ABPA Mimicking Pulmonary Tuberculosis - A Case Report

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Abstract: Allergic Broncho pulmonary Aspergillosis a immune mediated hypersensitivity reaction to inhaled spores of fungus Aspergillus fumigatus seen exclusively in patients with long standing Asthma. It is seen in about 20% of patients with asthma ABPA is frequently misdiagnosed as tuberculosis because of common presentation and radiological features. Here we present a case of ABPA misdiagnosed as Pulmonary Tuberculosis.

Keywords: ABPA (Allergic Bronchopulmonary Aspergillosis), Tuberculosis

1. Introduction

Allergic Broncho pulmonary Aspergillosis a hypersensitivity reaction to antigens of fungus Aspergillus fumigatus seen patients with long standing Asthma or Cystic fibrosis. It is seen in 20% of Bronchial asthma a patients(1). About half of ABPA has been initially mis-diagnosed as Tuberculosis. Early diagnosis of ABPA prevents the onset of Bronchiectasis. Here we present a 18year old female who has been mis-diagnosed as Pulmonary Tuberculosis and was on Anti-tuberculous Therapy but subsequently diagnosed as ABPA in our institute.

2. Case Report

A 19 year old female came with complaints of fever, cough with sputum, breathlessness, loss of appetite for 20 days. She had past history of asthma for which she has been taking inhalers since childhood. Her initial Chest x ray showed opacities in right middle lobe and left upper lobe. She was started on ATT on clinico-radiological basis with sputum CBNAAT negative for MTB. After 2 months ATT she has persistent symptoms, hence she was referred to our hospital for further evaluation.

We admitted in our institute, complete clinical examination was done. She had bilateral wheeze, Her initial Chest X-ray showed bilateral consolidation in right middle and left upper lobes. Since the pattern in x ray was suspicious of ABPA. CT chest was done which showed bilateral consolidation and suspicious high attenuation mucus plugs. Blood investigation were done with Esophageal eosinophil levels of 884 cells per microL (normal less than 500cells per microL), total IgE levels were 1058IU/mL (normal 100 IU/mL) and IgE specific for Aspergillus fumigatus was 39.4kUA/L (below 0.38.KUA/L). With the above lab investigations, on background of long standing Asthma the diagnosis of ABPA was made. Her sputum was sent for fungal culture which showed growth of Aspergillus species. Subsequently she was started on steroids and her symptoms improved.

3. Discussion

ABPA occurs due to hypersensitivity reaction to antigens of fungus Aspergillus fumigatus which colonizes in patients with long standing asthma. It manifests as shortness of breath due to bronchospasm, pulmonary infiltrates, low grade fever with a chronic course. Though pathogenesis is incompletely understood, inhaled Aspergillus spores in sufficient quantities behave as antigens. The antibodies IgG and IgE will be elevated in response to the antigens. In healthy individuals the fungal spores will be eliminated and Antibody levels will be very low. Th2 helper cells plays a critical role in atopic individuals leading to mast cell degranulation Aspergillus fumigatus protease causes release of proinflammatory cytokines IL8 leading damage to the epithelium and disruption of protective barriers. This leads to hypersensitivity reaction with further release of IL4, IL5 and IL 13 with increased blood and airway eosinophils.

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Criteria for diagnosing ABPA proposed by International Society of Human and Animal Mycology includes the following:

A. Predisposing conditions:
1) Bronchial Asthma
2) Cystic fibrosis.

B. Obligatory criteria (both):
1) Type 1 positive Aspergillus skin test, cutaneous Hypersensitivity to Aspergillus antigen or Elevated IgE Specific to Aspergillus.
2) Elevated total IgE levels. (>1000IU/mL).

C. Other criteria (any 2):
1) Presence of precipitating or IgG antibodies against Aspergillus fumigatus.
2) Radiologic features suggestive of ABPA
3) Total eosinophil count >500 cells/microL.

Radiologic features suggestive of ABPA includes transient consolidation, nodules fleeting opacities, toothpaste finger in glove appearance or permanent findings central bronchiectasis, ring shadows, or pleuro-pulmonary fibrosis.

Treatment of a patient with ABPA rules mainly on oral corticosteroids. Two regimens are given short or longer regimen. Shorter regimen 0.5mg/kg per day of oral prednisone for 1 to 2 weeks and then tapered over a period of 12 weeks. Longer regimen involves 0.75mg/kg tapered for a duration of 6 to 12 months. Adjunct to steroids antifungal therapy is used. Oral itraconazole reduces steroid dose, serum IgE levels, sputum eosinophil and also number of exacerbations. Duration of itraconazole is minimum of 4 to 6 months.

4. Conclusion

Although Tuberculosis is endemic in India, the diagnosis of TB will be questionable when sputum CBNAAT is negative and patient fails to improve with anti tuberculosis therapy. ABPA is one of the unrecognized disease, with increase in incidence in modern days. With availability of Antibody testing ABPA should be one of the differentials in top order in patients with standing Asthma with abnormal x ray. This case illustrates the same.

Conflict of Interest: None

References