Case Report

A Blunt Trauma Chest Causing Left Ventricular Pseudoaneurysm - An Unusual Presentation

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ABSTRACT

Left ventricular (LV)pseudoaneurysms occur as a complication of myocardial infarction, cardiac surgery and rarely due to thoracic trauma, infective pericarditis or iatrogenically due to accidental perforation of myocardium. Ventricular pseudoaneurysms are acquired by blood filled spaces outside the cardiac chambers communicating with the ventricle. We present a case of left ventricular pseudoaneurysm presented after a blunt non penetrating chest injury. The peculiarity in this case was his delayed presentation and significant lack of symptoms till a month before presentation. The patient underwent successful aneurysmectomy after detail investigations and is doing well in the subsequent follow ups.

Introduction :

LV aneurysms are of two types : true and false (or pseudo). True aneurysms are sequelae of transmural myocardial infarction. They vary widely in size and compliance, infrequently undergo progressive expansion and seldom rupture. False or pseudoaneurysms are rare complications of myocardial infarction, trauma or iatrogenic perforation and represent a contained myocardial rupture. It is important to recognize a pseudoaneurysm because likelihood of spontaneous rupture is high. Unlike a true aneurysm in which the wall consists of dense fibrous tissue with excellent tensile strength the wall of pseudoaneurysm is composed of organizing thrombus and parts of epicardium and pericardium[1]. Given the propensity for pseudoaneurysms to rupture leading to cardiac tamponade, shock and death compared with a more benign natural history for true aneurysms, an accurate diagnosis of these conditions is important. True aneurysms usually call for an elective surgery[2]. We present a case of LV Pseudoaneurysm as an unusual presentation of blunt chest trauma who responded well to surgical treatment.

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Case :

A 36 years old male, presented with complaints of class II dyspnoea on exertion and a pulsating mass on left side of chest. There was no history of hypertension, diabetes or symptoms suggestive of ischemic heart disease or any addictions. He, however, did give history of fall from second floor two years back for which he took treatment from local doctor. On examination he had tachycardia (heart rate 110 / min), BP 110/70 in right arm supine position and prominent neck pulsations. Local examination revealed a large pulsatile mass measuring approximately 10 x 10 cm present in epigastrium and left hypochondrium with a bruit over it. Per Abdomen examination showed mild hepatomegaly. Rest of examination was essentially unremarkable.

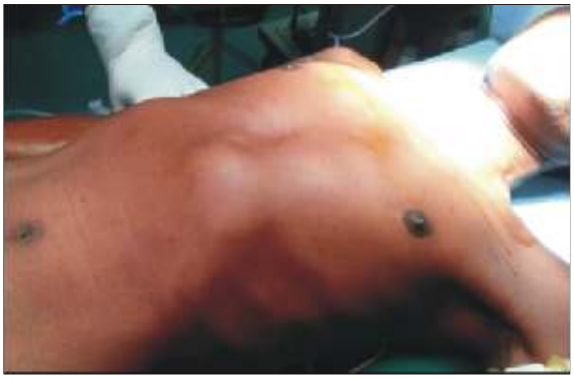

Figure 1

Routine investigations revealed Hb-9.6 gm.%, Blood sugar, kidney function tests, liver function tests including INR and cardiac enzymes were normal. HIV and HBsAg were negative. ECG showed non-specific ST-T changes. Chest x-ray showed a large mass occupying the lower left hemithorax with shifting of cardiac silhouette and mediastinum to right ***(Fig. 2).*** 2D Echocardiography and Doppler study revealed a dilated LV and presence of a large aneurysm of size 13.8 x 6.8 cm present at apicolateral wall of LV with a communicating neck measuring 2.7 cm in diameter ***(Fig. 3).*** Colour Doppler imaging revealed a turbulent bidirectional flow through the defect ***(Fig. 4).*** Biventricular systolic function was otherwise normal and there was no regional wall motion abnormality.

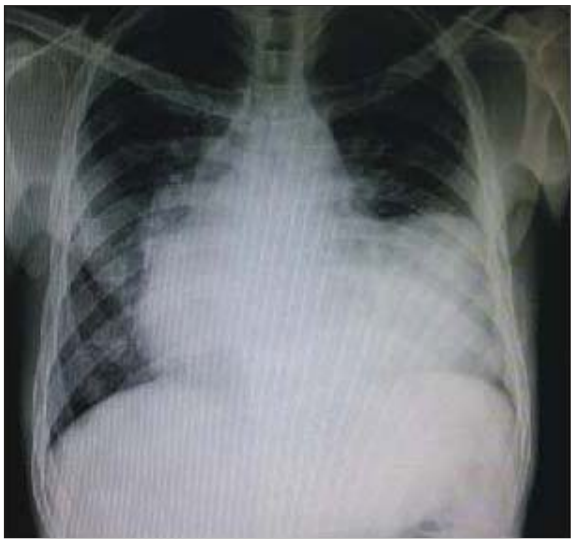

Figure 2 : **Chest X-Ray showing a large mass occupying the lower left hemithorax with shifting of mediastinum to right**

Patient was taken up for multislice cardiac CT which confirmed presence of large aneurysm communicating with LV cavity ***(Fig. 5).***

Cardiac MRI again showed a large 14.2 x 13.3 x 15.6 cm irregular, partially thrombosed aneurysm arising from LV apex. The neck measured 2.5 cm in diameter. The aneurysm extended upto lateral chest wall, anteriorly upto anterior chest wall and inferiorly displaced stomach, diaphragm and spleen ***(Fig. 6).***

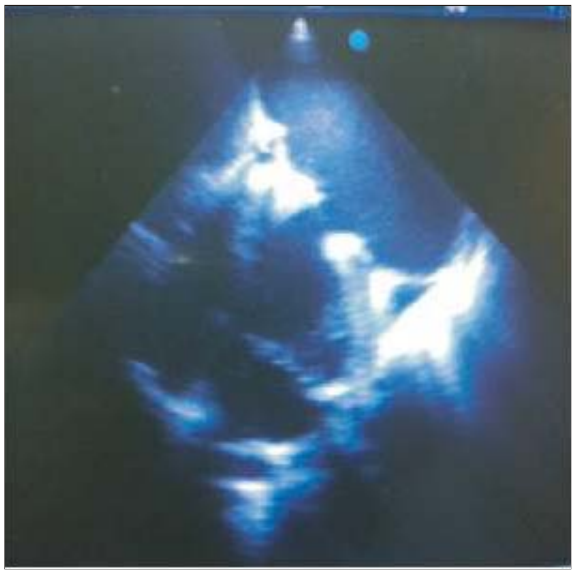

Figure 3 : **2D Echocardiographic image showing the communication between LV cavity and the pseudoaneurysm**

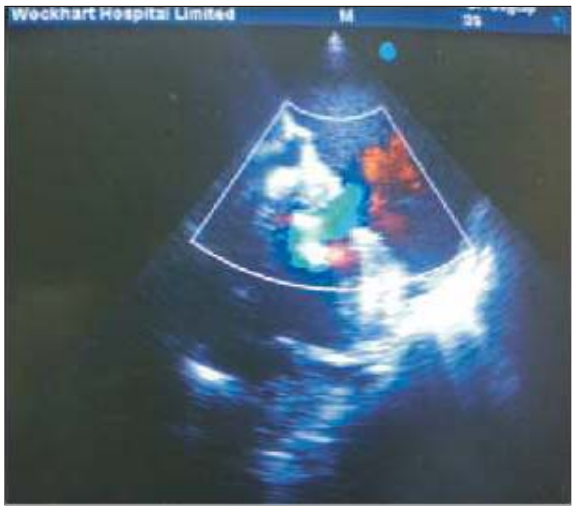

Figure 4 : **Colour Flow Image depicting the blood flow between the LV cavity and the pseudoaneurysm**

After initial stabilization the patient was taken up for surgical repair. Operative findings revealed severe pericardial adhesions over both the ventricles. Large aneurysmal sac of 15 x 15 x 14 cm size with clots within was seen with a communicating neck of size 3 x 3 cm over lateral wall of LV near apex. Aneurysmal wall was dissected, sac was opened and clots were removed. Neck was identified and linear

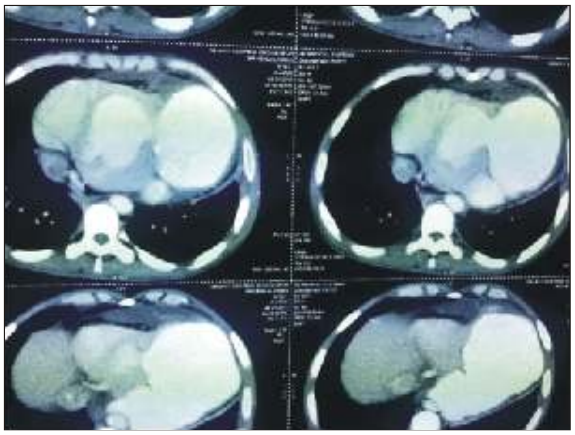

Figure 5 : **Multislice Cardiac CT showing the large out pouching and the communication between two cavities**

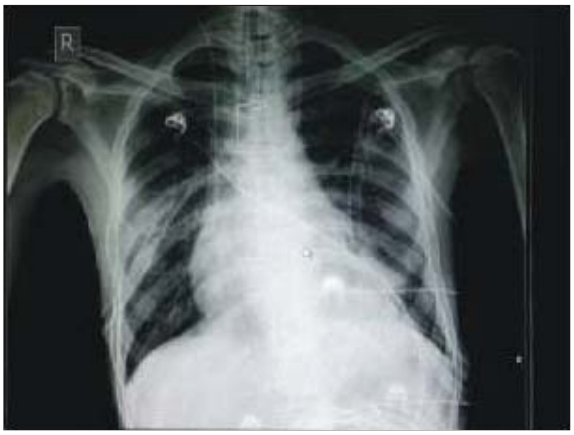

Figure 7 : **Post Operative CXR showing mild cardiomegaly and absence of opacity in left lower hemithorax**

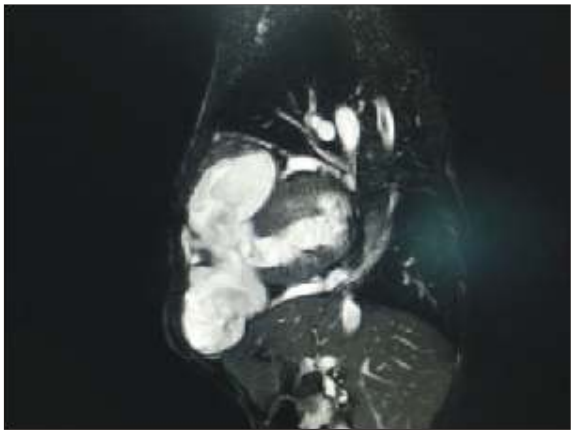

Figure 6 : **Cardiac MRI showing large, irregular, lobulated aneurysm displacing the abdominal viscera**

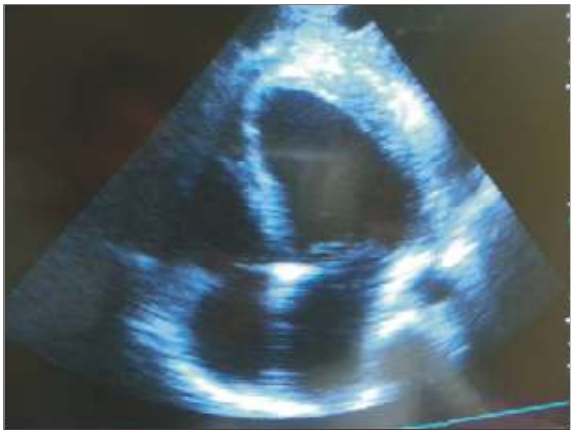

Figure 8 : **Post operative 2D Echocardiographic image showing well maintained LV geometry**

closure of neck was done maintaining LV geometry. The patient withstood the procedure well.

Post-operative chest x-ray and echocardiographic images showed normalization of cardiac structures ***(Fig. 7 & 8).*** Post-operative course was uneventful and the patient was discharged on ninth post-operative day. At subsequent follow-ups he was found to be in NYHA Class I with no recurrence of pseudoaneurysm.

Histopathological examination revealed sheets of RBCs along with fibrin and neutrophils i.e. fibrovascular tissue. No atypia or inflammation were seen. No cardiac muscle seen.

Discussion :

Post traumatic LV pseudoaneurysm is an unusual presentation of blunt trauma chest. This condition has been attributed to myocardial contusion or to a direct vascular lesion leading to myocardial necrosis[3]. LV pseudoaneurysms are prone to rupture (estimated risk 30% - 45% based on prior studies) akin to a "ticking time-bomb" and thus a surgical approach is often undertaken[4,5,6]. A study evaluated the clinical presentation, diagnostic accuracy of imaging modalities, outcomes and prognosis of 290 patients with LV psuedoaneurysm[7]. Congestive cardiac failure, chest pain, dyspnoea were the most frequently reported symptoms but > 10% patients

were asymptomatic. Physical examination revealed a murmur in 70% of patients.

The majority of patients had ECG abnormalities (usually nonspecific ST-T changes) and only 20% had ST segment elevation. More than 50% of patients may demonstrate the appearance of a mass on chest x-ray which provides an important clue to the diagnosis. Clinical features of chronic pseudoaneurysm are nonspecific and frequently detected incidentally[8].

Colour flow imaging or contrast echocardiography can be helpful in locating the site of free wall rupture. Negative echocardiographic findings should not exclude myocardial rupture if clinical suspicion is high. In this case another imaging technique such as Cardiac MRI should be considered[9].

A pseudoaneurysm is usually characterized by a small neck communication that connects LV and aneurysmal cavity (the ratio of the diameter of the entry and maximal diameter of pseudoaneurysm is usually less than 0.5) although some may have a wide neck. There is always to and fro blood flow through the rupture site that can be documented with Doppler and colour flow imaging[9].

Complications include Congestive Cardiac Failure, arrhythmias, mediastinal rupture, arterial embolization and even sudden cardiac death.

Surgery remains the treatment of choice in symptomatic patients. Untreated pseudoaneuryms have 30-45% risk of rupture. Surgical repair of post traumatic LV pseudoaneurysmshas reported mortality above 7%[5]. Surgical treatment in asymptomatic individuals however remains controversial. A patch is preferable in large defects near base of heart to avoid excessive traction on myocardium or distortion of left circumflex artery and coronary sinus. Direct primary suture repair is an effective approach in most due to absence of myocardial disease.

To conclude, it is prudent to keep in mind that an insignificant cardiac injury may have a delayed presentation of symptoms. In cases of blunt chest trauma suspected structural damage must be excluded by the simple and readily available modality that is echocardiography. Echocardiography should be repeated several days later to rule out possibility of delayed myocardial rupture. In the present era even extensive injuries to cardiac structures can be repaired with good results.

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