

## Myocarditis and Pulmonary Tuberculosis: A Case Report

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### Abstract

Tuberculosis is one of the leading causes of death, worldwide. The predisposing factor of Cardiovascular Disease (CVD) in tuberculosis is due to the direct effect of Mycobacterium tuberculosis on the myocardium and coronary arteries, immune activation, and cytokine storm. We are presenting a case study of a 51-year-old male who presented with shortness of breath, cough with expectoration, and fever. He had a history of chronic smoking. High fever persisted for the next 4 days when the patient developed pain in the chest radiating to the left side of the shoulder. The patient was critical and intubated. ECG showed T wave inversion. Troponin-T was positive. CK-NAC 360 U/L, CK-MB 36 U/L, SGOT 3303 U/L.

**Keywords:** Myocarditis, Pulmonary tuberculosis, CK-NAC, CK-MB, Troponin

### Introduction

Tuberculosis is the 13th leading cause of death and it is conserved as the second leading infectious killer after COVID-19 worldwide. In 2020, around 1.5 million death has been reported due to tuberculosis [1]. Worldwide the burden of tuberculosis and cardiovascular disease is enormous [2,3]. The predisposing factor of Cardiovascular Disease (CVD) in tuberculosis – the direct effect of Mycobacterium tuberculosis on the myocardium and coronary arteries, immune activation and cytokine storm, and cross-reactivity of anti-mycobacterial antibodies against cardiovascular tissue [4]. Hence it plays a role in the inflammatory mechanism of the atherosclerotic process.

### Case Presentation:

A 51-year male presented in the emergency ward with complaints of shortness of breath, cough with expectoration, and fever for the last 10 days. He had a history of smoking bidi for more than the last 30

years. There was no history of other comorbidities, no history of past tuberculosis. On auscultation bilateral Ronchi were present, left side Crepts were present, Heart rate 110 /min, BP 150/85 mmHg, pO<sub>2</sub> <93. ECG all 12 lead was normal. The liver function test, kidney function test, and Blood Sugar were normal. HIV, HBsAg, and Anti-HCV were non-reactive. Sputum for AFB was positive (3+).

Chest x-ray (Fig – 1) showed Chronic Obstructive Pulmonary Disease (COPD) with acute exacerbation and left mid-zone pneumonitis was diagnosed. HRCT chest (Fig – 2) was done and the finding was:

1. Pan-lobular emphysematous changes involving bilateral lung fields (mainly in the upper lobes).
2. Multiple lobulated cystic lesions involving the left upper lobe with the rest of the left upper lobe showing diffuse air space opacities with multiple air bronchograms, also involving the lingular segments – suggestive of multiple

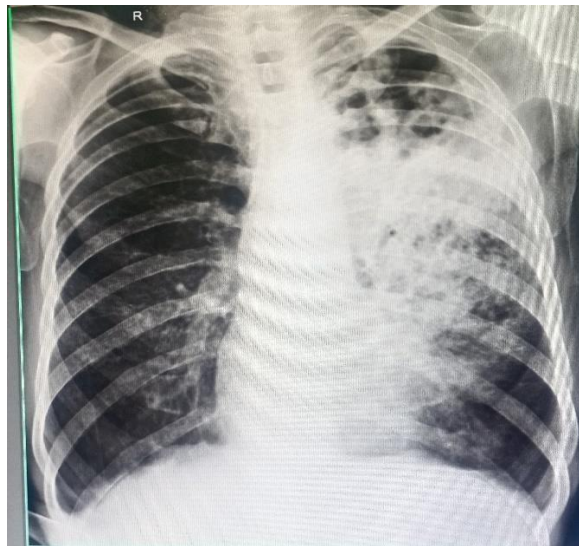
cavitary lesions with adjacent consolidatory changes. Above mentioned cavity also shows communication with the left upper lobe bronchus.

3. Patchy peribronchial infiltration with linear branching opacities gives a tree-in-bud appearance in the rest of the lung fields predominantly in the right middle lobe.
4. Mild left-sided pleural effusion

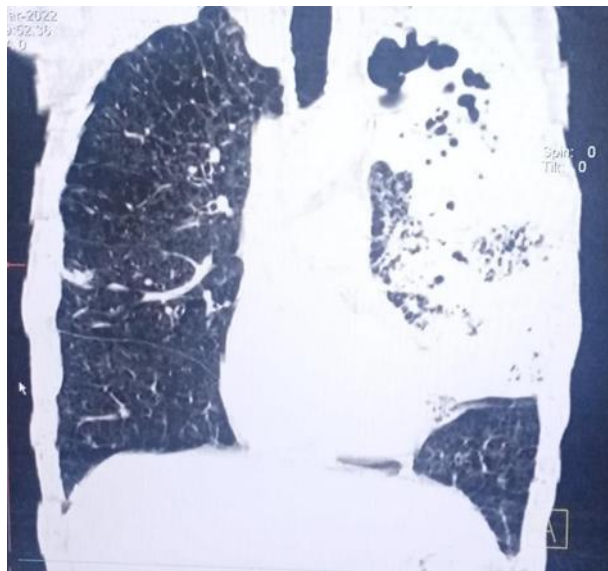
#### 5. Mediastinal lymphadenopathy

High fever persisted for the next 4 days when the patient developed pain in the chest radiating to the left side of the shoulder. The patient was critical and intubated. ECG (Fig – 3) showed T wave inversion. Troponin-T was positive. CK-NAC 360 U/L (Normal Range: 24 – 145 U/L) CK-MB 36 U/L (Normal Range: 0 – 24 U/L), SGOT 3303 U/L (Normal Range: 0 – 35 U/L)

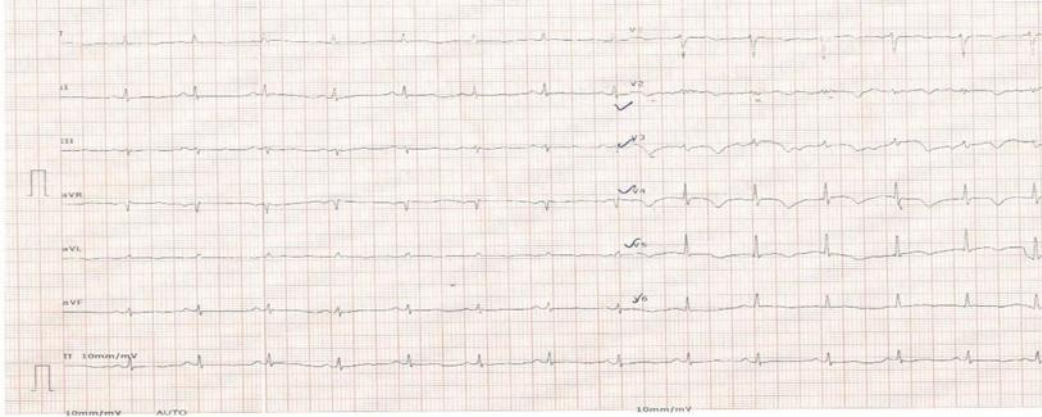
**Fig – 1: X-Ray Chest PA View**



**Fig – 2: HRCT Chest**



**Fig – 3: ECG shows T wave inversion V2, V3, V4, V5, and V6**



**Discussion:**

There have been several studies in Russia and India which support the relationship between coronary artery disease and tuberculosis [5]. Kinare, et al. reported a case of a 19-year-old male who ended fatally due to a large ventricular aneurysm obtained from myocardial infraction caused by tubercular coronaritis of the left anterior descending branch [6]. The intracellular pathogen involved in chronic and latent infection has been implicated in the development of cardiovascular diseases (CVD) [7]. There has been a strong relationship between infection and inflammation and immune reaction in patients with CVD [8]. The pro-inflammatory profile (activated macrophages, T cell, elevated cytokine) has been studied in a patient with tuberculosis [4]. Tuberculosis also induces auto-immunity through molecular mimicry of the heat-shock protein system [9]. Cases of sudden death and arthritis, a rare occurrence, is suggestive of the direct effect of myocardial tuberculosis on the myocardium and coronary artery [10].

According to WHO, the highest tuberculosis burden country in the world is India, which has an estimated incidence of 26.9 lakh cases in 2019 [11]. An estimated 17.9 million people died from CVDs in 2016, representing 31% of all global deaths. Of these deaths, 85% were due to heart attack and stroke [12].

**Conclusion:**

Individuals at risk of CVD may demonstrate raised blood pressure, glucose, and lipids as well as overweight and obesity. Identifying those at the highest risk of CVDs and ensuring they receive appropriate treatment can prevent premature deaths. However, tuberculosis can be one of the risk factors

for CVD and it should not be missed in the Indian population as India has the highest tuberculosis burden.

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