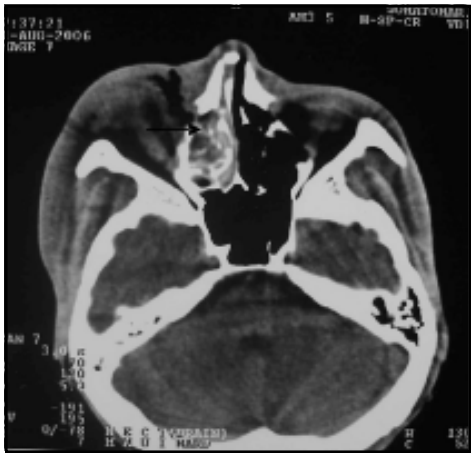

Fig-6.CT Scan: showing erosion of the nasal and orbital bone.

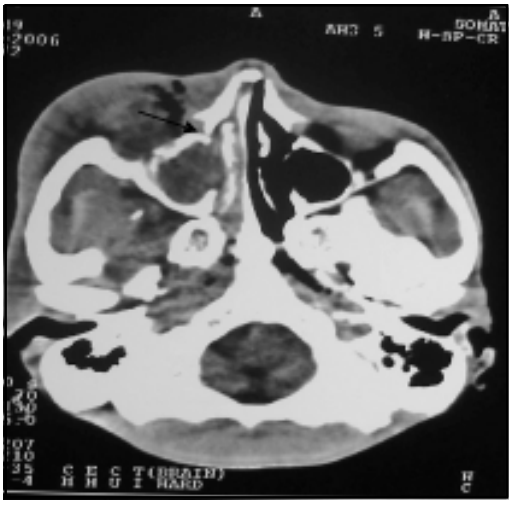

Fig-7. CT PNS: showing right-sided orbital involvement

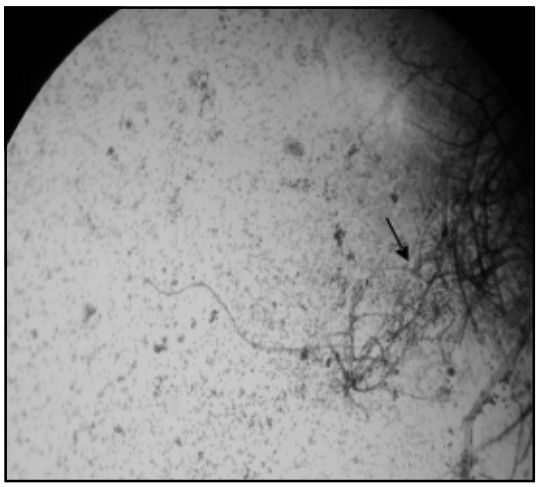

Fig-8. Direct microscopy from the pus shows broad non-septate hyline hyphae along with tissue material.

Fig-9. Culture of the pus & whitish materials on several Sabouraud dextrose agar, colonies shows floccose, dense and hairy appearance.

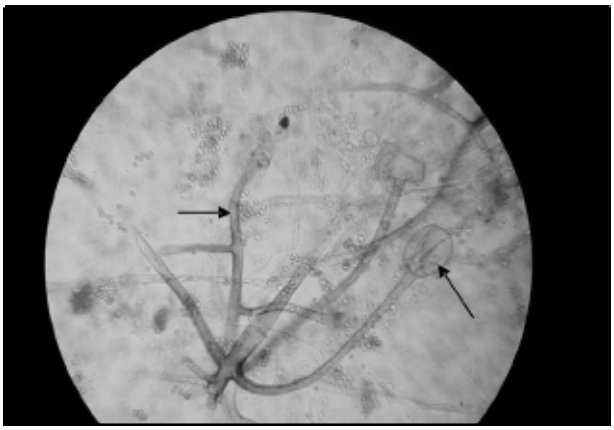

Fig-10. Culture form the Sabouraud dextrose agar under microscopy showed non-septate hyphae with rhizoids, sporangiophores and sporangia of Rhizopus species under microscopy.

Direct smear from the pus showed broad non-septate hyaline hyphae along with tissue material.

Culture of the pus on Sabouraud dextrose agar showed floccose, dense and hairy colonies.

Microscopic examination of the growth showed non-septate hyphae with rhizoids, sporangiophores and sporangia of Rhizopus species.

Discussion

Rhinoncerebral mucormycosis is rapidly progressive invasive fungal infection in a patient with immunological or metabolic compromised conditions. Number of deaths are increasing along with the rise in incidence of immunodeficiency and opportunistic infections, especially when recognized and treatment has been

delayed. Such infection is rare in routine practice. When one confronts with such cases, awareness will help to initiate immediate treatment.

Upper airway mucormycosis was first described in 1885 by Paltuf, who called it mycosis mucorina.(1) In 1943 Gregory *et al*, reported 3 patients of RCM with fatal diabetic ketoacidosis.(2) Cure of the disease was first reported in 1955 by Harris.(3) Mukharji *et al* reported three cases of mucormycosis with diabetes (4). Wattal C. *et al* in 2001 reported a case of mucormycosis (5).

The diabetic patients with ketoacidosis are disproportionately affected by mucormycosis. Rhizopus species have an active ketone reductase system and thrive in high glucose and acidotic conditions. These patients also have decrease phagocytic activity because of impaired glutathione pathway. Normal serum inhibits Rhizopus whereas serum of the diabetic ketoacidosis patients stimulates its growth.

Rhinocerebral mucormycosis begins with colonization of the nasal mucosa by air borne spores. Mucorales hyphae have a predilection for the growth into the artery and lymphatic systems. The fungi invade the nerves, fatty tissue and bone but muscles are usually spared. Angioinvation by the hyphae produces a fibrin reaction and the development of "mucor thrombi" which occludes the artery and lead to ischemia and infarction and consequence formation of black necrotic eschar of the skin and mucous which is characteristic of rhinocerebral mucormycosis. The infection spreads rapidly to adjacent sinuses and orbit and continue into the cranium via the ethmoid bone or orbital vessels (2). Our patient presented with ocular symptoms and CT scan revealed sinuses involvement without any evidence of CNS involvement; hence "Rhinoorbital mucormycosis" was the most acceptable terminology.

When clinical picture includes the presence of sinusitis with black discoloration in the nose and palate in addition to predisposing factors, a diagnosis of rhinocerebral mucormycosis should be highly suspected. A tissue biopsy is necessary to confirm the diagnosis. The microscopic examination of the nasal discharge or biopsy material in KOH wet mount shows characteristic irregular broad, aseptate ribbon-like hyphae (10-20µm across) with wide-angle or right angle (45-900) branching at irregular interval. These fungi are poorly stained with PAS,Gridely and Gram stain but very well seen in H & E and Groctt-Gomori methenamine silver stain.

Radiological finding are helpful in assessing the stages of disease rather than making a definite diagnosis as because in early stage radiological finding may be indistinguishable those of simple rhinosinusitis. Bony erosion is only the late feature of the disease.

The standard medical therapy for the rhinocerebral mucormycosis is amphotericin B in a dose of 1.0-1.5 mg/kg/day for a period of several weeks to several months depending on the clinical response and the degree of the drug side effect, especially nephrotoxicity. Less toxic form of amphotericin B such as liposomal amphotericin B, colloidal dispersion amphotericin B and amphotericin lipid complex may be safe(6). Other therapeutic modalities includes hyperbaric oxygen therapy and nasally nebulized amphotericin B.

The prognosis of the rhinocerebral mucormycosis depends on early diagnosis and resolution of the predisposing condition. Survival has been positively corrected with the time of diagnosis and initiation of treatment.

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