

## Case Report

# A case report of cardiac tamponade following blunt chest trauma

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### ABSTRACT

Traumatic cardiac tamponade due to blunt chest injury is a life threatening and time-critical emergency that requires early recognition and prompt management often alongside other resuscitative considerations. We present here a case of 25 year old male with history of blunt chest trauma with hypotension and raised central pressures. The patient was taken up for exploratory laparotomy which proved negative and the central tendon bulge of diaphragm was seen. Hence, the decision was made to examine the pericardial space via thoracotomy to find cardiac tamponade of 400 cc. Immediate intra- operative stabilization of vitals was seen on relieving the intra pericardial pressures. Any patient with severe blunt chest trauma, disproportionate hypotension that is not responding to fluid resuscitation along with elevated central venous pressures should be thoroughly evaluated for cardiac tamponade.

**Keywords:** Cardiac tamponade, Blunt chest trauma, Echocardiography

### INTRODUCTION

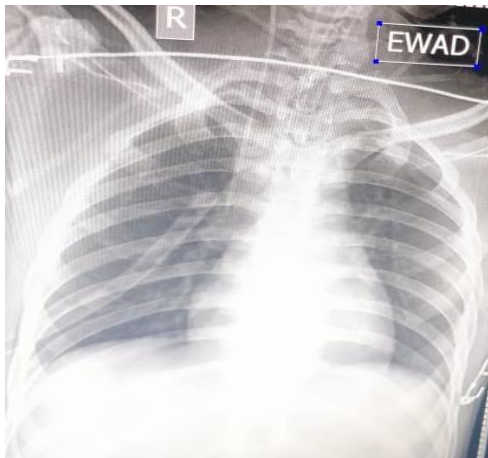
Cardiac tamponade due to blunt chest trauma is a rare entity. Traumatic cardiac tamponade is a life threatening and time-critical emergency that requires early recognition and prompt management often alongside other resuscitative considerations.<sup>1</sup> Structural cardiac injuries including chamber or vessel rupture carry a high mortality rate and patients usually die at scene or in-transit. The small remainders of patients who survive to hospital have tears under relatively low pressures and immediate diagnosis and surgery can help the survival. High grade of clinical suspicion is required. Any patient with severe blunt chest trauma, disproportionate hypotension that is not responding to fluid resuscitation along with elevated central venous pressures should be thoroughly evaluated for cardiac tamponade.

### CASE REPORT

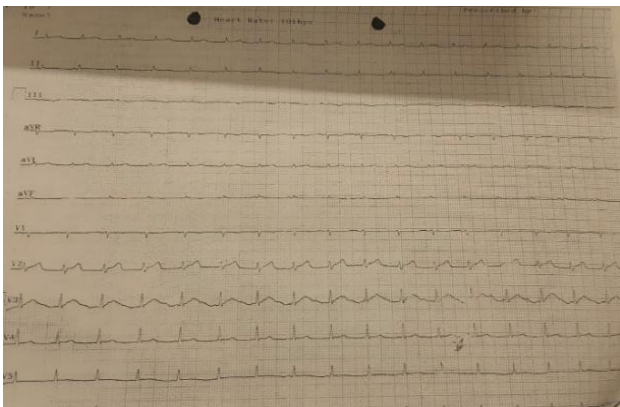
A 25 year old male presented to the emergency trauma ward of LTMGH within one hour of alleged history of fall from a running train. He had sustained blunt chest and abdominal trauma along with head injury. Head injury was associated with loss of consciousness. The patient had tachycardia of 120 beats per minute and hypotension of 70/40 mmHg. Air entry was decreased on right side with palpable rib fractures of 2/3/4<sup>th</sup> rib without subcutaneous emphysema. Heart sounds were normal on auscultation. GCS score of E3M5V4 was observed. Per abdominal palpation revealed tender abdomen without any hematuria. A large friction abrasion was noted on the sternum along with multiple small friction abrasions on the face.

Fluid resuscitation was started immediately after securing two wide bore intravenous lines Inter costal drain was immediately secured on the right side. Chest X-rays were

taken which established the presence of 2, 3, 4<sup>th</sup> rib fractures along with pneumothorax on the right side and confirmed the position of intercostals drain. Ultrasonography (FAST) was done where no free fluid was seen. The blood pressures continued to be sub optimal so inotropes were started along with blood transfusions. Patient was intubated. Central venous pressure of 8-10 cms H<sub>2</sub>O was noted. Ultrasonography (FAST) was repeated to reveal mild free fluid with internal echoes. Echocardiography of the heart suggested mild pericardial effusion of minimal thickness on right side with maintained ejection fraction of 55-60%. ECG showed low voltage waves.



**Figure 1: Chest X-ray confirming the placement of intercostal drain.**



**Figure 2: ECG showing low voltage waves.**

Patient was taken up immediately for an exploratory laparotomy of the abdomen under general anesthesia. Intra operative findings were 100cc of ascitic fluid mixed with blood. The bowel pathology was traced to be normal. Solid organs like spleen, liver, kidneys and pancreas were normal. A significant diaphragmatic bulge was seen on the central tendon region. A decision was made to perform a thoracotomy via median sternotomy. Around 400 cc of blood was evacuated from pericardial space. A 6-8 cm vertical rent was present at antero-inferior wall of right ventricle with no bleeding which

was repaired with pericardial patch reinforcement in two layers with polypropylene 6-0. Bilateral pleura were opened. Contusions were seen on thymus lobes, right atrial wall, right ventricular wall and diaphragm. Thymectomy was done on account of compromised blood supply. Thorough normal saline wash was given and hemostasis was ensured. A mediastinal drain and bilateral pleural drains were placed under a water seal. Bilateral abdominal drains were placed. Intra-operatively, patient had received 3 packed red cells. Patient was put on triple inotropic support with noradrenaline, dopamine and adrenaline. Towards the end of the procedure, patient was off all supports and was able to maintain blood pressure. Patient was not extubated and was shifted to recovery on ventilator support on volume A/C mode.



**Figure 3: Intra operative picture showing vertical rent on right ventricle.**

Post operatively, patient was treated with antibiotics, analgesics and intra venous fluids. He was extubated on postoperative day two and liquids were started orally on postoperative day three. The drain output progressively decreased and eventually removed serially. A pansystolic murmur was auscultated post operatively. Serial 2D echocardiography revealed tricuspid valve prolapsed with LV ejection fraction of 60% without any regional wall motion abnormality which was managed conservatively. After initial stabilization, CT (brain) was done to rule out any brain parenchymal injury and no abnormality was seen. Patient was discharged on Metoprolol 25 mg OD on postoperative day thirteen.

## DISCUSSION

The main cause of cardiac tamponade in trauma patients is penetrating chest injury (80-90%) and it occurs in only 10% of blunt chest trauma injuries.<sup>2</sup> Most of these cases present as contusion to the myocardium presenting as an arrhythmia and are frequently self-limiting. Blunt Cardiac Injuries (BCI), which in addition to blunt cardiac rupture also include injuries such as myocardial contusion, have been reported in 8 to 86% of cases of blunt chest trauma and are associated with high mortality rates.<sup>3</sup> In one

review, the reported mortality in those who were still alive in the emergency department was 89%.

Under normal circumstances, intrapericardial pressure is similar to intrapleural pressure, both being subatmospheric. As left ventricular volume increases by rapid infusion of blood during early diastole, intrapericardial pressure increases. Understandably, during this time the right ventricle is also filling. Since the right ventricle is thin walled, however, increases in intrapericardial pressure (or extrinsic compression from a clot) can exert a more deleterious effect upon this chamber. With tamponade, accumulation of fluid in the relatively non-compliant pericardial space (tough fibrous pericardium) causes the intrapericardial pressure to increase rapidly. Additional increases in intrapericardial fluid result in the loss of diastolic compliance and depressed cardiac function. As the consequences of tamponade progress, higher filling pressures are required for the ventricles to fill. Compensatory mechanisms include higher systemic and pulmonary artery pressures by vasoconstriction, tachycardia, time-dependent pericardial stretch, and blood volume expansion. This latter mechanism only aids in compensating for slow-growing pericardial effusions. Intrapericardial pressure rises initially to levels greater than left and right atrial pressures with the aforementioned compensatory mechanisms occurring. When right and left atrial, right and left ventricular, pulmonary artery wedge and intrapericardial pressures equalize, cardiovascular collapse ensues.

Diagnosis of blunt cardiac injury is difficult to make due to (a) associated injuries which divert physician's attention; (b) lack of physical findings; and (c) lack of specificity of non-invasive tests available in an emergency room.<sup>4</sup> Cardiac tamponade presents as hypotension and decreased cardiac output. The signs of cardiac tamponade are often subtle and can mimic other life-threatening conditions in trauma patients. Beck's triad of elevated jugular venous pressure, reduced arterial pressure and muffled heart sounds, as well as Kussmaul's sign are not practical or reliable in the primary survey of trauma patients.<sup>5</sup> Dilated neck veins will not manifest in the hypovolaemic patient and Kussmaul's sign is abolished in the intubated patient. The difficulty of diagnosis is often compounded by the presence of other life-threatening injuries such as bleeding sources.

Trueblood et al reported the commonest site of rupture to be the atrial appendage presumably due to it being thinnest and weakest area of the heart. He also reported three cases of cardiac rupture at the junction of atria and great veins. They felt that rapid movement of the heart into the left chest coupled with compression force, presumably led to tear in relatively fixed superior and/or inferior vena caval junction with the atria.<sup>6</sup> The mechanisms of cardiac rupture have been divided into seven broad categories.<sup>7-9</sup> The most common cause of ventricular rupture is direct force applied to the anterior

chest wall. This is thought to result in injury if it occurs during end-diastole, when the ventricles are maximally dilated. In acceleration/deceleration injuries, the heart is torn where the great vessels insert into the atria. Bidirectional compression, more common in patients with compliant chest walls and the probable mechanism in our patient, involves compression of the heart between the sternum and the thoracic spine. Less frequently encountered are blast, concussive, indirect, and combined mechanisms. The nature of the injury is dependent on the phase of the cardiac cycle during which the force is applied. Ventricular injuries occur at the end of diastole, and atrial injuries take place in late systole, when the atria are fully distended and the AV valves are closed.

With clinical signs and symptoms being masked in trauma so often, initial importance should be given to diagnostic facilities as echocardiography. It is an innocuous technique, highly reliable, and readily available at the patient's bedside.

Pericardial effusion appears as an echo-free space between the two layers of the pericardium. Echocardiographic assessment should be structured and focused to,

- Differentiate between global or localized effusion;
- Quantify the effusion;
- Describe fluid appearance;
- Analyze hemodynamic compromise.<sup>10</sup>

Collapse of right atrium and right ventricle is also present along with effusion as seen in different cardiac phases.<sup>11</sup>

Diastolic ventricular size variability with respiratory cycle is observed. During inspiration, RV filling is increased, while LV size in diastole decreases. The opposite scenario is present during expiration. This is a physiological phenomenon that can cause a variation of no more than 5% of the cardiac output in absence of tamponade.<sup>12</sup>

An important sign of tamponade seen in 2D echocardiography is dilatation of the IVC (>20 mm in an adult size heart) and hepatic veins. This is known as IVC plethora, and, although not very specific, it is a very sensitive sign of cardiac tamponade (92%).<sup>13</sup>

An inspiratory "bounce" of the interventricular septum toward the LV is a common, but not specific finding in cardiac tamponade.

Along with echocardiography, ECG may show low voltage QRS complexes, sinus tachycardia, electrical alternans and PQ segment depression.

Creatine kinase-myocardial bands (CK-MB) are evaluated as prognostic factors. Elevated preoperative CK-MB may be associated with a poor prognosis.<sup>14</sup> This enzyme is not specific for cardiac injury, but can reflect severe muscle injury and potentially predict

rhabdomyolysis. According to a 1998 study by Swaanenburg et al, among 38 patients with thoracic injuries, 18-30 had increased CK-MB levels, increased CK-MB activity, an elevated ratio of CK-MB activity to total CK, or an elevated ratio of CK-MB mass to total CK upon admission.<sup>15</sup> Although elevated CK-MB levels are not specific for cardiac injury, they can be indicative of severe combined injuries and be associated with a poor prognosis.

Associated injuries may be distracting in blunt chest trauma. As in this case, the diagnosis was not apparent until a negative abdominal exploratory laparotomy. Any patient with severe blunt chest trauma, disproportionate hypotension that is not responding to fluid resuscitation along with elevated central venous pressures should be thoroughly evaluated for cardiac tamponade.

## CONCLUSION

Traumatic cardiac tamponade due to blunt chest trauma is rarely encountered and requires high degree of clinical suspicion. With prompt management, many cases are salvageable. Echocardiography can be helpful in confirming the diagnosis but is not completely reliable.

In the case described here, the patient presented with a myriad of symptoms of polytrauma where chest injury could have been missed but high suspicion proved the patient to be salvageable.

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