Delayed diagnosis of cardiac tamponade following isolated blunt abdominal trauma

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Traumatic haemopericardium is an uncommon but life threatening condition. It is usually caused by penetrating cardiac injuries or cardiac rupture from blunt chest trauma. We report haemopericardium and cardiac tamponade in a young girl after blunt abdominal trauma. She presented with mild upper abdominal pain, tachycardia and hypotension having been kicked in the abdomen by a horse. No damage was found at laparotomy and she remained haemodynamically unstable. Further investigation found cardiac tamponade and haemopericardium. This was managed by insertion of a pericardial drain using transthoracic echocardiogram guidance, with later drainage in the operating theatre using guidance with a transoesophageal echocardiogram.

Case report
A 14-year-old girl presented at 17:30 to the emergency department of a peripheral hospital with a history of being kicked three times in the abdomen by a horse. She complained of epigastric and left upper quadrant pain. Initial examination revealed a tachycardia of 130 beats min⁻¹, arterial pressure of 95/65 mm Hg and mild epigastric tenderness. There was no open wound or evidence of chest injury. She declined analgesia. Intravenous access was obtained and fluid resuscitation commenced. Initial investigations revealed a haemoglobin (Hb) of 140 g litre⁻¹, normal plasma electrolytes and amylase. An electrocardiogram (ECG) and chest x-ray were not performed. The patient was admitted for observation. Two hours later her Hb was 125 g litre⁻¹. She remained hypotensive and tachycardic over the next 3 h with oliguria despite fluid administration. Because ultrasound and computed tomography studies were not available, laparotomy was done.

Anaesthesia was induced using fentanyl 100 μg and thiopentone 200 mg with suxamethonium 100 mg to achieve tracheal intubation. Anaesthesia was maintained with 0.8% isoflurane and 50% nitrous oxide in oxygen. Morphine 10 mg was given for intra- and postoperative analgesia. During the 2-h procedure, she was given 2 litres of Hartman’s solution and 500 ml of Haemacell. Her systolic arterial pressure remained between 90 and 100 mm Hg throughout the procedure and her urine output at 20–30 ml h⁻¹. The surgical findings included free peritoneal fluid, an oedematous gall bladder and duodenum but no evidence of a ruptured viscus or intra peritoneal haemorrhage. Increased pressure in the inferior vena cava (IVC) and congested hepatic and portal veins were noted. After an hour in recovery, she was transferred to the ward where she remained hypotensive, tachycardic and oliguric despite another 6 litres of fluid (3 litres of crystalloid and 3 litres of...
colloid). She was transferred to an intensive care unit (ICU) early the next morning.

On arrival in the ICU she was shocked, with a heart rate of 140 beats min⁻¹ and arterial pressure of 70/30 mm Hg. Heart sounds were present and normal. Chest examination was normal. The abdomen was tender in keeping with recent laparotomy.

There was minimal serosanguinous discharge from her abdominal drains. Fluid administration continued and a radial intra arterial cannula and right internal jugular central venous cannula were inserted. Infusions of norepinephrine and dopamine were commenced. Central venous pressure (CVP) was 22 mm Hg. An ECG showed sinus rhythm with normal size complexes, right ventricular dominance and peaked T waves. On chest x-ray, a right pleural effusion and normal cardiac outline were noted. Her Hb was 127 g litre⁻¹ and arterial blood gas analysis showed pH 7.13, $P_{CO_2}$ 59 mm Hg, $P_{O_2}$ 103 mm Hg, bicarbonate 18 mmol litre⁻¹ and base excess -11 mmol litre⁻¹ ($FIO_2$ 0.6). Plasma potassium was 6.5 mmol litre⁻¹ and 50 ml of 50% dextrose and 15 units of insulin were given. In view of the hypotension and raised CVP the possibility of cardiac tamponade was considered. An urgent trans-thoracic echocardiogram (TTE) revealed a large pericardial effusion with cardiac tamponade (Figs 1, 2).

Fig 1 Transthoracic echocardiogram showing the four cardiac chambers and surrounding haemopericardium.

Fig 2 Transthoracic echocardiogram showing showing right ventricle (RV) being compressed by haemopericardium.
2). A pericardial drain was placed using guidance by the TTE and 500 ml of fresh blood was aspirated cautiously with ease. There was very mild improvement in the haemodynamic condition but not complete drainage of the effusion. A cardiothoracic surgical opinion suggested a ruptured right ventricle as the likely diagnosis. The patient was urgently transferred to theatre with a view to sternotomy. In theatre, with a scrubbed surgical team in attendance, anaesthesia was induced using midazolam 4 mg, fentanyl 250 µg and rocuronium 50 mg to facilitate tracheal intubation. Anaesthesia was maintained with 0.5% isoflurane in oxygen/air. Phenylephrine 50 µg boluses were given in conjunction with the norepinephrine and dopamine infusions already being given. A trans-oesophageal echocardiogram showed a large pericardial effusion, tricuspid regurgitation and abnormal right ventricular wall motion consistent with myocardial contusion. Fresh blood (50 ml) was easily aspirated via the pericardial drain with no change in the size of the effusion and no improvement in cardiovascular state. Since blood was aspirated with ease, the effusion did not diminish in size and there was little improvement in haemodynamic state we considered that the drain could have inadvertently penetrated the ventricle. According to the TTE it was in the correct position but we could not see it with the TOE. To confirm pericardial placement of the pigtail catheter we injected 2 ml of Haemacell, which was seen on the TOE as echogenic bubbles within the pericardial sac. Using the TOE, a further 200 ml blood was aspirated from the pericardium until it was empty with marked improvement in the haemodynamic condition. Thirty minutes after complete drainage of the effusion no recurrence of the collection was seen on the TOE and we concluded that there was no ventricular rupture. Surgery was deferred and the patient was transferred to the ICU with the pericardial drain in place. A right intercostal catheter was placed and 850 ml blood drained. Serial TTE over the next 24 h showed no re-accumulation of fluid but there was significant abnormality of right ventricular wall motion consistent with contusion and mild tricuspid regurgitation. She was discharged from the ICU the next day. She made a good recovery over the next 2 weeks with discharge being delayed by an ileus.

Discussion
Cardiac injuries are the most commonly overlooked injuries in patients who die from trauma. The true incidence of cardiac trauma is difficult to quantify although the incidence of blunt cardiac trauma requiring treatment may be as low as 2.6–4.5%. Haemopericardium is usually caused by a penetrating cardiac injury or cardiac chamber rupture from blunt chest trauma. The literature abounds with such reports. The case we describe is unusual on three counts. First, the site of the trauma was the upper abdomen and not the chest. Our patient was most specific that the blows were to her epigastrium alone although there can be no doubt that a significant degree of force must have been transmitted up across the diaphragm as well as in an antero-posterior direction across the abdomen. Secondly there was an unusually large amount of blood (700 ml) present in the pericardium without evidence of cardiac rupture. Thirdly the effusion was successfully drained with TOE guidance.

Symptoms of cardiac tamponade are usually rapid in onset but depend on the rate and volume of pericardial fluid accumulation. It is a diagnosis that needs a high index of suspicion, in one review four out of five cases were initially missed in 16 patients who presented to an emergency department with penetrating cardiac trauma, and in 16 of 24 patients with atrial tears from blunt trauma the diagnosis was delayed by at least 1 h despite all 24 patients having an increased CVP or neck vein distension and hypotension. In our case report the patient presented with a mechanism of injury suggesting intra abdominal injury and her initial clinical findings supported this. She had no ‘classic’ signs of tamponade, but Beck’s triad (distended neck veins, muffled heart sounds and hypotension) is found in only 10% of trauma patients with tamponade. It was unfortunate that a chest x-ray was not done early in management, because a haemothorax may have aroused suspicions, although she had no symptoms related to her chest at that time. Even with a haemothorax, the clinical features of cardiovascular instability and blunt abdominal trauma would make an intra peritoneal bleed the most likely diagnosis. However, if a chest x-ray done after a negative laparotomy, with the patient still hypotensive, showed a haemothorax then the possibility of serious chest injury would be considerable.

Induction of anaesthesia in these circumstances is fraught with difficulty. As fluid collects rapidly in the pericardial space pressure can increase quickly in the relatively indistensible parietal pericardium. Pressure changes are transmitted to the ventricle and this decreases the atrio-ventricular pressure gradient during diastole and impedes ventricular filling, causing a reduced end-diastolic volume and stroke volume. Increased diastolic ventricular pressure will decrease the coronary perfusion pressure predisposing those with coronary artery disease to ischaemia, particularly if tachycardia is present. Compensatory mechanisms include increased sympathetic activity leading to increased heart rate and increased contractility, which help maintain cardiac output in the face of a reduced stroke volume and vasoconstriction to maintain central blood pressure and support the gradient for coronary flow. The anaesthetic aims are therefore to maintain adequate filling pressures, adequate heart rate and maintain contractility. Anaesthesia for the laparotomy was induced with thiopentone, which is not the agent of choice as this can cause hypotension by venodilation and decreased venous return decreasing cardiac output with potentially disastrous consequences as was reported by Cyna et al. Propofol is similarly contraindicated.
The patient remained relatively stable throughout her laparotomy with no haemodynamic deterioration. Laparotomy failed to provide a definitive cause for her cardiovascular instability although there were some clues. Haemoglobin concentration did not decrease and increased pressure on the IVC and congested hepatic and portal veins were noted. After surgery, the patient became increasingly shocked despite persistent administration of fluids and was extremely ill when she was transferred to intensive care.

Pigtail drainage catheter under echo guidance using local anaesthesia is the method of choice in the management of pericardial effusions and cardiac tamponade and prevents the need for diagnostic needle pericardiocentesis which has a high false positive and false negative rate. An unusually large amount of blood was aspirated (500 ml) with no diminution of flow, suggesting the possibility of right atrial or ventricular rupture. TOE probe placement is routine before all cardiac surgical procedures in our institution. This confirmed that the pigtail catheter was in the pericardial cavity and allowed the effusion to be drained under direct vision and to see if the effusion would reaccumulate. It also allowed the right ventricle, valves and interatrial septum to be seen more accurately than with the TTE. Because the effusion did not reaccumulate 30 min after drainage, we felt that cardiac rupture was not present and the patient could be managed conservatively without exploratory surgery. Without TOE, the patient would have undergone unnecessary surgery.

In summary pericardial tamponade is an uncommon but life threatening condition. It can mimic other conditions such as hypovolaemia particularly after trauma. A high index of suspicion must be retained if it is to be diagnosed and treated effectively.

References

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