

Pulsatile Tinnitus: A Multimodality Imaging Review of Etiology and Diagnostic Evaluation

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Abstract

Pulsatile tinnitus is a rhythmic, pulse-synchronous auditory perception that is frequently associated with an underlying vascular or structural abnormality. While tinnitus affects approximately 10–20% of adults, pulsatile tinnitus is relatively uncommon but clinically significant, with an identifiable and potentially treatable cause detected in over 70% of patients and in up to 90% in dedicated imaging cohorts. The etiologic spectrum is broad and includes vascular, structural, systemic, benign, and malignant conditions, some of which may result in serious morbidity if unrecognized. Imaging is central to diagnosis and management, with evaluation commonly performed using high-resolution temporal bone CT, CT angiography and venography, MRI with angiographic and venographic techniques, and digital subtraction angiography in selected cases. This pictorial review integrates multimodality imaging with representative case examples to provide a structured, physiology-based diagnostic approach. By emphasizing key imaging patterns across major etiologic categories, this review aims to enhance diagnostic confidence, streamline imaging workup, and facilitate recognition of actionable causes, thereby supporting timely intervention and improved patient outcomes.

Keywords: Pulsatile tinnitus; Multimodality imaging; CT angiography; MR angiography; Vascular abnormalities; Temporal bone imaging

1. Introduction

Tinnitus is the perception of sound in the absence of an external auditory stimulus and is a common condition, affecting approximately 10–15% of the population, with up to 70–75% of individuals experiencing tinnitus at some point during their lifetime [1]. While most cases are non-pulsatile and sensorineural in origin, pulsatile tinnitus (PT) represents a distinct and clinically important subtype characterized by rhythmic auditory perception synchronous with the cardiac cycle. Although PT accounts for less than 10% of tinnitus cases, it is of particular clinical significance because of its association with underlying vascular and structural abnormalities [2].

Unlike non-pulsatile tinnitus, which is typically subjective, PT may be subjective or objectively audible and is frequently related to altered or turbulent blood flow, with transmission of haemodynamic vibrations to the auditory apparatus [2]. Consequently, PT has a relatively high likelihood of an identifiable underlying cause. An aetiology can be established in approximately 70–80% of patients, with diagnostic yield increasing when a comprehensive multimodality imaging approach is employed [2,3].

The aetiological spectrum is broad and encompasses venous, arterial, and arteriovenous abnormalities, as well as structural and neoplastic conditions. Venous abnormalities, particularly venous sinus stenosis and conditions associated with intracranial hypertension, are increasingly recognised as important causes. Arterial atherosclerotic disease and vascular variants may also produce PT, while structural abnormalities and hypervascular tumours, such

as paragangliomas, constitute additional important differentials. Although less common, high-flow vascular lesions, particularly dural arteriovenous fistulas (dAVFs) and arteriovenous malformations (AVMs), require prompt recognition because of their potential neurological morbidity [2,3].

Clinical evaluation can provide useful clues to the underlying haemodynamic mechanism. A low-pitched sound that diminishes with ipsilateral jugular compression may suggest a venous origin, whereas a high-pitched bruit or persistent pulse-synchronous symptoms may raise suspicion for an arterial or arteriovenous source. Otoscopic examination may reveal a retrotympanic vascular or soft-tissue lesion. Nevertheless, clinical assessment alone is often insufficient to establish the underlying diagnosis, particularly when otologic examination is unremarkable [2].

Imaging, therefore, plays a central role in the diagnostic evaluation of PT. High-resolution temporal bone CT, CT angiography/venography, MRI, MR angiography/venography, and, when indicated, digital subtraction angiography provide complementary information for identifying the underlying abnormality and assessing its clinical significance [2,3]. This case-based pictorial review illustrates the multimodality imaging spectrum of PT across arterial, venous, arteriovenous, structural and osseous, neoplastic, systemic, and selected nonvascular mimics, emphasising characteristic imaging findings, important diagnostic pitfalls, and clinically relevant features that influence further investigation and management.

2. Imaging Modalities and Diagnostic Strategy

Imaging is central to the evaluation of pulsatile tinnitus (PT), as the underlying abnormality may involve the temporal bone, arterial or venous circulation, intracranial vasculature, or skull base. Because no single technique can adequately assess the full spectrum of PT etiologies, imaging should be tailored to the clinical presentation, otoscopic findings, and suspected pathophysiological mechanism. High-resolution CT (HRCT), CTA/CTV, MRI with MRA/MRV, and digital subtraction angiography (DSA) provide complementary anatomical and haemodynamic information, while dynamic techniques such as 4D-CTA and time-resolved MRA may further characterize suspected arteriovenous shunting. The principal indications, protocol considerations, and diagnostic strengths of each modality are summarised in **Table 1**.

Table 1. Imaging modalities and their roles in the evaluation of pulsatile tinnitus

Modality	Principal indications	Suggested protocol/components	Principal utility	diagnostic
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HRCT temporal bone	Retrotympenic lesion; suspected osseous abnormality or vascular variant	Submillimetre high-resolution algorithm; reformations, including Pöschl and Stenvers planes when indicated	acquisition; bone multiplanar including	Optimal assessment of temporal-bone anatomy, dehiscence, SS CD, otosclerosis, jugular bulb abnormalities, aberrant ICA, persistent stap edial artery, and ossicular erosion
CTA/CTV	Suspected arterial or venous pathology; persistent PT with normal otoscopy	Thin-section enhanced acquisition with arterial and/or venous phase imaging; multiplanar and vascular reconstructions	contrast-	Demonstrates arterial stenosis, aneurysm, dissection and FMD; evaluates venous sinus abnormalities and sigmoid sinus wall relationships; may demonstrate indirect features of arteriovenous shunting
4D-CTA	Suspected dAVF or AVM; characterization of arteriovenous shunting	Multiphase acquisition with reconstruction	dynamic temporal	Demonstrates early venous filling and shunt haemodynamics and can assist localization of arteriovenous shunts

MRI/MRA/MRV	Persistent PT with normal T1, T2, FLAIR and DWI; Comprehensive radiation-otoscopy; suspected post-contrast 3D T1; TOF-free evaluation; excellent intracranial, venous, MRA; MRV; time-resolved soft-tissue characterization; vascular or neoplastic MRA and ASL when evaluates IIH-associated pathology indicated findings, venous sinus stenosis, tumours and vascular abnormalities; advanced sequences may improve detection of arteriovenous shunting
DSA	Persistent suspicion of Selective catheter Reference standard for dAVF or other vascular angiography with dynamic characterization of dAVFs shunt despite assessment of arterial and and AVMs; defines arterial negative/equivocal non- venous phases feeders, fistulous point, invasive imaging; detailed venous drainage and cortical venous reflux; characterization and enables endovascular treatment planning
Carotid duplex ultrasound	Suspected extracranial Spectral and colour Doppler Rapid and noninvasive carotid disease; patients assessment of extracranial assessment of extracranial with relevant vascular risk carotid circulation carotid stenosis; limited for factors intracranial and skull-base pathology

Abbreviations: ASL, arterial spin labelling; AVM, arteriovenous malformation; CTA, computed tomography angiography; CTV, computed tomography venography; dAVF, dural arteriovenous fistula; DSA, digital subtraction angiography; DWI, diffusion-weighted imaging; FLAIR, fluid-attenuated inversion recovery; FMD, fibromuscular dysplasia; HRCT, high-resolution computed tomography; ICA, internal carotid artery; IIH, idiopathic intracranial hypertension; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; MRV, magnetic resonance venography; PT, pulsatile tinnitus; SSCD, superior semicircular canal dehiscence; TOF, time-of-flight.

The major etiological categories, representative entities, and proposed mechanisms of PT are summarised in **Table 2** and provide the framework for the case-based imaging review that follows.

Table 2. Classification of the major causes of pulsatile tinnitus

Category	Representative causes	Principal mechanism of PT
Arterial	Atherosclerotic carotid/vertebral stenosis; arterial dissection; fibromuscular dysplasia; ICA/vertebral aneurysm; aberrant ICA; persistent stapedial artery; AICA vascular loop	Turbulent arterial flow or direct transmission of arterial pulsations
Venous	IIH and venous sinus stenosis; sigmoid sinus diverticulum/dehiscence; jugular bulb abnormalities; IJV compression; cerebral venous sinus thrombosis; prominent emissary veins	Turbulent venous flow, altered venous drainage, or elevated intracranial venous pressure
Arteriovenous	Dural arteriovenous fistula; arteriovenous malformation; carotid–cavernous fistula	High-flow arteriovenous shunting and arterialization of venous drainage
Structural/osseous	Otosclerosis; semicircular canal dehiscence; cholesteatoma; cholesterol granuloma; petrous apex lesions; other temporal-bone defects	Altered sound conduction, third-window effects, or loss of normal osseous insulation
Neoplastic/hypervascular	Paraganglioma; endolymphatic sac tumor; hemangioma; meningioma; schwannoma; temporal-bone/skull-base metastasis	Tumor vascularity, vascular or venous compression, osseous erosion, or neurovascular displacement

Systemic/functional	Anemia; hyperthyroidism; pregnancy/high-output states; middle-ear or palatal myoclonus; spontaneous intracranial hypotension; Chiari I malformation	Increased systemic flow, muscular activity, or altered CSF/venous dynamics
Nonvascular mimics	Temporomandibular joint dysfunction; Eustachian tube dysfunction; somatosensory or rhythmic non-pulsatile tinnitus	Nonvascular mechanical or somatosensory phenomena perceived as pulsatile

Abbreviations: AICA, anterior inferior cerebellar artery; CSF, cerebrospinal fluid; ICA, internal carotid artery; IIH, idiopathic intracranial hypertension; IJV, internal jugular vein; PT, pulsatile tinnitus.

The diagnostic imaging strategy for PT is outlined in **Fig. 1**.

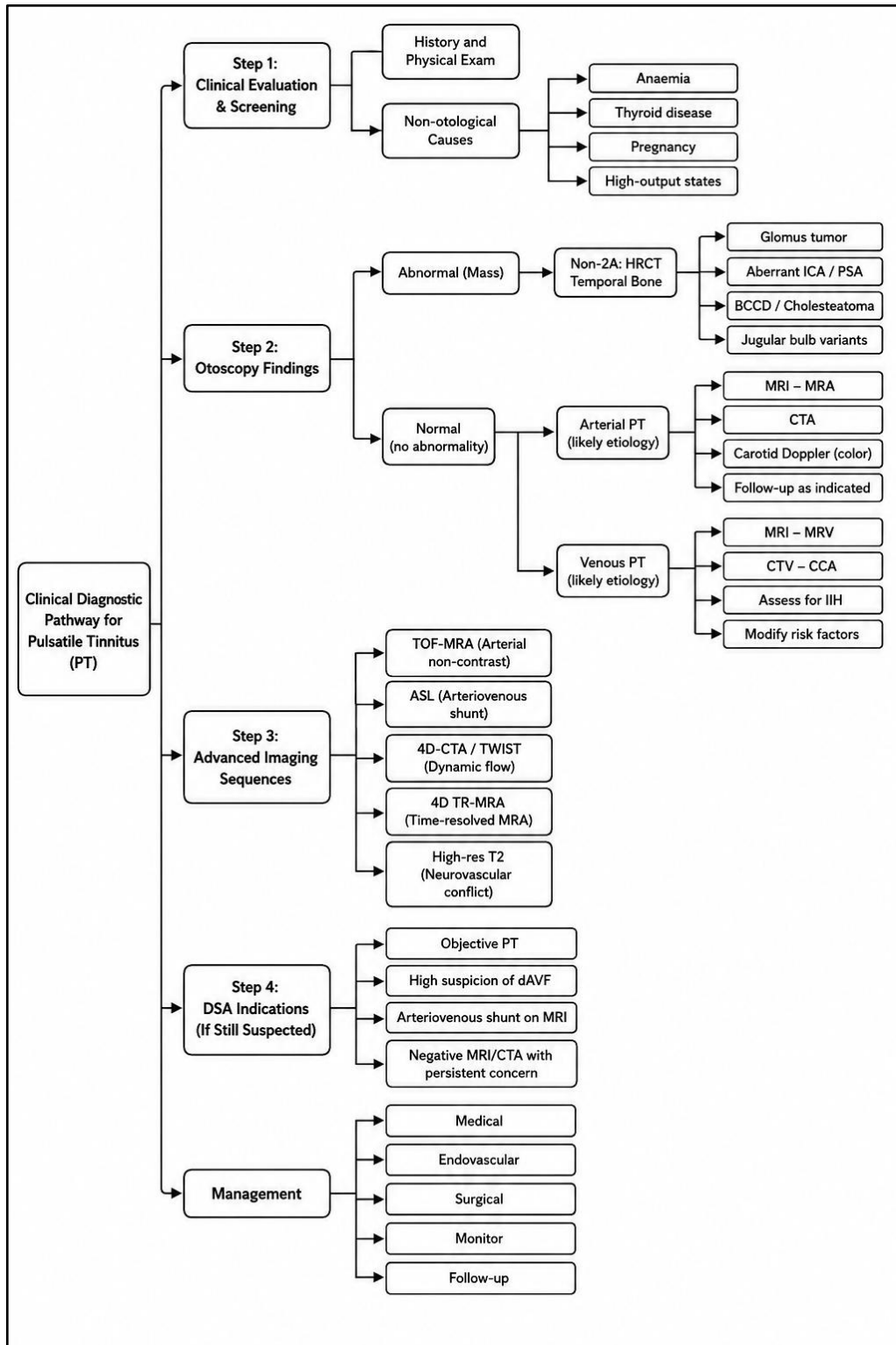


Fig. 1. Diagnostic imaging strategy for pulsatile tinnitus.

3. Case-Based Imaging Spectrum of Pulsatile Tinnitus

3.1. Arterial Etiologies

Arterial PT results from abnormal or turbulent arterial flow and may arise from stenosis, dissection, dysplasia, aneurysms, or congenital vascular variants. CTA and MRA are the principal non-invasive techniques for evaluating suspected arterial causes [4,5].

3.1.1. Atherosclerotic Carotid or Vertebral Artery Stenosis

Atherosclerosis of the extracranial ICA or vertebral arteries is one of the most common arterial causes of PT in the elderly. Progressive plaque formation—calcified, fibrofatty, or mixed—produces luminal narrowing and turbulent post-stenotic flow. CTA and MRA demonstrate focal stenosis, eccentric or concentric plaque, and post-stenotic dilatation, while Doppler ultrasound shows elevated PSV/EDV and spectral broadening (**Fig. 2**). PT may precede neurological symptoms, and identification of significant stenosis has management implications including antiplatelet therapy, statins, or revascularisation.

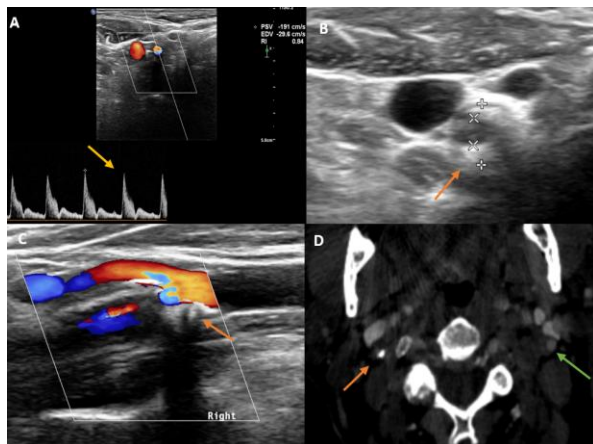


Fig. 2. (A to C) USG, Carotid Doppler images, (D) CT angiogram of neck. There is a post-stenotic elevated PSV at the right proximal ICA (golden arrow in A) caused by mixed calcific and soft plaque (orange arrow in B, C, D), causing significant (approximately 80%) diameter stenosis. Note the normal ICA on the left side (green arrow in D)

3.1.2. Carotid or Vertebral Artery Dissection

Cervical arterial dissection is an important cause of acute unilateral PT and may occur with headache, neck pain, or Horner syndrome [5,6]. CTA/MRA may demonstrate tapered stenosis, an intimal flap, double lumen, pseudoaneurysm, or occlusion, with an intimal flap directly visualised on CTA and TOF MRA (**Fig. 3**). Fat-suppressed T1-weighted MRI can additionally demonstrate mural haematoma [5].

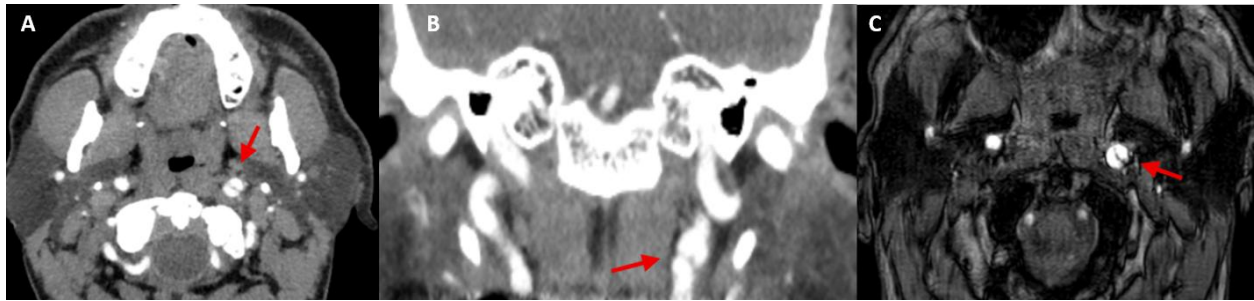


Fig. 3. (A, B) CT angiogram, (C) MRI TOF angiogram of head and neck. There is a hypodense focal intimal flap in the distal ICA at the C1 level of neck (red arrow in A and B). There is no thrombosis or differential contrast opacification between the true and false lumens on the CT angiogram of the head and neck. A thin curvilinear hypointense intimal flap is also seen on the MR TOF image (red arrow in C).

3.1.3. Fibromuscular Dysplasia

Fibromuscular dysplasia (FMD) is a non-atherosclerotic, non-inflammatory arteriopathy affecting medium-sized arteries, particularly the extracranial carotid and vertebral arteries. PT is a recognised manifestation of cerebrovascular FMD [7]. CTA or MRA typically demonstrates alternating areas of stenosis and dilatation, producing the characteristic “string-of-beads” appearance (**Fig. 4**). FMD is also associated with arterial dissection and aneurysm formation [7].

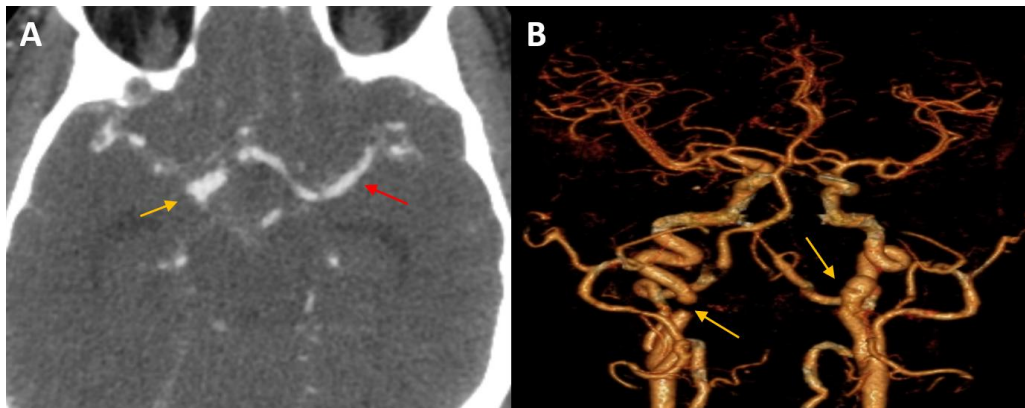


Fig. 4. (A) CT angiogram of the head shows multifocal narrowing in the left MCA (red arrow) and focal dilatation in the right MCA (golden arrow) in a proven case of FMD. (B) 3D MVR shows focal dilatation and stenosis in bilateral cervical ICA (golden arrow).

3.1.4. Aberrant Internal Carotid Artery

An aberrant ICA is a rare congenital vascular anomaly that may present with PT, conductive hearing loss, or a retrotympanic vascular mass. HRCT demonstrates the anomalous course of the vessel within the middle ear, associated

carotid canal abnormalities, and its close relationship to the cochlear promontory (**Fig. 5**) [8]. Preoperative recognition is essential because inadvertent biopsy or middle-ear surgery may cause severe haemorrhage.

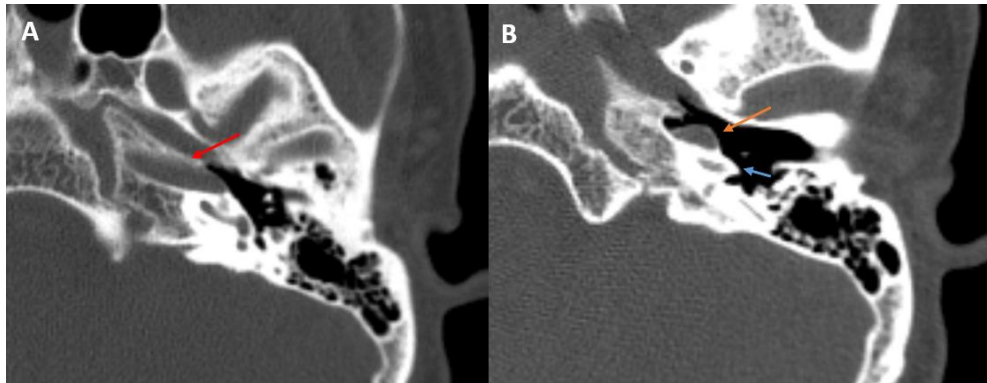


Fig. 5. (A and B) HRCT of left temporal bone axial sections show an aberrant course of petrous ICA anterior and medial to middle ear cavity (red arrow). It is entering the middle ear cavity with dehiscence and thinning of the bony canal at the lateral aspect (orange arrow). It also reveals a close association with the cochlear promontory (blue arrow).

3.1.5. Persistent Stapedial Artery

Persistent stapedial artery (PSA) is a rare embryologic vascular variant that may cause PT or conductive hearing loss. HRCT may demonstrate a small vascular channel traversing the middle ear, often between the facial nerve canal and cochlear promontory, with absence of the ipsilateral foramen spinosum (**Fig. 6**) [9,10]. Accurate recognition is important before middle-ear surgery because of the risk of vascular injury.

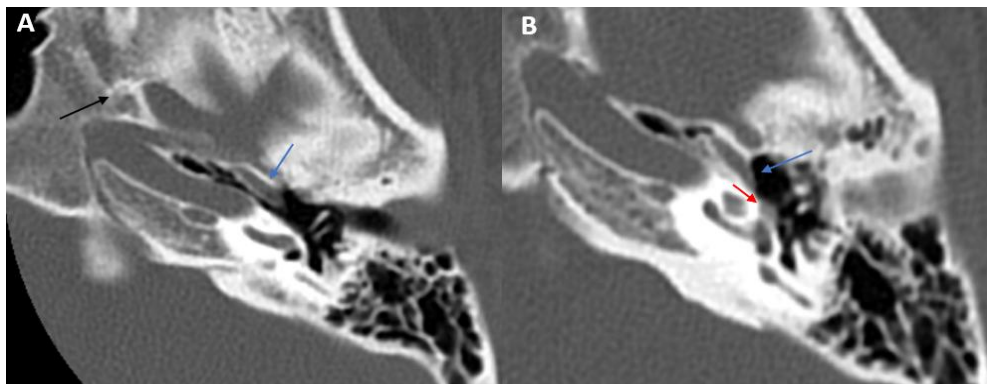


Fig. 6. (A and B) HRCT of temporal bone axial sections shows thin tubular soft tissue density (blue arrow) crossing the middle ear, extending between the tympanic segment of facial nerve and cochlear promontory. It is also seen exiting the middle ear cavity through the anterior wall near the tympanic portion of the internal carotid artery (red arrow). Absent ipsilateral foramen spinosum (black arrow). Features were suggestive of persistent stapedial artery.

3.1.6. Arterial Aneurysms

Aneurysms involving the ICA, vertebrobasilar system, or AICA may rarely cause PT through turbulent flow or transmission of arterial pulsations to adjacent auditory structures [5]. CTA and MRA characterise aneurysm location and morphology, including terminal and petrous ICA aneurysms (**Fig. 7**), while DSA provides detailed assessment when endovascular treatment is considered [5].

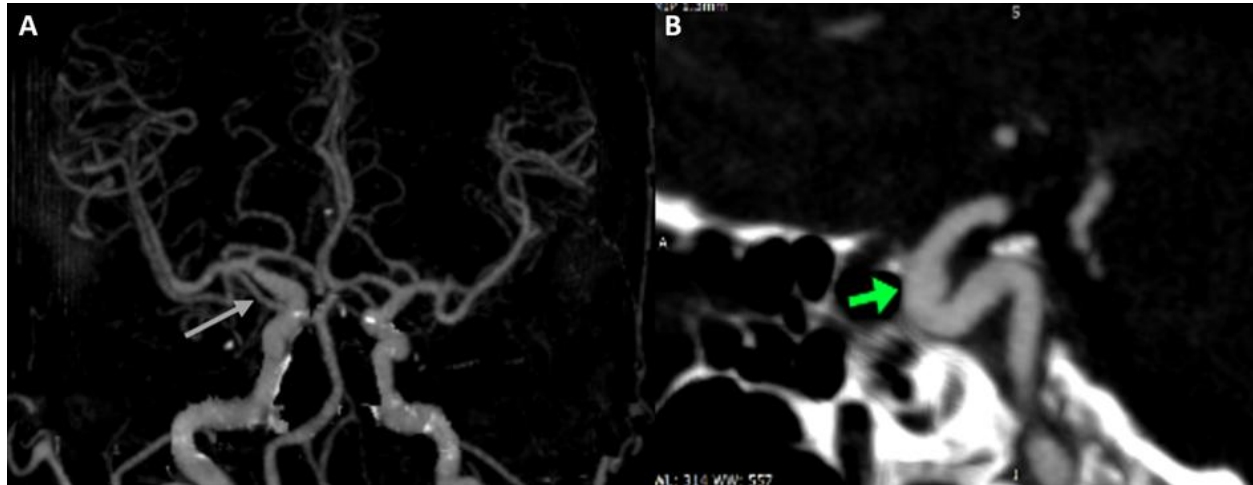


Fig. 7. (A) MRI TOF 3D Brain angiogram in a hypertensive patient with pulsatile tinnitus revealed a fusiform aneurysmal dilatation of the right terminal ICA without neck or thrombus, confirmed on DSA. (B) CT angiogram of the head, sagittal view of another patient with pulsatile tinnitus, showed a small saccular aneurysm in the petrous ICA abutting the petrous temporal bone.

3.1.7. Neurovascular Contact (AICA Loop)

Neurovascular contact between an anterior inferior cerebellar artery (AICA) loop and the vestibulocochlear nerve has been proposed as a cause of tinnitus through direct neural compression or transmission of arterial pulsations [11,12]. High-resolution 3D T2-weighted MRI sequences enable detailed assessment of vascular loops and their relationship to cranial nerve VIII (**Fig. 8**) [13]. However, vascular loops and neurovascular contact are common anatomical variants, and their association with tinnitus remains uncertain [12,13]. Importantly, a recent case-control study found no significant association between unexplained unilateral PT and the presence, depth, or angulation of internal auditory meatus vascular loops or vestibulocochlear neurovascular contact [13]. Therefore, these findings should be interpreted cautiously and only after more established causes of PT have been excluded.

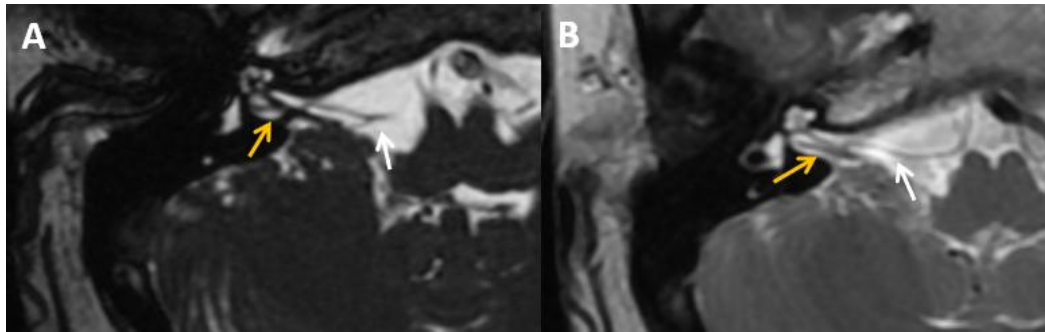


Fig. 8. (A) FIESTA 3D axial sequence, (B) T2W axial cut shows grade I neurovascular conflict with AICA vessel loop (white arrow) crossing over the VII and VIII nerves (orange arrow) indenting and mildly displacing them (white arrow).

3.2 Venous Etiologies

Venous abnormalities represent the most commonly identified causes of pulsatile tinnitus (PT) and include transverse and sigmoid sinus stenosis, sigmoid sinus wall abnormalities, jugular bulb anomalies, and abnormal emissary venous drainage [14]. Venous PT is typically perceived as a low-frequency humming or whooshing sound that may vary with head position and can be reduced or abolished by ipsilateral jugular compression, reflecting modulation of venous flow and pressure [14].

3.2.1. Idiopathic Intracranial Hypertension (IIH)

Idiopathic intracranial hypertension (IIH) is an important cause of venous PT, with PT reported in a substantial proportion of affected patients. Elevated intracranial pressure may contribute to transverse sinus stenosis, altered venous drainage, and turbulent flow, although the relationship between intracranial pressure and venous sinus stenosis may be bidirectional [14].

MRI findings supportive of IIH include a partially or empty sella, distension of the perioptic CSF spaces, tortuosity of the optic nerves, and flattening of the posterior globe (**Fig. 9A–D**). Additional findings may include prominence of Meckel's caves and other osseous or venous changes associated with chronically elevated intracranial pressure. Slow flow or thrombosis may coexist within the transverse sinus (**Fig. 9E**), while MRV or CTV frequently demonstrates transverse sinus stenosis, often involving the transverse–sigmoid junction (**Fig. 9F**) [14].

In patients with venous PT, including those without typical clinical manifestations of IIH, subtle or isolated venous sinus stenosis may be present. Advanced techniques such as arterial spin labelling (ASL) and 4D-flow MRI can further characterise altered cerebral perfusion and venous haemodynamics. Management of IIH includes weight reduction, acetazolamide, and other measures directed at reducing intracranial pressure, while venous sinus stenting may be considered in appropriately selected patients with significant venous sinus stenosis and a trans-stenotic pressure gradient. Improvement or resolution of PT following reduction of intracranial pressure or correction of venous outflow obstruction further supports the haemodynamic basis of the symptom

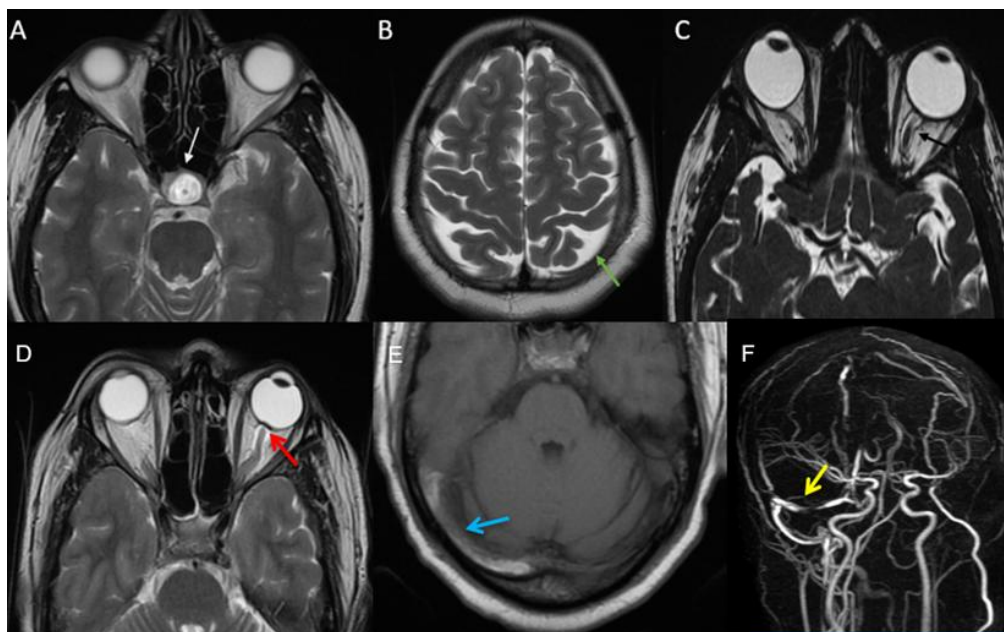


Fig. 9. (A and B) T2W axial sections of the brain reveal empty sella (white arrow) and prominent extra-axial CSF spaces in the posterior parietal region (green arrow). (C) FIESTA 3D axial sections show a prominent perioptic CSF cuff (black arrow) better appreciated on (D) T2W axial section with a tortuous optic nerve and inward bulge of sclera (red arrow). There was slow flow/thrombosis appearing as T1 hyperintense in the right transverse sinus (blue arrow). (F) MR venogram 3D reconstruction revealed stenosed sigmoid and transverse sinuses on the right side (yellow arrow) with filling defects. CSF pressure analysis and papilledema confirmed benign intracranial hypertension.

3.2.2 Cerebral Venous Sinus Thrombosis (CVST)

Cerebral venous sinus thrombosis (CVST) is a less common but clinically important venous cause of PT because tinnitus may occasionally precede more overt neurological manifestations. PT may result from disturbed venous haemodynamics caused by partial venous obstruction, recanalisation, residual post-thrombotic stenosis, or recruitment of collateral venous pathways. Slow flow or thrombosis may also coexist with venous sinus stenosis, as illustrated in **Fig. 9E–F**.

MRI can directly demonstrate thrombus and associated complications such as venous infarction or haemorrhage (**Fig. 10A–C**). Depending on thrombus age and the imaging sequence, the affected sinus may demonstrate altered signal intensity, while susceptibility-weighted imaging (SWI) can depict susceptibility related to thrombus within affected sinuses or cortical veins (**Fig. 10D, E**). MRV and CTV may demonstrate filling defects, non-visualisation of affected venous segments, irregular residual lumina, and collateral venous pathways (**Fig. 10F, G**)

Management is directed primarily at the underlying CVST, with anticoagulation forming the cornerstone of treatment in appropriate patients. PT may improve as venous drainage is restored; however, persistent symptoms may occur when residual stenosis or altered collateral venous flow remains [14].

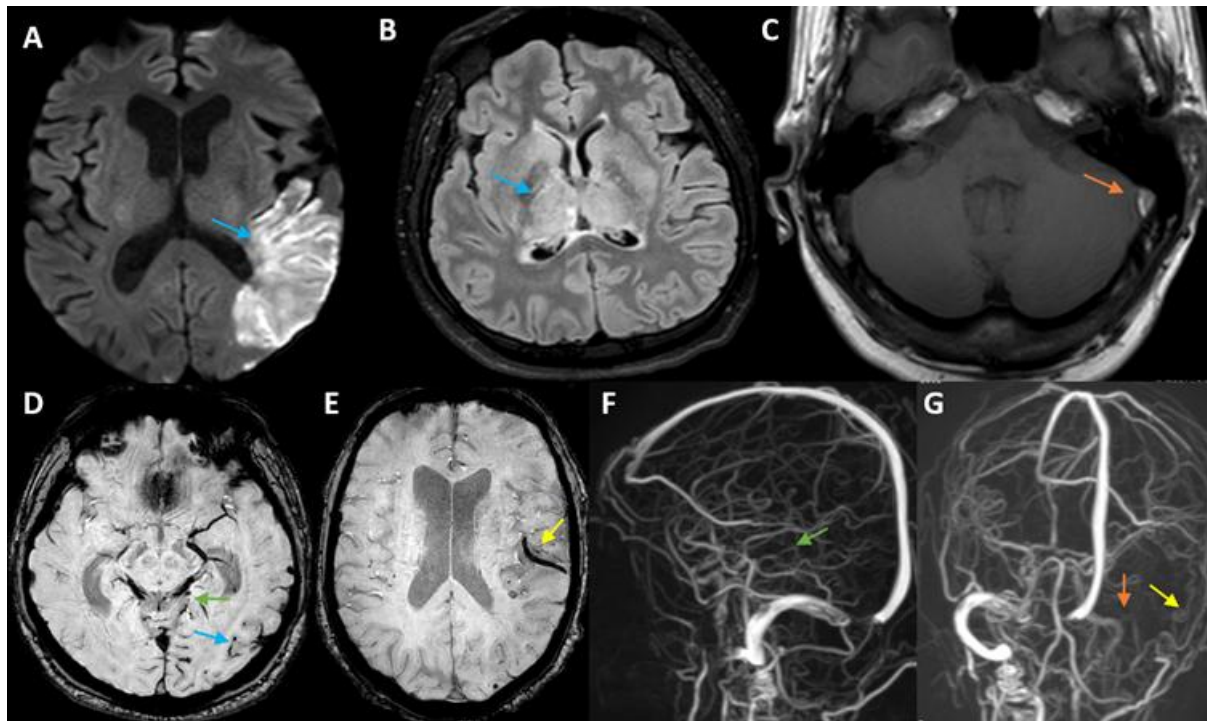


Fig. 10. (A, B) DWI and FLAIR images demonstrate acute infarcts involving the left parietal cortex and bilateral thalami (blue arrows). (C) T1-weighted image demonstrates hyperintense signal at the left transverse–sigmoid sinus junction, consistent with thrombus. (D, E) SWI demonstrates blooming related to thrombus involving the straight sinus, internal cerebral vein (green arrow), and a cortical vein draining towards the vein of Labbé (yellow arrow), with haemorrhagic foci within the left parietal infarct (blue arrow). (F, G) 3D TOF MR venography demonstrates non-visualisation of the left transverse and sigmoid sinuses, straight sinus, and internal cerebral vein, with collateral venous channels, consistent with acute-on-chronic cerebral venous sinus thrombosis.

3.2.3 Venous Sinus Stenosis and Post-Stenotic Diverticulum

Venous sinus stenosis is a common cause of venous pulsatile tinnitus (PT) and may occur in association with idiopathic intracranial hypertension (IIH) or as an isolated abnormality. Focal narrowing, particularly at the transverse–sigmoid junction, increases flow velocity and generates turbulent venous flow that may produce pulse-synchronous sound. Associated post-stenotic dilatation or diverticulum can further alter local haemodynamics and contribute to PT,

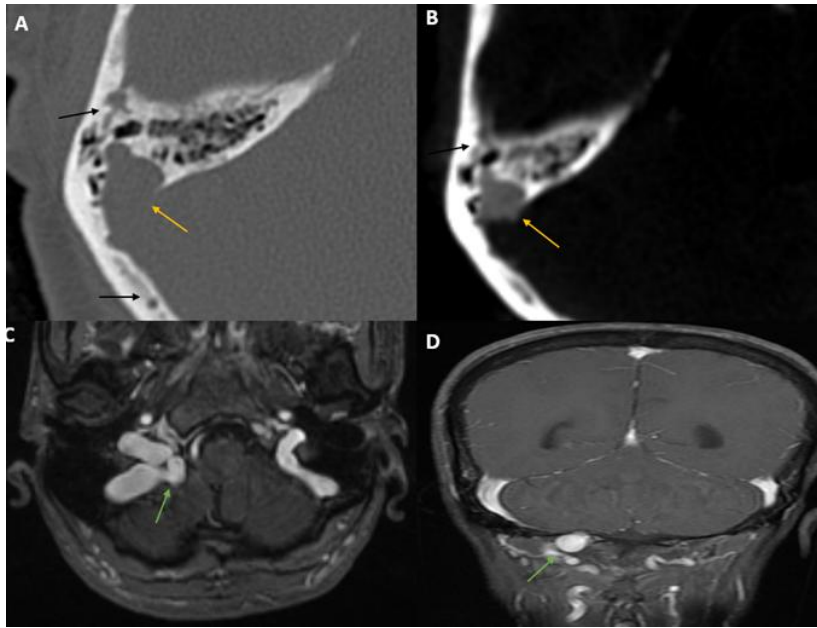


Fig. 11: (A) HRCT of temporal bone axial cuts, (B) with contrast shows a well-defined lytic lesion with sharp margins protruding into the mastoid with dehiscent posterior wall. It was confirmed to be a contrast-filled outpouching arising from the right sigmoid sinus (yellow arrow). Few small calibre trans osseous venous channels are seen adjacent to the diverticulum in the right parietotemporal bone, which are draining into the right sigmoid sinus. (C, D) Contrast-enhanced T1 MRI axial and coronal images of a different patient show contrast filled outpouching arising from the right sigmoid sinus (green arrow).

MRV and CTV demonstrate focal or segmental venous sinus narrowing, most commonly involving the transverse–sigmoid junction. Post-stenotic dilatation or sigmoid sinus diverticulum may coexist and further contribute to turbulent flow (**Fig. 11**). High-resolution CTV with multiplanar and oblique reformations is particularly useful for characterising the stenotic segment, associated diverticulum, and adjacent osseous anatomy [14].

Advanced techniques such as phase-contrast MRI and 4D-flow MRI can provide additional information on venous velocity, flow patterns, wall shear stress, and other haemodynamic alterations associated with venous PT. In patients considered for venous sinus stenting, catheter venography with venous manometry can assess the haemodynamic significance of the stenosis and trans-stenotic pressure gradient. Venous sinus stenting can result in substantial and sometimes rapid improvement or resolution of PT in appropriately selected patients by reducing the pressure gradient and abnormal turbulent venous flow [14].

3.2.4 Sigmoid Sinus Diverticulum and Dehiscence

Sigmoid sinus wall abnormalities, principally sigmoid sinus diverticulum and dehiscence, are recognised causes of venous pulsatile tinnitus (PT). A sigmoid sinus diverticulum represents a focal outpouching of the sinus, whereas dehiscence refers to focal absence of the bony plate separating the sigmoid sinus from the adjacent mastoid air cells.

These abnormalities are significantly more frequent in patients with PT than in controls; one HRCT study identified sigmoid sinus diverticulum/dehiscence in 23% of patients with PT compared with 1.2% of patients without PT [15]. More recent case-control data similarly demonstrate an increased prevalence of sigmoid sinus wall abnormalities in patients with PT [16].

High-resolution CT (HRCT) of the temporal bone is particularly useful for demonstrating focal defects or thinning of the sigmoid plate and defining the relationship of the sinus to the mastoid air cells (Fig. 11A,B). CT venography can additionally delineate a sigmoid sinus diverticulum and associated venous abnormalities (Fig. 11C,D). Loss of the normal osseous barrier may increase transmission of venous flow-related sound through the mastoid air-cell system, while a diverticulum may further contribute to local flow disturbance and PT [17].

Sigmoid sinus wall abnormalities may coexist with transverse sinus stenosis and imaging features associated with idiopathic intracranial hypertension, highlighting the importance of assessing the entire cerebral venous system rather than interpreting the sigmoid wall abnormality in isolation [18]. In appropriately selected symptomatic patients, transmastoid sigmoid sinus wall reconstruction or resurfacing can provide substantial and durable improvement. A recent long-term series reported complete or partial resolution in 83.8% of treated ears at more than 5 years of follow-up [19]. However, persistent or recurrent PT may occur, particularly when the wall defect is incompletely reconstructed or associated venous haemodynamic abnormalities remain unaddressed [19,20].

3.2.5. Jugular Bulb Anomalies

Jugular bulb variants are common and often incidental; however, a high-riding, dehiscent, or diverticular jugular bulb may cause venous pulsatile tinnitus (PT) when closely related to the middle or inner ear [21]. A high-riding jugular bulb is variably defined according to its relationship with temporal-bone landmarks, including extension to or above the basal turn of the cochlea or proximity to the internal auditory canal [21]. A dehiscent jugular bulb lacks the normal bony plate separating it from the middle ear, whereas a jugular bulb diverticulum represents a focal outpouching of the bulb.

High-resolution CT (HRCT) of the temporal bone is the preferred technique for defining these variants and their relationship to adjacent otic structures. HRCT readily demonstrates a high-riding jugular bulb and associated bony dehiscence into the middle ear cavity (**Fig. 12**), while CTV/MRV provides complementary assessment of venous anatomy [21,22]. As these variants also occur in asymptomatic individuals, clinicoradiological correlation is essential. Most incidental lesions require no treatment; in selected patients with disabling PT, surgical resurfacing/reconstruction or endovascular treatment may provide symptomatic improvement [22–24].

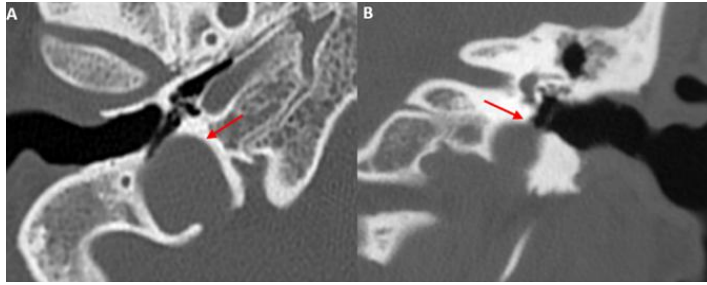


Fig. 12. (A and B) Axial and coronal sections of HRCT temporal bone show a high-riding jugular bulb with dehiscence of its anteriosuperior reaching the floor of the middle ear cavity (red arrow).

3.2.5. Internal Jugular Vein Compression

Styloidogenic jugular venous compression syndrome (SJVCS), sometimes termed the jugular variant of Eagle syndrome, results from extrinsic compression of the upper internal jugular vein (IJV) between the styloid process and the C1 lateral tubercle/transverse process, impairing cerebral venous outflow [25–27]. Patients may present with headache, pulsatile tinnitus, visual symptoms, or features of intracranial hypertension [25–27]. CTV or MRV demonstrates focal IJV narrowing and collateral venous drainage, with compression between the styloid process and C1 readily demonstrated on CT (**Fig. 13**). Dynamic venography with manometry may demonstrate position-dependent stenosis and a trans-stenotic pressure gradient [25,26]. In selected symptomatic patients, surgical decompression, typically styloidectomy and occasionally C1 decompression, may restore venous outflow; endovascular treatment may be considered when significant residual stenosis persists after adequate osseous decompression [25,27,28].

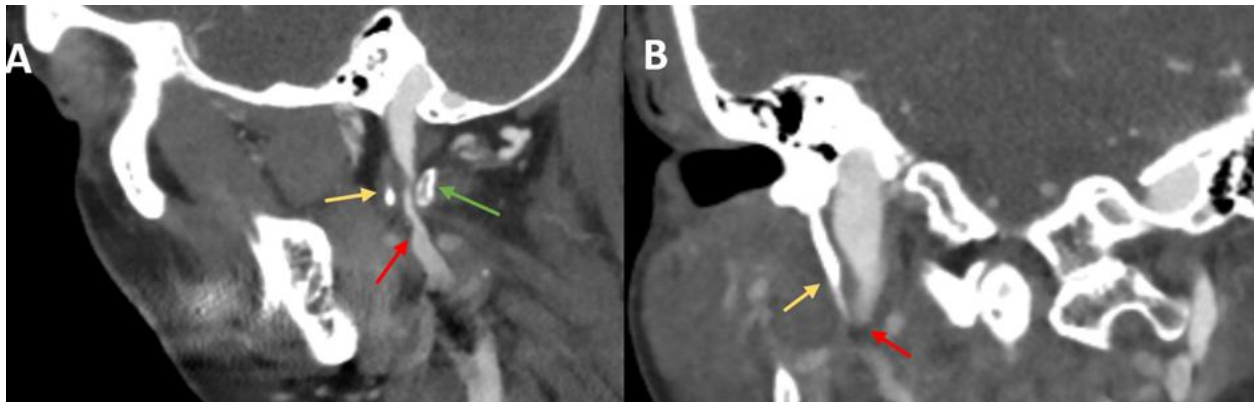


Fig. 13. (A, B) CT angiogram of neck shows IJV narrowing (red arrow) due to compression between elongated styloid process (yellow arrow) and C1 transverse process (green arrow)

3.3. Arteriovenous Shunts

Arteriovenous shunts are important, potentially high-risk causes of PT. Abnormal arterial-to-venous flow produces venous arterialization and turbulence, particularly when the transverse–sigmoid sinus is involved. The presence of

cortical venous reflux (CVR) is particularly important because it is associated with increased risk of intracranial hemorrhage and neurological complications [29,30].

3.3.1. Dural Arteriovenous Fistulas (DAVFs)

DAVFs are abnormal communications between dural arteries and dural sinuses or cortical veins and represent an important vascular cause of PT. Clinical risk is determined primarily by the venous drainage pattern, with CVR identifying higher-risk lesions [29,30]. MRI/MRA may demonstrate dilated vessels, prominent venous flow-related signal, or early venous filling, while CTA can reveal enlarged arterial feeders and abnormal venous channels [31]. ASL may improve detection by demonstrating abnormal venous signal from arterialized blood, and time-resolved MRA can provide additional information on shunt haemodynamics [32,33]. DSA remains the reference standard for defining the fistulous point, arterial supply, venous drainage and CVR, and permits Borden or Cognard classification and treatment planning [29,30,34].

3.3.2. Brain Arteriovenous Malformations (AVMs)

Brain AVMs are high-flow vascular lesions comprising a nidus connecting feeding arteries and draining veins without an intervening capillary bed. Although PT is uncommon, superficially located or posterior fossa AVMs may produce pulse-synchronous symptoms. MRI typically demonstrates serpiginous vascular flow voids, while CTA/MRA delineate the nidus, feeding arteries, and draining veins (**Fig. 14D, E**). DSA provides definitive angioarchitectural characterization and remains important for treatment planning. Surgical risk can be assessed using the Spetzler–Martin grading system [35]. Related arteriovenous and venous abnormalities that may enter the imaging differential, including sinus pericranii and dural arteriovenous fistula, are also illustrated in **Fig. 14A, B** and **Fig. 14F**, respectively.

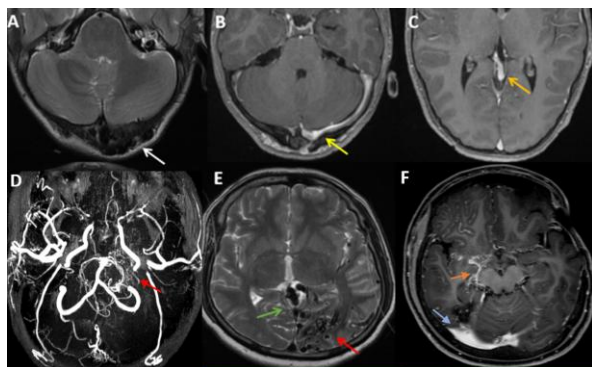


Fig. 14. (A) T2W MRI axial cut of the posterior fossa shows multiple dilated and tortuous venous channels in the occipital region (white arrow). (B) T1W contrast MRI section shows early contrast opacification of the veins draining into the prominent left sigmoid sinus (yellow arrow), straight sinus, and vein of Galen (orange arrow). A case of sinus pericranii. (D and E) TOF 3D head angiogram and T2 MRI axial sections of a different patient show multiple dilated and tortuous flow voids in the left posterior parietal and occipital lobe with arterial feeders arising from left ICA, MCA, and PCA (red arrow in D). Early draining veins are seen joining the dilated straight sinus (green arrow). Nidus

is visualized, suggestive of cerebral AVM. (F) MR angiogram axial section of brain reveals an abnormal fistulous communication between a tributary of straight sinus (blue arrow) and right MCA, PCA (orange arrow) without a nidus, suggestive of an acquired dural AV fistula secondary to chronic MCA, PCA occlusion confirmed on DSA.

3.4. Structural and Osseous Pathologies

Structural abnormalities of the temporal bone may cause PT through altered sound conduction or increased transmission of adjacent vascular pulsations. HRCT temporal bone is the principal imaging modality, supplemented by MRI for selected soft-tissue and labyrinthine abnormalities.

3.4.1. Otosclerosis (Otospongiosis)

Otosclerosis is an abnormal bone-remodelling disorder of the otic capsule, most commonly involving the fissula ante fenestram. HRCT demonstrates focal hypoattenuation at the fissula ante fenestram in fenestral otosclerosis (**Fig. 15A, B**) and pericochlear hypoattenuation in retrofenestral disease (**Fig. 15C, D**) [36]. PT is an uncommon manifestation and may relate to increased vascularity during active otospongiosis.

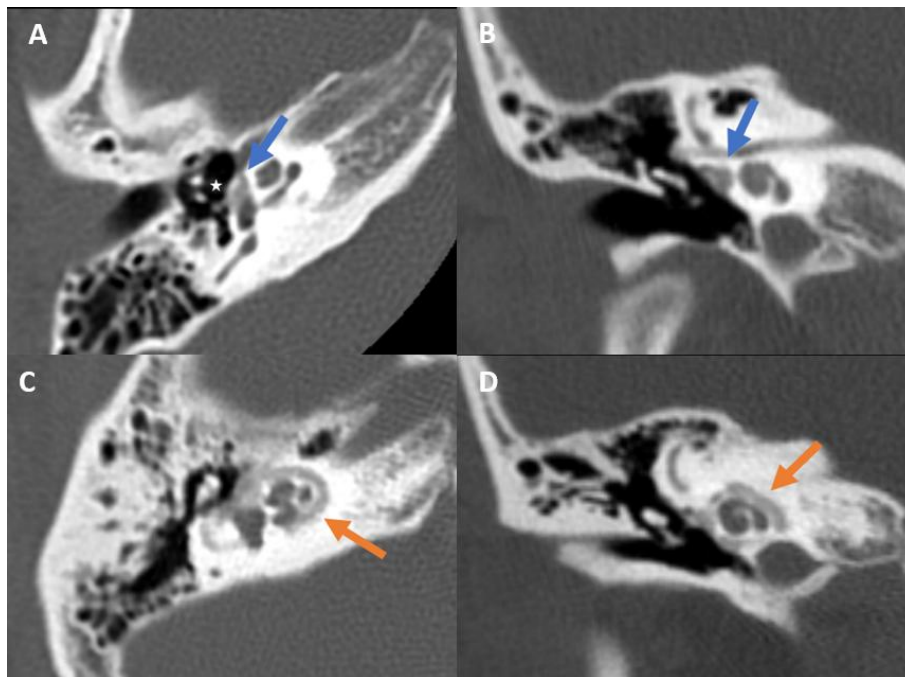


Fig. 15. (A and B) Axial and coronal HRCT sections of right temporal bone show hypodense lucent foci anterior to the oval window at the fissula ante fenestram (blue arrow in A) indicating bone mineralisation. The stapes footplate is thickened and fixed, consistent with stapes fixation (white star). (C and D) Axial and coronal HRCT sections of right temporal bone show diffuse spiral-shaped hypodensity around cochlea known as the “halo sign” (orange arrow) indicating demineralisation. In both cases, findings were present bilaterally, confirming fenestral and retrofenestral otosclerosis.

3.4.2. Superior Semicircular Canal Dehiscence

Superior semicircular canal dehiscence (SSCD) creates a third mobile window, producing autophony, conductive hearing loss, sound- or pressure-induced vertigo, and occasionally PT [37]. HRCT with Pöschl and Stenvers reformations best demonstrates the defect; imaging should be correlated with symptoms and physiological testing because apparent dehiscence may occur on CT [37].

3.4.3. Cholesteatoma

Cholesteatoma is a keratinizing lesion that progressively erodes middle-ear and mastoid structures. PT is uncommon but may occur with erosion adjacent to vascular structures. HRCT defines scutum, ossicular, labyrinthine, facial canal, tegmen, and sinus plate erosion and may demonstrate complications such as labyrinthine fistula formation (**Fig. 16**), while diffusion-weighted MRI is particularly useful for lesion detection and assessment of residual or recurrent disease [38].

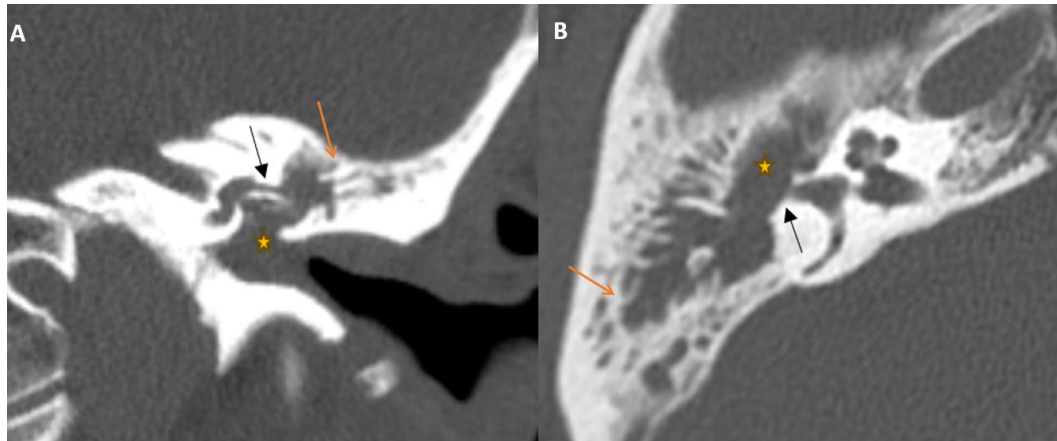


Fig. 16. (A, B) coronal and axial HRCT of temporal bone shows soft tissue density in the middle ear cavity (yellow star) extending to mastoid antrum and air cells (red arrow). Focal discontinuity in the anterior limb of lateral semicircular canal which is seen in continuity with the soft tissue in the middle ear cavity suggestive of lateral semicircular dehiscence forming a labyrinthine fistula. The soft tissue density was confirmed to be cholesteatoma post surgery in a patient with CSOM.

3.4.4. Cholesterol Granuloma

Petrous apex cholesterol granuloma is a benign expansile lesion that may cause hearing loss, vertigo, and tinnitus. CT typically demonstrates smooth expansion with bony remodelling and cortical thinning, while MRI characteristically shows high T1- and T2-weighted signal without significant enhancement or diffusion restriction (**Fig. 17**) [39].

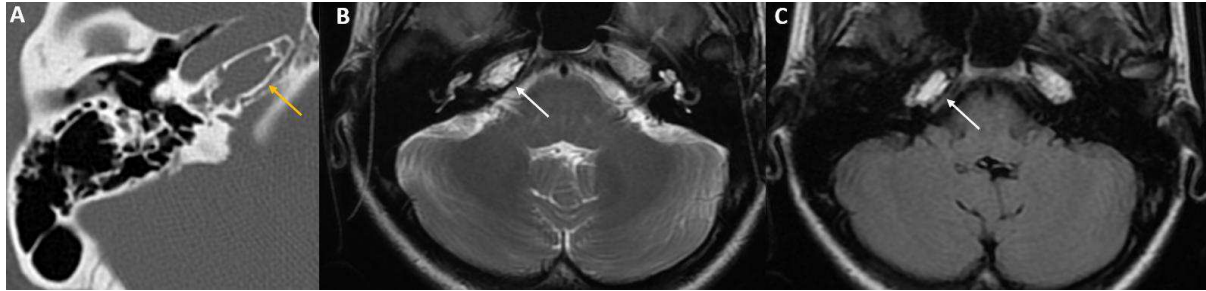


Fig. 17. (A) HRCT temporal bone axial section, (B & C) T1W and T2W MR axial temporal sections show a well-defined cystic/lytic expansile lesion with sharp margins in the right petrous apex causing bone remodeling and cortical thinning (yellow arrow). It appears T1 and T2 hyperintense (white arrows) with no diffusion restriction, indicating a cholesterol granuloma.

3.4.5. Labyrinthitis Ossificans

Labyrinthitis ossificans represents fibrosis and subsequent ossification of the membranous labyrinth, usually following infection, trauma, or haemorrhage. HRCT demonstrates labyrinthine ossification, often involving the basal cochlear turn (**Fig. 18**), whereas MRI may detect earlier fibrotic change through loss of normal labyrinthine T2 signal. Recognition is particularly important before cochlear implantation.



Fig.18. (A, B, C) Axial HRCT temporal bone cuts show calcific haziness predominantly in the basal turn of cochlea (green arrow), suggestive of labyrinthitis ossificans. Soft tissue in mastoid air cells eroding the sigmoid plate (blue arrow). Enlarged vestibular aqueduct (white arrow)

3.4.6 Less Common Structural and Osseous Causes

Several less common structural abnormalities may also be associated with PT through altered sound transmission, bony dehiscence, or increased exposure of adjacent vascular structures. These include sigmoid sinus plate thinning related to chronic otitis (Figure 18B), jugular bulb-associated bony defects, Paget disease, Langerhans cell histiocytosis, petrous apex cephaloceles, and enlarged vestibular aqueduct (Figure 18C). Although these entities are

uncommon causes of PT, their recognition on temporal bone imaging may help identify an underlying structural abnormality and guide further clinical evaluation.

3.5. Temporal Bone and Cerebellopontine Angle Lesions

Temporal bone, skull-base, and cerebellopontine angle (CPA) lesions may occasionally cause PT through neurovascular compression, altered venous drainage, or transmission of vascular pulsations. Their relationship to PT should be interpreted cautiously, particularly when lesions are small or incidental.

3.5.1. Arachnoid Cyst

Arachnoid cysts are benign CSF-filled extra-axial lesions that are usually incidental but may cause tinnitus, hearing loss, or vestibular symptoms when located in the CPA and producing mass effect [40]. MRI demonstrates CSF signal on all sequences, complete FLAIR suppression, absence of enhancement, and no diffusion restriction, distinguishing them from epidermoid cysts [40,41]. PT is uncommon and should be attributed to the lesion only when relevant neurovascular compression or displacement is demonstrated.

3.5.2. Epidermoid Cyst

Epidermoid cysts are congenital inclusion lesions that commonly involve the CPA and may cause cranial neuropathies, hearing loss, vertigo, or tinnitus [41]. MRI typically demonstrates T1 hypointensity, T2 hyperintensity, incomplete FLAIR suppression, and, characteristically, marked diffusion restriction on DWI (**Fig. 19A–C**), differentiating epidermoids from arachnoid cysts, which follow CSF signal and do not restrict diffusion (**Fig. 19D–F**) [41,42]. PT is uncommon but may occur when adjacent vascular structures are displaced or encased.

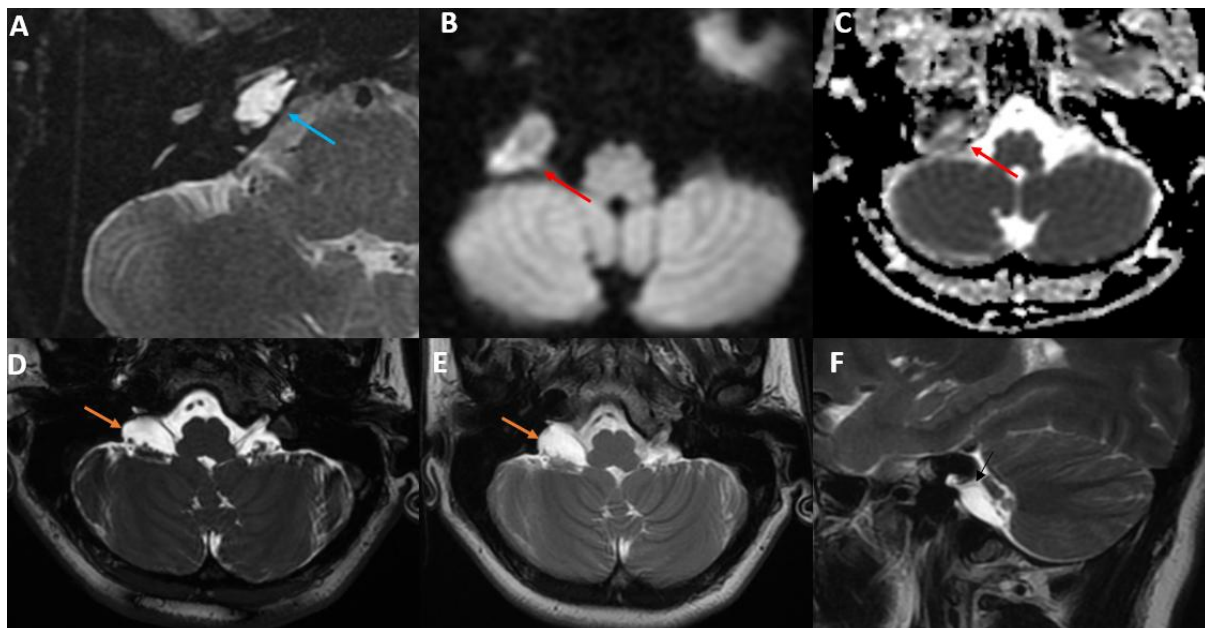


Fig.19. (A) FIESTA T2W MR 3D image axial section, (B) DWI and (C) ADC sections show a well-defined hyperintense lesion in the right petrous apex (blue arrow) showing diffusion restriction and corresponding low ADC values (red arrows) favouring an epidermoid cyst. (D) FIESTA T2W MR 3D image shows a well-defined extra-axial CSF intensity round lesion in right CPA angle (orange arrow) displacing the VII and VIII nerves and flocculus of the cerebellum. (E) Sagittal T2W MR image shows its thin hypointense wall with no communication (black arrow), suggestive of an arachnoid cyst

3.5.3. Benign Petrous Apex Lesions

Petrous apex effusions, mucocles, and cholesterol granulomas may cause auditory or vestibular symptoms, although PT is uncommon [43]. Effusions typically preserve osseous trabeculae on CT and demonstrate fluid signal on MRI, whereas mucocles are expansile lesions with smooth bony remodelling and variable MRI signal depending on protein content [43]. Cholesterol granulomas characteristically demonstrate high T1- and T2-weighted signal with smooth expansion and bony remodelling [39,43]. CT and MRI are complementary for lesion characterization and assessment of adjacent neurovascular structures.

3.6. Neoplastic and Hypervascular Tumors

Neoplastic and hypervascular lesions of the temporal bone, skull base, and cerebellopontine angle (CPA) are uncommon causes of PT. Symptoms may result from tumor vascularity, compression of adjacent vessels, altered venous drainage, or osseous erosion. Paragangliomas are the prototypical neoplastic cause of objective PT, while other relevant lesions include vascular malformations, endolymphatic sac tumors (ELSTs), meningiomas, schwannomas, and skull-base malignancies [44].

3.6.1. Paragangliomas

Head and neck paragangliomas are highly vascular tumours that may present with unilateral pulsatile tinnitus (PT), hearing loss, or cranial neuropathies. Glomus tympanicum typically arises at the cochlear promontory (**Fig. 20**), whereas glomus jugulare involves the jugular foramen with associated osseous erosion (**Fig. 21**). Other head and neck paragangliomas include vagal and carotid body tumors, with carotid body tumors characteristically splaying the internal and external carotid arteries (**Fig. 22**). CT delineates osseous involvement, while MRI demonstrates avid enhancement and may show the characteristic “salt-and-pepper” appearance in larger lesions [44].

Glomus vagale typically arises in the upper carotid space and appears as an intensely enhancing hypervascular mass, with displacement of adjacent vascular and pharyngeal structures (**Fig. 23C, D**). Other vascular tumors, including hemangiomas, may occasionally involve the temporal bone or middle ear and mimic paragangliomas clinically, particularly when presenting as a reddish pulsatile retrotympanic lesion (**Fig. 23A, B**).

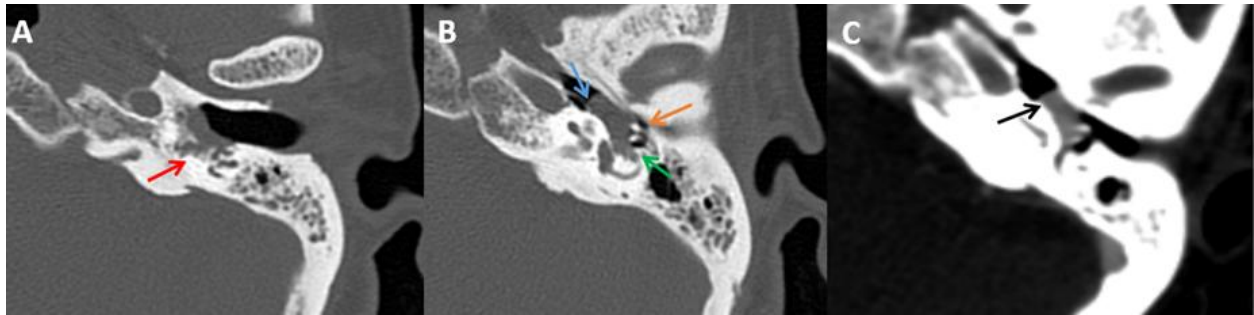


Fig. 20. (A, B) Axial cuts of HRCT of the temporal bone reveal a well-defined soft tissue density lesion in the middle ear cavity at the cochlear promontory (blue arrow), encasing the ossicles (orange arrow) and causing permeative/erosive changes at the anterior aspect of the petrous bone, sinus tympani (red arrow). The lesion is also causing anterior limb of lateral semicircular canal dehiscence (green arrow). (C) Axial HRCT cut of the temporal bone with contrast shows intense enhancement (black arrow) of the lesion with HU 140. Histopathologic examination post-surgery revealed glomus tympanicum.

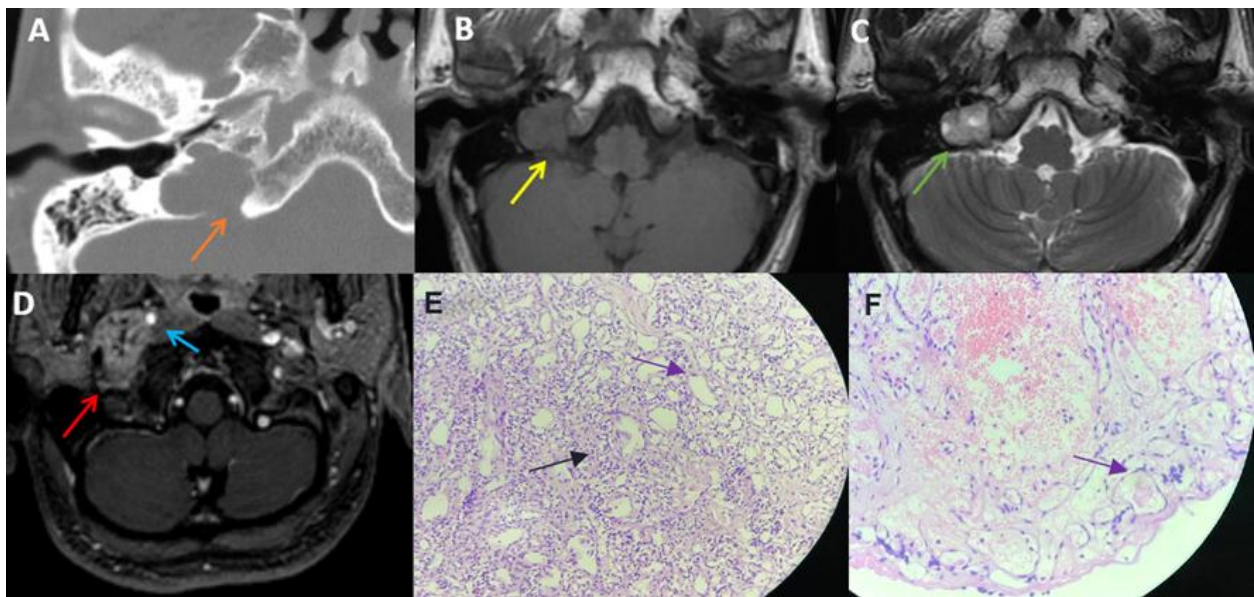


Fig. 21. (A) HRCT right temporal bone axial cut shows an expansile lytic lesion in the right jugular fossa with a well-defined sharp margin and cortical breach in the posterior petrous (red arrow). On MRI temporal bone axial cuts (B) T1W, (C) T2W, (D) T1W with contrast show the lesion appearing T1 hypointense (yellow arrow), T2 heterogeneously hyperintense with small cystic areas (green arrow). The lesion is pointing inferolaterally in the jugular fossa in continuity with the jugular vein, showing heterogeneous enhancement (red arrow), suggestive of glomus jugulare. Subtle hypo- and hyperintense areas are seen within the lesion on T1W, resembling salt and pepper appearance better seen on T1W contrast. The lesion is supplied by branches of ECA (blue arrow). (E) H&E 40X shows glomus cells (black arrow) interspersed with capillary spaces (purple arrow) in fibro-collagen stroma. (F) H&E 40X shows glomus cells (black arrow) interspersed with capillary spaces (purple arrow) in fibro-collagen stroma.

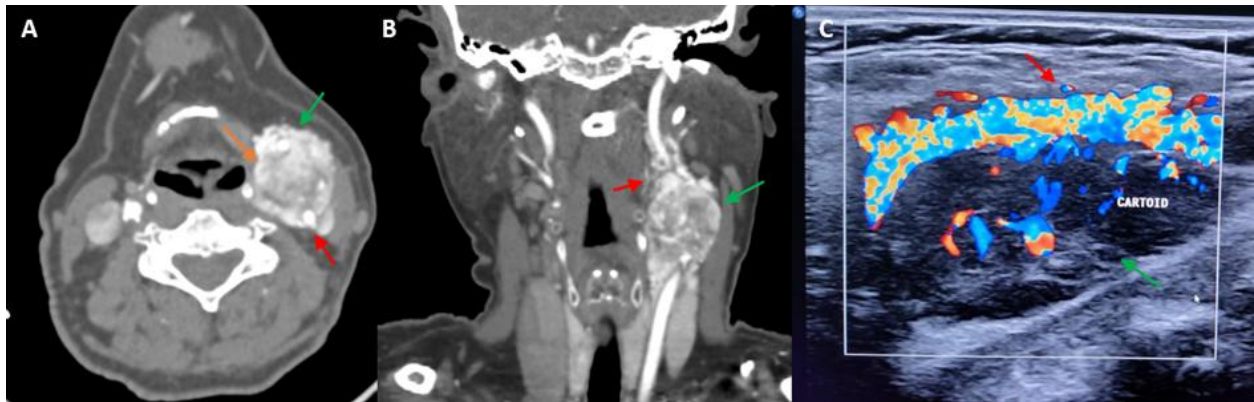


Fig. 22. (A, B) Axial and coronal CT neck with contrast shows an intensely enhancing lesion (green arrow) encasing the common carotid and splaying the ICA (red arrow) and ECA (orange arrow). (C) USG Neck with Doppler shows a hypoechoic lesion supplied by branches of CCA and proximal ICA (red arrow).

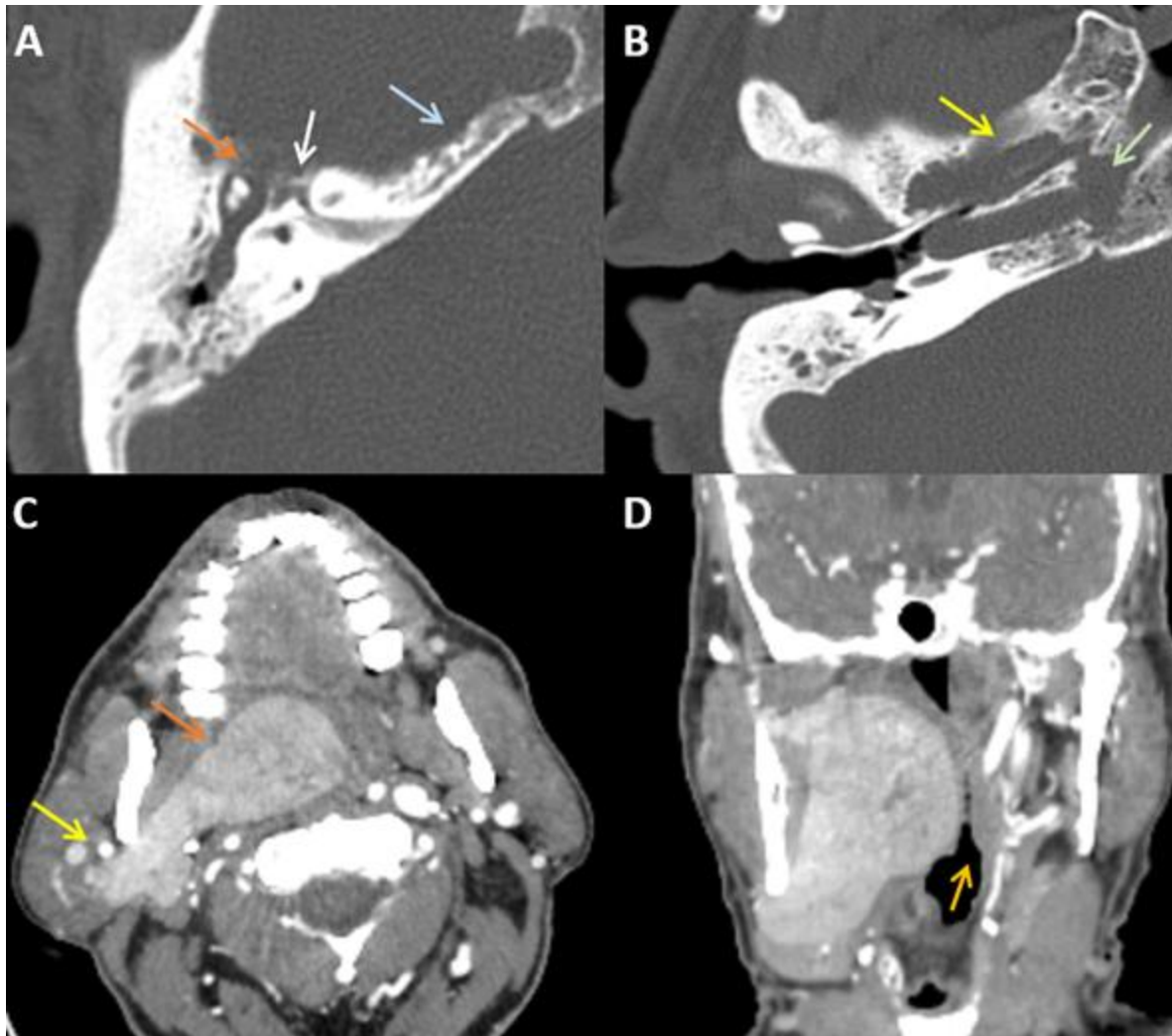


Fig. 23. (A and B) Axial HRCT temporal bone shows erosion and destructive changes due to a lytic soft tissue density lesion involving the genu of facial canal (white arrow), tegmen tympani, attic (red arrow), petrous temporal bone, apex (blue arrow) and carotid canal (green arrow). A well-defined lytic lesion with spiculation at the greater wing of the sphenoid (yellow arrow) is seen with extension to the pterygoid plate. The patient could not afford an MRI. Otoscopy revealed a reddish pulsatile area in the middle ear cavity. This was confirmed to be a hemangioma. (C and D) Axial CECT neck contrast arterial phase shows an oblong-shaped intensely enhancing lesion (red arrow) with prominent feeders and non-enhancing areas involving the deep parotid space (yellow arrow), carotid space, parapharyngeal space, oropharynx, causing lateral wall bulge and airway narrowing (orange arrow). This was confirmed to be a glomus vagale.

3.6.2. Facial Nerve Venous Malformation

Facial nerve venous malformations (FNVMs) are rare benign vascular lesions, most commonly involving the geniculate ganglion, followed by the internal auditory canal. HRCT may demonstrate facial canal expansion, erosion

or thinning, and characteristic intralesional calcifications or osseous spicules, sometimes producing a “honeycomb” appearance (**Fig. 23A, B**). MRI typically demonstrates T1 iso-/hypointensity, T2 hyperintensity, and avid, often heterogeneous enhancement [45,46]. Lesions historically termed facial nerve hemangiomas are now considered more consistent with venous malformations based on radiologic-pathologic and immunohistochemical correlation [45].

3.6.3. Endolymphatic Sac Tumors

ELSTs are rare tumours arising from the endolymphatic sac and have a strong association with von Hippel–Lindau disease [47]. Patients may present with hearing loss, tinnitus, or vestibular symptoms. CT typically demonstrates destructive posterior petrous-bone changes, while MRI shows heterogeneous signal and enhancement related to the tumor's vascular, hemorrhagic, and proteinaceous components (**Fig. 24A–E**) [47]. Histopathologic confirmation may demonstrate characteristic cystic spaces lined by cuboidal epithelium and containing colloid-like material (**Fig. 24F**).

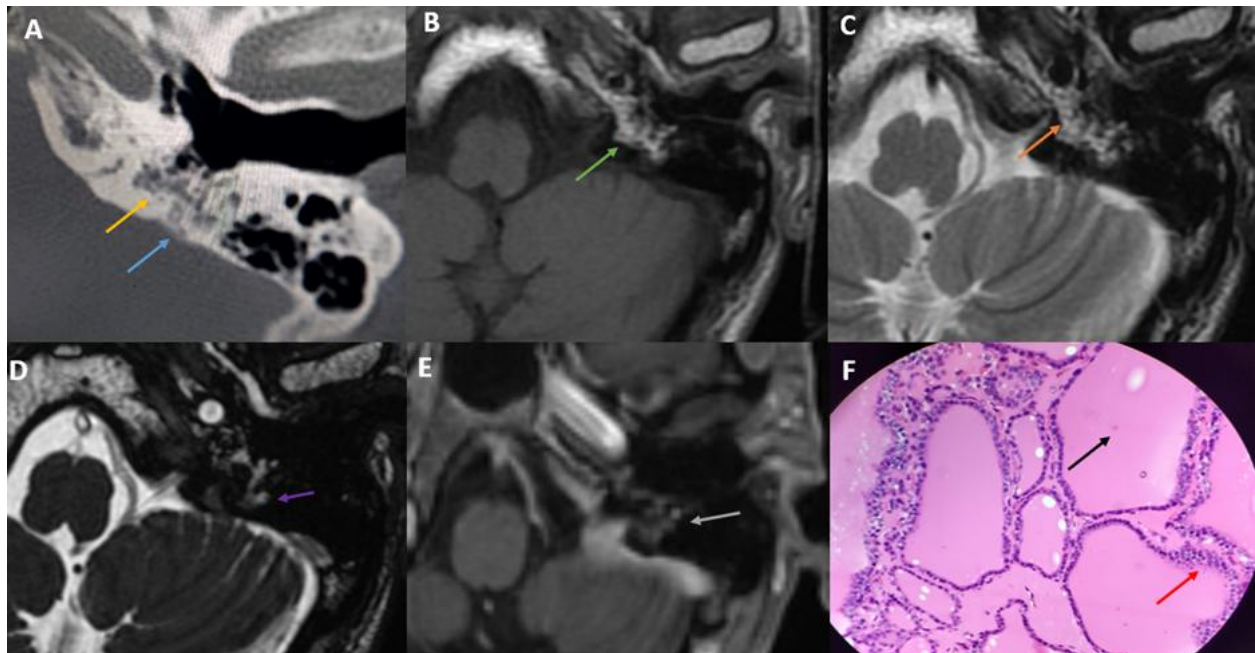


Fig. 24. (A) HRCT of temporal bone axial cuts shows a subtle moth eaten/ permeative pattern at the posterior petrous bone in proximity with the endolymphatic duct. The lesion appears hyperintense on T1W green arrow in B, hyperintense on T2W orange arrow in C, demonstrates Continuity with the endolymphatic sac (purple arrow in D) on FIESTA 3D with subtle enhancement on T1W contrast (grey arrow in E). Histopathologic exam H and E 40X confirmed a cystic type of endolymphatic tumor with cystic spaces lined by cuboidal epithelium (red arrow in F) filled with colloid-like material (black arrow in F). No spread to intracranial structures noted.

3.6.4. Meningiomas

CPA and petrous meningiomas may contribute to PT through compression or narrowing of adjacent venous sinuses. MRI typically demonstrates an avidly enhancing dural-based mass, sometimes with a dural tail and intracanalicular extension (Fig. 25), while CT may demonstrate calcification or hyperostosis.

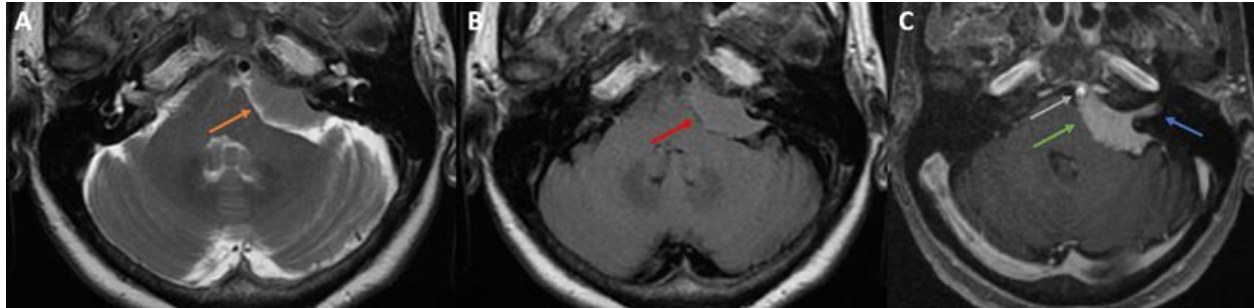


Fig. 25. (A) T2W (B) FLAIR (C) T1W with contrast axial cuts show an extra-axial homogeneously enhancing dural-based lesion (green arrow) at the left CP angle appearing slightly hyperintense on T2W (orange arrow), isointense on FLAIR (red arrow) with intracanalicular extension (blue arrow) suggesting meningioma. The lesion is abutting the middle cerebellar peduncle of the midbrain. It is abutting the basilar artery with less than 180-degree contact (grey arrow)

3.6.5. Schwannomas

Vestibular schwannomas typically arise within the internal auditory canal and extend into the CPA, although PT is an uncommon presentation. MRI demonstrates an enhancing IAC/CPA mass, with larger lesions occasionally showing cystic change (Fig. 26A–D). Facial nerve schwannomas follow the facial nerve canal and may produce smooth canal enlargement (Fig. 26E), while lower cranial nerve schwannomas may also occur within the CPA (Fig. 26F).

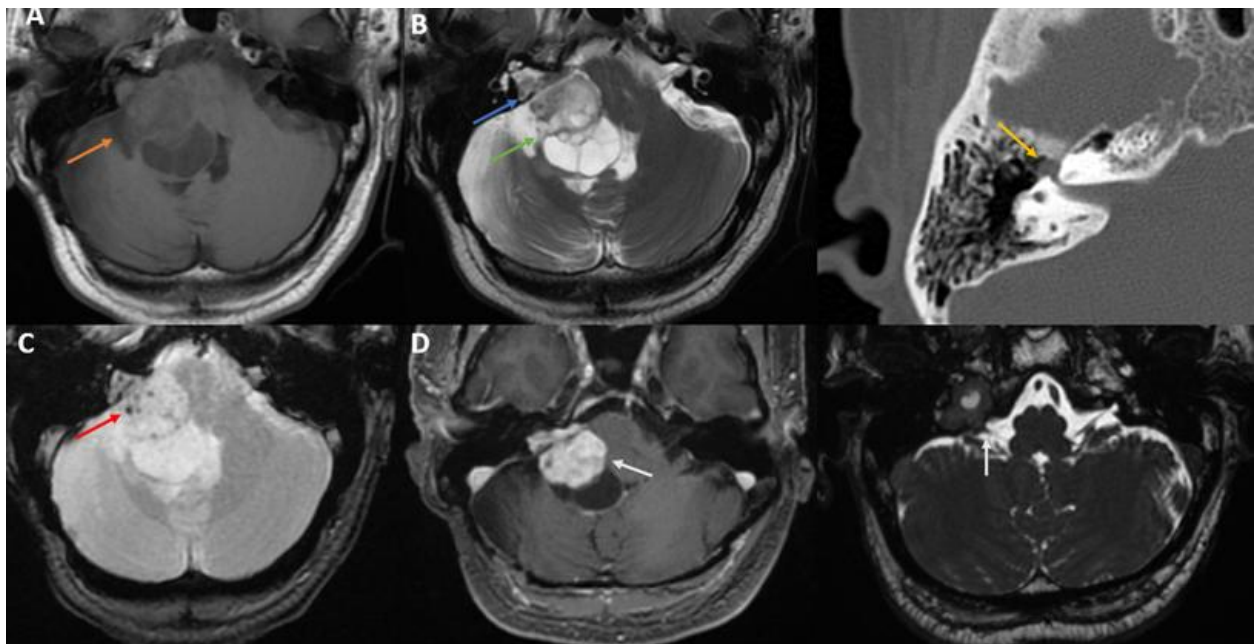


Fig. 26. (A) T1W, (B) T2W, (C) GRE and (D) T1W with contrast axial cuts show an extra-axial solid cystic lesion (green arrow). The solid component appearing iso to hypointense on T1W (orange arrow), heterogeneously hyperintense on T2W (green arrow), showing enhancement of the solid component (white arrow) and a few calcific specks on GRE blooming (red arrow) seen in the left CP angle cistern with intracanalicular (blue arrow) component causing widening of porus acusticus forming an ice cream cone appearance. (E) HRCT axial cuts of the temporal bone of another patient reveal a globular dilatation of the geniculate ganglion facial canal, which is protruding into the middle ear cavity. (F) FIESTA 3D sequence of another patient shows a hypointense lesion with a cystic component in continuity with the IX nerve (white arrow). All were confirmed to be schwannoma post-surgery.

3.6.6. Other Skull-Base Malignancies

Skull-base malignancies may occasionally produce PT through osseous destruction, vascular encasement, or altered local haemodynamics. CT demonstrates destructive osseous changes and local tumour extension (**Fig. 27**), while MRI is particularly useful for assessing marrow infiltration, perineural spread, and intracranial extension.

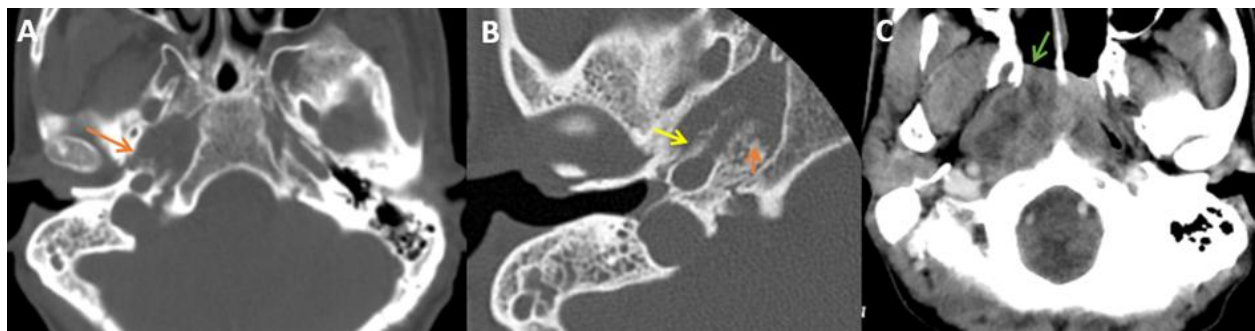


Fig. 27. (A and B) Axial HRCT temporal bone sections show a lytic lesion with a permeative pattern of destruction at the right petrous apex (red arrow) involving the carotid canal (yellow arrow) and greater wing of the sphenoid, pterygoid. (C) Soft tissue window with contrast CT of base of skull axial section shows enhancing soft tissue density lesion (green arrow) involving the nasopharynx, torus tubarius with extension to right parapharyngeal, carotid space and perineural invasion along 9th, 10th, 11th nerves. Features in favour of nasopharyngeal carcinoma with regional spread.

3.7. Miscellaneous, Systemic, and Functional Conditions

Several systemic and functional conditions may cause or mimic PT through increased vascular flow, altered CSF dynamics, or rhythmic muscular activity. Imaging is often primarily useful for excluding a focal vascular or structural cause.

3.7.1. High-Output Systemic States

High-output states, including anemia, hyperthyroidism, pregnancy, and high-output cardiac conditions, may increase vascular flow and produce bilateral or diffuse PT. When imaging excludes a focal cause, laboratory and cardiovascular assessment should be guided by the clinical context. Treatment of the underlying systemic disorder may improve symptoms [2].

3.7.2. Myoclonus and Muscular Causes

Middle-ear myoclonus results from involuntary contraction of the tensor tympani or stapedius muscles, producing rhythmic clicking, fluttering, or thumping that may mimic PT [48]. Palatal tremor can similarly produce rhythmic tinnitus. Diagnosis is primarily clinical; MRI is useful when a central neurological cause of palatal tremor is suspected [48].

3.7.3. Spontaneous Intracranial Hypotension

Spontaneous intracranial hypotension (SIH), usually caused by spinal CSF leakage, may occasionally produce tinnitus through altered CSF–venous dynamics. Brain MRI may demonstrate diffuse pachymeningeal enhancement, venous engorgement, brain sagging, and subdural collections (Fig. 28) [49]. Current consensus recommends contrast-enhanced brain MRI and whole-spine MRI as first-line imaging; CT or digital-subtraction myelography may subsequently be required to localise the leak [49].

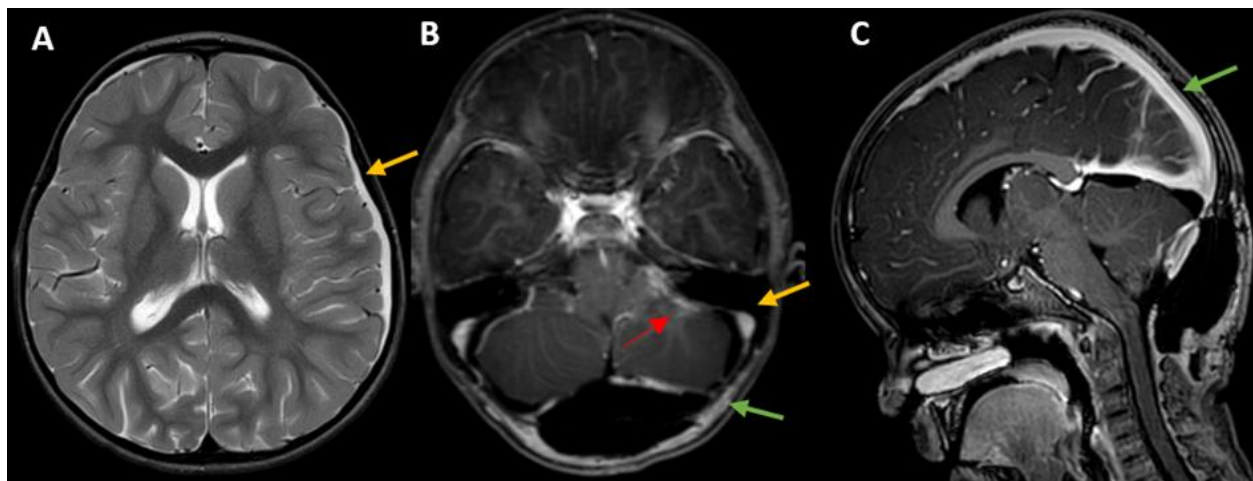


Fig. 28. T2 MR axial image shows hyperintense subdural fluid in the frontoparietal region (golden arrow in A). (B, C) Axial and sagittal T1 contrast images reveal pachymeningeal enhancement at the left CP angle (red arrows), posterior fossa subdural hygroma (golden arrow in B), and prominent dural venous sinuses (green arrow in B, C).

3.7.4. Chiari I Malformation

Chiari I malformation is characterised by caudal displacement of the cerebellar tonsils, conventionally ≥ 5 mm below the foramen magnum, although clinical significance depends on tonsillar morphology, craniocervical crowding, and

CSF dynamics rather than measurement alone [50,51]. MRI demonstrates tonsillar descent and associated abnormalities such as syringomyelia, while cine phase-contrast MRI can assess CSF flow in selected patients [50,51]. PT is uncommon, and alternative vascular or structural causes should be excluded before attributing symptoms to Chiari I malformation.

3.7.5. Temporomandibular Joint (TMJ) Dysfunction

Temporomandibular joint (TMJ) dysfunction may cause or exacerbate tinnitus, typically non-pulsatile, through somatosensory interactions between the trigeminal and auditory pathways [52,53]. Tinnitus may vary with jaw movement, clenching, or pressure over the TMJ and may be accompanied by jaw pain, clicking, or restricted movement [52,53]. Imaging is generally reserved for suspected structural pathology, with CT demonstrating osseous abnormalities (**Fig. 29**) and MRI evaluating the articular disc and surrounding soft tissues [54]. True pulse-synchronous tinnitus is uncommon in TMJ dysfunction; therefore, an underlying vascular cause should be excluded when PT is suspected.

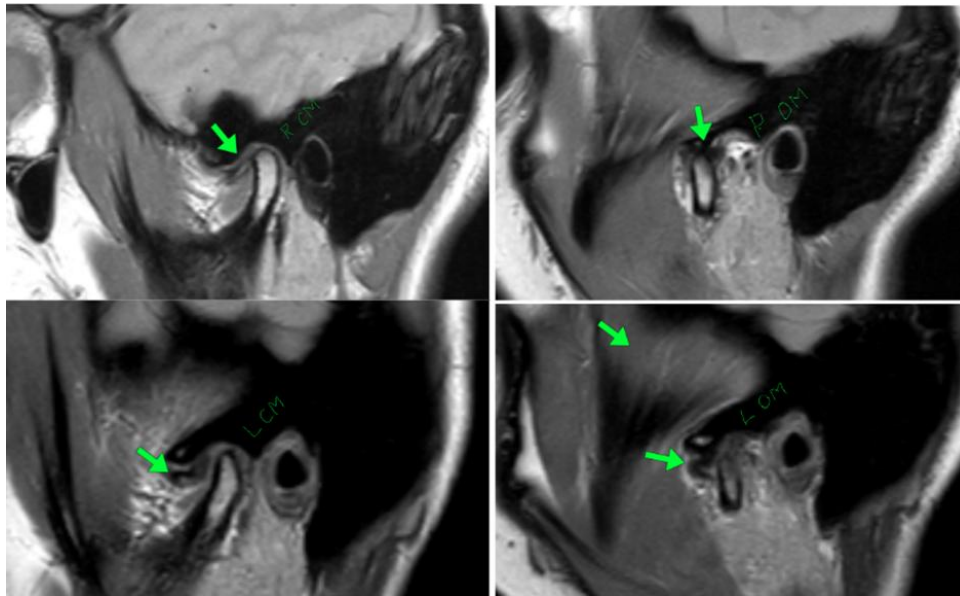


Fig. 29. Temporomandibular joint dysfunction. Sagittal oblique MR images of the temporomandibular joints demonstrate abnormal articular disc position/internal derangement (green arrows) in closed- and open-mouth positions (R- Right, L- left, CM- Closed mouth, OM-Open mouth).

5. Conclusion

Pulsatile tinnitus encompasses a broad spectrum of vascular, structural, neoplastic, systemic, and occasionally nonvascular conditions, many of which are treatable. A clinically directed multimodality imaging approach is therefore essential for identifying the underlying cause and distinguishing benign variants from potentially high-risk vascular lesions. HRCT is particularly valuable for temporal-bone and osseous abnormalities, whereas

MRI/MRA/MRV and CTA/CTV provide complementary assessment of intracranial, arterial, venous, and neoplastic pathology. Advanced techniques such as ASL, time-resolved MRA, and 4D-CTA can improve evaluation of suspected arteriovenous shunting, while DSA remains important when non-invasive imaging is inconclusive but clinical suspicion for a high-risk vascular lesion persists or detailed treatment planning is required. Careful integration of clinical findings with targeted imaging is central to accurate diagnosis and appropriate management of PT.

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