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Case Report

Cerebral Venous Sinus Thrombosis Resulting from Mucormycosis Infection: A Case Report

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Abstract:

Background: *Mucormycosis is a rapidly progressing and deadly form of fungal infection. It poses a significant threat to patients with uncontrolled diabetes or other underlying systemic conditions. This infection can manifest in various forms, including rhino-cerebral, pulmonary, gastrointestinal, cutaneous, or disseminated types. The underlying conditions can affect the clinical presentation and often delay diagnosis, leading to poor outcomes. A black necrotic eschar is the defining characteristic of mucormycosis. Most cases are considered acute surgical emergencies.*

Case Details*: We report a case of rhino-cerebral mucormycosis in a 64-year-old diabetic patient who presented with sinusitis and occasional nasal bleeding. Gradually, he developed cavernous sinus thrombosis, superior sagittal sinus thrombosis, and bilateral frontal lobe infarcts. Unfortunately, the patient died due to delayed diagnosis and initiation of definitive treatment.*

Conclusion*: Early diagnosis is crucial for survival and the reduction of complications. Clinician awareness, swift treatment initiation, and timely surgical intervention are effective strategies for managing the disease.*

Keywords: *Cerebral Venous Sinus Thrombosis, Mucormycosis, Case Report*

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Introduction:

Mucormycosis is caused by infection with fungi from the order Mucorales. Rhizopus species are the most common causative organisms.[1,2] Saprophytic aerobic fungi from the class Phycomycetes (order Mucorales) cause rhinocerebral mucormycosis, also known as phycomycosis. Phycomycetes are ubiquitous and are commonly found in decaying vegetation, soil, and bread mold. They grow rapidly and can release large numbers of airborne spores. Rhizopus has an enzyme called ketone reductase, which allows it to thrive in high-glucose, acidic conditions. Serum from healthy individuals inhibits the growth of Rhizopus, whereas serum from individuals in diabetic ketoacidosis stimulates growth.[3] Rhinocerebral and pulmonary mucormycosis are acquired by inhaling spores. In healthy individuals, cilia transport these spores to the pharynx, where they are cleared through the gastrointestinal tract. In susceptible individuals (e.g., those with diabetic ketoacidosis or neutropenia), spores germinate, hyphae develop, and usually begin in the nasal turbinates or the alveoli.[4] Mucormycosis is angioinvasive; thus, thrombosis and infarction occur in infected tissues.[5,6,7] A black necrotic eschar is the hallmark of mucormycosis.[8,9]

Case report:

Mr. X, a 64-year-old male with a history of uncontrolled diabetes mellitus, hypertension, and chronic kidney disease, was admitted to Square Hospital Ltd. in Dhaka with complaints of severe headache, nausea, and vomiting for three days, along with occasional nasal bleeding for the past 10 to 15 days. Upon examination, the patient exhibited signs of cavernous sinus thrombosis, including proptosis and congestion of the left eye; fundoscopy revealed papilledema.

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Subcutaneous low-molecular-weight heparin was initiated. An ENT consultation was sought for the nasal bleeding. Fibre optic video nasoendoscopy was performed, revealing a fleshy, granular, blackish mass at the anterior end of the left nasal cavity in the middle turbinate. A punch biopsy was taken and sent for histopathology, which revealed necrotic tissue containing numerous yeasts and hyphae of fungi. Gomori methenamine silver staining was positive for mucormycosis. Anidulafungin was initiated; however, the patient's condition continued to deteriorate. He was transferred to the ICU due to an altered level of consciousness and convulsions. MRI of the brain and MRV demonstrated features of cavernous sinus thrombosis, sagittal sinus thrombosis, and a frontal lobe infarct with hemorrhagic transformation. IV Amphotericin B was commenced, but unfortunately, the patient passed away.

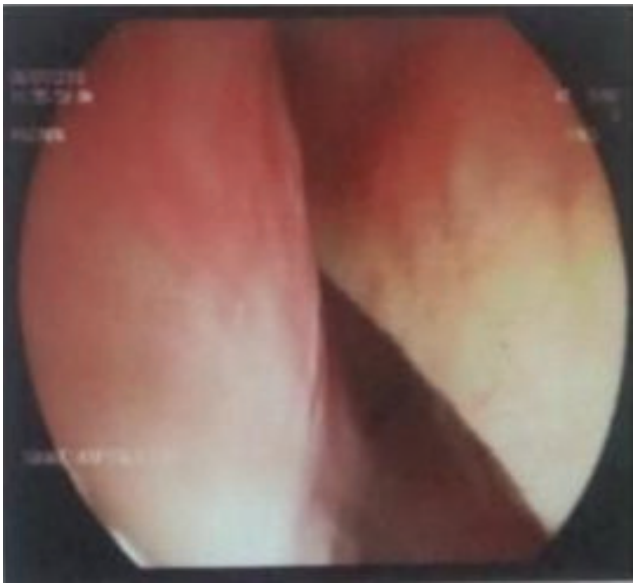

Fig.-1: *Normal right nasal cavity*

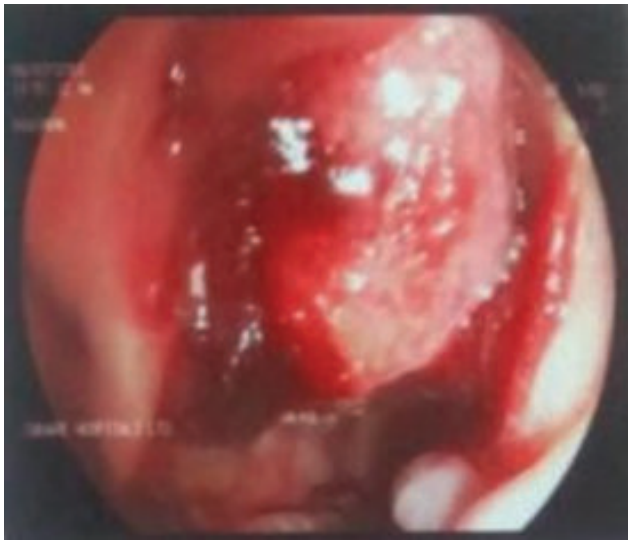

Fig.-2: *Left nasal cavity showing fleshy mass lesion*

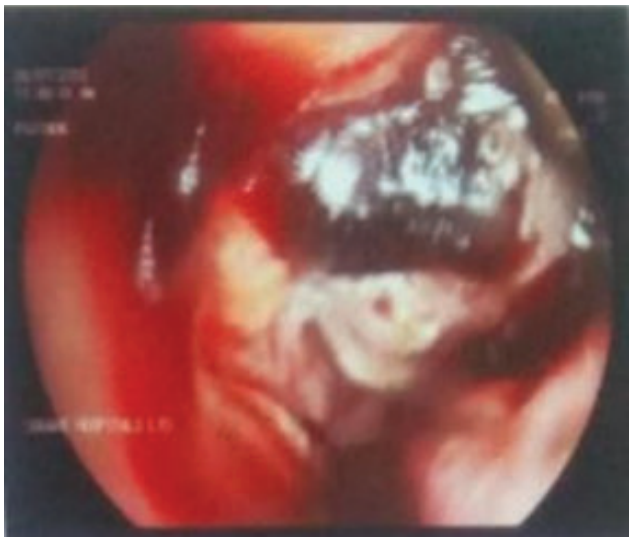

Fig.-3: *Left nasal cavity with blackish lesion suggestive of mucormycosis*

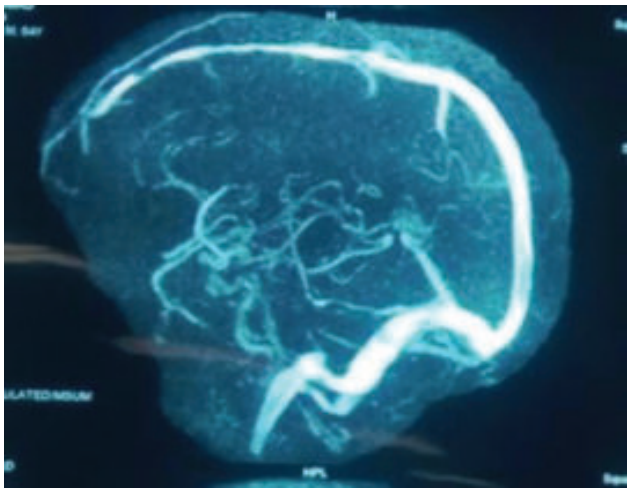

Fig.-4: *showing features of sagittal sinus thrombosis*

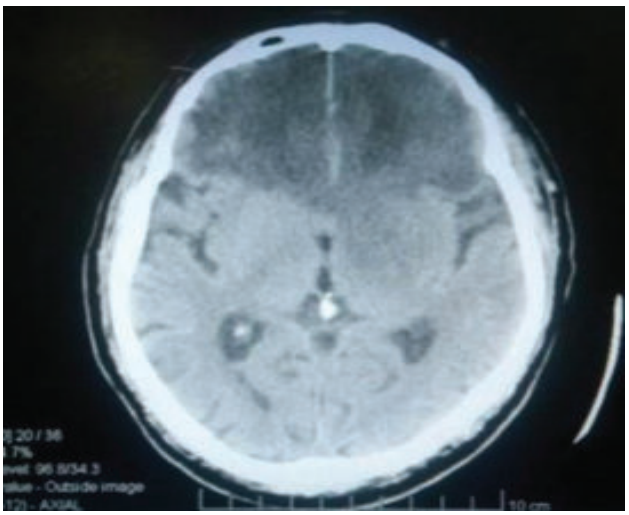

Fig.-5: *CT scan of the brain showing bilateral frontal lobe infarct*

Discussion

Mucormycosis is one of the most aggressive and lethal invasive mycoses.[7] The predisposing factors for mucormycosis are uncontrolled diabetes (particularly in patients having ketoacidosis), malignancies such as lymphomas and leukemias, renal failure, organ transplant, immunosuppressive therapy, deferoxamine administration, cirrhosis, and AIDS.[10] Mucorales species are vasotropic, causing tissue infarctions, and the mucormycosis spectrum ranges from cutaneous, rhinocerebral, and sinopulmonary to disseminated and frequently fatal infections, especially in immunecompromised hosts.[11] In diabetic patients, especially with elevated blood sugar levels, the spores germinate, hyphae develop, fungi begin to invade tissues, thrombosis occurs, resulting in tissue necrosis and fungi continue to grow in this devitalized tissue causing further damage to surrounding tissues.[12] Rhinocerebral mucormycosis (ROCM) is the most common form of mucormycosis in patients with diabetes mellitus. The invading fungus may spread inferiorly to invade the palate, posteriorly to invade the sphenoid sinus, laterally into the cavernous sinus to involve the orbits, or cranially to invade the brain, which can prove fatal.[13]

The infection typically presents as acute sinusitis, characterized by fever, nasal congestion, purulent nasal discharge, headache, and sinus pain. All of the sinuses become involved, spread to contiguous structures, such as the palate, orbit & brain, and usually progresses rapidly. The hallmarks of spread beyond the sinuses are tissue necrosis of the palate resulting in palatal eschars, destruction of the turbinates, perinasal swelling, and erythema & cyanosis of the facial skin overlying the involved sinuses &/or orbit. A black eschar, which results from necrosis of tissues after vascular invasion by the fungus, may be visible in the nasal mucosa, palate, or skin overlying the orbit.[14,15] Our patient had uncontrolled diabetes, which is a well-known predisposing factor for mucormycosis, along with the spread of the lesion from the palate into adjacent tissues.

A definitive diagnosis of mucormycosis can be made by tissue biopsy for direct microscopy or histopathological examination, or by culture on Sabouraud's dextrose agar that identifies the characteristic hyphae. The hyphae are broad, irregularly branched & rarely have septations. The detection of aseptate hyphae with right-angled branching is pathognomonic.[16] In contrast to the hyphae of Aspergillus, which are narrower, exhibit regular branching & have many septations. The lack of regular septations may contribute to the fragile nature of hyphae & the difficulty of growing the agents of mucormycosis from clinical specimens.

Treatments of mucormycosis need to be fast and aggressive because by the time even the presumptive diagnosis is made, often the patient has suffered significant tissue damage that cannot be reversed. The treatment involves reasonable control of blood sugar with reversal of diabetic ketoacidosis, early initiation of amphotericin B therapy and surgical debridement.[17,18]

Intravenous liposomal amphotericin B is the drug of choice for initial therapy.[19] The usual starting dose is 5mg/kg daily & may be increased to 10mg/kg daily in an attempt to control this infection. Posaconazole or isavuconazole is used as step-down therapy for patients who have responded to amphotericin B. Patients usually need antifungal treatment for an extended period (weeks to months), depending on the severity of the disease.[20]

Aggressive surgical debridement of involved tissues should be undertaken as soon as the diagnosis of any form of mucormycosis is suspected. In the case of rhinocerebral infection, debridement to remove all necrotic tissue will often be disfiguring, requiring removal of the palate, nasal cartilage, and the orbit.

Despite early diagnosis & aggressive treatment, the prognosis for recovery from mucormycosis is poor. The factors associated with poor survival in ROCM include delay in diagnosis and treatment, hemiparesis, bilateral sinus involvement and facial necrosis.[21]

Conclusion

Rhinocerebral mucormycosis is a rare disease with a high mortality rate. It should be included in the differential diagnosis of patients presenting with sinusitis, pain and oedema of the face, facial paraesthesia and facial paralysis with risk factors such as diabetes, haematological malignancies, prolonged corticosteroid intake or renal failure. Earlier recognition of the condition, prompt reversal of risk factors, adequate control of blood sugar, early initiation of medical therapy and meticulous surgical debridement may improve patient outcome.

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