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A Sequela of Interstitial Lung Disease in a Post Tubercular Lung Disease Patient: A Case Report

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ABSTRACT

Interstitial lung disease is a large group of disorders associated with lung scarring. It presents with a dry cough leading to progressive dyspnea. Pulmonary auscultation exhibits a crackle of inspiration. Interstitial lung disease is commonly associated with patients' occupations. This disease has a higher incidence in males and has a high mortality rate. Fungal and bacterial infections also have some role in interstitial lung disease. Although rare, patients with pulmonary tuberculosis can rarely develop interstitial lung disease. However, aspergillosis, airway infection, and atypical mycobacterial infection are common sequelae of pulmonary tuberculosis. A computed tomography scan is the radiological modality of choice. It shows an abnormal opacity pattern with nodular, linear, reticular, or reticulonodular strength. We had an elderly male diagnosed with interstitial lung disease confirmed with the clinical findings and radiological techniques. The patient had suffered from tuberculosis seven years back that was treated completely with anti-tubercular therapy. The patient experienced shortness of breath despite a high flow of oxygen. Despite giving nintedanib, the patient's condition did not improve, leading to death.

Keywords: Mycobacterium Tuberculosis; Anti-Tubercular Therapy (att); Interstitial Pulmonary Fibrosis; Post-Tubercular; Interstitial lung disease

INTRODUCTION

Interstitial lung disease is a broad term for disorders that present with exertional dyspnea, dry cough as symptoms and inspiratory crackles on auscultation. In general, the prevalence of interstitial lung disease is 20% higher in men (80.9 per 100,000) than in women (67.2 per 100,000).[1] Etiology includes occupational work exposure; fungal and atypical bacterial pneumonia are some of the common causes. An abnormal pattern of opacity on Computed Tomography (CT) scan in the form of linear, reticular, nodular and reticulonodular strengthen the diagnosis of interstitial lung disease. It is estimated that there are more than eight million new cases of tuberculosis and 1.3 million deaths yearly.[2] Common sequelae of pulmonary tuberculosis include secondary infection with aspergillosis, atypical mycobacteria, and secondary bacterial airway infection. Interstitial lung disease is sometimes misdiagnosed as pulmonary tuberculosis; however, in a rare scenario, patients can develop interstitial lung disease in a post tubercular lung patient.[3] In this report, we present the patient of a 54-year-old male diagnosed with interstitial lung disease.

CASE PRESENTATION

A 54-year-old elderly male with a family history of tuberculosis presented to the outpatient department with chief complaints of shortness of breath and cough with expectoration for the past year, which worsened in the past month. In addition, he also had bilateral chest pain for the past fifteen days. Her past medical history revealed he was diagnosed with pulmonary tuberculosis seven years back, for which he was taking an Anti-tubercular drug regimen and showed recovery. He has a negative history of other comorbid illnesses like diabetes and hypertension; he was also allergic to dust.

On admission, he was febrile (100.5°F), tachycardia (110 beats/minute), tachypnea (32 breaths/minute), oxygen saturation of 86% on room air, and blood pressure of 130/80 mmHg. He also had clubbing of grade 3; however, the rest systemic examination findings were unremarkable. His biochemical and hematological parameters were unremarkable except for leukocytosis.

His respiratory examination demonstrated the presence of fine bilateral crackles. His chest x-ray revealed white interstitial lung marking, suggestive of interstitial lung disease (Figure 1). A non-contrast CT of the chest was performed, which revealed interstitial lung disease diffuse crazy-paving, consolidation bilaterally, and focal parenchymal band in the right upper lobe of the lung (Figure 2).

Figure 1: White interstitial lung marking is shown by an arrow on the Posteroanterior view of the Chest x-ray.

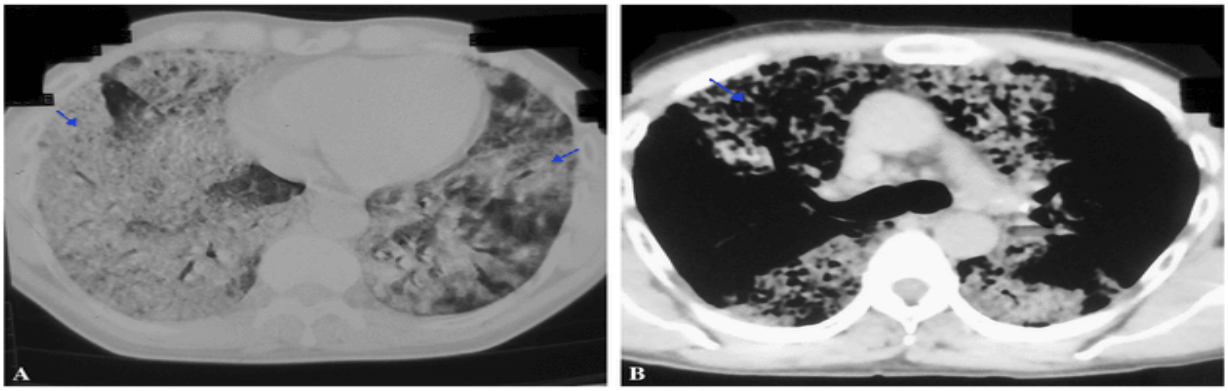

Figure 2: Diffuse crazy-paving, consolidation bilaterally (A) and focal parenchymal band in the right upper lobe of the lung (B).

He also underwent diagnostic tests like bronchoalveolar lavage, anti-Cyclic Citrullinated Peptides (anti-CCP) antibody, rheumatoid factor assay, culture and sensitivity for gram stains, fungal stain, and Acid-Fast Bacilli (AFB) were performed. Out of which, AFB culture and sensitivity were positive (>10 AFB oil immersions fields). The predisposition of pulmonary tuberculosis most likely worsens his interstitial lung disease.

The patient was given intravenous fluids, anti-tubercular therapy, methylprednisolone, and broad-spectrum antibiotics. A nasal cannula was started as respiratory support therapy. The patient maintained saturation Spo2 of 86% at 6L/min oxygen flow rates. His successive chest x-ray revealed his condition worsened despite treatment, and unfortunately, the patient succumbed to death (Figure 3).

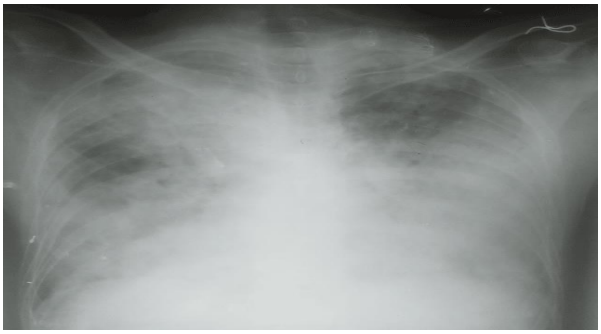

Figure 3: Progressive massive fibrosis on PA view of Chest x-ray.

DISCUSSION

We reported a case of a 54-year-old elderly male complaining of shortness of breath and cough with expectoration. The symptoms persisted for the past year, worsening in the past month. His radiological and clinical picture confirmed the existence of interstitial lung disease. The clinical patterns of ILD are heterogeneous and are further classified into different groups based on disease behaviors (reversible/irreversible, progressive/stable).[4] This heterogeneity in the clinical pattern is explained by variability in the immune responses to a pathogen among different individuals based on their genetic profile. Generally, in response to lung injury, our body generates an adequate response by producing the right amount of tissue to repair the damage. But in interstitial lung disease, an abnormal healing response causes the lung parenchyma to become scarred and thickened around the alveoli, limiting oxygen diffusion into our bloodstream. Environmental factors like smoking, occupational exposure to dust, toxic chemical gases, and some drugs can trigger interstitial lung disease. Following tuberculosis, patients exhibit a broad range of outcomes, from completely asymptomatic to severe respiratory distress. Drugs including chemotherapeutics, heart medications, anti-inflammatory drugs, and antibiotics can also cause injury to the respiratory epithelium.

Most interstitial lung diseases have a similar pathology involving structural remodeling of the distal airways causing impaired gaseous exchange. Earlier, this remodeling was thought to result from chronic inflammation; however, more recent studies have postulated that this is due to the tissue injury with an abnormal wound healing process resulting in collagenous fibrosis.[5] Even though most of the causes are unknown, various risk factors have been associated with interstitial lung disease. The most significant risk factors include smoking, hepatitis C, tuberculosis, history of pneumonia, and COPD. It was also elucidated that the risk of developing interstitial lung disease increases sharply with age and is more common in males than in females. It is challenging and tough to know the exact cause of the interstitial lung disease because it is multifactorial.

Interstitial lung diseases encompass a wide range of diseases. It requires an intense hematological, immunological, and radiological workup such as complete blood work, autoimmune workup, chest x-ray and CT scan to diagnose a case of interstitial lung disease. Since chest x-ray is less accurate in detecting the early degenerative changes, it is usually followed by a high-resolution CT chest, which is the investigation of choice for detecting interstitial lung disease. Spirometry can also be done to identify the type of respiratory abnormality (restrictive/ obstructive) and to assess the diffusion capacity of the lungs. But to obtain a definitive diagnosis of pulmonary fibrosis, microscopic examination of the lung tissue obtained from bronchoscopy, bronchoalveolar lavage, or surgical biopsy is necessary. Interstitial lung disease can lead to a series of life-threatening complications, including pulmonary hypertension, right-sided heart failure (cor pulmonale), respiratory failure, and secondary infections and may increase the risk of pulmonary tuberculosis. Granulomas formed in tuberculosis have a complex morphology with a necrotic material in the center walled off by Nature Killer (NK) cells, lymphocytes, and macrophages. TNF-alpha and IFN-gamma play

a major role in maintaining the granulomas intact. According to Takahisa Gono et al., TNF-alpha and IFN-gamma levels are higher during an acute exacerbation.[6] This rise in TNF-alpha and IFN-gamma could lead to the dislodgement of the well-formed granuloma containing mycobacterium, which poses a further risk of re-activation of Mycobacterium tuberculosis. The clinical management of active tuberculosis in a patient with underlying ILD is quite backbreaking and challenging because the interstitial process and the underlying fibrosis may mask the infectious foci.[7] The scarring in the interstitial lung disease is irreparable, and management might not always be effective in halting the disease progression. But certain medications may temporarily improve the manifestations and slow the disease's progression.

Medications include corticosteroid and nintedanib, an intracellular tyrosine kinase inhibitor with anti-fibrotic properties, and it was one of the novel drugs approved for the idiopathic pulmonary fibrosis treatment and lately, it has been recognized to be useful even in other chronic fibrosing interstitial lung disease with a progressive phenotype and systemic sclerosis-associated interstitial lung disease and medications that reduce stomach acid.[8] Supplemental Oxygen therapy and surgery can also be effective. Our patient was managed conservatively by giving intravenous fluids, anti-tubercular therapy, and nasal cannula as respiratory support therapy. The patient was maintaining saturation SpO2 of 86% at 6/liters of oxygen. The patient could not be given standardized treatment because of resource limitations. Unfortunately, the patient succumbed to death.

CONCLUSIONS

Interstitial lung disease is the lung scarring usually related to the patient's occupation. The patient usually presents with a dry cough with progression in shortness of breath and inspiratory crackles on lung auscultation. The opacity pattern on a CT scan is diagnostic for interstitial lung disease. Nintedanib is an approved therapeutic agent of choice with antifibrotic activities. More case reports are needed to confirm the relation between post-tuberculosis patients with interstitial lung disease.

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