A Case of Allergic Bronchopulmonary Aspergillosis (ABPA) in a Patient with a History of Cocaine Use and Tuberculosis

Noura Ayoubi  
*University of South Florida*, nayoubi@usf.edu

Samuel Jalali  
*University of South Florida*, samueljalali@usf.edu

Nikesh Kapadia  
*University of South Florida*, nrkapadi@usf.edu

Follow this and additional works at: [https://digitalcommons.usf.edu/intmed_facpub](https://digitalcommons.usf.edu/intmed_facpub)

Scholar Commons Citation  
[https://digitalcommons.usf.edu/intmed_facpub/128](https://digitalcommons.usf.edu/intmed_facpub/128)

This Article is brought to you for free and open access by the Internal Medicine at Digital Commons @ University of South Florida. It has been accepted for inclusion in Internal Medicine Faculty Publications by an authorized administrator of Digital Commons @ University of South Florida. For more information, please contact [scholarcommons@usf.edu](mailto:scholarcommons@usf.edu).
A Case of Allergic Bronchopulmonary Aspergillosis (ABPA) in a Patient with a History of Cocaine Use and Tuberculosis

Noura Ayoubi,1 Samuel Jalali,2 and Nikesh Kapadia3

1Morsani College of Medicine, University of South Florida, Tampa, FL, USA
2Department of Internal Medicine, University of South Florida, Tampa, FL, USA
3Department of Internal Medicine, University of South Florida, Tampa, FL, USA

Correspondence should be addressed to Noura Ayoubi; nayoubi@health.usf.edu

Received 17 May 2019; Revised 27 September 2019; Accepted 11 November 2019; Published 23 November 2019

Academic Editor: Stephen P. Peters

Aspergillosis refers to a spectrum of disorders that can occur due to colonization with the Aspergillus fungus. Aspergillus is unique in that it can result in either infectious or atopic manifestations. Types of aspergillosis diseases include allergic Aspergillus sinusitis, aspergilloma, invasive aspergillosis, fungal asthma, and allergic bronchopulmonary aspergillosis (ABPA) [1].

ABPA is an airway hypersensitivity reaction to the fungus that is almost exclusively seen in patients with cystic fibrosis or asthma [2, 3]. The estimated prevalence of ABPA among patients with asthma is approximately 1–2% [4]. The prevalence is slightly higher in patients with cystic fibrosis, estimating about 2–9% [5]. In rare cases, ABPA can be seen in lung transplant recipients or those with bronchiectasis, chronic granulomatous disease, or hyper-IgE syndrome [6].

1. Introduction

Aspergillosis refers to a spectrum of disorders that can occur due to colonization with the Aspergillus fungus. Spores are commonly airborne in the environment but only classically cause disease in immunocompromised patients. Aspergillus is unique in the fact that it can result in either infectious or atopic manifestations. Types of aspergillosis diseases include allergic Aspergillus sinusitis, aspergilloma, invasive aspergillosis, fungal asthma, and allergic bronchopulmonary aspergillosis (ABPA) [1].

ABPA is an airway hypersensitivity reaction to the fungus that is almost exclusively seen in patients with cystic fibrosis or asthma [2, 3]. The estimated prevalence of ABPA among patients with asthma is approximately 1–2% [4]. The prevalence is slightly higher in patients with cystic fibrosis, estimating about 2–9% [5]. In rare cases, ABPA can be seen in lung transplant recipients or those with bronchiectasis, chronic granulomatous disease, or hyper-IgE syndrome [6].

The International Society for Human and Animal Mycology (ISHAM) provides a proposed set of diagnostic criteria for ABPA (Table 1) [7]. These criteria are divided into three groupings: predisposing conditions, obligatory criteria, and other criteria. One of the following predisposing conditions must be present: asthma or cystic fibrosis. Both of the following obligatory criteria must be present: positive Aspergillus skin test or IgE levels against Aspergillus that are detectable and an elevated serum IgE concentration. At least 2 of the following “other criteria” must be present: precipitating Aspergillus serum antibodies, pulmonary opacities seen on chest radiograph, or a serum eosinophil count greater than 500 cells/microliter in patients who have not been on glucocorticoids.

Despite the requirement of one predisposing condition, ISHAM notes that a diagnosis of ABPA is still possible in patients without asthma or cystic fibrosis, albeit very rare [8]. The objective of this case report is to highlight a rare case of ABPA in a patient with a history of chronic cocaine use and tuberculosis and no history of asthma or cystic fibrosis.
2. Case Presentation

This patient is a 57-year-old male with a past medical history of tuberculosis and a significant social history of chronic cocaine use who presented to the emergency department due to progressively worsening dyspnea over the last three months. On physical exam, the patient had diffuse wheezing. He also endorsed a 20-pound weight loss over the last three months and increased urinary retention. The patient denied fevers, chills, recent travel, hemoptysis, and cigarette smoking.

During his hospital stay, the following labs were performed: Acid-fast bacilli smear, Mycobacterium polymerase chain reaction, Legionella antigen, urine drug screen, immunoglobulin E (IgE) levels, Aspergillus antigen, and neutrophilic cytoplasmic antibody. Acid-fast bacilli testing was negative, as well as mycobacterium polymerase chain reaction, Legionella antigen, and neutrophilic cytoplasmic antibody. Urine drug screen was positive for cocaine. Eosinophil count was elevated at 670 cells/μL (normal < 610 cells/μL). IgE level was also elevated at 2,378 kU/L (normal < 114 kU/L). Based on findings of elevated IgE levels, IgE against Aspergillus fumigatus was sought out and came back positive (0.56 kU/L).

Chest X-ray revealed hyperinflation, flattened diaphragm, and pleural thickening (Figure 1). Chest CT without contrast revealed some bronchial wall thickening and bronchiectasis in the bilateral lower lobes (Figure 2(a)). In the right upper lobe, there was also some bronchial wall thickening, bronchiectasis, and tree-in-bud nodularity (Figure 2(b)). A 1.0 x 2.2 x 1.8 cm solid nodule was visualized in the posterior medial right lung (Figure 2(c)). This was grossly stable in size from a chest CT 3 months prior.

The patient was first treated at an outside hospital for pneumonia with amoxicillin-clavulanate and a 7-day course of prednisone 10 mg at the first onset of his symptoms. He reported transient improvement with steroids. On presentation, prior to confirmation of ABPA, the patient was treated with 60 mg intravenous methylprednisolone BID and inhaled ipratropium bromide with albuterol sulfate. Once ABPA was confirmed, the patient was transitioned from methylprednisolone to a 4-day treatment course with 60 mg oral prednisone. The patient noted significant improvement prior to discharge.

Given the concern for malignancy due to significant weight loss and urinary retention, the prostate-specific antigen (PSA) was measured. This was elevated at 11.2 ng/ml (normal < 4.0 ng/ml). Urology was consulted, and outpatient follow-up with urology was recommended for a transrectal ultrasound- (TRUS-) guided biopsy.

3. Discussion

Aspergillosis refers to a spectrum of disorders that can occur due to colonization with a type of Aspergillus fungus, most commonly Aspergillus fumigatus. Spores are commonly airborne in the environment yet rarely cause disease in immunocompetent patients. On the other hand, immunocompromised patients are at risk of various presentations of disease due to inhalation of the fungus. Aspergillus is unique in the fact that it can result in either infectious or atopic manifestations. Types of aspergillosis infections include allergic Aspergillus sinusitis, aspergilloma, invasive aspergillosis, and allergic bronchopulmonary aspergillosis [1].

Allergic Aspergillus sinusitis (AAS) is a hypersensitivity reaction to Aspergillus that results in sinusitis. The reaction is similar to that of allergic bronchopulmonary aspergillosis; however, it occurs in the sinuses. Despite the similar mechanism of colonization, few cases report simultaneous presence of both diseases [9]. Patients at risk for developing AAS include those with a prior sensitization to the Aspergillus antigen who also have a past medical history of asthma or rhinosinusitis [10].
An aspergilloma is an intracavitary fungus ball that typically presents in previously colonized areas of the lung [11]. For this reason, many patients may have a history of colonization with *Mycobacterium tuberculosis*. [12]. Chest X-ray shows a cavitary lesion with a thickened wall. In most instances, this is an incidental finding, and patients remain asymptomatic. Rarely, patients can present with hemoptysis secondary to blood vessel invasion. *Aspergillus* sensitization has also been commonly reported in the setting of pulmonary tuberculosis cavitary disease [12]. Dhooria et al. reported that 16% of patients with a history of pulmonary tuberculosis had *Aspergillus* sensitization. Nevertheless, a diagnosis of tuberculosis is not a "predisposing condition" in the ISHAM criteria [12].

Invasive aspergillosis refers to systemic dissemination of the fungus. Normally, alveolar macrophages and epithelial cells work together to rid the lungs of infection before dissemination can occur [13]. In cases where this is inefficient, the fungi are able to latch onto blood vessels, invade the endothelium, and spread throughout the body. Risk factors for invasive aspergillosis include severe neutropenia, glucocorticoids given in high doses, AIDS, and immunosuppression [14]. Once the fungus becomes systemic, it can affect almost every organ, including the liver, kidneys, eyes, and brain [14].

Allergic bronchopulmonary aspergillosis (ABPA) is a hypersensitivity reaction to the *Aspergillus* fungus, similar to AAS. However, this reaction occurs in the airways [3]. ABPA is almost exclusively seen in patients with cystic fibrosis or asthma [2]. The estimated prevalence of ABPA among patients with asthma is approximately 1–2% [4]. The prevalence is slightly higher in patients with cystic fibrosis, estimating about 2–9% [5]. In rare cases, ABPA can be seen in lung transplant recipients or those with bronchiectasis, chronic granulomatous disease, or hyper-IgE syndrome [6].

Diagnosis of ABPA is often made using the ISHAM criteria (Table 1) [7]. This patient presented with both "obligatory criteria" (elevated serum IgE concentration and elevated IgE levels against *Aspergillus fumigatus*). He also met two of the "other criteria" (tree-in-bud nodularity and bronchiectasis and total eosinophil count > 500 cells/μL).
This case is unique in that the patient did not have any of the predisposing conditions. ISHAM notes that a diagnosis of ABPA can still be made despite not presenting with these conditions although this is rare.

ABPA is characterized by mucoid bronchial obstruction, resulting in wheezing and rhonchi. Given the background of hypersensitivity in this disease, patients typically have associated eosinophilia and eosinophilic pneumonia [15]. Despite the severity of Aspergillus colonization, the fungi do not invade bronchial walls. In most cases, the presence of fungal spores in the lungs results in the formation of IgG and IgA against the antigens. These are sufficient enough to clear the Aspergillus [16]. In patients with a history of atopy, additional IgE antibodies also form, manifesting as the hypersensitivity reaction of ABPA.

Similar to the pathogenesis of asthma, CD4+ helper T cells also play a role in the development of ABPA [17]. When exposed to fungus, helper T cells secrete interleukins (IL), which include IL-4, IL-5, and IL-13 [18]. In response to the interleukins, the body produces IgE and eosinophils, contributing even more so to the common finding of eosinophilia. Mucus plugs contain Charcot–Leyden crystals and eosinophils. Culture of these plugs may or may not grow the Aspergillus fungus [15].

Chronic airway hyperreactivity due to ABPA can result in the development of bronchiectasis. Chest radiographs often reveal thickened bronchial walls and atelectasis secondary to mucoid plugs [19]. Initial pulmonary function testing presents with an obstructive pattern of changes, including an increased residual volume (RV) and a decreased forced expiratory volume in one second (FEV1) [20]. Recommended treatment focuses on controlling inflammation and is usually a taper of systemic glucocorticoids over three to six months.

ABPA is rarely reported in patients without asthma or cystic fibrosis. This patient is unique in that he did not meet the ISHAM predisposing conditions usually required for diagnosis of ABPA. It is important to note that this is a single case of such association, which does not warrant enough evidence to claim that chronic cocaine use and previously treated tuberculosis increase risk for ABPA. Nevertheless, it is our hope to make healthcare providers aware of its potential presence in chronic cocaine users and patients with tuberculosis, both of which are not traditionally associated with this condition.

Disclosure

This case report was presented at the 2019 University of South Florida Health Research Day on February 22, 2019.

Conflicts of Interest

The authors have no conflicts of interest to declare.

References
