Learning objectives

Skull base tumors are complex clinical and radiological entities. They arise from the cranial base or reach it, either from an intracranial or extracranial origin. Sometimes nasosinusual or vascular tumors can reach intracranial cavity. Otherwise, some intracranial tumors can pass through skull base to reach extracraneal spaces. Radiologists are challenged determine more accurately the extend and origin and the extension of tumoral processes in this region.

The purpose of this work is:

- Review the anatomic relationships of head and neck tumors and skull base.

- Differential considerations and hallmark imaging features of different lesions.

- Highlight radiographic features critical for surgical planning and intraoperative guidance.

Background

1.- Introduction:

Skull Base is one of the most challenging anatomic regions to access surgically. Neuroradiologists play a key role in helping to identify important anatomic landmarks that are critical for surgical planning and intraoperative guidance. Imaging of the skull base requires a detailed knowledge of the skull base anatomy and it must be carefully scrutinized for distortions of anatomy. The relation with the cranial vessel and nerves is very important as many of the patients with skull base tumors will present with cranial nerve deficits as the first sign. So, the neurovascular structures entering and exiting the skull base through the basal foramina must be take into account by surgeons.

The imaging approach as well as the type of lesions that will be found will be different for the anterior, middle and posterior skull base.

CT and MRI are complementary in providing an appropriate differential diagnosis and in aiding surgical planning. CT better demonstrates bone destruction and intralesional calcifications. MRI better desmonstrate soft tissues and is superior to CT to evaluate
the relationship of skull base pathologies. MRI is more accurate in depicting the exact margins of intracranial tumor extension because of its multiplanar display and superior tissue contrast.

Management outcomes for skull base tumors are maximized when their treatment is approached in a multidisciplinary fashion, utilizing the knowledge base of varied medical, surgical, and radiotherapeutic specialists.

2.- Anatomy:

The new multi-detector row CT images bring us a high resolution anatomy of the skull, allowing a presurgical quality assessment of the skull base. (Fig. 1 on page 21) The skull base can be subdivided into 3 regions: the anterior (Fig. 2 on page 22, Fig. 3 on page 22 ), middle (Fig. 4 on page 22, Fig. 5 on page 23, Fig. 8 on page 24 ), and posterior cranial fosses. (Fig. 6 on page 23, Fig. 8 on page 24, Fig. 8 on page 24 )

3.- Radiological findings:

Skull base tumors may originate from the neurovascular structures of the base of the brain or the basal meninges (e.g., meningioma, pituitary adenoma, schwannoma, paraganglioma), from the cranial base itself (e.g., chordoma, chondrosarcoma), or from the subcranial structures of the head and neck (e.g., esthesioneuroblastoma, sinonasal carcinomas).

3.1:ANTERIOR SKULL BASE:

3.1.1: Esthesioneuroblastoma (Fig. 9 on page 25):
Esthesioneuroblastomas is an uncommon malignancy of the olfactory epithelium usually arises from the basal layer of olfactory mucosa in the region of the olfactory plate or superior nasal cavity.

As the symptoms and signs are non-specific, patients tend to present with advanced disease, with intracranial extension occurring about 25% of cases. Well differentiated tumors rarely metastasize, however given their propensity to recur locally, these lesions are considered malignant.

The new stage criteria are: T1 tumor that affects the nasal fossae and/or paranasal sinuses (excluding the sphenoid); T2 affects the nasal fossae and paranasal sinuses (including the sphenoid) with infiltration of the lamina cribosa; T3 extends to the orbit
or anterior cranial fossa without invasion of the dura mater; and T4 includes cerebral invasion.

**CT:**

1. Expansive homogeneous soft tissue mass occupying the ethmoid air cells that is invading the cribiform plate and breaking through to the anterior cranial fossa.
2. A relatively uniform enhancement
3. Occasional focal calcification may be present.
4. There is usually nasal bony remodeling and erosion at cribiform plate, rarely hyperostosis.

**MRI:**

1. Variable T1 and T2 weighted signal with some heterogeneity
2. Minimal to moderate gadolinium enhancement.
3. Large tumoral lesions may present solid and cystic components.
4. Enhanced images, particularly in the coronal plane, were very helpful in identifying intracranial extension.
5. The presence of small cysts at the tumor-brain interface are typical.
6. Although esthesioneuroblastomas are initially unilateral within the upper nasal cavity, (Fig. 9 on page 25) they subsequently expand the nasal cavity and ethmoid air cells bilaterally, before penetrating the orbit and cribiform plate. (Fig. 10 on page 26).

The **treatment of choice** is wide surgical excision which are known to recur insidiously even years later. Nowadays, expanded endonasal approach (EEA) resection of the tumor is the rule. In selected cases an open approach (bifrontal craniotomy) could be indicated. Stereotactic radiosurgery can result in increased tumor control and should be considered as a treatment option.

**3.1.2: Adenoid cystic carcinoma** (Fig. 11 on page 27, Fig. 12 on page 28):

Is an uncommon epithelial malignant tumor that accounts for less than 1% of all head and neck malignancies and about 10-22% of all salivary gland tumors. This neoplasm is characterized by its prolonged clinical course, infrequent lymphatic invasion, multiple local recurrence and delayed distant metastasis. The sinonasal location accounts for less than 4% of all head and neck adenoid cystic carcinomas. This tumor has a characteristic tendency to **spread along neurovascular bundles.** Parotid origin tumors can reach middle fossa through oval foramina by trigeminal perineural spread V3. (Fig. 11 on page 27). Also it may be extension though the middle cranial fossa via the superior orbital fissure (Fig. 12 on page 28)
CT:
1. Soft tissue attenuation mass invading the central skull base, sphenoid sinus, sinuous cavernous, with extension to the intracranial middle fossa.
2. Contrast enhancement may show areas of central necrosis.
3. Tumoral calcifications are rare.
4. Widening of skull base foramina (oval, rotundum...)
5. Aggressive bone destruction of the middle skull base is the rule in advanced tumors

MRI:
1. Relatively homogeneous and of intermediate signal on T1 and T2 weighted.
2. Moderate enhancement mass after contrast administration
3. Perineural enhancement along the neurovascular bundles
4. Focal signal inhomogeneity in areas of necrosis or hemorrhage.

The treatment of choice is a complete surgical resection but in large masses it can not be performed due to extensive involvement of the skull base, sinus cavernous or internal carotid artery and/or intracranial tumor extension. The use of high-dose conformal proton beam radiotherapy achieves a high rate of local control.

3.1.3. Rhabdomiosarcoma (Fig. 13 on page 30):

Is a mesenchymal malignant tumor that occurs more often in children.

40% arise in head and neck. The orbit, nasopharynx and nasosinusal sinusses are affected most often. This tumor can reach intracranial cavity (41%) by direct invasion from the orbit or sinus. Metastases are frequent (50%). Tumors of the orbit have a better prognosis; parameningeal (including nasal cavity tumors), nasopharyngeal, paranasal sinus, middle ear, mastoid, infratemporal fossa, and pterygopalatine fossa tumors have a worse prognosis due to the possibility of subarachnoid spread.

CT:
1. Usually show an aggressive infiltrative soft tissue mass
2. Variable enhancing soft-tissue mass
3. Areas of bone destruction are frequent

MRI:
1. Large infiltrating enhancing soft tissue mass lesion with bone destruction
2. The infra-cranial epidural invasion across the frontal base is best seen on coronal MRI.
3. Signal intensity is similar to the muscle on T1-weighted and hyperintense on T2-weighted
4. Diffuse often market and heterogeneous enhancement (Fig. 13 on page 30).

The **treatment of choice** is combined radio-chemotherapy. Surgery should be performed as the first treatment if it causes no functional or esthetic harm, followed by systemic chemotherapy.

### 3.1.4 Olfactory groove Meningioma (Fig. 14 on page 30):

Common benign intracranial tumors that arise from arachnoid cells of the duramater. Olfactory groove meningioma arises from the midline of the anterior fossa between the crista galli and the tuberculum sellae. They are usually bilateral but may be asymmetric and get a large size before causing symptoms. The most common presenting symptom is a subtle change in mental function, headache or seizure disorder.

**CT:**

1. Variable bone involvement (destruction, erosion or hyperostosis)
2. Hyperdense, homogeneous enhancing mass
3. Frequent dural tail (35%)
4. Calcifications (25%)

**MRI:**

1. Isointense on T1 weighted, iso or hyperintense on T2-weighted
2. Bright homogeneous contrast enhancement. (Fig. 14 on page 30).
3. Dural tail enhancement (80%)
4. Diffusion ADC may be restricted
5. MRs elevated levels Alanine

**MRI criteria of meningioma malignancy:** (a) significant peritumoral oedema, (b) absence of calcium deposition, (c) nonhomogeneous contrast enhancement, (d) cysts within the lesion and (e) poorly defined, irregular borders. (Fig. 14 on page 30)

Surgery is the **treatment of choice and** subfrontal, pterional o endoscopic approach may be used. Complete removal can be achieved in 90% of cases. In planning the operation, it is important to remember that the blood supply comes into the tumor through the bone in the midline of the anterior fossa from branches of the ethmoidal, middle meningeal, and ophthalmic arteries.

### 3.1.5 Skull base Malignant Hemangiopericytoma (Fig. 15 on page 31):
Hemangiopericytoma are malignant mesenchymal non-meningothelial tumors arising from pericytic cells and account for less than 1% of all vascular neoplasms. Mean age ranging from 37 to 44 years. They have an inevitable tendency to recur locally and metastasize distally.

The location of intracranial hemangiopericytomas is similar to that of meningiomas. Like meningiomas and other extraaxial masses, hemangiopericytomas are dural-based and show white matter "buckling".

CT:
1. Soft tissue hyperdense extraaxial mass
2. Low density cystic or necrotic areas are common
3. Bone erosion (90%) (Fig. 15 on page 31).
4. Peritumoral oedema is frequent.

MRI:
1. Isointense on T1 weighted, iso or hyperintense on T2-weighted with cortical gray matter.
2. Prominent internal serpentine signal voids, suggesting vessels are common. Cerebral angiography show intensive vascularization.
3. This tumor show heterogeneous enhancement on both contrast-enhanced CT scans and MR images
4. Dural tail in 50%.

treatment of choice is surgery resection and postoperative adjuvant radiotherapy. Long-term follow-up is paramount because local recurrences and distal metastases can develop sometimes years after the initial treatment.

3.1.6 Small cell undifferentiated carcinoma-neuroendocrine (Fig. 16 on page 32):

Small cell neuroendocrine carcinoma (SCNC) group II of the nasal or paranasal region is extremely rare. Neuroectodermal neoplasms may be divided into lesions showing primarily epithelial differentiation (Group I, neuroendocrine carcinomas) and a more diverse group (Group II) of nonepithelial neoplasms. Group II tumors discussed include olfactory neuroblastoma, malignant melanoma, and Ewing’s sarcoma. Clinical diagnosis is often a challenge. Immunohistochemical and molecular complex analysis are needed for definitive diagnosis. Mean age ranging from 40- 50. Clinical features are headache, epistaxis, chronic sinonasal sinusitis.

CT
1. Homogeneous soft tissue mass occupying the ethmoid air cells and nasal fossa.
2. Intracranial extension is often: the cribriform plate is the pathway through to the anterior cranial fossa.
3. Relatively uniform enhancement
4. There is usually nasal bony remodeling and erosion at cribiform plate.

**MRI**: *(Fig. 16 on page 32)*:

1. Variable T1 and T2 weighted signal with hemorrhagic areas.
2. Nasal poliposis mass with moderate gadolinium enhancement.
3. Large peripheral cystic components are frequent.

The **treatment of choice is**. combined surgical approach and chemoradiation.

### 3.1.7 Nasosinusal Lymphoma *(Fig. 17 on page 33)*:

Non-Hodgkin's lymphoma (NHL) is a rare tumor of the skull base. Nasal and paranasal sinus NHL with both skull base and intracranial involvement represents advanced-stage primary sinonasal disease.

**CT**:

1. Bulky, lobular soft tissue mass in nasal cavity and sinus.
2. Bone remodeling or destruction, nasal septal involvement
3. Isodense moderate homogeneous enhancing mass

**RM**:

1. Intermediate, homogeneous signal on T1
2. Low-intermediate on T2
3. Moderate, diffuse enhancement
4. Low ADC on DWI *(Fig. 17 on page 33)*

**Treatment**: Surgical biopsy before definitive treatment is recommended. Local radiation therapy provides local control; adjuvant chemotherapy after primary radiation therapy may be required for recurrent disease.

### 3.1.8 Fibrous Dysplasia *(Fig. 18 on page 34)*:

Fibrous dysplasia is a developmental anomaly that can affect any bone in the body. The skull and facial bones are the affected sites in 10-25% of patients with monostotic
fibrous dysplasia and in 50% of patients with polyostotic fibrous dysplasia. Sarcomatous degeneration is rare.

CT:

1. Expansive lesion of medullar bone space with variable attenuation: (fig.17) Pagetoid mixed patter (25 %); sclerotic "ground-glass" density (56 %) or cystic (20%). Reflecting his mixture fibroosseus nature and activity phases. Cystic areas may be present.
2. Affected bone encroaches on skull base canals and foramina
3. 3 presentations :Monostotic, polyostotic or diffuse (McCune-Albright syndrome)

RM: localized fibrous dysplasia on MR imaging often mimics a tumor because fibrous tissue can enhance brilliantly after the injection of contrast material.

1. Low signal on T1 and T2 images with more heterogeneous signal patter in "active" areas ( high signal )
2. Characteristic areas of variable to strong contrast enhancement

The treatment of choice is surgery when possible.

3.1.9. Primary Intraosseous Frontal Hemangioma (Fig. 19 on page 35):

Primary intraosseous cavernous hemangiomas (PICHs) of the skull base are extremely rare tumors. These lesions are most common in the frontal and parietal bones of the calvarium. These tumors are slow-growing lesions and typically occur in women in the fourth and fifth decades of life. Although hemangiomas of the skull base are slow-growing lesions, they can be expansive and involve neighboring structures.

CT:

1. Expansive, well-circumscribed area of rarefaction with a sunburst pattern of trabeculations radiating from a common center. (Fig. 19 on page 35)
2. There is usually no reactive sclerosis at the margins.
3. Honeycomb or soap-bubble configuration is characteristic
4. Enhancing soft-tissue masses with lytic bone destruction
5. Mastoid, jugular, magnun, lacerum foramen can be involved

MRI:

1. Variable. High signal intensity on T2 and low signal intensity on T1, with
2. Enhancement occurs with contrast administration
3. Hypervascular lesion and a delayed blush on dynamic sequences.

**Treatment:** The surgical treatment of choice is en bloc excision and establishment of normal bony margins by drill curettage.

**3.2: CENTRAL SKULL BASE:**

**3.2.1. Juvenile angiofibroma** *(Fig. 20 on page 36):*

Juvenile angiofibroma is an uncommon, highly vascular tumor that affects adolescent boys. Nasal obstruction, epistaxis are the initial signs and symptoms. The blood supply of these lesions is primarily from the external carotid artery (ECA) and in some cases from the internal carotid artery (ICA).

The site of origin is the region of nasopharynx and the sphenopalatine foramen. Once the tumor gain access to the pterygopalatine fossa, it may spread to the orbit and the middle fossa. It may have a very aggressive behavior, extending to adjacent tissues and causing bone destruction by compression. The intracranial involvement is relatively frequent (10% to 36%) but it rarely goes beyond the duramater.

The inferior orbital fissure is the usual site for intracranial extension.

**CT:**

1. Expansive soft tissue mass with homogeneously avid enhancing
2. Involvement of the pterygopalatine fossa is generally associated by anterior remodeling of the posterior wall of the maxillary sinus.

**MRI:**

1. Low signal intensity on T1-weighted with brightly homogeneous enhancement on T1 gadolinium
2. Hyperintense on T2-weighted images *(Fig. 20 on page 36).*
3. Internal foci of punctate low signal intensity represent tumor vessels.

**Recommended treatment** is surgery but with the highly vascular nature of the lesion there is a risk of extensive blood loss. Preoperative embolization and endoscopic surgical approaches facilitate removal in lower stages without increasing morbidity.

**3.2.2. Middle fossa Meningioma** *(Fig. 21 on page 38):*
Meningiomas are typically benign tumors that arise from arachnoidal cells of the meninges. They account for approximately 15% of all primary brain tumors.

**Localisation:** Sphenoid wings (50%), tuberculum sella and olfactory groove (40%). Posterior fossa (8%).

Meningiomas of the wings of the sphenoid bone are often subclassified into middle, medial and hyperostosing en plaque varieties. Extension though the lateral orbital wall is common and accounts for the classic clinical presentation of this lesion: slowly progressive, painless unilateral exophthalmos and decreased visual acuity (Fig. X). Any of the meningiomas of the sphenoid ridge can extend extracranially via skull base foramen or by destruction and direct extension of the middle cranial fossa (Fig 20).

**CT:**

1. Focal areas of destruction, erosion or hyperostosis (bone windows).
2. The soft-tissue component enhances intensely after the administration of contrast material.

**MRI:**

1. T1 and T2-weighted images show extraaxial mass similar to parenchymal intensity
2. Bright enhancement after Gd (Fig. 21 on page 38).
3. Extraaxial component is better seen on SET1 + Gd coronal plane

**Treatment of choice:** The decision of whether to, and how best to, treat a meningioma is based on multiple factors, including size and location of the tumor, symptoms, growth rate, and age of the patient.

In general, there are three basic options: Observation (in meningiomas with slow growing, increasing its size only 1-2 mm per year), surgical removal (including minimally invasive surgical endoscopic endonasal approach to reach meningiomas at the base of the skull) and radiotherapy.

**3.2.3. Invasive Macroadenoma** (Fig. 22 on page 38):

Pituitary adenomas account for approximately 10 to 15% of primary brain tumors operated upon in the Unites States. MRI of healthy subjects indicates that approximately 10% of the population harbors pituitary lesions. It is estimated that 5% of pituitary adenoma become invasive and may grow to gigantic sizes (>4 cm in diameter). They
may invade skull base, extending into anterior/middle/posterior fossae. They cannot distinguish from pituitary carcinoma in base of imaging.

CT:

1. Large invasive mass without separate identifiable pituitary mass extend inferiorly invading sphenoid.
2. May destroy upper clivus
3. Focal areas of bone destruction and erosion that can contain floccular calcification
4. Heterogeneous enhances intensely after the administration of contrast material

MRI: (Fig. 22 on page 38)

1. Isointense on T1 and on T2-weighted images
2. Extraaxial mass similar to parenchymal intensity
3. Early, intense, but heterogeneous enhancement after Gd
4. Cyst and hemorrhage are common

Treatment of choice: Surgical resection. Medical, stereotactic radiosurgery, or convencional radiotherapy.

3.2.4. Nasopharyngeal carcinoma: (Fig. 23 on page 39)

Nasopharyngeal carcinomas account for approximately 70% of all primary malignancies of the nasopharynx. Diagnosis is usually achieved with endoscopic guided biopsy. The mean age at diagnosis is 45-55 years. A minority of patients have submucosal disease, with normal appearing overlying mucosa. MRI is then essential in guiding biopsy. Nodal metastases are present in the vast majority of patients at the time of diagnosis (75 - 90%)

CT:

1. Large invasive mass in nasopharyngeal area
2. Large aggressive tumors may extend into any direction, eroding the base of skull and passing via the Eustachian tube, foramen lacerum, foramen ovale or directly through bone into the clivus, cavernous sinus and temporal bone
3. Bone has irregular margins where it has been destroyed

MRI:

1. Is more sensitive to perineural spread and for demonstrating early the bone marrow changes of infiltration (Fig. 23 on page 39)
2. Isointense on T1 and on T2-weighted images
3. Fluid in the middle ear is a helpful marker
4. Post contrast sequences should be fat saturated
5. Prominent heterogeneous enhancement is typical
6. Perineural extension should be sought

**Treatment of choice:** IMRT Radiotherapy offers the best chance of long-term control. Endoscopic endonasal approach (EEA) is an appropriate neurosurgical technique in recurrences, when the intracranial invasion is limited.

### 3.2.4. Chordoma (Fig. 24 on page 40):

Chordomas are rare, slow-growing malignant neoplasms of embryonic notochordal derivation. They account for less than 1% of all intracranial tumors. Approximately 35% affect the skull base. Clivus (spheno-occipital synchondrosis) is the most often localization, others are: sphenoid (30%) and petrous apex. Age at onset is 20-40 years. Clinical issues: include cranial neuropathies, headache, vision disturbances and otolaryngologic symptoms such as nasal obstruction or epistaxis.

**CT:**

1. Enhancing soft-tissue masses with lytic bone destruction (95%)
2. May extend into the epidural space and causing compression of the vertebrobasilar system and brainstem ("thumbs" the Pons).
3. Often shows areas of calcification, many of which are actually fragments of destroyed bone. (Fig. 24 on page 40)
4. Jugular, magnun, lacerum foramen can be involved

**MRI:** Isointense to slightly hypointense on T1-weighted

1. Classically hyperintense on T2-weighted images.
2. They also may contain areas of decreased signal intensity corresponding to calcification or hemorrhage (T2*)
3. Heterogeneous enhancement occurs with contrast administration (Honeycomb pattern).
4. Vascular encasement is the rule (80%)

**Treatment of choice:** Complete surgical resection followed by radiotherapy offers the best chance of long-term control. Endoscopic endonasal approach (EEA) is the neurosurgical technique when the intracranial invasion is limited. Chordomas are radioresistant, requiring high doses of radiation to be controlled. Therefore, highly focused radiation such as proton therapy is more effective than conventional x-ray radiation.
3.2.5. Chondroblastoma (Fig. 25 on page 41):

Chondroblastoma is a rare primary cartilaginous benign bone tumor, which accounts for 1% of all primary bone tumors. Skull base and facial bones are an extremely unusual site of occurrence. The squamous temporal bone is the commonest site of occurrence in the skull extending into the infratemporal fossa with time.

CT:
1. Expansive intermediate or low density mass
2. CT confirms the lytic nature of the lesion and shows areas of calcification.

MRI:
1. Cystic polylobulated lesion, hipointense on T1-weighted and hyperintense on T2-weighted. (Fig. 25 on page 41).

3.2.6. Chondrosarcoma (Fig. 26 on page 42, Fig. 27 on page 43):

Chondrosarcoma is a slow-growing, malignant cartilaginous tumor. Account for approximately 6% of all skull base lesions. They can arise in cartilage, endochondral bone or primitive mesenchymal cells in the brain, meninges, membranous bone of soft-tissue. Secondary chondrosarcoma can occur in bone diseases (Fibrous Dysplasia, Paget's disease, Ollier syndrome).

Specific sites of involvement include the parasellar region, cerebelopontine angle and facial region (sphenoethmoid and maxillary bone). The parasellar and facial lesions can cause extensive destruction of the skull base.

CT:
1. Characteristic Chondroid calcifications in the tumor matrix 50% (arc, ring-like, stippled and amorphous calcifications). (Fig. 26 on page 42, Fig. 27 on page 43)
2. Bone erosion and destruction (50%) sharply defined edges.
3. Variable enhancing soft-tissue mass.

MRI: (less specific)
1. Hypointense relative to the brain on T1-weighted images.
2. Hyperintense on T2-weighted images.
3. Heterogeneous internal areas of decreased signal represent calcification.
4. Enhancement is heterogeneous.

Treatment of choice: Includes careful preoperative evaluation and surgical resection or radiotherapy, particularly carbon ion radiotherapy which has been reported to achieve a
better outcome than simple local control. Endoscopic endonasal approach (EEA) is the neurosurgical technique when the intracranial invasion is limited.

3.2.7. Epidermoids and Dermoids cysts (Fig. 28 on page 44):

Epidermoids and dermoid cysts are benign congenital lesions of ectodermal origin. They account for approximately 1% of all intracranial tumors. Although these lesions are congenital, patients are usually not symptomatic until they are aged 20-40 years. Most occur in the region of the cerebellopontine angle and sphenoe-temporal suture (fig. 26). The distinction between dermoid and epidermoid lesions is important prognostically and may impact on surgical management as a subtotally resected dermoid is less likely to recur than its epidermoid counterpart. Intracranial dermoids usually contain a varying combination of lipid, liquid cholesterol, whorls of hair, calcifications and decomposed epithelial cells producing typical appearances.

CT:

1. Low attenuation mass similar to cerebrospinal fluid.
2. Decreased attenuation mass are most often extradural.
3. Fatty density areas within the mass, scattered subarachnoid fat droplets foci when rupture.
4. Enhancement is rare but can sometimes be seen around the margin of the tumor.
5. Geographic patter erosion of skull base
6. Calcification occurs in only 15-20% of cases.

MRI: (Fig. 28 on page 44):

1. Slightly hyperintense or isointense relative to gray matter on T1-WI (tends to vary with the lipid content, with the signal intensity being increased in lesions with a high lipid content and decreased in those with a low lipid content)
2. Isointense relative to CSF on T2-weighted images.
3. The center of the epidermoids usually has an internal architecture with areas of heterogeneity.
4. Enhancement of portions of the rim may be seen after the administration of contrast material.
5. Typically shows restricted diffusion (bright).
6. They are not completely suppressed on FLAIR sequence giving heterogeneous appearance in contrast to arachnoid cysts

Treatment of choice: Surgical resection is the treatment of choice. These tumors have a tendency to have a capsule that is densely attached to surrounding structures including the tiny, delicate vessels of the brain stem and cranial nerves. This can make complete
capsule resection quite difficult, without causing serious injury. In some patients it may be necessary to leave remnants of the capsule behind, which increases the risk of recurrence.

### 3.2.8. Cholesterol Cyst (Fig. 29 on page 45):

Cholesterol granuloma is an inflammatory lesion most commonly found in the central skull base (petrous apex, middle ear). This lesion typically forms when blood from bone marrow leaks into a nearby air cell. The trapped blood eventually breaks down and generates significant inflammation, resulting in a buildup of fluid in the air cell. This fluid-filled lesion slowly expands over time and causes progressive thinning of surrounding bone. Clinical findings: Hearing loss, Gradenigo syndrome (abducens nerve palsy, pain and facial nerve palsy) and hemifacial spasm.

**CT:**
1. Expansive bony changes with scalloping of surrounding bone.
2. Non enhancing soft-tissue masses with lytic bone destruction may involve the adjacent otic capsule and carotid canal.
3. Jugular, magnun, lacerum foramen can be involved.

**MRI:**
1. High signal intensity on T1 and low signal intensity on T2, with peripheral dark hemosiderin deposition ring (fig.27)
2. Non enhancement occurs with contrast administration.

**Treatment:** For surgical removal infrachoclear transtemporal approach or transsphenoidal endoscopic approach are options.

### 3.2.8. Schwannomas (Fig. 30 on page 46, Fig. 31 on page 47):

Schwannomas or neurilemmoma arise from the nerve sheath and consist of Schwann cells in a collagenous matrix. These lesions account for 6-8% of intracranial neoplasm. Slowly-growing.

Localization: vestibular schwannomas are the most common Localization, followed by trigeminal (Fig. 30 on page 46, Fig. 31 on page 47) and facial schwannomas and then glossopharyngeal, vagus, and spinal accessory nerve schwannomas.

1. Large, sharply demarcated, fusiform or dumbbell mass.
2. Iso - hypodenuating relative to brain parenchyma.
3. Calcification or areas of hemorrhage.
4. The enhancement pattern is typically homogeneous.
5. Bone-window images can demonstrate remodeling of the adjacent skull base.
6. Smooth enlargement of skull base foramina and fissures

**MRI:**

1. Isointense or slightly hypointense relative to gray matter on T1-weighted images
2. Hyperintense on T2-weighted images.
3. Gadolinium enhancement is typically homogeneous.
4. Intratumoral nonenhanced cystic components may be marked

**Treatment:** Total surgical removal of tumor in a single operation is the goal.

### 3.2.9. Triton tumor (Fig. 32 on page 48):

Malignant triton tumor (MTT) is extremely rare. In this tumor Schwann cells coexist with malignant rhabdomyoblasts. This tumor is characterized by their aggressiveness and poor prognosis.

**Localization:** usually occurs in the head and neck and trunk.

**Intracranial MTT** is very rare and the prognosis is poor

The diagnosis of MTT was mainly based on histopathological characteristic and immunohistochemical features.

**CT:**

1. Large soft tissue hypoattenuation mass invading the central skull base with extension to the intracranial middle fossa.
2. Contrast enhancement may show areas of central necrosis
3. Smooth enlargement of skull base foramina and fissures

**MRI:**

1. Hypointense on T1 and hyperintense on T2-weight
2. Bright homogeneous contrast enhancement (Fig. 32 on page 48).

**Treatment:** Early diagnosis and complete resection followed by radiation therapy is important for long-term survival.

### 3.3 POSTERIOR SKULL BASE:
3.3.1. Multiple myeloma (Fig. 33 on page 49):

Extramedullary plasmacytomas are rare lesions and can occur either as a part of a generalized disease (Multiple myeloma) or a local entity. Plasmacytoma account for less than 1% of all head and neck tumors. 80% these occur in the nasopharynx and the paranasal sinuses. When the skull is involved, most occur in the calvarium and the skull base is rarely affected. Neurologic symptoms due to plasmacytomas located either in the base of the skull or at intracranial locations are extremely rare. The cause of the neuropathy is the direct compression of nerves or nerve groups in their intracranial course.

CT:

1. Well demarcated solitary intraosseous lytic tumor with non-sclerotic margins.
2. Occasionally aggressive with bone destruction and involvement of adjacent structures. Lytic mass with scalloped, poorly margins, non-sclerotic margins. (Fig. 33 on page 49)
3. Multiple calcifications are often visible.

MRI:

1. ISO-hypointense on T1 weighted images. homogeneous.
2. Moderate signal intensity on T2 weighted images (high cellular tumor)
3. Moderate homogeneous contrast enhancement with central inhomogeneity.

Treatment of election: local irradiation is the primary mode of treatment for extramedullary plasmacytomas, occasionally followed by surgical resection when residual tumor. When multiple myeloma is diagnosed, the treatment of choice is systemic combination chemotherapy.

3.3.2. Skull Base Metastasis (Fig. 34 on page 50):

Metastasis to the skull-base particularly affects patients with carcinoma of the breast, lung, kidney and prostate. Clinically, the key feature is progressive ipsilateral involvement of cranial nerves. Five syndromes have been described according to the metastatic site including the orbital, parasellar, middle-fossa, jugular foramen and occipital condyle syndromes. MRI is nowadays the most useful examination to establish the diagnosis but plain films, CT scans with bone windows and isotope bone scans remain helpful to demonstrate bone erosion. Overall median survival is about 2.5 years.

CT:
1. Infiltrative soft tissue mass with bone erosion.
2. More frequent: Lytic mass with scalloped, poorly marginated, non-sclerotic margins.
3. May be Sclerotic (e.g Prostate) or expansive (thyroid and kidney)
4. Multiple calcificcations are often visible.

**MRI: (fig. 32)**

1. Iso-hypointense on T1 weighted images heterogeneous.
2. Moderate signal intensity on T2 weighted images (in high cellular tumors)
3. Moderate heterogeneous contrast enhancement with central necrosis.
4. Fat saturation necessary to distinguish enhancement from normal hyperintense marrow.

**Treatment of election:**

The treatment depends on the nature of the underlying tumor. Radiotherapy is generally the standard treatment, while some patients with chemosensitive or hormonosensitive lesions. Gamma Knife radiosurgery is sometimes a useful alternative, particularly for previously irradiated skull-base regions, and for small tumors (diameter < 30 mm).

### 3.3.3. Solitary fibrous tumor (Fig. 35 on page 51, Fig. 36 on page 52):

Is an uncommon spindle cell tumor that typically arises from the visceral pleura. Intracranial localization is rare and tend to show a dural attachment. In the central nervous system they form dural-based masses mimicking meningiomas. The usual sites are clinoid process, orbit, sellar- and parasellar area, middle cranial fossa, cerebellopontine angle, lateral ventricle and the falx cerebri.

Clinical manifestations: exophthalmos, loss of vision, hemiparesis, hearing loss, facial palsy and headache.

**CT:**

1. Discrete extra axial masses, lobulated, solid, with peritumoral cyst
2. Bone invasion is common.
3. Central hypoenhancing or nonenhancing areas may be seen in the tumor, which represent necrosis or cystic change.
4. Calcification is rare and can be seen in large benign or malignant tumors.

**MRI:**
1. Typically have intermediate signal intensity on T1-weighted images and heterogeneous low signal intensity with flow voids on T2-weighted images. (Fig. 35 on page 51, Fig. 36 on page 52)
2. Hypervascular areas enhance intensely, the hypocellular areas show moderate enhancement, and areas of necrosis or of cystic or myxoid degeneration do not enhance after contrast material administration.

**Treatment of choice:** Total excision is the treatment of choice. Radiotherapy is administered if the excision is subtotal or partial. The chemotherapeutic agent Toremifene citrate can also be administered if the proliferation rate is high. Recurrence and also malignant transformation have been reported.

**3.3.4. Schwannomas** (Fig. 37 on page 53):

See 3.2.8 Schwannomas

**3.3.5. Glomus Jugulare tumor** (Fig. 38 on page 54):

Glomus jugulare (jugular foramen paragangliom) is a slow growing vascular tumor located at the skull base. It is the most common tumor of the temporal bone. Clinical manifestation: pulsatile tinnitus, difficulty swallowing, hoarseness, hearing loss, facial weakness and hypoglossal neuropathy.

**CT:**

1. Well-defined enhancing soft-tissue masses within the jugular foramen
2. CT shows characteristic permeative-destructive bone changes: initially, erodes the superolateral margin of the jugular foramen and jugular spine with subsequent extension to the mastoid and adjacent occipital bone.
3. Significant intracranial and extracranial extension may occur, as well as extension within the sigmoid and inferior petrosal sinuses.
4. Neural infiltration is also common.
5. May extent superiorlateraly from jugular foramen into the middle ear.

**MRI:**

1. Highly vascular tumor on MRI is characteristic.
2. Increased T2W signal intensity and decreased T1W signal intensity, with intense enhancement after gadolinium injection.
3. Numerous, prominent internal flow voids can also be seen, resulting in what has been called a "salt-and-pepper" pattern.
4. Gradient recall echo (GRE) MR sequences are more sensitive to flow and can therefore demonstrate the blood vessels within the tumors better than spin echo sequences. (Fig. 38 on page 54).

**Treatment of choice:** Surgical resection. Embolization is a common technique used as the alone treatment option or as a precursor to surgical excision. Stereotactic radiosurgery is being used as primary or adjuvant therapy.

3.3.6. Rhabdomiosarcoma (Fig. 39 on page 55):

see 3.1.3. Rhabdomiosarcoma.

3.3.7. Foramen Magnun Meningioma: (Fig. 40 on page 56)

See 3.2.2. Middle fossa Meningioma

Images for this section:
Fig. 1: A. 3D CT of skull base B. Anatomical specimen

Fig. 2: Anterior cerebral fossa.

Fig. 3: CT & Specimen correlation
Fig. 4: Middle cranial fossa

Fig. 5: Middle cranial fossa
**Fig. 6:** Posterior cerebral fossa

**Fig. 7:** Clivus and foramen magnum area
Fig. 8: Correlation between CT and anatomical specimen
**Fig. 9:** Esthesioneuroblastoma. B. Coronal bone windows CT scan shows the lesion extending through the cribiform plate into the anterior cranial fossa (arrow). A. Skull base diagram. C Coronal T1-Weighted RM image postgadolinium and D. T2-weighted MR image shows the tumor filling the right nasal cavity. Note the obstructive phenomenon into the ethmoid sinus and the small tip intracranial component (arrows. In these cases endoscopic approach is preferred.
**Fig. 10:** Esthesioneuroblastoma. A. Coronal, T1-weighted MR image postgadolinium show a large bilateral nasal cavity tumor extending into the intracranial epidural space. Note the expansive frontal sinus mucoceles secondary to obstructed frontal infundibula B. Axial CT scan shows a destructive bone pattern with calcification. The bone is partially remodeled laterally, but the periorbita is the true barrier that prevents the lesion from extending into the orbit. C. Kull base diagram
Fig. 11: A. T2-weighted MR image show a well defined, bulky tumor in the deep parotid space. B. postcontrast Coronal T1-weighted MR image show the lesion extending through the foramen ovale into the right cavernous sinus in the middle cranial fossa.
Fig. 12: Nasosinusal carcinoma A. Coronal T1-weighted MR image reveals a relatively well-defined maxillary sinus mass. The enhancement is mild-to-moderate and heterogeneous. B. Skull base diagram C. Axial fat-suppression T2-weighted image shows a intermediate to high signal due to high cellularity. D. Axial NECT shows relatively well-
defined mass with areas of bone destruction and remodeling affecting maxillary sinus, nasal cavity and sphenoid bone. Some small foci of calcification are seen within the mass.

Fig. 13: Rhabdomyosarcoma A. Axial, B. Skull base diagram, C. Sagital and D. Coronal SE-weighted T1 MR postcontrast demonstrate a large infiltrating enhancing soft tissue mass lesion with bone destruction involving right frontal sinus, right nasal cavity and orbital roof not crossing of the midline. The intra-cranial epidural invasion across the frontal base is best seen on coronal imaging. This tumor can reach intranial cavity (41%) by direct invasion from the orbit or sinus.
Fig. 14: Olfactory meningioma A. T1-weighted, B. T2-Weighted coronal MR images shows a Meningioma of olphatory groove at the anterior cranial fossa extending through the cribiform plate and roof of the ethmoid into upper nasal cavity. The lesion also protrudes into the orbit. C. and D. Sagittal MRI T1-weighted postcontrast images better demonstrate the frontal sinus and bone involvement. Note the large cystic anormalities in the marging of the tumor mimicking olfatory neuroblastoma.
Fig. 15: Hemangiopericytoma Contrast enhanced CT shows skull base erosion and hyperdense heterogeneous enhancing extraaxial mass occupying the right middle fossa and anterior fossa. 3D-CT scan demonstrate severe bone erosion of anterior and middle fossa. the mass reach the anterior cranial fossa eroding the planum esphenoidale and the middle fossa throught optic canal and superior orbital fissure.
Fig. 16: Carcinoma neuroendocrino A. Sagital T1-weighted MR image postcontrast show tumor arising in the upper nasal cavity. Note the very large, intracranial cystic component of the tumor resembling the neuroblastoma pattern. C. Axial T2-weighted and D. Axial CT postcontrast images show the cystic component without edema previously interpreted as porencephalic cyst.
Fig. 17: Nasosinusal lymphoma. A. Axial SE-T1 weighted MR image shows a nasal cavity infiltrating mass involving the right ethmoid cells. Note the hyperintense signal in the ethmoid cells due to obstructive sinusitis. The medial wall of the left orbit is expanded by the tumor. B. Skull base diagram. C. Coronal T2-weighted MR image shows a homogenous hypointense infiltrating mass affecting nasosinusal area with a small tip extending through the cribiform plate. D. Diffusion weighted MR image (DWI) shows a strongly hypointense ADC map due to high cellularity of the mass.
Fig. 18: Skull Base Fibrous Dysplasia

A. and B. Axial Bone CT shows expansive bone lesion with variable attenuation. Centrally lucent lesions with thinned but sclerotic borders and ground-glass density are seen. B. 3D reformation view shows espheno-ethmoidal thickening due to fibrous Dysplasia.
Fig. 19: Frontal hemangioma. A. Axial and Coronal CT C+ shows a expansive, well-circumscribed area of rarefaction with a sunburst pattern of trabeculations radiating from a common center. The mass is originating in the right frontal bone and is extending into the right orbit in a patient presenting with proptosis. C. and D. radiography reveals a sunburst pattern resembling osteosarcoma.
Fig. 20: Juvenile Angiofibroma. A. Coronal T1-Weighted MR imaging shows an intensely enhancing mass originating as sphenoparatine foramen with extension to nasal cavity, nasopharynx, and infratemporal fossa. Coronal plane shows the JNA extension to the middle fossa, in close contact with the temporal lobe. B. Coronal CT scan shows bone...
remodeling and destruction C. Skull base diagram D. Coronal CT scan shows coana occupation and sphenoid lateral wall is expanded and eroded. Inferior Orbital fissure may be widening o Top Differential diagnoses: Rabdomyosarcoma

Fig. 21: Meningioma A. Coronal SE-T1 MRI shows a dumbbell-shaped homogeneous enhanced extraaxial mass. Note the cavernous sinus involvement medially. B. Skull base diagram C. Axial CT Scan at FCM level and D. at maxilary sinus level reveal the bulky extracranial parapharingeal component.
Fig. 22: Invasive Macroadenoma de Clivus. A. and D. Axial CT bone scan shows a large lytic expansive lesion of the sellar and parasellar region. Note the typical benign bone margins. B. Coronal C Sagital and E. Postcostras T1-weighted RM images show a large sellar mass with skull base invasion extending through cavernous sinus and clivus. Note de suprasellar uppwar extension and carotid enchasement. F. Skull base 3D view.
Fig. 23: Cavum carcinoma. A. Coronal T1 C+ MR reveals a mildly enhancing infiltrating mass arising in lateral pharyngeal recess destroying a small area of skull base bone around the oval and lacerum foramina. The ipsilateral cavernous sinus is infiltrated and carotid artery is entrapped. Masticador space muscles are also infiltrated. B. Skull base diagram C. Axial CT Scan shows the permeative pattern with oval foramen widening
Fig. 24: Chordoma A. Coronal T1-weighted postcontrast Fat suppression image shows a large rhinopharyngeal mass extending cerebral middle fossa through parasellar and oval skull base area. B. Skull base diagram. C. and D. Axial T1-weighted postcontrast image shows a large parapharyngeal mass eroding medially the petrous apex and lateral clivus bone. In the upper level expanding tumor invades and displaces laceral foramen and petrous apex laterally.
Fig. 25: Chondroblastoma of skull base. A. Axial T2-weighted RM image shows a high signal intensity erosive mass affecting retromaxilar space. B. Skull Base diagram. C. Sagital T1-weighted RM image shows a hypointense heterogeneous mass with middle fossa extension. D. Coronal T2-weighted RM image shows a extensive involvement of maxillary
**Fig. 26:** Chordrosarcoma A y B. Axial CT scan shows a destructive lytic lesion of clivus and petro-occipital fissure. Small foci of calcifications within the tumor matrix are shown. (note absence of choroid calcifications). The carotid canal is involved. B. Coronal SE-T2 MRI reveals a hyperintense tumor with small hypointense foci. D. Skull base diagram E. Axial Fat-suppression T2-weighted RM imagen shows the characteristic hyperintensity mass with medial extension into prepontine cistern
Fig. 27: Chondrosarcoma. A. axial B. coronal C. Sagital and D. 3D-CT: Show petroclival bone erosion and destruction with sharply defined edges. A more permeative pattern is seen along the clivus. No characteristic Chondroid calcifications are seen in the tumor matrix.
**Fig. 28:** Dermoid Tumor A. Coronal T1-Weighted and B. T2-Weighted MR images show a non-enhance large cystic appearance mass (hypo T1 and Hiper T2). The mass shows a well defined limits with extraaxial criteria extended into Silvian fissure. No edema was noted on T2. C. Coronal CT Scan shows the extensive skull base involvement with "remodeling", erosions and lytic appearance areas. D. Sagital T1-Weighted and E. Axial T1-Weighted MR images postcontrast show hiperintense periferal fatty foci. F. Skull base diagram
Fig. 29: Cholesterol Granuloma A. Axial Fat-supression T1-weighted RM imagen shows the characteristic hyperintensity cause the metahaemoglobin, inflammatory and cholesterol components. B. Skull base diagram C. Sagital T1-weighted RM imagen shows a hyperintese petroclival mass. D. After Gd the periferal tumoral enhancement obscure the mass boundaries. Note the involving of Meckel cave.
**Fig. 30:** V2 schwannoma A. T1-weighted MR image and B. Postcontrast T1-weighted MR image shows a large mass in the pterigopalatine fossa with extension into the left orbital apex through orbital fissure. Marked Peripheral enhancement of tumor with hypovascular cystic areas within the tumor. C. Fat-suppression T2-weighted MR image D. Skull Base diagram E. Coronal SE-T2 show a characteristic well defined high signal intensity mass remodeling sphenoid bone.
Fig. 31: Trigeminal (V2) Schwannoma. A. Posterior and B. Anterior 3D view show a remodelling and widening of inferior orbital fissure. C. Sagital CT scan shows also the pterygopatine fossa expansion. D. 3D diagram
Fig. 32: Triton tumor A and B. Axial CT scan shows a intermediate homogeneous infratemporal mass and enlarged oval foramen. The adjacent cigomatic bone is expanded by the tumor. C. skull base diagram D. Axial T1-weighted RM image poscontrast show a intense enhancement of the mass with foramen oval participation. E. Sagital Fat-Supression T2-weighted RM Image shows a non-homogeneous hyperintense mass. F. Sagital Fat-Supression T1-weighted RM image poscontrast show a intense enhancement. Note the intracranial extension through the oval foramen.
Fig. 33: Skull base Plasmocitoma A. Axial bone CT Scan shows a diffuse destructive-lytic areas in central skull base with sellar, basisphenoid and ethmoid involvement. B. Skull base Diagram C. and D. Axial postcontrast CT show a moderated enhancement infiltrative soft tissue mass involving both central skull base and right petrous bone. Multiple calcificications are also seen
Fig. 34: Skull base metastasis A. Sagital T1-weighted RM image shows a large hypointense infiltrating mass eroding the occipital bone. The tumor extends from clivus to yugular foramen. B. Skull base diagram C. Axial FLAIR reveals expanding to pontocerebellar angle D. Axial postcontrast T1-weighted RM image shows a heterogeneous enhance mass involving neurovascular structures in yugular foramen.
Fig. 35: Fibrous solitary tumor A. 3D-TC down view shows a lytic right occipital lesion B. Axial. C. Sagital and D. Coronal postcontrast T1-weighted Images show a large enhancing mass in posterior pontocerebellar angle with two components: one extraaxial in posterior fossa and other extracranial in parapharyngeal space.
**Fig. 36:** Fibrous solitary tumor. A. Axial FLAIR T2-weighted image shows a characteristic hypointense mass; B. DWI show a low signal with no restriction; C. Axial T1 and D. postcontrast T1-weighted images show a isointense mass with homogeneous enhancement.
Fig. 37: Schwanomma of CN XI A. Axial and B. Coronal postcontrast T1-weighted RM images show a well defined bright enhancement mass in the lower part of pontocerebellar angle passing through the yugular foramen. C. Skull base diagram D. Coronal T2-weighted RM imagen shows hypointense focal area within the mass related to intratumoral haemorrhage.
Fig. 38: Glomus yugular and Vagal A. Axial and B. Axial and C. postcontrast T1-weighted RM images show a space-occupying lesion destroying the petrous bone with intense enhancement expanding the yugular foramen. D. Dynamic contrast Fast-SPGR shows the hypervascular nature of the lesion. E. Sagittal postcontrast T1-weighted RM images show a second space-occupying lesion in the internal yugular space representing vagal glomus tumor. F. Skull base Diagram
Fig. 39: Neck Rhabdomyosarcoma with intracranial extension A. Dynamic Fast-SPGR map show a mass infiltrating and destroying occipital condile, lateral clivus and apex petrous. A small intracranial nodule contact with medulla. The rhinopharyngeal is envolved and the deep planes of parapharyngeal space are loos. B. Axial D. Coronal and D,E Sagital postcontrast T1-weighted RM images show a large space-occupying lesion destroying the petrous bone with diffuse, often marked, heterogeneous enhancement. Note the anterior displacement of carotid artery. Prevertebral and carotid spaces are also occupied. F. Skull base Diagram
**Fig. 40:** Skull Base Meningioma

A. Antero-posterior Radiographic shows a diffuse sclerosis of large and lesser wings of sphenoid bone. B. Axial CT postcontrast shows a large enhanced mass involving anterior, middle and posterior fossa. The tumor is extending into the left orbit with proptosis. C. Skull base diagram
Imaging findings OR Procedure details

Introduction

All patients with suspicious of skull base lesion require high quality imaging for disease staging. The choice of modality is relative accessibility of MR and CT. For most circumstances CT will provide sufficient information of skull base bone and vascular involvement.

MRI is particularly useful for soft tissue evaluation and intracranial invasion. In general, for skull base and nasopharyngeal tumours MRI is superior to CT. MRI is however more expensive, less available and takes significantly longer.

CT Protocols

Evaluation by three-dimensional (3D) computed tomography (CT) was performed in a Toshiba Aquilion multidetector 64 row CT-Scan. The skull base 3D reconstruction was analyzed in a Vitrea Workstation.

MRI protocols

Sagittal T1-weighted:
TR 460 ms, TE 9,7 ms, 256 x 192; 5mm think, 1.5 mm skip, 24x24 field of view (FOV), 1 number of excitation (NEX)

Axial T1 FLAIR weighted:
TR 2237 ms, TE 7,7 ms, T1 750, 256x256, 5mm think, 1.5 mm skip, 24x18 FOV, 2 NEX

Axial T2 FR-FSE fat suppression:
TR 4700 ms, TE 89,7 ms, 320 x 256, 5 mm think, 1.5 mm skip, 24x18 FOV, 3 NEX, fat sat

Coronal T2 FR-FSE:
TR 4320 ms, TE 90,1 ms, 320 x 256, 5 mm think, 1.5 mm skip, 24x18 FOV, 3 NEX.

Postcontrast Axial dynamic T1 FSPGR fat suppression:
TR 150 ms, TE 1,5 ms, 256 x 160, 5 mm think, 1.5 mm skip, 24x24 FOV, 1 NEX, fat sat

Postcontrast Axial FSE T1 fat suppression:
TR 600 ms, TE 9,4 ms, 256 x 192, 5 mm think, 1.5 mm skip, 24x24 FOV, 3 NEX, fat sat

Postcontrast Coronal FSE T1 fat suppression:
TR 600 ms, TE 9,7 ms, 256 x 192, 5 mm think, 1.5 mm skip, 24x24 FOV, 3 NEX, fat sat

Conclusion
There are a wide variety of tumors affecting the base of the skull. These tumors share overlapping clinical radiologic and pathologic features that may lead to diagnostic confusion and possible misdiagnosis. The value of imaging studies in the histopathologic diagnosis of these lesions cannot be overemphasized. Three-dimensional imaging of the skull base provides an excellent topographic visualization of the tumor extent. CT and MR imaging are crucial in the diagnostic evaluation, treatment planning, and follow-up, monitoring of the disease. Such imaging, especially when contrast is used, can accurately detect and state the extent of tumor involvement.

References


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