THROMBOSIS OF MECHANICAL VALVE PROSTHESIS: A CASE REPORT

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Abstract

Mitral valve prosthesis thrombosis is a rare but life-threatening complication associated with mechanical heart valves. It requires timely diagnosis and prompt intervention to prevent severe morbidity and mortality. We report the case with hemoptysis, severe dyspnea, and fever, two years after mechanical mitral valve replacement with a St. Jude Medical prosthesis. Despite consistent anticoagulation therapy, recent transition from warfarin to low-molecular-weight heparin during hospitalization for pneumonia may have contributed to prosthetic thrombosis. Echocardiography revealed significant mitral valve dysfunction with a mean pressure gradient of 45 mmHg and evidence of thrombus formation. Emergency surgery confirmed total prosthetic valve thrombosis and necessitated thrombectomy and replacement with a new mechanical valve. Postoperative recovery was uneventful, with improved hemodynamics and resolution of pulmonary edema. This case highlights the complexities of managing mechanical valve thrombosis in patients with multiple risk factors, including anticoagulation changes, atrial fibrillation, and recurrent pulmonary infections. The surgical approach remains the gold standard for treatment, though emerging evidence supports the potential role of thrombolysis in select cases. In conclusion, mitral valve prosthesis thrombosis represents a significant challenge requiring multidisciplinary management and strict anticoagulation monitoring, and the importance of developing standardized protocols for early diagnosis, anticoagulation management, and surgical intervention.

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Introduction

Heart defects with valvular dysfunction remain a major focus in cardiology and cardiac surgery, affecting over 100 million people worldwide and contributing significantly to disability and mortality.[1,2] Surgical intervention, including valve repair or replacement, is the cornerstone of treatment. In recent years, transcatheter techniques for valve repair and replacement have gained widespread adoption due to their minimally invasive nature.

However, all prosthetic heart valves, whether mechanical or biological, are inherently thrombogenic and associated with a risk of complications. These devices necessitate anticoagulant therapy, which may be required for a short or extended duration depending on the type of prosthesis and individual patient factors.

Prosthetic valve dysfunction can arise from multiple causes, including impaired leaflet mobility, incomplete closure, thinning of biological leaflets, alterations in the effective orifice area, or changes in transvalvular pressure gradients.[3] The underlying etiologies of prosthetic dysfunction include thrombosis, prosthetic endocarditis with vegetation formation, pannus formation, and leaflet degeneration in biological prostheses.[4] Notably, the risk of thrombus formation is significantly higher with mechanical prostheses compared to biological ones.[5] However, the choice of prosthesis type depends on specific patient characteristics and clinical indications, each with distinct advantages and limitations.

This report aims to present a severe complication of thrombosis involving a mechanical mitral valve prosthesis, highlighting the clinical challenges and considerations associated with this condition.

Case presentation

A 44-year-old male chef was urgently admitted to the intensive care unit

presenting with a two-day history of hemoptysis, severe dyspnea at rest, and fever reaching 39.5°C. His medical history revealed degenerative mitral valve insufficiency with chordae rupture, for which he underwent surgical replacement in 2019 using a St. Jude Medical mechanical mitral prosthesis (size 31). Concurrent tricuspid regurgitation was managed with De Vega annuloplasty during the same procedure. The patient had been on long-term warfarin therapy, maintaining therapeutic international normalized ratio (INR) levels. His history was further complicated by cardiac arrhythmias, including paroxysmal bradycardic atrial fibrillation, necessitating the implantation of a dual-chamber pacemaker. In 2022, worsening heart failure was documented, with a left ventricular ejection fraction (LVEF) reduced to 20% and a left ventricular end-diastolic volume (LVEDV) exceeding 200 mL. To address the progressive deterioration, the pacemaker system was upgraded to an implantable cardioverter defibrillator (ICD). Unfortunately, no additional detailed medical history or clinical records were available from the patient or his family.

The patient had a long-standing history of grade 3 arterial hypertension and was on a regimen of bisoprolol, spironolactone, and warfarin. Since late 2019, he had also experienced recurrent episodes of community-acquired pneumonia, requiring periodic hospitalizations. In recent weeks, he was admitted to a therapeutic department where anticoagulation therapy was transitioned from warfarin to subcutaneous low-molecular-weight heparin. However, his condition progressively worsened, culminating in signs of pulmonary edema. A chest X-ray (Figures 1) and computed tomography (CT) scan confirmed pulmonary edema with associated hypostatic changes. Transthoracic echocardiography (TTE) revealed significant mitral valve dysfunction, with a maximum pressure gradient across the valve of 68 mm Hg and a mean gradient of 45 mm Hg. The mobility of the prosthetic mitral valve's moving elements was markedly reduced, and echodense structures were visualized on its ventricular surface, indicative of thrombotic material.

Left ventricular volumetric analysis (M-mode, Teicholz method) demonstrated a left ventricular end-diastolic volume (LVEDV) of 183 mL, end-systolic volume (ESV) of 88 mL, and stroke volume (SV) of 95 mL, with a calculated left ventricular ejection fraction (LVEF) of 51%. Systemic diastolic pressure (SDP) was measured at 70 mm Hg (Figure 2).

A multidisciplinary team reviewed the findings and confirmed the diagnosis of mechanical mitral valve thrombosis, prosthetic dysfunction, and pulmonary edema. Given the severity of the condition, an emergency surgical intervention was deemed necessary.

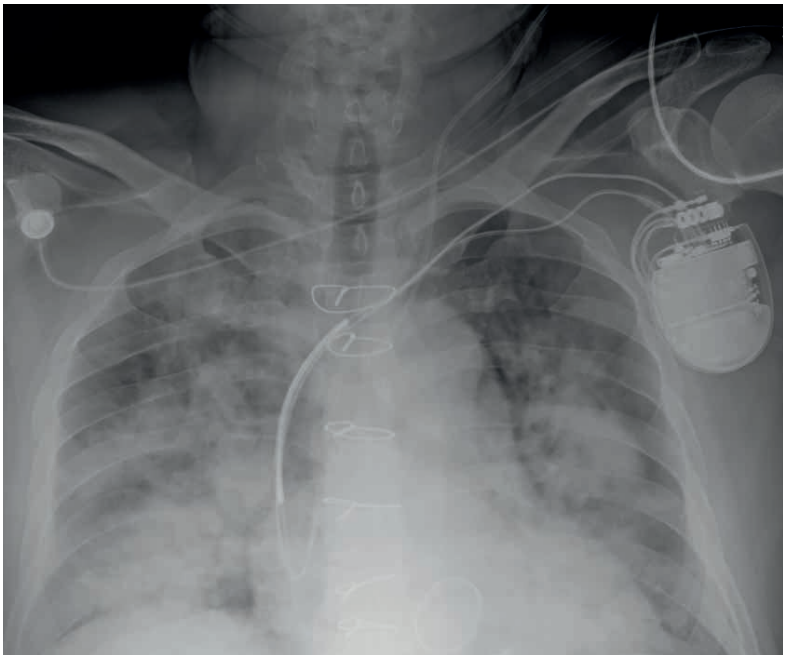
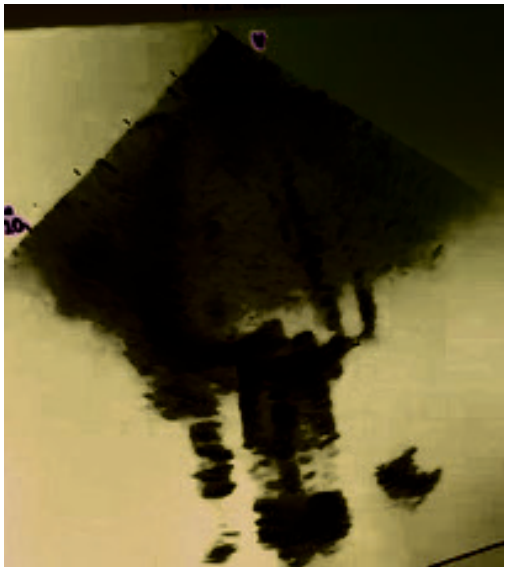


Figure 1.
The chest X-ray: pulmonary edema



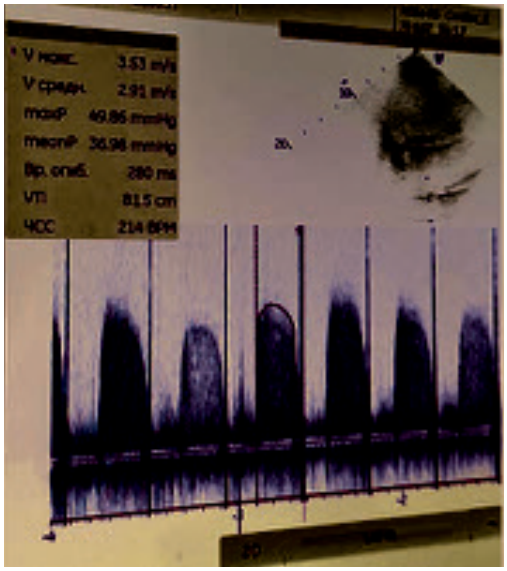


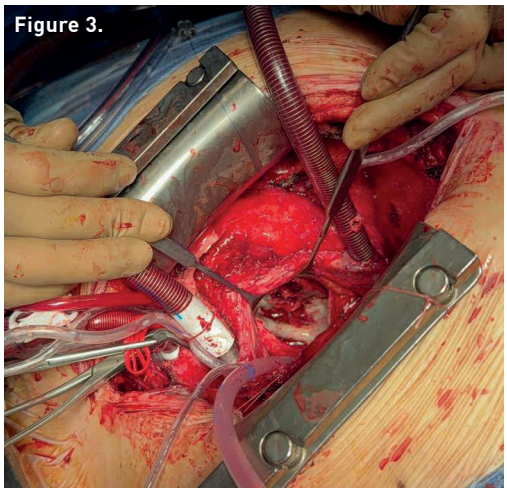
Figure 2.
Transthoracic echocardiography before intervention

The surgery commenced with a resternotomy. Upon reopening the sternum, extensive adhesive processes were encountered, necessitating a subtotal cardiolysis to free the cardiac structures. Standard cannulation of the ascending aorta was performed, along with separate cannulation of the superior and inferior vena cava. Cardiopulmonary bypass (CPB) was initiated, and the aorta was cross-clamped. Antegrade cold blood cardioplegia was delivered into the aortic root to achieve myocardial protection.

The left atrium was accessed through the Waterstone groove for revision. The left atrial cavity was notably enlarged; however, no thrombotic masses were identified in the left atrial appendage or at the pulmonary vein ostia. During examination of the mitral valve, complete thrombosis of the bi-leaflet mechanical prosthesis was observed (Figures 3-4). Both leaflets were encased in thrombotic material, comprising both fresh and organized thrombi, which severely impaired the mobility of the prosthetic components (Figures 5-6).

The thrombotic material was meticulously removed (Figure 4), and the dysfunctional prosthesis was explanted (Figure 5). A new St. Jude Medical mechanical prosthesis (size 29) was implanted in the intra-annular position using 14 "P"-shaped sutures with spacers to ensure secure fixation. The left atrium was subsequently closed. Following the release of the aortic clamp, spontaneous cardiac function was successfully restored.

The subsequent stages of the surgery, including weaning from cardiopulmonary bypass, placement of drains, and metal osteosynthesis of the sternum, proceeded without complications. The total duration of cardiopulmonary bypass was 184 minutes, with a cardiac arrest time of 127 minutes.



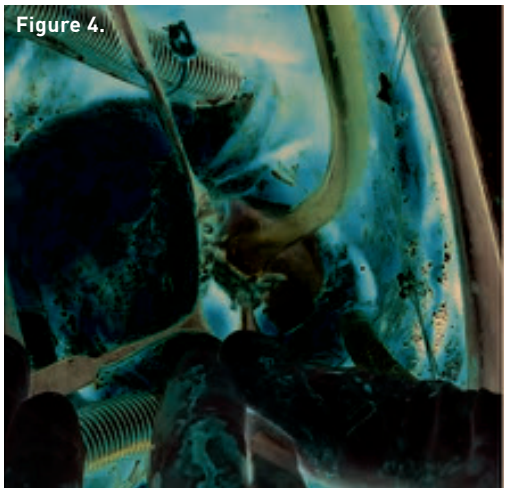
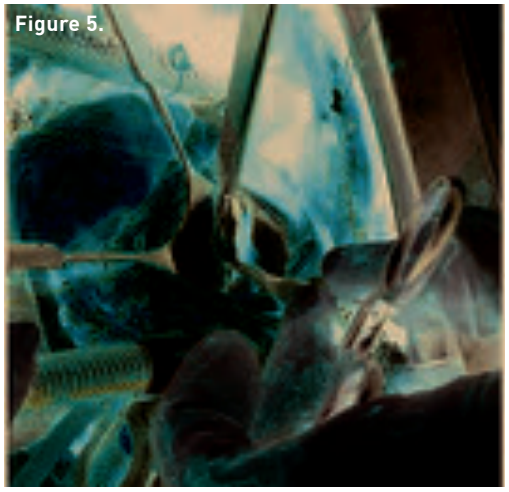


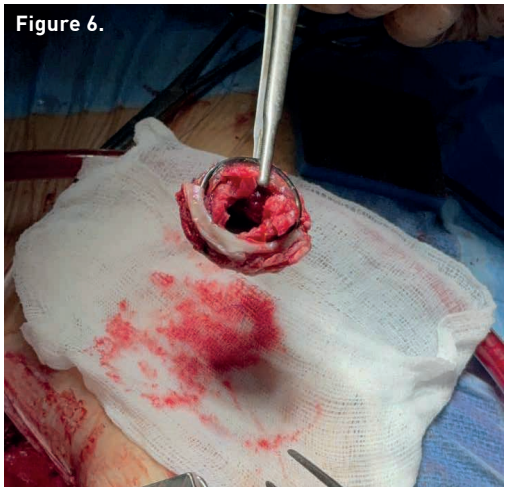
Figure 3.
Thrombosis of the mechanical mitral valve prosthesis

Figure 4.
Removal of the platelet concentrate

Figure 5.
Explantation of the mechanical prosthesis

Figure 6.
Dysfunction of prosthesis





After the surgery, the patient was transferred to the intensive cardiac care unit (ICU) on inotropic support with *Norepinephrine* 170 ng/kg/min and *Dobutamine* 7 µg/kg/min. The patient's condition was successfully stabilized postoperatively. A transthoracic echocardiogram was performed, showing the following results:

- Mean gradient across the mitral valve prosthesis: 5 mm Hg;
- Maximum gradient: 7 mm Hg;
- Left ventricle: End-diastolic diameter (EDD) 4.8 cm, End-systolic diameter (ESD) 3.2 cm;
- Left ventricular volumes (Teicholz method): End-diastolic volume (EDV) 106 ml, End-systolic volume (ESV) 40 ml, Stroke volume (SV) 66 ml, Ejection fraction (EF) 62% (66% by Simpson's method);
- Systemic diastolic pressure: 42 mm Hg;

There was positive progression in the chest X-ray results, with the resolution of pulmonary edema. The patient continues to receive intensive therapy in the ICU.

Discussion

All artificial heart valve prostheses are thrombogenic, posing significant risks of complications despite their life-saving benefits. Implantation imposes a rigorous treatment regimen on patients, requiring consistent management of risk factors, strict adherence to anticoagulant therapy, and regular clinical monitoring. Thrombosis of mitral valve prosthesis, while rare with proper therapy, remains a severe and life-threatening complication that demands an individualized approach, particularly in determining the timing of surgical intervention.[6] The complexity of this condition stems from its profound implications for systemic circulation, making prompt diagnosis and intervention crucial.

Surgical management, including thrombus removal and prosthetic valve reimplantation, continues to be the cornerstone of treatment. Survival rates correlate with the extent of thrombus involvement, with better outcomes observed in cases identified and managed early. Despite advancements in treatment, no standardized protocol for managing prosthetic valve thrombosis exists, underscoring the importance of case-specific, multidisciplinary team discussions for optimal decision-making.[7,8]

Emerging evidence on thrombolytic therapy for prosthetic valve thrombosis offers a potential alternative or adjunct to surgical management but requires further validation through robust clinical trials.[9] In this case, the surgical intervention was executed effectively, addressing the circulatory insufficiency caused by the thrombosis.[10]

Limitations. This case highlights multiple contributing factors to the development of mitral valve prosthesis thrombosis. However, significant gaps in clinical data limit a comprehensive understanding of the patient's condition. There is limited information regarding the patient's postoperative anticoagulation management, especially during the first year after mitral valve replacement. Additionally, the adequacy of warfarin therapy during this period is unclear. The presence of atrial fibrillation, ne-

cessitating pacemaker implantation, further compounded the patient's risk of thrombosis. The duration and extent of left ventricular systolic dysfunction also remain poorly defined. Other contributing factors, such as inflammatory lung processes and the transition from warfarin to low-molecular-weight heparin, likely played a role in thrombus formation. Furthermore, the precise factors leading to heart failure at the time of initial treatment and the pharmacological regimen that improves the ejection fraction remain unclear.

What's known? Studies indicate that the risk of thrombus formation is highest within the first three months post-implantation, emphasizing the critical importance of stringent anticoagulant therapy and INR monitoring during this period. Risk stratification tools like the CHA2DS2-VASc score can aid in tailoring management strategies.

What's new? Regular echocardiography evaluation, clinical assessment of thrombotic and bleeding risks and close monitoring allow to decrease of risk. As the number of patients with heart valve prostheses continues to rise, the development of standardized protocols for early risk factor identification, thrombus prevention, and safe, effective anticoagulation therapy is imperative.

Conclusions

Mitral valve prosthesis thrombosis is a rare but life-threatening complication requiring careful and comprehensive management. The presence of comorbidities such as atrial fibrillation and other implanted devices increases the likelihood of thrombotic events. This case underscores the need for ongoing research and multidisciplinary collaboration to improve outcomes for patients with prosthetic heart valves.

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