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RESEARCH ARTICLE

CHRONIC PULMONARY ASPERGILLOSIS IN AN OLD TREATED CASE OF TUBERCULOSIS: A CASE REPORT

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ABSTRACT

The diagnosis of Chronic Pulmonary Aspergillosis (CPA) is challenging and a high index of suspicion for CPA should be kept in mind in endemic areas of tuberculosis (TB) as the clinical features of both overlap. It can be life threatening if not intervened timely. We present a case of an elderly male; diabetic, alcoholic but reformed smoker and known case of treated pulmonary TB who developed CPA two years later. It's emphasized that proper clinical examination, microbiological evidence, radiological findings, medical or surgical intervention if needed can reduce the case fatality rate.

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INTRODUCTION

Chronic Pulmonary Aspergillosis (CPA) is a progressive pulmonary disease which affects predominantly immunocompetent individuals or subtly immunocompromised, particularly those with long-standing airway disease, such as bronchial Asthma or Cystic fibrosis, most commonly post-treatment tuberculosis (TB). [1] Aspergillus has a propensity to invade pulmonary blood vessels, resulting in hemoptysis and pulmonary infarction. In addition to TB, other immunosuppression conditions such as diabetes mellitus, malnutrition, alcoholism, connective tissue diseases, and prolonged corticosteroid therapy also increase the risk for development of CPA. [2] We present a case of an elderly male; diabetic, alcoholic but reformed smoker and known case of treated pulmonary TB who developed CPA two years later.

CASE PRESENTATION

A 51 year old male patient reported to the Medicine department of Adesh Medical College and Hospital, Shahbad (Ambala) with chief complaints of cough with expectoration, low grade fever on and off and dyspnoea since five months. The expectoration was greenish colored and associated with hemoptysis.

The patient was a reformed smoker, chronic alcoholic, diabetic, non-hypertensive and was a treated case of pulmonary tuberculosis two years back. On examination, the patient looked emaciated, was agitated but conscious to time, place and person. The vitals were: Blood pressure- 148/90 mmHg, Pulse Rate- 98 bpm and Respiratory rate was 24/ min. On auscultation, coarse crackles were heard on right side of chest and wheezing heard on both sides of chest. Abdomen, cardiovascular and neurological examination were unremarkable. A clinical diagnosis of bronchiectasis?Chronic Obstructive Pulmonary Disease(COPD) or relapse of tuberculosis was made. Sputum for Acid Fast Bacilli (AFB), Sputum for culture for Mycobacteria, Sputum for CBNAAT, Tuberculin test, ESR, Chest X-Ray and blood samples were sent as initial work up. Laboratory investigations revealed normocytic anaemia, lymphocytosis and mild eosinophilia, raised ESR (50 mm/hour) and HbA1C was 8.5%. Tuberculin test was positive. Sputum smears (two samples) for AFB were negative. CBNAAT report showed no mycobacteria. He was non reactive for hepatitis B, C and HIV. Chest X-Ray showed increased bronchovascular markings and consolidation in right upper lobe. HRCT was advised which revealed bronchial dilatation and diffuse upper right lobe consolidation and cavitation. (Figure 1). Serum IgG antibodies to Aspergillus fumigates was positive.

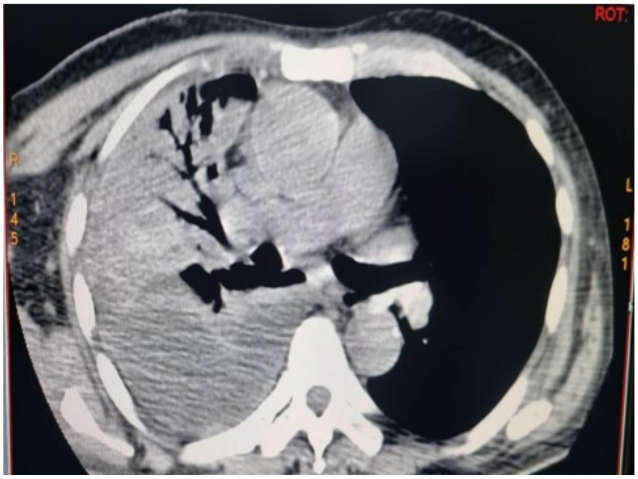

Figure 1. Diffuse right upper lobe consolidation seen on HRCT

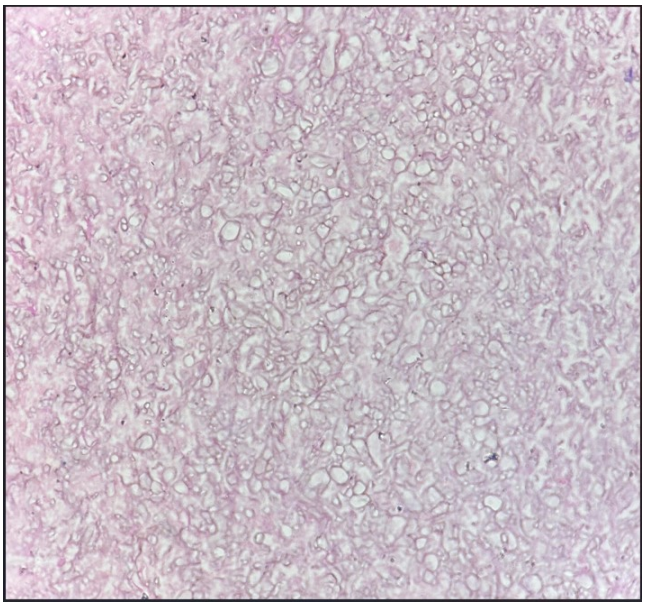

Figure 2. Large clusters of fungal hyphae with acute angle branching and spores

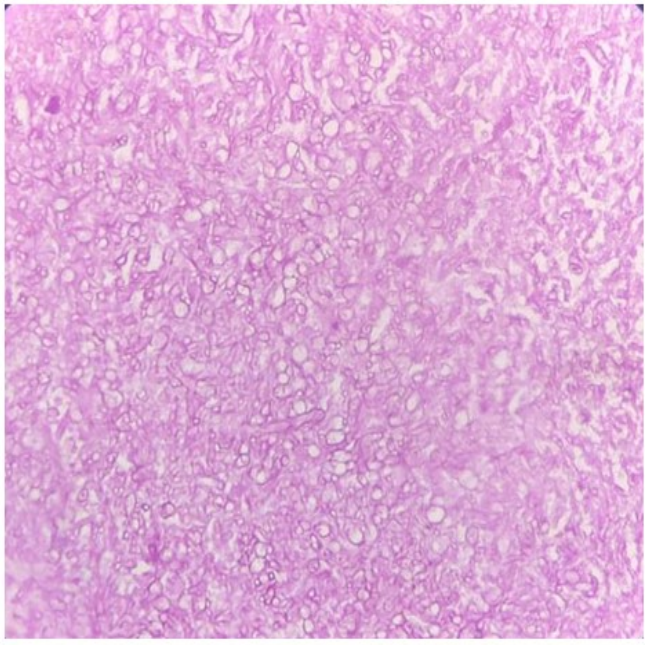

Figure 3. Fungal hyphae as seen in PAS stain

The patient was referred to CTVS department for further management. He underwent right upper lobectomy and the specimen was sent to the histopathology department. The specimen measured 12*9*3 cm. The external surface was unremarkable. Cut section showed few dilated bronchi. Microscopically, multiple sections examined revealed largely preserved bronchiolar lining, showing focal hyperplasia and ulceration at places. The bronchiolar lumen showed dilatation and presence of large clusters of fungal hyphae with acute angle branching and spores.(Figure 2). The peribronchial area showed dense chronic inflammation comprising of lymphocytes, plasma cells and few histiocytes. The interstitium showed numerous pigment laden macrophages and inflammatory cells along with presence of multiple thickened and congested blood vessels. No granuloma/atypia/malignancy was noted. PAS stain was positive for fungus.(Figure 3). The patient was subsequently put on antifungals along with cover of antibiotics to prevent superadded infection. He was advised for regular follow up after 2 months.

DISCUSSION

CPA affects an estimated 3 million people worldwide. More than half of cases of CPA occur as a complication of treated PTB. The criteria for CPA diagnosis are: illness of ≥ 3 months and all of:

Weight loss, persistent cough and/or haemoptysis; (2) chest images showing progressive cavitary infiltrates and/or a fungal ball and/ or pericavitary fibrosis or infiltrates or pleural thickening; and (3) a positive *Aspergillus* IgG assay or other evidence of *Aspergillus* infection. [3]

The spectrum of CPA ranges from Simple Aspergilloma, Chronic Cavitatory Pulmonary Aspergillosis (CCPA) to Chronic Fibrosing Pulmonary Aspergillosis (CFPA) when left untreated, and sub-acute Invasive Pulmonary Aspergillosis (SAIA), formerly called Chronic Necrotizing Pulmonary Aspergillosis. Even after treatment, it has a high case fatality rate. The clinical features of CPA overlaps with presentation of TB which makes the diagnosis challenging. CPA often presents with cough, chest pain or discomfort, weight loss, profound fatigue, severe shortness of breath, and life-threatening hemoptysis.[4] The TB patients have defective macrophages, monocytes, and T-cells function, as well as chemotaxis defects that predispose them to opportunistic fungal infections. Healed pulmonary TB can result in pulmonary cavities which can become infected with Aspergillus and an aspergilloma may then form after months or years of infection.[5]

A combination of clinical suspicion with radiological, microbiologic, and serologic findings are required for the diagnosis. Aspergillosis if diagnosed early can be treated and progression to fibrotic stage can be avoided efficiently. Microbiological testing principally for Aspergillus IgG antibodies should be employed to recognize early CPA in patients with residual pulmonary tuberculosis.[6]Serum IgG antibodies are rather helpful, but the results could be falsely negative in cases where the species is other than Aspergillus fumigatus or in patients under steroid treatment.[7] Surgery is curative in selected patients with localised disease and has been safely delivered in resource-poor settings.

Treatment with oral azole drugs can prevent clinical and radiological progression. [8] In our case, the patient had poorely controlled diabetes, chronic alcoholic and in addition past history of treated TB which became ground for Aspergillus infecton. Since the patient hailed from rural, low socioeconomic background and due to lack of awareness, he was irregular for consultation and hence his clinical improvement couldn't be followed up.

CONCLUSION

Post tubercular development of CPA is common in developing countries and can be responsible for high morbidity and mortality. A high clinical suspicion, early diagnosis and timely intervention can save the patient from devastating effects. Therefore, suitable measures should be taken to prevent coinfection of tuberculosis with opportunistic fungi in endemic areas of TB.

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