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Cardiac Tamponade - An Unlikely Cause Of Unexplained Hypotension In An Isolated "Minor" Blunt Chest Injury

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ABSTRACT

Cardiac tamponade after a minor blunt chest trauma is indeed rare. Here, we report on one such case presenting with unexplained hypotension at the Emergency Department. The problems of diagnosis and treatment are discussed. In short, the proper management of this life-threatening condition can only be rendered by a high index of suspicion and close vigilance.

Keywords: cardiac tamponade, blunt chest trauma

INTRODUCTION

Extensive blunt chest injury is associated with a high mortality rate of at least about 30%, even in patients who can survive to reach medical care. Death is usually attributed to associated injuries[1].

In comparison with penetrating thoracic trauma, blunt chest injury infrequently results in cardiac tamponade[2]. Needless to say, cardiac tamponade arising from an isolated minimal blunt trauma to the chest is extremely rare[3].

Here, we present an unusual case of a young woman with unexplained hypotension after a history of an apparently "minor" injury.

CASE HISTORY

An anaesthetic consult was made for a previously well 32-year-old female van driver who had presented with unexplained hypotension (systolic blood pressure of 65 mmHg) after her chest had hit the steering wheel as the result of a collision with a tree. Being conscious and devoid of neurological deficits, she had complained of some chest pain when she was first seen at the Emergency Department, some one hour after the accident.

Her heart rate was not elevated. Slight bruising over the sternum was noticed but there was no other soft tissue or skeletal injury. Her abdomen was soft and not distended. She did however have diffused swelling of the neck anteriorly with minimal congestion of superficial neck veins. Air entry was equal in both lungs and there was no muffling of heart sounds.

She was resuscitated with a total of 3 litres of crystalloids and colloids with no improvement of clinical signs. The chest X-ray and 12-Lead ECG showed normal findings. Her complete blood count, arterial blood gases and urea electrolytes results were also essentially normal.

However, the central venous pressure was 30 mmHg, with a monophasic trace. There was no pulses paradoxus. An urgent 2-dimensional echocardiography showed a pericardial effusion with a predominant collection around the right ventricle and right atrium, resulting in the collapse of the right ventricle. The left ventricular contractility however, appeared normal.

Open subxiphoid pericardial drainage was performed under ketamine-pancuronium midazolam anaesthesia. (The patient's trachea had been intubated earlier, prior to the arrival of the anaesthetist at the casualty department).

Intra-operatively, the blood pressure normalised rapidly to a systolic of 130 mmHg after the evacuation of 75 mL of unclotted blood from the pericardial sac. The central venous pressure also reduced dramatically to 10 mmHg with the rapid return of the polyphasic waveform.

Subsequently, minimal drainage was obtained from the pericardial space via a catheter in-situ. The patient recovered uneventfully. Serial myocardial isoenzyme fraction of creatinine phosphokinase (CKMB) was consistently lower than 5%.

There was also no change in the ST segments of the subsequent ECGs. The patient was extubated and discharged from the ICU after 3 days. A CT scan which was done before home discharge some 2 weeks after the injury showed a resolving haematoma with grossly intact mediastinal structures, including the ascending aorta.

DISCUSSION

Cardiac tamponade arising from intrapericardial bleeding can result from a variety of cardiac injury, ranging from minor contusions to cardiac rupture; the latter being more severe, will nevertheless have a poorer prognosis and may be rapidly fatal[4,5]. Judging from the clinical presentation and the favourable outcome, our patient most probably had self-limited extra cardiac haemorrhage of the pericardium.

Clinical evidence of cardiac tamponade after acute injury can be deceptive. Beck's triad (muffled heart sounds, hypotension and distended neck veins) is manifested in only a third of the patients[2]. Normally, the pericardial space contains less than 50 mL of fluid; the presence of as little as 100 mL of blood in the pericardial space has been shown to impair diastolic filling, increase venous pressure and decrease cardiac output[6].

As evident from our case, the evacuation of just 75 mL of unclotted blood from the pericardial space resulted in remarkable improvement of the haemodynamic parameters. The presence of a high central venous pressure, while suggestive of cardiac tamponade, is by no means diagnostic of the condition[3].

Cardiac tamponade typically produces a monophasic venous pressure trace in which the *y* descent is obliterated because of the inhibition of the early diastolic run-off from the atrium to the ventricle by the compressive pericardial effusion[7]. However, a low pressure cardiac tamponade may occur in the hypovolaemic patient with a lower equilibration of the right heart pressures[6].

Pulses paradoxus, the old classical sign of cardiac tamponade is not only hard to detect, but is also a non-specific finding[8]. Chest X-rays and ECGs are also not useful in diagnosis as both significant enlargement of the cardiac shadow and ECG changes occur rarely.

Echocardiography is a useful diagnostic tool for cardiac tamponade because the absence of intra-pericardial fluid

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effectively excludes the diagnosis. Moreover, additional causes and the degree of cardiac dysfunction can also be detected. Fluoroscopy, by revealing decreased or absent cardiac pulsation, may also help in the diagnosis of cardiac tamponade[9].

Although initial fluid resuscitation is the universally acceptable treatment to increase diastolic filling pressure and cardiac output, the extent of definitive treatment of cardiac tamponade due to blunt injury is open to dispute. While some authors believed that pericardiocentesis and observation to be the definitive management, others had advocated pericardiotomy and exploratory thoracotomy to be the treatment of choice[1].

In our patient, subxiphoid pericardiotomy and drainage was probably adequate as there was no myocardial dysfunction (including regional wall motion abnormality) detected and the heart had appeared to be intact. Moreover, there was no perceptible bleeding after the initial removal of the liquid blood. If there had been continued bleeding, a further exploration would have been emergently undertaken.

In summary, unexplained hypotension in a seemingly "minimal" blunt trauma to the chest must alert the clinician to the possibility of cardiac tamponade even though this is indeed a rare event under the circumstances described.

Finally, close vigilance is required as the classical findings of cardiac tamponade are often absent in these cases.

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