

MUCORMYCOSIS OF THE PALATE

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Abstract

Mucormycosis is an invasive, potentially lethal fulminant fungal infection that is caused by normally saprophytic fungus belonging to the class zygomycetes. Mucormycosis is characterized by its unrelenting progression towards vital organs with marked propensity towards arterial wall by direct extension producing vascular thrombosis leading to ischaemic necrosis. The maxilla rarely undergoes necrosis due to its rich vascularity. The case report here was a case of mucormycosis with extensive necrosis of maxilla in a 48 year old male patient with palatal perforation as the sole presentation of mucormycosis.

Key words: Mucormycosis, palatal perforation, fungal infection.

Introduction

Organisms of the class zygomycetes as documented by Ribes et al were first noted to cause disease in humans since 1800¹. First description of zygomycosis was reported by Platauff in a paper entitled "Mycosis Mucorina" in 1885^{1,2,3}. Mucormycosis is an opportunistic, fulminant fungal infection that is caused normally by saprobic organisms of class zygomycetes, including genera such as Absidia, Mucor, Rhizomucor and Rhizopus^{1,2}. Infection usually results from inhalation of spores through nose or mouth⁴. The fungus invades the blood vessels and subsequently spreads through them. Once fungal hyphae enter into the blood stream they can disseminate to other organs such as cerebrum or lungs which can be fatal for the patient⁵.

Case Report

A 48 year old male patient reported to the department of Oral Medicine and Radiology with the chief complaint of ulceration on the palate with nasal regurgitation of food since 15 days. History revealed that ulcer was on posterior part of palate which gradually increased in size and got perforated. He also gave history of intermittent fever for past 6 months.

On general physical examination patient was moderately

built, well nourished and febrile. Extraoral findings revealed bilateral submandibular lymphadenopathy and discharge from the nose (Fig -1).



Fig -1: Patient profile with nasal discharge.



Fig- 2: Lesion on palate measuring 5 x 4 cm

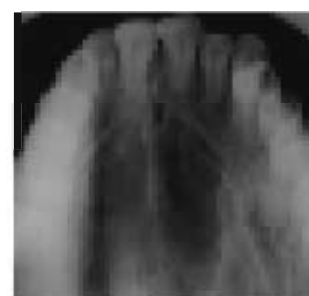


Fig- 3: Maxillary occlusal radiograph shows solitary radiolucency present on midline.

Intraorally, solitary deep burrowing oval shape ulceration at the posterior part of the hard palate which was perforated measuring 5x4 cm was noticed. The ulcer was necrotic, tender with a tendency to bleed and was covered with slough (Fig-2). Based on history and clinical examination, we arrived at a provisional diagnosis of midline lethal granuloma with the differential diagnosis of mucormycosis, Wegener's Granulomatosis, malignant ulcer, syphilitic gumma and tuberculous ulcer.

Routine hematological investigations revealed that hemoglobin was 10.7 gm % and erythrocyte sedimentation rate (ESR) was raised. VDRL (Venereal Disease Research Laboratory) test, Mantoux test and ELISA test for HIV (Human Immunodeficiency Virus) was negative. Maxillary occlusal

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radiograph showed solitary radiolucency in midline extending from incisive foramen to the most posterior region (Fig-3). Para nasal sinus radiograph showed haziness on right side of maxillary sinus (Fig-4). Computed tomography scan showed mild deviated nasal septum towards left side, diffuse asymmetrical mucosal thickening of bilateral maxillary

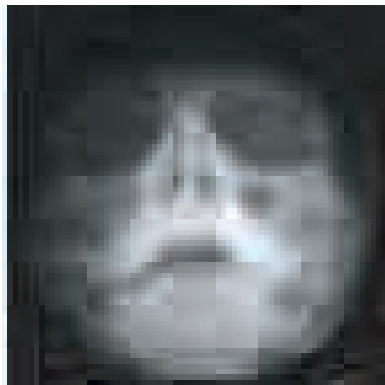


Fig- 4: Para nasal sinus radiograph shows haziness present on right side maxillary sinus.

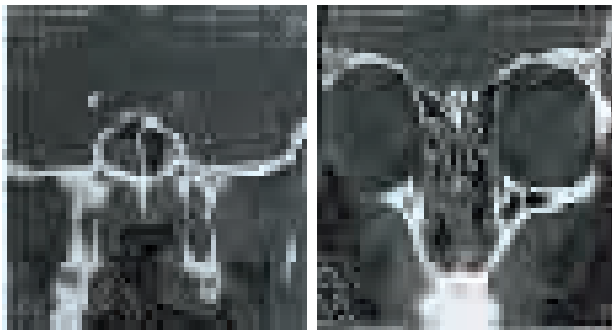


Fig - 5, 6: CT scan shows mild deviated nasal septum towards left side, diffuse asymmetrical mucosal thickening of bilateral maxillary sinus and destruction of posterior part of hard palate.

sinus and destruction of posterior part of hard palate (Fig-5,6). An incisional biopsy was performed and on histopathological examination, the mucosal connective tissue exhibited areas of necrosis. Interspersed between collagen fibers and necrosed tissue/abundant numbers of non septate hyphae of varying lengths were observed (Fig-7). Based on investigation final diagnosis of Mucormycosis of palate was made and patient was immediately admitted in hospital for management.

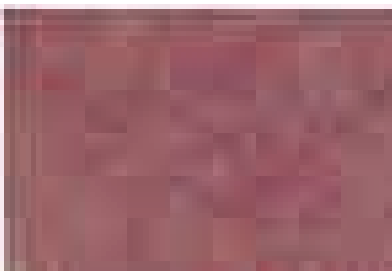


Fig: 7 Photomicrograph shows nonseptate hyphae branching at various angles in between fungal spores.



Fig-8,9: Fabrication of obturator was done.

Considering the seriousness of the disease under medical supervision, surgical debridement and antifungal therapy was instituted, Fabrication of obturator was done to cope up with oro-nasal regurgitation problem (Fig:8,9). But, even after providing proper medical and dental treatment the patient was demised uneventfully.

Discussion

Mucormycosis is an opportunistic, rapidly progressing and lethal form of fungal infection in human^{1,6,7}. It is caused by saprophytic fungus that occurs in soil or as a mould on decaying food⁸. Fungus is non-pathogenic for healthy individuals and can be cultured regularly from nose, throat and oral cavity representing opportunistic rather than true pathogen⁹. It has no geographic distribution, specific racial, sex or age predilection². Zygomycosis presents as a spectrum of diseases, depending on the portal of entry and the predisposing risk factors of the patient. The 5 major clinical forms are as follows: the most common type, rhinocerebral type, followed by pulmonary type, gastrointestinal type, cutaneous type and disseminated type^{1,7}. Rhinocerebral mucormycosis represents one-third to half of all cases of mucormycosis as reported by Pillsbury HC et al¹. Rhinomaxillary form of disease is a subdivision of the rhinocerebral form, which begins with the inhalation of fungus by a susceptible individual⁹.

Mucormycosis have been reported to occur in otherwise healthy individuals with various predisposing medical conditions which may also be responsible, such as uncontrolled diabetes, blood dyscrasias, Protein calorie malnutrition, corticosteroids and immunosuppressive therapy etc^{1,5}. Of these, Type I diabetes mellitus is most common and is associated with 40% of mucormycosis cases as documented by Tryfon et al⁴. Joseph TP et al reported that early stages of disease exhibits facial cellulitis, anaesthesia, nasal discharge, necrotic turbinates, fever, headache and weakness^{5,8}. Mucormycosis is characterized by angioinvasion, thrombosis, infarction and necrosis of involved tissue¹. Fungus invades arteries and causes damage secondary to thrombosis and ischaemia⁹.

Jones AC et al stated that the most common oral sign was ulceration of palate, which resulted from necrosis due to invasion of the palatal vessel^{1,9}. Extension from the sinuses into the mouth caused painful, black necrotic ulceration in the hard palate¹. Greenberg MS et al reported lesion was characteristically large and deep causing denudation of underlying bone⁹. In the present case all signs and symptoms were seen except cellulitis, anaesthesia and headache.

Plain radiograph of sinuses and orbits may demonstrate sinus mucosal thickening, sinus opacification without fluid levels and spotty destruction of the bony walls of

the paranasal sinuses^{1,4}. CT scan or MRI may demonstrate erosion or destruction of bone or sinuses and helps delineate the extent of the disease¹. Diagnosis of mucormycosis is easily made on tissue sections. Involved tissues demonstrate focal areas of infection, necrosis, hemorrhage and presence of numerous large fungal hyphae which are non-septate, having ribbon-like or empty cellophane tubes appearance with angle branching ranging from 450-900^{1,2}.

Successful treatment of mucormycosis consists of aggressive surgical debridement of necrotic tissue, systemic antifungal therapy and control of any underlying disease process⁹. Antifungal treatment is based upon systemic high dose amphotericin B and has a number of serious side effects, and renal toxicity is an almost invariable complication of therapy⁴. Recently, intravenous liposomal amphotericin, intravenous lipid complex and hyperbaric oxygen therapy has also been used¹.

Conclusion

Mucormycosis is considered as a possible diagnosis in spontaneous necrotic soft tissue lesions of the palate. Although it occurs rarely, mucormycosis is characterized by a high mortality rate if it is not diagnosed and

managed appropriately. Early diagnosis and adequate management not only increases the chance of survival but also reduces the size of defect.

References

1. Jayachandran S , Kritika C. Mucormycosis presenting as palatal perforation. *Ind J Dent Res* 2006; 17 (3): 139-142.
2. Pandey A, George J, Rao NN. Mucormycosis causing extensive destruction of maxilla. *Ind J Dent Res* 2006; 15 (2): 74-77.
3. Jayasheela M, Amberkar VS, Denny CE, et al. Mucormycosis of the palatal tissues: Case Report. *Journal of Indian Academy of Oral Medicine & Radiology* 2003 Oct-Dec; 15(4): 162-164.
4. Tugsel Z, Sezer B, Akalin T, et al. Facial swelling and palatal ulceration in a diabetic patient. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2004; 98 (6): 630-6.
5. Auluck A. Maxillary necrosis by mucormycosis. A case report and literature review. *Med Oral Patol Oral Cir Bucal* 2007; 12: E360-4.
6. Huang JS, Kok SH, Lee JJ, Hsu WY, Chiang CP, Kuo YS.. Extensive maxillary sequestration resulting from mucormycosis. *British Journal of Oral and Maxillofacial Surgery* 2005 Dec;43(6):532-4. E pub 2005 Jul 15.
7. Leitner C, Hoffmann J, Zerfowski M, Reinert S. Mucormycosis : Necrotizing soft tissue lesion of the face. *J Oral Maxillofac Surg* 2003 Nov; 61(11): 1354-1358.
8. Jones AC, Bentsen TY, Freedman PD et al. Mucormycosis of the oral cavity. *Oral Surg Oral Med Oral Pathol Oral Radiol* 1993; 75(4): 455-60.
9. Greenberg MS. *Burket's Oral Medicine Diagnosis and Treatment*. 10th ed. Canada: BC Decker Inc;2003. p. 78-79.