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Allergic bronchopulmonary aspergillosis with eosinophilic pleural effusion

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Abstract
Allergic bronchopulmonary aspergillosis (ABPA) in a patient without previously diagnosed asthma or cystic fibrosis with pleural effusion at presentation is uncommon and rare. In a high tuberculosis burden country like India, patients with pleural effusion are frequently misdiagnosed as tuberculosis even when there is no bacteriological confirmation. We report a case of a 48-year female who presented with left side pleural effusion not resolving on antitubercular treatment from two months. The pleural fluid was exudative with 20% eosinophils. Investigations revealed peripheral eosinophilia with HRCT thorax showing left lingular and right upper lobe bronchiectasis. Spirometry showed reversible moderate obstruction. Elevated serum immunoglobulin E, Precipitating serum antibodies to Aspergillus fumigatus were positive and the Aspergillus fumigatus immediate skin test yielded a positive reaction. A diagnosis of ABPA associated with eosinophilic pleural effusions was made and the patient was commenced on prednisolone. At review, the patient's symptoms had considerably improved and her pleural effusions had resolved.

Keywords: ABPA; Eosinophilic pleural effusion; Misdiagnosed TB

Introduction
Allergic Bronchopulmonary Aspergillosis (ABPA) is a hypersensitivity reaction to fungus Aspergillus fumigatus commonly affecting asthmatic and cystic fibrosis patients [1]. Patients typically present with migratory pulmonary infiltrates and bronchiectasis, though atypical presentations like paratracheal and hilar lymphadenopathy, collapse, pneumothorax, bronchopleural fistula, and allergic sinusitis have also been observed [2]. In all cases of eosinophilic, exudative pleural effusion, other causes like ABPA must be ruled out before attributing the effusion to tuberculosis as in our case.

Case Report:
A 48-year-old female, presented to the OPD of PGIMS Rohtak with complaints of dry cough, left-sided chest pain and shortness of breath (SOB) on exertion for the past 2 months. There was no history of hemoptysis, expectoration of mucus plugs, asthma, cystic fibrosis and trauma. She initially presented to a private practitioner with the above complaints who on chest xray and ultrasound thorax found mild left-sided pleural effusion. After diagnostic thoracentesis and pleural fluid analysis, she was started on ATT by the private practitioner. She came to our OPD as her symptoms worsened over the due course. On physical examination, she was found to have bilateral wheeze and diminished breath sounds in the left lower chest. Chest Xray (Figure-1) and ultrasound thorax showed mild left-sided pleural effusion. Diagnostic thoracocentesis was done and pleural fluid analysis revealed a cell count of 8000cells/mm3 with 40% lymphocytes, 30% neutrophils, 20% eosinophils and 10% monocytes with protein levels of 5.7 g/dL and glucose of 95 mg/dL. Pleural fluid adenosine deaminase levels (ADA) were 59.0 units/L. Stain for acid-fast bacillus (AFB) was negative and bacterial pyogenic culture was negative. The cytological examination was negative for malignant cells. Her Mantoux test was negative. We advised HRCT thorax for her which showed left lingular and right upper lobe bronchiectasis with centrilobular nodules in the right upper lobe and cystic changes in upper lobes of both lung fields.(Figure-2) On spirometry, she had a moderate obstruction. In view of the clinicoradiological presentation, she was then evaluated for ABPA. Her serum IgE (Immunoglobulin E) specific for Aspergillus fumigatus was 47.30 kUA/L (kilo units of antibodies per liter), total IgE was 9252 kUA/L, IgG specific for Aspergillus fumigatus 69.03 U/ml and absolute eosinophil count (AEC) was 1356 cells/mm3. A. fumigatus immediate skin test yielded a positive reaction. Sputum induction was done and was negative for AFB on microscopy and cartridge based nucleic acid amplification test (CBNAAT). A diagnosis of ABPA with eosinophilic pleural effusion was made. She was started on formoterol (6 mcg) and budesonide (400 mcg) inhaler 2 puffs BD with spacer, oral prednisolone 1.0 mg/kg for 4 weeks followed by 0.5 mg/kg for 2 weeks and itraconazole 200 mg BD for 2 months. On follow up after 3 months her symptoms were controlled, effusion subsided on chest Xray (Figure-3) and her AEC had reduced to 500 cells/mm3.

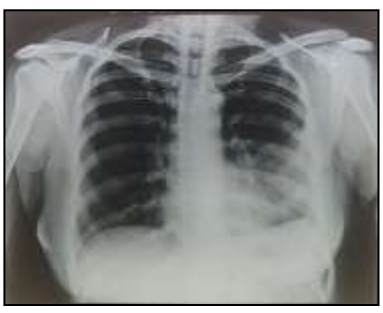

Fig 1: Chest radiograph shows left lower lung zone homogenous opaque

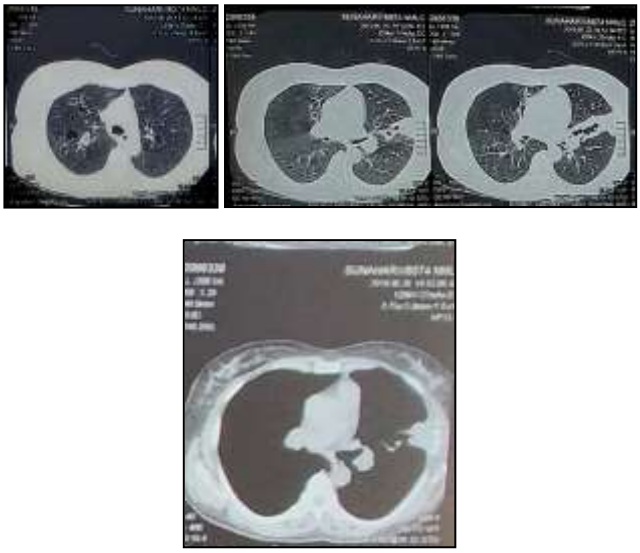

Fig 2: High-resolution computed tomography of the chest shows left lingular and right upper lobe bronchiectasis with centrilobular nodules in the right upper lobe and cystic changes in upper lobes of both lung fields and mild left side pleural effusion

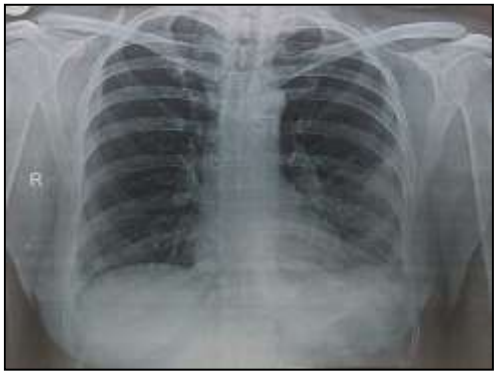

Fig 3: Chest radiograph shows left lower lung zone homogenous opacity (pleural effusion) in previous chest radiograph subsided

Discussion

ABPA usually complicates asthma and cystic fibrosis. Pleural involvement is uncommon and there are very few reported cases of ABPA with pleural involvement. In most of these reported cases, diagnostic thoracentesis revealed a predominant lymphocytic cell population. The mechanism of pleural effusion in ABPA includes an intense inflammatory response, with the release of cytokines and fungal translocation into the pleural space, leading to a local Th2-dependent inflammatory response or lung collapse, leading to "ex vacuo" pleural effusion [3]. Another case of ABPA with collapse lung and pleural effusion suggested that sterile exudative pleural effusion occurred due to increased negative intapleural pressure caused by collapse rather than translocation of fungus [4] Another postulated mechanism is the inflammatory pleural reaction, which occurs adjacent to inflamed lung tissue, leads to the development of an exudative effusion. Although there is a broad differential diagnosis of eosinophilic pleural effusions, the description of only two such cases highlights the rarity of ABPA as a reported cause of eosinophilic pleural effusions.

In a similar study by Madan et al, they highlighted the importance of aggressive investigation for tuberculosis in the patients of ABPA with pleural effusion before starting them on steroids [5].

Therefore in all cases of suspected ABPA, pulmonary tuberculosis needs to be aggressively ruled out to prevent the disastrous consequences later in life as these patients receive glucocorticoids as part of treatment.

Conclusion

ABPA rarely present with pleural effusion and must be considered in the differential diagnosis of patients presenting with an eosinophilic pleural effusion. Though these cases are rare still diagnosis is possible without any invasive procedures like thoracoscopy and bronchoscopy based on the clinic-radiological profile of the patient and serological tests. Regular follow-up is necessary for these patients for a better outcome.

Conflict of Interest: Authors have no conflict of interest.

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