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CASE REPORT

Spontaneous Papillary Muscle Rupture Coexisting with Chronic Mitral Regurgitation – A Case Report

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Summary

Given the low prevalence of coronary artery disease in the young, spontaneous papillary muscle rupture is an uncommon cause of severe mitral regurgitation and emergency admission in this age group. Spontaneous papillary muscle rupture is usually associated with features of acute mitral regurgitation. We highlight an incidental finding of a ruptured papillary muscle in a young adult with features of chronic mitral regurgitation. A 20-year-old male presented with a history of worsening symptoms of heart failure. Examination showed features of congestion, such as bilateral pedal oedema and tender hepatomegaly. The precordial examination revealed a displaced apex beat, gallop rhythm, and an apical pansystolic murmur. Transthoracic echocardiography showed thickened mitral valve leaflets, diastolic doming of the anterior mitral valve, ruptured papillary muscle with anterior mitral valve prolapse, restrictive mitral inflow pattern, severe mitral regurgitation, mild tricuspid regurgitation, and dilated cardiac chambers. At surgery, dilated cardiac chambers, avulsed anterolateral papillary muscle, and a prolapsed anterior mitral valve leaflet were seen. He had mitral valve replacement with a 31/33 ON-X mechanical mitral valve and left atrial ligation. He has since been on follow-up care. This case highlights that in rare cases, rheumatic heart disease can present with spontaneous papillary muscle rupture on a background chronic mitral regurgitation.

Keywords: Chronic Mitral regurgitation, Congestive Cardiac Failure, Rheumatic Heart Disease, Spontaneous Papillary Muscle Rupture, Valve replacement surgery.

Introduction

Spontaneous papillary muscle rupture (PMR) is a rare but life-threatening emergency that is most

commonly associated with acute myocardial infarction. [1–3] It is typically deadly and resistant to medical interventions. Spontaneous rupture of the papillary muscle seldom occurs without an acute myocardial infarction. [4] However, papillary muscle rupture can occur due to myocarditis, infective endocarditis, trauma, microvasculopathy, rheumatic valvular heart disease, collagen disease, or mitral valve apparatus calcification. [1,2] Acute rupture frequently results in severe mitral valve regurgitation, acute life-threatening cardiogenic shock, and pulmonary oedema. Non-ischaemic aetiologies may be associated with preserved left ventricular systolic function, resulting in a decreased burden of cardiogenic shock compared to ischaemic causes [1] Rupture of the anterolateral papillary muscle is commonly associated with rheumatic mitral valve changes. Rheumatic valve disease thickens the valve leaflet edges, and subvalvular apparatus, with attendant restricted leaflet motion. [5] Once a papillary muscle rupture has been diagnosed, urgent mitral valve replacement is required to correct the patient's hemodynamic deterioration. [4]. The objective of this report is to heighten awareness about this relatively rare but potentially fatal condition.

Case Description

A 20-year-old male was referred to ABUAD Multisystem Hospital, Ado-Ekiti, on account of dyspnoea on exertion for two months and bilateral leg swelling of two weeks duration. Dyspnoea was initially on exertion but later progressed to occur at rest; there were associated palpitations, orthopnoea, paroxysmal nocturnal dyspnoea, and nocturnal dry cough. About two weeks later, he noticed painless, progressive, bilateral leg swellings with associated right hypochondriac pain. The patient presented without any preceding fever or chest trauma. He had no history of childhood exercise intolerance, hypertension, diabetes mellitus, comparable hospital admissions or cardiac surgeries. Furthermore, he did not consume alcohol, smoke cigarettes, or utilise recreational drugs. He reported diminished urine output, facial swelling that regresses throughout the day, and nocturia.

On examination, he was conscious and alert, not in any obvious distress, afebrile (36.5°C), pale, anicteric, not dehydrated, acyanosed, with no finger clubbing, no asterixis, but with bilateral pitting pedal oedema up to the distal third of the legs. His percutaneous oxygen saturation was 99% in room air. He had a pulse rate of 104 beats per minute, regular, normal volume, synchronous with other peripheral pulses; the blood pressure was 122/81 mmHg, and the jugular venous pressure was not elevated. The apex beat was at the sixth left intercostal space, anterior axillary line but not heaving. On auscultation, he had a gallop rhythm, an apical pansystolic murmur, with a loud pulmonic component of the second heart sound. His respiratory rate was 28 cycles per minute, with reduced breath sound over the right lower lung zones. The abdomen was full, moved with respiration, and had tender hepatomegaly with no demonstrable ascites.

The laboratory investigations showed a white cell count of 7.4×10^9/L, a lymphocyte count of 64.7%, a granulocyte count of 27.5%, and a haemoglobin concentration of 13.8g/dL. Serum electrolytes, urea, and creatinine were essentially normal. Blood culture and sensitivity yielded no bacterial growth. Prothrombin time was 21.4 seconds, and INR was 1.5. The chest X-ray revealed a cardiothoracic ratio of 64.13%, with a straightened left heart border. There was no focal lung lesion, and the pleural recesses were free. The electrocardiography (ECG) showed sinus tachycardia, right axis deviation, right ventricular (RV) hypertrophy, prolonged QTc, and widespread non-specific T wave abnormalities and features of RV strain pattern (S1Q3T3) (Figure 1). Holter ECG revealed no significant atrial or ventricular arrhythmias.

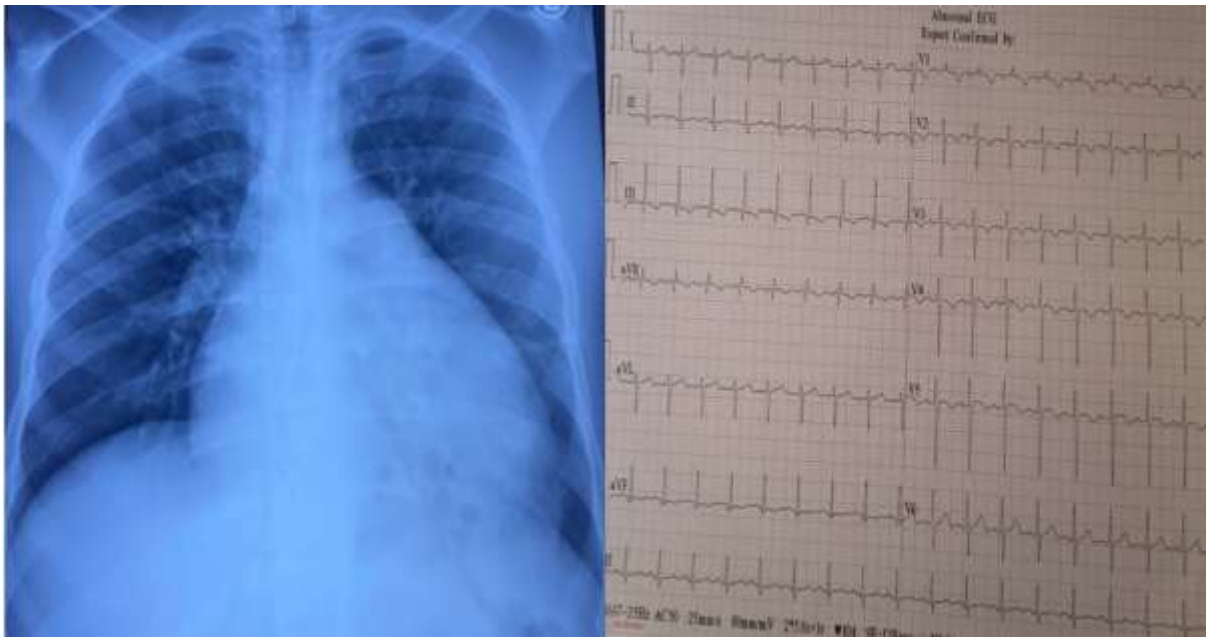

Figure 1: (a) Chest radiograph showing cardiomegaly with a straightened left heart border. (b) ECG showing sinus tachycardia, right axis deviation, and right ventricular hypertrophy.

The echocardiography showed borderline left ventricular (LV) systolic function (Simpson's biplane EF of 58.8%). All chambers were dilated, eccentric LV hypertrophy, thickened mitral valve leaflets, diastolic doming of the anterior mitral valve, ruptured papillary muscle with anterior mitral valve prolapse, restrictive mitral inflow pattern, severe mitral regurgitation, and mild tricuspid regurgitation (TRVmax = 2.8m/s). There was no aortic regurgitation or stenosis. Other findings were normal right ventricular systolic function, mildly elevated right ventricular systolic pressure of 36.36 mmHg, and mild circumferential pericardial effusion.

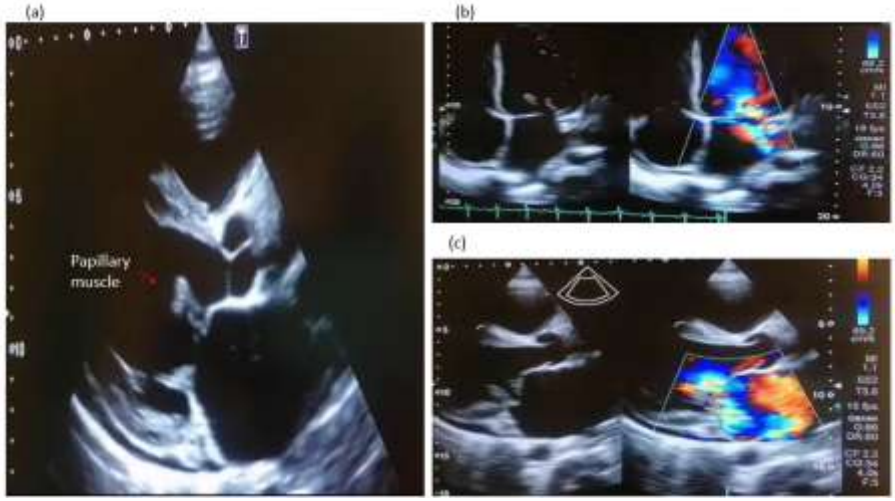

Figure 2. Transthoracic echocardiogram showing (a) thickened mitral valve leaflets, diastolic doming of the anterior mitral valve, some degree of thickening of the subvalvular apparatus, and a stump-like papillary muscle at the tip of the anterior mitral valve leaflet in the parasternal long axis view. (b and c) Colour-flow Doppler imaging shows a posteriorly directed jet of severe mitral regurgitation.

The clinical diagnosis was heart failure due to severe chronic primary mitral valve regurgitation, likely of rheumatic origin. The patient received intravenous furosemide 40mg every 12 hours, oral spironolactone 25mg daily, oral dapagliflozin 10mg daily, and oral ramipril 2.5mg daily. He was subsequently referred for mitral valve replacement surgery. During the surgical procedure, the findings revealed pericardial effusion, dilated cardiac chambers, a tense pulmonary artery, a normal aorta, an avulsed anterolateral papillary muscle, and a prolapsed anterior mitral valve leaflet. The patient underwent mitral valve replacement utilising a 31/33 ON-X mechanical mitral valve and left atrial ligation. Subsequently, he was prescribed warfarin at a dose of 2.5 mg daily, digoxin at 0.125 mg once daily, ramipril at 2.5 mg in the morning and 1.25 mg in the evening, bisoprolol at 1.25 mg once daily, and frusemide at 20 mg once daily. He has been on continuous outpatient follow-up care with meticulous INR monitoring.

Ethical considerations

This research was carried out following the Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans. Ethical clearance was obtained from the Afe Babalola University Multisystem Hospital Ethics and Research Committee (AMSH/REC/25/006). Informed consent was obtained from the patient to use his data in the research.

Discussion

Mitral regurgitation (MR) is the most prevalent valvular heart disease [6,7]. It can be due to abnormalities of the mitral valve and its apparatus (primary MR) or left ventricular dysfunction (secondary MR). [7] Rheumatic valve disease accounts for a significant cause of heart failure and chronic mitral regurgitation in Nigeria and Africa at large. [6,8] However, papillary muscle rupture is a rare and life-threatening cause of acute MR. [4] The definitive treatment of papillary muscle rupture is valve replacement. [4]

This index case had symptoms of about two months duration and signs of heart failure that included evidence of right ventricular dysfunction. The displaced apex beat, cardiomegaly on the chest x-ray, and dilated cardiac chambers on echocardiography are evidence of volume overload, suggesting a longstanding MR. [5] These contrast with the findings of sudden onset of dyspnea and features of haemodynamic instability, which characterise an acute MR. [7, 9] The identification of a prolapsed anterior mitral valve leaflet, ruptured papillary muscle, and severe mitral regurgitation with features of chronic LV volume overload and thickened leaflets on echocardiography and at surgery, suggests papillary muscle rupture as a contributory factor to the severe MR rather than being the only cause. [10]

Although the commonest risk factor for PMR is acute myocardial infarction, the index patient is young and has no known cardiovascular risk factors. [11] Also, the anterolateral papillary muscle was involved as opposed to the posteromedial papillary muscle, which is more commonly affected in acute myocardial infarction due to its single blood supply by the posterior descending artery. [12] There was no obvious regional wall motion abnormality on echocardiography. These put together make papillary muscle rupture of non-ischaemic origin likely, but this does not negate the need for coronary angiography in this patient, which could not be done at the same facility due to a lack of expertise at that time. Infective endocarditis was considered, but no vegetation was identified on the valves on echocardiography. The blood culture was also negative.

The findings of a prolapsed anterior mitral valve leaflet, thickened mitral valves exhibiting diastolic doming of the anterior leaflet, severe mitral regurgitation (MR), dilated cardiac chambers, and borderline left ventricular (LV) systolic function led to a diagnosis of non-ischaemic-chronic-primary-MR, likely due to rheumatic heart disease. It is important to consider that in our context, rheumatic heart disease is the most common cause of valvular heart disease in young individuals. [10] He started diuretics to alleviate congestion while awaiting definitive treatment—mitral valve replacement. A mechanical valve is preferred in young individuals, although lifelong vitamin K antagonist therapy is necessary as prescribed for this patient. [13,14]

Conclusion

This case illustrates that, in exceptional instances, rheumatic heart disease may manifest with spontaneous papillary muscle rupture, alongside concomitant characteristics of chronic mitral regurgitation.

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Authors' Contributions: OBO and OSO conceived the research idea. OBO, UPA and OOE analysed and interpreted the data. OBO, OOE, UPA and OSO drafted the manuscript. OSO, OOE, UPA and APT revised the draft for sound intellectual content. OSO, OOE, UPA, OBO and APT approved the final version of the manuscript.

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