

Tuberculosis and Dilated Cardiomyopathy-Case Report of a Rare Entity with Literature Review

Bhupen Barman¹, Animesh Mishra², Tony Ete^{1*}, Pravin Kumar Jha², Synrang Batngen Warjri¹, Kyrshanlang G Lynrah¹, Taso Beyong¹

¹Department of General Medicine, North Eastern Indira Gandhi Regional Institute of Health and Medical Sciences, Shillong, Meghalaya

²Department of Cardiology, North Eastern Indira Gandhi Regional Institute of Health and Medical Sciences, Shillong, Meghalaya

*Corresponding author: tonyete14@gmail.com

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Abstract Tuberculosis remains one of the most important cause of preventable mortality and morbidity worldwide. Cardiovascular involvement in tuberculosis, though relatively rare, can be hemodynamically life threatening if not identified or diagnosed appropriately. Here we are reporting a case of sputum positive pulmonary tuberculosis with dilated cardiomyopathy which improved after administration of antitubercular drug along with decongestive therapy.

Keywords: Tuberculosis, dilated cardiomyopathy

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1. Introduction

Tuberculosis (TB) is one of the most fatal infectious diseases, which continues to be a major global health problem. Cardiovascular involvement is very rare in Tuberculosis and represents 1-2% of total patients. Tuberculosis pericarditis represents the predominant form of cardiovascular TB. Involvement of myocardium is very rare with limited data in literature and is available mostly from autopsy series. Tuberculous myocarditis may occur by haematogenous or lymphatic spread or directly from the contagious structures like pericardium. If undiagnosed, it may lead to left ventricular failure, arrhythmias like atrial fibrillation, ventricular tachycardia and sometimes even to sudden cardiac death. We are reporting a case of sputum positive pulmonary tuberculosis who presented with dilated cardiomyopathy with severe left ventricular dysfunction and improved with antitubercular and decongestive therapy.

2. Case Report

A 32 year old female presented with low grade prolonged fever, cough with expectoration and progressively worsening dyspnoea for last one month. There was no past history of any major illness or history of exposure to any chronic disease. On admission, patient was febrile, had dyspnoea at rest and she was tachypneic. On examination, she was conscious, well oriented, had mild pallor, tachycardia (pulse rate 124/minute), bilateral ankle oedema, normotensive

(blood pressure 110/70 mmHg), respiratory rate 26/minute and raised JVP. There was no lymphadenopathy, clubbing or cyanosis. Chest examination showed bilateral inspiratory crepitation with diminished breath sounds. Cardiovascular examinations showed diffuse apical impulse with left ventricular S3 and a pansystolic murmur of mitral regurgitation. The abdomen was mildly distended with tender hepatomegaly. There was no neck stiffness or sign of meningeal irritation. Her oxygen saturation was 80-85% on pulse oximetry. Blood investigation revealed Hb-10.2 gm/dl, TLC-8300/mm³, DLC N83 L0, M0, ESR- 68 mm/hr. Liver function tests showed serum bilirubin-3.0mg/dl, direct-1.4mg/dl, indirect-1.6mg/dl, SGOT-293 U/L, SGPT-143 U/L, Alp-165, total protein- 5.6 gm/dl, albumin-2 gm/dl, globulin-3.6 gm/dl. Hepatitis serology and HIV were negative. Renal function test and coagulation profile were normal. Chest X-Ray showed non homogenous opacity in the left upper zone with cardiomegaly (Figure 1). Sputum for AFB was positive (2+). Ultrasonography abdomen showed hepatomegaly with mild ascites. ECG showed sinus tachycardia with non specific ST-T changes. Echocardiography was showing global hypokinesia with dilated chambers with moderate mitral regurgitation, moderate tricuspid regurgitation, severe left ventricular dysfunction with an ejection fraction of 25%; left ventricular end systolic and end diastolic dimensions were 37 mm and 57 mm and minimal pericardial effusion (Figure 2). A diagnosis of pulmonary tuberculosis with dilated cardiomyopathy was made and was treated with intravenous diuretics followed by oral, ACE inhibitor (ramipril 2.5 mg), carvedilol (3.125 mg twice daily), antibiotics and antitubercular therapy in the

form of isoniazid (300 mg), rifampicin (450 mg), ethambutol (800 mg) and pyrazinamide (1500 mg) for two months as intensive phase followed by isoniazid and rifampicin for four months as continuation phase. She responded well to treatment with reduction on severity of dyspnoea,

disappearance of peripheral oedema and chest clearance on auscultation. Follow up chest X-Ray showed mild cardiomegaly with normal lung fields (Figure 3) and there was improvement in ejection fraction in echocardiography (LVEF 35%).

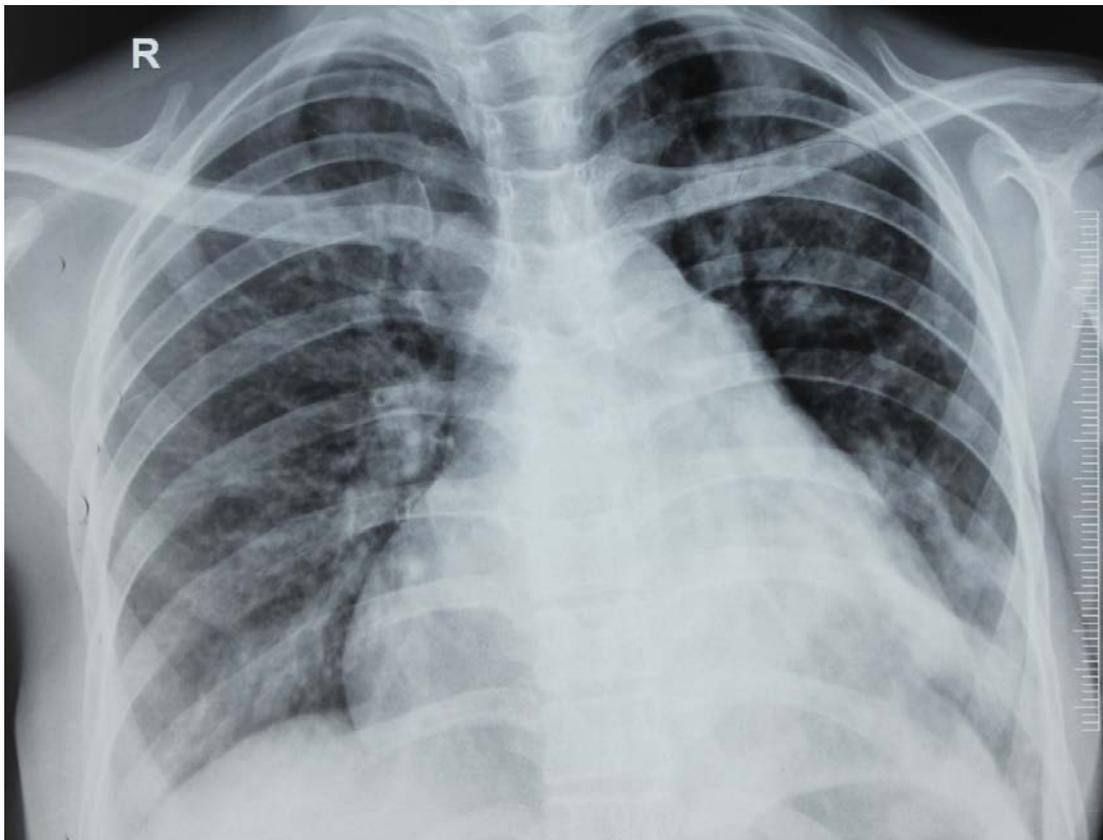


Figure 1. Chest X ray showing non homogenous opacity in left upper zone with cardiomegaly

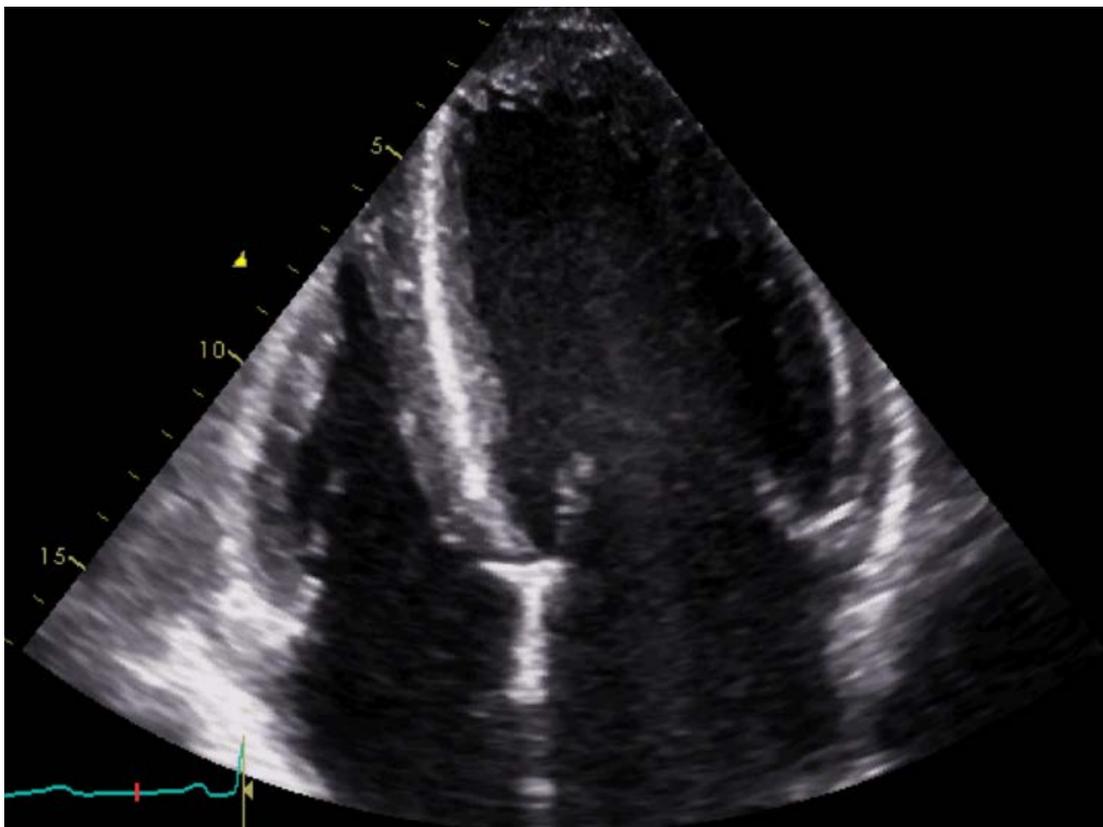


Figure 2. Echocardiography showing dilated chambers of the heart

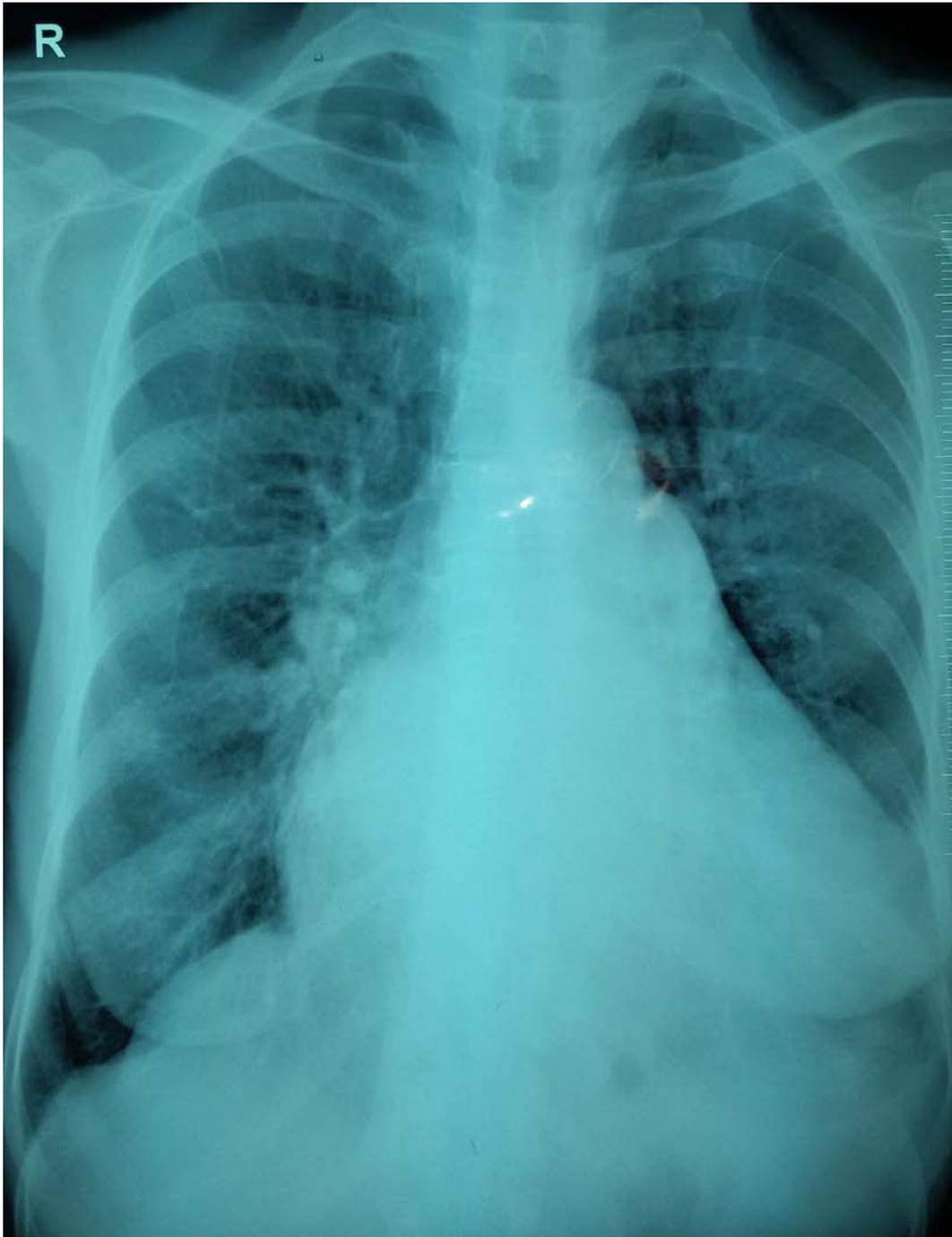


Figure 3. Chest X-Ray showing mild cardiomegaly with normal lung fields during follow up

3. Discussion

Despite being almost 100% curable, TB is still a leading cause of morbidity and mortality worldwide, representing second most common cause of death from infectious disease globally after HIV [1]. The latest World Health Organization (WHO) figures indicate that there are 9.0 million (range, 8.6 million -9.4 million) incident cases of TB, equivalent to 126 cases per 100000 population. India has about one quarter (24%) of global burden of TB with annual cases of about 2 million to 2.3 million [2]. Although TB can affect any organ system of the body, it is believed to spare four organ systems: heart, thyroid, skeletal muscle and pancreas [3]. Cardiovascular involvement is a relatively uncommon manifestation in patients with

TB. It is estimated that cardiac TB accounts for about 1-2% of all cases of TB in immunocompetent persons [4]. It mainly affects the pericardium and tuberculosis pericarditis is the commonest form of cardiovascular TB. Involvement of myocardium is very rare and usually TB occurs elsewhere in the body. Although cardiac TB was first described by Maurocordat in 1664 followed by Morgagni in 1761 [5], it was Laennec in 1826 who affirmed the existence of cardiac TB and described heart as thirteenth rank among organ affected by tuberculosis [4]. Tuberculous involvement of myocardium occurs more commonly by means of haematogenous spread, less often by retrograde lymphatic drainage from the tuberculous mediastinal lymph node. Rarely, there may be direct spread from contagious structures like tuberculosis pericarditis. Histopathologically, myocardial TB can be divided into three components: 1) nodular tubercles of the

myocardium that varies from pea to egg size with central caseation, 2) milliary tubercles of the myocardium complicating generalised milliary disease, and 3) diffuse infiltration associated with tuberculous pericarditis where myocardium is infiltrated by granulation tissues containing giant cells, endothelial cells and lymphocytes [6]. Clinically myocardial TB may remain asymptomatic or may present with congestive heart failure [7], various rhythm disturbances like ventricular and supraventricular tachycardia or varying degrees of heart block [8]. There are few isolated case reports of cardiovascular tuberculosis as right ventricular outflow tract obstruction [9], ventricular pseudoaneurysm, aortic regurgitation and coronary arteritis and even sudden cardiac death [10]. Myocardial TB is often not diagnosed during life, but with high degree of clinical suspicion diagnosis can be confirmed by an endomyocardial biopsy. Our case presented with prolonged fever with features of congestive cardiac failure, sputum was positive for acid fast bacilli, radiological evidence of cardiomegaly and echocardiographic evidence of dilated cardiomyopathy. The patient's clinical features improved significantly after antitubercular therapy.

4. Conclusion

Although rare, myocardial tuberculosis should be suspected as a cause of dilated cardiomyopathy in any patient with feature of tuberculosis elsewhere in the body. If it goes unrecognised, myocardial tuberculosis is often fatal; with proper early treatment, however, it is amenable

to cure. Increasing recognition of this entity may help us to detect more cases of this curable form of dilated cardiomyopathy in areas of high prevalence of tuberculosis like India.

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