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**Case Report**

# A case report on *Aspergillus fumigatus* causing pulmonary aspergillosis in an immunocompromised patient presenting as chronic obstructive pulmonary disease and pulmonary tuberculosis

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## ABSTRACT

Pulmonary aspergillosis (PA) is a complicated fungal infection caused by aspergillus species, predominantly affecting immunocompromised individuals. This case highlights the diagnostic complexities as it often mimics other respiratory diseases like tuberculosis (TB) and chronic obstructive pulmonary disease (COPD) and in a patient with advanced HIV. Case presentation: A 48-year-old male with HIV (on third-line antiretroviral therapy, CD4 count 150 cells/µL) presented with a one-week history of cough, wheezing, shortness of breath, and fatigue. Initial management for COPD and bacterial infection provided minimal relief. Extensive diagnostic workup, revealed elevated Aspergillus-specific IgG antibodies (122 U/ml) and IgE levels (67.60 KUA/l). High-resolution computed tomography (HRCT) of the chest revealed bilateral bronchiectasis, suggestive of fungal infection, confirming PA. Conclusion: This case underscores the importance of considering opportunistic fungal infections like PA in immunocompromised patients with non-resolving respiratory symptoms, even when classic radiological signs are absent or overlap with other pulmonary conditions.

**Keywords:** Pulmonary aspergillosis, *Aspergillus fumigatus*, HIV, Voriconazole

## INTRODUCTION

*Aspergillus fumigatus* is a thermotolerant fungus with worldwide distribution and frequently isolated from the outer environments i.e. soil, plant matter, food and debris and inner spaces such as hospitals.[1] PA is a spectrum of mycotic diseases caused by aspergillosis species which are ubiquitous throughout the world called *Aspergillus fumigatus* for which respiratory tract is the major portal of entry for infections.[1,2] A spectrum of clinical syndromes, notably invasive aspergillosis, Aspergillosis mycetoma (fungal ball), and allergic broncho PA (ABPA), are associated with aspergillus related pulmonary conditions.[3] Invasive PA (IPA) is a potentially fatal disease that can occur in people with severe immune deficiency and chronic respiratory disorders, chronic obstructive pulmonary disease (COPD). however, not only affect those with significant immunological deficiencies.[2] It is one of the fungal infections which can lead to secondary pulmonary infection in an immunocompromised patient and there are some cases have also been documented in non-immunocompromised individuals associated with severe pulmonary complications, for instance or such as severe influenza, pneumonia, TB, COVID-19, allergic asthma and COPD.[2,4] ABPA is a hypersensitivity reaction in the lungs caused by *Aspergillus fumigatus* predominantly seen in individuals with asthma or cystic fibrosis.[6] CD4 cells are typically low and other predisposal factors present concurrently in HIV positive patients with PA. Although in patients with HIV /AIDS,

there is a notable increasing incidence of PA.[6] Individuals considered most commonly at established risk groups includes those with Steroid use, pulmonary diseases, COPD, Hemodialysis, long term use of steroid, prolonged neutropenia as a result of chemotherapy and lung transplant.[7] Herein, in the presence of traditional or typical factors, we depict the case of a middle-aged man who was discovered to have comprehensive PA.

## CASER REPORT

A 48-year-old retroviral positive male patient presented to the hospital with the complaints of cough with expectoration, wheezing, general weakness, shortness of breath, loss of appetite, fatigue, swelling in foot, unable to walk in a week prior to admission (7 days). He is on anti-retroviral therapy for the last 22 years and presently on III$^{-line}$ ART (Darunavir 600 mg 1-0-1, Ritonavir 100 mg 1-0-1, Dolutegravir 50 mg 1-0-1). His social history showed not a smoker and he was reformed alcoholic. Nevertheless, his symptoms worsened and prescribed with inj. Dexamethasone 8 mg stat and 4 mg 1-1-1, inj. Furosemide 1-1-0, inj. cefuroxime and sulbactam 1.5 gm 1-0-1, tab. Oseltamivir 75 mg 1-0-1 to relieve symptoms. His medical history with indistinguishable complaints 7 years and 2 months ago, for which he received antitubercular therapy for duration of six months and was hospitalized multiple times due to systemic recurring symptoms. The patient was diagnosed with COPD and lower respiratory tract infection as well as treated with Inj. Dexamethasone 4mg, Inj. Cefuroxime and Sulbactam 1.5 gm, Furosemide, Tab. Trimethoprim and sulphamethoxazole, neb. Acetylcysteine prior to 30 days before admission and furthermore, he also prescribed with Inj. Inactivated Influenza vaccine-4 0.5 ml, Inj. Pneumococcal 13-Valent vaccine, Inj. pneumococcal polysaccharide vaccine IP 0.5ml for pneumonia and COPD. He was also a documented case of emphysema, smear negative pulmonary TB, Henoch-Schoenlein purpura, bronchiectasis was confirmed by CT thorax which showed bronchiectatic changes with wall thickening and cystic dilation in the segments of the right upper, middle, both lower lobes are described and enlarged mediastinal lymph nodes, HRCT was done to detect fibrosis, community acquired pneumonia, lower respiratory tract infection and COPD were confirmed by chest x ray, anterior ischemia (Normal sinus rhythm with T wave abnormality), WAARI and osteoporosis, Asthma, dengue, glaucoma, Koch's disease in the last 10 years and he also has undergone cataract surgery of left eye.

On examination, he was afebrile and found to have bilateral wheezing, Vital parameters were found to be 120/80 mm/Hg of blood pressure, pulse rate 86 bpm, respiratory rate 20/min, temperature 97.6ºF, oxygen saturation 100% and laboratory investigation showed hemoglobin 11 g/dl, C-reactive protein 12.3 mg/l, electrolytes were normal, CD4 count 150 cells, viral load 220000 copies/ml. However, further diagnostic evaluation showed, sputum CBNAAT was negative or non-reactive for TB, sputum nocardia was not detected, gram stain, bacterial culture sensitivity, fungal smear, gene expert, AFB culture sensitivity all were negative, HRCT chest scan showed bilateral extensive bronchiectasis especially in right middle and bilateral lower lobes with few centrilobular nodules in right upper lobe (Figure 1), chest X-ray indicated bronchiectasis cytopathology revealed lung capacity lesion (smear show few scattered inflammatory cells in hemorrhagic background, no typical cells seen), USG guided FNAC was done and there is no evidence of pneumothorax and he was further evaluated for PA and his immunoglobulin IgE, serum (FEIA) level was raised 67.60 KUA/l (Normal range<64.00 KUA/l), Aspergillus antibodies IgG, serum (CLIA) results showed positive (122 U/ml) (<40-Negative, ≥40- positive) indicates the presence of aspergillus-specific IgG antibodies in the blood, this indicate the exposure to Aspergillus fungus, allergy individual marker aspergillus was negative and his chest CT showed right middle lobe thick walled cavity with underlying bronchiectasis, likely fungal, aspergillosis (Figure 2).

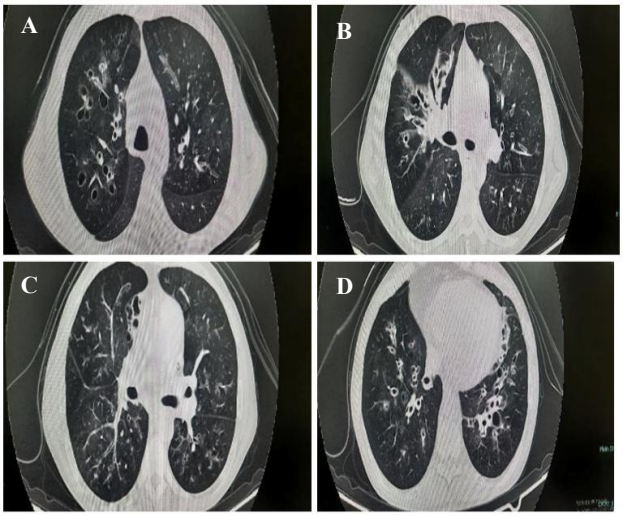


**Figure 1 (A-D): HRCT scan of the chest scan showing bilateral extensive bronchiectasis especially in right middle and bilateral lower lobes with few centrilobular nodules in right upper lobe.**

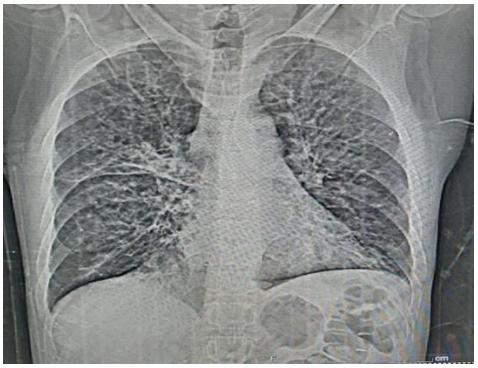

**Figure 2: Chest CT showed right middle lobe thick-walled cavity with underlying bronchiectasis, likely fungal, aspergillosis.**

Initially, the patient was suspected to have COPD and was it to have PA. The patient was treated with antifungal agents, voriconazole, 200 mg 1-0-1 for one month, followed by itraconazole (sustained-release), 130 mg 1-0-1 for 2 months. The patient was followed up, and currently patient is currently asymptomatic.

## DISCUSSION

Several previous research studies depicted that even patients who are immunocompetent individuals might contract PA when exposed to a host factor or high levels of aspergillus. An ideal marker to detect or diagnose PA is galactomannan antigen (GM) however it was not performed in this case and in addition, other criteria include Aspergillus antibodies IgG, serum (CLIA), immunoglobulin IgE, serum (FEIA), chest CT, HRCT, serum 1 to 3 β-D-Glucan (BDG), Lung biopsy, polymerase chain reaction (PCR) can be performed if necessary.[2] The diagnosis of PA is complex as it relies on the presence of clinical, radiological and microbiological criteria, which differ according to the type of PA (IPA or CPA) and the type of patient population. Managing PA poses significant challenges attributed to the increasing incidence of the limited number of treatment alternatives, concomitant medication effects, adverse effects and the emergence of antifungal resistance.[5] Multiple diagnostic criteria, procedure and prior screening should be implemented according to or based on patients' condition for early recognition or to rule out PA and also to avoid complications associated with mortality. Triazoles (itraconazole, voriconazole, posaconazole, isavuconazole) remains gold standard to treat PA followed by polyenes (liposomal amphotericin B) and Echinocandins (caspofungin, micafungin, anidulafungin). According to earlier clinical studies, voriconazole has increased survival and tolerability rate in patients with PA.[8] Additional diagnostic procedure includes bronchoscopy and open-lung biopsy. The diagnosis of PA remains complex. Timely identification of PA in severely immunocompromised individuals is obscure and in patients with risk factors require a high level of clinical suspicion. Itraconazole is metabolised by the cytochrome P450 (CYP) enzyme in the liver and therefore has potential for drug interactions.[9] It will interfere with anti-retroviral drug therapy, hence, closely monitor CD4 cells count and viral load.[10]

## CONCLUSION

These study findings emphasize the importance of considering opportunistic infections in immunocompromised patients, which can mimic other diseases, such as TB and COPD. It is crucial to check for rare infections, such as PA, in patients presenting with non-resolving respiratory symptoms or nonspecific symptoms, even in the absence of classical radiological or microbiological findings. Appropriate antifungal therapy remains critical to prevent further complications, morbidity, and improve prognosis in patients with complex medical histories. This case also illustrates the diagnostic challenge of PA in immunocompromised individuals, particularly those with concurrent pulmonary disorders like COPD and prior TB.

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