Case report - Valves

Acute aortic and mitral valve regurgitation following blunt chest trauma

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Abstract

Traumatic rupture of intracardiac structures is an uncommon phenomenon although there are a number of reports with regards to rupture of the tricuspid, mitral and aortic valves. We report the case of a 25-year-old patient who presented with acute aortic and mitral valve regurgitation of traumatic origin. Both lesions were seen separated by 2 weeks. Pathophysiology is reviewed. The combination of both aortic and mitral lesions following blunt chest trauma is almost exceptional.

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1. Introduction

Aortic valve regurgitation after blunt chest trauma is an uncommon condition. Although associated intracardiac lesions have been described aortic and subsequent sequential mitral regurgitation seems to be extremely rare [1,2]. This report refers to a young patient who developed both lesions after a frontal car crash, and underwent subsequent sequential aortic and mitral valve replacement in a period of 4 weeks.

2. Case report

A 25-year-old previously healthy man had a frontal high-velocity car accident resulting in blunt craniothoracic trauma at the end of January 2003. He was admitted to a community hospital. Plain chest X-ray showed fractures of the left clavicle, first and second ribs. A computed tomographic scan was performed showing no intracranial lesions, but anterior and posterior mediastinal contusion with suspected periaortic involvement, left pheumothorax and bilateral lung contusion. Pleural drainage and non-invasive ventilation failed to control respiratory insufficiency. Congestive heart failure (CHF) developed. Transoesophageal echocardiogram (TEE) showed severe aortic valve regurgitation due to perforation of the non-coronary cusp. No other intracardiac lesions were detected.

On day three he was referred to our department with the diagnosis of severe aortic valve regurgitation resulting in intractable CHF. Physical examination showed sinus tachycardia, a 4/6 aortic diastolic murmur and third sound. Surgery was performed on an emergency basis upon admission. Intraoperative gross evaluation revealed a normal aortic annulus, ascending aorta and arch. The non-coronary cusp was found to be almost totally detached from the native annulus (Fig. 1). There was a 3 X 3 mm² subannular hematoma in the area of the non-coronary cusp. A 23 mm Carpentier–Edwards bovine pericardial xenograft (Edwards Lifesciences, Irvine, CA, USA) was implanted. The early postoperative course was initially uneventful and he was transferred to the referring hospital on day 9 for convalescence.

A transthoracic echocardiogram performed on day 12 after surgery showed normal function of the aortic implant and severe mitral valve regurgitation. TEE confirmed severe mitral regurgitation due to chordal rupture of the anterior leaflet of mitral valve, corresponding to the A1–A2 segments of the Carpentier classification. Four weeks after the first operation, mitral valve repair was attempted on an elective basis. At reoperation, primary chordae of A1–A2 segments were found to be ruptured at the level of the tip of the anterior papillary muscle. The papillary muscle looked otherwise normal on inspection. There was a 3 X 3 mm² perforation at the level of the mitral annulus close to the anterior commissure, which was identified to be closely

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Traumatic rupture of intracardiac structures is an uncommon phenomenon after blunt chest trauma. The most frequent lesion observed in this scenario is myocardial contusion. Valve involvement is less common, the most frequent being the aortic valve [3], followed by the mitral [4] and tricuspid [5]. In recent years there has been a rise in the incidence of these lesions due to proliferation of motor vehicle accidents, although the real prevalence might be underestimated. Mitral valve injury following blunt chest trauma was then replaced with a 27 mm Carpentier–Edwards bovine pericardial xenograft using the intravalvular implantation technique. He was discharged after a new echocardiogram showed normal function of both replacement devices. He is currently doing well 7 months after surgery.

3. Comment

Traumatic rupture of intracardiac structures is an uncommon phenomenon after blunt chest trauma. The most frequent lesion observed in this scenario is myocardial contusion. Valve involvement is less common, the most frequent being the aortic valve [3], followed by the mitral [4] and tricuspid [5]. In recent years there has been a rise in the incidence of these lesions due to proliferation of motor vehicle accidents, although the real prevalence might be underestimated. Mitral valve injury following blunt chest trauma is rare, with around 40 cases surgically treated reported in the English literature until July 2001 [6]. The first mitral traumatic rupture was reported in 1936 and the first successful repair in 1964 [7]. The first description of aortic valve injury was made in a necropsy case in 1830 [8]. There are <100 cases of aortic valve injury reported until 2002 [9].

A well-known mechanism that may account for both traumatic mitral and aortic regurgitation is an increase in intracardiac pressure during a vulnerable phase of the cardiac cycle. For the aortic valve this period is early diastole, whereas a vulnerable period for mitral valve and subvalvular apparatus occurs during late diastole and early systole. Tear or avulsion from the annulus of one aortic valve cusp is the most frequently observed aortic valve lesion, usually the non-coronary cusp [1] as seen in our patient. The most common mitral lesion is rupture of the papillary muscles, followed by the chordae tendineae, and a leaflet tear [4]. In our case, subacute presentation of mitral regurgitation could be related to a partial chordal rupture. Although we have to speculate considering that there was probably insufficient echocardiographic information when the patient was referred as this was an emergency case, it may happen that the subannular aortic hematoma could lead to a perforation of the leaflet at its annular implantation. Subsequent stress on a possibly injured subvalvular apparatus led to chordal rupture after 2 weeks. There is the possibility that mitral regurgitation may have gone unnoticed in the early echocardiographic study. We are aware of just a previous report of similar characteristics [10].

Diagnosis of acute aortic insufficiency is suggested by heart failure and/or a new diastolic heart murmur and history of blunt chest trauma. Clinical findings of patients with traumatic mitral injury include a wide range of settings varying from asymptomatic patients to acute cardiogenic shock. In our case, the mitral lesion was an incidental finding in transthoracic echocardiography after aortic valve replacement since it was clinically silent and had not been detected before the first operation. Nowadays transesophageal echocardiography is the non-invasive test of choice to diagnose these conditions as it offers a higher resolution and real-time assessment of both function and morphology.

The indications for aortic valve replacement versus aortic valve repair depend on the extent of the lesion in the damaged cusp or number of cusps involved. For the mitral valve, many surgical strategies have been described including a variety of repairs and replacement. The choice for a particular approach must be based on the extent of damage, accurate analysis of the mitral apparatus and surgeon’s technical expertise. In our patient we chose a tissue valve as he could be non-compliant with anticoagulation medication according to the patient’s relatives. In conclusion, patients with recent or past history of high-energy blunt chest trauma must undergo echocardiography to rule out intracardiac lesions either isolated or in combination. The association of aortic and mitral lesions as the result of blunt chest trauma is almost exceptional.

References


