Post-traumatic left ventricular pseudoaneurysm

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Abstract

Left ventricular pseudoaneurysms (LVPs) occur as a complication of myocardial infarction, cardiac surgery and, rarely, due to thoracic trauma, infective pericarditis or iatrogenesis due to accidental perforation of the myocardium. Ventricular pseudoaneurysms are acquired by blood-filled spaces outside the cardiac chambers communicating with the ventricle. We present a case of LVP presented after a blunt non-penetrating chest injury. The patient underwent successful aneurysmorrhaphy.

Keywords: Cardiac • Trauma • Ventricular aneurysm

INTRODUCTION

Left ventricular pseudoaneurysms (LVPs) form when a cardiac rupture is contained by adherent pericardium or scar tissue. Myocardial infarction accounts for most LVPs, followed by cardiac surgery, trauma, infection and iatrogenesis [1]. Non-penetrating trauma of the chest can result in severe damage to the heart. In some patients, these injuries result in extreme symptoms, but in some they may be occult. We describe a case of post-traumatic LVP following blunt chest injury presented as cardiac failure, who underwent a successful repair of a pseudoaneurysm.

CASE REPORT

A 25-year old male presented with complaints of persistent chest pain, palpitations and shortness of breath on exertion. His history included a blunt injury on the left side of the chest from an automobile accident four months previously. He was haemodynamically stable without any pulmonary and cardiac abnormality, clinically as well as radiologically. The patient was managed conservatively, and no active intervention was required. Over the next few days the patient developed gradually worsening chest pain, palpitations and shortness of breath.

On examination, his vitals were normal. Cardiac examination revealed diffuse apical systolic thrust with a visible and palpable double impulse. A soft systolic murmur was heard over the precordium. The chest skiagram showed a cardiomegaly with a localized radio-opacity at the left border of the cardiac silhouette near the ventricular apex. Sinus tachycardia was present in electrocardiography. Echocardiography showed a large pseudoaneurysm of 6 × 14 cm lateral and posterior to the left ventricle connected by a narrow neck of 2 cm diameter to the posterolateral wall of the left ventricle. The ratio of the maximum internal diameter of the orifice to the maximum internal diameter of the cavity was 0.14. Doppler echocardiography showed turbulent bidirectional flow between the pseudoaneurysm and the left ventricle cavity. Multislice cardiac computerized tomography revealed a large pseudoaneurysm of 10.3 × 11.1 × 6.0 cm communicating with the left ventricle through a 20 mm defect in the left ventricular free wall (Fig. 1). The patient was referred for surgical intervention.

At operation through a median sternotomy, the aneurysmal sac was identified arising from the lateral wall of the left ventricle with dense pericardial adhesion. A standard mild hypothermic cardiopulmonary bypass instituted with antegrade blood cardioplegia. Pericardial adhesionolysis was done and the aneurysmal sac was opened. There were no blood clots in the sac, and a 2-cm diameter ‘neck’ of the aneurysm in the left ventricular wall was visualized. The direct closure of the ‘neck’ of the sac with a double layer of 3/0 Prolene sutures (Ethicon, Somerville, NJ, USA) was done using a piece of the aneurysmal wall as felt (Fig. 2). The patient came off bypass uneventfully. The patient was discharged from the hospital on the 6th post-operative day. Histopathological examination revealed that the sac wall was lined by thick fibrocollagenous tissue showing a loose area of myxoid degeneration covered with a layer of pericardial tissue. No cardiac muscle was seen in the section.

Repeat echocardiography before discharge and 6 months later revealed a normal cardiac function without any residual aneurysmal sac.
DISCUSSION

LVPs occur as a complication of myocardial infarction or after cardiac surgery. However, there are several cases of LVP formation after penetrating injuries, closed thoracic trauma, infective pericarditis and iatrogenic [1, 2]. A review of non-penetrating cardiac trauma indicated that 80% of blunt injuries resulted from automobile accidents [3, 4].

Rapid deceleration of the body with blunt chest trauma forces the heart against the chest wall and provokes excessive shear forces on cardiac structures. This can lead to excessive intraventricular pressure and can result in an acute rupture of the ventricular free wall, the ventricular septum or cusps of the aortic valve. The type and the extent of blunt cardiac injuries vary widely.

Congestive heart failure, chest pain and dyspnoea are the most frequently reported symptoms. Arterial embolizations and arrhythmias have also been described, while >10% of patients are asymptomatic [3]. Diagnosis may be missed because of an atypical presentation and/or a delayed presentation.

Electrocardiographic changes are usually non-specific in post-traumatic LVPs.

Cardiomegaly is the most common chest X-ray finding, while sometimes it showed a localized bulge on the cardiac silhouette. The echocardiographic features typical of pseudoaneurysms include a sharp discontinuity in the endocardial image at the site of communication of the pseudoaneurysm with the left ventricular cavity and an orifice that is relatively narrow in comparison with the diameter of the pseudoaneurysm. This finding differs from those in a true aneurysm, which typically has a large communicating neck. The ratio of the maximum internal diameter of the orifice to the maximum internal diameter of the cavity never exceeded 0.5 in the cases of a pseudoaneurysm, while this was between 0.9 and 1.0 in the cases of a true aneurysm [5]. Pulsed-wave and colour flow Doppler echocardiography features are visualization of the high-velocity, turbulent, bi-directional flow between the left ventricle and a pseudoaneurysm [6]. On multislice computed tomography, pseudoaneurysms are characterized by an abrupt disappearance of the myocardial wall at the border of the pseudoaneurysm [6]. These findings were present in our case also.

Magnetic resonance imaging [7] and contrast angiography [8] are the other methods used to detect pseudoaneurysms.

Most authors favour surgical intervention [1, 3, 9], since untreated pseudoaneurysms have a 30–45% risk of rupture. The surgical repair of post-traumatic LVPs presents several options with a reported mortality above 7% [6]. A patch is preferable in large defects or defects near the base of the heart, to avoid excessive traction on the myocardium or the distortion of the circumflex artery and coronary sinus. The direct primary suture repair is an effective approach in most post-traumatic LVPs due to the absence of myocardial disease. We did direct closure of the ventricular opening with a double layer of 3/0 Prolene sutures (Ethicon) using a piece of the aneurysmal wall as felt.

In conclusion, myocardial contusion is frequent in patients with severe non-penetrating chest trauma, and the increase in automobile accidents in recent times increases the possibility of cardiac injury without obvious external injuries. Once cardiac injury is suspected, structural damage must be excluded using echocardiography, which should be repeated after several days due to the possibility of the delayed rupture. Even extensive injury to cardiac structures can be repaired with good results.

Conflict of interest: none declared.
REFERENCES


eComment. Left ventricle pseudoaneurysm secondary to infective endocarditis

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In the interesting case report presented by Singh et al. [1], the patient underwent a successful aneurysmorrhaphy four months after a blunt non-penetrating chest injury. What is remarkable in their report is that the authors make no allusion in their discussion to a previous and well-known aetiology of left ventricle pseudoaneurysm which is infective endocarditis.

We [2] and others [3] have published case reports about false aneurysm arising from the left ventricle in patients with infective endocarditis. Myocardial abscess, pseudoaneurysm of the ventricle and cardiac rupture rarely complicate infective endocarditis. Presentation can vary from pericardial effusion to cardiac tamponade occurring in association with annular abscess or myocardial perforation [4]. Formation of a myocardial abscess may be secondary to multiple coronary septic emboli or to local spread from the infected endocardium to the nearby myocardium. Rupture of the myocardial abscess is often fatal, but when the rupture is contained by scar tissue, this entity is known as a post-infective endocarditis pseudoaneurysm. As long as the pseudoaneurysm remains contained, the haemodynamic state will remain good but the chances of survival without surgery remain slim [5].

Conflict of interest: none declared.

References