International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 17s, 2025
https://theaspd.com/index.php

Mucormycosis - A Case Report Of A Disease Of An Environmental Concern

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ABSTRACT: *Mucormycosis is a rare menacing fungal infection which affects the maxillary jaw more often. A potentially fatal invasive fungal infection, mucormycosis also known as zygomycosis or phycomycosis affects individuals who are immunocompromised such as poorly managed diabetes, hematological malignancies such as lymphoma, blood cancer, and organ transplant patients are traditional risk factors for mucormycosis. It is an angioinvasise disease caused by saprophytic fungi. Mucormycosis has a pronounced propensity to penetrate blood vessels, which can lead to thrombosis, necrosis, and tissue infarction.*
KEYWORDS: *Fatal fungal, Black fungus, Mucormycosis ,diabetic mellitus.*

INTRODUCTION: Tissue infarction and necrosis are hallmarks of the angioinvasive disease mucormycosis.Clinical symptoms of mucormycosis, such as diffuse mucormycosis, pulmonary, gastrointestinal, cutaneous, and rhino-orbital-cerebral (ROCM) presentations, are classified based on anatomic location (1).Based on its anatomical location, mucormycosis can be divided into six types: (1) rhino-orbital-cerebral mucormycosis (ROCM); (2) pulmonary; (3) cutaneous; (4) gastrointestinal (GI); (5) diffuse; and (6) mucormycosis of rare sites. The most common clinical presentation in diabetics is ROCM. On the other hand, disseminated and pulmonary illnesses are the most common problems among organ transplant recipients or patients with hematological malignancies (HemeM). Treatment results are significantly worse when treatment is postponed, even by a few days, because mucormycosis can spread rapidly (2).

Globally, the incidence rate of mucormycosis ranges from 0.005 to 1.7 cases per million.

An estimated 140 cases of mucormycosis per million people are reported in India, which is around 80 times more common than in developed nations. India is thought to have a 70-fold higher prevalence of mucormycosis than the rest of the world.

CASE REPORT: A 47-year-old female patient came to our oral and maxillofacial surgery department with a chief complaint of swelling in the right midface for past 1 month. There was no pain and history of dental extraction 5 years ago which was uneventful.

She had a mild asymmetry in the face on the right side, the skin over the midface was normal. There was mild tenderness on palpation on left side midface and the temperature over the skin was normal. The lymph nodes were not palpable. Paresthesia of infraorbital area was noticed on right side. The facial expressions were normal(**figure:1**).

Intraoral examination revealed mild diffuse discoloration on the entire palatal mucosa on extending from palatal aspect of 13 to 23. On palpation, the palatal mucosa was flabby and soft. There was no pus discharge. Tooth mobility was seen in relation to 1st quadrant and the maxillary alveolar bone was necrosed and exposed in relation to 1st quadrant.(**figure:2**).

CT revealed involvement of the maxillary sinus, and other sinuses like Frontal sinus and ethmoid air cell was not involved. There were observations of bone erosions on the right maxillary sinus walls extending towards zygoma bone involving zygomatic arch as well. Early involvement of the maxillary sinus sinuses was seen on CT scans as mucosal thickening, typically without air or fluid levels. There was evidence of bone loss / erosion on the right side of the maxilla extending upto zygomatic arch on 3DCT images. The CT scan revealed polypoidal thickening of the mucosa, as well as hyperdense foci and sinus wall rosions in the right maxillary sinus. The CT scan showed well defined porous erosions on zygomatic bone extending upto the zygomatic arch(**figure:3,4**).

Surgical Protocol:

The patient was referred to ENT department for FESS(Functional Endoscopic Sinus Surgery) to get the maxillary mucosal growth removed. This was done a week prior to the actual date of surgery.

Under General Anaesthesia the patient was intubated (nasotracheal intubation), Weber ferguson incision was given on the right side of the face. The incision was marked on the right side extending from upper lip to lower infraorbital margin. Using black silk the Superior labial artery was ligated. Incision was given using scalpel and blade No.15 and then the full thickness mucoperiosteal flap was raised to expose the necrotic bone(figure 5). Using round and 701 bur the necrotic bone was removed under copious irrigation until fresh bleeding was induced. The full thickness flap was sutured back(figure 7). The excised tissue was sent for histopathological examination and KOH mount (figure 6). The reconstruction to reconstruct the defect as the patient was not willing.

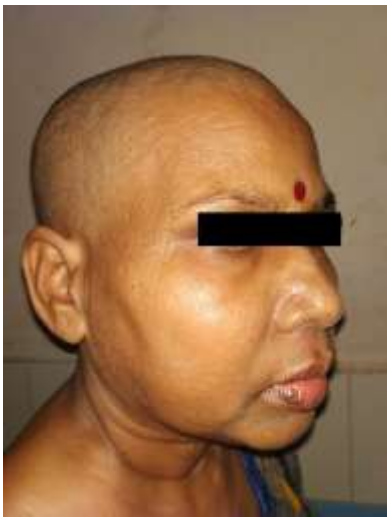

Figure 1: Extra oral right sided midface swelling

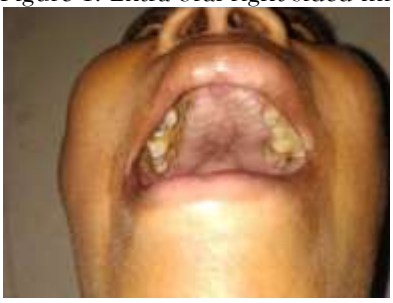

Figure 2 : Intra oral flabby palatal mucosa along with

Figure 3 : Bone erosions noted on right side of maxilla in 3D CT Scan

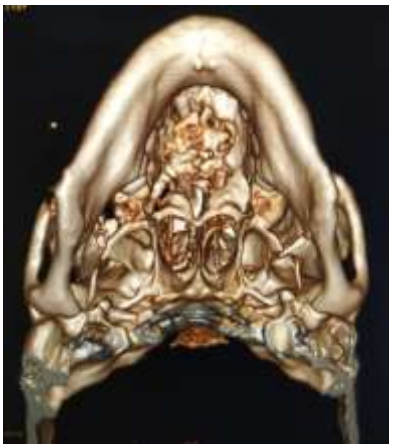

Figure 4 : Bone erosions on the palatal aspect

Figure 5 : Intra operative after reflecting full thickness flap

Figure 6 : Excised necrosed maxilla along with teeth

Figure 6 : Post operative after removing necrosed bone

Figure 7 : Post operative suturing

DISCUSSION:

Mucormycosis, a disease that belongs to the phycomycetes class is a deadly fungal disease caused by a saprophytic fungus. It's a severe opportunistic disease. Rhizopus accounts for 90% of cases of mucormycosis. This microbe can be grown in the throat, nasal passages, oral cavity, and feces of healthy people who do not show any clinical signs of infection (4). Inhaling sporangiospores can result in a mucormycosis infection. This infection is particularly common in individuals receiving iron chelation therapy, diabetics experiencing frequent episodes of ketoacidosis, burn and trauma patients, immunocompromised cancer patients, and chemotherapy recipients (5).

Adequate pathogen clearance is hampered by local variables such as dehydration, mucus stasis, and retentive anatomical niches. Laminin and collagen IV are exposed by tissue damage in diabetes or during cytotoxic chemotherapy, which binds mucoralean spores and makes evacuation difficult, causing internalization and germination [8]. Invading host cells with lytic enzymes and poisons including mucoricin, germinated spores cause necrosis and tissue thrombosis [9, 10]. In healthy people, infection is avoided by platelets and innate and adaptive immunity [11]. Epidermal growth factor pathways protect against mucormycosis and preserve the integrity of skin and mucocutaneous tissue [8].

There are two possible causes of oral mucormycosis. One is from direct wound contamination, which often causes infection to spread to other viscera, and the other is from a diffused infection, in which the nose acts as the entrance site. Palatal ulceration and necrosis can result from infections that start in the nose or PNS; the affected area usually looks black. When the infection spreads because of direct wound contamination, clinical symptoms may appear anywhere in the mouth cavity, including the mandible. One important difference between mandibular and maxillary infections is cavernous sinus thrombosis. This devastating outcome could be caused by a maxillary infection (7).

The gastrointestinal, disseminated, and miscellaneous forms are less common than the rhinocerebral, pulmonary, and superficial cutaneous forms in clinical presentations (8).Patients with uncontrolled diabetes mellitus are most frequently affected by the rhinocerebral (rhinomaxillary) form of infection (9).Rhinorebral mucormycosis can cause low-grade fever, headache, facial pain, edema, and malaise, among other clinical signs. The disease typically begins in the nasal mucosa or palate and progresses to the paranasal sinuses, followed by the angular, lacrimal, and ethmoidal vessels, among other vessels in the vicinity. Moreover, the retro-orbital region may potentially be directly affected by mucormycosis (10).If fungal hyphae reach the bloodstream and travel to other organs, such as the brain or lungs, they can be fatal to the patient (11).

Type of Mucormycosis	Some common symptoms
Rhinocerebral (sinus and brain) mucormycosis	Facial swelling Exposed bone Tooth mobility Nasal congestion Black lesions on nasal bridge or upper inside of mouth that quickly become more severe Fever
Pulmonary mucormycosis	Fever

	Severe Cough Chest pain Shortness of breath Hemoptysis
Cutaneous mucormycosis	Blisters Ulcers Blackish discoloration
Gastrointestinal mucormycosis	Abdominal pain Nausea and vomiting Gastrointestinal bleeding

Table 1: Types of Mucormycosis

DIAGNOSIS OF MUCORMYCOSIS:

Mucormycosis is diagnosed histopathologically and on KOH mount. Samples of tissues were inspected macroscopically, processed according to standard procedures, and routinely stained with Hematoxylin & Eosin (H&E). Periodic Acidic Schiff (PAS) and Gomori Methenamine Silver (GMS), two specialized fungus stains, were used to highlight the fungal cell wall and to confirm and/or distinguish the fungal species. A high index of suspicion is crucial because the condition is uncommon. Histological characteristics such as non-pigmented, broad (5–20 μm), thin-walled, ribbon-like hyphae with pauci septations or aseptate, and right angle branching were used to identify the genus Mucorales. A diagnosis is made by identifying risk factors, evaluating clinical symptoms, using imaging modalities as soon as possible, and starting diagnostic procedures based on advanced molecular, histopathology, and culture approaches as soon as possible(23).

MANAGEMENT OF MUCORMYCOSIS:

Surgery:

Surgery was the definitive option for the management of rhino-oculo-cerebral infections, comprehensive surgical debridement is required under general anaesthesia, involving all necrotic areas until the vital bone is found (bleeding spot), with the help of ENT unit for debridement of mucosal polyps via FESS (Functional Endoscopic Sinus Surgery). Complete debridement under general anaesthesia with adequate follow up is mandatory(24,25). In extensive cases a second KOH mount can be performed by a swab test after 3 months post operatively to know if there is any fungal growth.

Antifungal medication:

The antifungal medications with in vitro action against mucorales were posaconazole, amphotericin B (Amb), and its lipid formulations. The discovery of isavuconazole has added to the arsenal of antifungal medications(26). Drug therapy always starts with Amphotericin B (Amb) IV 1mg/kg/day for 14 days and then posted for surgical debridement.

Posaconazole has demonstrated anti-mucorale action both in vitro and in vivo;300 mg as a loading dose and then followed by 100 mg once daily for 3 months as a post operative care. However, there is no information available regarding the utilization of posaconazole therapy as first-line treatment. As a result, after initiating treatment with L-Amb, posaconazole has a role in the therapeutic arsenal for prevention or consolidation. There hasn't been any research done on how well posaconazole tablets or intravenous formulations work to treat mucormycosis. Finally, despite adequate serum concentrations, incidences of mucormycosis have been documented in individuals using posaconazole prophylaxis(27).

CONCLUSION:

Mucormycosis is an aggressive fulminant invasive fungal infection that can occur in patients with a number of conditions, such as uncontrolled diabetes, renal failure, organ transplants, long-term corticosteroid and immunosuppressive therapy, cirrhosis, burns, and AIDS-related malignancies such as lymphomas and leukemias. It can be brought on by minor dental procedures, such as tooth extractions, in patients with diabetes. More work should be done to identify this sickness early and treat the patient at the earliest.

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