Case Report

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Allergic Bronchopulmonary Aspergillosis: A Masquerade of Difficult to Control Asthma

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ABSTRACT

Background

Allergic bronchopulmonary aspergillosis (ABPA) is an immunological pulmonary disorder caused by hypersensitivity to a common fungus *Aspergillus* which colonizes the airways of patients with asthma and cystic fibrosis. Characterized by chronic bronchial inflammation, eosinophilia, airway remodeling, and bronchiectasis, it presents clinically with worsening symptoms of cough and breathlessness that is difficult to control. Early diagnosis and rapid implementation of proper management are critical to prevent complications and/or disease progression. Although latest criteria depends on positive serology and typical radiological findings associated with the disease, atypical presentation can occur thus a high index of suspicion is necessary. We describe a case of 16 year old female with difficult to control asthma diagnosed as ABPA in absence of any identifiable imaging findings.

Keywords: Allergic Bronchopulmonary Aspergillosis (ABPA); Peak Expiratory Flow (PEF); Immunoglobulin G (IgG); Immunoglobulin M (IgM)

Correspondence: Dr. Parishrut Prasad Pandey, Department of Internal Medicine, College of Medical Sciences, Bharatpur, Chitwan, Nepal. Email: Parishrut@hotmail.com Phone:+977-9851300556 **Article received**: 2025-12-26. **Article accepted**: 2026-02-11. **Article published**: 2026-03-31.

INTRODUCTION

Allergic bronchopulmonary aspergillosis (ABPA) is a complex hypersensitivity reaction in response to colonization of the airways with *Aspergillus fumigatus* that occurs almost exclusively in patients with asthma or cystic fibrosis (CF).[1] Aspergillus species comprise of over 100 species of molds that are present ubiquitously in the environment, especially in the organic matter but most of the illness is caused by *Aspergillus fumigatus, Aspergillus niger, Aspergillus flavus*, and *Aspergillus clavatus.*

In ABPA, Aspergillus colonization of the asthmatic airway leads to vigorous IgE- and IgG-mediated immune responses superimposed on the asthmatic milieu. Aroteolytic enzymes and mycotoxins released by fungi, in concert with Th2-mediated eosinophilic inflammation and IL-8-mediated neutrophilic inflammation result in airway damage, bronchiectasis, fibrosis and respiratory compromise.[2,3,4] The thick mucus in the airways of these patients makes it difficult to clear up the Aspergillus spores when inhaled. The prevalence of allergic bronchopulmonary aspergillosis (ABPA) among patients with persistent asthma is estimated at 1 to 2 percent, although rates up to 28 percent have been reported in the indian subcontinent with cases peaking during early winters and fall.[5] Radiographically manifesting as include fleeting opacities, bronchiectasis, and findings related to mucoid impaction and bronchial obstruction, it can manifest even in absence of any identifiable imaging abnormality.[6,7]

CASE PRESENTATION

A 16 year old girl, previously diagnosed as Asthma from her childhood from a local health center near her home presented to our center with complains of cough on and off since 6 months, associated with diurnal variation, increased in severity during the evenings. The cough was persistent since 6 months only partially relieved by medications. She was under inhaler medications Budesonide 200mg and Salbutamol 200mcg on as needed basis with periodic increase her doses as per requirement. She did not give history of hemoptysis, Fever, weight loss or night sweats.

She visited our out patient department with the above mentioned complains. Her investigations were normal with complete hemogram displaying normal eosinophil levels.

Chest Xray was unremarkable for any new lesions and cardiac investigations were normal. Her spirometry showed mild obstructive picture with reversibility and PEF showed a diurnal variation of 23%.

She was advised for a FeNO test but refused thus was deferred. With the diagnosis of Moderate persistent asthma she was started treatment based on the GINA guidelines of track 1 with combination of ICS- Formeterol as Maintenance and Reliever therapy. She was called back for follow up after a month.

On her follow up she showed improvement although she still complained of cough. Chest Examination was still positive for occasional wheezing. With Reinforcement of correct inhaler technique she was started on a mast cell stabilizer and again called for follow up after 1 month. She responded to the treatment on follow up with good control of her symptoms and increase in her exercise tolerance.

However, again after 2 months she presented tour center with similar complains of cough and Occasional SOB. Her HRCT chest was done which was normal and blood investigations did not reveal any new abnormalities. Absolute eosinophil count was within normal limit.

With possibility of ABPA she was counselled for testing. Although initially she refused with further reinforcement the patient party agreed. Her total IgE levels came out to be >3000 and serology tested positive for *Aspergillus fumigatus* IG M.

She was started on Steroids with tablet cortilone 0.5mg/kg for 2 weeks and taper over 3 months. However upon reduction of her steroid dose to 10 mg she again complained of increasing cough.

She was then again continued on her previous dose of cortilone (20mg) along with tab Voriconazole 200mg twice daily for 16 weeks. During the course

of treatment she gradually responded and at the end of treatment was able to function normally with no limitation of activity. She was continued on ICS-Formetrol for further 3 months then gradually it was tapered to as per needed basis.

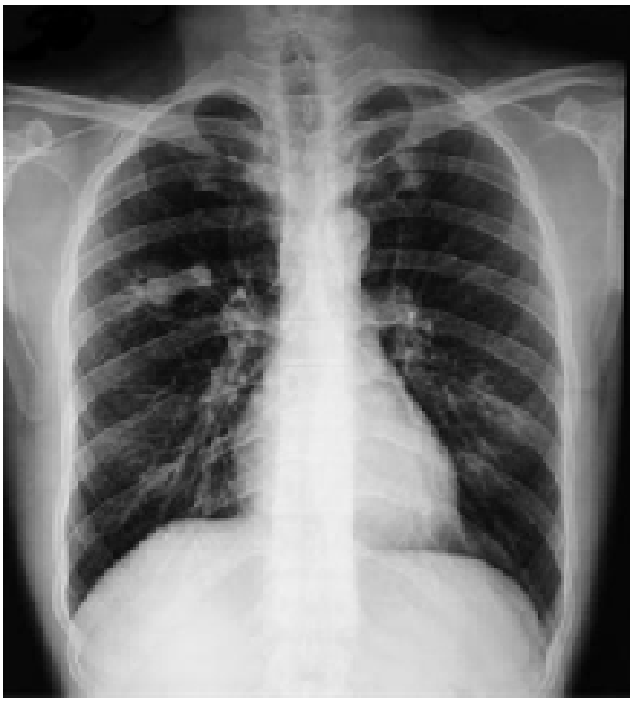

Figure 1. Chest radiograph showing a non-homogeneous opacity in the right mid zone with perihilar patchy infiltrates in the left mid and lower zones. Transient pulmonary infiltrates or fleeting shadows that are characteristic of allergic bronchopulmonary aspergillosis are visible (Figure 1).

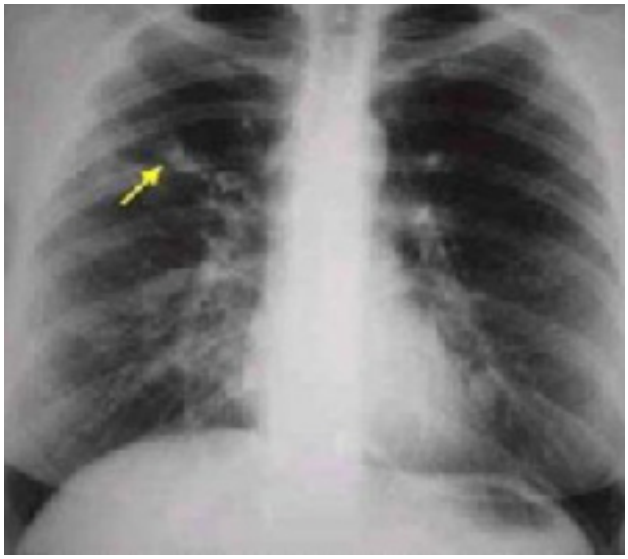

Figure 2. Chest xray showing finger in glove shadow (arrow) and modular ioacities in right middle third in ABPA

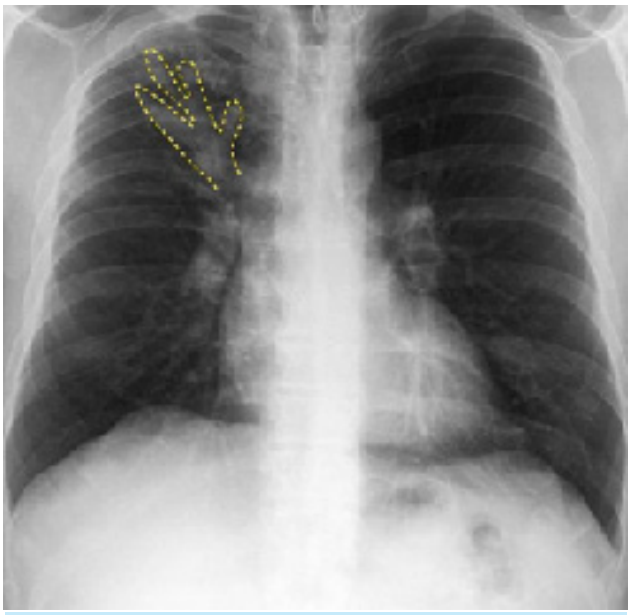

Figure 3. Glove like opacity in the right upper zone (yellow dotted line) represents sputum plugged bronchiectasis

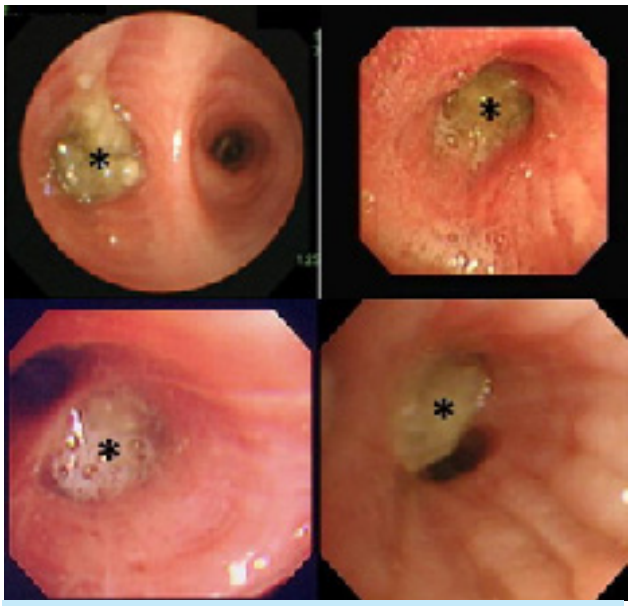

Figure 4. Showing extensive mucus plugging seen during bronchoscopy in ABPA.

DISCUSSION

ABPA was first described by Hinson and colleagues in 1952 and is often associated with chronic lower airway diseases (asthma and cystic fibrosis) which are associated with viscous mucus production and impaired mucociliary clearance, allowing inhaled *Aspergillus fumigatus* spores to persist in the airways. It may occur infrequently in patients without a history of bronchial asthma.

The small conidia of *Aspergillus fumigatus* can easily enter the airways. Exposure to large numbers of conidia may cause ABPA , but not all asthmatics develop ABPA despite being exposed to the same environmental factors. Genetic associationwith HLA-DR molecules DR2, DR5, and possibly DR4 or DR7 contribute has been shown to contibute to susceptibility; whereas, HLA-DQ2 contributes to resistance, and a combination of these may determine the outcome of ABPA and the adptive immune responses (Th2 CD4+ T cell responses) of the lung.[8]

A History of recurrent episodes of wheezing with cough, dyspnea, pleuritic chest pain, blood-stained sputum, or sputum with brown mucus plugs with ancillary symptoms like anorexia, fatigue, generalized aches, low grade fever should prompt the treating clinician to consider the possibility of ABPA.[9]

Radiologically Chest X-ray has 50% sensitivity for the diagnosis of ABPA. It can show parenchymal infiltrate and bronchiectasis changes mostly in the upper lobes; However, all lobes may exhibit involvement.

HRCT Chest is the investigation of choice to detect bronchiectasis distribution and other abnormalities that are undetectable on a chest X-ray, such as centrilobular nodules and tree-in-bud appearance.[10]

The following shadows may present radiologically:

'Finger in glove' opacity: suggestive of mucoid impaction in dilated bronchi.

'Tramline shadows': suggestive of parallel linear shadows extending from the hilum in bronchial distribution and reflecting longitudinal views of inflamed, edematous bronchi.

'Toothpaste shadows': representing mucoid impaction of the bronchi.

'Ring shadows': reflecting dilated bronchi with inflamed bronchial walls.

Sputum cultures for A. fumigatus is not diagnostic, but if it reveals an organism, then it helps in drug susceptibility test.

In areas with availability Bronchoscopy may show areas of Mucoid impaction and bronchial brushings may reveal mucus that contains aggregates of eosinophils, fungal hyphae, and eosinophil-derived Charcot-Leyden crystals. The finding of hyphae-filled mucus plugs is considered pathognomonic for ABPA

Diagnosis can be made by the criteria proposed by ISHAM working group which puts forth:

Predisposing conditions of 1. Bronchial asthma.
2. Cystic fibrosis.

Obligatory criteria (both should be present)'

Type I - positive Aspergillus skin test (immediate cutaneous hypersensitivity to Aspergillus antigen) or elevated IgE levels against Af.

Elevated total IgE levels (greater than 1000 IU/mL).

Other criteria (at least two of three)

Presence of precipitating or IgG antibodies against Af in serum.

Radiographic pulmonary opacities consistent with ABPA.

Total eosinophil count over 500 cells/microliter in steroid naive patients.

(If the patient meets all the other criteria, an IgE value less than 1000 IU/mL may be acceptable).

The treatment of ABPA mostly focusus on symptom control and cessation of lung damage. Systemic glucocorticoids are considered the mainstay of treatment of acute ABPA based on the results of case series. The optimal dosing of Prednisolone is not known. Although commonly used regimen use an initial dose of prednisone 0.5 mg/kg daily (or equivalent) for 14 days, followed by conversion to an every other day regimen of 0.5 mg/kg, and further tapering and discontinuation at three months . Some

patients may need a higher initial dose of prednisone (eg, 40 to 60 mg/day).

Antifungal therapy are reserved for patient who are unable to taper oral glucocorticoids or have an exacerbation of ABPA as seen with our patient. Typically Oral voriconazole is used for adults with a loading dose of 400 mg every 12 hours for two doses followed by a maintenance dose of 200 mg twice daily for 16 weeks. Liver function tests should be monitored closely for any evidence of hepatotoxicity. Alternatives include itraconazole in loading dose of 200 mg three times a day for three days, followed by 200 mg twice daily for 16 weeks but whether intraconazole is superior to voricaonzole remains unclear despite randomized control trials.[11,12,13]

Use of Biologics are reserved to patients who are unable to taper off steroids or have recurrent colonoization despite course of antifungals. Anti IGE therapy with omalizumab, Anti- IL5 agents and Anti IL -4 alpha agents are on the new trends with each showing good efficacy.

CONCLUSIONS

The Possibilty of ABPA in the patient of asthma should be always born in mind in patients whose symptom control do not reach satisfactory levels despite optimization of medical treatment despite the absence of typical radiological findings or biochemical clues which as seen in our case which was mimicking as acute exacerbation of asthma. This case highlights the challenge faced during the diagnosis of ABPA with atypical presentation.

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Authors' contributions

Conceptualization: Parishrut Prasad Pandey,

Data curation: Parishrut Prasad Pandey,

Formal analysis: Parishrut Prasad Pandey, Shaphal, Bibesh Dahal.

Investigation: Parishrut Prasad Pandey,

Methodology: Parishrut Prasad Pandey,

Supervision: Parishrut Prasad Pandey, Shaphal Gyawali.

Writing-original draft: Parishrut Prasad Pandey, Shaphal Gyawali, Bibesh Dahal.

Writing -review & editing: Parishrut Prasad Pandey, Shaphal Gyawali, Bibesh Dahal.

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