Case Report

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Traumatic Cardiac Tamponade – Relearning Old Lesions to Avoid Delay in Diagnosis and Management of a Life-Threatening Thoracic Injury

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Abstract

Cardiac tamponade is a life-threatening condition due to abnormal collection of fluid in the pericardial sac causing hemodynamic instability. In trauma it is blood that gets collected in the sac, most commonly due to penetrating chest injuries or less commonly, blunt chest trauma. If this is not diagnosed and intervened timely, this can be rapidly fatal. We are reporting a case of 35 years old male who was involved in a road traffic accident (RTA). He sustained steering wheel injury on his chest and was taken to multiple hospitals where he was managed only conservatively due to missed diagnosis, before presenting to our ED (Emergency Department). He was here diagnosed with cardiac tamponade with obstructive shock and was urgently taken to operation theatre (OT) for pericardial decompression and was saved.

Keywords: Cardiac Tamponade; Thoracic Injury; Hypotension; Obstructive Shock; Beck's Triad; Muffled Heart Sounds; Pulsus Paradoxus; Electrical Alternans; Kussmaul Sign; Road Traffic Accident (RTA); Controlled Fluid Resuscitation; Thoracotomy; Sternotomy; Pericardiectomy; Pericardiotomy; FAST Scan.

Introduction

Traumatic cardiac tamponade most commonly occurs in penetrating thoracic injuries, more specifically penetrating cardiac injuries. However blunt injuries can also produce tamponade commonly due to cardiac rupture, injuries of great vessels or pericardial vessels.

In atraumatic tamponade, fluid gets collected gradually in the pericardial sac over a period of weeks to months depending upon the cause and the body's compensatory mechanism keeps the hemodynamics stable for a longer period of time. This may be referred to as chronic tamponade and may collect as high as 1litre fluid [1].

However in trauma, blood gets collected in a short span of time usually minutes to hours causing severe rapid hemodynamic instability. This is acute tamponade or may be referred as surgical tamponade; as little as 150ml blood can be lethal [1].

Tamponade is defined as the decompensated phase of cardiac compression resulting from increased intrapericardial pressure [1]. This causes decreased venous return, decreased cardiac output, hypotension, obstructive shock, hypoperfusion, metabolic acidosis and multi-organ dysfunction syndrome (MODS).

Figure 1 shows how fluid or blood gets collected in the pericardial sac in tamponade.

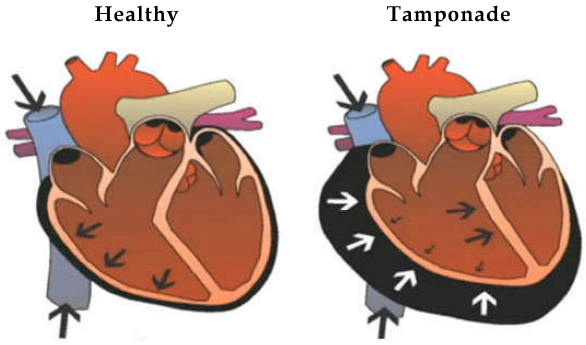
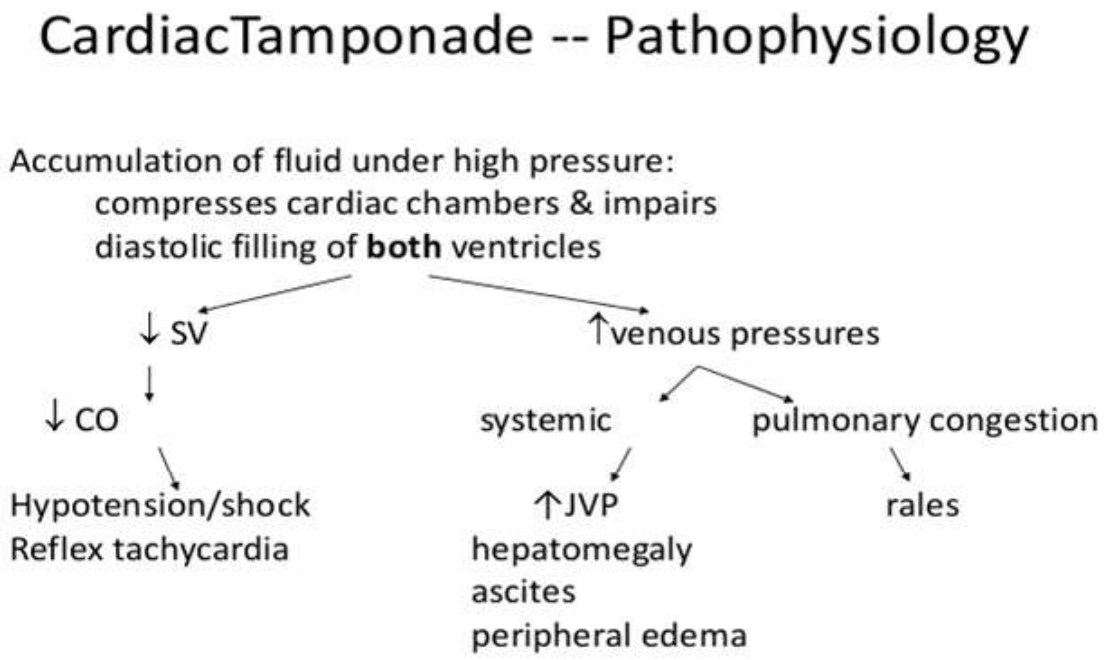


Fig. 1:



The pathophysiology of tamponade can be demonstrated pictorially as Above (Figure 2)

The underlying process for the development of tamponade is a marked reduction in diastolic filling, which results when transmural distending pressures become insufficient to overcome increased intrapericardial pressures [4]. Tachycardia is the initial cardiac response to these changes to maintain the cardiac output [4]. The amount of pericardial fluid needed to impair diastolic filling of the heart depends on the rate of fluid accumulation and the compliance of the pericardium. Rapid accumulation of as little as 150mL of fluid can result in a marked increase in pericardial pressure and can severely impede cardiac output, whereas 1000 mL of fluid may accumulate over a longer period without any significant effect on diastolic filling of the heart [4].

The typical features of tamponade popularly called Beck's triad are

- Hypotension
- Raised JVP or CVP
- Muffled heart sounds

Raised JVP may not be present many a times due to presence of severe hypotension.

Beck's triad is found only in 10% of patients with tamponade [2].

Some other clinical features [3] of cardiac tamponade are chest tightness, tachypnea, tachycardia, confusion/altered mental status, oliguria/anuria, cold clammy extremities, pulsus paradoxus (drop in systolic BP >10mmHg during inspiration) etc.

Kussmaul sign (Paradoxical increase in jugular venous pressure during inspiration) is also sometimes seen.

Tamponade is a medical emergency, the complications of which include pulmonary edema, shock, renal failure and death [4].

The overall risk of mortality depends on the speed of diagnosis, the treatment provided, and the underlying cause of the tamponade. If left untreated, the condition is rapidly and universally fatal [4].

Case Study

A 35 years young male was brought to our ED at around 12 midnight with an alleged h/o RTA 4 hours back. He was driving a car when his car hit head-on with another vehicle coming from opposite direction. There was apparently no loss of consciousness (LOC), seizures, vomiting, ENT bleed. The patient was then taken to 2 different nearby hospitals by the paramedics before being brought to our ED.

As per the notes of previous two hospitals, he was diagnosed as blunt chest injury and was managed conservatively after doing chest X ray and FAST scan

which were reported normal then. However the patient was deteriorating in terms of consciousness and this is when his attendants brought him to our hospital for further management.

He was immediately taken to a monitored bed and initial trauma evaluation done.

He was drowsy and not responding to verbal commands.

Airway was patent with C-collar in situ; no secretions or blood in oral cavity; trachea was in midline and the neck veins were not distended.

Breathing rate was rapid with RR 28/min and oxygen saturation was only 76% at room air which improved to 80% with high flow oxygen via facemask. Air entry were equal and clear bilaterally with minimal bony crepitus over midsternal region on palpation; however there was no external sign of injury on inspection.

In terms of circulation, his pulse rate was 125/min with feeble central pulses and non-palpable peripheral pulses; BP was not recordable; Capillary refill time (CRT) was more than 4 seconds, heart sounds were difficult to be appreciated in the noisy ED.

In terms of disability, his GCS score was E2V3M5 = 10/15; random blood sugars (RBS) was 114mg/dl; pupils were bilaterally equal and normally reacting to light and there were no lateralizing signs.

On exposure, peripheries were cold and clammy; swelling on mid-forehead of 2x2 cms; there was no other external sign of injury.

Ryle's tube was in situ; no bleeding seen.

Foley's catheter was in situ; only 30ml urine since last 3 hrs.

Trauma code was activated and 2 large bore IV canulae were inserted in cubital veins and samples taken for VBG, Blood groupingCrossmatching, complete blood count, kidney function tests and liver function tests.

Radiological imaging studies were ordered; CXR, NCCT Head, NCCT C-Spine, Pelvic Xray, and FAST scan.

On secondary survey, the only significant finding was mid-sternal deformity with bony crepitus.

ECG rhythm strip showed electrical alternans as shown below (Figure 3) and 12 lead ECG also showed only sinus tachycardia with electrical alternans.

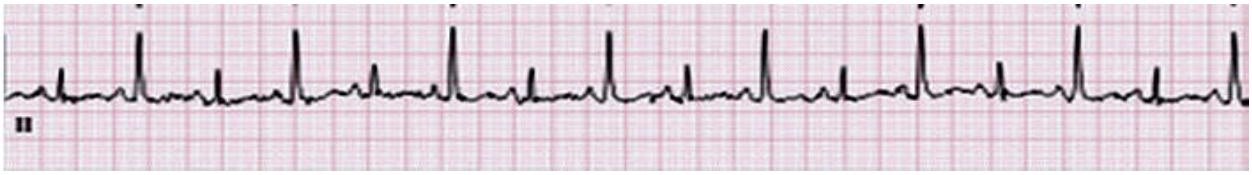


Fig. 3:

CXR showed mildly increased cardiac shadow and FAST scan revealed fluid in pericardial sac around 500-700ml causing tamponade effect.

Other radiology imaging were not done in the ED due to hemodynamic instability.

Patient's VBG showed ph = 7.206, pO_2 = 14.7 mmHg, PCO_2 = 48 mmHg, HCO_3 = 18.5 mmol/L, Na^+ = 146 meq/L, K^+ = 3.2 meq/L, Ca^{2+} = 1.01 mmol/L, Lactate = 3.9; he was in metabolic acidosis.

A provisional diagnosis of Cardiac tamponade with obstructive shock was made and the Cardiothoracic surgeon was informed immediately who after evaluation advised to shift the patient immediately to operation theatre (OT).

Controlled fluid resuscitation was given in order to avoid further worsening of the condition and just to get a palpable peripheral pulse.

BP came up to 70mmHg systolic but he was still in altered mental status.

Pt was taken for urgent thoracotomy/sternotomy.

Course in the Hospital and Outcome

Patient was electively intubated in the OT and was put on mechanical ventilator and general anaesthesia induced.

Intraoperatively, there was a complete fracture of mid-sternal region; sternotomy was done followed by pericardiectomy; 700ml blood clot was removed from the pericardial sac; diffuse bleeding found in the SVC region which was controlled and wound closed with 3 drains. Following pericardial decompression his pulse and blood pressure started settling down.

He was shifted to ICU early morning for observation with stable vitals with pulse of 90/min and BP of 90/60 mmHg.

His 1st set of laboratory reports showed urea of 25mg/dl and creatinine of 0.97mg/dl and samples taken just after the surgery showed increased creatinine of 1.48 mg/dl, meaning that he developed acute kidney injury (AKI).

When hemodynamically stabilized, he was sent for other radiological imaging studies including CT head, C-spine and thorax and multiple X rays which did not reveal anything significant.

On 1st post-op day, he was extubated and was conscious and oriented and vitals were stable without any inotropic support with good urine output.

Repeat echocardiography showed no pericardial fluid collection.

His renal function tests also improved when his hemodynamics got stabilized.

He was eventually discharged after 5 days of hospital stay in a stable condition; OPD follow up after 3 days was also satisfactory.

Discussion and Therapeutic Considerations

This case report illustrates a 65 years old male who sustained blunt chest trauma and presented with altered mental status and hypotension. He was misdiagnosed in previous two hospitals where he presented first and was then brought to our ED.

He was later diagnosed to have cardiac tamponade with obstructive shock and acute kidney injury.

He was immediately taken to OT for cardiac decompression afterwhich he improved.

Cardiac injuries are most commonly overlooked injuries in patients who die from trauma.

The case we describe here is unusual in 4 counts. First, around 700ml blood was removed from the pericardial sac without any evidence of cardiac rupture. Secondly, once the blood and blood clots were removed and the SVC laceration repaired, he improved quickly without any re-effusion later. Thirdly, although it was a high speed RTA, he had only isolated cardiac tamponade without any other injury. Fourthly, the AKI which developed due to hypotension improved quickly once the tamponade was relieved.

The initial CXR did not show any sign of tamponade and the initial FAST scan also was normal which means that the bleeding was more gradual over a period of 3-4 hours to cause the tamponade effect and hemodynamic instability.

Moreover the initial aggressive fluid resuscitation given to stabilize the BP might have worsened the condition of bleeding vessel (s). This emphasizes the importance of balanced resuscitation in trauma when there is hemodynamic instability and the source of bleeding is not yet identified and controlled.

At some point, it was thought that altered mental status could be due to head injury as there was a forehead hematoma which created suspicion and the hypotension could be due to spinal shock but with high degree of suspicion cardiac tamponade was correctly diagnosed and appropriate treatment was given and the patient was saved without any morbidities.

In 2009, Rastogi, described a case of a 50 years old man who was hit by a motorbike who was conscious and oriented with stable vitals but had only mild breathing difficulty without any external signs of injury; he was discharged after giving first aid. The man died after 7-8 hrs and his postmortem report revealed cardiac tamponade [5]. This report also clearly shows that any significant trauma to chest should be evaluated completely and monitored eventhough initial examination seem normal.

Conclusion

Diagnosis of cardiac tamponade is not always very easy.

Cardiac tamponade may take several hours to develop and to cause circulatory failure; therefore any chest trauma must be properly and completely evaluated before coming to any conclusion.

The physical findings of cardiac tamponade are not always apparent despite life-threatening acute cardiac tamponade after blunt trauma.

Focus should always be given to entire vital organs like the heart and the possibility of tamponade must be kept in mind.

Pericardiotomy or pericardiectomy via a thoracotomy or sternotomy is mandatory for life saving cardiac decompression in acute traumatic cardiac tamponade.

A prompt diagnosis using FAST scan and appropriate treatment are lifesaving.

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