Incidental Finding of Traumatic Papillary Muscle Rupture on Intraoperative Transesophageal Echocardiogram following a Motor Vehicle Accident

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INTRODUCTION

Traumatic papillary muscle rupture is an uncommon cause of acute mitral regurgitation (MR). Here we present imaging of acute severe MR secondary to papillary muscle rupture in a 23-year-old man who presented following a motor vehicle crash.

CASE PRESENTATION

The patient is a 23-year-old man with no known medical history who presented to our institution following a motor vehicle accident. He was driving at an unknown speed when he first crashed into a pole and then subsequently a building. His lower extremities were entrapped, and extrication took approximately 25 minutes. Glasgow Coma Scale was initially 12 but declined to 6; he was intubated at the scene. He was given two units of packed red blood cells and 700 mL isotonic fluid en route as blood was noted in his airway.

Upon arrival to the emergency department, initial focused assessment with sonography in trauma exam demonstrated a lack of lung sliding on the left, and a chest tube was emergently placed with subsequent rush of air, consistent with a tension pneumothorax. The remainder of the primary survey was unremarkable. Upon subsequent examination in the emergency department, there was concern for decreased right-sided breath sounds and a chest tube was placed. Chest x-ray revealed left flail chest and subcutaneous emphysema. The patient was admitted to the surgical intensive care unit upon stabilization. The patient became progressively difficult to oxygenate despite optimized ventilator settings. He was taken for a computed tomography scan of the chest, abdomen, and pelvis, which revealed normal left ventricular (LV) function with an LV ejection fraction of 67%-72% as well as normal right ventricular function. There was noted to be flail P2 and P3 segments of the posterior mitral valve leaflet along with evidence of rupture of the posteromedial papillary muscle (Figures 1 and 2A, Videos 1 and 2). The resulting MR was noted to be severe as evidenced by the size of the color Doppler jet (>50% of the left atrial area; Figure 2B, Videos 3 and 4). Additionally, there was noted to be systolic flow reversal in the pulmonary veins consistent with significant MR (Figure 3A and B). Of note, continuous-wave Doppler revealed that the MR jet was triangular with a rapid decline in late systolic velocity, reflecting the rapid rise in left atrial pressures (Figure 4). Traditional Doppler-based methods for quantification of chronic MR are not reported as these may not be accurate in acute MR. Of note, frothy pink sputum was suctioned from the endotracheal tube during the formal TEE.

Surgery was discussed, although deferred due to development of suspected hemorrhagic pulmonary edema following initiation of prophylactic heparin with bloody sputum seen in the endotracheal tube. The patient’s course was complicated by acute respiratory distress syndrome, acute limb ischemia, abdominal hypertension, acute renal failure, and worsening intraparenchymal hemorrhage, all of which led the patient’s family to transition to comfort measures.

DISCUSSION

Here we describe a case of traumatic papillary muscle rupture resulting in severe mitral valve regurgitation following a motor vehicle crash with blunt chest trauma. Although the primary survey only revealed pneumothorax, progressive difficulty with oxygenation even after placement of a chest tube was a clue to additional pathology, in this case acute MR.

Although acute MR following blunt chest trauma can be seen after motor vehicle accidents, the overall prevalence of acute MR following blunt chest trauma is low. One often cited study demonstrated that of 546 cases of cardiac injury from trauma, damage to the mitral valve was seen in just 0.01%. Cardiac contusion is more commonly seen following this mechanism of injury. Of the cardiac structures, the anterior structures are most vulnerable to injury, particularly the right ventricle and the tricuspid valve. Blunt injury may result in acutely increased intracardiac pressures through sudden compression of the right ventricle (which is situated anteriorly and immediately behind the sternum).

Mitrval valve injury following blunt chest trauma is thought to be secondary to an acute rise in intracardiac pressure, which ultimately
VIDEO HIGHLIGHTS

**Video 1:** Transesophageal echocardiogram midesophageal plane at 0° reveals flail P2 scallop of the mitral valve with rupture of the posteromedial papillary muscle.

**Video 2:** Three-dimensional multiplanar reconstruction of the mitral valve apparatus reveals flail P2 and P3 scallops of the posterior mitral leaflets.

**Video 3:** Transesophageal echocardiogram midesophageal plane at 151°. A2 and P2 scallops of the mitral valve leaflets are imaged in two dimensions. The large color Doppler jet of severe MR is visualized.

**Video 4:** Transesophageal echocardiogram midesophageal plane at 47°. The color Doppler shows the jet of severe MR.

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Figure 1 Transesophageal echocardiogram midesophageal view at 0° demonstrating flail P2 scallop of the mitral valve (*). Arrows indicate the flail gap, measuring 1.8 cm.

Figure 2 Transesophageal echocardiogram midesophageal view at 47°. Panel A reveals the ruptured posteromedial papillary muscle (*), and panel B reveals the accompanying color Doppler jet of severe MR.

Figure 3 (A) Pulsed-wave Doppler signal sampling the left upper pulmonary vein demonstrating systolic flow reversal (*). (B) Pulsed-wave Doppler signal sampling the right lower pulmonary vein, again revealing systolic flow reversal (*).
results in MR through rupture of the papillary muscle, chordae tendineae, or leaflets themselves. The acuity of the rupture and subsequent regurgitation does not allow for left atrial remodeling. The high regurgitant volume and subsequent decrease in stroke volume and cardiac output lead to hypoperfusion and hypotension, and patients generally present in acute congestive heart failure or cardiogenic shock. Even with a hyperdynamic ventricle, the LV cannot acutely compensate for this reversal of flow.

In our patient, the TEE shows acute severe MR with flail P2 and P3 scallops of the posterior mitral valve leaflet with evidence of rupture of the posteromedial papillary muscle. Not only is traumatic MR itself a rare condition, but a review of surgically corrected cases of traumatic MR demonstrates that about half of the cases were due to a ruptured papillary muscle, with only eight of those demonstrating rupture of the posteromedial papillary muscle as seen with our patient.

CONCLUSION

Our case highlights the imaging findings associated with a rare diagnosis of traumatic mitral valve damage due to papillary muscle rupture. It also emphasizes the role of echocardiography in assessing patients presenting with trauma.

REFERENCES