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Case report

Tuberculous pericardial effusion causing tamponade in a healthy young male: A diagnostic and therapeutic challenge of a rare manifestation of a fairly frequent disease in Sri Lanka: A Case Report

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Introduction

Tuberculous pericarditis with pericardial effusion, although considered exceedingly uncommon, is a well-recognized extrapulmonary complication of tuberculosis (TB) [1]. Tuberculosis is the most common cause of pericardial effusion in Sri Lanka but reports of tamponade are scarce [2,3]. Pericardial TB is seen in 1-2% of all cases of pulmonary tuberculosis. It accounts for 4% of all cases of acute pericarditis and 7% of cases of cardiac tamponade[3]. Tuberculous pericarditis accounts for 60-80% of cases of acute pericardial effusions in the developing world but only 4% in developed countries [4]. Africa, Asia and Latin America, where 95% of active tuberculosis is reported, accounts for 98% of all TB deaths [5].

Pulmonary TB accounts for the majority of cases of TB worldwide. However, extra-pulmonary TB, commonly seen among immunosuppressed populations, accounts for more morbidity and mortality [4]. With the rise of HIV, more cases of extra-pulmonary TB are being reported worldwide, pericardial TB being the most common site reported [6]. Prompt treatment is lifesaving in patients presenting with TB pericarditis with cardiac tamponade [5]. Diagnosis is challenging, due to the low diagnostic yield and difficulty in interpretation of the pericardial fluid analysis reports [5,7]. Mortality in the untreated approaches 85% at 6 months, with a mean survival of 3.7 months [4].

We report a case of tuberculous pericarditis in a young, healthy, immune-competent male who presented with tamponade. The case was diagnosed on pericardial biopsy

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and tuberculin skin test. Patient had a rapidly filling effusion that had to be repeatedly aspirated, later proceeding to pericardiostomy to maintain haemodynamic stability. Thereafter, he was started on anti-TB therapy and steroids with remarkable recovery.

Case Presentation

We report a case of a 35-year-old, previously healthy, Sinhalese army officer who presented to us with a one week history of bilateral lower limb oedema followed by facial puffiness and abdominal distention. He had progressive shortness of breath with postural dizziness and a dry cough with no haemoptysis for 2 weeks duration. He had no history of fever or nocturnal sweats prior to admission but had episodic fever during the ward stay.

On examination, he was thin built, severely tachypnoeic and pale with a tinge of icterus. He had gross lower limb oedema up to mid-calf level, gross abdominal distension and facial puffiness. His neck and upper limb veins were distended. There were a few small and scattered, bilateral lymph nodes in the neck and evidence of right lower zone effusion in chest. His jugular venous pressure (JVP) was elevated with a non-deviated, indistinct but palpable apex. Heart sounds were heard. Our patient did not have a pericardial rub nor muffled heart sounds and the apex beat was felt (possibly as the patient was thin built). Blood pressure which was initially 90/60mmHg later dropped to 70/50mmHg with a regular pulse of 110. There was no pericardial rub nor audible murmurs. His abdominal examination revealed mild hepatomegaly with evidence of free fluid.

His full blood count showed a haemoglobin of 8.8g/dL. The blood picture was normocytic, suggestive of a mixed deficiency anemia and was reactive. WBC was 4.45 × 10^9 with normal differential count and normal platelet count. ESR and CRP were 45mm/1^{st} hour and 52mg/L, respectively. Renal functions and electrolytes were in the normal range. Liver functions revealed a slightly elevated ALT and a marginally raised direct hyperbilirubinemia (ALT 164 U/L, AST 45U/L, Total bilirubin 27.8micmol/L, direct bilirubin 15.8micmol/L, ALP 140.7U/L). ECG showed small complexes. Chest X-ray showed an enlarged heart with blunted costophrenic angles. Cardiac margins were well defined (Figure 1).

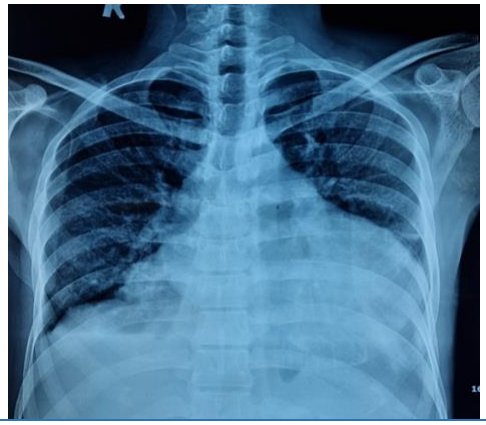

Figure 1: Chest radiograph showing evidence of cardiomegaly. The inferior cardiac border is well defined.

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2D-ECHO confirmed the presence of a thickened pericardium with multiple septations. There was a large pericardial effusion, largest at the infero-apical surfaces (29mm). There was no right atrial or ventricular dilatation, TRPG (tricuspid regurgitation peak gradient) was 17mm, with early diastolic RV collapse.

Pericardial fluid aspirate was blood-stained. Cultures, cytology, TB culture and Gene-Xpert studies were negative. Full report showed 3680/mm^3 RBC, WBC 65/mm^3 (65% polymorphs). Repeated aspirations were performed for rapidly refilling effusion to maintain h/aemodynamic stability. Initially, 900cc of fluid was removed. Following 3 aspirations due to repeated re-accumulation of fluid, the patient underwent subxiphoid pericardiostomy with concomitant biopsy. Pericardial biopsy revealed granulomata composed of epithelioid histiocytic lymphocytes and occasional multinucleated giant cells. Large areas of eosinophilic necrosis were seen with no atypical cells. The picture was compatible with chronic granulomatous inflammation with necrosis, possibly tuberculosis. AFB stains were negative, but the Mantoux test was 15mm (positive).

Ultrasound abdomen showed mild hepatomegaly with ascites. Immune markers (ANA, rheumatoid factor) were negative. TSH was 1.727 µmol/ml (0.44) LDH was 257 U/L

CECT chest showed a uniformly thickened pericardium with moderate effusion (after drainage of pericardial effusion) and right sided lung collapse with moderate pleural effusion. Lymphadenopathy was noted on either side of the diaphragm and supraclavicular area. There was no notable lung or liver masses or any evidence of skeletal metastasis (Figure 2).

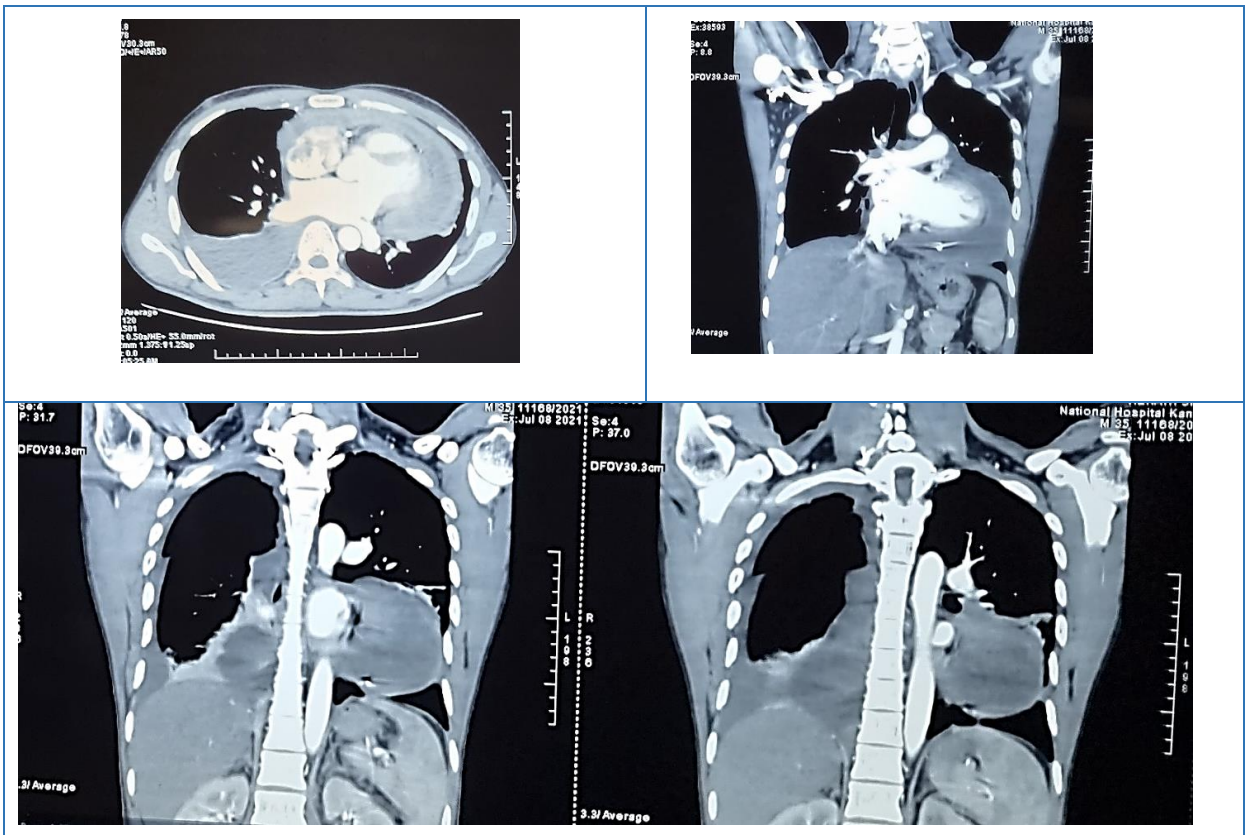

Figure 2: CECT Chest; Showing thickened pericardium, pericardial effusion and right sided pleural effusion.

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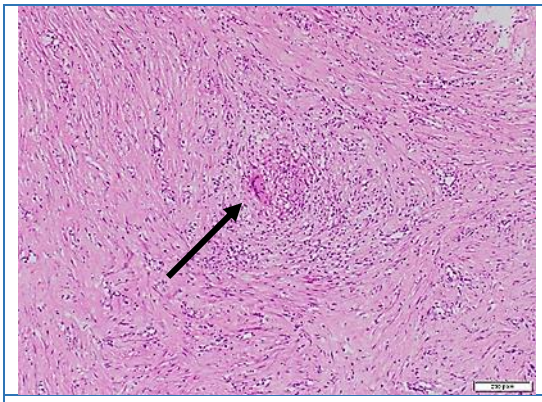
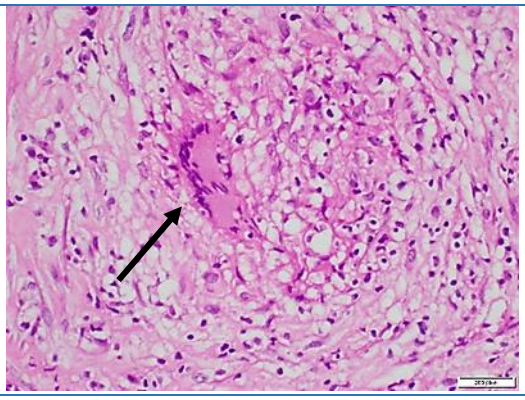
Following the biopsy report and the positive Mantoux test, anti-tuberculous treatment (ATT) was commenced with steroid cover while monitoring liver functions.

Patient improved dramatically. There was no re-accumulation of fluid following surgery. The supra clavicular lymph nodes were not palpable after starting ATT.

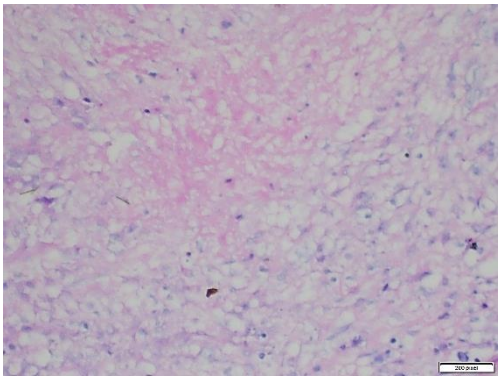
The patient was well during follow up visits and was continuing ATT at the usual regimen. Repeat 2DECHOs revealed resolution of effusion at 6 weeks and 6 months after commencing ATT.



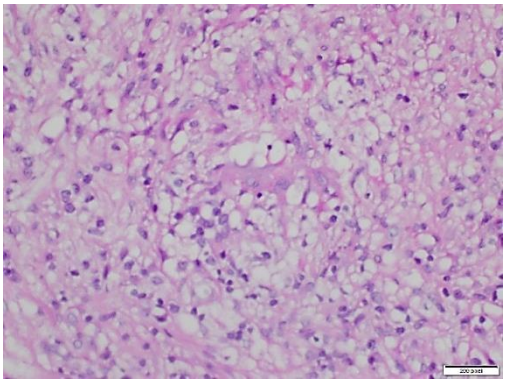
a. Arrowed is a granuloma composed of epithelioid histiocytic lymphocytes



b. arrow shows a multinucleated giant cell



c. AFB stains (negative



d. PAS stain (negative)

Figure 3: Histology

Discussion

Differentiation between cardiomyopathy, constrictive pericarditis, effusion or congestive cardiac failure was clinically ambiguous. The chest X-ray, showing cardiomegaly with clear lung fields, is supportive of an effusion but definitive diagnosis is via 2DECHO[8].

The clinical findings of effusion or tamponade do not always correlate with ECHO findings [7]. In a study conducted by Levine et al on patients with pericardial effusion, only 50% were clinically suspected to have tamponade concluding that 2DECHO detects tamponade far earlier than clinical examination [7].

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In haemodynamically compromised patients with large pericardial effusions, a malignant or tuberculous etiology was commoner than idiopathic pericarditis [9]. Thus TB, being treatable, should be excluded in a patient coming with a large effusion.

Our patient was compensating to maintain reasonable blood pressures despite the large effusion. Marked improvement of pressure and tachycardia following pericardiocentesis confirmed the presence of tamponade. Tamponade is not an "all or none" phenomenon. It has a clinical spectrum ranging from slight elevations of intrapericardial pressure with subtle haemodynamic changes to more severe haemodynamic compromise or even death [7]

Although commonly sent for analysis, pericardial fluid is not very useful in diagnosis. AFB smears of pericardial fluid are rarely positive and the culture yield is around 50-59%. Pericardial histology has a diagnostic yield of around 70-83% [10] . Diagnostic yield of pericardiocentesis can be as low as 7-19%, whereas the procedural risk amounts to around 5% [4,11]. None of the cultures or stains for AFB were positive ~~i~~in our patient.

A study by Reuter et al concluded that only 50 % of samples for histology confirmed caseating granulomas, even when the fluid culture was positive for TB. Mycobacterial culture yielded 56% positivity with a higher sensitivity compared to fluid smear [11]. Nevertheless, culture results take time, rarely assisting in decision making. The cell composition in TB pericardial fluid may vary, unlike TB pleural effusions with lymphocytic predominance. Thus, the cyto-diagnostic criteria with higher specificity in pleural TB is not as helpful in pericardial TB [11]. Our patient's pericardial fluid had a neutrophil predominance, contrary to the expected lymphocytosis.

TB PCR in the setting of TB pericarditis has a sensitivity as low as 15%-20% and a specificity of 96% -100%. Sensitivity is higher when pericardial tissue is used for PCR. IFN gamma assay above 50pg/ml has the best sensitivity and specificity profile of 92% and 100%, respectively, which further increases when combined with ADA and cytology [12]. Unfortunately, these investigations, especially IFN gamma, have financial constraints in our setting.

Our patient's diagnosis was confirmed by a combination of ECHO and CT features suggestive of chronic pericarditis, positive Mantoux test, biopsy positivity for granuloma (although there was no caseation) and a positive response to ATT.

Definitive diagnosis of TB pericarditis is by demonstrating tuberculous bacilli in pericardial fluid or in histology. Probable diagnosis is by evidence of TB elsewhere in the body with pericardial effusion, raised ADA levels and a positive response to ATT [3]. Criteria for diagnosis include one or more of the following 1. Isolating tuberculous bacteria from pericardial fluid or biopsy 2. Demonstrating granulomas on the pericardial biopsy 3. Isolation of *M. tuberculosis* from non-pericardial exudates in the presence of clinical or radiological evidence of TB, with a positive response to ATT in the absence of any other cause for the effusion [11]

Most cases of TB pericarditis in the literature were reported in patients with underlying immunosuppression, such as in HIV or medication [6,12]. Our patient had no evidence

of immunodeficiency, emphasizing the need to consider tuberculosis as a differential in healthy young patients coming with pericardial effusion in an endemic setting.

Management of tuberculous effusions largely depends on symptomatology. Pericardiocentesis may be helpful in both confirming the diagnosis and relieving symptoms [13]. The ideal procedure to drain an effusion should be easy to perform, low risk and ensure complete and permanent drainage with infrequent recurrences. A procedure giving samples for cytological, histological and microbiological diagnosis is an additional advantage [14]. The two primary modalities used to drain symptomatic effusions are transcutaneous pericardioscopy and open sub-xiphoid surgical drainage. Sub-xiphoid pericardiostomy is a surgical procedure where an opening is created in the pericardium to directly visualize and explore the pericardium and probe the cavity or insert a tube for complete drainage and obtain biopsy samples. Transcutaneous drainage helps in visualizing the pericardium and doesn't need large incisions or general anesthesia but needs high degree of expertise [14]. In our case, pericardiostomy was inevitable due to fluid re-accumulation despite repeated aspirations and gave us the advantage of obtaining tissue samples.

The concept of a pericardial window for permanent drainage of fluid into the pleural cavity is misleading as the hole created is sealed off fast by surrounding tissue [15]. Thus, the resolution of symptoms, although initially due to surgical drainage, later, was possibly due to the commencement of ATT with steroid cover.

Conclusion

Tuberculosis should be considered in cases of acute pericardial tamponade, even in a previously healthy, low risk individual especially in an endemic background. Diagnosis is challenging as pericardial fluid analysis is not sensitive or reliable. A combination of clinical findings, imaging and histology with a positive Mantoux and response to ATT may point to the diagnosis in the context of negative microbiology. Obtaining pericardial biopsy, although it helps greatly in confirming diagnosis, is not mandatory unless pericardiostomy is inevitable for symptom relief. Early initiation of ATT is the key in management and may need to be combined with surgical procedures to drain chronic effusions.

Acknowledgements

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