Acute ST-Segment Elevation Myocardial Infarction Complicated by Partial Papillary Muscle Rupture

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Abstract

Acute Mitral Regurgitation (MR) after an acute Myocardial Infarction (MI) can be a catastrophic complication of acute MI and requires early recognition and emergent surgical intervention. The classic presentation is that of acute pulmonary edema and cardiogenic shock with recent history of MI. However, we present a case in which a patient presented with acute myocardial infarction and within a few hours, after percutaneous intervention, developed pulmonary edema and shock; reminding readers that this mechanical complication of MI can present at any time and may present with varying severity of symptoms. A high clinical suspicion should be maintained in order to recognize this serious complication and to expedite definitive surgical and life-saving treatment.

Case Report

A 69 year old Caucasian male presented to the Emergency Department (ED) complaining of chest pain and shortness of breath. Over the preceding week, he had been experiencing intermittent chest discomfort at rest, however 15 hours prior to presentation he experienced acute onset of chest pressure which continued until he presented to the emergency room. Initial vital signs revealed a blood pressure of 119/76 mmHg, respiratory rate of 18 breaths per minute, a pulse of 136 beats per minute and oxygen saturation of 92% on room air. Cardiovascular exam was pertinent for sinus tachycardia without a murmur. Pulmonary exam was notable for bibasilar crackles. A 12-lead Electrocardiogram (ECG) revealed ST-segment elevation in the inferior leads as well as sinus tachycardia (Figure 1). The patient was emergently taken to the catheterization suite for coronary angiography which revealed an occlusion of the mid Right Coronary Artery (RCA) as well as a moderate to severe eccentric plaque in the proximal RCA (Figure 2). The patient received heparin as well as eptifibatide and subsequently three drug eluting stents were deployed to the RCA. TIMI 3 flow was restored. Left ventricular angiography demonstrated severe mitral regurgitation (thought to be functional in nature) and inferior wall hypokinesis (Figure 3). An intra-aortic balloon pump was placed and patient was transferred to the Intensive Care Unit (ICU) for further management. The patient tolerated the procedure well and had no complaints upon transfer to the ICU.

In the ICU, the patient began to decompensate with increasing oxygen requirements and hypotension requiring multiple vasopressors. The patient was electively intubated and a Transesophageal Echocardiogram (TEE) was performed which identified a partial postero- medial papillary muscle rupture, with an unsupported segment of the anterior mitral valve leaflet, and posteriorly directly severe mitral regurgitation (Figures 4A-4C).

The patient was referred for urgent surgical intervention. No further mechanical support was utilized as he was taken to the operating room very promptly after diagnosis. As the anatomy was unsuitable for repair, the patient underwent mitral valve replacement with a bio prosthetic valve. The patient gradually recovered over the next fourteen days and was discharged home in good condition.

Discussion

Acute Papillary Muscle Rupture (PMR) is a rare but serious complication of acute myocardial infarction with a mortality rate of 50% within the first 24 hours in those who do not undergo operative intervention [1,2]. Patient outcome is highly dependent on prompt recognition and referral to surgery for definitive repair. PMR, partial or complete, is a rare etiology of MR, occurring in 1% of patients with transmural myocardial infarction [3]. It typically occurs within a few days of an ischemic event [4]. The postero-medial papillary muscle is approximately 10 times more likely to rupture than the antero-lateral due to the solitary blood supply to the postero-medial head from the posterior descending artery [3,4]. The antero-lateral papillary muscle receives a dual blood supply from the left anterior descending and left circumflex arteries [3]. Both leaflets may be involved serving as a reminder that both the anterior and posterior leaflets have attachments to both papillary muscles [5]. In our patient, the partial tear involved the postero-medial papillary muscle yet the unsupported segment was the anterior leaflet with a posteriorly directed jet.

Risk factors for PMR include an infero-posterior acute MI, single-vessel disease, and absence of diabetes mellitus [4]. The clinical
Presentation of PMR typically presents with acute pulmonary edema and cardiogenic shock. A murmur may not be present due to a large regurgitant orifice and lack of pressure gradient between the left ventricle and atrium [1]. In addition, an initially audible murmur may become less audible as arterial pressure falls [3].

Diagnosis of complete rupture can be made by Tran Thoracic Echocardiogram (TTE) where flail mitral leaflet or papillary muscle head is seen to prolapse into the left atrium [6,7]. This may be technically challenging as the patient is usually tachycardic and in extremis. Partial PMR may be even more difficult to visualize. TEE may be necessary for complete visualization of the anatomic structures.

Initial medical management should be directed to improving hemodynamics (intra-aortic balloon pump, inotropes, and diuresis) [1]. ECMO or Impella for enhanced hemodynamic support may be considered, depending upon the individual patient compromise. Ultimately, the patient will require mitral valve repair or replacement with coronary revascularization therapy [8].

**Conclusions**

Papillary muscle rupture is a rare complication of myocardial infarction and usually presents with acute pulmonary edema and cardiogenic shock. There are many case reports that demonstrate this classic presentation however, our patient illustrates that partial papillary muscle rupture may not initially present with obvious signs of cardiogenic shock or pulmonary edema [5,9]. Therefore, the diagnosis of papillary muscle rupture should be considered at any point in the patient’s course with sudden and unexpected hemodynamic compromise even in the absence of a murmur. Diligent evaluation by TEE, with particular focus on the subvalvular mitral apparatus will provide prompt diagnosis of partial tears in the absence of obvious flail of the mitral leaflets or papillary muscle.
References


