

A case of myopericarditis

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ABSTRACT

Acute myopericarditis is an entity which can mimic presentation of acute myocardial infarction (AMI). We present a 22 years old, previously healthy man who came with history of chest pain, breathlessness, and low grade fever. On examination he was detected to have raised JVP, pulsus paradoxus and right sided pleural effusion. ECG revealed ST-segment elevation and PR segment depression in non-contiguous leads. Troponin T was elevated. Acute tuberculous myopericarditis was diagnosed on the basis 2 D ECHO findings, laboratory tests, as well as the changes observed in electrocardiograms (ECG) and in the cardiac enzyme levels. Pleural fluid aspiration revealed lymphocytic predominant, exudative fluid with ADA in suspect range. The case highlights the importance of taking proper history, thorough examination. High index of suspicion supported by Transthoracic 2 D ECHO and cardiac MRI confirms the diagnosis.



Fig. 1 : Raised JVP

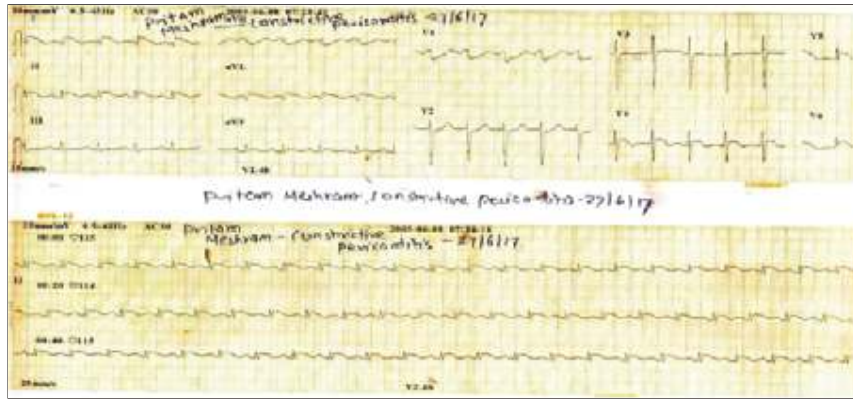


Fig. 2 : ECG showing ST and PR Segment deviation



Fig. 3 : Chest X-ray

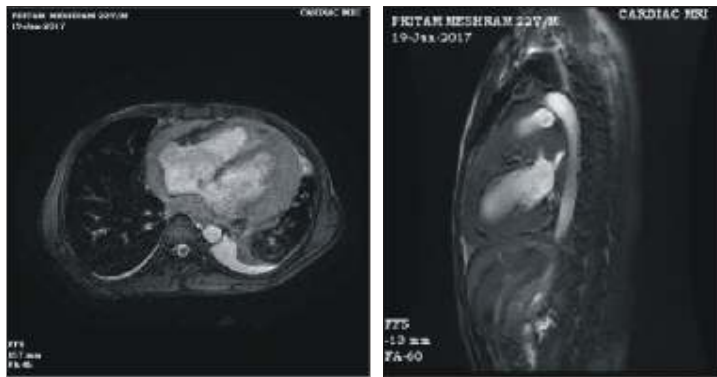


Fig. 4 & 5 : Cardiac MRI shows pericardial thickness

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Introduction :

A 22 years old male patient, laborer by occupation, presented with complaints of chest pain over left side of chest which was more in supine position and used to get relieved in sitting position, palpitations, breathlessness, dry cough and low grade fever with

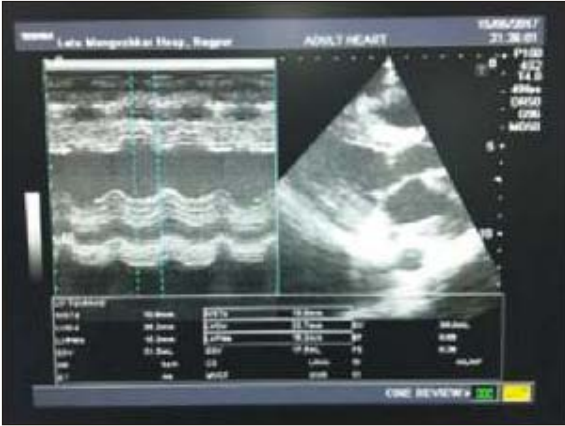


Fig. 6 : 2D ECHO showing thickened pericardium

evening rise and weight loss since one month. On examination he appeared sick and restless, was febrile. Pulse rate was 148/min. with pulsus paradoxus, RR 52/min. and BP 100/70 mmHg in right arm supine position. His JVP was raised and Kussmaul's Sign was positive. Rest of the general examination was unremarkable. His CVS examination revealed pulsatile precordium with diffuse apex beat, normal heart sounds with Pericardial knock. RS examination revealed pleural effusion on left side. Rest of the systemic examination was normal.

Investigations : ESR 38, Trop T +ve, Mantax Test -ve. Rest Blood investigations were within normal limits.

ECG : sinus tachycardia and with ST Elevation in Lead I, II, aVL, aVF and V3-V6 and depression of PR segment in aVR.

CXR (PA) view : cardiomegaly with blunting of left CP Angle.

Trans-thorasic 2-D Echocardiography : Increased echogenicity of whole lateral wall of left ventricle with akinesia, parietal pericardial thickening of 14 mm lateral to free wall of left ventricle and around 10 mm over free wall of right ventricle. There was thin rim of pericardial fluid with e/o cardiac tamponade, ejection fraction was normal. Thus there was evidence of myocardial and pericardial involvement.

Cardiac MRI : thickened pericardium of 1.9 cm along the left lateral wall, with thin rim of pericardial effusion.

Pleural fluid : showed exudative picture with

lymphocytic predominance and ADA (33.70), which was in suspect range.

On the basis of findings & investigations patient was labeled as a case of subacute constrictive myopericarditis secondary to tuberculosis. He was started on first line anti Koch's treatment (HRZE) and Injectable steroids followed by oral steroids to which patient showed significant response. Patient was referred to higher center for surgical management thereafter.

Constrictive pericarditis CP is a chronic thickening of pericardium, caused by pericardial fibrosis, fused pericardial membranes and eventually calcification, leading to an impaired filling of ventricles and consequently reduced ventricular functions. Constrictive pericarditis usually leads to chronic diastolic heart failure¹. Causes include idiopathic, iatrogenic, infections like tuberculosis, viral, fungal or parasitic infections and autoimmune disorders².

Normal pericardium does not produce electrical impulses that affect the electrocardiogram, and the ST-T changes seen in pericarditis are believed to be because of inflammatory injury of a thin layer of myocardium beneath the visceral pericardium which also raises the of cardiac markers.^{3,4}

European Society of Cardiology have suggested that "pericarditis" should be the term used when pericarditis is unaccompanied by any evidence of myocarditis; while "myopericarditis" be used when there is evidence, such as widespread ST-segment elevation of myocardial involvement, and "perimyocarditis" be used when pericarditis is the dominant feature associated with myocarditis.⁵

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