

Review of Skull Base Metastases

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Learning Objectives:

1. Review the epidemiology of skull base metastases
2. Revise the anatomy of the skull base and how it relates to clinical presentation

3. Understand the imaging features of skull base metastases on CT and MRI
 Background: Compared to other sites of skeletal metastases, skull base metastases are relatively rare with varying incidence reported in the literature ranging from 0.13-5.0% in an autopsy series [1] to up to 23% in certain cancer groups [2]. Haematogenous spread is most common and the most frequently implicated cancers are breast, prostate and lung carcinomas and lymphoma [1]. Presentation ranges from asymptomatic with metastases detected on screening CT or MRI, to symptomatic presentations with pain or cranial neuropathies which can form part of a spectrum of specific skull base clinical syndromes depending on the cranial nerve/s affected [3]. Most often, treatment is with targeted external-beam radiation therapy, either as a stand-alone modality or in combination with surgery or chemotherapy [4].

Imaging Findings and Procedure Details: The skull base forms the floor of the cranial cavity and separates the brain from the extracranial structures of the head and neck. Skull base anatomy is complex namely due to the multiple canals and foramina which allow for the passage of arteries, veins and cranial nerves between the two compartments. It is involvement of these canals and foramina where the cranial nerves traverse which frequently accounts for patient's symptoms. CN VI palsy has been described as the most frequently observed neuropathy [5], however other clinical syndromes such as Gasserian ganglion syndrome (CN V), Jugular foramen syndrome (CN IX, X, XI) and occipital condyle syndrome (CN XII) have also been described (3). Patients typically undergo both CT and MRI either as a screening examination for cranial metastases or to investigate clinical symptoms. Skull base metastases are most frequently found in the clivus, petrous apex and sphenoid bone due to the higher marrow content of these areas. On CT the metastases are often lytic, but may also have a sclerotic appearance. They are typically T1 hypointense due to replacement of the fatty marrow and can have variable T2 signal hyperintensity but usually demonstrate homogenous enhancement. Fat-suppressed T2 and T1 post gadolinium best demonstrates the lesions. Diffusion imaging has also been shown to be a useful adjunct in detecting skull base disease (2).

Five case examples will be provided to highlight the imaging features of metastases at various locations at the skull base.

Conclusion: Knowledge of normal skull base anatomy is essential in evaluating the imaging of asymptomatic patients with primary malignancies, as well as an understanding of cranial neuropathies in those that are symptomatic.

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Short case series of different causes of a cerebrospinal fluid rhinorrhoea

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Purpose/Objectives: Cerebrospinal fluid (CSF) rhinorrhoea refers to a CSF leakage extracranially into the paranasal sinuses, then into the nasal cavity, and exits via the anterior nares.

CSF rhinorrhoea is often seen whenever there is an osseous or dural defect of the skull base, mostly caused by head trauma or a post-operative complication of skull base surgery, or due to congenital defects.

Methods and materials: We present two cases of CSF rhinorrhoea caused by diverse etiology and treated with different surgical approaches.

Result: A 72-year-old woman with a 6-month history of left-sided intermittent nasal discharge and mild headache. This was identified to be CSF caused by intrasphenoidal meningoencephalocele due to the persistence of the lateral craniopharyngeal canal (Stenberg's canal). The brain MRI identified a herniated temporal lobe through a bony defect that communicates the middle cranial fossa with the lateral recess of the sphenoidal sinus. The patient underwent a functional endoscopic sinus surgery – with the resection of meningoencephalocele and closure of the defect with fascia lata lever. There were no complications related to the surgery and no recurrence of CSF leakage in 6 months.

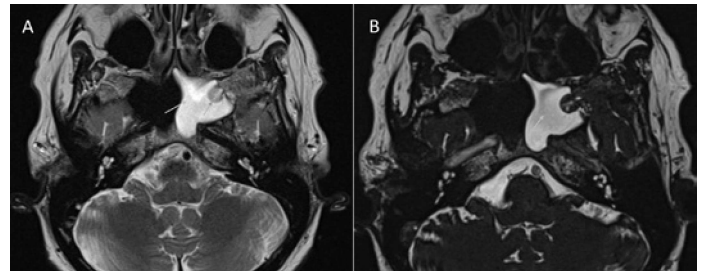


Image A: T2 weighted ax. and B:T2 3D CISS. The orange arrow marked left sphenoidal cavity with fluid, which is most likely CSF. An intrasphenoidal meningoencephalocele marked with a red arrow and the persistence of the lateral craniopharyngeal canal (Stenberg's canal) is marked with a blue arrow. [1]

A 41-year-old male with multiple skull and facial fractures (Le Fort III), a traumatic subarachnoid hemorrhage in the left frontal lobe, and traumatic brain injury resulting from altercations. He was primarily treated with subsequent facial bone osteosynthesis using mini and microplates. A month after this episode, the patient was re-admitted to another hospital due to progressive headache, subfebrile temperature, and persistent watery nasal discharge. Brain CT was repeated demonstrating diffuse pneumocephalus and cerebrospinal fluid circulation abnormalities with signs of transtentorial herniation and brain edema. Progressive widening of cerebral ventricles was noted, as well as periventricular edema, most likely, acute communicating hydrocephalus due to meningitis. The patient underwent a right ventriculostomy with an antimicrobial drain, followed by a ventriculoperitoneal shunting procedure. Shortly after, the CSF rhinorrhoea resolved.

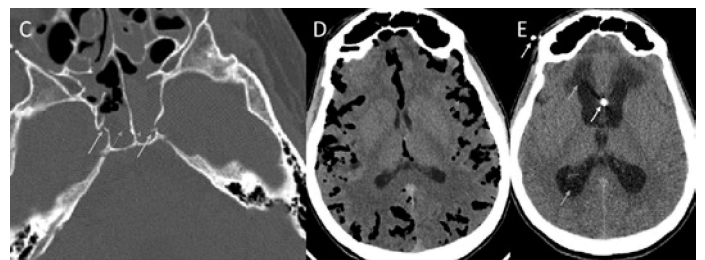


Image C. CT bone window, the yellow and red arrows shows the fractures of the walls of the sphenoid cavity with blood and CSF inside. D,E image. The blue arrow marks extensive pneumocephalus and the gray arrow at follow up images shows the developing communicating hydrocephalus which was treated with ventriculoperitoneal shunting- white arrows [2]

Conclusion: CSF rhinorrhoea is a relatively rare condition occurring secondary to different etiology, however, it can be clinically noticeable or insidious. A comprehensive diagnostic assessment of individuals clinically suspected of having CSF rhinorrhoea is critical, along with an understanding of CSF components and imaging abnormalities.

References:

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