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Case report

Allergic bronchopulmonary candidiasis

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Introduction

Allergic Bronchopulmonary Aspergillosis (ABPA) is an allergic disorder caused by fungi of *Aspergillus* species in patients of bronchial asthma. Over the years other fungal species have also been implicated in causing the disease. Hence the term ABPA has been replaced by the term Allergic Bronchopulmonary Mycosis (ABPM), although *Aspergillus* continues to be the commonest. *Candida albicans* rarely causes ABPM and a few case reports suggest dual aetiology (*Aspergillus* and *Candida albicans*). We report one such case of ABPM.

Case report

45-year-old lady, a homemaker had presented to the emergency with complaints of cough with expectoration of 30 days duration and chest pain of 15 days duration. The cough had an insidious onset and had been gradually increasing. She

noticed increased cough on waking up in the morning and it aggravated on lying down. It had been associated with scanty thick viscid – with an almost pellet like consistency – brownish–blackish sputum. She denied history of haemoptysis. She had also developed wheeze, which was intermittent and was more noticeable in the evenings. She also complained of sharp pricking pain below the right breast that was aggravated by deep inspiration and coughing. She had no fever, breathlessness, or any orthopnoea.

She had been diagnosed as a case of bronchial asthma since the age of 10 years. She was admitted repeatedly in the past for acute exacerbations. The last severe attack requiring ICU admission occurred 7 years back after exposure to household dust. The patient was on intermittent inhaled long acting β_2 agonists. Three years prior to presentation the patient was diagnosed as a case of pulmonary tuberculosis based on a history of two episodes of haemoptysis, chronic cough and the presence of bilateral lower zone non-homogenous opacities and some nodular opacities in the

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Fig. 1 – Chest radiograph on presentation: depicts a non-homogenous opacity in the right lower zone with an air bronchogram (arrow).

right upper zone on chest radiograph. The patient received 6 months of ATT. The patient was diagnosed with type 2 diabetes mellitus 2 years back and her blood sugar was well controlled with metformin and dietary restrictions.

Examination revealed a well-built overweight lady, who was tachypnoeic with a respiratory rate of 24 per minute. She was afebrile with a pulse of 88 per minute and a blood pressure of 140/90 mm of Hg. Examination of the chest revealed dull percussion note and bronchial breathing over the right infrascapular region and a polyphonic wheeze all over the chest.

Investigations revealed Leukocytosis (TLC 13,600/mm³) with eosinophilia (Eosinophil 12%) and an absolute eosinophil

count of 1480/mm³. The chest radiograph revealed a non-homogenous opacity with air bronchogram in the right lower zone (Fig. 1). The patient was started on tablet Amoxicillin + Clavulanate (500 + 150 mg) and nebulization with salbutamol and budesonide. A CECT done revealed bilateral central bronchiectasis with bronchoceles in partially collapsed right lower lobe (Fig. 2).

She was further investigated with spirometry which revealed mild obstruction with insignificant reversibility. A total serum IgE was obtained which was raised (3200 IU/L). Specific IgE against *Aspergillus fumigatus* revealed high titres (12.5 kUA/L). Sputum was sent for fungal cultures. Skin prick test for *Aspergillus* (or other fungi) was not undertaken due to non-availability at our centre.

Patient was diagnosed as a case of Allergic Bronchopulmonary Aspergillosis and was started on prednisolone. We planned to treat her with intensive regimen of prednisolone, 0.75 mg/kg for 6 week, followed by 0.5 mg/kg for 6 week, and then to taper the dose by 5 mg every 6 week with the aim to treat for a total duration of 6–12 months.

The patient reported decreased cough within a week of treatment and the pain also subsided. The sputum cultures for fungus revealed multiple colonies of *Candida albicans*. The diagnosis was revised to Allergic Bronchopulmonary Candidiasis (ABPC) with past sensitization to *Aspergillus*. Tab Itraconazole 200 mg once a day was added to the treatment with the aim to continue for 16 weeks.

The patient was followed up with 6 weekly total serum IgE levels. The levels dropped to 1573 IU/L at 6 weeks. The chest radiograph showed resolution of the opacities (Fig. 3).

Discussion

The prevalence of ABPM is estimated to be around 1%¹ in the general population and its prevalence in patients attending

Fig. 2 – CECT chest reveals consolidation with collapse right lower lobe, central bronchiectasis and intra bronchial plugging.

Fig. 3 – Chest radiograph after 6 weeks of therapy: shows near complete resolution of opacity with residual cystic and nodular opacities.

asthma clinics is around 7%. The prevalence increases to around 39% in patients with bronchial asthma admitted to ICU.² The non-specific nature of symptoms coupled with a low index of suspicion usually leads to the patient being symptomatic with asthma for about 10–12 years before the diagnosis of ABPM is considered.³

Though not an uncommon disease, our case had certain peculiarities. Although a history of poorly controlled asthma with frequent exacerbations should have merited consideration of the diagnosis of ABPA but the patient was exhibited ATT based on chronic respiratory symptoms, history of haemoptysis and radiological findings. About 45% of the cases are diagnosed as pulmonary tuberculosis and treated with anti-tubercular therapy as the radiological picture may mimic pulmonary tuberculosis.⁴ The long lead-in period to diagnosis again highlights low index of suspicion for this disorder.

Though *A. fumigatus* is the most common cause of ABPM, other fungi such as *Aspergillus niger*, *Alternaria*, *Cladosporium*, *Rhizopus* (*Mucor*), *Penicillium*, *Candida*, *Fusarium*, *Schizophyllum commune*, *Cryptococcus neoformans*, *Trisporonbeigelii* have all been reported to cause ABPM. Diagnosis of ABPM is based on Rosenberg–Patterson criteria⁵ which include asthma, fleeting pulmonary opacities, positive skin test, eosinophilia, precipitating antibodies (IgG) in serum, elevated serum IgE (>1000 IU/mL), central bronchiectasis and raised specific IgE. We were unable to carry out skin sensitivity testing due to administrative difficulties; however, our case satisfied five of the eight criteria which are highly suggestive

of the diagnosis of ABPM. Repeated sputum cultures in our patient grew colonies of *Candida albicans*. Though *Candida* has been reported in the literature as a cause of ABPM the exact prevalence remains unknown.⁶ Our case had ABPM due to both *Aspergillus* and *Candida albicans*. Most likely the patient got sensitized to *Aspergillus* in the past and the present episode was due to the airway colonization by *Candida albicans*. A study in Saudi Arabia reported a prevalence of ABPM due to both *Aspergillus* and *Candida* at 3%⁷ among patients of bronchial asthma.

Our case highlights the need to screen all patients of bronchial asthma for ABPM. We suggest that all patients of bronchial asthma should undergo skin prick test (SPT). Those with a positive result should have their total serum IgE levels evaluated. A level greater than 1000 IU/mL warrants further evaluation for ABPA. Those with total serum IgE levels between 500 and 1000 IU/mL should be followed up with IgE levels every 6 weeks if they test positive for specific IgE for *Aspergillus*. Those with total serum IgE levels less than 500 IU/mL require yearly estimation of total serum IgE. All other patients of bronchial asthma should be followed up with IgE levels every 2 years.

Conflicts of interest

All authors have none to declare.

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