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Multiple Emboli Caused by Mechanical Mitral Valve Thrombosis: A Case Report

Armando Cardosa-Aguilar[1], Luis E. Martínez-Bravo[2], Elizabeth Armijo-Yescas[1], Tania Hernández-Trejo[1], Carlos Augusto Contreras-Martinez[1]

[1] Department of Cardiology, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado, Hospital Regional de Alta Especialidad Bicentenario de la Independencia, Tultitlan, Estado de Mexico, Mexico

[2] Instituto Mexicano del Seguro Social, Hospital General de Zona N.º 4, Guadalupe, Mexico.

ABSTRACT

We present the case of a 51-year-old woman with a history of atrial fibrillation and rheumatic heart disease who underwent a prosthetic mitral valve replacement two years ago. Despite receiving anticoagulant therapy, this patient experienced embolisms, including embolic strokes and a coronary embolus, over a month. Following a cardiology evaluation, it was determined that mechanical valve thrombosis was the primary source of embolism, and embolisms ceased following a replacement. This case report illustrates the difficulty in diagnosing prosthetic valve thrombosis as a source of multiple emboli in patients receiving adequate anticoagulant therapy, a rare but potentially fatal complication of mechanical valve replacement.

KEYWORDS: Mechanic valve thrombosis, mitral valve, embolic stroke, myocardial infarction, atrial fibrillation, case report.

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INTRODUCTION

Prosthetic valve thrombosis (PVT) is a serious complication of valve replacement that occurs most frequently with mechanical valves. Mechanical valve thrombosis occurs at a rate ranging from 0.1% to 5.7% per year, with mitral prosthetic valve thrombosis occurring five times more frequently than aortic prosthetic valve thrombosis [1, 2]. The European Society of Cardiology (ESC) and the American Heart Association (AHA) both recommend vitamin K antagonists in order to avoid additional thrombosis after mechanical valve implantation [3,4]. Poor anticoagulation adherence and inadequate INR management are risk factors for PVT. The clinical appearance varies from non-obstructive patients like heart failure to obstructive situations with fatal consequences like cardiogenic shock. Herein, we present a case of numerous emboli caused by mitral mechanical valve thrombosis that occurred despite adequate anticoagulation these events were resolved after the valve was replaced.

CASE REPORT

A 51-year-old female with a history of permanent atrial fibrillation who underwent mechanical mitral valve replacement (Carbomedics 27 mm) for rheumatic heart disease and was on acenocoumarin therapy. She developed sudden left hemiparesis two years after valve replacement, and a CT scan of her brain revealed an acute ischemic area in the external capsule and putamen, indicating a right middle cerebral artery embolism. Her lab tests reported an INR of 2.5, and a transthoracic echocardiogram no signs of valvular thrombosis. She was discharged one week later after improvement. After one month, she presented with dyslalia. She went to the emergency room, where she was diagnosed with a transient ischemic attack; her brain CT scan revealed no new findings; and after five days of hospitalization, she complained of chest pain and dyspnea. Her heart auscultation revealed a decreased prosthetic click, her electrocardiogram showed atrial fibrillation with ST-segment elevation from V1-V3, a negative T-wave in the inferior and lateral leads, and an alternating bundle branch block (Figure 1), and a cardiology consultation was requested for acute coronary syndrome.

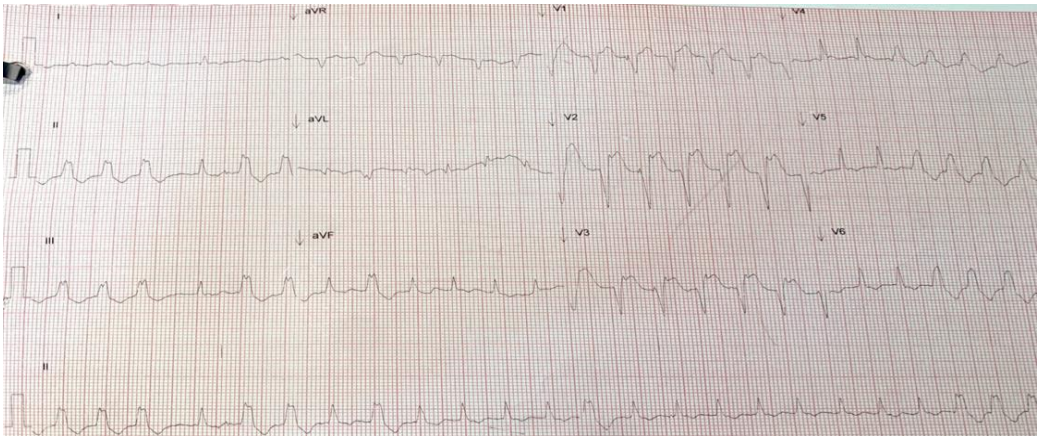

Figure 1. Electrocardiogram with anterior wall ST segment elevation, atrial fibrillation, and alternans left bundle-branch block.

Her transesophageal echocardiography shown global hypokinesia, a reduced ejection fraction of 20%, mechanical prosthesis in mitral position with a 15 mm filiform mobile image and reduced leaflet motion, culminating in a 15mm mitral valve thrombosis (Figure 2).

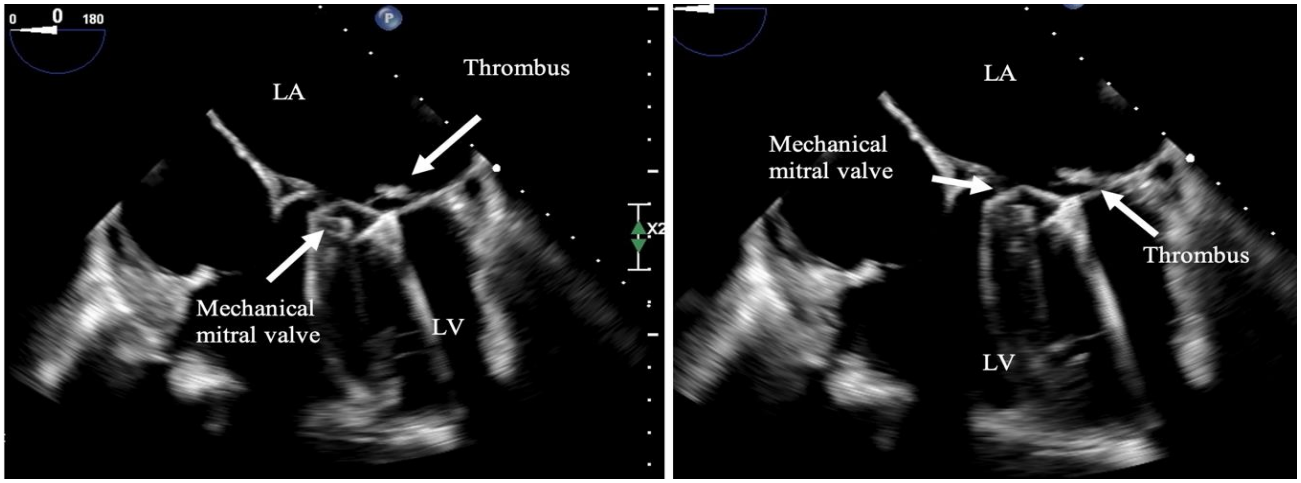

Figure 2. Transesophageal echocardiography reveals a mitral valve mass consistent with valve thrombosis. LA Left atrium. LV Left Ventricle.

After these findings, an emergency cardiac catheterization was performed, which revealed an anterior descending artery blockage with angiographic characteristics consistent with an embolic origin (Figure 3).

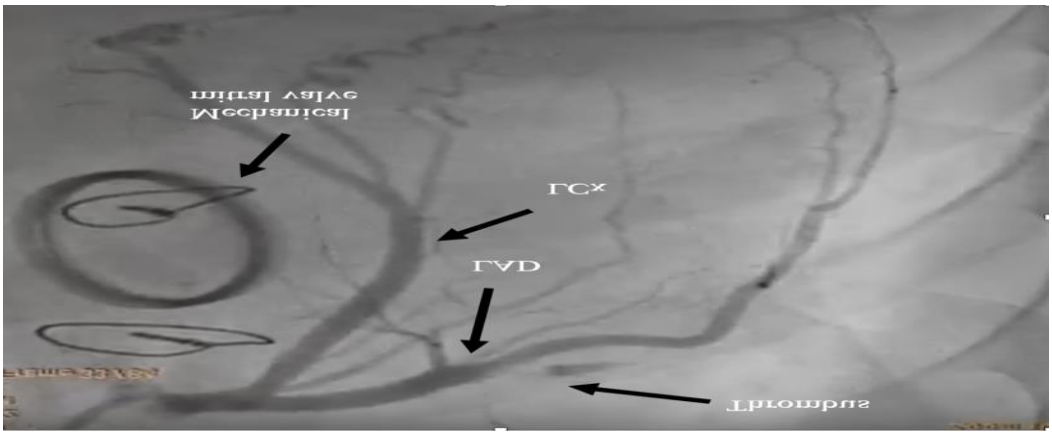

Figure 3. Emergency coronary angiography. On the left anterior descending artery, there is a lack of contrast filling; after resection, this was consistent with a coronary thrombus. LCx Circumflex left artery.

Thrombus aspiration during primary percutaneous coronary intervention was performed, obtaining a white thrombus, restoring coronary flow and an performing an angioplasty with Resulte Onyx Stent 2.5x38 mm with final TIMI 3 flow, due to intra-hemodynamic instability intra-aortic balloon counterpulsation (Arrow ACAT 2 Wave Intra-Aortic Balloon Pump) was implanted, and the patient was admitted to the coronary unit. Following a discussion between the heart team, the patient, and her family, it was decided to refer her for emergency mitral valve replacement (implanting a 27 mm carbomedic valve) the following day; her postoperative care consisted of the removal of the aortic balloon counterpulsation two days later without further complications, her embolic events ceased, and she was discharged on acenocoumarin treatment. In three months of follow-up, she has not developed any new embolic events.

DISCUSSION

PVT is multifactorial, following the classic Virchow's triad in which endothelial factors are crucial for biocompatibility between prostheses and cardiac tissue depending on the suture zone, cicatrization, and reendothelialization. Dysregulation of these factors predisposes to the development of fibrous pannus in 45 to 75% of valve thrombosis cases [5]. Hemodynamic factors include the characteristics of the prosthesis as well as the overall cardiac hemodynamic status; the localized regions of turbulent flow are the most likely to lead to stasis and thrombus formation; primary or secondary hypercoagulability is a relatively uncommon mechanism in prosthetic valve thrombosis; and acquired causes of hypercoagulability include comorbidities such as obesity, smoking, chronic kidney disease, and anemia, which were not present in our case [1].

PVT is classified according to valve leaflet mobility as obstructive prosthetic valve thrombosis and nonobstructive prosthetic valve thrombosis [6]. There is a broad spectrum of clinical manifestations resulting from obstructive valve with hemodynamic compromise and clinical symptoms consistent with reduced cardiac output. The presence of non-obstructive valve thrombosis in asymptomatic patients or as thromboembolic events is possible. The main finding on clinical examination is a reduced or absent prosthetic opening or closing sound (for mechanical valves), new heart murmur or signs of heart failure [2]. Acute cardiac failure, pulmonary edema, cardiogenic shock, embolic events such as cerebrovascular and acute coronary syndromes, and systemic embolism are complications of valve thrombosis. As many as three percent of acute coronary syndromes are attributable to coronary embolism. This embolism often arises by infective endocarditis, cardiac tumors, or left ventricular thrombus, among other conditions [7]. Many of coronary emboli involve the left coronary arteries, which could be explained by the preferential flow into the left artery related to coronary flow volume and aortic valve shape [8]. As with our patient, aspiration thrombectomy is a viable option for patients with a high thrombus burden [7].

Transthoracic echocardiogram, transoesophageal echocardiogram, fluoroscopy, and/or CT imaging is indicated according to AHA guidelines when PVT is suspected to assess valve function, leaflet motion, and the presence and extent of thrombus [4]. The transthoracic echocardiogram reveals an elevated transvalvular gradient (aortic Pmax > 50 mm Hg, mitral Pmean > 10 mm Hg), as well as the presence of a mobile occlusive mass on the prosthetic valve1. Using color Doppler, the presence of aberrant transprosthetic flow or central regurgitation indicates abnormal valve closure [5]. Transoesophageal echocardiogram may visualize the atrial side of mitral prostheses better than transthoracic echocardiogram; it facilitates the detection of the obstruction's cause and the dimension of the thrombus.

Fluoroscopy allows the evaluation of leaflet motion and the measurement of leaflet mechanical opening and closure angles [2]. In patients with unclear transthoracic and transesophageal echocardiography results, multidetector CT could provide precise evaluation of the structure and function of prosthetic valves. CT imaging can differentiate between thrombosis and fibrotic pannus, as well as evaluate the dynamic leaflets and assess the prosthetic valve's morphology [1].

Mechanical valve thrombosis is typically a subacute (3 days to 3 months) to chronic (> 3 months) event resulting in abnormal or absent motion of the valve leaflets, often associated with inadequate anticoagulation [1, 4]. Obstruction is more frequent on right-sided heart valves, with tricuspid mechanical prostheses being more frequent, followed by pulmonary, mitral, and aortic prostheses [1].

The treatment of mechanical valve thrombosis needs to involve the management of risk factors and the optimization of anticoagulation control. [9]. It also depends on the size of the thrombus, and the patient´s medical condition. For patients with NYHA I-III and a thrombus size <0.8mm, medical therapy is recommended [1,5]. Increasing the INR target range to 3.5–4.0 or adding daily low-dose aspirin (75–100 mg), with a Class 2a recommendation from the ESC and AHA guidelines [3,4]. For NYHA III-IV and thrombus size > 0.8mm low-dose fibrinolytic therapy or emergency surgery is recommended [1,4].

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Consent Statement: Written consent was obtained from the patient.

Declaration of interest: None

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