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(CASE REPORT)

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Acute coronary syndrome revealing aortic prosthetic valve dysfunction: A Case Report with Literature Review

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Abstract

Coronary embolism is an uncommon cause of ST-segment elevation myocardial infarction (STEMI), typically occurring in patients with an underlying embolic substrate. Mechanical prosthetic heart valves represent a major predisposing factor, particularly in the presence of prosthetic dysfunction. We report the case of a 61-year-old postmenopausal woman with a history of double mitral and aortic valve replacement using mechanical prostheses (ATS No. 29 in the mitral position and St. Jude Medical No. 17 in the aortic position), who presented with acute chest pain and apicolateral ST-segment elevation. Diagnostic investigations revealed coronary embolism secondary to prosthetic valve thrombosis with immobilization of one leaflet of the aortic prosthesis. This case highlights the pivotal role of multimodal cardiac imaging in establishing the etiological diagnosis and guiding optimal management.

Keywords: Coronary Embolism; Mechanical Heart Valve; Aortic Prosthetic Valve Dysfunction; STEMI; Cardiac Magnetic Resonance; Echocardiography

1. Introduction

Coronary embolism is a rare but potentially life-threatening cause of acute coronary syndrome, accounting for less than 3% of STEMI cases. It predominantly affects patients with embolic-prone conditions such as atrial fibrillation, infective endocarditis, or mechanical prosthetic heart valves. Prosthetic aortic valve dysfunction, particularly due to thrombosis, carries a substantial risk of systemic thromboembolic complications, including coronary embolization. Diagnosis relies on an integrated approach combining clinical presentation, coronary angiography, and advanced cardiac imaging modalities.

2. Case Report

We report the case of a 61-year-old woman whose only cardiovascular risk factor was menopause. Her medical history included double mitral and aortic valve replacement in 2009 using mechanical prostheses: an ATS No. 29 prosthesis in the mitral position and a St. Jude Medical (SJM) No. 17 prosthesis in the aortic position.

The patient was admitted for acute-onset constrictive chest pain. The electrocardiogram showed ST-segment elevation in the apicolateral territory, consistent with acute ST-segment elevation myocardial infarction.

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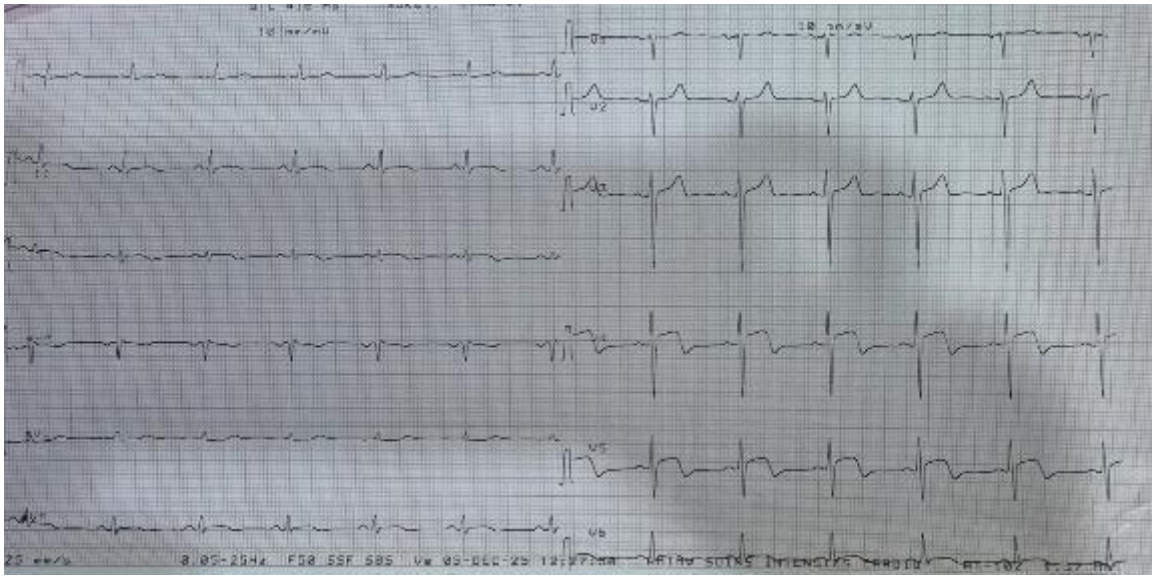

Figure 1 Electrocardiogram showing ST-segment elevation in the apicolateral leads**.**

On initial clinical examination, the patient was conscious and hemodynamically stable, with a blood pressure of 125/66 mmHg and a regular heart rate of 75 bpm. There were no signs of shock or acute heart failure. Cardiac auscultation revealed clearly audible mechanical prosthetic valve sounds, with a systolic murmur at the aortic area radiating to the neck vessels. Pulmonary examination was normal, with no crackles. There were no signs of systemic congestion or associated neurological deficits.

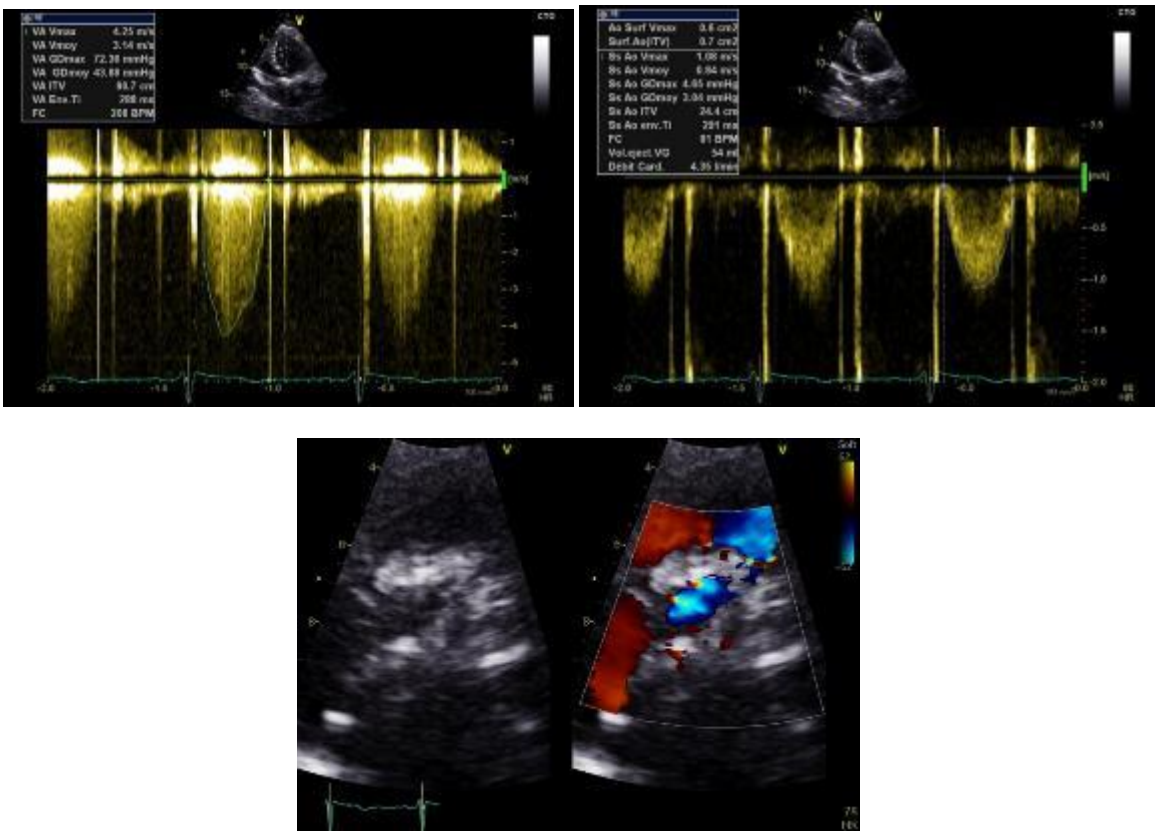

Figure 2 Transthoracic echocardiography showing a mechanical prosthesis with a blocked leaflet, a transprosthetic gradient of 43 mmHg, Vmax of 4.2 m/s, and an effective orifice area of 0.7 cm^2.

Initial laboratory tests showed a marked elevation of myocardial necrosis biomarkers, with a troponin level of 2073. The international normalized ratio (INR) at admission was 1.8.

Transthoracic echocardiography revealed apical akinesia with a left ventricular ejection fraction estimated at 50%. The aortic prosthesis appeared dysfunctional, with suspected leaflet immobilization, an effective orifice area of 0.7 cm^2, a peak velocity (Vmax) of 4.2 m/s, and a mean transaortic gradient of 43 mmHg.

Emergency coronary angiography revealed distal occlusion of the left anterior descending artery, consistent with coronary embolism, without significant atherosclerotic lesions in the remaining coronary tree.

Cardiac magnetic resonance imaging (MRI) demonstrated transmural late gadolinium enhancement of the apex, confirming the diagnosis of embolic myocardial infarction and allowing precise tissue characterization.

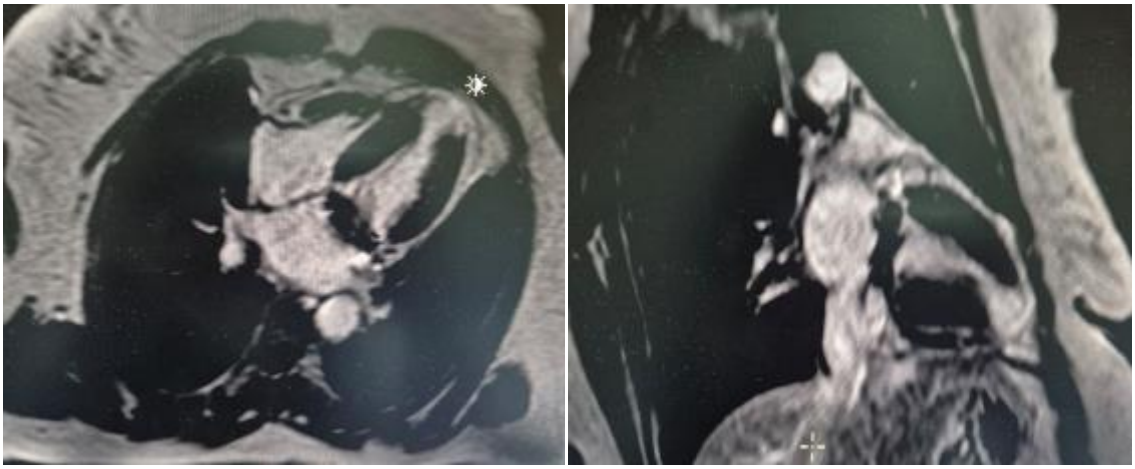

Figure 3 Late gadolinium enhancement sequences on cardiac MRI showing transmural enhancement at the apex of the left ventricle.

Transesophageal echocardiography (TEE), complemented by fluoroscopy, clearly demonstrated immobilization of one leaflet of the aortic prosthesis, consistent with prosthetic valve thrombosis, with no evidence of associated infective endocarditis.

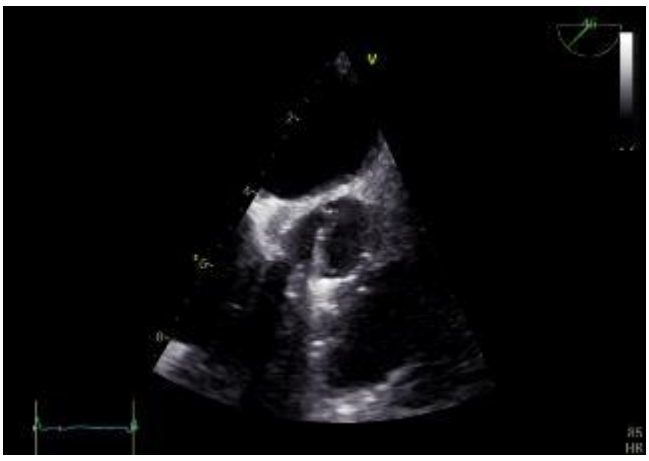

Figure 4 Transesophageal echocardiography showing a blocked leaflet of the aortic prosthetic valve.

Cardiac computed tomography (CT), performed as part of the etiological work-up, did not reveal significant coronary atherosclerotic plaques, further supporting a non-atherosclerotic mechanism. It also demonstrated abnormal motion of the mechanical aortic prosthesis, suggesting restricted opening of one leaflet, consistent with echocardiographic

findings, along with a hypodense mass attached to the ventricular side of the immobilized leaflet, compatible with thrombus.

The patient underwent surgical treatment with replacement of the aortic prosthesis, with an uncomplicated postoperative course.

3. Discussion

Coronary embolism is a rare but likely underdiagnosed cause of ST-segment elevation acute coronary syndrome. Angiographic series estimate its prevalence between 1% and 3% of myocardial infarctions, with a significant in-hospital mortality related to diagnostic delay and the severity of the underlying condition [1,2]. The main etiologies include atrial fibrillation, emboligenic heart diseases, infective endocarditis, and mechanical prosthetic valves [3].

In patients with mechanical valves, prosthetic valve thrombosis is a feared complication. It is favored by inadequate or unstable anticoagulation, prosthesis-related factors (small size, aortic position), and patient-related factors [4]. Leaflet immobilization, as observed in our patient, reflects obstructive or partially obstructive thrombosis and exposes patients to a high risk of systemic thromboembolic events, including coronary embolism [5].

The diagnosis of coronary embolism relies on a combination of clinical, angiographic, and contextual arguments. The criteria proposed by Shibata et al. include the absence of significant atherosclerotic lesions, distal coronary occlusion, the presence of an embolic-prone condition, and identification of a potential embolic source [1]. In our case, distal occlusion without associated atherosclerosis combined with documented aortic prosthetic dysfunction strongly supports an embolic mechanism.

Multimodal cardiac imaging plays a central role in the diagnostic approach. Transthoracic echocardiography is the first-line examination for evaluating ventricular function and transprosthetic gradients. However, transesophageal echocardiography remains the reference technique for morphological and functional assessment of mechanical prostheses, allowing detection of thrombus, pannus, or leaflet immobilization [6]. Fluoroscopy provides valuable complementary information by analyzing valvular kinetics.

Cardiac MRI provides major added value in this context. It allows precise myocardial tissue characterization, confirming the diagnosis of infarction and clarifying its mechanism. The transmural late enhancement of the apex observed in our patient is typical of abrupt coronary occlusion without prior collateral circulation, supporting an embolic rather than chronic coronary disease mechanism [7,8]. Furthermore, cardiac MRI offers prognostic assessment through evaluation of infarct size and residual left ventricular function.

Cardiac CT (multidetector computed tomography) has become an increasingly recognized tool for evaluating mechanical prosthetic valves, as a complement to echocardiography. Thanks to its high spatial resolution and ECG-gated multiphasic acquisition, it allows precise dynamic analysis of prosthetic leaflet motion and opening angles, facilitating the diagnosis of mechanical dysfunction [9].

Cardiac CT is particularly effective in differentiating prosthetic valve thrombosis from pannus formation, two entities with distinct therapeutic implications. Thrombosis typically appears as a hypodense, recently formed mass, whereas pannus corresponds to denser, fibrous tissue of chronic evolution [10]. This distinction can be challenging with echocardiography, especially in small prostheses or in patients with limited acoustic windows.

In our case, cardiac CT excluded significant coronary atherosclerotic disease, reinforcing the embolic hypothesis. It also demonstrated restricted opening of one leaflet of the aortic prosthesis, consistent with TEE and fluoroscopic findings, consolidating the diagnosis of prosthetic valve dysfunction. Thus, cardiac CT emerges as a key complementary examination in the diagnostic algorithm of mechanical aortic prosthetic valve dysfunction, particularly when echocardiographic findings are discordant or incomplete.

Therapeutic management of coronary embolism relies on urgent myocardial reperfusion, most often by percutaneous coronary intervention with or without thrombus aspiration, combined with rigorous optimization of anticoagulation [2,3]. In cases of mechanical prosthetic valve thrombosis, the therapeutic strategy (intensified anticoagulation, fibrinolysis, or surgery) depends on the obstructive nature, prosthetic location, and the patient's clinical status, in accordance with European guidelines [4,6].

Finally, this case highlights the importance of an integrated and multidisciplinary approach in patients with mechanical prosthetic valves presenting with acute coronary syndrome. Early recognition of coronary embolism not only allows adaptation of the reperfusion strategy but, more importantly, enables treatment of the underlying cause to prevent recurrent thromboembolic events.

4. Conclusion

Coronary embolism should be systematically considered in cases of STEMI occurring in patients with mechanical prosthetic heart valves. This case emphasizes the essential role of multimodal imaging—including echocardiography, fluoroscopy, and cardiac MRI—in identifying underlying aortic prosthetic valve dysfunction. Early diagnosis is crucial for optimal management and prevention of recurrent thromboembolic events.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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