*Concorso Pubblico, per titoli ed esami, per n. 1 posto di Dirigente Medico – Area della Medicina Diagnostica e dei Servizi - Disciplina di Neuroradiologia - da assegnare alla UOC Radiologia Diagnostica per Immagini - Neuroradiologia Intervenzionale*

**PROVA ORALE: DOMANDE INERENTI LA DISCIPLINA A CONCORSO**

1. RM intra operatoria: MRgFUS
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7. FLOW-DIVERTER INTRASACCULARI
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16. NEUROINTERVENTISTICA NELLA GESTIONE MAV SPINALI

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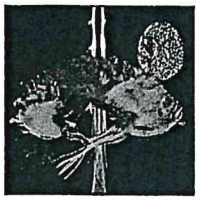

# Head and Neck: Skull Base Imaging

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There are a myriad of head and neck pathologies that extend from the extracranial to the intracranial compartment, traversing the skull base, and knowledge of the imaging appearance of this pathology is critical to practicing neurosurgeons. This article reviews some of the important inflammatory or acquired head and neck pathology along the skull base, neoplastic skull base lesions, and the intracranial extension of head and neck malignancy. Focus will be on the relevant anatomy, appropriate imaging protocols to evaluate these processes, as well as the differentiating imaging findings on computed tomography and magnetic resonance imaging.

**KEY WORDS:** Cerebrospinal fluid, Contrast-enhanced computed tomography, Gadolinium contrast, Idiopathic intracranial hypertension, Magnetic resonance imaging, Perineural tumor, Skull base

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The close proximity of the extracranial head and neck (HN) to the intracranial compartment makes knowledge of HN anatomy and disease processes critical for the neurosurgeon. In some locations, for example, the cribriform plate, only a millimeter of bone or less separates the nasal cavity from the extradural space. In this review, we present important HN lesions, essential anatomy, and stress imaging findings that help differentiate benign from malignant or aggressive disease processes.

For virtually all complicated sinus or skull base lesions, both computed tomography (CT) and magnetic resonance imaging (MRI) are indicated. A noncontrast sinus or skull base CT, which covers the mastoids, temporal bone, and entire skull base, is recommended, and intravenous contrast is not usually necessary as the MRI will provide soft tissue detail. For lesions that are completely intraosseous, the mass may only be appreciated on MRI (Figure 1). CT is superior to MRI for subtle cortical bone changes, but marrow processes are best characterized with non-Gadolinium (Gd) contrast-enhanced T1 MRI. If there is a contraindication to MRI, the sinus/skull base CT should be done with iodinated contrast. In this era of limiting ionizing radiation, there is no reason to do CT both without and with contrast, and the contrast-enhanced CT (CECT) will be all that is necessary.

Adequate history prior to MRI is essential to plan the correct study. In our practice, we have over a dozen different MR protocols for HN lesions that may have an osseous or intracranial component. For skull base lesions, MR technique should always include T1-W images without contrast or fat saturation (FS) in order to show marrow replacement by edema or tumor. Referring physicians, including neurosurgeons, frequently emphasize perceived need for Gd on all brain MRI, but in fact Gd may obscure lesions if the correct technique is not done. Communication between the neuroradiologist and neurosurgeon has a positive impact on patient care.

**ABBREVIATIONS:** **CECT,** contrast-enhanced CT; **CSF,** cerebrospinal fluid; **CT,** computed tomography; **FS,** fat saturation; **Gd,** gadolinium; **HN,** head and neck; **ICA,** internal carotid artery; **IIH,** idiopathic intracranial hypertension; **MRI,** magnetic resonance imaging; **PNT,** perineural tumor; **SI,** signal intensity

## SKULL BASE ANATOMY

Modern imaging has the unique advantage of being able to demonstrate the complex skull base anatomy in multiple planes. CT demonstrates the bony anatomy best, while MRI has superior soft tissue resolution.

The skull base is made of the paired frontal and temporal bones, as well as the ethmoid and occipital bones, and these bones form the floors of the anterior, middle, and posterior cranial fossa. The thin and complex anterior cranial fossa separates the frontal and ethmoid sinuses and orbits from the inferior frontal lobes and

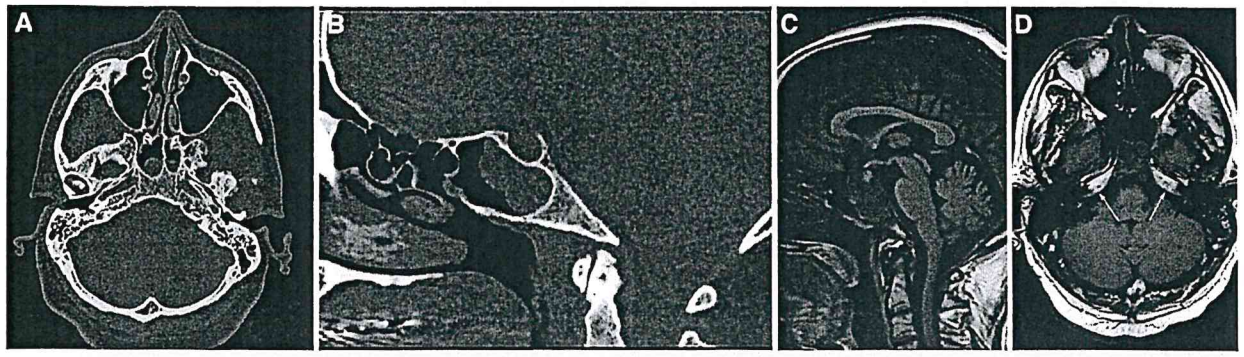


**FIGURE 1.** *Seventy-year-old male with a history of osseous sarcoma and multiple bone metastases, now with new headache.* **A,** *Axial bone algorithm CT shows normal central skull base. Soft tissue in both sphenoid chambers appears benign with no bone erosion.* **B,** *Sagittal reformation from axial data set shows normal clivus. Floor of sella turcica appears intact. Sphenoid sinus soft tissue has no malignant characteristics.* **C,** *MRI was obtained same day as CT. On this T1-weighted sagittal image, there is complete replacement of the superior two-third of the clivus (arrow), erosion of the sella floor (arrowhead), and intracranial extradural extra-osseous tumor in the retroclival location (short arrow). These findings were not seen on the bone window CT.* **D,** *Axial T1-weighted image shows normal SI in petrous bones (arrows) but replacement of marrow in clivus.*

olfactory bulbs, and is formed by the frontal and ethmoid bones, with the anterior border being the posterior table of the frontal sinus, the lateral border the orbital roof (or orbital plate of the frontal bone), and the medial border formed by the thin cribriform plates, lateral lamella, and ethmoid roof (or fovea ethmoidalis). The lesser wing of the sphenoid bone, with the clinoid process, tuberculum sella, and planum sphenoidale, form the posterior border, dividing the anterior and central skull base.

The central skull base contains the sella turcica, skull base foramina and cranial nerves II through VI, and the internal carotid artery (ICA). The skull base divides the intracranial structures from not only the sphenoid sinuses, but also the extracranial soft tissues deep to the skull base inferiorly, including the masticator, parotid, parapharyngeal, and pharyngeal mucosal spaces. The basi sphenoid portion of the clivus, the dorsum sella, and the superior petrous ridge of the temporal bone demarcate the junction of the central and posterior skull base. The posterior skull base is made up by the posterior temporal and occipital bones, and contains the foramina for cranial nerves VII to XII, the jugular vein and ICA, and the largest foramen of the skull base, the foramen magnum.

## INFLAMMATORY/ACQUIRED SKULL BASE PATHOLOGY

### Mucoceles

When a pneumatized air cell is obstructed, mucous accumulates and the cell walls gradually become thinned, deossified, and ultimately resorbed. The overall volume of the cell, whether it is the frontal, ethmoid, or sphenoid sinus, or at the petrous apex, increases and the sinus expands. An expanded, completely opacified cell is a *mucocele*. The obstruction to the sinus ostium may be benign, such as fibrous dysplasia, or malignant, as in a sinonasal squamous cell carcinoma.[1] Mucoceles, when infected, are termed pyoceles. The goals of CT or MRI in this setting include careful assessment of the sinus ostium to determine the cause of the obstruction, and assessment of the sinus or cell walls to detect intraorbital or intracranial extension.

CT may show marked bony thinning, and in fact the osseous wall may appear dehiscent (Figure 2). The content of the mucocele or the thinned wall usually has a smooth interface with the dura or periorbital fat. This characteristic lack of a feathery interface implies that the process may be intracranial or intraorbital, but is extradural and extraconal, without dural or intraconal extension.

Signal intensity (SI) within the mucocele on MRI is variable depending on how long the sinus or cell has been obstructed, and the relative water vs protein concentration of the contents.[2] Mucoceles that are relatively new are isointense on all sequences to cerebrospinal fluid (CSF). Mucoceles with decreasing water concentration vary from high signal on T1 and hyperintense signal on T2 (Figure 3) to high signal on T1-weighted images and markedly decreased SI on T2 images. In fact, the intensity on T2 can be so low that the cell appears air filled and not opacified. There is usually thin smooth enhancement of the mucoperiosteum lining the mucocele.

### Intracranial Complications of Sinusitis, Mastoiditis, and Facial Infections

Local complications of bacterial sinusitis, mastoiditis, and less commonly severe facial infections include osteomyelitis, epidural abscess, subdural empyema, meningitis, ventriculitis, and cerebritis. Ultimately, a discrete intra-axial brain abscess can develop, which may be in close proximity to the infected sinus or mastoid or even remote, as the infection can extend hematogenously. The initial imaging should be a skull base and

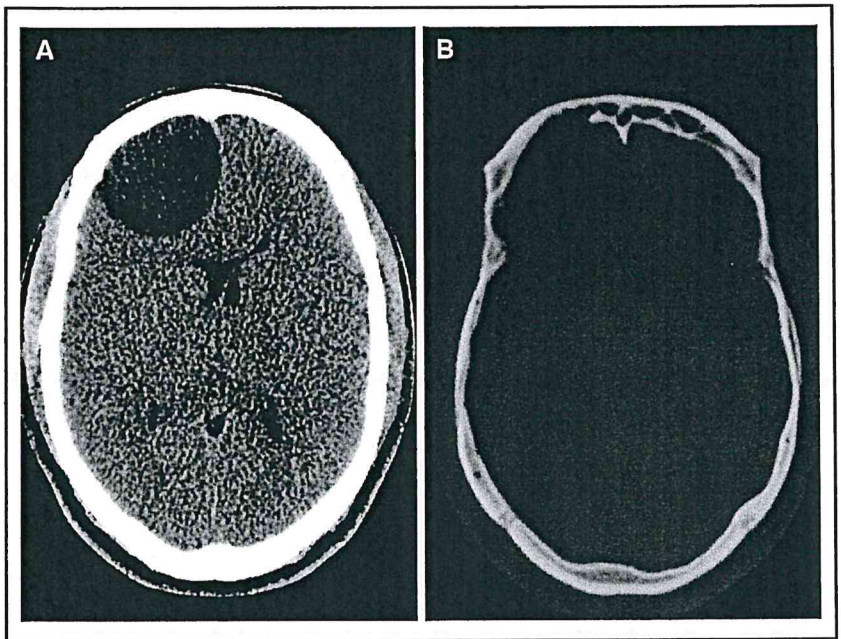


**FIGURE 2.** *Adult male with chronic sinusitis and headaches. Right frontal sinus mucocele.* **A,** *Axial noncontrast CT shows a well-circumscribed extra-axial right frontal mass that is homogeneously mucoid density.* **B,** *Bone algorithm axial image shows the walls of the mucocele (arrows) are thin, deossified, and probably completely dehiscent. Note opacified left frontal sinus, without expansion or dehiscence of the walls.*

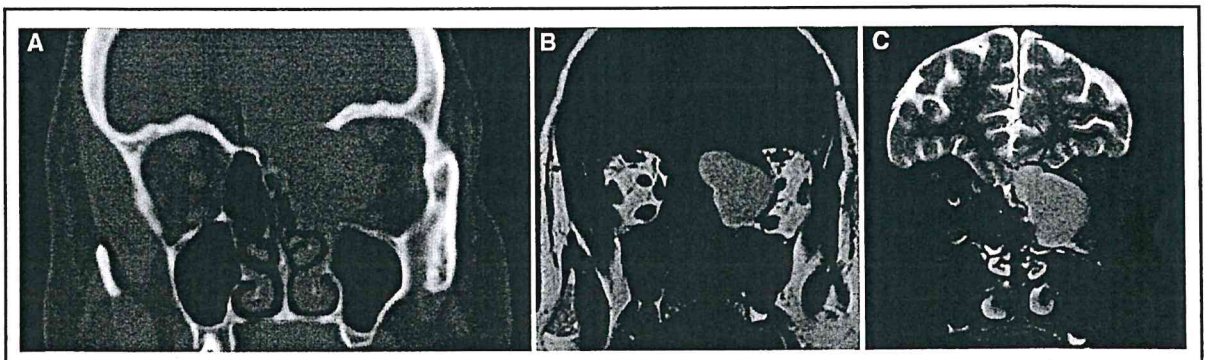


**FIGURE 3.** *Fifty-year-old male with left proptosis and headache. Left frontoethmoid mucocele.* **A,** *Coronal bone algorithm CT shows complete destruction of the left ethmoid roof, and lamina papyrecea, by a soft tissue mass. On the right, note the normal cribriform plate (small arrow) and ethmoid roof (longer arrow).* **B,** *Coronal T1 MR image shows the mass is well circumscribed and homogeneously high SI. There is a smooth interface with the brain suggesting the dura is intact. Note lateral displacement of the left medial rectus muscle (long arrow) and superior oblique muscle (arrowhead). The mucocele contents are proteinaceous, and therefore high SI on T1 MR without contrast.* **C,** *Coronal T2 MR with FS shows intact dura laterally as line between mucocele and brain (arrow) is present. More medially the dura is thinned and possibly dehiscent, as the black line is not preserved (small arrow). There is still a smooth interface with the gyrus rectus, and no vasogenic frontal lobe edema.*

sinus or temporal bone CT, and if an MR examination is anticipated, a CECT is not necessary.

Imaging findings are both extra and intra-axial, and include meningeal enhancement, cortical edema from cerebritis, and brain abscess (Figure 4). MRI with Gd shows thickened and enhancing leptomeninges, and ependymal enhancement when there is ventriculitis. The classic appearance of a brain abscess is a ring-enhancing mass, with a rim of low SI on T2-weighted images.[3] Diffusion-weighted imaging is particularly helpful, as empyemas and pyogenic brain abscesses have restricted diffusion

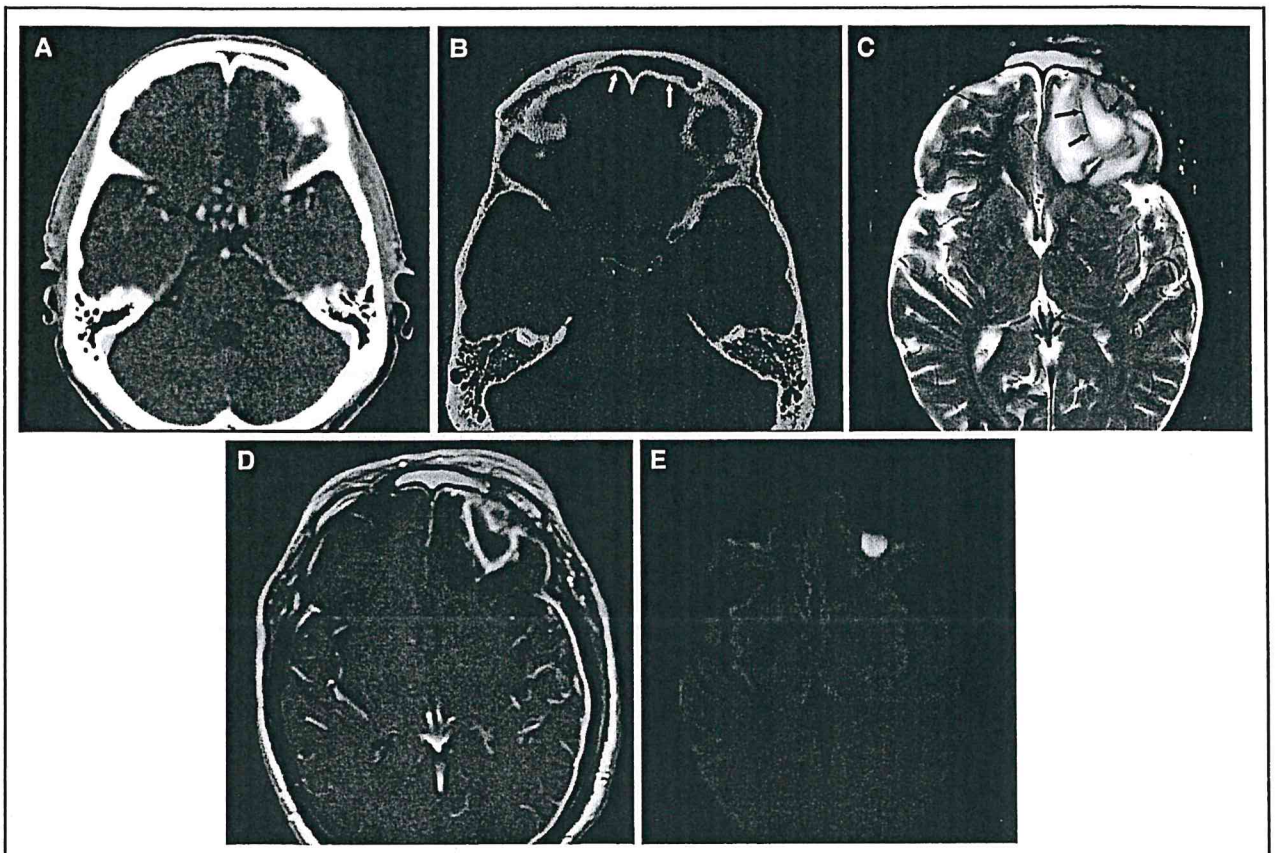


**FIGURE 4.** *Adult patient with several weeks of sinusitis, now with severe headache, seizures, and altered mental status. Bacterial frontal sinusitis with intracranial abscess.* **A**, *Axial CECT shows a peripherally enhancing left frontal intra-axial mass (arrows) with surrounding vasogenic edema (short arrows). Note opacified frontal sinus.* **B**, *Bone algorithm CT confirms opacified frontal sinus, but the posterior wall is intact (arrows). The infection can extend from the sinus intracranially presumably through venous channels, without destroying the posterior wall.* **C**, *Axial T2-W FS image shows boggy edema in the left forehead and the completely opacified frontal sinus, filled with hyperintense debris and pus. The abscess has a low SI rim (arrows), a characteristic appearance for brain abscess on T2 images.* **D**, *Axial T1-W Gd-enhanced image shows the peripheral enhancement of the abscess capsule, the central debris and pus, and surrounding vasogenic edema. There is diffuse thin nonnodular dural enhancement (arrows) of the entire left cerebral hemisphere, likely a combination of meningeal edema and possibly subdural pus.* **E**, *On this diffusion image notice the markedly restricted diffusion in the abscess (arrows), a characteristic of brain abscess. There is artifact at the posterior frontal sinus wall and the frontal lobe, a limitation of diffusion imaging at any bone–brain interface.*

(Figure 4E). Advanced imaging techniques, such as perfusion imaging and diffusion tensor imaging,[4] have been described but are usually not necessary as the clinical presentation is generally unequivocal.

Venous thrombosis, either cortical vein or major sinus, is a potential serious complication of bacterial sinusitis or mastoiditis. Expansion of the venous sinus with loss of flow and a filling defect are common imaging findings on both CECT and MRI.[5] Cavernous sinus thrombosis is suspected when the lateral dural wall is displaced or convex laterally, the sinus contents are heterogeneous from filling defects, and there is often narrowing or spasm of the ICA (Figure 5)[6-8]. Because sinus thrombosis is often catastrophic, with cerebral edema, hemorrhage, and infarctions as potential complications, early suspicion and imaging is stressed.[9] Treatment, in addition to appropriate antibiotics, includes surgical drainage of the involved sinus or mastoid complex. Use of thrombolytics and even mechanical clot removal are controversial. Because venous thrombosis and especially cavernous sinus thrombosis are rare, prospective comparisons of treatment are not available.

Skull base osteomyelitis is suspected in a diabetic patient with headache and poorly controlled glucose. The process may begin in the external auditory canal, and is often a *Pseudomonas* species. A "routine" brain MRI may be normal early in the disease. The MRI technique in this setting is critical, as noncontrast T1 images in all planes without FS are essential, and Gd images without