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Case Report

Post tuberculosis obstructive airway disease: an underdiagnosed complication of pulmonary tuberculosis.

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Abstract

Pulmonary infection with *Mycobacterium tuberculosis* is a well-known morbidity in Sri Lanka, where tuberculosis (TB) is endemic, and it poses huge challenge to the healthcare system. A number of pulmonary complications occur following pulmonary TB. Post TB obstructive airway disease is one of them but under diagnosed in susceptible individuals, which results in presentation of patients at advanced disease with its complications, leading to poor patient outcome and quality of life. Here we report a case history of a patient who had past history of bacteriologically positive pulmonary TB, presented to us with advanced obstructive airway disease and its complications.

Key words

Post TB obstructive airway disease, pulmonary fibrosis, COPD

Introduction

Tuberculosis is caused by the bacilli *Mycobacterium tuberculosis*. About ⅓rd of the world population is affected by TB (1). Among them those with pulmonary TB have high risk for developing TB related pulmonary complications such as lung fibrosis, cavitatory lung lesion, bronchiectasis, aspergilloma and obstructive airway disease. Pulmonary complications after TB infection are demonstrated even after successful completion of treatment and elimination of the organism from the site (2)

Case report

A 55-year-old male with the past history of bacteriologically positive pulmonary TB and completed six months of anti TB treatment, fifteen years ago, admitted to the casualty surgical unit with fever and abdominal pain of one week duration. He was diagnosed with multiple liver abscess and found to be hypoxic with the SpO_2 67% on room air. His chest x-ray revealed extensive upper lobe pulmonary fibrosis bilaterally. Arterial blood gas analysis revealed decompensated type 2 respiratory failure. After commencement of appropriate antibiotic therapy for liver abscess he was transferred to casualty medical unit for further evaluation and management of type 2 respiratory failure.

He had chronic cough with expectoration of scanty sputum for past one year for which he consulted local GP on few occasions and received courses of antibiotic therapy. He also reported exertional shortness of breath mMRC grade III/IV and tiredness which worsened over last two months with wet cough and expectoration of purulent sputum without hemoptysis. He also had unintentional weight loss of 8 Kg and poor appetite over a year. He did not complain of night sweats neither did he have urinary symptoms. He frequently consumes toddy, but never smoked. He did not have history of industrial dust exposure. He did not have any pets or poultry at home. There is no personal history of atopy. His past medical history was not significant, and he was not on any medication for chronic illnesses. He is a manual worker.

On examination his BMI was 19 Kg/m^2. He was alert, anxious and tachypneic. He was not pale or icteric. He did not have central or peripheral cyanosis, clubbing, cervical lymphadenopathy or hypertrophic osteoarthropathy. He had flaps, bilateral pitting ankle oedema and his JVP was elevated. His pulse rate was 90 beats per minute which was regular with good volume, blood pressure was 126/92 mmHg. Cardiac auscultation revealed loud pulmonary second heart sound and pan systolic murmur best heard at left lower sternal edge. Respiratory system examination revealed

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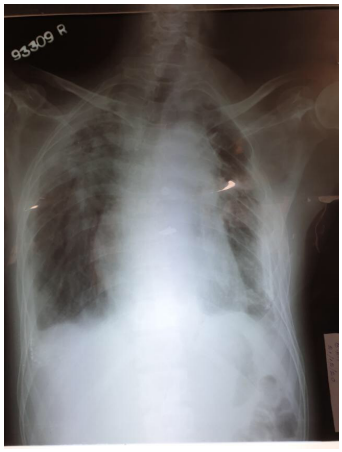
equal breath sounds in both sides with inspiratory crackles and expiratory wheeze heard bilaterally. On abdominal examination, there was tenderness over right hypochondrium. There was no hepatosplenomegaly or free fluid identified clinically.

On investigation, complete blood count revealed neutrophil leukocytosis with normal hemoglobin and platelet count, with raised inflammatory markers which were tallying with ongoing infection and inflammation. Initial arterial blood gas analysis revealed PH of 7.26 with the PCO_2 of 89.1 mmHg. He had mild impairment of liver function. His renal function test, serum electrolytes, coagulation profile, serum amylase, troponin I and urine microscopy were normal. ECG revealed right axis deviation with evidence of pulmonary hypertension. Sputum for AFB, gram stain, bacterial culture and gene expert were negative. Aspergillus specific IgG was negative. Chest X-ray revealed bilateral lung fibrosis and evidence of bronchiectasis. Ultrasound abdomen revealed multiple liver abscess. Transthoracic echocardiogram revealed moderate pulmonary hypertension with TR and TRPG of 48 mmHg, ejection fraction was 70%. HRCT chest revealed both right and left upper lobes cystic bronchiectasis with underlying fibrosis with mediastinal shift to the left and bilateral pleural effusion. There was no identifiable intracavitatory bodies or aspergilloma.

He was treated with noninvasive ventilation (NIV) with other supportive management such as IV antibiotic, systemic steroids, nebulization with inhaled bronchodilators, nutritional support and intravenous fluid to supplement his oral intake. With serial arterial blood gas analysis monitoring NIV support was gradually weaned off (time dependent) with significant clinical improvement. He was started on inhalers with tiotropium, a long-acting muscarinic antagonist (LAMA) and salmeterol/fluticasone, a combination of long-acting beta-2 agonist/inhaled corticosteroid (LABA / ICS). On discharge his inhaler technique was assessed and optimized. A month after discharge spirometry was done which revealed obstructive pattern with negligeable bronchodilator reversibility and severe airflow limitation with the FEV_1 / FVC ratio of 0.65, FEV1 of 0.85 compatible with the diagnosis of TB associated obstructive pulmonary disease (TOPD).



Chest X-ray: Extensive fibrosis of upper lobes of both lungs

With the commencement of appropriate pharmacological and non-pharmacological management, significant improvement was noted in his clinical condition and exercising capacity, with stable PCO_2 in arterial blood gas, on clinic follow up.

Discussion

Chronic obstructive airway disease (COPD) is the 3rd most common cause of death worldwide. According to GOLD 2020 guidelines, COPD is defined as FEV_1/FVC of less than 0.7 in spirometry after bronchodilators (5).

Obstructive airway disease is a well-known complication following pulmonary TB and it was reported time to time (1). Delay in commencement of treatment, inadequate treatment , recurrent infection, tobacco smoking, exposure to household smoke and industrial exposure are considered to be risk factors for the development of post TB obstructive airway disease (4). According to studies, extensive lung parenchymal damage starts from 6 months to 18 months from the diagnosis of primary pulmonary infection (1,3). Another important fact to note is, the individuals who were satisfactorily treated also developed post TB lung damage including obstructive airway diseases (2). It brings up the concern about the pathogenesis of post TB obstructive airway disease which is mainly due to the host response to the infection, including production of various cytokines which resulted in lung damage.

Significant number of patients are affected by pulmonary TB in Sri Lanka. Pulmonary fibrosis associated with restrictive lung disease, bronchiectasis and aspergilloma are the frequently diagnosed complications of post pulmonary TB in Sri Lanka. Even though post TB obstructive airway disease is frequently demonstrated in studies, it is under diagnosed (5) in clinical setup because spirometry is required for the definitive diagnosis, which is not freely available except tertiary centers, in Sri Lanka. Those who are not diagnosed, present to the health care center once the disease is well advanced with its complications, which causes significant reduction in the life span of the patient. Treatment modalities available for advanced COPD are not affordable by significant number of patients in Sri Lanka, because of their poor socio-economic background. It further rises the rate of adverse outcome.

Our patient presented with post pulmonary obstructive airway disease complicated with type 2 respiratory failure and pulmonary hypertension. He is a nonsmoker and no additional occupational risk factors to note. Household exposure to smoke especially from firewood could be an added risk factor for him to develop post TB obstructive airway disease. Firewood is the most used fuel for cooking by the population with poor socio-economic background in Northern part of Sri Lanka.

Conclusion

Obstructive airway disease is a well-known complication of post pulmonary tuberculosis but being under diagnosed in Sri Lanka. Early detection of complications and initiation of appropriate treatment improves patient outcome and alleviate financial burden to the healthcare system.

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