Non-traumatic acute temporal bone diseases.

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Learning objectives

to recognize multimodality imaging signs of most common non-traumatic diseases affecting the temporal bone in the acute setting and to perform a correct differential diagnosis, in order to decrease the time it takes to perform correct clinical care.
Background

The temporal bones comprise the lateral skull base, forming portions of the middle and posterior fossae. Each temporal bone is composed of five osseous parts: the squamous, mastoid, petrous, tympanic, and styloid portions.

Temporal bone represents a crucial site of skull base: within temporal canals and foramina are located vascular and nervous structures (petrous internal carotid artery and 7th and 8th cranial nerves), middle ear structures (tympanic membrane, ossicles) and inner ear components (bony and membranous labyrinth, perilymphatic spaces).

Temporal bone is also contigous to very important structures (i.e dural sinuses, meninges, cerebral lobes, cerebellum): pathologic processes involving temporal bone may spread to these structures resulting in dangerous complications.

Non-traumatic acute temporal bone lesions represent a frequent indication for imaging after clinical evaluation, especially to exclude intracranial, potentially fatal, disease or complications. The most common clinical symptoms are: otalgia (with or without otorrhea), sensorineural hearing loss, facial nerve disorders, vertigo and dizziness.

Sudden sensorineural hearing loss (SSNHL) is defined as sudden hearing impairment, of at least 30 dB at three consecutive frequencies occurring over 3 days, and its incidence has been estimated as 5±20/10,000 individuals per year. Although the exact etiology of SSNHL is still debated (such that it is classified as idiopathic), viral infections, immunological diseases, and impairment of the vascular microcirculation have been suggested as possible causes.

Because stroke, malignancies and vestibular schwannoma can give rise to SSNHL, as obvious potential causes they should be addressed in the initial and follow-up management course in all patients with SSNHL.

Vertigo is defined as a false sense of motion; vertigo may be persistent or episodic, with or without hearing loss. Vertigo and dizziness can be symptoms of various diseases, such as: labyrinthitis, Meniere disease, benign paroxysmal positional vertigo, vestibular neuritis, perilymphatic fistulas, migraine, stroke, intracranial tumors. When imaging is performed the most important aim is to identify possible central cause.

Computed tomography (CT) has revolutionized imaging of the temporal bone; multissection CT scanners allow acquisition of high resolution volumetric data that enable image reformation in any plane (multiplanar reconstruction, MPR).

CT is an essential imaging test for the initial evaluation of temporal bone, as a result of its high resolution (HR) for bone assessment and its short acquisition time. CT is usually performed without contrast media administration; intravenous constrast media may be
considered when infectious complications or neoplastic conditions are suspected. CT is very useful because it quickly allows to extend the scan to the whole head (ie intracranial complications).

In many cases it is possible to reach a diagnosis or, at least, to reduce the range of pathological possibilities.

After antero-posterior and lateral topograms, axial CT images are acquired through the temporal bones, with scan excursion from the arquate eminence through the mastoid tip.

At our institution, 0.5 mm axial sections are then reprocessed and reformatted into magnified coronal and sagittal images and displayed at 0.25 mm intervals with overlap. (Figg.1,2,3)

In addition to the traditional axial and coronal views, it has become routine at our institution to also perform reformation in the Stenvers and Pöschl projections. The Stenvers projection is the plane parallel to the long axis of the petrous bone, and the Pöschl projection is the plane perpendicular to the long axis of the petrous bone. (Figg.4,5)

These additional planes are useful for evaluating the structures of the middle and inner ear, which may not be as well seen in the standard axial and coronal planes.

**Magnetic Resonance Imaging (MRI)** is a second level modality to evaluate temporal bone. It is superior to CT in differentiating soft tissue and represents the gold standard to evaluate the fluid-filled spaces of inner ear, nervous structures (facial and vestibulocochlear nerves), that pass through cerebellopontine angle (CPA) and the internal auditory canal (IAC), and the brainstem. MRI is also useful in differentiating soft tissue (ie cholesteatoma) in middle ear and mastoid, petrous apex and external auditory canal (EAC).

MRI is performed using at least 1.5 Tesla magnet with iv gadolinium administration, using sequences and parameters listed in Fig.6.

Axial thin-section heavily T2-weighted imaging sequences and constructive interference in steady state (CISS) MR imaging are obtained through the IAC and pons. This sequence associates high signal with high spatial resolution, thus obtaining excellent visualization of nervous structures lying in fluid spaces (liquor, perilymph and endolymph). CISS sequence allows MPR on coronal and parasagittal planes (perpendicular to IAC long axis). Parasagittal images best demonstrate the facial nerve (CN7) and the vestibulocochlear nerve (CN8).

Non-echo-planar Diffusion-weighted imaging (DWI) sequences, on axial and coronal planes, are superior to conventional echo-planar DWI in temporal bone evaluation, since they minimize susceptibility artifacts at the skull base and increase sensitivity for detection
of lesions as small as 2 mm. DWI technique is indicated when an abscess is clinically suspected and to exclude brain stroke.

CT and MRI are complementary imaging modalities in the evaluation of temporal bone. Radiological approach usually comprises:

- CT for bone structures: external auditory canal, middle ear, osseous labyrinth, osseous canal of cranial nerves

- MRI for soft/fluid structures: fluid filled spaces of inner ear, cranial nerves, cerebellopontine angle, brainstem and soft tissue in middle ear, mastoid and petrous apex aerated cells.
## MULTI-DETECTOR CT SCANNING PARAMETERS

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**Fig. 1:** Multi-detector CT scanner parameters.

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Fig. 2: Standard coronal CT dataset.

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Fig. 3: Standard sagittal CT dataset

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Fig. 4: Stenvers reformats are made in a plane perpendicular to the roof of the SSC.

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Fig. 5: Poschl reformats are made in plane parallel to the axis of the summit of the SSC.
### MRI Sequences & Parameters

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**Fig. 6:** MRI sequences and parameters (1.5 Tesla scanner)

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Findings and procedure details

INFECTIONOUS-INFLAMMATORY LESIONS

Several inflammatory conditions may affect the temporal bone.

The inflammatory diseases of the temporal bone are generally classified according to the site of origin: external ear, middle ear and mastoid, petrous apex and inner ear.

**Necrotizing or malignant otitis externa** occurs most commonly in elderly diabetic patients and other patients in immunocompromised states, as an infection usually caused by *Pseudomonas aeruginosa* high mortality rate. The patients present with severe otalgia and otorrhea. Infection can then spread via fissures of Santorini into the soft tissues beneath the skull base, leading to skull base osteomyelitis.

On images, soft-tissue thickening in the external canal is noted, often with bone destruction and inflammatory changes in the mastoid.

MR and CT are complementary modalities for evaluation of this entity, with the bone windows at CT showing the destructive process to greater advantage, and MR imaging better demonstrating associated soft tissue complications. (Figg. 7, 8, 9, 10, 11)

**Acute otitis media (AOM) and mastoiditis** are primarily diseases of infants and young children, defined as infectious processes of the middle ear, involving a variable portion of the mastoid air cell system. Patients present with fever, otalgia, and a red bulging tympanic membrane.

AOM is often subsequent to a viral upper respiratory infection disrupting the mucosal barrier, thus allowing bacterial inception and growth. Reflux of infected secretions from the nasopharynx to the eustachian tube allows bacteria to reach the middle ear.

Imaging is usually not necessary in uncomplicated acute otitis media, but plays a crucial role in evaluation of intratemporal or intracranial complications.

Noncontrast CT scan shows opacification of the middle ear cavity and mastoid air cells with possible fluid levels; nonspecific debris is seen in acute uncomplicated otitis and mastoiditis. The integrity of osseous structures, including the ossicles, mastoid cortical bone, and mastoid trabeculae, is preserved.
Contrast-enhanced CT and/or MRI are performed when a complication is suspected, including the whole head in the field-of-view. (Figg. 12, 13, 14, 15)

**Petrous apicitis** is an infectious process that occurs in the setting of a pneumatized petrous apex (present in 30% of the population), caused by medial extension of acute otitis media. It is characterized by septal and cortical destruction, osteitis, and adjacent meningeal inflammation. Patients usually present with an acute febrile illness and some or all of the symptoms of the classic Gradensigo 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.

Because the petrous apex cannot be directly visualized, imaging evaluation is mandatory and often critical for patient care, as treatment approaches depend on the specific disease process and the nearby structures involved. (Figg. 16, 17, 18, 19, 20, 21).

**Labyrinthitis** refers to inflammatory disease of the perilymphatic spaces of the inner ear that results in secondary changes within the endolymphatic spaces (membranous labyrinth). It can occur as a result of infections (viral or bacterial) or may be autoimmune. The most common symptoms are SNHL and vertigo.

The evolution of labyrinthitis is characterized by three stages: acute, fibrous, and ossification. In the acute stage, the CT scan is normal, while contrast-enhancement of the inner ear may be noted on T1-weighted MR images; this enhancement is typically faint. In the intermediate fibrous stage there is loss of fluid signal intensity on heavily T2-weighted sequence images with persisting gadolinium enhancement, while the CT scan may still appear normal. In the late ossific stage, the normal cochlea, vestibule, and/or semicircular canals are replaced by bone attenuation on CT scans and appear as low-intensity signal on T2w MRI. (Figg. 22, 23, 24, 25, 26, 27, 28)

**IDIOPATHIC LESIONS**

**Bell's palsy** refers to acute facial nerve paralysis with no identifiable cause. It was long thought to be idiopathic; strong evidence is now present to implicate reactivation of herpes simplex virus or varicella zoster virus (Ramsay Hunt syndrome) with latent infection in the geniculate ganglion.

Contrast-enhanced MRI may demonstrate thickening and increased signal of the facial nerve in the fundal portion of the internal auditory canal or uniformly linear enhancing facial nerve. (Figg. 29, 30)
Perilymphatic fistulas (PLF) is an abnormal communication between the perilymphatic space of the inner ear and the air space of the middle ear, most commonly occurring at either the round or oval window. Patients with PLF have sudden hearing loss, fluctuating sensorineural deficit, vertigo, tinnitus and headache. When a PLF exists, perilymph in the inner ear escapes, driven by the hydrostatic pressure of the cerebrospinal fluid (CSF), and is replaced by CSF. This can also result in lower than normal levels of CSF fluid around the brain. A PLF should be considered in pediatric patients with recurrent meningitis, and the option of middle ear exploration should be pursued. Imaging evaluation is performed to exclude other causes of associated symptoms.

At CT evaluation inner ear anomalies must be reported. Demonstration of pneumolabyrinth is the most important sign, especially if associated to middle ear effusion; careful attention to the stapes superstructure is required, in order to look for any anatomical variants.(Fig.31)

Semicircular Canal Dehiscence (SCD)

Superior semicircular canal dehiscence (SSCD): The SSCD syndrome was first described by Minor et al in 1998 and is characterized by the formation of a "third opening" or "third window" between the superior semicircular canal and the middle cranial fossa, secondary to a bony defect in the canal, with disruption of endolymphatic homeostasis particularly with loud auditory stimulations. It is characterized by vertiginous symptoms, oscillopsia, and rotatory and vertical nystagmus occurring in the setting of loud noises (Tullio phenomenon).

SSCD can be identified with CT, demonstrating absence of bone forming the roof of the superior surface of the canal, best seen at high resolution 0.5 mm collimation and MPR in the plane of the superior canal (Pöschl views) and in a perpendicular plane to the same canal (Stenvers views). The bone defects have been described at the level of the arcuate eminence or in the region of the superior petrosal sinus. Studies have found that magnetic resonance imaging (MRI) is also very useful for diagnosing SSCD syndrome, but it is usually not performed in the acute setting.(Figg.32,33)

Posterior semicircular canal dehiscence (PSCD) is rarely reported compared to SSCD, and has been reported in association with a high jugular bulb or a aberrant arachnoid granulation. PSCD and SSCD may be associated on the same side and this suggests to be due to a congenital maldevelopment of the inner ear.(Figg.34,35)

Spontaneous Petrous Carotid Artery Dissection

Spontaneous dissection of cervical vessels is a well-documented cause of ischemic stroke in young adults.
Spontaneous dissection can occur especially in patients with fibromuscular dysplasia (FMD), cystic medial necrosis, intimal fibroelastic aberrations or connective tissue disorders, e.g. Marfan syndrome.

Dissection is characterized by subintimal (subintimal dissection) or subadventitial (subadventitial dissection) penetration of circulating blood causing vessel narrowing and leading to thromboembolism, pseudo aneurysm formation or both.

Non-contrast brain CT is insensitive for dissection but may demonstrate ischaemic changes within the brain. CT Angiography (CTA) may demonstrate enlargement of the dissected artery and abnormal vessel contour. Classical appearance is: narrowed eccentric lumen surrounded by a crescent-shaped mural thrombus and thin annular enhancement; intimal flap; dissecting aneurysm (pseudoaneurysm). MRI shows: high signal crescent sign within the wall of the vessel (best seen with T1 fat saturation or T2 weighted images), absent flow-void, abnormal vessel contour on MR Angiography (MRA) and evidence of cerebral ischaemia. (Fig.36, 37)

**MISCELLANEOUS**

**Petrous apex aerated lesion**

In Fig.38 is reported the case of a young man who was admitted at the emergency department for left trigeminal palsy and headache, with suspected cervical artery dissection; no significant trauma was referred. CTA was negative for acute vascular lesions and brain stroke, but the left petrous apex showed air bubbles (not present in a 2 years previous CT examination), which compress the left Meckel cave. After 6 months, next CT scan showed air resorption and resolution of trigeminal palsy.

**Iatrogenic lesion**

In Fig.39 is reported the case of a 15 years old boy with pneumococccic meningitis after middle cranial fossa hadrontherapy for recurrent/residual sellar lesion (previously surgically treated - pituitary macroadenoma). Imaging demonstrate destruction of cortical bone of the right petrous apex which allowed intracranial bacterial spread from temporal bone and CSF leak into the middle ear, found at clinical evaluation.

**Surgical screws mobilization**

In Fig.40 is reported the case of a 58 years old woman with right retroauricular swelling who received cochlear implant 7 years before. CT scan demonstrated subgaleal fluid collection of temporal and retroauricular region on the right side, with mobilization of surgical screws. The Patient underwent surgical toilette of the fluid collection and cochlear implant reprise (Fig.41).
Fig. 7: Necrotizing otitis externa: axial (A) and coronal (B) CT images (bone window) show soft tissue filling left EAC, thickening of the tympanic membrane and diffuse otomastoid debris, in a diabetic patient.

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**Fig. 8:** Necrotizing otitis externa (same patient in Fig. 7): axial post contrast CT image (A) shows pathologic heterogenous enhancement of rhino-pharinx spreading to both masticatory spaces, demonstrating fluid collections (abscesses), due to inflammatory extension from the left ear to skull base. Axial T2 TSE FS (B) and axial post contrast VIBE (C) images better demonstrate reactive bone edema (clivus and occipital condyles) with thickening and contrast uptake of the dura. In B fluid effusion in left temporo-mandibular joint is also evident.

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**Fig. 9:** Necrotizing otitis externa (same patient in Fig. 7): axial post contrast CT images show left otomastoid debris and irregular thinning of antero-lateral aspect of petrous carotid canal, best seen with bone window (A).

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Fig. 10: Necrotizing otitis externa (same patient in Fig. 7): coronal (A) and parasagittal (B) MIP MR angiography images show regular patency and symmetrical lumen of intracranial ICAs.

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Fig. 11: Necrotizing otitis externa (same patient in Fig. 7): A: sagittal post contrast CT image on the left side shows fluid effusion in the temporo-mandibular joint, best seen under articular eminence. B: coronal post contrast VIBE image demonstrates diffuse extension of inflammatory process to rhinopharinx, peri-vertebral and masticatory spaces. C: coronal T2 TSE FS image reveals fluid collection within the palate mucosa and adjacent muscle edema.

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Fig. 12: Acute oto-mastoiditis: Axial CT image (bone window, A) shows left oto-mastoiditis with cortical bone erosion of the sigmoid plate of left temporal bone. B: Axial post-gadolinium VIBE image in the same Patient confirms intracranial extension of the infectious disease, seen in the left cerebellum, and well demonstrate linear contrast enhancement of left IAC segment of 7th and 8th cranial nerves.
**Fig. 13:** Intracranial complications of otomastoiditis (same Patient in Fig.12): Axial post contrast CT image (A) shows multiple foci of meningitis and cerebritis (gyriform iuxtacortical contrast enhancement), in both cerebral hemispheres. Axial FLAIR (B) and VIBE images (C) confirm intracranial extension of the infectious disease and better demonstrate right frontal subdural empyema and diffuse dural hyperemia.
**Fig. 14:** Otomastoiditis with intracranial complications: A, B: axial and sagittal CT images (bone window) show right oto-mastoid debris, loss of mastoid septations and erosion of tegmen tympani and tegmen antri. C: axial post contrast VIBE reveals a non enhancing defect within the lumen of right sigmoid sinus, representing sinus thrombosis. D: sagittal FLAIR image show area of increased signal in the right temporal lobe, due to cerebritis.

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Fig. 15: Otomastoiditis with intracranial complications (same Patient in Fig.14): A, B, C, D: coronal multiparametric MRI images show area of restricted diffusion in the inferior aspect of right temporal lobe with heterogeneous enhancement representing cerebral abscess. E, F: coronal CT images (E, soft tissue, and F, bone window) show oto-mastoid debris and erosion of tegmen tympani and area of hypodensity in the adjacent temporal lobe due to infectious edema.

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Fig. 16: Left otomastoiditis with apical petrositis (Gradenigo’s syndrome): axial ADC and DWI (A,B) show area of restricted diffusion on left petrous apex that represent an abscess, with intense peripheral enhancement on axial post-gadolinium VIBE (D). Axial T1SE-weighted image(C) demonstrates asymmetry of intracranial ICAs, with left side narrower than right one.

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Fig. 17: Left otomastoiditis with apical petrositis - Gradeneigo's syndrome (same patient in Fig. 16): axial (A) and coronal (B) post-contrast VIBE images show clival enhancement (osteitis) and swelling and enhancement of trigeminal ganglion in left Meckel cave (and its mandibular division), due to bone and perineural extension of flogistic process.

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**Fig. 18:** Left otomastoiditis with apical petrositis (same patient in Fig. 16): A: MRI angiography in the acute phase of Gradenigo’s syndrome shows asymmetric but patent intracranial ICAs, with the left side narrower than the right one. B: MRI angiography performed after 3 weeks shows symmetric lumen of both ICAs.

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Fig. 19: Left otomastoiditis with apical petrositis - Gradenigo’s syndrome (same patient in Fig. 16). A: Axial CT image (bone window) after 1 week of antibiotic and steroid therapy shows opacification of petrous apex and decreased bone density due to osteopenia. B: Axial CT image (bone window) 7 months later shows aerated petrous apex cells and restoration of bone density.

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Fig. 20: Right otomastoiditis with apical petrositis: axial DWI and ADC (A,B) show area of restricted diffusion on right petrous apex with fluid signal on axial T2 TSE image (C) and intense peripheral enhancement on axial post-gadolinium VIBE (D), representing an abscess.

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Fig. 21: Right otomastoiditis with apical petrositis (Same Patient in Fig. 20): CT images (bone window) show cortical disruption of right petrous apex (A), involving posterior wall of horizontal segment of petrous ICA canal (B), anterior wall of IAC (A) and clivus (D).

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**Fig. 22:** Labyrinthitis: axial CISS (A) and post contrast VIBE (B) images through inner ear show left vestibular low signal intensity of the fluid filled space and marked contrast enhancement.

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Fig. 23: Labyrinthitis (same Patient in Fig.22): axial CISS (A) and post contrast VIBE (B) images through inner ear show basal turn of the left cochlea and Posterior Semicircular Canal low signal intensity of the fluid filled space and marked contrast enhancement.

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Fig. 24: Labyrinthitis (same Patient in Fig.22): coronal CISS (A) and post contrast VIBE (B) images through inner ear show left vestibular and Superior Semicircular Canal low signal intensity of the fluid filled space and marked contrast enhancement.

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Fig. 25: Labyrinthitis ossificans (same patient in Fig.22): axial CT (bone window) through Lateral Semicircular Canal (LSC), before (A) and 7 months after (B) bilateral cochlear implant in Patient with rapidly progressive earing loss and rheumatologic pathology (systemic vasculitis); in (B) bilateral LSCs show almost complete ossification of the fluid-filled spaces.

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Fig. 26: Labyrinthitis: Coronal CISS (A) and post contrast VIBE (B) reformatted images demonstrate low signal intensity of the basal turn of the right cochlea which enhances after gadolinium administration.

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Fig. 27: Labyrinthitis (same Patient in Fig. 26): para-sagittal right side CISS (A) and post contrast VIBE (B) reformatted images demonstrate low signal intensity of the basal turn of the cochlea which enhances after gadolinium administration.

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**Fig. 28:** Labyrinthitis (same Patient in Fig. 26): axial post contrast VIBE demonstrates intense contrast enhancement of the basal turn of the right cochlea.

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Fig. 29: Bell's palsy: Axial CISS (A) and post-gadolinium VIBE (B) images show symmetrical appearance and course of facial nerves, with linear contrast uptake of the left facial nerve.

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**Fig. 30:** Drug neuropathy in a Patient who develops acute right sensorineural hypoacusia after pharmacological therapy with high doses of aminoglycosides. Axial post-gadolinium VIBE images before (a) and 20 days after (b) pharmacological therapy: in b there is marked linear contrast uptake of the right IAC segment of C7 and C8 nerves.

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Fig. 31: Stapes anomaly in a Patient with suspected Perilymphatic fistulas: Axial CT images (bone window) al level of oval window show asymmetry of the two crura of the right stapes (A), with abnormal archway appearance (the anterior crus is greater than the posterior one) best demonstrated in magnified image (C). On the left ear (B, D) the stapes is normal.

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**Fig. 32:** SSC dehiscence: multiplanar CT images (bone window) of right ear show cortical defect at the superior surface of SSC (red arrows).

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**Fig. 33:** SSC dehiscence: multiplanar CT images (bone window) of left ear show focal cortical defect at the posterior surface of SSC (red arrow in A and B).

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**Fig. 34:** PSC dehiscence: multiplanar CT images (bone window) of right ear show ectatic jugular bulb with focal cortical defect at the inferior surface of PSC (red arrow in A and C).

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**Fig. 35:** LSC dehiscence: Multiplanar CT images of right ear show chronic otitis media with focal cortical defect of the anterior surface of LSC (red arrows).

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Fig. 36: Petrous Carotid Artery Dissection: CT Angiography reformatted images on axial and coronal planes demonstrate parietal lesion of the petrous segment of left internal carotid artery, which appears narrower than the right side, thus determining vascular stenosis.

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Fig. 37: Petrous Carotid Artery Dissection (same Patient in Fig.36): CT Angiography reformatted images on paracoronal and sagittal planes demonstrate parietal lesion of the petrous segment of left internal carotid artery, which appears narrower than the right side, thus determining vascular stenosis.

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**Fig. 38:** Petrous apex aerated lesion: CT reformatted images (bone window) on coronal plane through petrous apex documented air bubbles on the left side (B), probably compressing Meckel cave, which were absent 2 years before (A) and disappeared 6 months later (C).

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Fig. 39: Iatrogenic lesion: axial post contrast VIBE image (A) documents sellar lesion extending on left parasellar region and, posteriorly, in the prepontine cistern. Axial post contrast CT images (soft tissue window B - bone window C) show cortical bone destruction of the right petrous apex (red arrow in B and C).

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Fig. 40: Surgical screws mobilization: axial CT (bone window A, C) and axial MIP (B) images clearly demonstrate that surgical screws are not fixed in right the temporal bone due to inflammatory swelling of extracranial soft tissue (D).

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**Fig. 41:** Surgical screws mobilization (same Patient in Fig. 40): A: Waters X-ray view; B: Modified Stenvers X-ray view; C: Lateral X-ray view. After surgical toilette of the right temporal sieroma and cochlear implant reprise, X-ray evaluation demonstrates correct position of implant and surgical screws.

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Conclusion

In the acute scenario radiologist plays a central role in clinical decision making regarding non-traumatic temporal bone lesions and complications.
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