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Pseudo-endocarditis secondary to ruptured posteromedial papillary muscle with anatomical variation: A Case Report

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ABSTRACT

Myocardial infarction is the leading cause of papillary muscle rupture. This complication occurs in up to 5% of cases post MI and although rare, it constitutes a cardiac emergency if left untreated.

On this basis, a 59-year-old male presented with low-grade fever and atypical chest pain with raised inflammatory markers and troponin levels. He was treated for infective endocarditis after echocardiography revealed a mass on the mitral valve, which was presumed to be a mitral valve vegetation and so he completed a 6-weeks course of antibiotics followed by elective mitral valve replacement surgery.

During surgery, it was discovered that there was no endocarditis. Instead an unusually small muscle head of one of the posteromedial papillary muscle groups had ruptured secondary to an inferior myocardial infarction. This ruptured muscle head was highly mobile and mimicked a mitral valve vegetation. The mitral valve was successfully repaired, and the right coronary artery grafted.

He made a full recovery but developed new-onset atrial fibrillation for which he is awaiting elective cardioversion.

One should have a high index of suspicion for diagnosing papillary muscle rupture as it may mimic valvular vegetation on echocardiography, especially if the papillary muscle involved is an anatomical variant.

Keywords: Papillary muscle, rupture, endocarditis, mitral valve

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List of abbreviations

MI: myocardial infarction
ECG: electrocardiography
CRP: c-reactive protein
TTE: Transthoracic echocardiography
TOE: Transoesophageal echocardiography
RCA: Right Coronary Artery
AL: anterolateral
PM: Posteromedial
LV: left ventricle

Introduction

Myocardial infarction (MI) is the primary cause of papillary muscle rupture and can lead to severe mitral regurgitation [1]. The incidence of papillary muscle rupture is approximately 0.5-5% and it tends to occur 4-7 days post MI [2,3]. Patients with papillary muscle rupture may demonstrate clinical findings that result in misleading diagnoses especially in conjunction with fever and raised infection markers. The aim of this case report is to demonstrate how ruptured papillary muscle secondary to inferior MI was misdiagnosed as infective endocarditis. There have only been a few case reports in the literature documenting papillary muscle rupture mimicking endocarditis. Albeit, this is the first case report where a papillary muscle anatomical variant was the cause for echocardiographic findings suggesting endocarditis.

Case Description

A 59-year-old male with no comorbidities presented with sharp left-sided chest pain and dyspnoea for two hours which started at rest. These symptoms had been intermittently triggered by exertion and relieved by rest over the past 7 days, but he denied other symptoms of heart failure, chest infection or weight loss and had no risk factors of venous thromboembolism. He is an ex-smoker (stopped 25 years ago) and a social drinker with no family history of cardiovascular disease. He had a low-grade fever and hypoxia with otherwise normal observations. Physical examination revealed a new apical pansystolic murmur and fine crepitations to the lung bases with a raised jugulovenous pressure and mild pedal oedema.

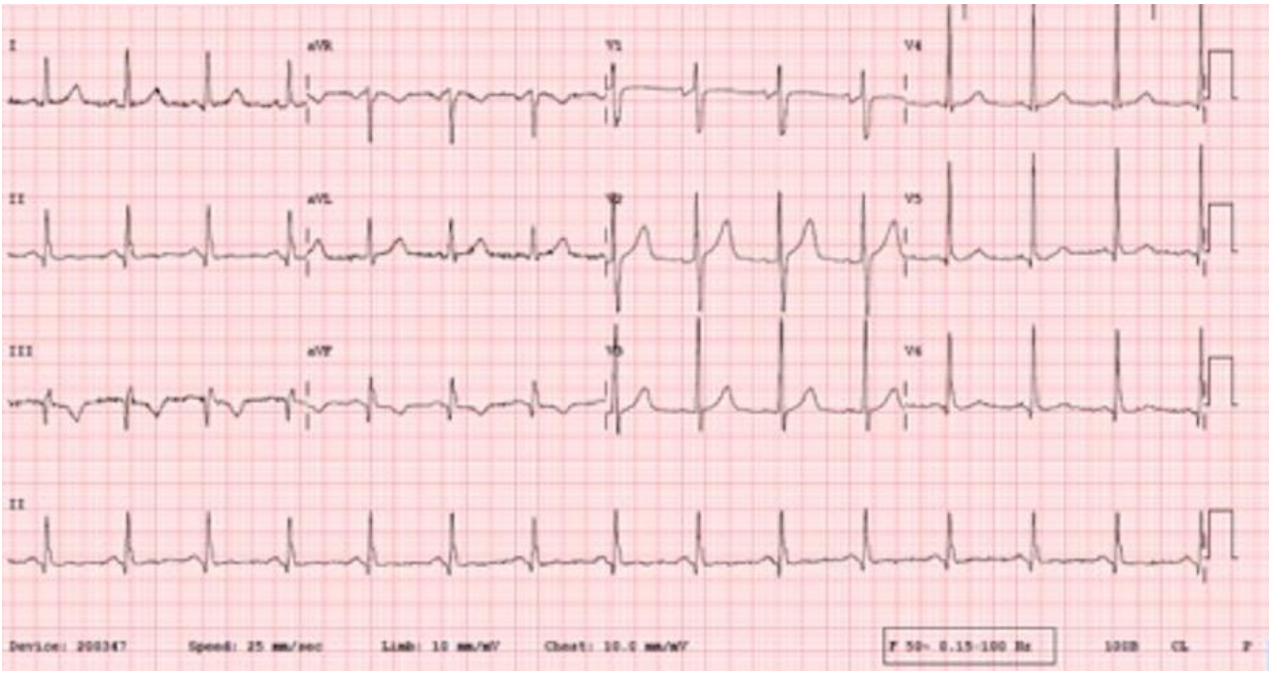

Figure 1: ECG demonstrating Q waves and T wave inversions in the inferior leads.

Electrocardiography (ECG) demonstrated ischaemic changes in the inferior leads [figure1] with baseline and 1-hour Troponin I level of 786 ng/L and 904 ng/L respectively (<12ng/L). Other significant blood results were a raised c-reactive protein (CRP) of 124 mg/L (< 5mg/L) and a white cell count of 11 x 10^9g/L (4-10 x10^9g/L). He had predominantly right sided infiltrates suggestive of cardiogenic pulmonary oedema on chest x-ray [figure 2].

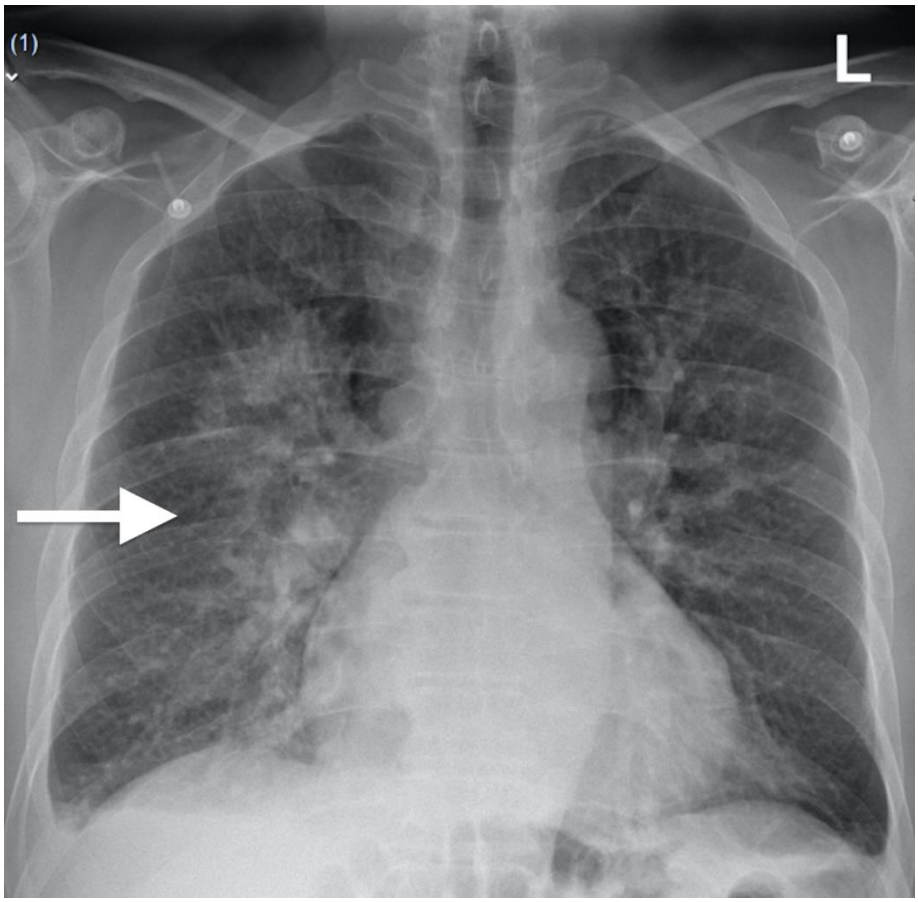

Figure 2: Antero-posterior chest xray demonstrating predominantly right-sided pulmonary oedema (see arrow).

Urgent transthoracic (TTE) and subsequent transoesophageal (TOE) echo revealed a mobile mass attached to anterior mitral valve leaflet with features of severe eccentric mitral regurgitation (which accounted for the pulmonary oedema and resultant hypoxia) [figure 3a and 3b].

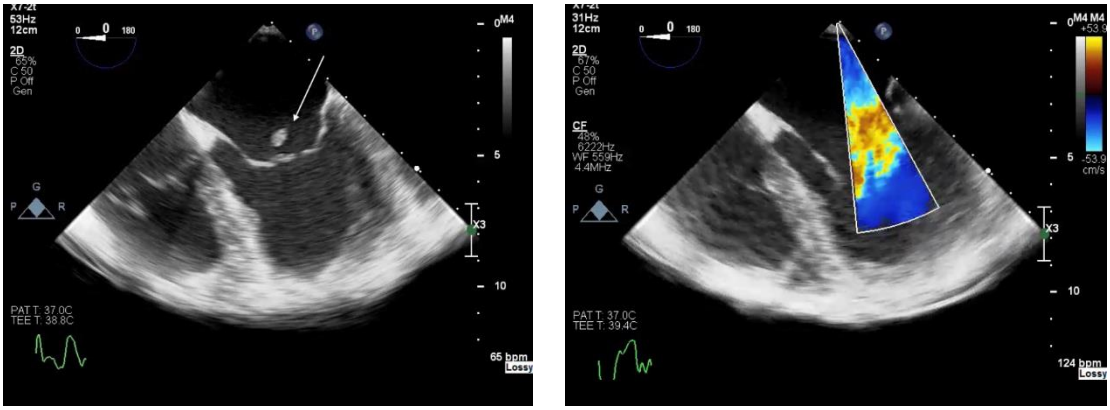

Figure 3a: Transoesophageal echocardiogram demonstrating a mass attached to the tip anterior leaflet of the mitral valve (see arrow).

Figure 3b: Transoesophageal echocardiogram demonstrating mitral regurgitation with an eccentric jet.

He was diagnosed as infective endocarditis with acute heart failure secondary to severe mitral regurgitation and possible delayed presentation MI. He was commenced on aspirin, ticagrelor, bisoprolol, ramipril and furosemide. A set of 3 blood cultures were taken and he was started on intravenous amoxicillin 2g 4-hourly and gentamicin 1mg/kg 12-hourly which was changed to ceftriaxone 2g once-daily after 72 hours as per the Microbiologist's advice as he continued to have temperature spikes. All blood cultures were negative but antibiotic treatment continued for a

possible culture negative endocarditis based on the modified Duke's criteria. Additionally, inpatient coronary angiogram demonstrated 99% stenosis of the right coronary artery (RCA) in keeping with a delayed presentation MI [figure 4].

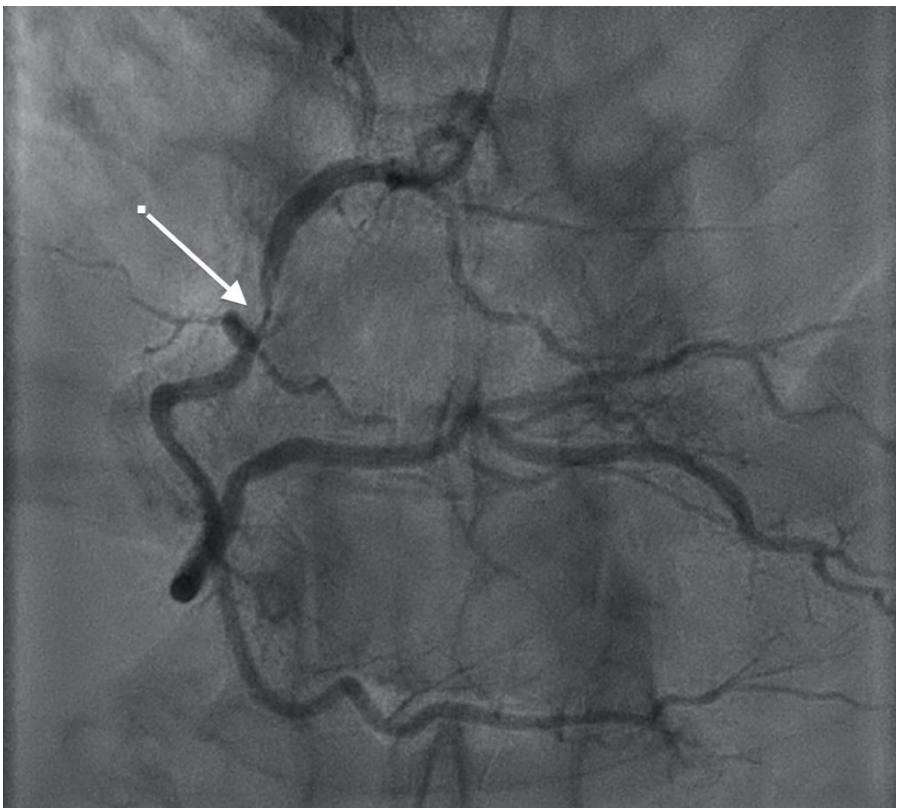

Figure 4: Coronary angiogram demonstrating a 99% stenosis of the proximal right coronary artery (see arrow).

He successfully completed a 6-weeks course of antibiotics with weekly outpatient TTE and blood tests, while awaiting elective mitral valve replacement surgery. During surgery, it was discovered that there was no endocarditis but complete rupture of one of the 3 posteromedial papillary muscle groups secondary to RCA infarction. The ruptured posteromedial papillary muscle head was unusually small which was an anatomical variant. It was this ruptured muscle head that was mimicking a mitral valve vegetation. The mitral valve was successfully repaired by resuspending the flailing part of the P3 division of the mitral valve with neochords and closing the posterior medial commissure. The right coronary artery was also grafted. His recovery was uneventful except for new-onset atrial fibrillation for which he is awaiting elective cardioversion.

Discussion

The mitral valve consists of anterolateral (AL) and posteromedial (PM) papillary muscles, with variable morphology due to incomplete delamination of the trabecular ridge during embryologic development [2,4]. The left ventricular (LV) papillary muscles are usually conical and broad-based with the PM muscle having 2 or more muscle heads [5]. The PM muscle has a single blood supply from the RCA and is 6-10 times more susceptible to ischaemia and rupture [2-3]. It is most commonly associated with inferior MI and rupture can occur even with small infarcts [2-3,5]. Complete papillary muscle rupture involves the entire muscle and causes cardiogenic shock while incomplete papillary muscle, as in our patient, involves one of the muscle heads and results in mitral regurgitation and pulmonary oedema [4-5]. Papillary muscle rupture accounts for 0.4-5% of deaths after a MI and if left untreated, it has an 90% mortality rate by 1 week [5]. Within the context of this case report it was believed that the anatomical variation of the ruptured papillary muscle was the reason why the patient did not have more acute and severe symptoms.

Echocardiography is the imaging modality of choice in assessing cardiac abnormalities. TOE has a diagnostic sensitivity of 95-100% for papillary muscle rupture as it is better able to visualise the mitral valve apparatus [6]. With regards to our patient, the anatomical variation of the ruptured muscle head posed a challenge in diagnosing papillary muscle rupture with TOE. This was further confounded by the presence of fever and raised inflammatory markers. However, myocardial necrosis may trigger a sterile cytokine-mediated inflammation with fever and leucocytosis 2-5 days post MI [7]. This was the likely explanation for our patient as his blood cultures were persistently negative. In incomplete papillary muscle rupture surgical repair of the mitral valve is preferred as vascularity, innervations and continuity with ventricular musculature remains intact [4].

Conclusion

Our case report demonstrated that papillary muscle rupture may present atypically, and the anatomical variation of the ruptured papillary muscle head made the diagnosis with echocardiography difficult. One should have a high index of suspicion for papillary muscle rupture as a delay in diagnosis and treatment may be associated with significant morbidity and mortality. Mitral valve repair is the gold standard for the surgical treatment of incomplete papillary muscle rupture.

Acknowledgement

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Statement of Ethics

The subject of this case report has given written informed consent to publish this case (including publication of images).

Disclosure Statement

The authors have no conflicts of interest to declare

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