

## Case Report

# The great masquerader: Papillary muscle rupture, a diagnosis forgotten?

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**Acute papillary muscle rupture (PMR) often results in acute, torrential mitral regurgitation and florid pulmonary edema. This catastrophic consequence of a subacute presentation of myocardial infarction is associated with a high mortality rate if not identified quickly. The incidence of PMR has decreased in the era of early percutaneous interventions for acute coronary syndromes. However, the importance of prompt recognition and intervention for this condition is paramount. We present a classic case of PMR and the associated hemodynamics, diagnosis and therapeutic management. This case serves as a guide in unmasking this fatal condition.**

**Key words:** Papillary muscle rupture (PMR), diagnosis, myocardial infarction, pulmonary edema.

## INTRODUCTION

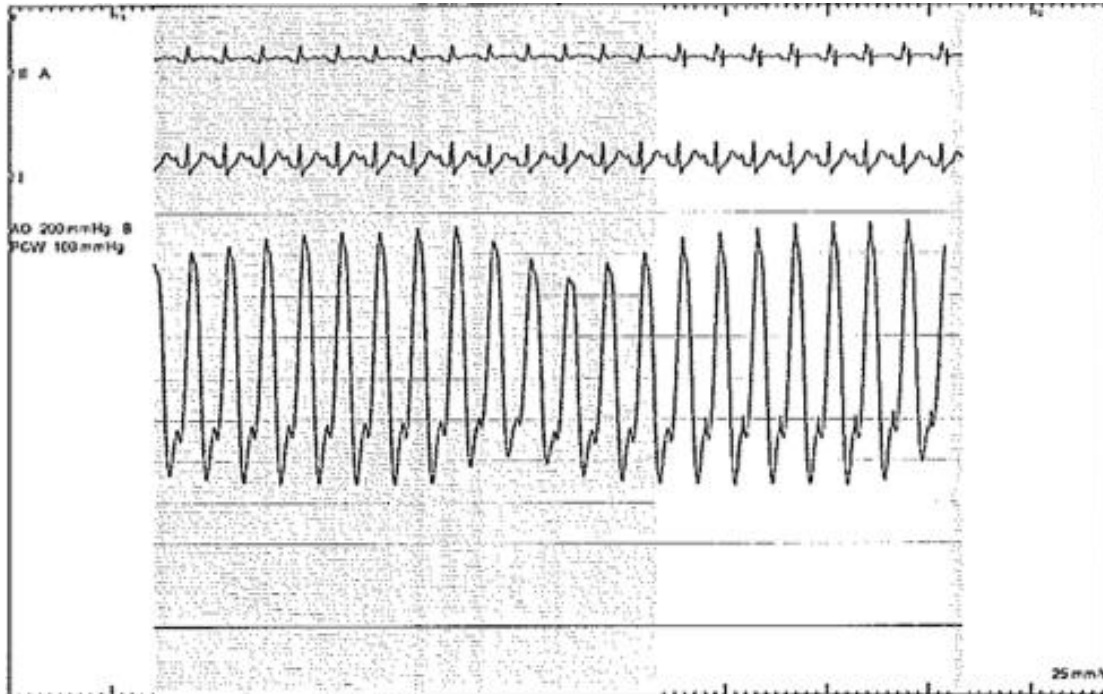
Acute myocardial infarction (AMI) is an entity, which must be diagnosed and treated with seasoned clinical skill as the measure of morbidity and mortality are a race against the clock. Associated with this diagnosis are multiple life threatening complications, all of which may be overlooked even in the hands of the most experienced physicians. Acute organic mitral regurgitation (MR), secondary to papillary muscle rupture (PMR) is seen in 1 to 5% of cases of AMI (Kishon et al., 1992). Myocardial infarction is frequently associated with MR, and it is estimated that 20 to 30% of patients with coronary artery disease also have associated MR. PMR may result in acute pulmonary edema and/or cardiogenic shock. PMR can often be elusive and a challenge to diagnose. If PMR is not treated as a surgical emergency, the mortality rate that would be reported will be as high as 80% in the first 24 h even in the best hands (Kishon et al., 1992).

## CASE REPORT

A 51-year-old male presented to a small community hospital with a chief complaint of left substernal chest discomfort. He stated he has

been coughing and breathless for the past three days. Upon arrival, he was diaphoretic, pale and unable to complete sentences due to severe dyspnea at rest. He was hypotensive and tachycardic. The physical examination revealed elevated jugular venous distension, marked rales on pulmonary auscultation, and tachycardic heart rate with no additional sounds. A 12 lead electrocardiogram suggested an acute inferior myocardial infarction. A chest X-ray revealed pulmonary vascular congestion consistent with pulmonary edema. He was then transferred to a tertiary care hospital for emergent cardiac catheterization for a suspected ST elevation myocardial infarction (STEMI). Upon arrival, the patient was unable to lay flat during the procedure. Subsequently, he was intubated requiring mechanical ventilation secondary to acute respiratory failure. Coronary angiography was performed revealing severe three-vessel coronary artery disease. The left main coronary artery was without significant disease. There was a 90% lesion in the mid left anterior descending artery, a 90% lesion in the right coronary artery, and the left circumflex artery gave rise to one small marginal branch which was 100% occluded. A left ventricular angiogram was performed revealing severe mitral regurgitation with retrograde filling of the pulmonary veins. A right heart catheterization was then performed and it revealed a mean right atrial pressure of 12 mmHg, right ventricular pressure of 80/20 mmHg, pulmonary artery pressure of 80/40 mmHg, and a mean pulmonary capillary wedge pressure of 60 mmHg. The "a" wave was 49 mmHg while "giant v-waves" were identified measuring 95 mmHg (Figure 1). The systemic level "v" waves suggested severe, acute mitral regurgitation. An intra-aortic balloon pump was placed and the patient was further evaluated with transesophageal echocardiography. A transesophageal echocardiogram confirmed severe mitral

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**Figure 1.** “Giant V waves” illustrated on pulmonary capillary wedge tracing.

regurgitation with a ruptured posteromedial papillary muscle and a flail anterior leaflet. The patient was taken to the operating room within 10 h of presentation. He underwent a mitral valve replacement with a #29 St. Jude bioprosthetic valve and three vessel bypass grafting. A bioprosthesis was chosen in this patient secondary to noncompliance. The patient was constantly relocating secondary to his occupation and had no physician that he followed up with consistently. The risk of inadequate anticoagulation appeared to be greater than the potential for repeat valve replacement in the future. Postoperative pathology confirmed a ruptured posteromedial papillary muscle head attached to chordae (Figure 2). The patient recovered successfully in the cardiovascular intensive care unit and was discharged after two weeks in stable condition.

## DISCUSSION

The first documented case of PMR was reported in 1948, subsequently, Austen et al. (1957) reported the first successful mitral valve repair (MVR) performed in 1965. Reports have shown that the short-term mortality rate of patients post-operatively following MVR due to PMR is 19 to 27%. However, without surgical intervention, the mortality rate can be as high as 80% within the first 24 h with only 6% surviving more than two months (Kishon et al., 1992; Chen et al., 2002; Tavakoli et al., 2002).

The mitral valve is a complex structure with function that is assisted by the anterolateral and posteromedial papillary muscles. The arterial blood supply to the papillary muscles consists of small coronary vessels, which arise from the large epicardial arteries. The anterolateral papillary muscle originates from the

anterolateral wall of the left ventricle and provides chordae to the anterolateral half of both mitral leaflets. It has a dual blood supply from the left anterior descending and the diagonal or a marginal branch of the left circumflex artery. Large anterolateral infarcts can result in anterolateral PMR; however, this is rare secondary to the redundant blood supply (Tauke and Eysmann, 1997; Kalra et al., 2000). The posteromedial papillary muscle has a single blood supply from the posterior descending artery. The posterior descending artery arises from the right coronary artery in majority of the population; however, there is a subgroup in which the posterior descending artery arises from the left circumflex artery. Due to the fact that the posteromedial papillary muscle has a single blood supply, it is involved in PMR 6 to 12 times more frequently as compared to the anterolateral papillary muscle (Austen et al., 1965; Masuda et al., 1997).

Acute organic MR can be disguised as a great masquerader as patients often present with shock following AMI. The typical holosystolic murmur of MR is frequently absent due to the torrential reversal of flow into the left atrium during ventricular systole. The hemodynamics may reveal “giant v waves” on the pulmonary wedge tracings. The sudden increase in left atrial pressure leads to pulmonary edema and at times cardiogenic shock. The electrocardiogram may not show the typical findings of STEMI, but rather nonspecific ST/T wave abnormalities. The chest radiograph often reveals pulmonary edema without cardiomegaly.

Management of acute organic mitral regurgitation secondary to PMR is a critical issue that requires rapid



**Figure 2.** Mitral valve leaflet and chordae attached to ruptured posteromedial papillary muscle head.

diagnosis and treatment. Surgical repair or replacement of the mitral valve and coronary artery bypass grafting has been the therapy of choice to decrease mortality. Medical therapy alone has an 80% mortality rate, while rapid surgical intervention can reduce this to <20% (Kishon et al., 1992; Chen et al., 2002; Tavakoli et al., 2002). Data regarding this clinical situation is limited to case studies as a randomized controlled trial is not feasible. However, this has been the accepted modality by the ACC/AHA guidelines. Support with an intra-aortic balloon pump (IABP) is often initiated if the diagnosis is made in the Catheterization laboratory. It is suggested that hemodynamic support be maintained for at least 24 h after surgical treatment (Stout and Verrier, 2009). The addition of left ventricular assist devices such as the Impella 2.5 give operators another option. However, the data for using this device for acute mitral regurgitation secondary to PMR is limited.

The incidence of PMR in the setting of AMI has markedly reduced in the era of early percutaneous interventions. Physicians must maintain an awareness of this structural complication on late presenting acute coronary syndromes to make rapid diagnosis of this catastrophic condition. Recognizing the hemodynamics and management strategies of acute mitral regurgitation can be life saving (DeBusk et al., 1970). Thus, we present a classic case of PMR with diagnostic right and left heart catheterization hemodynamics as well as transesophageal echocardiography. We also illustrate the effectiveness of rapid IABP insertion and surgical intervention.

## REFERENCES

- Austen WG, Sanders CA, Averill JH, Friedlich AL (1965). Ruptured papillary muscle. Report of a case with successful mitral valve replacement. *Circulation* 32:597-601.
- Chen Q, Rlymple-Hay MJ, Alexiou C, Ohri SK, Haw MP, Livesey SA, Monro JL (2002). Mitral valve surgery for acute papillary muscle rupture following myocardial infarction. *J. Heart Valve Dis.* 11(1): 27-31.
- DeBusk JF, Kleiger RE, Ebnoter CL, Daily PO, Herrison DC (1970). Successful early operation for papillary muscle rupture. *Chest* 58(2).
- Kishon Y, Oh JK, Schaff HV, Mullany CJ, Tajik AJ, Gersh BJ (1992). Mitral valve operation in post infarction rupture of a papillary muscle: immediate results and long-term follow-up of 22 patients. *Mayo Clin. Proc.* 67(11):1023-1030.
- Masuda Z, Hata T, Tsushima M, Matsumoto M, Uchita S, Hamanaka S (1997). A surgical case of total anterior papillary muscle rupture after acute myocardial infarction. *Nihon Kyobu Geka Gakkai Zasshi* 45(6):869-873.
- Sanders RJ, Neuburger KT, Ravin A (1957). Rupture of papillary muscles: occurrence of rupture of the posterior muscle in posterior myocardial infarction. *Chest* 31(3):316-323.
- Stout KK, Verrier ED (2009). *Valvular Heart Disease: Changing Concepts in Disease Management. Acute Valvular Regurgitation.* *Circulation.* 119:3232-3241.
- Tavakoli R, Weber A, Vogt P, Brunner HP, Pretre R, Turina M (2002). Surgical management of acute mitral valve regurgitation due to post-infarction papillary muscle rupture. *J. Heart Valve Dis.* 11(1):20-25.
- Tauke JT, Eysmann SB (1997). Ruptured papillary muscle. *Circulation* 96:698-699.
- Kalra P, Ohri S, Morgan J (2000). Mitral regurgitation secondary to ruptured papillary muscle. *Heart* 84(1):13.