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Allergic Bronchopulmonary Aspergillosis in Chronic Obstructive Pulmonary Disease

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Abstract

Allergic bronchopulmonary aspergillosis (ABPA) is a well-recognised entity which complicates the course of bronchial asthma and cystic fibrosis. The occurrence of ABPA in other pulmonary conditions like COPD is rare. Here we are reporting a case of 52 year old male who presented with acute exacerbation of COPD and haemoptysis due to co-existent ABPA.

Keywords: COPD, ABPA, Bronchial asthma

Introduction

Allergic bronchopulmonary aspergillosis (ABPA) is a most frequently recognised manifestation of pulmonary aspergillosis. Being an immunologically mediated disease, it usually occurs in atopic individuals and is caused by hypersensitivity reaction to aspergillus fumigatus. Patients with chronic obstructive pulmonary disease (COPD) have mucus hypersecretion and may favour occurrence of ABPA. However, ABPA is rarely reported in other lung disorders including COPD. Here we are reporting a case of COPD complicated by ABPA.

Case Report

A 52-year old ex-smoker (pack year 15) was diagnosed to have chronic obstructive pulmonary disease (COPD) 4 years back. Diagnosis of COPD was considered based on symptoms of cough and breathlessness with a history of exposure to risk factor and confirmed on spirometry (post-bronchodilator FEV1/FVC <0.7 and postbronchodilator FEV1 59% without reversibility). His symptoms were well controlled on inhaled bronchodilators since the time of diagnosis. He came to the outpatient of chest department with the complaint of worsening cough and breathlessness of 2 weeks duration. There was no history of fever, anorexia and weight loss. He was having history of repeated exacerbation in the past 3 months with response to steroids. Examination revealed respiratory rate of 24 breaths per minute. Chest auscultation revealed generalised decrease in breath sounds, prolonged expiratory phase and ronchi. Rest of the examination was unremarkable. Saturation on room air was 94%. He was initially managed with antibiotics, theophylline, inhaled steroids and bronchodilators. But he had one episode of haemoptysis of moderate amount. He was evaluated further for the aetiology of haemoptysis.

Chest radiograph was normal (Figure 1). Sputum for acid fast bacilli was also negative for three consecutive days. Computed tomography (CT) thorax was done as a work-up of haemoptysis. CT thorax showed bilateral central bronchiectasis. Haematological and biochemistry investigation were normal except absolute eosinophil counts of 2850 cells/µl. Skin testing was done to exclude the diagnosis of allergic bronchopulmonary aspergillosis (ABPA). Both skin prick tests (type 1 and type 3) were positive. Subsequently total serum IgE was also found to be elevated at 1636 IU/ml (normal 1-87 IU/ml). Aspergillus specific IgE and IgG were 19 kU/l (normal 0-0.35 kU/l) and 28.2 (normal <40 IU/ml) respectively. Detailed history did not reveal any history of allergic rhinitis, allergic conjunctivitis and urticaria. There was no significant family history suggestive of any allergic disorder.



Figure 1: CT thorax shows bilateral central bronchiectasis.

Thus a diagnosis of ABPA in a COPD patient was made and started on oral prednisolone at the dose of 0.5 mg/kg/day, inhaled bronchodilators and steroids. On follow up at 2 weeks, there was a subjective and clinical improvement.

Discussion

ABPA is an immune mediated disease caused by hypersensitive reaction to fungus aspergillus fumigatus. Current criteria require the presence of bronchial asthma and cystic fibrosis for the recognition of ABPA [1]. The Rosenberg-Patterson criteria are most often used for the diagnosis of ABPA [2,3] and presence of six out of eight major criteria provides a relatively firm basis for the diagnosis. Our patient meets five out of these eight major criteria for the diagnosis of ABPA.

The first step in the pathogenesis of ABPA is the development of aspergillus hypersensitivity, which is followed by exaggerated host response with resultant eosinophilic pulmonary inflammation and lung damage [4]. It is believed that in genetically predisposed individuals, A. fumigatus conidia are trapped in the mucus and narrowed airway of patient with asthma/Cystic fibrosis where they germinate to form hyphaev [5]. They then release soluble and particulate antigens which

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cause marked airway inflammation. The epithelial damage results in diffusion of soluble antigen and mycelial fragments into interstitium with further release of inflammatory mediators, and influx of inflammatory cells [5]. The antigens are also presented to Th2 cells which lead to total and A. fumigatus specific IgE synthesis, mast cell degranulation and promotion of a strong eosinophilic response. The inflammatory cells lead to tissue injury and characteristic pathology of ABPA [5].

Patients with COPD have mucus hypersecretion and impaired mucociliary clearance which may predispose these patients to the colonisation of aspergillus fumigatus resulting in development of ABPA. Till date only one such case was reported from Chandigarh [6]. A case control study from the same place also showed high probability of aspergillus hypersensitivity/ABPA in patients of COPD [7].

Conclusion

ABPA can also complicate the patients with COPD and besides bronchial asthma or cystic fibrosis; COPD can also be taken as major criteria for the diagnosis of ABPA.

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