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Case Report

Non-resolving pneumonia in an elderly diabetic: Unmasking pulmonary mucormycosis

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Abstract

Mucormycosis is a rare but life-threatening fungal infection caused by fungi of the order Mucorales, predominantly affecting immunocompromised individuals. Pulmonary mucormycosis (PM), the second most common form, is often misdiagnosed due to its nonspecific clinical presentation, which includes fever, cough, dyspnea, and haemoptysis. Early diagnosis and treatment are critical to improving outcomes in this highly invasive infection.

We report the case of a 71-year-old male with uncontrolled diabetes mellitus, who presented with non-resolving pneumonia. Despite empirical antibiotic therapy, his symptoms persisted, and imaging revealed a cavitary lesion in the left upper lobe. Bronchoscopy with bronchoalveolar lavage and biopsy confirmed the diagnosis of pulmonary mucormycosis caused by Rhizopus species. The patient was treated with intravenous liposomal amphotericin B, followed by oral posaconazole. Glycaemic control was optimized, and surgical intervention was planned but deferred due to clinical improvement.

This case highlights the importance of considering mucormycosis in non-resolving pneumonia, especially in patients with diabetes and other risk factors. It underscores the need for early diagnostic evaluation, prompt initiation of antifungal therapy, and multidisciplinary management to achieve favourable outcomes in this often-fatal condition.

Keywords: Pulmonary mucormycosis, Uncontrolled diabetes mellitus, Non-resolving pneumonia, Amphotericin-B, Posaconazole

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1. Introduction

Mucormycosis is a rare but serious fungal infection caused by fungi of the order Mucorales, commonly affecting immunocompromised individuals.[1] Pulmonary mucormycosis (PM), the second most common form after rhino-cerebral mucormycosis, occurs via inhalation of fungal spores, leading to angioinvasion, tissue necrosis, and potential dissemination.[2]

Key risk factors include uncontrolled diabetes mellitus, hematological malignancies, and prolonged immunosuppression. Clinical presentation is nonspecific, with symptoms like fever, cough, dyspnea, and haemoptysis, often mimicking other pulmonary infections. Diagnosis requires high clinical suspicion, imaging and microbiological confirmation.

Management includes early initiation of liposomal amphotericin B, surgical debridement when necessary, and control of underlying conditions. This case underscores the importance of prompt recognition and targeted treatment of pulmonary mucormycosis in non-resolving pneumonia, particularly in patients with diabetes and other comorbidities.

2. Case Presentation

A 71-year-old male, a known case of Parkinson's disease, diabetes mellitus (DM), and hypertension (HTN), presented to Pulmonary Medicine OPD as a referred case from a local hospital. He had history of fever of one week duration. Fever was of low-grade, intermittent type. There was associated cough with mucoid expectoration, breathlessness, and generalized tiredness. Patient was initially admitted and treated in the local hospital with empirical intravenous antibiotics (ceftriaxone and clarithromycin). Initial chest X-

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ray revealed non-homogenous opacity in left upper zone (**Figure 1**A). Despite one week of antibiotic therapy, the patient showed no significant clinical improvement. Sputum for acid-fast bacilli (AFB) was negative, and sputum culture revealed normal flora. Sputum KOH mount was also negative. A repeat chest X-ray revealed a cavitary lesion in the left upper zone (**Figure 1**B). Hence the patient was referred to our centre for further expert evaluation and management.

On general examination, there was no pallor, icterus, cyanosis, clubbing, or lymphadenopathy, and his vitals were stable, except for mild desaturation (SpO_2 94%). Auscultation revealed crepitations in left side. The patient was admitted under the Pulmonology department for further management. Initial laboratory investigations showed leucocytosis, elevated erythrocyte sedimentation rate (ESR), elevated blood sugar levels, and a high glycosylated haemoglobin (HbA1c), with other parameters within normal limits.

To further evaluate the condition, a contrast-enhanced computed tomography (CECT) of the chest was performed, revealing a mildly enhancing thick-walled cavitary lesion with surrounding ground-glass opacity in the apico-posterior segment of the left upper lobe (**Figure 2**A and B). The antibiotics were escalated (to piperacillin-tazobactam). Given the lack of improvement and inconclusive sputum studies, the patient underwent bronchoscopy. Bronchoscopy revealed inflamed mucosa with nodular lesions in the left upper lobe bronchus. Bronchoalveolar lavage (BAL) and biopsy samples were obtained for further analysis. Endocrinology consultation was taken for proper control for blood sugar levels.

Lactophenol cotton blue stain (**Figure 3**A) and KOH mount examination (**Figure 3**B) of the BAL sample demonstrated broad, non-septate hyphae. BAL culture and biopsy specimen cultures showed moderate growth of Rhizopus fungus (**Figure 3**C), confirming mucormycosis.

Cytology and CBNAAT were negative. A diagnosis of pulmonary mucormycosis was made.

The patient was initiated on intravenous liposomal amphotericin B (5 mg/kg/day) with serial monitoring of renal function tests and serum electrolytes to assess for nephrotoxicity. Blood sugar levels were closely monitored and controlled. Cardiothoracic vascular surgery consultation was obtained, and left upper lobe lobectomy or wedge resection was planned following adequate medical management of the fungal infection. Serial chest X-rays were performed to monitor progress.

The patient showed gradual clinical and radiological improvement (**Figure 4**) with amphotericin B therapy. After one week of treatment, he was transitioned to oral posaconazole therapy (300 mg twice daily as a loading dose, followed by 300 mg daily). The patient was discharged on oral posaconazole therapy, after significant clinical improvement and was closely follow-up. A chest X-ray performed after three months of posaconazole therapy showed compete resolution of the disease (**Figure 5**).

This case highlights a non-resolving pneumonia that was diagnosed as pulmonary mucormycosis upon further investigation. The patient responded well to antifungal therapy, underscoring the importance of early diagnosis and targeted treatment in such cases.

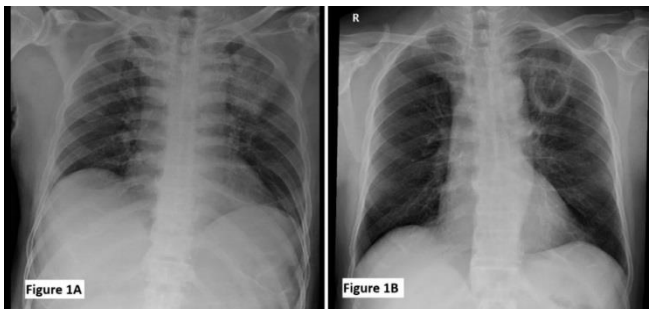

Figure 1: Chest x-ray showing consolidation in left upper zone; **A:** which has later progressed to a thick walled cavitary lesion **B**.

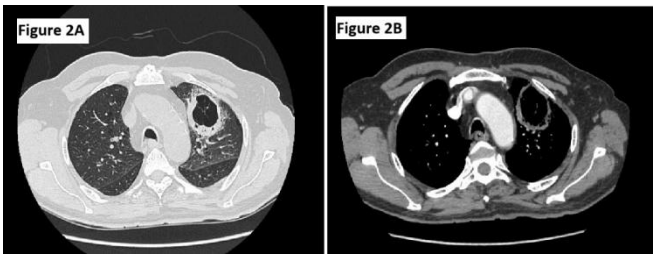

Figure 2: Contrast enhanced computed tomography images showing thick walled cavitary lesion in left upper lobe.

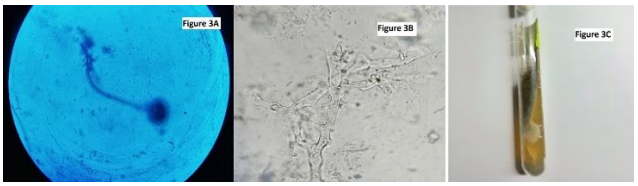

Figure 3: Lactophenol cotton blue stain showing long, aseptate hyphae bearing large, round, darkly stained sporangia; A: Potassium hydroxide mount showing broad, ribbon-like, non-septate hyphae of mucor; **B** and Black fluffy Rhizopus colony on Sabouraud Dextrose Agar medium **C**.

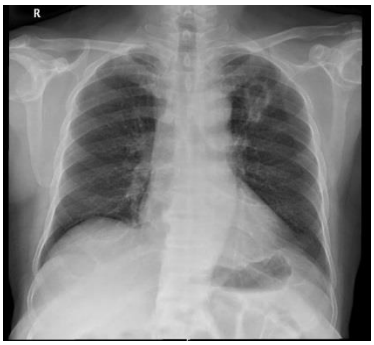

Figure 4: Chest x-ray showing shrinking cavity, after one week of amphotericin-B therapy.

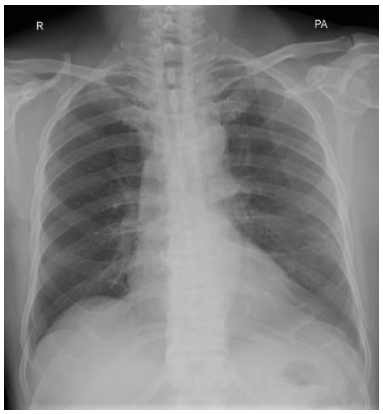

Figure 5: Chest x-ray showing completely resolved disease, after three months of posaconazole therapy.

3. Discussion

Mucormycosis is a potentially fatal fungal infection caused by moulds known as mucormycetes, classified under the order *Mucorales*.[1,2] The primary causative species include *Rhizopus*, *Actinomucor*, *Apophysomyces*, *Cunninghamella*, *Lichtheimia* (formerly *Absidia*), *Saksenaea*, and *Syncephalastrum*.[1] These fungi are widespread in nature and are commonly found in soil, decomposing organic matter, compost, and contaminated food sources.[1]

This opportunistic infection typically affects individuals with significant underlying conditions such as poorly controlled diabetes mellitus, hematologic malignancies, HIV/AIDS, severe malnutrition, or those receiving immunosuppressive therapy.[3] As seen in our case, uncontrolled diabetes—especially in the presence of diabetic ketoacidosis—predisposes individuals to mucormycosis. The fungi utilize a ketone reductase enzyme, enabling them to flourish in a high-glucose environment. Furthermore, increased iron availability enhances fungal proliferation, and diabetic patients exhibit elevated serum iron levels due to impaired transferrin binding, contributing to their susceptibility.[4]

Mucormycosis presents in various forms, including rhino-cerebral, pulmonary, cutaneous, gastrointestinal, disseminated, and rarer manifestations.[2] Pulmonary mucormycosis (PM) is the second most prevalent type after rhino-orbital involvement.[2]

Inhalation of fungal spores is the primary mode of infection in PM.[1] Upon entry into the respiratory tract, these spores germinate into hyphae, leading to angioinvasion, tissue necrosis, and potential hematogenous spread to other organs. [1] As in our case, the clinical presentation of PM is nonspecific, making differentiation from other pulmonary fungal infections challenging.[4] Symptoms commonly include fever, chest pain, dyspnea, and haemoptysis, which can be severe and life-threatening due to vascular invasion by fungal hyphae.[1]

Early detection is critical for prompt intervention and improved patient outcomes.[1] The diagnosis of mucormycosis is confirmed by identifying broad, ribbon-like, aseptate hyphae with tissue invasion on histopathology, supported by fungal culture from affected sites.[1] This distinct morphology differentiates *Mucorales* from other pathogenic fungi, such as *Aspergillus* spp.[1] Fungal cultures typically require three to five days for growth.[5]

Pulmonary mucormycosis exhibits a range of nonspecific radiological findings, often mimicking other fungal pneumonias.[1,4] A hallmark imaging feature is the *reverse halo* sign on chest CT, characterized by central ground-glass opacity surrounded by a dense consolidative ring, indicating central lung infarction with peripheral haemorrhage.[1] Common fungal biomarkers such as (1,3)-β-D-glucan and galactomannan have low sensitivity and specificity for *Mucorales*. Currently, no reliable serum-based diagnostic tests are available for mucormycosis.[1,5]

Management of PM involves antifungal therapy and, in some cases, surgical intervention.[5] Amphotericin B (AmB) remains the most potent antifungal agent against *Mucorales* and is the first-line treatment.[1] However, conventional amphotericin B deoxycholate (AmB-D) is associated with significant toxicity, including nephrotoxicity and infusion-related reactions. Liposomal amphotericin B (L-AmB), a lipid-based formulation, offers a better safety profile while maintaining efficacy.[6] The recommended dosage for L-AmB in mucormycosis is 5 mg/kg/day, whereas AmB-D is administered at 1–1.5 mg/kg/day.[1,6] Step-down or salvage therapy generally involves oral posaconazole or isavuconazole.[1,7] Currently, no standardized treatment duration exists; therapy is individualized and discontinued based on clinical and radiological improvement, negative fungal cultures, and resolution of immunosuppression.[8] In our case, the patient received an initial one-week course of L-AmB, followed by a transition to oral posaconazole.

Surgical debridement or resection of infected tissue is another key component of treatment, as it has been shown to enhance survival rates.[1,9] In our case, a left upper lobectomy

was initially planned but later deferred due to the patient's significant clinical improvement with antifungal therapy.

Early clinical suspicion and rapid diagnosis are crucial for effective management of mucormycosis.[1] Addressing predisposing factors, such as optimizing blood sugar levels in diabetic patients, is a fundamental aspect of treatment. [1] When complete elimination of risk factors is not feasible, such as in transplant recipients, efforts should be made to minimize immunosuppression as much as possible. Persistent immunosuppression presents a significant challenge in managing this aggressive fungal infection. [1]

4. Conclusion

Pulmonary mucormycosis remains a rare but serious infection that requires a high index of suspicion, particularly in patients with uncontrolled diabetes and non-resolving pneumonia. Early evaluation with imaging and microbiological confirmation is crucial for timely diagnosis and treatment initiation. Our case highlights the effectiveness of a stepwise antifungal approach, where initial L-AmB therapy was successfully transitioned to oral posaconazole, allowing outpatient management without the need for prolonged hospitalization or intensive renal function monitoring. This underscores the importance of considering mucormycosis in persistent pulmonary infections and the role of oral posaconazole as a practical step-down therapy. A multidisciplinary approach, including infectious diseases specialist, endocrinology, radiologist, microbiologist, and surgical teams, is crucial for optimizing mucormycosis management, as it facilitates early diagnosis, individualized treatment strategies, and improved patient outcomes.

5. Source of Funding

None.

6. Conflicts of Interest

The authors declare no conflicts of interest.

7. Acknowledgements

None.

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