

## Clinico- Radiological Imaging Features of Covid-19 Associated Rhino-Orbital-Cerebral Mucormycosis in a Single Tertiary Care Centre: A Case Study of 20 Patients

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Received: 03-06-2021 / Revised: 23-07-2021 / Accepted: 28-08-2021

### Abstract

**Introduction:** Even after more than one year following the origin of the pandemic the pathogenesis of COVID-19 remains partially understood and understanding of the same continues to evolve with time. As the highly infectious virus continues to give rise to new cases globally, one of the biggest concerns is superadded infections. Superadded infections are deemed to occur as the pandemic tends to overlap with geographical realm and seasonality for few infections or due to the encounter with other community or nosocomial pathogens (during hospitalization for COVID-19). However, information on superadded infections in COVID-19, especially with relation to invasive fungal infections, is lacking. **Materials and Methods:** We evaluated the Retrospective, descriptive imaging and clinical data of 20 patients (thirteen males and seven females). Between 30 to 72 years old, with mucormycosis of the craniofacial areas. Patients were selected for study if the diagnosis of mucormycosis was established by means of computed tomography (CT) scans and magnetic resonance (MR) BOSS (Brain-Orbit-Sinus study with IV contrast) protocol imaging. Biopsy & culture were available for confirmation of diagnosis. All of the patients were immunocompromised. Patients with impaired renal function were excluded. **Results:** Of the 20 patients who had CT scans available for review, 12 patients (60%) had hyperdense in relation to muscle/brain in the noninvasive sinusitis suggesting secondary obstructive changes (inspissated secretions). Only 8 patients (40%) had iso to hypodense lesions relative to muscle/brain. On Magnetic Resonance Imaging, 12 patients (60%) had hyperintense lesions relative to brain on T1-weighted images & 13 patients (65.00%) had hypointense on T2-weighted images. **Conclusion:** We propose that, patients with COVID-19 infection are susceptible to mucormycosis because of impairment of barrier defense, dysfunction of phagocytes and lymphocytes and the use of immunosuppressive medications such as steroids, tocilizumab, high ferritin levels, use of prolonged oxygenation & COVID-19 virus itself as a risk factor. Treating clinicians need to be aware of the possibility of mucormycosis, in such patients particularly in those with underlying comorbidities. Early diagnosis and treatment of secondary fungal infections can substantially reduce morbidity and mortality. In our opinion, the severe immunocompromised state secondary to poorly controlled diabetes made the patient susceptible to both severe COVID-19 and mucormycosis.

**Keywords:** Mucormycosis, COVID-19, CAROCM, steroids, tocilizumab, CT, MRI BOSS Protocol.

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### Introduction

Even after more than one year following the origin of the pandemic the pathogenesis of COVID-19 remains partially understood and understanding of the same continues to evolve with time. As the highly infectious virus continues to give rise to new cases globally, one of the biggest concerns is superadded infections. Superadded infections are deemed to occur as the pandemic tends to overlap with geographical realm and seasonality for few infections or due to the encounter with other community or nosocomial pathogens (during hospitalization for COVID-19). However, information on superadded infections in COVID-19, especially with relation to invasive fungal infections, is lacking.

Multiple cases of invasive pulmonary aspergillosis (CAPA - COVID-

19 associated pulmonary aspergillosis) and invasive candidiasis (CAC-COVID-19 associated candidiasis) have been reported in patients with severe COVID-19[1]. This has been linked to the use of steroids, long-term antimicrobials, prolonged mechanical ventilation, and the extended duration of hospital stay in these patients. There is growing concern over the increase in cases of invasive mucormycosis in COVID-19 (CAROCM - COVID-19 associated Rhino- Orbital-cerebral mucormycosis) as well. Mucormycosis is an angio-invasive disease caused by opportunistic fungi of the order Mucorales in immunocompromised patients[2]. Untreated mucormycosis is almost always fatal. It remains to be seen if this increasing incidence of mucormycosis in COVID-19 is related to the illness itself, the steroids and immunomodulators administered for treatment, or the worsening of underlying predisposing factors in the socio-economic upheaval caused by the pandemic. Further, it is unknown how the co-existence of COVID-19 would affect the natural history and treatment of patients with mucormycosis.

However, when brain Invasion has occurred, mortality is greater than 80%. Because of its lethal nature, it must be recognized early and

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treated aggressively. We retrospectively reviewed the neuroimaging findings in a series of Twenty patients with rhino-orbital-cerebral mucormycosis to establish common radiological patterns that may be useful in predicting the early diagnosis of this infection, evaluation & management.

#### Materials and Methods

We evaluated the imaging and clinical data of 20 patients (thirteen males and seven females; Table:2), between 30 to 72 years old (Table:1), with mucormycosis of the craniofacial areas. Patients were selected for study if the diagnosis of mucormycosis was established by means of computed tomography (CT)PNS scans, magnetic resonance (MRI) BOSS (Brain-Orbits-Sinus Study with IV contrast) Protocol, biopsy & culture were available for confirmation. All of the patients were immunocompromised. Clinical information about the presentation, management, and evolution of disease was obtained from medical history in all cases (Table:3,4,5,7& 8). Nineteen had diabetes mellitus (Table:9), and one had chronic lymphocytic leukemia. All patients had MRI BOSS with IV Contrast Protocol imaging on a 1.5-T MRI system. Both T1- and T2-weighted images were obtained as well as T1-weighted images after intravenous injection of gadopentetate dimeglumine (0.1 mmol/kg). All 20 patients had CT PNS Scans available for review. Images were evaluated for density, signal intensity, and contrast enhancement characteristics. The CT density was evaluated in non-enhanced images and compared with muscle/brain. The MR signal intensity was compared with gray matter on the T1- and T2-weighted images. Gadolinium Enhancement was graded on a scale from none to marked (Table:10). All studies were reviewed by two radiologists, and the anatomic structures involved by the infection were defined by consensus.

#### Results

##### Computed Tomography Findings

Of the 20 patients who had CT scans available for review, 12 (60.00%) had hyperdense lesions (Fig.1) to muscle/brain in the noninvasive sinusitis suggesting secondary obstructive changes (inspissated secretions). Only 8 patients (40%) had iso to hypodense lesions (Fig.3) relative to muscle/brain. Thinning & erosions of bony margins (Fig. 2,4,8) is seen in 13 patients (65.00%) with periantral fat plane infiltration (Fig. 3,5). On Magnetic Resonance Imaging, 12 patients (60%) had hyperintense lesions (Fig. 9,10) relative to brain/muscle on T1-weighted images & 13 patients (65.00%) had hypointense lesions (Fig. 12) on T2-weighted images. Abnormal areas of diffusion restriction with ADC reversal (Fig. 13) are noted in 12 patients (60.00%). Variable patterns of enhancement ranging from None (Fig. 14,18) in 6 patients (30.00%), peripheral rim enhancement (Fig. 13,15,19) in 12 patients (60.00%) & mild homogenous enhancement (Fig. 20,27) in 2 patients (10.00%). Nonenhancing devitalized turbinates (S/o "Black Turbinate sign"; Fig. 14,15) in 12 patients (60.00%) (Table-10).

##### Anatomic Involvement

A particular pattern of anatomic involvement affecting the nasal cavity, maxillary sinus, and ethmoid cells was consistently observed in 16 of the twenty patients (Figs. 1–5). Additionally, there was variable involvement of sphenoid & frontal sinuses, orbit, cavernous sinus, carotid artery, skull base floor, and intracranial structures (Fig.16-32) as described.

##### Clinical Evolution

Orbital exenteration, ethmoidectomy, medial maxillectomy, and debridement of the nasal vault were performed in all patients. More extensive debridement of necrotic tissue was performed as required in each particular case according to surgical findings. All patients received amphotericin-B parenterally. Twelve patients (60.00%) recovered, while seven patients (35.00%) expired & one patient (5.00%) lost to follow up (Table:11).

Table 1: Age Distribution

S. No	Age (in years)	No of patients	Percentage
1	30-43 years	4	20.00%
2	44-57 years	7	35.00%
3	58-72 years	9	45.00%

Table 2: Gender Distribution

S. No	Sex	No of patients	Percentage
1	Male	13	65.00%
2	Female	7	35.00%

Table 3: History of Steroid usage

S. No	History of Steroid usage	No of patients	Percentage
1	Yes	18	90%
2	No	2	10%

Table 4: Oxygen Requirement

S. No	Oxygen Requirement	No of patients	Percentage
1	Yes	17	85%
2	No	3	15%

Table 5: Ferritin levels

S. No	Ferritin levels	No of patients	Percentage
1	Normal	9	45.00%
2	Elevated	11	55.00%

Table 6: Radiological Evidence

S. No	Radiological Evidence Both CT & MRI	No of patients	Percentage
1	Yes	20	100%
2	No	0	0%

Table 7: Clinical presentation

S. No	Clinical presentation	No of patients	Percentage
1	Right eye pain and chemosis	2	10.00%
2	Right eye swelling	2	10.00%
3	Chemosis with loss of vision in right eye	1	5.00%
4	Epistaxis, loss of vision left eye	1	5.00%
5	Left side facial pain and nose block	2	10.00%

6	Dryness and crusting in nasal cavity	4	20.00%
7	Holocranial headache	6	30.00%
8	Right eye pain and decreased vision	1	5.00%
9	Impairment of left eye vision	1	5.00%

**Table 8: History of immunosuppressive medications**

S. No	History of immunosuppressive medications	No of patients	Percentage
1	Yes	18	90.00%
2	No	2	10.00%

**Table 9: History of diabetes mellitus**

S. No	History of Diabetes mellitus	No of patients	Percentage
1	Yes	19	95.00%
2	No	1	5.00%

**Table 10: Radiological Imaging Findings**

S. No	Age/Sex		CT		MRI				
	Age	Sex	Density	Bone erosions/t thinning	T <sub>1</sub> W Signal Intensity	T <sub>2</sub> W Signal Intensity	Diffusion restriction	Post contrast T <sub>1</sub> W	Black Turbinate sign
1	30	M	Hyperdense	Yes	Hyper	Hypo	Yes	Peripheral	Yes
2	61	F	Isodense	No	Hypo	Hyper	No	homogenous	No
3	39	F	Hypodense	Yes	Hypo	Hyper	No	None	No
4	65	M	Hyperdense	No	Hyper	Hypo	Yes	Peripheral	Yes
5	43	M	Hypodense	Yes	Hypo	Hyper	No	None	No
6	50	M	Hyperdense	Yes	Hyper	Hypo	Yes	Peripheral	Yes
7	55	F	Hyperdense	Yes	Hyper	Hypo	Yes	Peripheral	Yes
8	68	M	Hypodense	No	Hypo	Hyper	No	None	No
9	35	M	Hypodense	No	Iso	Hyper	No	Peripheral	No
10	72	M	Hyperdense	Yes	Hyper	Hypo	Yes	Peripheral	Yes
11	41	F	Hyperdense	Yes	Hypo	Hyper	No	homogenous	No
12	69	M	Hyperdense	No	Hyper	Hypo	Yes	Peripheral	Yes
13	44	M	Hyperdense	Yes	Hyper	Hypo	No	None	Yes
14	57	M	Isodense	No	Hyper	Hypo	Yes	Peripheral	Yes
15	48	F	Hyperdense	Yes	Hyper	Hypo	Yes	Peripheral	Yes
16	58	F	Isodense	No	Hypo	Hyper	Yes	Peripheral	No
17	47	M	Hyperdense	Yes	Hyper	Hypo	No	None	Yes
18	63	F	Isodense	Yes	Hypo	Hypo	Yes	Peripheral	No
19	59	M	Hyperdense	Yes	Hyper	Hypo	Yes	Peripheral	Yes
20	53	M	Hyperdense	Yes	Hyper	Hypo	Yes	None	Yes

**Table 11: Clinical Outcome**

S. No	Clinical Outcome	No of patients	Percentage
1	Improved	12	60.00%
2	Death	7	35.00%
3	Lost to follow up	1	5.00%

**Radiological Imaging Findings:** - "One Picture is worth more than Ten thousand words"



**Fig 1:** Coronal plain CT soft tissue window shows few internal Hyperdensities within mucosal thickening of bilateral maxillary sinuses

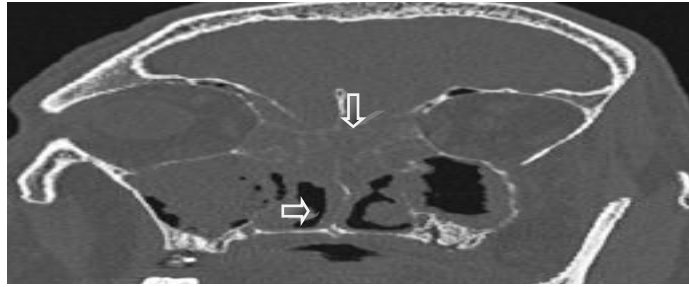


Fig 2: Coronal plain CT bony window shows ossicular thinning and erosions involving frontal-maxillary -ethmoidal sinuses



Fig 3: Axial CT plain imaging soft tissue window shows right periantral fat infiltration

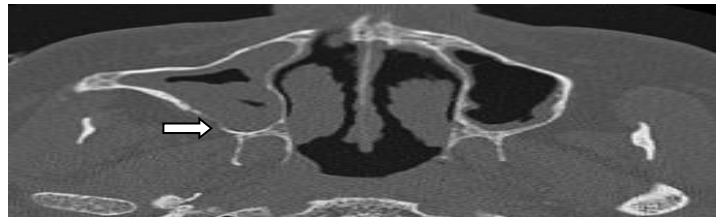


Fig 4: Axial CT plain imaging bony window shows thinning and erosions of the postero-lateral wall of right maxillary sinus.

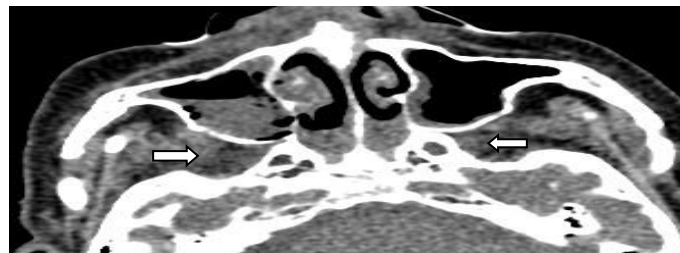


Fig 5: Axial plain CT imaging soft tissue window shows bilateral periantral fat infiltration



Fig 6: Axial Plain CT imaging soft tissue window shows left pterygopalatine canal infiltration



Fig 7: Coronal CT plain imaging soft tissue window shows right extra/intraconal orbital infiltration and myositis.



Figure 8: Axial CT PNS Bone window shows extensive thinning & erosions of bony walls of bilateral maxillary sinus (Whiteblock arrow) extending to involve zygomatic arches (White arrow).

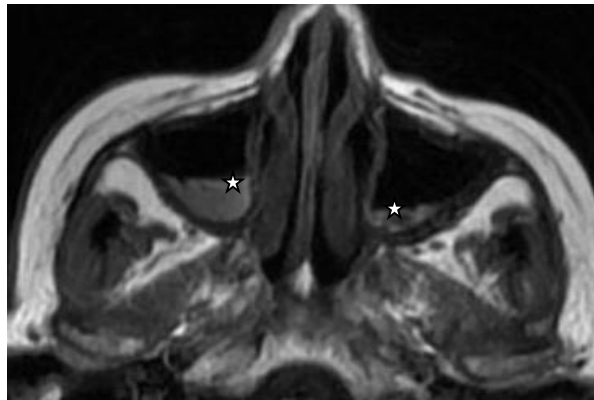


Fig 9: Axial T1W MR imaging shows hyperintense mucosal thickening in bilateral maxillary sinuses.

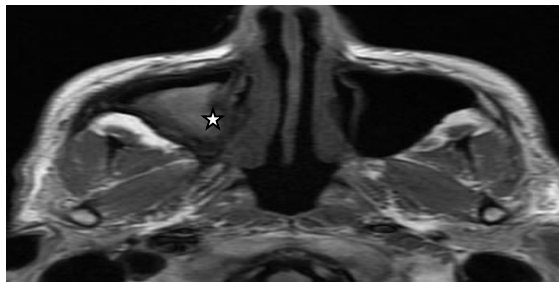


Fig 10: Axial T1W MR Image shows internal hyperintense mucosal thickening of right maxillary sinus.

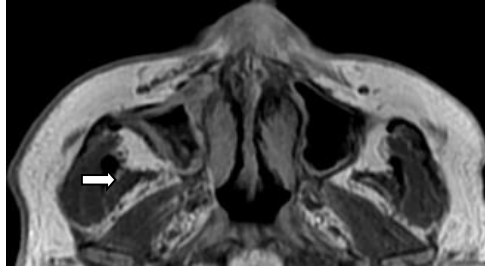


Fig 11: Axial T1W MR Image shows cortical erosion of the posterior lateral wall right maxillary sinus.

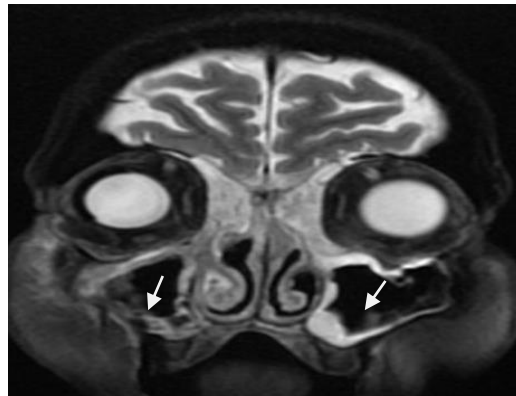


Fig 12: Coronal T2W MR Image shows few internal hypointense foci within/adjacent to mucosal thickening of B/L maxillary sinuses consistent with fungal elements.

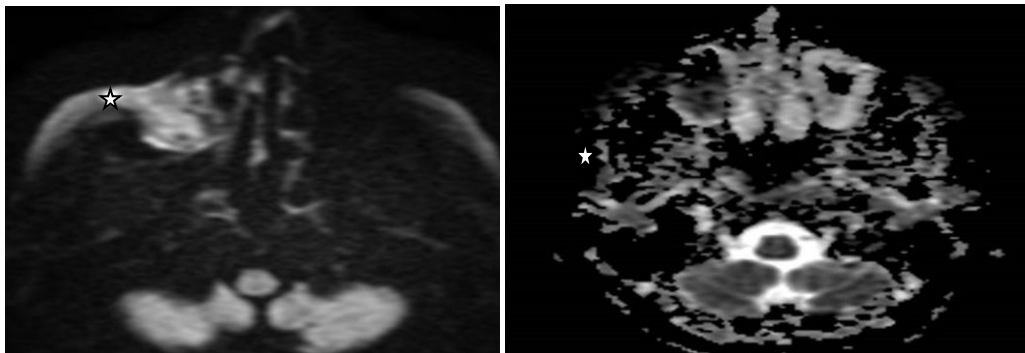


Fig 13: Axial MR Image of brain shows abnormal diffusion restriction with ADC reversal in right maxillary sinus.

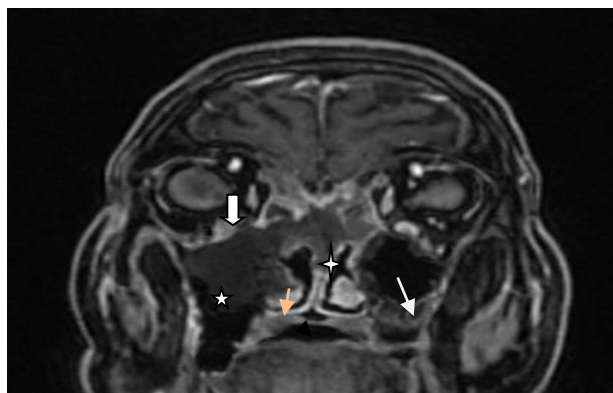


Fig 14: Coronal post contrast T1W MR image shows non-enhancing (star) to peripheral rim-enhancing (white arrow) devitalized mucosal thickening involving bilateral maxillary sinuses infiltrating into adjacent turbinates (orange arrow), nasal septum (twinkle) & right orbit (white bold arrow).

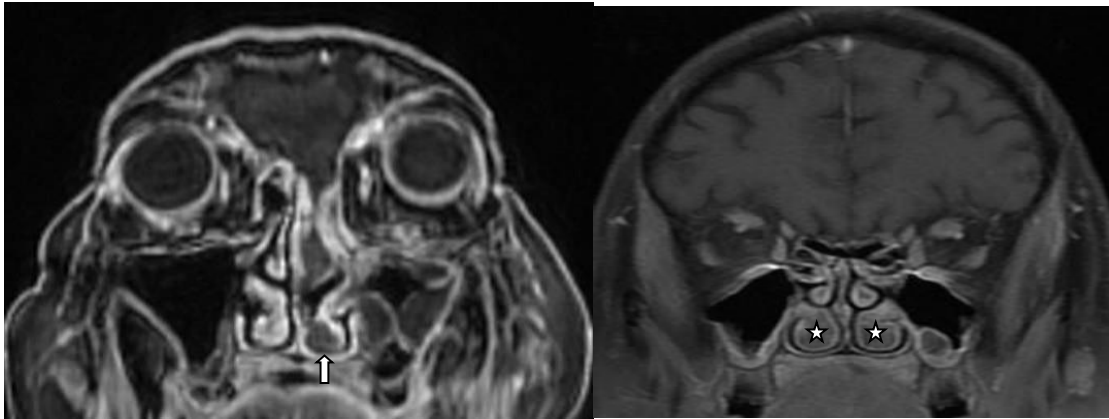


Fig 15: Coronal post contrast T1W images shows “black turbinate sign” involving inferior turbinates suggesting devitalized necrotic tissue.

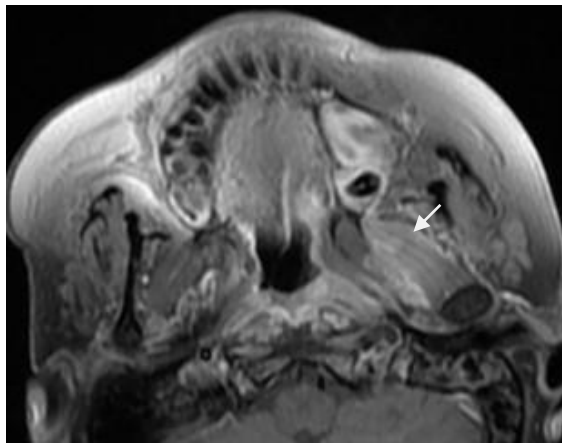


Fig 16: Axial post contrast T1W image shows left Pterygoid muscle involvement.

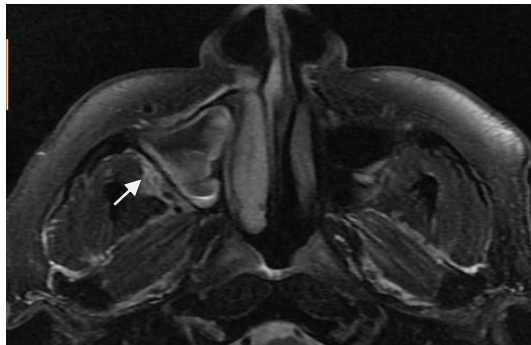


Fig 17: Axial fat suppressed T2W image shows right periantral fat plane infiltration.

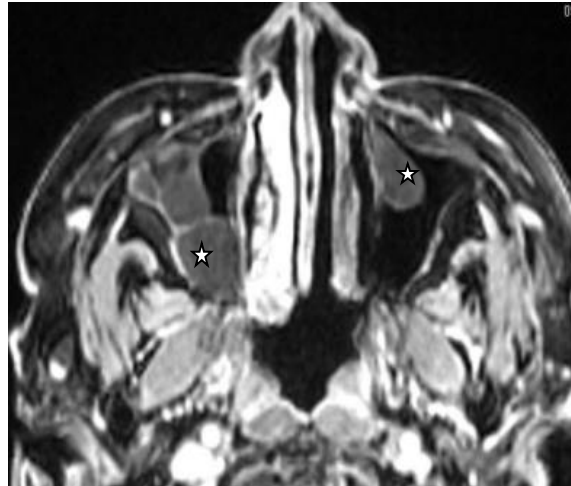


Fig 18: Axial post contrast T1W image shows non enhancing devitalized mucosal thickening partially opacifying bilateral maxillary sinuses.

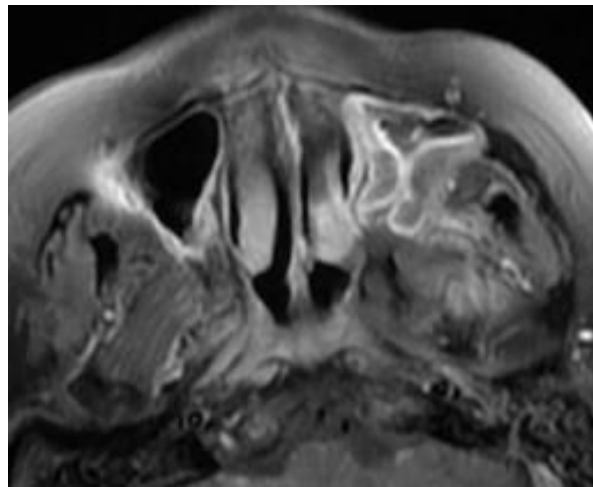


Fig 19: Axial post contrast T1W image shows invasive peripheral rim enhancing central necrotic mucosal thickening causing near total opacification left maxillary sinus.

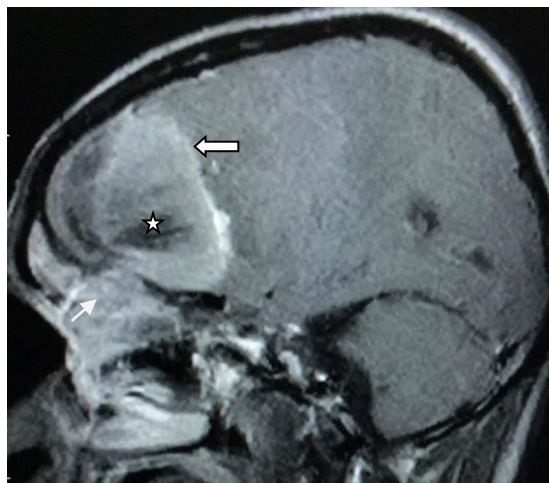


Fig 20: Sagittal post contrast T1W imaging shows mild homogeneously enhancing infiltrative soft tissue lesion with internal hypointense foci (fungal elements: Star) involving frontal, ethmoidal sinuses (white arrow) infiltrating into adjacent frontal lobe (solid white arrow).

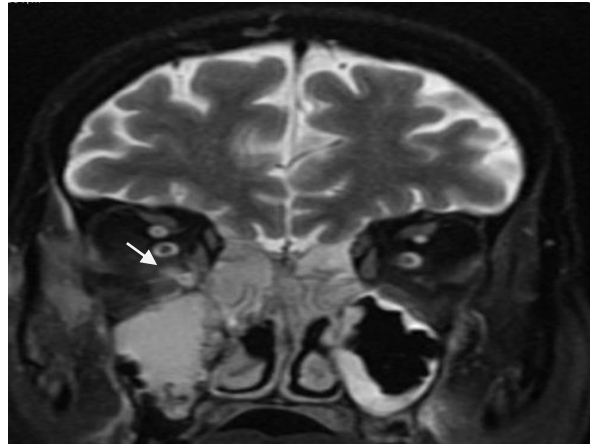


Fig 21: Coronal T2W plain MR image shows infiltration into intra/extraconal compartment of right orbit

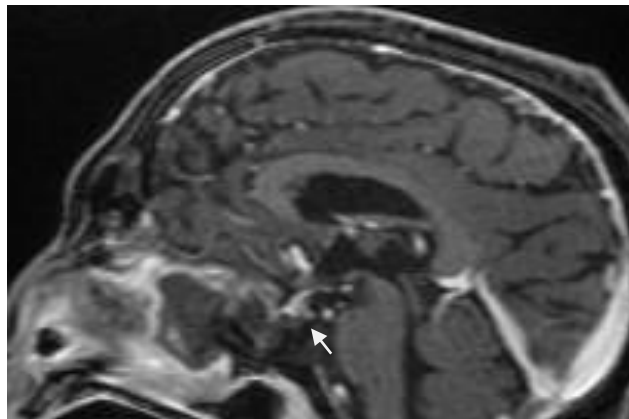


Fig 22: Sagittal T2W MR image shows sellar infiltration by Nonenhancing mucosal thickening.

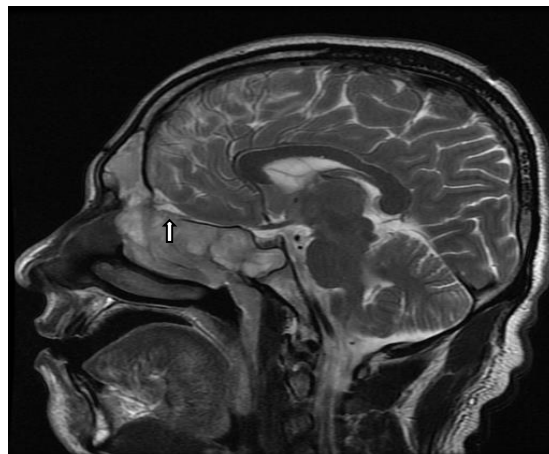


Fig 23: Sagittal T2W MR image shows base erosions and intracranial extension.

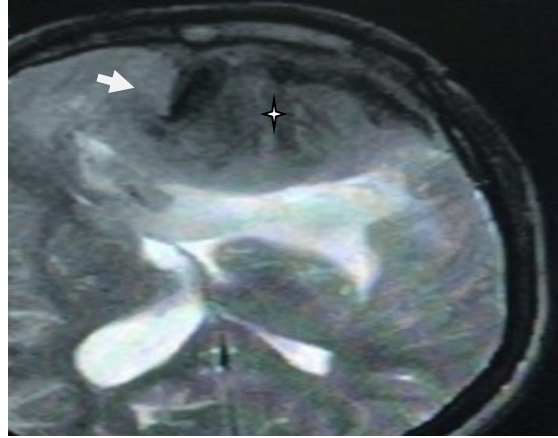


Fig 24: Axial T2W plain MR image shows ill-defined heterogeneously hyperintense mass lesion (solid white arrow) with multiple internal hypointensities (fungal elements-star) involving frontal sinus and infiltrating adjacent frontal lobe.

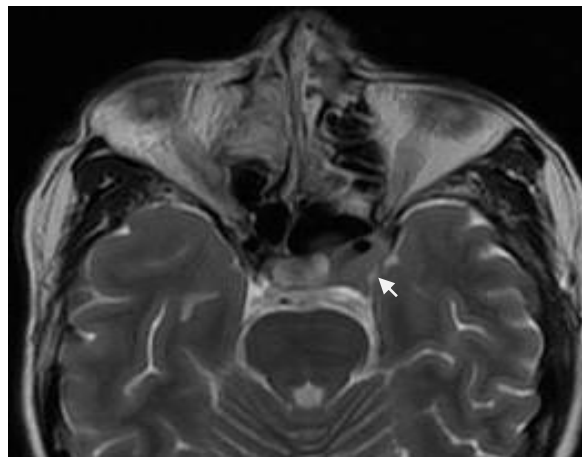


Fig 25: Axial T2W plain MR image shows left cavernous sinus infiltration without causing any ICA narrowing.

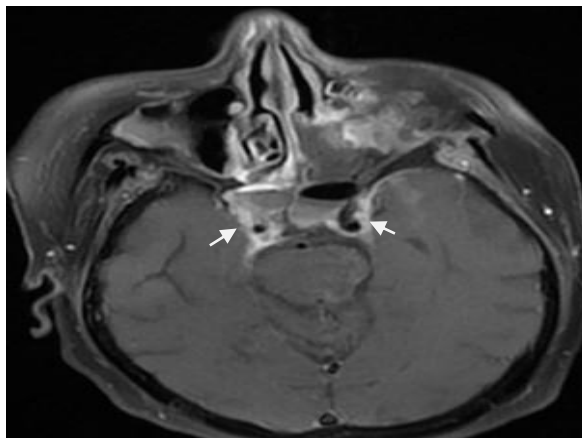


Fig 26: Axial post contrast T1W MR image shows bilateral cavernous sinus infiltration causing narrowing of right ICA.

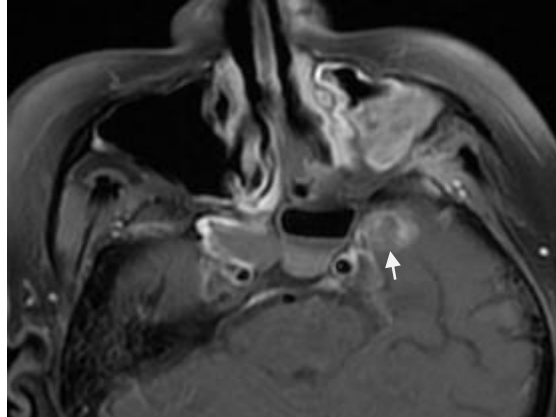


Fig 27: Axial post contrast T1W MR Image shows left anterior temporal lobe infiltration.

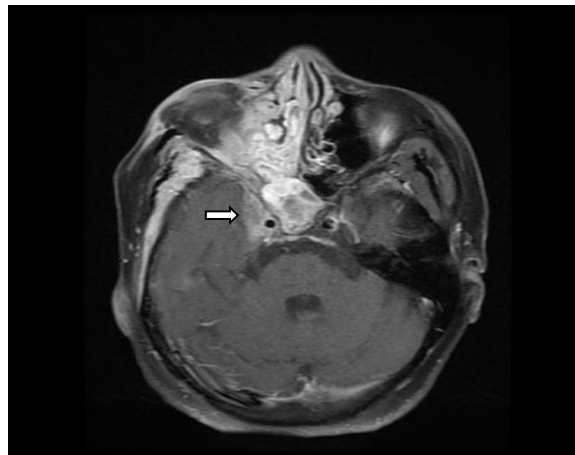


Fig 28: Axial post contrast T1W image MR image shows right cavernous sinus infiltration and Dural spread along the right temporal convexity.

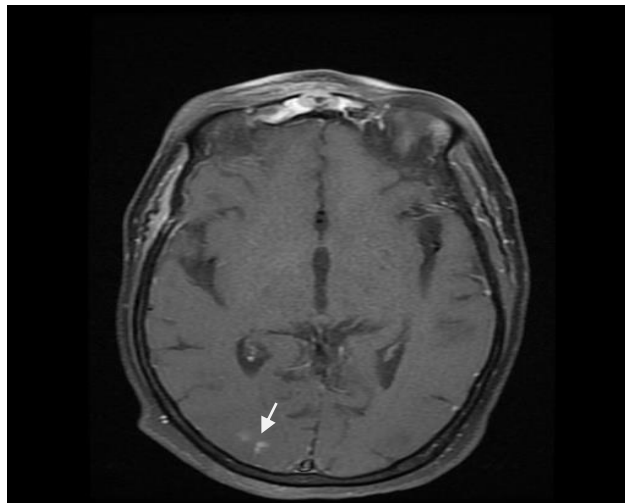
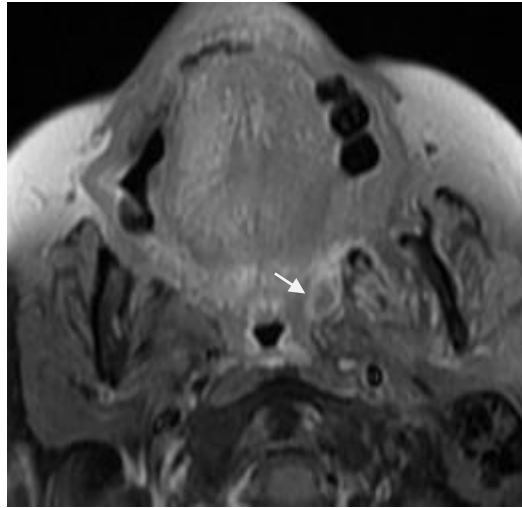


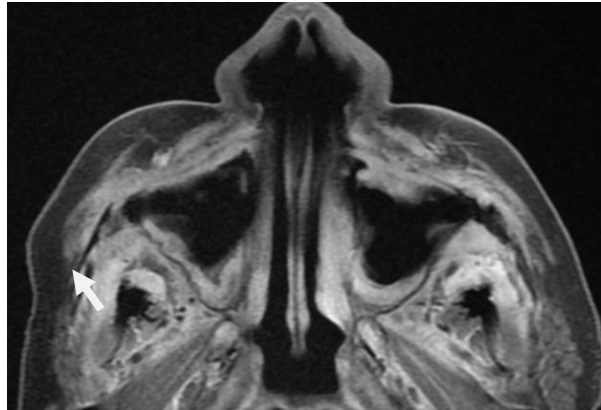
Fig 29: Axial post contrast MR image shows leptomeningeal spread of mucormycosis along the sulcal spaces of right occipital lobe.



**Fig 30: Coronal T2W plain MR image shows infiltration into the right orbital apex(Orbital Apex syndrome)with resultant optic perineuritis, myositis of extraocular muscles and engorgement of superior ophthalmic vein.**



**Fig 31: Axial post contrast T1W MR image shows left parapharyngeal space infiltration.**



**Fig 32: Axial T1 Post contrast imaging at the Maxillary sinus level shows invasive fungal sinusitis infiltrating up to zygomatic arches (Block arrow).**

#### **Discussion**

Mucormycosis, also known as zygomycosis and phycomycosis, was first described by Paultauf in 1885[4]. Phycomycetes are ubiquitous fungi occurring in soil, air, skin, body orifices, manure, spoiled food, and dust[5,6]. Inoculation occurs by inhalation, when spores reach the nasal cavity and/or nasopharynx. The fungus may then spread to the paranasal sinuses and subsequently to the orbit, meninges, and brain by direct extension[7]. Orbital involvement results from spread through the nasolacrimal duct and medial orbital wall. Such invasion

is facilitated by the thinness of the lamina papyracea, congenital dehiscence often presents along the medial wall, and the perforations of the medial wall by arteries and veins[8]. Mucormycosis invades the walls of the blood vessels resulting in vascular occlusion, thrombosis, and infarction, as well as dissemination to the central nervous system from the primary focus[9,10]. Spread to the brain may occur via the orbital apex, orbital vessels, or via the cribriform plate. Generally, the presenting symptoms are low-grade fever, cephalgia, sinusitis, facial swelling, orbital apex syndrome with

blurred vision, and cranial palsies from cavernous sinus involvement in an immunocompromised patient[11]. Early visual loss would favor the diagnosis of rhino-orbital-cerebral mucormycosis over bacterial cavernous sinus thrombosis in which blindness is a much later finding[12]. We found that MRI signal intensity of mucormycosis lesions tends to be hyperintense on T1WI & hypointense in T2WI sequences. After the administration of gadolinium, the lesions had variable enhancement patterns ranging from homogeneous to heterogeneous peripheral rim enhancing or non-enhancing at all. We think that contrast-enhanced T1-weighted images are helpful in delineating the intracranial spread when meningeal enhancement is present as well as in identifying invasion of the cavernous portion of the internal carotid artery by the disease. This had been previously described by Mohindra and associates who showed that MRI can detect cavernous sinus invasion and vascular complications such as ischemia[13].

An aggressive Sino nasal and orbital inflammatory process was observed in 8 of our patients. This behavior was also reported previously by Mnif and colleagues, who suggested that the association between orbital cellulitis and sinusitis in the context of immunosuppression should prompt the diagnosis of mucormycosis. However, the differential diagnosis should consider other fungi, Graves' disease, carotid cavernous fistula and thrombosis, bacterial cellulites, inflammatory pseudotumor, and paranasal sinus tumor. The aggressive clinical features of the disease can suggest the diagnosis of fungal infection, but the final diagnosis depends on the anatomopathological demonstration of fungal tissue invasion. Tissue cultures may be positive, allowing the identification of specific species[14]. Our series demonstrated a mortality rate of 35.0%, in agreement with other series which show up to 50% mortality without intracranial manifestation, and up to 90% mortality if there is intracranial involvement. The survival rates in those with invasive mucormycosis reflect the variance in the ability to reverse the underlying predisposing cause. Immunocompromise can vary from diabetic ketoacidosis to leukemia. Up to 80% of nondiabetic patients survive whereas fewer than 50% of diabetic patients survive. In our series, 1 out of the 7 deaths were due to nondiabetic conditions, namely chronic lymphocytic leukemia[15]. These mortality rates exemplify the aggressive nature of this disease process. Mucormycosis has earned its reputation as the most acutely fatal fungal infection known to humans. It is rapidly growing, and within 24 hours, cultures of mucor can grow to the top of the culture plate. It is important that if the diagnosis is suspected, the suspicion be acted on immediately.

#### Conclusion

We propose that, patients with COVID-19 infection are susceptible to mucormycosis because of impairment of barrier defense, dysfunction of phagocytes and lymphocytes and the use of immunosuppressive medications such as steroids and tocilizumab. Treating clinicians need to be aware of the possibility of mucormycosis, in such patients particularly in those with underlying comorbidities. Early diagnosis by using imaging modalities like CT PNS & MRI BOSS (Brain-Orbit-Sinus study with IV Contrast),

Nasal endoscopy, biopsy and treatment of secondary fungal infections can substantially reduce morbidity and mortality.

In our opinion, the severe immunocompromised state secondary to Poorly controlled diabetes with Steroid/immunosuppressive treatment as well as history of prolonged oxygenation, elevated serum ferritin levels & Severe COVID-19 Patients itself made the patient susceptible to mucormycosis. MRI BOSS (BRAIN-ORBITS-SINUS STUDY with IV contrast) protocol imaging in correlation with CT PNS screening are the best mode of choice in early detection, evaluation & management of Rhino-orbital-cerebral mucormycosis.

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**Conflict of Interest: Nil**

**Source of support: Nil**