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Review Article

Sudden sensorineural hearing loss treatment options: a short literature review

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ABSTRACT

Sudden sensorineural hearing loss (SSNHL) remains a complex, urgent and challenging condition in otology, often presenting without warning and with limited time for intervention. Time is of the essence. Prompt diagnosis and immediate initiation of treatment are crucial to optimizing hearing recovery and minimizing long-term auditory deficits. Patients must be adequately counselled on the emergency nature of SSNHL and the prognosis, which depends on the underlying etiology and early management. Available literature reinforces the critical role of timely therapeutic strategies in preserving auditory function, preventing permanent disability and enhancing the overall quality of life.

Keywords: Sudden sensorineural hearing loss, Corticosteroid therapy, Intratympanic steroid injection, Hyperbaric oxygen therapy, Hearing recovery

INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) is defined as sensorineural hearing loss of ≥30 dB affecting ≥3 consecutive audiometric frequencies within a 3-day time frame.[1-5] It is an emergency requiring prompt identification and management.[1] If not treated in time, persistence of the SNHL can affect quality of life.[6] Annually, the incidence is from 5-20/100,0003 however the incidence increases with age, from 1.2/100,000 for ages less than 9 years to 77/100,000 for ages more than 65 years.[1] It is most common between the age groups of 30-50 years.[7] Bilateral SSNHL (BiSSNHL) though rare is a worrisome entity with a reported proportion of 8.09% in all cases of SSNHL.[8] BiSSNHL is associated with more systemic diseases.[8] Various causes of SSNHL are reported but majority of cases are idiopathic and management is largely empirical aimed at mitigating the cochlear injury and inflammation rather than targeting a specific etiology.[2,3] The modified Siegel criteria are used to quantify final hearing outcomes.[9,10] According to these criteria, complete recovery is defined as a final pure tone average (PTA) threshold better than 25 dB. Partial recovery refers to a hearing gain of 15 dB with a final PTA threshold between 25 and 45 dB. Slight improvement is considered when there is a gain of 15 dB and the final PTA threshold ranges between 46 and 75 dB.

No improvement is defined as either a hearing gain of less than 15 dB or a final PTA threshold between 76 and 95 dB. A non-serviceable ear is identified when the final PTA threshold is worse than 95 dB.

METHODS

This article was designed as a narrative literature review focusing on evidence related to the etiology, evaluation and management of SSNHL. Emphasis was given on treatment modalities and prognostic factors. A comprehensive literature search was conducted using PubMed/MEDLINE, Scopus and Google Scholar databases. The search strategy included the following

keywords: sudden sensorineural hearing loss, idiopathic SSNHL, bilateral sudden hearing loss, corticosteroids, intratympanic steroid injection, hyperbaric oxygen therapy, and prognosis in SSNHL.

Priority was given to recent peer-reviewed articles published between 2017 and 2025, including randomized controlled trials, cohort studies, systematic reviews, meta-analyses, clinical practice guidelines and large observational studies. Older publications were only included where relevant definitions, classification systems or criteria were mentioned.

Articles were selected based on relevance to clinical practice, clarity of methodology and applicability to adult and paediatric SSNHL cases. Case reports and abstracts without full texts were excluded. Data were extracted quantitatively and synthesized narratively. No formal statistical pooling or meta-analysis was undertaken. The narrative approach was chosen to provide a clinically oriented, up-to-date overview of SSNHL management, integrating emerging evidence with established treatment options to support informed decision making in routine practice.

DISCUSSION

Etiology and symptomatology

SSNHL has a standard definition, but the aetiology of the disease is varied and difficult to pin-point in most cases. Most cases are thus invariably labelled as idiopathic due to absence of identifiable causes and broad-spectrum treatment must be undertaken to prevent further damage.[2,3,7]

According to Aldè et al, otological (39.3%), infectious (21.4%), autoimmune (7.1%), vascular (5.4%) and neoplastic 1.8% factors were identified as major contributors to SSNHL.[1] They further inferred that Meniere's disease, isolated cochlear endolymphatic hydrops, Herpes Simplex 1 virus and Epstein-Barr virus as the most common causes overall.

The rate of recovery is low in patients having co-morbidities.[7] Patients with a high CHADS2 or Framingham risk score are at higher risk of SSNHL and severe hearing loss.[6] Hyperglycaemia due to diabetes mellitus and dyslipidaemia have an impact on microcirculation and this entails a poorer prognosis in patients.[7]

The labyrinthine artery, with its three branches, is the sole vascular supply to the inner ear.[6] Vestibulo-cochlear artery (VCA) syndrome typically presents with high-frequency hearing loss and vertigo, often resulting from the artery's course through a narrow bony canal.[6,11]

BiSSNHL is another condition and these patients are likely suffering from co-morbidities or autoimmune diseases.[2-8] In a study by Celik, 42% of patients had a history of smoking.[3] He et al reported that 62% of their cases of BiSSNHL were idiopathic.[8] The most common identifiable causes were vascular conditions. They report 11% paediatric patients and head trauma followed by large vestibular aqueduct syndrome were the most common causes in these paediatric patients.[8] The prognosis is worse in bilateral SSNHL with poor recovery.[2-8] There are two variants; Simultaneous and Sequential. Simultaneous BiSSNHL is when the hearing loss in both the ears occurs within 72 hours while the Sequential variant is when symptoms occur after 72 hours in the other ear.[2-8]

SSNHL is most commonly unilateral.[1] Patients often report sudden hearing loss within 72 hours, with or without associated symptoms such as tinnitus, aural fullness and hyperacusis. Tinnitus is the most frequently observed symptoms.[2-8] Dizziness is seen in 30% of patients and is associated with poor prognosis.[7] Acute low-tone SNHL (ALHL) is described as having hearing loss in only the low frequencies and is more common in females of 30-40 years of age.[5] Symptoms include ear fullness and tinnitus with hearing loss and may further progress to Meniere's disease in some cases.[5]

Management

Aldè et al propose a comprehensive diagnostic protocol, incorporating audiological tests, high-resolution 3D-FLAIR delayed magnetic resonance imaging (MRI), cone beam computed tomography (CBCT), and screening for coagulation, infectious, and autoimmune diseases which they report leads to aetiology identification in 75% of cases, thus facilitating personalized management.[1]

Pure tone audiometry is initially done and shows the characteristic SNHL of >30dB in 3 consecutive audiometric frequencies. In a cohort study involving 200 ears, He et al studied observed mild sensorineural hearing loss in 6.5%, moderate loss in 18%, severe loss in 28.5% and profound loss in 47% ears.[8] Tympanometry, stapedial reflex, along with a battery of vestibular tests, are further recommended.[1] It is reported that hearing improvement was better in low frequencies than at high frequencies.[3]

Blood tests and thrombophilia screening includes a complete blood count, prothrombin time, activated partial thromboplastin time, protein C, protein S, homocysteine, folic acid and antibodies to beta-2 glycoprotein-1 and cardiolipin.[1] It is suggested that Neutrophil-Lymphocyte ratio (NLR) and Platelet-Lymphocyte ratio (PLR) could be useful biomarkers to detect Idiopathic SSNHL.[6,12] An infectious cause can be determined by screening for anti-bodies to Herpes Simplex 1 and 2, Cytomegalovirus, Epstein-Barr virus, Borrelia, Severe acute respiratory syndrome coronavirus-2 (SARS-CoV2).[1]

MRI plays a crucial role and is recommended. An "MRI abnormality directly related to SSNHL" is given by

AAO-HNS 2019, which mentions presence of “labyrinthine hemorrhage" and "cochlear inflammation".[5] MRI showing labyrinthine hemorrhages, seen in patients with coagulation defects or viral labyrinthitis, or white matter lesions, seen in those with cardio-vascular risk or in the elderly, are associated with poorer prognosis.[5,6]

Sun et al studied inner-ear 3D-FLAIR MRI in patients of SSNHL.[13] They state that 16.07% of these patients had auditory nerve enhancement on MRI and these patients had lower cure rates than those without enhancement. These patients also had a more severe hearing loss and aggravated vestibular function injury.

Prompt initiation of treatment is essential and should not be delayed while awaiting diagnostic results. Age of hearing loss, severity, frequency of impact, simultaneous vertigo and the time lag till medical treatment is started are the major influencing factors for recovery.[7] This underscores the importance of immediate start to the treatment. At the same time, it is reported that spontaneous recovery is seen in around 32-65% cases; most resolving within two weeks after onset however, waiting for spontaneous recovery is not recommended.[14]

Corticosteroids remain the cornerstone of therapy due to their role in reducing cochlear inflammation and edema.[3] Prednisolone, dexamethasone and methylprednisolone are the most commonly used.[7] The recommended dosage is oral prednisolone at 1mg/kg/day in a single daily dose for 10-14 days with tapering after 1 week.[3,14] Intravenously, methylprednisolone is given at a dose of 0.8mg/kg/day or dexamethasone 10mg/day for 7-14 days with tapering is administered.[7] (The dosing regimens vary across studies and institutional protocols; the doses mentioned reflect commonly reported therapeutic ranges) Systemic corticosteroids along with intra tympanic steroid injections (ITSI) injections provide additional therapeutic benefit.[5]

ITSI causes steroid diffusion into the inner ear through the round window membrane.[7] ITSI is further recommended as initial option of treatment in those with contraindications to systemic steroids.[5] 0.4-0.8 ml of Dexamethasone (4-10 mg/ml) or Methylprednisolone (30-40 mg/ml) can be injected in 4-6 sessions over a two week period.[7] Adverse effects of ITSI include transient dizziness, infections, persistent tympanic membrane perforation and the possibility of injuring the middle ear structures.[6,7] In the paediatric age group, ITSI is done as a salvage therapy after systemic steroids.[7] Cheng et al treated patients with concurrent intravenous dexamethasone 10 mg/day and intratympanic dexamethasone 5 mg/day for 5 days, which was then tapered off with oral methylprednisolone.[8] They report that the combination therapy led to better results. They add that treatment within 14 days of symptoms led to better hearing improvement.

Hyperbaric oxygen therapy (HBOT), introduced in 1979, increases oxygen in the blood, in turn increasing oxygen in the cochlea, causing the reduced oxygen pressure in the perilymph in SSNHL patients to increase by around 450%.[3] HBOT is suggested as an option, in combination to steroid therapy, and to be used in the acute phase (two weeks since symptom onset).[5] There is positive evidence for the use of HBOT6 and combination therapy has shown better outcomes than monotherapy.[7]

Aldè et al treated patients with oral corticosteroids and hyperbaric oxygen treatment (8-24 sessions at 2.5 atm) while Celik and Akil treated patients with intravenous steroids and hyperbaric oxygen (20 sessions at 2.5atm).[1,3] Combined therapy resulted in better treatment outcomes.[3]

Vasoactive treatments are also suggested to alleviate any vascular condition of the inner ear.[5,6] Prostaglandin E1 (PGE1), an arachidonic acid metabolite with vasodilatory effects, may be used in cases of cochlear ischaemia as it improves inner ear circulation.[5,6] Intravenous dose of 20-60 µg/day as a continuous infusion can be given with corticosteroids as a combination therapy as monotherapy with PGE1 has failed to show a benefit in the literature.[6,7] The combination therapy was reported to be more effective in patients with female, age ≥65 years old, latency to begin treatment <4 days, presence of vertigo and female gender.[15]

Additional treatment modalities include Carbogen (a mixture of 95% O2 and 5% CO2) inhalation, oral nifedipine. Antioxidants like a combination of Vitamin E (600mg/day) and Vitamin C (1,200 mg/day) or just high dose Vitamin C can be supplemented. Other supplements include Zinc, Magnesium, N-acetyl-cysteine and Ginkgo biloba.[7]

He et al treated patients with intravenous or intratympanic dexamethasone for 3-5 days or 5 alternate day sessions respectively along with HBOT, vitamin B12 and ginkgo biloba supplementation and found favorable outcomes.[8]

Bilateral SSNHL had an effective rate of 32% with few patients having progressive hearing loss even during treatment.[8] There was no difference in efficacy between injected steroids and systemic steroids in cases with BiSSNHL.[2] With literature reporting the higher chances of BiSSNHL in those with systemic conditions, we recommend a control of these comorbidities as a preventive measure for SNHL.[2,8] Kim et al suggest that treatment results in Idiopathic SSNHL are significantly poorer for patients of chronic kidney disease on dialysis.[4] Similar results have been found in patients with other comorbidities. While the majority of patients experience significant recovery, rehabilitation strategies are vital for those with persistent hearing deficits. Hearing aids or cochlear implants are suggested if the hearing recovery after treatment is not adequate.[1] These can be selected as

per the patient's preference, their occupation as well as level of hearing thresholds after therapy.

CONCLUSION

Sudden sensorineural hearing loss (SSNHL) is a complex otologic emergency that necessitates prompt diagnosis and the immediate initiation of treatment to maximise hearing recovery. While the majority of cases remain idiopathic, current literature suggests that a combination of systemic and intratympanic corticosteroids, often supplemented with hyperbaric oxygen therapy, gives most benefit these patients. Furthermore, the proactive management of underlying comorbidities plays a critical role in preventing progressive auditory deficits and improving overall prognosis. Ultimately, early intervention and personalised therapeutic protocols are essential for best clinical outcomes.

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