Shortness of breath after blunt chest trauma: consider acute aortic valve insufficiency

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Abstract - This case report describes a patient who developed symptoms of dyspnoea early after a high velocity car accident. Her symptoms were progressive and 25 days after the trauma massive aortic insufficiency was diagnosed. Operation showed a tear in the commissure between the right and the non-coronary cusps with a tear in the intima. Uncomplicated aortic valve replacement followed. Traumatic aortic insufficiency is a rare and sometimes difficult diagnosis to make. Clinical presentation, pathophysiology, diagnosis and treatment are described.

Keywords - Blunt chest trauma, acute aortic valve insufficiency.

Introduction
Patients complaining of shortness of breath after blunt chest trauma are a diagnostic challenge due to the myriad of potential underlying disorders, non-specific presentation and the presence of coexisting injuries. Traumatic aortic valve insufficiency is a rare but potentially fatal complication of blunt chest trauma. Although a potential surgical emergency, its diagnosis can be extremely difficult. We describe a patient with severe traumatic aortic valve insufficiency and a prolonged delay in diagnosis. We discuss the existing literature regarding underlying mechanisms, diagnosis and treatment options.

Case report
A 79-year-old woman was brought to our emergency department after a high velocity car accident. She had no relevant previous medical history, apart from bilateral hip replacement. On arrival she had a blood pressure of 120/80mmHg, heart rate of 100 bpm, oxygen saturation of 95-99% with 15 l O₂/min with a non-rebreathing mask and a maximum Glasgow Coma Scale score. The secondary survey showed fractures of the right occipital condyl, transverse processes C4, C6, C7 on the right and L2 on the left, ribs 1, 4, 6, 7 on the left and 1 on the right, manubrium sterni, right humerus and proximal phalanx of the third left digit.

On chest x-ray bilateral lung contusion was diagnosed (Figure 1). The electrocardiogram (ECG) was normal but because of the fractured manubrium a cardiologist was consulted. Troponin I was <0.20 ug/l and the transthoracic echocardiogram (TTE), which was of moderate quality, showed normal left and right ventricular function, minimal aortic valve regurgitation, moderate tricuspid valve insufficiency and no pericardial effusion.

She was admitted to the medium care unit for observation and the next day she was transferred to the orthopaedic ward.

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Figure 1. Chest x-ray on admission
hypertension (61/21mmHg). Pro-BNP was 13105 pg/ml. With increasing creatinine levels (from 29 to 65 mmol/l) diuretics were stopped despite persistent pulmonary oedema. On Day 20 she developed atrial fibrillation and was transferred to the Coronary Care Unit for rhythm control and treatment with dobutamine, ACE inhibition and loop diuretics was started. Physical examination by the cardiologist now showed a systolic and a diastolic murmur. On Day 22 a CT angiography was made to exclude pulmonary embolism. On Day 25 the symptoms of congestive heart failure were progressing and a pulmonary artery catheter showed the following values: pulmonary artery pressure 73/41mmHg (mean 41), pulmonary capillary wedge pressure 39 mmHg, right atrial pressure 21 mmHg and a cardiac index of 2.37 l/min/m². The same day severe respiratory insufficiency developed and she was transferred to our Intensive Care unit for intubation and mechanical ventilation. Transoesophageal echocardiography showed massive aortic insufficiency and a defect between the right- and non-coronary cusps (Figure 3), normal left ventricular function without dilatation and moderate mitral and tricuspid insufficiency. There were no signs of a dissecting aneurysm. Treatment with milrinone and norepinephrine was started. The next day, surgery was performed which revealed a traumatic aortic valve insufficiency due to rupture of the commissure between the right and the non-coronary cusp with an associated intima tear (Figure 4). The intima was sutured and the aortic valve replaced with a bioprosthesis (Edwards Perimount 23 mm). Her postoperative course was uneventful. Inotropic agents were stopped and she was extubated on Day 33. She was transferred to the general ward three days later and was discharged to a nursing home for further rehabilitation on Day 50.

**Discussion**

We describe a patient with traumatic aortic valve insufficiency after blunt chest trauma and a prolonged delay in diagnosis. Although on initial chest X-ray on the emergency department bilateral lung contusion was diagnosed, it is more compatible with a diagnosis of acute pulmonary oedema. The progressive signs of congestive heart failure and the repeated chest x-ray one week after admission clearly suggest that severe aortic valve insufficiency was present from the beginning. Unfamiliarity with traumatic aortic valve insufficiency and the moderate quality of the TTE probably explain the delay in diagnosis.

Traumatic aortic valve insufficiency was first described in 1830 by Penderleath [1]. Although the incidence of traumatic aortic valve lesion has increased with the prevalence of high-speed accidents, it is still extremely rare. To date fewer than 150 case reports concerning traumatic aortic valve injury have been published [2-6]. During surgery various anatomical lesions may explain acute aortic valve regurgitation including commissure rupture with or without an associated intima tear, or cusp avulsion. In the latter usually only one cusp is damaged, and the non-coronary cusp is usually involved. A few case reports describe rupture of two or three cusps [2,3]. Sometimes a proximal dissecting aneurysm explains the acute valve insufficiency. The most likely mechanism of aortic valve injury is a sudden increase in intrathoracic pressure induced by the trauma during the early diastole. This is the most vulnerable phase with the highest pressure gradient over the aortic valve, which may lead to rupture of the cusp or commissure [2,4-7]. The mitral valve is most vulnerable during late diastole or early systole due to a volume-loaded ventricle but traumatic lesions of the mitral or tricuspid valves are even less common than the aortic valve injury [5].

**Figure 2**: Chest x-ray on Day 8 showing extensive pulmonary oedema

**Figure 3**: Transoesophageal echocardiography with massive aortic insufficiency

AO = aorta, AR = aortic regurgitation jet, LV = left ventricle, LA = left atrium.
The clinical presentation of a patient with an acute aortic insufficiency is comparable with that of acute mitral valve insufficiency and differs from the more commonly found chronic aortic valve insufficiency. In chronic aortic valve insufficiency there will be dilatation of the left ventricle with adaptation of forward stroke volume and cardiac output despite the regurgitant volume. In acute onset aortic valve insufficiency, the normal left ventricle size cannot compensate for the regurgitant volume which leads to an increase in end diastolic pressure, increase in pulmonary capillary wedge pressure and the subsequent development of pulmonary oedema. Cardiac output decreases and cardiogenic shock can develop. Symptoms can vary from mild dyspnoea to haemodynamic instability with shock and may become progressive in time (from hours to weeks) after the trauma [6]. If dyspnoea is the only symptom, patients can be easily misdiagnosed as having a non-cardiogenic pulmonary problem.

Echocardiography is indispensable in confirming the presence and severity of aortic valve regurgitation. Severe aortic valve insufficiency is based on the following echocardiographic findings: vena contracta (narrowest central flow region of a jet) >6 mm (sensitivity 95%, specificity 90%), pressure ½ time < 200 ms, holodiastolic flow reversal in abdominal aorta, premature mitral valve closure and normal left ventricular size and function. Colour Doppler on TTE may underestimate regurgitation severity, particular when the jet is eccentric. However, with moderate quality visualization and persistent signs of congestive heart failure TEE is the most appropriate examination [2-8].

Acute traumatic aortic valve insufficiency accompanied by signs of pulmonary or haemodynamic instability, is an indication for immediate surgery. Depending on the anatomical diagnosis, valvuloplasty may be an option, but the long-term results appear unfavourable with a high rate of recurrence. This makes valve replacement the treatment of choice in most cases [2-8]. In our case, valve replacement was chosen because of co-morbidity and the age of the patient, which made re-operation for recurrence unattractive.

In conclusion acute aortic valve injury, although rare, must be considered in every patient with symptoms of congestive heart failure after blunt chest trauma. TTE should be part of the initial investigation and followed by TEE if visualization is inadequate and to assess severity once a diagnosis of aortic valve regurgitation is made.

References

1. Penderleath D. Case of death from rupture of one of the semilunar valves of the aorta. London Medical Gazette 1830;7:109

Figure 4: Rupture of the commissure between the right and the non-coronary cusps with associated intima tear

RCC = right coronary cusp, NCC = non-coronary cusp. Arrows indicate the rupture and associated intima tear.