



## ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS (ABPA) – NEVER UNDERDIAGNOSE

### Respiratory Medicine

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### ABSTRACT

**Introduction:** Allergic bronchopulmonary aspergillosis (ABPA) is an idiopathic inflammatory disease of the lung, characterized by an allergic inflammatory response to colonization of the airways by *Aspergillus fumigatus* or other fungi. The lack of distinction between ABPA, asthma, mold-sensitive asthma and TB, delays the diagnosis of patients with long-standing disease; however, it is clear that ABPA is a relatively common entity. Estimates are that true ABPA complicates approximately 7% to 14% of cases of chronic steroid-dependent asthma. **Case History:** Here is a 60yr old male known hypertensive, T2DM, and COPD, reformed smoker, farmer by occupation came with complaints of breathlessness, cough with expectoration, and weight loss since 2 yrs. On auscultation - bilateral coarse crepts present. Serum IgE was > 1000 IU/ml. HRCT thorax showed bilateral hyperinflated lung with consolidation, air trapping and bronchiectasis with peri bronchiolar thickening – suggestive of infective bronchiectasis. S. IgE specific to aspergillosis came positive. Patient was diagnosed with ABPA and was started on steroids and antifungal. On second month follow up patient symptoms were reduced and was clinically better. **Results:** Though ABPA is commonly seen the prevalence is unknown due to variability in diagnostic criteria used in various studies, often misdiagnosing it with asthma, TB and NTM. ABPA commonly presents with refractory asthma. So the diagnosis of ABPA remains challenge and requires strong presumption. If recognised early and managed aggressively, ABPA is treatable and may remit indefinitely; progressive lung damage can be avoided. **Conclusion:** We present a case of an old man with breathlessness, cough with expectoration, weight loss since 2 yrs, diagnosed with ABPA and managed with steroids and antifungal. Pt significantly improved with each followup with steroids and antifungal treatment which is a definitive form of management.

### KEYWORDS

ABPA- Allergic bronchopulmonary aspergillosis, bronchiectasis, *Aspergillus fumigatus*, steroids, antifungals

### INTRODUCTION

ABPA is an idiopathic lung inflammatory condition where the immune system reacts allergically to *Aspergillus fumigatus* or similar fungi colonizing the airways. Its exact prevalence remains uncertain due to varied diagnostic criteria across studies, overlap with mold-sensitive asthma, and delays in diagnosing longstanding cases. Despite this variability, ABPA is commonly encountered. The disease spectrum ranges widely. Patients can exhibit no symptoms, have mild-to-moderate asthma, or suffer from severe forms that may necessitate lung transplantation. Early recognition and aggressive management of ABPA are crucial as it can be effectively treated and may enter long-term remission, thereby preventing progressive lung damage [1].

### CASE REPORT:

A 60-year-old elderly male, belonging to the low middle class and working as an arcanut farmer, presented with a medical history of hypertension, type 2 diabetes mellitus, and chronic obstructive pulmonary disease (COPD). He is an reformed smoker. He complained of progressive breathlessness (graded MMRC 1 to 3), productive cough exceeding 100ml/day, significant weight loss (12kg) over the past two years, with decreased appetite, and disturbed sleep.

Upon clinical examination, the patient was conscious with a BMI of 14.7 kg/m<sup>2</sup>, blood pressure of 140/90 mmHg, pulse rate of 98 bpm, oxygen saturation of 90% on room air, respiratory rate of 18 breaths per minute, and unraised jugular venous pressure. He exhibited grade 3 clubbing, poor oral hygiene with dental caries, and congested post-pharynx. His chest appeared barrel-shaped with reduced bilateral chest movements, supraclavicular hollowing, and use of accessory muscles. Palpation revealed reduced chest expansion (<1cm bilaterally), increased tactile vocal fremitus, and vocal resonance in all lung fields. Percussion indicated hyperresonance across all lung fields. Auscultation revealed bilateral coarse crepitations with normal vesicular breath sounds and was otherwise unremarkable.

The clinical diagnosis was acute infective exacerbation of COPD with bronchiectasis. Sputum acid-fast bacilli (AFB) and Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) were negative, while sputum culture showed growth of normal oropharyngeal flora. Sputum potassium hydroxide (KOH) mount did not reveal fungal elements. Serum immunoglobulin E (IgE) level was elevated (>1000 IU/l). Chest X-ray showed bilateral hyperinflated lungs with increased bronchial markings. High-resolution computed tomography (HRCT) of the chest revealed bilateral hyperinflated lungs with areas of consolidation, air trapping, bronchiectasis, and peribronchial/peribronchiolar thickening

suggestive of infective bronchiectasis. Echocardiography (2D echo) showed no regional wall motion abnormalities at rest, good left ventricular function with mild tricuspid regurgitation and mild pulmonary arterial hypertension (PAH), and an ejection fraction (EF) of 62%. Pulmonary function tests (PFT) indicated severe obstructive and restrictive patterns with no significant bronchodilator response (BDR).

The patient was initiated on low-dose steroids, bronchodilators, and treatment for COPD exacerbation. Despite initial therapy, his condition deteriorated. IgE specific to aspergillosis was subsequently tested and returned positive, leading to a diagnosis of allergic bronchopulmonary aspergillosis (ABPA) complicating an acute exacerbation with secondary infection. Treatment was intensified with intravenous antibiotics, bronchodilators, high-dose steroids (0.5 to 1 mg/kg), and antifungal medication (itraconazole 200 mg twice daily).

During follow-up at two months, the patient reported reduced symptoms and overall clinical improvement.



HRCT Thorax.



Chest X-ray-PA



HRCT Thorax

**DISCUSSION**

Allergic bronchopulmonary aspergillosis (ABPA), first described in 1952 by Hinson and colleagues, commonly occurs in conjunction with chronic lower airway diseases like asthma and cystic fibrosis, characterized by thick mucus and impaired mucus clearance, allowing *Aspergillus fumigatus* spores to persist in the airways. Although less frequent, ABPA can also affect individuals without a history of asthma[2]. Approximately 2.5% of adults with asthma, totaling about 4.8 million people worldwide, are estimated to have ABPA. It can develop regardless of age or gender[3]. Typical symptoms include wheezing upon auscultation and occasionally sputum with brown mucus plugs, though not always present[4].

Diagnosis primarily relies on immunological tests, with elevated *Aspergillus fumigatus*-specific IgE levels (>0.35 kUAl) being the most sensitive indicator. The *Aspergillus* skin test, though fairly sensitive (88-94%), may miss some cases. Serum total IgE measurement is useful for diagnosis and follow-up, but lacks specificity as a screening tool [5]. High-resolution chest CT is the preferred imaging method, revealing characteristic central bronchiectasis, which can extend peripherally in a significant number of cases. MRI's role in ABPA diagnosis is still under evaluation and is not currently recommended routinely [6]. Several diagnostic criteria exist, with the Rosenberg-Patterson and ISHAM-ABPA criteria being widely recognized in asthma-related ABPA diagnosis. The Japan ABPM research program has recently proposed ten new diagnostic criteria for ABPM/ABPA specifically for patients who do not have cystic fibrosis[7].

**ISHAM Criteria:**

Predisposing factors	Asthma, Cystic fibrosis
Obligatory Criteria(both should be present)	1)Type I <i>Aspergillus</i> skin test positive (immediate cutaneous hypersensitivity to <i>Aspergillus</i> antigen) or elevated IgE levels against <i>Aspergillus fumigatus</i> 2) Elevated total IgE levels (> 1000 IU/mL)
Other criteria (at least two of three)	1)Presence of precipitating or IgG antibodies against <i>A. fumigatus</i> in serum 2) Radiographic pulmonary opacities consistent with ABPA 3)Total eosinophil count > 500 cells/IL in steroid naive patients.

Treatment for ABPA focuses on managing asthma and cystic fibrosis symptoms, reducing lung inflammation, preventing exacerbations, and slowing disease progression. Prompt and thorough treatment is crucial[9]. Many patients experience significant recovery with treatment, including reduced serum IgE levels, resolution of lung infiltrates, and symptom improvement. However, relapses can occur [7]. Corticosteroids are essential in ABPA treatment, targeting the inflammatory response triggered by *Aspergillus fumigatus*. The optimal oral steroid dose remains uncertain, with lower doses linked to higher recurrence rates [7]. Inhaled corticosteroids are key in managing persistent asthma symptoms but do not effectively treat ABPA alone [7]. Antifungal therapies complement treatment by reducing fungal colonization and inflammation, potentially decreasing the need for prolonged systemic steroids [7]. The long-term prognosis varies, with untreated patients at risk of irreversible lung damage and respiratory failure.

**Conflicts Of Interest:**

The authors declare that they have no conflict of interest.

**Acknowledgements:**

The authors declare that they have no competing interest.

**CONCLUSION**

ABPA commonly presents with refractory asthma. Radiological Central bronchiectasis with elevated S.IgE (>1000IU/ml) and IgE specific for aspergillosis confirmed ABPA diagnosis in our case. Though ABPA is commonly seen, the prevalence is unknown due to variability in diagnostic criteria used in various studies, often misdiagnosing it. If recognised early, ABPA is treatable, progressive lung damage can be avoided [1].

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