

## Medical and Clinical Case Reports

## Acute Severe Mitral Valve Regurgitation with Posterior Leaflet Torn Post Motor Vehicle Accident: A Case Report and Review

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## ABSTRACT

Cardiac injuries are rare complications after blunt chest trauma, and when encountered, myocardial contusions and aortic valve involvement account for most of these injuries. Nevertheless, acute severe mitral valve regurgitation due to a torn leaflet is an infrequently reported life-threatening type of injury in the literature. Hereby, we present a case of acute severe mitral regurgitation post-blunt chest trauma in MVA, highlighting the ICU role in early detection and prompt pharmacological management to prevent heart failure until surgical correction ensues.

**Keywords**

Mitral Valve, Motor Vehicle Accident, Blunt chest Trauma, Mitral regurgitation, Nitrates, Inotrope.

**Abbreviation list**MVA: Motor vehicle accidents, ICU: Intensive care unit, ER: Emergency department, ATLS: Advanced Trauma Life Support, FiO<sub>2</sub>: Fraction of inspired oxygen, TV: Tidal volume, IR: Interventional radiology, PEEP: Positive end-expiratory pressure.**Introduction**

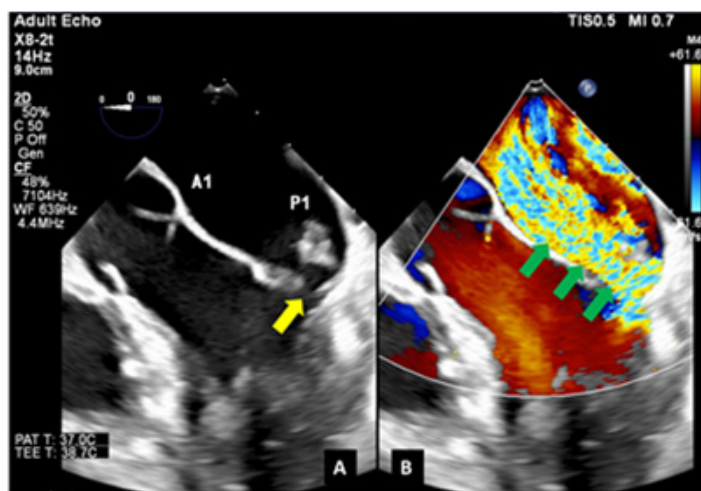
Cardiothoracic blunt trauma following MVA occurs in 30% of civilian trauma cases. In particular, mitral valve injury with regurgitation is infrequently reported in the literature [1]. The mitral valve apparatus traumatic injury could involve papillary muscle rupture or dysfunction, chordae tendineae rupture, or leaflet tears [2]. This report presents an acute severe mitral valve regurgitation due to a torn posterior leaflet, post blunt chest trauma. We will describe the intensive care unit (ICU) approach in managing the critical stages of hemodynamic instability following acute severe traumatic mitral insufficiency and preventing heart failure by pharmacological bridging therapy until surgical correction ensue.

**Case Report**

A 29-year-old male with no medical or surgical history was involved in a high-velocity car collision as a driver. He sustained brain contusion and subgaleal hematoma formation, multiple long bones and hip fractures, hepatic and pancreatic lacerations, and hematoma formation due to active bilateral internal iliac arteries bleeding. He also sustained thoracic injury with multiple rib fractures and tension pneumothorax. The patient was stabilized through the Advanced Trauma Life Support (ATLS) protocol in the emergency department (ER) and then underwent urgent bilateral iliac arteries embolization. The patient was admitted to the ICU intubated, mechanically ventilated on volume-controlled mode and sedated with Propofol, Midazolam, in addition to Fentanyl infusions. He required a fraction of inspired oxygen (FiO<sub>2</sub>) of 100% and positive end-expiratory pressure (PEEP) of 12 mmHg to maintain oxygenation. He was in cardiogenic shock with tachycardia of 145 BPM initially and required infusions of norepinephrine and dobutamine and nitroglycerin.

Cardiovascular system examination demonstrated a pansystolic murmur radiating to the left parasternal area. Electrocardiogram was unremarkable except for sinus tachycardia. Transthoracic echocardiogram showed a tear at the lateral scallop of the posterior

mitral valve leaflet, with the involvement of posteromedial commissure, causing severe mitral regurgitation. Furthermore, high-resolution images using transesophageal echo with mid-transesophageal 2D and 3D views with Doppler were obtained. Figure 1 displays a midesophageal 2D view of the mitral valve with ruptured lateral scallop (P1) of the posterior mitral leaflet (yellow arrow) and a Doppler image showing severe mitral regurgitation (green arrows). Figure 2 displays a transesophageal 3D image of the surgical view of the mitral valve, the ruptured P1 scallop of the posterior mitral leaflet (A), and the ventricular aspect showing the ruptured scallop and capitation gap (B).

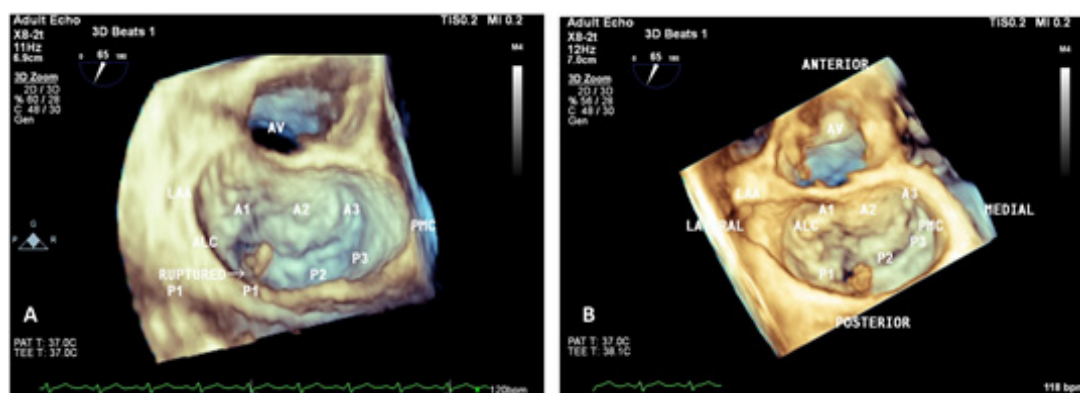


**Figure 1:** Mid-Transesophageal 2D and Doppler images of the mitral valve demonstrating ruptured lateral scallop (P1) of the posterior mitral leaflet (yellow arrow) and Doppler images showing severe mitral regurgitation (green arrows).

Because of the hemodynamic condition, the patient was started on nitroprusside and dobutamine infusions on day 2. The vasodilator effect of Nitroprusside infusion in decreasing pulmonary congestion and regurgitation volume and the Dobutamine's inotropic effect decreased the end-systolic volume, increased stroke volume and improved the overall cardiac function significantly. One of the main challenges with this case was weaning off sedation and ventilation and counteract the increased Systemic vascular resistance (SVR)

consequences on the heart. The ventilator settings were adjusted to reach a  $\text{FiO}_2$  of 40%, along with a PEEP of 10 mmHg. Propofol infusion was switched with dexmedetomidine infusion and remifentanyl infusion. On day 4, respiratory culture was positive for *E. coli*, and blood culture was positive for *Acinetobacter*, for which he was started on Antibiotics. On day ten, nitroprusside infusion stopped, and captopril began at 25 mg TID. The patient started to develop a picture of pulmonary edema on day 15, for which he was started on a low-dose furosemide infusion. On day 16, dobutamine infusion was weaned off, and norepinephrine was running at a rate of 0.03 mcg/kg/min. Another Echo study was done on day 17; Ejection Fraction >55%, dilated LV 60/40 mm compared with 53/38 mm in the previous report, LA was mildly dilated, mitral valve with ruptured lateral scallop (P1) of the posterior mitral leaflet with severe mitral valve regurgitation, no pericardial effusion, no wall motion abnormalities. The patient was off inotropic support on day 22 and had a bedside tracheostomy on day 23. Thereafter, a trial of ATC versus mechanical ventilation with low fio2 of 25% and 21%. The patient did not go into LV failure despite the persistent tachycardia reaching 119 BPM, for which esmolol infusion was started on day 24. Around day 27, he was off mechanical ventilation and on room air. He had agitated delirium, for which he received ketamine and quetiapine. On day 28, his medications were adjusted to captopril 12.5 twice a day, along with metoprolol with a heart rate target of 80 bpm and enoxaparin 40 mg daily. On day 30, he was discharged from ICU for conservative management at the ward, and he was on room air, not in distress, and hemodynamically stable off inotropes and with GCS 15/15 with captopril and metoprolol as anti-heart failure medications. Unfortunately, this patient was not a surgical candidate, as the surgical intervention, including mitral valve clip, IABP or replacement, will accompany a possibility of cardiovascular collapse intra-operatively due to the increased risk of bleeding with heparinization owing to his polytrauma. Therefore, the surgical intervention was deferred to allow time for recovery as the patient is compensating for medical therapy.

After one week in the ward, the patient was discharged on captopril 12.5 mg and metoprolol 50 mg. He was informed that the surgery could be done electively once he is stable, fit, and recovered



**Figure 2:** Transesophageal 3D image of the surgical view of the mitral valve showing the ruptured P1 scallop of the posterior mitral leaflet (A) and ventricular aspect showing the ruptured scallop and capitation gap (B).

from polytrauma, especially since chronic mitral regurgitation can be tolerated for many years. Also, the patient was educated concerning the warning signs and symptoms (congestive heart failure, chest pain) of mitral regurgitation and when to come to ER before symptoms progress. One month after discharge, The patient had follow-up appointments with the cardiac surgery clinic to discuss surgical options which fit his current condition.

## Discussion

Traumatic mitral valve injury is rare, it was first reported in the early 1960s and collected to reach around 82 cases in the literature by 2010; they were analyzed, allowing for a better understanding of the epidemiology, etiology, natural history, pathology, and treatment of this rare condition as It is crucial to delivering the appropriate management plan [6]. The mitral valve apparatus traumatic injury may manifest as papillary muscle rupture or dysfunction, chordae tendineae rupture, or leaflet tears [2]. Mitral valve injury occurs when extreme pressure is applied at early systole during the isovolumic contraction between the mitral valve's closure and the aortic valve's opening [7]. In our case, the patient was the driver and suffered a high-speed car collision, complicated by a tear at the lateral scallop of the posterior mitral valve leaflet, with the involvement of posteromedial commissure, causing severe mitral regurgitation. Investigational studies have shown that an intraventricular pressure greater than 320 mm of Hg is needed to cause any cardiac wall or valve rupture [14]. Severe mitral regurgitation is diagnosed when the regurged blood reaches 50% of the total left ventricular stroke volume [8]. Unfortunately, the diagnosis of such injury can be significantly delayed secondary to the presence of other injuries. The following findings should raise suspicion of this injury: hemodynamic instability, elevated jugular venous pressure, alternating heart tones, pulse, and blood pressures that change dramatically with body position [9]. The clinical picture of patients with mitral valve injury may be none to cardiogenic shock. These patients commonly have tachycardia and new pansystolic murmurs radiating to the left parasternal area. In the reported case, the patient was hemodynamically unstable with tachycardia and hypotension, for which he was on norepinephrine and dobutamine infusions. His cardiac examination revealed a pansystolic murmur radiating to the left parasternal area. No distended neck veins were noticed. It is worth mentioning that traumatic mitral insufficiency, if not detected early and treated correctly, can get complicated and progress to congestive heart failure and cardiogenic shock [12]. Chest X-ray may show cardiomegaly. Electrocardiogram (Echo) frequently reveals sinus tachycardia, as mentioned in the case. The echocardiogram is the primary diagnostic modality of mitral valve injuries. The transthoracic echocardiogram (TTE) has a limited role in polytrauma patients, as there will be a poor window due to dressing, interference from pneumothorax, and inability to give the proper position to the patient. The transesophageal echocardiogram (TEE) has higher resolution sonography and is not affected by the previously mentioned factors [13]. Following the abnormal examination findings in this case, a prompt TTE echo was requested and reported severe mitral regurgitation caused by a tear at the lateral scallop of the posterior mitral valve leaflet. For

which a more detailed TEE was requested to confirm the diagnosis and it revealed a ruptured P1 scallop of the posterior mitral leaflet (A), and the ventricular aspect showing the ruptured scallop and capitation gap (B).

Pharmacological bridging therapy for critically ill patients anticipating surgical intervention has been reported in the literature and followed successfully in many centers worldwide. The mainstay of treatment is preload and afterload reduction, particularly in mitral regurgitation with pulmonary edema as in our case [15]. By starting this patient on nitroprusside and dobutamine infusions, the vasodilator effect of Nitroprusside infusion decreased pulmonary congestion and regurgitation volume; besides, the dobutamine's inotropic effect alleviated the end-systolic volume and, therefore, increased stroke volume and improved the overall cardiac function significantly. Nitrates and diuretics reduce filling pressures, and inotropes and an intra-aortic balloon pump are helpful in the background of hypotension and hemodynamic instability. Even though the American College of Cardiology/American Heart Association Guidelines (2014, 2017) did not include acute mitral regurgitation, they mainly provided guidance for managing chronic primary and secondary MR [11]. Shammass et al. case report of traumatic mitral regurgitation that was treated successfully through medical therapy is worth mentioning as it highlighted the crucial part of the pharmacological management of these patients during the acute stages [16]. Our patient's condition was stabilized medically as he had a normal left ventricular function. His medications were titrated according to his hemodynamic response and eventually to a low dose of captopril and metoprolol orally, as mentioned previously. Unfortunately, he was at risk of bleeding and hemodynamic deterioration if he proceeded to surgical correction due to the high dose of anticoagulation therapy with the associated injuries of multiple bone fractures. However, the definitive treatment of mitral regurgitation remains surgery. Valve repair has been reported in a few cases [10], and valve replacement is the recommended operative procedure. However, when mitral valve replacement is warranted, a choice is made between transcatheter minimally invasive valve replacement and surgical valve replacement with a mechanical or bioprosthetic valve. Repair is usually available only to those whose condition has a nonrheumatic, noninfectious, and non-ischemic cause; as in our case, unfortunately, the hemodynamic and mental instability and the unexpected critical deterioration has highlighted as not a surgical candidate, further follow-ups with cardiac surgery team were arranged upon discharge to discuss the surgical options according to his progression which included mitral clip, or mitral valve replacement.

## Conclusion

Acute severe mitral regurgitation due to posterior leaflet rupture is a rare but potentially fatal complication of blunt chest trauma. Timely diagnosis, early pharmacological and possible mechanical support with IABP and prompt surgical repair of the MV are critical components of management.

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