CASE REPORT

Delayed post-traumatic tamponade together with rupture of the tricuspid valve in a 15 year old boy

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Abstract

With the increase in the number of high speed motor vehicle accidents, blunt, non-penetrating trauma to the heart has become an important health problem. An unusual case is reported of a 15 year old boy urgently referred with cardiac tamponade and a new systolic murmur four months after a car accident. The problems of the diagnosis and possible causes of late cardiac tamponade and tricuspid regurgitation following this type of accident are discussed.

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Keywords: blunt cardiac trauma; delayed cardiac tamponade; traumatic tricuspid regurgitation

A 15 year old boy with no medical history was admitted in August 1999 to hospital following a road traffic accident. He had bilateral extensive pulmonary contusion, liver laceration, and a fracture of the fifth metacarpal bone. During his admission no signs of cardiac involvement were noted and there was no evidence of major vessel disruption. After one week he was discharged in good clinical condition.

Some weeks later he noticed progressive tiredness and dyspnoea during exercise. Four months after the accident when the patient was suffering from flu like symptoms, his general practitioner discovered a systolic murmur.

In the days before the admission, the patient's temperature rose to 39°C and he had severe dyspnoea at rest.

On arrival at a second hospital, the patient was pale, tachypnoeic, and tachycardic, his blood pressure was 110/70 mm Hg, a pulsus paradoxus was noted, and central venous pressure was increased. Echocardiography showed a large pericardial effusion. The patient was immediately referred to our department for emergency pericardiocentesis.

Transoesophageal echocardiography just before the puncture showed severe tricuspid regurgitation due to a flail valve caused by a chordal rupture. Left ventricular function was completely normal. Severe dilatation of the right ventricle was noted (fig 1).

A total of 650 cm³ (sero) sanguineous fluid (with obvious signs of haemolysis) was removed with immediate normalisation of the patient's clinical and haemodynamic situation. Lactate dehydrogenase and total protein content in the pericardial fluid were, respectively, 4369 U/l and 51 g/l (concentrations in serum 856 U/l and 55 g/l, respectively).

Further testing showed mild signs of inflammation. C reactive protein 108 mg/l (normal < 1) and fibrinogen (5.21 g/l, normal range 1.8–4.0) were increased at the time of admission.

Some weeks later the tricuspid valve was surgically repaired. Inspection of the valve showed a rupture of the posterior papillary muscle and partial laceration of the chordae tendineae, causing deinsertion of anterior and posterior leaflets (fig 2).

The posterior papillary muscle was reinserted at its base, the chordae tendineae of the anterior leaflet were reimplanted on the head of

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Figure 1 Transthoracic echocardiography showing severe dilatation of the right ventricle resulting from flailed tricuspid valve caused by chordal rupture.

Figure 2 Intraoperative picture of the disorganised tricuspid valve. The top of the posterior papillary muscle is visible.

the anterior papillary muscle, and a tricuspid annuloplasty using a Carpentier Edwards ring was done.

The patient's postoperative course was uneventful and he was discharged on day 6.

Subsequent examination of the patient during the year following the accident showed no recurrences of pericardial effusion. Echocardiography showed very mild tricuspid regurgitation with no signs of pericardial constriction.

Discussion

With the recent increase in high speed motor vehicle accidents, blunt cardiac trauma is seen more frequently. Several types of injuries to the heart following this type of trauma have been described.

We report two unusual complications of blunt chest trauma: delayed cardiac tamponade and severe tricuspid regurgitation. Cardiac tamponade is frequently reported after blunt chest trauma as a result of early haemopericardium or pneumopericardium. However, delayed cardiac tamponade caused by late haemopericardium is rare (in contrast to delayed cardiac tamponade after cardiac surgery).

Post-traumatic haemopericardium can be caused by coronary artery laceration, cardiac rupture, or diffuse myocardial haemorrhage. As the signs of cardiac tamponade are dependent on the rate of fluid accumulation, the quantity of pericardial fluid, and the compensatory mechanisms maintaining cardiac output, the fact that the tamponade became evident after four months made those causes unlikely. The exact cause of the pericardial effusion in our case is speculative.

Several hypotheses are postulated in the literature. In one case slow bleeding into the pericardial space was suggested in a 9 year old child presenting with delayed cardiac tamponade one week after suffering a blunt trauma.¹ Others postulate that it may result from the displacement of thrombus that had temporarily closed the cardiac wound.² Another suggestion is that the adhesion that formed at the time of injury was torn.³ Still other authors postulate that delayed cardiac tamponade can be caused by exudative non-haemorrhagic pericardial effusions caused by postcardiac injury syndrome and is then attributed to autoantibodies against the pericardium or myocardium.⁴ Another possibility is that the pericardial effusion has gradually developed and that haemolysis of an earlier pericardial haematoma is responsible for accumulation of additional fluid in the pericardial space.⁵ We believe that this last mechanism was probably the cause of the delayed cardiac tamponade in our patient.

A second consideration is the severe tricuspid regurgitation. Intracardiac lesions of the tricuspid valve induced by blunt chest trauma are rare. As traumatic rupture is often well tolerated haemodynamically, its frequency may be

underestimated. A review in 1994 found a median duration between the trauma and surgery of 17 years with a postoperative median survival of 12 years.⁶

During the past few years an increasing number of reports have been published, mainly because of the increase in road accidents and because of increased awareness and the availability of better diagnostic techniques, especially transthoracic and transoesophageal Doppler echocardiography.⁷

Because of its position in the chest (immediately behind the sternum), the right ventricle has a predisposition for an anteroposterior compression type of injury. An increase in hydrostatic pressure results in an increase in intracardiac pressure. With this sudden increase in right ventricular intracavitary pressure during the end diastolic phase, when both the pulmonary and tricuspid valve are closed, major traction on both the valvar and subvalvar apparatus is generated.⁸ If subvalvar damage, rupture or avulsion of one or both papillary muscles (most often the anterior papillary muscle), or rupture of the chordae tendineae is present, the clinical picture seems to become more rapidly symptomatic.⁹

Damage to the leaflets themselves, either by laceration or by rupture near the annulus, leads to a more insidious onset of symptoms.¹⁰ In our case rupture of the posterior papillary muscle was seen, together with rupture of the chordae tendineae supporting the anterior leaflet.

This case emphasises the need for physicians to be aware of possible late complications after blunt chest trauma in order to intervene in a timely fashion. Follow up examination of all patients with chest trauma for several months after the injury is recommended for detection of such delayed sequelae.

The patient who has suffered a blunt cardiac trauma has to be educated and instructed to return when symptoms of chest pain or dyspnea become evident.

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