INTRODUCTION

Pseudoaneurysm occurs as an incomplete rupture of heart when it is sealed with thrombus and pericardium. It is called pseudo as its wall does not contain any myocardial element. It occurs as a rare complication of myocardial infarction, infective endocarditis or chest trauma, etc. It is to be treated urgently by surgical repair as it has high risk of rupture. Few cases so far are reported with tuberculosis as a cause for it.

CASE HISTORY

Sixteen years boy presented to us with complaints of gradually increasing bulging of left side of chest wall (precordium) since 3 years. He had localized chest pain on left side with dyspnoea on severe exertion since 3 years. He had high grade fever with chills for the last 30 days. He had no history of chest trauma, joint pain or swelling, skin, eye or genital lesion, no seizure episode.

On general physical examination patient had blood pressure of 110/80mmHg and pulse rate of 96/min regular, normal volume and character. He had oral temperature of 101°F.

On cardiovascular system examination chest wall was asymmetrical with precordium bulge in 4th-6th intercostal space, 4x4 cm in size in mid clavicular line. Over the above mentioned area there was see-saw movement. Apex beat felt in 5th intercostal space just lateral to mid clavicular line, diffuse occupying 3cm² area without parasternal heave or thrill. Localised pericardial rub heard over 5th intercostal space in mid clavicular line. Heart sounds were normal with no murmur. There was soft non-tender splenomegaly 5 cm below costal margin. Respiratory, nervous system and GIT examination were normal.

On investigations Hb-8.3g%, TLC-15000/cumm, ESR-65, liver, renal function tests and urine examination were normal. PBF for malarial parasite and Widal test were negative. Blood culture was positive for coagulase positive staphylococcus aureus. Fundus examination was normal. On X-ray chest there was cardiomegaly with cardiothoracic ratio 2.73 : 2. Ultrasonography showed splenomegaly. In sequential ECG there was ST depression with T-wave inversion in V5-V6 leads, serial cardiac enzymes were normal. 2D-Echo cardiography showed large pseudoaneurysm at inferolateral wall of left ventricle with sac 45mm x 45mm and neck 40 mm in size, there was pedunculated vegetation 20 mm x 6 mm size, highly mobile at the neck of pseudoaneurysm with mild LV dysfunction. However possibility of blood clot was also kept as differential diagnosis.

CT angiography of thorax showed large pseudoaneurysm arising from left posterolateral wall of left ventricle (Figs. 1a and 1b) measuring about 6cm in diameter with peripheral egg shell calcification and partial intraluminal thrombus. There was left pleural effusion with collapse of left lower lobe of left lung.
Multiple partially calcified mediastinal hilar and cervical lymph nodes were present.

Biopsy cervical lymph node – showed granuloma consisting of caseous necrosis surrounded by epithelioid cells and Langhan’s type of giant cells.

Patient was put on antibiotics as per infective endocarditis guidelines. During the course of treatment patient developed ischemic infarct in right MCA territory leading to left hemiparesis. Final diagnosis was pseudoaneurysm of left ventricle, tubercular etiology with infective endocarditis. Patient was immediately transferred to cardiovascular surgeon, where urgent surgery was advised with moderate risk. There patient refused for operation and left against medical advice with the risk of sudden death explained.

**DISCUSSION**

Pseudoaneurysm occurs as an incomplete rupture of heart when organizing thrombus and hematoma together with pericardium seal the rupture of left ventricle. Its wall is composed of only hematoma and pericardium with no myocardial element which differentiates it from true aneurysm, whose wall contains myocardial element.

Pseudoaneurysm maintains its communication with cavity of left ventricle by a narrow neck. Pseudoaneurysm may be larger than the ventricle itself. It can occur as a complication of myocardial infarction, cardiac surgery, infective endocarditis, chest trauma, tuberculosis, rheumatoid arthritis, Kawasaki’s disease, Behcet's syndrome.

**Mechanism of formation** When there is transmural infarction there is localized pericarditis causing adhesion of visceral and parietal pericardium, infarcted myocardium ruptures causing extravasation of blood in adherent pericardium causing progressive enlargement of aneurysmal sac with development of thrombus within the sac. Mechanism of formation of pseudoaneurysm in tuberculosis is by rupture of tubercular myocardial abscess with hematoma formation around it which is covered with pericardium.

Pseudoaneurysms have higher risk of rupture and sudden death as compared to true aneurysm. Signs and symptoms of pseudoaneurysm are bulging of chest wall over precordium with see - saw movement of chest wall, There may be chest pain, dyspnoea, signs of CHF, arrhythmias, embolism, to and fro murmur and pansystolic murmur. Investigations to confirm pseudo- aneurysm are 2D echocardiography, contrast angiography, CT and MRI.

When confirmed pseudoaneurysm should be repaired urgently with the correction of defect as it has high risk of rupture.

**REFERENCES**