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Allergic Bronchopulmonary Aspergillosis (ABPA) in pre existing chronic obstructive pulmonary disease (COPD)-A case report

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ABSTRACT

Allergic bronchopulmonary aspergillosis (ABPA) is an entity of immunological pulmonary disease characterised by airway hyper-responsiveness to Aspergillus fumigatus colonised in the airways, as well as distinct radiological and serological findings. The case reported here is a 53-year-old farmer who was diagnosed with COPD seven years back presented with cough with expectoration, shortness of breath, wheeze. The radiological investigation revealed fleeting shadows, and a CT scan revealed central cystic bronchiectatic features, which are typical with ABPA. On further investigation the sputum examination revealed Aspergillus. Eosinophilia was found in peripheral blood smears, as well as elevated total IgE and fungus specific IgE levels in serum. As a result, ABPA was determined to be the cause. Management with steroids, antifungals improved patient symptoms and partial improvement in pulmonary infiltrates.

Keywords: Aspergillosis, asthma, COPD, eosinophilia, bronchiectasis, eosinophilia

1. INTRODUCTION

Allergic bronchopulmonary aspergillosis (ABPA) is a clinicopathological and immunological pulmonary condition characterised by a wide range of clinical symptoms produced by the respiratory epithelium's hyper-responsiveness to *Aspergillus fumigatus* (Flood-Page, 2018). Patients with asthma have a prevalence of 1–2%, steroid-dependent asthma has a frequency of 7–14 percent, and patients with cystic fibrosis have a prevalence of 2–15 percent. Hinson et al., (1952) first described it in 1952, although the diagnostic criteria have since been revised. Characteristic features in ABPA include coughing up thick mucus plugs and wheezing. Aspergillus antigen hypersensitivity, elevated total IgE levels and fungus specific IgE levels, peripheral and

pulmonary eosinophilia, pulmonary infiltrates seen on radiography and mucus impingement or bronchiectasis on radiological finding (Flood-Page, 2018). The case presenting here is a patient of COPD who developed ABPA after being exposed to a high concentration of fungal spores. Treatment with steroids and antifungals was attributed to complete symptom relief and partial remission of bronchiectatic changes.

2. CASE REPORT

A 53-year-old farmer having history of COPD since 7 years taking regular medication metered dose inhaler with spacer tiotropium 9mcg+formeterol 6mcg 2 puffs twice a day. Patient had history of occasional exacerbation episodes in winter which relieved on consulting local practitioner. Currently patientpresented to this hospitalwith a history of cough with expectoration containing mucous plugs, shortness of breath limiting moderate physical activities and wheeze since 3 weeks. Patient's symptoms developed after exposure to dust in farm. Symptoms were of nocturnal variation becoming worst at night. History of 3 episodes of intermittent fever and 1 episode of hemoptysiswas present. Patient had no history of any comorbidity such as diabetes, hypertension, and tuberculosis.

On admission patient was afebrile having heart rate 90/minute with blood pressure 120/80mm Hg, respiratory rate of 23cycles/minute, spo2-94% on room air. On systemic examination there was audible expiratory wheeze on bilateral lung fields along with scattered coarse crepitations. His peak expiratory flow was 360 L/min. CXR showed Bilateral hyperinflated and hyperlucent lung fields, Flattening of bilateral hemidiaphragams, Blunting of right cardiophrenic angle, Bronchiectatic changes in lower zones of bilateral lung fields as shown in Figure 1.

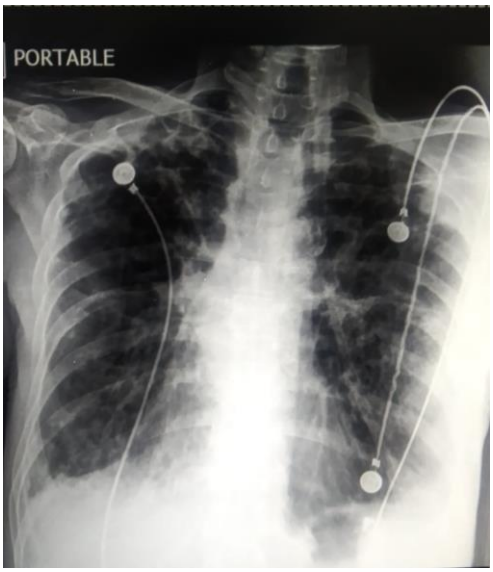

Figure 1 showing chest x ray revealing Bilateral hyperinflated and hyperlucent lung fields, Flattening of bilateral hemidiaphragams, Blunting of right cardiophrenic angle, Bronchiectatic changes in lower zones of bilateral lung fields

HRCT Thorax revealed bilateral central bronchiectatic changes, few subpleural bullae noted in right upper lobe, cavity with internal soft tissue density area noted in anterior and apicoposterior segment of left lower lobe in the upper lobes as shown in Figure 2a and b.

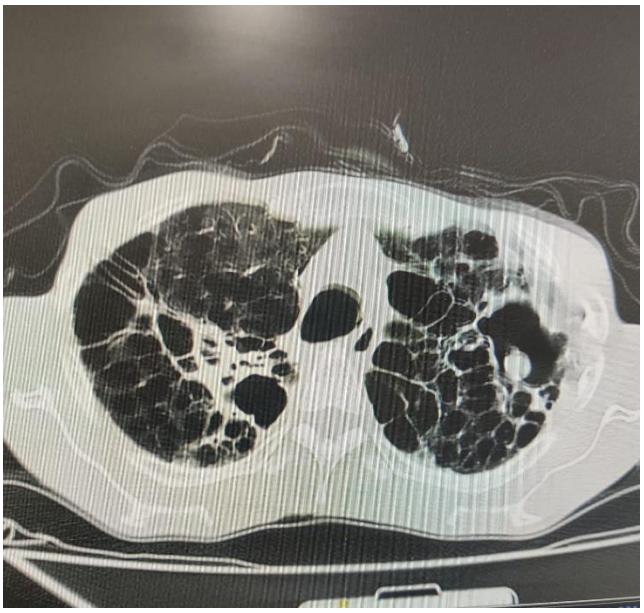

Figure 2a showing HRCT Thorax revealed Bronchiectatic changes in right upper lobe, few subpleural bullae noted in right upper lobe, cavity with internal soft tissue density area noted in anterior segment of left lower lobe

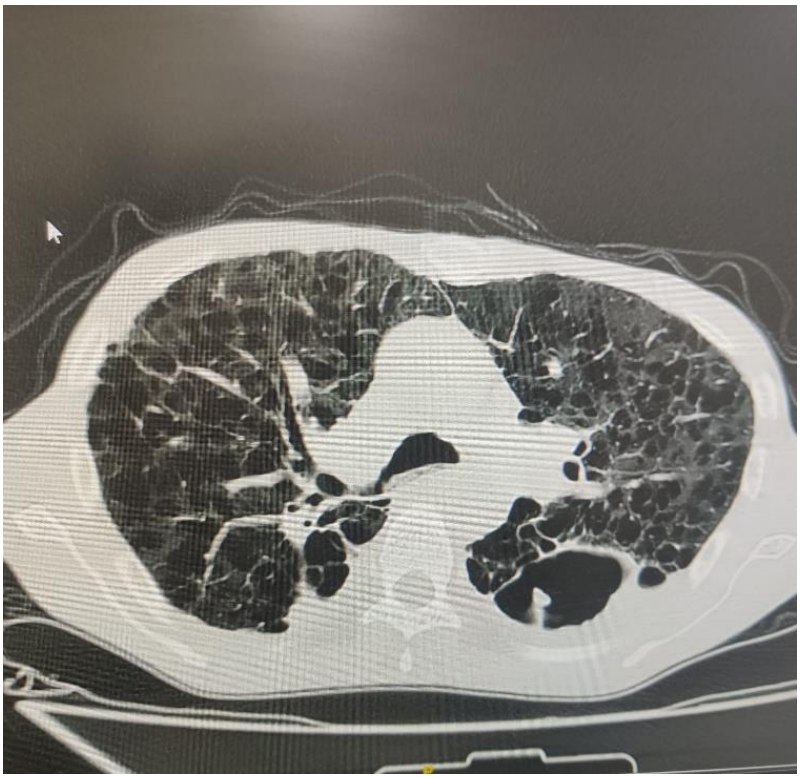

Figure 2b showing HRCT Thorax revealed cavity with internal soft tissue density area noted in apicoposterior segment of left upper lobe

Hematological evidences suggestive of ABPA are elevated total IgE levels 1264 kU/L (ULN 81.0), *Aspergillus fumigatus* specific IgE 31.8kU/L (ULN < 0.35) and eosinophilia 0.9 × 109 /L (ULN 0.4 × 109 /L), Sputum culture grew *Aspergillus fumigatus* showed septate hyaline fungal hyphae with dichotomous branching as shown in figure 3.

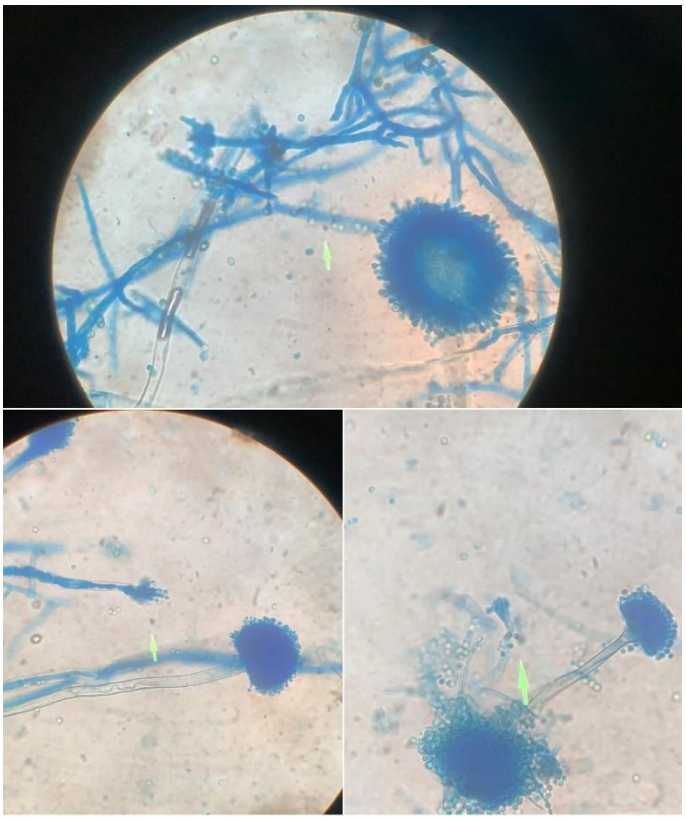

Figure 3 showing sputum culture by LPCB mount from growth showing septate hyaline hyphae with dichotomous branching

Based on clinical, haematological, radiological evidences ABPA and treatment started with oral Prednisolone 30 mg followed by 20mg for 6 weeks and tapered to 5mg every week and Itraconazole 200 mg bd, tablet azithromycin 250mg thrice weekly for 2 months, foracort inhaler 6 mcg twice a day, hypertonic saline nebulization and antihistaminics. Patient improved symptomatically, wheeze resolved rapidly with improvement in lung functionPEF 650 L/min and clearance of CXR changes. Total IgE decreased substantially to 333kU/L after 8 weeks.

3. DISCUSSION

After inhaling *Aspergillus fumigatus* spores, saprophytic development in the hyphal form occurs, resulting in an immune response that leads to ABPA. It's unclear what survival mechanisms *Aspergillus fumigatus* has, or what anomalies in bronchial mucus it has, that allow it to thrive rather than as clearing seen in all other asthma patients who don't acquire ABPA. Many allergens have enzymatic activity, including a ribonuclease which iscytotoxic by inhibiting synthesis of protein, a fibrinogen-binding protein that favours cellular injury, a metalloprotease and manganese superoxide dismutase etc. (Upadhyay et al., 2014; Greenberger, 2002). Many of these allergens bind to IgE or IgG antibodies, causing T and B cells to become activated. The mechanisms in bronchial wall damage, bronchiectasis, bronchiolitis obliterans (which occurs distally in ABPA), and promoting *A fumigatus* proliferation are not well known. Mast cells, epithelial cells, lymphocytes, macrophages, and eosinophils interact with antigenic glycoproteins and enzymes (perhaps collagenase) which is favoured by *Aspergillus fumigatus* in bronchial mucus (Walicka-Serzysko & Sands, 2015; Agarwal et al., 2020).

In susceptible patients, defective clearance of aspergillus conidia led to germination into hyphae which expose fungal proteins which are recognized by phagocytes through their receptors and exocytosis of these proteins causes relase of cytokines activates T helper cell immune response which eliminates fungi by phagocytosis. On other hand immune response occur withrelease of interleukins generates inflammatory reaction by mast cell degranulation causes characteristic immunological and pathological findings of ABPA. Persistence of inflammation leads to bronchiectasis (Greenberger, 2002; Agarwal et al., 2020; Knutsen et al., 2012; Kramer et al., 1989).

Antibody formation, cytokine synthesis, cellular proliferation (*A fumigatus* can act as a growth factor for eosinophils in vitro), and effector molecules all combine to create a complex set of immunologically driven events. Interleukins may be secreted by these cells, promoting IgE and IgG1 synthesis, eosinophil growth and survival, and mast cell proliferation (Greenberger, 2002; Agarwal et al., 2020; Pool et al., 2013; Allmers et al., 2000). Although oral steroids and Itraconazole relieve symptoms and reduce disease

progression in many patients, bronchiectatic changes are not expected to be reversible once they have developed. By this case report we could emphatically state thatearly diagnosis and management are associated to complete clinical remission of symptoms and partial reversal of bronchiectatic changes. Despite the fact that Aspergillus-specific serological tests have significantly decreased and are continuing to decline, they remain elevated. Our patient remained symptom-free for over a year following therapy termination, indicating that complete cure with normalisation of serology and prolonged bronchiectasis resolution is attainable.

4. CONCLUSION

Regardless of the level of control asthmatics or COPD patients in specialised clinics should have a high index of suspicion for ABPA. The pathophysiology of this condition is centred on host susceptibility, and host immunologic responses are the key determinants of clinical, biological, pathological, and radiological characteristics. To prevent the progression, it must be recognised early and managed effectively.

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Author contribution

VVSSS- Development of Manuscript

SA- Diagnosed the case and initiated the idea of case report and contributed in development of Manuscript

AA- Relevant data imported

SS and SK- Reviewed and edited the Manuscript

CVSA- Relevant data collection

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Conflict of interests

The authors declare that there are no conflicts of interests.

Data and materials availability

All data associated with this study are present in the paper.

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