Case Report:
Tuberculosis Presenting as Myocarditis

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Abstract

A case of tuberculosis presenting with features of myocarditis is being reported on account of its rarity.

Key Words: Tuberculosis – Myocarditis.

Introduction

TUBERCULOSIS is a fairly common cause of pericardial disease but myocardial involvement though known is often not diagnosed during life. Tuberculosis myocarditis is seen mostly in association with pericardial disease but isolated myocardial tuberculosis is an unusual finding; the prevalence has been reported as 0.14%, 0.2% and 2% in varies series, we present here a rare case of myocardial tuberculosis with clinical presentation as congestive heart failure.

Case Report

An Indonesian female, 28 years old, presented to Erfan Hospital on the 26th of June 2009, complaining of long history of night fever, loss of weight, pallor and sweating.

On clinical examination, upon admission, blood pressure was 90/60mmHg, heart rate was 140/min, pulse was regular, equal on both sides and of big volume. The patient was dyspneic and severely pale but her temperature was normal. Examination of the heart revealed S3 apical gallop and chest examination revealed bilateral fine basal crep. Examination of the abdomen revealed the presence of moderate amount of ascites. No other abnormalities could be found from clinical examination.

The following investigations were done for the patient:

12-lead EKG revealed sinus tachycardia and X-ray heart and chest and CT chest revealed the presence of cardiomegaly, pulmonary venous congestion, left apical dense infiltrate, parahilar and basal infiltrates.

Laboratory work-up revealed:

- Urine: Normal.
- Stool: Normal.
- Hb.8.8, Hct.28.5, WBCs 19.7, platelets 324,000, reticulocytes 3%, ESR45.
- Blood picture showed the presence of anisocytosis, poikilocytosis, microcytosis and hypochromacib. Sickling test was negative.
- CRP 8.7 IU/L.
- LDH 444IU/L, AST 57, ALT 46IU/L.
- Iron 21.7ug/dl, ferritin 1765ng/ml.
- Blood group was B+ve.
- Serum bilirubin total 0.63mg/dl, direct 0.48 and indirect 0.15.
- CK total 242IU/L, CKMB 5.2IU/L, Troponin T 0.01ng/ml.
- Free T4 0.48 ng/dl, TSH 4.32 IU/ml.
- Tumor markers: Negative.
- Sodium 129mmol/L, potassium 3.9mmol/L, chloride 98mmol/L, bicarbonate 15.5mmol/L, BUN 21mg/dl, creatinine 0.7mg/dl, calcium 6.3mg/dl, phosphorus 2.9mg/dl, magnesium 1.4mg/dl, uric acid 6.5mg/dl.
Virology study revealed Dengue IgG and IgM were negative.

CMV IgM negative, IgG positive.

Epstein Barr Virus IgG and IgM negative.

ANA negative.

Sputum culture positive for gram positive cocci and blood culture revealed the presence of staphylococcus hominis.

Urine culture negative.

Sputum smear for AFB was positive but PPD was negative.

Cortisol 23.9 mcg/dl.

Blood gases revealed the presence of hypoxia on admission but later on corrected.

Echocardiogram revealed the presence of very poor global left ventricular systolic function, severe mitral regurge and moderately severe tricuspid regurge. Ejection fraction was between 8 and 10%.

Radiology study: Pelvi-abdominal ultrasonography revealed the presence of moderate amount of pelvi-abdominal fluid, mild hepatomegaly, bilateral minimal amount of pleural effusion.

One hour after admission the patient has fast sinus tachycardia and she developed paroxysmal atrial tachycardia and started to be shocked that was why the patient was transferred to the ICU where she was maintained on inotrope support and mechanically ventilated because of severe pulmonary congestion and acute pulmonary edema.

The patient received one unit of packed cells. She started to improve and inotropic support was weaned and mechanical ventilation was weaned off.

Few days later, the patient was currently hemodynamically stable and lab. works were corrected. Her sputum for AFB was negative after receiving anti-tuberculous drugs and she was discharged to outpatient Clinic for follow-up after a week’s time.

The possibility of tuberculosis myocarditis has been raised in this case based on the following:

1- History.

2- Clinical examination.

3- Investigations including:

   a- High ESR.

   b- The sputum culture for acid fast bacilli.

   c- Radiological study including CT chest which proved the presence of apical dense infiltrates, para-hilar and basal infiltrates.

   d- The dramatic improvement in the clinical condition and the ejection fraction of the heart (more than 50% after days) after receiving the specific anti-tuberculous treatment.

Discussion

Isolated tuberculosis myocarditis is a rare disease. The first such case was reported by Maurocadat in 1664 and second case in 1761 by Morgagni after a gap of 97 year [1].

The reasons for its rarity are not known. It has been suggested that constant movement of myocardium is not conducive to lodgement of tubercle bacilli but Raviart has proposed that lactic acid produced by muscular activity offers protection to cardiac muscle against the tubercle bacilli [2].

The spread of tubercle bacilli to myocardium is either through haematogenous route or direct extension from pericardium or retrograde lymphatic spread from bronchial lymphnodes due to endobronchial tuberculosis. The presentation of this case as of congestive heart failure is also rare because myocardial tuberculosis itself is asymptomatic, rarely diagnosed during life. However, there are only four case reports in literature of presentation as congestive heart failure [3,4].

Other uncommon manifestations are valve dysfunction [5,6], rhythm disturbance and sudden cardiac death [7].

Tuberculous myocarditis is rare and occurs as a complication of tuberculosis elsewhere. The myocardium is affected by direct extension or, less often, by retrograde lymphatic drainage from tuberculous mediastinal nodes. Infection via the haematogenous route may develop in miliary disease. Direct spread from tuberculous pericarditis can also occur [8].

Horn and Saphir [2] have described three histological types of myocardial tuberculosis: (1) nodular tubercles (tuberculomas)? of the myocardium, varying from pea to egg size, with central caseation, (2) miliary tubercles of the myocardium complicating generalised miliary disease and (3) the uncommon diffuse infiltrative type usually associated with tuberculous pericarditis in which the myocardium is diffusely infiltrated by granulation tissue containing giant cells, endothelial cells and lymphocytes.
Fig. (1): These Figs. Represent the Ecco the Heart of the Patients.
Myocardial tuberculosis is rarely diagnosed during life and most of the literature is based on autopsy reports. A clinical picture of disseminated miliary tuberculosis is usually evident and cardiovascular symptoms, when present, are mainly in the form of rhythm disturbances such as supraventricular arrhythmia, ventricular tachycardia or atrioventricular block [9]. Sudden death has been reported and has been attributed to a fatal ventricular arrhythmia or a conduction defect [8].

It has been suggested that myocardial involvement should be suspected in a patient with tuberculosis if CHF supervenes [10,11] or valve dysfunction develops [6].

References
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