

CASE REPORT

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A rare case of allergic bronchopulmonary aspergillosis in a patient with chronic obstructive pulmonary disease

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Abstract

Allergic bronchopulmonary aspergillosis (ABPA) is known to complicate asthma and cystic fibrosis, but an association of ABPA with chronic obstructive pulmonary disease (COPD) has also been found lately. The mechanism through which ABPA operates is still an enigma. With the discovery of ABPA in COPD, interest in the theory that asthma and COPD could share a common etiology has been rekindled. Herein, we present the case of a 60-year-old male, diagnosed case of COPD, who reported exacerbations during the monsoons, which mandated frequent courses of systemic steroids. He was diagnosed to have co-existing ABPA based on positive skin tests to *Aspergillus niger*, *A. flavus*, *A. fumigatus*, and *A. tamarii* and raised serum total IgE and specific IgE against *Aspergillus* spp. His CT scan showed centrilobular and panacinar emphysema along with central bronchiectasis. The spirometry showed a significant response to bronchodilators. The patient fulfilled all the minimal essential requirements for the diagnosis of ABPA, except asthma. The patient responded well to systemic steroids. This report makes an important observation that species of *Aspergillus* other than *A. fumigatus* could be instrumental in causing ABPA in patients with COPD, unlike that seen in asthma. The possible etiologic mechanisms underlying the development of ABPA in COPD and also the evolution of ABPA from its serological to bronchiectasis variant are discussed in this report.

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Full Text

Introduction

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity disorder to airborne allergens of *Aspergillus* spp., which has classically been associated with asthma, [1],[2] cystic fibrosis, [3] and cavitary lung disease. [4] Data from the

western hemisphere estimate ABPA to be prevalent in 1-2% cases of bronchial asthma, [5] but reports from India show a much higher prevalence, ranging from 7.6% to 27.2%. [6],[7],[8],[9] Although the criteria to diagnose ABPA require that the patient have bronchial asthma, [10] there exist cases of ABPA where a background of asthma was absent. [1],[11],[12],[13],[14],[15],[16],[17],[18],[19] Lately, cases of ABPA have been reported in patients with chronic obstructive pulmonary disease (COPD). [20],[21],[22] Where on one hand, COPD and asthma are two distinct disorders, on the other hand, they share some similarities as well. [23] Thus, it seems likely that these two disorders represent two ends of a spectrum of inflammatory airways disease and should this be true, the occurrence of ABPA in COPD might be explained. Through this document, we present the case of a 60-year-old male, diagnosed case of COPD, who was discovered to have ABPA and discuss the possible etiologic mechanisms behind this association.

Case Report

A 60-year-old fisherman from Delhi, India was referred to the Vallabhbhai Patel Chest Institute in October 2011 with the chief complaints of progressive exertional dyspnea, productive cough since 2 years. The patient had been smoking up to 40 'beedis' (flakes of tobacco (*Nicotiana tabacum*) rolled up in a tendu (*Diospyros melanoxylon*) leaf) since the age of 15 years. The history of the patient was punctuated by exacerbations during June to October when his symptoms were poorly controlled with inhaler therapy alone and would not be controlled unless systemic steroids and antibiotics were administered for the increased dyspnea and purulent expectoration. He denied having been administered inhaled long acting β agonists and corticosteroid combinations on a regular basis but admitted to the frequent use of inhaled salbutamol and systemic steroids for his persistent symptoms, which he was using upon presentation to our institute as well. The patient did not give any history of remissions of symptoms, or symptoms suggestive of any atopic disorder. The family history was non-contributory. The physical examination of his chest revealed bilateral vesicular breath sounds with prolonged expiration associated with bilateral infrascapular coarse crackles and polyphonic wheeze. Pallor, cyanosis, and clubbing were absent. Based on the history and clinical findings, a clinical diagnosis of late-onset asthma was formulated with COPD as a close differential diagnosis. However, the latter was later confirmed by his investigations also.

The hemogram of the patient showed a hemoglobin level of 13.9 g/dL, total leukocyte count of 9,500 cells/ μ L, which comprised of 76% polymorphs, 12% lymphocytes, 8% monocytes, and 4% eosinophils. The absolute eosinophil count of the patient was 350 cells/ μ L, and serum biochemical parameters were within normal limits. Sputum was negative for the presence of pyogenic and acid-fast organisms. The spirometry of the patient showed pre-bronchodilator FEV₁/FVC, FVC, and FEV₁ of 63%, 1.67 L (62% of predicted), and 1.06 L (48% of predicted) while the post-bronchodilator-values were 63%, 1.88 L (69% of predicted), and 1.18 L (54% of predicted), respectively, which suggested severe airflow limitation with significant bronchodilator response (increase in FVC by 12% and 210 mL).

A radiograph of the chest showed a low-lying diaphragm and increased vascular markings bilaterally [Figure 1]. A contrast-enhanced, high-resolution computed tomogram (HRCT) of the thorax was obtained, which showed predominantly upper-lobe paraseptal and centrilobular emphysema and signs of 'early' central bronchiectasis [Figure 2]. Corroborating the history of exacerbations during the monsoons (the period from July to September being the rainy season in Northern India) with the central bronchiectasis, the patient was investigated for mold hypersensitivity.[Figure 1]{Figure 2}

A skin prick test was performed, which showed a type I reaction to *A. niger*, *A. flavus*, *A. fumigatus*, and *A. tamarii*. Also, a type III reaction was elicited against *A. flavus*. The total serum immunoglobulin E (IgE) of the patient was 7744 IU/mL. The specific IgE against *A. terreus*, *A. flavus*, and *A. niger* were raised 1.9, 1.4, and 1.3 times more than the control, respectively. Serum precipitins (specific IgG) against *Aspergillus* spp. were negative, and no fungi could be isolated from the sputum. [Table 1] below summarizes the findings in our case and how the same differ from the diagnostic criteria proposed by Rosenberg. [10]{Table 1}

A diagnosis of ABPA with COPD was made as our case met all parameters of the minimal essential criteria, [24] except bronchial asthma. The patient was initiated on tapering doses of prednisolone starting at a dose of 1 mg/kg along with inhaled steroids with bronchodilators and oral methylxanthines. The patient reported symptomatic relief, but repeat measurement of his serum total IgE levels was not available as the patient dropped-out of follow-up a month and a half after initiation of treatment.

Discussion

The occurrence of ABPA in asthmatics and patients of cystic fibrosis is well-known, but the affection of COPD patient with ABPA is relatively new-found, with cases having been reported over the past 5 years from India only. [20],[21],[22] The exact pathophysiological mechanism underlying ABPA is an enigma, even 60 years after its description. [5] The fact that not all asthmatics go on to develop ABPA suggests that this disease could be the outcome of interplay between genetic susceptibility of the host and his environment. Inflammation of the airways leads to mucus hypersecretion, which creates an environment conducive to trapping and germination of fungal spores so ubiquitously present in the environment. [25] While under normal circumstances, antigen processing would activate both Th1 and Th2 pathways; [26] in ABPA, the latter tends to predominate leading to B-cell activation and eosinophilic inflammation mediated by IL-4, IL-5, and IL-13. [27] The pathogenicity of the fungus is enhanced by the proteolytic nature of the fungal antigens that allows greater access of the germinating spore to the epithelial and subepithelial layers of the bronchial wall where these enzymes produce cytotoxic effects. [28],[29] Further, homology of fungal enzymes with human enzymes probably results in the host immune system acting against self. [29]

Asthma and COPD, in spite of sharing common features such as airway inflammation, airflow obstruction, and mucus hypersecretion differ significantly from each other. Where the inflammation in asthma is eosinophilic and amenable to treatment through inhaled steroids, that in COPD is predominantly neutrophilic making the latter relatively steroid-resistant. [23] However, this distinction fades on comparing severe asthma with COPD, both of which have neutrophilic inflammation. [30] Similarly, some studies show that smokers share certain features with asthmatics such as airway hyper-reactivity, [31],[32] hyper-gammaglobulinaemia E, [33] eosinophilia, [34] and eosinophilic inflammation [35] responsive to systemic glucocorticoids. [36] Thus, a common origin of the two disorders appears plausible, and the same was promulgated for the first time through the Dutch-hypothesis. [37] ABPA is frequently regarded as a severe form of asthma, but the pulmonary inflammation seen therein is predominantly eosinophilic. [38],[39] However, bronchial neutrophilia is also observed in ABPA to an extent greater than in asthma alone and also correlates with the degree of tissue damage. [40] Whether the same is the case with ABPA in COPD is not known yet; should that be the case, it may contribute in explaining the development of ABPA in COPD through pathways akin to those in asthma.

In our case, the history of smoking, exertional type of progressive dyspnea and productive cough of 2 years, absence of a completely symptom-free interval, constant need of medication between exacerbations, and no history of allergic diathesis favors a diagnosis other than asthma. The radiology of the patient showed hyperinflation of the lung, which can also be seen in asthma, [41] but the demonstration of paraseptal and centrilobular emphysema on the HRCT clinched COPD as the diagnosis. However, our patient demonstrated a bronchodilator response, which favors asthma as the diagnosis but does not preclude the diagnosis of COPD either. [42],[43],[44] On the other hand, with regards to the diagnosis of ABPA, the Rosenberg criteria [10] require that 6 out of 8 major criteria be present, but in the case presented above, only 4 namely, type I cutaneous hypersensitivity, raised total serum IgE, and specific IgE against *Aspergillus* spp. were present. The patient also had lesions that appeared as early central bronchiectasis. However, the minimal essential requirements for diagnosing ABPA [24] were met, except for the presence of asthma. No remarks could be made on the fleeting nature of pulmonary opacities, as previous radiographs of the patient were not available. Negativity of serum precipitins can be attributed to more than one factor such as the patient being in a phase of remission, [45] having received systemic steroids in the recent past, [46] or probably as an outcome of the inherent insensitivity of the Ochterlony's immunodiffusion test. [47] Peripheral blood eosinophilia, another major diagnostic criterion, was not observed in our patient probably because of the past intake of steroids. It has been shown that eosinophilia has a limited role in the diagnosis of ABPA. [48] Moreover, in the case presented above, though the HRCT showed lesions suggestive of central bronchiectasis, the same were subtle.

Our case shares many similarities with the previously reported cases [20],[21],[22] such as presence of COPD in the place of asthma, type I and type III cutaneous hypersensitivity against *A. fumigatus* and *A. flavus*, and positive IgE antibodies specific to *Aspergillus* spp. Total IgE in our case was also raised as in the previously reported cases but to a much higher level. Nevertheless, our case also differed from these two cases in that our patient demonstrated significant bronchodilation on spirometry and did not demonstrate the presence of serum precipitins. In the case presented above, signs suggestive of 'early' central bronchiectasis were observed, but were not very definitive of the same. Out of the 4 cases reported previously, [20],[21],[22] central bronchiectasis was seen in only 2 cases, [20],[22] implying thereby that the serological variant of ABPA (ABPA-S) can also be seen in COPD. Since it is well-known that central bronchiectasis develops later in the course of ABPA-S, [49] it is likely that our patient started as ABPA-S and is currently undergoing this transition. A more definitive picture can be obtained by following-up this patient regularly for the development of central bronchiectasis variant of ABPA (ABPA-CB).

The above-mentioned comparisons go on to imply that mechanisms similar to those behind ABPA in asthmatics may be operative in the ABPA of COPD as well. Assuming that the agent and environment factors remain constant in patients of asthma and COPD, it is likely that host factors play an important role in the pathogenesis of ABPA. Alterations in certain genes such as major histocompatibility antigen (MHC) genes (HLA-DR2, HLA-DR5, HLA-DQ2), interleukins (IL-4RA, IL-10), surfactant protein A2 (SP-A2), toll-like receptor (TLR), and cystic fibrosis transmembrane conductance regulator

(CFTR) have been proposed as predisposing factors for the development of ABPA. [50] With *Aspergillus* sensitization having been brought to light in patients with COPD, [21] further work needs to be done to determine in COPD the: (a) prevalence of *Aspergillus* hypersensitivity, (b) prevalence of ABPA, (c) etiopathogenesis of ABPA, (d) histopathology of the lung, (e) difference in clinical presentations and laboratory results between the ABPA of asthma and that of COPD, and (f) differences in response to treatment. Moreover, though *A. fumigatus* is the most commonly involved agent in the causation of ABPA, the reactivity on skin tests and serology to antigens of other *Aspergillus* spp. as seen in all these reports suggests that these species too could be important in causing ABPA in COPD. Thus, it would be lucrative to study the role of species of *Aspergillus* other than *A. fumigatus* and also non-*Aspergillus* molds in causing allergic bronchopulmonary mycosis in association with COPD.

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Figure 1: Chest X-ray of the patient demonstrating a low-lying diaphragm and increased vascular markings



Figure 2: HRCT of the patient showing centrilobular and paraseptal emphysema and central bronchiectasis

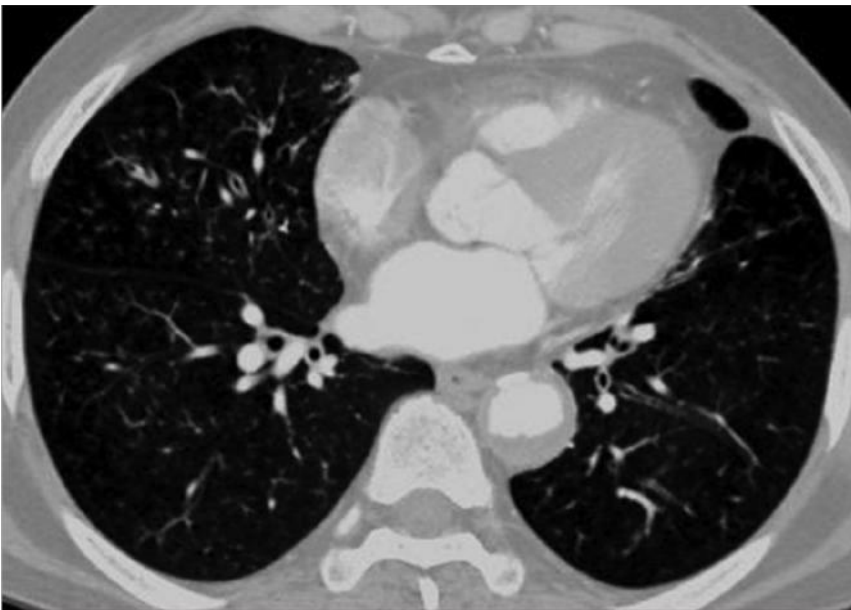


Table 1: Summary of the Rosenberg criteria for the diagnosis of ABPA in the presented case

Diagnostic criteria	Findings in the present case
Major criteria	
Bronchial asthma	-
Type I cutaneous hypersensitivity	+
Eosinophilia	-
Serum total IgE	+
Serum precipitins against <i>Aspergillus</i>	-
Specific IgE/IgG against <i>Aspergillus</i>	+
Fleeting pulmonary opacities	-*
Central bronchiectasis	+
Minor criteria	
Expectoration of sputum plugs	-
Isolation of fungus from sputum	-
Type III cutaneous hypersensitivity	+

*Previous radiographs of the patient were not available, ABPA - Allergic bronchopulmonary aspergillosis