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A Rare Presentation of Allergic Bronchopulmonary Aspergillosis

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Abstract

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity reaction to *Aspergillus fumigatus*, primarily affecting individuals with asthma or cystic fibrosis.[1] It is characterized by chronic airway inflammation, mucus plugging, and central bronchiectasis. Patients often present with wheezing, cough, breathlessness, and expectoration of brownish mucus plugs[1].

Diagnostic findings include elevated serum IgE, eosinophilia, and positive Aspergillus-specific IgE/IgG antibodies. HRCT typically shows central bronchiectasis and mucus impaction. Due to its overlap with asthma and other allergic conditions, ABPA is often underdiagnosed. Prompt recognition and treatment with corticosteroids and antifungal agents are essential to prevent irreversible lung damage.

Anaphylaxis is a serious, potentially life-threatening allergic reaction that occurs rapidly after exposure to an allergen, such as certain foods, insect stings, medications, or latex. [2]It involves multiple organ systems and can cause symptoms like difficulty breathing, swelling, low blood pressure, rash, and even unconsciousness.[2]

This case highlights an unusual presentation of ABPA initially misdiagnosed as recurrent anaphylaxis.

Keywords: NIL

Introduction

A 50-year-old male presented to DY Patil Hospital, Navi Mumbai, with complaints of breathlessness, urticaria, and a persistent cough for the past 1–2 years, which had worsened significantly over the preceding 15 days. The patient had a known history of ischemic heart disease with a left ventricular ejection fraction (LVEF) of 15–20%, systemic hypertension, and was status-post percutaneous transluminal coronary angioplasty to the left anterior descending artery. Additionally, the patient had a confirmed history of COVID-19 infection in August 2021, diagnosed via RT-PCR.

Of particular note, the patient had experienced multiple episodes of similar symptoms in the past, which were previously managed as recurrent anaphylactic reactions at an external hospital, where he received injectable corticosteroids and Adrenaline.Patient is watchman by occupation and having fungal exposure in his working place.

On current admission, investigations were initiated to explore the underlying etiology of his persistent respiratory and allergic symptoms:

1. **Complete Blood Count:** Hemoglobin: 11.9 g/dL; WBC: 8130/µL; Platelets: 217,000/µL
    a. Eosinophils: 5.6% (Absolute eosinophil count: 1200/µL)
    b. Neutrophils: 46.2%; Lymphocytes: 35.6%; Monocytes: 13.3%; Basophils: 0.7%
2. **Serum Total IgE:** 2715 IU/mL (elevated)
3. **Aspergillus-specific IgE:** 2.22 (positive)
4. **Aspergillus-specific IgG:** 41.7 (positive)
5. **Allergy Testing:**
    a. High reactivity: *Aspergillus fumigatus*
    b. Moderate: *Homia tropicalis*, mosquito, banana
    c. Low: Bohia grass, mustard, honey bee

6. **Skin Prick Test:** Positive for *Aspergillus fumigatus*, *A. flavus*, mosquito, cotton dust, and others
7. **ANA Blot, C-ANCA, P-ANCA:** Negative
8. **Pulmonary Function Test (PFT):** Small airway obstruction with good bronchodilator response
9. **Fractional exhaled Nitric Oxide (FeNO):** No eosinophilic airway inflammation
10. **HRCT Thorax:** Central bronchiectasis, mucus plugging in the right bronchus intermedius, fibrotic changes, and adjacent pleural thickening

Based on these clinical, laboratory, and radiological findings, a definitive diagnosis of ABPA was made.

The patient was treated with oral itraconazole 100 mg twice daily and injectable corticosteroids. He was discharged on tapering oral corticosteroids and itraconazole with a follow-up scheduled in 15 days. At the follow-up, the patient reported significant symptomatic improvement. The patient's IgE levels showed a significant decrease over three months, dropping from 2715 IU/mL to 1890 IU/mL, and further down to 502 IU/mL, indicating improved allergic control

Discussion

This case highlights a rare and diagnostically challenging presentation of ABPA that initially mimicked recurrent anaphylactic reactions. The patient's prior episodes of acute breathlessness and urticaria were managed as anaphylaxis without clear identification of allergen exposure. Despite receiving appropriate anaphylaxis treatment, including corticosteroids and antihistamines, his symptoms recurred, prompting further evaluation.[3]

Anaphylaxis is typically an IgE-mediated hypersensitivity reaction that manifests rapidly following exposure to a known allergen. It is characterized by hypotension, bronchospasm, skin manifestations such as urticaria and angioedema, and occasionally gastrointestinal symptoms. In this case, although hypotension was present—a feature more commonly associated with anaphylaxis—the absence of gastrointestinal involvement, the chronicity of respiratory symptoms, and the identifiable exposure to fungal allergens raised clinical suspicion for an alternative diagnosis, ultimately leading to the consideration of allergic bronchopulmonary aspergillosis (ABPA), where hypotension is an uncommon but notable finding.[4]

The comprehensive immunologic workup revealed significant eosinophilia, markedly elevated total IgE, and positive Aspergillus-specific antibodies. HRCT thorax findings of central bronchiectasis and mucus plugging are considered pathognomonic for ABPA, particularly when coupled with asthma and immunologic markers.

Misdiagnosis of ABPA as anaphylaxis is uncommon but plausible in cases where skin and respiratory symptoms coexist. Additionally, a history of occupational exposure to fungal spores, as reported by this patient, further supports the development of hypersensitivity to *Aspergillus fumigatus*.

Management of ABPA involves two primary goals: suppressing immune hyperreactivity and reducing fungal colonization. Corticosteroids remain the first-line therapy to attenuate inflammation, while antifungals like itraconazole help decrease the fungal burden. Serial monitoring of IgE levels and imaging is crucial to track disease progression and response to treatment.[4]

This case underscores the importance of integrating clinical features with immunologic and radiologic findings in diagnosing ABPA. Timely recognition and treatment can prevent complications such as irreversible bronchiectasis and chronic respiratory failure.

Conclusion

Allergic bronchopulmonary aspergillosis remains an underrecognized yet significant cause of chronic pulmonary symptoms in asthmatic and atopic individuals. This case illustrates how ABPA can masquerade as anaphylaxis, leading to misdiagnosis and delays in appropriate therapy. Persistent symptoms despite standard anaphylaxis management, coupled with elevated IgE levels, eosinophilia, and specific antibody positivity, should prompt further investigation for ABPA.

Clinicians should maintain a high index of suspicion in patients with recurrent asthma exacerbations or unexplained allergic manifestations, especially when imaging shows features like central bronchiectasis. Early diagnosis and initiation of corticosteroids and antifungal agents can lead to substantial clinical

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improvement and prevent long-term pulmonary damage. This case highlights the need for increased awareness and multidisciplinary management in atypical presentations of ABPA.

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