



International Journal of Current Research in Medical Sciences

ISSN: 2454-5716

P-ISJN: A4372-3064, E-ISJN: A4372-3061

www.ijcrims.com



Case Report

Volume 4, Issue 7 -2018

DOI: <http://dx.doi.org/10.22192/ijcrms.2018.04.07.009>

Allergic Bronchopulmonary Aspergillosis (ABPA) misdiagnosed as Pulmonary Tuberculosis

¹ Verma, Vishal;

Senior Resident , Chest and TB department, Government Medical College, Amritsar, India

² Rana, Srijna*;

Junior Resident, Chest and TB department, Government Medical College, Amritsar, India

³ Gupta , Sandeep;

Assistant Professor, Chest and TB Department, Government Medical College, Amritsar, India

⁴ Singh, Daljit ;

Senior Resident , Chest and TB department, Government Medical College, Amritsar, India

⁵ Kajal, NC;

Professor, Chest and TB Department, Government Medical College, Amritsar, India

⁶ N.S.Neki;

Professor of Medicine, Govt. Medical College, Amritsar, India

*Corresponding author: **Dr. Srijna Rana**

Room no: 57, NRI hostel, Government Medical College - Amritsar

Circular road Amritsar-143001

E-mail: srijnarana@yahoo.com

Abstract

Allergic Bronchopulmonary Aspergillosis (ABPA) is a syndrome associated with asthma, manifesting as transient pulmonary infiltrates and eosinophilia which can progress to severe proximal bronchiectasis, and pulmonary fibrosis. Clinikoradiologically it is similar to pulmonary tuberculosis hence most cases of ABPA are usually misdiagnosed as pulmonary tuberculosis resulting in delay in diagnosis as well as treatment which predisposes them to complications and potentially toxic anti-tubercular therapy (ATT). Here we report a case of ABPA masquerading as tuberculosis in a 67 year old female .

Keywords: ABPA, misdiagnose, ATT.

Introduction

Aspergillus is a ubiquitous fungus which causes a variety of clinical syndromes. Allergic bronchopulmonary Aspergillosis (ABPA) is the best recognized manifestation of *Aspergillus*-associated hypersensitivity to *Aspergillus* antigens in patients with long standing atopic asthma. It is characterized by the presence of asthma, peripheral blood eosinophilia, immediate type I skin reactivity and serum precipitin antibodies to *Aspergillus fumigatus*, elevated total serum IgE, increased levels of *Aspergillus* specific IgE and IgG, pulmonary infiltrates, and central bronchiectasis. Most of the ABPA cases are initially misdiagnosed as pulmonary tuberculosis [1]

Case Report

A 67 year old female patient presented with the chief complaint of haemoptysis since 1 week. She was given symptomatic treatment for the same. She also gave history of intermittent fever and cough with minimal expectoration for last four months. Patient had taken antituberculosis treatment (Rifampicin, Isoniazid, Ethambutol, Pyrazinamide) two times in the last two years with no significant improvement. On further evaluation, patient also gave history of episodic breathlessness with wheezing for last 12 years. On physical examination, the patient was afebrile with a pulse rate of 80/min, respiratory rate of 21/min and blood pressure of 120/80 mmHg. There was no pallor, cyanosis or clubbing. On chest examination there was no abnormality on inspection, palpation and percussion. On auscultation bilateral crepitations and ronchi were audible. Examination of other systems was unremarkable.

Routine investigation showed; hemoglobin: 10.4 gm%, total leucocyte count: 8700/mm³ with on differential count - eosinophils were raised, platelet count: 3.3 lacs/mm³ and Erythrocyte sedimentation rate: 35 mm/hr. Mantoux test

showed an induration of 6mm at 72 hours. But sputum samples for acid-fast bacilli were found to be negative. The skin prick test for *Aspergillus fumigatus* and *Aspergillus flavus* were positive. The total IgE and eosinophil count were 267 IU/mL and 760 cells/ μ L, respectively. Galactomannan antigen for *Aspergillus* was 3.36.

Chest radiograph revealed heterogenous opacities in right upper and mid zones with a fibrotic band and cavitations on right side and partial segmental collapse on left upper lobe [Figure 1a] Contrast enhanced computed tomography (CECT) of the chest showed fibroconsolidative changes in the right upper lobe and apical segment of right lower lobe alongwith collapse consolidation in the right upper lobe [Figure 2].

On Bronchoscopy mucus plugging was seen in both the bronchus

Skin prick test with *Aspergillus fumigatus* antigen showed a positive reaction for type I and also late type III (Arthus) hypersensitivity in comparison to positive control (Histamine Phosphate 1mg/ml). Total IgE was 2234 IU/ml (reference range 0 to 100 IU/ml). Specific IgG and IgE against *A. fumigatus* by ELISA were 4.43 U/ml and 3.57 U/ml. Spirometry showed features of mild obstruction (FEV1 67%, FEV1/FVC ratio 72) with significant bronchodilator reversibility (post bronchodilator FEV1 72% [% change 16]). Thus a diagnosis of ABPA was established.

It was decided to keep the patient under observation while treatment for ABPA with oral methylprednisolone, starting with a daily single dose of 16mg, voriconazole 200 mg along with inhaled fluticasone with formoterol in two divided doses and as and when required. Patient responded very well to this treatment and her serial radiography of the chest showed resolution of the persisting opacity, after 2 months of the therapy. [Figure 1b]



Figure 1a – Chest x-ray PA view showing heterogenous opacities on the right and partial segmental collapse on the left

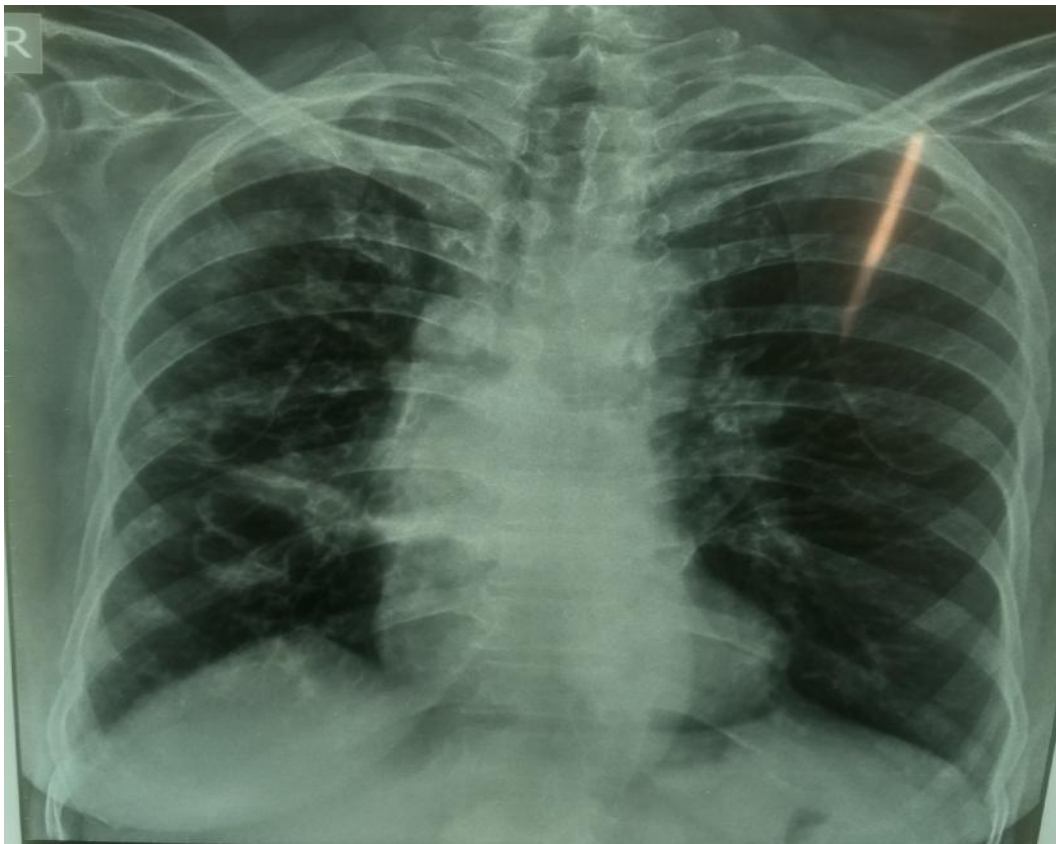


Figure 1b - Chest x-ray PA view after two months of treatment showing resolution

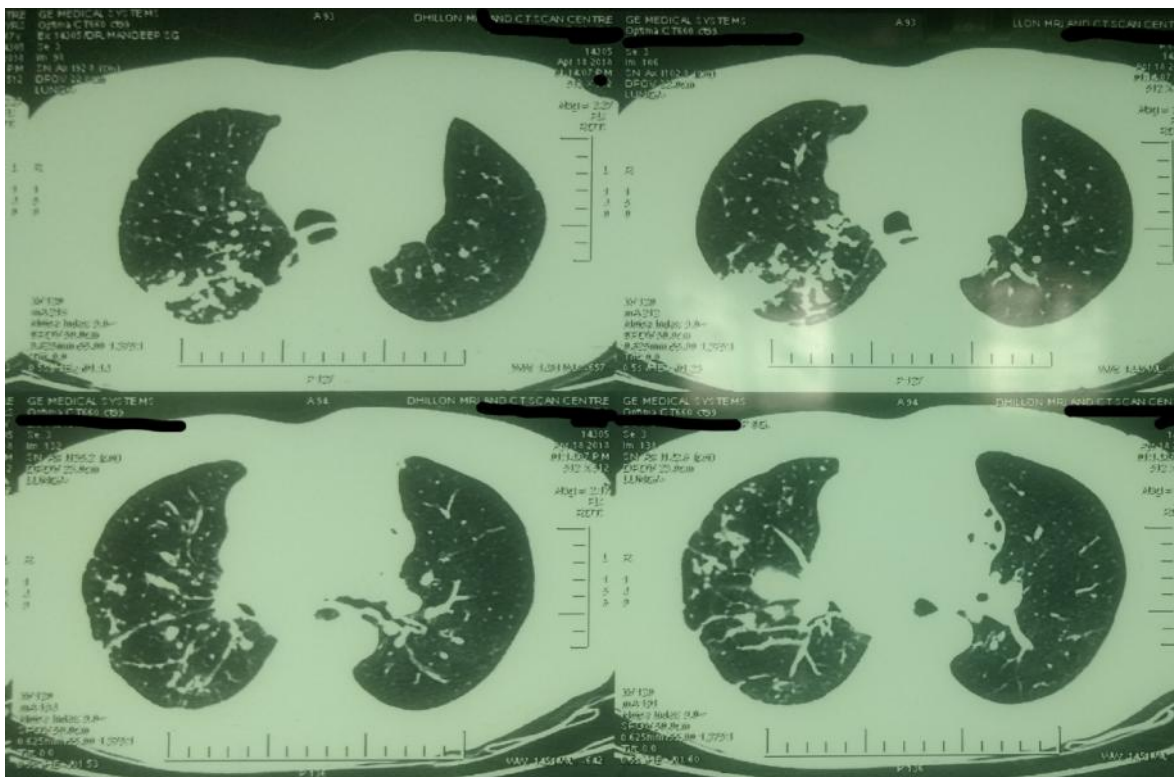


Figure 2 - CECT of the chest showed fibroconsolidative changes in the right upper lobe and apical segment of right lower lobe alongwith collapse consolidation in the right upper lobe

Discussion

Aspergillus causes a variety of clinical syndromes in the lung, ranging from aspergilloma in patients with lung cavities, to chronic necrotizing aspergillosis in those who are mildly immunocompromised or have chronic lung disease but most commonly presents as ABPA. A wide variety of chest radiographic changes are known to be associated with ABPA. These changes include normal chest radiography, nodules, avascular areas, various infiltrate patterns, consolidation, parallel lines and ring shadows, hyperinflation, band shadows and tramline shadows, “honey combing” “toothpaste” shadows, “gloved finger”, changes like fibrosing alveolitis, lobar shrinkage and atelectasis as well as pseudohilar adenopathy and pleural thickening. [2-8]

In the present case, the diagnosis of ABPA was established on clinical, hematological, immunological and radiological grounds. Two of the most common differential diagnoses of ABPA include bacterial pneumonia [9] and pulmonary

tuberculosis [10, 11] which should be given a great caution because of the high prevalence.

Usually misdiagnosis is associated with similar clinical and radiological pattern of lung diseases, misinterpretation of chest X-ray and detection of AFB in sputum. Several features are common to both ABPA and Pulmonary Tuberculosis; clinical findings, raised ESR and even radiological appearance (upper lobe infiltrates and cavities). Patient with pulmonary tuberculosis present with chronic cough, fever, weight loss and hemoptysis, and ABPA presents with asthma symptoms with associated fever, weight loss, hemoptysis. Due to similarity in clinical symptoms, endemicity of tuberculosis in India and radiological opacities, it becomes difficult to diagnose or even suspect ABPA on initial outpatient visits. If the patient is started on ATT without actual disease, many problems may occur. First and foremost is wrong treatment, together with wrong data collection, and more importantly no actual treatment was given to patient, hence, leading to more chances of fibrotic lung disease and disability and increased morbidity.

Conclusion

Awareness of entity, careful history taking and astute clinical examination may give important clinical clues to evaluate further. 'All wheezes are not asthma' and 'Every cough more than 2 weeks should be evaluated for tuberculosis but should not be treated in haste as tuberculosis'. Its important to take these into consideration as most of the cases are treated as a case of pulmonary tuberculosis. Other differential diagnoses include sarcoidosis, cryptogenic organising pneumonia, chronic eosinophilic pneumonia and carcinoma lung. More awareness is needed so as to curb wrong diagnosis as well as treatment.

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How to cite this article:

Verma, Vishal, Rana, Srijna, Gupta Sandeep, Singh, Daljit , Kajal, NC, N.S.Neki. (2018). Allergic Bronchopulmonary Aspergillosis (ABPA) misdiagnosed as Pulmonary Tuberculosis. Int. J. Curr. Res. Med. Sci. 4(7): 42-46.

DOI: <http://dx.doi.org/10.22192/ijcrms.2018.04.07.009>