


A Rare Case of Partial Aortic Mechanical Valve Thrombosis With Intact Mitral Mechanical Valve Presenting With ST-Elevation Myocardial Infarction Patients

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Abstract

The incidence of mechanical valve thrombosis (MVT) is around 0.4 per 100 patient-years. Mitral valve thrombosis has a higher incidence than aortic valve thrombosis with a nearly 5-fold increase. Various factors contribute to MVT. The most common cause of valve thrombosis is poor adherence/disruption of anticoagulation therapy. Low cardiac output is known to increase the risk of prosthetic valve thrombosis. Other factors such as diabetes, hypertension, and other patient comorbidities might also play a role. Decreased flow promotes hypercoagulability. Lower pressure in the left atrium (and higher velocities in the left ventricle) can partially contribute to the higher incidence of mitral MVT versus aortic MVT. The presenting symptoms usually depend on the severity of the valve thrombosis; nonobstructive valve thrombosis patients have progressive dyspnea, signs of heart failure, and systemic embolization with strokes being the most common complication. In this article, we present a case of a middle-aged woman with a history of mitral and aortic mechanical prosthesis who presented with an ST-segment elevation myocardial infarction and pulmonary edema due to mechanical aortic valve prosthesis thrombosis. She had an isolated mechanical aortic valve prosthesis thrombosis with intact mitral valve, which, to the best of our knowledge, has not yet been described. We performed a literature review by searching PubMed and Embase using the keywords “mechanical valve,” “thrombosis,” “aortic,” and “mitral,” our search did not show similar cases.

Keywords

mechanical aortic valve, thrombosis, STEMI, embolization

Introduction

The incidence rate of mechanical valve thrombosis (MVT) is around 0.4 per 100 patient-years. Mitral valve thrombosis has a greater incidence compared with aortic valve thrombosis with a nearly 5-fold increase (0.5 per 100 patient-years vs 0.1 per 100 patient-years, respectively).¹ While rare, MVT is a serious problem that requires reoperation. The thrombus formation is explained by the triad of stasis, endothelial injury, and hypercoagulability.²

The St. Jude Medical (SJM) bi-leaflet mechanical valve (St. Jude Medical, Inc) is the most commonly used valve in aortic valve replacement (AVR) since it was first introduced to the market in 1978.^{3,4} Numerous studies have reported excellent hemodynamics, durability, and lower complication rates in patients who underwent AVR with SJM mechanical valves.⁵⁻⁸

In this article, we present a case of a middle-aged woman with a history of mitral and aortic mechanical prosthesis who presented with an ST-segment elevation myocardial

infarction (STEMI) and pulmonary edema due to mechanical aortic valve prosthesis (MAVP) thrombosis. She had an isolated MAVP thrombosis with an intact mitral valve, which, to the best of our knowledge, has not yet been described before.

Case Presentation

A 56-year-old female of Russian descent was brought to the emergency department with a complaint of chest pain. She described the pain as retrosternal, dull in nature, 9 out of 10

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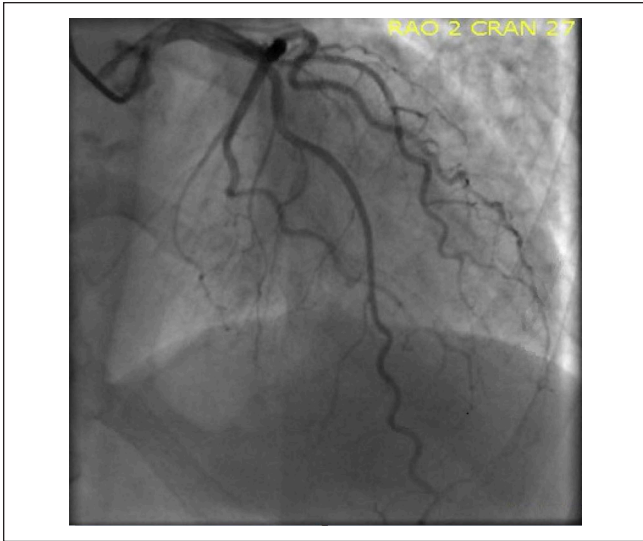


Figure 1. Coronary angiogram from 2016 showing normal left anterior descending coronary artery.

in intensity, non-radiating, and was associated with shortness of breath. Her medical history included hypertension, diabetes, hyperlipidemia, heart failure with preserved ejection fraction, chronic obstructive pulmonary disease, hypothyroidism, paroxysmal atrial fibrillation, and rheumatic mitral valve disease status post metallic mitral and AVR for which she was on warfarin. The patient stated that while she has been taking her medications religiously, she had had nosebleeds recently and contacted her doctor who advised to pause warfarin therapy. In 2016, the patient was admitted to our hospital with complaints of shortness of breath and had elevated troponins for which she had an angiogram that showed normal coronaries (Figure 1).

On arrival to the emergency room, she was still in pain on a non-rebreather mask with oxygen saturation of 98%. Her heart rate and blood pressure were normal. Her examination was remarkable with a healed longitudinal scar over her sternum, audible click in the mitral area, and lower lung crackles on lung auscultation. Her electrocardiogram showed ST-segment elevation in leads aVL and V1 with reciprocal ST depressions in the inferolateral leads (Figure 2). She was taken for an emergent coronary angiogram that revealed 100% thrombotic occlusion of left anterior descending and first diagonal (D1) arteries (Figure 3). The other vessels were normal. A successful fetch thrombectomy was performed.

Given the patient's shortness of breath, high oxygen requirement, and the diffuse thrombosis of her vessels, we suspected coronavirus disease 2019 infection (COVID-19); hence, we performed balloon angioplasty and opted to defer coronary stenting pending COVID-19 testing. She was given 80 mg of furosemide intravenously and started on tirofiban and heparin infusion intravenously. She was given aspirin and prasugrel and was transferred to the coronary care unit. Her laboratory values were remarkable for elevated serum

creatinine of 1.5 (normal 0.3-1.1 mg/dL), elevated troponin level 0.44 (0.00-0.04 ng/mL), and subtherapeutic INR (international normalized ratio) 1.3 (normal 0.9-1.2). We sent 2 nasal swabs for a polymerase chain reaction, and both were negative for COVID-19. The antibody test was negative for IgG and IgM. We obtained her old operative report from 2016, which revealed that she had AVR with SJM regent series, size 19 mm, and mitral valve replacement with SJM Masters series, size 27 mm, and aortoplasty using bovine pericardium.

We next performed an echocardiogram that showed moderately decreased left ventricular systolic function with ejection fraction 31% to 35%, abnormal wall motion in the anterior septum, mid and apical inferior septum, and anterior apical segment. She had a severely dilated left atrium (LA) with an LA volume index 52.61 mL/m²; mechanical prosthetic mitral valve with a mean gradient of 9 mm Hg; mechanical prosthetic aortic valve (MPAV) with a dimensionless index of 0.29; and moderate to severely elevated pulmonary artery systolic pressure. Her elevated gradients raised a suspicion for valve thrombosis. Fluoroscopy revealed normal motion on the leaflets of the mechanical mitral prosthesis and only one mobile leaflet in the MPAV (Figures 4 and 5). Video 1 (available online) shows a fluoroscopy clip taken from a left anterior oblique and caudal projections of a normally functioning mitral prosthesis (as shown in the lower right aspect of the video) with a single leaflet motion in the aortic valve (shown in the left upper aspect). We made a diagnosis of aortic valve thrombosis, and her STEMI was thought to be due to coronary embolization from the MPAV. Meanwhile, the patient's new-onset systolic heart failure was treated with intravenous diuretics. Cardiothoracic surgery was consulted, and they recommended therapeutic anticoagulation and repeat fluoroscopy in a week to assess the motion of the MPAV leaflets. A transesophageal echocardiogram was done for better evaluation of the valvular pathology. It showed thrombus within the prosthetic aortic valve prolapsing into the left ventricular outflow tract as well as toward the ascending aorta. Flow across the valve was limited to the posterior portion of the lumen consistent with immobilization of the anteriorly positioned leaflet. A small mobile thrombus at the anterolateral mitral annulus was suspected. The patient's clinical condition improved, and her oxygenation was normal on room air. Repeat fluoroscopy showed similar results with persistence of the MPAV leaflet motion restriction. She was discharged in a stable condition on a therapeutic dose of warfarin, aspirin (81 mg), and plans to follow with her cardiothoracic surgeon for evaluation of reoperation.

Discussion

Mechanical valve thrombosis is defined as any obstruction of the prosthetic heart valve by a noninfective thrombotic material.⁹ Despite having a low incidence, MVT remains a challenging complication. The most common cause of valve

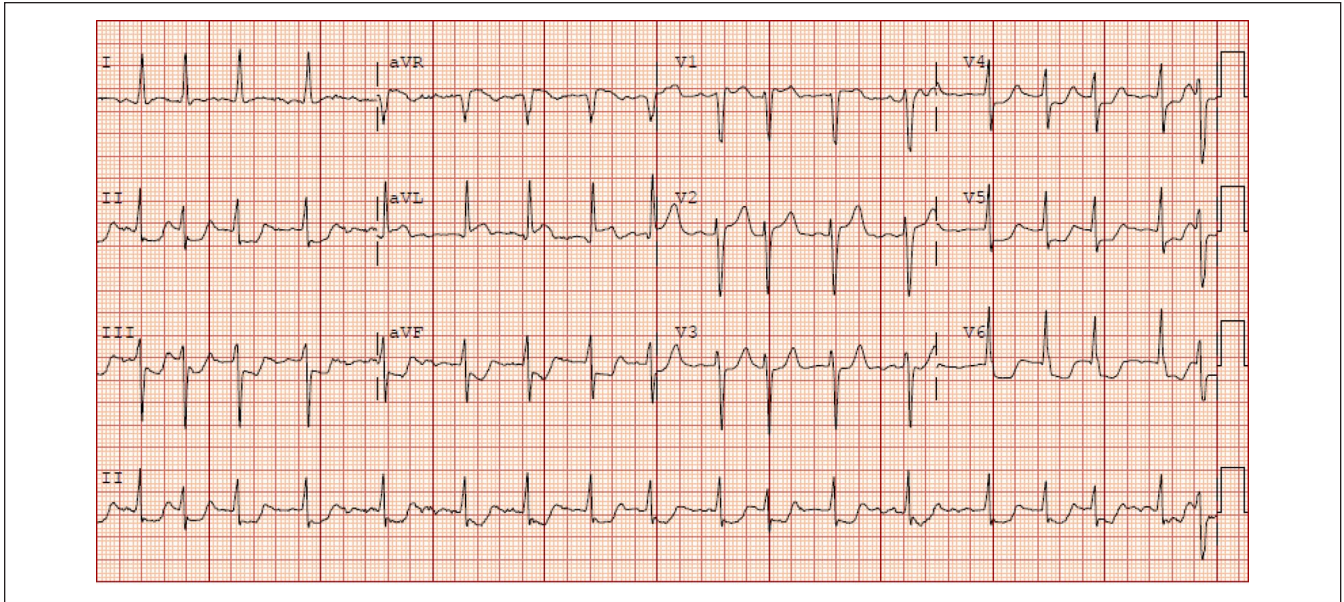


Figure 2. The electrocardiogram shows ST-segment elevation in lead aVL and V1 with reciprocal ST-segment depressions in inferolateral leads.



Figure 3. Coronary angiogram showing a large thrombus in the proximal left anterior descending artery.

thrombosis is poor adherence/disruption of anticoagulation therapy. Low cardiac output is a known factor that increases the risk of prosthetic valve thrombosis due to reduced leaflet motion.¹⁰ The decreased flow promotes hypercoagulability by reducing the clotting factors washout and limiting the inhibitor's flow in the adjacent area. The lower pressure encountered in the left atrium (and the higher velocities in the left ventricle) can partially contribute to the higher incidence of mitral MVT versus aortic MVT.¹¹ In addition, the turbulent flow promotes platelet adhesion to the valve surface and delays endothelialization, which affects the malposition of

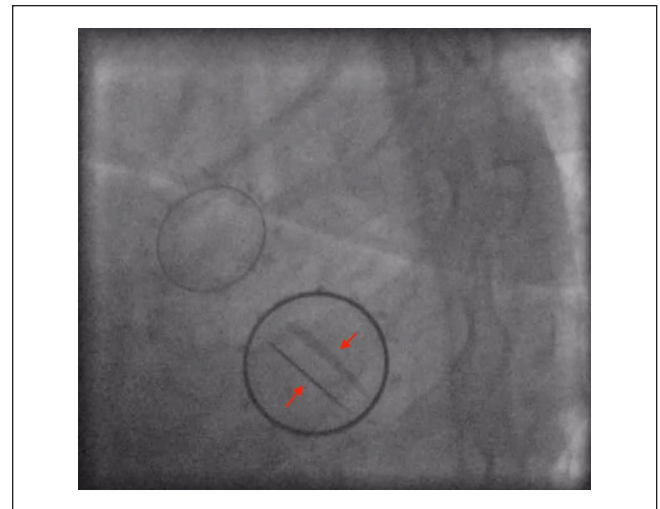


Figure 4. Fluoroscopy image showing mechanical mitral valve prosthesis with bi-leaflet opening as shown by the red arrow.

prosthetic valves. Other factors, such as diabetes, hypertension, and other patient comorbidities, might play a role.¹²

The presenting symptoms usually depend on the severity of the valve thrombosis; nonobstructive valve thrombosis patients usually have progressive dyspnea, signs of heart failure, and systemic embolization. Strokes are the most common complication.² Importantly, patients with delayed identification of obstructive valve thrombosis may present with cardiogenic shock. The diagnosis is made through a combination of the clinical picture, fluoroscopy, or echocardiography. Cardiac catheterization is rarely necessary to make a

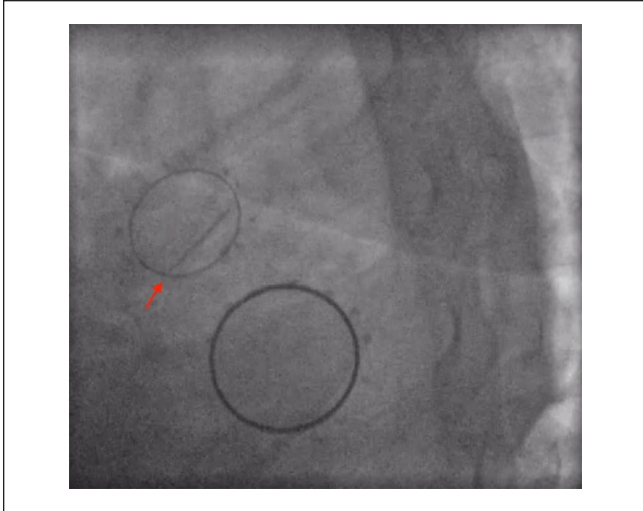


Figure 5. Fluoroscopy image of a mechanical aortic valve prosthesis with a single leaflet opening as shown by the red arrow.

diagnosis. The gold standard for treatment of MVT remains reoperation with better results demonstrated in patients without hemodynamic compromise. Reoperation for both aortic and mitral valve mechanical prosthesis carries an equal early operative mortality.¹³ Emergent and urgent surgery are associated with a mortality rate of 7.1% to 69%—these rates lead to thrombolysis being tried with a success rate of 80% to 91% and carrying a lower mortality rate of 4% depending on the obstruction and functional class.¹⁴

A recent study examined patients with SJM double valve replacement with a mechanical prosthesis, and only 1% of the patients needed reoperation for valve thrombosis.¹⁵ Thrombolysis has been tried with promising results; in fact, trials have demonstrated that low-dose thrombolytics were as effective as high doses.¹⁶ Interestingly, in our patient, the mitral valve leaflets had normal motion, while the aortic leaflet was thrombosed. While pannus formation is a known cause of MAV obstruction, it does not explain the cause of the STEMI that the patient suffered. The likely explanation, given her clinical presentation and subtherapeutic anticoagulation, is a thrombosed aortic valve leaflet with embolism into the left coronary artery. A similar case has been reported with a right coronary artery thrombus leading to acute coronary syndrome necessitating thrombolytic administration. The patient had hemodynamic instability and a large thrombus burden.¹⁷ We could not explain the paradox of the normal motion of the mitral valve with thrombosis of the aortic valve—this contradicts the norm.

Conclusion

It is important to include valve thrombosis in the differential in the presence of mechanical prosthesis and STEMI—especially in the setting of heart failure and/or angiographic characteristics compatible with large thrombus burden. We

also highlight the possibility of isolated MAVP thrombosis with an intact mechanical mitral valve. More research is needed to identify similar cases and the etiology of such a unique case.

Declaration of Conflicting Interests

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Ethics Approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed Consent

Written informed consent was obtained from the patient for his anonymized information to be published in this article.

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Supplemental Material

Supplemental material for this article is available online.

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