



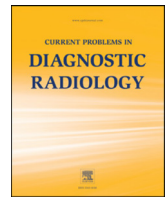
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CT Imaging Features in Acute Invasive Fungal Rhinosinusitis- Recalling the Oblivion in the COVID Era

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Objective: Recent pandemic of COVID19 infection has witnessed a re-emergence of invasive fungal sinusitis especially of the *Mucor* species, which has been a rare entity in the pre covid era. Covid associated mucormycosis (CAM) is one of the dreaded and fatal complications which has surfaced up and early diagnosis is critical for management and survival. It is identified to affect both subset of patients, those with active COVID-19 infection and those who have recovered from the disease in the last 4-6 weeks. Imaging features suggestive of early invasion with supportive imaging examples and relevance of these findings in clinical decision making is presented.

Methods: This paper reviews the various imaging signs of early invasion in CAM. A comprehensive checklist for clinically relevant and quick reporting is also presented.

Results: Emphysematous or ulcerative mucosal changes in the nasal cavity is an early imaging feature of CAM. Periantral soft tissue and soft tissue within the pterygopalatine fossa are important imaging signs to indicate extrasinus invasion. Disease within pterygopalatine fossa may lead to multidirectional spread and is an important check site. These findings are seen even in absence of bony erosions owing to the neurovascular spread of disease. Intra orbital and intracranial extensions were found to be fairly common and must be sought for.

Conclusion: The knowledge of early subtle signs of CAM on imaging can aid in prompt diagnosis of this fatal entity in the pertinent clinical setting. Imaging signs of spread of disease and delineation of its extent as inferred from CT imaging aids in prognosis and appropriate surgical management.

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Introduction

Fungi are ubiquitous organisms known to cause opportunistic infections in immunocompromised hosts.¹ Of these, acute invasive fungal rhinosinusitis (AIFR) is a fulminant infection resulting in high morbidity and mortality. The mortality rate has been reported to be as high as 50 to 80 percent.² This has been a rare disease with a reported incidence of around 2% in immunocompromised patients, most susceptible group being those with haematological diseases.³ The recent COVID 19 pandemic has witnessed a high surge in cases of AIFR especially of the *Mucor* species, which was the less common cause of AIFR in the pre covid era, second to *Aspergillus*.⁴ The Indian subcontinent witnessed a dramatic spurt in cases of COVID associated mucormycosis (CAM) and by June 7, 2021, a spate of 28,252 cases of mucormycosis were reported, 86% having a history of COVID-19 infection.^{5,6}

COVID associated mucormycosis (CAM) is seen to affect both subset of patients, those with active COVID 19 infection and those who have recently recovered in the last 4-6 weeks. An ideal environment in the clinical setting of COVID 19 is offered by low oxygen (hypoxia),

high glucose (diabetes, new onset hyperglycemia, steroid-induced hyperglycemia), acidic medium (metabolic acidosis, diabetic ketoacidosis), high iron levels (increased ferritins) and decreased phagocytic activity of white blood cells due to immunosuppression (SARS-CoV-2 mediated, steroid-mediated or background comorbidities) along with prolonged hospitalization with or without mechanical ventilation.⁷

The pathogenesis involves inhalation of ubiquitous fungal spores which proliferate and invade the nasal mucosa of the susceptible host, and spread to the paranasal sinuses with subsequent involvement of the orbit and brain, if not treated early.

CT is considered the first line imaging modality for assessment of AIFR or CAM. CT aids both in early diagnosis as well as delineating the extent of disease. In addition, it provides a roadmap for surgical approach and management. MR is usually reserved for cases with features of orbital apex syndrome or intracranial involvement such as mental status changes, seizure, or stroke.⁸

In a study in 2015 by Middlebrooks E et al.⁹ a seven variable model of imaging findings was devised on CT to diagnose AIFR. The seven variables included periantral fat, bone dehiscence, orbital invasion, septal ulceration, pterygopalatine fossa, nasolacrimal duct and lacrimal sac. They found that the presence of abnormality in a single variable in the model had an 87% positive predictive value (PPV), 95% negative predictive value (NPV), 95% sensitivity, and 86% specificity. The presence of abnormality in two variables increased PPV and specificity to 100%. Thus, CT was a robust tool for diagnosing AIFR.

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TABLE 1
Checklist for reporting cases of acute invasive fungal rhinosinusitis

Anatomical regions involved	Imaging findings to look for
1. Nasal cavity and nasopharynx	<ul style="list-style-type: none"> • Hypertrophy or ulceration of mucosa • Emphysematous soft tissue content within nasal cavity • Nasal septum ulceration • Necrotic turbinates
2. Paranasal sinuses- maxillary, ethmoid, frontal, sphenoid	<ul style="list-style-type: none"> • Mucosal hypertrophy or emphysematous soft tissue contents • Hypodense contents with hyperdense fungal hyphae
3. Periantral region	<ul style="list-style-type: none"> • Fat stranding and heterogeneity in preantral and retroantral region • Soft tissue contents or abscess formation in preantral and retroantral region
4. Sphenopalatine foramen, pterygopalatine fossa, infratemporal fossa	<ul style="list-style-type: none"> • Fat stranding and heterogeneity • Soft tissue contents with complete obliteration of fat
5. Deep spaces- Masticator, buccal, parotid	<ul style="list-style-type: none"> • Fat stranding and heterogeneity • Complete obliteration of intramuscular fat planes • Granuloma/ abscess formation
6. Nasolacrimal duct and sac	<ul style="list-style-type: none"> • Soft tissue infiltration in nasolacrimal duct and sac
7. Hard palate	<ul style="list-style-type: none"> • Mucosal hypertrophy, ulcerations and bony erosions
8. Bones	<ul style="list-style-type: none"> • Rarefaction and bony erosions of walls of sinuses, orbit, base of skull
9. Orbit	<ul style="list-style-type: none"> • Fat stranding/soft tissue in extraconal compartment • Fat stranding/soft tissue in intraconal compartment • Intraorbital abscess/Subperiosteal collection • Bulky Extraocular Muscle • Optic nerve thickening and stretching • Thickening of coats of globe • Posterior globe tenting • Orbital apex involvement
10. Intracranial compartment	<ul style="list-style-type: none"> • Meningitis • Extra-axial collection • Neural foramina widening • Intraparenchymal granulomas/abscess
11. Vascular complications	<ul style="list-style-type: none"> • Cavernous sinus thrombosis • Arterial narrowing/thrombosis/pseudoaneurysm • Infarct

In this review we emphasize the subtle CT findings of CAM for early diagnosis and CT features of disease extension with representative illustrative examples. The relevance of these findings to clinical decision making is also emphasized. A comprehensive checklist is presented to ensure clinically relevant reporting of imaging findings (Table 1).

Clinical Signs and Symptoms

Symptoms of early CAM that is limited to nose and paranasal sinuses include nasal obstruction, nasal crusting and nasal discharge that could be brownish or blood tinged. Patients characteristically present with a painless, necrotic nasal septal ulcer called eschar.¹⁰

Intraorbital spread seen in moderately advanced disease manifests clinically as proptosis and periorbital swelling, diplopia, eye pain, ptosis and loss of vision. Infraorbital nerve involvement may result in loss of sensation on face /cheek. Inferior spread into the upper alveolus may cause loosening of teeth, discoloration/ulceration of palate and palatal perforation. Swelling and ulcerative lesions on gums can be seen before frank alveolar involvement. Disease spread to infratemporal fossa can cause pain in jaw movement and decreased mouth opening due to involvement of pterygoid muscles.

Advanced disease with intracranial infection may present with III-VI cranial nerve palsy, hemiparesis and hemiplegia, altered sensorium, seizures and coma.

As initial symptoms like nasal obstruction and discharge are non-specific and can mimic “run of the mill” rhinosinusitis, imaging aids in detection of subtle signs of this highly morbid and potentially fatal disease entity, enabling early diagnosis and prompt initiation of therapy. Hence, imaging plays a key role in reducing the morbidity and mortality from this disease.

CT Imaging Protocol

For the complete evaluation of sinuses and surrounding structures, CT scan is performed, using thin collimation ranging from 0.5 to 1 mm in axial plane and multiplanar reconstructions are obtained in coronal and sagittal planes. Both soft tissue and bone algorithm reconstructions are obtained for evaluation of extrasinus extension and bony rarefactions/erosions. Non-ionic iodinated intravenous contrast is administered unless contraindicated, at a maximum dose of 1-1.25 mL/kg body weight via the peripheral intravenous route. This helps in delineation of intraorbital and intracranial extension. The field of view includes superior extent of frontal sinus cranially, the upper jaw inferiorly, ala of nose anteriorly and cavernous sinuses posteriorly.

Imaging Features

Sinonasal Disease

Nasal Cavity

Unilateral mucosal thickening of the nasal cavity has been considered to be a consistent and somewhat specific feature seen in AIFR.¹¹ However, a large subset of patients with CAM present with bilateral involvement. In a recent study, it was found that AIFR caused by *Aspergillus* had predominantly unilateral findings whereas disease due to *Mucor* species, the species most often associated with CAM, had greater bilateral sinonasal involvement.⁴

Early findings include emphysematous soft tissue or ulcerative mucosal changes in the nasal cavity (Fig. 1a,b). The middle turbinate is the most common site of involvement in the nasal cavity. There

may be features of non enhancement of the turbinate and/or bone destruction. This lack of contrast enhancement on imaging is a result of angioinvasive nature of these fungi causing tissue necrosis. This gives the characteristic ‘black turbinate sign’ initially described on MRI. Although MRI gives a superior depiction, the sign can very often be appreciated on CT (Fig. 1c). Severe cases may reveal destruction of the nasal septum (Fig. 2a).

Soft tissue may also be seen extending along the nasolacrimal duct from the inferior meatus to lacrimal sac along the inferomedial orbit. Posterior extension to involve the nasopharynx in the form of mucosal thickening or ulceration may also be seen (Fig. 2b).

Paranasal Sinuses

Hypodense mucosal thickening/soft tissue opacification of sinuses may be seen on non-contrast CT scan which often demonstrate

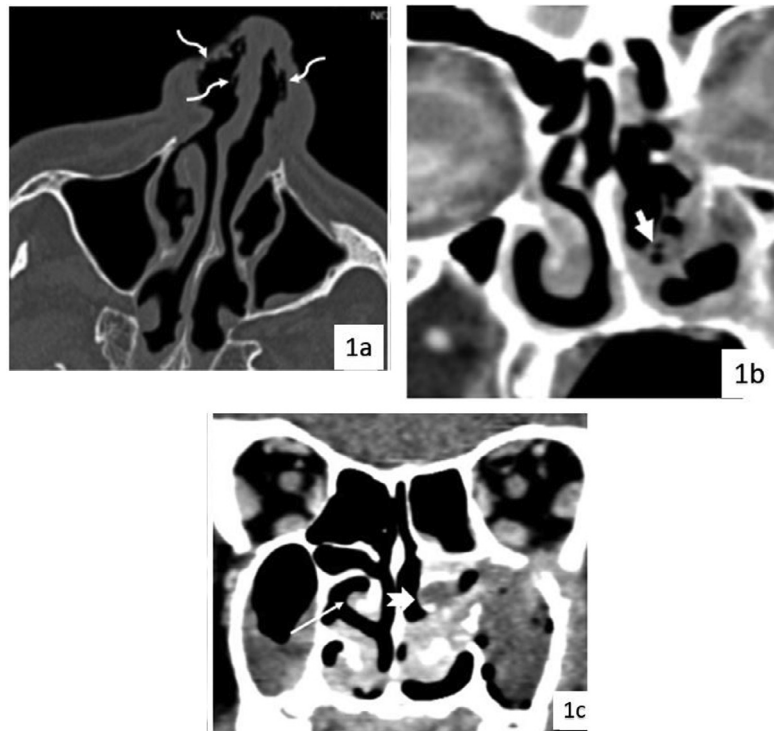


FIG. 1. Covid associated mucormycosis in three different patients. Axial bone window CT (a) and coronal soft tissue reformatted images (b,c) depict ulcerated mucosal lining involving vestibule and nasal septum (curved arrows in a), emphysematous nasal cavity mucosal thickening (short straight arrow in b) and necrotic left middle turbinate (notched arrow in c). Contralateral normal enhancing right middle turbinate is noted (long straight arrow).

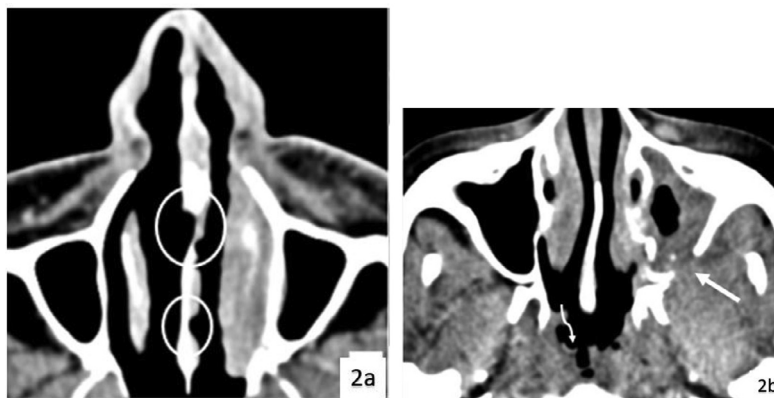


FIG. 2. Covid associated mucormycosis in a 43 year old male patient 4 weeks after recovery from COVID 19. Axial contrast enhanced CT (a) shows ulcerated nasal septum mucosa (white circle). Ulcerative mucosal changes seen involving the nasopharynx (curved white arrow in b) in another patient with CAM with associated destruction of posterior wall of maxillary sinus as well as retroantral and masticator space involvement (straight white arrow in b).

hyperdense fungal hyphae within the sinus. Although the mucosal thickening may be mild, presence of signs of invasion on CT point to the correct diagnosis. Obliteration of periantral fat with stranding or soft tissue thickening is another subtle sign of early AIFR (Fig. 3). These findings are often seen with intact intervening bone owing to spread of disease through perivascular channels.

Apart from the finding of peri antral soft tissue, presence of soft tissue within the pterygopalatine fossa (PPF) is an important imaging sign to indicate extrasinus invasion. Disease spreads from sphenopalatine foramen along sphenopalatine artery into PPF. This is a major neurovascular crossroad in head and neck region through which a perivascular/perineural spread of disease may be seen in multiple directions (Fig. 4).

- Anteriorly, the inferior orbital fissure is the gateway to the orbit.
- Medially, the sphenopalatine foramen communicates with the nasal cavity.
- Laterally, the pterygomaxillary fissure serves as a pathway of spread to the infratemporal fossa.
- Inferiorly, it is connected to the oral cavity via the greater palatine canal.
- The foramen rotundum and pterygoid canal connects the PPF to the middle cranial fossa.

The PPF has been described as a major reservoir for AIFR and its debridement may effectively seal off the gap for the spread of disease.¹²

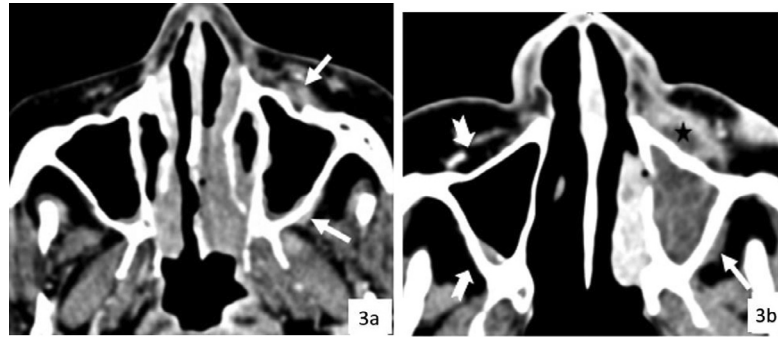


FIG. 3. Covid associated mucormycosis in two patients with a history of prolonged use of steroids. Contrast-enhanced axial CT images depict different extent of periantral fat involvement. Mild left peri antral soft tissue (arrows in a) is seen in the first patient. Rim enhancing abscess in left pre antral soft tissue (star) with mild retro antral soft tissue (arrow in b) is seen in the second patient. Contralateral pre antral and retroantral fat is normal (notched arrows in b).

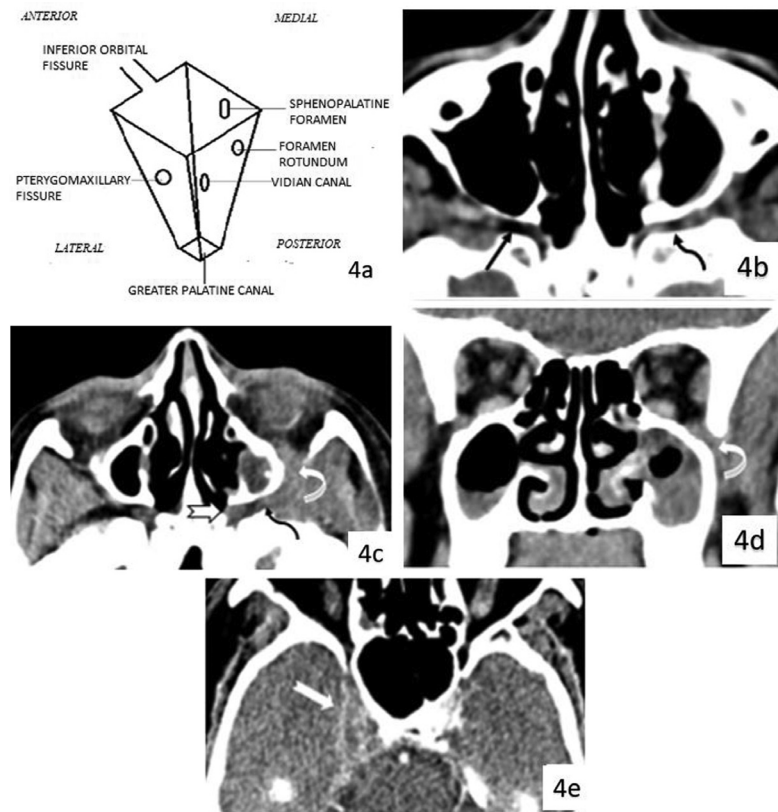


FIG. 4. Illustration (a) depicting the pterygopalatine fossa (PPF, inverted truncated cone) and routes of its multidirectional spread. Three different patients with CAM depicting multidirectional spread via PPF. Axial contrast enhanced computed tomography (CECT) image (b) depicts minimal soft tissue in left pterygopalatine fossa (curved black arrow in b), contralateral PPF is normal (straight black arrow). Axial and coronal CECT images in another patient (c,d) depict soft tissue contents within posterior nasal cavity extending via sphenopalatine foramen to PPF (notched white arrow in c), further extending into infratemporal fossa via pterygomaxillary fissure (curved black arrow in c). Spread to the orbit is seen via inferior orbital fissure (circular white arrow in c and d). Axial CECT image (e) reveals perineural extension of disease to middle cranial fossa via foramen rotundum (notched white arrow in e).

Bone changes: Bone dehiscence or erosion is a highly specific marker for AIFR albeit with low sensitivity as it is generally considered to be a delayed imaging appearance.^{9,13} In their retrospective case control study, Middlebrooks E et al⁹ reported that bone dehiscence was a variable which had 100% specificity for AIFR but a low sensitivity of about 40%. Another study by DelGaudio et al¹³ found bony erosions in only 35% of their patients. These bone changes may be evident in sinus walls, turbinates or the hard palate giving a mottled appearance (Fig. 5).

Clinical Relevance

The knowledge of the early imaging features and signs of invasion is critical for early diagnosis for CAM.

Knowledge of extension of disease has indispensable role in management of CAM along with the surgical approach. Disease limited to the nose and paranasal sinuses can be cleared endoscopically through the nose. Detection of disease in PPF on imaging is crucial for management, although clinically hidden behind intact posterior wall of the maxillary sinus. Involvement of the pterygopalatine fossa and infratemporal fossa requires a different approach to management

(Denker's procedure). When the maxilla/upper alveolus is affected, an open sublabial approach is preferred.

Pattern of extrasinus spread of disease: It is crucial to understand the pattern of spread of disease outside the sinuses, as this has a bearing on prognosis and management of the patient. The following are the recognised patterns of spread.

Deep neck spaces: The presence of buccal space, masticator space and parotid space adjacent or near the paranasal sinuses make these an understandable site of spread of disease. The involvement of these spaces can be evident by soft tissue heterogeneity and fat stranding within these spaces or formation of granulomas/ abscesses. Spread may be witnessed as contiguous involvement from sinuses with frank bone destruction or may be seen with intact bony walls indicating a perivascular/perineural spread, the latter being a commoner pathway of spread. Multidirectional spread of disease from pterygopalatine fossa has already been described (Fig. 4).

Orbit: It has been described to be the most common site of extrasinus involvement in mucormycosis.^{14,15} Orbital spread may occur via congenital dehiscence/ destruction of lamina papyracea or through various foraminal gateways. The latter has been found to be commoner pathway of spread.¹⁶ The anterior and posterior ethmoidal foramina may transmit the disease to the medial quadrant,

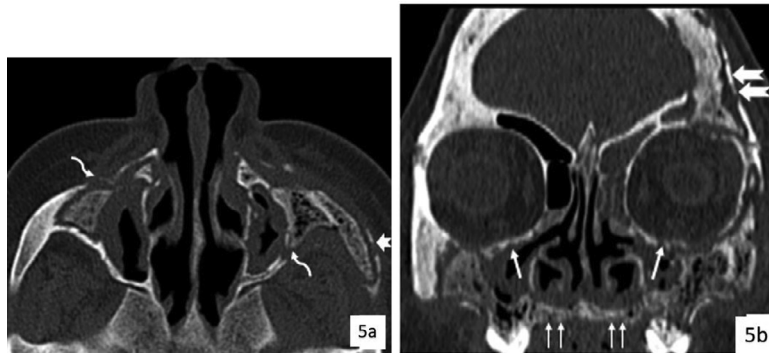


FIG. 5. A 50-year-old female with uncontrolled hyperglycaemia and CAM. Axial (a) and coronal reformatted (b) CT images demonstrate bony erosions of walls of bilateral maxillary sinuses (curved arrows), left zygomatic bone (notched arrow), left frontal bone (2 notched arrows), floor of bilateral orbits (Thin straight arrows) and hard palate (2 thin straight arrows).

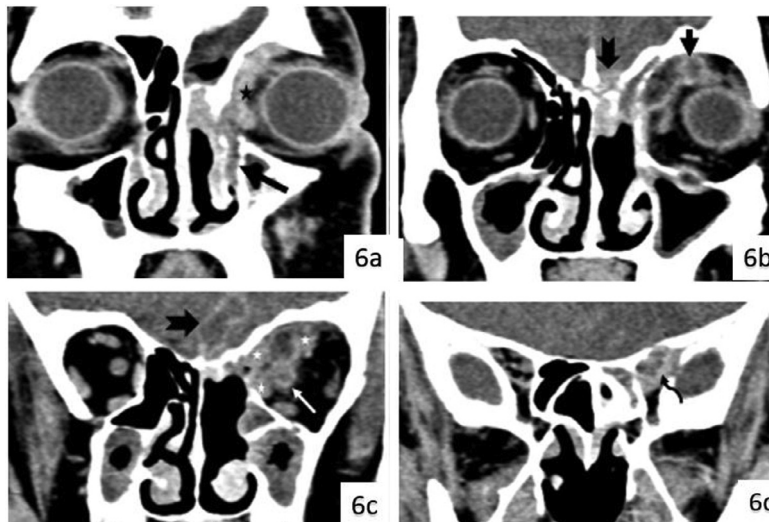


FIG. 6. Two patients with CAM with orbital involvement. Coronal contrast enhanced CT of the first patient shows (a) infiltration of soft tissue into left nasolacrimal duct (black arrow), lacrimal sac and medial orbital quadrant (black star). The patient underwent left orbital exenteration and pus filled lacrimal sac was found intra-operatively. In another post functional endoscopic sinus surgery (FESS) CAM patient and no perception of light in left eye, coronal CT images (b-d) show abscess in superomedial compartment of left orbit (black arrow in b) extending posteriorly to involve the optic nerve which shows thickening and rim enhancement (white arrow in c). Adjacent extraocular muscles are bulky (white stars in c). The disease extends to the orbital apex (curved black arrow in d). Intracranial extension is also depicted through the eroded cribriform plate (notched black arrows in b and c).

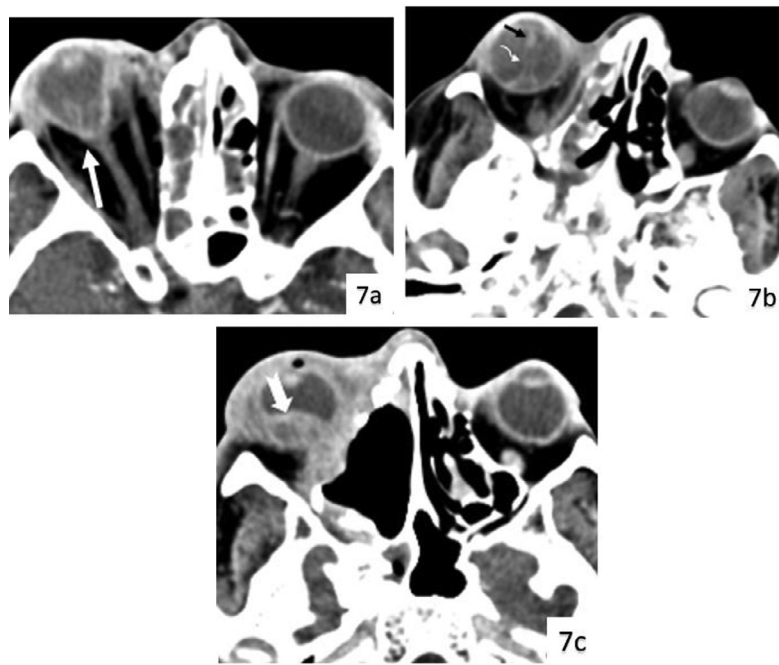


FIG. 7. Three different patients with CAM and globe involvement with complete loss of vision in right eye. Axial contrast enhanced CT images reveals proptosis of right eye with optic nerve stretching and conical deformity of posterior globe giving the “Guitar pick” sign (straight white arrow in a), choroid detachment (curved white arrow in b) and lens dislocation (black arrow in b) and heterogeneously enhancing soft tissue involving the coats of the globe and extending into the globe (notched white arrow in c).

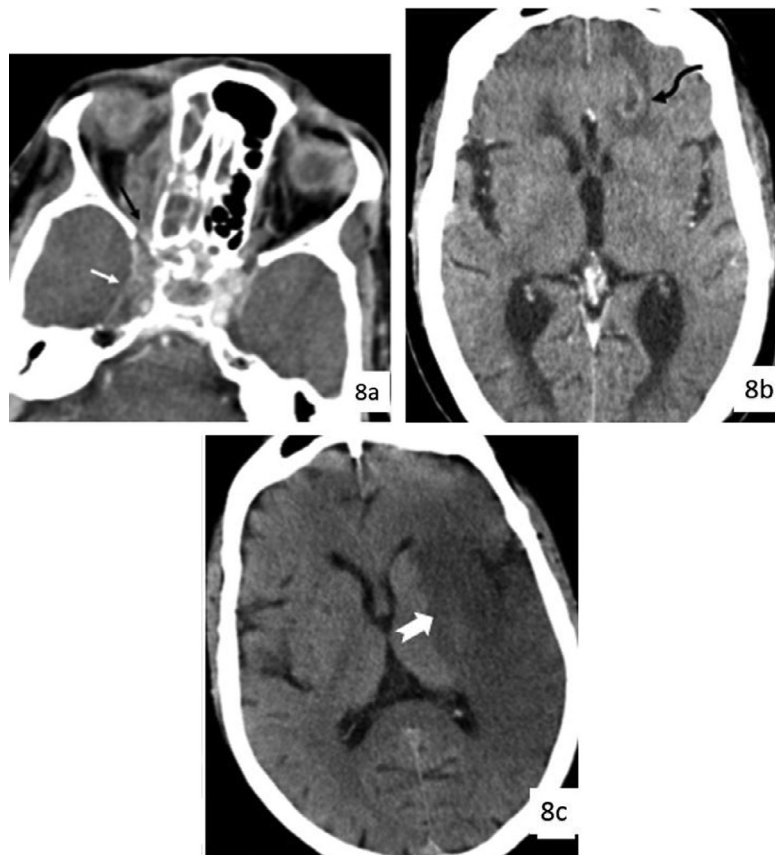


FIG. 8. Three different patients with CAM with intracranial involvement. Contrast enhanced CT axial images reveals heterogenous enhancing soft tissue thickening in right sided sinuses with extension into the right orbital apex (black arrow in a) with contiguous spread to the cavernous sinus and its thrombosis (straight white arrow in a), rim enhancing abscess in left frontal lobe (Curved black arrow in b) and infarct in territory of left middle cerebral artery (notched white arrow in c).

infraorbital foramen and inferior orbital fissure to inferior and inferolateral quadrant respectively and nasolacrimal duct to the inferomedial quadrant. Orbital involvement on imaging may be seen as intra or extraconal fat stranding or as intra orbital soft tissue content /abscess. The involvement of the extraocular muscles is suggested by their heterogenous and bulky appearance. Extension into the orbital

apex is a critical finding as disease here can easily spread intracranially to cavernous sinus region (Fig. 6).

While optic nerve sheath complex and globe involvement is better evaluated on MR, observations of optic nerve thickening and encasement by orbital soft tissue can be seen. In cases of raised intra-orbital pressure, stretching of the optic nerve with conical deformity

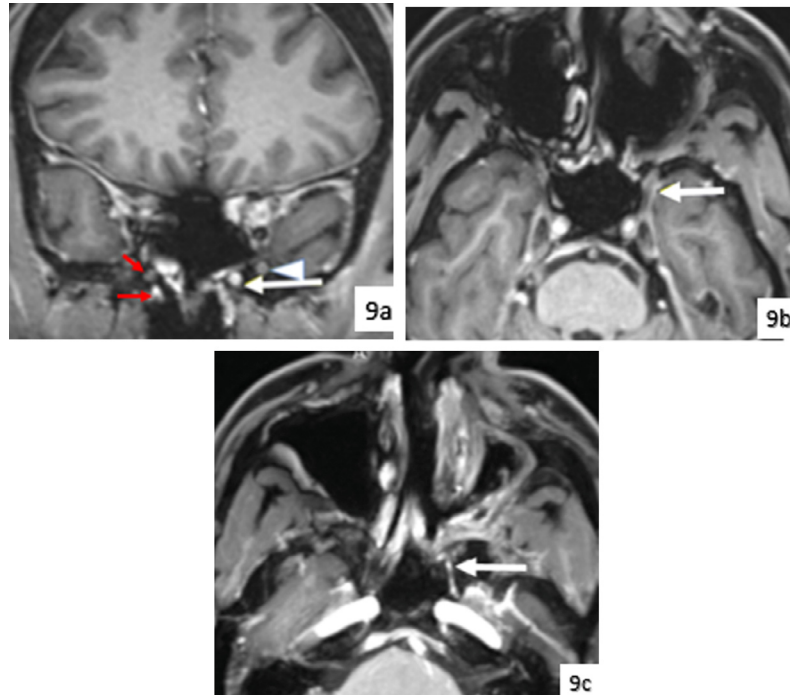


FIG. 9. A 43-year-old patient with CAM. Post contrast coronal (a) and axial MR images (b,c) depict enlarged left foramen rotundum and vidian canal (white arrowhead and white arrow in a respectively) with thickened and enhancing left maxillary (arrow in b) and vidian nerves (arrow in c). Normal contralateral foramina are depicted by red arrows in a. (Color version of figure is available online.)

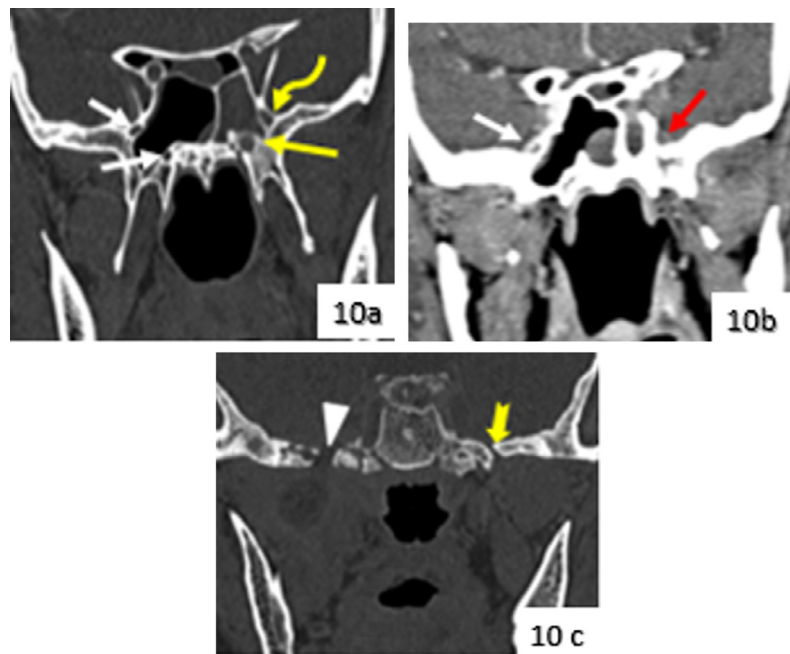


FIG. 10. A 58-year-old diabetic with CAM; Coronal reformatted CT at bone (a) and soft tissue (b) windows demonstrate perineural spread of disease, seen as subtle widening of vidian canal (yellow arrow in a) and foramen rotundum (curved yellow arrow in a) with loss of juxtaforaminal fat pad (red arrow in b). Normal contralateral foramina are depicted by white arrows. Coronal reformatted (c) CT image in another case of CAM depicts widened and eroded right foramen ovale (white arrowhead in c). Note made of normal left foramen ovale (notched yellow arrow in c). (Color version of figure is available online.)

of posterior globe (guitar pick sign) can be suggestive findings on CT (Fig. 7a). The involvement of globe may be seen as direct involvement, with soft tissue abutting/extending into it with thickening of its coats or deformation (Fig. 7b,c).

Intracranial Spread

Intracranial spread may occur by various routes - via the orbital apex from the orbit, direct extension from the sphenoid sinus and through the roof of ethmoid sinus. The intracranial structures involved are the meninges with imaging correlate of pachymeningitis; cavernous sinus involvement seen as cavernous sinus thrombosis (Fig. 8a); brain parenchyma involvement demonstrated as a granuloma or abscess (Fig. 8b); and vascular involvement visualised as an arterial infarct (Fig. 8c). Extra axial collections as epidural or subdural abscess may also be seen.

A perineural mode of spread may also be observed. Although enhanced/ thickened nerves are seen better with MRI (Fig. 9), certain CT features can point toward perineural spread of disease. CT can help detect subtle foraminal widening, loss of juxtaforaminal fat pad, eroded foramina (Fig. 10) and secondary denervation changes in the muscles supplied by the nerve. In the head and neck region, these typically are the muscles of mastication supplied by the mandibular division of trigeminal nerve which can show edema and enhancement in the acute stage of denervation.

Although MRI has superior contrast resolution for intracranial findings, contrast enhanced CT can also aid in detecting these. Altering window settings on the viewing stations can further aid in revealing the less obvious findings such as cavernous sinus thrombosis, the most common form of intracranial involvement encountered.

Clinical Relevance

The awareness of the various patterns of spread of extrasinus disease is crucial as it helps to manage and prognosticate the patients. In addition to clinical symptomatology, the extent of orbital and brain involvement plays a key role in making surgical decisions. Minimal periorbital disease in medial and inferior compartment can be easily cleared endoscopically by removing lamina papyracea. Frank orbital involvement or orbital apex involvement with blind eye needs orbital exenteration. All patients having intracranial brain parenchymal disease need neurosurgical intervention.

Conclusion

AIFR especially of the *Mucor* species has witnessed a re-emergence in the present COVID 19 pandemic. The detection of early subtle signs of CAM on imaging can aid in prompt diagnosis of this fulminant and fatal entity. A checklist for reporting CAM is provided (Table 1) that can aid in exact delineation of disease on imaging and help in prognosticating as well as deciding the course of appropriate surgical management.

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