CASE REPORT
ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS WITHOUT BRONCHIAL ASTHMA: A RARE OCCURRENCE

Urvinder Pal Singh, Pooja Aneja, Aditi

ABSTRACT
Allergic bronchopulmonary aspergillosis (ABPA) is a complex clinical entity resulting from an allergic immune response to Aspergillus species and most often occurs in patients with asthma and cystic fibrosis (CF). ABPA is rarely observed in the absence of asthma which is in fact the principal criterion for its diagnosis. We report a case of a 31-yr-old young man who had all other criteria except Bronchial asthma and was diagnosed as a case of ABPA.

Key Words: ABPA, Allergic bronchopulmonary Aspergillosis, Allergic lung disease.

INTRODUCTION:
Allergic Bronchopulmonary Aspergillosis (ABPA) is an allergic lung disease caused by hypersensitivity reactions to antigens of Aspergillus species, which is a genus of fungi with worldwide distribution. Diagnostic criteria for ABPA include the presence of bronchial asthma, immediate skin test reactivity to Aspergillus fumigatus, elevated total and A.fumigatus specific IgE levels, pulmonary infiltrates (transient or fixed), central bronchiectasis, peripheral blood eosinophilia and presence of precipitins against Aspergillus antigen. Bronchial Asthma is considered to be essential for diagnosis and is thought to play an important role in the development of the disease. Majority of the patients of ABPA have asthma or cystic fibrosis, however there are several case reports of ABPA without coexisting bronchial asthma or cystic fibrosis.

CASE REPORT:
A 31-yr-old man visited our hospital for evaluation of chronic cough which had persisted for eight months with minimal expectoration coupled with an intermittent low-grade fever and myalgia. He had been taking treatment from private doctors and now had been advised to take anti tubercular treatment. He came to us for confirmation of his diagnosis of pulmonary tuberculosis. The patient reported no history of wheezing or dyspnea nor did he have any personal or family history of atopy. The patient did not smoke and had no previous history of diseases like bronchial asthma or pulmonary tuberculosis. His chest was clear on auscultation. Other physical examination showed no abnormal findings.

On investigations the patient had a total leukocyte count of 8,300 cells/mm³ with 47% neutrophils, 21% lymphocytes and 32% eosinophils. His sputum & sputum collected by induction method both were negative for AFB. Intradermal tests with Aspergillus fumigatus spp elicited strong type I hypersensitivity reaction. His total serum IgE level estimated using fully automated chemiluminiscence system was 11583 IU/mL (N~ 100 IU/mL). Specific IgE against A.fumigatus was >100 KU/L (N~0.24KU/L).


The patient's chest radiograph revealed a heterogenous opacity in the left upper lobe with bronchiectatic changes in both lung fields (Fig I). A chest computed tomography scan displayed consolidation in the left upper lobe, central bronchiectasis and mucous impacted dilated bronchi in bilateral upper lobes (Fig II). Air space nodules with few of them forming tree in bud pattern were also seen in posterior segment of right upper lobe and anterior basal segment of left upper lobe.

A diagnosis of ABPA was thus made as the patient fulfilled majority of the criteria given by Rosenberg et al\(^1\). He was treated with prednisolone 0.75 mg/kg for 6 weeks, 0.5 mg/kg for next 6 weeks, and then tapered by 5 mg every 6 week to continue for a total duration of 9 months\(^9\). The patient showed marked improvement in his condition and there was marked clearing of his X-ray after 2 months of treatment (Fig III). His total serum IgE after two months of treatment decreased significantly to 4784 IU/ml. The patient is still visiting this hospital for follow up every month. No symptoms or signs of asthma have appeared till now.

**DISCUSSION:**
ABPA is a hypersensitivity disorder induced by Aspergillus species colonizing the lung cavity predominantly in patients with asthma\(^10\). It is the most frequently recognized manifestation of allergic aspergillosis occurring worldwide. ABPA was first reported in the United Kingdom in 1952\(^11\) and since then has been reported worldwide. From India the first 3 cases were reported in 1971\(^12\). Although Rosenberg-Patterson criteria\(^1,13\) are most often used for the diagnosis, there is still no consensus on the number of criteria needed to diagnose ABPA and patients in different stages may not fulfill these criteria\(^14\).

Despite the fact that ABPA is usually seen in association with bronchial asthma, ABPA has also be known to be an important complication of pulmonary disease associated with cystic fibrosis. ABPA has been variously reported in 1 to 15% of CF patients. The diagnosis of ABPA in CF is more complicated and disagreement exists in the literature regarding the diagnostic criteria. The difficulty lies in the fact that the usual criteria for ABPA and the common signs and symptoms of CF overlap\(^15\).

Bronchial asthma has classically been considered an essential diagnostic criterion for ABPA and has also been believed to play a crucial role in its development\(^1\). It is considered that role of bronchial asthma in the development of ABPA is to make the expectoration of Aspergillus difficult and colonization easy by the viscous sputum\(^5\). ABPA is rarely thought of in the absence of asthma as this is the first criterion for diagnosis. However several cases without bronchial asthma have been reported\(^4-8\). Glancy et al\(^4\) reported that 11 out of 42 patients with ABPA had no evidence of bronchial asthma. A
systematic MEDLINE search performed by Agarwal et al for the occurrence of ABPA without bronchial asthma, were able to record 36 cases reported across the globe. Because of the absence of bronchial asthma, these cases are often mistaken initially for other pulmonary disorders like bronchogenic carcinoma or pulmonary tuberculosis. Furthermore, the remarkable radiologic similarity to pulmonary tuberculosis has important clinical implications in high tuberculosis prevalent areas.

Absence of bronchial asthma in our case was a reason for the delay in diagnosis of ABPA. Peripheral blood eosinophilia prompted further investigations which clinched the diagnosis of ABPA without bronchial asthma. The patient showed marked improvement with steroids and is symptom free for past one year. It has been stated that asthma may not be the initial criteria but may appear in the later stages of the disease. Absence of asthma can be a good prognostic factor showing the early stages of the disease.

To conclude, we suggest that although ABPA most commonly occurs in patients with pre-existing bronchial asthma, a high index of suspicion should be maintained in the absence of asthma. ABPA without clinical asthma can and does pose diagnostic difficulties as in our case. Thus ABPA should be kept as a diagnostic possibility in patients with radiographic abnormalities and peripheral eosinophilia but no history or symptoms related to bronchial asthma.

REFERENCES:
Legends

Fig I: Xray chest showing heterogenous opacity in the left upper zone and bronchiectatic changes in both lung fields.

Fig II: CT chest showing consolidation in the left upper lobe, central bronchiectasis and mucous impacted dilated bronchi in bilateral upper lobes.

Fig III: Xray chest after 8 weeks showing marked clearing of the lesions. Allergic bronchopulmonary aspergillosis (ABPA) is a complicating factor of cystic fibrosis which can result in a devastating combination as lung disease progresses. The overlap between the signs and symptoms of the two conditions makes diagnosis problematic.