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Thrombotic mitral valve obstruction during venoarterial extracorporeal membrane oxygenation with left ventricular venting following complex valve surgery

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Abstract

Background: Left ventricular (LV) unloading during veno-arterial extracorporeal membrane oxygenation (VA-ECMO) is essential for managing refractory low cardiac output after complex cardiac surgery. However, direct LV venting is associated with an increased risk of intracardiac thrombosis, particularly when anticoagulation is delayed.

Case presentation: A 69-year-old woman with hypertrophic cardiomyopathy and severe left ventricular outflow tract obstruction presented with exertional dyspnea. Echocardiography showed severe mitral regurgitation with marked annular calcification, moderate aortic regurgitation, and systolic anterior motion of the mitral valve. She underwent complex cardiac surgery including septal myectomy, aortic and mitral valve replacement with bioprostheses, and tricuspid annuloplasty. Intraoperative rupture at the anterior mitral commissure required re-clamping, patch repair, and reimplantation of the prostheses. Prolonged cardiopulmonary bypass led to postoperative circulatory failure, necessitating VA-ECMO via the right axillary artery and femoral vein, with left ventricular venting through the right superior pulmonary vein. On postoperative day 4, ECMO was successfully

weaned after recovery of contractility; however, the patient developed acute pulmonary edema and cardiogenic shock within hours, prompting urgent ECMO reinstitution. Transesophageal echocardiography on day 9 revealed thrombotic obstruction of the mitral bioprosthesis, confirmed intraoperatively. Both prosthetic valves and the left ventricle were covered with extensive white and red thrombi, which were completely removed. Cardiac function recovered after reoperation, and ECMO was successfully discontinued. Despite subsequent bilateral cerebral infarctions, the patient survived and was gradually weaned from mechanical ventilation at 1-month follow-up.

Conclusion: Thrombotic complications in patients supported with VA-ECMO after valve replacement result from multiple interacting factors, including surgical trauma, prosthetic material, altered flow dynamics, and delayed anticoagulation. This case highlights the diagnostic challenges of assessing prosthetic valve function during VA-ECMO and underscores the need for individualized decisions regarding LV unloading strategies and anticoagulation management. Careful ECMO weaning and close echocardiographic surveillance may help reduce the risk of catastrophic valve-related complications.

Keywords: ECMO; left ventricular unloading; prosthetic valve thrombosis

1. Background

Left ventricular (LV) unloading has become a pivotal adjunct strategy in managing patients with cardiogenic shock or low cardiac output syndrome (LCOS) following cardiac surgery who are supported with veno-arterial extracorporeal membrane oxygenation (VA-ECMO). Common unloading methods include intra-aortic balloon pump (IABP), percutaneous left ventricular assist devices (pLVADs), atrial septostomy, and surgical left ventricular venting. We report a rare case of thrombotic obstruction of a prosthetic mitral valve associated with left ventricular venting during VA-ECMO support following complex valve surgery.

2. Case presentation

A 69-year-old woman with a history of hypertension, well-controlled on bisoprolol, presented with exertional dyspnea. Transthoracic echocardiography revealed hypertrophic cardiomyopathy with severe left ventricular outflow tract (LVOT) obstruction. The interventricular septum measured 15 mm at the basal segment, 13 mm at the posterior wall, and 16 mm at the apex. Doppler imaging demonstrated a peak flow velocity of 4.4 m/s

across the LVOT with a pressure gradient of 76 mmHg. Marked mitral annular calcification accompanied by severe mitral regurgitation (regurgitant area 9.9 cm^2) and moderate aortic regurgitation (regurgitant area 1.5 cm^2) were present. Estimated pulmonary artery systolic pressure was 29 mmHg based on tricuspid regurgitant jet velocity. Systolic anterior motion (SAM) of the mitral valve anterior leaflet was noted. The ascending aorta was mildly dilated (39 mm). Left ventricular end-diastolic diameter was 46 mm, end-systolic diameter 26 mm, and left atrial diameter 49 mm. Coronary angiography showed a 30-50% localized stenosis in the mid-left anterior descending artery. The patient underwent complex cardiac surgery under general anesthesia with cardiopulmonary bypass (CPB), including surgical septal myectomy (25 mm * 25 mm area, 8 mm thick), aortic valve replacement (AVR) with a bioprosthesis, mitral valve replacement (MVR) with a bioprosthesis using the skirt technique, and tricuspid valve annuloplasty. Active pericardial bleeding was observed during the first attempt to wean from CPB, leading to re-initiation of cardiopulmonary bypass. Following re-clamping of the ascending aorta, antegrade Del-Nido cardioplegia was administered again to ensure myocardial protection. Surgical exploration revealed a ventricular rupture at the anterior commissure of the mitral valve.

The implanted aortic and mitral bioprostheses were removed. A bovine pericardial patch was used to repair the ventricular rupture: the patch was sutured along the residual mitral leaflet tissue, meticulously closing the annular and myocardial defects, and extended to reinforce surrounding normal myocardium and the fibrous trigone. The mitral bioprosthesis was reimplanted using the skirt technique, and a new aortic bioprosthesis was sewn in place. Total CPB time was 557 minutes, and aortic cross-clamp time was 372 minutes.

Weaning from CPB remained difficult, prompting initiation of veno-arterial extracorporeal membrane oxygenation (VA-ECMO) for circulatory support. Given the high risk of re-rupture due to the fragility of the repaired ventricular myocardium, a left ventricular (LV) venting cannula was placed via the right superior pulmonary vein. The chest was left open for delayed sternal closure. VA-ECMO was established via the right axillary artery and right femoral vein. On the first postoperative day, due to significant coagulopathy and bleeding, anticoagulation was withheld and blood products including coagulation factors and red blood cells were administered. Once bleeding was controlled and coagulation parameters normalized, intravenous heparin infusion was started

(5–10 U/kg/h), titrated according to activated clotting time (ACT) and activated partial thromboplastin time (APTT). On postoperative day 2, the LV venting cannula was removed and the chest closed. VA-ECMO support was continued. On day 4, with improved left ventricular contractility, VA-ECMO weaning was initiated per ELSO guidelines (1), with flow reduced by 500 mL every 30 minutes and maintained at 1 L/min while cardiac function was repeatedly evaluated via bedside echocardiography. Arterial blood gas analysis was performed hourly, showing normal lactate levels. With satisfactory hemodynamics and contractility, both the LV vent and VA-ECMO were successfully removed approximately 4 hours after initiating the weaning process. However, within 4 hours of ECMO removal, the patient developed progressive hypotension, tachycardia, and hypoxemia (SpO_2 85–92%). High-dose vasopressors were required, and copious frothy sputum was suctioned from the endotracheal tube. An intra-aortic balloon pump (IABP) was inserted for circulatory support, but severe hypoxemia persisted, with persistent pink frothy sputum from the airway. Arterial blood gas analysis showed: PaO_2 53 mmHg, $PaCO_2$ 54 mmHg, base excess +3.4 mmol/L, lactate 1.0 mmol/L. Ventilator settings included FiO_2 100% and PEEP 8 cmH_2O. Bedside chest X-ray revealed marked bilateral pulmonary infiltrates Despite IABP

and vasopressors, effective circulation could not be maintained, prompting emergent re-initiation of VA-ECMO. Following successful cannulation, SpO_2 rapidly increased to >95%, mean arterial pressure stabilized at 60–70 mmHg, and vasopressor requirements were significantly reduced.

After ECMO weaning, the patient experienced rapid hemodynamic deterioration and severe hypoxemia. Fluid overload was excluded as a potential cause. Pulmonary artery CTA ruled out pulmonary embolism but demonstrated bilateral pulmonary edema (**Figure 1(A&B)**), indicating acute left ventricular failure. On day 9, transesophageal echocardiography (TEE) was performed for further assessment and revealed prosthetic mitral valve thrombosis with obstruction and mild regurgitation (**Figure 1(C&D)**). The patient underwent urgent surgical exploration on day 10. Intraoperatively, thrombus was found extensively covering the mitral valve prosthesis and subvalvular apparatus. One leaflet of the prosthesis was immobile due to thrombus. White thrombi were seen on the atrial and ventricular surfaces. The aortic bioprosthesis was also covered in white thrombus, and red thrombi were identified in the LV outflow tract. All thrombi were meticulously removed. The patient returned to the surgical ICU under combined

VA-ECMO and IABP support. Two days postoperatively, with satisfactory cardiac function, VA-ECMO was weaned successfully, and IABP support was continued. 5 days later, echocardiography showed a recovered left ventricular ejection fraction (LVEF) of 50%, and chest radiography demonstrated significant resolution of pulmonary infiltrates, allowing IABP removal. However, the patient subsequently developed altered consciousness and somnolence. Brain CT confirmed bilateral cerebral infarctions. The patient was transferred to a neurological center under mechanical ventilation and underwent tracheostomy. At 1-month follow-up, the patient was alive and undergoing gradual weaning from ventilatory support. The timeline and relevant perioperative laboratory results are presented in **Figure 2**.

3. Discussion

This patient with hypertrophic obstructive cardiomyopathy and multivalvular disease underwent LVOT septal myectomy and bioprosthetic AVR/MVR. The intraoperative left ventricular rupture and subsequent repair resulted in severe ventricular dysfunction necessitating VA-ECMO with direct LV venting for unloading. Postoperative bleeding delayed initiation of systemic anticoagulation, while the presence of prosthetic valves combined

with intracavitary venting cannula created a highly thrombogenic milieu. Importantly, postcardiotomy patients supported with VA-ECMO are intrinsically predisposed to thrombus formation because of surgical trauma, blood–artificial surface interaction, and altered intracardiac flow patterns, irrespective of the unloading strategy employed (2). In this context, the thrombotic event observed in our patient should be interpreted as the result of multiple converging risk factors rather than a direct causal consequence of LV venting alone.

Under VA-ECMO support, altered flow dynamics may promote valve stasis through different mechanisms. Both low-flow states during ECMO weaning and high ECMO flow–related increases in left ventricular afterload may promote prosthetic valve stasis through different mechanisms, thereby facilitating thrombosis in susceptible patients. Beyond increasing thrombotic risk, these unfavorable hemodynamic conditions may also hinder early detection of prosthetic valve dysfunction, as reduced pulsatility and limited transvalvular flow can mask early functional deterioration on routine echocardiography. Nevertheless, this prothrombotic environment, shaped by surgical trauma, delayed anticoagulation, prosthetic material, and unfavorable flow conditions, likely

contributed to the development of catastrophic prosthetic mitral valve thrombosis and subsequent cerebral infarction.

1. When to Combine IABP?

During VA-ECMO support, LV unloading is often necessary to reduce pulmonary congestion, prevent ventricular distension, and promote myocardial recovery. Indications include hemodynamic instability (e.g., pulmonary capillary wedge pressure >18 mmHg, pulse pressure <15 mmHg), echocardiographic signs (progressive LV dilation, stasis, thrombus, closed aortic valve, low LVOT Velocity-Time Integral (VTI) <10 cm), and clinical symptoms like refractory pulmonary edema or malignant arrhythmias (3). Compared to percutaneous devices, the unloading effect of intra-aortic balloon pump (IABP) is relatively limited. Moreover, in VA-ECMO patients with pulse pressure <10 mmHg, IABP may paradoxically reduce cerebral and systemic perfusion (4), making it more suitable for those with preserved LV contractility.

Although the IABP-SHOCK II trial demonstrated no survival benefit of IABP in myocardial infarction-related cardiogenic shock, this study did not include patients following valve surgery (5). In patients receiving VA-ECMO after valve surgery, adjunctive IABP use has been associated with a reduced risk of thrombus formation

and lower in-hospital mortality (6). Although direct comparisons between IABP and LV venting cannulation are lacking, some studies comparing IABP and Impella in VA-ECMO patients suggest that Impella may increase bleeding risk, while ECMO+IABP showed a survival benefit over ECMO alone (log-rank P = 0.005), but ECMO+Impella did not (log-rank P = 0.66) (7).

In this case, surgical LV rupture prompted early unloading via direct cannulation, primarily to lower LV pressure, reduce the risk of re-rupture, and control ventricular dilation. On ECMO day 4, partial myocardial recovery was noted. After initial weaning failure, IABP was added to reduce afterload and mitigate acute pulmonary edema, enhancing the likelihood of successful recovery. Thus, in VA-ECMO patients with residual LV function, IABP may represent a reasonable unloading option, as it can reduce ventricular load, promote aortic valve opening, and potentially lower thrombotic risk by minimizing intracardiac foreign bodies. Nevertheless, compared with microaxial pumps such as Impella, the unloading effect of IABP is indirect and limited, and the choice of unloading strategy should be guided by clinical urgency, bleeding risk, and device availability.

2. Thrombosis Associated with Surgical LV Unloading

Surgical unloading involves adding a 16–20 Fr drainage cannula via a Y-connector into the ECMO venous limb, with insertion sites including the LV apex, pulmonary vein, or pulmonary artery. This method is especially effective for patients with severe ventricular dilation, cardiogenic shock, and minimal native ejection (3).

Danial et al. analyzed 152 patients who developed cardiogenic shock after valve surgery and required VA-ECMO, 9 of them developed prosthetic valve thrombosis (6). Consistent with prior evidence, valve surgery combined with VA-ECMO support represents a profoundly thrombogenic condition, even in the absence of direct LV venting (2). Although direct LV venting can effectively reduce LV preload and mitigate intracavitary blood stasis, the proximal aortic root may still experience sluggish flow, which predisposes it to thrombus formation (8). According to ELSO guidelines, anticoagulation during VA-ECMO should be monitored by laboratory assays such as aPTT or anti-Xa activity, with a target anti-Xa level of 0.3–0.7 IU/mL.

In our patient, anticoagulation was initiated once bleeding was controlled and titrated according to standard ACT and aPTT targets; however, whether conventional anticoagulation goals are

sufficient in postcardiotomy VA-ECMO patients with prosthetic valves remains uncertain. Besides, these markers fail to capture the real-time interactions between blood, endothelium, and artificial surfaces—limiting our understanding of true thrombotic risk (9). Notably, acute thrombosis of mitral bioprostheses has been reported even in the early postoperative period without ECMO or mechanical unloading, underscoring the inherent thrombogenicity of recent valve replacement itself (10). A similar case was reported by Imada et al. (11), where a patient developed mitral valve bioprosthesis thrombosis after mitral valve replacement and VA-ECMO support with LVAD insertion.

Additionally, during ECMO weaning, current guidelines recommend gradual flow reduction to about 1 L/min, followed by comprehensive evaluation prior to decannulation. However, the low-flow period increases thrombotic risk both in the patient and the ECMO circuit. Although intensified anticoagulation may be theoretically indicated during this phase, no consensus exists on optimal targets. Transesophageal echocardiography appears valuable during weaning for detecting intracardiac thrombus and assessing prosthetic valve function. More frequent or continuous TEE surveillance during critical phases of ECMO support and

weaning may help anticipate valve-related complications in high-risk patients.

4. Conclusion

In patients supported with VA-ECMO after valve replacement surgery, thrombotic complications result from a complex interplay of surgical trauma, altered flow dynamics, delayed anticoagulation, and prosthetic material. In cases of severe ventricular dysfunction requiring surgical LV venting, heightened awareness of thrombotic risk is essential. During ECMO weaning, careful flow modulation, individualized anticoagulation management, and transesophageal echocardiographic monitoring may improve procedural safety.

Abbreviations

ACT	Activated Clotting Time
aPTT	activated Partial Thromboplastin Time
anti-Xa	Anti-Factor Xa Activity
AVR	Aortic Valve Replacement
CPB	Cardiopulmonary Bypass
CTA	Computed Tomography Angiography
ELSO	Extracorporeal Life Support Organization

FiO_2	Fraction of Inspired Oxygen
IABP	Intra-Aortic Balloon Pump
LCOS	Low Cardiac Output Syndrome
LVEF	Left Ventricular Ejection Fraction
LV	Left Ventricular
LVEF	Left ventricular ejection fraction
LVOT	Left Ventricular Outflow Tract
MVR	Mitral Valve Replacement
$PaCO_2$	Partial Pressure of Carbon Dioxide in Arterial Blood
PaO_2	Partial Pressure of Oxygen in Arterial Blood
PEEP	Positive End-Expiratory Pressure
pLVAD	Percutaneous Left Ventricular Assist Device
SAM	Systolic Anterior Motion (of the mitral valve)
TEE	Transesophageal Echocardiography
VA-ECMO	Veno-Arterial Extracorporeal Membrane Oxygenation
VTI	Velocity-Time Integral

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Figure legend

Figure 1. Perioperative imaging findings.

(A, B) Chest CT images demonstrate bilateral pulmonary edema.

(C, D) Transesophageal echocardiography reveals thickening of one mitral prosthetic leaflet with decreased echogenicity (*arrows*), suggestive of thrombus formation.

TEE = transesophageal echocardiography; CT = computed tomography.

Figure 2. Time line and periopeartive laboratory results.

aPTT (s) = activated partial thromboplastin time;

cTNT (pg/mL) = cardiac troponin T;

ECMO = extracorporeal membrane oxygenation;

IABP = intra-aortic balloon pump;

LV = left ventricle;

NT-proBNP (pg/mL) = N-terminal pro B type natriuretic peptide;

PLT (*10^9/L) = platelet count;

TTE = transthoracic echocardiography;

WBC (*10^9/L) = white blood cell count.

Author contributions

J.H., L.L and Y.X. wrote the main manuscript text, J.H. and T.S. prepared figures 1-2. All authors reviewed the manuscript.

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Data availability

The datasets used and/or analysed during the current study are available from

the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The study involving human participant was reviewed and approved by Ethics Committee of Guangdong Provincial People’s Hospital.

Consent for publication

Informed written consent was obtained from the patient for

publication of this report and any accompanying images.

Competing interests

The authors declare no competing interests.