

Case Report

A rare case of cardiac tamponade due to tuberculosis

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ABSTRACT

Pericardial effusion is an abnormal accumulation of fluid in the pericardial cavity. Because of the limited amount of space in the pericardial cavity, fluid accumulation leads to an increased intrapericardial pressure which can negatively affect heart function. A pericardial effusion with enough pressure to adversely affect heart function is called cardiac tamponade. Pericardial effusion usually results from a disturbed equilibrium between the production and re-absorption of pericardial fluid, or from a structural abnormality that allows fluid to enter the pericardial cavity. Tuberculosis involvement of the pericardium is well-known and can result in pericardial tamponade apart from other sequelae like constrictive pericarditis. Here we report a case of 60 years old female of pleuropericardial effusion presented with acute onset breathlessness, palpitation, tachypnea. Urgent echocardiography shows cardiac tamponade. Pericardiocentesis was performed immediately and more than one litre hemorrhagic fluid drained. Patient was put on anti-tubercular treatment with oral steroid after adenosine deaminase positivity in exudative pericardial fluid. Patient is doing well in follow-up visit.

Keywords: Tuberculosis, Pericardial effusion, Pericarditis

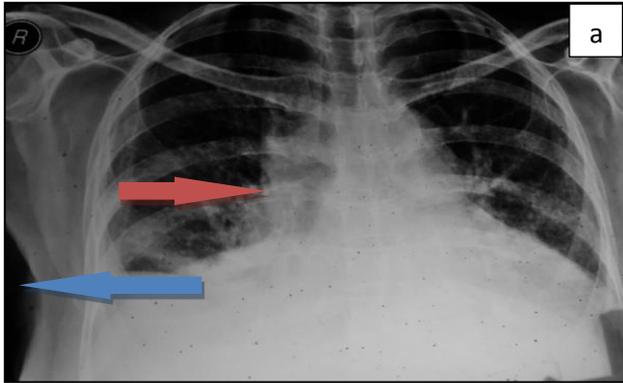
INTRODUCTION

India has the highest burden of Tuberculosis in the world, accounting for approximately one fifth of the global incidence. Cardiovascular involvement is a relatively uncommon manifestation in patients with tuberculosis and has been described in one to two percentage of patients.¹ A recently published literature suggests that, in the developed world, TB is a relative rare cause of pericardial diseases in HIV negative, immunocompetent persons and accounts for 2% of the cases of acute pericarditis, 2% of cardiac tamponade.^{2,3} Tuberculous involvement of the pericardium is well-known and can result in pericardial tamponade apart from other sequelae like constrictive pericarditis. Despite this, large pericardial effusions are uncommon, and manifestation as cardiac tamponade is rare. Here we report a case that cardiac tamponade caused by rapidly increasing pericardial effusion due to tuberculosis pericarditis.

CASE REPORT

Here we report a case of 60 years old female patient who was nonsmoker, nondiabetic, nonhypertensive and came with the complaints of breathlessness, which was insidious onset, progressive, even at rest, more in laying down, and loss of appetite since 1 month. She also complaint of swelling in both legs and abdominal distension since 25 days. She was admitted for the same complaints in private hospital where she was diagnosed with pleuro-pericardial effusion on basis of chest x ray and echocardiography and started on first line anti-tubercular drugs (isoniazid, rifampicin, pyrazinamide, ethambutol) and oral steroid empirically. She was diagnosed case of hypothyroidism since 1 year but she was not on medication. The condition of the patient deteriorated in last 24 hours and she was brought to AIIMS. At the time of admission, general condition of the patient was poor, she was restless, tachypneic (RR-

35/min), blood pressure (94/80 mmHg with pulsus paradoxus), hypoxic (SpO₂-86%) at room air and pulse (144/min) was raised. On systemic examination, there was dull note on right side with reduced air entry suggestive of right side pleural effusion along with raised Jugular venous pressure, tender hepatomegaly, pedal edema and muffled heart sound secondary to large Pericardial effusion. Chest X ray shows cardiomegaly with right pleural effusion (Figures 1a).



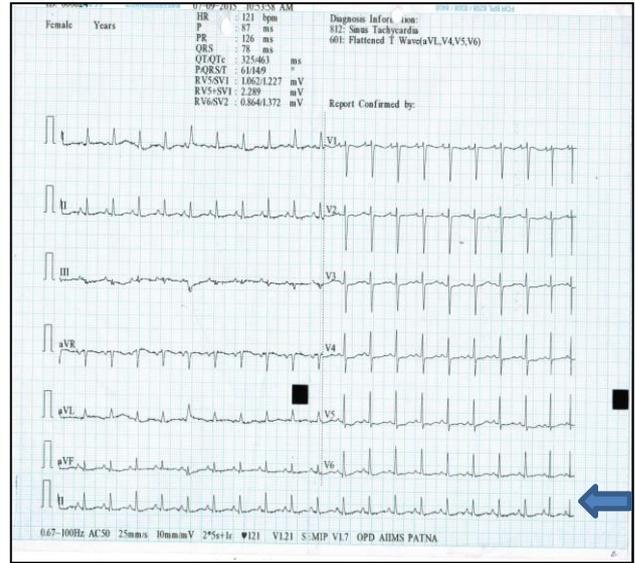
Red arrow- Cardiomegaly; Blue arrow-Blunting of costophrenic angle.

Figures 1: CXR shows cardiomegaly with right pleural effusion. a) at the time of admission; b) at time of discharge.

ECG features sinus tachycardia with low voltage complexes (Figure 2). Urgent 2D-ECHO was done which show diastolic collapse of right ventricle and atrium along with large amount of pericardial fluid which was suggestive of pericardial tamponade. Consent was obtained and Immediately pericardiocentesis was done with pig tail catheter in situ, 600ml of hemorrhagic fluid was drained and catheter remain placed placed for 2 days with total drainage of 1500 ml.

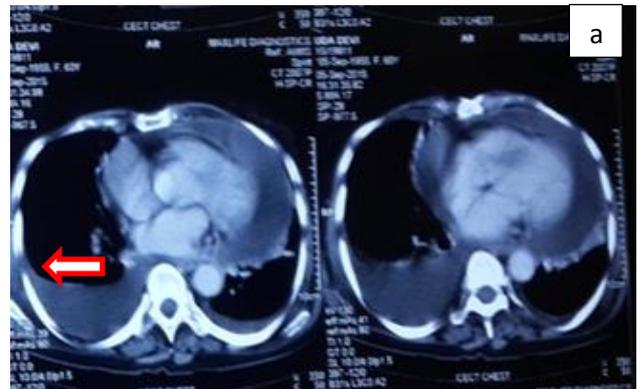
The pericardial fluid was sent for routine investigation. The fluid contained 1200 leukocytes (60% lymphocytes), but microbiology and cytology yielded negative results. Sputum and pericardial fluid was negative for acid-fast bacilli and malignant cell however adenosine deaminase

(59 IU/L) was supportive for tubercular origin. Pericardial fluid was negative for TB PCR (polymerase chain reaction).



Blur arrow: Low voltage complex (<10mm in chest leads and <5mm in limb leads).

Figures 2: Electrocardiogram show sinus tachycardia with low voltage, T wave inversion.



Figures 3: CECT shows bilateral pleural effusion (R>L) with pericardial effusion. a) Arrow showing right side pleural effusion. b) Arrow showing fluid density surrounding heart.

Viral serology for hepatitis and human immunodeficiency virus (HIV) were negative. Contrast enhanced computed tomography thorax was also performed which shows right mild pleural effusion and left mild to moderate pleural effusion along with moderate to gross pericardial effusion, pericardial thickening, necrotic lymph node in pretracheal and precarinal region (Figure 3).

On further investigation, she has deranged liver function? Drug induced hepatitis for which ATT was optimized by holding isoniazid, rifampicin, pyrazinamide and adding streptomycin (0.75g), levofloxacin (750mg). CBC and LFT repeated, which shows mild anemia (Hb-10 g%) and improvement in liver enzymes. One unit blood transfusion was done along with sequential reintroduction of first line anti-tubercular drugs after normalization of liver enzymes. Chest X-ray was performed on discharge which shows complete disappearance of cardiomegaly and pleural effusion (Figures 1b). A patient was discharged on Anti tubercular treatment HRZE daily regimen and oral 40mg prednisolone with tapering schedule. Follow-up ECHO done which did not reveal pericardial effusion and patient was doing well on 3rd month follow up visit.

DISCUSSION

Tubercular pericarditis can develop at any age but commonly occurs in middle age. In one of the case series⁴ more than 80% patients were older than 35 years with nearly half of them being more than 55 years old. Like other forms of tuberculosis, it is more common in patients who are immunosuppressed. In another case series⁵ of 47 patients with large pericardial effusion, Tamponade is frequent with large tuberculous and chronic idiopathic effusions.

The quantity and quality of the effusion were similar. Fever and pericardial rub were more frequent with tuberculosis as also deposits and strands on echocardiography and patients had a strongly positive skin test. Mediastinal lymph gland enlargement on chest CT was found only and in all with tuberculous effusion. Our patient's age was sixty years without any evidence of immunosuppression. The clinical manifestations of tuberculous pericarditis are wide and variable. Chest pain, cough and dyspnoea are common manifestation, non-specific constitutional symptoms, including fevers, night sweats, weight loss, and fatigue, may also arise.⁶ Patients may present subacutely with the development of constrictive pericarditis, or acutely with pericardial fluid accumulation leading to cardiac tamponade.⁷ The three most common causes of tamponade are neoplastic disease, idiopathic pericarditis, and renal failure. Tamponade may also result from bleeding into the pericardial space after cardiac operations, trauma, and treatment of patients with acute pericarditis with anticoagulants.⁸ In this case unusual presentation is

development of pericardial tamponade after slowly accumulating fluid over a period. The source of the mycobacterium in tuberculous pericarditis remains uncertain, with less than half of patients having evidence of pulmonary tuberculosis.⁹ There may be other contiguous foci of infection, such as the mediastinal or hilar nodes, spine, sternum, or, less commonly, miliary spread.¹⁰ In present case, patient also had pleural effusion with necrotic mediastinal lymph node involvement. Pericardial fluid high in ADA activity (>40 U/L) is of great value in early diagnosis of tuberculous pericarditis, with a sensitivity of 94% and a specificity of 97% for tuberculous pericarditis.¹¹ In our patients pericardial fluid had high ADA (59 U/L) favour for diagnosis of tuberculosis cause. In developed country, the most common cause of bloody pericardial effusion in patients with signs or symptoms of cardiac tamponade is now iatrogenic disease. Of the non-iatrogenic causes, malignancy, complications of acute myocardial infarction, and idiopathic disease predominated.¹²

CONCLUSION

Tuberculosis diseases is endemic in our country. Pericardial involvement and pericardial effusions are well-documented which may result in pericardial tamponade. Despite this, large pericardial effusions are uncommon, and manifestation as cardiac tamponade is rare. There should be high degree of suspicion in cases of rapidly developing pericardial effusion.

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