Delayed rupture of the mitral valve complicating blunt chest trauma

A D Farmery, P H Chambers, A P Banning

Abstract

A case is described of acute torrential mitral regurgitation resulting from avulsion of both papillary muscles 12 hours after a blunt chest and abdominal injury. The intensive care and surgical management are discussed.

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Keywords: mitral regurgitation; chest trauma

Case report

A 38 year old male driver was involved in a head on road collision while wearing a seat belt. Recovery of the patient from the scene of the accident took one hour. On arrival in hospital, his Glasgow coma score was 15, he was able to maintain patency of his airway, had a respiratory rate of 35 breaths/min and was cyanosed, with evidence of a flail segment in the left chest and reduced intensity of breath sounds on the right. His pulse rate was 130 beats/min, blood pressure of 190/90 mm Hg, and heart sounds were normal. The abdomen was rigid and "splinted". Chest radiography confirmed a haemopneumothorax on the left and pneumothorax on the right with bilateral rib fractures. A 12 lead electrocardiogram showed sinus tachycardia with T wave inversion in leads V₅ and V₆.

Bilateral chest drains were inserted and a repeat chest film confirmed re-expansion of both lungs and some pulmonary shadowing. An urgent laparotomy demonstrated 1 litre of blood in the peritoneal cavity and several lacerations in the liver which were sutured and packed. The patient was transferred ventilated and haemodynamically stable to the intensive care unit (ICU) but three hours later he

suddenly became shocked with a fall in blood pressure and a rise in the central venous pressure. Hypovolaemia from continued abdominal bleeding was thought to be unlikely and this was supported by results of an abdominal ultrasound examination. A transthoracic ultrasound, performed at this time by the radiologist, was reported as showing good left ventricular function with no significant pericardial effusion or evidence of tamponade.

Despite inotropic support the patient remained hypotensive and further chest radiography demonstrated pulmonary oedema. Repeat clinical examination now revealed a palpable systolic thrill and a loud pansystolic murmur and further transthoracic echocardiography (TTE) was performed which demonstrated prolapse of the anterior mitral valve leaflet, severe mitral regurgitation, and a poorly defined echogenic mobile mass within the left ventricular cavity. Left ventricular function was good.

The patient was transferred urgently to a cardiothoracic centre where transoesophageal echocardiography (TOE) demonstrated severe mitral regurgitation, rupture of both mitral valve leaflets, and a ruptured papillary muscle attached to the chordae tendinae prolapsing between the left ventricular and left atrial cavities (fig 1). Surgical examination demonstrated myocardial contusion of the right ventricle and flail mitral valve leaflets both with ruptured haemorrhagic papillary muscles. The valve was unsuitable for repair and a Carbomedics 29 mm metal prosthesis was implanted. Over the next 24 hours the patient's condition was unstable with hypotension and oliguria, requiring intra-aortic balloon pumping and haemofiltration. He was extubated on day 11 and discharged from ICU on day 17. On day 22 he was discharged fit and well from hospital.

Discussion

Injuries to the heart and great vessels should always be considered after blunt chest trauma. This case illustrates the limitations of TTE in the emergency situation where imaging is commonly suboptimal. This is due to the problems of positioning patients who are ventilated, instrumented, and may have pneumothoraces or lung contusion. The presence of a tachycardia also makes the interpretation of colour Doppler signals difficult, and in the context of severe valvular regurgitation where the ventricle "flutters to deceive", the clinician may be falsely reassured by dynamic systolic function. TOE is the investigation of choice when traumatic papillary muscle rupture is suspected as it allows the diagnosis to be made with 100% sensitivity and specificity.²

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Figure 1 Four chamber horizontal plane transoesophageal echo image during left ventricular (LV) systole. A ruptured papillary muscle can be seen in the body of the LV cavity (outlined) with prolapse of the anterior mitral valve leaflet (arrow) in the left atrium (LA).

Delayed papillary muscle rupture is well documented.¹ The forces exerted on the heart from dynamic chest trauma are variable and depend on the elasticity of the thorax and the intra-abdominal pressure. A further variation depends on the points in the respiratory and cardiac cycles at which the insult occurs. The most vulnerable points are at maximal inspiration, and during isovolumic systole when all the valves are closed and the cavities hold the maximum amount of blood. The pathophysiology of the muscle rupture is not clear but it has been suggested that the intramural blood flow redistribution caused by local oedema, fibre rupture, and haematoma produces sufficient endocardial ischaemia to cause papillary muscle necrosis. Papillary muscles, being projections from the ventricular wall, are particularly prone to ischaemia in this way as they cannot benefit from a collateral supply. The subsequent proteolytic process reaches a maximum

over 24 hours that accounts for the delayed presentation.

In conclusion this case illustrates the need for the possibility of cardiac trauma to be considered in patients who sustain a blunt chest injury. This consideration should be a dynamic one, and the diagnosis should be reconsidered after any change in the patient's condition even where cardiac trauma was previously excluded. Although TTE may be useful in some cases, it has limited sensitivity compared with TOE and consideration should be given to more widespread application of the latter in ICUs.³

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- 2 Spangenthal EJ, Sekovski B, Bhayana JN, *et al.* Traumatic left ventricular papillary muscle rupture: the role of transesophageal echocardiography in diagnosis and surgical management. *J Am Soc Echocardiogr* 1993;6:536–8.
- 3 Townsend JN, Hutton P. Transesophageal echocardiography in anaesthesia and intensive care. *Br J Anaesth* 1996;77:137–9.

Fatal flecainide intoxication

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Abstract

Flecainide acetate is a potent class 1C antiarrhythmic agent used mainly for the treatment of supraventricular arrhythmias. Acute overdose of this drug is rare but frequently fatal. The clinical course of a patient that ingested a large quantity of flecainide as a suicide attempt is described and current therapeutic strategies discussed.

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Keywords: flecainide; toxicity

Case report

A previously healthy 36 year old man presented to the accident and emergency department having taken a deliberate overdose of approximately 100, 100 mg flecainide acetate tablets six hours previously. On arrival his clinical observations showed a systolic blood pressure of 140 mm Hg and a Glasgow coma scale (GCS) score of 15/15. His electrocardiography (ECG) monitoring strip showed a polymorphic ventricular tachycardia at a heart rate of 140 beats/min (fig 1).

Ten minutes after his arrival the patient had an episode of pulseless ventricular tachycardia which was treated successfully with a single unsynchronised shock at 200 joules. Subsequently his observations were stable with a systolic blood pressure of 170 mm Hg and GCS score of 15/15. ECG monitoring displayed sinus rhythm at a rate of 75 beats/min.

Analysis of 12 lead ECG showed the QRS duration to be prolonged at 0.2 sec (fig 2). Urea and electrolyte measurements were within normal limits. Arterial pH was 7.374.

Advice from a regional poisons information unit recommended gastric lavage, administration of activated charcoal, and infusion of sodium bicarbonate to raise the arterial pH to 7.5. The patient refused gastric lavage and activated charcoal but allowed treatment with hypertonic sodium bicarbonate. He agreed to come into hospital and was therefore admitted to the coronary care unit for monitoring and treatment with sodium bicarbonate 1.26%. While in coronary care the patient's ECG monitoring continued to demonstrate sinus rhythm at approximately 75 heartbeats/min with prolonged QRS duration.

Ten hours after admission the patient's condition deteriorated with an episode of hypotension secondary to ventricular tachycardia. This reverted spontaneously before treatment was instituted and the systolic blood pressure returned to 120 mm Hg. Electrolyte analysis was again normal and arterial pH was 7.404. Two hours later the patient deteriorated to a pulseless electrical activity cardiopulmonary arrest. Full resuscitation following Advanced Life Support guidelines was performed but was unsuccessful. The patient was pronounced dead 12.5 hours after admission. At postmortem examination a quantity of granular material was identified within the deceased's stomach indicating that absorption was still continuing up to the time of death. Serum flecainide concentration was 3.32 mg/l at postmortem examination. The usual therapeutic plasma concentration is 0.2–1 mg/l.

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Figure 1 ECG monitoring strip.