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REVIEW ARTICLE

Idiopathic sudden sensorineural hearing loss: current insights into etiology, diagnosis, and management

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Summary

Idiopathic sudden sensorineural hearing loss (ISSNHL) is a distinct otologic emergency characterized by an abrupt onset of unilateral sensorineural hearing loss, typically occurring within 72 hours and lacking any identifiable cause. Although its pathogenesis remains incompletely understood, several mechanisms have been proposed, including cochlear microvascular insufficiency, viral or autoimmune-mediated inner ear inflammation, intracochlear membrane rupture, and stress-induced cellular responses.

The diagnostic algorithm requires prompt audiological assessment, exclusion of alternative etiologies, and, where indicated, neuroimaging, particularly utilizing advanced modalities such as three-dimensional fluid-attenuated inversion recovery (3D-FLAIR) magnetic resonance imaging (MRI). Systemic corticosteroids remain the mainstay of treatment, while intratympanic steroid injections and hyperbaric oxygen therapy are increasingly employed as adjunctive or salvage therapies. Treatment outcomes vary considerably and are influenced by multiple prognostic factors, including patient age, severity of hearing loss, presence of vertigo, timing of therapeutic intervention, and underlying vascular or metabolic comorbidities.

This review synthesizes current evidence on the etiopathogenesis, clinical presentation, diagnostic approach, and management strategies for ISSNHL, drawing on recent systematic reviews, clinical guidelines, and original research. Special attention is given to pediatric and bilateral cases, as well as to emerging diagnostic and therapeutic modalities. A more refined understanding of the heterogeneous nature of ISSNHL is essential for guiding individualized clinical decision-making and optimizing patient outcomes.

Keywords: idiopathic sudden sensorineural hearing loss, sensorineural hearing loss, corticosteroid, hyperbaric oxygen therapy, 3D-FLAIR MRI, prognosis

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INTRODUCTION

Idiopathic sudden sensorineural hearing loss (ISSNHL) is defined as a rapid-onset sensorineural hearing loss of at least 30 dB across three contiguous frequencies occurring within 72 hours, without an identifiable cause. It represents a unique diagnostic and therapeutic challenge, given its abrupt nature and multifactorial pathogenesis (1). Although ISSNHL has been recognized for several decades, its precise etiology remains unresolved, and management strategies continue to evolve.

The annual incidence is estimated at 5 to 20 per 100,000 individuals, although this may be an underestimation due to undiagnosed or spontaneously recovering cases (2). The condition typically affects one ear, most commonly in middle-aged individuals, though pediatric and elderly populations are not exempt. The challenge lies in the lack of causal therapy for ISSNHL, with corticosteroids, which are broadly used in various medical conditions, remaining the treatment of choice. However, their efficacy is restricted in specific patient populations, and a considerable body of literature critically examines and analyzes the effectiveness of this treatment (3). When hearing recovery does not occur, individuals may experience long-term consequences, including impaired communication, diminished quality of life, and negative impacts on mental health. It is essential to refer such patients for auditory rehabilitation, whether through the use of hearing aids or cochlear implants, to improve their auditory function and overall quality of life.

In recent years, there has been renewed interest in the pathophysiological mechanisms of ISSNHL, as well as in the optimization of diagnostic protocols and therapeutic interventions. This narrative review synthesizes current understanding and emerging insights into ISSNHL, with a focus on etiology, clinical presentation, diagnostic work-up, treatment modalities, and prognosis.

METHODS

This narrative review was conducted in accordance with the journal's guidelines for narrative reviews. A comprehensive literature search was performed in the PubMed and Google© Scholar databases—the search period covered from the earliest available publications to June 2025. The keywords used included: idiopathic sudden sensorineural hearing loss, ISSNHL, sudden deafness, pediatric ISSNHL, bilateral ISSNHL, treatment, diagnosis, prognosis, as well as their equivalents in Serbian. Inclusion criteria comprised peer-reviewed articles, clinical guidelines, systematic reviews, and book chapters in English and Serbian focusing on the pathophysiology, clinical presentation, diagnostic methods, treatment modalities, and prognosis of ISSNHL. Exclusion criteria included conference abstracts without full text and studies not relevant to the scope of this review. References were identified through database searches and by manually reviewing the bibliographies of included articles to ensure completeness.

PATHOPHYSIOLOGY AND ETIOLOGIC HYPOTHESES

Despite extensive research, the pathogenesis of ISSNHL remains elusive. As the term idiopathic implies, no definitive cause can be identified in the majority of cases. Nonetheless, several hypotheses have been proposed to explain the abrupt onset of hearing loss, including vascular insufficiency, viral infection, autoimmune dysfunction, intracochlear membrane rupture, and stress-induced cellular response mechanisms.

Vascular Hypothesis

Vascular compromise of the cochlea is one of the oldest and most widely accepted theories. The cochlea is supplied by a single terminal artery - typically the labyrinthine artery - which lacks collateral circulation, rendering it highly susceptible to ischemic injury. Microvascular occlusion, vasospasm, or thrombotic events may lead to acute hypoperfusion and cochlear infarction (4). These mechanisms align with findings of increased cardiovascular risk burden and cerebral small vessel disease in ISSNHL patients, as demonstrated in a recent systematic review and meta-analysis (5).

Recent neuroimaging studies have provided further support for this vascular hypothesis. A significantly higher prevalence of white matter lesions (WMLs) has been reported in patients with ISSNHL compared to age-matched healthy controls. These lesions, which are typically associated with cerebral small vessel disease, may serve as imaging biomarkers of systemic microangiopathy involving both cerebral and cochlear circulations. The inner ear's end-arterial configuration and high metabolic demand further predispose it to ischemic damage from even subtle reductions in perfusion. Moreover, an inverse correlation between WML burden and hearing recovery has been observed, suggesting that microvascular pathology may influence both the onset and prognosis of ISSNHL (6-9).

Viral and Post-Viral Inflammation

Viral infections, particularly those involving neurotropic viruses such as herpes simplex virus (HSV) and varicella-zoster virus (VZV), have long been implicated in cochlear and retrocochlear inflammation. Reactivation of latent viruses within the spiral ganglion or cochlear nerve may lead to neuritis, resulting in sudden sensorineural hearing loss (SSNHL). The seasonal variation of ISSNHL incidence further supports a viral etiology (10).

Recent studies have also linked SARS-CoV-2 to sudden hearing loss, potentially through viral invasion of the cochlea or inflammation of vascular structures. SARS-CoV-2 may induce vasculitis, impairing cochlear blood flow and leading to ischemic damage. Some studies have reported hearing loss following SARS-CoV-2 infection, suggesting that virus-induced damage or immune responses may contribute to SSNHL (11-14). Moreover, the role of SARS-CoV-2 vaccination in triggering hearing loss has been explored, with some studies indicating potential autoimmune mechanisms (11). SARS-CoV-2 infection has been linked to bilateral SSNHL, highlighting severe hearing impairment, likely due to virus-induced cochlear damage or immune-mediated vascular effects (15-17)

Autoimmune hypothesis

Autoimmune mechanisms are considered an important factor in the pathogenesis of SSNHL. In some cases, cross-reactivity between viral antigens and inner ear structures may trigger immune responses that lead to cochlear inflammation and subsequent hearing loss. This hypothesis is supported by studies demonstrating the presence of circulating autoantibodies - particularly against inner ear antigens - in a subset of patients with SSNHL (3, 18). Moreover, immune-mediated attack may exacerbate cochlear damage through pro-inflammatory cytokines and direct cytotoxicity (19).

Intracochlear Membrane Rupture

In his seminal 1968 paper, Simmons proposed the membrane rupture theory as a potential mechanism underlying SSNHL (20). He hypothesized that spontaneous ruptures of the cochlear membranes - particularly Reissner's and the basilar membranes - could lead to the mixing of endolymph and perilymph, resulting in acute cochlear dysfunction and sudden hearing loss. This theory was based on clinical observations and histopathological findings, suggesting that mechanical disruptions within the inner ear could precipitate ISSNHL in the absence of identifiable vascular or infectious causes.

Stress Response Theory

A novel etiologic model, the stress response theory, has been proposed as an alternative to traditional vascular or infectious hypotheses. Merchant et al. (21) conducted a histopathological analysis of 17 temporal bones from patients with ISSNHL, which revealed no consistent evidence supporting commonly proposed etiologies such as vascular occlusion, membrane rupture, or viral infection. Instead, their findings suggest that cellular stress-response mechanisms, particularly involving nuclear factor kappa B (NF-κB) activation, may play a key role in the pathophysiology of ISSNHL.

This hypothesis is further elaborated by Masuda and Kanzaki (22), who proposed the "stress response theory" as a potential explanatory model. According to this theory, various systemic stressors- such as psychological or physical stress, infections, and inflammation- can activate the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system. This leads to the release of catecholamines and pro-inflammatory cytokines, which may activate NF-κB in the cochlear lateral wall, resulting in local inflammation, increased vascular permeability, and dysfunction of cochlear homeostatic mechanisms.

CLINICAL PRESENTATION AND DIAGNOSTIC ALGORITHM

ISSNHL is classically defined as a rapid-onset hearing loss of at least 30 dB across three consecutive frequencies within 72 hours, occurring without an identifiable cause (1,23). Clinically, ISSNHL most commonly presents unilaterally, accompanied by tinnitus, aural fullness, and frequently vertigo or imbalance (10,24). While tinnitus is reported in up to 80–90% of cases, vestibular symptoms are present in approximately one-third of patients and are generally considered a negative prognostic factor (24, 25). In the study by Čvorović 2021 et al. (26), it was found that tinnitus triggered by ISSNHL was more frequent in patients with better contralateral hearing and of a younger age, irrespective of the severity of hearing loss on the affected side or the asymmetry between the ears.

The initial diagnostic work-up should include otomicroscopic examination to rule out external and middle ear disease, followed by a complete audiological assessment (23). Pure-tone audiometry is essential not only for confirming the presence, degree, and configuration of sensorineural hearing loss in ISSNHL, but also for identifying specific audiometric patterns - such as ascending, descending, flat, or profound curves - that may carry prognostic significance (25); in addition, speech audiometry is particularly valuable in patients with usable residual hearing to assess speech discrimination capacity and monitor functional outcomes during follow-up, while tympanometry assists in ruling out middle ear pathology as a confounding factor (27,28). In addition, the auditory brainstem response (ABR) may be helpful in differentiating cochlear from retrocochlear pathology, particularly when magnetic resonance imaging (MRI) is unavailable. ABR testing is particularly relevant in the diagnostic work-up of ISSNHL, as it enables the detection of retrocochlear pathology by identifying abnormalities in neural conduction time, which may indicate the presence of vestibular schwannoma or demyelinating and ischemic lesions at the brainstem level (7,8).

Neuroimaging plays a critical role in excluding structural lesions of the cerebellopontine angle and inner ear. Gadolinium-enhanced MRI, particularly with three-dimensional fluid-attenuated inversion recovery (3D-FLAIR) sequences, is considered the modality of choice for visualizing inner ear pathology. Hyperintense signals observed on delayed FLAIR imaging - especially in the basal turn of the cochlea or vestibular apparatus - have been associated with inflammatory or vascular insults and may carry prognostic value (8,9,29).

Advanced 3D-FLAIR MRI is particularly effective in detecting subtle endolymphatic signal abnormalities that are not visible on conventional sequences. This technique has demonstrated clinical utility in identifying cochlear and vestibular enhancement in patients with suspected labyrinthine inflammation or ischemia. However, its sensitivity is time-dependent, with optimal detection typically achieved 4 to 24 hours after gadolinium administration.

The presence of nonspecific white matter lesions - commonly observed in older individuals or those with vascular comorbidities - may confound image interpretation and should be considered in the differential diagnosis (8,9).

In addition to its diagnostic value in detecting vascular and inflammatory injury, 3D-FLAIR MRI contributes to the differential diagnosis of various labyrinthopathies. For example, diffuse enhancement of vestibular structures may suggest viral labyrinthitis, whereas focal enhancement limited to the cochlea is more consistent with an ischemic etiology. In autoimmune inner ear disease, bilateral and symmetric enhancement is typically observed. This imaging modality also assists in distinguishing the acute phase of disease—characterized by active inflammation and contrast enhancement—from the chronic phase, where atrophic changes may predominate in the absence of significant enhancement. Nonetheless, several limitations must be acknowledged, including variability in interpretation, the need for standardized timing of imaging, and the fact that the absence of enhancement does not exclude underlying functional impairment.

For patients with vertigo or imbalance, vestibular assessment is warranted. A comprehensive battery, including video head impulse test (vHIT), caloric testing, and vestibular evoked myogenic potentials (VEMP), can reveal patterns of labyrinthine dysfunction that may correlate with hearing loss severity and outcome (24,28).

Laboratory testing is not universally indicated (23) but may be performed in selected cases to evaluate potential autoimmune, infectious, or thrombotic etiologies. Specific markers such as antinuclear antibodies, syphilis and Lyme serology, and thrombophilia panels may be considered based on clinical suspicion (28,30).

The diagnostic approach to ISSNHL is multifaceted, requiring prompt audiologic confirmation, targeted neuroimaging, and- in selected cases- vestibular and laboratory investigations. Early identification of prognostically relevant findings, such as vestibular involvement or cochlear enhancement on MRI, can inform both therapeutic decisions and patient counseling (3,31).

Table 1 summarizes the diagnostic modalities available and utilized in the evaluation of ISSNHL cases.

NATURAL HISTORY AND PROGNOSTIC FACTORS

Spontaneous Recovery Rates

ISSNHL exhibits a highly variable natural course, with spontaneous recovery reported in a significant proportion of cases. Rates of spontaneous improvement have been estimated between 32% and 65%, with the majority of recovery typically occurring within the first two weeks after symptom onset (29, 32-34). However, the extent and completeness of recovery vary widely. Complete recovery is observed in only a subset of patients, while others may experience partial or no improvement. In a landmark prospective, double-blind, placebo-controlled study, Wilson et al. reported that 65% of patients in the

Table 1. Diagnostic methods in Idiopathic sudden sensorineural hearing loss

Diagnostic Method	Purpose / Indication	Notes
Otomicroscopic examination	Exclude external and middle ear pathology	Standard initial examination
Pure-tone audiometry	Confirm SNHL and determine audiogram configuration	Key diagnostic tool
Speech audiometry	Assess speech discrimination and follow-up	Useful for monitoring rehabilitation
Tympanometry	Exclude conductive hearing loss	Helps in differential diagnosis
Auditory Brainstem Response	Differentiate cochlear vs retrocochlear pathology (when MRI is unavailable)	Useful when MRI is not available or contraindicated
3D-FLAIR MRI	Visualize inflammatory or ischemic changes	Optimal 4–24h after gadolinium injection
Standard MRI (T1/T2 sequences)	Rule out cerebellopontine angle tumors	Should be contrast-enhanced to detect tumors
Vestibular testing (vHIT, VEMP, calorics)	Assess vestibular function and prognosis	Vertigo is a negative prognostic factor
Laboratory analysis	Evaluate autoimmune, infectious, or vascular causes	Not routine; based on clinical suspicion

SNHL- sensorineural hearing loss; MRI- magnetic resonance imaging; 3D-FLAIR- three-dimensional fluid-attenuated inversion recovery, vHIT- video head impulse test; VEMP- vestibular evoked myogenic potentials

placebo group exhibited some degree of spontaneous recovery, with 32% achieving complete recovery within one month (35). While spontaneous recovery can occur in a subset of patients, current clinical guidelines discourage a wait-and-see approach. Instead, prompt initiation of treatment - most commonly with corticosteroids - is universally recommended for all patients, with the option for corticosteroid intervention emphasized within two weeks of symptom onset (23,36).

Prognostic Factors in ISSNHL

Numerous clinical and audiometric parameters have been identified as predictors of hearing recovery in ISSNHL. The most consistently reported prognostic indicators include the initial severity of hearing loss, with milder losses generally associated with more favorable outcomes (25,37). Audiogram configuration also plays a significant role; patients presenting with low-frequency or upward-sloping audiograms typically exhibit better recovery compared to those with flat or profound hearing loss patterns (25). Another well-established factor is the timing of treatment initiation- early intervention, ideally within seven days of symptom onset, is strongly correlated with improved auditory outcomes regardless of the therapeutic approach used (25,36,38,39). Additionally, the presence of vertigo at presentation is considered a negative prognostic factor, potentially reflecting more extensive inner ear or vestibular system involvement (25).

Beyond these clinical indicators, emerging evidence suggests that certain systemic comorbidities and cerebral microvascular changes may adversely influence hearing recovery in ISSNHL. Metabolic syndrome, diabetes mellitus, and hypertension have been associated with poorer outcomes, likely due to their detrimental effects on cochlear microcirculation (40,41). Supporting this vascular hypothesis, recent neuroimaging studies have identified a significant association between ISSNHL and cerebral WMLs, which are considered radiological markers of cerebral small vessel disease. The presence, distribution, and extent of WMLs- particularly in periventricular and subcortical regions- correlate with worse auditory outcomes, suggesting that ISSNHL in some patients may reflect a systemic microvascular pathology affecting both cerebral and cochlear microcirculation (6-9).

MANAGEMENT AND TREATMENT MODALITIES

Corticosteroid Therapy

Systemic Corticosteroids

Systemic corticosteroids are the mainstay of treatment for ISSNHL. Prednisone and methylprednisolone are most commonly used, though dexamethasone may also be employed in specific protocols. According to current guidelines, including those from the American (23), Spanish (27), and Japanese (31) societies, early initiation of systemic corticosteroids - preferably within 72 hours and no later than 14 days after symptom onset - is associated with better outcomes (2,23,27,31,42).

While the Spanish consensus guideline recommends systemic corticosteroids as standard therapy, and the Japanese guideline supports high-dose regimens - such as intravenous methylprednisolone 500–1000 mg/day for three consecutive days in severe cases (31) - the 2019 American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS) guideline presents systemic corticosteroid therapy only as an option, rather than a strong recommendation, thereby allowing individualized decision-making (23). The typical oral regimen includes prednisone at 1 mg/kg/day, up to 60 mg daily, methylprednisolone at 48 mg/day, or dexamethasone at 10 mg/day, administered for 7 to 14 days, followed by tapering over 5 to 7 days, depending on clinical response and patient tolerance (23,27).

Regular monitoring is recommended during corticosteroid therapy, including audiometric evaluation at the end of treatment and at delayed intervals, as well as clinical surveillance for potential adverse effects such as hyperglycemia, hypertension, and neuropsychiatric symptoms (23).

Intratympanic Corticosteroids

Intratympanic (IT) corticosteroids are used either as primary treatment in patients with contraindications to systemic therapy or as salvage therapy in those who fail to achieve satisfactory recovery with systemic corticosteroids. The most commonly used agent is dexamethasone, in concentrations ranging from 10 mg/mL (stock solution) to 24 mg/mL (compounded). Typical administration includes injecting 0.4 to 0.8 mL into the middle ear, up to four times over 2 weeks (23,31,43). Methylprednisolone, at 30–40 mg/mL, is also used with comparable protocols (31).

In contrast to systemic corticosteroids, which are presented as an option for initial treatment, the 2019 AAO-HNS guideline formally recommends intratympanic steroid therapy as salvage treatment for patients with incomplete recovery between 2 and 6 weeks after symptom onset (23). Spanish and Japanese guidelines also support IT therapy, particularly in non-responders, although protocols vary in terms of concentration, frequency, and timing (27,31).

Meta-analyses suggest that combined systemic and intratympanic steroid therapy may provide better hearing outcomes than systemic therapy alone, especially in salvage situations (43,44). Nevertheless, variability in treatment response and limitations in evidence quality have led some authors to advocate for a more individualized approach (45).

While corticosteroids - administered either systemically or intratympanically - remain the most commonly used first-line therapy for ISSNHL due to their anti-inflammatory and immunosuppressive actions, current guidelines stress the importance of timely intervention, appropriate case selection, and, when necessary, referral to centers capable of providing intratympanic or adjunctive therapies such as hyperbaric oxygen therapy (23).

Hyperbaric Oxygen Therapy

Hyperbaric oxygen therapy (HBOT) is frequently used as an adjunctive treatment in ISSNHL, particularly in the early stages of the disease or in patients who fail to improve with corticosteroids alone. The typical HBOT protocol includes administration of 100% oxygen at 2.0 to 2.5 atmospheres absolute (ATA), for 60 to 90 minutes per session, once daily, with a total of 10 to 20 sessions, depending on response and patient tolerance (46-48).

HBOT is most effective when initiated within 2 weeks of symptom onset, preferably as early as possible. Multiple studies and guidelines emphasize that the therapeutic benefit of HBOT diminishes significantly after 14 days from the onset of hearing loss (46,47). However, some authors suggest that a modest benefit may still be observed when HBOT is initiated within the first 30 days, particularly in combination with intratympanic corticosteroid therapy, though evidence beyond the two-week window is less robust. Outcomes are generally less favorable (48,49).

HBOT is gaining traction as an adjuvant modality aimed at improving cochlear oxygenation. Meta-analyses demonstrate that HBOT, when combined with corticosteroids, may yield superior outcomes compared to medical therapy alone (46-48). Nevertheless, access and protocols remain heterogeneous across institutions and countries (23,27,31).

Other Therapies

Other pharmacologic approaches - such as vasodilators, antioxidants, antiviral agents, and anticoagulants - have been explored but lack consistent evidence of efficacy. A systematic review found no conclusive benefit of vasodilator therapy compared to placebo (50).

Antioxidant vitamins, when used as adjuvant therapy, may offer a modest benefit in improving hearing recovery in patients with ISSNHL, particularly when administered early in the treatment course (51). Nevertheless, heterogeneity in antioxidant types, dosages, and treatment durations limits broader recommendations.

A Cochrane review found no compelling evidence that antivirals provide significant benefit in the treatment of ISSNHL compared to placebo (52).

Although anticoagulants have been considered due to the potential role of vascular occlusion in the pathophysiology of ISSNHL, existing studies do not provide robust evidence to support their routine use. The absence of conclusive benefit and the associated risk of bleeding complications further limit their clinical application (53).

A systematic review and meta-analysis of non-steroid pharmacologic treatments highlighted methodological inconsistencies and a lack of standardization in study protocols. These reviews failed to demonstrate any consistent benefit of such therapies over placebo (53,54).

Table 2 presents the therapeutic options used in ISSNHL management.

SPECIAL POPULATION AND EMERGING TOPICS

Pediatric Cases

ISSNHL in children is a rare condition, with epidemiological studies estimating an annual incidence of approximately 20-30 cases per 100.000 children, which is about 10-20 times lower than in adults (55). Pediatric patients may exhibit fewer classical symptoms than adults, such as tinnitus or vertigo, which can delay diagnosis and intervention. In some cases, hearing loss may be first suspected because of behavioral changes, a decline in school performance, or parental observation of communication difficulties. Despite this, several studies suggest that children tend to have better hearing recovery outcomes than adults, particularly when treated promptly.

A systematic review and meta-analysis by Franz et al. reported favorable outcomes in pediatric patients, especially with early administration of corticosteroids. However, heterogeneity in diagnostic criteria and small sample sizes were noted as limiting factors (56).

Table 2. Therapeutic options in Idiopathic sudden sensorineural hearing loss

Therapeutic option	Timing	Comment / Recommendation
Systemic corticosteroids	Ideally, within the first 72h, up to 14 days	Standard therapy; recommended in all guidelines
Intratympanic corticosteroids	Primary option if systemic therapy is contraindicated; used as salvage therapy 2–6 weeks after onset	Recommended as adjunct or alternative therapy
Hyperbaric oxygen therapy	Most effective within 14 days; modest benefit up to 30 days	Adjunctive therapy with potential benefit
Antioxidants	Early in the course of the disease	Possible minor benefit as an add-on therapy
Vasodilators	Unproven efficacy	No evidence of benefit over placebo
Antiviral drugs	No proven benefit	Cochrane review: no proven benefit
Anticoagulants	Insufficient data, bleeding risk	No evidence of benefit; potentially harmful

Further supporting this, a pooled analysis by Reading et al. emphasized that early steroid therapy remains the cornerstone of treatment and is associated with higher recovery rates. MRI was recommended to exclude retrocochlear pathology in unclear cases. The study also pointed out the need for standardized protocols to guide clinical decision-making in pediatric ISSNHL (57).

More recently, Barron et al. reviewed bilateral sudden sensorineural hearing loss in children and identified that autoimmune, infectious, and genetic causes should be considered in the differential diagnosis. Their work underscores the importance of comprehensive etiological evaluation, particularly in bilateral or recurrent cases, and stresses the need for multidisciplinary management (58).

In both unilateral and bilateral presentations, prompt recognition, early corticosteroid therapy, and thorough diagnostic work-up - including MR imaging and serologic testing - remain the foundation of care in pediatric ISSNHL. Rehabilitation strategies should be developmentally appropriate, with early fitting of hearing aids or cochlear implantation when indicated, combined with auditory-verbal therapy to support language acquisition.

Bilateral Cases

The prognosis of bilateral ISSNHL is generally poorer than that of unilateral cases, with lower rates of complete recovery and a higher likelihood of persistent functional impairment (59,60). Prompt recognition and initiation of treatment are crucial; high-dose systemic corticosteroids remain the cornerstone of therapy, often combined with intratympanic steroid injections and, in selected cases, hyperbaric oxygen therapy (23,59).

In such cases, clinicians should actively investigate underlying conditions, including autoimmune disorders (e.g., systemic lupus erythematosus, Cogan's syndrome), infectious diseases (e.g., syphilis, Lyme disease, HIV), vascular etiologies (e.g., vasculitis, antiphospholipid syndrome), and neoplastic or paraneoplastic syndromes (23). In pediatric bilateral ISSNHL, which is exceedingly rare, autoimmune, infectious, and genetic etiologies are proportionally more common; multidisciplinary management is recommended (57,58).

Long-term auditory rehabilitation planning is essential, as bilateral involvement can significantly impair speech perception, spatial hearing, and quality of life. Early fitting of hearing aids or consideration of cochlear implantation in cases of profound, irreversible loss should be integrated into the treatment plan.

LONG-TERM OUTCOMES

Role of Hearing Aids and Cochlear Implantation

When conservative treatment fails to achieve satisfactory hearing recovery, auditory rehabilitation with hearing aids and, if necessary, cochlear implantation becomes essential. Hearing aids are effective for patients with residual moderate to severe unilateral hearing loss, improving speech perception, spatial awareness, and overall quality of life. Cochlear implantation, however, is typically reserved for patients with profound, irreversible bilateral hearing loss (15). While rarely required for ISSNHL, cochlear implants may be indicated in cases of bilateral involvement or persistent severe functional impairment despite conventional rehabilitation.

Psychosocial Impact

ISSNHL can have a significant psychosocial impact, especially in patients with permanent hearing loss. Increased levels of anxiety, depression, social withdrawal, and reduced quality of life are commonly reported (61). Early psychological support combined with auditory rehabilitation- including the use of hearing aids or cochlear implants- plays a crucial role in improving long-term outcomes and patient well-being.

CONCLUSION

ISSNHL remains a heterogeneous clinical entity with multiple proposed etiologies and no single causal therapy. Early recognition and prompt initiation of treatment, primarily with corticosteroids, are critical to optimizing outcomes. Adjunctive options such as intratympanic steroid injections and hyperbaric oxygen therapy may provide additional benefit in selected patients, particularly when used early or in salvage settings.

Pediatric and bilateral presentations require special attention due to their diagnostic complexity and prognostic implications. In children, subtle or atypical symptoms can delay recognition, while bilateral cases often reflect underlying systemic disease and carry a poorer prognosis.

Advanced magnetic resonance imaging, particularly three-dimensional fluid-attenuated inversion recovery (3D-FLAIR) sequences, enhances detection of subtle cochlear and vestibular pathology, facilitates prognostication, and guides therapeutic decisions.

Clinical practice should integrate early referral for hearing rehabilitation, including hearing aids or cochlear implantation when indicated, to mitigate long-term communicative and psychosocial consequences.

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IDIOPATSKA IZNENADNA SENZORINEURALNA NAGLUVOST: SAVREMENI UVIDI U ETIOLOGIJU, DIJAGNOZU I LEČENJE

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Sažetak

Idiopatska iznenadna senzorineuralna nagluvost (AISNN) predstavlja urgentno stanje u otologiji, koje se karakteriše naglim nastankom jednostrane senzorineuralne nagluvosti u roku od 72 sata, bez jasno utvrđenog uzroka. Iako etiopatogeneza ostaje delimično razjašnjena, predloženi mehanizmi uključuju mikrovaskularnu insuficijenciju kohlee, virusno ili autoimuno posredovano zapaljenje unutrašnjeg uha, rupturu intrakohlearnih membrana i ćelijske odgovore na stres.

Dijagnostički postupak podrazumeva hitnu audiološku procenu, isključenje mogućih uzroka i, po potrebi, neuroimidžing – naročito uz primenu naprednih tehnika kao što je 3D-FLAIR magnetna rezonanca. Sistemski kortikosteroidi predstavljaju standardnu terapiju, dok se intratimpanične aplikacije i hiperbarična oksigenacija sve češće koriste kao dopunske mere, posebno u slučajevima koji ne odgovaraju na inicijalno lečenje. Ishod lečenja je izrazito varijabilan i zavisi od više prognostičkih faktora, uključujući starost pacijenta, stepen gubitka sluha, prisustvo vrtoglavice, vreme započinjanja terapije i postojanje vaskularnih ili metaboličkih komorbiditeta.

Ovaj pregledni rad sintetizuje aktuelne dokaze o etiopatogenezi, kliničkoj prezentaciji, dijagnostičkom pristupu i terapijskim strategijama u lečenju AISNN, oslanjajući se na sistematske preglede, kliničke vodiče i originalna istraživanja. Posebna pažnja posvećena je pedijatrijskim i bilateralnim slučajevima, kao i novim dijagnostičkim i terapijskim modalitetima. Dublje razumevanje etiološke i kliničke heterogenosti ovog poremećaja ključno je za uvođenje individualizovanog pristupa i poboljšanje terapijskih ishoda.

Ključne reči: idiopatska iznenadna nagluvost, senzorineuralni gubitak sluha, kortikosteroidi, hiperbarična oksigenacija, 3D-FLAIR MRI, prognoza

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