Journal of Current Medical Research and Opinion
CMRO 08 (11) 4506-4510 (2025)
DOI: https://doi.org/10.52845/CMRO/2025/8-11-3
www.cmro.in
CMRO
ISSN (O) 2589-8779
(P) 2589-8760

Case Report

Rapid Response

Dr. Stephen Maharaj[1], Dr. Sophina Baksh[2], Professor Giovanni Teodori[3], Mrs. Natasha Rahaman[4], Dr. Risshi Rampersad[5]

[1]Cardiology Resident, Cardiology Department, Caribbean Heart Care Medcorp Ltd., St. Clair Medical Centre, Port of Spain, Trinidad and Tobago.
[2]Surgical Resident, Cardiology Department, Caribbean Heart Care Medcorp Ltd., St. Clair Medical Centre, Port of Spain, Trinidad and Tobago.
[3]Head of Cardiac Surgery, Surgical department, Caribbean Heart Care Medcorp Ltd., St. Clair Medical Centre, Port of Spain, Trinidad and Tobago.
[4]Cardiac Surgeon, Surgical Department, Caribbean Heart Care Medcorp Ltd., St. Clair Medical Centre, Port of Spain, Trinidad and Tobago.
[5]Head of Department, Cardiology Department, Caribbean Heart Care Medcorp Ltd., St. Clair Medical Centre, Port of Spain, Trinidad and Tobago.

Abstract

A 51-year-old hypertensive male presented with worsening shortness of breath, orthopnea and paroxysmal nocturnal dyspnea. X-ray imaging revealed cardiomegaly, consolidation and bilateral pleural effusions, suggestive of pneumonia. He eventually became hemodynamically unstable. Echocardiography revealed severe mitral regurgitation secondary to complete papillary muscle rupture. Coronary angiography showed triple vessel coronary artery disease. The patient underwent urgent mitral valve replacement with a mechanical prosthesis and coronary artery bypass grafting (CABG). Post-operative recovery was uneventful and transthoracic echocardiography showed good mitral valve and systolic function.

Keywords: papillary muscle rupture, mitral regurgitation, myocardial infarction, mitral valve replacement, coronary artery disease, coronary artery bypass grafting.

Introduction

Case Report

A 51-year-old male, known hypertensive, presented to a medical center with a four-day history of worsening shortness of breath. He also reported paroxysmal nocturnal dyspnea, orthopnea and minimal pedal edema. Chest X-ray findings included bilateral infiltrates and cardiomegaly. CT chest showed cardiomegaly with bilateral pleural effusions and consolidations suggestive of pneumonia. Electrocardiogram at the time showed a sinus tachycardia with non-specific ST depressions in V_4 - V_6. He was treated with intravenous (IV) ceftriaxone 1g, IV furosemide stat, atenolol and hydrocortisone and allowed home. He reported no significant improvement of symptoms with these medications and later that day presented to the emergency department (ED) of a private hospital.

In the ED, he had acute shortness of breath (New York Heart Association Class IV) and denied any chest pain, nausea, or vomiting. On examination blood pressure (BP) was 104/72, heart rate (HR) was 117 bpm. On auscultation, S1, S2 heard and

Corresponding Author: Yassine Merad, Surgical Resident, Cardiology Department, Caribbean Heart Care Medcorp Ltd., St. Clair Medical Centre, Port of Spain, Trinidad and Tobago.
E-mail: stephenmaharaj21@yahoo.com

Received: 22.08.2025 | **Revised:** 29.10.2025 | **Accepted:** 16.11.2025 | **Published:** 19.11.2025

there was a pansystolic murmur heard best at the apex, radiating to the left axilla. Air entry was reduced bilaterally, consistent with pulmonary edema up to mid-lung fields and small bilateral effusions. On transthoracic echocardiogram (TTE), a preserved ejection fraction of 60 to 65 per cent was noted. Laboratory results included white blood cell count 21 x 10^9/L, C reactive protein 136.6 mg/L, erythrocyte sedimentation rate 30 mm/hr.

The patient was admitted, and treatment began with furosemide, spironolactone, clopidogrel, trimetazidine, aspirin, bisoprolol, digoxin, and enoxaparin. He was also started a course of antibiotics. Regular laboratory tests were done, and the patient had improvement in clinical markers, and hemodynamic stability was maintained. His inflammatory markers plateaued seven days after presentation to the ED. Of note, there was no bacterial growth after five days on blood cultures.

At midnight on day eight of admission, he became acutely short of breath. BP 100/70, HR 98 bpm, SPO_2 80 per cent that increased with re-breather mask to 96-98 per cent, Temp 38.1 C. On examination, he was in flash pulmonary edema with severe mitral valve regurgitation by bedside TTE. A nitroglycerin infusion of 50 mg in 50 ml was started at 5 ml/h and dopamine infusion 200 mg in 50 ml started at 3 ml/h to maintain a MAP > 70 mmHg. He was treated as suspected infective endocarditis with severe mitral regurgitation secondary to a ruptured posterior mitral valve leaflet, as well as type II myocardial infarction in the setting of sepsis, tachycardia, severe mitral regurgitation and pulmonary edema. At this time, the patient required treatment in a high-dependency unit or Intensive Care Unit (ICU), including bilevel positive airway pressure/continuous positive airway pressure (BiPAP/CPAP) ventilator support. As such, he was transferred for management at our institution.

The patient was admitted to ICU and started on propofol, nitroglycerin, esomeprazole, furosemide and continued treatment with intravenous antibiotics (gentamycin, doxycycline, cefuroxime). On TTE, severe mitral regurgitation secondary to complete papillary muscle rupture was seen. These findings were then confirmed on transesophageal echocardiogram (Fig.1) with no vegetations noted and a preserved ejection fraction (EF) of 75 per cent. He was subsequently taken for a formal coronary angiogram and findings included triple vessel coronary atherosclerotic disease and anomalous origin of the right coronary artery (RCA). There was total occlusion of the distal segment of the RCA, a 70 per cent calcified lesion in the mid-segment of the left anterior descending artery, and an 80 to 90 per cent stenotic lesion of a large caliber obtuse marginal 3 artery (Fig.2).

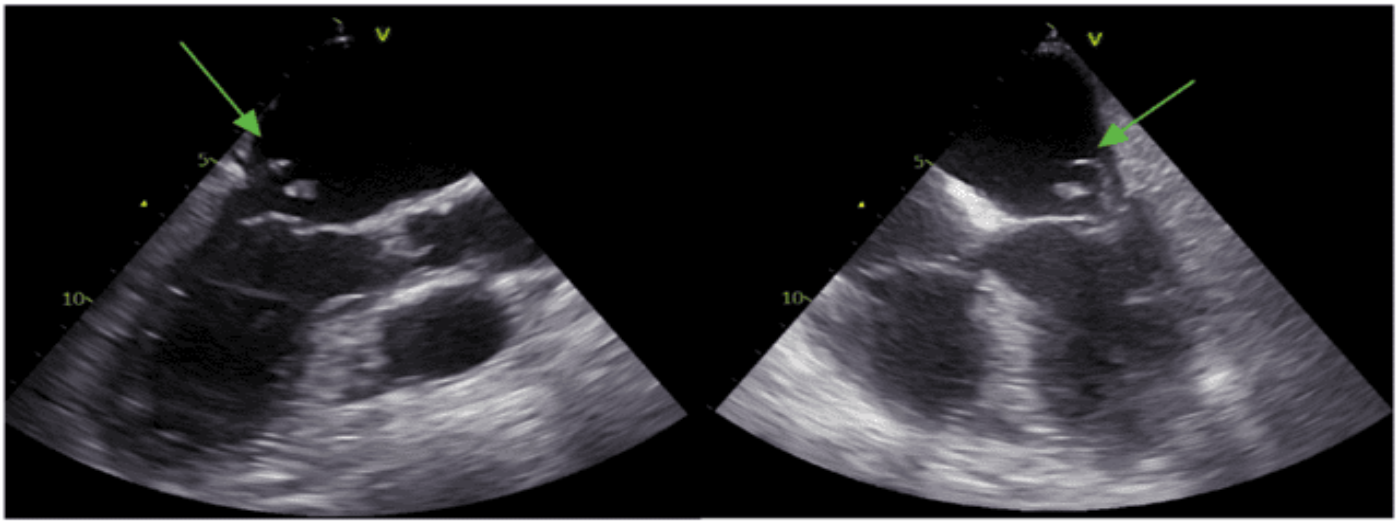

Figure 1. Transesophageal echocardiogram showing complete papillary muscle rupture and a flail posterior mitral valve leaflet.

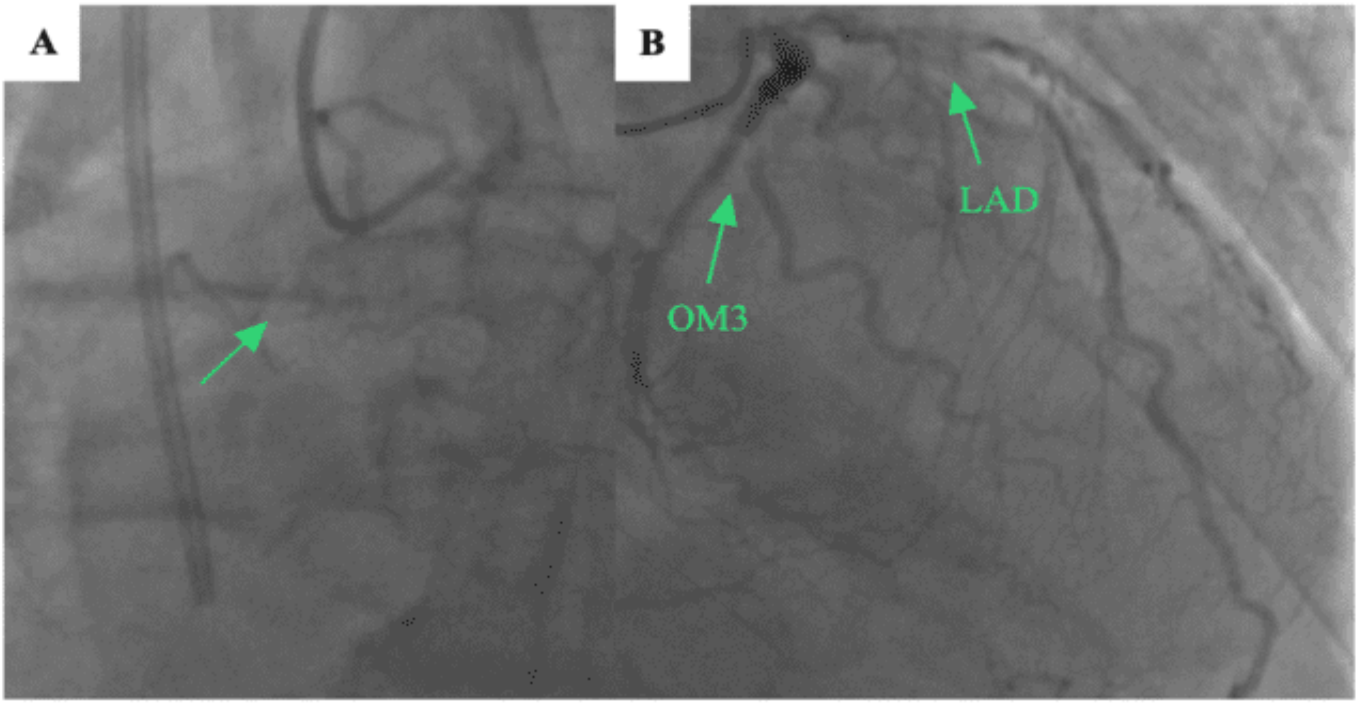

Figure 2. Coronary angiography showing: (A) Occlusion in the distal segment of the right coronary artery, (B) Stenotic lesion in the proximal segment of obtuse marginal artery 3 (OM3) artery and, a calcified lesion in the mid-segment of the left anterior descending artery (LAD).

The patient then underwent urgent coronary artery bypass grafting and mitral valve replacement. Under cardiopulmonary bypass with ascending aorta and vena cava cannulation, the following were performed: left internal mammary artery to left anterior descending artery and long saphenous vein to the first obtuse marginal artery. The mitral valve which showed chordal rupture, was resected (Fig. 3) and a 31/33 mm On-X mechanical valve was implanted. The patient was vasoplegic with low systemic arterial pressure despite very high doses of inotropes and vasoconstrictors. After more than two hours of observation and treatment of the hemodynamic status, an acceptable stability was achieved, with low values of oxygenation and blood pressure despite high fraction of inspired oxygen and high positive end-expiratory pressure.

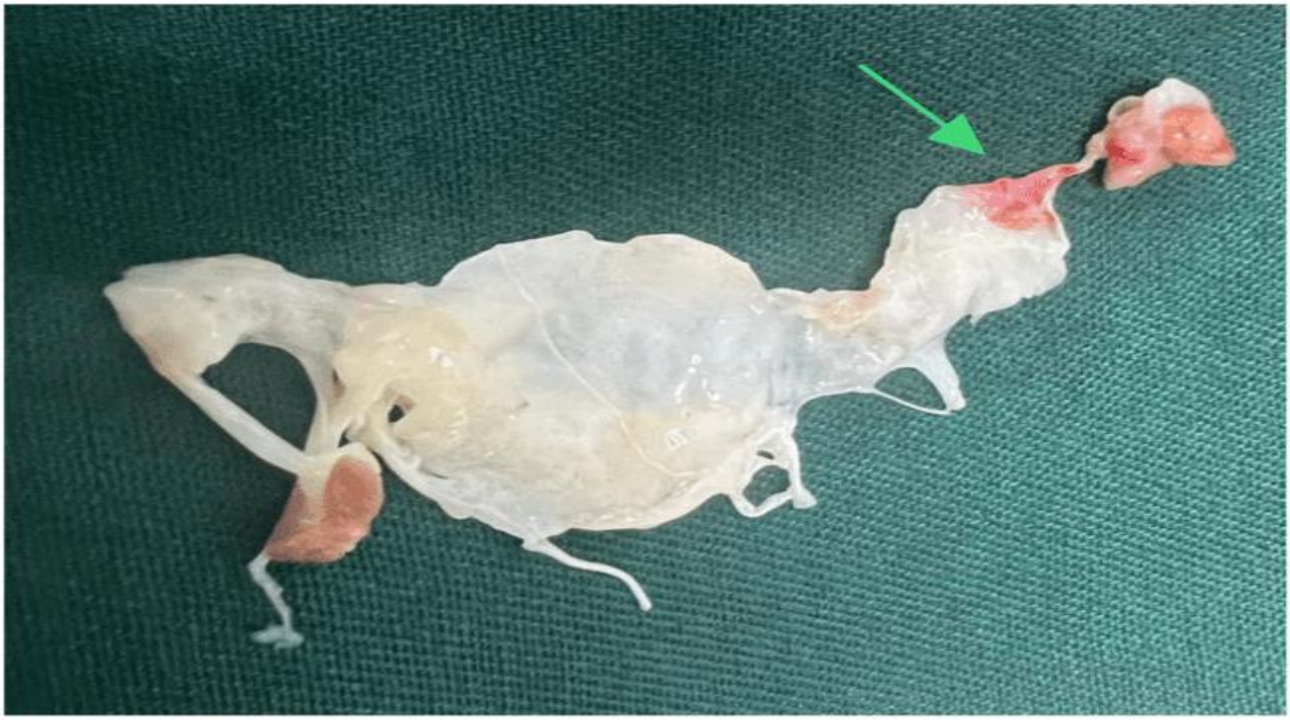

Figure 3. Photograph of the resected mitral valve with chordal rupture

In the following days, the patient improved haemodynamically and went on to have an uneventful post-operative stay. Daily laboratory results were within normal limits. Post-operative echocardiogram showed mechanical mitral valve with normal function and EF 67 percent. He was discharged ten days after surgery with appropriate secondary prevention medications.

Discussion

Papillary muscle rupture (PMR) is a serious, life-threatening complication of acute myocardial infarction (AMI) with an incidence between 0.05 and 0.26 per cent[1,2]. PMR is a mechanical complication that most often occurs within a week after an inferior myocardial infarction but can manifest up to three months later[1, 3]. Acute PMR can lead to severe mitral valve regurgitation and subsequent sudden clinical decline secondary to cardiogenic shock, pulmonary oedema and heart failure[3,4]. Risk factors for PMR include older age, female sex, history of heart failure, chronic kidney disease and a delay in presentation of first AMI[2,6].

Mitral valve competence is maintained by the anterolateral and posteromedial papillary muscles which are attached to mitral valve leaflets via the chordae tendineae[2]. The posteromedial papillary muscle has a solitary blood supply from the posterior descending artery, a major branch of the right coronary artery, whereas the anterolateral papillary muscle is supplied by both the left anterior descending and circumflex arteries. As such, posteromedial PMR is more likely to occur post AMI[1,5]. PMR can be partial or complete, occurring at one of the heads of the papillary muscle or at the papillary trunk respectively[1,3]. Patients with complete PMR tend to present more hemodynamically unstable as compared to partial or incomplete rupture[1]. Complete PMR had a greater risk of operative mortality than partial PMR, with early mortality rates of 31.5% and 10.9% respectively[1].

In most cases of PMR, patients present with an inferior ST-segment elevation myocardial infarction[1,5]. Coronary angiography confirms culprit vessel occlusion and commonly shows single or double vessel coronary artery disease[6]. Echocardiography is the preferred diagnostic tool which shows a mobile mass attached to a mitral valve leaflet prolapsing into the left atrium[2,3,6]. A preserved ejection fraction is often seen in these patients[5,6]. The proposed mechanism by which PMR occurs is as a result of normal contractility with no compensation for the compromised papillary muscle, leading to rupture[5].

Early diagnosis and prompt surgical intervention are vital for patient survival given the hemodynamically unstable nature of PMR[1,3]. Initial medical management focuses on hemodynamic support, respiratory support, and preload and afterload reduction. Therapeutics include diuretics, vasoactive agents such as norepinephrine or dopamine, and use of bilevel positive airway pressure or mechanical ventilation[3,6]. Medical treatment only of acute mitral regurgitation secondary to PMR has a mortality rate of about 80 per cent within the first 24 hours of rupture[3,7]. Several studies have reported mortality rates between 9 and 45 per cent for mitral valve repair or replacement[1,6]. In the SHOCK (SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK) Trial Registry, mitral valve surgery had half the in-hospital mortality rate compared to medical treatment only for PMR with cardiogenic shock[8].

Chord-sparing mitral valve replacement is the preferred treatment option because the operation is predictable with an established durability[6]. Of note, large series data showed that mitral valve replacement patients with chord-sparing had similar operative mortality rates to those without, 21.8 per cent and 27.1 per cent respectively. Mitral valve surgery with concomitant CABG is recommended due to significant improvement in long-term survival[4,6]. However, acute revascularisation did not affect 30-day mortality, with multiple large series reporting similar operative mortality rates with concomitant CABG (about 20 per cent) and with valve surgery only (19.8 to 22.2 per cent)[1,4,6]. As such, the risks of lengthening the operation with revascularisation must be considered compared to the long-term

benefit with CABG[6]. Nevertheless, the high risk nature of ischemic PMR and the significant fatal outcome of medical management alone demand no delay in surgical intervention with an approach that focuses on ameliorating mitral regurgitation[4,8]

Conclusion

This case highlights the severity of papillary muscle rupture (PMR) as an important complication of acute myocardial infarction. Prompt recognition and immediate intervention are essential to successful clinical outcomes due to the high mortality rate and hemodynamic unstable nature of complete PMR. Medical management only or delayed mitral valve surgery has a significantly high mortality rate. As such, timely surgical intervention is critical for survival as demonstrated in this patient who underwent mitral valve replacement and coronary artery bypass grafting.

Ethics Approval and Consent to Participate

This case report has been approved by the Ethics Review Board under Medcorp Ltd. with reference number CHCMSM9. Written informed consent was obtained from the patient for publication of this case report and images used.

Acknowledgement

We would like to acknowledge Dr Ravi Ramlal for his contributions to this case, that is, clinical input and management of the patient.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest. Although affiliated with Caribbean Heart Care Medcorp Ltd., the judgments in data interpretation and writing were not influenced by this relationship.

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