Case report

A previously healthy 42-year-old man was admitted to our emergency department after a horse had kicked him in his left chest 2 h earlier. At the trauma scene he was conscious but confused and dyspnoeic. A left thoracostomy was performed and he was transferred to our hospital. Upon arrival, he was awake with a heart rate of 120 beats/min, palpable peripheral pulses, normal breath sounds, and bruises across his lower sternum and left ribcage.

Shortly after arrival, he became restless with increasing shortness of breath. The systolic blood pressure decreased to 70 mmHg and his heart rate increased to 140 beats/min, while peripheral pulse oximetry indicated significant oxyhaemoglobin desaturation. An endotracheal intubation was performed and the patient developed massive frothy lung oedema fluid. Chest radiography was interpreted as bilateral lung contusion (Fig. 1) and his electrocardiogram showed sinus rhythm, 75 beats/min, with STT depression in inferolateral leads (Fig. 2). Due to progressive hypoxia, mechanical ventilator pressures were increased. His haemodynamic state further deteriorated into pulseless electrical activity requiring high-dose inotropes and two episodes of cardiopulmonary resuscitation. Transthoracic echocardiography was performed but was complicated because of difficulties in obtaining accurate windows and because of the nervous tension in the emergency department resuscitation room. Finally pericardial tamponade was excluded and no other abnormalities were observed, but colour Doppler was not used. The patient was transferred to the ICU with the diagnoses of bilateral lung contusion and possible myocardial contusion. Because of further respiratory deterioration he was placed in a prone position and a transoesophageal echocardiography (TEE) was performed. This time, a massive mitral insufficiency was observed due to a complete avulsion of the anterolateral papillary muscle (Figs. 3 and 4). Mitral valve replacement was readily carried out. Because of massive pulmonary congestion the patient failed to wean from cardiopulmonary bypass and veno-arterial extracorporeal membrane oxygenation (ECMO) was instituted until haemodynamic stabilisation at day 4. Unfortunately, there was no neurological recovery after cessation of sedation and a brain CT scan revealed massive bilateral ischaemic stroke. After excluding other causes for his neurological state, further therapy was withheld and the patient died.

Discussion

In this case report we describe a patient with a traumatic mitral valve rupture. His acute respiratory insufficiency was
Fig. 1 Chest X-ray at admittance showing bilateral pulmonary congestion, which was first assumed to be bilateral lung contusion.

Fig. 2 ECG at admittance showing sinus rhythm with STT depression in inferolateral leads.

Fig. 3 Mid-oesophageal four-chamber view showing ruptured anteropapillary muscle.
first misdiagnosed as bilateral lung contusion. Although this injury is much more common after blunt chest trauma, the clinical presentation with massive pulmonary oedema and the congestion on chest X-ray was more compatible with acute left-sided heart failure. Transthoracic echocardiography was performed as recommended in the Advanced Trauma Life Support guidelines. However, because of unfamiliarity with this condition and the stress in the resuscitation room, colour Doppler was not used and the correct diagnosis was not established. After a delay of 4 h, the correct diagnosis was made and patient underwent surgery.

Traumatic mitral valve incompetence due to blunt chest trauma was first described by Glendy and White in 1936 [1]. Compared with myocardial infarction, which is a well-recognized cause of papillary muscle rupture, blunt chest trauma is a rare cause of this condition. Parmley et al. described 24 cases of papillary muscle rupture in 546 autopsy cases of nonpenetrating trauma to the chest [2]. Most commonly the papillary muscles are affected, followed by the chordae tendineae and mitral valve leaflets [3]. The presumed mechanism leading to these lesions is a sudden deceleration or compression of the heart leading to an increase in intracardiac pressure. At end diastole and during the isovolumetric contraction, this may damage the closed atrioventricular valve or subvalvular apparatus.

The clinical picture is always virtually acute and includes forward and backward failure. Although in general the nature of the damage to the valve and subvalvular structures dictates the best mode of repair, there is no consensus in the literature for the best surgical strategy. The most common treatment reported is mitral valve replacement [3].

In comparable cases of acute cardiac failure, ECMO is increasingly being used. Veno-arterial ECMO can completely overtake the circulation and respiratory function while the underlying cause can be treated [4, 5].

Clinicians should be alert for traumatic valve lesions in every case of acute respiratory insufficiency after blunt chest trauma. An echocardiographic evaluation with the use of colour Doppler is warranted in every patient with dyspnoea after blunt chest trauma. When transthoracic evaluation is not conclusive it should be followed by transoesophageal echocardiography. After diagnosis of traumatic papillary muscle rupture, mitral valve replacement or repair should be carried out as soon as possible.

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