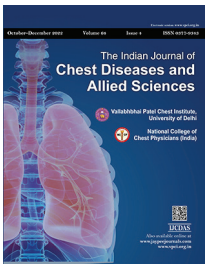
CASE REPORT

Combined Allergic Bronchopulmonary Aspergillosis and Tropical Pulmonary Eosinophilia: A Rare Co-occurrence



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Abstract

Allergic bronchopulmonary aspergillosis (ABPA) and tropical pulmonary eosinophilia (TPE) are common lung diseases presenting with peripheral blood eosinophilia. Although these have been widely reported both from India and outside, simultaneous co-occurrence of the two diseases has not been reported so far. We hereby present a case of an elderly male, a known case of asthma, who was diagnosed to have concurrent ABPA and TPE. Partial clinical response as well as the persistence of eosinophilia after ABPA treatment raised the suspicion that subsequently led to the diagnosis of TPE. The concurrent treatment of both conditions led to satisfactory clinical and serological improvement.

Keywords: Allergic bronchopulmonary aspergillosis, Asthma, Filaria, Tropical pulmonary eosinophilia.

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Abbreviations used in this Article

ABPA = Allergic bronchopulmonary aspergillosis; TPE = Tropical pulmonary eosinophilia; OPD = Outpatient department; IgE = Immunoglobulin E; IgG = Imunoglobulin G; HRCT = High-resolution computed tomography; AEC = Absolute eosinophil count; ANCA = Antineutrophil cytoplasmic antibody; DEC = Diethylcarbamazine; TID = Three times a day; CT = Computed tomography.

Introduction

Eosinophilic lung diseases constitute a diverse group of disorders characterized by eosinophil-mediated lung inflammation that involves lung parenchyma or airways or both. Allergic bronchopulmonary aspergillosis and TPE are two such eosinophilic lung disorders with different etiologies, overlapping presentations but different management. While eosinophils are the primary cells causing bronchial inflammation in asthma/ABPA, TPE is characterized by tissue eosinophil infiltration. Although isolated presentations of the two diseases are well documented in the literature,[1] their coexistence is not known.

Case Description

A 60-year-old non-smoker male presented to the outpatient department (OPD) of Department of Pulmonary Medicine with complaints of intermittent fever (around 100 F) for 4 months, dry cough, and progressive shortness of breath for 2 months. The patient was diagnosed case of bronchial asthma for the last 10 years and was on treatment with a combination of inhaled bronchodilators and corticosteroids, montelukast, and theophylline. Despite treatment, the disease was uncontrolled with daily use of a rescue inhaler (levo-salbutamol) reported by him. Chest auscultation revealed normal air entry with bilateral polyphonic inspiratory and expiratory wheeze. Chest X-ray was grossly normal with mildly increased bronchovascular markings (Fig. 1). Complete

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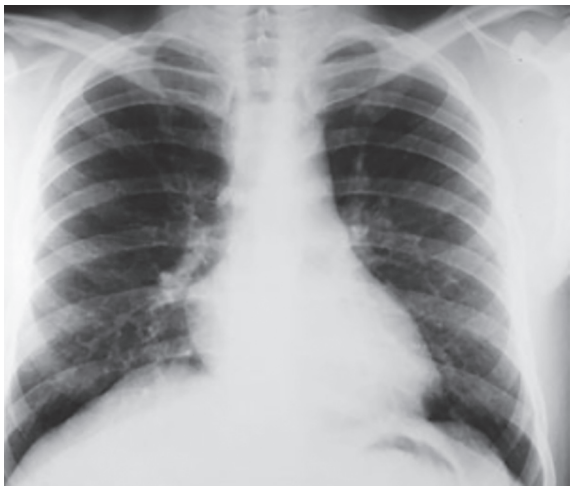

Fig. 1: Chest X-ray showing mildly increased bronchovascular markings

Figs 2A and B: (A) Computed tomography (CT) thorax at level of carina showing non-specific airspace patch in left upper zone; (B) CT thorax at level of carina shows small pleural based thickening bilaterally

blood count revealed hemoglobin of 13.5 gm/dL, a total leucocyte count of 19200/µL with elevated eosinophils (16%). The patient was suspected to have ABPA and serum immunoglobulin E (IgE) specific for *Aspergilus fumigatus* was sent which came higher (4.19 IU/mL) than the normal cut-off value. Subsequently, total IgE (9885 IU/mL) and serum precipitins immunoglobulin G (IgG) for Aspergillus were also found to be raised confirming the diagnosis of ABPA. High-resolution computed tomography (HRCT) thorax was done that showed a non-specific airspace patch in the left upper zone and small pleural-based thickening bilaterally (Fig. 2). The patient was started on oral steroids (prednisolone 0.5 mg/kg/day) and the asthma treatment was continued.

The patient showed initial improvement but the low-grade fever and nocturnal cough persisted at 8 weeks of treatment. Serum IgE levels decreased to 5664 IU/mL, but absolute eosinophil count (AEC) was significantly high (2100/mm^3). So, the patient was evaluated for other potential causes of eosinophilia. A stool examination was done that was negative for cysts, ova, and parasites. The antineutrophil cytoplasmic antibody (ANCA) profile was negative but the serum for microfilaria antigen came out to be positive. So, he was diagnosed to have concurrent ABPA and TPE. Diethylcarbamazine (DEC) in the dose of 100 mg three times a day (TID) was added to the treatment. After 3 weeks, the patient showed a significant improvement in symptoms with a further decrease in serum IgE levels (2356 IU/mL) and blood eosinophils (AEC 450/mm^3).

Discussion

Allergic bronchopulmonary aspergillosis is an inflammatory respiratory disorder triggered by hypersensitivity to the fungus *A. fumigatus*. The disease manifests clinically as chronic asthma, transient pulmonary infiltrates, and bronchiectasis. Allergic bronchopulmonary aspergillosis frequently muddles the course of patients with asthma,[2] presenting with difficult-to-control symptoms.

Allergic bronchopulmonary aspergillosis is diagnosed using a set of criteria that include predisposing conditions, asthma, cystic fibrosis, presence of two obligatory parameters (immediate cutaneous hyper-reactivity to aspergillus antigens or IgE above 0.35 kUA/L and total IgE above 1000 IU/mL) along with at least 2 out of 3 other criteria (peripheral blood eosinophil count above 500 cells/µL, transient pulmonary infiltrates on chest radiograph, and presence of precipitins (IgG) against *A. fumigatus*).[3] The present case also fulfilled all criteria for ABPA, however, there was an absence of bronchiectasis or high attenuation mucus on CT that are specific for ABPA. The treatment of ABPA encompasses the use of oral glucocorticoids as anti-inflammatory agents to suppress the underlying immune hyper-reactivity, as was also given in our patient. Antifungal agents (Itraconazole) are also recommended as add-on therapy to reduce the fungal burden, particularly in patients with ABPA relapse.[4]

Tropical pulmonary eosinophilia is another important eosinophilic lung disease caused by a hypersensitivity response to microfilariae of the parasites, *Wuchereria bancrofti* and *Brugia malayi*.[5] The disease is commonly seen in filariasis endemic areas with symptoms of paroxysmal nocturnal cough, breathlessness, and high eosinophil counts. The present patient was also a resident of the endemic area but remained unsuspected for TPE due to overlapping clinical presentation with ABPA. Confirmation is done by positive serology for microfilariae (elevated serum antifilarial IgE, IgG, or microfilaria antigen).[5] In the present case, serum for microfilaria antigen came out to be positive. Although the HRCT scan did not show ground glass opacities suggestive of TPE, the institution of DEC led to a marked improvement in clinical and hematological parameters that validated the causative role of microfilaria in the dual pathology.

The coexistence of these two diseases seems to be a rare phenomenon. We tried to search for a similar case presentation in literature by using different combinations of keywords ABPA, TPE, ABPA on Google Scholar and PubMed, but could not find any such report. The two diseases have been widely described individually in historical reports from India.[1] Chhabra et al. evaluated three patients with respiratory symptoms and eosinophilia and confirmed them as TPE but during the assessment of these patients, specific IgE levels against *A. fumigatus* were found to be elevated indicating fungal hypersensitivity to *A. fumigatus*.[4] On the similar ground, another report demonstrated airway hyper-responsiveness in two patients with TPE.[6]

The present case brings to light the rare coexistence of these two eosinophilic lung diseases. Although infrequent, the co-occurrence of these two diseases is very much possible, especially in endemic

countries like India where both diseases are widely prevalent. A patient presenting with eosinophilia and raised IgE levels should be thoroughly investigated and all differentials should be kept in mind to detect and appropriately manage any coexisting disorder. A high index of suspicion is needed to detect TPE in patients of ABPA, particularly those not responding to treatment.

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