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# SILENT SPREAD AT THE SKULL BASE;A PICTORIAL JOURNEY THROUGH OSTEOMYELITIS

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**ABSTRACT**

Skull base osteomyelitis (SBO) is a potentially life-threatening infection that predominantly affects immunocompromised individuals and elderly diabetic patients. Often, the clinical diagnosis is difficult to arrive at, resulting in intracranial complications that are difficult to treat. Early recognition of the condition is challenging, owing to the nonspecific clinical manifestations. Imaging, therefore, becomes crucial for diagnosis as well as for determining the extent of the disease, thereby guiding and monitoring response to therapy. This pictorial essay showcases the diverse imaging spectrum of SBO on MRI and non-contrast CT, focusing on the differences in extent of skull base involvement based on the etiopathogenesis of infection.

## INTRODUCTION

Skull base osteomyelitis is an infective process which may predominantly involve the middle cranial fossa alone, or spread to involve all three anatomic components of the base of skull. Although conventionally SBO has been described as a complication of malignant otitis externa, there are two different varieties based on aetiology .(1,2)

- Typical (otogenic) SBO: with identifiable otological source.
- Atypical (central) SBO: without clear otologic source

Otogenic skull base osteomyelitis affects the elderly with long-standing diabetes mellitus and usually presents clinically with severe otalgia and otorrhea along with varying degrees of hearing loss; the symptoms often being disproportionate to the findings of clinical examination. (1,4,8).Concomitant involvement of the facial nerve (cranial nerve VII) as it traverses the petrous temporal and mastoid bone, is also commonly associated resulting in various patterns of facial palsy. Cranial nerves V to XII may be also affected by contiguous spread to posterior cranial fossa(4,6,9). Microbiologically, the aerobic bacterium *Pseudomonas aeruginosa* is the predominant causative pathogen, although in non-diabetic immunocompromised individuals, fungal organisms may be involved in etiopathogenesis. The pathway of otogenic spread is from the external and middle ear, through the petrous apex and clivus to the cisternal segments of the cranial nerves to begin with, and subsequently cerebral parenchymal involvement with abscess formation, if left untreated. (2,4,6,9)

Atypical or Central skull base osteomyelitis is usually the infection of younger, middle-aged individuals and may not be associated with diabetes mellitus or immunocompromised states(2,4,6,7). The clinical presentation is usually with insidious and nonspecific symptoms, such as headache, atypical facial pain, sudden onset of sensory or motor symptoms of cranial neuropathy, or even sinonasal symptoms in a subset of patients(4,6,7). It is observed that the cranial nerve VI and the lower cranial nerves (IX and X) are more often involved than the facial nerve(7). *Staphylococcus aureus* is reported to be the causative pathogen more frequently than *Pseudomonas aeruginosa* in this type. The primary site of disease is usually the central skull base, particularly the sphenoid bone and clivus, with or without extension to the paranasal sinuses, deep fascial spaces, or the oral cavity (2,3)

Symptoms are often subtle, leading to delayed diagnosis. Early recognition becomes of utmost importance owing to the silent spread of the disease, resulting in risk of intracranial complications.

### Clinical Presentation

- Persistent otalgia or headache: Severe, persistent otalgia or deep-seated headache is common and may be disproportionate to the local clinical findings
- Otorrhea (in typical SBO): Persistent purulent otorrhea is characteristic of typical SBO, usually secondary to malignant otitis externa, but may be absent in atypical SBO
- Cranial nerve palsies (VII, IX, X, XI, XII): Cranial neuropathies may occur with disease extension, with CN VII commonly involved in typical SBO and CN VI and lower cranial nerves (IX–XII) more frequently affected in atypical SBO.
- Elevated inflammatory markers (2, 4): ESR and CRP are typically elevated, although leukocytosis may be absent, particularly in chronic or atypical disease.

### Imaging Modalities

Imaging plays a key role in diagnosis of SBO and its complications, with CT as well as MRI having significant value. Radiological imaging is not only essential for early diagnosis but is equally important for:

- Mapping disease extent
- Guiding biopsy
- Monitoring therapeutic response

### Computed Tomography (CT)

CT helps in evaluating the erosion and destruction of the skull base more effectively.

### The Key findings enabling accurate diagnosis on CT acquisition at bone window settings are-

- Bone erosion
- Soft tissue thickening in the external auditory canal and middle ear cavity.
- Mastoid air cell opacification

### Challenge:

Early disease shows no changes in the bones and therefore an incomplete diagnosis of otitis media alone, without identifying early SBO, may be made, further delaying the appropriate treatment.

### Magnetic Resonance Imaging

MRI is the modality of choice for early detection of both intracranial complications as well as inflammatory changes in the involved bones of the skull base.It is a crucial addition to CT, providing enhanced assessment of soft-tissue involvement, marrow infiltration, and meningeal as well as parenchymal spread. A thorough protocol involving T1-weighted, T2-weighted, STIR, diffusion-weighted imaging (DWI), and post-contrast fat-suppressed T1-weighted

sequences is essential for proper evaluation of the extent and complications.

**MR Imaging Findings**

During the initial phases of otogenic SBO, MRI may show altered signal intensity and enhancement in the external auditory canal, along with significant edema and inflammatory changes in the nearby auricular soft tissues. On T1-weighted images, the soft-tissue inflammation is seen as hypointensity compared to muscle, with the loss of normal retromandibular, parapharyngeal, and preclival fat. On T2-weighted images, these alterations present as hyperintense signal and indicate a mix of inflammation, edema, and phlegmon formation. Inflammatory soft tissue shows heterogeneous enhancement with gadolinium contrast administration. In SBO of fungal etiology, especially mucormycosis, the enhancement is heterogeneous due to a mix of enhancing inflammatory and non-enhancing devitalized (necrosed)tissue(3,9). Osseous involvement is marked by the substitution of normal fatty marrow, leading to T1 hypointensity and matching hyperintensity on STIR sequences, together with non-uniform post-contrast enhancement. Initial marrow alterations may be discreet and necessitate careful assessment using fat-suppressed sequences, particularly in areas like the mastoid, petrous apex, and occipital bone. As infection progresses, necrosis of the marrow can happen, possibly resulting in abscess development, which manifests as a T2 hyperintense, well-marginated intraosseous collection with peripheral rim enhancement. Restricted diffusion supports the presence of abscess formation.

Unchecked, the infection disseminates anteromedially, engaging deeper structures adjacent to the skull base. Disease advancement can lead to greater bone involvement, often reaching the clivus and occasionally bilateral changes. (5,7,9)

In Central SBO, MRI findings are broadly similar; however, the epicenter of disease is typically located in the central skull base, particularly the sphenoid bone. Marrow signal abnormalities most commonly involve the clivus, with extension to the sphenoid wing and petrous apices. Soft-tissue inflammatory involvement in the preclival region may appear more symmetric, producing diffuse nasopharyngeal fullness. Significant extension of infection to the nasopharyngeal region might be seen and resemble an infiltrative tumor (5,7). Diffusion-weighted imaging can be particularly useful in this situation to rule out primary neoplastic nasopharyngeal pathology. Apparent diffusion coefficient (ADC) values aid in differentiating infection from neoplasm, with higher ADC values seen in SBO compared to ADC values in nasopharyngeal carcinoma or lymphoma. Moreover, nasopharyngeal inflammatory phlegmon typically shows peripheral rim enhancement,where as nasopha ryngeal carcinoma tends to enhance more uniformly.

**Imaging findings of associated complications of skull base osteomyelitis**

- Cranial neuropathy: Affected cranial nerves appear thickened with T2-weighted hyperintensity and abnormal post-contrast enhancement, particularly at the skull base foramina and root exit zones. Associated inflammatory soft tissue often obliterates the normal fat planes around the neural foramina and extends along the expected course of the nerves. High-resolution heavily T2-weighted sequences such as 3D-CISS/FIESTA and post-contrast fat-suppressed T1-weighted images are particularly useful for detecting subtle neural involvement and perineural abnormalities.(4,6,9,11)
- Intracranial abscess: Abscesses typically appear as ring-enhancing lesions with a centrally non-enhancing cavity that demonstrates marked diffusion restriction on diffusion-weighted imaging (DWI) due to viscous purulent contents. Extensive surrounding vasogenic edema is usually evident on T2-weighted and FLAIR images and may produce significant mass effect.(4,6,9,11)
- Meningitis: Meningeal involvement occurs due to direct extension of infection through the skull base foramina or adjacent osteomyelitic bone. Contrast-enhanced MRI demonstrates smooth or nodular leptomeningeal and/or pachymeningeal enhancement, often involving the posterior fossa and skull base cisterns. Associated FLAIR hyperintensity within the subarachnoid spaces may reflect inflammatory exudates(4,6,9,11).
- Venous sinus thrombosis: Dural venous sinuses adjacent to the skull base, particularly the sigmoid, transverse, and cavernous sinuses, may show thrombus formation. MRI demonstrates loss of the normal flow void, abnormal intrinsic signal intensity within the affected sinus depending on the age of the thrombus, and absent or irregular enhancement following contrast administration. (4,6,9,11)
- Carotid artery involvement: Inflammatory extension to the internal carotid artery is an uncommon but potentially life-threatening complication of skull base osteomyelitis. MRI with contrast and MR angiography may demonstrate circumferential vessel wall thickening and enhancement, reflecting infectious arteritis. Progressive disease can result in luminal narrowing, thrombosis, pseudoaneurysm formation, or complete arterial occlusion, increasing the risk of ischemic stroke ((4,6,9,11).

**Other imaging modalities that can be relied upon for diagnosis of SBO are :**

**i)Nuclear Medicine Imaging with Technetium-99m Bone Scan and Gallium-67 Scan**

**Technetium-99m bone scan**

- Highly sensitive but nonspecific because increased radiotracer uptake reflects osteoblastic activity and increased bone turnover, which can occur with infection as well as malignancy, trauma, fracture, or other inflammatory conditions.
- Useful for early detection of: skull-base osteomyelitis, particularly before definite structural bone destruction becomes apparent on CT. (10,11)

**Gallium-67 scan**

- More specific for active infection/inflammation than a bone scan because Gallium accumulates at sites of increased inflammatory activity and leukocyte-mediated infection.
- Useful for detecting active skull-base infection, assessing its extent, and monitoring treatment response
- Gallium scans are more Helpful for monitoring treatment response.(10,11)

**Treatment Overview**

- Long-term targeted antibiotic therapy
- Antifungal therapy in selected cases
- Surgical debridement (limited role) (4,6,8)

Imaging changes often lag behind clinical improvement, and this is a limitation for MRI as well as CT studies. This is because osseous abnormalities may persist for several weeks to months even after there is a reduction in inflammatory soft-tissue changes. PET-CT is particularly valuable in follow-up imaging. (9,10,11)

**Table 1: Features assisting in differentiating SBO from other conditions involving skull base**

| Condition | Key Distinguishing Features |
|---|---|
| Nasopharyngeal carcinoma | Soft tissue mass with associated nodal involvement |
| Cholesteatoma | Non-enhancing middle ear mass showing restricted diffusion |
| Sarcoidosis | Associated Systemic findings |
| Metastases | Multifocal lesions, instead of contiguous involvement |

## CONCLUSION

Recognition of the characteristic patterns of osseous, marrow and soft-tissue involvement on CT and MRI, together with careful assessment for cranial-nerve, vascular and intracranial complications, is essential for early diagnosis and accurate delineation of disease extent. Familiarity with the distinction between typical and atypical/central forms can help avoid diagnostic delay and inappropriate treatment.

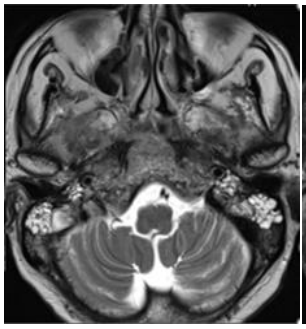

**Fig 1a**

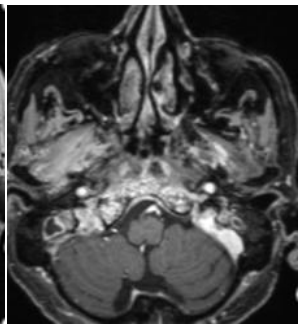

**Fig 1b**

Central(atypical) skull base osteomyelitis.Images showing T2 hyperintense signals in the petrous and mastoid parts of bilateral temporal bones, basisphenoid, basi-occiput, and clivus (Fig 1a) and heterogeneous post-contrast enhancement (Fig 1b).

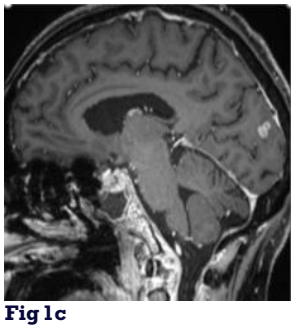

**Fig 1c**

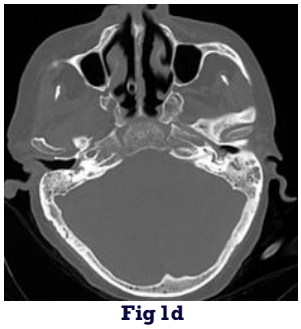

**Fig 1d**

CT and MR Images showing enhancing soft tissue thickening involving the prevertebral space extending from the basisphenoid to the lower border of C1. Note the conglomerated ring enhancing lesions in the occipital lobe(Fig 1c). Non contrast CT head shows diffuse rarefaction of the involved bones without evidence of any cortical breach (Fig 1d).

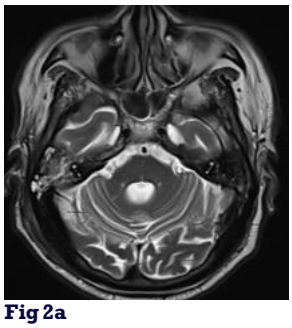

**Fig 2a**

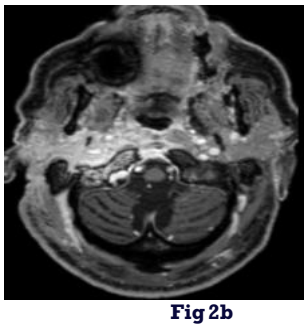

**Fig 2b**

Otogenic/Typical skull base osteomyelitis. Images showing T2 hyperintense soft tissue in the right middle ear cavity extending into the mastoid antrum with no intracranial extension (Fig 2b). On post-contrast T1-weighted images, there is enhancing soft tissue in right tympanic cavity with extension into prevertebral, pharyngeal mucosal, posterior cervical space and ipsilateral parapharyngeal space (Fig 2b).

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