ORIGINAL RESEARCH ARTICLE

Magnetic Resonance Imaging in Rhino-Orbito-Cerebral Mucormycosis

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A B S T R A C T

Introduction: Rhino-orbital-cerebral mucormycosis (ROCM) is an acute and aggressive fungal infection that occurs in immunocompromised patients with diabetes. The disease originates in the sinonasal mucosa and extends rapidly to neighboring structures, including orbit and sometimes brain. Study aimed to evaluate the diagnostic accuracy of MRI in Mucormycosis and to investigate the imaging findings of Mucormycosis.

Materials and methods: This hospital based retrospective study was carried out over a period of 3 months from April 2021 to June 2021 in 30 patients with symptoms of Mucormycosis who underwent MRI at Department of Radiodiagnosis at Santhiram General hospital, Nandyal.Patients who met inclusion and exclusion criteria were included in the study.

Results: Our study shows the infection is more common in males than females. Unilateral involvement of sinus more common than Bilateral involvement.Ethmoid sinus is most commonly involved paranasal sinus in our study followed by maxillary sinus.The combination of maxillary+ethmoid+sphenoid sinuses was frequently seen.The involvement of paranasal sinuses with intraorbital extensionis more common.

Conclusion: Magnetic resonance imaging is highly useful imaging modality for the diagnosis of ROCM and shows T2-W hyperintense signal intensity in sinonasal mucosa and infiltrating lesion in orbit. MRI determines the extent of invasion very well.

Keywords: Mucormycosis, Magnetic Resonance Imaging (MRI)

INTRODUCTION

Rhino-orbital-cerebral mucormycosis (ROCM) is a life threatening infection caused by saprophytic fungi belonging to genera Mucor,Rhizopus,Absidia[1].Rhino-orbital-cerebral mucormycosis (ROCM) is an acute and uncommon aggressive fungal infection that occurs in immunocompromised patients including those with diabetes.Ketoacidosis in diabetes enhances susceptibility to ROCM.The disease originates in the sinonasal mucosa and extends rapidly to neighboring structures, including orbit and sometimes brain. ROCM is characterized by a very high residual morbidity and mortality due to angioinvasive property of the fungus, which causes vascular occlusion resulting in extensive tissue necrosis. Early diagnosis and timely intervention is key to successful treatment[2].

Study aimed to evaluate the diagnostic accuracy of MRI in Mucormycosis and to investigate the imaging findings of Mucormycosis.

MATERIAL AND METHODS

This hospital based retrospective study was carried out over a period of 3 months from April 2021 to June 2021 in 30 patients with symptoms of Mucormycosis who underwent MRI at Department of Radiodiagnosis at Santhiram medical college and general hospital, Nandyal.

Inclusion criteria

1. Patient willing to participate in the study and willing to give written and informed consent.
2. Patients with history of COVID 19 ,diabetes, steroid use.
3. Patients with symptoms of suspected Mucormycosis

Exclusion criteria

1. Patient not willing to participate in the study and not willing to give written and informed consent.

Method of study

- MR imaging examination of the patients done under a 1.5 T Siemens Magnetom_Essenza, sygno version-sygno $VH_{21}ASL_{36}P_{43}$ machine TIM+DOT System
- For each patient the following MRI protocol is followed as shown in Table 1.

RESULTS

Our study shows the infection is more common in males(73.3%) than females(26.6%)(Table 2). Unilateral(70%) involvement of sinus more common than Bilateral(30%) involvement(Table 3).Ethmoid sinus(96%) is most commonly involved paranasal sinus in our study followed by maxillary sinus(90%).The combination of maxillary+ethmoid+sphenoid sinuses (60%) was frequently seen(Table 4).The involvement of paranasal sinuses with intraorbital extension(66.6%)is

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Acquisition Details	
Preferred field strength	1.5 or 3.0 Tesla MRI
Preferred planes of imaging	Axial and coronal planes
Preferred sequences	2D Spin echo or fast spin echo sequences with T1W, T2W, STIR or fat saturated T2W,and fat saturated postcontrast T1W images Diffusion imaging, MR angiogram
Coverage	Axial images: Teeth to top of frontal sinus Coronal images: Nasal cartilages to pons
Preferred Slice Thickness	2-3 mm
Role of Individual Pulse Sequences	
T2 and T1 weighted in axial and coronal plane	Delineate soft tissue and bone involvement
Short tau inversion recovery (STIR)/Fat-saturated T2W images	Most sensitive sequence to demonstrate pathology - Should be acquired at least in one plane
Fat-saturated T1W images with intravenous gadolinium	Best for delineating the extent of pathology and areas of avascular necrosis
MR angiogram	To look for angioinvasion in cases with skull base or cavernous sinus involvement
Diffusion imaging	To detect areas of cerebral and optic nerve infarction

Table-1: Ideal MRI protocol in a suspected case of ROCM 3

Sex	**Number of patients**
Male	22(73.3%)
Female	8(26.6%)

Table-2: Distribution of Sex

Sinus involvement	**Number of patients**
Unilateral	21(70%)
Bilateral	9(30%)

Table 3: Distribution of Unilateral or bilateral involvement of sinuses

Sinus involved	**Number of patients**
Maxillary	27(90%)
Ethmoid	29(96.6%)
Sphneoid	20(66.6%)
Frontal	18(60%)
Maxillary+ Ethmoid	16(53.3%)
Ethmoid+Sphenoid	5(16.6%)
Maxillary+ Ethmoid+Sphenoid	18(60%)
Pansinusitis	12(40%)

Table 4: Distribution of involvement of sinuses in mucormycosis infection

Area involved	**Number of patients**
Nose and paranasal sinus alone	5(16.6%)
PNS with intraorbital involvement	20(66.6%)
Intracranial involvement	10(33.3%)

Table -5: Distribution of Area involved in Mucormycosis infection

more common(Table 5).The different patternsof extrasinus involvement is shown in table 6.

DISCUSSION

Sinonasal involvement[3]

Normal paranasal sinuses are air-filled structures which are

Type of involvement	**Number of patients**
Soft tissue infiltration and fat stranding	20(66.6%)
Orbital cellulitis	20(66.6%)
Optic neuritis	12(40%)
Skull base lytic destruction	1(3.3%)
Cavernous sinus involvement	2(6.6%)
Brain (Cerebritis,Abscess,Infarcts)	10(33.3%)
Black turbinate sign	9(20%)

Table-6: Distribution of MRI pattern of Mucormycosis

hypointense on all sequences.The normal MRI imaging findings of sinuses, orbit, and brain are listed in Table 7. The common imaging features of ROCM are listed in Table 8. In fungal sinusitis, opacification of the sinuses by soft tissue is seen. Multiple sinus involvement is seen in about half the cases of ROCM. The contents of the sinuses have varying signal characteristics on MRI. The T2Wsignal intensity is determined by the extent of necrosis (causing hyperintensity) and the presence of paramagnetic elements such as iron and manganese within the fungal hyphae (causing hypointensity). The findings on diffusion-weighted imaging are variable with one series reporting restricted diffusion. On postcontrast scans,the contents of the sinuses may show a variety of appearances ranging from: 1. intense homogenous enhancement, 2. Variable enhancing and nonenhancing areas, and 3. complete central nonenhancement with or without a thin irregular rim of peripheral enhancement. A characteristic imaging feature of invasive fungal sinusitis on postcontrast T1W images is the absence of enhancement in areas that normally enhance.This finding is secondary to the angioinvasive nature of thefungus, causing microthrombosis and tissue necrosis in the affected regions.This appearance – termed as the "Black Turbinate sign"(Fig 1) – is the imaging counterpart of the necrotic eschar seen on clinical orrhinoscopic examination. Recognitionof this sign may aid in early diagnosis of ROCM.

Extrasinus extension[3]

Extension beyond the sinuses is one of the most important

Nasal Cavity and Paranasal Sinuses				
Anatomical structure	T1W images	T2W images	Fat-Sat T2W images	Postcontrast T1W images
Mucosal lining	Isointense	Thin, linear hyperintensity	Thin, linear hyperintensity	Thin, smooth enhancement
Air within sinuses	Absent signal (signal void)	Absent signal (signal void)	Absent signal (signal void)	-
Bony Structures				
Anatomical structure	T1W images	T2W images	Fat-Sat T2W images	Postcontrast T1W images
Cortical bone	Hypointense	Hypointense	Hypointense	No enhancement
Bone marrow	Hyperintense	Intermediate signal	Hypointense	Mild heterogeneous enhancement
Orbit				
Anatomical structure	T1W images	T2W images	Fat-Sat T2W images	Postcontrast T1W images
Aqueous and vitreous of globe	Hypointense - similar to CSF	Hyperintense - similar to CSF	Hyperintense- similar to CSF	-
Coats of the globe	Intermediate signal	Hypointense	Hypointense	Thin linear enhancement of the choroid
Retroorbital fat	Hyperintense	Hyperintense	Hypointense	No enhancement
Extraocular muscles	Intermediate signal	- Intermediate signal	Intermediate signal	Intense homogenous enhancement
Optic nerve	Isointense to cerebral white matter	Isointense to cerebral white matter	Isointense to cerebral white matter	No enhancement
Other Structures				
Anatomical structure	T1W images	T2W images	Fat-Sat T2W images	Postcontrast T1W images
Periantral fat	Hyperintense	Hyperintense	Shows suppression - hypointense	No enhancement
Muscles	Intermediate signal	Intermediate signal	Intermediate signal	Mild homogenous enhancement
Cavernous sinus	Isointense	Variable signal based on blood flow	Variable signal based on blood flow	Homogenous enhancement
Patent blood vessels	Absent signal (flow voids)	Absent signal (flow voids)	Absent signal (flow voids)	Variable enhancement

Table-7: Imaging appearances of normal structures on MRI[3]

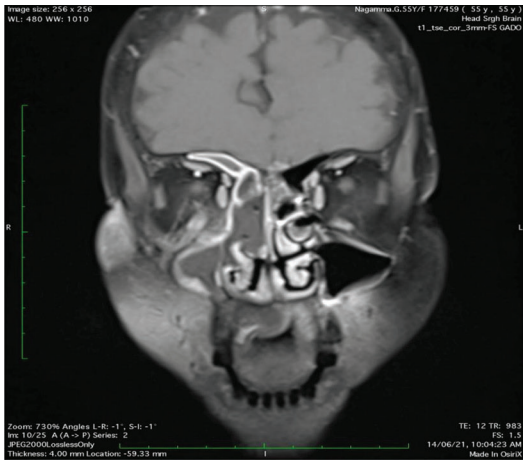

Figure-1: Coronal CE T1 weighted MRI image showing non enhancing middle and superior turbinates-Black turbinate sign seen in Mucormycosis.

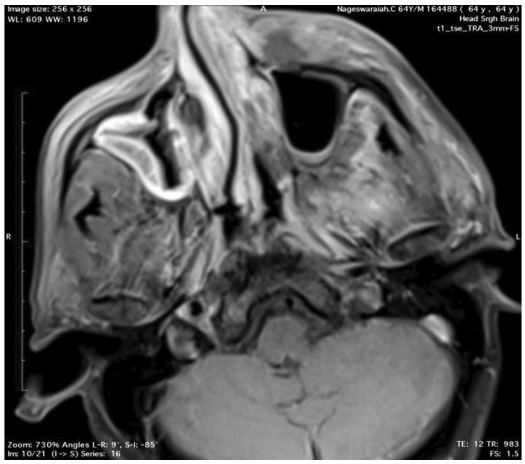

Figure-2: Axial CE T1 weighted MRI image in case of Mucormycosis showing left premaxillary and retro maxillary soft tissue swelling with mild enhancement .

indicators suggesting fungal etiology. Therakathu et al. showed that the orbit was the most common site of extrasinus involvement followed by the face. Other sites of involvement include the masticator space, palate, skull base, orbital apex, pterygopalatine fossa, cavernous sinus, cranial nerves, internal carotid artery, and the brain. Middlebrooks et al. described a

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Imaging finding	Best sequence to visualize the finding	Importance of recognition
Mucosal thickening with T2 hypointense components	T2W images	Suspicion of fungal etiology
Nonenhancement of involved mucosa/ soft tissue	Post contrast T1W images	Suspicion of fungal etiology
Marrow edema and enhancement of adjacent bones and skull base	Fat- saturated T2W and postcontrast T1W images	Bony invasion
Edema and enhancement of fat planes surrounding the maxillary antrum	T1W, Fat-saturated T2W and postcontrast T1W images	Periantral soft tissue invasion
Edema of retroorbital fat and enhancing soft tissue in the orbit with or without involvement of the extraocular muscles	T1W, Fat-saturated T2W and postcontrast T1W images	Orbital extension
Diffusion restriction within the optic nerve	Diffusion-weighted images	Optic nerve infarction
Edema and enhancing soft tissue within the orbital apex and pterygopalatine fossa	T1W, Fat-saturated T2W and postcontrast T1W images	High risk of cavernous sinus involvement and intracranial extension
Internal carotid artery narrowing without or with arterial wall enhancement	MR angiogram and postcontrast T1W images	Arterial wall invasion
Meningeal enhancement, cerebral parenchymal signal changes with peripheral enhancement	T2W, diffusion-weighted and postcontrast T1W images	Cerebral parenchymal invasion/ abscess formation
Cerebral parenchymal signal changes with diffusion restriction	T2W and diffusion-weighted images	Acute infarction

Table 8: Imaging findings in ROCM3

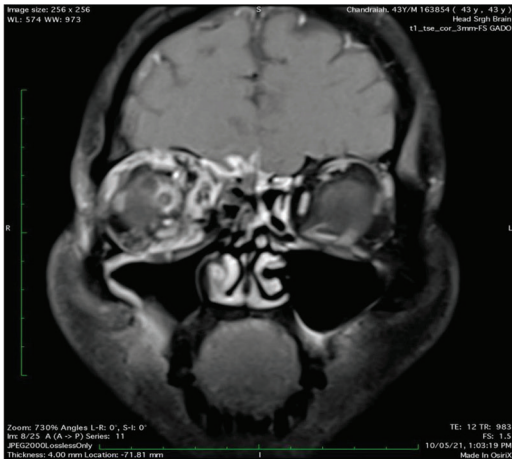

Figure-3: Coronal CE T1 weighted MRI image showing enhancement in intraconal and extraconal compartment of right orbit medial to optic nerve with thickening of medial and inferior rectus muscles and peripheral enhancement of right optic nerve-S/o Orbital invasion in case of Mucormycosis

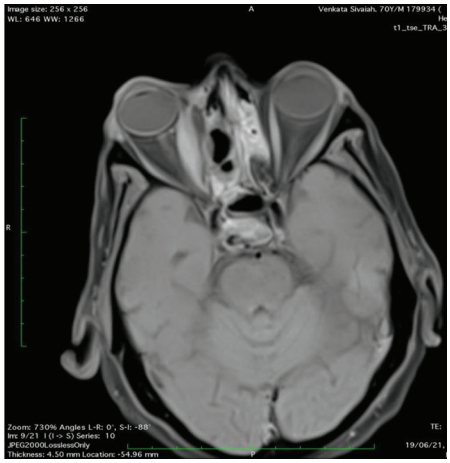

Figure-4: Axial CE T1 weighted MRI image in case of Mucormycosis showing thickening of right medial rectus muscle.

CT-based seven-variable diagnostic model to predict acute invasive fungal sinusitis. The presence of any one of the seven variables (extension of disease into the periantral fat, orbits, pterygopalatine fossa, sphenopalatine foramen, nasolacrimal duct, lacrimal sac, and bone dehiscence) had a 95% sensitivity and 86% specificity for fungal etiology. The presence of any two variables gave 88% sensitivity, 100% specificity, and 100% positive predictive value for the diagnosis of invasive fungal sinusitis.The pattern of extension of infection beyond the margins of the sinuses is best delineated on fat-suppressed T2W and fat-suppressed postcontrast T1W images, as edema and enhancement of bony walls. On MRI, extension into the periantral fat is seen as signal changes and enhancement within the premaxillary and retroantral fat(Fig 2). Further extension into the infratemporal fossa is seen as edema and enhancement within the muscles of mastication. Extension of the pathology into the pterygopalatine fossa is seen as replacement of normal fat signal surrounding the branches ofthe internal maxillary artery and presence of enhancing soft tissue.

Orbital involvement[3]

Orbital invasion in mucormycosis commonly occurs through pathways of least resistance, which include lamina papyracea, nasolacrimal duct, ethmoid foramina, and perforations of the medial orbital walls by vascular channels.Early orbital infection shows soft tissue infiltration and edema of the retroorbital fat around the extraocular muscles(Fig 3).Infiltration of the retroorbital fat is best appreciated on fat-saturated T2W sequences. As orbital invasion commonly

occurs through the medial wall, inflammatory tissue or abscess formation may be seen along the medial aspect of the orbit with lateral displacement and edema of the medial rectus muscle [Fig. 4].Optic nerve involvement may be seen. Sudden onset of blindness can be due to central retinal artery or ophthalmic artery occlusion, optic nerve infarction, or direct infiltration of the optic nerve. Direct invasion of the optic nerve results in swollen optic nerve with high T2 Signal intensity.(Fig 5)Isolated involvement of the optic nerve suggests spread of infection through branches of the ophthalmic artery, which is an indication for initiation of aggressive treatment.Diffuse orbital infection may present with severe proptosis and tenting of the globe . Thickening and enhancement of the extraocular muscle.

Figure-5: Axial CE T1 weighted MRI image in case of Mucormycosis showing bulky right optic nerve with peripheral enhancement-S/o Optic neuritis.

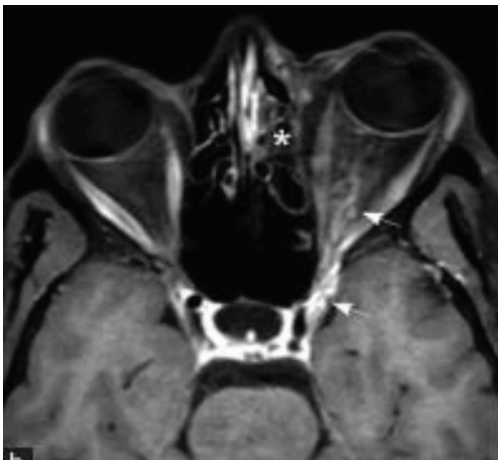

Figure-6: Fat-saturated postcontrast T1W image through the orbit shows left ethmoidal sinusitis (asterisk). There is retroorbital fat stranding and heterogeneously enhancing soft tissue at the orbital apex.

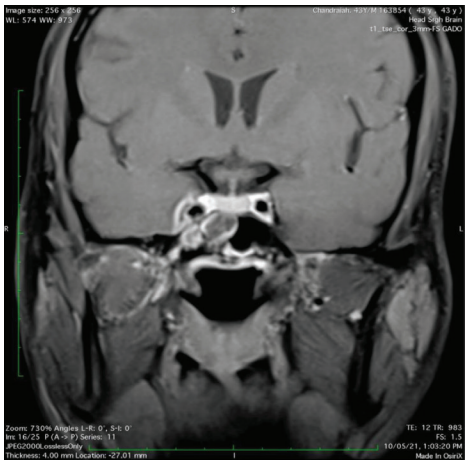

Figure-7: Coronal CE T1 weighted MRI image in case of Mucormycosis showing mild enlarged right cavernous sinus with patchy heterogenous enhancement.

Orbital apex involvement[3]

Enhancing soft tissue at the orbital apex extending into both the optic canal and superior orbital fissure may present as orbital apex syndrome. When imaging findings of sinusitis are associated with orbital apex syndrome suspicion of fungal etiology must be raised.Infection can spread from the orbital apex posteriorly through the superior orbital fissure into the cavernous sinus(Fig 6) and through the inferior orbital fissure across the pterygopalatine fossa into the infratemporal fossa .

Cavernous sinus and major arterial involvement[3]

In ROCM, heterogenously enhancing soft tissue may be seen extending from the superior orbital fissure to involve the cavernous sinus. The lateral walls of the sinuses are normally concave laterally or straight on coronal and axial images. Loss of concavity of the cavernous sinus is a sign of involvement. Inearly stages, bulky cavernous sinus with convexity of the lateral wall is seen [Fig. 7]. Postcontrast images show filling defects within the sinus. Occlusion of the superior ophthalmic vein may occur due to extension of pathology along the vein or due to soft tissue compression at the orbital apex. On imaging, the thrombosed vein is seen as a dilated cord-like structure superior to the optic nerve, crossing from the medial to lateral side. The lumen of the vein shows loss of normal flow void and filling defects on postcontrast images. Cavernous segment of internal carotid artery may be encased by the soft tissue or thrombus in the cavernous sinus causing narrowing of its lumen. Alternatively, the fungus can invade the arterial wall, causing occlusion of its lumen. Areas of arterial wall invasion may be demonstrated as wall enhancement on vessel wall imaging.Vascular invasion and occlusion of the cavernous segment of the internal carotid artery are the most common causes leading to cerebral infarcts. [Fig. 8]

Intracranial extension[3]

Intracranial involvement in mucormycosis commonly occurs by direct spread across the cribriform plate, walls of the ethmoid, and frontal sinuses. Extensions into the middle cranial fossa from the pterygopalatine fossa and along internal carotid artery are also seen.Perineural spread from the cavernous sinus, along the trigeminal nerve, can lead to predominant posterior fossa involvement. Early intracranial spread is better appreciated on contrast-enhanced T1W images when there is meningeal enhancement. Other intracranial manifestations include abscesses and infarcts. Fungal invasion of the brain parenchyma appears as ill-defined areas of altered signal intensity, usually T2 hyperintensity, in nonvascular distribution. Minimal perilesional edema and

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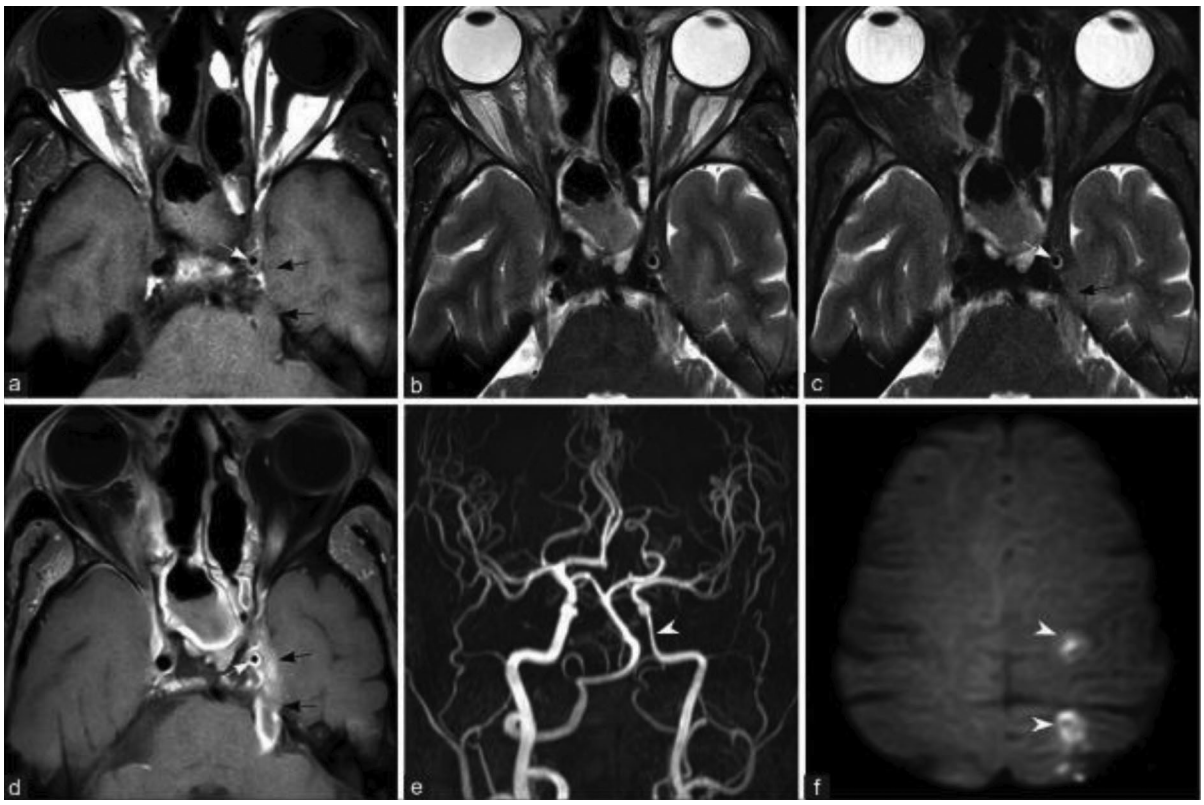

Figure-8: Perineural spread to the cerebral parenchyma and arteritis. T1W (a), T2W (b) images, fat-saturated T2W (c), and fat-saturated postcontrast T1W (d) images show perineural extension from the left cavernous sinus, along the trigeminal nerve up to the pons (black arrows). Thickening and enhancement of the wall of the left internal carotid artery is seen (white arrows). Arterial narrowing on MR angiogram (e) sand left cerebral watershed infarcts (f) (arrowheads) are seen

Figure-9: Axial CE T1 weighted MRI image in case of Mucormycosis showing peripherally enhancing lesion in right basi frontal lobe-Evolving Abscess indicating intracranial extension

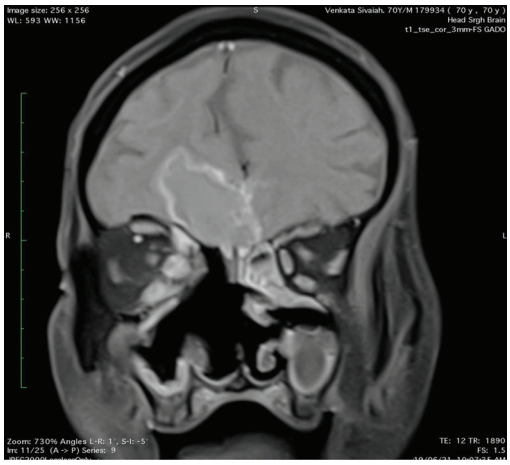

Figure-10: Coronal CE T1 weighted MRI image of case of mucormycosis showing enhancement in intraconal and extraconal compartment of right orbit medial to optic nerve with thickening of medial and inferior rectus muscles and peripherally enhancing lesion in right basi frontal lobe -S/o Orbital and intracranial extension.

peripheral enhancement are present(Fig 9) . Development of a well-delineated mass with liquified central T2 hyperintense core showing diffusion restriction indicates abscess formation.Abscesses in ROCM may show the characteristic well-defined rim enhancement (Fig 10).

Skull base involvement[3]

Skull base osteomyelitis is a rare complication, usually seen in the late stages of the disease because angioinvasive nature of the fungus facilitates extensive spread of infection into the deep soft tissues through the perivascular channels even before bone destruction. Early involvement of the bone marrow can be picked up on T1W images, which show loss of normal fat signal. The marrow appears hypointense on T1W images and hyperintense on STIR images with postcontrast images showing heterogenous enhancement. In advanced stage, there is extensive heterogeneously enhancing soft

Proposed Staging of Rhino-Orbito-Cerebral Mucormycosis (ROCM)

Staging of Rhino-Orbito-Cerebral Mucormycosis	Symptoms	Signs	Primary Assessment	Confirmation of Diagnosis
Stage 1: Involvement of the nasal mucosa 1a: Limited to the middle turbinate 1b: Involvement of the inferior turbinate or ostium of the nasolacrimal duct 1c: Involvement of the nasal septum 1d: Bilateral nasal mucosal involvement	Nasal stuffiness, nasal discharge, foul smell, epistaxis	Foul-smelling sticky mucoid or black-tinged, or granular or haemorrhagic nasal discharge, nasal mucosal inflammation, erythema, violaceous or blue discoloration, pale ulcer, anaesthesia, ischemia, eschar	Diagnostic nasal endoscopy, Contrast-enhanced MRI (preferred) or CT-scan	Deep nasal swab or endoscopy-guided nasal swab or nasal mucosal biopsy for direct microscopy, culture and molecular diagnostics; nasal mucosal biopsy for rapid histopathology with special stains
Stage 2: Involvement of paranasal sinuses 2a: One sinus 2b: Two ipsilateral sinuses 2c: > Two ipsilateral sinuses and/or palate/oral cavity 2d: Bilateral paranasal sinus involvement or involvement of the zygoma or mandible	Symptoms in Stage 1 + facial pain, facial edema, dental pain, systemic symptoms (malaise, fever)	Signs in Stage 1 + unilateral or bilateral, localized or diffuse facial edema, edema localized over the sinuses, localized sinus tenderness	Diagnostic nasal endoscopy, Contrast-enhanced MRI (preferred) or CT-scan	Same as Stage 1 + sinus biopsy for direct microscopy, culture and molecular diagnostics and rapid histopathology
Stage 3: Involvement of the orbit 3a: Nasolacrimal duct, medial orbit, vision unaffected 3b: Diffuse orbital involvement (>1 quadrant or >2 structures), vision unaffected 3c: Central retinal artery or ophthalmic artery occlusion or superior ophthalmic vein thrombosis; involvement of the superior orbital fissure, inferior orbital fissure, orbital apex, loss of vision 3d: Bilateral orbital involvement	Symptoms in Stage 1 and 2 + pain in the eye, proptosis, ptosis, diplopia, loss of vision, infraorbital and facial V1 V2 nerve anesthesia	Signs in Stage 1 and 2 + conjunctival chemoses, isolated ocular motility restriction, ptosis, proptosis, infraorbital nerve anesthesia, central retinal artery occlusion, features of ophthalmic artery occlusion and superior ophthalmic vein thrombosis. V1 and V2 nerve anesthesia, and features of III, IV and VI nerve palsy indicating orbital apex/superior orbital fissure involvement.	Diagnostic nasal endoscopy, Contrast-enhanced MRI (preferred) or CT-scan	Same as Stage 2 + orbital biopsy if indicated and if feasible (if the disease is predominantly orbital) for direct microscopy, culture and molecular diagnostics and rapid histopathology
Stage 4: Involvement of the CNS 4a: Focal or partial cavernous sinus involvement and/or involvement of the cribriform plate 4b: Diffuse cavernous sinus involvement and/or cavernous sinus thrombosis 4c: Involvement beyond the cavernous sinus, involvement of the skull base, internal carotid artery occlusion, brain infarction 4d: Multifocal or diffuse CNS disease	Symptoms in Stage 1 to 3 + bilateral proptosis, paralysis, altered consciousness, focal seizures	Signs in Stage 1-3 (some features overlap with Stage 3) + V1 and V2 nerve anesthesia, ptosis, and features of III, IV and VI nerve palsy indicate cavernous sinus involvement. Bilaterality of these signs with contralateral orbital edema with no clinico-radiological evidence of paranasal sinus or orbital involvement on the contralateral side indicate cavernous sinus thrombosis. Hemiparesis, altered consciousness and focal seizures indicate brain invasion and infarction.	Diagnostic endoscopy, Contrast-enhanced CT Scan, MRI (preferred)	Same as Stage 3

tissue with infiltration into the bones.Obliteration of normal adjacent fat planes with T2 hyperintense soft tissue edema, perineural, and intracranial spread may be seen. Abscess formation can occur appearing as fluid signalintensity area with central diffusion restriction and peripheral rim enhancement.

Staging of ROCM[4]

MRI for follow up[3]

MR imaging also plays an important role in follow up of patients who are on treatment. In patients with clinical suspicion of ROCM, where nasal endoscopy and initial MR imaging studies are noncontributory, it is recommended

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to perform follow-up imaging after 72 h. The progression of the disease and development of more classic imaging findings provide more evidence for fungal etiology (probable ROCM). It should be remembered that imaging findings may take longer to resolve than clinical signs.

CONCLUSION

Magnetic resonance imaging is highly useful imaging modality for the diagnosis of ROCM and shows T2-W hyperintense signal intensity in sinonasal mucosa and infiltrating lesion in orbit. MRI determines the extent of invasion very well.Cavernous sinus thrombosis and internal carotid artery narrowing is well depicted by MRI. DWI may add specificity to the diagnosis by showing restricted diffusion in the path of fungal invasion[2].

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