Tuberculous Myocarditis: An Unusual Presentation of the Disease

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-ABSTRACT-

Tuberculosis (TB) is a common public health problem in Pakistan. It can affect any part of the body but generally spares skeletal muscles, thyroid, pancreas and myocardium. Here we report an unusual case of tuberculous myocarditis in a young female who was treated with antituberculous therapy and steroids.

Keywords: Tuberculosis; Myocarditis; Antituberculous therapy

INTRODUCTION

Extra-pulmonary tuberculosis is very common in Pakistan and has been reported extensively [1]. The disease can affect any organ of the body but there are certain body sites that are rarely affected. Tuberculous myocarditis is a very rare which usually complicates miliary tuberculosis [2]. The definite diagnosis can be made only with biopsy and in few of the earlier case reports, it had been diagnosed at autopsy [3, 4]. Here we report a case of a 23-year-old female patient with miliary tuberculosis who presented with fever and congestive heart failure along with echocardiographic abnormalities. She diagnosed with tuberculous myocarditis and showed excellent recovery on antituberculosis chemotherapy with marked improvement in cardiac function.

CASE REPORT

A 23-year-old female presented in the outpatient clinic with complains of on and off cough for 1 year and fever for 4 months. Initially, the cough was nonproductive for which she had received various oral antibiotics and antihistamines without any improvement. Four months prior to presentation, her cough became productive with purulent sputum and vomiting with coughing. She developed fever up to 103°F. She was given a course of oral antibiotics and anti-pyretics which settled her fever but her cough did not resolve. Tuberculous contact history was positive. A chest X-ray was ordered which showed reticular nodular shadowing in both lung fields with dense and prominent left hilum, as

shown in Figure 1.

The patient was started on first line antituberculous therapy with four drugs (isoniazid, rifampicin, ethambutol and pyrazinamide). She developed side effects of abdominal discomfort and vomiting and stopped taking her medications and discontinued followup. Two months later, she presented in the emergency room with complaints of fever, cough, pedal swelling and exertional dyspnea. She had a heart rate of 120 beats/min, blood pressure 90/50 mmHg, respiratory rate 32 breaths/min, skin pallor, bilateral pitting edema, and elevated jugular venous pressure. She had a pansystolic mumur at the mitral area radiating to her axilla with diminished breath sounds and decreased vocal resonance in right lower lung zone.

Laboratory workup showed hemoglobin 10.9 gm/dL, white blood cells 9.6x109/L, platelets 235x109/L, erythrocyte sedimentation rate 99 mm/hr, C-reactive protein 10 mg/L. Blood urea nitrogen, serum creatinine, serum electrolytes and liver function tests were in the normal reference range. Troponin-I was <0.01 ng/mL. ECG showed sinus tachycardia with T-wave inversions in lead II, lead III and AVF.

Ultrasound chest showed mild right sided pleural effusion with septations and underlying collapsed lung and minimal left sided pleural effusion. Echocardiography showed ejection fraction of 20% with dilated left ventricle, left atrium and right ventricle; left ventricular systolic dysfunction with moderate mitral and tricuspid regurgitation and right ventricular systolic blood pressure of 50-55 mmHg. The pericardium was normal and no pericardial fluid was seen. The

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Figure 1: X-ray showing reticular nodular shadowing in both lung fields with dense and prominent left hilum



chest X-ray is shown in Figure 2. Patient was diagnosed with tuberculous myocarditis based on clinical and laboratory data.

She was started on treatment for heart failure with diuretics. ACE inhibitors and beta blockers. Anti-tuberculous treatment was re-started with 4 drugs containing rifampicin, isoniazid. ethambutol and pyrazinamide for 2 months along with high dose prednisolone (1 mg/kg which was tapered in 6 weeks). This was followed by rifampicin and isoniazid for 6 months. The patient showed significant clinical improvement at 2 months of treatment. The echocardiography showed ejection fraction of 50-55% and normal dimensions of the cardiac chambers. The X-ray is shown in Figure 3.

Figure 2: X-ray showing normal peri-cardium and no pericardial fluid



DISCUSSION

Tuberculous myocarditis is a rare disease with only a few reported cases in the literature. Cardiac manifestations have been rarely seen in patients with tuberculosis. It is estimated that 1% of all cases of tuberculosis have cardiac involvement [5]. Tuberculosis mostly affects the pericardium; involvement of valvular structures and myocardium is very rare [5, 6].

Tuberculous myocarditis results from hematogenous spread, direct seeding from the overlying pericardium or from lymphatic spread via the mediastinal lymph nodes. In our patient, it was thought that the miliary tuberculosis lead to hematogenous seeding of the myocardium with mycobacterium tuberculosis [7].

Definite diagnosis of myocardial tuberculosis can only be made by endomyocardial biopsy [8], but because this procedure is very risky, it was not

Figure 3: X-ray showing improvement in our patient's condition



attempted in our patient. Histopathologically, 3 types of myocardial involvements have been described i) tuberculomas of the myocardium with central caseation; ii) miliary tubercles of the myocardium complicating miliary disease and iii) the uncommon diffuse infiltrative type associated with tuberculous pericarditis [7].

Tuberculous myocarditis has a variable presentation from being clinically asymptomatic with a diagnosis being made at autopsy to intractable ventricular arrhythmias, long QT syndrome, heart block, valve dysfunction, obstruction of the superior vena cava, right

ventricular outflow tract or pulmonary vein, congestive heart failure and even sudden cardiac death [9]. Our patient had military TB along with clinical and echocardiographic features of congestive cardiac failure so the probability of tuberculous myocardial involvement was very high. Although cardiac MRI is an investigation of choice for diagnosing infiltrative disorders of the heart, it was not performed because of unavailability of this investigation at our hospital. Antituberculous therapy with steroids is the cornerstone of treatment of this serious condition. Although rare, this condition should be suspected in any person who presents with military TB and heart failure or arrhythmias. Our patient responded very well to 8 months of antituberculous drugs and tapering dose of steroids with an uneventful recovery.

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