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Sudden Sensorineural Hearing Loss: Early Diagnosis and Evidence-Based Management

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Abstract

Sudden sensorineural hearing loss (SSNHL) is an otological emergency characterized by an abrupt reduction in hearing, usually affecting one ear and frequently accompanied by tinnitus, aural fullness, or vertigo. Early recognition and timely specialist assessment are essential because the diagnostic and therapeutic window may influence hearing recovery. Although several infectious, vascular, inflammatory, autoimmune, and neurological mechanisms have been proposed, the majority of cases remain idiopathic despite appropriate investigation.

SSNHL is conventionally defined as a sensorineural hearing loss of at least 30 dB across three consecutive audiometric frequencies developing within 72 hours. Diagnosis requires differentiation from conductive hearing loss, prompt audiometric confirmation, assessment for neurological or other modifying features, and appropriate investigation for retrocochlear pathology. Clinical examination, tuning-fork tests, pure-tone audiometry, speech audiometry, tympanometry, and magnetic resonance imaging have complementary roles in the diagnostic pathway.

Systemic corticosteroids may be offered as initial treatment within two weeks of symptom onset after consideration of contraindications and patient preferences. Intratympanic corticosteroids are recommended as salvage treatment for incomplete recovery, particularly within two to six weeks after onset. Hyperbaric oxygen therapy combined with corticosteroids may be considered in selected patients within specified treatment windows. Routine use of antivirals, vasodilators, thrombolytics, and indiscriminate laboratory testing is not supported by current guideline recommendations.

This narrative review discusses the epidemiology, proposed pathophysiology, clinical presentation, diagnostic evaluation, evidence-based treatment, prognosis, and rehabilitation of patients with SSNHL. It emphasizes the importance of urgent referral, shared decision-making, audiometric follow-up, and individualized care by the ENT specialist.

Keywords: Sudden sensorineural hearing loss, otological emergency, pure-tone audiometry, systemic corticosteroids, intratympanic steroids, hyperbaric oxygen therapy, hearing rehabilitation, otolaryngology.

1. Introduction

Sudden sensorineural hearing loss is a potentially disabling condition that requires prompt recognition and appropriate clinical management. Patients may describe waking up with reduced hearing, experiencing an abrupt decline during routine activities, or noticing sudden difficulty understanding speech on the telephone. Tinnitus, a sensation of ear blockage, and vertigo may accompany the hearing loss. Because these symptoms can resemble cerumen impaction, Eustachian tube dysfunction, or middle-ear disease, the condition may initially be misdiagnosed, delaying specialist evaluation.

SSNHL is conventionally defined as a sensorineural hearing loss of at least 30 decibels (dB) at three consecutive frequencies developing within 72 hours. This definition is widely used in clinical studies and guidelines, although patients with smaller but functionally significant sudden hearing changes may also require urgent assessment. The condition predominantly affects one ear, and an identifiable cause is found in only a minority of cases. The term *idiopathic sudden sensorineural hearing loss* is used when an appropriate clinical evaluation does not identify an underlying cause. [1,2]

The American Academy of Otolaryngology–Head and Neck Surgery Foundation (AAO-HNSF) updated its clinical practice guideline in 2019 to improve diagnostic accuracy, reduce unnecessary investigations, clarify treatment windows, and support appropriate follow-up. The guideline emphasizes distinguishing conductive from sensorineural hearing loss at initial presentation, obtaining audiometry promptly, evaluating for retrocochlear pathology, and discussing the benefits and limitations of available treatments. [1]

The pathophysiology of idiopathic SSNHL remains incompletely understood. Proposed mechanisms include viral or post-infectious injury, cochlear microvascular disturbances, immune-mediated inflammation, and cellular dysfunction within the cochlea. These hypotheses have informed treatment strategies, but no single mechanism adequately explains all cases.

Management is complicated by spontaneous recovery, variable severity, differences in treatment protocols, and uncertainty concerning the magnitude of benefit from some interventions. Nevertheless, prompt assessment is important because treatment options have defined time windows, particularly for corticosteroids, intratympanic salvage therapy, and hyperbaric oxygen therapy. The ENT specialist must balance urgency with careful diagnosis, assessment of contraindications, and informed patient choice. [1–3]

OBJECTIVES

The objectives of this review are:

1. To describe the clinical definition, epidemiology, and proposed pathophysiology of SSNHL.
2. To discuss clinical presentation and differential diagnosis.
3. To outline the roles of clinical examination, audiometry, and imaging.
4. To review evidence-based medical treatment and the timing of therapeutic interventions.
5. To discuss prognosis, follow-up, hearing rehabilitation, and patient counselling.

6. To identify limitations in the current evidence and future research priorities.

REVIEW METHODOLOGY

This article is a narrative review based on major clinical practice guidelines, peer-reviewed reviews, randomized controlled trials, and systematic reviews relevant to SSNHL. The principal reference framework is the AAO-HNSF *Clinical Practice Guideline: Sudden Hearing Loss (Update)*, supplemented by studies comparing systemic and intratympanic corticosteroids and literature addressing contemporary management. [1–5]

The literature was selected for clinical relevance to adult patients, particularly those with idiopathic SSNHL. This review did not use a prospectively registered protocol, exhaustive reproducible database search, formal risk-of-bias assessment, or quantitative synthesis. It should therefore be interpreted as a narrative synthesis rather than a systematic review or meta-analysis.

DEFINITION AND EPIDEMIOLOGY

SSNHL refers to an abrupt reduction in auditory function caused by dysfunction of the cochlea, auditory nerve, or related sensory pathways. The commonly used audiometric definition requires a reduction of at least 30 dB across three consecutive frequencies within 72 hours. However, the clinical presentation and degree of hearing impairment vary considerably.

The 2019 AAO-HNSF guideline reports an estimated incidence of approximately 5–27 cases per 100,000 people annually, with substantial uncertainty related to population characteristics, case definitions, and ascertainment. [1] SSNHL can occur at different ages, although it is most frequently encountered in adults. It is usually unilateral, while bilateral or recurrent episodes are less typical and warrant particular attention.

The consequences extend beyond auditory thresholds. Patients may experience communication difficulties, social isolation, anxiety, impaired work performance, and persistent tinnitus. Severe or profound loss may substantially affect quality of life, particularly when the contralateral ear also has impaired hearing.

PATHOPHYSIOLOGY

The pathogenesis of idiopathic SSNHL remains uncertain. Several mechanisms have been proposed, and more than one may contribute in individual patients.

1. Viral and post-infectious mechanisms

Viral infection or a post-infectious inflammatory response has been proposed as a cause of cochlear injury. However, an association with a recent respiratory illness does not establish a viral cause in an individual patient. Evidence has not supported routine antiviral treatment for otherwise typical idiopathic SSNHL. [1,2]

2. Vascular mechanisms

The cochlea has a high metabolic requirement and depends on a delicate microvascular supply. Disturbance of cochlear perfusion has therefore been proposed as a mechanism of sudden hearing loss. Nevertheless, a vascular mechanism cannot generally be confirmed from symptoms alone, and routine treatment with vasodilators, thrombolytics, or vasoactive agents is not recommended for idiopathic SSNHL. [1]

3. Inflammatory and immune-mediated mechanisms

Inflammation, immune dysregulation, and autoimmune inner-ear disease may contribute to auditory dysfunction in selected patients. Bilateral, fluctuating, recurrent, or otherwise atypical presentations may prompt further investigation for systemic or immune-mediated disease.

4. Cellular and cochlear injury

Oxidative stress, cellular metabolic dysfunction, and damage to sensory hair cells or auditory neural structures have been investigated as potential mechanisms. These processes provide a biological rationale for several proposed treatments, but mechanistic plausibility alone does not establish clinical effectiveness.

In many patients, the underlying mechanism remains unidentified. Consequently, management of idiopathic SSNHL is based primarily on clinical evidence, the urgency of presentation, and an individualized assessment of potential benefits and risks.

CLINICAL PRESENTATION

The typical presentation is sudden unilateral hearing reduction developing over minutes to hours, or noticed on waking. Some patients describe distortion of speech rather than complete deafness.

Common symptoms

1. **Sudden hearing impairment:** The principal symptom, ranging from mild to profound loss.
2. **Tinnitus:** May be continuous or intermittent and can persist even when hearing improves.
3. **Aural fullness:** Often described as pressure, blockage, or a sensation of fluid in the affected ear.
4. **Vertigo or imbalance:** May indicate associated vestibular involvement and can increase functional disability.
5. **Communication difficulty:** Patients may struggle to localize sound, understand speech in noise, or use the telephone.

The absence of pain or visible ear abnormalities does not exclude SSNHL. Likewise, an apparent sensation of ear blockage should not automatically be attributed to wax or Eustachian tube dysfunction.

The history should establish the precise onset, progression, affected ear, previous episodes, tinnitus, vestibular symptoms, recent infections, trauma, noise exposure, medication exposure, and relevant neurological symptoms. The clinician should also assess previous hearing impairment, vascular risk factors, autoimmune disease, and any symptoms suggesting a systemic disorder.

Bilateral sudden hearing loss, recurrent episodes, severe vertigo with neurological symptoms, facial weakness, diplopia, ataxia, or other focal neurological findings require urgent assessment for alternative or additional diagnoses. Suspected stroke or another acute neurological emergency requires immediate emergency evaluation.

DIAGNOSTIC EVALUATION

1. Otoscopy and clinical examination

Otoscopy is essential to identify potentially reversible conductive causes, including impacted cerumen, external-ear disease, tympanic membrane perforation, and middle-ear effusion. The ENT specialist should examine the tympanic membrane and middle ear and perform an appropriate cranial nerve and neurological examination.

Normal otoscopy in a patient with abrupt hearing loss should increase suspicion of a sensorineural cause, although it does not establish the diagnosis by itself.

2. Tuning-fork tests

The 512-Hz tuning fork is commonly used for bedside assessment when audiometry is not immediately available.

- **Weber test:** Sound lateralization toward the better ear may support sensorineural loss in the opposite ear, while lateralization toward the affected ear may suggest conductive loss.
- **Rinne test:** Air conduction greater than bone conduction is generally consistent with normal hearing or sensorineural loss; bone conduction equal to or greater than air conduction may suggest conductive loss.

These tests are useful for initial triage but have limitations, particularly in mild, bilateral, or mixed hearing loss. They must not delay formal audiometry or specialist referral.

3. Pure-tone audiometry

Pure-tone audiometry is the key investigation for confirming and characterizing SSNHL. It measures air- and bone-conduction thresholds across frequencies, establishes the degree and configuration of hearing loss, and helps distinguish sensorineural from conductive pathology.

The AAO-HNSF guideline recommends obtaining audiometry as soon as possible and within 14 days of symptom onset. This is an outer guideline time frame, not a reason to defer testing when an earlier assessment is available. [1]

Speech audiometry provides additional information about speech recognition and may help explain functional communication difficulties. Tympanometry can assist in evaluating middle-ear function when indicated.

4. Magnetic resonance imaging

Patients with confirmed SSNHL should be evaluated for retrocochlear pathology, commonly using magnetic resonance imaging (MRI) of the internal auditory canals and cerebellopontine angles with an appropriate protocol. MRI can help identify vestibular schwannoma and other structural abnormalities that may not be evident on routine clinical examination.

Auditory brainstem response testing may be considered when MRI is contraindicated or unavailable, although it is less sensitive for some small retrocochlear lesions. Imaging should not unnecessarily delay time-sensitive treatment decisions. [1]

Routine computed tomography of the head is not recommended in the initial evaluation of otherwise typical SSNHL. Targeted temporal-bone CT may be appropriate when a specific bony or traumatic pathology is suspected.

5. Laboratory investigations

Routine broad laboratory screening is not recommended for typical idiopathic SSNHL because indiscriminate testing has low diagnostic yield. Investigations should be guided by clinical findings and suspected underlying conditions.

Targeted testing may be appropriate when the history or examination suggests autoimmune disease, infection, metabolic disease, or another systemic cause. Bilateral or recurrent hearing loss, systemic symptoms, and atypical clinical findings may justify a more focused investigation.

6. Differential diagnosis

Important differential diagnoses include:

- Conductive hearing loss due to cerumen, otitis media, or middle-ear effusion.
- Ménière disease, particularly when hearing loss is accompanied by episodic vertigo and fluctuating auditory symptoms.
- Vestibular schwannoma and other retrocochlear lesions.
- Ototoxic medication-related auditory injury.
- Acoustic or head trauma.
- Autoimmune inner-ear disease and selected infectious disorders.

- Posterior circulation ischaemia or other neurological conditions when associated signs are present.

The purpose of the diagnostic pathway is not merely to confirm hearing loss but also to identify conditions requiring specific treatment or urgent intervention.

EVIDENCE-BASED MANAGEMENT

The management of SSNHL requires urgent assessment, informed decision-making, and appropriate treatment within clinically relevant time windows. The choice of treatment should account for the severity of hearing loss, time since onset, comorbidities, contraindications, patient preference, and the limitations of available evidence.

1. Initial management and referral

Any patient with suspected sudden sensorineural hearing loss should receive urgent clinical assessment and prompt referral to an ENT specialist or an appropriate emergency pathway. Conductive causes should be excluded without delaying audiometric confirmation.

The initial assessment should document the onset and severity of hearing loss, associated symptoms, relevant medical history, and neurological findings. Treatment options should be discussed promptly because the potential benefit of corticosteroid treatment is time-dependent.

Patients should be informed that hearing recovery varies and that spontaneous improvement may occur. No available intervention guarantees complete restoration of hearing.

2. Systemic corticosteroids

Systemic corticosteroids are widely used as initial treatment for idiopathic SSNHL. Their proposed benefit relates to anti-inflammatory effects and modulation of immune responses within the inner ear. However, evidence regarding the magnitude of benefit is limited by spontaneous recovery, differences between studies, and variation in treatment protocols.

The AAO-HNSF guideline states that clinicians *may offer* corticosteroids as initial treatment within two weeks of symptom onset. This is an option rather than a strong recommendation, reflecting uncertainty in the evidence. [1]

Oral prednisone or prednisolone is commonly used in clinical practice, with the regimen individualized according to local protocols, patient factors, and specialist judgment. Treatment decisions should consider diabetes mellitus, hypertension, peptic ulcer disease, glaucoma, active infection, psychiatric vulnerability, and other relevant contraindications or precautions.

Patients receiving systemic corticosteroids should be counselled regarding potential adverse effects, including hyperglycaemia, sleep disturbance, mood changes, gastrointestinal symptoms, and blood

pressure elevation. Appropriate monitoring is particularly important in patients with pre-existing comorbidities.

Corticosteroid treatment should be considered promptly when appropriate, but clinicians should avoid presenting it as a guaranteed cure or delaying referral while attempting prolonged empirical treatment.

3. Intratympanic corticosteroids

Intratympanic corticosteroid therapy involves administration of a corticosteroid, commonly dexamethasone or methylprednisolone, into the middle ear through the tympanic membrane. The drug reaches the inner ear through diffusion across the round-window membrane, allowing targeted treatment while reducing systemic exposure.

Intratympanic therapy has two principal roles:

- As an alternative or individualized initial approach when systemic corticosteroids are unsuitable or their risks are substantial.
- As salvage treatment when hearing recovery following initial management is incomplete.

The AAO-HNSF guideline recommends offering or referring patients for intratympanic steroid therapy when recovery is incomplete two to six weeks after symptom onset. [1]

A randomized non-inferiority trial by Rauch and colleagues compared oral prednisone with intratympanic methylprednisolone in patients presenting with unilateral SSNHL and found intratympanic treatment non-inferior to oral treatment under the trial's prespecified criteria. The findings support intratympanic therapy as a reasonable treatment option in appropriately selected patients, although they do not establish that every route or protocol is equivalent in all clinical settings. [3]

Potential adverse effects include transient pain, dizziness, otitis media, and persistent tympanic membrane perforation. Patients should understand the procedural nature of the treatment, the possibility of incomplete recovery, and the need for follow-up audiometry.

4. Hyperbaric oxygen therapy

Hyperbaric oxygen therapy (HBOT) involves breathing oxygen in a pressurized chamber to increase oxygen availability in body tissues. It has been investigated as an adjunctive treatment for SSNHL based on the hypothesis that improved oxygen delivery may support cochlear function.

The AAO-HNSF guideline states that clinicians may offer HBOT combined with corticosteroids within two weeks of onset as initial treatment, or as salvage treatment within one month of onset. [1]

HBOT requires access to an appropriately equipped facility and assessment of treatment suitability. Potential disadvantages include cost, repeated chamber sessions, middle-ear barotrauma, claustrophobia, and, rarely, oxygen-related complications.

The evidence suggests potential benefit in selected circumstances, but uncertainty remains regarding the magnitude of improvement, ideal patient selection, and the most effective treatment protocol. HBOT should therefore be discussed as an adjunct rather than a universal requirement.

5. Treatments not recommended for routine use

The AAO-HNSF guideline recommends against routinely prescribing antivirals, thrombolytics, vasodilators, or vasoactive substances for idiopathic SSNHL. Routine laboratory testing and indiscriminate head CT are also discouraged. [1]

These recommendations are important because a biologically plausible mechanism does not necessarily translate into demonstrated clinical benefit. Treatment should prioritize interventions supported by appropriate clinical evidence and should avoid unnecessary cost, adverse effects, and diagnostic delay.

Antioxidants and other proposed adjunctive treatments have also been investigated, but evidence is insufficient to justify their routine use as standard treatment for all patients.

PROGNOSIS AND FACTORS AFFECTING HEARING RECOVERY

The prognosis of SSNHL is variable. Some patients experience substantial spontaneous or treatment-associated improvement, while others have persistent hearing impairment despite timely management.

Prognosis may be influenced by the initial severity of hearing loss, audiometric configuration, time to presentation, age, associated vertigo, and other clinical characteristics. However, these factors should be interpreted as prognostic indicators rather than definitive predictors for an individual patient.

Profound hearing loss and associated vestibular symptoms may be associated with less favorable outcomes in some clinical series. Delayed presentation may reduce opportunities to use time-sensitive treatments, although late presentation should still prompt appropriate specialist assessment and discussion of relevant options.

The clinician should avoid guaranteeing recovery or interpreting early partial improvement as proof that no further follow-up is required. Repeat audiometry is necessary to determine the final degree of recovery and guide rehabilitation.

FOLLOW-UP AND HEARING REHABILITATION

1. Audiometric monitoring

Audiometric assessment should be repeated at the conclusion of treatment and again within six months, as recommended by the AAO-HNSF guideline. [1] Earlier reassessment may be appropriate when symptoms change or when results would influence further treatment decisions.

Follow-up should document pure-tone thresholds, speech recognition where appropriate, tinnitus, vestibular symptoms, and the patient's functional hearing needs. Residual loss should be explained clearly, including its implications for communication and safety.

2. Management of persistent tinnitus

Tinnitus may persist even when hearing improves. Patients should be asked about its impact on sleep, concentration, mood, and daily functioning. Supportive measures may include tinnitus education, hearing rehabilitation, sound-based approaches, and psychological interventions where indicated.

Persistent distress warrants appropriate assessment and referral rather than reassurance alone.

3. Hearing aids and assistive technology

Patients with residual hearing loss should receive counselling regarding audiological rehabilitation. Depending on the degree and configuration of hearing loss, options may include conventional hearing aids, assistive listening devices, remote microphone systems, and communication strategies.

For selected patients with persistent severe-to-profound unilateral sensorineural hearing loss, assessment for a bone-conduction device or cochlear implantation may be appropriate. Candidacy depends on the clinical situation, audiological findings, local criteria, and specialist assessment.

Rehabilitation is an essential component of care and should not be withheld merely because the initial treatment did not restore normal hearing.

4. Patient counselling

Patients should be informed about the natural history of SSNHL, uncertainty surrounding treatment effectiveness, potential adverse effects, and the importance of follow-up. Written information and clear communication may help reduce anxiety and improve adherence.

Counselling should also address communication difficulties at work and home, strategies for listening in noisy environments, and practical safety considerations when hearing is reduced on one side.

DISCUSSION

Sudden sensorineural hearing loss remains a challenging condition in otolaryngology because it combines diagnostic urgency with uncertainty regarding its underlying mechanisms and the effectiveness of individual treatments. The most important clinical priority is early recognition. Patients frequently present with nonspecific complaints of ear blockage or muffled hearing, making it essential to distinguish conductive from sensorineural loss at the first clinical encounter. [1,2]

A structured diagnostic pathway improves clinical decision-making. Otoscopy and tuning-fork testing provide rapid bedside information, while formal audiometry establishes the degree and type of hearing loss. MRI or an appropriate alternative investigation is important for evaluating retrocochlear pathology.

Routine laboratory panels and non-targeted CT scanning do not replace this pathway and may lead to unnecessary testing. [1]

Corticosteroids remain central to contemporary treatment, although the evidence should be communicated accurately. The guideline supports offering initial corticosteroids within two weeks as an option, rather than implying universal benefit. This distinction is important when counselling patients about spontaneous recovery, potential adverse effects, and the possibility of incomplete hearing restoration.

Intratympanic corticosteroids are particularly relevant as salvage treatment. Their localized administration offers an alternative for patients who cannot tolerate systemic treatment and provides an evidence-supported option when initial recovery is incomplete. The randomized trial by Rauch and colleagues supports intratympanic treatment as a viable alternative to oral prednisone in the studied population, while differences in protocols and clinical circumstances should be considered when applying its findings. [3]

HBOT may provide an additional option for selected patients, but accessibility, treatment burden, and uncertainty about optimal patient selection limit universal application. The evidence-based approach is to discuss its potential role within the recommended time window, taking into account local availability and individual circumstances. [1,4]

An additional challenge is the management of persistent symptoms. Hearing loss and tinnitus may continue despite appropriate treatment, with significant effects on communication, mental well-being, and occupational functioning. Audiometric follow-up and timely rehabilitation are therefore integral to management, not merely secondary considerations. [1]

Future research should focus on identifying patients most likely to benefit from specific treatments, clarifying optimal dosing and timing, standardizing outcome measures, and comparing interventions through adequately powered randomized trials. Research into biomarkers and cochlear pathophysiology may eventually support more individualized treatment, but current clinical practice should remain grounded in established guidelines and transparent communication about uncertainty.

LIMITATIONS OF THE REVIEW

This article is a narrative review and does not represent a systematic review or meta-analysis. The literature was selected for clinical relevance, without a prospectively registered protocol, exhaustive reproducible database search, formal independent screening, or structured risk-of-bias assessment.

The available evidence is heterogeneous in terms of patient selection, baseline hearing loss, treatment timing, corticosteroid regimens, outcome definitions, and follow-up duration. Spontaneous recovery complicates the interpretation of treatment effects, while variation in clinical practice may limit the generalizability of individual trials.

The review focuses primarily on adult idiopathic SSNHL and does not provide a comprehensive evaluation of pediatric cases, all identifiable secondary causes, or every emerging therapeutic intervention.

Recommendations should be interpreted alongside current local guidance and individual clinical circumstances.

CONCLUSION

Sudden sensorineural hearing loss is a time-sensitive otological condition in which prompt recognition, accurate diagnosis, and informed treatment decisions are essential. ENT specialists should prioritize the exclusion of conductive causes, obtain audiometric confirmation as early as possible, assess for modifying clinical features, and arrange appropriate evaluation for retrocochlear pathology.

Systemic corticosteroids may be offered as initial treatment within two weeks of onset, while intratympanic corticosteroids are an important salvage option for incomplete recovery within the recommended two-to-six-week interval. Hyperbaric oxygen therapy combined with corticosteroids may be considered in selected patients within appropriate treatment windows. Routine use of unsupported pharmacological agents and indiscriminate investigations should be avoided.

Follow-up audiometry, patient counselling, and hearing rehabilitation are essential components of comprehensive care. Although current treatment cannot guarantee complete recovery, a timely, evidence-based, and individualized approach offers patients the best opportunity to make informed decisions and address the functional consequences of hearing loss.

DECLARATION BY AUTHORS

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