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Case Report

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Fatal Pulmonary Mucormycosis in a Diabetic Patient: A Case of Missed Early Diagnosis and Delayed Antifungal Therapy

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ABSTRACT

Background: Mucormycosis is a fast-progressing, angioinvasive fungal infection primarily affecting immunocompromised individuals, especially those with uncontrolled diabetes mellitus. Pulmonary mucormycosis presents diagnostic challenges due to its radiologic and clinical overlap with bacterial and other fungal pneumonias. This case highlights the clinical challenge and need for early recognition and management of pulmonary mucormycosis in high-risk diabetic patients with cavitating pneumonia.

Methods: We presented the case of a 66-year-old male with long-standing diabetes and hypertension who was admitted with fever, breathlessness, altered mental status, and signs of diabetic ketoacidosis. Initial assessments exposed bilateral pneumonia with cavitary lesions and ground-glass opacities.

Results: Despite empirical broad-spectrum antibiotics and antifungals, the patient's respiratory condition deteriorated. BAL and sputum cultures eventually revealed MDR *Klebsiella* and budding yeast cells. Liposomal amphotericin B was started late on Day 7, but the patient succumbed to refractory sepsis and respiratory failure on Day 8. Histopathology from transbronchial lung biopsy confirmed pulmonary mucormycosis with broad aseptate hyphae. The case underlines diagnostic delays in fungal sepsis in high-risk diabetics in the absence of early positive fungal cultures.

Conclusion: The presence of DKA, cavitary lung lesions, and unresolving pneumonia despite antibiotics should prompt suspicion of mucormycosis. Early empirical liposomal amphotericin B may be life-saving. Prompt recognition and aggressive treatment are essential for better outcomes, especially in diabetic patients with cavitary lesions unresponsive to standard antibacterial therapies.

Key-words: Pulmonary mucormycosis, Fungal sepsis, Diabetes mellitus, Necrotising pneumonia, Diabetic ketoacidosis, Fungal pneumonia, MDR Klebsiella

INTRODUCTION

Fungal sepsis, specially caused by invasive fungi such as *Mucorales*, represents a life-threatening clinical entity. Its diagnosis and management remain particularly challenging in immunocompromised individuals, particularly patients with uncontrolled diabetes mellitus [1]. Infections caused by *Mucor*, *Rhizopus*, and related species are grouped under mucormycosis, an angioinvasive infection associated with high illness and death.

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This opportunistic fungal infection has gained more attention in the post-COVID era, frequently triggered by immunosuppression, corticosteroid use, or hyperglycaemia [2].

In clinical practice, differentiating fungal sepsis from bacterial sepsis becomes difficult due to overlapping clinical and radiological features. The symptoms of mucormycosis include rapid progression, vascular invasion, and tissue necrosis, making early diagnosis and prompt antifungal therapy critical [3]. Pulmonary mucormycosis, though rare compared to rhino-cerebral involvement, can present as cavitating pneumonia, non-resolving infiltrates, or haemoptysis, and may mimic tuberculosis or bacterial necrotising pneumonia [4].

Patients with diabetic ketoacidosis present a predominantly favourable environment for mucormycosis to prosper. Acidosis impairs phagocytic

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activity, while hyperglycaemia and iron overload support fungal proliferation. Moreover, the angioinvasive nature of the fungi facilitates rapid tissue destruction and systemic dissemination [5].

This case report establishes a terminal episode of pulmonary mucormycosis in a 66-year-old diabetic and hypertensive male, and the diagnostic and therapeutic tasks [6]. Despite broad-spectrum antibiotics and antifungal therapy, delayed recognition of the fungal aetiology leads to fatal results, emphasising the importance of initial suspicion and destructive involvement [7].

The case sputum culture and radiological imaging may initially be non-conclusive or confusing, also emphasizing the limitations of conventional diagnostic modalities. In such high-risk patients, initial bronchoscopy and tissue biopsy, joined with empirical antifungal therapy, might improve outcomes [8].

Mucormycosis diagnosis is preferably established via histopathology or culture of tissue samples, see-through broad, aseptate hyphae with angioinvasion. However, delays in diagnosis are frequent due to the lack of specific non-invasive biomarkers. Management primarily includes initial administration of liposomal amphotericin B and surgical debridement if possible [9].

This report also brings attention to multidrug-resistant bacterial co-infections, which frequently accompany fungal sepsis in immunocompromised patients, further complicating the clinical course and antibiotic stewardship [10,11].

Eventually, this case emphasises the need for heightened clinical suspicion, rapid diagnostic workup, early initiation of appropriate antifungal agents, and a multidisciplinary method of management. It serves as an instructive example for clinicians' management of critically ill diabetic patients presenting with cavitary lung lesions and systemic deterioration [12].

CASE REPORT

A 66-year-old male with a history of type 2 diabetes mellitus and hypertension for the past 10 years, accomplished with oral hypoglycaemic agents, presented to the Emergency Department of a tertiary care hospital, with a history of fever for one-month, and nausea on the day of admission and advanced breathlessness for two weeks, and acute onset different mental status, abdominal pain.

On examination, the patient was febrile (100°F), with a blood pressure of 120/70 mmHg, respiratory rate of 28 breaths per minute, and SpO_2 of 86% on room air. He was cachectic and found to have a deep, infected foot ulcer. The central blood glucose was 598 mg/dL, and arterial blood gas analysis exposed pH 7.43, PaO_2 58.1 mmHg, $PaCO_2$ 18.6 mmHg, and HCO_3^- 16.5 mmol/L, suggestive of metabolic acidosis. Urine ketones were positive (++), representing diabetic ketoacidosis.

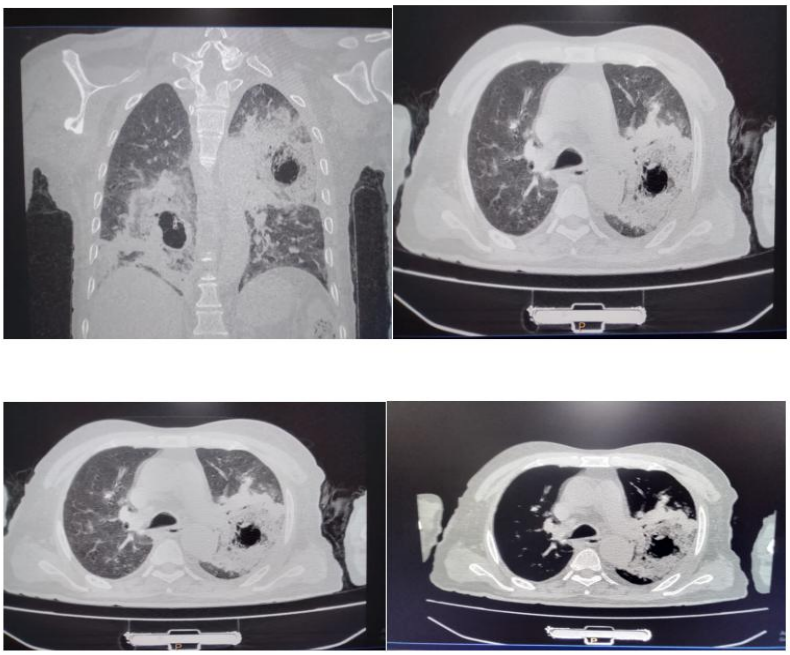

Fig 1: Bilateral thick-walled cavity with surrounding ground-glass opacity which likely to be tuberculosis as per the radiologist's impression

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Initial laboratory investigations exposed important abnormalities: haemoglobin was 9.1 g/dL, serum urea was 104 mg/dL, and creatinine was 2.7 mg/dL, total leukocyte count was 17,600/mm³ with 89% neutrophils. Serum sodium was 130 mEq/L, and potassium was 3.2 mEq/L. Albumin was disparagingly low at 1.8 g/dL. Markers of simple infection and inflammation were elevated: procalcitonin at 29.91 ng/mL and C-reactive protein at 99.98 mg/L. His glycaemic control was poor, as established by an HbA1c of 14.5%. Chest auscultation revealed bilateral crepitations. Radiological assessment established bilateral thick-walled cavities with surrounding ground-glass opacities on chest imaging, results that elevated suspicion for necrotising infections or fungal involvement.

He was immediately started on empirical intravenous piperacillin-tazobactam, teicoplanin, fluconazole, fluid resuscitation, and insulin infusion. Over the next 48–72 hours, the patient remained febrile, tachypneic, and hypoxic, necessitating intermittent non-invasive ventilation. They are prompting escalation of antibiotics to meropenem and replacement of fluconazole with voriconazole due to increasing suspicion of fungal pneumonia, was no important clinical improvement.

By Day 4, the patient presented signs of partial development. He became afebrile and maintained suitable oxygen saturation on 6 L/min oxygen via face mask. Blood pressure and urine output stabilised, and representing correction of ketoacidosis, urine ketones became negative. However, repeat laboratory assessment exposed a drop in haemoglobin to 6.8 g/dL and a reduction in TLC to 9,900/mm³. Serum urea and creatinine improved to 42 mg/dL and 1.7 mg/dL, respectively. Procalcitonin levels decreased to 2.88 ng/mL. Despite these biochemical improvements, chest X-ray results continued unchanged, and the patient continued to exhibit rising apprehensions about a continuing or inadequately given pathology determined tachypnoea and elevated inflammatory markers.

On Day 5, in view of non-resolving respiratory symptoms and lack of radiological improvement, a diagnostic bronchoscopy with bronchoalveolar lavage was performed. Bronchoscopic visualisation revealed mucopurulent secretions in the right lower and left upper lobes, along with an edematous, necrotic bronchial mucosa that bled upon touch. Whitish necrotic patches were noted, raising the thought of invasive fungal infection. BAL samples were sent for CBNAAT, which returned negative for Mycobacterium tuberculosis.

On Day 6, requiring continued NIV support, the patient's oxygenation status was criticised. BAL cultures revealed the presence of multidrug-resistant *K. pneumoniae*, which was sensitive only to a combination of ceftriaxone-sulbactam-EDTA and fosfomycin. The fungal stains remained negative despite determined clinical suspicion. In response, nebulised colistin was introduced, and antibiotic therapy was tailored to culture sensitivity. Arterial blood gas analysis showed deteriorating parameters with PaO_2 of 73.4 mmHg, $PaCO_2$ of 26.2 mmHg, pH of 7.44, and HCO_3^- of 17.4 mmol/L.

By Day 7, a transbronchial lung biopsy was performed due to persistent clinical deterioration and radiographic progression. Histopathological examination of the biopsy specimen revealed broad, aseptate, ribbon-like hyphae suggestive of mucormycosis, confirming the clinical suspicion. The patient required continuous NIV, with a marked increase in oxygen demand and deteriorating urine output. ABG showed additional deterioration: PaO_2 was 50.3 mmHg, $PaCO_2$ was 34.9 mmHg, pH was 7.48, and HCO_3^- rose to 25.0 mmol/L. Inflammatory markers were again elevated; TLC increased to 14,800/mm³, and procalcitonin surged to 43.77 ng/mL. Sputum culture sent on the same day revealed numerous short, determined gram-negative bacilli and occasional budding yeast cells, pointing toward a polymicrobial infection with probable invasive fungal involvement. Considering the new findings and strong clinical suspicion, liposomal amphotericin B was initiated.

Inappropriately, on Day 8, the patient rapidly deteriorated. He became tachypneic and restless, followed by bradycardia. He was intubated emergently but suffered a cardiac arrest. Despite full resuscitative efforts, the patient could not be revived. Final ABG results showed PaO_2 of 55 mmHg, $PaCO_2$ of 50 mmHg, pH of 7.158, and HCO_3^- of 19.2 mmol/L. His TLC was 12,100/mm³, serum urea increased to 119 mg/dL, and creatinine rose to 3.1 mg/dL. Fluid input/output over the last 24 hours was 1470/1215 mL.

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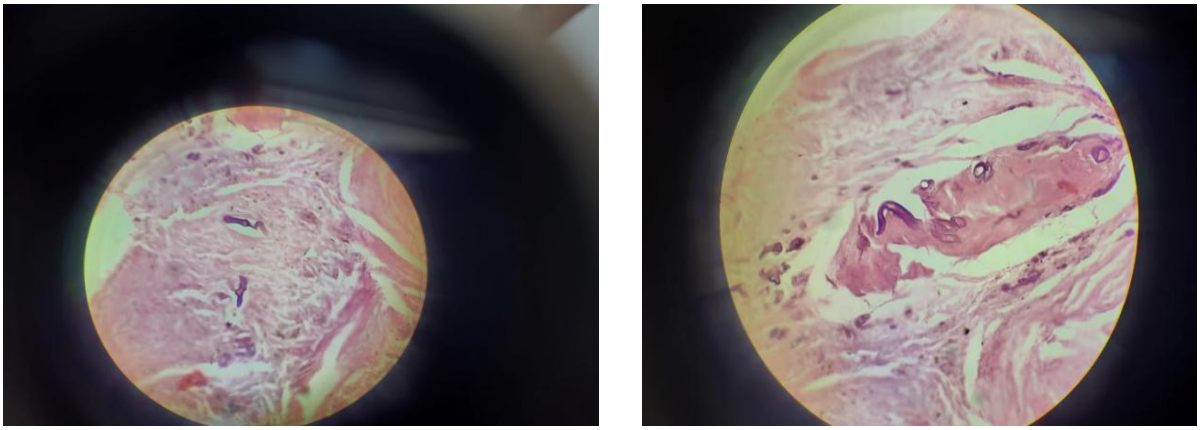

Fig. 2: Broad aseptate hypha suggestive of mucor mycosis

Definitive diagnosis was made based on transbronchial lung biopsy findings, which demonstrated broad, aseptate fungal hyphae consistent with Mucorales species based on the detection of broad, aseptate fungal hyphae characteristic of *Mucorales* species, including Radiological review and histopathology have long established the diagnosis of pulmonary mucormycosis. The definitive diagnosis was made too late to influence therapy, as the liposomal amphotericin B had been initiated only hours before the incurable incident.

This case emphasises numerous missed opportunities. The patient had simple, uncontrolled diabetes, complicated by DKA and cachexia, ideal conditions for mucormycosis. Despite evident clinical signs and imaging consistent with fungal involvement, initial fungal cultures were negative, and BAL/sputum studies were delayed, leading to a late diagnosis. Even though voriconazole was empirically initiated, it is ineffective against mucormycosis. Amphotericin B, the drug of choice, was introduced only on Day 7. Moreover, no surgical debridement or biopsy was attempted, possibly due to the patient's fragile condition.

In critical need to suspect mucormycosis initially in diabetic patients presenting with non-resolving pneumonia and cavitary lung lesions. In such cases, empirical initiation of liposomal amphotericin B may be lifesaving. BAL cultures and histopathological confirmation remain essential, but should not delay treatment if clinical suspicion is high. Initially, aggressive therapy with appropriate antifungal agents and multidisciplinary care is important to improving survival in this lethal disease.

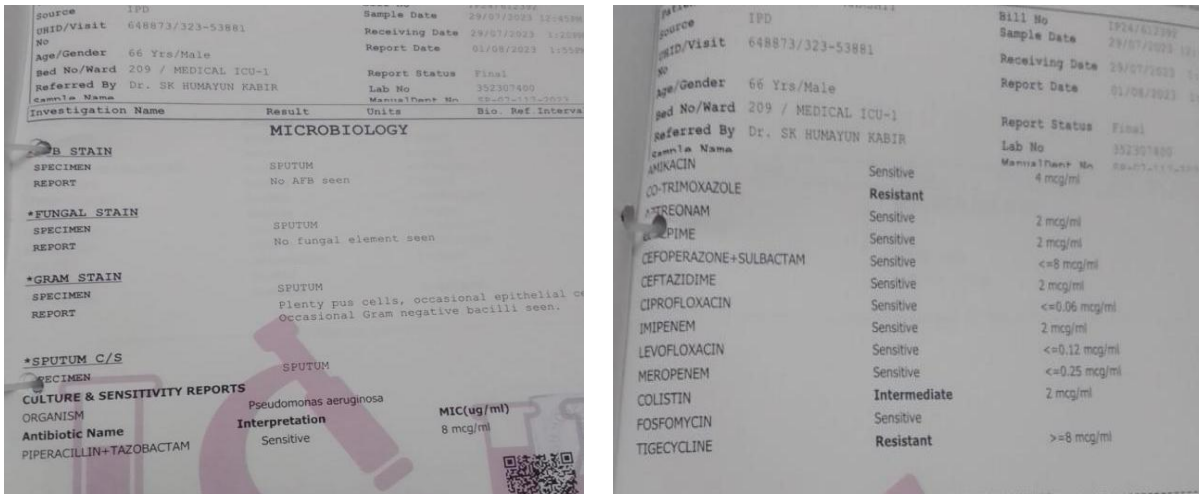

Fig. 3: Microbiological Findings of the presented case

DISCUSSION

Pulmonary mucormycosis typically affects immuno-compromised individuals, a rare but rapidly advancing opportunistic fungal infection. The disease is characterised by aggressive tissue invasion, vascular thrombosis, and necrosis, which contribute to its high illness and death rates. In this case,

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the patient presented with classical risk factors, especially those with uncontrolled diabetes mellitus, haematological malignancies, or who are on immunosuppressive therapy, including poorly controlled diabetes with ketoacidosis, and clinical results of non-resolving pneumonia and systemic sepsis, ultimately confirmed postmortem as pulmonary mucormycosis.

Mucormycosis is caused by fungi have its place to the order *Mucorales*. These organisms are abundant in the environment, found in soil, decaying organic matter, and even hospital air systems. In healthy individuals, primarily *Rhizopus*, *Mucor*, and *Rhizomucor* species, the immune system, predominantly neutrophils and macrophages, prevents fungal invasion. However, in immunocompromised states such as diabetes mellitus, mainly in the situation of ketoacidosis, these defence mechanisms are impaired. Acidosis disturbs iron-binding proteins, leading to increased free iron, which supports fungal growth. In addition, hyperglycaemia impairs neutrophil chemotaxis and phagocytosis, enabling rapid fungal proliferation and tissue invasion [13-15].

This development of uncontrolled diabetes, ketoacidosis, and cachexia case establishes the ideal situation for mucormycosis. These are important to lung tissue infarction and subsequent systemic sepsis, creating a permissive environment for the angioinvasive properties of *Mucorales*.

Pulmonary mucormycosis presents non-specifically with symptoms such as fever, cough, dyspnoea, chest pain, and haemoptysis. Radiologically, it can mimic bacterial pneumonia, tuberculosis, or other fungal infections like invasive aspergillosis. Common imaging findings include cavitary lesions, consolidation, nodules with halo or reverse halo signs, and pleural effusions [16-18].

In this case, the initial chest X-ray and clinical picture were consistent with bilateral pneumonia and cavitary lesions, which could be misinterpreted as necrotising bacterial infection or pulmonary tuberculosis, both prevalent conditions in the Indian subcontinent. The radiotherapist initially did not identify any classic signs, such as the halo or reverse halo sign, which made the diagnosis more complex. Although CT imaging would have been more specific, financial or logistical limitations might have limited its use.

Bronchoscopy on Day 5 revealed necrotic, bleeding bronchial mucosa and whitish patches, raising clinical suspicion of invasive fungal infection. However, the BAL sample was negative for fungal elements, which is not unusual in mucormycosis. Culture and microscopy are frequently negative due to difficulty in isolating the organism and its fastidious nature. In addition, serologic tests like galactomannan and β-D-glucan, useful in aspergillosis, are typically negative in mucormycosis [19]. Thus, a high index of clinical thought is paramount.

In this patient, fungal infection was suspected but not aggressively pursued until late in the disease course. Empirical voriconazole was started early; however, this agent lacks activity against *Mucorales* and might even exacerbate mucormycosis by inhibiting competing fungal flora [20]. Amphotericin B, th treatment of choice, was only introduced on Day 7, too late to change the clinical trajectory.

One distinguished feature in this case is the polymicrobial nature of the infection. The patient's BAL culture revealed MDR *K. pneumoniae*, which complicated management and may have masked or delayed the diagnosis of fungal sepsis. Coinfections with bacteria are not uncommon in mucormycosis and can deteriorate outcomes by contributing to septic shock, further immunosuppression, and multiorgan failure [6]. The delayed adjustment of antibiotics and late initiation of colistin may have compounded the disease severity.

The budding yeast cells, suggesting the onset of fungal proliferation and sputum on Day 7 showed gram-negative bacilli. This makes even with results from other studies that describe mucormycosis as a "late diagnosis" infection, frequently picked up after empirical treatment for bacterial causes fails [5].

The foundation of treatment for mucormycosis includes initial initiation of intravenous liposomal amphotericin B, surgical debridement of infected tissue, and reversal of fundamental risk factors such as hyperglycaemia, acidosis, and neutropenia [21]. Liposomal amphotericin B is preferred due to its better acceptability and the ability to deliver higher doses. However, its efficacy is time-sensitive. A delay of more than 6 days in starting antifungal treatment is related to an important increase in death [22].

Inappropriately, in this case, liposomal amphotericin B was started only after the infection had significantly progressed. Surgical involvement was not possible given the rapid clinical decline and generalised sepsis. Even though metabolic parameters such as blood glucose and ketoacidosis were initially controlled, the determined

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hypoxia and radiographic non-resolution were not aggressively investigated early enough.

Alternative agents such as posaconazole or isavuconazole are used as step-down therapy or salvage therapy, but were not employed here due to the late stage of disease recognition. This case underlines the need for rapid escalation to definitive antifungal therapy when fungal pneumonia is suspected in a vulnerable patient, regardless of initial BAL or culture results.

Numerous important clinical lessons can be derived from this case. First, mucormycosis must be included in the differential diagnosis of cavitary lung lesions in diabetic patients, especially when they do not respond to standard antibacterial or anti-tubercular therapy. Second, a negative fungal stain does not exclude invasive fungal disease. Clinical judgment, bronchoscopy findings, radiology, and host risk factors must be weighed together.

Third, empirical antifungal therapy should be considered early in critically ill patients with radiological and clinical signs suggestive of invasive fungal infection. Delay in initiating amphotericin B, the only fungicidal drug against mucormycosis, remains a major contributor to poor prognosis. Fourth, voriconazole should not be used empirically if mucormycosis is suspected, as it lacks activity against *Mucorales*. Finally, emphasise the overlap between fungal and bacterial sepsis, the need for a comprehensive microbial workup, the high procalcitonin level and marked inflammatory response in this case.

This case also establishes that the limitations in resource-limited situations, even in the absence of laboratory confirmation, are serious in such environments where rapid molecular diagnostics, advanced imaging, and antifungal stewardship may not be readily accessible. Training clinicians to recognise red-flag signs of mucormycosis.

Pulmonary mucormycosis transmits a grim prognosis, with mortality rates ranging from 50% to 80% depending on mass immunity and timeliness of therapy [23]. Dispersed mucormycosis, as seen in this patient, has near-universal death. Surgical resection of affected lung lobes improves survival but is rarely feasible in critically ill patients [24].

This should be actively considered in diabetic patients presenting with non-resolving pneumonia and signs of sepsis case reinforces that mucormycosis, however rare. Initial bronchoscopy, imaging review, empirical antifungal initiation, and aggressive supportive care are vital. Multidisciplinary is essential for optimising consequences involvement, including infectious disease specialists, pulmonologists, intensivists, and microbiologists. Most importantly, clinicians must not delay initiating treatment while pending definitive culture or biopsy results in suspected cases of invasive fungal disease.

CONCLUSIONS

Pulmonary mucormycosis is a rare but fatal opportunistic infection, particularly in diabetic patients presenting with sepsis and cavitating lung lesions. Clinical recognition is challenging and delayed diagnosis often results in lethal consequences. In this case, despite early empirical antibiotics and antifungals, the absence of specific early diagnostic markers and false-negative cultures led to a missed therapeutic window. Voriconazole, though commonly used for fungal infections, is ineffective against Mucorales and may worsen outcomes if not promptly replaced with amphotericin B upon clinical suspicion. In resource-limited settings, clinical judgment, radiological findings, and awareness of risk factors must guide early intervention. Empirical use of liposomal amphotericin B in critically ill diabetic patients showing signs of pulmonary mucormycosis could be lifesaving. A multidisciplinary approach involving infectious disease specialists, pulmonologists, and intensivists is essential. Ultimately, early suspicion, rapid antifungal therapy, and aggressive supportive care remain the cornerstones for improving survival in mucormycosis-related fungal sepsis.

CONTRIBUTION OF AUTHORS

One author has only contributed to this article.

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