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Pulmonary Mucormycosis with Endobronchial Involvement Complicated by Pleural Effusion

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ABSTRACT

Background: Pulmonary mucormycosis is an infection caused by a fungus with a severe and often fatal course, with a mortality rate exceeding 50%. It can spread through blood or lymphatic fluids or by inhalation of sporangiospores. Immunocompromised patients are especially susceptible to this high mortality rate. Histopathology is used to identify nonseptate hyphae in tissue, and culture is used to confirm the diagnosis. Treatment usually involves a combination of aggressive surgery and medication.

Case Report: A patient with diabetes mellitus received corticosteroid treatment for facial paralysis, which led to the diagnosis of pulmonary mucormycosis. Without requiring surgical resection, the patient was successfully treated with intravenous antifungal medication, along with intrabronchial and intrapleural liposomal amphotericin B.

Conclusion: Pulmonary mucormycosis requires prompt diagnosis and treatment. Close monitoring of such infections may minimize the need for invasive procedures and lower mortality, particularly when endobronchial involvement accompanied by effusion is present.

Keywords: Amphotericin, bronchoscopy, fungal infection, pleural effusion, pulmonary mucormycosis.

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INTRODUCTION

Fungal infections have become more common as a result of the growing use of chemical agents, immunosuppressive medications, broad-spectrum antibiotics, and organ transplantation. A serious fungal infection, mucormycosis usually strikes immunocompromised people as an opportunistic infection.[1]

The diagnosis of mucormycosis is based on the identification of non-septate hyphae in tissue through histopathology, followed by culture verification. Antifungal therapy and rigorous surgical debridement of the afflicted tissues are typically used in combination for treatment.[2]

This case concerns a diabetic patient who developed pulmonary mucormycosis (PM) after receiving systemic corticosteroid treatment for facial paralysis. This report's objectives are to help physicians identify and diagnose PM, especially when pleural effusion is present, and to offer practical treatment options that can lower death rates.

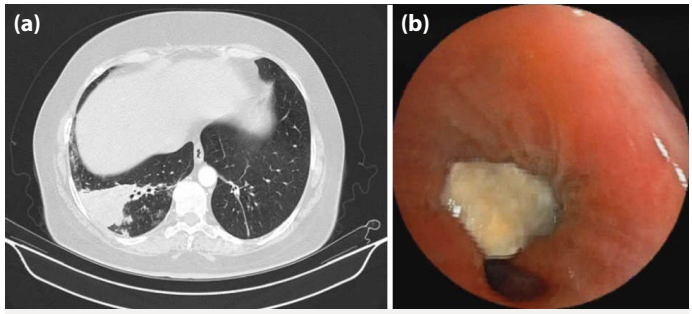

Figure 1. (a) Chest computed tomography showing consolidation in the right lower lobe. **(b)** White plaques observed during bronchoscopy on the entrance of the right lower lobe segments.

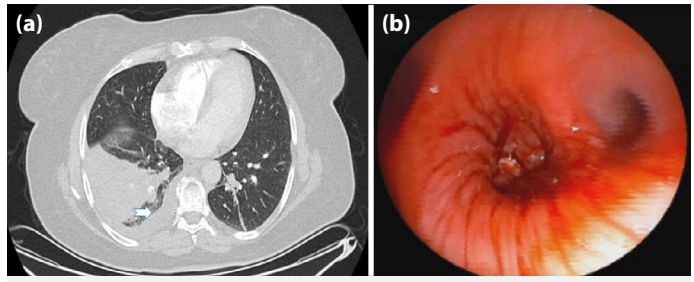

Figure 2. (a) Progression of the consolidation on the right lower lobe of the lung with pleural effusion (white arrow). **(b)** Bronchoscopic image showing obstruction of the entrance to the right lower lobe, with the mucosa appearing hyperemic and edematous.

The patient in this manuscript has given written informed consent to publication of their case details including images. The authors declare that this article is intended for a case report article

CASE REPORT

After receiving antibiotic treatment at another hospital, a 53-year-old woman with known diabetes mellitus came to our outpatient clinic with dyspnea. She had received methylprednisolone for a total of 15 days (beginning with 80 mg daily and titrated with decreasing doses of 16 mg every 3 days) after being diagnosed with facial paralysis two months earlier.

Routine blood tests performed upon admission revealed the following unusual findings: 10.6% of glycated hemoglobin (HbA1c), glucose 254 mg/dL, C-reactive protein 52.83 mg/L, and white blood cell count 24,570/µL. The right lower lobe showed consolidation on chest computed tomography (CT) (Fig. 1a). A white plaque was discovered at the entrance of the right lower lobe's basal segments during a flexible fiberoptic bronchoscopy (FFB) that same day (Fig. 1b). Under a microscope, hyphae were seen on the bronchoalveolar lavage (BAL) smear. The patient was started on intravenous liposomal amphotericin B (LAmB) in light of these findings.

There were signs of mucormycosis and hyphae that were consistent with a fungal infection in the transbronchial lung biopsy. BAL cultures showed no signs of bacterial or mycobacterial growth. Subsequent chest X-rays revealed a blunted right costophrenic sinus, a sign of pleural effusion, even though the patient was still on LAmB. During a thoracentesis, we determined the effusion to be exudative and drained it accordingly. Nevertheless, it re-accumulated.

A follow-up chest CT scan after three weeks of antifungal and antibiotic treatment revealed that the consolidation and pleural effusion had progressed (Fig. 2a). A second FFB was performed because of the chest CT's deteriorating results. We discovered that the mucosa was edematous and hyperemic, obstructing the entrance to the right lower lobe (Fig. 2b).

Following the addition of posaconazole to the treatment, we noticed a reduction in acute-phase reactants. The infiltration and effusion did not, however, resolve as anticipated radiologically. We chose to perform FFB and gave 50 mg of LAmB intrabronchially (50 mg diluted in 20 mL of saline). One week later, we administered an additional 25 mg of LAmB (diluted in 20 mL of saline) intrabronchially. A week after, we administered an additional 25 mg intrabronchially, along with 50 mg of LAmB (50 mg diluted in 20 mL of saline) intrapleurally.

The patient was discharged on oral posaconazole (400 mg twice daily). However, the patient returned with dyspnea two weeks later. While the consolidation and effusion in the right lower lobe were resolving, a follow-up chest CT scan showed that the left lower lobe had developed new infiltration. After readmission, the patient received an intrabronchial dose of 25 mg of LAmB.

We performed a second FFB one week later, administering 25 mg of LAmB intrabronchially to both the right and left lower lobes. The following week, we administered 50 mg of LAmB intrapleurally.

During follow-up in the outpatient clinic, the patient continued to improve. Significant regression was seen in both the left and right lower lobes on a follow-up CT scan two months later (Fig. 3a). While closely monitoring the patient, we conducted an investigatory FFB and found no fungal plaques, indicating a significant improvement. Additionally, there were no indications of pleural effusion or infiltration on the X-ray (Fig. 3b, c).

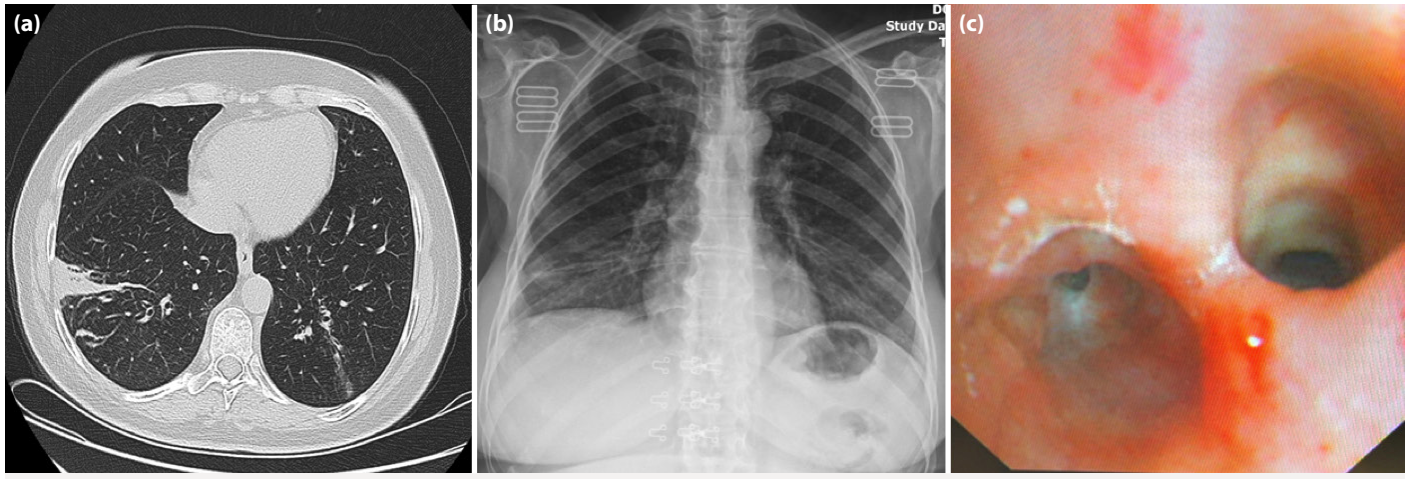

Figure 3. (a) Chest tomography showing significant regression of consolidation on both lower lung lobes. **(b)** Chest X-ray showing no infiltration nor pleural effusion. **(c)** No fungal plaques seen on the follow-up bronchoscopy.

DISCUSSION

Mucormycosis is a rapidly progressing fungal infection caused by filamentous fungi belonging to the Mucorales order. Rhino-orbital-cerebral mucormycosis is the predominant variant of the disease, succeeded by cutaneous and pulmonary forms. In immunocompromised individuals, infections are highly prevalent and usually arise from the inhalation of spores. The predominant underlying condition is inadequately controlled diabetes, with a considerable number of patients showing documented ketoacidosis.[3]

The pathogenic fungus poses challenges for prompt and accurate diagnosis owing to the nonspecific clinical and imaging characteristics of PM.[3] Prior to arriving at our clinic, the patient received treatment for pneumonia due to consolidation in the right lower lobe. Nonetheless, the recommended treatment failed to yield any alleviation. The nonspecific clinical and radiological findings in the patient's results may have led to a delay in diagnosis.

The most effective treatment for PM involves a combination of medical management, surgical intervention, and addressing the underlying predisposing condition. Despite these interventions, mortality rates remain high.[4] Nattusamy et al.[5] demonstrated the effective administration of 25 mg of LAmB via the intrabronchial route in a patient with endobronchial PM, who had shown no response to one month of intravenous LAmB treatment. A reduction in endobronchial growth was noted following three weeks of intrabronchial LAmB administration. The patient received oral posaconazole (400 mg twice daily) for eight weeks during the follow-up period, and a subsequent bronchoscopy did not indicate any recurrence. Our patient did not demonstrate improvement with intravenous LAmB alone and subsequently developed pleural effusion. We decided to deliver 50 mg of LAmB (diluted in 20 mL of saline) intrabronchially due to the presence of endobronchial mucormycosis plaques noted during FFB. LAmB was not aspirated following its administration as a bolus to exert its effects. We conducted the procedure an additional four times, administering 25 mg of diluted LAmB in 20 mL of saline on each occasion. In light of the advancement in the left lower lobe, we administered the concluding dose to both lower lobes of the lung. The follow-up revealed a notable enhancement.

Pleural effusion may occur in approximately 8% of all cases of PM, as observed by Liu et al.[6] The case they presented had pneumonia complicated by hydropneumothorax. The patient received antifungal therapy and effusion drainage, without surgical intervention. Pleural effusion resulting from PM has been addressed with intrapleural LAmB in several cases. A 56-year-old renal transplant recipient with diabetes was diagnosed with PM and subsequently underwent a right lung pneumonectomy, along with intrapleural LAmB (100 mg daily, diluted in 100 mL of saline, administered via a chest tube clamped for two hours over seven days).[7]

In our case, we initially sought to drain the effusion while administering intravenous antifungal therapy. We administered intrapleural LAmB (50 mg diluted in 20 mL of saline) twice within a two-week interval as the effusion reaccumulated. We delivered LAmB as a bolus into the pleural space via an injector and permitted it to take effect. The pleural effusion was successfully managed, and no additional accumulation was noted after the administration of intrapleural LAmB.

CONCLUSION

PM is a severe and life-threatening disease that is increasing in prevalence as a result of the rise in immunocompromised patients and advancements in medical treatment. The disease's vague and nonspecific presentations complicate the diagnosis, which in turn contributes to the high mortality and morbidity rates. It is imperative to address predisposing factors, surgical interventions, and early and effective medical management in order to reduce mortality associated with PM. Aggressive procedures such as repeated bronchoscopic and intrapleural drug injections, along with close monitoring, may be necessary as part of the treatment.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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