

CASE REPORT

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Idiopathic posterior papillary muscle rupture; a case report

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Abstract

Background: Papillary muscle rupture is a rare condition. Its clinical presentation, diagnosis and management can be very challenging for the clinician.

Case presentation: A 73-year-old woman with hypertension presented with chest pain, ST-segment changes, and elevated serum troponin levels. Coronary angiography was normal. Echocardiography revealed normal ventricular function, flail posterior mitral leaflet, and severe mitral regurgitation. She underwent emergent mitral valve replacement.

Conclusion: The diagnostic and management strategies of this uncommon presentation are discussed.

Keywords: Papillary muscles, Mitral valve, Shortness of breath, Valve repair

Background

Papillary muscle rupture (PMR) is itself uncommon, with incidence rates of 0.029% in patients presenting with MI, and few case reports of non-MI etiology [1]. Compared to patients without PMR, those with PMR had significantly higher in-hospital mortality rates (5.3% vs 36.3%, $p < 0.001$) [1]. Papillary muscle rupture usually occurs within 1 week post infraction. These patients present acutely with signs of acute hypoxemic respiratory failure due to severe pulmonary edema. Hypotension secondary to cardiogenic shock has also been reported. Urgent surgery is the recommended approach for such patients given the high mortality rate of this condition [2].

Timeline

Emergency department	Chest Pain + ST depressions II-III-AVF, V4-V6, ST elevation in aVR + positive troponins, Cath Lab Activation
Cath lab	-No evidence of coronary arterial disease -Ventriculogram and subsequent ECHO indicative of severe mitral regurgitation -IABP placement and intubation
Cardiology intensive unit	Patient diuresed and stabilized prior to surgery
Surgery	-Posterior papillary muscle rupture -Mitral valve replacement with bioprosthetic Epic 31 mm
Cardiothoracic surgery intensive care unit	Patient weaned off IABP and inotropes

Case presentation

A 73-year-old woman with a medical history of hypertension presented to the emergency department after 30 min of severe pressure-like substernal chest pain that radiated to her left arm and jaw at rest. The pain began 30 min prior to presentation. Her review of systems was positive for nausea and acute shortness of breath at rest. Pertinent negatives included any history of these symptoms, dyspnea on exertion at baseline, absence of trauma,

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initiation of new medications, exposure to sick contacts, or recent travel.

Patient's past medical history was significant for hypertension, generalized anxiety disorder, major depressive disorder, hypertension, and severe osteoarthritis. Patient was noted to have atypical polymyalgia rheumatica for which she was placed on 6 months taper course of steroids. She has been under the care of rheumatology with autoimmune and inflammatory markers all negative, indicative of osteoarthritis. Patient was on buspirone, duloxetine, hydrochlorothiazide, valsartan and meloxicam as needed. Patient's family history was significant for a father with sudden cardiac death due to dilated cardiomyopathy. No other risk factors or high-risk behaviors were reported.

On physical examination, the patient was normothermic and tachycardic with a rate of 112 beats per minute. Her blood pressure was 88/55 mm of Mercury (mmHg) and peripheral oxygen saturation was 88% on 5 L of supplemental oxygen via nasal cannula. She was in acute distress, diaphoretic, with jugular venous distension to the mid-neck, and bibasilar crackles. Normal S1 and decreased S2 were noted, with a holosystolic murmur radiating to her axilla; well-perfused extremities with +1 lower extremity edema to the knees were also noted.

Initial electrocardiogram was indicative of diffuse ST-segment depressions and ST-segment elevation in lead

aVR (Fig. 1). Initial blood investigations were notable for lactate of 1.7 mg/dl and troponin of 1.33 ng/dL. She was given aspirin, ticagrelor, and heparin infusion in the emergency department. Catheter angiography was negative for coronary artery disease with less than 30% of obstruction of all coronary vessels (Additional file 1 and 2: Videos 1 & 2). Patient's left ventricular end-diastolic pressure was elevated at 31 mmHg and left ventriculography was indicative of severe (4+) mitral regurgitation (MR) with dilated left atrium (Additional file 3: Video 3). Transthoracic echocardiogram (TTE) in the cath lab showed a left ventricular ejection fraction (LVEF) of 65%, severe MR, flail posterior mitral valve leaflet as well as flail head of posterior papillary muscle (Additional file 4: Video 4).

An intra-aortic balloon pump (IABP) was placed in the cardiac catheterization laboratory and patient was admitted to the cardiac intensive unit for diuresis. She was intubated in the cath lab due to severe acute hypoxemic respiratory failure. She improved with diuresis and hemodynamic support. She was taken for operative repair on hospital day 3. Intraoperative transesophageal echocardiogram (TEE) revealed severe eccentric MR (Additional file 5: Video 5). After standard initiation of cardiopulmonary bypass and cardiac arrest, direct visual inspection revealed a hemorrhagic posteromedial papillary muscle. The decision was made

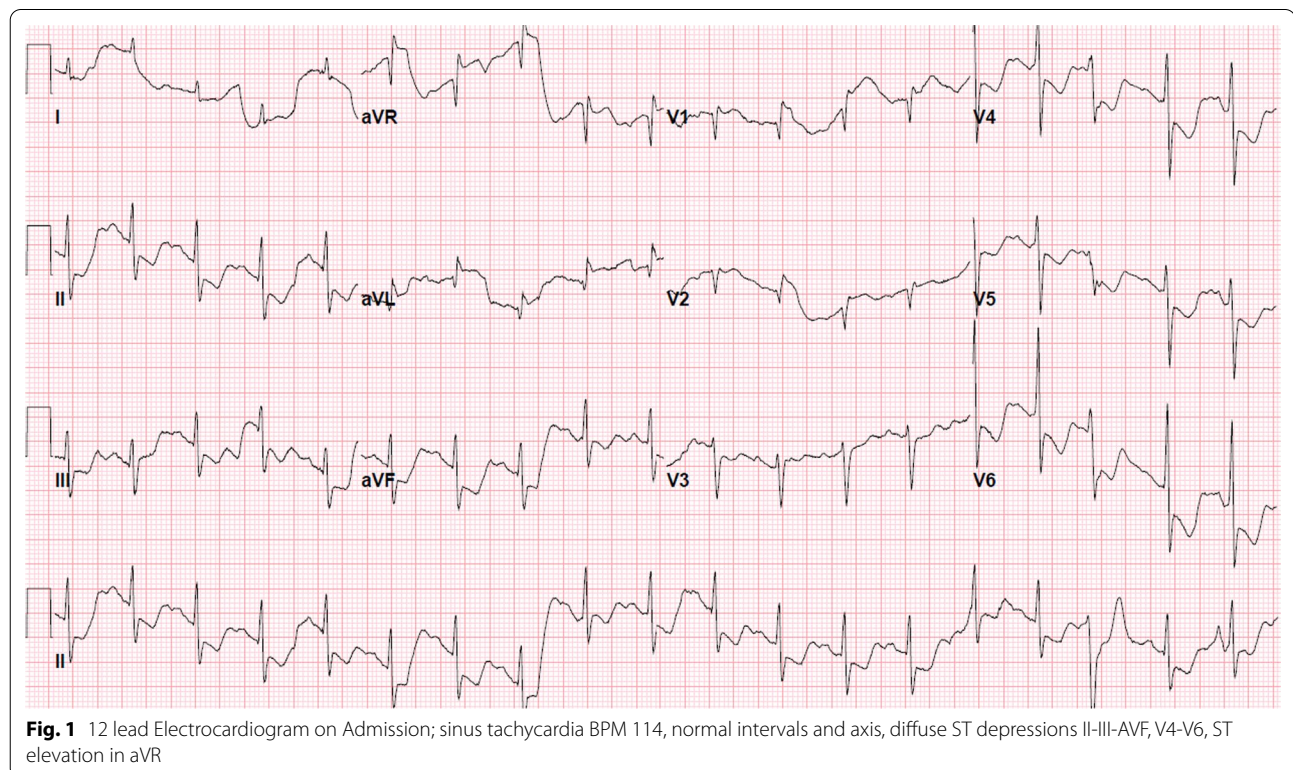


Fig. 1 12 lead Electrocardiogram on Admission; sinus tachycardia BPM 114, normal intervals and axis, diffuse ST depressions II-III-aVF, V4-V6, ST elevation in aVR

to replace the irreparably injured valve, and a 31 mm Epic™ bioprosthetic valve was placed. Postoperative TEE showed no evidence of residual MR and mean gradient of 2 mmHg (Additional file 6: Video 6). Pathologic specimen revealed ruptured posterior papillary muscle head, without any vegetations, calcifications, perforations; chordae tendinae appeared normal (Fig. 2). Microscopic pathologic evaluation revealed ruptured papillary muscle with myocyte necrosis and hemorrhage secondary to the rupture (Figs. 3a–d).



Fig. 2 Macroscopic specimen figure, ruptured papillary muscle head along with posteromedial mitral valve leaflet

Patient was diagnosed with acute severe mitral valve regurgitation secondary to posterior papillary muscle rupture (PMR). Given patient's presentation consistent with acute myocardial ischemia, myocardial infarction (MI) complicated by PMR was higher in our differential. Nonetheless, coronary angiogram showed no evidence of coronary artery disease. Coronary spasm, myocardial infarction without coronary artery occlusion (MINOCA) or acute coronary artery thrombosis followed by spontaneous recanalization could not be ruled out at this point. The patient's unstable hemodynamic status did not allow for a magnetic resonance imaging (MRI) prior to surgery. A diagnosis of idiopathic papillary muscle rupture leading to severe acute mitral regurgitation was made.

The patient had a prolonged postoperative hospitalization complicated by respiratory failure and ventricular cataplexy requiring prolonged support with IABP and milrinone. Cardiac output and cardiac index did improve to normal, she was weaned off all support and was discharged from the hospital. There was no MR or decreased LVEF on follow up transthoracic echo.

Discussion and conclusion

We present a rare case of idiopathic papillary muscle rupture causing severe MR requiring urgent surgical correction with good outcome. Spontaneous PMR in the absence of coronary artery stenosis is rarely reported in the literature [3–5], however, it has been associated with endocarditis, blunt chest trauma, Takotsubo cardiomyopathy, and acute pancreatitis [6, 7]. Coronary arterial

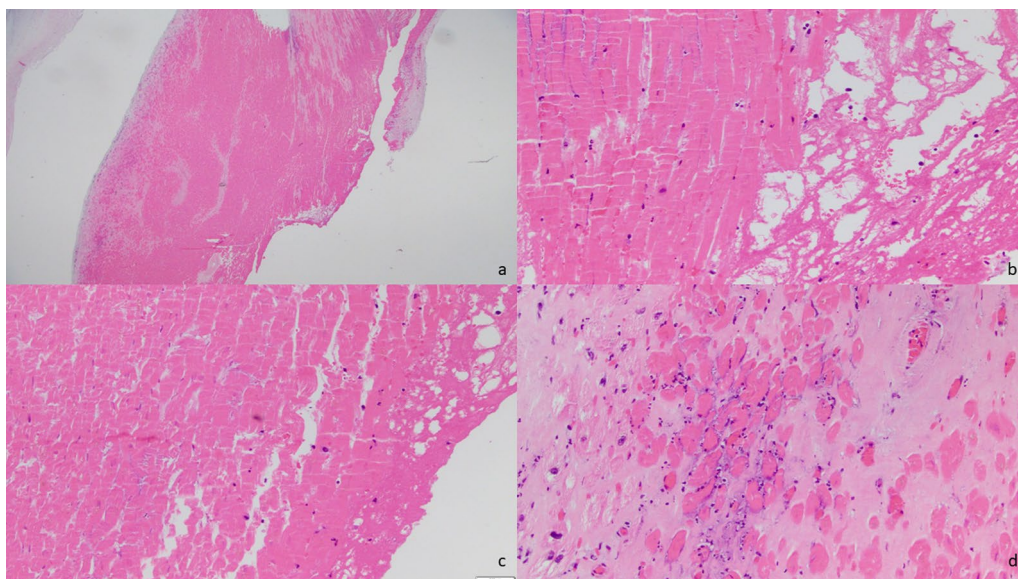


Fig. 3 **a** Low power image (1.25x) showing the resected papillary muscle. **b, c** Higher power images show eosinophilic myocytes consistent with myocyte necrosis, and adjacent hemorrhage. **d** A higher power image demonstrating myocyte necrosis with associated neutrophilic infiltration

spasm manifesting as ischemia has also been reported with PMR in a single case report [8]. Nonetheless, myocardial ischemia is the most common mechanism of reported PMR. The papillary muscles are highly vulnerable to damage induced by hypoxia or ischemia, and the posterior papillary muscle is most commonly affected given its typically poorly-collateralized arterial supply by branches of right posterior descending or right posterolateral artery or the left coronary obtuse marginal arteries [9]. The coronary angiogram of our patient did not demonstrate any vessel obstruction or evidence of coronary artery disease. Myocardial infarction without coronary artery occlusion or chronic microvascular ischemia could be plausible etiologies for our patient as demonstrated in other case reports, albeit unlikely given the lack of chronic ischemic symptoms in our patient's history [3]. The vulnerability of the papillary muscle is emphasized by the fact that recent or old papillary muscle infarction were detected in 25% of autopsy cases [10]. Cardiac MRI is a useful tool in differentiating ischemic tissue from other processes; unfortunately the acuity of our case did not allow for this study.

Initial medical management of PMR includes diuretics, and oxygenation delivered with non-invasive and invasive mechanical ventilation. Mechanical support with IABP should be considered in patients presenting with cardiogenic shock [2]. Nevertheless, prompt diagnosis and early surgical management are the cornerstones of treatment. Mortality rates stemming of retrospective surgical cohorts in the past 2 decades are between 25 to 30% (30 day post-operatively) [11]. Current data on catheter-based edge-to-edge techniques for this clinical entity are not robust, with few case reports in the literature [11].

Acute mitral regurgitation as a complication of myocardial infarction has a very poor prognosis if left untreated. We present a rare case of idiopathic papillary muscle rupture causing severe MR requiring urgent surgical correction with good outcome.

Abbreviations

MI: Myocardial infarction; PMR: Papillary muscle rupture; MINOCA: Myocardial infarction without coronary artery occlusion; MR: Mitral regurgitation; TTE: Transthoracic echocardiogram.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12872-022-02570-4>.

Additional file 1: Video 1. RAO CRAN view of Right Coronary Artery; Less than 30% occlusion

Additional file 2: Video 2. RAO Caudal view of Left Main, Circumflex and Anterior Descending Arteries; less than 30% occlusion in all territories

Additional file 3: Video 3. Ventriculogram of left ventricle showing +4 regurgitation jet in a severely dilated left atrium

Additional file 4: Video 4. Posterior Long Axis View, Transthoracic ECHO; Flail posteromedial mitral valve cusp, eccentric regurgitant jet, dilated left atrium

Additional file 5: Video 5. Transesophageal ECHO, midesophageal 3 chamber view; dilated LA, eccentric regurgitant jet, vena contracta 0.6 cm

Additional file 6: Video 6. Intraoperative Transesophageal ECHO Post Mitral Valve Replacement, midesophageal 4 chamber view; 31 mm Epic bioprosthetic valve, no regurgitation

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None.

Authors' contributions

IM, MJ and HH analyzed the data and wrote the manuscript. ME performed the histological examination. SF and MP analyzed the data and provided guidance to the rest of the authors. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Written informed consent was obtained from the patient for publication of this Case report and any accompanying images or videos. A copy of the written consent is available for review by the editor of this journal.

Competing interests

No competing interests applies to this study.

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