SINO NASAL MUCORMYCOSIS – CAUSING HEMIPLEGIA A CASE REPORT

D. Sridhara Narayanan\textsuperscript{1*}, Merlyn Ann Varghese\textsuperscript{2}, P. M. Hari\textsuperscript{3}

\textsuperscript{1}Associate Professor, Department of ENT, Sree Balaji Medical College and Hospital, Bharath University, Chennai, Tamilnadu.

\textsuperscript{2}MS Postgraduate, Department of ENT, Sree Balaji Medical College and Hospital, Bharath University, Chennai, Tamilnadu.

\textsuperscript{3}Professor and HOD, Department of ENT, Sree Balaji Medical College and Hospital, Bharath University, Chennai, Tamilnadu.

ABSTRACT

Fungal infection of paranasal sinuses is an increasingly recognized entity both in normal and immunocompromised individuals. Paranasal mycosis manifests as two distinct entities, a benign or noninvasive infection and more serious invasive infection which occurs in immunocompromised individuals and characterized by its rapid onset, ability to invade tissues and cause of destruction. Early diagnosis is vital in these infections because delay in initiation of treatment can be life threatening due to propensity of fungi to invade adjacent blood vessels and to embolize to distant organs including brain. In this article we report a rare case of sinonasal mucormycosis causing hemiplegia.

KEYWORDS: mucormycosis, hemiplegia.

INTRODUCTION

Fungal rhino sinusitis is being recognized and reported with increasing frequency over the last two decades worldwide. It occurs in two distinct forms - the fulminant invasive disease, which is predominantly seen in patients with some form of immunosuppression and the chronic fungal rhino sinusitis in apparently healthy hosts. Apart from the species of Aspergillus which is isolated from a majority of such cases, dematiaceous hyphomycetes, Pseudoallescheria boydii, candida, fusarium, hyalohyphomycetes and Zygomycetes are also reported. The changing terminology for mucormycosis and of its causative agents has
complicated data retrieval and confused clinicians. All the agents of mucormycosis belong to the order Mucorales.

Pathologically, mucormycosis is characterized by vascular invasion with hyphae, infarction and necrosis of tissue and by an acute or sub acute course. Because mucormycosis is encountered as a secondary disease or an opportunistic infection, the distribution of the various clinical forms is based on predisposing factors rather than age, sex, race or geography. Based on histopathological findings, five categories of fungal rhinosinusitis disorders are recognized, each having characteristic presentation. The two broad categories are Invasive and Non-invasive. The tissue invasive fungal rhino sinusitis can be of 3 types: Acute necrotizing, fulminant fungal rhinosinusitis, Chronic Invasive fungal rhinosinusitis and Granulomatous Invasive (Indolent) fungal rhinosinusitis. The non-invasive category are of 2 types: fungal ball (sinus mycetoma) and AFS (Allergic fungal sinusitis).

CASE REPORT
A 65-year-old female patient presented with history headache for 20 days and purulent nasal discharge for 20 days. She also had facial swelling for 7 days and nasal obstruction for 3 days. She is a known diabetic on irregular treatment. Patient was diagnosed to be hypertensive and was started on amlodipine. On examination there was tenderness over right maxillary sinus and black eschar on both nasal cavities. Oral cavity also showed black eschar with erosion of palate (Fig 1). Diagnostic nasal endoscopy showed black eschar in both nasal cavities. Diagnosis of mucormycosis was made and patient admitted in intensive care unit (ICU). Patient’s diabetes was brought under control by physician. There was no evidence of cavernous sinus thrombosis. ECG showed sinus arrhythmia and patient was put on Tablet metaproterenol sulfate (alupent).

Two dimension Echo was normal. Hematological investigations revealed hemoglobin: 14.5 g% and WBCs: 9800 cells/mm3. X-ray chest was normal. CT scan of para-nasal sinuses showed iso-dense, non-enhancing soft tissue swelling in right maxillary sinus, in right nasal cavity and in left nasal cavity with erosion of hard palate (Fig 2). Orbits were not involved. The patient was diagnosed as having chronic invasive fungal sinusitis. The patient was taken up for surgical debridement. The eschar was removed endoscopically and the perforation of nasal septum and palate was noted. Middle meatus was normal and the maxillary sinus mucosa was also normal. Ethmoids were not involved. Post-operatively, the patient was put on ciprofloxacin and insulin was continued. Following surgical debridement the patient
showed dramatic improvement. Histopathology report confirmed it as fungal sinusitis; the patient was started on amphotericin – B injection at dose of 1.5 gm I.V. on alternate days for 2 weeks. Hematological and renal profile along with other vital parameters was monitored. On the fourth post-operative day the patient developed left sided hemiplegia. Patient was treated for hemiplegia by the physicians. Patient’s condition improved and her hemiplegia was gradually recoved. She was discharged and is under follow-up

Fig 1. Black eschar with erosion of palate in oral cavity

Fig 2. CT nose and paranasal sinuses (PNS) showing isodense, non-enhancing soft tissue swelling in both nasal cavity and right maxillary sinus
Histopathology—mucormycosis
KOH mount revealed presence of broad aseptate hyphae (Fig 3). Fungal culture was done on Sabourads’ dextrose agar by incubating at 25°C. Growth was observed within 3 days of incubation. Dense cottony fluffy growth was observed which was initially white and then became grey. Lactophenol cotton blue mount of the fungus showed the presence of aseptate hyphae, rhizoids and sporangia containing brown spores. The isolate was identified as mucor (Rhizopus microsporum).

DISCUSSION
Mucormycosis is a fulminant and often fatal mycotic infection in human beings. It occurs in poorly controlled diabetes mellitus with ketoacidosis, leukemia, lymphoma, leukopenics, severe burns, renal diseases, carcinoma, severe cachexia, profound dehydration and septicemia in heroin addicts, children with severe sinusitis, immunosuppressed patients, recipients of renal transplants and supraphysiological doses of adrenal corticosteroids and azathioprine. The underlying disease influences the portal of entry and poorly controlled diabetes mellitus is the major condition in paranasal infections. A review of predisposing factors in 179 cases of paranasal sinus mucormycosis found that 126 patients had diabetes mellitus and only 8 had no known underlying disease. These fungi are found almost ubiquitously in soil, decaying vegetables, seeds, fruits, compost piles, animal excreta and old bread. The fungus invades the wall of the blood vessels, causing mechanical and toxic damage to the intima leading to thrombosis, later it invades the lymphatics and veins.[1] Mucormycosis starts in the nose and paranasal sinuses and spreads to the eye via the angular vein, lacrimal or ethmoid vessels as well as direct extension from the paranasal sinuses. Intracranial involvement occurs from invasion by way of superior orbital fissure, ophthalmic vessels,
cribriform plate and not uncommonly, through carotid artery.[3,4] In such patients, MRI has the advantage of detecting early vascular and intracranial invasion. In the orbital apex syndrome the prognosis is good if treated early. There is mucosal invasion and hence radical debridement and antifungal therapy is the treatment. Progression of the disease results in loss of corneal sensation, sluggish or absent papillary response to light, visual blurring retinal pallor, exophthalmos, complete ophthalmoplegia and with occlusion of the retinal and ciliary artery and blindness.[2] Also, numbness of the infraorbital skin (damage to infraorbital branch of the Vth cranial nerve) with skin necrosis, haze in the vitreous humor (invasion of the globe), superior extension from the ethmoid sinus crossing the dura into the frontal lobe causing obtundation may occur. Sphenoidal sinus extension, thrombosis of cavernous sinus or carotid-cavernous fistula, infection of VIIth cranial nerve (facial palsy) and temporal lobe, cerebral infarction, sudden catastrophic loss of cerebral function may ensue. Death in coma is usual. Rare manifestation of mucormycosis originating in paranasal sinuses include extension along the sphenoid ridge into the middle ear, hematogenous dissemination to the lung or an indolent course extending over many weeks. Ordinarily, death occurs in untreated cases within 4 weeks of onset. The other clinical manifestations of mucormycosis include pneumonia, skin and wound mucormycosis. However, rhinocerebral disease is the commonest, with the rhino-orbito-cerebral type having the highest mortality. The rhinomaxillary type runs a benign course without central nervous system involvement. In our case whether the hemiplegia was due to dissemination of fungi intracerebrally or was it a coincidental infarction is not known.

CONCLUSION
Mucormycosis is usually encountered as a secondary disease or an opportunistic infection due to numerous predisposing factors; commonly uncontrolled diabetes mellitus. Although hemiplegia is rare in rhinocerebral mucormycosis, two cases have been mentioned in literature. In our case whether the hemiplegia was due to dissemination of fungi intracerebrally or was it a coincidental infarction is not known. This case is reported for the occurrence of hemiplegia following mucormycosis of paranasal sinus.

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