Original Article

Central nervous system mucormycosis in diabetic patients: clinical features, neuroimaging and outcomes from a tertiary care hospital in Bangladesh

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ABSTRACT

Background: *Central nervous system (CNS) mucormycosis is a rare but life-threatening fungal infection that predominantly affects patients with uncontrolled diabetes mellitus. Data on its clinical characteristics, neuroimaging findings, management, and outcomes in Bangladesh remain limited. This study aimed to describe the clinical profile and outcomes of CNS mucormycosis in diabetic patients at a tertiary care hospital.*

Methods: *We conducted a retrospective cohort study of adult diabetic patients (e"18 years) with confirmed CNS mucormycosis admitted between 1 July 2021 and 30 June 2025. Data on demographics, comorbidities, clinical presentation, laboratory parameters, microbiological or histopathological findings, neuroimaging features, treatments, and outcomes were collected from medical records. Follow-up data up to three months after discharge were also analyzed.*

Results: *A total of 27 patients with CNS mucormycosis were included. Type 2 diabetes mellitus was present in 23 (85%), and type 1 diabetes mellitus in 4 (15%). Diabetic ketoacidosis occurred in 2 (7%), and chronic kidney disease in 7 (26%). HbA1c ranged from 9.5% to 16.4%, indicating poor glycemic control. The most frequent presenting symptoms were headache (24, 90%), fever (22, 82%), and periorbital swelling (21, 76%). Neurological features included ophthalmoplegia (14, 52%), cranial nerve palsy (11, 42%), and altered consciousness (4, 16%). Seizures and hemiplegia were less common (4, 16% and 3, 12%, respectively). Microbiological or histopathological confirmation was achieved in about half of the cases. MRI showed sinusitis (15, 56%), orbital extension/mass (13, 47%), cavernous sinus thrombosis (4, 16%), intracerebral space-occupying lesions (4, 14%), cerebral infarction (2, 8%), and skull base osteomyelitis (1, 3%). Treatment included conventional amphotericin B (13, 48%), liposomal amphotericin B (8, 30%), and posaconazole (6, 22%). Surgical interventions included functional endoscopic sinus surgery (11, 41%), maxillary/palatal debridement (3, 11%), and craniotomy (1, 4%). Hospital stay ranged from 13–28 days. In-hospital mortality was 52% (14/27). Among survivors, 8 (30%) had persistent neurological deficits at discharge. At three-month follow-up (n = 13), recurrence occurred in 6 (46%), persistent neurological deficits in 5 (38%), and complete recovery in 2 (15%).*

Conclusions: *CNS mucormycosis in diabetic patients is associated with high morbidity and mortality. Early diagnosis, prompt antifungal therapy, surgical intervention, and strict glycemic control are essential to improve outcomes. The high recurrence and neurological complication rates highlight the need for close follow-up in this high-risk population.*

Key words: *Central nervous system, mucormycosis, diabetes mellitus, rhino-orbital-cerebral mucormycosis, neuroimaging, amphotericin B, outcomes.*

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INTRODUCTION

Mucormycosis, caused by fungi of the order Mucorales, is the second most common invasive mold infection after aspergillosis, and its incidence has been increasing globally. It primarily affects individuals with uncontrolled diabetes mellitus, immunosuppression, or severe trauma, with clinical manifestations varying by host factors. Diabetic patients commonly develop rhino-orbital-cerebral mucormycosis (ROCM), whereas pulmonary and cutaneous forms are more frequently seen in hematological malignancies and trauma. Central nervous system (CNS) involvement is one of the most severe complications due to aggressive angioinvasion and tissue infarction, resulting in high morbidity and mortality.[1,2]

Most estimates of CNS mucormycosis are derived from case reports and small series, while population-based studies report an overall mucormycosis incidence of 0.43-1.73 per million, with increasing trends over time. The frequency of CNS involvement varies across populations and is more common in regions with diabetes-associated sinus disease, such as the Indian subcontinent, whereas hematologic malignancies are prominent risk factors in cancer-dominant cohorts.[3,4] Uncontrolled diabetes, particularly diabetic ketoacidosis (DKA), remains the leading risk factor for ROCM, often with cerebral extension. In developing countries, mucormycosis may also be the first manifestation of previously undiagnosed diabetes.[5,6]

CNS mucormycosis usually develops following inhalation of fungal spores, with spread from the paranasal sinuses or via hematogenous dissemination. Angioinvasion mediated by fungal ligands such as spore coat homolog (CotH) proteins binding to glucose-regulated protein 78 (GRP78) receptors on endothelial cells leads to vascular thrombosis, tissue necrosis, and brain invasion, while increased iron availability further enhances fungal growth and virulence.[7,8] ROCM typically begins in the nasal turbinates or paranasal sinuses and may initially mimic sinusitis but can rapidly progress to orbital and intracranial involvement, causing ophthalmoplegia, cranial nerve deficits, cavernous sinus thrombosis, hemiparesis, seizures, and altered consciousness.[9]

Radiologically, ROCM often shows sinus mucosal thickening, soft-tissue infiltration, and the characteristic "black turbinate sign" on magnetic resonance imaging (MRI), with possible orbital extension or bone destruction. Intracranial involvement may present as cavernous sinus thrombosis, brain infarction, or carotid artery occlusion, although these findings can overlap with other orbital or intracranial pathologies.[10,11] Other forms include isolated cerebral mucormycosis, commonly reported in intravenous drug users with basal ganglia involvement, and intracranial granulomatous mucormycosis, frequently described in diabetic patients in the Indian subcontinent presenting with tumor-like masses and focal neurological deficits.[12]

Definitive diagnosis requires histopathological or microbiological confirmation, demonstrating characteristic broad, ribbon-like, non-septate hyphae using special stains such as Gomori methenamine silver (GMS) or periodic acid–Schiff (PAS). Since no reliable circulating biomarkers are available, indirect diagnosis often relies on tissue sampling from involved sites, although polymerase chain reaction (PCR)-based and immunohistochemical techniques are emerging as promising diagnostic tools. [13]

Management requires early antifungal therapy with liposomal amphotericin B or isavuconazole, aggressive surgical debridement, and correction of underlying metabolic or immunological abnormalities, with prolonged treatment often required for several months.[14]

In Bangladesh, the rising prevalence of diabetes may increase the burden of central nervous system (CNS) mucormycosis, a condition associated with high morbidity and mortality. However, local data on its clinical characteristics, neuroimaging findings, and outcomes are limited. This study aims to evaluate the clinical presentation, neuroimaging features and outcomes of CNS mucormycosis in diabetic patients in Bangladesh.

METHODS

This retrospective cohort study was conducted at BIRDEM General Hospital, a tertiary care referral center in Bangladesh, between 1 July 2021 and 30 June 2025, and included adult hospitalized diabetic patients (e"18 years) with confirmed CNS mucormycosis. Patients with other CNS fungal infections or incomplete medical records were excluded.

Relevant data including demographic characteristics, glycemic status, clinical presentation, neuroimaging findings, microbiological or histopathological

confirmation, treatment received, and outcomes were collected from medical records. Diagnosis was based on clinical suspicion supported by brain and orbital magnetic resonance imaging (MRI) or computed tomography (CT) and confirmed through endoscopic or surgical biopsy with histopathology and fungal culture.

The primary outcome was in-hospital mortality. Secondary outcomes included survival to discharge (with or without neurological deficits), leaving against medical advice (LAMA), and three-month follow-up status. Survivors were assessed clinically at discharge and re-evaluated at three months through outpatient visits. Follow-up outcomes were categorized as recurrence of mucormycosis, persistence of neurological deficits, or complete recovery. Data were analyzed using descriptive statistics, with categorical variables presented as frequencies and percentages.

RESULTS

A total of 27 adult diabetic patients with confirmed CNS mucormycosis were included in the study. The mean age was 44 ± 8 years (range 30–58 years), with 13 males and 14 females. Baseline characteristics, including co-morbidities and hematological parameters, are summarized in Table I. The most common presenting symptoms were headache (90%), fever (82%), and periorbital swelling (76%). Other clinical features at presentation are summarized in Table II. Diagnostic confirmation of CNS mucormycosis was achieved through histopathology and direct microscopy. Histopathology was positive in 15 patients (56%), while direct microscopy was positive in 14 patients (52%). Specimens included nasal swabs (16), sinus tissue (12), orbital tissue (2), and brain biopsy (1). The detailed findings are summarized in Table III. MRI findings demonstrated involvement of the paranasal sinuses in 15 patients (56%), with orbital extension or mass in 13 patients (47%). Less common findings included cavernous sinus thrombosis (16%), intracranial space-occupying lesions (ICSOL) (14%), cerebral infarction/stroke (8%), and skull base osteomyelitis (3%). The detailed findings are summarized in Table IV. Treatment included conventional Amphotericin B in 13 patients (48%), liposomal Amphotericin B in 8 (30%), and Posaconazole in 6 (22%). Surgical interventions were performed in 15 patients, including FESS (11, 41%), maxilla/palate debridement (3, 11%), and craniotomy with debridement (1, 4%). The average hospital stay ranged from 13 to 28 days (Table V). In-hospital mortality was 52% (14 patients). Among survivors (12, 44%), 8 (30%) had persistent neurological deficits at discharge, while 4 (15%) recovered without deficits. One patient (4%) left against medical advice (Table VI). At three months, 13 patients were followed up either by outpatient clinical review or structured phone interview. Recurrence was observed in 6 patients (46%), persistent neurological deficits in 5 patients (38%), and full recovery in 2 patients (15%). The detailed findings are summarized in Table VII.

Table I. Baseline characteristics (N=27)

Variable	n (%) / Range
Type 2 DM	23 (85)
Type 1 DM	4 (15)
Diabetic ketoacidosis (DKA)	2 (7)
Chronic kidney disease	7 (26)
Renal transplantation	1 (4)
Bronchial carcinoma	1 (4)
Hemoglobin (g/dL)	11 – 13
TLC (/µL)	6,400 – 19,000
Random blood sugar (mmol/L)	17
HbA1c (%)	9.5 – 16.4
CRP (mg/L)	64 – 226
ESR (mm/hr)	65 – 120

Table II. Clinical features at presentation

Symptom	n (%)
Headache	24 (90)
Fever	22 (82)
Periorbital swelling	21 (76)
Ophthalmoplegia	14 (52)
Cranial nerve palsy	11 (42)
Facial pain	13 (48)
Blurred vision	11 (40)
Nasal crusting	7 (26)
Altered consciousness	4 (16)
Seizures	4 (16)
Hemiplegia	3 (12)

Table III. Microbiology and histopathology findings in patients with CNS mucormycosis

Specimen / tests	n (%)
Nasal swabs	16
Sinus tissue	12
Orbital tissue	2
Brain biopsy	1
Histopathology positive	15 (56)
Direct microscopy positive	14 (52)

Table IV. MRI findings showing sinus, orbital, and intracranial involvement in CNS mucormycosis

Imaging Feature	n (%)
Sinusitis	15 (56)
Orbital extension / mass	13 (47)
Cavernous sinus thrombosis	4 (16)
ICSOL	4 (14)
Cerebral infarction / stroke	2 (8)
Skull base osteomyelitis	1 (3)

Table V. Treatment and surgical interventions

Treatment3	n (%)
Conventional Amphotericin B	13 (48)
Liposomal Amphotericin B	8 (30)
Posaconazole	6 (22)
FESS	11 (41)
Maxilla/palate debridement	3 (11)
Craniotomy with debridement	1 (4)
Average hospital stays (days)	13–28

Table VI. Patient outcomes during hospital stay and discharge

Outcome	n (%)
In-hospital mortality	14 (52)
Left against medical advice (LAMA)	1 (4)
Survived to discharge	12 (44)
– With neurological deficits	8 (30)
– Without deficits	4 (15)

Table VII. Three-month follow-up outcomes (n=13)

Recurrence	6 patients (46%)
Persistent neurological deficits	5 patients (38%)
Full recovery	2 patients (15%)

Follow-up outcomes were assessed either by outpatient clinical review or structured phone follow-up.

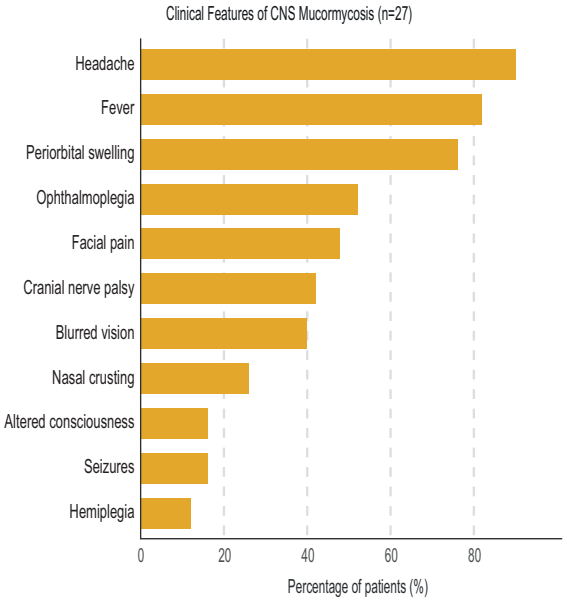

Figure 1. Clinical features of CNS mucormycosis

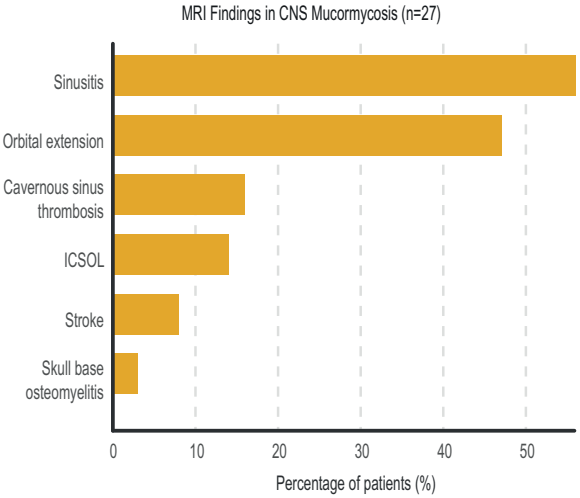

Figure 2. MRI findings in CNS mucormycosis

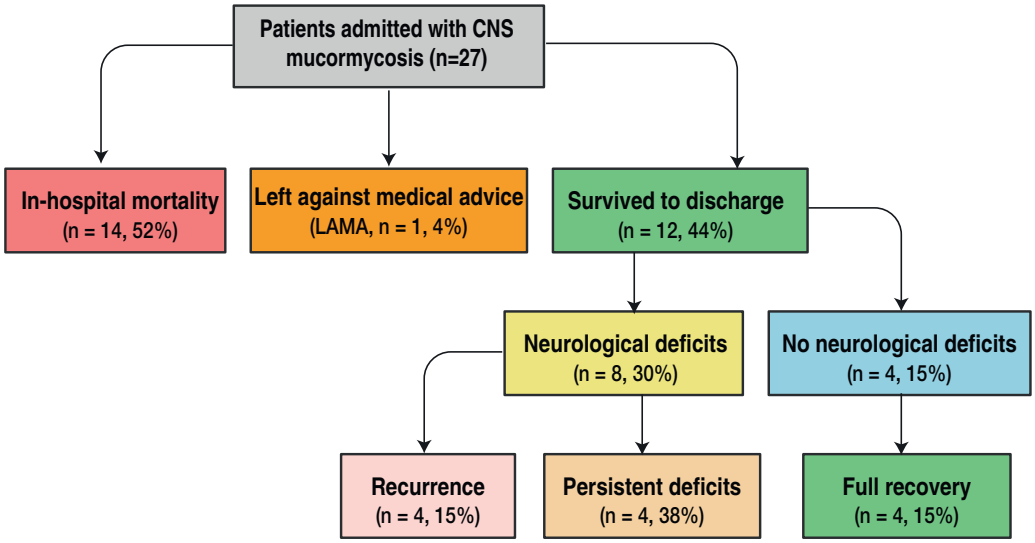

Figure 3. Flowchart of patient outcomes

DISCUSSION

CNS mucormycosis is a rare but devastating manifestation of invasive mucormycosis, particularly in patients with uncontrolled diabetes mellitus.[15] In this study of 27 patients, we observed that the majority (85%) had type 2 diabetes mellitus, with markedly elevated HbA1c values (9.5–16.4%), underscoring poor glycemic control as a key predisposing factor. A smaller proportion presented with diabetic ketoacidosis (7%) or chronic kidney disease (26%), conditions previously recognized as amplifying the risk of mucormycosis. These findings are consistent with global reports, where diabetes remains the single most important risk factor for mucormycosis, especially in South Asian populations.[16,17]

The clinical presentation in our study was dominated by headache (90%), fever (82%), and periorbital swelling (76%), which reflect early sino-orbital involvement. More advanced features, including cranial nerve palsy (42%), ophthalmoplegia (52%), and altered consciousness (16%), suggest progression into CNS structures as shown in figure 1. Seizures and hemiplegia, though less frequent, highlight the potential for cortical invasion and ischemic injury. These findings align with previous series where orbital and cranial nerve involvement often precede catastrophic intracranial complications.[18-21]

Microbiological and histopathological confirmation was achieved in just over half of the cases, highlighting the persistent diagnostic challenge of mucormycosis. Direct microscopy and histopathology yielded positive results in 52% and 56% of patients, respectively. This rate is comparable to other reports, where culture sensitivity remains limited and histopathology remains the cornerstone of diagnosis. Brain biopsy was performed in only one patient, reflecting the practical difficulties of obtaining intracranial tissue in critically ill patients.

Radiologically, sinusitis (56%) and orbital extension (47%) were the most common findings, while intracranial space-occupying lesions, cavernous sinus thrombosis, and infarctions were less frequent as shown in figure 2. The low yield of classical signs, such as the "black turbinate" sign, further emphasizes the non-specificity of imaging and the importance of combining clinical suspicion with microbiological or histopathological confirmation.[22,23]

Treatment patterns in this study reveal both therapeutic challenges and resource limitations. Nearly half of the patients received conventional amphotericin B, while only 30% received liposomal amphotericin B, which is better tolerated but less accessible in low- and middle-income countries due to cost. Adjunctive therapies included posaconazole (22%) and surgical debridement in selected cases, such as functional endoscopic sinus surgery (41%) and craniotomy (4%). Multimodal treatment is considered the standard of care, yet surgical

access to CNS lesions is often constrained by anatomical and clinical considerations.[24,25]

Despite aggressive therapy, outcomes were poor, with an in-hospital mortality rate of 52%. Among survivors, neurological sequelae were common (30%), underscoring the high morbidity of this disease. Follow-up at three months revealed recurrence in nearly half (46%), with only 15% achieving full recovery as shown in figure 3. These figures are in line with existing literature reporting mortality rates of 40–80% for CNS mucormycosis, even in high-resource settings. The high recurrence rate in our series may reflect suboptimal antifungal penetration into CNS tissue, delays in diagnosis, and the difficulty of achieving radical surgical clearance.[26,27]

Our findings highlight several important clinical implications. First, early recognition and aggressive management of sinonasal mucormycosis in diabetic patients are crucial to prevent intracranial extension. Second, access to liposomal amphotericin B and advanced surgical interventions remains limited in resource-constrained settings, likely contributing to poor outcomes. Third, long-term neurological disability is common among survivors, suggesting a need for structured follow-up and rehabilitation services.

This study has limitations inherent to its single-center setting, including the small sample size and potential selection bias. Microbiological confirmation was not achieved in all patients, and follow-up data were incomplete for some cases. Nevertheless, this original study on CNS mucormycosis in diabetic patients from Bangladesh provides valuable insights into the clinical spectrum, management challenges, and outcomes in this high-risk population.

Conclusion

CNS mucormycosis in diabetic patients carries alarmingly high mortality and morbidity, with delayed diagnosis and limited treatment options being major contributors. Early suspicion, timely initiation of liposomal amphotericin B, combined surgical intervention, and strict glycemic control remain critical for improving outcomes. Future studies should focus on early biomarkers, advanced imaging strategies, and improving access to safer antifungal formulations in resource-limited settings.

Authors' contribution: Islam MR designed the study, reviewed the literature, and performed data analysis; Rahman T reviewed the literature and assisted with data analysis; Islam J, Khan MSK, Alam D, and Akter S collected data and reviewed the literature; Habib R reviewed the literature

Conflict of interest: Nothing to declare.

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