

## Invasive fungal sinusitis: A case report

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### ABSTRACT

Fungal sinusitis is the mucosal inflammation of paranasal sinuses (PNSs) due to fungus etiology. The entity remained underdiagnosed earlier because of the non-existence and non-availability of imaging modalities. There is a wide range of clinical symptomatology. Fungal sinusitis requires precise evaluation and decision in type and diagnosis for the correct and appropriate management by an otorhinolaryngologist. The reason for this is being unique radiological features in fungal sinusitis in computerized tomography (CT) and magnetic resonance (MRI). We present the case of a 16-year-old boy who presented with a chronic history of nasal blockage and running nose of 3 months duration and now with fever and headache for the past 2 weeks. He underwent plain X-ray, NCCT, and MRI of PNS. He was diagnosed as having invasive fungal sinusitis on the basis of clinical and classical CT findings supplemented with MRI. He had further been advised surgical contemplation for the total cure as medical management is not the answer. Radiological modalities can precisely differentiate the different subtypes of fungal sinusitis. Otorhinolaryngologists make the management decision and precision on the type of fungal sinusitis keeping in view of involving the neighboring structures.

**Key words:** Fungal sinusitis, Otorhinolaryngologist, Paranasal sinuses, Underdiagnosed

**F**ungal sinusitis is a rare entity that can affect both healthy and immunocompromised individuals. The diagnosis is not made in the early stages because of concealed symptomatology and not able to undergo cross-sectional imaging investigations. Fungal sinusitis can be presented in different ways as allergic fungal sinusitis (AFS), fungal ball, chronic indolent fungal sinusitis, and invasive variety. Approximately 65% of cases of AFS had an association with asthma.[1] The frequency pattern of involvement is ethmoid, maxillary, frontal, and sphenoid sinuses. The management depends on the correct diagnosis as per radiological evaluation against the use of respective modality. The immunological and histopathological evaluation further adds to the confirmation of the pathology.[2,3]

### CASE REPORT

A 16-year-old boy reported with complaints of nasal blockage, running nose, and anosmia for the past 3 months. He gave a history of off and on fever with a headache for 2 weeks. There was no

history of vertigo, seizures, or any other neurological deficit and no history of any chronic illness such as diabetes, hypertension, or tuberculosis. He did not have any history of previous surgery or interventional procedure. He had been taking symptomatic treatment in the form of nasal decongestants and analgesics but without any permanent cure.

On examination, he was well preserved without any physical deformity. Systemic examination was non-contributory. All the vitals were well preserved and the temperature was normal at the time of examination. On direct rhinoscopy, the left nostril had hypertrophied congested mucosal surface obstructing the nasal space totally.

All the biochemical parameters were within normal limits. The routine baseline ultrasonography was unremarkable. The patient was evaluated radiologically by plain radiography of the paranasal sinuses (PNS) which had shown opaque left maxillary sinus, mucosal hypertrophy of the right maxillary sinus, and deflected nasal septum toward the right (Figure 1a-c). He was further subjected to computerized tomography (CT) scan and magnetic resonance imaging (MRI) of the PNS to know more details about the pathology. Non-contrast CT (NCCT) and contrast CT were done to see precisely the extension of the invasive nature of the pathology. NCCT showed an inhomogenous mass with increased

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density in the left sinonasal complex. There was no calcification seen (Figure 2a-d). Contrast-enhanced CT (CECT) showed enhancement of the non-necrotic part (Figure 3a-f). MRI was also carried out to see precisely more anatomical details, angiography, and venography. MRI showed T2WI hyperintense fungal lesion in the left maxillary sinus traversing through the left nostril and extending into the intracranial compartment after the destruction of the bone. The right maxillary sinus simply showed mucosal hypertrophy. There was no evidence of any vascular invasion or sinus thrombosis (Figures 4-6).

The patient was clinicoradiologically diagnosed with an invasive form of fungal sinusitis with intracranial extension and bone erosions. He was advised for surgical management as it is not possible to manage medically like acute fungal sinusitis.

## DISCUSSION

Rhinosinusitis is the most appropriate term to be used rather than sinusitis as there is a continuation of sinus mucosa with the nose. The severity could be acute lasting <1 month, subacute lasting from 1 to 3 months, and chronic which is more than 3 months.[4] Fungal sinusitis is divided into two groups comprising non-invasive and invasive. The non-invasive group includes acute fulminant fungal sinusitis, granulomatous invasive, and the invasive group includes AFS.[5] AFS is more common in the young immunocompromised populations and more prevalent

in a warm and humid climate. The underlying etiological factor is allergic reaction to aerosolized fungus which causes Type I and IgE-mediated hypersensitivity reaction. The most common fungi responsible are dematiaceous and hyaline molds.[6,7] *Aspergillus* falls in the latter category.[8] Those who undergo surgical manipulation constitute 5–10% and have chronic rhinosinusitis symptoms. In histopathological evaluation, they all had hypertrophic mucosal surfaces.[9] They show the opacification of the sinuses in radiological evaluation. These require precise differentiation from the invasive variety of the disease. The disease could be unilateral or bilateral.

The exact evaluation of the entity requires diagnostic techniques ranging from plain PNS radiograph to CT and MRI. Plain radiograph of PNS shows simple opacification of sinuses to air-fluid levels. Air-fluid levels are seen in bacterial sinusitis in 60% of cases and not in other types. Mucosal hypertrophy can be seen in 90% of cases but remains non-specific finding. This is in the form of an opacified line seen along the bony margins of the sinuses. Retention cysts and polypoid masses can be visualized in addition to other findings. It is very rare to see the bone erosion unless deep penetration study is done to visualize the adjacent bone.[10] CT evaluation is the choice of investigation as this can show the anatomical and pathological extension of the disease. This is helpful in chronic cases but not of much importance in acute cases. Coronal sections are very useful in making the correct diagnosis. CECT displays an excellent view

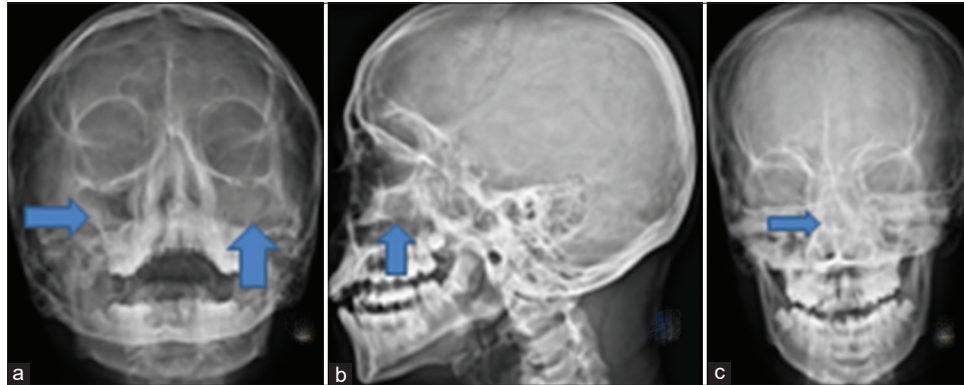


Figure 1: Plain X-rays paranasal sinuses (PNS) and skull (SXR). (a) Waters view for PNS shows opacified left maxillary sinus (vertical blue arrow) and mucosal hypertrophic lining in the right maxillary sinus (horizontal blue arrow). (b) SXR lateral view shows superimposed opaque sinuses (vertical blue arrow). (c) Anteroposterior view of SXR shows deviated nasal septum to the right (horizontal blue arrow)

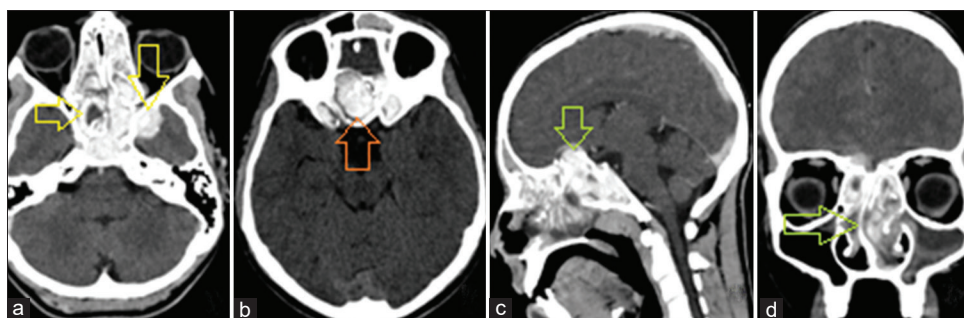


Figure 2: Computerized tomography of paranasal sinuses. (a) Axial section shows hyperdense lesion extending from the left nostril to intracranial compartment (vertical yellow arrow) and the right side also shows hyperdense lesion in the right nostril (horizontal yellow arrow). (b) Axial section shows hyperdense ethmoid group (red arrow). (c) Contrast sagittal section shows enhancing lesion with superior extension (green arrow). No evidence of cerebritis seen. (d) Coronal section shows right DNS (green arrow) with expanded left nasal space

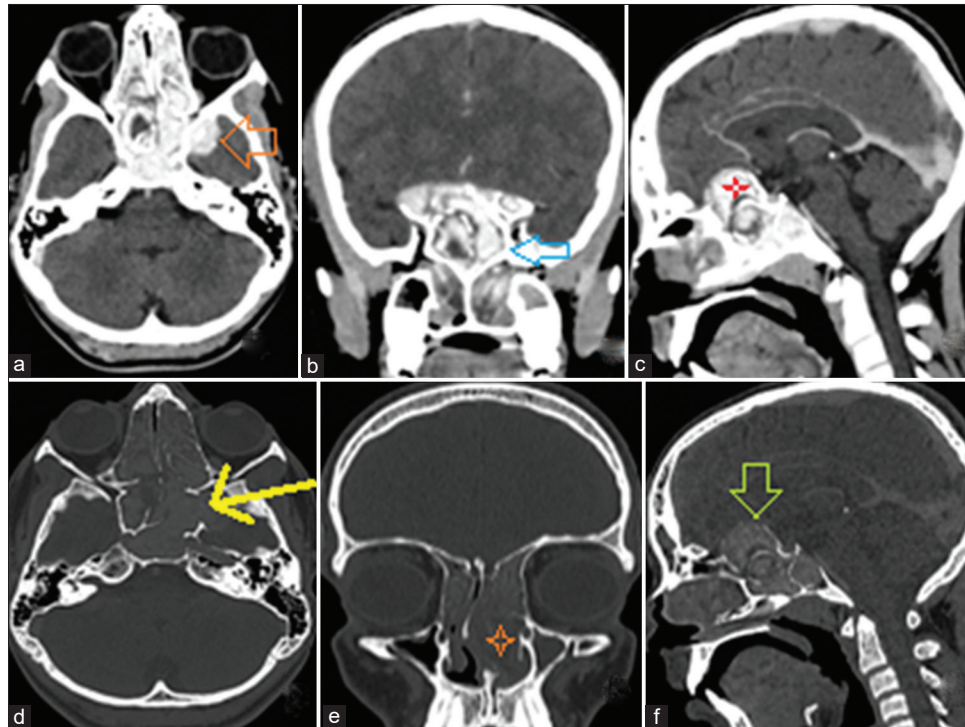


Figure 3: Contrast-enhanced computed tomography and bone window sections. (a) Axial section with intracranial enhancing fungal mass (red arrow). (b) Coronal section shows extension of infection in the ethmoidal region (blue arrow). (c) Sagittal section shows superior extension of fungal mass (red star). (d) Axial section in bone window shows expansion with erosion of bony outline (yellow arrow). (e) Coronal section shows expansion of the left nasal space (red star). (f) Sagittal section shows intracranial extension with bone erosion and destruction (green arrow)

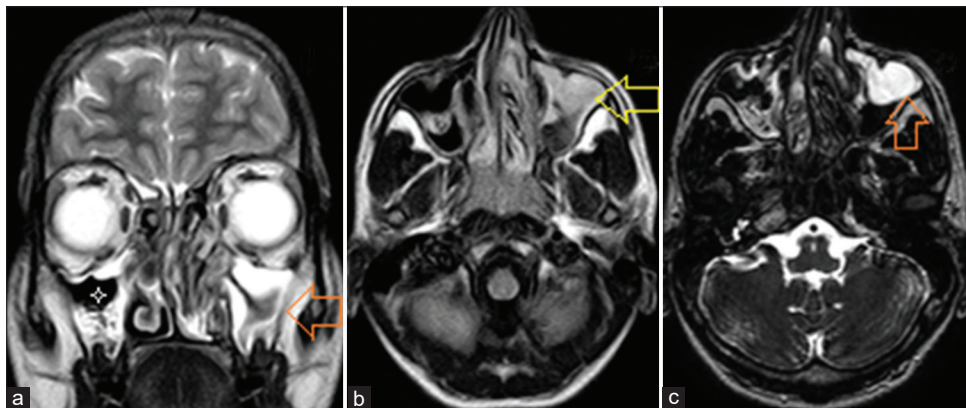


Figure 4: Magnetic resonance imaging of peripheral nervous system. (a) T2WI coronal section shows hyperintensity in the left maxillary sinus extending through the left osteomeatal complex (red arrow) with mucosal hypertrophy in aerated right maxillary sinus (white star). (b) Fluid-attenuated recovery axial section highlights the hyperintense fungal mass (yellow arrow). (c) T2WI drive shows the abnormal hyperintense fungal areas

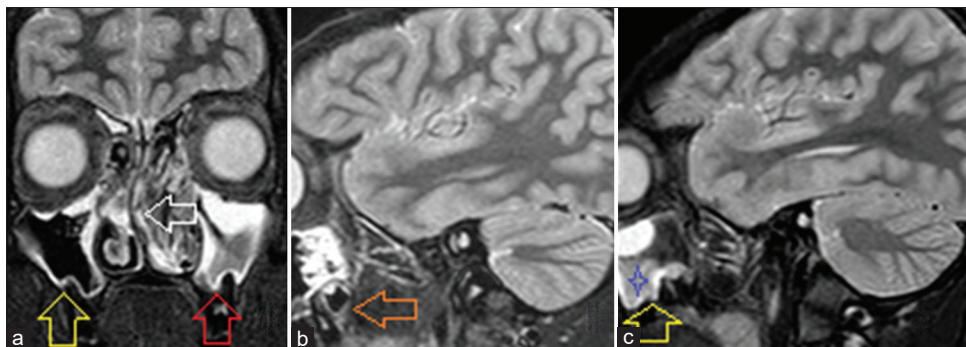
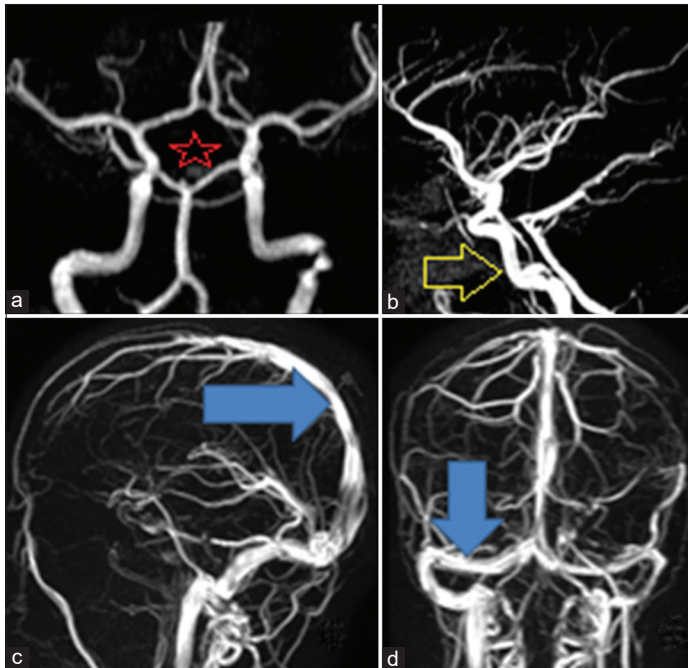


Figure 5: Magnetic resonance imaging short-tau inversion recovery sequences. (a) Coronal section shows peripheral hyperintensity with central hypointensity in the left maxillary sinus (red arrow). The right maxillary sinus shows mucosal hypertrophy (yellow arrow). (b) Sagittal section through the right maxillary sinus shows mucosal hypertrophy (red arrow) and (c) through the left maxillary sinus, reflects the hyperintense lesion with central hypointensity (yellow arrow)



**Figure 6: Magnetic resonance vascular mapping. (a and b) Time-of-flight angiography shows normal Circle of Willis (red star) and internal carotid vessels (yellow arrow). There was no angioinvasion noticed. (c and d) Venography shows normal superior sagittal sinus (blue arrow) and sigmoid venous sinuses (inverted blue arrow)**

of soft tissue and bone details. NCCT shows hyperdensity in the center of the lesion with surrounding hypodense region. Contrast studies are not required in these plain cases.[11,12] The anatomical road map is most suitable for the functional endoscopic sinus surgery (FESS).[13] 2D and 3D CT are very useful in making the diagnosis more suitable for the appropriate management.[14] MRI is further useful in knowing the invasion and extension to the surrounding structures as was our present case. There are classical findings in the form of peripheral T2WI hyperintensity with the central hypointensity of the fungal sinusitis. Hypointensity is because of minerals deposited in that region. T1WI is not of many advantage as the pathology remains isointense or hypointense. Post-gadolinium T1WI further confirms the enhancement in the peripheral part.[15]

The division of medical and surgical treatment can be made by the type of sinusitis. Invasive and chronic sinusitis is treated surgically and others are managed medically. The revolution of FESS has changed totally the guidelines of surgical management. Sinusitis results after the obstruction of the ostium of the maxillary sinus and hiatus semilunaris where an anterior group of sinuses drain. The main principles of FESS are to clear these obstructions responsible for chronic sinusitis.[16] The detailed anatomy by CT and MRI plays a pivot role in FESS surgery and follow-up.

## CONCLUSION

Radiological modalities can precisely differentiate the different subtypes of fungal sinusitis. Otorhinolaryngologist makes the management decision of FESS as per the type and involvement of the neighboring structures. Fungal sinusitis remains undiagnosed for a long period and becomes invasive as was in our case. Acute cases can be managed medically without surgical contemplation. The delay in diagnosis leads to complications which makes surgical management still more difficult.

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