American Journal of Medical Case Reports, 2025, Vol. 13, No. 6, 31-33
Available online at http://pubs.sciepub.com/ajmcr/13/6/1
Published by Science and Education Publishing
DOI:10.12691/ajmcr-13-6-1

Coronary Embolism in a Patient with Mechanical Mitral Valve: The High Cost of Anticoagulation Discontinuation

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Received May 20, 2025; Revised June 22, 2025; Accepted June 30, 2025

Abstract: **Coronary embolism** is a non-atherosclerotic cause of acute coronary syndromes. It is frequently observed in patients with atrial fibrillation or prosthetic heart valves. We report the case of a 40-year-old woman with no cardiovascular risk factors but a history of mechanical mitral valve replacement, who had discontinued her vitamin K antagonist (VKA) therapy without medical supervision. She presented with sudden-onset, typical anginal chest pain at rest, described as retrosternal pressure radiating to the left arm and associated with dyspnea. Transthoracic echocardiography revealed an abnormality on the mitral prosthesis, and transesophageal echocardiography confirmed the presence of a thrombus. Coronary angiography identified a subocclusive thrombotic lesion in the mid-left anterior descending (LAD) artery, consistent with coronary embolism. The patient underwent successful manual thrombectomy without stent implantation.

Keywords: *Coronary embolism, Prosthetic heart valve, Thrombectomy, Valve thrombosis*

Cite This Article: Laoufi Z, Lokman H, Benyass A, Mouine N, and Lakhal Z, "Coronary Embolism in a Patient with Mechanical Mitral Valve: The High Cost of Anticoagulation Discontinuation." *American Journal of Medical Case Reports*, vol. 13, no. 6 (2025): 31-33. doi: 10.12691/ajmcr-13-6-1.

1. Introduction

Acute coronary syndrome (ACS) is most commonly caused by atherosclerotic plaque rupture, which can lead to myocardial ischemia. However, non-atherosclerotic causes such as coronary embolism, though rare, should not be overlooked — particularly in patients with low cardiovascular risk profiles. Coronary embolism is an underrecognized cause of ACS, accounting for approximately 2.9% to 4.0% of cases in angiographic and autopsy series [1]. It can present with the same clinical and electrocardiographic features as classic atherothrombotic myocardial infarction, making diagnosis particularly challenging [2,3].

Atrial fibrillation, endocarditis, intracardiac thrombi, and the presence of mechanical prosthetic valves are conditions that predispose to coronary embolism. In patients with prosthetic heart valves, continuous anticoagulation is essential to prevent thromboembolic events. Poor adherence to anticoagulation therapy significantly increases the risk of coronary embolism.

Although rare, coronary embolism is associated with significant morbidity and a potentially severe prognosis, particularly in the absence of prompt diagnosis and management. Delayed recognition may result in larger infarcts, mechanical complications, or sudden cardiac death.

We report the case of a 40-year-old woman with a history of mechanical mitral valve replacement who had discontinued anticoagulation therapy for over three years. She presented with sudden-onset typical anginal chest pain at rest and was diagnosed with an acute coronary syndrome. Coronary angiography revealed a thrombotic occlusion of a coronary artery, that was successfully treated with manual thrombectomy. A diagnosis of coronary embolism was established.

2. Case Presentation

A 40-year-old woman with a history of mechanical mitral valve replacement for rheumatic mitral stenosis and concomitant tricuspid valve plasty performed nine years earlier was admitted for the management of acute chest pain at rest. The pain had started suddenly five hours before admission. It was described as severe, constrictive retrosternal discomfort radiating to the left arm, and was associated with dyspnea and sweating. The pain was continuous and not relieved by rest. Notably, the patient had discontinued her vitamin K antagonist (VKA) therapy three years earlier due to heavy menstrual bleeding. No medical consultation had been sought at the time, and she was not taking any other medications.

On examination, she was hemodynamically stable. Cardiac auscultation revealed an irregular rhythm and an audible mechanical valve click. The electrocardiogram (ECG) showed atrial fibrillation at 95 beats per minute, with negative T waves in the septal, lateral, and inferior leads. According to the patient, her atrial fibrillation had

preceded the surgery, likely as a complication of rheumatic mitral stenosis and left atrial enlargement.

Troponin levels were elevated to 400 times the normal value, with the rest of the laboratory results being normal (Table 1).

Table 1. Initial Laboratory Findings at Admission

Parameters	Results	Reference Range
Complete Blood Count (CBC)		
Hemoglobin	13.2 g/dl	12-16 g/dl
Hematocrit	39.5 %	36-45 %
Red Blood Cells (RBC)	4.5 T/L	4.2-5.4 T/L
White Blood Cells (WBC)	6.8 G/L	4-10 G/L
Neutrophils	4.2 G /L	1.5-7.5 G/L
Lymphocytes	1.9 G/L	1-4 G/L
Platelets	300 x 10^3 /L	150-400 X10^3 /L
Electrolyte Panel		
Sodium (Na+)	139 mmol/L	135-145 mmol/L
Potassium (K+)	4.1 mmol/L	3.5-5 mmol/L
Chloride (Cl-)	102 mmol /L	78-107 mmol/L
Bicarbonate (HCO3-)	24 mmol/L	22-28 mmol/L
Renal Function Panel		
Urea	5 mmol/L	2.5-7.1 mmol/L
Creatinine	70 µmol/L	45-90 µmol/L
Cardiac Marker		
Troponin I (High sensitivity)	6156 ng/L	<14 ng/L
Coagulation Profile		
Prothrombin Time (PT)	12.5 s	11-13.5 s
INR (International Normalised Ratio)	1.1	0.2-1.2

Given the clinical presentation suggestive of acute coronary syndrome (ACS), the patient was managed according to standard protocols. She received a loading dose of aspirin and clopidogrel. Serial troponin measurements were performed throughout her hospitalization. (Figure 1).

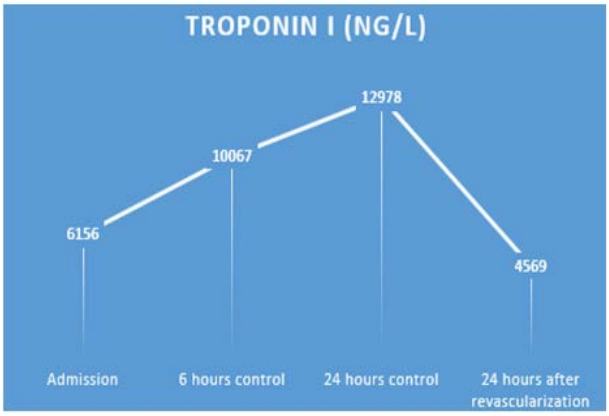

Figure 1. Graph showing the kinetics of the patient's troponin levels during hospitalization.

Transthoracic echocardiography demonstrated left ventricular dilatation with apical akinesia and a moderately reduced ejection fraction estimated at 39%. Dysfunction of the mechanical mitral valve was suspected. Both atria were dilated, whereas the right ventricle exhibited normal dimensions and preserved systolic function. Transesophageal echocardiography identified a mobile echogenic mass attached to the ventricular side of the mitral prosthesis, highly suggestive of a thrombus (Figure 2).

Coronary angiography revealed a subocclusive thrombotic lesion in the mid-left anterior descending (LAD) artery, with preserved distal flow, suggestive of coronary embolism (Figure 3).

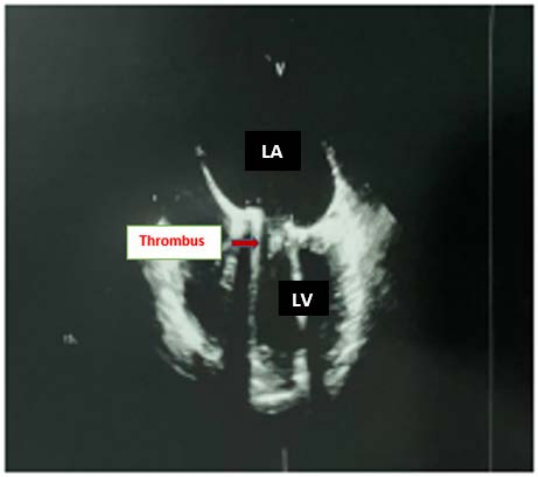

Figure 2. Transesophageal echocardiographic images showing a mobile thrombus attached to the ventricular side of the mechanical mitral valve. LA: Left Atria, LV:Left ventricle

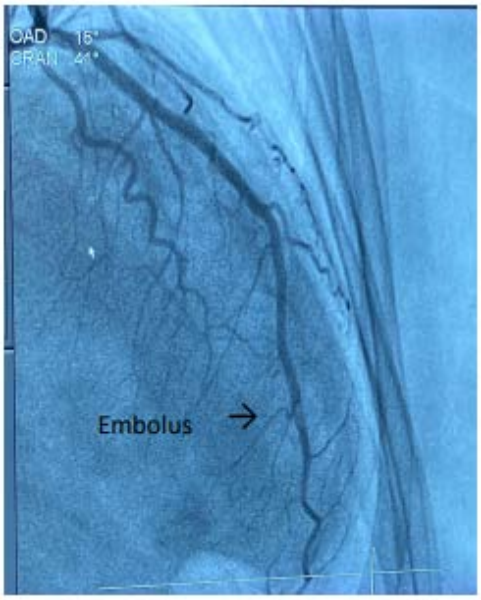

Figure 3. Coronary angiography showing a subocclusive thrombotic stenosis of the mid-left anterior descending (LAD) artery

The remainder of the coronary tree appeared angiographically normal. A manual thrombectomy was performed in the LAD, resulting in successful thrombus extraction and restoration of normal coronary flow, along with the administration of a GPIIb/IIIa receptor inhibitor. No stent was implanted, as no underlying atherosclerotic lesion was identified. Anticoagulation therapy with a vitamin K antagonist (VKA) was reinstated, accompanied by thorough patient education and regular INR monitoring.

3. Discussion

Embolic phenomena in the coronary circulation are rare under normal physiological conditions due to the protective anatomy of the coronary arteries and the shielding effect of the aortic valve cusps during systole. However, the risk of coronary embolism increases significantly in patients with atrial fibrillation, prosthetic heart valves, infective endocarditis [4], or hypercoagulable

states. In such cases, ineffective anticoagulation with International Normalized Ratio (INR) levels outside the therapeutic range or discontinuation of vitamin K antagonists (VKAs) may predispose to thrombus formation. Our patient presented a high thrombotic risk due to the presence of a mechanical mitral valve, atrial fibrillation, and a history of anticoagulation discontinuation. Clinically, coronary embolism typically presents as an acute coronary syndrome in patients with low cardiovascular risk but known thrombotic risk factors. Biologically, a marked elevation in cardiac biomarkers is often observed. However, electrocardiography (ECG) and transthoracic echocardiography (TTE) generally do not allow differentiation between coronary embolism and atherosclerotic acute coronary syndrome.

Transesophageal echocardiography (TEE) remains the key diagnostic tool, allowing detailed visualization of the heart valves and detection of intracardiac thrombi. Cardiac catheterization may reveal atypical findings, such as occlusion of one or more coronary vessels in the absence of underlying atherosclerosis — specifically, no evidence of unstable plaques, dissections, or mural thrombi.

To date, no established treatment guidelines exist [5]. In our patient's case, thrombectomy was carefully performed. Coronary thrombectomies are infrequently performed due to the technical expertise required and the significant risk of complications associated with the procedure. Common complications include coronary dissection, failure in cases of adherent thrombus with a no-reflow phenomenon, systemic embolism, and thrombus migration [6]. The addition of a glycoprotein IIb/IIIa receptor inhibitor is often indicated [7] to enhance thrombectomy effectiveness.

Another key consideration in managing these patients is whether or not to place a stent [8]. In cases of failure or partial success, balloon angioplasty combined with stent placement, alongside anticoagulant and antiplatelet therapy, can be beneficial [9].

In our patient's case, no stent was implanted, as the coronary vessels appeared smooth and showed no signs of atherosclerosis. Furthermore, distal flow beyond the lesion was preserved (TIMI III). Stent implantation would have necessitated lifelong anticoagulation, which posed a significant risk given the patient's history of treatment interruption.

Maintaining effective anticoagulation therapy is essential to prevent embolic events, especially in patients with atrial fibrillation [10]. In our patient's case, direct oral anticoagulant (DOAC) therapy was proposed due to its proven efficacy and ease of management compared to vitamin K antagonists (VKAs) [11], but the treatment was declined by the patient because of financial constraints. Recent studies and meta-analyses [12] have demonstrated that DOACs are as effective as VKAs in preventing thromboembolic events in patients with atrial fibrillation secondary to valvular heart disease, while offering a comparable or improved safety profile—particularly with a lower risk of intracranial hemorrhage—and greater ease of use.

4. Conclusion

Coronary embolism should be suspected in any case of acute coronary syndrome occurring in a patient with low cardiovascular risk but a high thrombotic risk . Accurate diagnosis relies on integrating the clinical context with advanced imaging modalities, such as transesophageal echocardiography, which can identify intracardiac sources of emboli, and coronary angiography, which remains the gold standard for definitive confirmation. The management of coronary embolism must be carefully individualized, taking into account the patient's coronary anatomy and the angiographic features of the culprit lesion.

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