



## Clinical Case Report

## Pancardiac tuberculosis – a case report

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## ABSTRACT

Tuberculous involvement of the heart though not common has been reported in literature. Pericardium is commonly involved followed by the myocardium. Endocardial involvement is rare. We would like to report a case of a 14-year-old female with florid and extensive involvement of all the layers of the heart by tuberculosis.

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## 1. Introduction

Tuberculosis is one of the leading causes of death in developing countries. It is said to spare thyroid, pancreas, heart, and skeletal muscle. Cardiac involvement by tuberculosis has been reported in literature. The overall incidence of involvement of heart in patients with tuberculosis is 1–2%. Pericardium is commonly involved by tuberculosis due to retrograde lymphatic spread from peritracheal, peribronchial, and mediastinal lymph nodes or by hematogenous spread. Tuberculous pericarditis accounts for 69.5% of cases in developing countries, whereas only 4% of cases in developed countries [1]. Myocardial involvement by tuberculosis is mostly seen in association with pericardial disease. Isolated myocardial involvement is rare. The prevalence of isolated myocardial disease reported is 0.14% to 2% in few studies [2]. Myocardium may be rarely involved by tuberculosis either due to the constant movement which makes it nonconductive to the lodgment of tubercle bacilli or the lactic acid which is produced by the muscular activity [3]. We report a rare case of pericardial, myocardial, and endocardial involvement by tuberculosis.

## 2. Case history

A 14-year-old female was admitted to our hospital with complaints of progressive dyspnea, chest pain, decreased appetite, and weight loss since 1 year. She weighed 27 kg for an expected weight of 41 kg. Her pulse was 90/min; respiratory rate was 16/min. There was bilateral

pedal edema. Her hemoglobin was 9.4 gm%; total count was 6100/cm<sup>2</sup> with 80% neutrophils, 20% lymphocytes. Her liver function tests and renal function tests were in normal limits. X-ray chest was performed and showed cardiomegaly. However, there was no calcification noted in the pericardium. A 2D echo showed a thick pericardium about 10 mm, right atrial enlargement, and depressed left ventricular function; Left ventricular ejection fraction [LVEF] was 40% suggestive of constrictive pericarditis. Her condition deteriorated, and she expired on the second day of hospital stay.

A complete postmortem was performed. She was an averagely build and poorly nourished female. She had mild pallor and bilateral pedal edema. Examination of chest cavity showed 300-ml pleural effusion on right side. Both the lungs showed 1-mm pinhead sized tubercles on the pleural as well as the cut surface. Heart was enlarged, globular, and weighed 1440 g. Visceral and parietal pericardiums were thickened and opaque, adherent to each other and to the lungs and diaphragm suggestive of pericarditis (Fig. 1). On cut section, pericardium was 10-mm thick whitish opaque and firmly adherent to the myocardium. There were multiple diffused white nodular lesions with caseation seen in the pericardium, myocardium reaching up to the endocardium with focal ulceration of the endocardium (Figs. 2 and 3). All the chambers of the heart along with the interventricular septum showed involvement by similar lesions. Mediastinal lymph nodes were enlarged and matted together; cut surface showed caseation. Spleen and liver also showed multiple miliary tubercles. Brain showed cerebral edema. All the rest of the organs were unremarkable.

Histopathological examination of the heart showed extensive areas of caseation with epithelioid cells, Langhan's giant cells, and lymphocytes in the pericardium, myocardium, and endocardium (Fig. 4). Epithelioid cell granulomas with and without caseation were seen in the mediastinal lymph nodes, lungs, spleen, and liver. Acid fast bacilli [AFB] stain was done on the section of the heart which was positive for acid-fast bacilli. This case had disseminated tuberculosis with pan cardiac involvement.

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Fig. 1. Shows thickened pericardium areas of caseation necrosis and adherent lung.

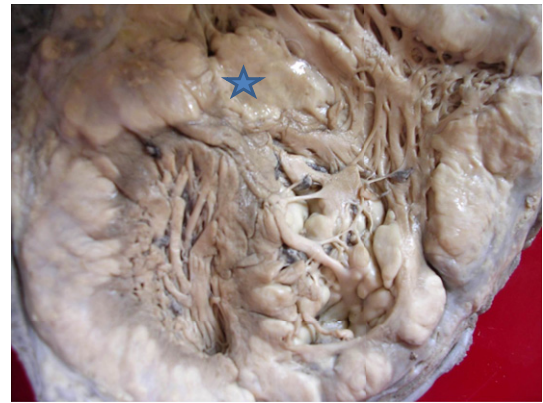


Fig. 3. (\*) Plague-like lesions seen in the ventricular endocardium.

### 3. Discussion

Cardiac involvement by tuberculosis is seen in 1–2 % patients with pulmonary tuberculosis. The earliest reports are by Maurocordat in 1664 and by Morgagni, 1761 [4]. Pericardial involvement may be in form of effusion, constrictive pericarditis, or a combination of both.

Horn and Saphir have described three histological types of myocardial tuberculosis: (a) nodular tubercles (tuberculomas) of the myocardium, varying from pea to egg size, with central caseation; (b) miliary tubercles of the myocardium complicating generalized miliary disease; and (c) the uncommon diffuse infiltrative type usually associated with tuberculous pericarditis in which the myocardium is diffusely infiltrated by the granulation tissue containing the giant cells, endothelial cells, and the lymphocytes [5]. The clinical presentation of myocardial tuberculosis can be asymptomatic or can present as sudden death, intractable ventricular arrhythmia, long QT syndrome, high heart block, or congestive cardiac failure. Echocardiography and MRI help in morphologic and hemodynamic assessment, whereas endomyocardial biopsy is the gold standard in diagnosis of myocardial tuberculosis.

Endocardial involvement has been reported in literature in form of mass lesion or in the form of valve endocarditis [6,7]. Our case showed

multiple plague-like patches of caseation necrosis in the left ventricular endocardium. A single case report of pancardiac tuberculosis has been reported in a literature in a 25-year-old female [8]. Our case had extensive involvement by tuberculosis involving all the three layers as well as all the four chambers of the heart in the form of pancardiac tuberculosis.

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Fig. 2. Coronal section of heart showing extensive involvement of the pericardium, ventricular myocardium, and endocardium by white caseation necrosis. Interventricular septum shows similar lesions.

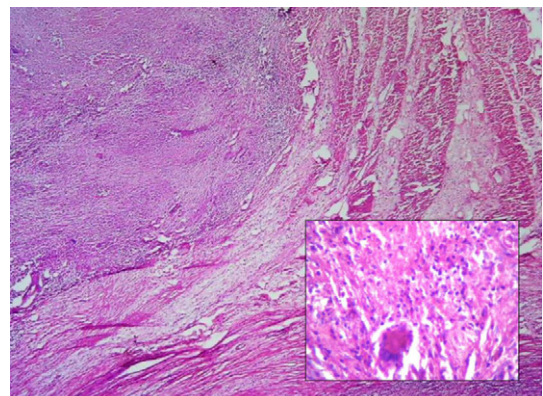


Fig. 4. (10× Hematoxylin eosin [H&E] stain) Extensive areas of caseation necrosis seen in the myocardium. Inset showing (40×) epithelioid granuloma.