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Case Report Open Access

Tuberculosis Pericarditis: Case Report

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Abstract

Tubercular pericarditis manifesting as cardiac tamponade with large pericardial effusion is relatively rare. We report a case of cardiac tamponade due to tubercular pericardial effusion with severe left ventricular systolic dysfunction.

Keywords: Cardiac tamponade; Tuberculous pericarditis; Severe left ventricular systolic dysfunction; Emergency pericardiocentesis; PCR in Tb pericarditis

Introduction

In endemic regions, *Mycobacterium tuberculosis* (MTb) is the commonest causative agent of pericardial disease which is found in 1-2% instances of pulmonary tuberculosis and is known to cause long term disability as well as high mortality even with Anti-Tubercular Therapy (ATT) [1-7]. MTb is responsible for up to 4% of acute pericarditis and 7% cases of cardiac tamponade which is in contrast to first world countries [7,8].

In spite of availability of effective ATT, HIV pandemic has caused increased burden of tuberculosis (TB) cases as well as Extra-Pulmonary Tuberculosis (EPTB) cases, which in turn poses a great challenge to physicians, microbiologists and pathologists in early diagnosis and prompt treatment [8,9].

MTb affecting pericardium can result in pericardial effusion apart from other sequel like cardiac tamponade and constrictive pericarditis [10].

Case Report

A 64 year-old male presented to cardiac care unit with a 1 month history of worsening breathlessness, generalized weakness and 5 days history of chest pain associated with sweating & palpitation at 2 am on 10th August 2018.

Patient is a farmer by occupation, non-smoker & non-alcoholic; not a known case of any significant illness.

At the time of admission, general condition of patient was poor; he was restless, tachypnoeic (RR-30/min), in hypotension (BP-80/60 mmHg) with tachycardia (HR-102/min).

On examination, patient heart sounds were muffled and neck veins were distended. An ECG showed, low voltage complexes in all leads with sinus tachycardia [Figure 1].

Immediate bed-side echocardiography revealed a large circumferential pericardial effusion measuring 24 mm with evidence of cardiac tamponade & severe LV systolic dysfunction (EF-10%). Bedside Echo guided percutaneous pericardiocentesis was performed to achieve hemodynamic stability; a total of 1500 mL straw coloured pericardial fluid aspirated and pericardial pigtail catheter was kept *in situ*. A 150 mL fluid was aspirated on 2 consecutive days. Chest X-ray revealed right paracardiac patchy consolidation with cardiomegaly [Figure 2].

A serology for HIV, HBsAg and HCV was negative. Pericardial fluid analysis was shown in Table 1.

	Pericardial fluid	Serum
Adenosine deaminase	74.9 IU/L	--
Lactate dehydrogenase	1609 U/dL	332 U/dL
Protein	4.3 mg/dL	6.1 mg/dL
Sugar	19 mg/dL	--
Cytology	Neutrophils-80% Lymphocytes-15% Epithelial cells-5% No malignant cells	--

Table1: Biochemical analysis of pericardial fluid and serum.

Pericardial fluid was negative for microscopy, AFB and culture yielded no bacterial or fungal growth after 48 hrs.

MTb culture on Lowenstein-Jensen (LJ) media yielded growth of *Mycobacterium* species after 39 days of incubation.

PCR analysis was positive for Tb complex (includes MTb, *M. bovis*, *M. microti*, *M. africanum*).

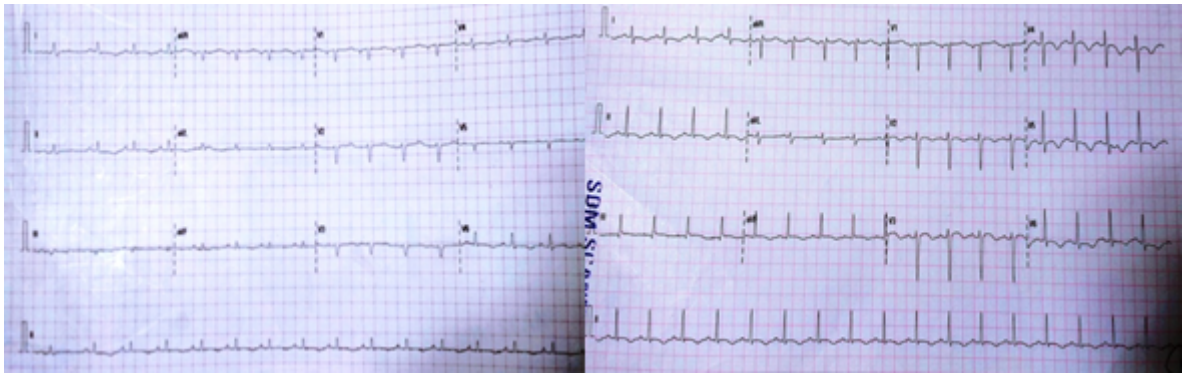

Figure 1: Showing low voltage complexes in all leads with sinus tachycardia on admission in first ECG; second ECG at the time of discharge was showing significant improvement.

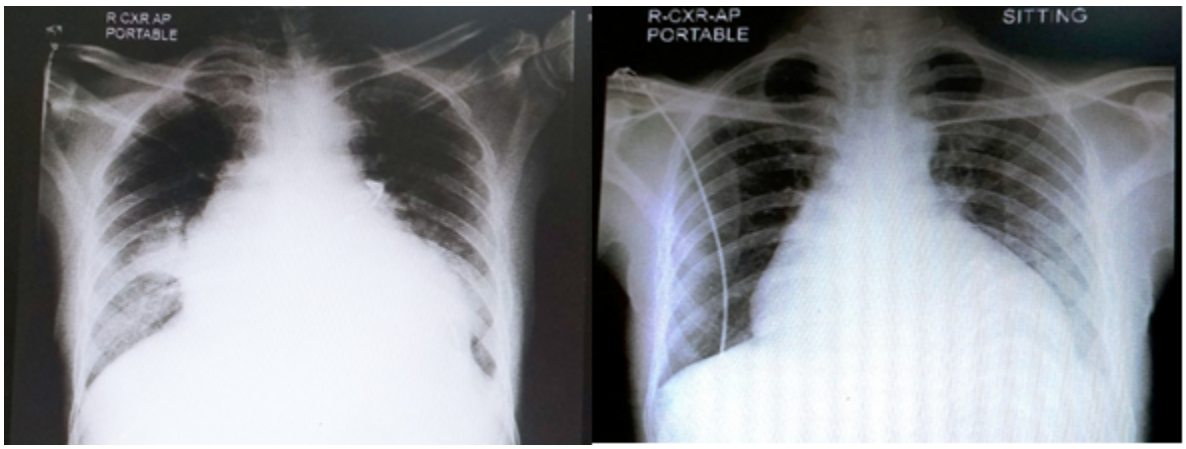

Figure 2: Showing right paracardiac patchy consolidation with cardiomegaly on admission in first chest X-ray; second chest X-ray showing significant improvement in lung shadows with sharp costophrenic angles on repeat chest X-ray after 4 days.

In view of altered liver function test, patient was put on alternate regimen of ATT i.e.

t-Ethambutol 800 mg OD

t-Levofloxacin 500 mg OD

Inj. Streptomycin 500 mg IM OD.

Patient's severe LV dysfunction with EF 10% on admission improved to 35% over a period of 4 days. A significant improvement noted in lung shadows of patient on repeat chest X-ray [Figure 2]. Patient was discharged after 7 days with alternate regimen of ATT. Patient's Echocardiography study showed normal LV function without pericardial effusion and constrictive physiology on follow-up visit after 8 days to cardiac OPD.

Discussion

Cardiac tamponade was by Richard Lower [11] as, "*the walls of the heart are compressed by fluid settling everywhere so that they cannot dilate sufficiently to receive blood; then the pulse becomes exceedingly small until it becomes utterly suppressed by the great inundation of fluid, whence succeed syncope, and death itself*".

A penetrating trauma to the heart or ventricular wall rupture after myocardial infarction leads to acute cardiac tamponade, whereas causes like infection, autoimmune diseases, neoplasm, uremia and other inflammatory diseases leads to a slower growing effusion [12].

Transkei was an unrecognized state in the southeastern region of South Africa, where Tb pericarditis was the second most common cause of heart failure following rheumatic heart disease.

In the Transkei it was so frequently found that it was referred to as the "*Transkei Heart*." The prognosis in tuberculous pericarditis is invariably bad and death may take place within one or two years but more often within a few months by Michael Gelfand [13].

Transkei heart may present as gradual pericardial effusion or constrictive pericarditis or a combination of effusion and constriction often having obscure and non-specific history such as fever, night sweats, fatigue, weight loss, chest pain, cough, dyspnoea [13,7]. In case of increasing effusion causing tamponade is characterized by tachycardia, pulsus paradoxus, hypotension, a low pulse pressure, raised central venous pressure and poor perfusion of vital organs due to impaired filling of the ventricles and reduction of stroke volume [13].

In our case, presentation was unusual as massive pericardial effusion led to severe heart failure without classical history of tuberculous pericarditis. The mode of transmission of MTb to pericardium remained uncertain in our case. According to literature less than half of the patients show evidence of pulmonary tuberculosis at the time of presentation [14]. We applied Light's criteria to analyze pericardial fluid and labeled it as exudative in nature. LDH value of >1000 U/L is best marker to differentiate exudates from transudes than pericardial fluid/serum LDH ratio, and pericardial fluid/serum protein ratio [15].

A definitive diagnosis of Tb pericarditis is most often by demonstrating tubercle bacilli in pericardial fluid or biopsy specimen by culture or in histology section of pericardium, whereas a probable diagnosis is based on the proof of Tb elsewhere in body with pericardial effusion, raised ADA levels (ref. range of 30-60 IU/L has 90% sensitivity and 70% specificity) with lymphocytic exudates and/or response to ATT [16,17].

In our case pericardial fluid culture on LJ media grew colonies suggestive of Mycobacterium species, PCR positive for Tb complex and raised ADA levels confirming tubercular origin of pericardial effusion. Patient was started on alternate regimen of ATT following PCR results. Thus PCR can be used as screening diagnostic test along with microscopy to overcome the limitations of conventional diagnostic tests which have negative impact on patient care due to low sensitivity and delay in results.

In our case, patient was not initiated on adjuvant corticosteroid therapy in view of co-morbidities but showed no signs of pericardial effusion and constrictive physiology on follow-up visits. A recent study involving 1400 cases in large randomized trial showed adjuvant prednisolone did not improve the combined all cause death outcomes and supplemental analysis showed a small mortality benefit in HIV negative cases [18].

Conclusion

Diagnosis of tuberculous pericarditis is challenging. It requires strong clinical suspicion especially in endemic regions.

Massive tuberculous pericardial effusion can present with severe heart failure.

In heart failure with pericardial effusion, treatable infections have to be ruled out based on available findings and investigations.

The role of adjuvant corticosteroid therapy in tuberculous pericarditis needs further evaluation.

e. Evidence based clinical practice, TB PCR helps to provide more information toward early diagnosis tuberculous pericarditis.

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