

# AUDITORY THROMBOSIS

Authored by  
**mohammad looti**

November 7, 2025

## RECOMMENDED CITATION

mohammad looti (2025). *AUDITORY THROMBOSIS*. PSYCHOLOGICAL SCALES.  
Retrieved from <https://scales.arabpsychology.com/?p=66067>

## AUDITORY THROMBOSIS

**Primary Disciplinary Field(s):** Otolaryngology, Neurology, Vascular Medicine

### 1. Core Definition and Pathophysiology

Auditory thrombosis refers specifically to the occlusion of the internal auditory artery (also known as the labyrinthine artery) due to the formation or lodging of a blood clot (thrombus or embolus). This vascular event leads to acute ischemia of the delicate inner ear structures, particularly the cochlea and the vestibular apparatus. The internal auditory artery is typically a branch of the anterior inferior cerebellar artery (AICA) or, less commonly, the basilar artery, and it is the sole vascular supply for the peripheral auditory and vestibular systems. Given its status as an end artery with minimal collateral circulation, any blockage immediately compromises blood flow, leading to rapid cell death and dysfunction.

The resulting lack of oxygen and nutrients (hypoxia and anoxia) causes irreversible damage to the highly metabolic hair cells within the organ of Corti. Since these sensory cells are crucial for converting sound vibrations into neural signals, their demise manifests clinically as sudden and often profound sensorineural hearing loss (SSHL). While not all cases of sudden deafness are attributed to vascular causes, auditory thrombosis represents one of the most severe and definitive etiologies. The mechanism involves the clot obstructing the narrow lumen of the artery, halting perfusion, and initiating an ischemic cascade that culminates in functional loss within hours.

Understanding the pathophysiology requires appreciating the intricate microvasculature of the inner ear. The internal auditory artery divides into the common cochlear artery and the anterior vestibular artery. If the occlusion occurs proximal to this division, both hearing and vestibular function may be lost. If the blockage is distal, only specific branches may be affected, potentially leading to isolated cochlear or vestibular dysfunction. Because the cochlea is particularly sensitive to fluctuations in blood flow, the rapid onset of severe hearing impairment is the hallmark clinical presentation of auditory thrombosis, distinguishing it from gradually progressive forms of hearing loss.

### 2. Etiology and Risk Factors

The formation of an auditory thrombus is generally linked to systemic conditions that predispose an individual to vascular disease and hypercoagulability. Although the condition is relatively rare, its occurrence is highly correlated with cardiovascular risk factors common in the general population. Primary systemic disorders such as severe atherosclerosis in the vertebral or basilar arteries, which supply the AICA, can lead to local thrombosis that propagates into the labyrinthine artery. Furthermore, conditions causing inflammation of the blood vessel walls (vasculitis), such as polyarteritis nodosa or systemic lupus erythematosus, can trigger local clot formation and

subsequent occlusion.

A significant number of cases arise from embolic events, where a clot originating elsewhere in the circulatory system travels and lodges in the internal auditory artery. Common sources of such emboli include cardiac conditions like atrial fibrillation, valvular heart disease, or recent myocardial infarction, which produce mural thrombi that break off. Paradoxical emboli, where a venous clot crosses into the arterial circulation through a patent foramen ovale (PFO), also represent a critical, though less common, risk factor, especially in younger patients without typical atherosclerotic disease. The narrow caliber and tortuous path of the internal auditory artery make it a prime location for the impaction of even small emboli.

Beyond structural heart disease, several hypercoagulable states significantly increase the risk of thrombosis throughout the body, including the inner ear circulation. These include inherited or acquired thrombophilias such as Factor V Leiden mutation, deficiencies in protein C or S, antiphospholipid syndrome, and elevated homocysteine levels. Lifestyle factors such as uncontrolled diabetes mellitus, long-standing hypertension, hyperlipidemia, and heavy smoking dramatically accelerate the underlying process of atherosclerosis, making the vascular endothelium prone to plaque rupture and subsequent thrombus formation. Therefore, a comprehensive evaluation of a patient presenting with suspected auditory thrombosis must include a thorough assessment of their systemic vascular health and coagulation status.

### 3. Clinical Presentation and Diagnosis

The defining characteristic of auditory thrombosis is the abrupt onset of hearing loss, often noted immediately upon waking or during routine daily activities. The hearing loss is typically unilateral and severe to profound in magnitude. Patients frequently describe the onset as instantaneous, distinguishing it clearly from gradual loss. This acute presentation is often accompanied by other symptoms indicative of inner ear damage, reflecting the shared vascular supply to the auditory and vestibular organs.

The most common accompanying symptom is tinnitus, a persistent ringing or buzzing sound in the affected ear, which occurs in the vast majority of cases. Approximately 40-50% of patients will also experience significant vertigo or dizziness, sometimes severe enough to cause nausea and imbalance. If the vestibular symptoms are prominent and prolonged, it strongly suggests a more proximal occlusion of the internal auditory artery, affecting both cochlear and vestibular branches. Diagnosis relies heavily on objective audiometric testing, which confirms the sensorineural nature and magnitude of the loss, typically showing a drop of at least 30 dB across three consecutive frequencies within 72 hours.

While audiometry confirms the functional loss, diagnosis of auditory thrombosis as the specific etiology requires exclusion of other causes and sometimes relies on imaging studies. Magnetic

Resonance Imaging (MRI), particularly diffusion-weighted imaging (DWI), is crucial for detecting concurrent ischemic events in the brainstem or cerebellum, which would confirm a proximal vascular origin (such as AICA occlusion). However, directly visualizing a small thrombus in the labyrinthine artery itself is technically challenging. Therefore, the diagnosis of auditory thrombosis is often presumptive, based on the clinical picture of acute, severe, isolated SSHL combined with known systemic vascular risk factors, especially when other common viral or idiopathic causes have been ruled out.

#### 4. Differential Diagnosis (Vascular vs. Non-Vascular Causes of SSHL)

Auditory thrombosis falls under the umbrella term of Sudden Sensorineural Hearing Loss (SSHL), a condition where the specific cause remains idiopathic (unknown) in up to 90% of cases. Therefore, a critical step in managing a patient with SSHL is to differentiate a true vascular event like thrombosis from other possible etiologies. Non-vascular causes of SSHL include viral infections (e.g., herpes simplex virus), autoimmune disorders (e.g., Cogan's syndrome), inner ear trauma, and perilymphatic fistulas. The high rate of idiopathic diagnosis reflects the difficulty in definitively isolating the underlying pathology in the inner ear.

Distinguishing between vascular and idiopathic SSHL is clinically significant because their prognoses and treatments may vary. Vascular etiology, particularly confirmed auditory thrombosis or embolism, often presents with specific indicators. For instance, the presence of severe vertigo, particularly if accompanied by subtle neurological deficits (even transient ones), significantly raises suspicion for a vascular event involving the AICA territory. Furthermore, patients with pre-existing cardiovascular disease or established hypercoagulable states are more likely to have thrombosis as the root cause.

The primary challenge lies in the fact that many idiopathic cases are hypothesized to have a microvascular or transient ischemic basis that cannot be proven by current imaging techniques. This overlap complicates the differentiation. Therefore, clinical guidelines often recommend a comprehensive workup to screen for high-risk vascular factors and systemic disease. If the clinical presentation is consistent with a profound, isolated loss in a patient with significant atherosclerotic risk, and infectious or inflammatory markers are negative, auditory thrombosis remains a leading working diagnosis, guiding the initiation of treatments aimed at improving blood flow and reducing clot burden.

#### 5. Treatment Strategies

The treatment of auditory thrombosis is aligned with protocols for managing acute SSHL, prioritizing rapid intervention to salvage viable inner ear structures. The standard initial therapy for most SSHL cases, regardless of suspected etiology, involves high-dose systemic glucocorticoids

(corticosteroids). These medications are administered orally or via intratympanic injection, aiming to reduce inflammation and edema within the cochlea that may exacerbate ischemic injury or swelling in the tight confines of the bony labyrinth. Early administration is crucial, usually within the first two weeks of onset, as delayed treatment yields significantly poorer results.

When auditory thrombosis is strongly suspected due to coexisting risk factors or neurological findings, treatment often incorporates strategies aimed at addressing the underlying vascular compromise. Anticoagulation therapy (e.g., heparin followed by warfarin or novel oral anticoagulants) or antiplatelet therapy (e.g., aspirin or clopidogrel) may be initiated to prevent further clot formation or progression, particularly if a cardioembolic source is identified. However, the use of systemic anticoagulation for isolated auditory thrombosis remains controversial and requires careful balancing of the risk of bleeding versus the potential benefit of reperfusion in a highly vascularized area.

Additionally, ancillary treatments focused on improving inner ear oxygenation and blood flow are sometimes employed, though evidence for their efficacy varies. These include hyperbaric oxygen therapy (HBOT), which aims to increase oxygen diffusion into the ischemic tissues, and vasodilators, intended to widen the internal auditory artery. Ultimately, the therapeutic approach is often multi-faceted, combining anti-inflammatory agents (steroids) with vascular risk factor modification and, in selected cases, targeted interventions for confirmed systemic cardiovascular disease or hypercoagulable states contributing to the thrombus formation.

## 6. Prognosis and Long-Term Outcomes

The prognosis for hearing recovery following auditory thrombosis is generally considered guarded, and it often depends heavily on the severity of the initial occlusion and the speed of intervention. Since the internal auditory artery is an end artery, the ischemic insult is usually profound and results in irreversible damage to the sensitive spiral ganglion neurons and hair cells. Patients who experience profound hearing loss and significant accompanying vertigo--suggesting a complete or near-complete occlusion of the proximal artery--typically have the worst outcomes regarding hearing recovery.

However, spontaneous partial recovery can occur in some cases, particularly if the initial thrombus was small or if collateral circulation, though limited, manages to partially restore perfusion before complete necrosis occurs. If recovery is achieved, it usually begins within the first few weeks after the event. The most significant predictor of poor outcome is delayed presentation (more than four weeks after onset) and the presence of severe comorbidities such as uncontrolled diabetes or severe vascular disease, which suggests a broader microcirculatory compromise.

Long-term management focuses on rehabilitation. For those with permanent profound unilateral hearing loss, interventions such as hearing aids, contralateral routing of signal (CROS) devices, or

cochlear implantation may be considered to improve communication ability and reduce the debilitating effects of single-sided deafness. Furthermore, long-term preventative care is mandatory, focusing on aggressive modification of systemic risk factors (blood pressure control, lipid management, smoking cessation) to prevent future thromboembolic events in the remaining ear or other vital organs, such as the brain.

## 7. Historical Context and Terminology

The recognition of vascular causes of sudden deafness dates back to early 20th-century otologic literature, but the specific concept of auditory thrombosis became prominent as clinicians sought to categorize the seemingly idiopathic cases of SSHL. Early theories posited that vascular insufficiency, hemorrhages, or specific thrombotic events were responsible for a significant fraction of sudden hearing loss long before modern imaging techniques could confirm the exact pathology. The term "auditory thrombosis" provides a clear, mechanistic explanation for the underlying physiological failure: localized blood clotting.

While the term remains clinically useful, in contemporary medical literature, the diagnosis is often subsumed under the broader, more cautious classification of Sudden Sensorineural Hearing Loss (SSHL) with suspected vascular etiology. This shift reflects the difficulty in obtaining definitive pathological proof of thrombosis in the labyrinthine artery during life. However, when SSHL is confirmed to be part of a larger stroke syndrome involving the AICA, the term is strongly validated, reinforcing the principle that the inner ear is essentially a peripheral extension of the cerebrovascular system, subject to the same thrombotic and embolic risks as the brain itself.

### Further Reading

[Sudden sensorineural hearing loss \(Wikipedia\)](#)

[Labyrinthine Artery \(Internal Auditory Artery\) \(Wikipedia\)](#)

[Thrombosis \(Wikipedia\)](#)