Lesions of the Petrous Apex: Classification and Findings at CT and MR Imaging

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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 sequelae, which typically result from mass effect or direct invasion of the cranial nerves, brainstem, or internal carotid artery. Because the petrous apex is not amenable to direct examination, cross-sectional imaging with computed tomography and magnetic resonance (MR) imaging plays an important role in diagnosis and characterization of lesions occurring there. Petrous apex lesions can be classified on the basis of their origin into the following categories: developmental lesions, inflammatory lesions, benign tumors, malignant tumors, vascular lesions, and osseous dysplasias. The most common lesions arising in the petrous apex are cholesterol granulomas, which can be reliably diagnosed with MR imaging due to their high signal intensity on both T1-weighted images and T2-weighted images. In addition, one should also be familiar with anatomic variants or pseudolesions in the petrous apex that can mimic pathologic processes.

Introduction

The apex of the petrous bone lies in a complex anatomic region that contains a number of critical neural and vascular structures. Therefore, lesions arising in or spreading to the petrous apex cause varied and occasionally severe clinical sequelae, which typically are the result of mass effect or direct invasion of the cranial nerves, brainstem, or internal carotid artery (ICA). Because the petrous apex cannot be directly visualized, imaging plays a crucial role in evaluation of lesions located there and is often critical for patient care, as treatment approaches depend on the specific disease process and the nearby structures involved (1,2). Recent developments in computed tomography (CT) and magnetic resonance (MR) imaging have greatly improved diagnosis and management of petrous apex lesions by allowing earlier detection and more accurate preoperative diagnosis (3,4).

Abbreviations: CISS = constructive interference in the steady state, CSF = cerebrospinal fluid, IAC = internal auditory canal, ICA = internal carotid artery, LCH = Langerhans cell histiocytosis

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In this article, we present concepts related to petrous apex imaging that one should be familiar with when evaluating patients with suspected disease in this region. We describe the anatomy of the petrous apex and surrounding structures, discuss imaging techniques for evaluating the petrous apex, review the clinical features of petrous apex lesions, and present an overview of lesions arising from or commonly involving the petrous apex. We conclude with a review of anatomic variants that may be confused with pathologic conditions by inexperienced radiologists.

Anatomy of the Petrous Apex
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 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. CT and MR imaging appearances of the normal petrous apex are illustrated in Figures 1–3.

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 (Fig 1b). The Dorello canal extends through the posteromedial portion of the petrous apex and contains the abducens nerve (cranial nerve VI) (Fig 3a).

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
Figure 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 (asterisk) 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.

Figure 3. Normal petrous apex canals. (a) Axial MR image obtained with the constructive interference in the steady state (CISS) sequence shows the cisternal segments of the abducens nerves entering the skull base through the Dorello canals (arrowheads), which are located along the posteromedial walls of the petrous apices. The Meckel caves (arrows) can also be seen bilaterally, coursing over the superior margins of the petrous apices just anterolateral to the Dorello canals. (b) Axial CT image shows the subarcuate canal (arrow) coursing laterally in the superior portion of the petrous apex between the crura of the superior semicircular canal. The subarcuate canal can occasionally be mistaken for a fracture. (c) Axial CT image at the level of the IAC shows the singular canal (arrow), which extends from the posterior wall of the IAC to the ampulla of the posterior semicircular canal. Note also the pneumatized anterior portion of the petrous apex.

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 (3,4,7).

junction of the ampulla of the posterior semicircular canal and the vestibule (Fig 3c). It contains the singular nerve, which is a division of the inferior vestibular nerve that innervates the ampulla of the posterior semicircular canal (2,6).

Along the anterior superior aspect of the petrous apex is a smooth depression that serves as the floor of the Meckel cave (Fig 3a). 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) (2).
Methods of Examination

MR imaging of the temporal bone should be performed with small surface coils or a dedicated multichannel head coil. T1-weighted sequences should be performed before and after intravenous administration of gadolinium contrast material; an axial three-dimensional gradient-echo sequence with 1-mm (or even submillimeter) section thickness or thin-section (section thickness ≤2 mm) axial and coronal spin-echo sequences can be used. Fat saturation techniques should be applied after contrast material administration. A heavily T2-weighted sequence, usually a three-dimensional turbo or fast spin-echo sequence with submillimeter section thickness or a balanced steady-state free-precession sequence (such as CISS or fast imaging employing steady-state acquisition), should also be performed.

Diffusion-weighted imaging is also a useful technique in the petrous apex, particularly for distinguishing cholesteatomas from other developmental lesions and characterizing the relative cellularity of neoplasms in the region. At our institution, diffusion-weighted imaging is performed by using a multisection spin-echo echo-planar imaging sequence identical to the sequence used routinely in the brain, with b values of 0 and 1000 sec/mm².

CT of the temporal bone can be performed by using helical or direct sequential axial and coronal imaging with submillimeter collimation from the top of the petrous bone to the tip of the mastoid. The advantages of helical acquisition are lower overall radiation dose and the ability to perform multiplanar reformation of the dataset, but the reformatted images in nonaxial planes tend to be less sharp than directly acquired sequential images. Images should be reconstructed with a submillimeter section thickness and use of a bone reconstruction algorithm to provide optimal visualization of the bony anatomy of the temporal bone.

In patients who are also undergoing MR imaging, we do not typically perform temporal bone CT with contrast material. However, in patients with a suspected petrous apex lesion and contraindications to MR imaging, we administer intravenous contrast material with the addition of soft-tissue windows to help delineate the extent of enhancing tumors.

Clinical Features and Choice of Imaging Technique

Patients with petrous apex lesions are often asymptomatic or present with nonspecific symptoms, such as headache, retro-orbital pain, or ear pain. Large lesions or those close to cranial nerves (eg, in the Meckel cave, cavernous sinus, or IAC) may cause cranial neuropathies, which can include sensorineural hearing loss, tinnitus, or vertigo (cranial nerve VIII); diplopia (cranial nerves III, IV, or VI); facial pain (cranial nerve V); or facial weakness (cranial nerve VII). The trigeminal and abducens nerves are the most commonly affected cranial nerves in the setting of petrous apex disease, as they are separated from the petrous apex by only a thin layer of dura mater and are therefore particularly susceptible to compression by adjacent disease processes (1–4).

Patients are commonly referred for dedicated imaging of the petrous apex because of cranial neuropathies or to further characterize lesions detected incidentally with other imaging studies. We generally recommend MR imaging as the first-line study for evaluation of suspected petrous apex disease because its higher contrast resolution provides excellent delineation of the soft-tissue extent of lesions, nicely demonstrates invasion of the cavernous sinus and marrow space, and depicts the relationship of lesions to nearby cranial nerves and vessels. MR imaging may also allow definitive diagnosis of certain petrous apex lesions that have unique signal intensity characteristics, such as cholesterol granulomas and petrous apex cephaloceles (discussed later).

In our practice, CT is primarily a problem-solving tool reserved for situations in which better characterization of mineralization and osseous involvement is needed, as CT is superior to MR imaging in this regard (7–10). At CT, lesions within or involving the petrous apex may be characterized as aggressive or nonaggressive on the basis of the associated pattern of bone erosion or remodeling. Some tumors may also demonstrate specific patterns of matrix mineralization (osteoid, chondroid, or fibrous) that may significantly narrow the list of differential diagnostic considerations. Another distinct advantage of CT is its ability to demonstrate the proximity of petrous apex lesions to certain critical structures such as the otic capsule and ossicles with submillimeter accuracy (4,8,9).

Both MR imaging and CT can also be used for biopsy site guidance as well as planning for surgery (4,7,11,12).

Classification of Petrous Apex Lesions

Several methods have been proposed for classifying lesions of the petrous apex. For instance, lesions may be classified as cystic or solid, as surgically treatable or not surgically treatable, as aggressive or nonaggressive, or as those that primarily arise within the petrous apex or those that spread secondarily to the region (1–4,7). We
prefer to categorize petrous apex abnormalities on the basis of their specific cause or origin into developmental lesions, inflammatory or infectious lesions, neoplastic (benign or malignant) lesions, vascular lesions, or osseous dysplasias (Table). In the remainder of this article, we discuss some of these lesions in detail.

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<th>Classification of Petrous Apex Lesions on the Basis of Origin and Imaging Characteristics</th>
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Note.—CSF = cerebrospinal fluid, DWI = diffusion-weighted imaging, LCH = Langerhans cell histiocytosis, T1WI = T1-weighted images, T2WI = T2-weighted images.
Developmental Lesions

Cholesterol Granuloma

Cholesterol granulomas are the most common lesions arising in the petrous apex. Classically, they occur in patients with a pneumatized petrous apex and a long-standing history of otitis media. The cysts are filled with viscous brown fluid, granulation tissue, and cholesterol crystals, which are often contained within a thick fibrous capsule that lacks a true epithelial lining.

The pathogenesis of cholesterol granulomas is still unknown, but the classic theory is that they are caused by inadequate ventilation of petrous apex air cells, which results in negative pressure, reabsorption of air, mucosal edema, and hemorrhage, with local tissue breakdown and cholesterol formation leading to a foreign-body reaction. A newer theory suggests that aggressive pneumatization of the petrous apex leads to exposure of bone marrow. The exposed marrow hemorrhages, causing obstruction of the apical outflow tract, which in turn leads to breakdown of the blood, a foreign-body reaction, and subsequent cyst formation and bone expansion (2,4).

Cholesterol granulomas can be large at the time of diagnosis. Bone expansion caused by growth of these cysts may lead to “bone gaps” caused by long-standing severe bone remodeling that may be mistaken for bone destruction. A diagnosis of cholesterol granuloma can be made reliably with MR imaging. Whereas most other petrous apex lesions have low or intermediate signal intensity on T1-weighted images, cholesterol granulomas are usually hyperintense on both T1- and T2-weighted images (13) (Fig 4). Because pneumatization of the petrous apex is typically symmetric in distribution, the presence of a highly pneumatized contralateral petrous apex also supports the diagnosis of cholesterol granuloma. Cholesterol granulomas do not enhance with contrast material, and the presence of enhancement should lead to an alternative diagnosis (14–16).

The chief differential diagnostic consideration at MR imaging is asymmetric petrous apex pneumatization (which is discussed in detail later). In these instances, fatty bone marrow also appears hyperintense on T1-weighted images and may be mistaken for a lesion. Fat-suppressed imaging allows differentiation of the two entities, as cholesterol granulomas will remain hyperintense after fat suppression (13,17). Asymptomatic small cholesterol granulomas are managed conservatively, whereas large lesions in symptomatic patients are treated with surgical drainage or cyst resection (2).

Cholesteatoma (Epidermoid)

Petrous apex cholesteatomas and epidermoids make up 4%–9% of all petrous apex lesions. Cholesteatomas may be classified as acquired or congenital, with congenital cholesteatomas of the petrous apex being more common. Congenital cholesteatomas arise from aberrant ectoderm that is trapped during embryogenesis; at histologic analysis, they consist of cysts lined with stratified squamous epithelium and filled with keratinous debris (17). These lesions classically occur in children and young adults.
At CT, cholesteatomas appear as nonenhancing, expansile petrous apex lesions that cause variable degrees of bone destruction. When no or minimal bone destruction is present, they cannot be distinguished from cholesterol granulomas with CT alone. At MR imaging, cholesteatomas generally have intermediate to low signal intensity on T1-weighted images. On T2-weighted and fluid-attenuated inversion-recovery images, they generally have high signal intensity. Enhancement is not seen after contrast material administration. Diffusion-weighted imaging is useful in diagnosis of cholesteatomas, as the lesions often show restricted diffusion, a feature that can be particularly beneficial in detection of recurrent cholesteatomas after surgical resection (4).

Petrous Apex Mucoceles

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. Symptomatic mucoceles are usually drained surgically, whereas asymptomatic mucoceles may be followed with serial imaging (2,4,18).

**Petrous Apex Cephalocele**

Petrous apex cephaloceles are rare lesions representing protrusions of arachnoid or dura mater, usually from the Meckel cave, into the petrous apex. They are thought to be caused by chronically increased intracranial pressure that is transmitted into the Meckel cave through a patent porus trigeminus. Petrous apex cephaloceles are associated with empty sella and Usher syndrome, are usually bilateral, and occur more often in women than in men (19,20). The lesions may be incidental findings but occasionally may erode into the otic capsule or pneumatized petrous apex cells, resulting in headaches, hearing loss, or CSF otorrhea (21).

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 (Fig 5). 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 (19–22).
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. Coronal or sagittal T2-weighted imaging is particularly useful for demonstrating extension of a cephalocele through the skull base (Fig 6). Iatrogenically induced cephaloceles can also be seen after surgery of the petrous apex.

Inflammatory Lesions

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 (23).

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 (24–26). Abscesses demonstrate ring enhancement and restricted diffusion on diffusion-weighted images (12). CT demonstrates opacification of petrous air cells in the early stage of the disease and bone destruction in later stages (Fig 7). Gallium single-photon-emission computed tomography (SPECT) is useful for evaluating the response to therapy (27).

Petrous Apex Osteomyelitis
Skull base osteomyelitis involving the petrous apex is distinguished from petrous apicitis in that the former can occur in a nonpneumatized 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 (2). Patients frequently have a predisposing condition such as diabetes mellitus, and *Pseudomonas* is by far the most common causative organism (2,25,26,28).

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
Figure 7. Petrous apicitis. (a) Axial contrast-enhanced CT image shows opacification of the right mastoid, middle ear, and anterior petrous air cells. There is cortical erosion along the posterior margin of the petrous apex (arrow). Note the pneumatization of the left petrous apex. (b) Gadolinium-enhanced fat-suppressed T1-weighted MR image shows diffuse enhancement throughout the right mastoid and middle ear, anterior petrous apex, and clivus. There is also a focal rim-enhancing fluid collection in the anterior petrous apex (arrowhead), a finding consistent with an abscess.

Figure 8. Osteomyelitis of the petrous apex and skull base in a patient with otitis externa. (a) Unenhanced T1-weighted MR image shows an infiltrative process involving the central skull base and temporomandibular joint. Note the loss of normal fatty marrow signal intensity in the clivus and left petrous apex. There is fluid in the left mastoid. (b) 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. Note the enhancement of the left external auditory canal and periauricular soft tissues (arrow).

In addition to common bacterial causes of petrous apex infection, atypical organisms such as Aspergillus can gain access via longitudinally arranged air cell tracts connecting the base of the petrous bone to the apex (29). Tuberculous disease is usually due to hematogenous spread of mycobacteria through venous plexuses or retrograde transmission via cell tracts from the mastoid (30). The MR imaging and CT features of these atypical infections are nonspecific and indistinguishable from those of other causes of petrous apicitis and osteomyelitis.

Bone scanning with technetium 99m SPECT is highly sensitive in detection of skull base osteomyelitis and is helpful in prediction of long-term outcome, as osteomyelitis extending across the midline tends to have a less favorable prognosis. Gallium SPECT is typically used to monitor the response to antibiotic therapy (25,26). Tissues or intracranial involvement (26) (Fig 8).
Inflammatory Pseudotumor
Inflammatory pseudotumor is a rare nonneoplastic inflammatory lesion that falls within the spectrum of focal fibrohistiocytic proliferations. It occurs most commonly in the lung but can affect the head and neck, with the larynx, trachea, and pharynx accounting for most of these cases. Involvement of the temporal bone is exceedingly rare, but one-third of patients with inflammatory pseudotumor of the temporal bone demonstrate petrous apex involvement (31).

At imaging, there is a locally aggressive, enhancing soft-tissue mass in the temporal bone that causes extensive bone erosion. There are no specific imaging features, and these lesions often mimic an aggressive tumor or infection. Definitive diagnosis requires biopsy, which demonstrates fibroblastic proliferation and mixed inflammatory cell infiltrates at histologic analysis. Surgical resection is the treatment of choice (2,31,32).

Wegener Granulomatosis
Wegener granulomatosis is a rare necrotizing vascular disorder characterized by the triad of necrotizing granulomas in the respiratory tract, a necrotizing vasculitis of small arteries and veins, and glomerulonephritis. The peak prevalence is in the 5th decade, and males are affected twice as often as females. Although the sinonasal cavity is the most commonly affected site in the head and neck, middle ear and mastoid involvement is seen in 40%–70% of patients and lesions may involve the petrous apex. Temporal bone involvement can also predispose patients to additional infectious complications such as petrous apicitis (33,34).

The imaging appearance of the lesions of Wegener granulomatosis is nonspecific; however, the combination of temporal bone lesions and erosive sinonasal disease strongly suggests the diagnosis. At CT, granulomas in Wegener granulomatosis appear as destructive soft-tissue masses. At MR imaging, the lesions are hypointense on T1-weighted images and hyperintense on T2-weighted images, with enhancement on gadolinium-enhanced images (33,34).

Benign Tumors

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 (35).

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 (Fig 9) (1,36).

Schwannoma
Petrous apex schwannomas usually originate from the fifth, seventh, or eighth cranial nerves. At CT, schwannomas are usually isodense 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. Occasionally, intracranial schwannomas may be cystic and contain fluid levels, findings suggestive of the diagnosis (37,38) (Fig 10).

Trigeminal nerve schwannomas usually arise in the Meckel cave or from the cisternal segment of the nerve. They may grow antegradely or retrogradely along the nerve and its branches, causing expansion of the neural foramina and superior indentation of the petrous apex. They may override the petrous apex and extend into the middle and posterior cranial fossae (2) (Fig 10).

Schwannomas of the eighth cranial nerve usually originate from the vestibular division, around the vestibular ganglion near the fundus of the IAC, and appear as well-defined masses filling the IAC. When large, the tumors tend to enlarge the porus acusticus and extend into the cerebellopontine angle. Rarely, vestibular schwannomas may extend laterally into the labyrinth (intralabyrinthine schwannomas) or even into the middle ear cavity (1,2,4).

Facial nerve schwannomas are rare. They usually originate from the geniculate ganglion and progress proximally or distally, causing expansion of the geniculate fossa and facial nerve canal (2). A rare type of petrous apex schwannoma is the primary intrasosseous schwannoma, which is found in the petrous carotid canal and is thought to arise from the deep petrosal nerve of the periarterial carotid plexus (39).
Paraganglioma
Paragangliomas may invade the petrous apex via preformed air cell tracts from their sites of origin in the jugular foramen or 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 (40). 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 (40) (Fig 11).

Chondroma
Chondromas are rare benign cartilaginous tumors that can manifest as solitary or multifocal tumors. They are associated with Ollier disease and Maffucci syndrome. Chondromas of the petrous apex are extremely uncommon and usually arise at the junction of the petrosphenoidal, petroclival, and spheno-occipital synchondroses (41). These tumors are slow growing, and symptoms result from compression of nearby structures.

At CT, chondromas classically appear as lytic bone lesions containing arc- and ringlike chondroid matrix calcification, but visible matrix calcification may be absent. At MR imaging, chondromas have low signal intensity on T1-weighted images and high signal intensity on T2-weighted images, with variable enhancement on contrast-enhanced images. Matrix calcification is evident as foci of low signal intensity on both T1- and T2-weighted images (41).

Malignant Tumors
Chondrosarcoma
Chondrosarcomas are malignant cartilaginous tumors that typically manifest in the 2nd and 3rd decades of life. In the skull base, they are much more common than their benign counterpart, the chondroma. Similar to chondromas, chondrosarcomas are associated with a number of syndromes, including Ollier disease, Maffucci syndrome, and Paget disease. They are believed to occur as a

result of degeneration of remnants of enchondral cartilage along skull base synchondroses. Therefore, chondrosarcomas involving the petrous apex typically originate at the petroclival and petrosphenoidal synchondroses (2,42).

Chondrosarcomas have an appearance similar to that of chondromas, and the two entities are essentially indistinguishable at imaging. CT demonstrates a destructive petrous apex mass containing arcs and rings of calcification, which reflect the chondroid nature of the tumor. At MR imaging, the lesions have low to intermediate signal intensity on T1-weighted images and high signal intensity on T2-weighted images relative to that of brain tissue. Signal heterogeneity can be seen and may be due in part to the presence of mineralized chondroid matrix (Fig 12). These tumors dem-
Figure 13. Chordoma. (a) Axial CT image shows a destructive mass involving the clivus, nasopharynx, left masticator space, and left petrous apex. Small foci of calcification in the lesion may represent residual bone. (b) Gadolinium-enhanced fat-suppressed T1-weighted MR image shows avid enhancement of the mass. Some areas have a honeycomb pattern of enhancement (arrow).

Chordoma
Chordomas are rare tumors that originate from embryologic remnants of the notochord and can occur anywhere from the skull base to the sacrum. Skull base chordomas are typically midline lesions arising in the clivus but may extend laterally to involve the petrous apex. Chordomas are most frequently seen in males younger than 40 years but can occur at any age. In the pediatric population, skull base chordomas are typically more aggressive and show a wider range of clinical features, atypical morphologies, and a greater prevalence of metastases (1,3).

At CT, skull base chordomas appear as locally destructive soft-tissue masses centered in the clivus. Calcifications are often evident and represent residual bone trabeculae; true tumor calcifications may occur in the chondroid variant of chordoma. Low-attenuation areas are occasionally seen and represent portions of the tumor containing gelatinous material. At MR imaging, chordomas are typically hypointense on T1-weighted images and hyperintense on T2-weighted images. After contrast material administration, they demonstrate variable enhancement and may have a characteristic honeycomb enhancement pattern (44) (Fig 13).
Endolymphatic Sac Tumor

Endolymphatic sac tumors are locally aggressive tumors arising from the proximal rugose portion of the endolymphatic sac, which is situated halfway between the IAC and jugular foramen. Therefore, these tumors are characteristically located along the posterior wall of the temporal bone and may extend into the mastoid or the anterior portion of the petrous apex. They are usually sporadic, but bilateral endolymphatic sac tumors are associated with von Hippel–Lindau syndrome (45).

At CT, endolymphatic sac tumors appear as soft-tissue masses with prominent intratumoral calcification that cause permeative bone erosion along the posterior surface of the petrous bone. At MR imaging, they have heterogeneous signal intensity on both T1- and T2-weighted images. Up to 88% of the lesions demonstrate areas of high signal intensity on T1-weighted images due to deposition of methemoglobin, hemosiderin, and cholesterol crystals from repeated intratumoral hemorrhage (Fig 14). Complete surgical resection of these tumors is the treatment of choice (45–47).

Metastasis

Metastases to the petrous apex are most commonly seen in patients aged 50–70 years. The petrous apex is the most common site for metastases in the temporal bone (83% of cases) and is the sole site of temporal bone involvement in 31% of cases. The most common tumor to metastasize to the petrous apex is breast cancer, followed by lung, prostate, and renal cell carcinomas (4). Metastatic tumor involvement of the petrous apex may be due to hematogenous spread from distant tumors, direct extension of an extra- or intracranial tumor, or leptomeningeal extension of a distant or intracranial tumor. Susceptibility of the petrous apex to hematogenous metastases is thought to be due to slow blood flow through the petrous apex marrow, which allows filtering and deposition of tumor cells (1,40).

The imaging characteristics of petrous apex metastases are nonspecific. Frequently, they demonstrate significant bone destruction and marked enhancement. CT usually shows an aggressive lytic lesion destroying the skull base. MR imaging depicts the soft-tissue extent of metastases, which usually have low to intermediate signal intensity on T1-weighted images and variable signal intensity (usually intermediate to
high) on T2-weighted images; enhancement is also variable (48,49) (Fig 15). Highly vascular metastases from melanoma, renal carcinoma, and thyroid carcinoma may mimic a glomus tumor on MR images because of the presence of intratumoral flow voids or hemorrhage. Treatment of petrous apex and skull base metastases is primarily palliative (1,48).

**Plasmacytoma**

Plasmacytomas of the temporal bone are rare. They may develop as intraosseous plasmacytomas from the marrow-rich petrous bone or as extramedullary plasmacytomas arising from the middle ear or mastoid mucosa that subsequently invade the petrous apex. At CT, they appear as lytic lesions without sclerotic rims. At MR imaging, they have low to intermediate signal intensity on T1-weighted images and are isointense on T2-weighted images, with intense enhancement.

Similarly to paragangliomas, plasmacytomas may show intratumoral flow voids. They may also exhibit restricted diffusion with low apparent diffusion coefficients because of their high cellularity. Radiation therapy is the primary treatment for these highly radiosensitive tumors, with surgery considered the second-line therapy (40,50).

**Figure 15.** Metastases to the petrous apex in a patient with breast cancer. (a) Axial non–fat-suppressed T2-weighted MR image shows an expansile lesion of mixed signal intensity (arrow) in the left petrous apex. Compare the signal intensity of the lesion to that of the high-signal-intensity fatty marrow in the normal contralateral petrous apex (arrowhead). (b) Corresponding gadolinium-enhanced T1-weighted MR image shows that the lesion has intense homogeneous enhancement. (c) Axial CT image obtained in a patient with metastatic leiomyosarcoma shows a lytic metastasis (arrow) centered in the left petrous apex.
Figure 17. Rhabdomyosarcoma in a young child. (a) Axial CT image shows a permeative lesion of the right middle ear and mastoid that extends into the petrous apex (arrow). (b) Corresponding gadolinium-enhanced T1-weighted MR image shows that the lesion (arrow) has homogeneous enhancement.

Lymphoma
Lymphoma involving the central skull base is rare and has nonspecific imaging features. It demonstrates strong enhancement, a permeative pattern of bone destruction at CT, and intermediate or slightly low signal intensity on T2-weighted images. Low apparent diffusion coefficients are typical because of the high degree of cellularity in these lesions (51).

Nasopharyngeal Carcinoma
Nasopharyngeal carcinoma occasionally spreads to the petrous apex by direct extension and can cause symptoms similar to those of the Grade-nigo triad (52). Infiltration into the IAC can also occur, leading to compression of the eighth cranial nerve and symptoms of vertigo, sensorineural hearing loss, or tinnitus (53).

Nasopharyngeal carcinoma demonstrates intermediate to high signal intensity (usually
greater than that of muscle) on T2-weighted images, low signal intensity on T1-weighted images, and moderate enhancement on contrast-enhanced images (Fig 16). Lymphadenopathy is present in up to 90% of patients, with the retropharyngeal chain often being the first involved nodal site (52).

Rhabdomyosarcoma
Rhabdomyosarcoma is a malignant childhood tumor arising from skeletal muscle or undifferentiated mesenchymal tissue. It has a bimodal age distribution, with one peak occurring during the 1st decade of life and the second occurring during adolescence. It is the most common soft-tissue malignancy in children and the most common primary malignancy of the temporal bone (54). Rhabdomyosarcomas usually involve the external auditory canal, middle ear cavity, and facial nerve but may extend medially into the petrous apex. Central nervous system extension and metastases can occur.

CT demonstrates an aggressive soft-tissue mass in the temporal bone that causes osseous destruction. At MR imaging, these tumors have intermediate signal intensity on T1-weighted images, variable signal intensity on T2-weighted images, and variable enhancement (54,55) (Fig 17). Differential diagnostic considerations for a destructive enhancing temporal bone lesion in a child include metastatic neuroblastoma and LCH.

Langerhans Cell Histiocytosis
LCH is an idiopathic neoplastic disorder characterized by proliferation of mature eosinophils and Langerhans cells. Classically, three major subtypes of the disease were described—eosinophilic granuloma, Hand-Schuller-Christian disease, and Letterer-Siwe disease—but these disorders are now all classified as LCH. Temporal bone involvement is seen in 15%–61% of cases of LCH, and the temporal bone is the most common site of skull base involvement (56). The process most frequently affects the mastoid, with the petrous apex, squamous bone, and middle ear being less often involved. Solitary lesions are typically treated with curettage or excision (2,40).

At CT, LCH appears as a well-circumscribed destructive lesion of the temporal bone. The margins of the lesion are usually nonsclerotic. Erosion of the bony labyrinth and ossicular chain can occur. At MR imaging, LCH lesions appear as focal soft-tissue masses in the temporal bone surrounded by extensive ill-defined bone marrow and soft-tissue edema. Marked enhancement is seen at both CT and MR imaging (56,57) (Fig 18).

Vascular Lesions
Petrous Carotid Aneurysm
Aneurysms of the petrous ICA segment are rare and may be traumatic, congenital, or postinfectious in origin (58). They are usually found incidentally but may be brought to attention due to tinnitus, cranial nerve palsies, or Horner syndrome (1,4). Most petrous ICA aneurysms are large and fusiform, and 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 (Fig 19). The degree of flow in the aneurysm may be underestimated with time-of-flight MR angiography because of turbulence. Contrast-enhanced MR angiography can be useful for delineating the size, patency, and extent of the aneurysm. Endovascular interventions or surgical trapping procedures are considered for symptomatic patients (17,58).

**Figure 21.** Polyostotic fibrous dysplasia. Axial CT image through the temporal bones shows diffuse enlargement of the central and left lateral skull base, including the petrous apex, with sparing of the otic capsule. The involved bone has a characteristic ground-glass appearance, a finding indicative of a combination of turbulent flow and likely mural thrombus.

**Figure 20.** Intraosseous dural arteriovenous fistula. Axial T1-weighted MR image shows a large multicompartmental lesion that consists of multiple hypointense flow voids. The lesion replaces the clivus and extends into both petrous apices, the masticator space, and the posterior cranial fossa.

**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 (59).

At CT, intraosseous dural arteriovenous fistulas appear as osteolytic lesions. MR imaging demonstrates multiple intraosseous flow voids (Fig 20) 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.

**Osseous Dysplasias**

**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. Classically, fibrous dysplasia is characterized as monostotic or polyostotic. The polyostotic form is associated with craniofacial involvement in roughly 50% of cases and can cross suture lines. Although rare, petrous apex involvement can occur and may cause narrowing of neural or vascular foramina, resulting in cranial neuropathies or vascular insufficiency (2,4).

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 (60) (Fig 21). 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 (60,61).

**Paget Disease**

Paget disease is a disorder of bone remodeling most commonly seen in white males over the age of 50 years. Possibly caused by an indolent viral infection, Paget disease is characterized by production of dense fragile bone formed by alternating episodes of excessive abnormal osteoclastic and osteoblastic activity. The disease may be polyostotic or monostotic and usually involves the calvaria, skull base, and temporal bones. Otic capsule involvement can also occur. Unlike fibrous dysplasia, Paget disease typically spares the facial bones (1,40,60).

Paget disease can be divided into three phases: osteolytic, mixed, and sclerotic. In the temporal bones, a fourth remodeling phase is described, in which inactive fragments of new bone undergo remodeling to produce lamellar bone containing haversian canal systems and new marrow (62). 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 (Fig 22). 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 (60).

**Normal Variants**

(“Leave Me Alone” Lesions)

**Asymmetric Petrous Apex Pneumatization and Sclerosis**

As noted earlier, petrous apex pneumatization may be asymmetric in up to 10% of patients; in these instances, the presence of marrow signal intensity in the contralateral nonpneumatized apex can be misinterpreted as a mass at MR imaging (4,10). Bone marrow in nonpneumatized petrous apices can normally show variations in signal intensity depending on the patient’s age. In younger patients, marrow can be of intermediate signal intensity with conventional sequences because of the high concentration of red marrow. In adults, red marrow becomes replaced by fatty marrow, which has higher signal intensity on T1-weighted images.
When asymmetric, the high-signal-intensity fatty marrow may be mistaken for a cholesterol granuloma on T1-weighted images. Lack of mass effect and close observation of the signal intensity with other pulse sequences usually lead to the correct diagnosis. Particularly helpful is the fact that signal from normal fatty bone marrow becomes suppressed on fat-suppressed images. CT demonstrates normal trabeculated bone in the nonpneumatized petrous apex with attenuation similar to that of marrow-containing bone elsewhere in the skull base (2,63) (Fig 23).

Petrous apices can also be sclerotic in 7% of cases. When sclerosis is asymmetric, high-signal-intensity marrow in the nonsclerotic apex can also mimic a lesion, similar to cases of asymmetric pneumatization. Overall, these appearances are seen in less than 5% of individuals (3).

Petrous Apex Effusion
As in the mastoids, effusions can develop within pneumatized petrous apex cells and can occasionally mimic masses. At CT, effusions can be distinguished from mass lesions on the basis of the lack of bone remodeling and preservation of bone septa in the petrous apex when a simple effusion is present (4,18). Petrous apex effusions have low to intermediate signal intensity on T1-weighted images (unlike cholesterol granulomas, which have high signal intensity); on T2-weighted images, effusions have very high signal intensity similar to that of CSF (Fig 24). Effusions should not enhance after contrast material administration, although a thin rim of mucosal enhancement can occasionally be observed.

Rarely, patients may have a giant air cell, which is defined as a petrous apex air cell larger than 1.5 cm in its largest diameter. When filled with air, these cells are incidental findings of no clinical significance, but when completely filled with fluid they may mimic a petrous apex mucocele (2,64).

Subarcuate Canal Pseudofracture
In patients presenting with head trauma, the subarcuate canal can occasionally be mistaken for a fracture at CT (5). This pitfall can easily be avoided by recognizing the characteristic course of the canal, which travels from the posterior margin of the temporal bone to the mastoid antrum between the limbs of the superior semicircular canal (Fig 3b).
Pseudolesions in Infants
In infants under 4 months of age, it is not uncommon to see a low-attenuation focus in the anterior otic capsule extending toward the petro-occipital fissure. This hypoattenuation is a normal finding that may represent incompletely mineralized bone or cartilage and should not be mistaken for an abnormality in young children. In older subjects, however, lucency around the otic capsule should raise concern about a pathologic condition such as retrofenestral otosclerosis (65).

Postprocedure Findings
Trigeminal rhizotomy is occasionally performed for trigeminal neuralgia. In this procedure, very hyperattenuating tantalum-impregnated glycerol is injected in the region of the gasserian ganglion. CT performed even years after rhizotomy can show hyperattenuating material in the Meckel cave that extends to just above and posterior to the petrous apex (1,2).

Conclusions
Cross-sectional imaging plays an important role in diagnosis and management of petrous apex lesions. In addition to providing clues to the origin of a lesion, MR imaging and CT provide valuable information about the extent of a lesion as well as its relationship to important surrounding structures, such as the ICA and nearby cranial nerves. 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. In addition, by recognizing normal variant anatomy that can occasionally be mistaken for a pathologic condition, the astute radiologist can prevent patients from undergoing additional unnecessary and potentially invasive diagnostic procedures or treatments.

References


Lesions of the Petrous Apex: Classification and Findings at CT and MR Imaging
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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 (2).

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Large lesions or those close to cranial nerves (eg, in the Meckel cave, cavernous sinus, or IAC) may cause cranial neuropathies, which can include sensorineural hearing loss, tinnitus, or vertigo (cranial nerve VIII); diplopia (cranial nerves III, IV, or VI); facial pain (cranial nerve V); or facial weakness (cranial nerve VII). The trigeminal and abducens nerves are the most commonly affected cranial nerves in the setting of petrous apex disease, as they are separated from the petrous apex by only a thin layer of dura mater and are therefore particularly susceptible to compression by adjacent disease processes (1–4).

Page 156 (Figure on page 156)
A diagnosis of cholesterol granuloma can be made reliably with MR imaging. Whereas most other petrous apex lesions have low or intermediate signal intensity on T1-weighted images, cholesterol granulomas are usually hyperintense on both T1- and T2-weighted images (13) (Fig 4).

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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 (23).

Page 170 (Figure on page 170)
When asymmetric, the high-signal-intensity fatty marrow may be mistaken for a cholesterol granuloma on T1-weighted images. Lack of mass effect and close observation of the signal intensity with other pulse sequences usually lead to the correct diagnosis. Particularly helpful is the fact that signal from normal fatty bone marrow becomes suppressed on fat-suppressed images. CT demonstrates normal trabeculated bone in the nonpneumatized petrous apex with attenuation similar to that of marrow-containing bone elsewhere in the skull base (2,63) (Fig 23).