The Journal of International Advanced Otology

J Int Adv Otol 2025; 21: 1–7 • DOI: 10.5152/iao.2025.251923

Review

Pulsatile Tinnitus: A Narrative Review

Andrea Salgado Alvear[1], Gerardo Arrieta Limón[1], Laura Michelle Reyes Martínez[1], Raymundo Solís Gómez[2], Fabiola Eunice Serrano Arias[1]

[1]Department of Education, National Institute of Neurology and Neurosurgery, Mexico City, Mexico
[2]Postgraduate Studies and Research Section, National Polytechnic Institute School of Medicine, Mexico City, Mexico

ORCID IDs of the authors: A.S.A. 0000-0002-0232-2578, G.A.L. 0009-0008-3056-4807, L.M.R.M. 0009-0007-8821-0377, R.S.G. 0000-0003-4185-8655, F.E.S.A. 0000-0002-0350-3477.

Cite this article as: Alvear AS, Limón GA, Martínez LMR, Gómez RS, Arias FES. Pulsatile tinnitus: A narrative review. *J Int Adv Otol.* 2025, 21(4), 1923, doi: 10.5152/jiao.2025.251923.

Pulsatile tinnitus (PT) is a symptom consisting of the perception of sound without an external stimulus, synchronized with the patient's heartbeat. It accounts for 4% of all tinnitus cases. The most common etiologies are vascular, including carotid stenosis, idiopathic intracranial hypertension, sinus stenosis, aneurysms, and arteriovenous malformations. The diagnostic approach involves a complete history and clinical examination to determine if PT is of arterial or venous origin and to guide imaging studies. Treatment ranges from lifestyle modifications and pharmacological therapy to minimally invasive procedures like endovascular interventions and surgery. Minimally invasive endovascular procedures offer promising outcomes. This narrative review analyzes the etiologies, diagnostic approaches, and management strategies of PT, providing updated information to guide its approach. Most patients with PT have a treatable cause; however, despite a thorough diagnostic approach, a specific etiology is not found in approximately 30% of cases. Although most etiologies of PT are not life-threatening, it affects the patient's quality of life as it provokes psychological disturbances.

KEYWORDS: tinnitus, endovascular treatment, neurotology, idiopathic intracranial hypertension

INTRODUCTION

Tinnitus is a symptom consisting of the perception of sound without an external auditory stimulus. It comes from the Latin word ¨tinnire¨ and refers to a ringing or tinkling sound.[1] Nearly 10 to 15% of United States adults experience tinnitus, normally occurring in the seventh decade of life.[2]

It can be classified as objective or subjective and pulsatile or non-pulsatile. Objective tinnitus is also detected by the examiner during auscultation, while subjective tinnitus is only perceived by the patient, accounting for 80% of the cases.[1] In contrast with non-pulsatile tinnitus, pulsatile tinnitus (PT) is synchronous with the patient's heartbeat and occurs only in 4% of patients.[3]

Most patients presenting with pulsatile tinnitus have a treatable cause, but regardless of a thorough approach, no etiology is identified in 30% of cases.[4] Even though most etiologies are not life-threatening, tinnitus may be life-changing, leading patients to psychological and physical consequences like anxiety, depression, and insomnia.[5]

Pulsatile tinnitus is classified by etiology into two groups, vascular and nonvascular. Vascular causes account for the majority of cases; the synchronic sound originates from arterial or venous structures secondary to increased flow or stenosis causing turbulence, which propagates through bones into the cochlea.[6] Another plausible explanation for nonvascular causes is an increased perception of normal body flow sounds as a result of increased bone conduction.[4]

ETIOLOGIES

Vascular

The most common vascular etiologies are paragangliomas, dural arteriovenous fistulae (dAVFs), idiopathic intracranial hypertension (IIH), atheromatous arterial disease, and venous anatomical variations.[7] Vascular causes are classified by their origin into venous, arteriovenous, and arterial.

Corresponding author: Andrea Salgado Alvear, e-mail: andysalg99@gmail.com
Received: February 18, 2025 • **Revision Requested:** March 4, 2025 • **Last Revision Received:** March 16, 2025 • **Accepted:** March 25 2025 • **Publication Date:** July 21, 2025
Available online at www.advancedotology.org

Venous

Venous PT is perceived during diastole and tends to be softer and milder than arterial tinnitus. In contrast with arterial PT, venous PT can be relieved or exacerbated with neck manipulation.[5] The most common venous causes of PT are described below.

Idiopathic Intracranial Hypertension (IIH) and Transverse Sinus Stenosis (TSS)

Intracranial hypertension is the most common cause of venous PT. It is described as increased intracranial pressure without a structural cause and normal cerebrospinal fluid.[8] It has an incidence of 1 to 2 adults per 100 000 annually and 20 per 100 000 overweight women between 20 and 44 years.[5,9] Common symptoms include papilledema, headache, visual disturbances, cranial nerve palsies (V, VI, and VII), and PT, which is present in 65% of patients.[4] Several physiopathological theories have been proposed, including an intrinsic mechanism consisting of enlarged arachnoid granulations and anomalies of the glymphatic system.[9,10]

Transverse sinus stenosis (TSS) is present at magnetic resonance venography in 94% of patients with IIH and it is usually bilateral, but it is not clear if it is a cause or a consequence of IIH. TSS etiologies are divided into intrinsic and extrinsic. A common intrinsic cause is enlarged arachnoid granulations, which cause a focal stenosis and can be associated with IIH when the obstruction is bilateral or from the dominant venous system. The aforementioned are more frequently seen in older patients.[11] Other intrinsic causes include intrasinus tumors or thrombosis. Extrinsic TSS is normally diagnosed in younger patients, it affects long segments, and it is normally associated with IIH.[1,11] Both intrinsic and extrinsic stenoses cause a turbulent flow, which transmits to the cochlea as PT.[9] Transverse sinus stenting results in the resolution of PT in 95% of patients with or without IIH.[8]

Characteristic radiological findings of IIH include empty sella syndrome, optic nerve tortuosity, excess cerebrospinal fluid along the nerve sheaths causing distension mostly of the optic nerve, and posterior ocular globe flattening.[9,10] Diagnosis is confirmed by an increased cerebrospinal fluid pressure (>250 mm H_2O) in a lumbar puncture.[1]

Jugular Vein Abnormalities

Jugular vein abnormalities are believed to be acquired and are seen in 10% to 15% of asymptomatic patients. This includes jugular dehiscence and diverticulum, high-riding jugular bulb (HRJB), and stenosis, which are normally located on the right side.[9] PT is a common symptom of a high or dehiscent jugular bulb (JB).[8] When the JB lies at the level of the hypotympanum instead of below the middle ear, and is associated with a thin jugular plate, turbulent flow is transmitted to the mastoid air cells and cochlea, producing PT.[5,9] It is important to state that this may manifest as a retrotympanic bluish mass on otoscopy, which can be associated with hearing loss, decreased membrane mobility, and temporal bone bruits.[7,8] Dehiscence of the JB may be secondary to bone erosion produced by an HRJB.

JV stenosis can also present with PT, specifically when the contralateral side is hypoplastic. Other common symptoms include headache, dizziness, vision disorders, and memory decline. Causes of JV stenosis may be divided into internal and external. An example of internal stenosis is thrombosis, while external stenosis is normally secondary to compression of the JV by the styloid process.[5]

Recent studies suggest that jugular vein anomalies may not be a direct cause of PT. Li et al[12] in a retrospective study including 195 patients with PT who underwent CT angiography and venography, found that sigmoid sinus anomalies were present in 65.6% of patients, while ipsilateral HRJB and dehiscent jugular bulb were found in 54.9% and 14.4%, respectively. Regardless of the incidence of ipsilateral jugular vein anomalies found in the study, there was no significant statistical correlation between these findings and the laterality of PT (HRJB: $P = .187$, dehiscent jugular bulb: $P = .858$), suggesting that the causative role of JV anomalies may be limited.

Sigmoid Sinus Dehiscence and Diverticulum (SSDD)

Some studies report that SSDD are the most common causes of venous PT; they account for 20% of cases.[5] Sigmoid sinus dehiscence is defined as an absence of the temporal bone that separates the sigmoid sinus from the middle ear, while a diverticulum is an outpouching secondary to a bone defect.[9] Most SSDD involve the right side, probably secondary to the dominance of the right brain venous system.[13]

Sigmoid Sinus Dehiscence and Diverticula are associated with transverse sinus stenosis (TSS) and **IIH**. A suggested theory states that TSS causes a turbulent flow that gradually destroys and debilitates the sinus wall, generating a diverticulum.[8] The relationship between these entities is not well understood. Lansley et al[13] in a retrospective study suggested that SSDD or transverse sinus stenosis are not directly responsible for PT in patients with IIH.

Marginal Sinus Stenosis

Marginal sinus stenosis has been identified as a recent contributor to PT, showing similar symptoms to TSS. Given its anatomical association with the temporal bone, the pathophysiology of PT likely parallels that of TSS, characterized by turbulent blood flow secondary to venous stenosis. It predominantly affects the right side (63%) and can be detected on magnetic resonance. Resolution of PT has been achieved through marginal sinus stenting.[14]

Other novel causes of vascular PT described in small case series include the diaphragm of the internal carotid artery and diploic arteriovenous fistulas. The first is described as a translucent endoluminal web perpendicular to the arterial wall, and the second as an

MAIN POINTS

- Pulsatile tinnitus (PT) is a rare form of tinnitus, often caused by vascular abnormalities. Most etiologies are not life-threatening, but it affects patients' quality of life.
- A complete clinical history, physical examination, and specific imaging studies are crucial for diagnosing PT and differentiating between arterial and venous etiologies.
- Minimally invasive endovascular procedures show promising outcomes for managing specific PT etiologies, offering high resolution rates with low complication rates compared to surgical interventions.

arteriovenous fistula that drains into the diploic veins between the inner and outer tables of the skull.[15,16]

Emissary Veins

Emissary veins are valveless vessels that connect the extracranial and intracranial venous systems. They have a bidirectional blood flow, which regulates intracranial pressure. The mastoid vein, posterior condylar vein, and petrosquamosal vein are associated with PT.[5] The mastoid vein connects the transverse or sigmoid sinus with the posterior auricular or cervical vein, passing through the mastoid foramen. The posterior condylar emissary vein links the jugular bulb to the external vertebral venous system. The petrosquamosal vein is an anatomical variant present in some patients that bridges the transverse sinus with the retromandibular vein and pterygoid venous plexus.[9]

When an intracranial venous occlusion or arteriovenous shunt is present, the emissary veins receive increased blood flow, causing vasodilation, consequently the flow becomes turbulent, and its transmission to the cochlea may present as PT.[5,9]

Eliezer et al[17] reported in a retrospective study including 6 patients with PT secondary to mastoid emissary veins (MEV) that endovascular treatment is effective in patients with clinical venous PT, demonstrating that MEV can be a source of PT and the technical success of endovascular embolization.

Arteriovenous

Dural Arteriovenous Fistula (dAVF)

A dAVF is an acquired attachment between venous sinuses and dural vessels, secondary to a dural venous sinus thrombosis. They account for 15% of all intracranial arteriovenous malformations.[6] PT is the earliest symptom in 10% of patients and is characterized by being objective and loud.[4] Dural AVFs of the transverse sinus (70%), hypoglossal canal (10%), and middle cranial fossa (6.7%) are most frequently involved in PT.[18]

Carotid Cavernous Fistula (CCF)

A CCF is an acquired vascular malformation consisting of the communication between the ICA and the cavernous sinus. The etiology of CCF may be spontaneous or most often secondary to trauma. Common clinical manifestations include subjective bruit, blurred vision, headache, diplopia, proptosis, and chemosis. If the fistula is located in the posterior segment of the cavernous sinus, PT may also be a symptom.[8]

Arteriovenous Malformation (AVM)

An AVM is a congenital abnormal vascular configuration consisting of ecstatic and dilated vessels that connect the arterial and venous systems. It is an uncommon condition present in only in 0.1% of the population.[19] They are a rare cause of PT, contributing to less than 1% of the cases.[20]

Arterial

Arterial pulsatile tinnitus is perceived during the systolic phase of the cardiac cycle, and in contrast with venous etiologies, it has no changes in volume or character with neck position or compression of cervical vascular structures.[8] The most common arterial causes of PT are described below.

Carotid Stenosis

Carotid atherosclerotic disease is the most common cause of PT in geriatric patients, with a prevalence of 8-20% in the general population. Besides PT, patients may present with neck pain, headache, ischemic strokes, transient ischemic attacks, or retinal ischemia, but PT may be the first manifestation.[1,7] Risk factors include age ≥80 years (odds ratio [OR], 8.11; 95% confidence interval [CI], 3.45-18.93), hypertension (OR, 1.72; 95% CI, 1.21-2.45), and hyperlipidemia (OR, 1.84; 95% CI, 1.30-2.62).[21] It is essential to auscultate the neck, searching for carotid bruits; this finding is present in 70-89% of patients with a 2-mm luminal narrowing.[22] The most common screening test for assessing carotid stenosis is duplex ultrasound.[7]

Internal Carotid Artery Dissection

Carotid artery dissection is a condition where the ICA intima tears. It may be spontaneous or traumatic, and it is the most common cause of stroke in young adults. This leads to turbulent blood flow, which can lead to PT. PT develops in 5% to 15% of patients with ICA dissection, but it is rarely the only presenting symptom.[8] Griffin et al[23] in a retrospective observational cohort study including 63 patients with ICA dissection, found that the most common presenting symptoms were headache (49.2%), cerebral infarction (31.8%), Horner syndrome (28.6%), neck pain (23.8%), incidental finding (19.1%), and transient ischemic attack (11.1%). PT was present in 11.1% of patients.

Fibromuscular Dysplasia

Fibromuscular dysplasia (FMD) is a non-atherosclerotic and non-inflammatory angiopathy affecting middle-sized vessels. It can cause vascular narrowing, tortuosities, and dissections and it is the second most common cause of extra-cranial carotid stenosis, primarily affecting women aged 20-60 years.[6] According to Dicks et al[24] PT is present in 37.2% of patients with FMD. Other signs and symptoms include headache, dizziness, vertigo, and cervical bruit. Some patients may present with transient ischemic attacks or stroke.[25]

Aneurysms

Aneurysms of the internal carotid artery (ICA), vertebral arteries, or anterior inferior cerebellar arteries induce turbulent flow; nevertheless, they are a rare cause of PT. Specifically, petrous carotid aneurysms present with symptoms such as hearing loss, epistaxis, and headache.[8] Symptoms of dissecting aneurysms consist of pain, subarachnoid hemorrhage, cranial neuropathies, and transient ischemic attack.[6]

Aberrant Carotid Arteries

Patients with an aberrant course of the carotid artery demonstrate an absence of the normal vertical segments and growth of the inferior tympanic and caroticotympanic arteries located in the middle ear.[26] A retrotympanic mass may be seen during otoscopy, and patients may be asymptomatic or present with hearing loss, PT, pain, and aural fullness.[27]

Persistent Stapedial Artery

It is an uncommon congenital anomaly present in 0.05% of the population, consisting of the persistence of the embryological stapedial artery.[26] It is often associated with an aberrant internal carotid artery and the absence of foramen spinosum. Patients may be

asymptomatic or suffer from PT due to turbulent flow in the persistent stapedial artery.[18]

Tortuosities

Carotid

Carotid artery tortuosity is the elongation of the ICA presenting on imaging as coiling, looping, or kinking. Patients are normally asymptomatic (80%), but they may also present with PT, dizziness, or stroke.[28] It is often seen in older adults and normally coexists with an audible bruit.[6]

Occipital

Arganbright et al[29] reported a case of a patient with PT secondary to an anomalous course of the occipital artery. A computed tomography angiography (CTA) was obtained, and a tortuous occipital artery was seen over the lateral segment of the mastoid. The patient's PT was completely resolved after ligation and resection of the anomalous portion of the artery.

NonVascular and Other Causes

Superior Semicircular Canal Dehiscence

Superior semicircular canal dehiscence (SSCD) is an abnormal communication between the semicircular canal and the middle cranial fossa. This enhances bone conduction, which may manifest as PT. Other symptoms of SSCD include vertigo, Tullio´s phenomenon, hearing loss, and chronic disequilibrium.[8]

Paragangliomas

Paragangliomas are benign tumors of the skull base presenting in the jugular bulb and middle.

ear with a high vasculature.[4] Tympanic and jugular paragangliomas usually manifest with subjective PT and conductive hearing loss. They may also present with lower cranial nerve dysfunction.[3,6] They may be identified during otoscopy or diagnosed using imaging studies of the temporal bone.[3]

Less frequent nonvascular causes of PT include systemic or regional conditions associated with increased blood flow such as pregnancy, anemia, thyrotoxicosis, osseous dysplasia, Paget disease, otosclerosis, cholesteatomas, and superior semicircular canal dehiscence.[1]

Figure 1 represents a schematic image summarizing the most common causes of PT described in the previous paragraphs.

Evaluation

The diagnostic approach of PT includes a rigorous and detailed history and examination, which are the most important primary tools, along with an audiological workup, blood tests, and imaging.[8,30] According to the American Academy of Otolaryngology and Head and Neck Surgery Foundation (AAO-HNS), a rigorous anamnesis and an extended physical exam should be performed before any imaging.[31]

History

The examiner should investigate the following characteristics of PT: chronicity, location or laterality, character (pulsatile or continuous), exacerbating and alleviating factors, association with other symptoms, and the impact on the patient's daily living.[3,30] The last characteristic may be evaluated by the Tinnitus Handicap Inventory, the Tinnitus Questionnaire, and the Hearing and Tinnitus Survey.[2] In order to evaluate the character of tinnitus objectively, the examiner may ask the patient to imitate the sound. A complete otologic history is also essential to detect associated symptoms such as hearing loss, vertigo, otalgia, otorrhea, and aural fullness.[3]

Since PT may be associated with medical conditions, a thorough drug and past medical history should also be acquired, including

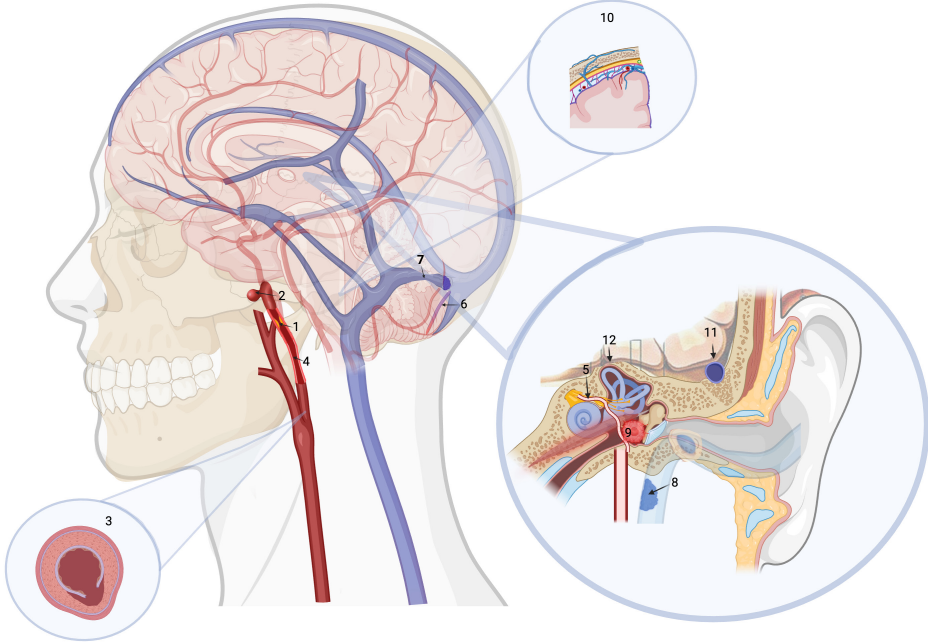

Figure 1. Common causes of PT: 1. Proximal Internal Carotid Atherosclerotic Disease 2. Internal Carotid Artery Aneurysm 3. Internal carotid Artery Dissection 4. Internal Carotid Stenosis Secondary to Fibromuscular Dysplasia 5. Persistent stapedial artery 6. Dural arteriovenous fistula from the Transverse Sinus to the Occipital Artery 7. Idiopathic Intracranial Hypertension (IIH) and Transverse Sinus Stenosis (TSS) and Thrombosis 8. Jugular Vein Abnormalities (Internal Jugular Vein Thrombosis) 9. Tympanic paraganglioma 10. Mastoid Emissary Vein 11. Sigmoid Sinus Dehiscence 12. Superior Semicircular Canal Dehiscence

history of strokes, cancer, thyroid disorders, vascular malformations, and chronic diseases.[30] Atherosclerotic carotid artery disease should be suspected in older patients with PT and a history of hypertension, diabetes, smoking, cerebrovascular disease, and dyslipidemia.[9,6] On the other hand, young and obese women with hearing loss, aural fullness, and headache should be evaluated for IIH.[30]

Clinical Examination

Clinical examination is crucial for the diagnostic approach of PT and should include a complete neurological and head and neck examination, auscultation, provocation and rotation maneuvers, otoscopy, and audiological evaluation.[3]

Searching for symptoms suggesting intracranial hypertension, such as headache, visual disturbances, papilledema, and nausea is an essential step of the neurological examinaton. If the aforementioned symptoms are present, a lumbar puncture should be performed. A cranial nerve examination should also be conducted.[4]

Auscultation of the ear canal, periauricular region, orbits, neck, and chest is essential to identify objective PT, bruits, and heart murmurs.[6] Particular emphasis should be given while auscultating the postauricular and upper neck regions since these locations are frequently associated with dural AVFs. Any bruit in these areas should be presumed to be an intracranial vascular abnormality until confirmed otherwise. This examination should ideally be ideally performed in a very quiet setting, such as an audiology booth, preferably using a modified electronic stethoscope.[8] When objective PT is present, its rate should be compared to the arterial pulse, and light digital pressure on the ipsilateral IJV should be applied.[6,30]

Venous PT can often be alleviated by using maneuvers that decrease the jugular blood flow; these maneuvers include gentle compression of the ipsilateral IJV, the Valsalva maneuver, or turning the head towards the affected side since the IJV is compressed by the sternocleidomastoid and the atlas. In contrast, compressing the contralateral jugular vein may increase PT intensity by redirecting venous flow. The aforementioned maneuvers have a 93% sensitivity in identifying venous PT.[1] Tinnitus may worsen with physical exertion and lying down, especially in quiet environments.[6,30]

Otoscopy is crucial to exclude middle-ear pathology, as it may show a retrotympanic mass.[6] Pathologies that present with this clinical finding include a high or exposed jugular bulb, intratympanic glomus tumors (paragangliomas), and aberrant carotid arteries.[7] Patients with tensor tympani myoclonus may reveal rhythmic movements in the tympanic membrane, soft palate, or pharynx. Opening the oral cavity widely could potentially cease the myoclonic contractions of the soft palate.[6]

In order to identify any hearing impairment, all patients with PT should undergo a complete audiological exploration, including the Weber and Rinne tests, and audiometry. A computed tomography of the temporal bone is suggested for all individuals with conductive hearing loss.[26]

Imaging Studies

AAO-HNS strongly recommends conducting a thorough physical examination and audiometry to follow an appropriate diagnostic pathway and request the adequate studies depending on the potential etiology of tinnitus. The guidelines highlight that imaging studies are not recommended when PT is bilateral, non-pulsatile, and is not associated with focal neurological abnormalities or asymmetric hearing loss.[31]

Imaging assessment should be patient-specific depending on the anamnesis and physical examination findings, including the presence or absence of a retrotympanic mass, objective PT, papilledema, or specific maneuvers suggesting an arterial or venous origin.[6]

When a mass is detected during otoscopy, a computed tomography (CT) scan to evaluate the middle ear is required. This study can diagnose conditions such as arterial anomalies, an exposed jugular bulb, or intratympanic tumors, most commonly paragangliomas.[6] Glomus tumors appear as bright red retro-tympanic nests in the inferior middle ear.[8] CT is particularly useful for assessing the extent of glomus tumors due to their permeative erosion of the skull base. A magnetic resonance imaging (MRI) can also be done to complement the diagnostic approach; typical findings include a salt and pepper appearance on T2.[8,30]

On the contrary, when otoscopy is normal, magnetic resonance venography and angiography should be initially obtained.[6]

Another proposed initial study in patients with normal otoscopy is a combined computed tomography angiography and venography (CTA/V) with 100 mL contrast and a fixed delay of 25 seconds. It searches for middle ear, skull base, and vascular conditions in a single examination.[7] The image should range from the aortic arch to the vertex.[1]

Lynch et al[32] in a retrospective study including 251 patients with PT found that MRI achieved a 100% diagnostic rate for neoplasms, while CT detected middle and inner ear pathology in 89% of cases. MRA and CTA diagnosed arterial PT at rates of 81% and 89%, respectively, concluding MRI and CT-based images are both successful in diagnosing PT.

In idiopathic intracranial hypertension (IIH), magnetic resonance venography (MRV) and MRI are the suggested initial studies. More than 90% of cases show bilateral venous sinus stenoses on MRV.[1,7] Imaging is used to exclude other causes of intracranial hypertension, but diagnosis is confirmed by measuring the opening pressure with a lumbar puncture. Radiological signs that can be observed include an empty sella, flattening of the posterior globes, and distension of the perioptic arachnoid spaces.[7]

If non-invasive imaging does not reveal the cause of arterial tinnitus, conventional angiography should be considered to rule out a small dural arteriovenous fistula (dAVF), which can also be detected on CTA/V, 3D TOF MRI sequences, or post-gadolinium MRI/MRA studies.[7,30] Angiography is also recommended in patients with an initial strong suspicion of AVF or AVM and in surgical candidates.[1]

Carotid atherosclerosis, which is responsible for 8-20% of PT cases, is typically evaluated with duplex ultrasound.[7] This study should be the first to be performed since it may be the only test required to detect carotid disease.[6] Fibromuscular dysplasia, which is the second most common cause of extracranial carotid artery stenosis, shows arteries with a "string of beads" appearance on CTA or angiography.[7,8]

Management

Management should focus on resolving the main cause of PT.[25] It is important to emphasize the significance of behavioral and psychological treatment, such as tinnitus retraining therapy and cognitive behavioral therapy.[5]

The following outlines the treatment of the most prevalent PT causes.

Venous

In IIH, the first step in treatment involves dietary changes and weight loss, which eliminate PT in most patients.[25] Adding acetazolamide to the lifestyle changes reduces even more CSF production, but this drug has several side effects, making adherence to the treatment difficult for patients.[5] When the aforementioned does not resolve the symptoms, invasive procedures such as therapeutic lumbar puncture or surgical/endovascular interventions may be performed.[9]

For those experiencing worsening vision, persistent headaches, and severe PT, a lumbar-peritoneal shunt might be suggested; this procedure can be counterproductive in morbidly obese patients due to shunt blockage caused by increased abdominal pressure. Following the CSF diversion, most patients experience amelioration of their symptoms; however, the procedure has a high revision and complication rate of 43% and 33%, respectively.[5]

Nicholson et al[33] in a systematic review and meta-analysis including 20 studies and 474 patients with IIH who underwent venous sinus stenting found that endovascular treatment has a high resolution and low major complication rate. Most patients experienced resolution of their symptoms, including papilledema (93.7% (95% CI 90.5% to 96.9%)), PT (90.3% (95% CI 83.8% to 96.70%)), and headache (79.6% (95% CI 73.3% to 85.9%)). After an 18-month follow-up, only 9.8% (95% CI 6.7% to 13%) of patients faced recurrence of their symptoms and 1.9% (95% CI 0.07% to 3.1%) suffered major complications.

Symptomatic dehisced jugular bulbs and diverticulum can be repaired using bone dust, perichondrium, tragal cartilage, conchal cartilage, and mastoid cortical bone.[25] Endovascular treatments, such as coiling, stent-assisted coiling, and WEB embolization, have shown effectiveness in case reports. However, jugular vein stenting has a considerable risk of stent migration, stent thrombosis, and lower cranial neuropathy.[5,9]

Coil embolization of sigmoid sinus diverticulum is generally well-tolerated and leads to relief of symptoms in most cases.[5,9] Sigmoid sinus dehiscence is treated surgically with resolution of symptoms in 74% of cases, with a complication rate of 24%, including CSF leak and sinus thrombosis.[5]

In patients with symptomatic isolated venous sinus stenosis without IIH, stenting is the primary treatment option.[9] When stenosis is extrinsic, several authors suggest using a larger stent to avoid further compression beyond the stent.[34] Stenting is also effective in marginal sinus stenosis, achieving a complete resolution of PT without complications in case series.[14]

Yang et al[35] in a literature review of 21 studies involving 41 patients who underwent endovascular treatment for sinus stenosis and diverticula, found that 39 patients (95.1%) achieved a complete resolution of PT, with only 1 complication and no mortality associated. The effectiveness and safety suggest considering this as first-line treatment when medical management fails.[35,36]

Coil embolization is also an effective treatment for patients with emissary vein anomalies. To determine if an emissary vein is causing PT, a balloon test occlusion is performed. If PT stops while the balloon is inflated and the vein is not critical for collateral pathways, the vein is sacrificed using a coil.[5,9]

Arterial and Arteriovenous

Treating arterial and arteriovenous causes of PT is not only intended to mitigate or resolve symptoms but also to reduce the risk of ischemic or hemorrhagic stroke.[5]

In dAVF, the primary goal is reducing the hemorrhagic stroke risk. When it presents with a low risk and symptoms are manageable, conservative treatment is suggested. On the other hand, when the fistula is symptomatic and has a high risk of stroke, endovascular treatment is recommended as the first line of treatment.[5] The same management is proposed for carotid cavernous fistulas.[8]

Most cases of internal carotid artery dissection (46-90%) are self-limiting. However, when the lesion is progressive or causes hemodynamic instability, angioplasty and stenting are advised. In patients with FMD, asymptomatic patients should be treated with antiplatelet therapy only. In contrast, several case reports indicate that stenting resolves PT in symptomatic patients or those with recurrent ischemic events.[8]

Surgery and stereotactic radiosurgery are considered the gold standard for treatment of paragangliomas due to their high success rates and low recurrence and complications. PT is resolved in up to 100% of patients who undergo surgery.[8,25]

Nonvascular

Available treatments for patients with tympani, stapedial, and levator veli palatini myoclonus include sectioning the muscle or applying botulinum toxin. Recent studies suggest botulinum toxin injection is a more appropriate treatment.[25]

Finally, SSCD treatment consists of resurfacing or plugging the dehiscent superior semicircular canal via a subtemporal craniotomy or a transmastoid approach. Surgical interventions provide the highest rates of full symptom resolution.[8]

CONCLUSION

PT is a symptom associated with diverse pathologies. Although most etiologies are not life-threatening, tinnitus may be life changing, leading patients to psychological consequences and causing a significant morbidity. Clinical history and physical examination are crucial for an accurate diagnosis and serve as a basis for the efficient use of imaging studies. Minimally invasive endovascular procedures offer promising outcomes and a lower complication and mortality rate than surgical interventions. However, further randomized controlled clinical trials are needed to compare treatment success and complication rates.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Design – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Supervision – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Resources – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Materials – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Data Collection and/or Processing – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Analysis and/or Interpretation – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Literature Search – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Writing Manuscript – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.; Critical Review – A.S.A., G.A.L., L.M.R.M., R.S.G., F.E.S.A.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study has received no financial support.

REFERENCES

1. Abdalkader M, Nguyen TN, Norbash AM, et al. State of the art: venous causes of pulsatile tinnitus and diagnostic considerations guiding endovascular therapy. *Radiology*. 2021;300(1):2-16. [CrossRef]
2. Dalrymple SN, Lewis SH, Philman S. Tinnitus: diagnosis and management. *Am Fam Physician*. 2021;103(11):663-671.
3. Chari DA, Limb CJ. Tinnitus. *Med Clin North Am*. 2018;102(6):1081-1093. [CrossRef]
4. Hofmann E, Behr R, Neumann-Haefelin T, Schwager K. Pulsatile tinnitus: Imaging and differential diagnosis. *Dtsch Arztebl Int*. 2013;110(26):451-458. [CrossRef]
5. Narsinh KH, Hui F, Duvvuri M, Meisel K, Amans MR. Management of vascular causes of pulsatile tinnitus. *J NeuroInterv Surg*. 2022;14(11):1151-1157. [CrossRef]
6. Sismanis A. Pulsatile tinnitus. *Otolaryngol Clin N Am*. 2003;36(2):389-402. [CrossRef]
7. Madani G, Connor SEJ. Imaging in pulsatile tinnitus. *Clin Radiol*. 2009;64(3):319-328. [CrossRef]
8. Pingree GM, Fleming C, Reavey-Cantwell J, Coelho DH. Neurosurgical causes of pulsatile tinnitus: contemporary update. *Neurosurgery*. 2022;90(2):161-169. [CrossRef]
9. Essibayi MA, Oushy SH, Lanzino G, Brinjikji W. Venous causes of pulsatile tinnitus: clinical presentation, clinical and radiographic evaluation, pathogenesis, and endovascular treatments: a literature review. *Neurosurgery*. 2021;89(5):760-768. [CrossRef]
10. Nicholson P, Kedra A, Shotar E, et al. Idiopathic intracranial hypertension: Glymphedema of the brain. *J Neuroophthalmol*. 2021;41(1):93-97. [CrossRef]
11. Lenck S, Nicholson P. Cerebral venous wall diseases: the other side of the picture. *AJNR Am J Neuroradiol*. 2021;42(2):297-298. [CrossRef]
12. Li P, Li L, Ma X, et al. Anatomical variations of the jugular foramen region in patients with pulsatile tinnitus. *J Neurol Surg B*. 2021;83:248-253.
13. Lansley JA, Tucker W, Eriksen MR, Riordan-Eva P, Connor SEJ. Sigmoid sinus diverticulum, dehiscence, and venous sinus stenosis: potential causes of pulsatile tinnitus in patients with idiopathic intracranial hypertension? *AJNR Am J Neuroradiol*. 2017;38(9):1783-1788. [CrossRef]
14. Cortese J, Eliezer M, Guédon A, Houdart E. Pulsatile tinnitus due to stenosis of the marginal sinus: diagnosis and endovascular treatment. *AJNR Am J Neuroradiol*. 2021;42(12):2194-2198. [CrossRef]
15. Lenck S, Labeyrie MA, Mosimann PJ, Saint-Maurice JP, Houdart E. Diaphragm of the internal carotid artery: a novel cause of pulsatile tinnitus. *J Neurol*. 2013;260(8):2185-2187. [CrossRef]
16. Guédon A, Eliezer M, Houdart E. Pulsatile tinnitus revealing a diploic arteriovenous fistula. *Clin Neuroradiol*. 2022;32(2):571-575. [CrossRef]
17. Eliezer M, Freitas RK, Fantoni M, Guedon A, Houdart E. Selective embolization of the mastoid emissary vein for pulsatile tinnitus treatment: when is it indicated? *J Neurointerv Surg*. 2020;12(10):999-1001. [CrossRef]
18. Kumar R, Rice S, Lingam RK. Detecting causes of pulsatile tinnitus on CT arteriography-venography: a pictorial review. *Eur J Radiol*. 2021;139:109722. [CrossRef]
19. Al Afif AA, Alamoudi U, Al-Sayed AA, Bance M. Multiple venous malformations as a cause of pulsatile tinnitus. *Case Rep Otolaryngol*. 2020;2020:8867963. [CrossRef]
20. Mohr JP, Kejda-Scharler J, Pile-Spellman J. Diagnosis and treatment of arteriovenous malformations. *Curr Neurol Neurosci Rep*. 2013;13(2):324. [CrossRef]
21. Woo SY, Joh JH, Han SA, Park HC. Prevalence and risk factors for atherosclerotic carotid stenosis and plaque: a population-based screening study. *Medicine*. 2017;96(4):e5999. [CrossRef]
22. Grotta JC. Clinical practice. Carotid stenosis. *N Engl J Med*. 2013;369(12):1143-1150. [CrossRef]
23. Griffin KJ, Harmsen WS, Mandrekar J, Brown RD, Keser Z. Epidemiology of spontaneous cervical artery dissection: population-based study. *Stroke*. 2024;55(3):670-677. [CrossRef]
24. Dicks AB, Gornik HL, Gu X, et al. Association of Fibromuscular Dysplasia and Pulsatile Tinnitus: a report of the US registry for fibromuscular dysplasia. *J Am Heart Assoc*. 2021;10(17):e021962. [CrossRef]
25. Sismanis A. Pulsatile tinnitus: contemporary assessment and management. *Curr Opin Otolaryngol Head Neck Surg*. 2011;19(5):348-357. [CrossRef]
26. Cunnane MB. Imaging of tinnitus. *Neuroimaging Clin N Am*. 2019;29(1):49-56. [CrossRef]
27. Hosokawa S, Mizuta K. Pulsatile tinnitus with a whitish mass in the middle ear. *Eur Ann Otorhinolaryngol Head Neck Dis*. 2021;138(5):413-414. [CrossRef]
28. Raidah A, Jaramillo N, Serena G, Lev S, Ramanathan A. Tortuous carotid arteries and their clinical implications: a report of two cases. *Cureus*. 2023;15(3):e36953. [CrossRef]
29. Arganbright J, Friedland DR. Pulsatile tinnitus. *Otol Neurotol*. 2008;29(3):416-416. [CrossRef]
30. Liyanage SH, Singh A, Savundra P, Kalan A. Pulsatile tinnitus. *J Laryngol Otol*. 2006;120(2):93-97. [CrossRef]
31. Jain V, Policeni B, Juliano AF, et al. ACR appropriateness criteria tinnitus: 2023 update. *J Am Coll Rad*. 2023:20-91.
32. Lynch P, Mitton T, Killeen DE, Kutz JW, Newcomer M. Diagnosing pulsatile tinnitus: a review of 251 patients. *Otol Neurotol*. 2022;43(1):128-136. [CrossRef]
33. Nicholson P, Brinjikji W, Radovanovic I, et al. Venous sinus stenting for idiopathic intracranial hypertension: a systematic review and meta-analysis. *J NeuroIntervent Surg*. 2019;11(4):380-385. [CrossRef]
34. Lenck S, Vallée F, Labeyrie MA, et al. Stenting of the lateral sinus in idiopathic intracranial hypertension according to the type of stenosis. *Neurosurgery*. 2017;80(3):393-400. [CrossRef]
35. Yang IH, Pereira VM, Lenck S, et al. Endovascular treatment of debilitating tinnitus secondary to cerebral venous sinus abnormalities: a literature review and technical illustration. *J NeuroIntervent Surg*. 2019;11(8):841-846. [CrossRef]
36. Labeyrie MA, Fantoni M, Vever U, et al. Intracranial venous sinus stenting for the treatment of lateral sinus stenoses: an analysis of 200 patients. *Diagn Interv Imaging*. 2021;102(10):619-627. [CrossRef]