Case Report

Pulmonary aspergillosis in a renal transplant recipient

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Abstract

Aspergilloma, one of the spectrum of pulmonary aspergillosis, is the most common and best-recognised form of pulmonary involvement by Aspergillus species. It usually develops in a pre-existing cavity in the lung secondary to tuberculosis. Here, we present a case of live non related renal transplant recipient with aspergilloma with bronchiectasis with tuberculosis who underwent left upper lobectomy with antitubercular treatment and voriconazole with complete resolution.

Keywords: Renal transplant, aspergilloma, tuberculosis

Introduction

Aspergilloma, one of the spectrum of pulmonary aspergillosis, is the most common and best-recognised form of pulmonary involvement by Aspergillus species. It usually develops in a pre-existing cavity in the lung secondary to tuberculosis. One of the risk factor is immunosuppressive condition such as renal transplantation. Here, we present a case of live non related renal transplant recipient with aspergilloma with bronchiectasis with tuberculosis.

Case report

A 52-year-old male, a renal transplant recipient presented in the outpatient department with history of fever for 5 days and loose stool for 2 days. He had undergone live non related renal transplant (LNRRTx) for end stage renal disease secondary to diabetes mellitus 6 months back. His wife was the donor with HLA mismatch of 5/6.The patient had received induction immunosuppression with antithymocyte globulin. He was under maintenance immunosuppression of tacrolimus, mycophenolate mofetil, and corticosteroid. He also received trimethoprim/sulfamethoxazole for 1 year and valganciclovir for 3 month as prophylaxis.

His fever was high grade, recorded upto 102^0F for which he took antibiotic, amoxicillin-clavulanic acid for 5 days. After which he developed loose stools about 4-5 episodes per day associated with blood and mucus for 2 days. He did not give history of cough, hemoptysis or chest pain. There was also no history of weight loss or loss of appetite.On examination, he was febrile with temperature of 100^0F with pulse rate 110/min and blood pressure of 100/60 mm hg. On ausculation, his chest revealed course crepitations in his left suprascapular area. All other systemic examination findings were within normal limits. So, with a provisional diagnosis of live non related renal transplant with Left sided pneumonia and Acute gastroenteritis with Diabetes mellitus, we admitted the case. He was empirically started on ceftriaxone and metronidazole.

The investigation reports at our hospital revealed hemoglobin 10.7 gm%, leukocyte count 2300/cmm with differential count of 56% neutrophil and 37% lymphocytes and platelet count 2,55,000/cmm. His serum creatinine 1 mg/dl. Stool routine and microscopic examination was normal. However, his chest X-ray revealed ill defined non homogenous opacity in the left middle zone, fibrotic changes with traction bronchiectasis in right upper zone and calcified granuloma in bilateral upper zone. High resolution CT Chest was done, which showed patchy consolidation with surrounding ground glass opacities and air bronchogram in anterior segment of left upper lobe, likely due infective pathology with suspicious invasive semi solid nodule with ground glass opacities, differential diagnosis of Invasive Aspergillosis. His sputum culture did not reveal any organism and was negative for any acid fast bacilli. His sputum GeneXpert MTB/RIF test was also negative

and his sputum KOH stain showed no fungal hyphae. Polymerase chain reaction (PCR) assay of the serum for cytomegalovirus DNA was negative.

After consultation with Cardiovascular and thoracic surgeon, left upper lobe lobectomy was done. The lung biopsy showed Aspergilloma with bronchiectasis and emphysema with a separate nodule showing necrotizing granulomatous lesion consistent of tuberculosis.

The patient was started on antitubercular treatment consisting of isoniazid, rifampicin, pyrazinamide, ethambutol and voriconazole for a total of 1-year duration. On follow up, the patient was asymptomatic with a serum creatinine of 0.9 mg/dl.

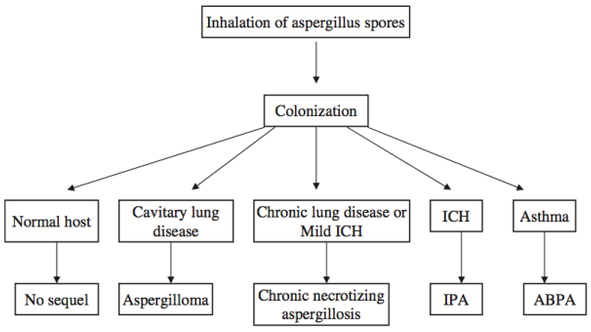

Figure 1. The clinical spectrum of conditions resulting from inhalation of aspergillus spores. ICH, immunocompromised host; IPA, invasive pulmonary aspergillosis; ABPA, allergic bronchopulmonary aspergillosis. Chest 2002;121:1988-1999.

Discussion

Aspergilloma is the most common and best-recognised form of pulmonary involvement by Aspergillus species. It usually develops in a pre-existing cavity in the lung. The aspergilloma (fungus ball) is composed of fungal hyphae, inflammatory cells, fibrin, mucus, and tissue debris. The most common species of Aspergillus recovered from such lesions is A. fumigatus. However, other fungi, such as Zygomycetes and Fusarium, may cause the formation of a fungal ball.

Many cavitary lung diseases are complicated by aspergilloma, including tuberculosis, sarcoidosis, bronchiectasis, ankylosing spondylitis, neoplasm[1,2]. Of these, tuberculosis is the most common[3]. In a study of 544 patients with pulmonary cavities secondary to tuberculosis, 11% had radiological evidence of aspergilloma[4].

The fungus ball may move within the cavity, but it does not usually invade the surrounding lung parenchyma or blood vessels, although exceptions have been noted [5,6]. The lesion remains stable in the majority of cases, but it may decrease in size or resolve spontaneously without treatment in 10% of cases[7]. Rarely, the aspergilloma may increase in size.

Most patients with aspergilloma are asymptomatic. When symptoms are present, most patients experience mild haemoptysis, but severe and life-threatening haemoptysis may occur, particularly in patients with underlying tuberculosis[8]. The mortality rate from haemoptysis related to aspergilloma ranges between 2–14%[9,10,11,12,13]. The source of bleeding is usually the bronchial blood vessels, and it may be caused by local invasion of blood vessels lining the cavity, endotoxins released from the fungus, or mechanical irritation of the exposed vasculature inside the cavity by the moving fungus ball[5,14,15]. Less commonly, patients may develop cough, dyspnoea that is probably more related to the underlying lung disease and fever that could be secondary to the underlying disease or bacterial superinfection.

Risk factors for poor prognosis of aspergilloma include the severity of the underlying lung disease, increase in size or number of lesions as seen on chest radiographs, immunosuppression (including corticosteroid therapy, renal transplantation and HIV infection), increasing Aspergillus-specific IgG titres, recurrent large volume haemoptysis and underlying sarcoidosis[16].

The diagnosis of pulmonary aspergilloma is usually based on clinical and radiographic features along with serological or microbiological evidence of Aspergillus spp. Chest radiography may show presence of a mass in a pre-existing cavity. Aspergilloma appears as an upper-lobe, mobile, intracavitary mass with an air crescent in the periphery[17]. Chest CT scan may be necessary to visualise aspergilloma that is not apparent on chest radiograph[18] (figure 2). These radiological appearances may be seen in other conditions such as neoplasm, abscess, hydatid cyst and granulomatosis with polyangiitis (Wegener's granulomatosis). Aspergilloma may also coexist with any of the above-mentioned conditions[19,20]. Sputum cultures for Aspergillus spp are positive only in 50% of cases[21]. Serum IgG antibodies to Aspergillus are positive in most cases but may be negative in patients on corticosteroid therapy[6].

Treatment is considered only when patients become symptomatic, usually with haemoptysis. There is no consensus on the best treatment approach. Inhaled,

intracavitary and endobronchial instillations of antifungal agents have been tried and reported in small numbers of patients, but without consistent success[12,22,23]. CT-guided percutaneous administration of amphotericin B can be effective for aspergilloma, especially in patients with massive haemoptysis, and can lead to resolution within few days[24,25]. The role of intravenous amphotericin B is uncertain; small studies failed to show a benefit[26].

Itraconazole may be useful in the management of selected patients with aspergilloma because it has a high tissue penetration. Oral itraconazole has been used with radiographic and symptomatic improvement in one-half to two-thirds of patients. Occasionally patients have a complete response[27,28,29]. The major limitation of itraconazole is that it works slowly and would not be useful in cases of life-threatening haemoptysis[30]. The role of newer antifungal azoles such as voriconazole in the treatment of aspergilloma has yet to be determined.

Surgical resection of the cavity and removal of the fungus ball is usually indicated in patients with recurrent haemoptysis, if their pulmonary function is sufficient to allow surgery. It is associated with relatively high mortality rates, ranging from 7– 23% [31,32,33,34,9,10,15]. The most common causes of death post-operatively are severe underlying lung disease, pneumonia, acute myocardial infarction, and Invasive pulmonary Aspergillosis[34,12].

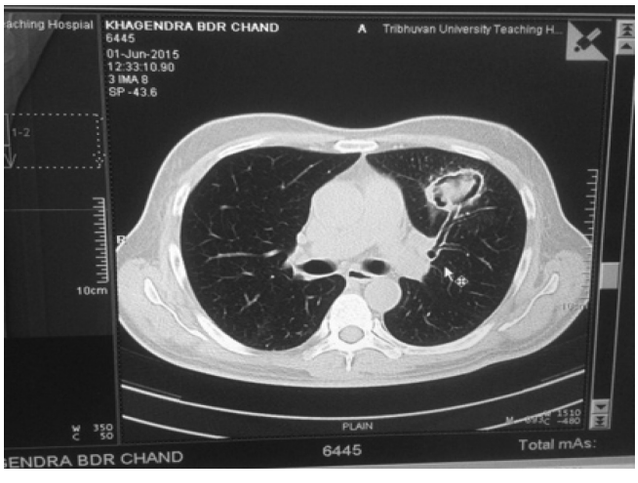

Figure 2. CT chest showing thin walled cavity with intracavitary body in anterior segment of left upper lobe, likely Aspergilloma.

Conclusion

In this case report, we present a case of live non related renal transplant recipient with aspergilloma with bronchiectasis with tuberculosis who underwent left upper lobectomy with antitubercular treatment and voriconazole with complete resolution.

Conflict of interests: None Declared

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