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Tubercular Pericarditis- A Case Series

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ABSTRACT
Tuberculosis is responsible for approximately 70% of cases of large pericardial effusion and most cases of constrictive pericarditis in developing countries. A definite or proven diagnosis is based on demonstration of tubercle bacilli in pericardial fluid or on histologic section of the pericardium. A probable or presumed diagnosis is based on proof of tuberculosis elsewhere in a patient with otherwise unexplained pericarditis, a lymphocytic pericardial exudate with elevated biomarkers of tuberculous infection, and/or appropriate response to a trial of antituberculosis chemotherapy.
Here we report 3 cases of tubercular pericarditis of which 2 cases survived and one died due to cardiac arrest.
The objectives of this case series is to emphasize that tuberculous pericarditis is a dangerous disease with a mortality of 17% to 40%; constriction occurs in a similar proportion of cases after tuberculous pericardial effusion. Early diagnosis and institution of appropriate therapy are essential to prevent mortality.
Keywords: *Tuberculosis,pericardial effusion, antituberculosis chemotherapy.*

INTRODUCTION

The incidence of tuberculous pericarditis among patients with pulmonary tuberculosis ranges from about 1% to 8%. Tuberculosis was diagnosed in only 4% of acute cases of acute pericarditis[1] Maltezou et al.[2] reported 102 children with extrapulmonary tuberculosis; only one had tuberculous pericarditis. The incidence of tuberculosis is frequent in Africans and Asians[3]. Tuberculous pericarditis usually develops by retrograde spread from peribronchial, peritracheal or mediastinal lymph nodes. Less commonly, it occurs during miliary tuberculosis, but it may also develop from a focus in the lung, spine or sternum[4]. The onset may be abrupt, resembling acute idiopathic pericarditis, with weight loss, cough, dyspnoea, chest pain, ankle oedema, fever, tachycardia, weakness, anorexia and night sweats[5]. Physical examination usually shows fever, tachycardia, pericardial friction rub, hepatomegaly, ascites, peripheral oedema and weak and distant heart sounds. The chest x-ray shows cardiomegaly. Sometimes a pleural effusion may be detected in about half of the patients. However, the apices and hila of the lung are usually normal and pulmonary infiltrates or calcification are only present in a minority of the patients. The tuberculin skin test (PPD) may be negative in as many as 30% of patients with tuberculosis, owing to anergy[6]. Pericardial fluid aspiration is important for diagnosis. A definitive diagnosis can be made by isolation of the bacillus

from the pericardial fluid, or by pericardial biopsy. Acid-fast bacilli are rarely found in pericardial effusions. Cultures of pericardial fluid are positive in only 50% of cases.

CASE REPORTS

The first case was a 8 year old boy who was a known case of sickle cell trait and this time presented with fast breathing and respiratory distress for last 7 days. Chest radiography showed cardiomegaly (Fig 1) and pericardial biomarker showed Adenosine deaminase value 96 IU/L (normal < 40). ECHO cardiography showed constrictive pericardial effusion.

The second case was a 3 year old female child who was admitted with complains of fever for last 10 days. All routine investigations were normal. Chest radiography showed cardiomegaly and non homogenous opacity on right lung (Fig 2). Pericardial biomarker showed Adenosine deaminase value 110 IU/L (normal < 40) and mononuclear pleocytosis. ECHO cardiography showed pericardial effusion.

The third case was that of a 14 year old boy who was admitted with complains respiratory distress for 7 days. There was history of low grade fever for 6 months with swelling of abdomen and feet. There was a history of contact with an open case of Tuberculosis. His neck veins were prominent and heart sounds were muffled. All routine investigations including renal function test and liver function test were normal. ESR was 110, CBNAAT was positive. Chest radiography showed cardiomegaly and opacities on right lung (Fig 3).

The first 2 patients were started on antitubercular drugs along with steroids and they showed improveement in 2 weeks. Their pericardial effusion decreased gradually and clinical condition improved rapidly. But the third patient died within 1 day of admission because of cardiac arrest.

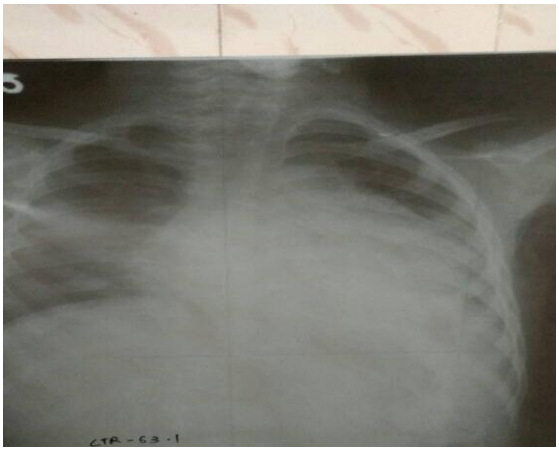

Fig 1 showing cardiomegaly (63%)

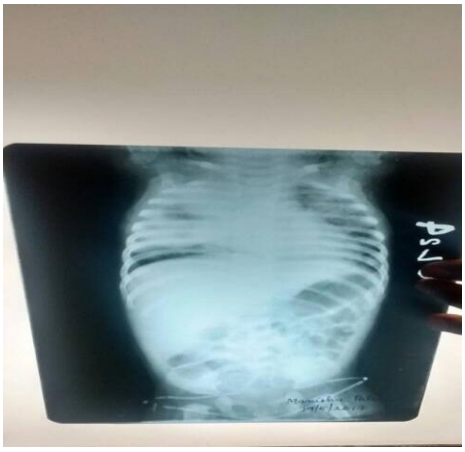

Fig 2 showing cardiomegaly with right side Non homogenous opacity

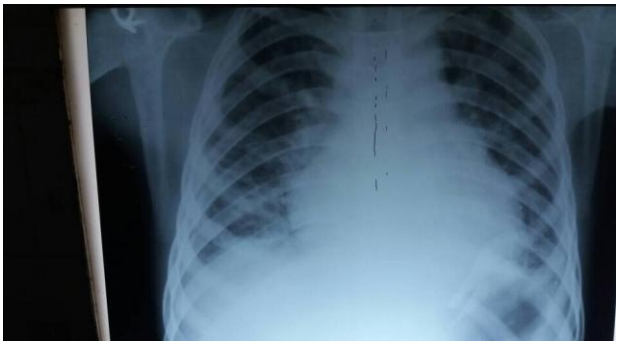

Fig 3 showing cardiomegaly and opacity on right lung

DISSCUSSION

The predominant symptoms of tuberculous pericarditis are cough, dyspnea, and chest pain. Night sweats, orthopnea, weight loss, and ankle edema are also common. As for signs, the most frequent are cardiomegaly, pericardial rub, fever, and tachycardia. Other findings may include pulsus paradoxicus, hepatomegaly, distended neck veins, pleural effusion, and distant heart tones [7,8].

Our third patient manifested almost all of these symptoms and signs. Current understanding of the pathogenesis of tuberculosis suggests that proinflammatory cytokines may be responsible for the symptoms of fever, weight loss, and weakness. The cytokine most clearly implicated is TNF-α [9,10]. Numerous other infectious and noninfectious causes can have a presentation similar to that of tuberculous pericarditis. A positive tuberculin skin test result may increase the suspicion of tuberculous pericarditis, but a negative skin test result does not exclude this diagnosis. In all three cases had mantoux was negative. Tuberculous pericarditis must be differentiated from the effusion of chronic renal failure or rheumatic disease.

CONCLUSION

Tuberculosis should be considered as a possibility in many cases of pyrexia of unknown origin (PUO) Our patients had fever for quite a long time. It may be necessary to make a presumptive clinical diagnosis of tuberculous pericarditis in severely ill patients with a large pericardial effusion and systemic symptoms. In such patients, antituberculous treatment must be started as soon as proper diagnosis is obtained. Clinical improvement usually occur after antituberculosis chemotherapy along with steroid.

In any case of pericardial effusion unless and until proved otherwise, the tubercular etiology should be kept in mind.

CONTRIBUTORS

Dr Nasreen Ali- conception, design and drafting

Dr Sunil Kumar Agarwalla-revising it critically for important intellectual content.

CONFLICT OF INTEREST

There was no conflict of interest and no funds received.

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