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Case Report

Resolution of allergic bronchopulmonary aspergillosis preceded by allergic fungal rhinosinusitis, without glucocorticoid therapy in an immunocompetent individual: a case report with narrative literature review

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ABSTRACT

Allergic bronchopulmonary aspergillosis (ABPA), seen predominantly in patients with uncontrolled bronchial asthma, cystic fibrosis, and immunocompromised patients, can uncommonly occur in an immunocompetent host. Rarely, an ABPA patient can have a preceding or concomitant history of its upper respiratory counterpart, allergic fungal rhinosinusitis (AFRS). The traditional mainstay of ABPA treatment is systemic glucocorticoids. We report a case of ABPA in a woman with a history of AFRS and recurrent atopic rhinitis, but with no previous definitive history of bronchial asthma or cystic fibrosis, that resolved without systemic glucocorticoid therapy. A 52-year-old female patient, a non-smoker, previously operated for AFRS, presented to the outpatient department with fever with chills, right-sided pleuritic type of chest pain, and cough with brown-black expectoration. The absolute eosinophil count was 1360. Serum baseline IgE was >2500. A chest X-ray revealed consolidation in the right middle zone. High-resolution computed tomography (HRCT) of the thorax revealed linear branching hyperdense content in a few of the dilated bronchi. Fiber-optic bronchoscopy confirmed thick mucus plugging at the lateral segment of the right middle lobe, pathognomonic of ABPA with high-attenuation mucus (ABPA-HAM). Oral Itraconazole monotherapy 200 mg twice daily for one day, followed by 100 mg twice daily for a total period of four months' duration, had no significant adverse events during treatment. C-reactive protein decreased significantly by 2 weeks, total serum IgE levels by 2 months; and repeat chest X-ray at 6 months showed complete resolution of pulmonary infiltrates. This case report corroborates recent revised 2024 ISHAM-ABPA working group guidelines advocating Itraconazole monotherapy alone for treating acute ABPA.

Keywords: Allergic bronchopulmonary aspergillosis, Allergic fungal rhinosinusitis, Glucocorticoid therapy, Antifungal monotherapy, Immunocompetent host

INTRODUCTION

Fungal pulmonary infections can be caused by endemic or opportunistic fungi. Endemic fungal infections can cause disease in otherwise healthy individuals especially in specific geographic regions. In contrast, opportunistic fungal infections typically cause disease in individuals with compromised immune systems, altered microbiota, or those who have disrupted integumentary barriers, but only rarely in the immunocompetent host.[1]

The most frequent pathogen associated with fungal pulmonary infections is the ubiquitous *Aspergillus* species (most commonly *A. fumigatus*), which can manifest, depending on the host-fungus interaction, in three clinical presentations: chronic pulmonary aspergillosis (CPA), invasive pulmonary aspergillosis (IPA), and allergic bronchopulmonary aspergillosis (ABPA).[2]

CPA is due to local lung invasion in patients with chronic pulmonary disease. IPA is a severe acute/subacute disease in immunocompromised or non-neutropenic critically ill patients, chronic obstructive pulmonary disease (COPD) or liver cirrhosis. ABPA, caused by hypersensitivity to *Aspergillus fumigatus*, is most frequently associated with severe, uncontrolled asthma or cystic fibrosis.[2] Rarely, ABPA is identified in the absence of asthma or cystic fibrosis, in which cases, COPD and post-tuberculous fibrocavitary disease may be predisposing conditions.[3]

However, other fungi (*Bipolaris, Curvularia* and *Schizophyllum* commune species) can cause a syndrome similar to ABPA, called allergic bronchopulmonary mycosis (ABPM), in the absence of underlying asthma or cystic fibrosis. Individuals with ABPA could also suffer from other allergic conditions such as atopic dermatitis, urticaria, allergic rhinitis and sinusitis.[4]

Systemic (oral) glucocorticoids have been the traditional mainstay of treatment for ABPA. We report a case of ABPA in a woman with a history of allergic fungal rhinosinusitis (AFRS) and recurrent atopic rhinitis, but with no previous definitive history of bronchial asthma or cystic fibrosis, that resolved without glucocorticoid therapy.

CASE REPORT

A 52-year-old female patient, non-smoker, an office employee residing in Ernakulam in Kerala state, South India presented in medical out-patient department with complaints of fever with chills and right sided pleuritic type of chest pain with occasional dry cough for a duration of about two weeks. She was admitted in medical ward for further evaluation and management. She subsequently developed cough with brown-black expectoration. Her medical history suggested recurrent episodes of allergic rhinitis from early childhood. She had undergone functional endoscopic sinus surgery bilaterally for AFRS around five years back.

She was evaluated in detail with routine blood investigations which was suggestive of an inflammatory syndrome with a total count of 12730, differential count of neutrophils 62 lymphocytes 23, eosinophils 11 and C-reactive protein (CRP) of 180. Absolute eosinophil count was 1360. Chest X-ray revealed right middle zone consolidation (Figure 1).

She was started initially on intravenous Meropenem and oral Clarithromycin in view of persistent respiratory symptoms.

On further evaluation with HRCT thorax she was found to have a wedge shaped consolidation without air bronchogram involving the right middle lobe with minimal collapse and adjacent ground glass opacity with interstitial thickening. It also showed evidence of fluid bronchogram with linear branching hyperdense content in few of the dilated bronchi with minimal central bronchiectatic changes noted in the anterior and posterior segment branch of right upper lobe with adjacent fibrotic changes. She also had minimal fibrotic changes in the left lower lobe (Figures 2 and 3).

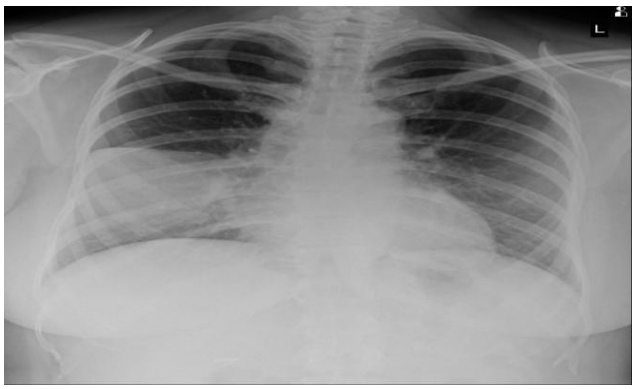

Figure 1: Chest X-ray showing consolidation of right middle and lower zones.

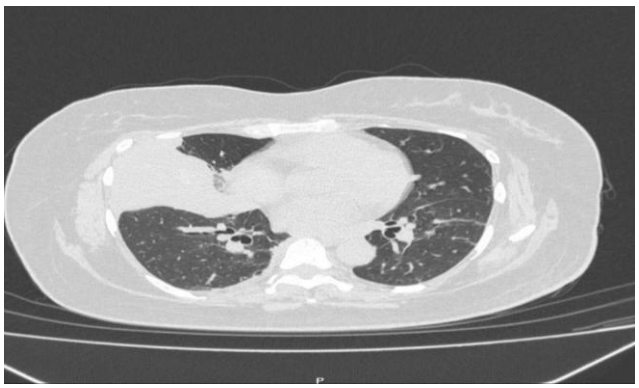

Figure 2: HRCT thorax showing a wedge shaped consolidation without air bronchogram involving the right middle lobe with minimal collapse and adjacent ground glass opacity. It also showed evidence of fluid bronchogram with linear branching hyperdense content in few of the dilated bronchi with minimal central bronchiectatic changes noted in the anterior and posterior segment branch of right upper lobe with adjacent fibrotic changes.

Fiber optic bronchoscopy was done which showed thick mucus plugging at lateral segment of right middle lobe (Figures 4a and b).

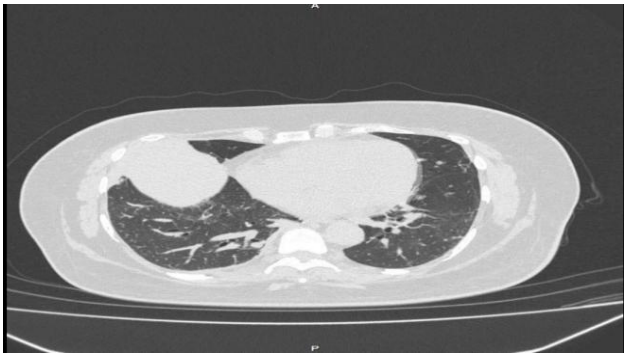

Figure 3: HRCT chest showing fluid bronchogram with linear branching hyperdense content in few of the dilated bronchi.

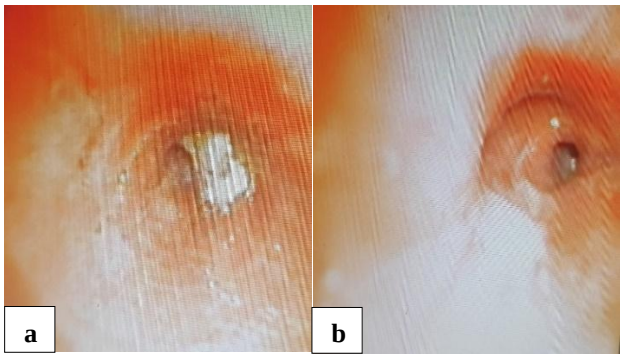

Figure 4 (a and b): Fiber optic bronchoscopy showing thick mucus plugging noted at the lateral segment of right middle lobe.

Serum baseline IgE was evaluated and was found to be >2500.

She was then started on oral Itraconazole 200 mg twice daily for one day followed by 100 mg twice daily for a total period of four months' duration with monitoring of liver function tests. Patient was followed up after two weeks of medication and was found to have a CRP of less than 10 with normal total counts. X ray showed beginning of resolution of pulmonary infiltrates which was suggestive of clinical improvement (Figure 5).

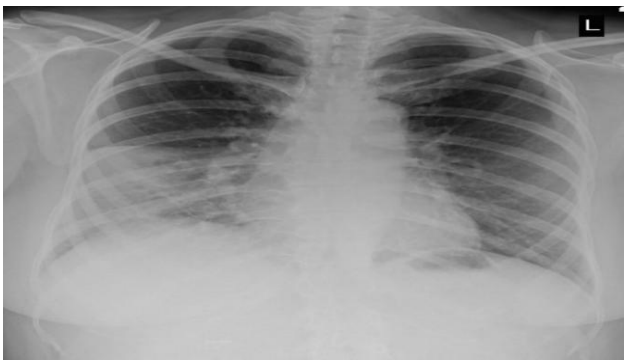

Figure 5: Chest X-ray after two weeks of initiation of antifungal therapy showing clearance of pulmonary infiltrates.

Total serum IgE levels decreased to 1875 and 970 after 1 and 2 months of treatment respectively. Repeat chest X-ray at 6 months showed complete resolution of the pulmonary infiltrates. No significant adverse events were reported during treatment.

DISCUSSION

ABPA, seen almost exclusively in patients with uncontrolled bronchial asthma, cystic fibrosis and immunocompromised patients, can also rarely occur in patients with bronchiectasis, chronic granulomatous disease, hyperimmunoglobulinemia E, and in lung transplant recipients.[5] It usually presents in patients in their third to fifth decade of life.

The normal human host eliminates A. fumigatus efficiently by innate and adaptive immune mechanisms. *Aspergillus* sensitization (AS) occurs in atopic individuals, defined by an immediate cutaneous hyperreactivity against *A. fumigatus* antigens or by elevated levels of serum *A. fumigatus*-specific IgE.[6] When not eliminated by the innate immune response, the typical adaptive response to *Aspergillus fumigatus*, skews from type 1 to type 2.

T helper type 2 (Th2) cells play an important role in the hypersensitivity reaction that occurs and manifests as Ig E production, eosinophilia and bronchiectasis which is relevant in the diagnosis of ABPA. Eosinophils are key mediators of inflammation in ABPA, releasing galectin-10 and forming Charcot–Leyden crystals. They undergo cell death, forming histone-rich extracellular traps and increasing mucus plug viscosity. Th2 responses lead to IL-4, IL-5, and IL-13 secretion, causing mast cell degranulation, eosinophilic inflammation and mucus hypersecretion.[7] Intense inflammatory reaction causes accumulation of eosinophilic mucin containing the fungal hyphae in the airways.

The pooled prevalence of ABPA is about 13% and 9% in asthma and cystic fibrosis respectively, with the prevalence of ABPA complicating asthma in the last decade reported to be higher in India compared to other countries.[8-10] ABPA without asthma has been shown to be a distinct subset (7%) of ABPA, with a better lung function and fewer exacerbations despite underlying bronchiectasis in the majority (97%); these features contributing to its initial misdiagnosis as bronchogenic carcinoma, pulmonary tuberculosis and others.[10,11]

Patients of ABPA usually presents with symptoms of cough with sputum- either blood stained or sputum with mucus plugs, pleuritic chest pain and non-specific complaints such as generalized tiredness and low-grade fever, as in this case. Patient may also present with symptoms like dyspnea, recurrent episodes of wheezing and some patients present with an asymptomatic pulmonary consolidation.

ABPA may occur with allergic fungal sinusitis having symptoms of chronic sinusitis, as in our case. AFRS is a specific subtype of chronic rhino sinusitis (CRS) defined as an intense, localized allergic/eosinophilic inflammatory sinus disease. The pathophysiology of AFRS is most consistent with chronic, intense Th2 allergic inflammation directed against colonizing fungi. [12] AFRS has been proposed to be the upper respiratory tract equivalent of ABPA. However, despite sharing similar immunopathogenetic features, patients with history of both ABPA and AFRS are uncommon. Some ABPA subjects can have a history of AFRS, but their concomitant occurrence is quite rare, with about 20 cases reported in the literature, of which 14 were from India.[13] ABPA with AFRS masquerading as granulomatous small–vessel vasculitis is extremely rare, with only a single case report –from India.[14]

AFRS is diagnosed by the demonstration of all of the five criteria proposed by Bent and Kuhn, *viz.* type I hypersensitivity; nasal polyposis; characteristic CT findings; eosinophilic mucin without invasion; and positive fungal stain of sinus contents removed during surgery.[15] AFRS usually requires endoscopic surgery and postoperative medical treatment including steroids, antifungals and immunotherapy.[16]

The diagnosis of ABPA is based on clinical, radiological and immunological findings. An elevated total serum Ig E level >1000 IU/ml and peripheral blood eosinophilia >500 are useful investigations in the diagnosis of this condition. In our case, the total serum Ig E was >2500 IU/ml and absolute eosinophil count was 1496. Other blood investigations include elevated specific serum Ig E and Ig G to *A. fumigatus*. *Aspergillus* skin testing revealing positive type 1 hypersensitivity reaction is typical of ABPA and represents the presence of Ig E antibodies specific to *A. fumigatus*, as in our case.

As per the recent 2024 published Revised International Society for Human and Animal Mycology (ISHAM)-ABPA working group consensus criteria for diagnosing ABPA, our case fulfilled the 2 essential criteria [serum total IgE ≥500 IU/ml and a positive type 1 skin test (instead of *A. fumigatus*-specific IgE ≥0.35 kUA/l)] and 2 other components [blood eosinophil count ≥500 cells/µb and Thin-section chest CT consistent with ABPA].[17]

HRCT of the chest is the initial investigation of choice in ABPA. ABPA was earlier (2011) classified as ABPA-S (mild) when HRCT is normal as in about one third cases; ABPA-CB (moderate) when HRCT shows central bronchiectasis; and ABPA-CB-HAM (severe) when HRCT shows high-attenuation mucus (HAM) pathognomonic of ABPA as in about one fifth cases.[18,19] The sensitivity and specificity of HAM are 35% and 100%, respectively.[20]

The 2024 published Revised ISHAM-ABPA working group has reclassified ABPA into five -Serological ABPA (ABPA-S), ABPA with bronchiectasis (ABPA-B), ABPA with mucus plugging (ABPA-MP), ABPA with high-attenuation mucus (ABPA-HAM), ABPA with chronic pleuropulmonary fibrosis (ABPA-CPF).[17] The hyperdensity of the HAM plugs has been attributed to the presence of calcium oxalate crystals.[21] The distinctive, if not pathognomonic, finding of linear branching hyperdense content in few of the dilated bronchi with minimal central bronchiectatic changes corresponding to ABPA-CB-HAM/ABPA-HAM was noted in our case. HAM on HRCT is pathognomonic of ABPA and confirms ABPA diagnosis even if all other criteria are not fulfilled.[7]

Further, on bronchoscopy, the finding of hyphae filled mucus plugging, considered pathognomonic for ABPA, was observed in our case.[22] Of the available 5 major diagnostic criteria for ABPA, the modified ISHAM criteria (2021) is most reliable and validated. The other recently proposed criteria by Asano and colleagues (2021) which diagnoses probable and definite ABPA, has the advantage of being useful in diagnosing ABPA sans asthma. However, the disadvantage is the need for bronchoscopy in the algorithm. [8]

There are five stages of ABPA: acute, remission, exacerbation, corticosteroid-dependent asthma, and fibrotic lung disease. Treatment is based on the disease stage.[23]

The main aim treatment in these patients is to control the symptoms of acute inflammation and to prevent progressive lung injury.

The main modalities of treatment include corticosteroids and antifungal therapy. The steroids help in relieving symptoms and decrease airflow obstruction, decrease peripheral eosinophils and serum Ig E. For new ABPA infiltrates, prednisone can be started at 0.5 mg/kg/day for 1-2 weeks, then on alternate days for 6-8 weeks followed by tapering by 5-10 mg every 2 weeks.[24]

However, in our case, glucocorticoids were not started while Itraconazole was started, continued and monitored for 4 months. Despite this, it is noteworthy that the patient showed significant improvement without glucocorticoids as mentioned earlier. While oral glucocorticoids are a primary treatment for ABPA, spontaneous clearance of consolidation in ABPA is known to occur, particularly in mild cases.[25] Spontaneous resolution of ABPA can also occur rarely when presenting as cough variant asthma. [26]

Itraconazole is the commonly used antifungal agent in treatment of ABPA which acts by reducing fungal load and thus reduces inflammatory activity. Itraconazole 200 mg for 16 weeks can be given reducing Ig E levels, improving pulmonary function and resolving pulmonary infiltrates. Monitoring of liver function tests should also be done in patients with itraconazole therapy due to its interference with hepatic metabolism. Itraconazole, without oral glucocorticoids, due to its significant steroid sparing

effect, has been documented to induce remission especially for steroid-dependent or recurrent cases of ABPA.[22]

The revised ISHAM-ABPA working group clinical practice guidelines (2024) recommend oral prednisolone or itraconazole monotherapy for treating acute ABPA (newly diagnosed or exacerbation). Prednisolone and itraconazole combination is recommended only for treating recurrent ABPA exacerbations; or as a short course of glucocorticoids (<2 weeks) to be used as initial therapy along with oral itraconazole.[17]

Voriconazole has been shown to have distinct advantages to itraconazole, such as improved gastrointestinal tolerance and bioavailability.[3] but has photosensitivity and poorer patient tolerance. Also, prednisolone decreases the plasma concentration of voriconazole in a dose-dependent fashion.[17] Hence, oral voriconazole, posaconazole and isavuconazole are not recommended as first-line agents for treating acute ABPA, but only if itraconazole therapy fails or steroids are contraindicated. Biological agents are also not recommended as first line agents due to lack of data.[17]

Antibiotic therapy for secondary bacterial infection; and biological agents such as anti Ig-E antibody (Omalizumab), interleukin (IL)-5 agents (mepolizumab, benralizumab) or dupilumab (anti-IL-4 subunit antibody) for uncontrolled asthma/cystic fibrosis can also be used in treatment. For ABPA in remission, Serum total IgE levels are recommended to be monitored every three to six months since, an increase may be a pointer to lung opacities on diagnostic imaging and peripheral eosinophilia. Pulmonary function tests should be performed to assess changes in symptoms and annually on follow-up.[3]

Chest CT is the recommended diagnostic investigation of choice at baseline for diagnosis, assessment and prognostication for ABPA especially with bronchiectasis.[17] Although magnetic resonance imaging is a radiation-free alternative, the resolution does not extend beyond the third or fourth generation bronchi/sub-segmental level/4th order, making it less ideal for ABPA with bronchiectasis, but probably a better choice for ABPA with cystic fibrosis.[27,28] Chest radiography is appropriate and sufficient for follow-up.[17]

The prognosis of ABPA is distinctly better if central bronchiectasis is absent, at diagnosis. Lung function is preserved in the majority with ABPA, despite occasional exacerbations. Unpredictably however, progression to pulmonary fibrosis is relentless, despite few symptoms and exacerbations, in the rare minority.[3]

CONCLUSION

This is a case report of ABPA in a woman with a history of AFRS and recurrent atopic rhinitis, but no definitive history of bronchial asthma, cystic fibrosis or other chronic pulmonary comorbidities, which resolved with antifungals and antibiotics only. This case, while highlighting the need for early diagnosis and treatment to prevent long term complications due to irreversible changes that can occur with untreated cases, also corroborates recent revised 2024 ISHAM-ABPA working group guidelines advocating itraconazole monotherapy alone for treating acute ABPA.

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