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Seminars in ULTRASOUND CT and MRI

Imaging of Nontraumatic Temporal Bone Emergencies

Nitesh Shekhrajka, MD and Gul Moonis, MD

This section aims to cover the non-traumatic pathologies affecting the temporal bone including external auditory canal, middle ear and inner ear which usually need emergent clinical attention. Many of the conditions in this section are secondary to infections in different clinical settings with resultant complications which may leave temporary or permanent sequelae if not suspected, timely diagnosed or treated.
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External Auditory Canal

Malignant Otitis Externa

This is a more aggressive form of acute otitis externa which affects the elderly, diabetic, and immunocompromised patients.[1] The term "Malignant" is a misnomer used to describe the aggressive clinical nature and high mortality in this condition. It is also referred to as necrotizing otitis externa (NOE). In most cases, the causative pathogen is *Pseudomonas aeruginosa*[2] which is not normally found in the external acoustic meatus (EAC) but *Aspergillus fumigatus* is also implicated in immunocompromised patients.[3]

The infection begins as an area of granulation at the junction of the bony and cartilaginous portions,[4] followed by chondritis and osteomyelitis. The infection then spreads to involve the structures outside the EAC such as middle ear, mastoid air cells, temporomandibular joint, and suprahyoid neck spaces (eg, parotid, masticator, and parapharyngeal spaces). Infection can also extend superiorly to involve the skull base and intracranial structures.[4]

Imaging: magnetic resonance imaging (MRI) and computed tomography (CT) are complementary modalities with CT showing the bone details to greater advantage and MRI better delineating associated soft tissue complications. Bony erosions, foraminal involvement, demineralization, and later sclerosis of the involved bones and skull base can be visualized on CT.

Department of Radiology, New York Presbyterian Hospital/Columbia University Medical Center, New York, NY.
Address reprint requests to Nitesh Shekhrajka, Department of Radiology, New York Presbyterian Hospital/Columbia University Medical Center, New York, NY. E-mail: nitesh1703@gmail.com

There is abnormal soft tissue thickening and enhancement along the margins of the EAC, auricle, and periauricular soft tissue. There is effacement of the fat planes around the stylomastoid foramen and infratemporal fossa[5]. The involvement of the stylomastoid foramen may result in facial nerve involvement. Opacification of middle ear cavity and mastoid air cells are frequently seen (Fig. 1).

If the disease extends inferiorly to involve the subtemporal soft tissues, parotid, masticator, and parapharyngeal spaces, imaging will demonstrate abnormal soft tissue enhancement, diffuse enlargement of the surrounding muscles, parotid enlargement, and effacement of the fat planes with or without abscess. Involvement of the temporomandibular joint can be seen as widening of the joint space and irregularity of the articular margins.

In the later stages of the disease, infection may involve the skull base bones leading to osteomyelitis. On CT osteomyelitis can manifest as demineralization, bony erosions, and periosteal bone reaction.[6] Increase in bone density can also be seen in chronic or treated cases. MRI, however, is the best imaging modality to evaluate suspected osteomyelitis because of its ability to evaluate fatty bone marrow. Diffuse T1 hypointensity with corresponding T2 hyperintensity and enhancement within the skull base is suggestive of osteomyelitis.[7]

Aural Foreign Body

Foreign body in the ear especially in the pediatric age group is commonly encountered by the emergency department. Direct otoscopic visualization is usually sufficient for diagnosis. Imaging is only necessary for evaluating complications in cases of chronic and deep lodged foreign bodies.

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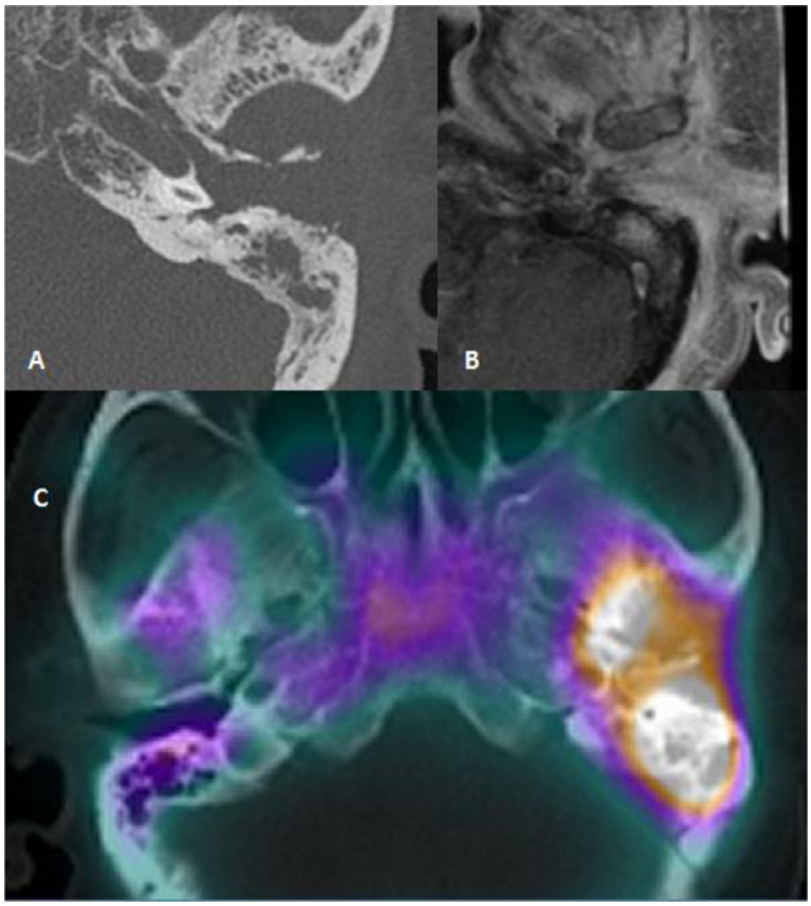

Figure 1 Malignant otitis externa: 74-year-old female with history of diabetes mellitus presents with sepsis and fever. Axial CT of the left temporal bone (A) demonstrating opacification of the mastoid air cells and external auditory canal (EAC) with soft tissue edema and destruction of the anterior and posterior wall of the EAC. Axial T1-post contrast fat sat MRI (B) showing extensive florid enhancement in the left EAC, periauricular, and retro-mastoid soft tissue, around the left temporomandibular joint extending into the left infratemporal and masticator region. Technetium-99 m methylene diphosphonate SPECT-CT (C) shows increased radiotracer uptake in the left EAC and surrounding soft tissue. The findings are consistent with malignant otitis externa. (Color version of figure is available online.)

Middle Ear and Mastoid

Acute Coalescent Mastoiditis

This occurs when acute infection leads to bone involvement with lysis and resorption of the septae which normally separate the mastoid air cells.[8] A dedicated CT of the temporal bone is needed to appreciate the osteoclastic dissolution of the septations[9] (Fig. 2).

Subdural Empyema

Direct spread of the infection through the erosion of temporal bone may result in peripherally enhancing crescent shaped subdural empyema which may demonstrate diffusion restriction on MRI (Fig. 3). Similar collection can also be seen in the epidural space.

Cerebral Abscess

Subdural empyema or direct spread may eventually lead to meningitis, cerebritis, and a frank abscess especially in the temporal lobe or cerebellum. Cerebral abscess manifests as ring enhancing and diffusions restricting lesion with central necrosis (Fig. 4).

Subperiosteal Abscess

Subperiosteal abscess of the mastoid protuberance is one of the commonest complications of coalescent mastoiditis secondary to mastoid cortical erosion and extrusion of the pus beneath the periosteum.[10] Other possible routes of spread of infection to subperiosteal space is through tympano-mastoid suture and/or through the vascular channels. CT and MRI with contrast can identify and delineate the abscess however CT is important to differentiate between reactive periostitis and a frank abscess (Fig. 5).

Bezold Abscess

The infection can erode through the mastoid cortex medial to the attachment of sternocleidomastoid muscle, at the attachment site of posterior belly of digastric muscle and can spread along the sternocleidomastoid muscle into the deep neck spaces. The abscess is usually confined to the posterior cervical

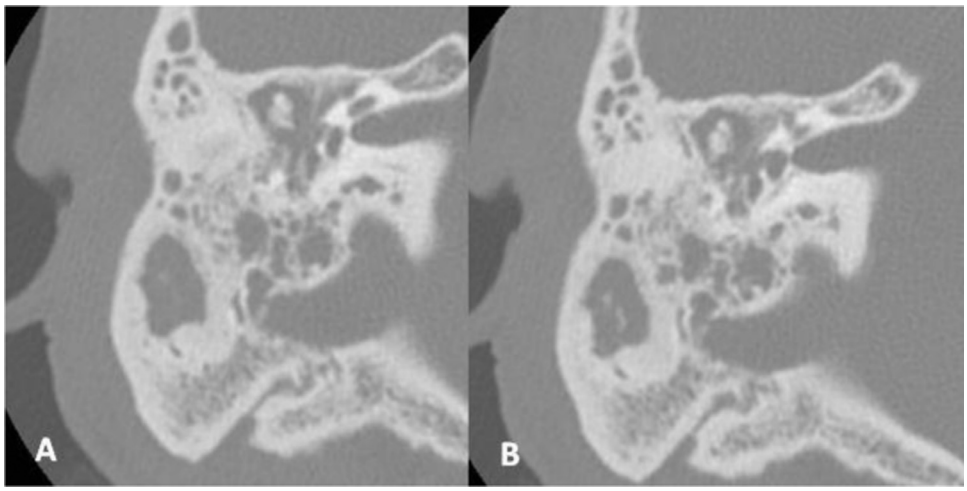

Figure 2 Acute coalescent mastoiditis: 20-years-old male with congenital HIV presents with fever, sepsis, and bacteremia. Axial CT of the right temporal bone (A and B) demonstrating opacification of the right mastoid air cells with destruction of the internal mastoid septations resulting in a large mastoid cavity. Also note the inflammatory changes in the tympanic cavity.

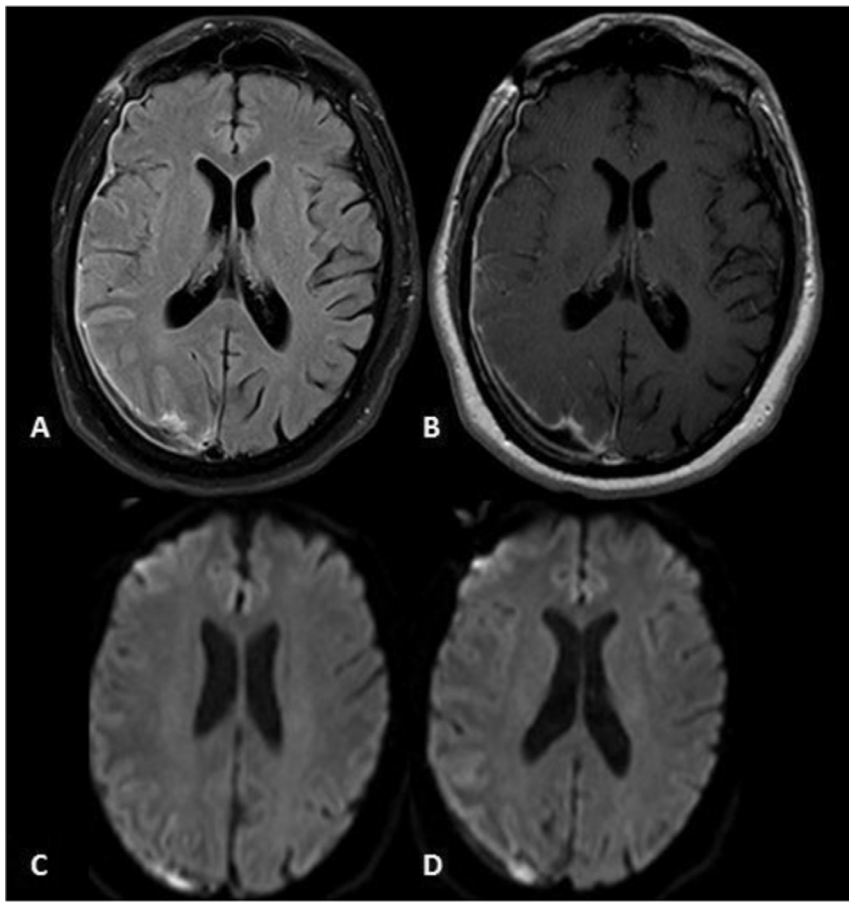

Figure 3 Subdural empyema: 39-years-old male with recent history of right sided mastoiditis presents with neurological symptoms. MRI of the brain, axial FLAIR (A) showing a FLAIR mixed intensity right frontoparietal subdural collection. Axial T1-post contrast (B) demonstrates peripheral enhancement corresponding to the right frontoparietal subdural collection. Axial diffusion-weighted images (C and D) demonstrates diffusion restriction in the right frontoparietal subdural space. Findings are consistent with subdural empyema.

and perivertebral spaces by the pharyngobasilar fascia and the deep layer of deep cervical fascia.[11] Bezold abscess is more common in adults due to thinner cortex of the pneumatized mastoid air cells.[11] CT shows erosion of the tip of the mastoid with peripherally enhancing abscess inferior to the mastoid and deep to the sternocleidomastoid muscle (Fig. 6).

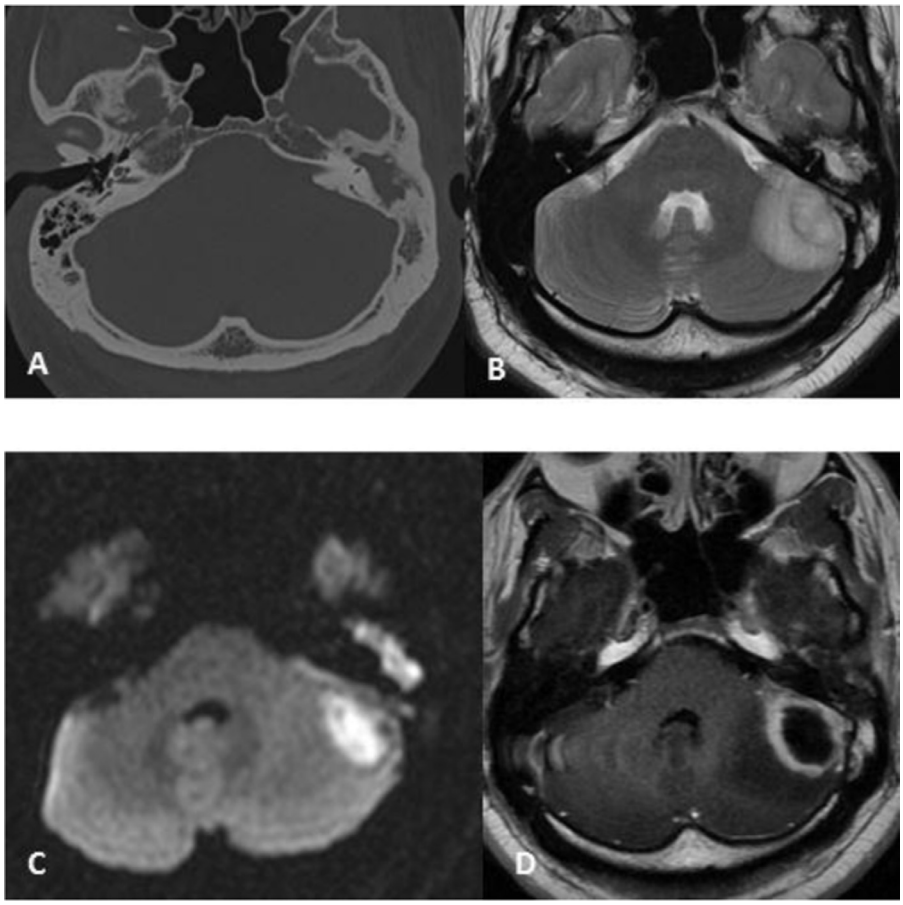

Figure 4 Cerebral abscess: 48-years-old male with fever, left sided hearing loss, and focal neurological symptoms. CT Temporal bones (A) demonstrating left mastoid effusion with loss of internal septations consistent with acute coalescent mastoiditis. Axial T2-weighted sequence of the brain (B) showing an area of abnormal T2 hyperintensity in the left cerebellum. Also note the left mastoid and middle ear effusions. Axial DWI sequence (C) showing corresponding diffusion restriction in the left cerebellum and Axial-T1 post contrast sequence (D) showing a large ring enhancing lesion in the left cerebellum, consistent with an abscess in the setting of acute coalescent mastoiditis.

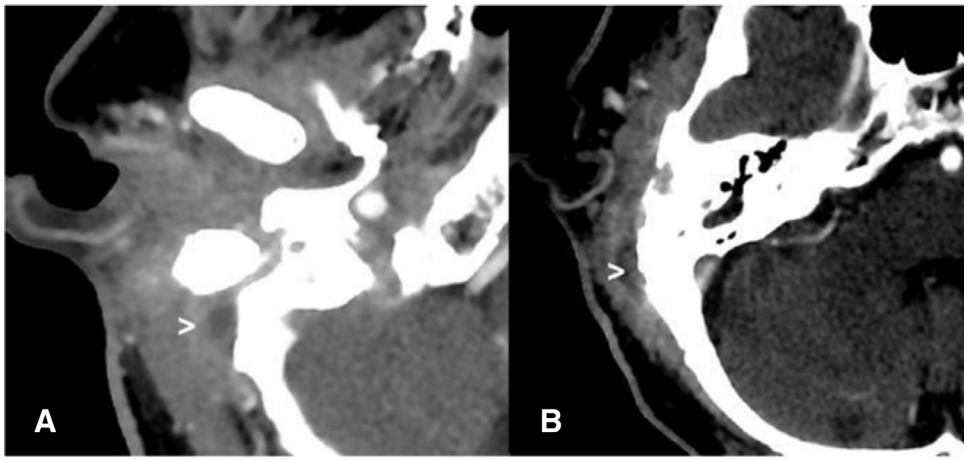

Figure 5 Subperiosteal abscess: 80-years-old male with poorly controlled diabetes mellitus presented with fever, sepsis, right ear pain, and swelling. Contrast enhanced CT of the right temporal bone (A and B) showing diffuse soft tissue thickening and enhancement along the right EAC, in the pre- and post auricular soft tissue and in the region of right temporomandibular joint. A small peripherally enhancing hypodensity is noted abutting the outer margin of the mastoid apex (arrowhead) with underlying erosive bony changes, consistent with a subperiosteal abscess in the setting of malignant otitis external and acute otomastoiditis.

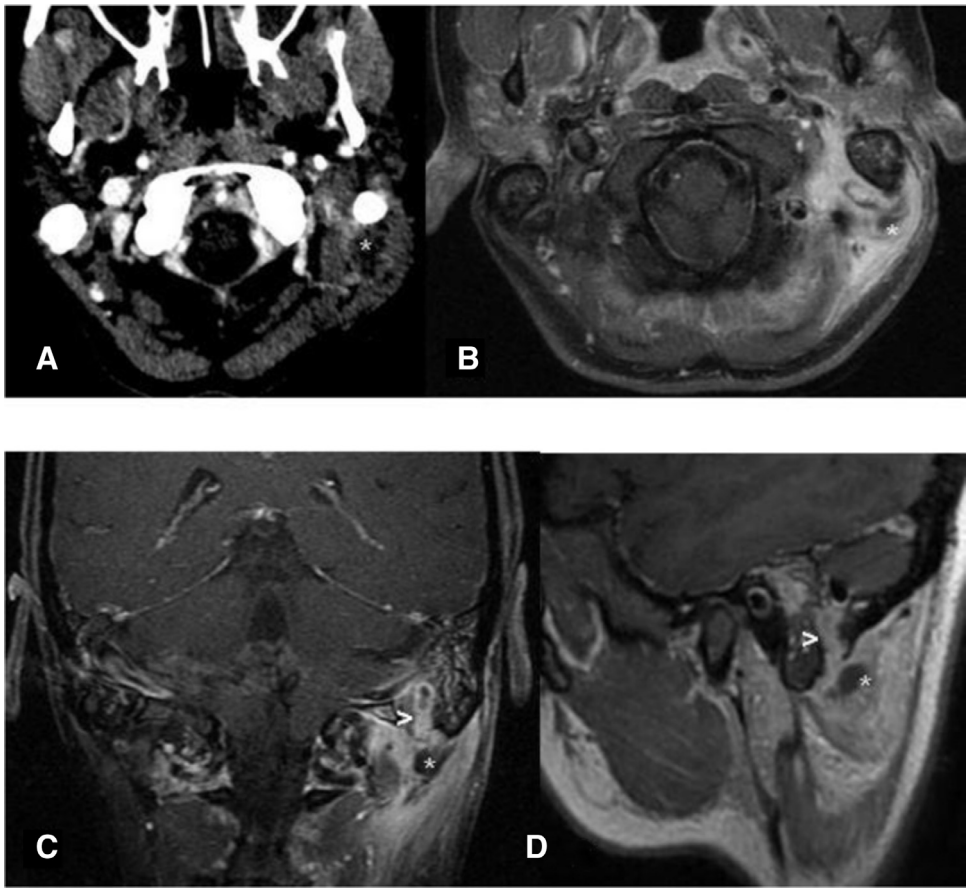

Figure 6 Bezold abscess: 26-years-old female with left sided acute mastoiditis. Contrast enhanced axial CT of the temporal bones (A) demonstrating a peripherally enhancing hypodense collection inferior to the left mastoid apex, medial to the sternocleidomastoid muscle (asterisk). MRI of the maxillofacial region, axial T1-post contrast fat sat sequence (B), Coronal T1-post contrast fat sat (C) and Sagittal T1-post contrast (D) showing a non enhancing collection (asterisk) in the infratemporal region, medial to the sternocleidomastoid muscle with diffuse surrounding soft tissue thickening and enhancement. Also note a defect in the left mastoid apex (arrowhead) and opacification/enhancement of the left mastoid air cells. A constellation of findings consistent with Bezold abscess in the setting of acute mastoiditis.

Petrous Apicitis

Petrous apex is pneumatized in approximately 30% of the population. When pneumatized, it is connected to the mastoid air cells through numerous supralabyrinthine and infralabyrinthine air channels that facilitate spread of infection from the mastoid air cells and middle ear to the petrous apex, resulting in petrous apicitis. Opacification of the petrous apex with septal and cortical destruction can be visualized on CT while MR can better show adjacent meningeal inflammation and enhancement.

While petrous apicitis occurs in pneumatized air cells, petrous apex osteomyelitis can occur in both nonpneumatized and pneumatized petrous apex. Infection may spread directly from malignant otitis externa or through the petrous carotid canal venous plexus in case of thrombophlebitis. On MRI extensive soft tissue enhancement is noted in the external auditory canal, petrous apex, and skull base soft tissue with bony erosion and replacement of the fatty bone marrow with enhancing soft tissue attenuation in the temporal bone and petrous apex (Fig. 7).

Patients can develop sixth nerve palsy and deep retroorbital pain in the V1 distribution in association with petrous apicitis due to close proximity of petrous apex to these nerves. A constellation of these findings is termed Gradinego syndrome.[12]

Venous Sinus Thrombosis

Thrombosis of the venous sinuses specially sigmoid sinus and internal jugular vein may be secondary to direct spread of infection from the temporal bone and mastoid.[13] On CT angiographahy and magnetic resonance angiography sinus thrombosis can be seen as filling defects in the involved sinuses. A peri sinus abscess can be difficult to differentiate from venous sinus thrombosis (Fig. 8).

Inner Ear

Bell's Palsy

Bell's palsy or idiopathic facial nerve paralysis is characterized by sudden onset unilateral or less commonly bilateral facial

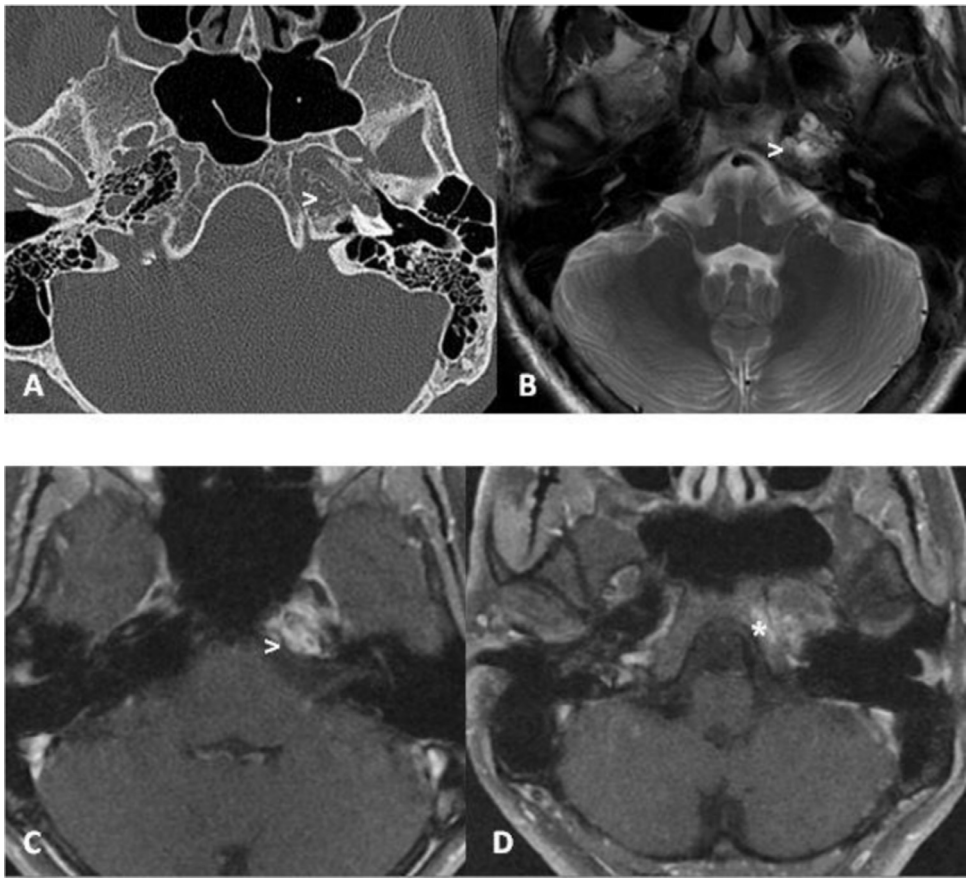

Figure 7 Petrous apicitis: 24-year-old male presents with left sided headaches. Axial CT of the temporal bones (A) demonstrating opacification of the left petrous apex air cells (arrowhead) with destruction of the internal septations (note the preserved septations on the right). MRI of the skull base, Axial T2-weighted sequence (B) demonstrating abnormal T2 hyperintensity in the left petrous apex air cells (arrowhead). Axial T1-post contrast fat sat sequences (C and D) showing abnormal enhancement (arrowhead) in the petrous apex (C), extending into the right side of the clivus (asterisk) (D). The findings are consistent with petrous apicitis.

nerve paralysis with subsequent spontaneous resolution within 6 months. As there are many causes of acute onset facial nerve palsy, Bell's palsy remains a diagnosis of exclusion supported by typical clinical presentation. Inflammation and edema secondary to unknown etiology is the thought to be the pathogenesis of Bell's palsy and corticosteroids significantly improve the outcome due to anti-inflammatory effects.[14]

Imaging is usually performed to rule out other cases of facial nerve palsy. Contrast-enhanced MRI in Bell's palsy may show varying degree of linear enhancement of the canalicular, labyrinthine, and tympanic segments of the facial nerve with may sometimes persists even beyond the clinical improvement. However, care should be taken not to mistake normal facial nerve enhancement (anterior and posterior genu as well as some enhancement of the tympanic and mastoid segments) as pathologic[15] (Fig. 9).

Ramsay Hunt Syndrome

Ramsay hunt syndrome or herpes zoster oticus is an acute onset unilateral peripheral facial nerve palsy accompanied by burning pain in the ear and an erythematous vesicular rash on the pinna, external acoustic meatus, and tympanic membrane. It is secondary to reactivation of varicella zoster virus in the geniculate ganglion. There may be involvement of the cranial nerve (CN8) due to its close proximity to geniculate ganglion and bony facial canal resulting in tinnitus, sensorineural hearing loss, nausea, vomiting, vertigo, and nystagmus.

MRI with contrast shows enhancement in the distal meatal segments of the CN VII and VIII, labyrinthine segment of CN VII, and occasionally enhancement of geniculate ganglion and tympanic and mastoid segments of CN VII (Fig. 10). Enhancement of the cochlea, vestibule, and semicircular canals as well as blister lesions of the external acoustic meatus may also be present.[16,17]

Facial nerve palsy is more severe and less likely to recover completely compared to Bell's palsy. Treatment with corticosteroids and Acyclovir may improve the outcome.

Labyrinthine Hemorrhage

No identifiable pathology is found in over 80% of the cases of sudden onset sensorineural hearing loss.[18] Labyrinthine hemorrhage is one of the causes of sudden sensorineural hearing loss which occurs usually in patients with hematological disorders (such as leukemia, sickle cell anemia,

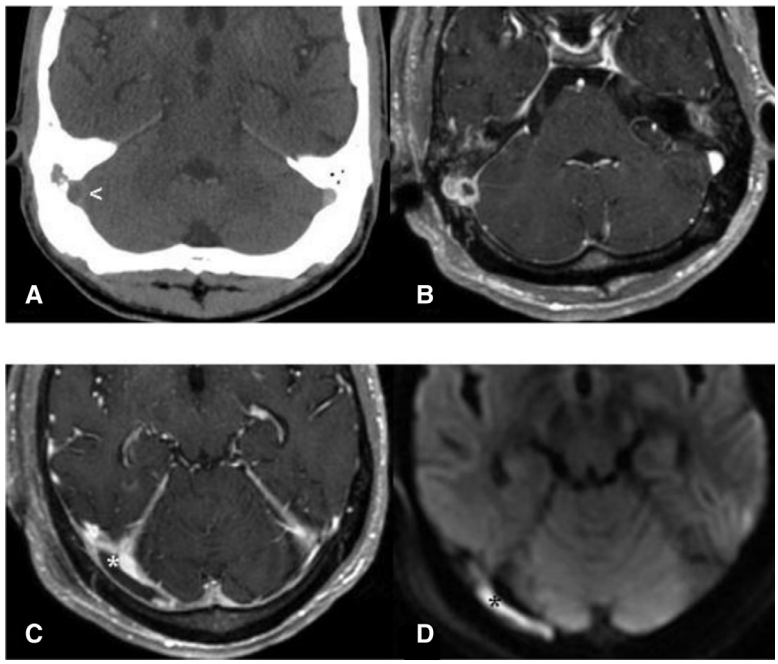

Figure 8 Venous sinus thrombosis: Contrast-enhanced axial CT of the head (A) showing opacified right mastoid air cells in a patient with coalescent mastoiditis with a defect in the medial cortex and contrast filling defect in the subjacent sigmoid sinus (arrowhead). Axial T1-post contrast fat sat MRI (B) demonstrating enhancement in the region of right mastoid with filling defect in the right sigmoid sinus. Axial T1-post contrast fat sat MRI cranial to the level of sigmoid sinus (C) showing a large filling defect (asterisk) in the right transverse sinus with corresponding diffusion restriction on DWI sequences (D). The findings are consistent with dural venous sinus thrombosis secondary to direct spread of infection from the acute coalescent mastoiditis.

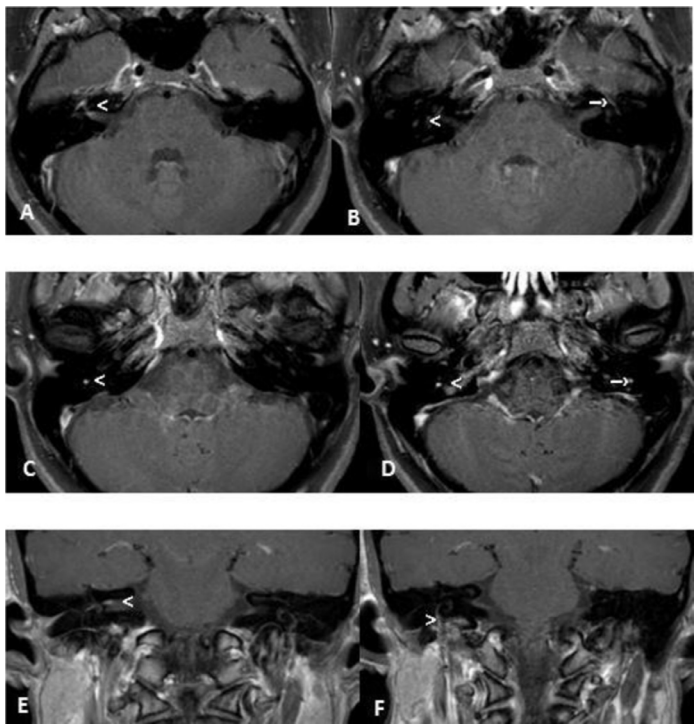

Figure 9 Bell's palsy: 36-years-old female with right sided facial palsy. Axial T1-post contrast fat sat sequences (A through D) through the internal auditory canals demonstrating asymmetrically increased enhancement along the canalicular and labyrinthine segments, first genu, and proximal tympanic segment (A), distal tympanic segment (B), mastoid segment (C-D) of the right facial nerve (arrowheads). Note the normal physiological enhancement of the tympanic (B) and mastoid (D) segments of the left facial nerve (arrow). On coronal T1-post contrast fat sat sequences (E-F) enhancement is seen in the canalicular (E) and mastoid (F) segments of the right facial nerve (arrowheads).

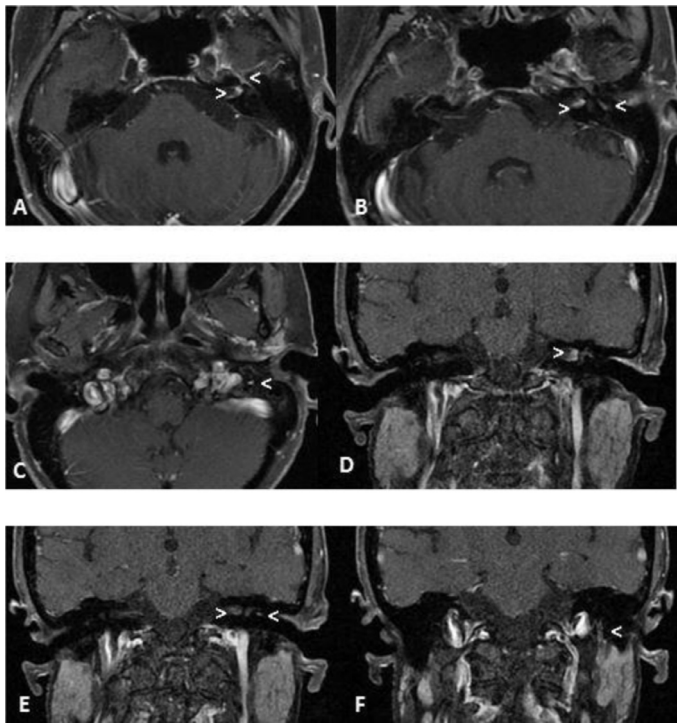

Figure 10 Ramsay hunt syndrome: 56-F with recent history of shingles presents with left facial paralysis. Axial T1-post contrast fat sat MRI through the skull base (A-D) demonstrating abnormal enhancement (arrowheads) along the posterior wall of the left internal auditory canal (IAC), distal aspect of the left IAC, canalicular segment, and first genu of the left facial nerve (A), tympanic segment of the left facial nerve (B), mastoid segment of the left facial nerve (C). On coronal T1 -post contrast fat sat sequences (D-F) similar finding of enhancement in the distal aspect of the left IAC (D), tympanic segment (E), and mastoid segment of the left facial nerve extending through the left stylomastoid foramen (F) are noted. Given the clinical history of recent shingles, the findings are most consistent with Ramsay hunt syndrome.

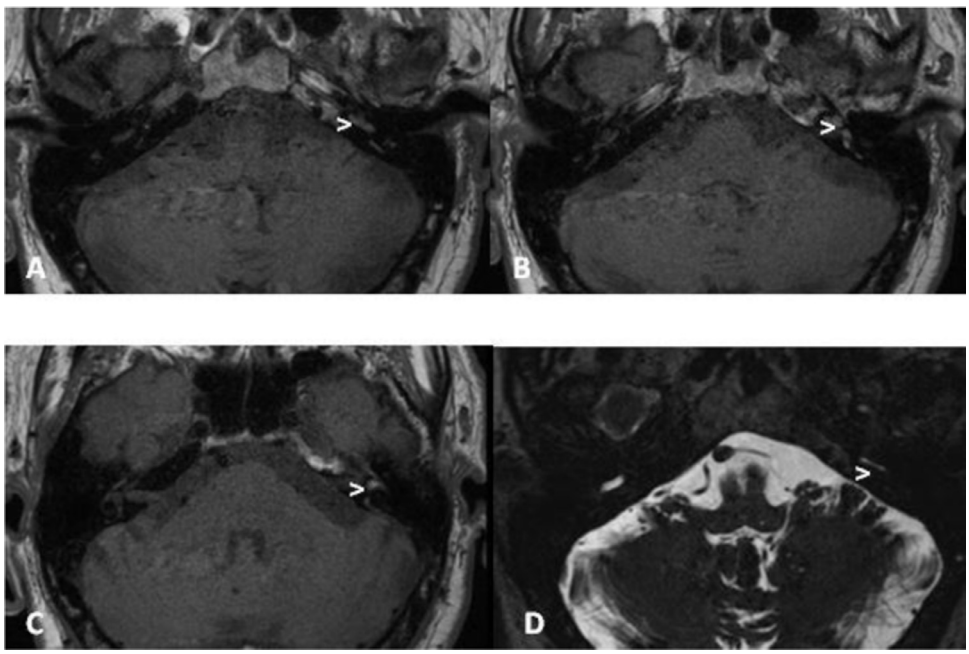

Figure 11 Labyrinthine hemorrhage: 74-year-old male with sudden onset left sided sensorineural hearing loss. Axial T1-pre contrast MRI through the internal auditory canals (A-C) demonstrating subtle increase in T1 signal (arrowheads) in the basal turn of left cochlea (A), Middle/apical turns of left cochlea and vestibule (B) and anterior and posterior limbs of the left superior semicircular canal (C). Axial 3D-FIESTA sequence through the IACs (D) showing decreased fluid signal in the basal turn of left cochlea (arrowhead).

pernicious anemia, or hyperviscosity disorders), anticoagulation medications, trauma, tumors, or labyrinthitis. On MRI hyperintense T1 signal in the membranous labyrinth is visualized which may persist up to 6 months after the onset of symptoms, due to the extended lifespan of erythrocytes in perilymphatic fluid. T2 signal can be variable depending on the age of hemorrhage (Fig. 11).

Acute Labyrinthitis

Acute inflammation of the membranous labyrinth may be secondary to infection (bacterial or viral), trauma or autoimmune process. The route of infection spread may be tympanogenic (middle ear/mastoid infection spread through round/oval window or through a fistula between middle ear and lateral semicircular canal), meningogenic (spread of meningeal/CSF infection through cochlear aqueduct), or hematogenic (via cochlear vasculature).

In acute stage, the perilymphatic space is filled with inflammatory cells which in chronic stages progress to fibroblastic reaction and bone formation. Acute labyrinthitis is considered a medical emergency as the potentially reversible acute stage may rapidly progress to irreversible chronic stage or labyrinthitis ossificans, which precludes the option of cochlear implantation.[19]

Enhancement of the membranous labyrinth is seen in the acute stages, which may however be difficult to appreciate due to intrinsic T1 shortening of the inflammatory secretions within the perilymph. In later stages, when ossification sets in, loss of fluid signal is noted on T2-weighted imaging and calcifications are seen within the labyrinth on CT.[19]

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