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Case Report **Cardiology**

Unveiling the Nexus: Myocardial Infarction Secondary to Coronary Embolism in a Patient with Mitral Stenosis and Atrial Fibrillation: Case Report

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Abstract

Myocardial infarction resulting from coronary embolism is a rare type of acute coronary syndrome, often going unnoticed. Distinguishing it from acute coronary syndromes arising from atherosclerosis is important, as it significantly influences the treatment approach. It is frequently associated with conditions that increase the risk of thromboembolism, such as infective endocarditis, atrial fibrillation, mitral valve disease, neoplasia and cardiac surgeries. We would like to present a case of coronary embolism involving a 42-year-old male with a history of rheumatic valve disease and atrial fibrillation who was admitted for acute chest pain. The diagnosis was confirmed through coronary angiography.

Keywords: Coronary embolism, myocardial infarction, acute coronary syndrome, Atrial fibrillation.

INTRODUCTION

Coronary embolism (CE) is a rare cause of acute myocardial infarction (MI), warranting consideration in patients with embolism-linked conditions due to its high rate of mortality [1]. CE has been linked to various clinical conditions such as aortic and mitral prosthetic heart valves, atrial fibrillation (AF), dilated cardiomyopathy, neoplasia, infective endocarditis, atrial septal defect, or intracardiac tumor [3]. In many instances, an underlying hypercoagulable state may be identified as a potential predisposing factor for thrombosis [2].

Diagnosing acute coronary syndrome (ACS) in relation to CE poses a considerable challenge for cardiologists. This is primarily due to its infrequent occurrence and the lack of agreement on both diagnosis and treatment protocols [4]. We showcase a case involving myocardial infarction resulting from a coronary embolism.

CASE PRESENTATION

A 42-year-old male was admitted at Mohammed V Military Hospital's emergency department for sudden and prolonged chest pain that commenced an hour prior to his arrival. The pain was accompanied by excessive sweating. His medical history included rheumatic mitral valve stenosis and atrial fibrillation for the past 5years for which he was on acenocoumarol. He had no personal risk factors that could be modified, and there was no family history of premature coronary artery disease. The patient admitted to poor adherence to his prescribed treatment and hardly showed up for review.

Upon physical examination, the patient was alert and oriented, with stable vital signs, including a blood pressure of 125/78 mmHg and a heart rate of 109 bpm. Lung auscultation revealed clear sounds with no crackles or wheezing. An irregular heart rhythm was noted, along with a diastolic murmur at the mitral region and an early diastolic rumble at the aortic region.

The electrocardiogram (ECG) showed atrial fibrillation with a ventricular rate of 102 bpm, a right bundle branch block and ST-segment elevation in leads V2, V3, V4 (Fig 1).

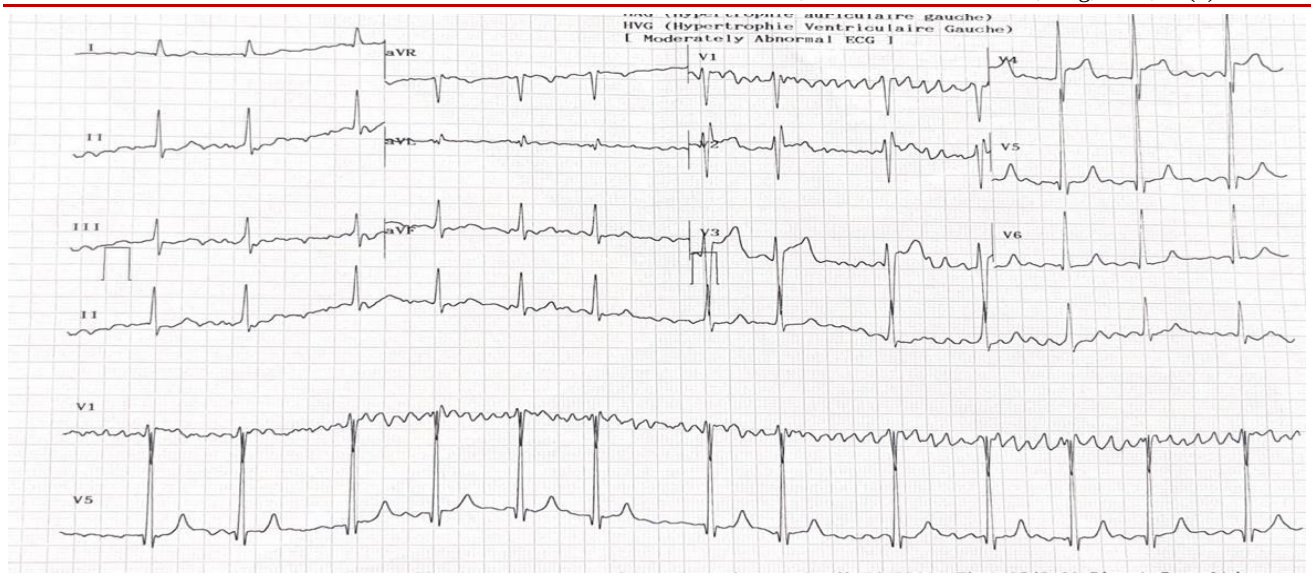

Fig 1 ECG - ST-elevation in V2, V3 and V4 leads

Blood testing revealed normal hemoglobin level 14,3 (normal values, Nv. 11-17g/dl), C reactive protein 3.1mg/l (nv. <5), platelets 132000(nv 150000-350000), INR 1.15(nv 2.0-3.0)

A transthoracic echocardiography (TTE) revealed significant mitral stenosis, with an estimated mean gradient of 10 mmHg across the mitral valve and a mitral valve area of 1.1 cm^2. Severe aortic valve regurgitation was also observed. Both atria were dilated, and sludge was present in the left atrial appendage. The right ventricle was dilated, and the pulmonary artery systolic pressure (PASP) measured 37 mmHg. Additionally, anterior and anterolateral wall hypokinesia and moderate left ventricular dysfunction were noted (LVEF; 38%).

A transesophageal echocardiography (TEE) confirmed the mitral stenosis and the presence of sludge in the left atrial appendage. There were no vegetations or images suggestive of neoplasia.

Given the clinical symptoms and ECG findings, an acute coronary syndrome was suspected, and the patient was given a loading dose of aspirin (300mg) and clopidogrel (300mg) as well as heparin infusion. He then underwent cardiac catheterization at the 5th hour from the onset of the pain, which revealed occlusion of the left anterior descending artery (LAD) with a significant thrombus burden. An aspiration thrombectomy was done but unsuccessful necessitating a balloon angioplasty. At the end of the procedure, a TIMI grade 1 flow persisted in the LAD, along with thrombus and distal embolization of the LAD. There was no evidence of dissection (Fig 2).

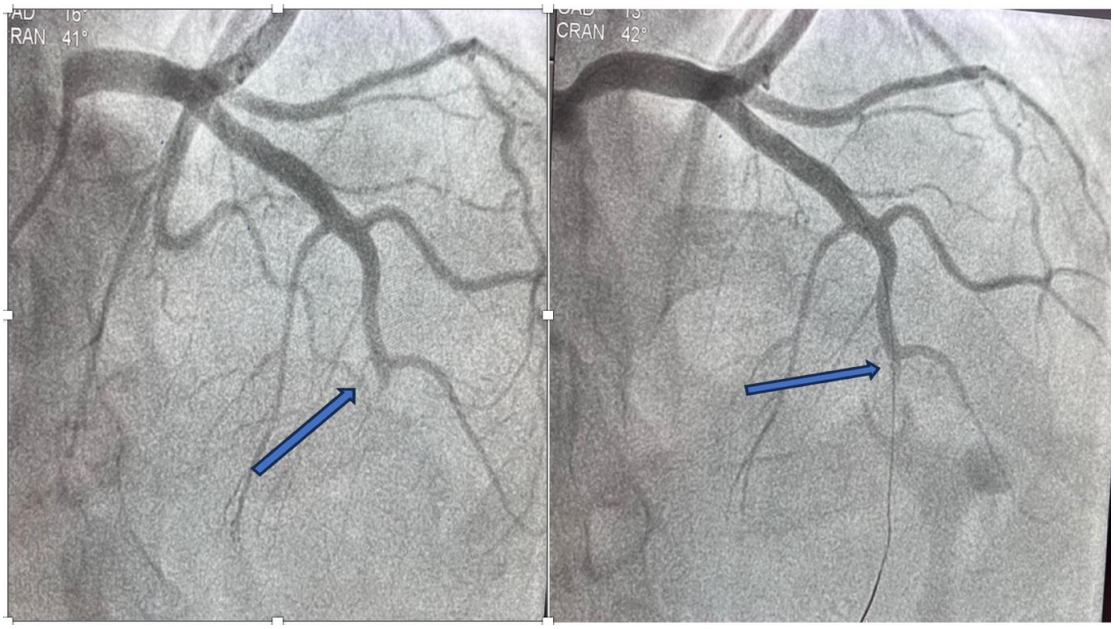

Fig 2 Coronary angiogram findings: Left coronary angiogram showing mid LAD thrombus without atherosclerotic plaques

The patient was transferred to the intensive care unit for further care, with continued unfractionated heparin (UFH) infusion and tirofiban for 48 hours which was later replaced with low molecular weight heparin.

A repeat coronary angiography demonstrated reduced thrombus burden in the LAD and the presence of a small thrombus in the distal LAD, with restoration of a TIMI grade 2 flow (Fig 3). Subsequent ECGs showed the resolution of ST-segment elevation in anterior leads and deep inverted T waves in leads V4 and V5 (Fig 4). The patient was prescribed clopidogrel and acenocoumarol.

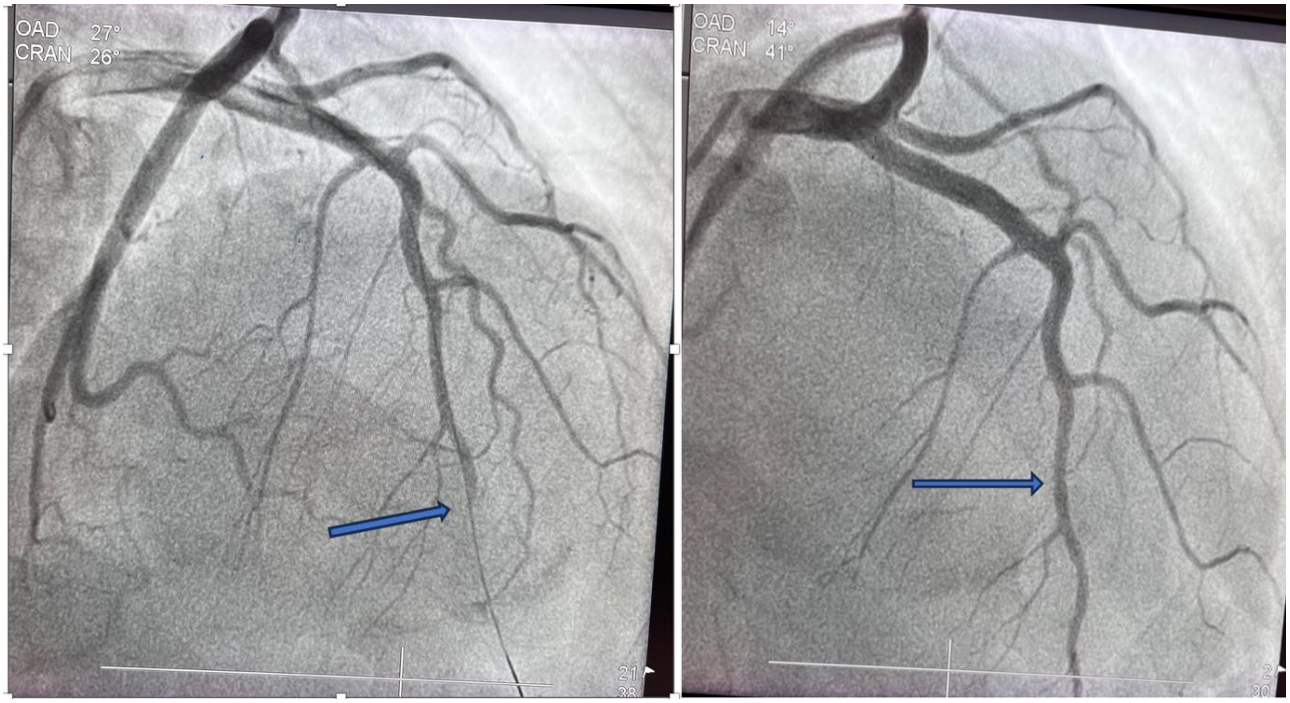

Fig 3. Control coronary angiography after balloon angioplasty and anti thrombotic treatment showing a TIMI 2 flow.

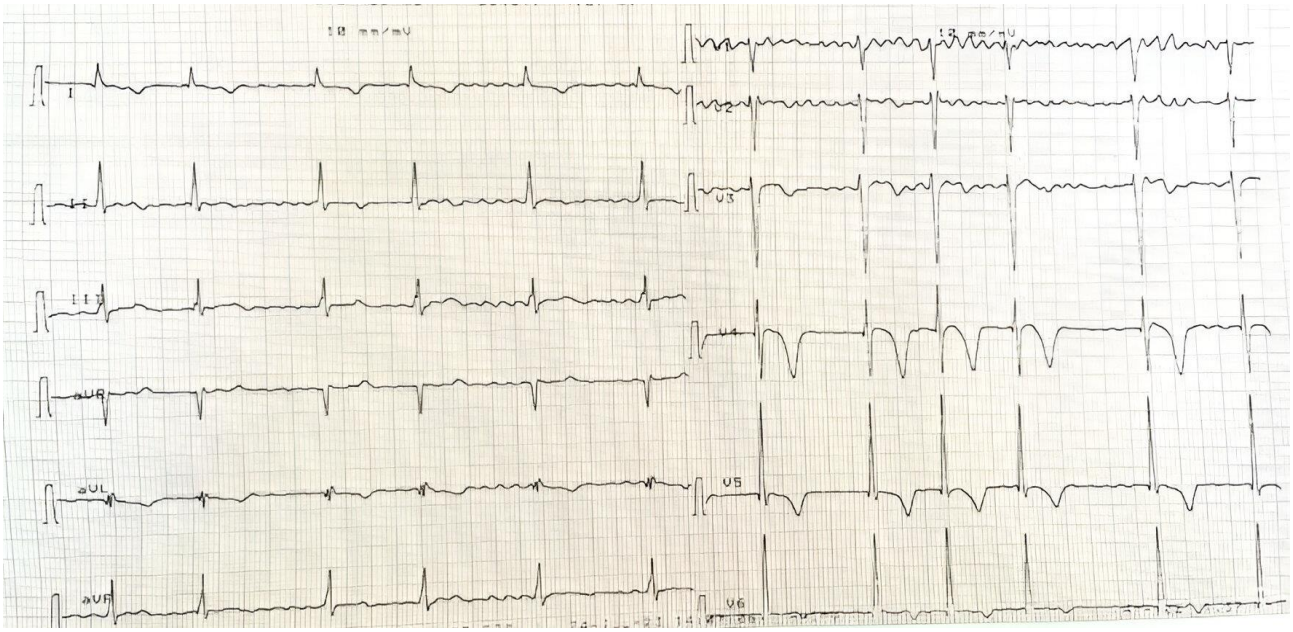

Fig 4 ECG - Resolution of ST-segment elevation and the apparition of deep inverted T waves after intervention

The patient had a favorable clinical outcome with no reported adverse effects. Subsequently, he received counseling regarding the significance of anticoagulation and preventive measures for infective endocarditis. It was advised that he undergo mitral and aortic valve repairs. He was then transferred to the cardio thoracic surgical ward where he had an aortic and mitral valve replacements. He was discharged after 14 days with a good outcome.

DISCUSSION

Coronary embolism is a rare cause of acute myocardial infarction, and its suspicion is warranted when myocardial infarction occurs in individuals with conditions linked to embolism [3]. Myocardial infarction resulting from coronary embolism accounts for approximately 3% of cases and is often not readily diagnosed [5]. Coronary embolism can be categorized into three types: direct, paradoxical, and iatrogenic, with some overlap between these classifications [6]. Direct coronary emboli typically originate from the left atrial appendage, left ventricle, aortic or mitral valves, or the proximal coronary artery. The embolic material may consist of thrombus, valvular debris, or even neoplastic tissue.

Paradoxical emboli traverse through an open foramen ovale, atrial septal defect, or pulmonary arteriovenous malformations, moving from the venous circulation into the systemic circulation. These emboli are most frequently derived from deep veins, often associated with deep venous thrombosis.

Iatrogenic emboli can arise following medical procedures, with valve replacements and coronary interventions being the usual culprits [6]. Popovic *et al*., [2] and Shibata *et al*., [7] in their respective studies, concluded that atrial fibrillation (AF) is the leading cause of coronary embolism, with prevalence rates of 46% and 73%, respectively [7]. The most recent study by Popovic *et al*., also concluded that AF is the leading cause of myocardial infarction secondary to CE with a prevalence of 20% [8].

The primary diagnostic tool for coronary embolism is coronary angiography, capable of revealing abrupt occlusions in distal, small-caliber, epicardial coronary arteries, even in the absence of underlying atherosclerotic disease [5]. Some patients may present with "bystander atherosclerosis," complicating the diagnosis [1]. Shibata and colleagues introduced the clinical diagnostic criteria for coronary embolism at the National Cerebral and Cardiovascular Center (NCVC). These criteria encompass three primary and three secondary indicators to determine the likelihood of a CE diagnosis as either probable or definite [7]. In our case, we assembled a primary criterion and two secondary criteria, enabling us to confirm a diagnosis of CE.

Table 1: Proposed criteria for the clinical diagnosis of coronary artery embolism (CE) according to Shibata, *et al*., [7]

Major criteria • Angiographic evidence of coronary artery embolism and thrombosis without atherosclerotic components • Concomitant coronary artery embolization at multiple sites* • Concomitant systemic embolization without left ventricular thrombus attributable to acute myocardial infarction
Minor criteria • <25% stenosis on coronary angiography, except for the culprit lesion • Evidence of an embolic source based on transthoracic echocardiography, transesophageal echocardiography, computed tomography, or MRI • Presence of embolic risk factors: atrial fibrillation, cardiomyopathy, rheumatic valve disease, prosthetic heart valve, patent foramen ovale, atrial septal defect, history of cardiac surgery, infective endocarditis, or hypercoagulable state
Definite CE: Two or more major criteria, or one major criterion plus ≥2 minor criteria, or three minor criteria **Probable CE**: One major criterion plus 1 minor criterion, or two minor criteria CE: Coronary artery Embolism, MRI: Magnetic Resonance Imaging. *Indicates multiple vessels within 1 coronary artery territory or multiple vessels in the coronary tree.

The left anterior descending artery (LAD) is the most frequently affected vessel (75%), as seen in our case, followed by the right coronary artery, as described in the case by Faraj *et al*., [4].

Additional investigations are necessary to pinpoint the source of the embolism. Initial etiological assessment typically involves transthoracic echocardiography, which helps rule out the presence of cardiac chamber thrombi, valvular heart diseases, infective and non-infective endocarditis as well as other cardiac tumors [5]. TTE is crucial for identifying the etiologies and the differentials. A simultaneous agitated saline study can exclude interatrial shunting. Transesophageal echocardiography is useful for eliminating the possibility of left atrial appendage thrombi. Cardiac computed tomography and cardiac MRI play essential roles in identifying intracardiac thrombi [5]. In our case, both transthoracic and transesophageal echocardiograms were conducted as further investigations, revealing severe mitral stenosis and the presence of sludge in the left atrial appendage.

To date, there are no treatment guidelines, leaving management at the discretion of the treating physician [9]. Treatment of the underlying pathology, particularly valvular diseases, atrial fibrillation, endocarditis and other associated hypercoagulable state as seen in cancer patients represent the cornerstones of its therapeutic strategy.

The initial management aligns with that of any suspected ACS case, involving dual antiplatelet therapy (DAPT), pain relief, and expedited or emergency coronary angiography [6]

While aspiration thrombectomy is a matter of debate, it is recommended in cases with a substantial thrombotic burden. When possible, thrombus analysis after manual thrombectomy has been suggested to improve diagnosis [5].

In the Popovic cohort, 40% of the 53 CE cases underwent manual thrombectomy; histopathological analysis was performed in 5 cases, identifying septic embolism (n=2), neoplasia tissue (n=1) and thrombus (n=2) [2]. Aspiration provides a more comprehensive evaluation of the underlying coronary artery. If aspiration results in a complete flow restoration with a normal angiographic appearance, intravascular ultrasound or optical computerized tomography (OCT) may be used to further assess the vessel, determining whether it is a result of isolated plaque erosion or an embolic event. In instances where the artery appears angiographically normal, balloon angioplasty and stent placement may not be necessary [6].

Anticoagulation forms the foundation of medical therapy for coronary embolism. In the absence of established guidelines, the decision regarding the duration of anticoagulant treatment is at the discretion of the treating clinician, who carefully balances the risk of bleeding against the risk of subsequent thromboembolic events. Specific considerations come into play for individuals who have experienced recent trauma, undergone hospitalization, or used certain medications like oral contraceptives, as they may exhibit a reversible procoagulant condition. Patients with predisposition to thrombosis, especially those with active cancer or antiphospholipid antibody syndrome, warrant recognition. In the absence of risk factors, short-term anticoagulation for three months should be explored. If the patient has documented risk factors, lifelong anticoagulation should be contemplated [7].

CONCLUSION

Coronary embolism is a rare type 2 myocardial infarction (MI), demanding a comprehensive evaluation that includes a detailed medical history, physical examination, and specialized diagnostic tests to uncover its root cause. In patients with STEMI and AF, CE accounts for a significant portion of the pathogenesis, marked by a distinct clinical presentation and therapeutic approach, as well as a worsened prognosis. This often-overlooked clinical entity warrants increased attention in the future to potentially improve outcomes.

Authors Contribution

Frederick Nana Yeboah: Study concept, Writing the paper.
Faid Soumia: Study concept, Writing the paper.
Lahmouz Youssef: Study concept, Writing the paper.
Djamba Lutundula Marc: Study concept, Writing the paper.
Ely Sidi Sidi M'hamed; Study concept, Writing the paper
Aatif Benyass: Supervision and data validation

Additional Information

Disclosures

Consent: written consent for submission and publication of this case report including images has been obtained from the patient.

Conflict of interest: None

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