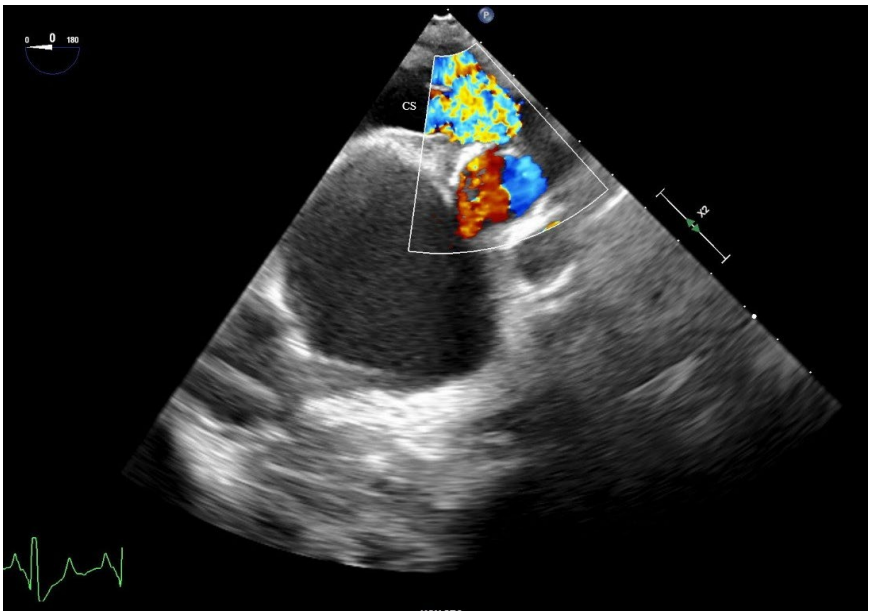


Figure 2. Color doppler of coronary sinus (CS) showing turbulent flow juxtaposed to the left circumflex artery with possible regions of fistulous flow.

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#FR 3 – Kiosk 23 – Poster 5

Thermal Deformation and Functional Loss of a Pulmonary Artery Catheter During Surgical MAZE Procedure: A Rare Intraoperative Event

Presenting Author: Anna Arakelyan, Resident, University of Arizona1

Introduction

The surgical MAZE procedure is an established adjunct to cardiac surgery for the treatment of atrial fibrillation. It improves long-term rhythm control by creating controlled atrial lesions using radiofrequency (RF) or cryothermal energy. Although generally safe, complications may include bleeding, conduction abnormalities, esophageal injury, & damage to surrounding structures. Interactions between intracardiac devices and ablation energy are rarely reported, and thermal injury to pulmonary artery (PA) catheters remains an exceptionally uncommon complication. We present a patient whose PA catheter became deformed & functionally obstructed during concomitant MAZE & CABG, resulting in immediate hemodynamic monitoring failure.

Case Presentation

A 71-year-old man with hypertension and paroxysmal atrial fibrillation on apixaban underwent elective CABG ×2 (LIMA–LAD and SVG–diagonal) with a biatrial MAZE procedure. A standard PA catheter was placed without difficulty prior to sternotomy. Initial hemodynamics were appropriate. Shortly after initiation of cardiopulmonary bypass and during early MAZE lesion creation, the anesthesia team observed a sudden and extreme increase in mean PA pressures from 11/8 mmHg to 250–300 mmHg. Simultaneously, the PA distal port and balloon lumen became nonfunctional, with inability to flush or draw back. CVP remained stable, the venous infusion port remained patent, and there was no evidence of left ventricular distention or catheter migration on TEE. The catheter was left in place until separation from bypass and then removed with the surgical team prior to chest closure. Inspection showed the distal catheter flattened, melted, and partially ablated near the distal thermistor region. After multidisciplinary discussion, a new PA catheter was placed and functioned normally for the remainder of the perioperative period. The patient recovered without hemodynamic or pulmonary sequelae.

Discussion

Thermal injury to a PA catheter during MAZE procedure is rare but clinically important. Thermal injury to a PAC during MAZE procedure is rare but can be clinically significant. Proposed mechanisms include:

- direct thermal injury from RF or other energy sources used in ablation, causing melting & loss of integrity
- mechanical entrapment within ablation devices, which can be compounded by thermal effects.

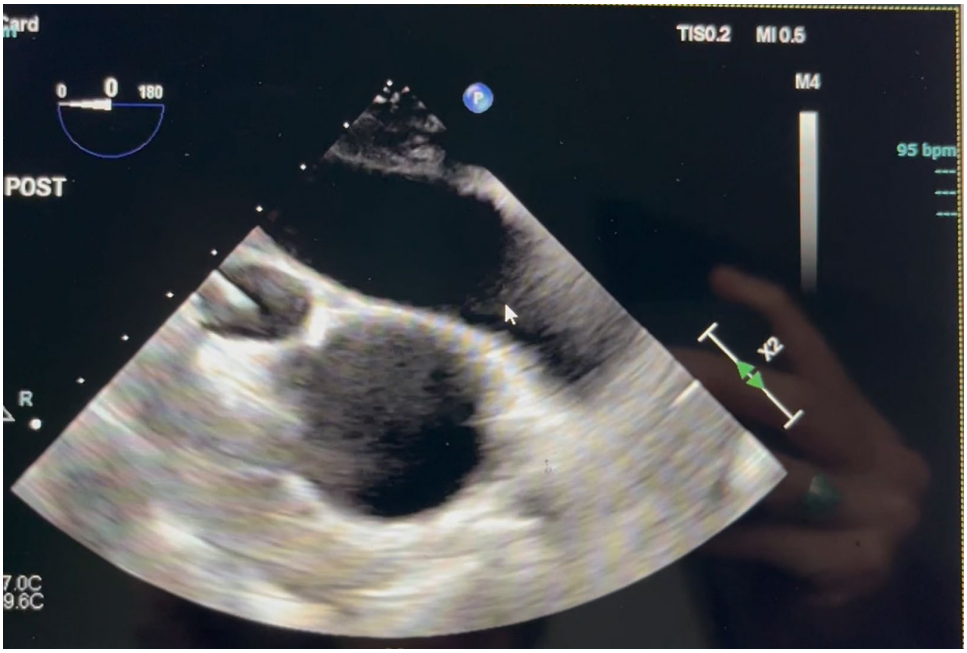
A review of the literature identified 223 distinct incidents of PA catheter entrapment reported across 77 case reports and 3 retrospective studies. Entrapment occurred most often during mitral valve surgery and was unrecognized until the postoperative period in 77% of cases. Inadvertent fixation to cardiac structures was the most common mechanism, and 75% of affected patients required redo sternotomy for retrieval, with

prolonged hospitalization compared with STS database averages. This case emphasizes the need for continuous vigilance even when patient is on bypass as well as correctly interpreting & discussing the information we receive from our many different monitoring devices. Strategies to mitigate risk include temporarily withdrawing the catheter during MAZE lesion sets, confirming catheter tip location with TEE, and establishing clear communication between surgical & anesthesia teams during ablation.

Images



PA catheter_damage



Normal PA catheter position on TEE



Chart with PA pressures

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#FR 3 – Kiosk 23 – Poster 6

Anesthetic Management of Catastrophic Embolization During Vacuum-Assisted Thrombectomy: A Case of Unrecognized Tumor Thrombus

Presenting Author: Kevin Truong, MD, Fellow, UT Houston

Introduction

Tumor thrombus extending into the right atrium presents high risk for life-threatening complications such as embolization and hemodynamic collapse. Use of AngioVac for tumor thrombus debulking, while increasingly supported by data, carries known risks of fragmentation and pulmonary embolization. We present a case of embolization of an unrecognized tumor thrombus requiring massive cardiopulmonary support and resuscitation.

Case Presentation

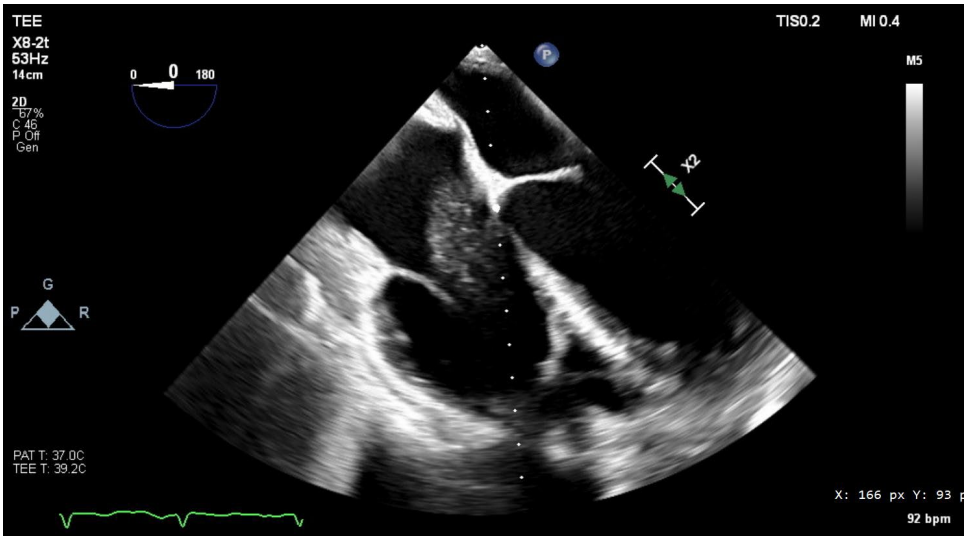
A 25-year-old male with metastatic chondrosarcoma was referred for thrombectomy of presumed iliocaval thrombus extending into the right atrium. Preoperative MRI suggested bland thrombus, and the procedure was planned accordingly. Under general anesthesia with arterial line, central access, and perfusion standby, intraoperative TEE revealed a large, mobile right atrial mass extending through the tricuspid valve into the RV. The AngioVac system was used for aspiration under continuous TEE guidance. During debulking, a portion of the mass was seen embolizing in real time, followed by abrupt hemodynamic collapse and three episodes of PEA arrest. CPR achieved ROSC each time. VA-ECMO was emergently initiated via femoral cannulation for circulatory support. After stabilization and ICU care, ECMO was weaned over five days.Pathology confirmed metastatic tumor thrombus, and the patient was discharged home for palliative management.

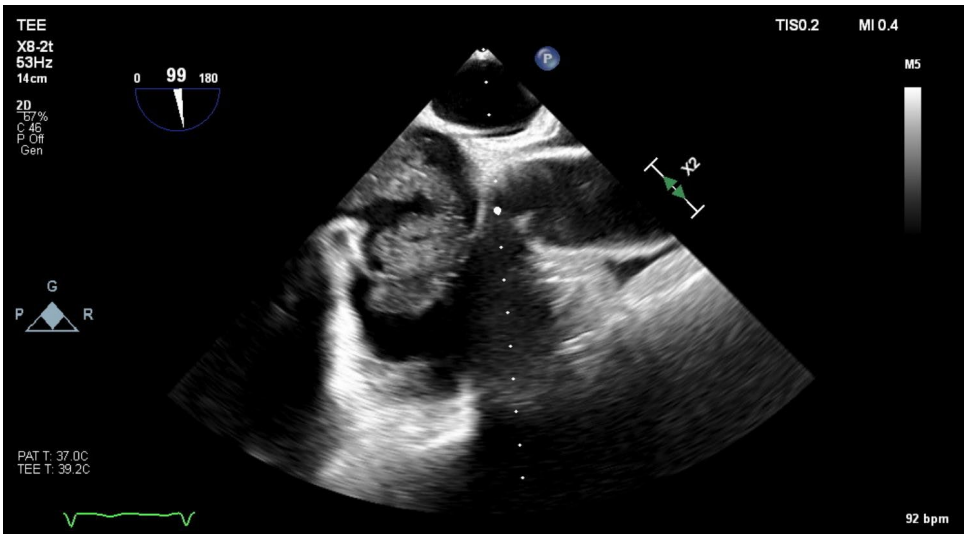
Discussion

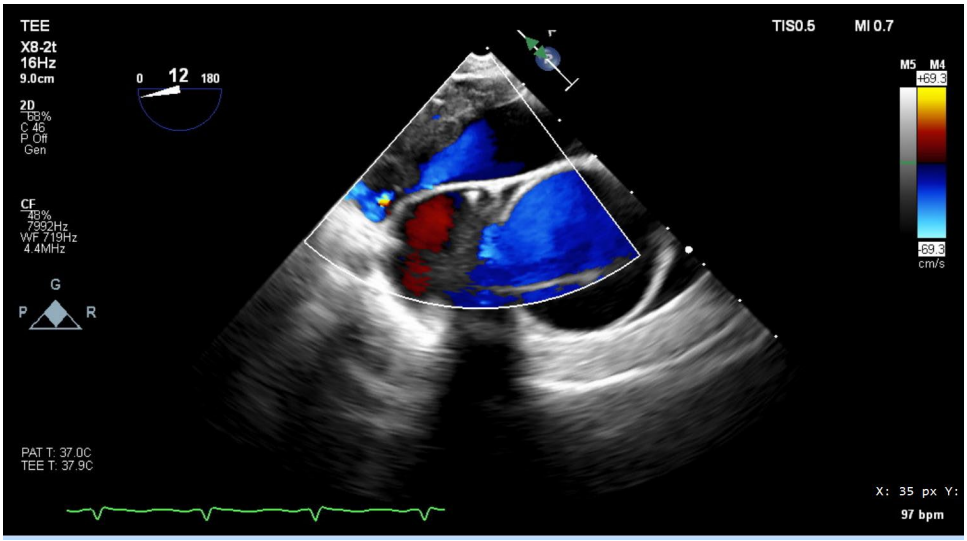
Cardiac tumors are rare and may be primary or metastatic. Primary cardiac tumors occur in only 0.001–0.3% of the population, while metastatic involvement is far more common at 2.3–18.3% [1]. Management typically involves open surgical removal, with prognosis dependent on tumor type. The AngioVac system is a minimally invasive suction-based device designed to remove thrombi or soft material from the venous system or right heart [2]. It is not intended for tumor extraction; its funnel-shaped tip may promote fragmentation, and can lead to unintended embolization. When embolization occurs, clinical effects depend on embolus size, mobility, and lodgment site. Pulmonary artery obstruction can rapidly cause obstructive shock, hypoxemia, and cardiac arrest. Mortality for massive pulmonary embolism remains high at 25–65%, depending on obstruction severity and hemodynamic status [3]. Intraoperative TEE is critical for real-time detection of thrombus migration, right ventricular strain, and acute circulatory collapse. Management of massive embolic obstruction prioritizes oxygen delivery and circulatory support while the obstruction is treated through catheter-directed therapy, thrombectomy, or systemic interventions when appropriate. When hemodynamic collapse occurs, venoarterial ECMO has become an important salvage strategy, providing immediate circulatory and oxygenation support while definitive therapy proceeds. Survival rates of

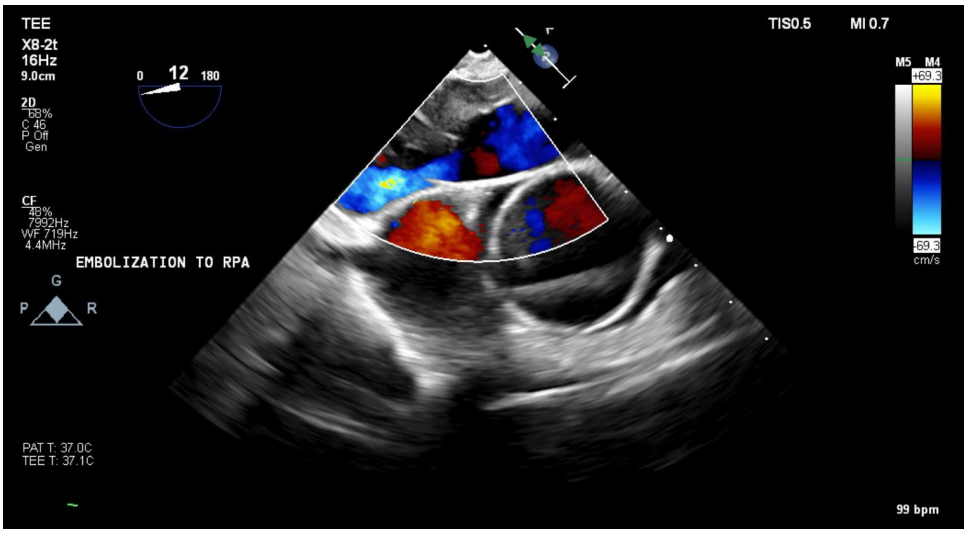
~40–60% have been reported in massive PE requiring ECMO [4]. Our case highlights the importance of anesthetic vigilance, continuous TEE monitoring, and early coordination with perfusion teams when the nature of an intravascular mass is unclear. Rapid initiation of ECMO was lifesaving after recurrent PEA arrest, underscoring its expanding role in catastrophic embolization during minimally invasive thrombectomy

Images









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#FR 3 – Kiosk 23 – Poster 7

Inverted Valve Placement During TAVR: A Complex Cardiac ICU Patient

Presenting Author: Adrina Grossman, MD, Fellow Physician, Washington University in St. Louis

Introduction

Transcatheter aortic valve replacement (TAVR) is an established therapy for severe aortic stenosis in high-risk patients. Although procedural safety has improved, complications can be devastating[1]. Patients presenting for TAVR already carry the burden of comorbidities that typically precludes them from undergoing open surgical valve placement. Complications therefore often lead to poor outcomes. This case's positive outcome can provide some guidance.

Case Presentation

A 68 y/o male severe aortic stenosis and an ejection fraction (EF) of 30% with extensive cardiovascular and systemic comorbidities including HTN, HLD, CHF s/p AICD placement, CAD s/p multiple PCI and CABG x3, COPD, CKD stage III and type 2 diabetes. The patient presented to an OSH for elective TAVR. Multiple vascular access attempts were made, including femoral and axillary approaches; following repeated failures, a trans-carotid approach was pursued. During deployment, the prosthetic valve misfired and was placed inverted within the aortic annulus, resulting in disruption of aortic flow and cardiac arrest. ACLS was initiated, while a balloon angioplasty sheath was passed into the aorta, balloon-stenting the mispositioned valve into the left ventricle, restoring aortic flow. The patient was then transferred to BJC for higher level care. On arrival to the CTICU, MTP was initiated due to the patient's starting hemoglobin of 5.9, profound hypotension, and expanding hematomas at his TAVR access sites. At that time, the patient's only central access was a single 5 Fr introducer catheter in the left femoral vein which was being used for vasopressors. The right carotid access site was enlarged, precluding further right neck vascular access. An 8 Fr Cordis and a quadruple lumen 7.5 Fr catheter were therefore placed into the left internal jugular vein. POCUS demonstrated new further severely reduced EF and new severe iatrogenic aortic insufficiency. The patient was emergently taken for redo sternotomy for retrieval of the percutaneously placed valve, new surgical AVR, and VA ECMO cannulation due to EF of 15% and severe pulmonary edema. His ICU course was complicated by AKI (Cr 4.9 mg/dL), requiring CRRT. The patient remained on VA ECMO for 5 days, converted to VV ECMO on day six because of persistent hypoxemia, and subsequently decannulated after ten days. After ten days of CRRT, the patient was transitioned to IHD. After three weeks in the ICU, the patient was discharged to the cardiac stepdown unit.

Discussion

Though TAVR is now the standard of care for high-risk candidates, the PARTNER Trial found the incidence of major (15.3%) and minor (11.3%) complications in TAVR amounted to approximately a quarter of all cases[2]. Though a case of inverted aortic valve placement has not previously been reported, this case accentuated those early interventions that allowed for his successful discharge home: i) early stenting of the inverted prosthetic valve out of the aortic outflow tract along with ii) early POCUS to assess cardiac function and

degree of AI and iii) the decision to proceed with surgical AVR and VA ECMO cannulation[3]. This case shows the importance of reporting unusual TAVR complications to refine protocols and improve care for TAVR patients.

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#FR 3 – Kiosk 24 – Poster 1

A Cool Case of Deep Hypothermic Circulatory Arrest

Presenting Author: Joana Abed Elahad, MD, Fellow, Keck Medicine of USC

Introduction

Deep hypothermic circulatory arrest (DHCA), developed in the 1960s, consists of systemic hypothermia (18-20°C) to preserve organ function during cessation of circulation1. It has been established as the primary neuroprotective mechanism, as it slows injury-inducing pathways by limiting cerebral metabolism2. However, prolonged DHCA has been associated with worse neurologic outcomes, requiring the use of antegrade and retrograde cerebral perfusion (ACP and RCP)2. Most patients tolerate 30 minutes of DHCA without significant neurologic dysfunction; there is an increased risk of brain injury at longer durations, and at 60 minutes patients may suffer irreversible brain injury1.

Case Presentation

A 29-year-old woman with history of bicuspid aortic valve (AV) status-post aortic valve replacement (AVR) at 16 years, complicated by endocarditis requiring redo mechanical AVR 5 years later, developed aortic stenosis (AS) and presented to Keck Hospital for a planned third-time AVR. Other medical history consists of coronary artery disease with stent to left anterior descending (LAD) artery, poststreptococcal glomerulonephritis status-post failed kidney transplant, currently on hemodialysis. On the day of surgery, the patient endorsed emesis from acid reflux 3 times weekly, which had not been previously evaluated. After successful rapid sequence induction and intubation, central venous and pulmonary artery catheters were placed. Intraoperative transesophageal echocardiogram (TEE) revealed left ventricular ejection fraction (EF) of 47%, and mildly dilated right ventricle (RV) with mild dysfunction. The aortic valve was difficult to visualize due to significant shadowing. Cannulation was established via femoral vein to right axillary artery. The patient was then cooled to 18°C and sternotomy was performed. As anticipated, this resulted in damaging the aortic valve graft; however DHCA time was kept as short as possible, for a total of 13 minutes. ACP was performed and central cannulation was achieved; then the aorta was clamped with anterograde cardioplegia. The coronary buttons were significantly calcified, which resulted in vein grafts being created to the left circumflex, LAD, and right coronary arteries. A ventricular septal defect was created and the pulmonary artery was injured, which were repaired. The patient was then rewarmed to 36°C, the aortic clamp was removed, and the patient was successfully weaned from bypass on an epinephrine drip and inhaled nitric oxide (iNO). TEE showed an improved EF of 55-60% and a moderately dilated RV with moderately reduced function. She was transferred to the intensive care unit on an epinephrine drip at 6mcg/min and iNO at 20ppm. She had delayed sternal closure 3 days later and was extubated the following day with no neurologic deficits.

Discussion

DHCA is beneficial in redo cardiac surgeries, especially in high-risk cases when there is concern for structural damage during sternotomy. If planned appropriately, it can be a beneficial option to help preserve

organ function and decrease the risk of neurologic injury. This technique requires careful planning and coordination with the surgical team to minimize time in DHCA. This case report helps to show that an appropriate high-risk cardiac surgery can result in a successful outcome with DHCA.

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#FR 3 – Kiosk 24 – Poster 2

From Cardiac Catheterization to Bypass Surgery: The Case of the Trapped Wire

Presenting Author: Miar Elaskandrany, MD, Resident, Brigham and Women's Hospital

Introduction

Guidewire entrapment is a serious complication of percutaneous coronary intervention (PCI), with reported incidences ranging from 0.2% - 0.8% in standard procedures and higher rates in complex interventions.[1] There are no official society-endorsed guidelines for the management of retained or dislodged guidewires. Management includes percutaneous retrieval, conservative therapy, and surgical removal, depending on the location, feasibility, and risk.

Case Presentation

A 75-year-old man with exertional angina and multivessel coronary artery disease underwent cardiac catheterization. During the case, an instantaneous wave-free wire pressure wire became trapped within a severe left anterior descending (LAD) stenosis. A Turnpike LP microcatheter was used to navigate the lesion, but the wire separated despite gentle traction. Snare retrieval attempts were unsuccessful, and a 3.5-mm balloon partially retrieved the wire, leaving a < 5-mm fragment in the diagonal branch. Optical coherence tomography confirmed no vessel injury. Due to the flow-limiting disease in the left main, left circumflex, and LAD coronary arteries, coronary artery bypass grafting (CABG) was planned with aortotomy for surgical extraction of the retained wire fragment, visible in the aortic root on transesophageal echocardiography (TEE) (Figure 1). The wire was retrieved with gentle traction. The patient underwent successful coronary revascularization and he was discharged on post operative day five after an uncomplicated course.

Discussion

Guidewire entrapment is a rare complication of PCI, associated with calcified or tortuous coronary segments, excessive torquing, or complex anatomy.[1] The distal flexible tip of intracoronary wires is susceptible to fracture where mechanical stress is greatest.[2] First-line strategy is percutaneous retrieval with snaring, balloon trapping, microcatheter extraction, and specialized maneuvers, though this carries risks including dissection, perforation, thrombosis, or embolization of the retained fragment.[1] Conservative management may be considered when a radiopaque fragment remains in a small side branch without clinical consequences, but retention still carries risks of thrombosis, perforation, migration, or embolization.[3] If percutaneous attempts fail, surgical retrieval may be required, particularly during CABG or when the fragment poses high risk; however, surgery carries risks.[1] In this case, an aortotomy was performed solely for wire removal. TEE offers 3D localization allowing precise, targeted arteriotomy and reducing the risk of inadvertent injury, while fluoroscopy provides only a 2D luminal view and is limited intraoperatively. Guidewire entrapment remains a rare but clinically significant complication. When percutaneous retrieval is unsuccessful, surgical extraction is an effective strategy. This case highlights the utility of TEE in intraoperative localization and removal of retained fragments. Routine consideration of TEE in similar scenarios may enhance procedural precision and improve patient outcomes. Despite technological and

technical advances, wire entrapment still occurs and lacks standardized management guidelines. Systematic study is needed to guide prevention and develop evidence-based management algorithms to improve patient safety.

Images

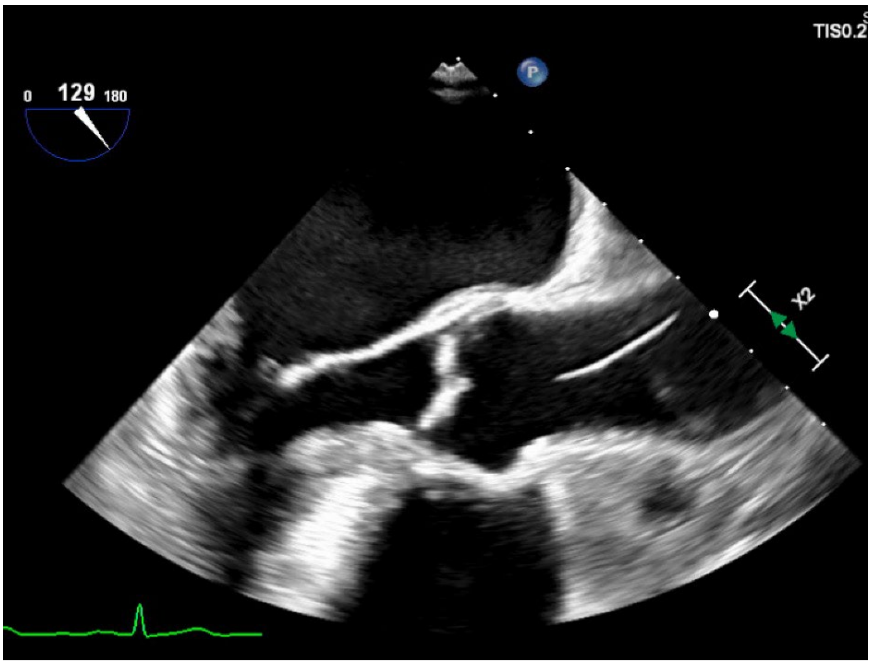


Figure 1. Mid-esophageal long axis view with retained wire visible in aortic root

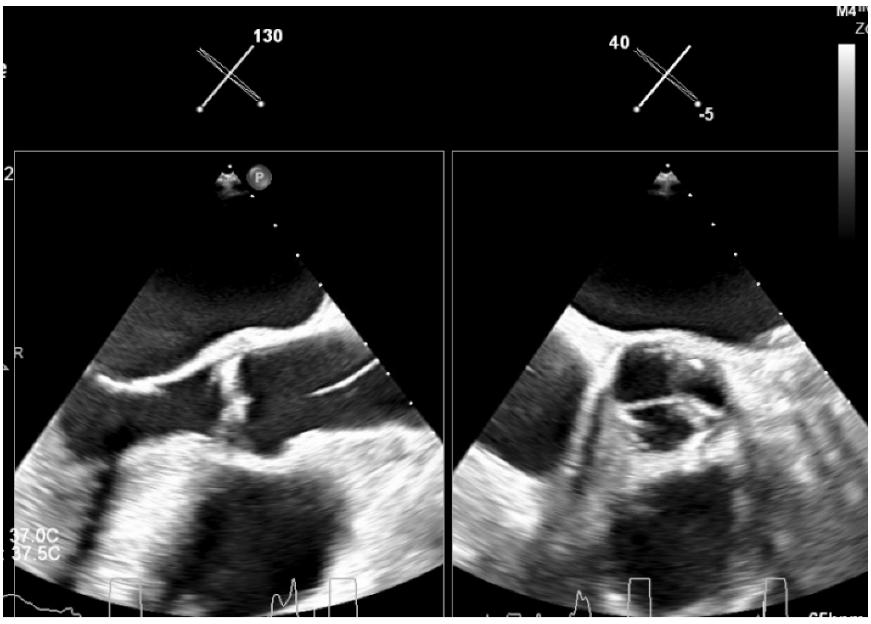


Figure 2. Mid-esopheageal long axis view with orthogonal X-Plane mid-esophageal aortic valve short axis view demonstrating retained wire protruding from left coronary ostium.

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#FR 3 – Kiosk 24 – Poster 3

Orthotopic Heart Transplantation for Giant Left Ventricular Pseudoaneurysm: Unique Perioperative Challenges

Presenting Author: Faizan Ahmed, MD, Resident, University of Louisville

Introduction

Left ventricular pseudoaneurysms following myocardial infarction carry rupture rates of 30-45% within the first year. Unlike true aneurysms which retain myocardial wall integrity, pseudoaneurysms are contained solely by thin fibrous tissue and pericardium. Giant pseudoaneurysms (>4cm) with severely reduced ejection fraction are not amenable to surgical repair, making orthotopic heart transplantation (OHT) the only viable option. However, OHT in this population presents unique challenges distinct from standard transplantation. We present systematic management of these complexities in a patient with a 7.5cm LV pseudoaneurysm.

Case Presentation

A 49-year-old female developed a giant LV pseudoaneurysm (7.5×3.6cm, 5.4cm ostium) nine months post-CABG×4, with progressive heart failure (EF 15%) despite home dobutamine. Catheterization showed elevated pressures (PAWP 23mmHg), CI 2.1 L/min/m^2 (INTERMACS 3). Pseudoaneurysm size precluded repair; transplantation via redo sternotomy was pursued.

Perioperative Management:

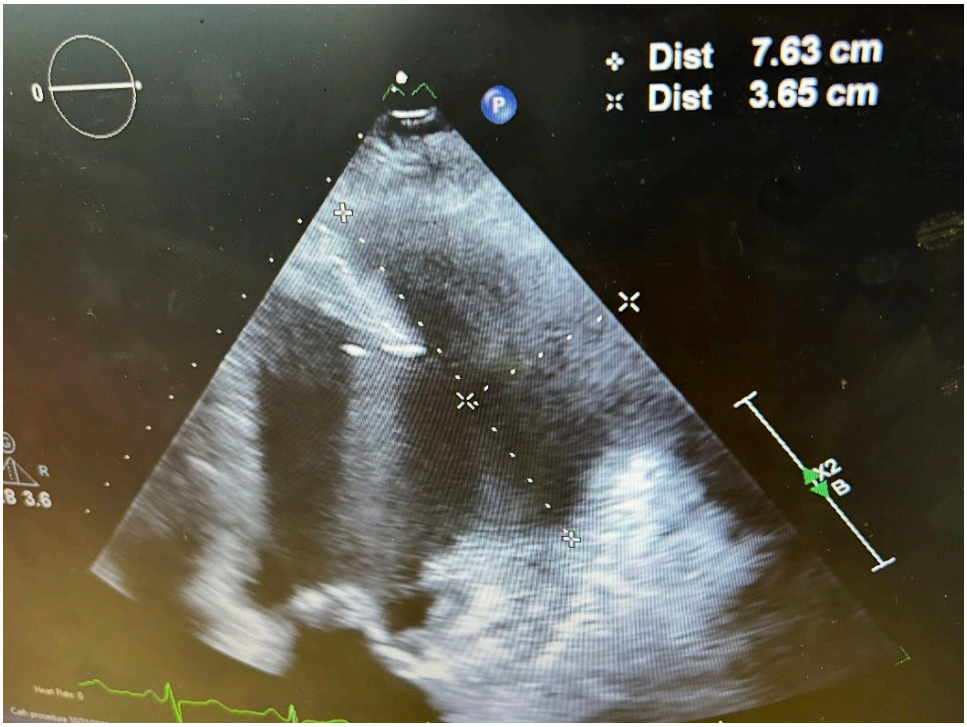
- Hemodynamic support with epinephrine, milrinone, iloprost, isoproterenol during high-risk explantation
- Intraoperative VF during dissection required immediate defibrillation—pseudoaneurysm-specific risk from manipulating scarred myocardium pre-bypass
- Dense adhesions from recent CABG surrounding thin-walled pseudoaneurysm; peripheral cannulation prepared for emergent bypass
- PCC, Factor VIIa, 900mL cell saver addressed coagulopathy
- Post-separation echo showed normal LV (EF 60-65%), mild RV dysfunction managed with inotropes/pulmonary vasodilators
- AKI (Cr 0.7→2.01mg/dL) required CRRT→hemodialysis

Postoperative Course: Encephalopathy requiring reintubation, MSSA pneumonia, pneumothorax complicated recovery. Serial echo demonstrated excellent allograft function. Patient weaned from support, discharged with normal function.

Discussion

Giant pseudoaneurysms create unique OHT challenges. The wall lacks myocardial support, risking catastrophic rupture before bypass. Dense adhesions compound risk; peripheral cannulation must be planned for emergent bypass. Pre-bypass arrhythmias distinguish these cases. Scarred myocardium creates re-entrant substrate; manipulation triggers malignant arrhythmias before bypass—demanding immediate defibrillation from chest entry. Giant pseudoaneurysms erode into adjacent structures (LA, pulmonary veins, lung). Our patient's anterolateral/inferolateral involvement placed structures at risk during cardiectomy. Thromboembolic risk exists; echo noted possible thrombus requiring minimal manipulation before cross-clamping. Massive hemorrhage is inevitable given large ostium, adhesions, anticoagulation, CPB coagulopathy—requiring hemostatic resuscitation beyond routine OHT. Successful outcome despite arrest, massive transfusion, and organ dysfunction demonstrates that anticipating pseudoaneurysm-specific complications enables excellent results. OHT for giant pseudoaneurysm differs fundamentally: the native heart threatens rupture with every manipulation. Preparation for emergent cannulation, arrhythmia management, massive transfusion, and organ injury must guide planning.

Images



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#FR 3 – Kiosk 24 – Poster 4

The Wiggly Wall: An Overly Mobile Septum Causing Trouble

Presenting Author: Saad Ali, MD, Fellow, Robert Wood Johnson Medical School

Introduction

Atrial septal aneurysm (ASA) is a rare structural abnormality characterized by bulging septal tissue. Although often incidental, it can be associated with an atrial septal defect (ASD) and predisposes to embolic events. Uncommonly, an ASA may result in symptomatic functional obstruction. Echocardiographically, an ASA is typically defined as interatrial septal excursion ≥10–15 mm beyond the plane of the septum (1). ASAs may also act as an arrhythmogenic substrate, predisposing patients to atrial tachycardia, flutter, or fibrillation (2). In combination with impaired atrial hemodynamics, this can increase thromboembolic risk. Because not all ASAs are deleterious, selecting patients for surgical intervention is challenging. We present a case of symptomatic ASA causing hemodynamic compromise, whose repair subsequently resulted in a new postoperative arrhythmia.

Case Presentation

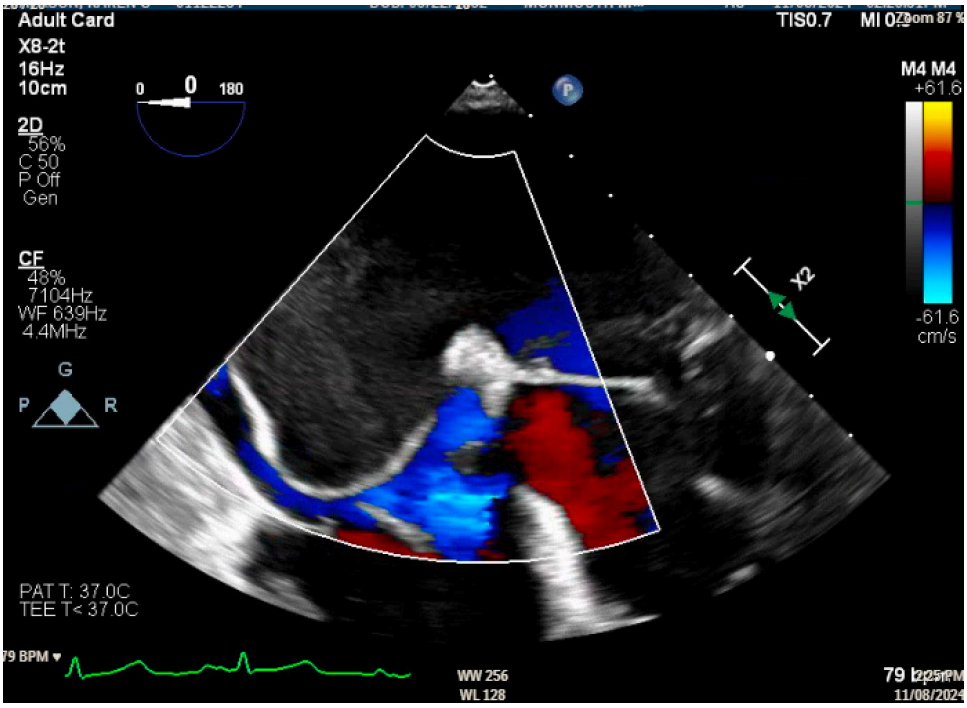
KJ, a 73-year-old woman with hypertension, diabetes, and prior brain aneurysm repair, presented with three recurrent “drop” episodes marked by abrupt lightheadedness and loss of consciousness with immediate recovery. Medical management was insufficient. A TTE seven years earlier had reported an ASD; however, repeat TTE identified an ASA causing inflow obstruction. Holter monitoring showed atrial tachycardia. TEE confirmed a mobile ASA with severe spontaneous echo contrast. She was started on apixaban and scheduled for surgical repair. Under general anesthesia, intraoperative TEE demonstrated a highly mobile septum that, with Valsalva, intermittently obstructed either atrioventricular valve depending on loading conditions. The septum was plicated in two layers and the left atrial appendage (LAA) was ligated. Repeat TEE showed resolution of aneurysmal motion and an absent LAA. Although she remained in sinus rhythm intraoperatively, she developed new rate-controlled atrial fibrillation postoperatively, which was treated with amiodarone and converted to sinus rhythm. She was discharged on amiodarone but without anticoagulation. She remained asymptomatic for 58 days until a single syncopal episode with a slower, more gradual onset, differing from her previous “drop” attacks. This was likely related to hypoglycemia or higher-dose metoprolol rather than recurrent obstruction or arrhythmia, as she remained in normal sinus rhythm.

Discussion

Anticoagulation was difficult to balance, as spontaneous echo contrast suggested thromboembolic risk while frequent falls increased harm. Surgical repair with LAA ligation was therefore pursued. ASA was likely congenital and progressed with comorbidities. With impaired diastolic function, even modest obstruction may produce hypotension and syncope. ASA provides an arrhythmogenic substrate, and its excision may convert focal tachycardia into a more disorganized rhythm. This case illustrates how congenital abnormalities may remain silent for decades before manifesting due to cumulative structural and electrical

atrial remodeling, creating challenges in choosing between medical and surgical management.

Images



Midesophageal 4 chamber view of atrial septal aneurysm (ASA) with spontaneous echo contrast and obstruction of outflow from Right Atrium to Right Ventricle

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#FR 3 – Kiosk 24 – Poster 5

Open embolectomy for pulmonary embolism in transit across patent foramen ovale in a 44 year old female with history of hydrocephalus and recent attempt at repair of cerebrospinal fluid leak

Presenting Author: Bryan Lynn, MD, Fellow, Emory

Introduction

Surgical pulmonary embolectomy (SPE) for acute pulmonary embolism (PE) was originally described by 1908 and first successfully performed in 1924. With advances in surgical technique and improvements in perioperative care, SPE is now considered a safe and effective technique. 2019 European Society of Cardiology (ESC) guidelines for management of acute PE include SPE as an alternative to rescue thrombolysis in patients with contraindications, and as an equivalent therapy to percutaneous catheter-based techniques. Other indications for SPE include clot-in-transit, massive clot burden in the pulmonary vasculature, and failed catheter-directed intervention.

Case Presentation

44 yo F with history of hydrocephalus and sinonasal cerebrospinal fluid (CSF) leak, had recent endoscopic repair using a pericranial flap. She was readmitted with PE and found to have embolus in transit across a patent foramen ovale (PFO). She underwent emergent open embolectomy, extraction of intracardiac thrombi, and PFO closure. Given her recent CSF leak procedure, and evidence of continued sinonasal CSF leak, her airway was managed as a rapid sequence intubation. Cardiopulmonary bypass was weaned with the assistance of inotropes given her moderate right ventricular dysfunction at baseline. She was extubated on postoperative day (POD) #1, had an inferior vena cava (IVC) filter placed on POD #7, then was discharged home with oral anticoagulation.

Discussion

Surgical intervention of acute PE has emerged as a safe and effective modality of treatment. One retrospective analysis showed that SPE had decreased mortality, bleeding complications and neurological events when compared to thrombolytic therapy. This study also found no difference in 5-year outcomes between surgical intervention and thrombolysis. Our patient had clot-in-transit (CIT), defined as mobile echogenic material in the right heart. Right sided clot is a rare finding in the absence of structural heart disease, atrial fibrillation, or an indwelling catheter. About 4% of patients with acute PE have a mobile embolus present in the right heart; these two findings are associated with a 40% mortality. Type A CIT is the most common and has a snake-like appearance. It carries the greatest risk of embolization either systemically if a PFO/atrial septal defect (ASD) exists, or into the pulmonary vasculature. Type B CIT are broad based, adherent to a chamber wall and are thought to originate from the atrium or ventricle. Type C CIT are the rarest subtype of thrombi, appear similar to cardiac myxomas, and are very prone to migration. Anesthetic management of PE patients is dependent on the planned intervention (catheter-based or open), and overall hemodynamic stability of the patient. Transesophageal echocardiography (TEE) is vital for risk stratification, detection of intracardiac thrombi, characterization of CIT, and identifying intracardiac shunts.

Patients may require inotropes, pulmonary artery vasodilators, or advanced mechanical circulatory support (MCS), dependent on baseline hemodynamics and TEE findings. As SPE becomes a more common modality of treatment, it is important that cardiac anesthesiologists can identify and effectively communicate pertinent findings to help guide management.

Images

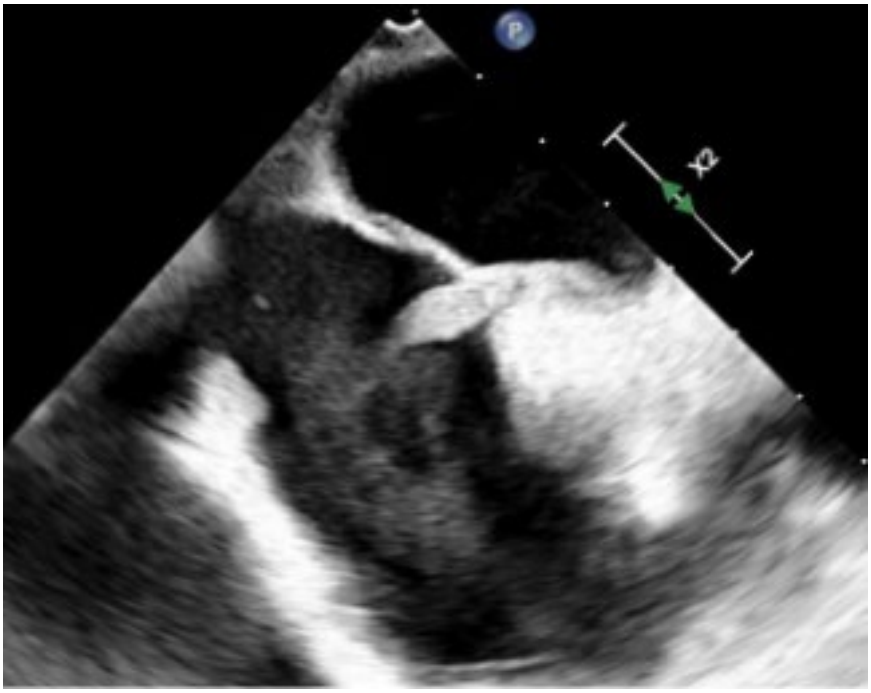


Figure 1 Midesophageal bicaval view showing thrombus across PFO



Figure 1 Thrombus after open resection

References

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#FR 3 – Kiosk 24 – Poster 6

Team Dynamics and Pharmacodynamics: Managing Coagulopathy After Tenecteplase Administration In An Acute Type A Aortic Dissection

Presenting Author: Simran Mand, MD, Stanford Healthcare

Introduction

In patients with suspected ischemic stroke without exclusion criteria, administration of IV thrombolysis (IVT) within 3 hours of symptom onset is standard of care. However, IVT is contraindicated in known or suspected type A aortic dissection (TAAD) due to risk of aortic rupture and uncontrolled coagulopathy during open aortic repair.[1] While aortic repair often re-establishes cerebral perfusion, select patients may benefit from undergoing endovascular intervention first. The decision to proceed with aortic surgery after recent IVT and the order in which to intervene on carotid malperfusion both require multidisciplinary decision making. We present a case highlighting these dilemmas in a patient with acute TAAD complicated by cerebral malperfusion and severe coagulopathy due to IVT administration.

Case Presentation

A 76-year-old female with no past medical history presented to an outside hospital with left hemiparesis, headache, and nausea. As last known normal was 1.5 hours prior and head CT showed no hemorrhage, IVT was initiated with 22.5 mg of Tenecteplase (TNK). Subsequent CT angiography of the head and neck unexpectedly revealed a TAAD extending into the right internal carotid artery (R ICA), and she was emergently transferred to our institution for surgical management. On arrival to our OR 2.75 hours after TNK administration, she was placed under general anesthesia. Initial thromboelastogram (TEG) was severely abnormal (Figure 1A), and aortogram showed static malperfusion of the R ICA. A multidisciplinary decision was made to first stent the R ICA to restore cerebral perfusion while attempting to correct the coagulopathy. Cryoprecipitate, fresh frozen plasma, and platelets were administered with repeat TEG showing improvement (Figure 1B). Additional products were administered prior to sternotomy, and she successfully underwent supracoronary ascending aorta and hemiarch replacement with correction of the IVT-associated coagulopathy prior to weaning from cardiopulmonary bypass (Figure 1C). Post-operative course has been notable for persistent left hemiplegia and respiratory failure requiring tracheostomy, and she remains inpatient at the time of submission.

Discussion

Multidisciplinary communication is essential in complex clinical scenarios. In this case, the presence of static cerebral malperfusion led the surgical team to stent the carotid prior to aortic repair while the anesthesiology team worked to treat IVT-associated coagulopathy.2 Repeated discussions were held regarding the risks and benefits of proceeding with open repair while severely coagulopathic; given concern for impending aortic rupture, both teams agreed to proceed. Open communication was as crucial as aggressive management of coagulopathy in this case, with both teams listening and exchanging information freely as the case evolved.

Images

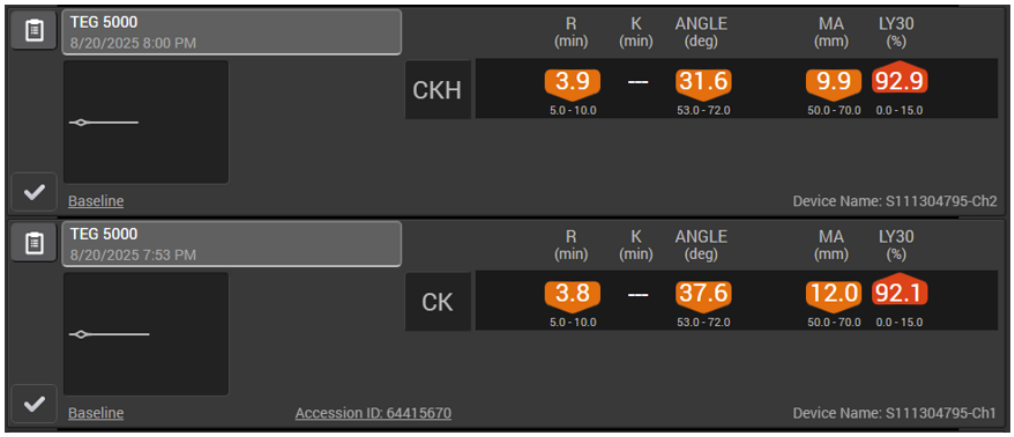


Figure 1A: Baseline TEG obtained upon patient arrival in the operating room.

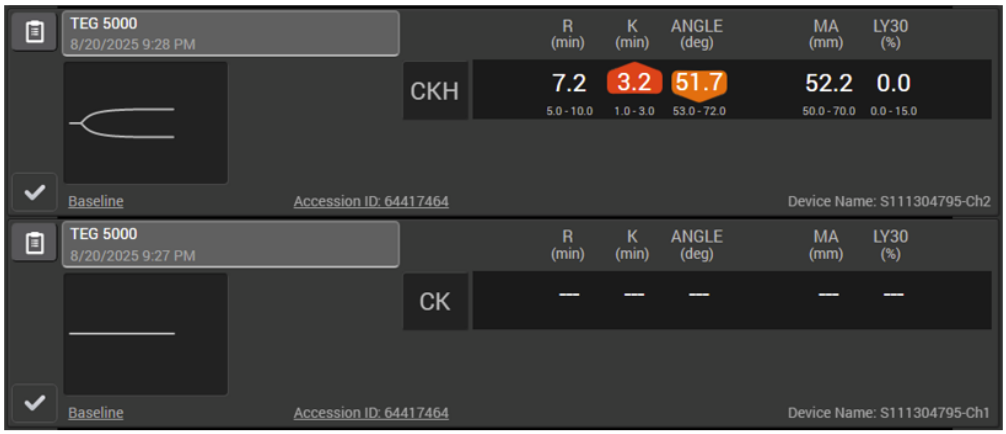


Figure 1B: Repeat TEG after administering 4 units fresh frozen plasma, 10 pooled units cryoprecipitate, and 1 unit platelets. The difference between the citrated kaolin (CK) and citrated kaolin with heparinase (CKH) channels reflects IV heparin administration for carotid stent placement.

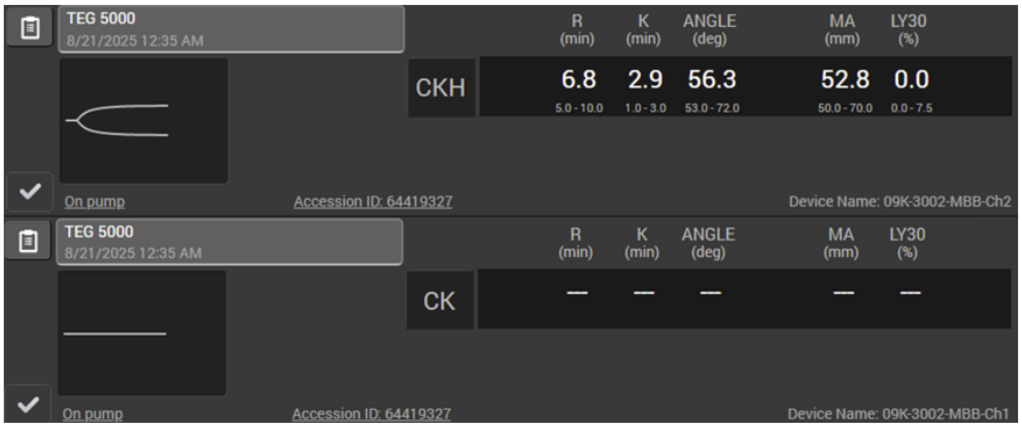


Figure 1C: Final TEG performed near the end of the cardiopulmonary bypass run while still fully heparinized; the CKH channel shows correction of the underlying TNK-associated coagulopathy.

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#FR 3 – Kiosk 24 – Poster 7

Profound right-to-left shunt during transcatheter mitral valve replacement: The power of pressure gradients

Presenting Author: Marc D. Locke, DO, Anesthesia Resident, University of Connecticut

Introduction

Transcatheter mitral valve replacement (TMVR) requires transseptal access of the left atrium (LA) for valve deployment, often resulting in an iatrogenic atrial septal defect (iASD) that can persist in 24-50% of patients1. Individuals with mitral valvulopathy frequently exhibit sequelae such as pulmonary hypertension, right-sided heart failure, and/or tricuspid regurgitation (TR). placing them at increased risk for clinically significant right-to-left shunting and rapid hemodynamic decompensation.

Case Presentation

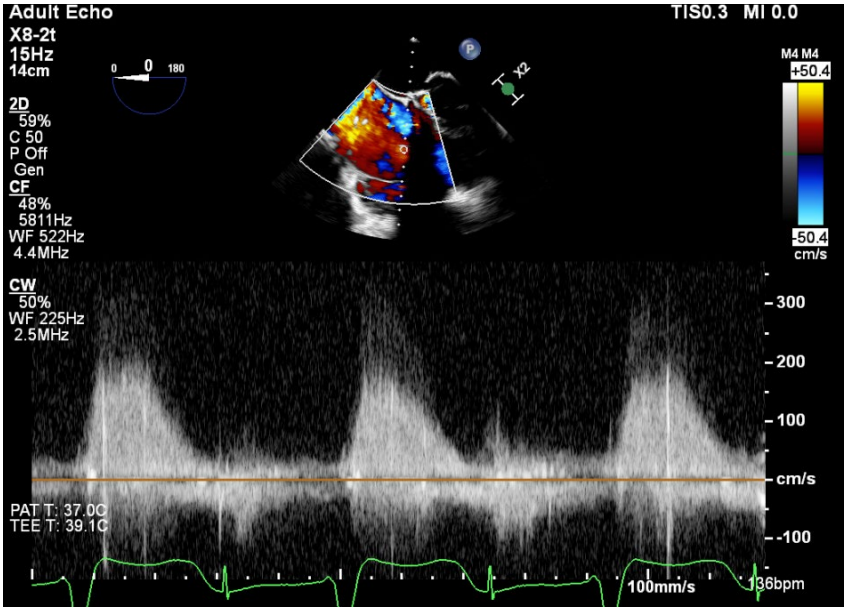
The patient is an 83-year-old female with a history of mitral valve disease and prior bioprosthetic mitral valve replacement in 2013 who presented for planned TMVR after worsening exertional fatigue and dyspnea. Transesophageal echocardiography (TEE) showed a bioprosthetic valve with moderate stenosis (area 1.4 cm^2, mean gradient 8 mmHg), normal left ventricular ejection fraction (LVEF), a severely enlarged right ventricle (RV), and severe TR (Figure 1). Left heart catheterization demonstrated normal coronaries (Figure 2), while right heart catheterization confirmed pulmonary hypertension with right-sided heart failure (Figure 3). She was not considered a candidate for an open heart procedure given her RV dysfunction. Standard ASA monitors were applied, and anesthesia was induced with propofol, fentanyl, rocuronium, and vasopressin, followed by uneventful endotracheal intubation. An arterial line was placed, anesthesia was maintained with sevoflurane, and hemodynamic support was provided with a norepinephrine infusion. $ETCO_2$ was maintained at 30 mmHg to prevent further elevation in pulmonary vascular resistance. An RV pacing wire was inserted, and heparin was given for anticoagulation. Under TEE guidance, septostomy and valve deployment during rapid pacing were completed without complication. Upon removal of the delivery system, the patient experienced a sudden drop in $ETCO_2$ and SpO_2, followed by hypotension. TEE revealed a moderate-to-severe iASD with significant right-to-left shunting (Figure 4). Despite septal occlusion, prolonged CPR took place without return of spontaneous circulation.

Discussion

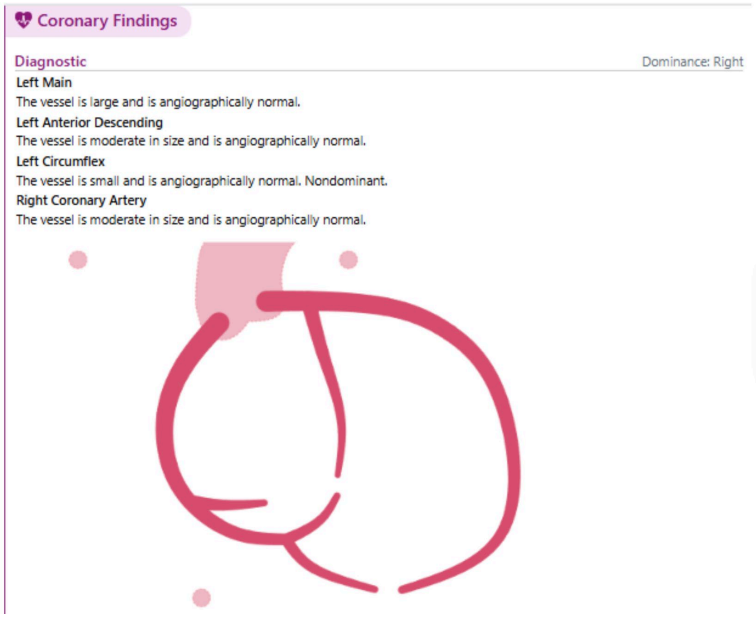
Although often clinically insignificant, iASDs can become hemodynamically dangerous in individuals with elevated right-sided pressures, pulmonary hypertension, or RV dysfunction. The sudden decrease in $ETCO_2$ and SpO2 with initial hemodynamic stability immediately after removal of the delivery system strongly suggested acute intracardiac shunting rather than valve failure or ischemic insult. While recurrent ventricular fibrillation suggested that severe and sustained hypoxemia likely precipitated myocardial ischemia and electrical instability. Once critical impairment of systemic oxygen delivery occurred, meaningful recovery became increasingly unlikely despite prompt mechanical correction of the shunt. Although emergency cardiopulmonary bypass is an option in similar situations, the patient and family

agreed to avoid emergency open heart surgery in the setting of uncontrollable hemodynamic instability. This case underscores the importance of careful preprocedural assessment of RV function and pulmonary pressures, early recognition of shunt-related instability, and consideration of proactive strategies to minimize or promptly close iASDs in high-risk TMVR patients.

Images



Pre-operative TEE midesophageal 4-chamber view demonstrating severe tricuspid regurgitation on color Doppler.



Coronary Findings

Diagnostic — Dominance: Right

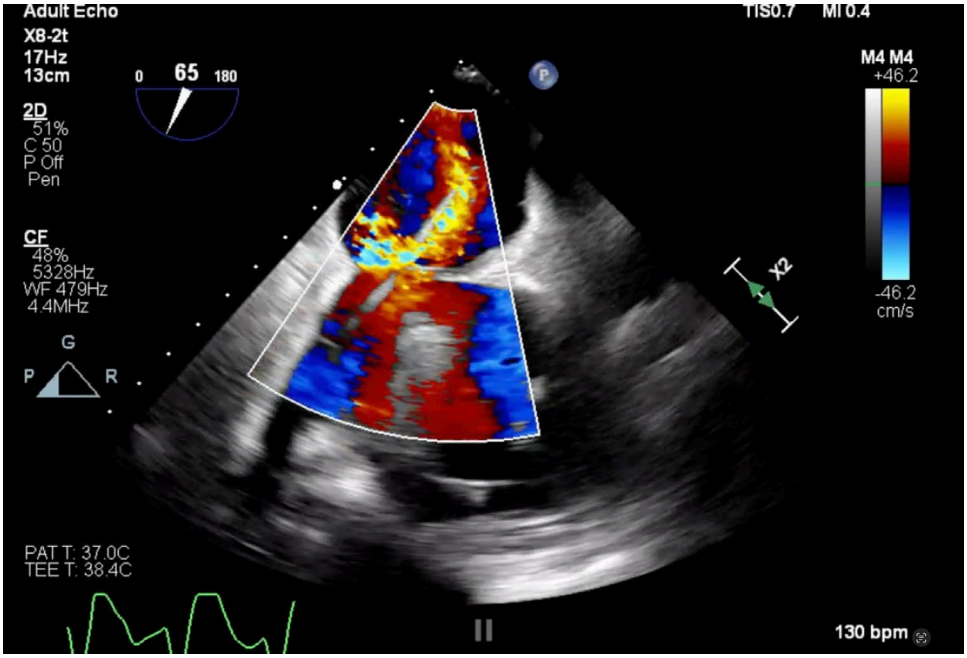
Left Main
The vessel is large and is angiographically normal.
Left Anterior Descending
The vessel is moderate in size and is angiographically normal.
Left Circumflex
The vessel is small and is angiographically normal. Nondominant.
Right Coronary Artery
The vessel is moderate in size and is angiographically normal.

Left heart catheterization demonstrating normal coronary arteries

Pressures AIR REST

	Systolic (mmHg)	Diastolic (mmHg)	Mean (mmHg)	A Wave (mmHg)	V Wave (mmHg)	EDP (mmHg)
RA			27	34	42	
			26	34	26	
RV	41	12				22
PA	46	20	35			
	47	24	35			
PW			23	28	27	
			23	27	26	
AO	111	58	79			
	110	58	79			
	0	0	0			
	64	49	56			
	114	58	80			
LV	114	4				24
	117	9				19
	119	7				16
IVC	19	42	27			

Left heart catheterization demonstrating pulmonary hypertension



Post-valve deployment TEE midesophageal modified bicaval view demonstrating iASD with right-to-left shunt

References

Schueler R, Öztürk C, Wedekind JA, Werner N, Stöckigt F, Mellert F, Nickenig G, Hammerstingl C. Persistence of iatrogenic atrial septal defect after interventional mitral valve repair with the MitraClip system: a note of caution. JACC Cardiovasc Interv.2015;8:450–459. doi: 10.1016/j.jcin.2014.10.024

#FR 3 – Kiosk 25 – Poster 1

A Right-Sided Aberration: Endocarditis of the Pulmonic Valve Amidst Multivalvular Disease

Presenting Author: Alessandro Sarno, DO, Anesthesiology Resident, University of Kentucky

Introduction

Infective endocarditis (IE) has high morbidity and mortality. Its risk factors include valvular abnormalities, prosthetic valves, indwelling catheters, cardiac devices, immunosuppression, poor dentition, and intravenous drug use (IVDU). The mitral valve (MV) is most frequently affected, followed by the aortic valve (AV), tricuspid valve (TV) which is classically involved in patients with IVDU, and least commonly, the pulmonic valve (PV). PV involvement is rare due to low shear stress, larger orifice, favorable flow, reduced

venous bacterial load, and rare baseline valvulopathy. We present a 45-year-old man with complex IE involving the PV, AV, and TV, who successfully underwent multivalve surgery.

Case Presentation

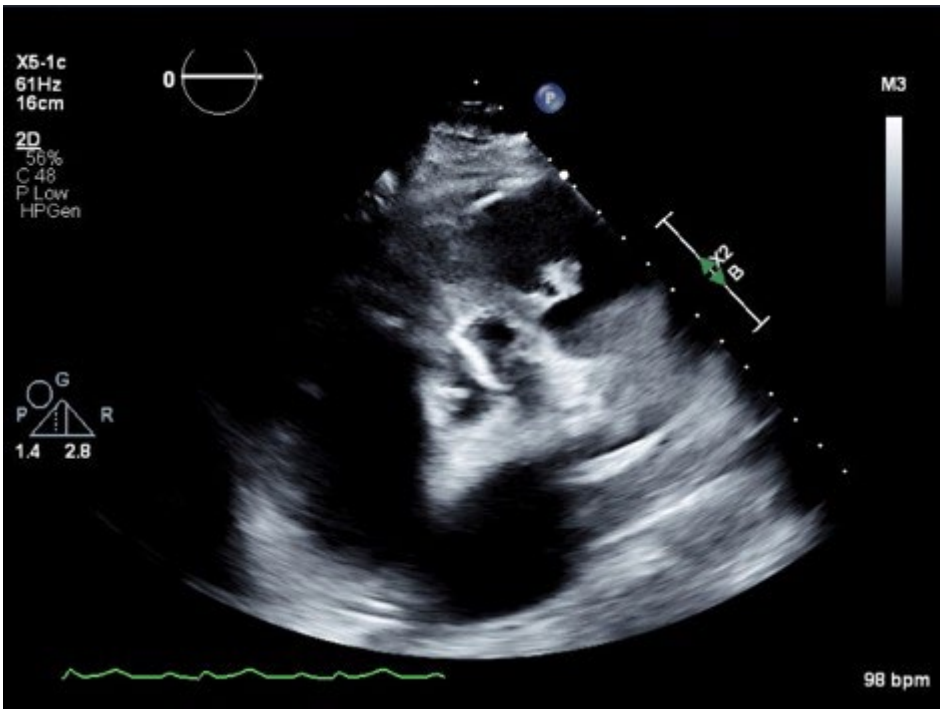
A 45-year-old man with a history of ventricular septal defect with spontaneous closure, hypertension, anxiety, and hyperlipidemia who presented with dyspnea, fatigue, lower extremity edema, and fevers. These symptoms began a few months after a ventral hernia repair. Initial laboratory work-up revealed anemia and acute kidney injury. CT imaging revealed pulmonary emboli with infarcts and pulmonary nodules with cavitary changes (Image 1). Transthoracic echocardiography (TTE) showed severe aortic regurgitation (AR) with an echo-free space anterior to the AV, concerning for perivalvular abscess, mild tricuspid regurgitation, and a large PV vegetation (Image 2). Preoperative transesophageal echocardiography (TEE) confirmed a periaortic abscess with erosion of the non-coronary cusp of the AV, severe AR (Images 3 and 4), a vegetation on the ventricular side of the TV with moderate regurgitation (Image 5), and a large PV vegetation with severe regurgitation (Image 5). Blood cultures grew Streptococcus mutans. Intraoperative TEE also demonstrated a small patent foramen ovale (PFO) which was closed. The patient underwent successful replacement of the AV, TV, and PV and was discharged on postoperative day 16.

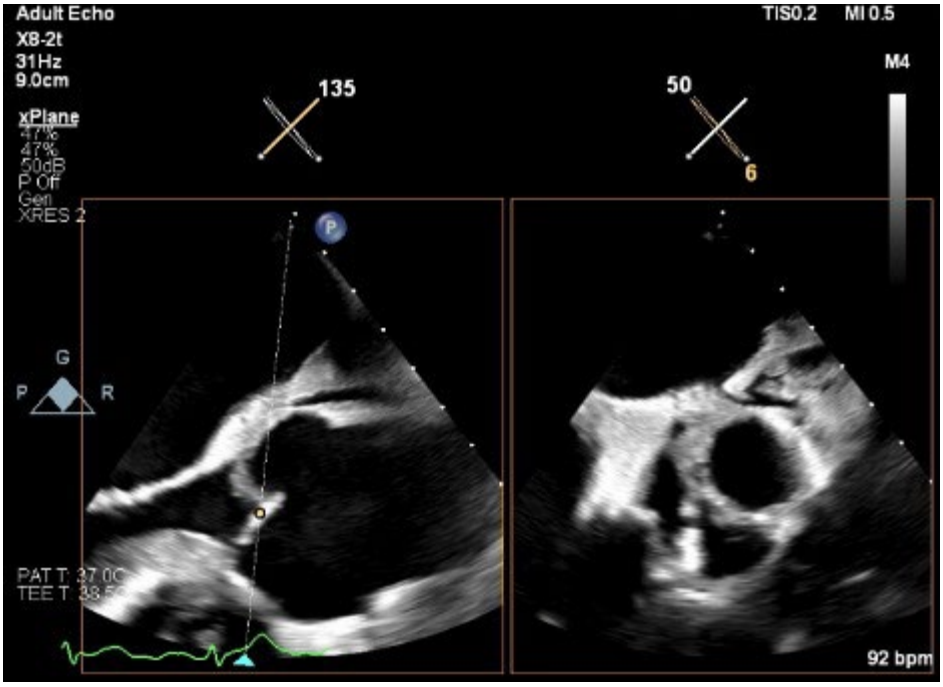
Discussion

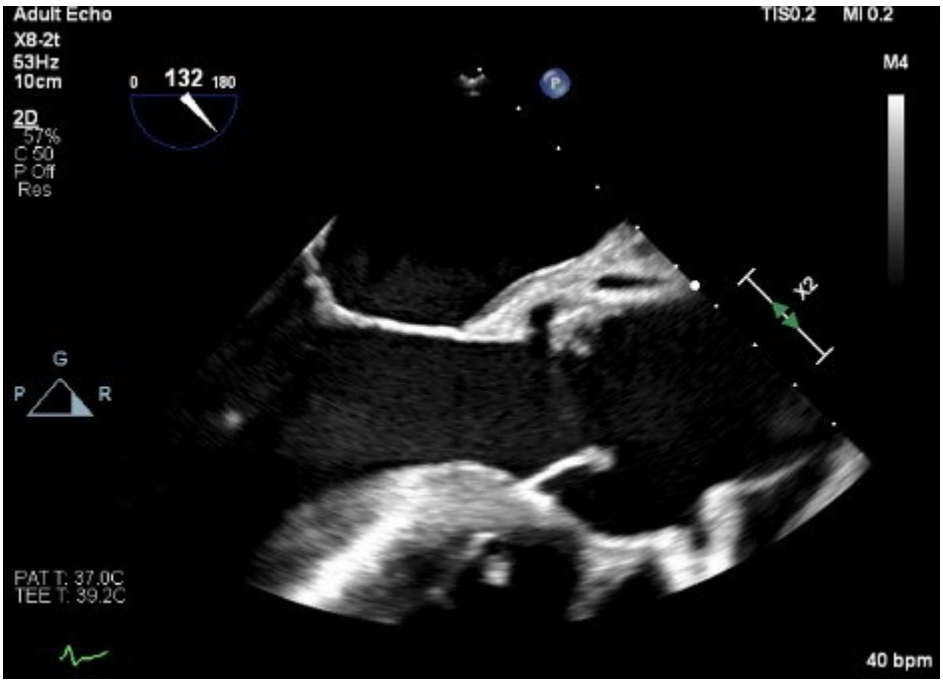
IE is a serious infection of the endocardium. The most frequent etiologic agents are Streptococcus (50–80%) and Staphylococcus (20–30%) species [1]. Risk factors include age, sex, structural heart disease, IVDU, congenital cardiac abnormalities, prior IE, and poor dentition. The incidence of IE is increasing, rising from 11 to 15 cases per 100,000 people between 2000 and 2011 [2]. IE commonly involves cardiac valves, with the MV affected most frequently, while PV involvement is exceedingly rare [1]. Multivalvular IE is also uncommon, occurring in 18% of cases, whereas PV involvement is reported in 1–2% of cases [2]. PV IE is often associated with congenital cardiac abnormalities, cardiac devices, central venous catheters, and IVDU [3]. This case represents a rare presentation of multivalvular endocarditis with PV involvement. Streptococcus mutans typically associated with left-sided IE has been reported to cause right-sided inoculation in the presence of intracardiac shunts [3]. Given this patient's history of PFO and VSD, shunt physiology is a plausible mechanism for bacterial seeding. Outcomes are poorer when multiple valves need to be replaced: mortality rises from ~18% for single-valve replacement to 20–25% when three or more valves require replacement.

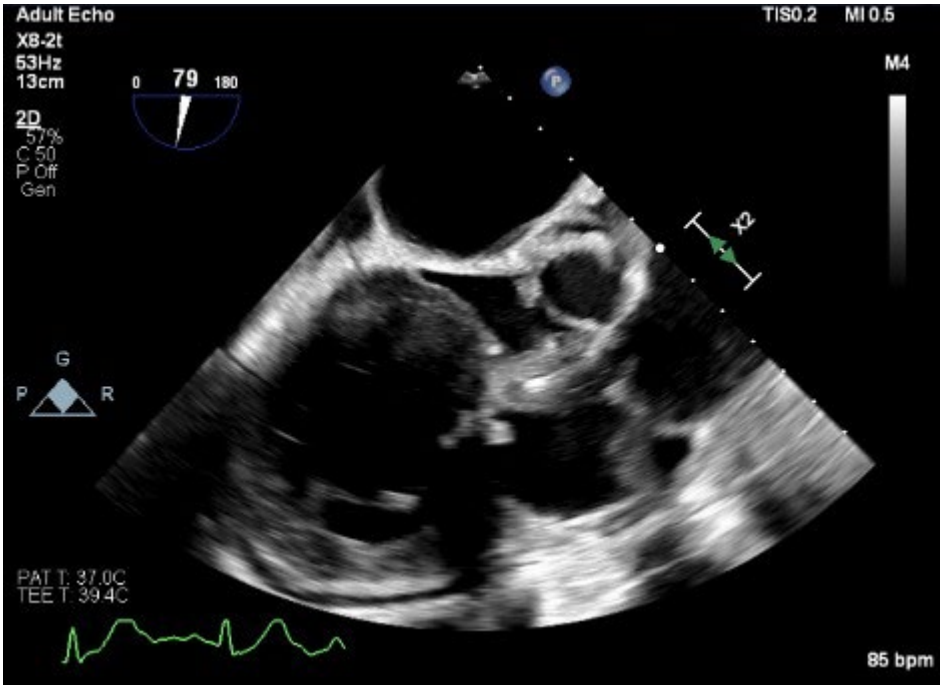
Images




X5-1c
61Hz
16cm
2D
56%
C 48
P Low
HPGen
0
M3
G
P
R
1.4
2.8
98 bpm


Adult Echo
X8-2t
31Hz
9.0cm
xPlane
47%
47%
50dB
P Off
Gen
XRES 2
TIS0.2
MI 0.5
M4
135
50
6
G
P
R
PAT T: 37.0C
TEE T: 38.5
92 bpm


Adult Echo
X8-2t
53Hz
10cm
2D
57%
C 50
P Off
Res
TIS0.2
MI 0.2
M4
0
132
180
X2
G
P
R
PAT T: 37.0C
TEE T: 39.2C
40 bpm



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#FR 3 – Kiosk 25 – Poster 2

Intraoperative Anesthetic Considerations for Repair of a Pulmonary Artery Aneurysm in a Marfan Syndrome Patient with Severe Pulmonary Insufficiency

Presenting Author: Dania Hussein, MD, New York Presbyterian Hospital Weil Cornell

Introduction

Marfan Syndrome is a connective tissue disorder with frequent cardiovascular involvement. Dilation of the main pulmonary artery (PA) occurs in up to 69% of affected individuals, although true pulmonary artery aneurysms (PAAs) are very rare [1]. This case discusses a patient with Marfan Syndrome and a history of previous cardiac surgeries presenting with severe pulmonary insufficiency (PI) and PAA.

Case Presentation

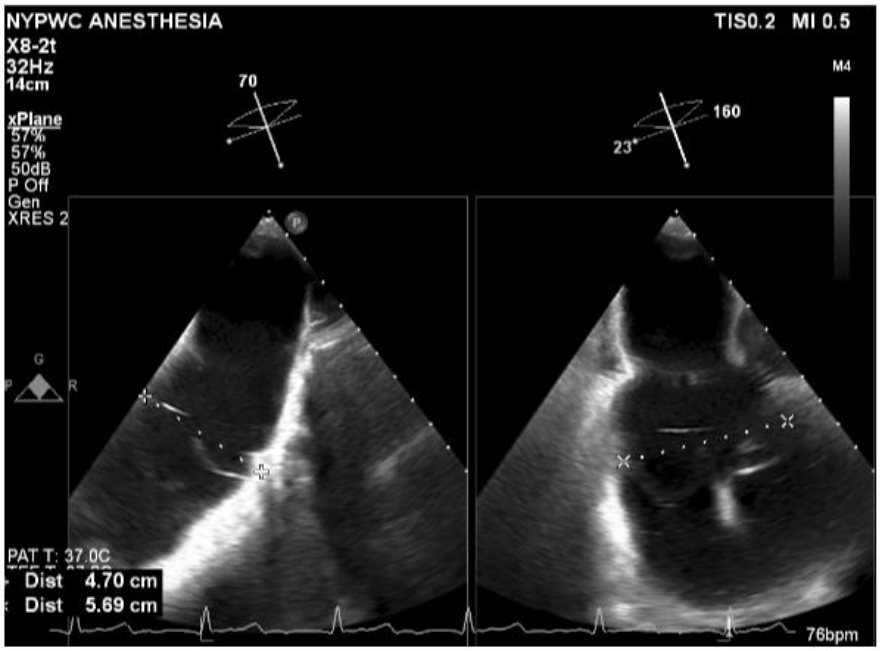
63 yo M with PMH Marfan Syndrome, atrial fibrillation and pulmonary hypertension (pHTN) who had a previous mitral valve repair and valve-sparing aortic aneurysm repair with subsequent severe aortic insufficiency requiring a re-operation with a mechanical composite graft. He presented with increasing shortness of breath and was found to have a 5.2 cm PAA in the main PA with severe PI and severe right ventricular (RV) dilation. The patient underwent a re-operation, PA repair, pulmonary valve replacement, aortic valve replacement, and a tricuspid valve repair. On pre-bypass transesophageal echo (TEE), the patient's PAA measured 5.69 cm and his RV was severely dilated with mildly reduced function. Given the patient's large pulmonary annulus, an inverted bioprosthetic mitral valve was used for the PV replacement. Goal-directed transfusion was used intraoperatively to avoid overloading the RV and the mean pressure was maintained to ensure adequate RV perfusion. Importantly, PA catheter derived cardiac outputs may not be accurate in these patients and were not used to direct therapy. Post-bypass TEE revealed a well-seated valve with no regurgitation. The main anesthetic goal was to manage systemic, pulmonary and right heart pressures given his PAA, documented pHTN, and RV size/function. His anesthetic included fentanyl, milrinone infusion, and dexmedetomidine. Since the patient's PA pressures were not severely elevated throughout the case and his post-bypass function was normal (>55%) inhaled nitric was not used. He was started on 4 mcg/min of epinephrine to support his biventricular function. His intraoperative course was complicated by persistent arrhythmias requiring 2 amiodarone boluses, an infusion and multiple defibrillations. In the intensive care unit the patient's chest tube output acutely increased requiring a chest exploration and blood product resuscitation.

Discussion

There are many anesthetic considerations for a surgical repair of a PAA. Intraoperative management goals include avoiding increases in the pulmonary vascular resistance (PVR) and stress on the PA walls while supporting RV function. Additionally, emphasis is placed on monitoring RV size and function, the new valve function/placement and possible presence of new tricuspid regurgitation using TEE, central venous pressures and PA pressures. [2] RV support is possible using inotropic agents such as milrinone or by decreasing PVR through agents such as inhaled nitric oxide. Additionally, to avoid increases in PVR, care

should be taken to avoid hypoxemia, hypercapnia, hypothermia, and acidosis. While PAAs are rare and dangerous, careful surgical and anesthetic planning can lead to proper and safe patient care and in our case, despite severe RV dilation there was minimal tricuspid regurgitation and the patient only required moderate RV support.

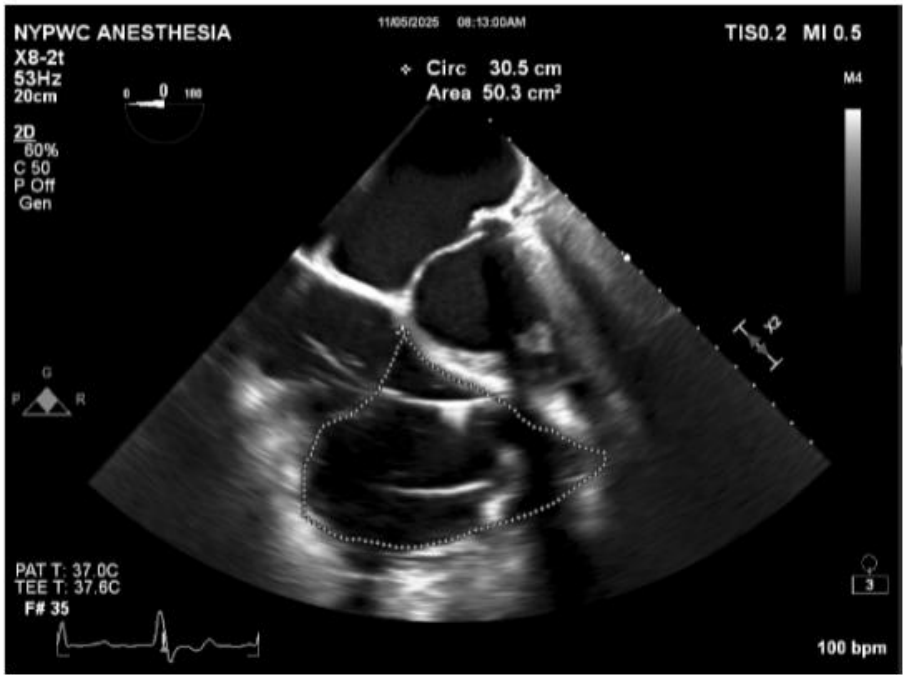
Images



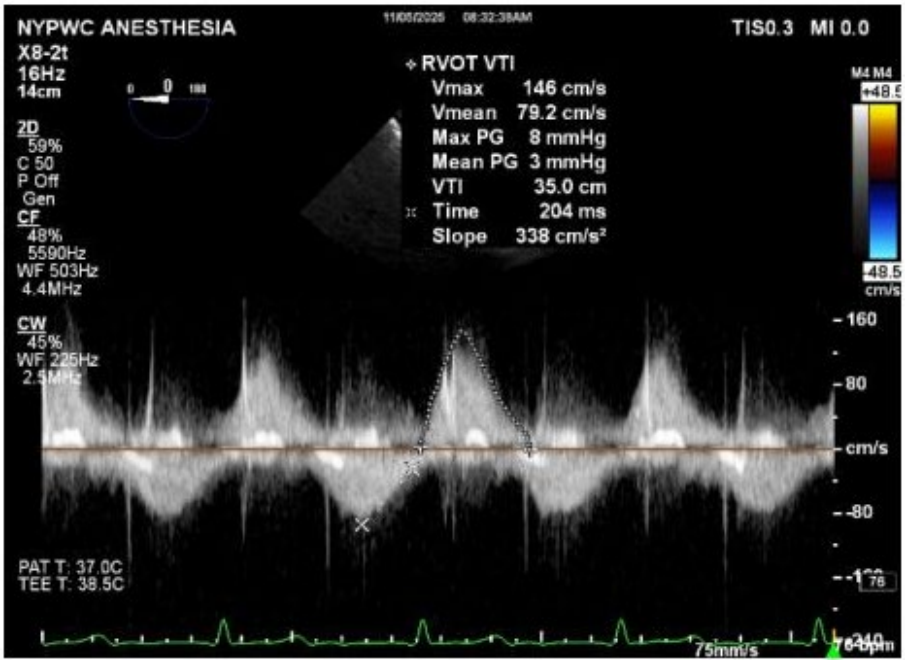
PV Annulus and PA Diameter



Tricuspid Annulus



Preoperative RV Size



RVOT VTI

References

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Balcı E, Demir A, Özgök A, Karadeniz Ü. Anaesthesia Management in Idiopathic Pulmonary Artery Aneurysm Surgery. Turk J Anaesthesiol Reanim. 2019 Dec;47(6):503-506. doi: 10.5152/TJAR.2019.21447. Epub 2019 Dec 1. PMID: 31828249; PMCID: PMC6886819.

#FR 3 – Kiosk 25 – Poster 3

"In the Groove": Surgery for Aortomitral Mass Complicated by Atrioventricular Groove Disruption

Presenting Author: Allyson F. Tragesser, MD, Resident Physician, University of Florida

Introduction

Intraoperative management of complex valvular pathology involving the aortomitral curtain poses significant anesthetic and surgical challenges. We present a 71-year-old female with severe aortic and mitral valve pathology requiring surgical intervention. Her surgery was complicated by atrioventricular groove dissociation (AVD) requiring extensive repair. Final pathology revealed IgG4-related disease (IgG4-RD). This case underscores the diagnostic uncertainty, intraoperative complexity, and multidisciplinary management associated with valvular manifestations of systemic inflammatory disease.

Case Presentation

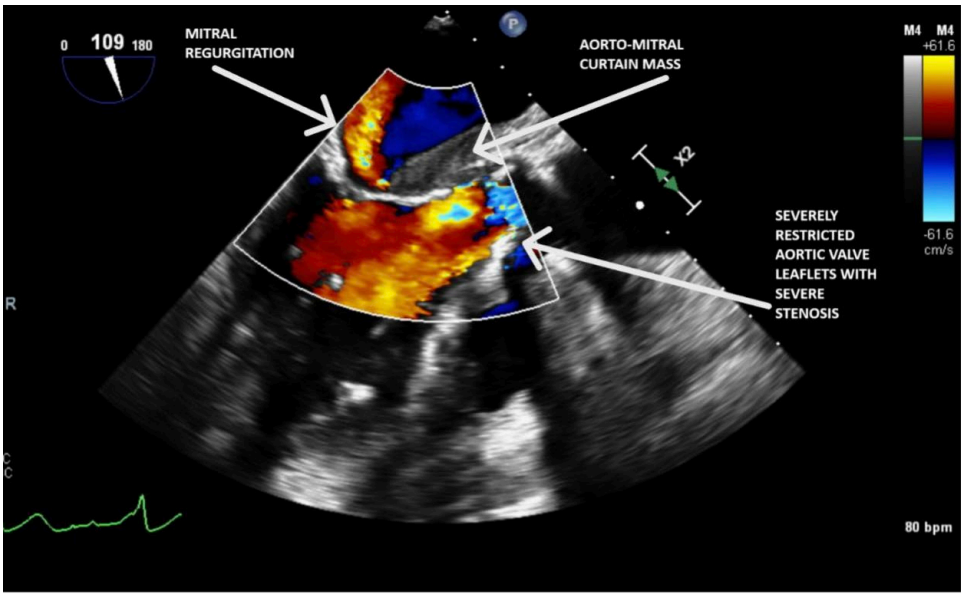
A 71-year-old female with hypertension, hypothyroidism, and rheumatoid arthritis presented with worsening dyspnea on exertion and recent complete heart block status post permanent pacemaker. Echocardiography revealed moderate aortic stenosis/regurgitation and moderate-to-severe mitral regurgitation. Cardiac MRI demonstrated a mass around the aortic annulus. She was transferred to Shands for aorto-mitral curtain reconstruction. Following uneventful induction and line placement, sternotomy revealed thick, immobile leaflet cusps and aortomitral curtain. Frozen pathology showed inflammatory cells and fibrosis without mitotic activity. Intraoperative TEE findings were discussed with the surgeon, and AVR/MVR were performed without curtain reconstruction. Upon weaning from bypass, bleeding was noted between the left atrium and ventricle, consistent with AVD. The patient was placed back on bypass, cooled, and underwent explantation and reimplantation of both valves after bovine pericardial patch repair of the groove via an internal approach after failed external repair. Multiple blood products and prothrombin complex concentrate were given. Final TEE demonstrated normal prosthetic valve and biventricular function. Pathology revealed IgG4-positive plasma cells. She was diagnosed with IgG4-RD, treated with steroids and rituximab, and discharged after ICU recovery.

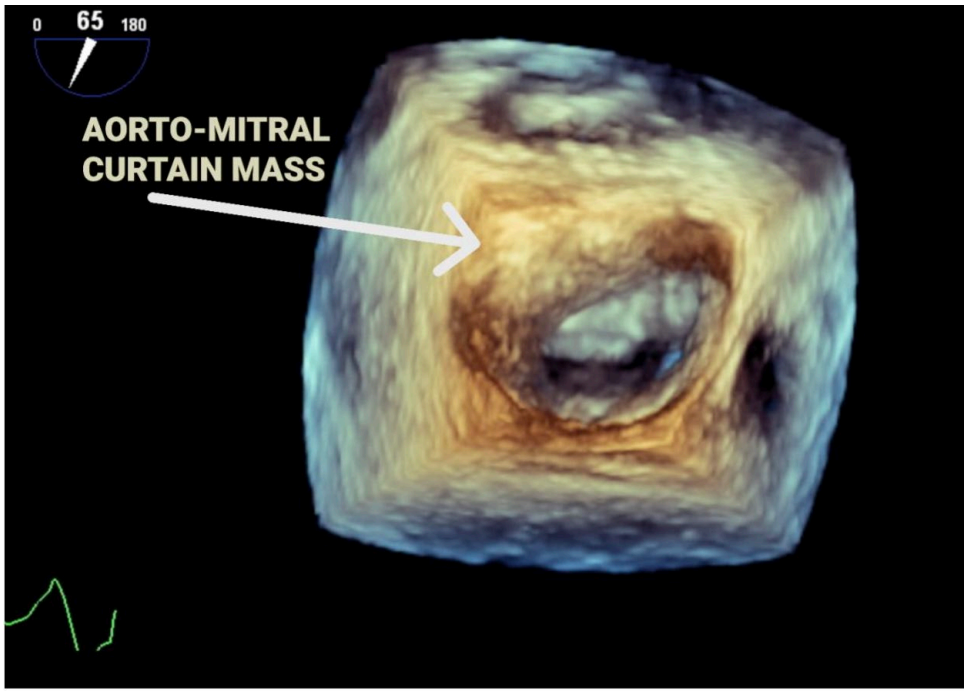
Discussion

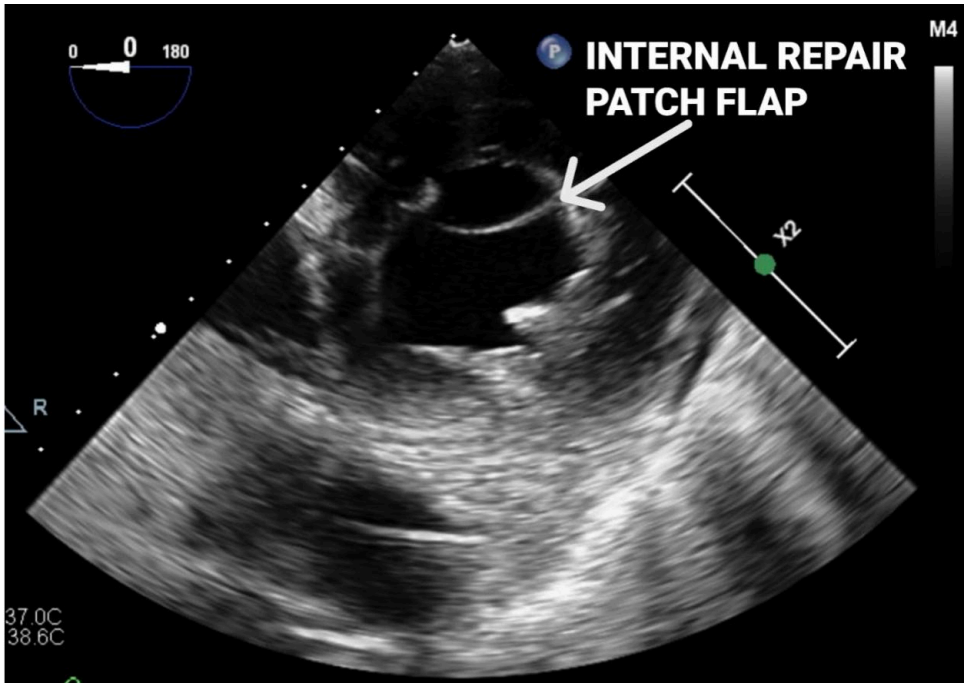
This case highlights the need for a broad differential when evaluating aortomitral masses. Potential etiologies include infectious endocarditis, Lambl's excrescences, papillary fibroelastoma, nonbacterial thrombotic endocarditis, valvular myxoma, and calcified amorphous tumors. IgG4-RD is a rare systemic fibroinflammatory disease (prevalence » 5.3 per 100,000) with exceptionally uncommon cardiac involvement[1]. It often mimics autoimmune and connective tissue disorders, though typically affects males over 50[1]. AVD is an infrequent but highly lethal complication of mitral valve surgery, with reported incidence ranging from 0.4–2.3% and mortality approaching 90% if untreated[2]. In a multicenter review, AVD occurred in 0.42% of mitral valve cases, with 30-day mortality of 15% and hospital mortality of 23%. Risk factors include age >60, mitral valve surgery, and tissue fragility[2,3]. Internal and external repairs have been described; the internal approach, successful in this case, is technically demanding[3], but enabled successful groove

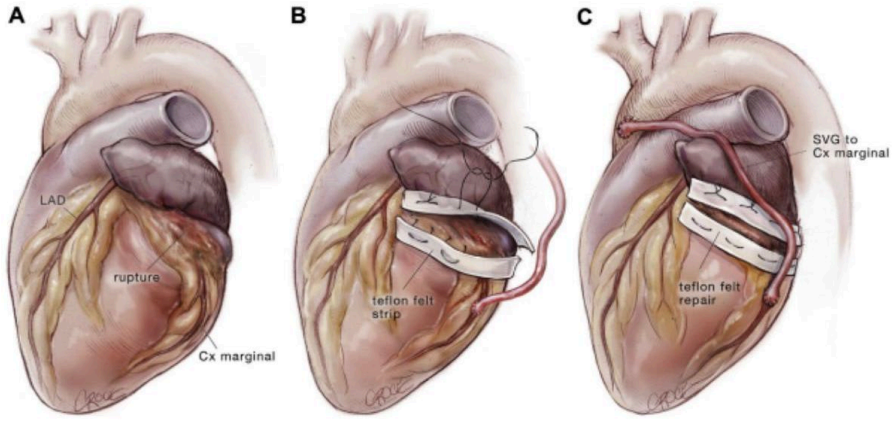
reconstruction and valve reimplantation. This case emphasizes the need for anesthesiologists to anticipate hemodynamic instability, manage complex transfusion requirements, and collaborate closely with the surgeons during prolonged bypass runs and high-risk cardiac repairs.

Images

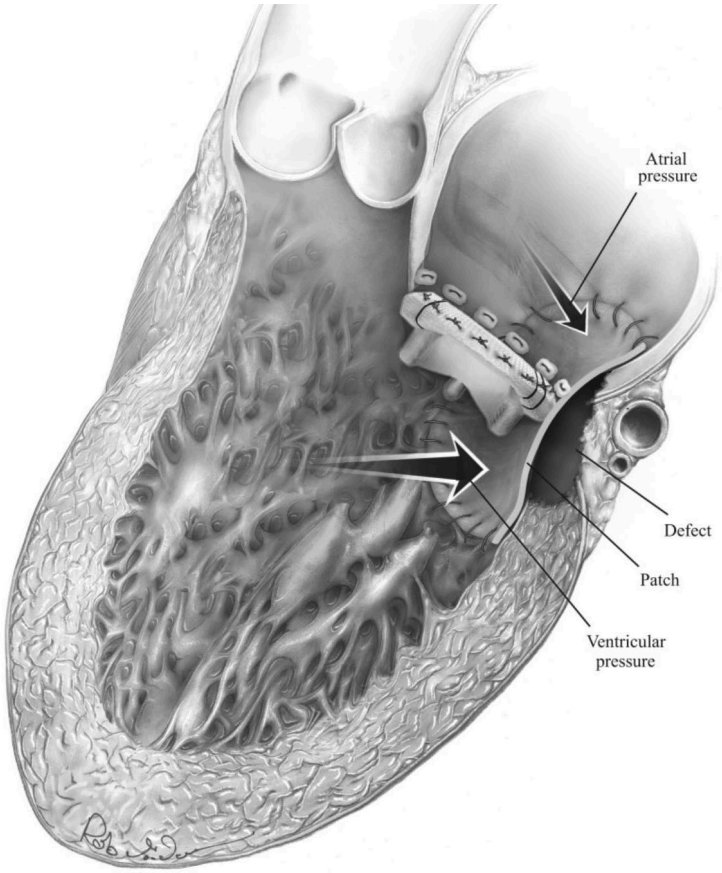








External repair of AVD with vein graft to left circumflex artery. Taken from https://doi.org/10.1016/j.athoracsur.2016.08.024



Internal AV D repair with patch over defect with required MVR. Taken from https://doi.org/10.1053/j.optechstcvs.2015.07.002

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#FR 3 – Kiosk 25 – Poster 4

Pump Problems: A Case Report of the HeartMate II Short-to-Shield Complication

Presenting Author: Esther Yoo, Resident, Montefiore Medical Center

Introduction

While the HeartMate III has largely supplanted the HeartMate II LVAD based on its lower complication profile, the HM2 continues to support a substantial portion of end-stage heart disease patients. As such, recognition and management of device complications are a critical part of care for this population. The short-to-shield (STS) phenomenon is one such uncommon but life-threatening complication. Here, we present a case of HM2 failure requiring emergent LVAD exchange to discuss the mechanism and clinical implications of STS, and highlight anesthetic considerations in managing this unusual complication.

Case Presentation

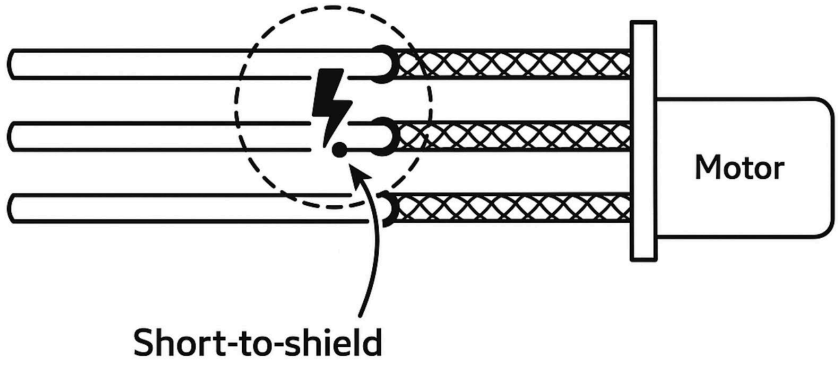
A 76-year-old woman with extensive cardiac history on long-standing HM2 support presented with severe driveline fraying and two days of LVAD alarms associated with presyncope. Upon admission, she experienced ten overnight pump stoppages, prompting emergent intervention via HM2-to-HM3 exchange. A pre-induction arterial line was placed; anesthesia was induced with etomidate, fentanyl, and rocuronium and maintained with isoflurane and fentanyl. TEE was utilized for real-time monitoring. Cardiopulmonary bypass was initiated without difficulty; a brief episode of ventricular tachycardia on bypass resolved with defibrillation. The HM2 inflow was removed from an intact apical cuff, the driveline transected at the abdominal wall, and the HM3 implanted using the prior cuff and outflow graft pathway. During CPB weaning, inhaled nitric oxide with dobutamine and epinephrine supported RV function, and HM3 speed was gradually titrated.

Discussion

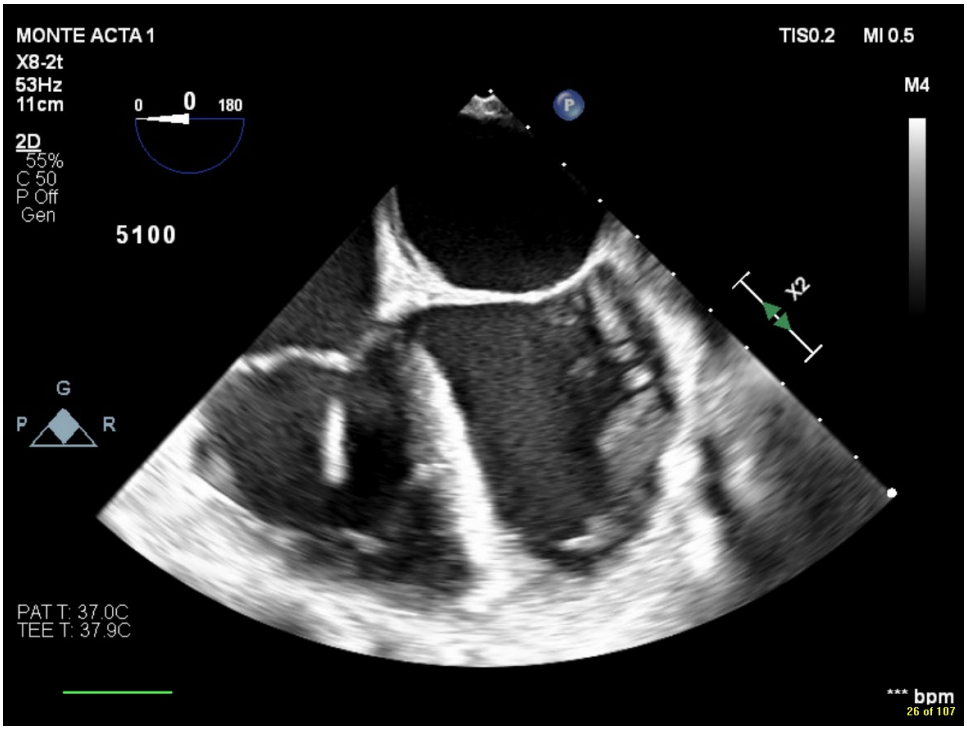
This case highlights an uncommon HM2 complication and its treatment, including key perioperative considerations during LVAD exchange. The HM2's axial-flow design and driveline architecture are associated with a more pronounced long-term complication profile, including pump thrombosis, hemolysis, and electrical defects such as the short-to-shield phenomenon. Its driveline contains multiple redundant conductors within a metallic shield, separated by a thin layer of insulation; over time, mechanical stress can expose a conductor to the grounded shield, diverting current from the motor and causing abrupt pump stoppage. In patients with minimal native cardiac output, even transient loss of device function can precipitate hemodynamic collapse. By contrast, the HM3's centrifugal pump and revised driveline design markedly reduce pump thrombosis and eliminate short-to-shield failure, making exchange particularly advantageous in patients with HM2 degradation. Intraoperatively, transitioning to HM3 support required optimization of RV performance to ensure adequate preload delivery. Inhaled nitric oxide reduced pulmonary vascular resistance, while dobutamine and epinephrine augmented RV contractility and perfusion during the transition to nonpulsatile flow. Stepwise titration of HM3 speed prevented excessive septal shift or suction events, with TEE guiding septal position, cannula orientation, and hemodynamic

response. As the HM2 cohort continues to age, anesthesiologists may increasingly encounter complications related to device deterioration requiring urgent HM3 replacement. Understanding the differences between these two devices is central to safely managing these high-risk patients and exchanges.

Images



Short-to-shield phenomenon is a result of the driveline internal wiring insulation breaking, which leads to a wire to touch the grounded metal shield, resulting in a short circuit that can stop the pump



Exchanged HM3 LVAD

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#FR 3 – Kiosk 25 – Poster 5

A Life-Threatening Complication After Watchman Device Implantation

Presenting Author: Rachna Dhanjal, MD, Lahey Hospital & Medical Center

Introduction

The Watchman left atrial occlusion device has shown efficacy and safety in preventing embolic complications of atrial fibrillation since its approval by the FDA in 2015. (1) Early and late migration of these devices has been described, (2) including immediate complications that may ultimately lead to cardiovascular collapse in the first few hours after implantation. (3) We report the post-procedural dislodgement of a Watchman device with migration into the left ventricle, causing cardiogenic shock and necessitating emergency surgical extraction with cardiopulmonary bypass.

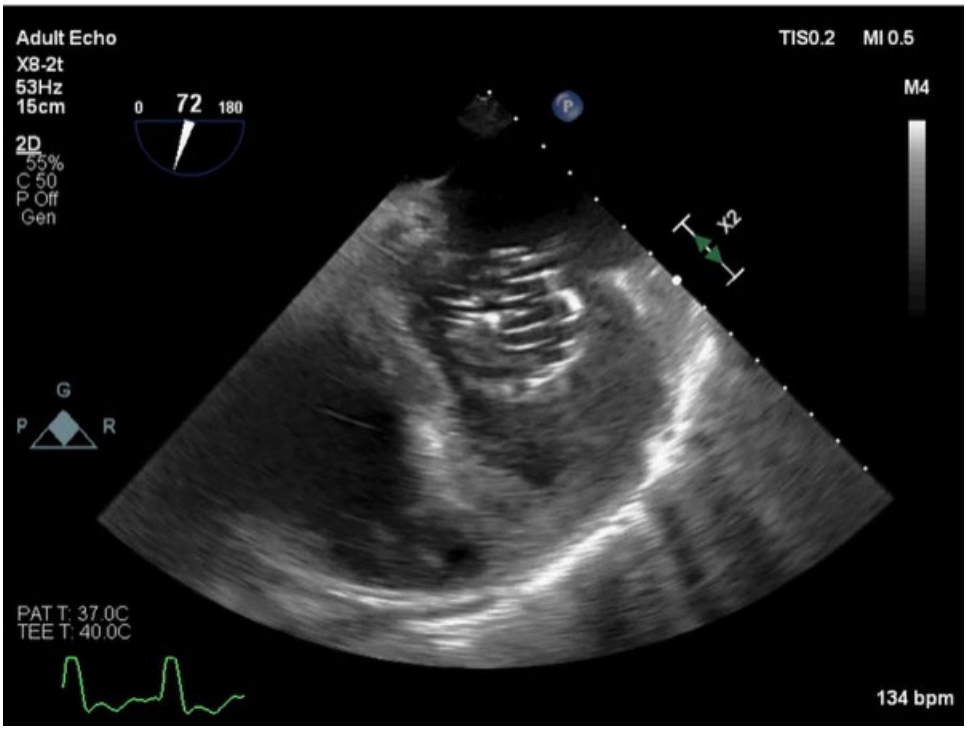
Case Presentation

A 79-year-old male with a medical history notable for essential hypertension, non-rheumatic aortic and mitral valve insufficiency, and permanent atrial fibrillation presented for Watchman implant. The procedure was performed under general anesthesia with transesophageal echo (TEE) and fluoroscopic guidance. While eating saltines in the recovery area, the patient complained of chest pain and was noted to be diaphoretic and hypotensive. Initial bedside transthoracic echocardiography (TTE) showed no evidence of pericardial effusion but revealed migration of the Watchman device to the cavity of the left ventricle, where it was entrapped in the subvalvular apparatus of the posterior leaflet of the mitral valve. The foreign body was creating moderate to severe mitral stenosis and a critical decrease in cardiac output. The patient was started on a norepinephrine infusion and brought expeditiously to the operating room for surgical extraction of the device. In the setting of worsening cardiogenic shock and progressive right ventricular failure, the patient required intermittent boluses of intravenous epinephrine throughout the induction of general anesthesia and the emergent initiation of cardiopulmonary bypass (CPB). Bicaval cannulation was used to facilitate atrial septostomy and open removal of the Watchman device. Post-bypass intraoperative TEE revealed new, severe mitral regurgitation (MR) resulting from a flail portion of the P3 segment of the posterior leaflet. The surgeon elected to defer definitive mitral valve repair or replacement to another setting. Separation from CPB was uneventful, and the patient was transported to the ICU and was extubated approximately 12 hours later. At the time of writing, he is well, with his MR responding to medical management.

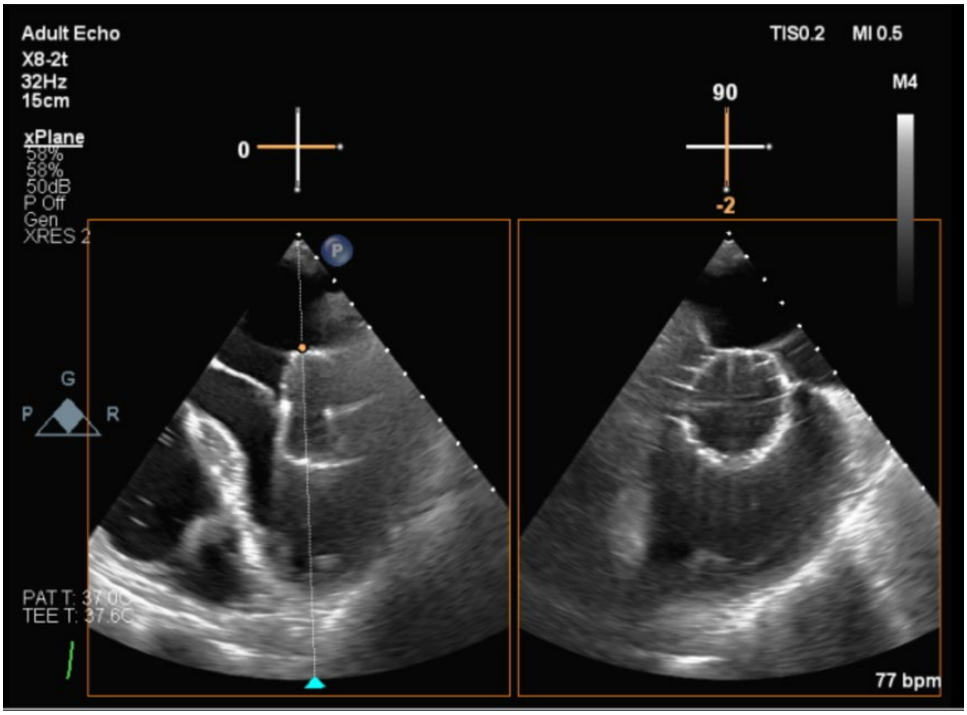
Discussion

While Watchman devices have been proven to be relatively safe, this case highlights a critical complication that may arise in the immediate post-implantation period. The early use of bedside TTE to rapidly diagnose possible underlying etiologies of shock and acute heart failure is of the utmost importance. The case also illustrates the critical role of intraoperative TEE in diagnosing new or residual cardiac pathology that may preclude successful weaning from CPB or that may require further surgical intervention.

Images



TTE picture



TEE picture

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#FR 3 – Kiosk 25 – Poster 6

Complex Arterial Vascular Disease Causing Cannulation Challenges, Malperfusion, and LIMA–LAD Coronary Steal During Emergent CABG and ECMO

Presenting Author: Fares Alghanem, MD, MBA, Resident Physician, Henry Ford Hospital

Introduction

Patients with arterial vascular disease present multiple hazards during cardiac surgery, including difficulty with central cannulation, misleading arterial-line hemodynamic monitoring, and unexpected coronary physiology. We describe a case in which critical arterial stenoses produced profound perfusion abnormalities during cardiopulmonary bypass (CPB) and VA-ECMO. These vascular lesions resulted in both regional hyperperfusion of the right arm due to brachiocephalic artery stenosis and coronary steal through a newly constructed LIMA graft due to proximal left subclavian artery occlusion.

Case Presentation

A 76-year-old woman with COPD, prior breast radiation, extensive atherosclerosis, and severe arterial vascular disease, including aorto-bifemoral bypass, presented with a left-main STEMI. Emergent CABG was undertaken with a right radial arterial line for hemodynamic monitoring. Intraoperatively, the ascending aorta was circumferentially calcified, precluding cross-clamping and central cannulation; right axillary cannulation was used for CPB. Failure to wean from CPB necessitated initiation of VA-ECMO (left femoral vein to right axillary artery). Striking discrepancies between arterial line pressure transducers were observed: right radial MAP 150 mmHg, left femoral 70 mmHg, and left axillary 15 mmHg, with the right upper extremity appearing hyperperfused. Angiography demonstrated severe right brachiocephalic artery stenosis that diverted both CPB and ECMO flows preferentially into the right arm, producing falsely reassuring right radial pressures while systemic perfusion remained critically low. Balloon angioplasty restored a more uniform distribution. Concurrently, angiography revealed complete proximal occlusion of the left subclavian artery. The new LIMA-to-LAD graft was functioning as a conduit for retrograde flow from the LAD into the underperfused left arm, creating a coronary steal phenomenon. Because the left subclavian artery could not be recanalized, PCI to the left main and LAD was performed, followed by coil embolization of the LIMA graft to eliminate the steal. Although coronary perfusion improved and pulsatility returned, the patient remained ECMO-dependent with multiorgan dysfunction.

Discussion

This case illustrates the extreme physiologic consequences of combined radiation-associated porcelain aorta and advanced arterial vascular disease. The inability to use central cannulation forced reliance on axillary inflow, which, in the setting of severe brachiocephalic artery stenosis, delivered support predominantly to the right arm and created dangerously misleading arterial-line pressure values. Multi-site monitoring ultimately revealed the extent of regional malperfusion. The proximal left subclavian occlusion produced an unusual postoperative LIMA–LAD steal, necessitating coil embolization of a newly created graft. Awareness of asymmetric perfusion patterns, early suspicion of arterial disease, and use of multiple

arterial pressure sites are essential to avoid catastrophic underestimation of systemic hypoperfusion in similar high-risk patients.

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#FR 3 – Kiosk 25 – Poster 7

Airway and Ventilatory Management of Large Tracheal Tear

Presenting Author: Tyler Brookshire, MD, Anesthesiology Resident Physician, UNC Health Department of Anesthesiology

Introduction

Tracheobronchial injuries are potential life-threatening emergencies with significant morbidity and mortality. Common etiologies include blunt or penetrating trauma and iatrogenic causes. Early recognition is important for timely intervention. We highlight a clinical scenario in which a large tracheal injury presented unique airway and ventilatory management considerations.

Case Presentation

Background

43 y.o. male with PMHx of Hodgkins lymphoma and prior traumatic brain injury who presented to an outside hospital with acute altered mental status, fever, and seizures. Intubated for airway protection upon admission.

Clinical Course

Day 3, Global hypokinesis LVEF~30% otherwise normal. Day 4, suspected cuff leak. Uncomplicated ETT exchange without improvement. Day 7, CTA chest concerning for tracheal tear. Transferred to tertiary center. Day 8, bronchoscopy revealed a posterior tracheal tear extending superiorly from the carina. CT Chest notable for pneumomediastinum with 5cm by 1.7cm defect along the posterior trachea. Progression to acute spontaneous loss of tidal volumes (>95%). External sternal pressure applied with temporary improvement of tidal volumes. ETT tube replaced with left sided DLT under fiberoptic visualization in ICU. The tracheal cuff was inflated with dual lung ventilation at adequate tidal volumes on pressure control ventilation with 8 cmH_2O PEEP.

Intraoperative Management

Day 9, scheduled exploratory thoracotomy. Anesthetic maintenance with propofol and isoflurane. Hemodynamically stable throughout on norepinephrine infusion. Single left lung ventilation with adequate tidal volumes on pressure control ventilation. 8cm tracheal tear identified. DLT exchanged for 8.0 SLT with exchange catheter and video laryngoscopy to facilitate proximal tear closure during surgery. Inability to seat right bronchial blocker necessitated intermittent apnea for proximal tear closure.

Discussion

This case features unique challenges and management of a tracheal injury. Suspected etiology of this tracheal injury was iatrogenic trauma from intubation given insidious onset of smaller tidal volumes. Incidence of tracheal injuries from intubation is 0.005%.[1] Positive pressure ventilation and PEEP can effectively worsen tracheal injuries.[2] Spontaneous ventilation is preferred, but due to the patient's

encephalitis, controlled ventilation was required and likely contributed to tracheal injury progression. External sternal pressure was emergently applied to improve tidal volumes by compressing the membranous portion of the tracheal defect. This approach has not previously been documented. A left sided double lumen ETT tube was urgently placed to facilitate ventilation. We suspect the larger ballon cuff extended beyond the tracheal defect providing partial occlusion. DLT permitted one lung ventilation as an emergent ventilatory strategy. Intraoperative ETT exchange in the left lateral position was required to facilitate surgical repair. Sequential steps, assigned roles, and back-up ventilatory plans were reviewed prior to the exchange. Transparent and close communication between all care teams involved was vital to ensure timely management of a dynamic clinical course.

Images

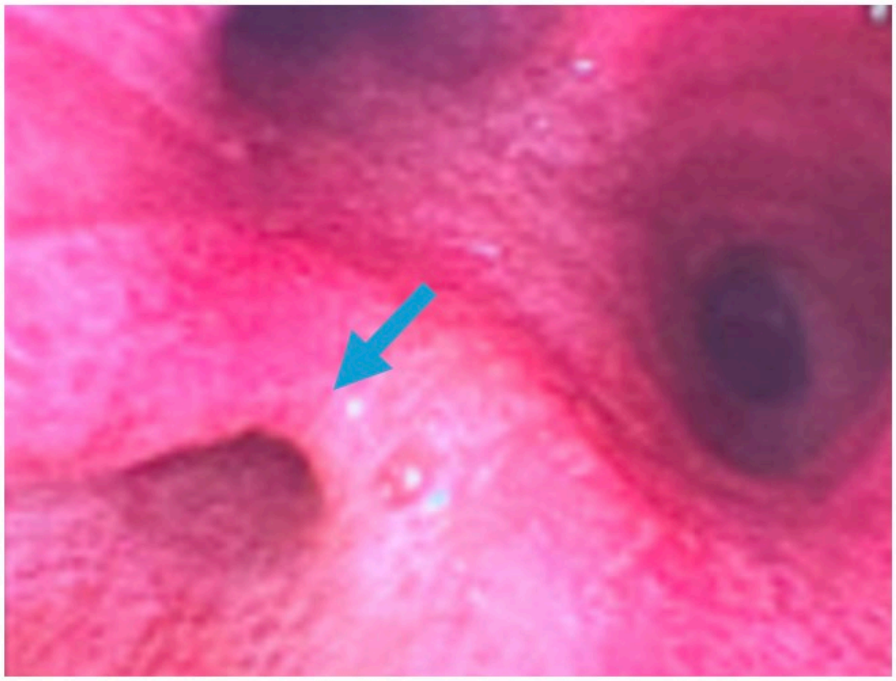


Figure 1: Pre-operative bronchoscopy with posterior tracheal defect (blue arrow)

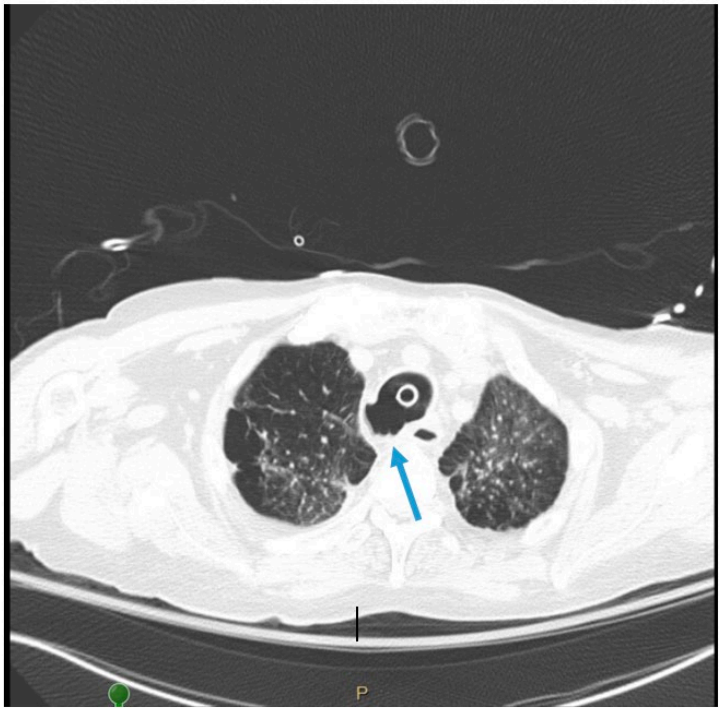


Figure 2: Pre-operative CT chest with tracheal defect (blue arrow)



Figure 3: Intraoperative tracheal defect (blue arrow)

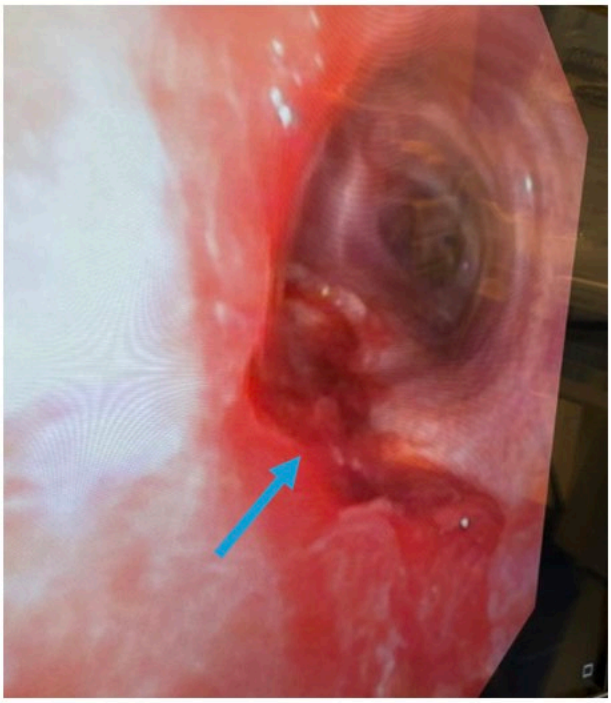


Figure 4: Post-operative bronchoscopy with intact suture line (blue arrow)

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#FR 3 – Kiosk 26 – Poster 1

Transfusion Management for Heart-Lung Transplantation in IgA Deficiency

Presenting Author: Steven Szymanski, MD, Resident, University of Chicago Department of Anesthesia/Critical Care

Introduction

Selective IgA deficiency is the most common primary immunodeficiency, with a prevalence ranging from 1 in 150 to 1 in 1000[2]. Exposure to IgA-containing blood products can cause severe anaphylaxis in patients who develop anti-IgA antibodies, which presents a major challenge during cardiothoracic procedures with potential for extensive blood transfusion[1]. This case highlights the detailed preparation and coordination required to safely perform combined heart–lung transplantation (HLT) in a patient with IgA deficiency.

Case Presentation

A 60-year-old female with pulmonary sarcoidosis complicated by pulmonary hypertension and right ventricular failure was listed for combined HLT. Due to known IgA deficiency (serum IgA < 7 mg/dL), the transfusion medicine team was consulted to coordinate a multidisciplinary transfusion plan. When suitable donor organs became available, the anesthesia and surgical teams coordinated preparation of 4 units of washed packed red blood cells (pRBCs) with the blood bank, with an additional 4 units of pRBCs becoming available from a nearby region within 8 hours. Previously, the patient made an autologous donation of plasma that would be available for use. The patient was brought to the operating room for en bloc HLT. General anesthesia was induced and the patient was intubated with a 37F L sided double lumen endotracheal tube. Monitors included an arterial line, transesophageal echo, and central venous access with a pulmonary artery catheter. After clamshell thoracotomy was performed, the patient was heparinized and cardiopulmonary bypass (CPB) was instituted. The patient's heart and lungs were excised and donor organs were placed en bloc. Intraoperatively she received 2 units of washed pRBCs, without evidence of transfusion reaction or hemodynamic compromise. She was weaned from CPB uneventfully and transported to the ICU where she was extubated on post operative day 4 and weaned off inotropic support by day 5.

Discussion

Proper transfusion management in patients with IgA deficiency requires multidisciplinary coordination. In this case, the patient received a preoperative transfusion medicine consultation outlining the various options for minimizing IgA exposure and mitigating the risk of anaphylaxis through various contingency pathways. The preferred method is to obtain blood products from IgA deficient donors, which can be difficult in patients with rare blood types such as AB[1]. Corticosteroids, antihistamines, and epinephrine should be readily available, and premedication should be used if giving non-IgA deficient blood products emergently. The process of washing red blood cells and platelets is performed to reduce the levels of IgA, though it can be timely and post washed platelets have a short shelf life[3]. Plasma must be collected from IgA deficient donors or collected autologously, which our patient was able to complete in advance. Recombinant factor VII can be used if IgA deficient donors cannot be located in the setting of hemorrhage. In the setting of organ

transplantation, an important consideration is the possibility of ex-vivo perfusion used on donor organs. Should this occur, this must be done with IgA deficient blood.

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#FR 3 – Kiosk 26 – Poster 2

Intraoperative Aorto-Atrial Fistula Following Aortic Valve Replacement: Critical Recognition by TEE and Emergent Bentall Reconstruction

Presenting Author: John C. Raynak, MD, Anesthesiology Resident (CA-2), Mayo Clinic- Arizona

Introduction

Aorto-atrial fistula is a rare complication after aortic valve replacement (AVR). Prompt intraoperative detection via transesophageal echocardiography (TEE) is essential to guide emergent surgical and anesthetic interventions. This case highlights TEE-guided recognition and perioperative anesthetic management.

Case Presentation

A 52-year-old male with bicuspid aortic valve, moderately enlarged ascending aorta (48–50 mm), moderate tricuspid valve regurgitation, and proximal LAD stenosis (60%) presented for surgical intervention. The patient was asymptomatic and his preoperative TTE showed preserved LV systolic function (EF 55%), mild mitral regurgitation, moderate tricuspid regurgitation, and borderline enlarged sinus of Valsalva. The patient was induced under general anesthesia and the case proceeded without issue. Initially, the surgeon placed a 25 mm mechanical Aortic valve and 32 mm aortic graft. After the first attempt to wean from cardiopulmonary bypass, TEE revealed continuous color doppler flow from the aortic root to the left atrium, consistent with an aorto-atrial fistula. Hemodynamics remained stable. The patient returned to bypass; the TEE images that showed the aorto-atrial fistula helped direct the surgeon on location of the fistula. Inspection revealed a suture tear above the noncoronary cusp communicating with the left atrium. The surgeon tried to repair it with sutures, but the fistula remained when attempting to wean off bypass. The decision was made to remove the mechanical valve and replace it with a Bentall aortic root reconstruction with bovine pericardial patch and 23 mm mechanical conduit. The patient was able to be weaned off cardiopulmonary bypass successfully with temporary epicardial pacing, minimal inotropes, and vasopressors. Total cross-clamp and bypass times were 397 and 530 minutes, respectively. Postoperatively, a dual-chamber pacemaker was placed on POD 6 for complete heart block. By POD 9, he was discharged home on supplemental oxygen, anticoagulation management, and outpatient cardiac rehabilitation.

Discussion

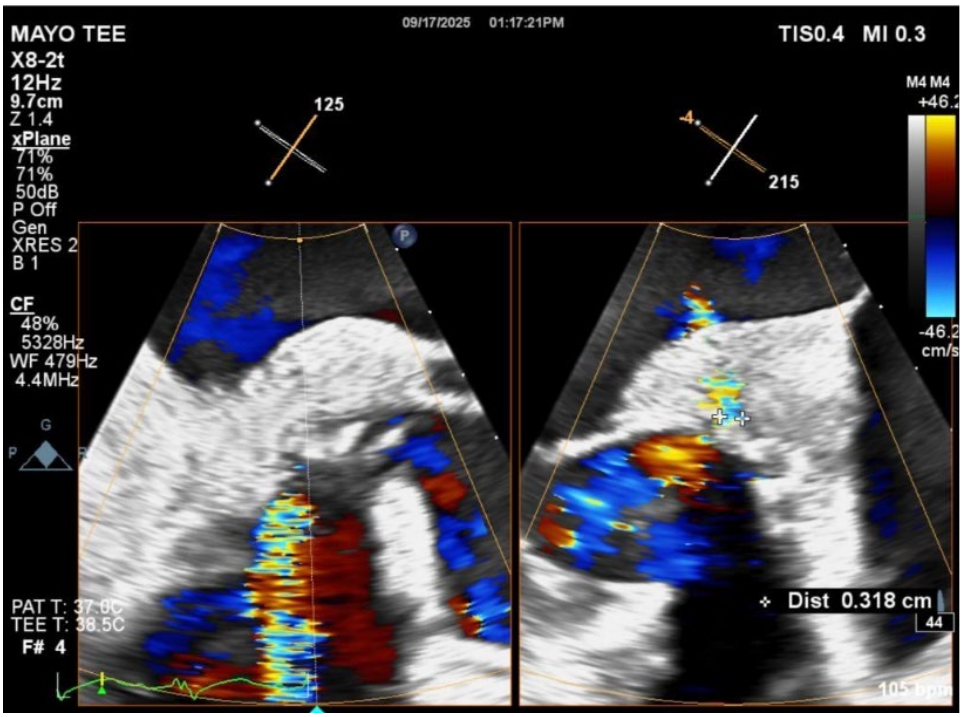
Aorto-atrial fistula can result from annular debridement, fragile aortic tissue, or technical injury, particularly in bicuspid valves with aneurysmal aorta. Key teaching points include:

- TEE Surveillance: Early detection of continuous Doppler flow from the aortic root to the left atrium enabled rapid return to bypass and emergent repair.
- Anesthetic Management: Maintenance of hemodynamic stability, coordination for transfusion, and careful anticoagulation monitoring were essential.
- Surgical Coordination: Multi-stage repair, including Bentall reconstruction, required seamless collaboration between anesthesia and surgical teams.

- Postoperative Considerations: Conduction disturbances, pacemaker dependency, and pulmonary optimization highlight ongoing anesthetic and perioperative vigilance.

This case underscores the importance of preoperative imaging, intraoperative TEE surveillance, and coordinated anesthetic-surgical decision-making in managing catastrophic aortic complications.

Images



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#FR 3 – Kiosk 26 – Poster 3

When Both Fail Early: Accelerated CAD and Valve Degeneration After TAVR

Presenting Author: Bushra Shehzad, M.D., Resident Physician, Wayne State University School of Medicine

Introduction

Ten-year data demonstrate that transcatheter aortic valve replacement (TAVR) prostheses are generally durable, with dysfunction most often attributable to leaflet degeneration, fibrotic or calcific stiffening, thrombus formation, endocarditis, or progressive patient–prosthesis mismatch rather than intrinsic structural failure (1). Approximately half of TAVR candidates also have concomitant coronary artery disease (CAD), and although a causal link between valve pathology and CAD progression has not been definitively established, a subset of patients can exhibit unexpectedly rapid acceleration of coronary disease (2). We present a case of valvular stenosis and rapidly progressive coronary artery disease necessitating surgical valve explantation and CABG three years after an initially successful TAVR.

Case Presentation

A 63-year-old female with HTN, CAD, and COPD underwent TAVR with a 29 mm Evolut Pro+ for severe AS. Pre-procedural angiography demonstrated mild CAD not requiring intervention. Her immediate post-TAVR course was excellent with rapid symptom resolution, stable pressure gradients, and normal leaflet motion confirmed by echocardiography. By the second year, echocardiography showed a mean gradient rise from 10 to 23 mm Hg with a stable EOA of 2 cm2. In the third year, the patient presented with dyspnea and angina and was found to have severe TAVR valve stenosis with a mean gradient of 72 mm Hg and an EOA of 0.5 cm2 in addition to critical left main coronary artery stenosis. The patient underwent TAVR explant with SAVR, aortic root enlargement, and CABG. Intraoperative TEE demonstrated thickened and restricted leaflets and the surgeon reported an “aortically positioned” valve and a porcelain aortic root. Her postoperative course was complicated by coagulopathy, renal failure, and a debilitating CVA, and she ultimately did not survive.

Discussion

TAVR valve degeneration and CAD progression are generally slow processes, making the rapid onset of severe prosthetic stenosis and advanced coronary disease in this patient atypical. Accelerated CAD can arise from a combination of patient-specific factors including metabolic and inflammatory drivers, elevated lipoprotein(a), and biologically active non-obstructive plaque that is prone to rapid expansion (2). In parallel, several device and valve-related factors may hasten bioprosthetic dysfunction, such as incomplete valve expansion, patient–prosthesis mismatch, or subclinical leaflet thrombosis (3). Our case illustrates how rapid coronary progression and early prosthetic dysfunction can arise despite initially mild angiographic findings. The mechanism was likely multifactorial, and any or all of the following may have contributed: inflammatory pathways, altered aortic root flow from the “aortically positioned” frame, and neoleaflet interactions that shifted shear forces near the coronary ostia. Additionally, chronic coronary flow impairment from frame overlap, leaflet position, or commissural misalignment may have further accelerated left main disease. This case underscores the importance of maintaining vigilance for interval changes in

symptoms, gradients, and coronary flow, including in patients whose TAVR course appeared uncomplicated and whose coronary disease was initially mild.

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#FR 3 – Kiosk 26 – Poster 4

Acute Mechanical Mitral Valve Thrombosis Resulting in Obstructive Shock and Emergent Replacement

Presenting Author: Katherine Lukshin, MD, Resident, Virginia Mason Medical Center

Introduction

Thrombosis is a well-known complication of prosthetic heart valves. Mechanical mitral valve prostheses are particularly susceptible to this phenomenon; the incidence of thrombosis in bileaflet mechanical mitral valves is estimated to be 0.2-0.5% per patient-year.1,2 Patients with obstructive mitral valve thromboses present at significantly elevated surgical risk, with perioperative mortality rates reported to be as high as 14.9-27.7%.3 Herein describes the successful management of a patient with sudden, severe mechanical mitral valve stenosis from an acute valve thrombosis causing obstructive shock requiring mechanical circulatory support and emergent surgical mitral valve replacement.

Case Presentation

A 45-year-old woman with a history of congenital mitral valve stenosis status post surgical replacement with a 27mm St. Jude mechanical valve on warfarin, paroxysmal atrial fibrillation, and alcohol use disorder presented with a two day history of nausea, vomiting, and sudden onset, severe dyspnea at rest. Initial work-up was notable for an acute kidney injury, transaminitis, elevated brain natriuretic peptide and troponin, and subtherapeutic INR of 1.7. TEE revealed severe mitral stenosis with a transmitral gradient of 17mmHg and a 3.2 cm x 2.3 cm echodensity consistent with thrombus. Heart catheterization revealed pulmonary artery pressure of 78/45 mmHg with mixed venous oxygen saturation of 29% and cardiac index of 0.9 L/min/m2. An intra-aortic balloon pump and Swan-Ganz catheter were placed and the patient was emergently transported to the OR. The patient was noted to be increasingly cyanotic and tachypneic during transport, and on arrival to the OR was found to have suprasystemic pulmonary artery pressures. Peripheral Veno-arterial (VA) ECMO was initiated simultaneously with induction of general anesthesia to bridge to cardiopulmonary bypass. The thrombus and subsequently mitral valve were removed without complication and replaced with a 25 mm ON-X mechanical mitral valve. The patient was successfully weaned off cardiopulmonary bypass and postoperative TEE revealed improved bi-venticular function but still required VA ECMO. The patient was weaned off VA ECMO two days after the surgery. She was eventually extubated, weaned off vasopressors, and discharged home 17 days postoperatively.

Discussion

This case serves as a framework to discuss the perioperative considerations and intraoperative challenges of managing a patient on the brink of cardiopulmonary collapse from obstructive physiology. The potential for rapid deterioration in this population highlights the significant morbidity and mortality of patients presenting with mechanical valve thromboses. Furthermore, this case underscores the importance of close coordination with the surgical team to successfully navigate emergent situations.

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#FR 3 – Kiosk 26 – Poster 5

A near miss: Ascending Aortitis Mistaken for Aortic Intramural Hematoma

Presenting Author: Marissa Jones, DO, Anesthesiology Resident, Virginia Mason Franciscan Health

Introduction

Aortic intramural hematomas (IMHs) have a lower incidence than aortic dissections, but pose similar mortality rates. Current evidence supports early surgical repair for large IMHs involving the ascending aorta, and conservative medical management for lesions involving the descending thoracic and abdominal aorta.1 Aortitis has similar radiographic features to IMHs and most commonly involves the abdominal aorta. Ascending aortitis is rare, but has been associated with IgG4-related disease (IgG4-RD) in multiple case reports.2,3 Typically, the diagnosis is made after postoperative histiologic analysis, thus exposing patients to unnecessary, high-risk surgical interventions. Herein, we describe a case where a patient presented for open surgical repair of a presumed IMH involving the ascending aorta but ultimately avoided intervention after uncovering aortitis.

Case Presentation

A 77-year-old woman with a history of obesity, diabetes, hypertension, and recurrent pleural effusions presented for right-sided chest pain and dyspnea in the setting of several months of worsening fatigue and general malaise. Initial imaging revealed a complex right pleural effusion and a new 13 mm crescentic hyperdensity along the lateral wall of the ascending aorta and aortic arch, consistent with IMH. She was started on antibiotics for possible parapneumonic effusion, and was also noted to have an acute kidney injury on admission. Thus, the decision was made to postpone surgical repair until these conditions were optimized. She was admitted to the ICU for close hemodynamic monitoring and titration of anti-impulse therapy. Over the next week, her renal function continued to worsen. Renal ultrasound revealed bilateral hydronephrosis, with subsequent CT KUB demonstrating diffuse retroperitoneal fibrosis. Bilateral ureteral stents were placed without improvement, and she ultimately required hemodialysis. Her pleural effusion progressed to bilateral effusions despite broad-spectrum antibiotics and initial attempts at diuresis. Diagnostic thoracentesis showed an exudative process but was unrevealing of infection nor malignancy. She began to endorse visual disturbances, and a CT head revealed pachymeningitis of the right optic nerve. Further investigation into her atypical imaging findings revealed an association between retroperitoneal fibrosis and optic pachymeningitis in patients with IgG4-RD. A PET scan of her chest demonstrated multifocal FDG uptake throughout her aorta consistent with aortitis, and uptake in her pleural effusions consistent with pleuritis. Rheumatology was consulted and additional workup supported the diagnosis of IgG4-RD. She symptomatically improved and had renal recovery after starting steroids, and was discharged on hospital day 34 without surgical intervention. Outpatient imaging demonstrated radiographic improvement of her aortic lesion and complete resolution of her pleural effusions.

Discussion

This case constitutes a near-miss event and highlights the importance of keeping aortitis on the differential

in patients with IMH presenting for surgical repair given the radiographic similarities—especially in patients with unexplained abnormalities across multiple organ systems that might be indicative of an autoimmune process.

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#FR 3 – Kiosk 26 – Poster 6

Use of Left Atrial Venoarterial ECMO as Bridge to LVAD in a Patient with STEMI, Cardiogenic Shock, and Left Ventricular Thrombus

Presenting Author: Alan Boiangu, MD, Fellow, Cleveland Clinic

Introduction

In patients presenting with biventricular failure, the use of temporary mechanical circulatory support can provide a means to transition to a long term, durable device or transplant. Certain contraindications may limit the utility of these devices in some patients.[1] Left atrial veno-arterial (LAVA) ECMO provides an emerging alternative strategy for unloading the left ventricle.[2] We describe the successful use of LAVA-ECMO as a bridge to LVAD implantation in a patient presenting with cardiogenic shock complicated by LV thrombus.

Case Presentation

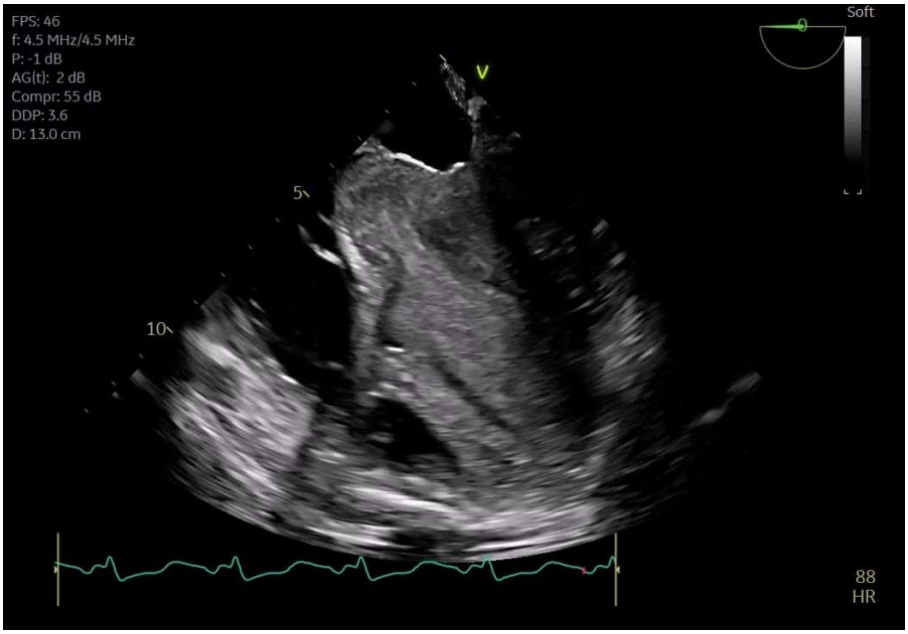
Our patient is a 31 year old male with past medical history of asthma, hypertension, and scoliosis who presented to our facility with 1 week of persistent dyspnea and generalized fatigue. Workup revealed a STEMI. PCI revealed 100% occlusion of his LAD with thrombus in his left circumflex system which was revascularized and an intra-aortic balloon pump was placed. He was transferred to the ICU, where he was initially asymptomatic however soon developed V-fib arrest; he was defibrillated once before achieving ROSC. Bedside surface echo revealed severe LV global hypokinesis, dysfunctional RV, and a large thrombus in the LV. He was intubated and a PA catheter was placed revealing reduced cardiac index and elevated PA pressures. The decision was made to place the patient on peripheral VA-ECMO. Given his large LV thrombus, he was not a candidate for traditional Impella; instead his circuit was reconfigured for trans-septal LV offloading (LAVA-ECMO). Given his persistent cardiogenic shock, he was evaluated for heart transplant. However, workup revealed an untreated HIV diagnosis and he was delisted. He was approved for durable LVAD as a potential bridge to future heart transplant. The patient underwent LVAD implantation at which time his iatrogenic ASD was closed, LV thrombus was removed, and RVAD was placed. He underwent RVAD removal approximately 5 days later. He was discharged to cardiac rehabilitation 2 months after his initial presentation.

Discussion

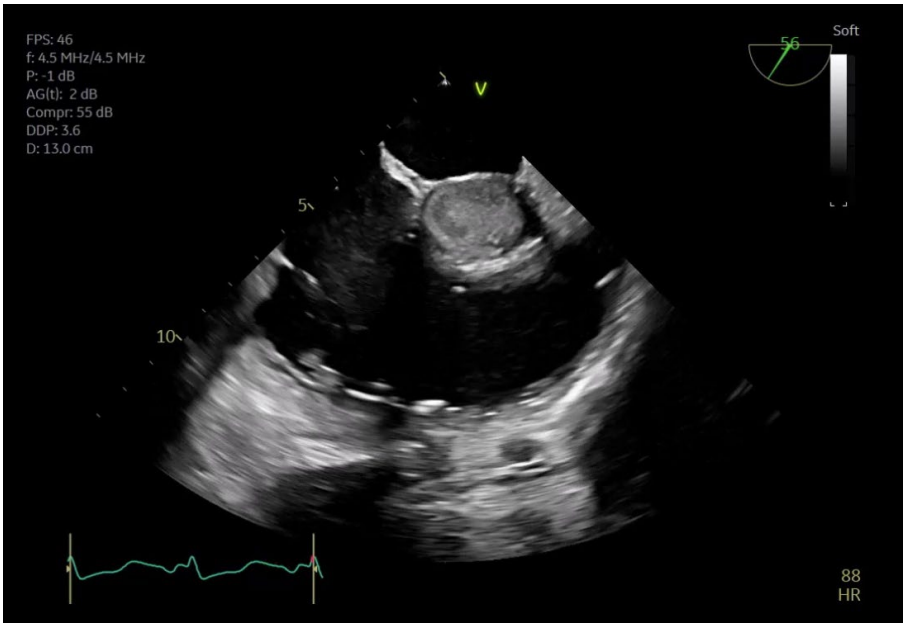
Our case highlights the utility of LAVA-ECMO in patients with cardiogenic shock. In traditional VA-ECMO, retrograde flow can worsen myocardial oxygen demand, wall stress, and LVEDP. The use of a traditional LV support device such as Impella was contraindicated in our patient due to the embolic risk. Therefore, an alternative such as LAVA-ECMO provided adequate support with minimized risk. Studies have shown that, independent of contraindications, the probability of survival is greater in patients being offloaded by LAVA-ECMO when compared to traditional Impella devices.[1] This case also highlights some of the considerations when evaluating patients for high risk LVAD implantation, such as threshold for implanting an RVAD in these patients.[2] Due to worsening right-sided pressures in our patient, adequate LV offloading was critical for

successful LVAD implantation.[3] The benefit of LAVA-ECMO in these patients has also been demonstrated with higher ECMO weaning rates, higher transplantation rates, and has been shown to be more cost-effective; avoiding the need for additional devices and further arterial access.[4] Further research into this emerging form of circulatory support and its anesthetic implications is needed.

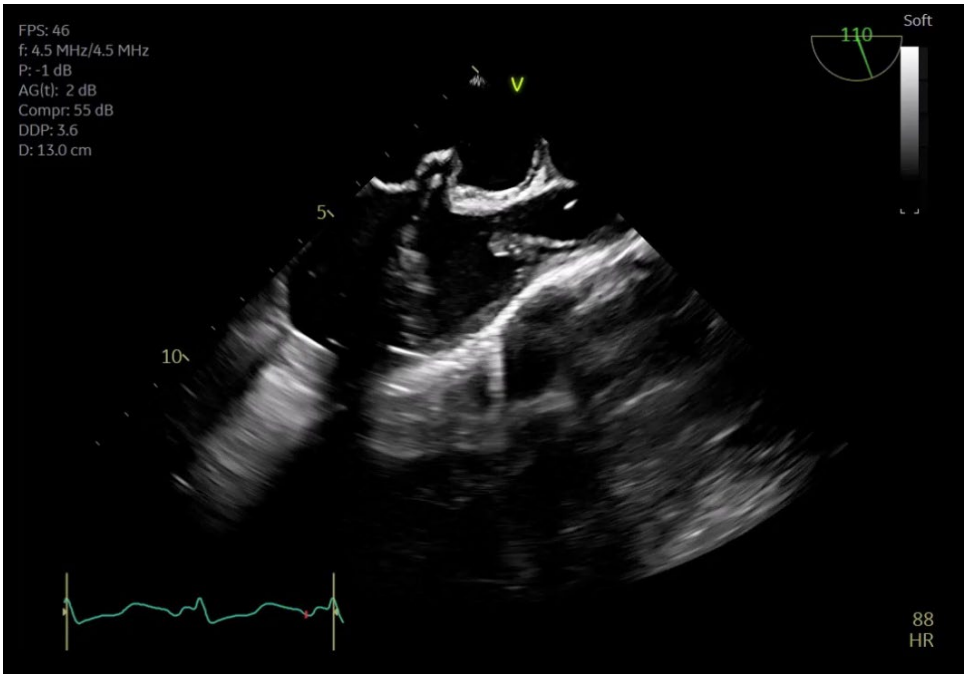
Images



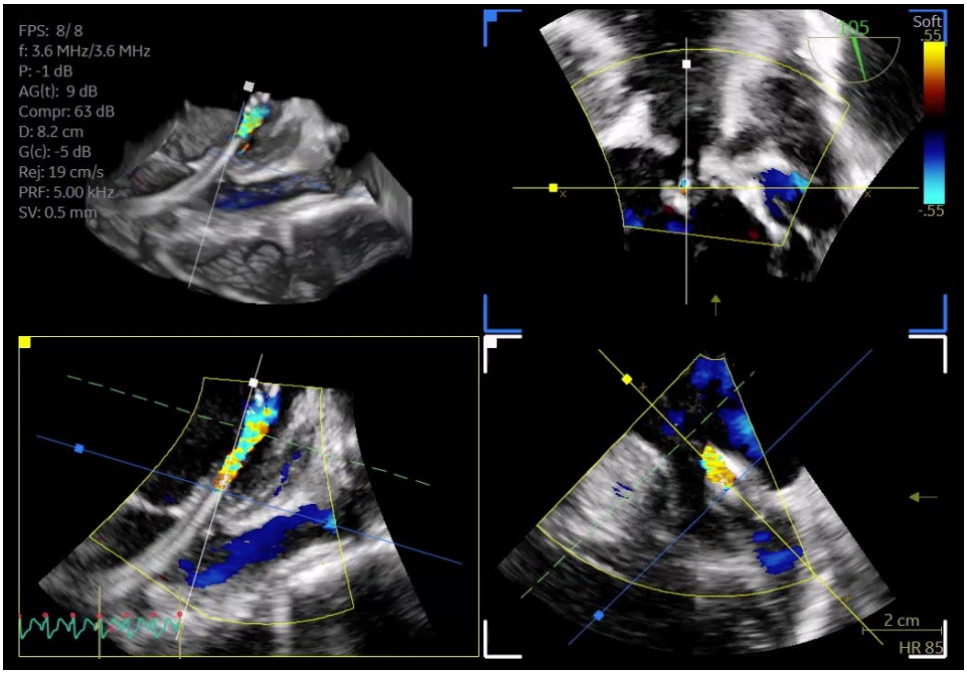
Mid esophageal TEE view of LV thrombus



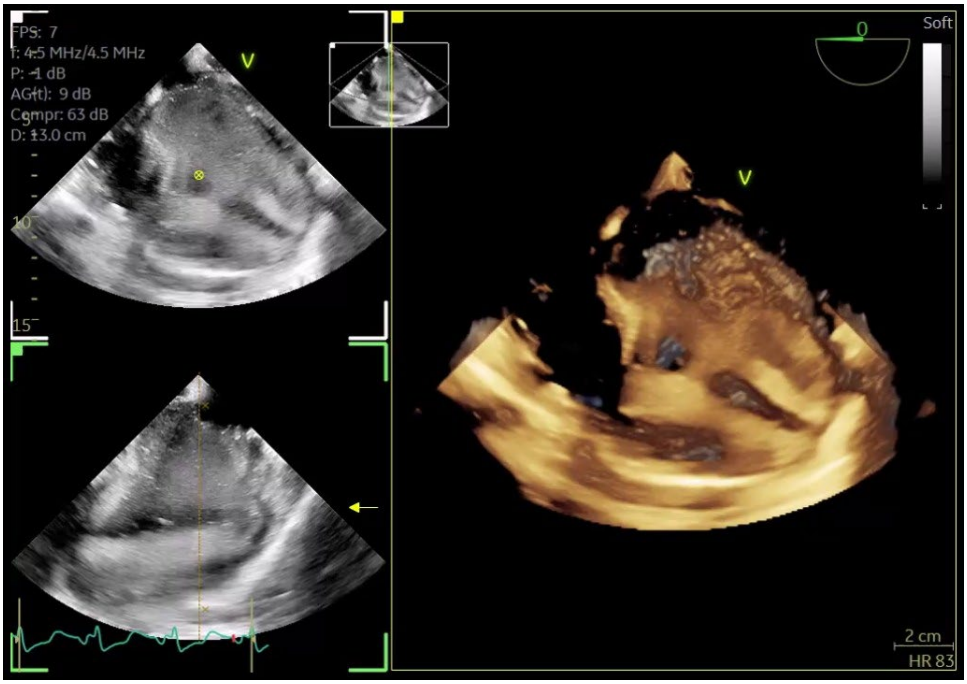
AV SAX view of Thrombus in LVOT



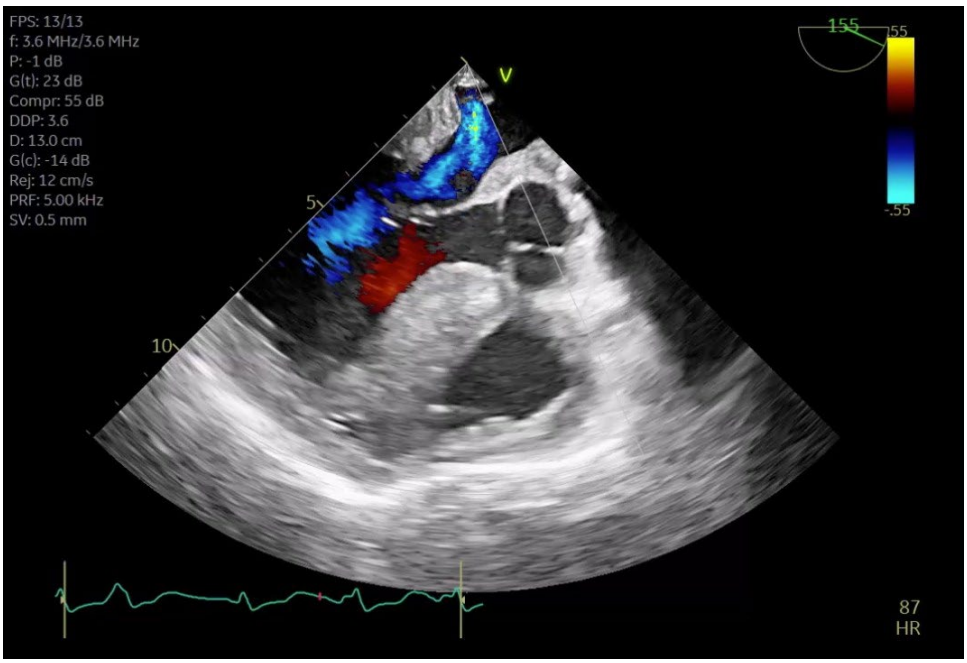
LA Cannula traversing the interatrial septum



Flow through interatrial cannula



3D image of LV thrombus



Offloading of LV using left atrial cannula

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#FR 3 – Kiosk 26 – Poster 7

Case Report: Takotsubo Cardiomyopathy Triggered Postoperatively in a Patient Undergoing Knee Arthroplasty

Presenting Author: Yang Yu, Identification of Physician's Qualification, Fuwai Hospital

Introduction

Takotsubo syndrome (TS), also known as stress-induced cardiomyopathy or "broken heart syndrome," is characterized by acute transient left ventricular systolic dysfunction, often mimicking acute myocardial infarction (AMI). It typically occurs following emotional or physical stressors, leading to left ventricular apical ballooning and acute heart failure. We present a rare case of TS in a 64-year-old female after bilateral knee arthroplasty.

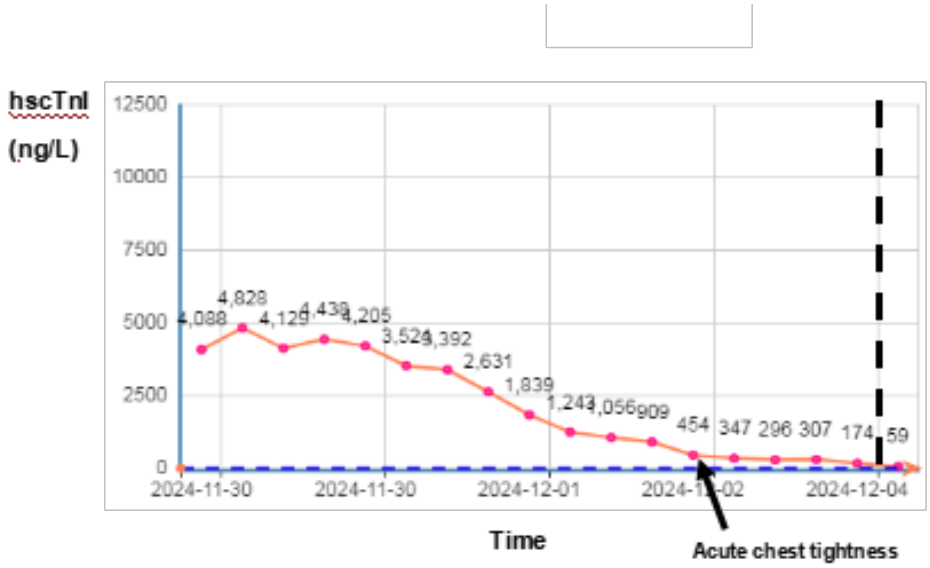
Case Presentation

A 64-year-old woman with no prior cardiovascular history developed TS postoperatively following robot-assisted knee arthroplasty. Intraoperatively, she experienced suspected coronary artery vasospasm under general anesthesia, followed by hemodynamic instability requiring ICU admission. Bedside echocardiography revealed apical hypokinesis with "takotsubo" morphology. Coronary angiography excluded obstructive coronary artery disease (LCX stenosis < 30%, TIMI 3 flow), confirming TS.

Discussion

The specific pathophysiological mechanism of Takotsubo syndrome (TS) remains unclear. Evidence suggests the involvement of the brain-heart axis, which regulates increased catecholamine levels at both systemic and presynaptic levels, thereby triggering microvascular constriction and direct myocardial injury. Findings from a murine study support that altered blood flow regulation in the heart can induce TS. TS occurs most frequently in postmenopausal women, where estrogen deficiency leads to endothelial dysfunction, resulting in sympathetic nervous system predominance and subsequent coronary microvascular spasm. Early studies indicated that TS is primarily triggered by emotional factors. Recent research, however, suggests that physical triggers may also contribute, and in some cases, no clear trigger can be identified. A prospective study in Japan involving 43 TS patients reported physical triggers in 52%, emotional triggers in 31%, and no identifiable triggers in 17% of cases. A retrospective study of 1,750 patients in Western countries found physical triggers in 36%, emotional triggers in 27.7%, a combination of both in 7.8%, and no identifiable trigger in 28.5% of patients. In the present case, follow-up inquiry by the attending physician revealed that the patient experienced specific stressors prior to surgery. The patient reported that two roommates significantly disrupted her sleep—one frequently moved around after bedtime, and the other had poor hygiene, requiring the patient to clean the bathroom before use, causing considerable distress. Additionally, the patient found the hospital meals unappealing and experienced poor dietary intake and sleep quality for three days following admission.

Images



Dynamic changes of hscTnI in the patient

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