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Case Report

Silent Acute Myocardial Infarction and Anterolateral Papillary Muscle Rupture: A Rare Complication in Elderly Patients

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Abstract

Mitral regurgitation (MR) following acute myocardial infarction (AMI) is a common occurrence, often resolving without significant complications. However, severe MR leading potentially to acute heart failure or cardiogenic shock is rare. It can result from papillary muscle rupture (PMR) which is an extremely uncommon and life-threatening mechanical complication of AMI. We present a case of a 77-year-old male with a late-presenting high-risk acute coronary syndrome (ACS) complicated by acute MR due to anterolateral PMR. The patient exhibited atypical symptoms, including dyspnea and cough, and was diagnosed with a STEMI equivalent. Echocardiography revealed severe MR secondary to papillary muscle rupture. The patient underwent successful coronary artery bypass graft (CABG) and mitral valve replacement surgery. We discuss the risk factors, management and complications of papillary muscle rupture, emphasizing the importance of early recognition and intervention. This case underscores the potential for atypical ACS presentations in the elderly and highlights the significance of specific ECG patterns, such as ST segment elevation in leads aVR and/or V1. Despite advancements in therapy, the mortality associated with papillary muscle rupture remains high, underscoring the critical need for preventive measures and heightened clinical awareness.

Introduction:

Mitral regurgitation (MR) is a common finding in patients after acute myocardial infarction (AMI), with an incidence of 17% on cardiac auscultation and 40-70% on echocardiography [1]. It is usually asymptomatic and resolves within a few hours. However, in rarer cases, significant MR can occur, leading to acute heart failure and potentially cardiogenic shock. Two distinct mechanisms can contribute to this. Ischemic dysfunction of a papillary muscle may cause a dynamic form of MR, which is mild at rest but becomes severe with increased ventricular loading. Less frequently, partial or complete rupture of a papillary muscle can result in hemodynamic instability and carries a high mortality rate.

In this paper, we present a case of late-presenting high-risk acute coronary syndrome (ACS) complicated by acute MR due to anterolateral papillary muscle rupture. Our manuscript was written in accordance with the CARE guidelines [2].

Case presentation:

A 77-year-old male was admitted to the cardiology department due to worsening angina persisting for six days. He had no known cardiovascular risk factors and no significant medical history. The patient had been experiencing angina consistent with Canadian Cardiovascular Society (CCS) Class II severity since the previous year. It had worsened over the

past week, occurring with minimal exertion and accompanied by intensified symptoms such as sweating and vertigo. However, he did not report prolonged chest pain at rest. Additionally, he mentioned the onset of mild dyspnea and coughing with frothy sputum two days before admission.

Upon admission, the patient was fully alert, afebrile, and had a normal body mass index (BMI) of 20 kg/m^2. He was hemodynamically stable with a blood pressure of 99/65 mmHg and a heart rate of 92 bpm. The patient exhibited mild orthopnea, was polypneic with a respiratory rate of 20 cpm, and had a saturation of 88% on room air. Cardiac auscultation revealed a harsh holosystolic murmur, best heard at the apex, radiating to the left sternal border. Pulmonary auscultation detected bilateral crackles at the bases, predominantly on the right side.

The electrocardiogram (ECG) displayed a regular sinus rhythm with diffuse ST segment depression and ST segment elevation in leads aVR and V1 (Figure 1). Additional right and basal leads showed negative T waves in the basal territory (Figure 2).

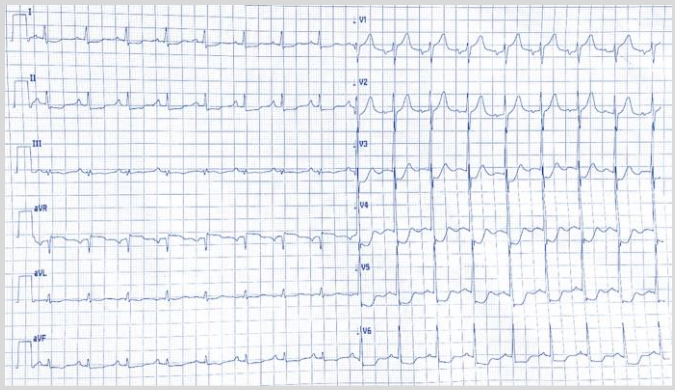

Figure 1: Twelve-lead ECG performed upon admission showing ST segment elevation of 1.5 mm and 1 mm respectively in leads aVR and V1, and ST segment depression of more than 1 mm in seven leads (leads V3 to V6, I, II, and aVF).

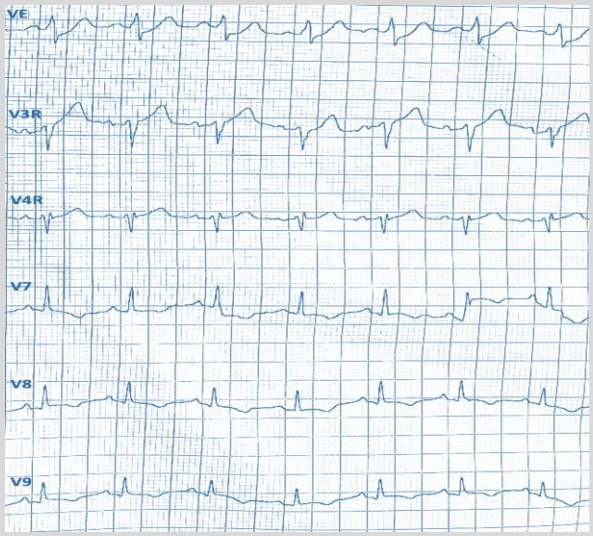

Figure 2: Right and basal leads showing negative T waves in the basal territory.

Initial laboratory findings showed elevated troponin I levels at 84 times the upper limit, slightly altered renal function with urea at 0.87 g/l (Normal range, NR: 0.15-0.55) and creatinine at 14.9 mg/l (NR: 7.2-12.5), and a normal Cell Blood Count (CBC) with hemoglobin and platelet levels respectively at 14.8 g/dl (NR: 13-16.5) and 248,000 elements/mm^3 (NR: 150,000-400,000). The metabolic workup revealed pre-diabetes with glycated hemoglobin (HbA1c) at 6.2%.

Chest X-ray showed a normal cardiac silhouette with mild bilateral pulmonary edema, predominantly on the right side (Figure 3). Transthoracic echocardiography (TTE) showed a hyperdynamic non-dilated left ventricle with regional wall motion abnormalities (akinesia of the basal and medial segments of the lateral wall) and a 70% ejection fraction. Severe mitral regurgitation due to flail of the anterolateral leaflet was observed, with a mobile element identified as a portion of the papillary head (Figures 4 and 5). The mitral regurgitation was eccentric, mainly directed towards the right pulmonary veins (Figure 6). Additionally, the patient exhibited high pulmonary pressure, elevated filling pressure, and B-lines on lung ultrasound, predominantly on the right side, indicative of pulmonary edema.

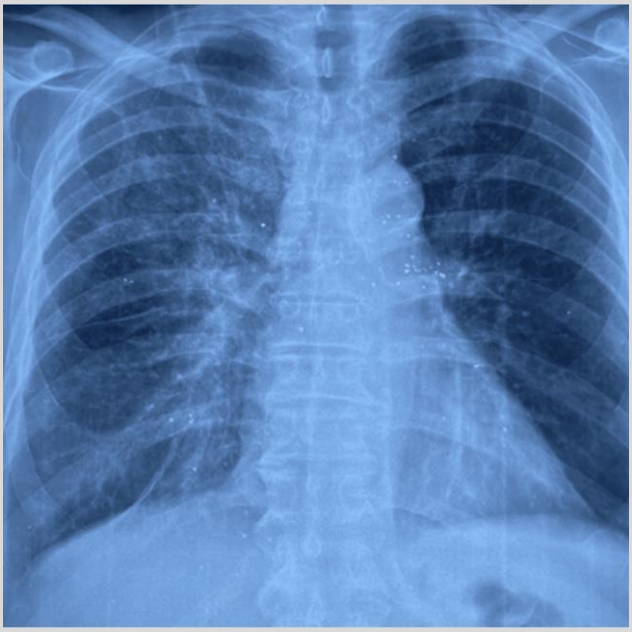

Figure 3: Chest X-ray showing pulmonary edema mainly on the right side.

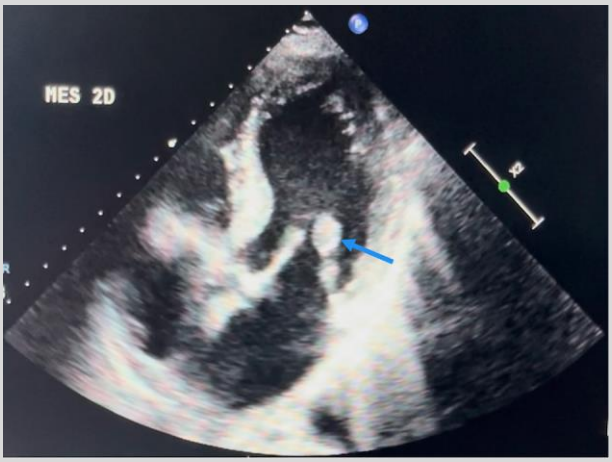

Figure 4: Apical five-chamber view during diastole showing an element attached to the anterolateral mitral leaflet identified as a portion of the papillary head (blue arrow).

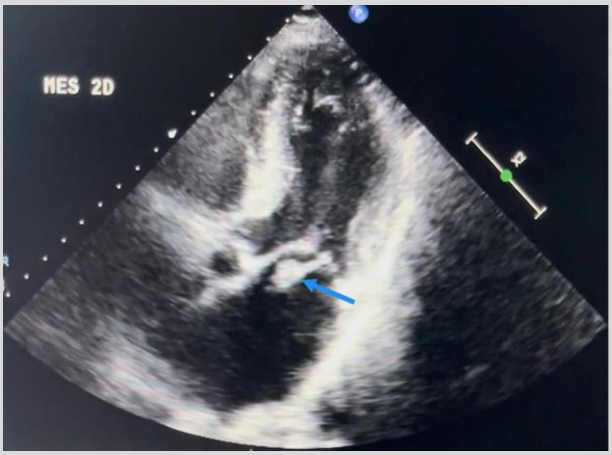

Figure 5: Apical five-chamber view during systole showing mitral valve prolapse with a portion of the ruptured papillary head attached to the anterolateral mitral leaflet (blue arrow).

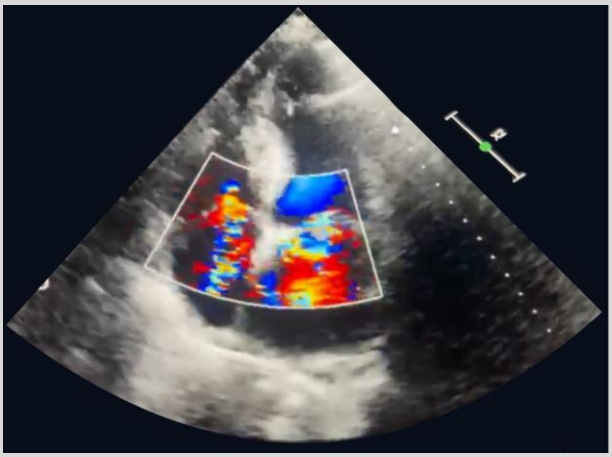

Figure 6: Color-Doppler showing severe eccentric mitral regurgitation.

The diagnosis was a STEMI equivalent complicated with mild left-sided heart failure secondary to acute mitral regurgitation due to partial papillary muscle rupture. The patient underwent diagnostic angiography, revealing a critical heterogeneous lesion of the proximal left main (LM) artery and an occlusion of the second obtuse marginal (Figure 7). The patient was urgently transferred to the surgical department for coronary artery bypass graft (CABG) and mitral valve replacement surgery. Meanwhile, he received aspirin, enoxaparin, diuretics, and proton pump inhibitors. Beta blockers and Angiotensin-converting enzyme (ACE) inhibitors were not initiated due to congestive heart failure and low blood pressure. He had no postoperative complications. The ruptured papillary muscle was identified as the anterolateral one during surgery (Figure 8).

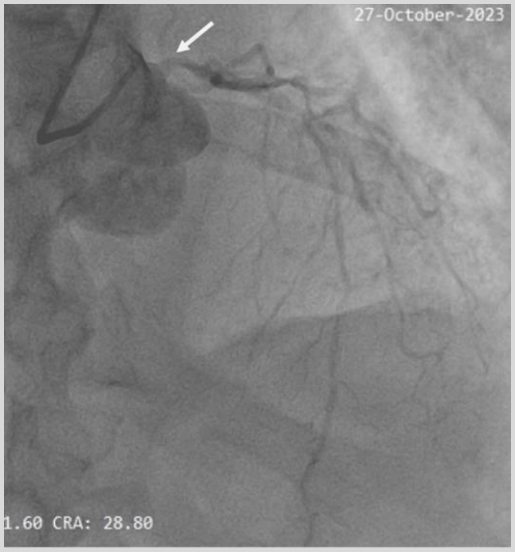

Figure 7: Cranial coronary angiography view showing a tight lesion of the proximal left main artery (white arrow).

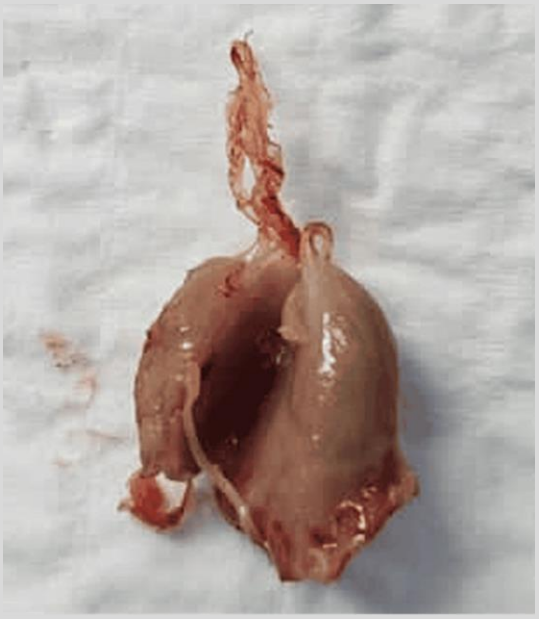

Figure 8: Ruptured anterolateral papillary muscle.

The postoperative follow-up echocardiography revealed preserved left ventricular function with a mitral bioprosthesis functioning appropriately (Figure 9).

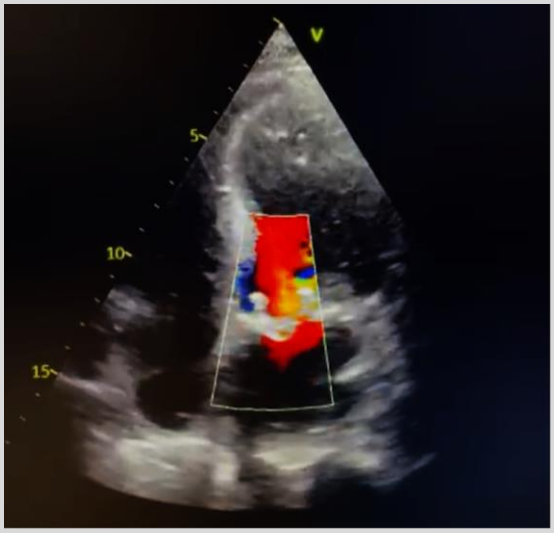

Figure 9: Postoperative echocardiography showing a properly functioning mitral bioprosthesis.

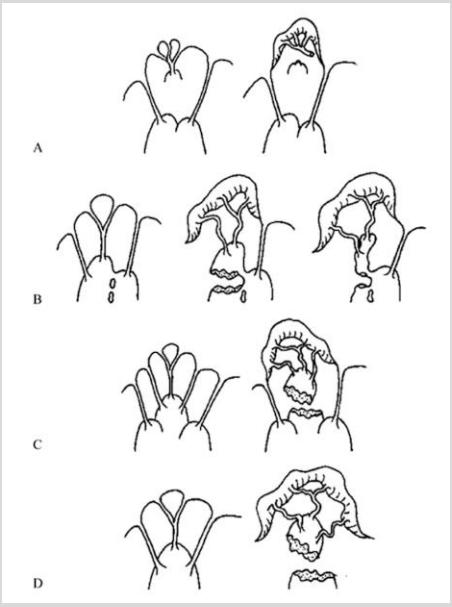

Figure 10: Diagram showing the different mechanisms of ischemic mitral valve prolapse as described by Jouan et al. [10]. A: Rupture of a commissural cord, B: Partial rupture (necrosis of a single head), C: Incomplete rupture (detachment of the main head), D: Total rupture.

Discussion:

PMR, ventricular septal rupture, and free wall rupture are severe and potentially lethal mechanical complications of acute myocardial infarction (AMI). They share a similar underlying pathophysiology: the rupture or tearing of necrotic myocardium due to myocardial infarction (MI) [3]. We will discuss the risk factors, management, and complications of PMR.

PMR is an extremely rare but often fatal complication of transmural AMI. Like other mechanical complications of AMI, its incidence has significantly decreased with the widespread use of reperfusion therapy, occurring in only 0.1-1% of the cases [4]. However, reported in-hospital mortality rates remain high: between 10-30% with surgical intervention and 50-75% without surgical intervention [5-7]. It usually occurs within two to seven days post-MI and results in acute severe MR secondary to prolapse of the mitral leaflet. Unlike other mechanical complications, the infarct size is relatively small in 50% of cases, as the preserved ejection fraction allows for a higher shear force to be exerted on the ischemic papillary muscle, predisposing it to rupture [8,9].

Various lesions producing ischemic mitral valve prolapse have been described, notably by Jouan et al. (Figure 10) [10]. Complete transection of a left ventricular papillary muscle is usually incompatible with life, as the sudden onset of massive mitral regurgitation (MR) that develops cannot be tolerated. A more frequent occurrence, however, involves the rupture of a segment of the papillary muscle, typically at its tip or head, leading to severe MR, which may be serious but not immediately life-threatening [4,10]. In our case, there was partial rupture of the papillary muscle, involving a single head of the muscle.

The posteromedial papillary muscle is by far the most commonly injured, accounting for 66-91% of cases, which is six to 12 times more common than anterolateral PMR [6,10]. To understand this discrepancy, two main factors must be taken into account: blood supply and stresses exerted on the structures. Indeed, the posterior papillary muscle is more sensitive to ischemia because its blood supply relies on a single main artery: the posterior descending coronary artery, furnished either by the right coronary or the circumflex artery depending on dominance. Moreover, it is subjected to greater shear force because of its deep location within the left ventricular wall. Conversely, the anterior papillary muscle's blood supply is provided by both the first obtuse marginal artery, originating from the left circumflex artery, and by the first diagonal branch, originating from the left anterior descending artery. Furthermore, it has a superficial location making the tension exerted by the chordae on this papillary muscle relatively low [5,6,9,10].

Clinically, patients who develop rupture of a papillary muscle manifest with pulmonary edema and/or cardiogenic shock. It's important to highlight that, in cases of highly eccentric leaks, pulmonary edema can be unilateral, depending on the papillary muscle affected. In our case, the MR demonstrates an eccentric pattern, predominantly directed towards the right pulmonary veins, resulting in primarily right-sided pulmonary edema. On cardiac auscultation, the murmur of MR due to prolapse can be harsh, unlike the typical murmur of MR. It is usually mid-to-late systolic but may also be holosystolic. In cases of severe acute MR, the murmur may be unimpressive or even absent due to rapid pressure equalization between the left atrium and left ventricle [9].

Immediate echocardiographic assessment is indicated in ACS when a mechanical complication is suspected [11]. The contrast between a precarious hemodynamic state and a hypercontractile left ventricle on echocardiography should raise suspicion of a mechanical complication such as PMR or ventricular septal rupture. In this context, echocardiography can promptly recognize MR secondary to partial or complete PMR and distinguish it from other, generally less severe forms of MR that occur with AMI. Color flow Doppler imaging is particularly valuable in distinguishing acute MR from ventricular septal defects in the context of AMI. However, diagnosing acute severe MR with transthoracic echocardiography can be challenging in cases featuring narrow eccentric jets and rapid equalization of pressures. In such instances, transesophageal echocardiography should be used, especially when suspicion is high, as it offers greater diagnostic accuracy [4].

PMR in the context of AMI requires urgent valvular surgery and CABG. While awaiting surgery, patients should receive diuretics and intravenous vasodilators. In cases of hemodynamic instability, inotropic support or mechanical assistance with an intra-aortic balloon pump (IABP) should be considered. Mitral valve replacement is more commonly performed than repair, primarily because it is quicker, and secondly, because suturing necrotic tissue is technically challenging. The operative mortality rate is 20-40% [12], but long-term survival for patients after the initial phase is generally good [8].

The case we reported is notable because it describes a rare mechanical complication of AMI: papillary muscle rupture (PMR), involving the anterolateral head, which is extremely uncommon. Additionally, it highlights that older patients may present with atypical and subtle manifestations of AMI, as our patient did not report acute chest pain and exhibited only mild orthopnea. Interestingly, the case also demonstrates that PMR can result in unilateral pulmonary edema, further emphasizing the atypical presentation. Finally, with respect to the ECG, this case reinforces that ST segment elevation in leads aVR and/or V1, accompanied by diffuse ST segment depression, should be managed as STEMI, in line with the latest 2023 ESC guidelines [11].

Conclusion:

PMR is a rare and life-threatening complication of AMI. The diagnostic challenges and high mortality rates associated with this condition emphasize the need for prompt recognition and intervention. Management involves urgent valvular and CABG surgery, with considerations for mechanical circulatory support, notably IABP. However, in-hospital mortality rates remain high. Therefore, the optimal approach is prevention through early reperfusion and should include careful attention to atypical clinical and electrical presentations of ACS, especially in the elderly.

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