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Intracranial Mucormycosis: A Rare And Devastating Case Report And Review Of Literature

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Abstract:
Mucormycosis is an aggressive opportunistic fungal infection with high morbidity and mortality, particularly in immunocompromised individuals. We present a rare case of a 51-year-old male with newly diagnosed Type 2 Diabetes Mellitus and hypertension, who developed intracranial mucormycosis with a right temporal lesion. The patient underwent decompressive craniotomy and antifungal therapy, with histopathology confirming mucormycosis. This case emphasizes the importance of early recognition and aggressive management in central nervous system fungal infections, highlighting the need for prompt diagnosis and treatment to improve outcomes.

Keywords: *Mucormycosis, Fungal Brain Infection, Craniotomy, Type 2 Diabetes Mellitus*

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I. Introduction:

Mucormycosis, formerly known as zygomycosis, represents a rare but highly aggressive form of fungal infection. It is caused by filamentous fungi belonging to the order Mucorales, including species such as *Rhizopus*, *Mucor*, and *Lichtheimia* [1]. These organisms are ubiquitous in nature, often found in soil, decaying organic matter, and even hospital environments. Despite being commonly present in the environment, they rarely cause disease in immunocompetent individuals. However, they become highly pathogenic in immunocompromised hosts, especially those with poorly controlled diabetes mellitus, diabetic ketoacidosis, neutropenia, hematologic malignancies, or prolonged immunosuppressive therapy, such as corticosteroid use. [2,3.]

The clinical presentation of mucormycosis varies depending on the anatomical site of involvement. The rhino-orbito-cerebral form is the most frequently reported, particularly among diabetic patients, while pulmonary mucormycosis is more common in hematological malignancy and transplant recipients. Other forms include cutaneous, gastrointestinal, disseminated, and less commonly, isolated cerebral mucormycosis[4]. The central nervous system (CNS) involvement is particularly uncommon and usually occurs secondary to local spread from adjacent paranasal sinuses or via hematogenous dissemination from a distant site.

Pathophysiologically, mucormycosis is characterized by rapid tissue invasion, with a hallmark feature being its Angio invasive nature. Upon inhalation or inoculation, the fungal spores invade blood vessels, leading to thrombosis, infarction, and extensive tissue necrosis[5]. This characteristic makes early diagnosis and intervention vital to preventing widespread systemic complications. In cases of CNS involvement, the clinical manifestations are often nonspecific and may include altered mental status, cranial nerve palsies, seizures, hemiparesis, or visual disturbances[6]. Such presentations can easily be mistaken for other neurological disorders, making diagnostic delays common.

Due to its aggressive nature, mucormycosis requires immediate treatment with antifungal agents—most notably liposomal amphotericin B—as well as surgical debridement of necrotic tissues wherever feasible. Delay in initiating appropriate therapy significantly worsens prognosis, often leading to fatal outcomes[7]. Therefore, high clinical suspicion, especially in high-risk individuals, combined with prompt imaging and histopathological confirmation, is essential to ensure timely diagnosis and intervention.

II. Clinical Case:

A 51-year-old male presented to the emergency department of a tertiary care center with symptoms as sudden-onset shortness of breath, generalized body weakness, slurring of speech, and acute right eye vision loss. The patient had a history of hypertension for the past three months and was recently diagnosed with Type 2

Diabetes Mellitus (T2DM). There was no prior history of steroid use, immunosuppressive drugs or known malignancy.

Upon initial examination, the patient was drowsy but arousable and disoriented with Glasgow Coma Scale (GCS) E2V1M4. His vitals were within normal limits , but neurological examination revealed right-sided upper motor neuron facial palsy and right eye papilledema. The patient was admitted , evaluated and investigated . Non contrast computed tomography (NCCT) of the head showed a well-defined heterodense lesion measuring approximately 3.5 × 3 × 3 cm in the right medial temporal region. Surrounding perilesional hypodensity suggested significant cerebral edema. NCCT PNS (paranasal sinuses) included mucosal thickening in the bilateral maxillary and ethmoidal sinuses, which raised suspicion of an underlying infectious etiology. To further delineate the nature of the lesion, a contrast-enhanced magnetic resonance imaging (CE MRI) of the brain was done which reveals large ill-defined lesion around 6.1 x 5.3 x 5.7 cm showing T2 heterogeneously hyperintense, T1 iso to hypointense and on FLAIR intermediate signal showing few cystic areas within noted in right temporal lobe including right perisylvian region, corona radiata on right side and right insular cortex. Few areas of haemorrhage are noted within with patchy and confluent areas of diffusion restriction within. On post-contrast study, no obvious enhancement is seen. Mild to moderate surrounding edema with mass effect is seen in the form of effacement of adjacent sulcal spaces, right lateral ventricle and third ventricle with midline shift measuring approximately 4.8 mm towards left side. Multiple areas of recent infarcts in right ganglio-capsular region, corona radiata on right side, pons laterally on right side and posteriorly and right cerebellar hemispheres (Fig 1). Routine laboratory investigations were within normal range. However, inflammatory markers such as ESR and CRP were elevated, consistent with an infective-inflammatory pathology.

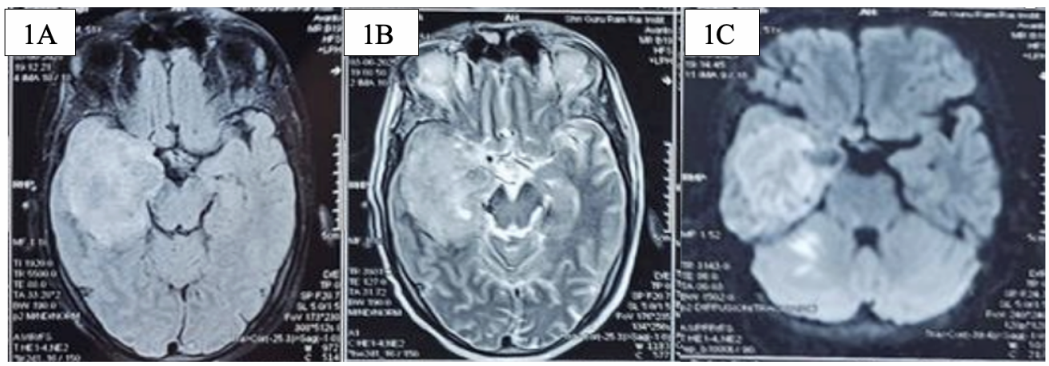

FIG 1 : CEMRI BRAIN (1A) T1w axial image showing large iso to hypointense ill regular marginated area around 6.1*5.3*5.7 cm in the right temporal lobe including right perisylvian region and insular cortex . (1B) T2w image hetergenously hyperintense lesion (1C) On DWI diffusion restriction is noted with few areas of hemorrhage

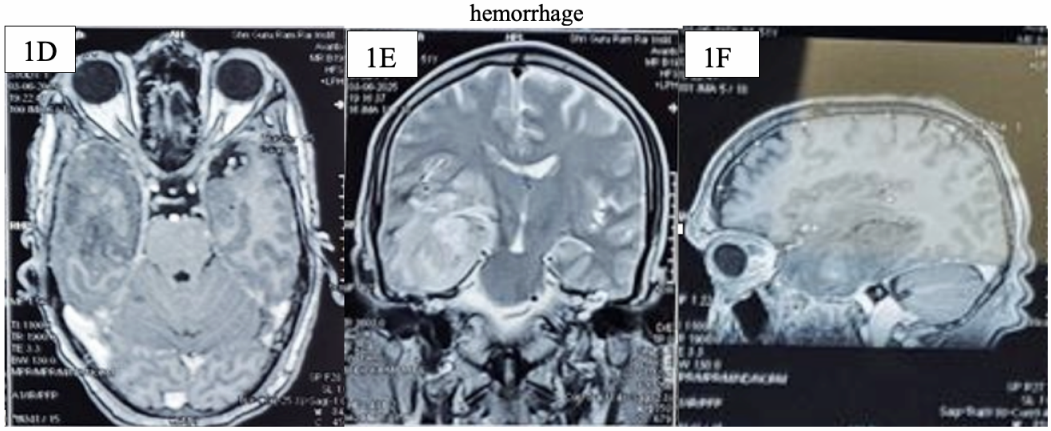

FIG 1 : (D,E,F) On Post Contrast axial , coronal and saggital image showing no obvious contrast enhancement .

The patient was taken up for surgery Right Fronto-Temporo-Parietal Craniotomy with microsurgical excision of lesion under general anesthesia (fig 2) .Intraoperatively, grey-black necrotic tissue was noted in the lesion site, extending to the tentorial incisura and adjacent middle cerebral artery territory . The entire necrotic mass was carefully excised and thorough debridement of devitalized brain tissue was performed.

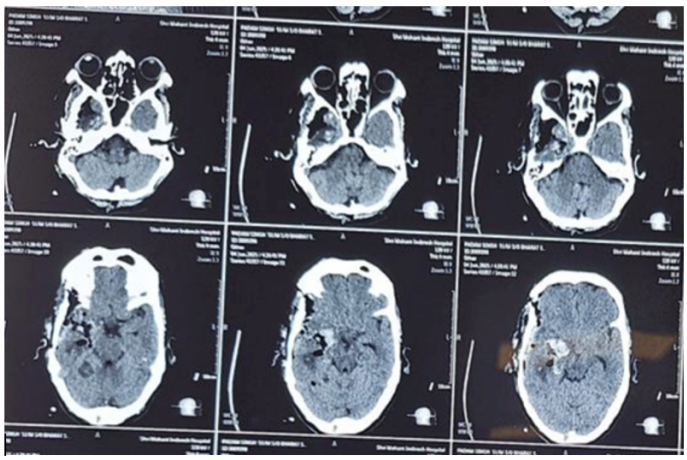

FIG 2: Post op NCCT Head image showing craniotomy defect with replaced bony flap is seen in the right fronto parieto temporal region with associated post operative changes .

The excised specimen was sent for histopathological examination. Histopathological analysis of the excised tissue revealed broad, aseptate fungal hyphae with right-angled branching, surrounded by extensive areas of necrotic brain parenchyma, neutrophilic infiltration, and granulomatous inflammation (Fig3). The findings were consistent with a diagnosis of mucormycosis. No malignant cells were detected.

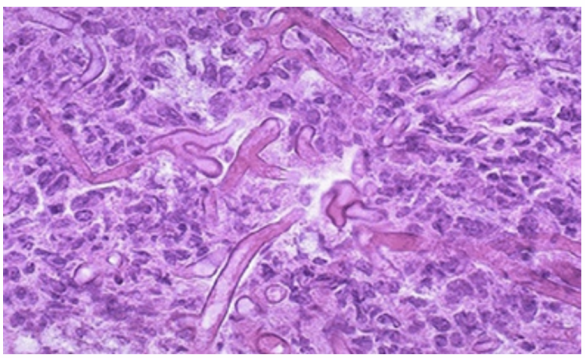

Fig3: Histopathology smear showing broad nonseptate hyphae of Mucormycosis

The patient was continued on ventilator support postoperatively in the neurosurgical ICU . On the fifth postoperative day, due to failure to wean off the ventilator, a tracheostomy was performed. The patient was started on antifungal therapy was initiated with liposomal amphotericin B at a dose of 5 mg/kg/day. Additional supportive therapy included IV fluids, broad-spectrum antibiotics, antiepileptics, anti-edema measures and blood transfusions were done. A multidisciplinary team comprising neurosurgeons, infectious disease specialists, intensivists, and ENT surgeons was involved in the patient's care. Despite intensive efforts, neurological improvement was minimal, and the prognosis remained guarded and patient expired on post op day 15th

III. Discussion:

Cerebral mucormycosis is an uncommon but highly lethal condition with diagnostic and therapeutic complexities. While mucormycosis more commonly affects the sinuses and lungs, its isolated or secondary spread to the CNS is exceedingly rare[8]. The rising incidence of mucormycosis in India, particularly post-COVID-19, has been attributed to rampant corticosteroid usage, uncontrolled hyperglycemia, and prolonged ICU stays[9]. However, this case was unrelated to COVID-19 and highlights the inherent risk posed by undiagnosed or poorly managed diabetes alone. Mucormycosis causes angioinvasion and tissue necrosis, as evidenced in this case by histopathological findings and the presence of infarcts on imaging[10]. Hyperglycemia impairs neutrophil chemotaxis and phagocytosis, while ketoacidosis facilitates iron availability, creating a conducive environment for fungal proliferation. Diagnosis often relies on neuroimaging, but definitive diagnosis requires tissue biopsy and fungal staining techniques such as GMS and PAS[11]. The cornerstone of therapy includes early and complete surgical debridement along with high-dose antifungal therapy. Liposomal

amphotericin B is the treatment of choice, while newer agents like posaconazole and isavuconazole are considered in refractory cases[12]. Verma *et al*[13] reported 30 cases of isolated cerebral mucormycosis, of which 17 were notable for history of intravenous drug abuse. Diabetes mellitus is the strongest risk factor for development of other mucormycosis infections, and in the meta-analysis of 929 cases mentioned above, 36% of patients with mucormycosis had a history of diabetes. It is believed that diabetic patients are susceptible to mucormycosis due to the fungal ketoreductase system allowing the organisms to metabolize ketone bodies. In addition, the hyperglycemia and acidosis often found in the setting of diabetes reduce neutrophil chemotaxis and adhesion to fungal hyphae as well as impair the inhibition of Mucorales spores and mycelia by alveolar macrophages[14]. In the present case, the patient had a history of diabetes mellitus, carried an increased susceptibility to mucormycosis infection due to these immunosuppressive mechanisms. Cerebral mucormycosis often manifests with a range of symptoms, including orbital and neurological signs, as well as headache. Systemic symptoms like fever frequently accompany neurological deficits. Our patient's presentation with headache, hemiplegia, and fever is consistent with reported cases. A review of 13 isolated cerebral mucormycosis cases found altered mental status (54%) and headache (51%) to be common symptoms. Considering the presenting symptoms of our patient and those of patients discussed in the literature, it is apparent that these nonspecific signs and symptoms are largely responsible for the challenges faced by clinicians in diagnosing cerebral mucormycosis, as many other conditions must be considered in the differential diagnosis. The treatment of patients with mucormycosis is challenging, and even in adequately treated patients, the associated mortality rate is still high. Standard treatments include AmB and its lipid formulation and surgical debridement[16,17]. In most retrospective studies, AmB and its lipid formulation are the preferred treatment and are part of any potential combination therapy. Posaconazole in combination with AmB is reported to offer treatment benefits when compared to antifungal monotherapy[18]. In some reports, posaconazole may be a more advantageous treatment strategy when used as the first line treatment for cerebral mucormycosis in diabetic patients with antifungal-resistant infections[19,20]. In our patient, surgical intervention and amphotericin B therapy were promptly initiated, but the advanced stage of disease and delayed neurological presentation significantly impacted prognosis. Hence we reviewed with literature which emphasize the need for heightened awareness, particularly in high-risk populations[13].Multidisciplinary coordination, timely radiological evaluation, aggressive surgical excision, and prolonged antifungal therapy are all essential to improving outcomes in such cases. A literature review suggests that the risk factors for death of patients with cerebral mucormycosis can be categorized as follows: (1) Susceptibility to infection (*e.g*., history of diabetes and long-term use of broad-spectrum antibiotics.(2) Delay in diagnosis and treatment (*e.g*., studies finding that a treatment delay of longer than 6 days[21] or 1 week[22] was associated with increased mortality).

IV. Conclusion:

This case reiterates the importance of maintaining a high index of suspicion for mucormycosis in diabetic patients presenting with new-onset neurological symptoms. Imaging modalities such as MRI play a pivotal role in identifying early cerebral lesions, but histopathology remains the gold standard for definitive diagnosis. Prompt surgical intervention coupled with aggressive antifungal therapy forms the mainstay of treatment. However, given the high mortality rate, particularly in cases with CNS involvement, early diagnosis remains the most critical determinant of survival. Written informed consent was obtained from the patient's legal guardian for publication of this case report and accompanying images.

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