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CASE REPORT

BEGINNER

HEART CARE TEAM/MULTIDISCIPLINARY TEAM LIVE

Papillary Muscle Rupture Complicating Acute Myocardial Infarction

A Tale of Teamwork

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ABSTRACT

A 52-year-old man presented with acute onset of chest pain and was found to have an inferolateral ST-segment elevation myocardial infarction and acute mitral regurgitation due to papillary muscle rupture. This case describes a rare, potentially fatal mechanical complication of acute myocardial infarction. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2020;2:2283–8) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 52-year-old man with a past medical history of obesity, hypertension, and pre-diabetes presented with sudden onset of chest pain for 1 h and was found to be in sustained ventricular tachycardia (Figure 1A). Blood pressure was 99/62 mm Hg, and physical examination was notable for a patient in moderate distress, with no appreciable cardiac murmur. Following administration of amiodarone, he converted to atrial fibrillation, revealing inferolateral ST-segment elevations and inferior Q waves (Figure 1B).

LEARNING OBJECTIVES

- To recognize the pathophysiology of PMR.
- To understand the breadth of clinical manifestations of PMR, including in physical examination, cardiac rhythm, hemodynamics, and imaging.
- To understand the utility of a multidisciplinary approach in the prompt diagnosis and management of PMR in order to decrease mortality.

QUESTION 1: WHAT IS THE DIFFERENTIAL DIAGNOSIS, AND WHAT FURTHER INFORMATION CAN BE DERIVED FROM THE PATIENT'S PRESENTATION WITH VENTRICULAR TACHYCARDIA?

Answer 1: The differential diagnosis includes ST-segment elevation myocardial infarction, primary ventricular tachycardia with secondary transmural ischemia, viral myocarditis, and pericarditis. The patient underwent emergent coronary angiography, which revealed a chronic total occlusion (CTO) of

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ABBREVIATIONS AND ACRONYMS

AMI = acute myocardial infarction

CTO = chronic total occlusion

LCx = left circumflex

MR = mitral regurgitation

PCWP = pulmonary capillary wedge pressure

PMR = papillary muscle rupture

RCA = right coronary artery

TEE = transesophageal echocardiogram

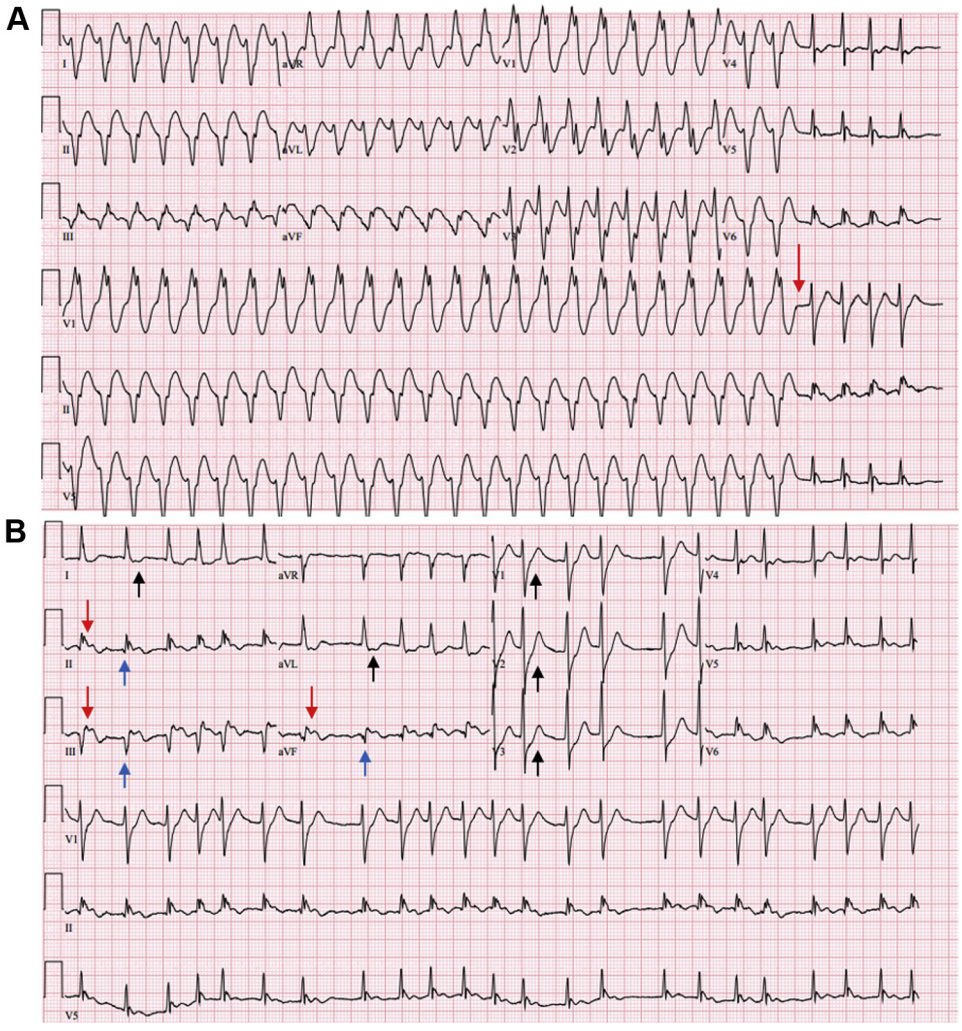
TTE = transthoracic echocardiogram

the proximal right coronary artery (RCA), with left-to-right collaterals, and a culprit subtotal thrombotic occlusion of the mid left circumflex (LCx) coronary artery (Figures 2A to 2C).

Our patient's presenting rhythm of ventricular tachycardia initially obscured the recognition of underlying ST-segment elevation. However, ventricular tachycardia is not commonly associated with ischemia, and is more likely related to underlying scar. The superior axis of the patient's ventricular tachycardia and inferior Q waves in atrial fibrillation indicate preexisting inferior wall scar substrate, ultimately explained by an RCA CTO noted on angiography. Notably, atrial fibrillation is also not a typical ischemic rhythm, but may suggest markedly increased left atrial loading conditions in the context of acute myocardial infarction (AMI) or mitral regurgitation (MR) (1).

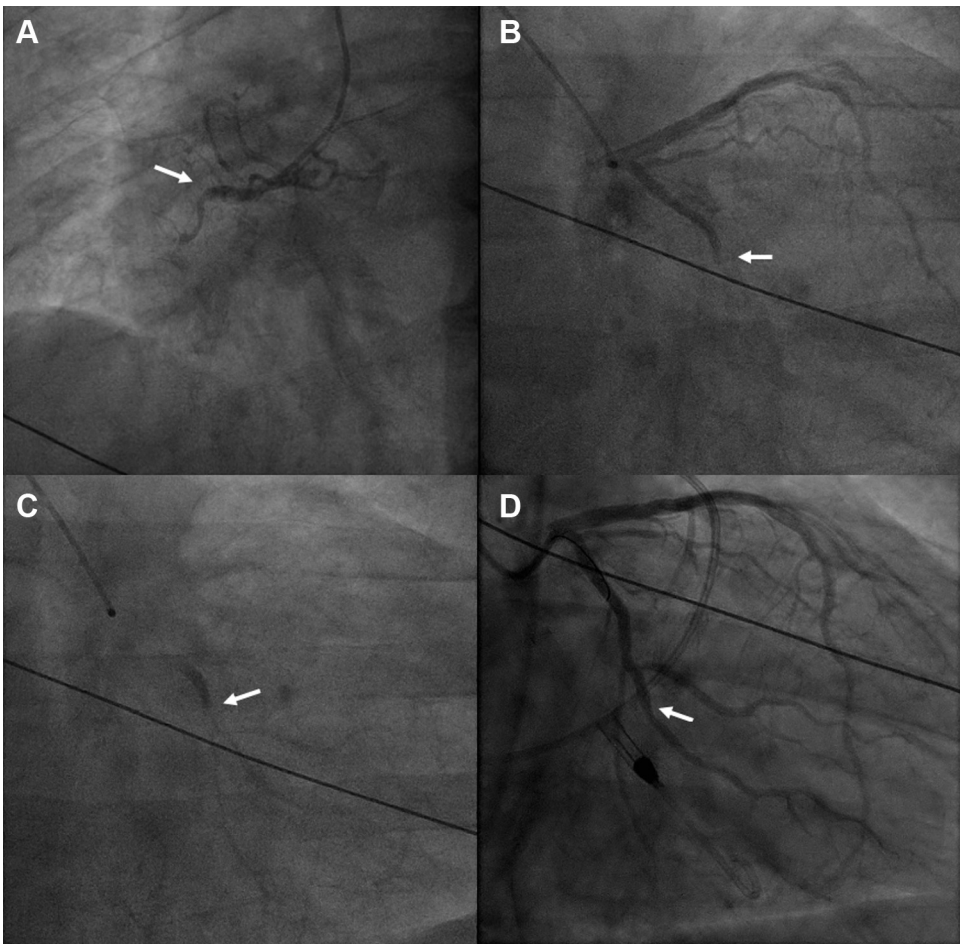
After coronary angiography, the patient developed increasing respiratory distress and worsening hypotension. Therefore, right heart catheterization was performed, demonstrating a right atrial pressure of 17 mm Hg, pulmonary capillary wedge pressure (PCWP) of 25 mm Hg with V waves to 55 mm Hg (Figure 3), cardiac index of 1.2 l/min/m^2, and cardiac output of 3.1 l/min. Concomitant with these findings, the patient's respiratory distress increased.

FIGURE 1 Electrocardiograms



(A) Monomorphic ventricular tachycardia, positive in V_1 and with a superior axis, consistent with left ventricular inferior wall exit. Conversion to atrial fibrillation can be noted **(red arrow)**. **(B)** Atrial fibrillation with inferior ST-segment elevations **(red arrows)**, anterolateral upsloping ST-segment depressions **(black arrows)**, and inferior Q waves **(blue arrows)**.

FIGURE 2 Coronary Angiogram



(A) Chronic total occlusion of the right coronary artery **(arrow)**. **(B)** Subtotal occlusion of the mid left circumflex artery **(arrow)**. **(C)** "Contrast staining" in the left circumflex artery **(arrow)**, indicating acute thrombus. **(D)** Following intervention, there was no residual stenosis of the left circumflex artery **(arrow)**.

QUESTION 2: GIVEN THESE CLINICAL, ANGIOGRAPHIC, AND HEMODYNAMIC FINDINGS, WHAT ARE THE APPROPRIATE NEXT STEPS?

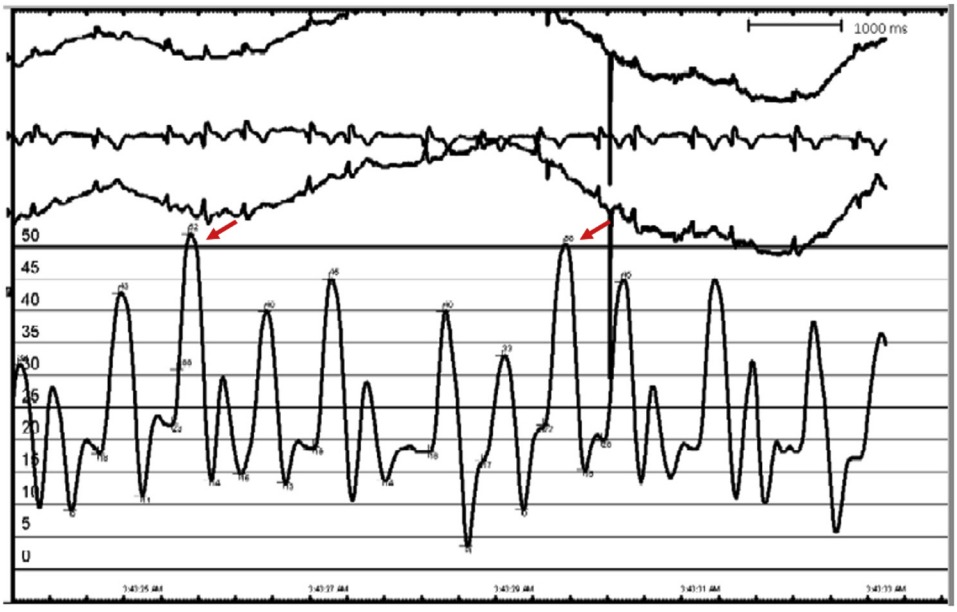
Answer 2: Given hemodynamic findings of cardiogenic shock and respiratory distress, mechanical support and stabilization of the patient's respiratory status before percutaneous coronary intervention is indicated. An Impella-CP (Abiomed Inc., Danvers, Massachusetts) was placed for mechanical support, and the patient was intubated.

With mechanical support unloading the left ventricle, the patient's respiratory status improved. Percutaneous revascularization and stenting of the LCx was then performed with return of TIMI 3 flow (**Figure 2D**). Transthoracic echocardiogram (TTE) demonstrated a left ventricular ejection fraction of 30% with akinesis of the basal to mid inferior and inferolateral walls, and a possible flail posterior mitral valve leaflet with MR, concerning for papillary muscle rupture (PMR) (Video 1A and 1B).

QUESTION 3: WHAT IS THE SIGNIFICANCE OF THE HEMODYNAMIC FINDINGS NOTED ON RIGHT HEART CATHETERIZATION?

Answer 3: Before intubation, the patient's right heart catheterization findings demonstrated cardiogenic shock in addition to elevated PCWP with large V waves, which are classically associated with severe MR (2). With intubation, mean arterial blood pressure immediately rose from 75 mm Hg to 150 mm Hg. The patient developed refractory

FIGURE 3 Pulmonary Artery Catheter Wedge Tracing



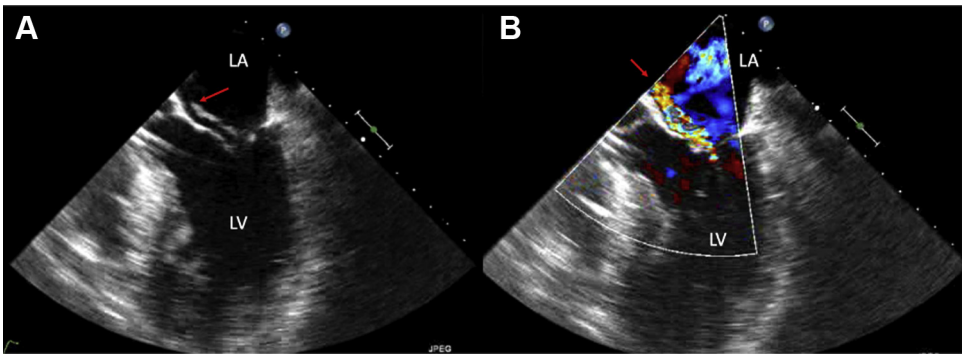
Pulmonary capillary wedge pressure of 25 mm Hg with V waves **(red arrows)** to >50 mm Hg.

hypoxia in the setting of acute increase in blood pressure. In this scenario, increased ventricular pressure reflects backward through an acutely incompetent mitral valve into an unaccommodating left atrium, and ultimately into the vasculature of the lungs, causing more pulmonary vascular congestion than would be expected from a similar jet in chronic MR (3). Diminishing the retrograde pressure load with systemic blood pressure reduction improved his pulmonary edema.

QUESTION 4: WHICH PHYSICAL EXAMINATION FINDINGS WOULD BE EXPECTED IN PMR, AND HOW MIGHT THESE FINDINGS BE EXPLAINED BY UNDERLYING PHYSIOLOGY?

Answer 4: In patients with PMR, physical examination and hemodynamic findings are normally consistent with that of severe MR, and may include a pansystolic murmur loudest at the apex, jugular

FIGURE 4 Transesophageal Echocardiogram



(A) Transesophageal echocardiogram demonstrates the tip of the papillary muscle **(red arrow)** prolapsing into the left atrium. **(B)** Color Doppler over the mitral valve shows anteriorly directed mitral regurgitation **(red arrow)**.

venous distention, and respiratory distress. Invasive hemodynamics may often demonstrate elevated pulmonary arterial pressure and PCWP, with large V waves (4).

Interestingly, our patient did not have an appreciable murmur on presentation, a notable absence in someone later found to have severe MR. In the context of PMR with "wide open" MR, the absence of a murmur may belie equalization of pressures between the left atrium and left ventricle, with decreased regurgitant volume through the valve in the setting of cardiogenic shock. This has been termed "silent MR" (5,6) and highlights the importance of maintaining a high index of suspicion and understanding various physiologic presentations of PMR.

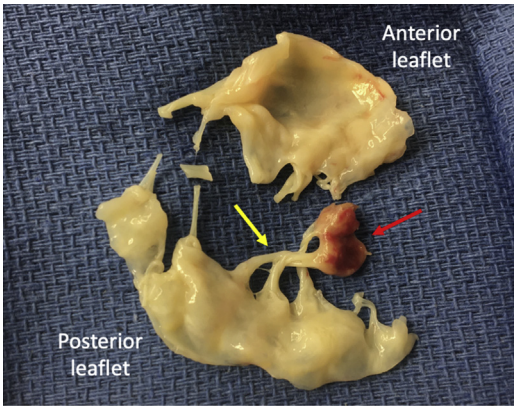
QUESTION 5: UNDER WHICH CIRCUMSTANCES IS PMR MOST LIKELY TO OCCUR?

Answer 5: Acute MR secondary to PMR is a rare, but potentially fatal, mechanical complication of AMI. Although the incidence of PMR has decreased significantly from 1% to 5% to <0.03% in the era of primary percutaneous coronary intervention, the mortality rate remains high (7, 8). PMR results in acute, severe MR, which leads to flash pulmonary edema, hypotension, and cardiogenic shock often requiring mechanical support (7). PMR more commonly involves the posteromedial papillary muscle, given its solitary blood supply from the posterior descending artery. Rupture of the anterolateral papillary muscle is less common due to its dual blood supply from the left anterior descending artery and the LCx (2). Therefore, PMR is most common in the setting of infarcts involving the patient's dominant coronary artery. Although our patient had a right dominant circulation, his CTO of the RCA with left-to-right collaterals resulted in de facto left-sided dominance. Acute occlusion of the LCx led to ischemia of the RCA territory, including the posteromedial papillary muscle. In addition, the near simultaneous presentation of AMI and PMR is unusual, as PMR usually occurs several days after AMI (2,3). The preexisting CTO of the RCA and inferior scar may have compromised the tensile strength of the papillary muscle and contributed to its early rupture.

QUESTION 6: WHAT ADDITIONAL INVESTIGATIONS ARE NEEDED TO COMPLETE THE DIAGNOSIS OF PMR?

Answer 6: Although hemodynamic findings can make the diagnosis of severe MR, structural diagnosis of PMR must be made with echocardiogram. Here, TTE demonstrated a probable flail posterior mitral valve leaflet with severe anteriorly directed MR, for which PMR was the most worrisome diagnosis. TTE is often limited in critically ill patients, and it is only with transesophageal echocardiogram (TEE) that one can adequately visualize the ruptured papillary muscle head prolapsing into the left atrium erratically, independent of ventricular wall motion (9). In this case, subsequent TEE confirmed the diagnosis of posteromedial PMR with severe, anteriorly directed MR (Video 2, **Figures 4A and 4B**).

FIGURE 5 Surgical Specimen



Posterior mitral valve leaflet with ruptured tip of the posteromedial papillary muscle **(red arrow)** and intact chordae **(yellow arrow)**.

QUESTION 7: WHAT IS THE MANAGEMENT OF PMR?

Answer 7: Once the diagnosis of PMR is confirmed, the cornerstone of treatment is early surgical intervention. With medical treatment alone, in-hospital mortality may be close to 80% (10). The mortality with surgical intervention is lower but has been described as high as 55% (7). Therefore, early diagnosis and urgent surgical management are paramount in reducing mortality.

Following TEE, the patient was taken for emergent mitral valve surgery. Intraoperatively, the ruptured posteromedial papillary muscle, which caused prolapse of the medial segment of the posterior mitral valve leaflet (P2), was identified (**Figure 5**). His native valve was replaced with a 33-mm St. Jude mechanical valve, as well as undergoing coronary bypass of his chronically occluded RCA using a saphenous vein. Postoperatively, the patient was

liberated from Impella-CP, but required inotropic support for several days. The patient was extubated on postoperative day 7. On repeat TTE, his mechanical mitral valve was functioning normally, and there was no residual MR. Given his myocardial scar and ventricular tachycardia, a secondary prevention cardiac defibrillator was placed, and he was ultimately discharged on postoperative day 19 on guideline-directed medical therapy for heart failure.

PERSPECTIVES

Although mechanical complications of AMI such as PMR are well described in the literature, they are extremely rare. As the pathophysiology of PMR may result in varied physical examination, cardiac rhythm, hemodynamic, and imaging findings, it is necessary to have an understanding of the breadth of presentations and use a multidisciplinary approach to ensure timely diagnosis and management.

AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS mitral valve, myocardial infarction, papillary muscles

APPENDIX For supplemental videos, please see the online version of this paper.

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