

Title : A Case of Tuberculous Pericardial Effusion

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Abstract

A 75-year-old male presented with severe breathlessness and was found to have a Clinical features of massive pericardial effusion leading to cardiac tamponade. There was neither previous exposure to tuberculosis nor any suggestion of immunosuppression. Repeated analysis of pericardial fluid established a tuberculous origin.

Keywords:

Pericardial effusion, pericardiocentesis, tuberculosis, cardiac tamponade

Case report

A 75-year-old male with no past history of Respiratory or cardiac disease Presented with breathlessness and tiredness following a recent chest infection. He had no past history of asthma or tuberculosis. On examination, he was afebrile, pulse rate 97/min, high volume, pulses paradoxus (30mm of Hg), blood pressure 130/86 mmHg, respiratory rate 44/min, JVP raised and heart sounds were muffled. There was no peripheral oedema, hepatosplenomegaly or lymphadenopathy. Laboratory results were normal apart from serum albumin 27 g/l (35–50), globulin 37 g/l (18–36), ESR 55 mm (1–20) and CRP 80 mg/l (-5.0). ECG showed low voltage complexes, sinus tachycardia and inferior lateral ischaemia (fig-1). Chest X-ray indicated left basal consolidation and pericardial effusion. Cardiac silhouette was slightly enlarged and globular. Echocardiogram showed a massive pericardial effusion with fibrin strands and visceral pericardial thickening. There was cardiac tamponade. An ultrasound guided single lumen catheter was inserted. Over 1st 24 hours 750 ml and next 48 hours 150 ml of straw-coloured fluid was drained. Samples were sent daily for analysis. The fluid was an exudate. Samples sent on the 6th and 7th day stained positive

for AFB. Culture identified Mycobacterium tuberculosis sensitive to all anti-tuberculous drugs. He was managed with first line anti-tuberculous Therapy for six months (2EHRZ + 4HR) along with short course of steroids (The dose for pericarditis is prednisolone 60 mg daily tapered off over four to eight weeks).^[1]

Discussion : Extrapulmonary tuberculosis occurs in 20% of patients with tuberculosis^[2], which rises to over 50% in people with HIV. Tuberculous pericarditis is seen in 1–8% of these patients. The route of spread to pericardium is usually from mediastinal or hilar lymph nodes by lymphatic or from lung by haematogenous spread and rarely as part of miliary tuberculosis. Tuberculous pericarditis can present with recurrent pericardial effusion without any history or symptoms of tuberculosis.

Fig. 2 : Showing bilateral CTEV

Thickened pericardium & fibrinous strands^[3] on echocardiography, predominant lymphocytes and high adenosine-deaminase activity^[4] in pericardial fluid are useful indicators in diagnosing tuberculous effusion. Exudative pericardial coating is sensitive but less specific for tuberculosis (found in malignant and purulent effusions). PCR of pericardial fluid for mycobacterium tuberculosis is the gold standard diagnostic test in cases where other tests are negative^[5].

Treatment of tuberculous pericarditis is by standard antituberculous chemotherapy. Corticosteroids help in rapid improvement of symptoms and reduce mortality, but do not influence the resolution of pericardial effusion^[6].

Early cardiac tamponade is a good predictor of subsequent constrictive pericarditis^[7] presenting in up to 50% of patients. Where complete drainage of fluid by pericardiocentesis is not possible, transcatheter intra pericardial urokinase^[8], if given early, helps fibrinolysis and drainage.

Incidence of tuberculosis is rising due to HIV, migrant population and drug resistance. Tuberculous pericardial effusion in an elderly patient with no risk factors or previous exposure to tuberculosis is rare. Our case demonstrates the need for a high index of suspicion for tuberculosis in any case of pericardial effusion. Repeated samples for analysis may be more productive than a single sample. When available PCR should be the choice of investigation. PCR testing may

decrease the number of cases classified as idiopathic.

Early diagnosis, antituberculous chemotherapy and high dose steroids should decrease the incidence of pericardial constriction and mortality following tuberculous pericarditis.

Key points .

- Tuberculous pericardial effusion though uncommon is being increasingly found, especially in immunosuppressed individuals.
- Thickened pericardium and fibrinous strands on echocardiography are suggestive of infective etiology.
- Tuberculous pericarditis can present without any previous history of exposure to tuberculosis. A high index of suspicion should be maintained in every case of pericardial effusion.
- Repeated samples for analysis may be more productive than a single sample. PCR is diagnostic and is the gold standard.

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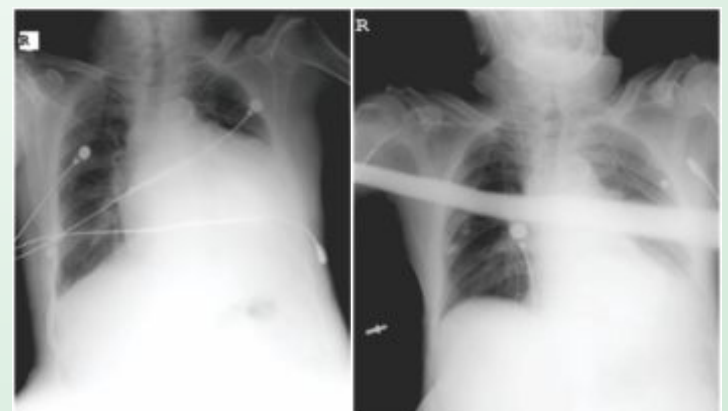
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Fig 1 Low Voltage ECG before pericardiocentesis



Fig 2 ECG after pericardiocentesis. Note improved voltage



X-ray Chest PA view Fig 3 before Pericardiocentesis,
Fig 4 after Pericardiocentesis

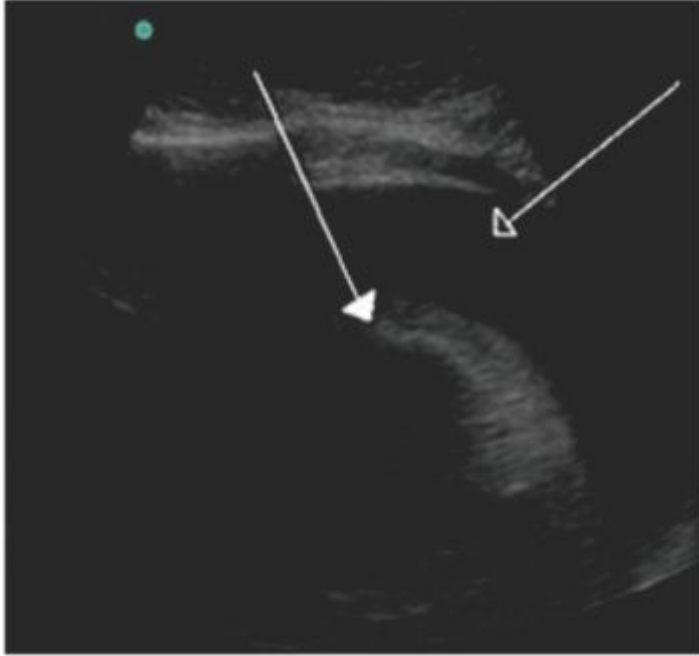


Fig 5- 2D Echo showing Pericardial Effusion and Right Ventricular Diastolic Collapse



Fig 6 and 7- Pericardiocentesis being done and pericardial drain in the Medical ICU of Padmashree Dr. Vikhe-Patil Memorial Hospital, Ahmednagar