Lesions of the petrous apex: an algorithmic diagnostic approach

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Learning objectives

The current exhibit aims to:

- To review anatomy of the petrous apex

- To propose an algorithm that helps the radiologists narrow the differential diagnosis when encountered with a lesion within the petrous apex

- To discuss most typical imaging findings of commonly encountered PA lesion
Background

The petrous apex is a complex region of the central skull base that is surrounded by a number of important vascular and neural structures and can be home to a wide range of disease processes.

Lesions arising in or spreading to the petrous apex cause varied and occasionally severe clinical squealer. Cross-sectional imaging with computed tomography and magnetic resonance (MR) imaging plays an important role in diagnosis and characterization of lesions occurring there.

The petrous apex is a pyramid-shaped structure that is formed by the medial portions of the temporal bone. It is obliquely positioned within the skull base, with its apex pointing anteromedially and its base located posterolaterally. The petrous apex is bounded by the inner ear structures laterally, the petro-occipital fissure medially, the petrosphenoidal fissure and internal carotid artery (ICA) anteriorly, and the posterior cranial fossa behind.

The superior surface is formed by the middle cranial fossa, Meckel cave, and ICA. Along the inferior surface are the jugular bulb and inferior petrosal sinus. The internal auditory canal (IAC) bisects the petrous apex into a large anterior portion that typically contains bone marrow and a smaller posterior portion that is derived from the otic capsule.

A number of identifiable vascular and neural channels are contained within the petrous apex. The petrous carotid canal and IAC are the largest channels traversing or bordering the petrous apex, but the Dorello canal, subarcuate canal, singular canal, and Meckel cave are smaller channels that are also seen reliably on high-resolution thin-section CT or MR images.

The IAC is located within the midportion of the petrous apex and houses the vestibulocochlear and facial nerves. It is generally directed anterolaterally from the cerebellopontine angle cistern, with its cisternal opening (the porus acusticus) located along the posteromedial edge of the petrous bone (Fig 1).

The petrous carotid canal lies within the anterior portion of the petrous apex and contains the horizontal portion of the petrous segment of the ICA, which passes over the foramen lacerum. The Dorello canal extends through the posteromedial portion of the petrous apex and contains the abducens nerve (cranial nerve VI).

The subarcuate canal (also referred to as the petromastoid canal) courses between the crura of the superior semicircular canal within the superior portion of the petrous apex. It contains the subarcuate artery, which supplies blood to the bony labyrinth, facial canal, and mastoid antrum.

The singular canal is a small channel that extends from the posterior margin of the IAC to
the junction of the ampulla of the posterior semicircular canal and the vestibule. It contains the singular nerve, which is a division of the inferior vestibular nerve that innervates the ampulla of the posterior semicircular canal.

Along the anterior superior aspect of the petrous apex is a smooth depression that serves as the floor of the Meckel cave.

The Meckel cave is a dura-lined diverticulum that contains the trigeminal (or gasserian) ganglion and the rootlets of the trigeminal nerve (cranial nerve V).

The anterior portion of the petrous apex is filled with marrow in approximately 60% of temporal bones (Fig 2), pneumatized in 33%, and sclerotic in 7%. Pneumatization of the petrous apex results from extension of air cells along infralabyrinthine, anterior, superior, posteromedial, or subarcuate tracts that communicate directly with the mastoid or middle ear cavity and provide direct pathways for disease spread from the mastoid bone or middle ear to the petrous apex. Pneumatization is asymmetric in 5%-10% of individuals.
**Fig. 1**: Normal petrous apex at CT. Axial CT images of the right temporal bone, obtained at the level of the IAC (A) and slightly more inferiorly at the level of the horizontal petrous carotid canal (B), show the normal petrous apex, which is bounded anteriorly by the petrosphenoidal fissure (arrows in B) and middle cranial fossa (MCF in A), posteriorly by the posterior cranial fossa (PCF), and medially by the petroclival fissure (arrowhead). The IAC divides the petrous apex into an anterior portion that usually contains marrow and a denser posterior portion that is derived from the otic capsule. C in A = cochlea, ICA in B = ICA canal, V in A = vestibule.

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Fig. 2: Fig. 2 Normal petrous apex at MR imaging. Axial unenhanced T1-weighted MR image at the level of the IACs shows the normal petrous apex. Note the high-signal-intensity fatty marrow in the anterior petrous apex (*) and the low signal intensity of the denser posterior petrous apex (arrowheads). In pneumatized or sclerotic apices, the anterior portion may normally demonstrate low signal intensity due to the absence of fatty marrow.

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Findings and procedure details

Petrus apex lesions can be classified on the basis of their origin into the following categories: developmental lesions, inflammatory lesions, benign tumors, malignant tumours, vascular lesions, and osseous dysplasias. The most common lesions arising in the petrous apex are cholesterol granulomas. In addition, one should also be familiar with anatomic variants or pseudolesions in the petrous apex that can be mistaken for pathologic conditions.

1- Normal Variants :

a) Petrous apex pneumatization:

Pneumatization can be highly variable involving a large portion of the petrous temporal bone or only a small posterolateral segment.

The air cells of the petrous apex are susceptible to similar pathologic processes that occur in the mastoid segment including obstruction, opacification, inflammation, and infection.

b) Asymmetric fatty marrow in the petrous apex:

Typically, normal marrow contains significant adipose tissue, and signal characteristics parallel those of scalp or orbital fat.

Fatty marrow is hyperintense on routine T1- and T2-weighted sequences. Confirmation is made by observing the complete loss of signal with fat-saturation techniques, such as a STIR sequence and frequency-selected fat-suppressed sequences.

2- Developmental Lesions :

a) Cholesterol granuloma:

Cholesterol granulomas of the temporal bone can occur in the mastoid segment, the middle ear, and the petrous apex. In fact, cholesterol granulomas are the most common primary petrous apex lesions.

It can extend in multiple directions from the petrous apex, most commonly posterolaterally into the mastoid segment, the internal auditory canal, or the middle ear. Lesions can also extend into the clivus, cerebellopontine angle, and middle cranial fossa.

Temporal bone CT reveals an expansile, sharply defined, and often rounded mass of the petrous apex with cortical thinning and trabecular breakdown.
The general appearance is that of a slowly progressive benign process.

There is central soft-tissue density without an internal matrix, a calcification, or residual septations. If the lesion is sufficiently enlarged, frank bony dehiscence is observed.

On MRI, cholesterol granulomas are typically hyperintense on both T1 and T2 sequences because of the accumulation of blood breakdown products and proteinaceous debris.

Small lesions may be relatively homogeneous, whereas large lesions show more heterogeneity. Often cholesterol granulomas have a distinct hypointense peripheral rim on T2-weighted images due to hemosiderin deposition.

After contrast administration, there may be subtle peripheral enhancement secondary to inflammatory response but no central enhancement that would indicate solid tissue.

b) Cephaloceles:

Petrous apex cephalocele (PAC) is a rare cystic lesion that is often misinterpreted as intrinsic inflammatory lesion of the petrous apex.

PAC arises outside the petrous apex from the Meckel's cave and secondarily erodes rather than expands the petrous apex. The important discriminators between PAC and cystic inflammatory lesions of the petrous apex are the eccentric location in the petrous apex, the continuity with the posterolateral aspect of the Meckel's cave, and the absence of inflammatory changes.

At imaging, petrous apex cephaloceles are situated just above the anterior petrous apex and are continuous with the Meckel cave. They are smoothly marginated and have the same signal intensity characteristics as CSF with all MR imaging sequences.

CT may show extensive nonaggressive erosion of the petrous apex with a smooth or scalloped border. Obliteration of the cyst cavity with fat or muscle is recommended for symptomatic lesions, and serial imaging is used for asymptomatic lesions.

Coronal or sagittal T2-weighted imaging is particularly useful for demonstrating extension of a cephalocele through the skull base.

Occasionally, cephaloceles of the petrous apex may arise through other defects in the skull base and dura mater. These may occur incidentally or in association with neurofibromatosis type 1.

Iatrogenically induced cephaloceles can also be seen after surgery of the petrous apex.

c) Cholesteatoma:
Congenital cholesteatomas are slow-growing lesions and may be asymptomatic for years.

On temporal bone CT, there is a smooth expansile lesion of the petrous apex (Fig 3). The central portion of the lesion shows no calcification or bony matrix.

On MRI, cholesteatoma typically is hypointense on T1, hyperintense on T2, and intermediate in signal on FLAIR images. Diffusion restriction is reported to be characteristic of this lesion.

After contrast administration, there may be subtle peripheral rim enhancement.

d) Mucocele:

Petrous apex mucoceles are uncommon. Similarly to mucoceles found elsewhere in the head and neck, they are most likely caused by postinflammatory obstruction of a pneumatized petrous apex air cell.

CT of mucoceles shows a smoothly expansile bone lesion that may cause septal erosion and can be difficult to distinguish from a cholesterol granuloma.

MR imaging is particularly helpful in distinction between these two entities, as mucoceles typically have low to intermediate signal intensity on T1-weighted images. Mucoceles are hyperintense on T2-weighted images and do not enhance after contrast material administration.

3- Inflammatory Lesions:

a) Effusion of petrous apex air cells:

Petrous apex air cells communicate variably with the middle ear and are susceptible to similar pathologic processes that affect the middle ear and mastoid air cells including inflammation, infection and obstruction.

Trapped fluid within petrous apex air cells is thought to be the sequela of previous otitis media, which fails to drain due to obstructed communicating channels. It often presents as in incidental imaging finding in patients with no otologic complaints.

In general, trapped effusion is a common incidental finding on MRI resulting in asymmetric signal of the petrous apex. T2-weighted images show hyperintense fluid signal within an otherwise normal-appearing, non expanded petrous apex. T1-weighted images typically show hypointensity related to simple fluid.

The T1 signal, however, can be intermediate or hyperintense depending on the protein content of the residual fluid. It is this feature more than any other that leads to potential confusion.
T1 hyperintensity can be seen in other lesions with high protein content including cholesterol granuloma and mucocele.

Additionally, intrinsic T1 hyperintensity might be mistaken for "enhancement" on contrast-enhanced images and may mimic neoplasm or petrous apicitis if careful comparison with unenhanced images is not made.

Trapped effusion should not show enhancement with gadolinium administration.

Temporal bone HRCT can be complementary to MRI in the diagnosis of trapped effusion and is recommended in equivocal cases.

Trapped effusion shows isolated opacification of the petrous apex without evidence of bony expansion, cortical disruption, or trabecular erosion.

b) Petrous apicitis:

Petrous apicitis is an infectious process caused by medial extension of acute otitis media into a pneumatized petrous apex. Subsequent obstruction of drainage from the petrous apex to the middle ear may result in formation of a purulent abscess. Streptococcus pneumoniae, Haemophilus influenzae, and Staphylococcus aureus are the most common causative organisms.

Patients with petrous apicitis usually present with an acute febrile illness and some or all of the symptoms of the classic Gradenigo triad (ear pain, palsy of the sixth cranial nerve, and facial pain).

Possible complications of petrous apicitis include meningitis, cerebral abscess formation, and venous sinus thrombosis.

The MR imaging findings of petrous apicitis are high signal intensity on T2-weighted images, low signal intensity on T1-weighted images, and contrast enhancement in a pneumatized anterior petrous apex. There may be associated enhancement of the adjacent dura mater and cranial nerves due to meningitis. Abscesses demonstrate ring enhancement and restricted diffusion on diffusion-weighted images.

CT demonstrates opacification of petrous air cells in the early stage of the disease and bone destruction in later stages.

c) Petrous Apex Osteomyelitis:

Skull base osteomyelitis involving the petrous apex is distinguished from petrous apicitis in that the former can occur in a non pneumatized petrous apex. It is usually caused by direct medial extension of necrotizing otitis externa or by retrograde spread of thrombophlebitis along the venous plexus of the petrous carotid canal.
Patients frequently have a predisposing condition such as diabetes mellitus, and Pseudomonas is by far the most common causative organism.

Early in the course of the disease, CT may show only soft tissue in the external auditory canal and loss of normal fat planes beneath the skull base.

In the later stages, there is bone erosion and fragmentation, bone sclerosis is also possible. MR imaging demonstrates soft-tissue replacement in the marrow spaces of the temporal bone and petrous apex and typically shows extension of the process into the adjacent soft tissues or intracranial involvement (Fig 9).

4- Vascular Lesions:

a) Petrous internal carotid artery aneurysm:

Aneurysms of the petrous segment of the ICA are rare, especially when compared with aneurysms of more distal intracranial segments.

Petrous ICA aneurysms are typically asymptomatic and may be incidentally discovered during workup for unrelated symptoms.

CT shows expansion of the carotid canal in the anterior petrous apex. The walls of the canal may be thin or even dehiscent, and the aneurysms may mimic cholesterol granulomas at unenhanced CT.

However, contrast-enhanced studies show marked enhancement in the lumen of a nonthrombosed aneurysm.

At MR imaging, mixed signal intensity or heterogeneous enhancement can be seen in the aneurysm lumen due to turbulent flow or mural thrombus. Endovascular interventions or surgical trapping procedures are considered for symptomatic patients.

b) Intraosseous Dural Arteriovenous Fistula:

Intraosseous dural arteriovenous fistulas are rare variants of dural arteriovenous fistulas. These lesions differ from the classic type of dural arteriovenous fistula in that the vascular nidus is situated nearly entirely within bone. They have been reported to occur in the clivus and petrous apex and are supplied predominantly by meningeal branches of the external carotid artery and ICA.

At CT, intraosseous dural arteriovenous fistulas appear as osteolytic lesions.

MR imaging demonstrates multiple intraosseous flow voids and contrast-enhanced images show serpentine enhancement in the affected diploic space, findings suggestive of intradiploic venous hypertension.
Time-of-flight MR angiography demonstrates flow-related enhancement within intraosseous vessels and a dilated intraosseous venous pouch. These lesions may be treated surgically or with endovascular occlusion.

5- Benign Tumours:

a) Meningioma:

Petroclival and cerebellopontine angle meningiomas are the most likely to involve the petrous apex.

Petroclival meningiomas originate from the medial aspect of the petrous apex and course over its wall or enter the Dorello canal. Cerebellopontine angle meningiomas arise from the dura mater along the posterior surface of the petrous apex and can extend into the IAC.

Meningiomas appear as dural-based masses that are typically slightly hyperattenuating to brain tissue on CT images, iso- to hypointense on T1-weighted images, and iso- to hyperintense on T2-weighted images.

They usually enhance avidly after contrast material administration. Meningiomas may cause hyperostosis of the petrous apex, a finding that is generally most evident at CT; at MR imaging, the hyperostotic bone has low signal intensity on T1- and T2-weighted images.

b) Paraganglioma:

Paragangliomas may invade the petrous apex via preformed air cell tracts from their sites of origin in the jugular foramen or middle ear.

Glomus tympanicum tumours (also known as paragangliomas of the middle ear) are highly vascular tumours that arise from paraganglia in the middle ear.

On CT images produced with a bone algorithm, paragangliomas show typical moth-eaten or permeative bone changes around the jugular foramen as a result of tumor infiltration through the haversian canal system.

At MR imaging, large paragangliomas may have a characteristic salt-and-pepper appearance on T1-weighted images.

The hypointense "pepper" represents high-velocity flow voids of feeding arterial branches in the tumor (also evident on T2-weighted images), while the less commonly seen hyperintense "salt" represents underlying foci of hemorrhage. These tumors usually enhance intensely at both CT and MR imaging after contrast material administration (Fig 4, 5).
C) Schwannoma:

Petrous apex schwannomas usually originate from the fifth, seventh, or eighth cranial nerves. At CT, schwannomas are usually isoattenuating to brain tissue and enhance after contrast medium administration.

At MR imaging, they generally appear as well-circumscribed, smoothly expansile masses that are iso- to hypointense on T1-weighted images and hyperintense on T2-weighted images, with enhancement on gadolinium-enhanced images (Fig6).

6) Malignant Tumours:

a) Chondrosarcoma:

Chondrosarcoma of the skull base is a rare neoplastic lesion with potentially lethal outcome.

HRCT and MR imaging play a crucial role in visualization.

CT of chondrosarcoma depicts a dense soft tissue structure with moderate enhancement after contrast material application. The number of calcifications which may appear in ring-like structures is variable.

MR imaging shows low signal intensity using T1-weighted sequences and high signal intensity on T2-weighted images. The tumour shows enhancement after gadolinium application.

Partly there can be depicted matrix mineralization causing signal heterogeneity.

b) Chordoma

Chordomas are rare locally invasive tumors arising from embryonic remnants of primitive notochordal elements.

CT reveals an expansile, lytic mass of the clivus that variably invades the sella, sphenoid sinus, and cavernous sinus.

Most authors argue that chordomas are more typically midline in location compared with chondrosarcomas.

CT often reveals scattered hyperdense foci representing residual bony sequestra or calcification within chordomas.

MRI better characterizes the soft-tissue invasive features associated with chordoma.
On MRI, chordomas can have variable signal on T1-weighted images including localized areas of hyperintensity due to hemorrhage or mucoid material.

Like chondrosarcomas, chordomas are characteristically hyperintense on T2-weighted sequences and tend to have well-defined margins (Fig 7).

T2 signal may be heterogeneous as well because of the presence of hemorrhage, calcification, residual bone fragments, or high proteinaceous mucus pool.

There is generally prominent but heterogeneous enhancement.

Chordomas often invade the cavernous sinus and encase the ICA without causing significant narrowing of the ICA.

As the tumor expands, it can extend exocranially into the nasopharynx or intracranially into the prepontine or cerebellopontine angle cisterns.

c) Metastasis:

As part of the skeletal system, the skull base is susceptible to hematogenously spread metastatic disease. Within the temporal bone, the petrous apex is the most commonly affected site.

Adenocarcinoma is the most common cell type and primary breast malignancy is the most common source. Other metastases occur in patients with lung, prostate, skin (melanoma), or kidney cancer.

If the lesion is solitary, differentiating the lesion from myeloma, chondrosarcoma, chordoma, invasive or intraosseous meningioma, or even petrous apicitis may be difficult.

CT findings in patients with hematogenous metastasis can be extremely variable. Metastatic disease can cause insidious infiltration of the marrow space with little, if any, change in the trabecular bone or cortex. More often, symptomatic tumor reveals a soft-tissue mass destroying cortical and cancellous bone of the apex, ranging in appearance from sclerotic to permeative to frankly lytic.

Unenhanced and contrast-enhanced fat-suppressed MR images reveal an infiltrating enhancing mass replacing normal marrow fat signal with or without extraosseous extension (Fig 8).

c) Endolymphatic sinus tumour:

An endolymphatic sac tumour is a rare locally aggressive papillary adenomatous neoplasm that originates from the epithelium of the endolymphatic sac and duct. Although
most endolymphatic sac tumors occur sporadically, there is a known association with von Hippel-Lindau syndrome.

Most commonly, it grows posteriorly to involve the cerebellopontine angle.

This tumor usually presents with symptoms of endolymphatic hydrops (gradual-onset, low-frequency hearing loss, tinnitus, fullness, and vertigo). Less commonly, patients present with acute hearing loss due to acute intralabyrinthine hemorrhage and associated inflammation.

On CT, an endolymphatic sac tumour presents as a soft mass with aggressive bone erosion in the retrolabyrinthine petrous area.

Internal amorphous calcifications are present in almost all cases.

Because of the high frequency of intratumoral hemorrhage, the lesion can show variable T1 hyperintensity.

On T2-weighted sequences, the lesion is generally hyperintense.

On contrast-enhanced images, there is intense nodular enhancement.

Because of its hypervascularity, this tumour, particularly large (> 2 cm) tumors, is frequently associated with flow voids.

For the same reason, this tumour shows tumour blush on angiography.

7- Osseous Dysplasias:

a) Fibrous Dysplasia:

Fibrous dysplasia is a mesenchymal disorder in which the normal process of converting immature woven bone to mature lamellar bone is disrupted. The result is a disorganized arrangement of bone trabeculae mixed with fibrous tissue and cysts.

CT is usually diagnostic and shows benign expansion of involved bones with relative preservation of cortical integrity and, frequently, a characteristic ground-glass internal matrix. Occasionally, fibrous dysplasia can manifest as a radiolucent bone lesion that mimics a more aggressive process.

At MR imaging, fibrous dysplasia can demonstrate alarming imaging features and can be mistaken for a malignant process. Typically, fibrous dysplasia appears as an expansile lesion with variable signal intensity and areas of low signal intensity on both T1- and T2-weighted images. Variable enhancement is seen after contrast material administration.

b) Paget Disease:
Paget disease is characterized by production of dense fragile bone formed by alternating episodes of excessive abnormal osteoclastic and osteoblastic activity. Unlike fibrous dysplasia, Paget disease typically spares the facial bones.

At CT, the early or osteolytic phase is characterized by bone lysis and demineralization, particularly in the petrous apex. In the intermediate or mixed phase, CT shows multiple mixed areas of lysis and sclerosis, which produce a mottled appearance. In the late sclerotic phase, this appearance progresses to the presence of thickened dense bone with an irregular cortical surface and poor corticomedullary differentiation. MR imaging shows heterogeneous signal intensity on T1- and T2-weighted images with heterogeneous enhancement in involved bone.
Fig. 3: Congenital cholesteatoma of left petrous apex in a 32-year-old woman who presented with left sided hearing loss. Axial CT scan shows a cholesteatoma surrounding the left labyrinth and extending to the jugular foramen.

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Fig. 4: Glomus tympanicum tumour in a 54-year-old woman with pulsatile tinnitus. (A) Axial CT image, (B) Axial T1-weighted image, (C) Axial T2-weighted image, (D) axial Gadolinium-enhanced T1-weighted image show an expansile lesion abutting the cochlear promontory and projecting in the middle ear cavity. This lesion is isointense on T1-weighted image, hyperintense on T2-weighted image and with contrast enhancement.

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Fig. 5: Glomus jugulare in a 56-year-old woman with peripheral facial paralysis. (A) Axial CT image in bone window and (B) soft-tissue window demonstrates left jugular foramen mass with irregular margins and lytic changes of surrounding temporal bone. MRI (C) Axial flair T2-weighted image (D) Axial and (E) coronal gadolinium-enhanced T1-weighted image enhancing mass within the right jugular foramen which expands and erodes the adjacent petrous bone and extends to the cerebello-pontine angle cistern and parapharyngeal space.

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**Fig. 6:** Fig 6 Vestibular schwannoma: 57 year old woman with recent onset left-sided hearing loss and slight occasional imbalance. MRI (A) Axial T2-weighted image and (B) axial Gadolinium-enhanced T1- weighted image show "ice cream cone" mass in the right cerebellopontine angle with homogenous avid enhancement post contrast.

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Fig. 7: Chordoma, 50-year-old man with right surdity: MRI (A) Axial T2-weighted images, (B) diffusion, (C) flair, and (D) axial contrast-enhanced fat-saturated T1-weighted images: Large heterogeneously enhanced mass centered arises from the clivus with posterior extension into the pontine cistern and laterally in the temporal lobe and the petrous apex.

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**Fig. 8:** Fig 8 Metastasis of breast cancer, 54 year old woman followed for breast cancer with and consults for headache and a right-sided hearing loss. MRI (A,B,C) Axial and (D) sagittal contrast-enhanced fat-saturated T1-weighted images show extra-axial, right petrous apex mass extending to the clivus and cerebello-pontine angle cistern and enhancing intensively after injection of contrast. It associates with diffuse enhancement leptomeninges from caused by leptomeningeal carcinomatosis.

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**Fig. 9:** Osteomyelitis of the petrous apex and skull base in a patient with necrotizing otitis externa. (A, B) Axial CT image in bone window, MRI (C) Axial T1-weighted image (D) Axial T2-weighted image (E) axial and (F) coronal Gadolinium-enhanced fat-suppressed T1-weighted image show an infiltrative process involving the central skull base, temporo-mandibular, the clivus and left petrous apex. There is fluid in the left mastoid. Corresponding gadolinium-enhanced fat-suppressed T1-weighted MR image shows intense enhancement of the left petrous apex and clivus as well as of the soft tissues of the nasopharynx and left masticator space.

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Conclusion

Radiologists interpreting images of this complex region provide expertise that is indispensable for patient care. For this reason, they should have a firm understanding of the anatomy of the petrous apex and the myriad disease processes that can occur there.
References

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