Objective

The petrous apex is a pyramidal shaped, variably pneumatized structure of the skull base that forms a unique intersection between the supracyoid neck and the intracranial compartment. Given its location, the petrous apex is susceptible to multiple pathologic processes including intrinsic lesions of bone, pneumatized air cells, or the petrous internal carotid artery; invasive “downgoing” intracranial processes; or invasive “upgoing” infiltrating nasopharyngeal or sinonasal lesions. Clinical presentations of these lesions, therefore, can be quite variable and depend largely on involvement of numerous intimately adjacent intra- and extracranial structures, especially the cranial nerves. Given this variability, petrous apex lesions cannot be diagnosed accurately on the basis of clinical findings alone. Fortunately, many of these lesions have characteristic MRI and CT appearances that can often allow a precise diagnosis. The purpose of this pictorial review is to emphasize these unique imaging features in association with focused clinical presentations and brief management outlines.

Conclusion

This article concisely reviews the anatomic architecture of the petrous apex including its critical relationships with adjacent structures. A variety of pathologic processes affecting the petrous apex are presented and discussed including the epidemiology, clinical presentations, imaging appearances, and management. On completion of this pictorial review and the associated self-assessment module, the reader will have acquired knowledge to reliably use MRI and CT to diagnose common petrous apex lesions.

Introduction

The petrous apex is the pyramidal, medial projection of the petrous portion of the temporal bone. The normal petrous apex is relatively simple in form with only one principal variation: the degree of pneumatization. That is, the apex may be variably pneumatized with aerated connections to the middle ear or may contain predominantly marrow fat.

What makes the petrous apex anatomically complex is its medial location in the skull base and its intimate relationship to other clinically important structures including the cavernous sinus, Dorello canal, and Meckel cave. The petrous apex represents a unique intersection between the supracyoid neck and the intracranial compartment. Hence, the petrous apex is susceptible to a variety of pathologic processes, including intrinsic lesions of bone, pneumatized air cells, or the petrous internal carotid artery (ICA); intracranial processes with inferior extension; or superiorly invasive nasopharyngeal or sinonasal lesions.

Clinical presentations of these lesions, therefore, can be quite variable and depend largely on involvement of numerous intimately adjacent intra- and extracranial structures, especially the cranial nerves. Given this variability, petrous apex lesions cannot be diagnosed accurately on the basis of clinical findings alone. Fortunately, many of these lesions have characteristic MRI and CT appearances that can often allow a precise diagnosis.

The primary goal of this article is to emphasize these characteristic imaging appearances in association with suggestive clinical presentations. Representative images will further highlight the important concepts of a particular lesion. There is brief discussion of lesion management as well.

Anatomy and Critical Relationships to Other Structures

The petrous apex of the temporal bone is located anteromedial to the inner ear within the angle created by the greater wing of the sphenoid bone anteriorly and the occipital bone posteriorly. The anterior margin of the petrous apex forms the medial posterior wall of the middle cranial fossa. The most inferior and medial exocranial margin of the petrous apex is separated from the clivus by an ovoid horizontal gap, the foramen lacerum, which contains a bridge of dense fibrous tissue and cartilage (Fig. 1). Above the foramen lacerum, the ICA exits the medial opening of the carotid canal on its way to the cavernous sinus.

The most superior and medial endocranial surface of the petrous apex contains an important landmark, a shallow depression called the “trigeminal impression” on which lies the trigeminal ganglion within Meckel cave. This close rela-
Petrous Apex Lesions

Just medial to the trigeminal impression, the tip of the petrous apex gives rise to a discrete fibrous tissue bundle that crosses medially across the petroclival fissure, within the meningeal and periosteal dural layers, to the base of the ipsilateral posterior clinoid process. This bundle is termed the “petrosphenoidal ligament” or “Gruber’s ligament.” Although not typically identified with routine imaging, this structure is important because it demarcates the superior boundary of a dural invagination called “Dorello canal.”

Dorello canal is an anatomic channel from the dural margin along the petroclival junction to the posterior cavernous sinus and contains the abducens nerve (sixth cranial nerve) along with portions of the basilar plexus and inferior petrosal sinus. Lesions of the petrous apex or petroclival junction are notorious for invading or compressing the Dorello canal and causing sixth cranial nerve palsy and diplopia. High-resolution T2-weighted MRI sequences of the skull base can identify the sixth cranial nerve and associated dural sleeve within the proximal aspect of the Dorello canal.

The inferior exocranial surface of the petrous apex is intimately related to the nasopharynx. The petrous apex is connected medially to the clivus by dense fibrocartilaginous tissue of the foramen lacerum that merges with the cartilaginous portion of the eustachian tube. Invasive neoplasms of the nasopharynx, thus, can traverse the sinus of Morgagni and readily gain access to the bony skull base including the petrous apex.

Petrous Apex Pneumatization

In general, the petrous apex is composed of dense bone and bone marrow. Pneumatization of the petrous apex occurs when epithelium-lined air cells develop as medial communications from the mastoid air cells. This occurs in 9–30% of patients, and in general there is a positive correlation between the degree of mastoid segment pneumatization and aeration of the petrous apex [1]. 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.

Of the pneumatized petrous bones, 4–7% are asymmetrical [1]. This asymmetry can have two important effects. First, it can create an asymmetric appearance during

---

Fig. 1—Three-dimensional anatomy of exocranial surface of petrous apex of healthy 27-year-old woman. Image shows exocranial surface of petrous apex (1); right foramen ovale (2); foramen lacerum (3), the ovoid gap between petrous apex and clivus (bracket). Carotid canal opening (4), through which internal carotid enters into petrous carotid canal, and right jugular foramen (5) are also depicted.

Fig. 2—Three-dimensional anatomy of endocranial surface of petrous apex of healthy 27-year-old woman that includes schematic representation of its relationship with important adjacent skull base structures. Image shows 3D reformation of high-resolution CT of normal skull base. On left side, important skull base structures are shown schematically in relation with left petrous apex. Segment 3 (lacerum segment) of left internal carotid artery (1) immediately beyond intrapetrous course and course of left trigeminal nerve (2) are shown. Trigeminal nerve trunk approaches toward petrous apex with gradual expansion as it forms trigeminal ganglia at Meckel cave immediately above petrous apex. There are three branches from trigeminal ganglion, V1 and V2 (medial two branches) and V3 (lateral branch). Slightly lateral course of left abducens nerve (3) toward left petrous apex through Dorello canal, the small space located between petrous apex and petrosphenoidal ligament (4) are identified.

AJR:196, March 2011
Asymmetric Fatty Marrow in the Petrous Apex

Asymmetric pneumatization is related to another normal variation: asymmetric fatty marrow within the petrous apex. This finding is a common incidental finding on brain, skull base, and soft-tissue neck MRI studies obtained for evaluation of nonotologic complaints. 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 (Fig. 3). Fat density can also be confirmed on high-resolution CT (HRCT).

Diagnostic difficulty occurs in two situations. First, asymmetric fatty infiltration of the apex may be observed as conspicuous asymmetric high signal on contrast-enhanced images and could be mistaken for an enhancing pathologic lesion. Correlation with unenhanced T1-weighted images and use of fat-saturation techniques are necessary to avoid this pitfall. Second, other lesions with T1 shortening (i.e., bright T1 signal) such as cholesterol granuloma might be casually disregarded as normal marrow if fat-saturation techniques are not used. No follow-up imaging is necessary [1].

Bony defects: cephaloceles—Petrous apex cephaloceles are relatively uncommon. Grossly, petrous apex cephaloceles represent cystic expansion and herniation of the posterosilateral portion of Meckel cave into the superomedial aspect of the petrous apex. As such, they are CSF-filled structures lined by dura and arachnoid of variable thickness and may contain some prolapsed fibers of the trigeminal nerve. Unlike cephaloceles of other areas of the base of the skull, petrous apex cephaloceles do not contain any brain tissue. Although these lesions have been described variably in the literature as “meningoceles” [2] or “herniation of Meckel cave” [3], the most popular term is “petrous apex cephalocele.” The exact cause is not known, but petrous apex cephaloceles likely represent developmental dehiscence occurring gradually when chronic CSF pulsation combines with congenitally thin bone along the roof of the petrous apex [3].

Petrous apex cephaloceles are generally considered incidental, “don’t-touch” lesions [3]. There are rare case reports, however, in which lesions were associated with symptoms of fifth cranial nerve dysfunction, otorrhea, recurrent meningitis, or pulsatile tinnitus [4]. Surgical intervention has been reported in some cases, but most instances were reported before widespread recognition of the lesion and its key imaging features. Most petrous apex cephaloceles are unilateral but can be bilateral as well [3]. Occasionally, bilateral lesions can be seen in association with diffuse skull base thinning related to chronic elevation of intracranial pressure, as in pseudotumor cerebri.

HRCT of the skull base using a bone algorithm shows smoothly margined, lobulated, cystic expansion of the petrous apex. MRI likewise shows a lobulated nonenhancing cystic lesion that follows CSF signal on all MRI sequences. A coronal high-resolution T2-weighted sequence is the best sequence to show superomedial communication with Meckel cave (Fig. 4). In equivocal cases, CT cisternography may be performed to show the communication with Meckel cave. However, the sensitivity of CT cisternography for petrous apex cephalocele is not high.

Vascular lesions: 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 segment aneurysms are thought to originate from congenitally weak areas in the arterial wall at the origin of several embryonic vessels [5, 6].

Petrous ICA aneurysms are typically asymptomatic and may be incidentally discovered during workup for unrelated symptoms. They may be surprisingly large at initial diagno-

Fig. 3—Asymmetric fatty marrow of left petrous apex in 45-year-old woman being evaluated for multiple sclerosis. A, Unenhanced non–fat-saturated T1-weighted image reveals smoothly margined T1 hyperintense lesion in left petrous apex. There is intermediate signal on non–fat-suppressed T2-weighted sequence (images not shown). B, However, fat-saturated T2-weighted image shows intermediate T2 signal (arrow) is completely reversed, thus confirming fat content of left petrous apex. Cholesterol granuloma, a common close differential diagnosis, also has high T1 signal but signal of cholesterol granuloma does not reverse with fat-suppression technique. On CT image (not shown), there was nonpneumatization of left petrous apex without any evidence of bone remodeling.
sis. Occasionally, such aneurysms may present with headache, diplopia, Horner syndrome, or pulsatile tinnitus.

On bone CT, there is focal or fusiform dilatation of the bony carotid canal. The margins may be smooth and scalloped or may show peripheral irregularity related to bony erosion. Nonuniform calcification in the aneurysm wall or within peripheral plaque may contribute to the irregularity and may suggest an invasive mass. CT angiography (CTA) is the best overall study for diagnosing petrous ICA aneurysm, typically showing confluent enhancement of the aneurysm lumen during arterial bolus injection (Fig. 5). CTA can readily evaluate the degree of intraluminal thrombus as well.

The MRI appearance of a petrous ICA aneurysm can be quite variable. The signal characteristics are influenced by multiple factors including the size of the aneurysm, the degree of thrombus, and complex flow dynamics within the aneurysm. On both T1- and T2-weighted images, the aneurysm can appear as a complex or mixed-signal mass with central hypointensity related to the patent lumen. Flow voids may be layered or arranged in a “swirl” pattern. After gadolinium contrast administration, the aneurysm can appear as an irregular enhancing mass [5] (Fig. 5). MR angiography (MRA) can be diagnostic but may not reliably distinguish flow-related signal within the lumen from T1 hyperintense signal within mural thrombus. Motion artifact from flow within the patent lumen of aneurysms is an important clue to the diagnosis and is found most commonly (56%) on contrast-enhanced T1-weighted sequences [7].
Asymptomatic small aneurysms may require no treat-
ment and can be safely followed up by serial imaging [6].
Symptomatic aneurysms or progressively enlarging aneu-
rysms should be treated [6].

Inflammatory Lesions

Effusion of petrous apex air cells—Petrus apex air cells
communicate variably with the middle ear and are suscep-
tible to similar pathologic processes that affect the middle
ear and mastoid air cells including inflammation, infection,
and obstruction. Opacification of these air cells can occur
in conjunction with otitis media and can persist despite reso-
lution of middle ear disease and of clinical symptoms due
to obstructed drainage related to adhesive fibrosis along
communicating air channels [1]. Such persistent fluid in the
apex has been termed “trapped effusion” and appears to
have no further clinical relevance. Pathologic and microbi-
ologic evaluations have shown the fluid to be nonpurulent in
nature and to contain no microorganisms.

Trapped effusion of the petrous apex rarely creates a radi-
ologic diagnostic dilemma. In general, trapped effusion is a
common incidental finding on MRI resulting in asymmetric
signal of the petrous apex. T2-weighted images show hyperin-
tense fluid signal within an otherwise normal-appearing, non-
expanded petrous apex (Fig. 6A). T1-weighted images typi-
cally 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 hyperin-
tensity can be seen in other lesions with high protein content
including cholesterol granuloma and mucocele. Additionally,
intrinsic T1 hyperintensity might be mistaken for “enhance-
ment” on contrast-enhanced images and may mimic neoplasm
or petrous apicitis if careful comparison with unenhanced im-
ages is not made. Trapped effusion should not show enhance-
ment with gadolinium administration.

Temporal bone HRCT can be complementary to MRI in the
diagnosis of trapped effusion and is recommended in equivocal
cases (Fig. 6B). Trapped effusion shows isolated opacification
of the petrous apex without evidence of bony expansion, cor-
tical disruption, or trabecular erosion. The cortical margins of
the native petrous apex air cells are easily discerned despite
opacification. These observations are typically straightforward.
Occasionally, the question arises regarding the possibility of
(and subjective perception of) “subtle” or “early” expansion
or cortical thinning of the petrous apex. Anecdotally, if
one cannot decide if the petrous apex is “expanded,” it prob-
ably isn’t. Otherwise, lingering concerns should be mitigated
by the absence of localized clinical findings, such as ipsilateral
sixth cranial nerve palsy, and by follow-up examinations to
document stability.

Mucocele—A mucocele technically is an obstructed air

cell containing respiratory epithelium that maintains its
ability to secrete mucus. The continued production and
accumulation of mucoid material within the obstructed air

cell, along with the associated inflammatory response, lead
to expansion, remodeling, and ultimately permissive ero-
sion or destruction of the bony margins.

A primary mucocele developing within a pneumatized pe-
trous apex is extremely rare. An expanding mucocele in this
location may cause localized pain or ipsilateral cranial neu-
ropathy. As the petrous apex mucocele enlarges, it erodes
bony septations and coalesces with adjacent air cells. Temporal
bone CT shows opacification of petrous apex air cells with
associated expansion of the cortical margins. MRI signal can
be complex, with hyperintense T2 signal and variable T1 sig-
nal dependent on protein content and the degree of inspissa-
ation of the mucoid material. There is no central enhance-
ment, but peripheral enhancement can occur secondary to
inflammatory response. Mucoceles typically do not show dif-
fusion restriction. The differential would include choleste-
toma; cholesterol granuloma; trapped effusion; and, if suffi-
ciently complex, a petrous ICA aneurysm.

Cholesterol granuloma—Cholesterol granulomas of the
temporal bone can occur in the mastoid segment, the mid-
dle ear, and the petrous apex. In fact, cholesterol granulo-

Fig. 7—Right petrous apex cholesterol granuloma in
34-year-old man who presented with hearing loss.
A, On unenhanced T1-weighted image, there is
significant remodeling of right petrous apex. Lesion
is heterogeneously hypointense on unenhanced
T1-weighted sequence because of extracellular
methemoglobin.
B, On fat-suppressed T2-weighted image, there is
persistent hyperintensity at center of lesion. Dark
peripheral rim (arrows) on medial aspect of lesion is
due to peripheral hemosiderin. Of note is that bright
T1 signal from fat at left petrous apex of image A,
is completely saturated in this fat-suppressed T2-
weighted sequence.
mas are the most common primary petrous apex lesions [1, 8]. The exact mechanisms that initiate formation and allow perpetuation of the cholesterol granuloma are not clearly understood. The prevailing hypothesis suggests an initial obstruction to an air cell; development of a vacuum phenomenon; and repeated cycles of hemorrhage, inflammatory response, and gradual expansion due to bone remodeling and resorption [5]. The advancing margin represents a fibrous capsule without true epithelial lining.

The resulting expansile mass 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. Patients may present with a variety of symptoms caused by mass effect and compression. The most common symptom is hearing loss, followed by vertigo and headache [9]. Other less common manifestations are tinnitus, otalgia, diplopia, trigeminal neuralgia, or facial spasm [10]. On otoscopy, it may appear as a “blue lesion” [11].

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 (Fig. 7). 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 [10].

Given the extensive use of neuroimaging for brain, sinus, and neck pathology, small cholesterol granulomas of the petrous apex can occasionally be discovered in the absence of associated symptoms. Serial follow-up examinations are recommended in these patients because lesions can remain stable for long periods of time. Otherwise, symptomatic or progressively enlarging lesions should be treated with surgi-

Fig. 8—Congenital cholesteatoma of left petrous apex in 32-year-old man who presented with left-sided hearing loss.
A, On T2-weighted image, there is expansion of left petrous apex and lesion appears very bright.
B, Diffusion-weighted image shows characteristic diffusion restriction associated with low apparent diffusion coefficient (ADC) on ADC map (not shown).

Fig. 9—Left petrous apicitis in 27-year-old man who presented with history of left middle ear infection, hearing loss, and sixth cranial nerve palsy.
A, On T2-weighted image, there is heterogeneously hyperintense signal at left petrous apex associated with expansion of bone. Note fluid collection on left mastoid and middle ear cavity.
B, On contrast-enhanced T1-weighted image, there is heterogeneous enhancement of left petrous apex. Posterior aspect of lesion enhances intensely. Also note subtle enhancement of left mastoid and middle ear cavity.
Chapman et al.

Surgical intervention with the intent to drain and aerate [10]. Multiple surgical approaches have been used including transphenoidal endoscopic drainage.

Cholesteatoma—Cholesteatomas of the petrous apex are synonymous with epidermoid cysts. These lesions are considered to be congenital in origin and are ectopic rests (or abnormal persistence) of embryologic epithelial tissue in the petrous apex. This tissue forms a cyst lined by stratified squamous epithelium. As internal desquamation occurs from the lining, keratinized debris accumulates centrally with a highly organized structure. Enlargement of the cholesteatoma occurs gradually as the advancing epithelium combined with host inflammatory response results in surrounding bone resorption. Congenital cholesteatomas are slow-growing lesions and may be asymptomatic for years. Common clinical symptoms are caused by progressive mass effect and include hearing loss, cranial nerve palsies according to location, and headache. Hearing loss is sensorineuronal and due to involvement of the retrocochlear auditory apparatus.

On temporal bone CT, there is a smooth expansile lesion of the petrous apex [11]. The central portions 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 (Fig. 8A). Diffusion restriction is reported to be characteristic of this lesion (Fig. 8B). After contrast administration, there may be subtle peripheral rim enhancement.

Surgical excision or exteriorization is the treatment of choice [5].

Apical petrositis—Overt infection of the petrous apex (i.e., apical petrositis) is a relatively rare complication that occurs when infectious otomastoiditis extends medially into the petrous apex usually via pneumatized air cells. Initially, intact petrous apex air cells are opacified with purulent exudate. With progressive infection, the epithelium is invaded and destroyed, and the supporting bony trabeculae and inner cortical margins undergo demineralization and resorption. Infection then spreads beyond the air cells to the adjacent marrow space of the petrous apex, essentially forming a localized osteomyelitis of the skull base.

At this point, infection can spread directly to the outer cortical margins of the petrous apex and cause permeative and destructive changes. This process can also involve the cortical margins of the carotid canal and can lead to vasospasm or arteritis of the ICA. Infectious exudate can also invade and traverse multiple intraosseous veins that communicate directly with the dural sinuses and veins of the skull base, resulting in thrombophlebitis and sinus thrombosis. Ul-

![Fig. 10](image-url) — Left petrous apex chondrosarcoma in 71-year-old man who presented with deep headache and sixth cranial nerve palsy.
A, On high-resolution CT scan of skull base, there is expansion and permeative change of left petrous apex associated with trabecular destruction. Note that lesion is off midline, and note lack of intratumoral calcification or residual bony fragment.
B, On T2-weighted image, lesion has characteristic T2 hyperintensity; bony expansion is also evident.
C, Lesion enhances heterogeneously on contrast-enhanced T1-weighted image.
Petrous Apex Lesions

...ultimately, meningitis, encephalitis, and intracranial abscess can develop.

As expected, symptoms can be highly variable and depend on the stage of disease. Most patients present with severe otalgia and otorrhea with associated deep facial or retroorbital pain. Occasionally, patients present with the classic Gradenigo triad: otomastoiditis, deep facial pain secondary to trigeminal neuropathy, and lateral rectus palsy and diplopia secondary to sixth cranial nerve palsy. The classic triad, although more often the exception than the rule, is explained by the unique relationship of the petrous apex to Meckel cave and Dorello canal [5]. Please see the earlier anatomic discussion.

In children, petrous apicitis may occur as an acute primary process, whereas in adults petrous apicitis often occurs in the setting of chronic otomastoiditis or recent mastoid surgery. The most common offending organisms are considered to be the same as those that cause otomastoiditis, but culturing and identifying a specific organism are difficult given the previous use of antibiotics in most of these patients [5]. Historically, such an invasive infection of the skull base was considered to nearly always be fatal. With marked improvements in diagnosis and antibiotic therapy, however, most patients survive without significant deficit.

Fig. 11—Chordoma of left petrous apex and clivus in 33-year-old woman who presented with deep headache and diplopia.

A, T2-weighted image shows expansile heterogeneously T2 hyperintense lesion (arrowhead) of left petrous apex that has encroached into clivus. Round T2 hyperintense lesion at left pons is due to partial volume artifact from large posterior fossa component of lesion (not shown). Also note congestion of left mastoid (arrow).

B, On contrast-enhanced T1-weighted sequence, there is intense enhancement of lesion. Note small ringlike enhancement of part of large posterior fossa component of tumor.

Fig. 12—Left petrous apex metastasis from breast cancer in a 50-year-old woman being evaluated for bony metastasis from known breast cancer. On fused PET/CT scan, there is moderate-to-intense uptake of FDG in left petrous apex lytic lesion.

Fig. 13—Paraganglioma of left petrous region in 54-year-old woman who presented with pulsatile tinnitus and hoarseness.

A, On contrast-enhanced T1-weighted image, there is brightly enhancing lesion in left petrous bone associated with multiple intratumoral flow voids due to high vascularity of tumor. Lesion arises from left jugular foramen (not shown) and secondarily involves petrous apex.

B, On T2-weighted image, lesion is isointense to cerebellum. Salt-and-pepper appearance of tumor is due to prominent intratumoral vascular flow voids.
Temporal bone CT shows opacification of preexisting apical air cells in conjunction with otomastoiditis. There is typically mild expansion of the petrous apex. The hallmark feature is permeative destruction of the cortical and cancellous bone in this region. It should be remembered, however, that detection of bone loss occurs relatively late, requiring 30–50% demineralization before lysis is evident on CT. Early in the infectious process, bone changes may be subtle or may even be absent despite severe symptoms. If contrast-enhanced CT is performed, petrous apicitis can appear as a heterogeneously enhancing infiltrating mass and may mimic neoplasm.

MRI is complementary to CT in the diagnosis of apical petrositis and provides better visualization of the intimately associated soft tissues. Fluid of variable complexity is noted in the petrous apex and middle ear. After contrast administration, the opacified, irregular air cells of the petrous apex may show heterogeneous enhancement related to phlegmon or an irregular coalescent fluid collection with peripheral enhancement (Fig. 9). Enhancement and thickening of the adjacent dura can occur. Inferiorly, infection and edema can spread to the levator palatine muscle and adjacent tissues of the nasopharynx. Other complications including cavernous sinus thrombosis, leptomenigitis, cranial neuritis, and intracranial abscesses are best detected with MRI. If an abscess has formed, it is well visualized on diffusion-weighted imaging [12]. Gallium-67 SPECT is complementary to CT scan and MRI. At times, it increases the sensitivity of skull base lesion and is particularly helpful to monitor treatment response.

**Tumors**

*Chondrosarcoma*—Chondrosarcomas of the skull base are slow-growing, moderately to well-differentiated neoplasms that typically originate off the midline from cartilaginous remnants of the petrooccipital fissure. However, up to 28% of cases, it may arise from the clivus [13]. Chondrosarcomas are lobulated tumors that invade locally by advancing medially to involve the clivus and superiorly to involve the cavernous sinus. These tumors also have a propensity to extend laterally and inferiorly to involve the petrous apex and the foramen lacerum. This feature often results in broad contact with and effacement of the petrous ICA. Clinical presentation depends on the overall size of the lesion and associated cranial nerve involvement. Clinical presentations vary from nonspecific headache or craniofacial pain to specific cranial neuropathies, especially abduces (sixth cranial nerve) palsy.

Thin-section CT reconstructed with a bone algorithm is best for assessing the effects of a tumor on the central skull base, evaluating the potential foraminal involvement, and assessing the internal matrix. CT generally shows an expansile lobulated mass that expands and destroys the petrous apex with lesser involvement of the basisphenoid (Fig. 10A). The transition zone with adjacent normal bone is relatively narrow and may be permeative or smooth but typically is not sclerotic. The internal architecture or matrix of chondrosarcoma is variable and ultimately depends on the amount of chondroid tissue present. Classically, chondroid matrix calcification results in the so-called “arcs and whorls,” which are variably oriented rounded or curvilinear calcifications. This appearance is seen in approximately 50% of cases [14]. The remaining lesions may contain nondescript calcifications or no calcification at all.
MRI can best evaluate the soft-tissue characteristics of a petroccipital fissure chondrosarcoma. The mass may be rounded or may contain multidirectional lobular extensions resulting in a “cauliflower” shape. Often the main axis of the tumor parallels the longitudinal axis of the petrous apex. Chondrosarcomas are of hypo- or intermediate intensity on T1 sequences and are characteristically hyperintense on T2-weighted sequences (Fig. 10B). Intrinsic calcification may produce foci of hypointensity within the mass. These tumors generally show significant enhancement that may be heterogeneous or uniform (Fig. 10C).

The mainstay of treatment of chondrosarcoma is surgical resection followed by radiation therapy.

Chordoma—Chordomas are rare locally invasive tumors arising from embryonic remnants of primitive notochordal elements [15]. Chordomas of the skull base constitute one third of all such tumors and arise most frequently from the sphenoid synchondrosis of the clivus. Chordomas grow gradually and can enlarge dramatically within the clivus before producing significant symptoms. Like many other lesions in the central skull base, chordomas most often present with nonspecific craniofacial pain or focal cranial nerve deficit. Diplopia secondary to sixth cranial nerve palsy is common.

CT reveals an expansile, lytic mass of the clivus that variably invades the sella, sphenoid sinus, and cavernous sinus [15]. Most authors argue that chordomas are more typically midline in location compared with chondrosarcomas. Petrous apex involvement would then occur secondarily as the midline tumor invades superiorly and laterally. Other reports, however, question the usefulness of this distinction and document significant overlap in the appearances and locations of chordomas and chondrosarcomas [16]. CT often reveals scattered hyperdense foci representing residual bony sequestrum 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. This hyperintensity has been ascribed to the presence of characteristic vacuolated (“physaliphorous”) tumor cells that contain intracytoplasmic mucous droplets. T2 signal may be heterogeneous as well because of the presence of hemorrhage, calcification, residual bone fragments, or high proteinaceous mucus pool [15] (Fig. 11A). There is generally prominent but heterogeneous enhancement (Fig. 11B). 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. As with CT, it may be difficult to distinguish chondrosarcomas from chordomas of the central skull base with MRI.

Complete surgical treatment is the treatment of choice with or without additional radiation therapy for chordoma.

Hematogenous 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 and is involved in 80% of reported cases [5]. Adenocarcinoma is the most common cell type and primary breast malignancy is the most common source [5]. Other metastases occur in patients with lung, prostate, skin (melanoma), or kidney cancer. Metastatic disease to the petrous apex often occurs late in the course of malignant disease and may be “incidental” and asymptomatic in a patient with evidence of multifocal skeletal involvement. In these cases, the diagnosis is seldom in doubt. Rarely, petrous apex involvement may cause presenting symptoms of headache, sixth cranial nerve palsy, or hearing loss in a patient without known cancer. Careful search of the initial CT or MRI examination is necessary to identify or exclude additional marrow lesions of the skull base, calvaria, upper cervical spine, or facial bones. 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.

On PET, FDG uptake is variable but can be significant. Uptake in the skull base may be partially obscured by physiologic uptake in adjacent brain and may escape detection. Surgical intervention is rarely indicated for metastatic disease in the petrous apex but is occasionally undertaken when tissue diagnosis is necessary (Fig. 12).

Plasmacytoma and multiple myeloma—Plasmacytoma is a solitary tumor of neoplastic monoclonal plasma cells in either soft tissue (extramedullary) or in bone occurring in the absence of a clinical diagnosis of multiple myeloma. Plasmacytoma is generally regarded as benign unless multiple myeloma develops. Multiple myeloma represents a malignant multifocal proliferation of plasma cells with established clinical, radiologic, and laboratory criteria for diagnosis. Plasma cell tumors of the petrous apex may represent solitary plasmacytomas or myelomatous lesions in the setting of multiple myeloma. Both lesions have a similar radiologic appearance. CT shows an expansile, intraosseous soft-tissue mass with lytic destruction of the petrous apex. On MRI, lesions are generally isointense on T1 and iso- to hyperintense on T2 (compared with gray
cells and results in mixed permeative and sclerotic changes in the marrow space and any pneumatized air containing calvaria may be observed with close inspection.

Paraganglioma—The inferior and lateral aspect of the petrous portion of the temporal bone contains the external opening of the carotid canal as well as the jugular fossa, which contains the jugular bulb. Occasionally, paragangliomas (i.e., glomus jugulare or jugulotympanic tumors) of the jugular fossa can erode the medial margin of the fossa and invade the occipital bone and adjacent petrous apex. Paragangliomas can occur sporadically or as part of a familial syndrome. Up to 40% of head and neck paragangliomas are familial and tend to present earlier than the sporadic type and are more commonly multicentric [18]. The most common clinical presentation of jugular foramen paraganglioma is hoarseness due to vagal neuropathy followed by pulsatile tinnitus with or without hearing loss [19]. Only 3% of all head-neck paragangliomas secrete cat echolamines. Jugulotympanic paragangliomas are usually benign; only 5% are malignant [18].

On CT, a paraganglioma appears as ahypervascular enhancing soft-tissue mass that enlarges and erodes the jugular bulb with peripheral permeative changes of the petrous temporal bone and occipital bone. On MRI, paraganglioma shows marked enhancement after contrast administration and may show intraparenchymal flow voids [20] (Fig. 13A). The signal changes can be heterogeneous on T1- and T2-weighted images (Fig. 13B). The so-called “salt and pepper” appearance has been applied to the heterogeneous appearance due to markedly prominent intratumoral vascularity on multiple sequences. There may be avid uptake of FDG on FDG PET scan, and the lesion may be confused with more aggressive or malignant lesions including metastatic disease.

The best imaging clues to the diagnosis of paraganglioma are location at the jugular foramen, hypervascularity, and identification of additional paragangliomas in the head and neck. Conventional angiography can be useful to document the typical hypervascular appearance of paraganglioma but is performed most often in conjunction with preoperative embolization.

Meningioma—Meningiomas can affect the petrous apex in four principal patterns. First, given the variable appearance of the apex, the thin cortical shell, and the effects of volume averaging, petroclival meningiomas can often appear on MRI and CT to invade the apex proper when, in fact, they do not. Second, petroclival meningiomas can result in hyperostosis of the apex, with bony sclerosis and expansion. Third, meningiomas along the apex can directly invade the underlying cortical and trabecular bone. The invasive soft-tissue component obliterates the marrow space and any pneumatised air cells and results in mixed permeative and sclerotic changes in the apex. Fourth, a primary intraosseous meningioma can rarely originate within the petrous apex, mimicking a primary or metastatic bone tumor. Obviously, then, the CT and MRI appearances will vary considerably according to the type of bone involvement and degree of tumor invasion. The best clue to the diagnosis in most cases is identifying a dural-based extraosseous soft-tissue component that otherwise has features suggesting a meningioma such as CT hyperdensity, homogeneous enhancement, and a dural tail shown on MRI [5] (Fig. 14).

Endolymphatic sinus tumor—An endolymphatic sac tumor 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 [21]. This is a slow-growing tumor with four principal growth patterns. Most commonly, it grows posteriorly to involve the cerebellopontine angle. Less commonly, the tumor can grow laterally to involve the middle or external ear through the mastoid air cells, superiorly to the middle cranial fossa, and finally along the petrous ridge to involve the clivus and other central skull base structures [22]. 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 tumor presents as a soft mass with aggressive bone erosion in the retrolabyrinthine petrous area. Internal amorphous calcifications are present in almost all cases [23]. Because of the high frequency of intratumoral hemorrhage, the lesion can show variable T1 hyperintensity. On T2-weighted sequences, the lesion is generally hyperintense (Fig. 15A). On contrast-enhanced images, there is intense nodular enhancement (Fig. 15B). Because of its hypervascularity, this tumor, particularly large (> 2 cm) tumors, is frequently associated with flow voids [23]. For the same reason, this tumor shows tumor blush on angiography.

The treatment of choice for endolymphatic sac tumor is complete surgical excision with a wide surgical margin.

Conclusion

This article succinctly reviews the complex anatomy of the petrous apex and its relationships to adjacent structures of the skull base to provide a complete understanding of the anatomic bases of different clinical presentations of petrous apex lesions. Classic imaging appearances of the most common petrous apex abnormalities are emphasized in this article in addition to the necessary concise comments on demographics, clinical presentation, and management. This review article and the associated self-assessment module provide the information necessary to confidently diagnose common petrous apex abnormalities.
Petrous Apex Lesions

References


FOR YOUR INFORMATION

The reader’s attention is directed to the Self-Assessment Module for this article, which appears on page S40.