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Sudden Sensorineural Hearing Loss: Current Evidence of Etiology, Management, and Prognosis

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Review Article

Sudden Sensorineural Hearing Loss: Current Evidence of Etiology, Management, and Prognosis

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Abstract

Sudden sensorineural hearing loss (SSNHL) is an otologic emergency because if not recognized and promptly treated, it can result in permanent hearing loss, tinnitus, and reduced quality of life. Although 70% of SSNHL cases are idiopathic, it is potentially linked to vascular impairment, especially migraines. Currently, there is a standard guideline from the American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS) for SSNHL. However, the effectiveness of treatment is still uncertain. Moreover, there are still knowledge gaps in many aspects of SSNHL, such as the lack of a standardized measurement for hearing improvement and the lack of a definite timeline for salvage therapy. In this review, the current evidence of etiology, management, and prognostic factors of SSNHL is elaborated.

Keywords: review, sudden sensorineural hearing, sensorineural hearing

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Introduction

Sudden sensorineural hearing loss (SSNHL) is defined as a rapid onset of hearing loss of at least 30 decibels in three consecutive frequencies within 72 hours. This disorder affects approximately 5 to 27 per 100,000 people annually worldwide[1]. Studies in Europe and the United States have reported that SSNHL typically occurs at ages 40 to 50 years[2], while studies in Japan have reported onset at 60 to 70 years[3]. SSNHL is considered an otologic emergency because, if not recognized and promptly treated, it can result in persistent hearing loss, tinnitus, and reduced quality of life[1].

Although SSNHL is considered idiopathic in 70% of cases[4], recent studies have demonstrated that vascular impairment, especially in the cochlea, is linked to the idiopathic etiology of SSNHL[5,6]. Moreover, there is a rising trend of studies indicating that migraines, a vascular disease, might be linked with SSNHL in terms of incidence, symptoms, and histopathology[7-10].

The current treatment of SSNHL is based on the standard guideline of the American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS)[1]. Options for treatment are systemic steroids (within 2 weeks as a primary treatment), intratympanic steroids, and hyperbaric oxygen therapy (as adjunct or salvage treatment). However, the effectiveness of the treatment is still contradictory and uncertain, as some studies have shown improved hearing, while others show no difference in hearing compared to a placebo. The evidence is still low due to methodological limitations, such as small sample sizes. Moreover, the standardized criteria for initiating salvage treatment or assessing treatment outcomes are still missing.

The prognostic factors for hearing recovery in SSNHL, according to the standard guideline of the American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS), are patient age, presence of vertigo at onset, degree of hearing loss, audiometric configuration, and time between onset of hearing loss and treatment. Data regarding other factors, such as accompanying symptoms (tinnitus), underlying disease (metabolic syndromes), and investigation (inflammatory markers), are not mentioned in the standard guideline.

As mentioned above, there are still gaps in the knowledge and evidence of sudden sensorineural hearing loss (SSNHL). Our study aimed to summarize the current evidence and update the etiology, treatment, and prognosis of SSNHL. We also wanted to highlight potential

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areas for researchers to investigate, thereby helping to close the evidence gap. Finally, we wanted to provide data to clinicians in order to assist them when making evidence-based clinical decisions.

Etiology and diagnosis

SSNHL is considered idiopathic for 70%[5]of etiologies; only 30% can be identified: vascular impairment (AICA stroke, atherosclerosis), infection (herpes simplex, varicella zoster, CMV, HIV, Lyme disease)[11,12], autoimmune disease (Cogan syndrome, SLE, and Wegener's granulomatosis)[13-15], and inner ear or central nervous system pathology. Etiologies must be identified and addressed because these etiologies require specific management. However, routine (nontargeted) laboratory testing is not recommended, as it may lead to the unnecessary evaluation of false-positive results and increased financial costs.

The standard guideline of the American Academy of Otolaryngology–Head and Neck Surgery (AAO-HNS) recommends clinicians look for clinical features, such as bilateral or fluctuating SNHL and focal neurologic deficit, because these features are likely to be non-idiopathic. Bilateral or fluctuating sensorineural hearing loss should raise suspicion for infection and autoimmune disease, while focal neurologic deficit indicates central nervous system involvement.

MRI is recommended for the workup of retrocochlear pathology in the setting of SSNHL. The recent study demonstrated that 4.5% of patients with SSNHL have an abnormality on MRI[16] (The standard guideline mentions 2.7–10.2%)[1]. The most common abnormality was vestibular schwannoma[16]. This MRI abnormality is considered as causal of SSNHL rather than an incidental finding. The possible mechanism for SSNHL in these cases was related to the mechanical compression of the cochlear nerve, microvascular compromise in the cochlear, endolymphatic hydrops, and biochemical changes of the inner-ear fluid[17]. Even though the most common symptom of vestibular schwannoma is hearing loss, only 7–20% have sudden onset[18]. Another option to detect retrocochlear pathology is the auditory brainstem response (ABR), which can be offered to patients who do not wish to undergo an MRI. However, ABR may miss up to 42% of small (subcentimeter) or intracanaliculi tumors[19]. Routine CT of the head/neck or brain (thickness 5 mm) is definitely not recommended in the setting of SSNHL because of its low resolution, risk of exposure to radiation or IV contrast, and high cost. But it can be useful to detect possible intracranial hemorrhage or acute stroke in suspected cases.

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There are 4 possible mechanisms that could explain the idiopathic etiology of SSNHL: inner ear vasospasm, inner ear membrane rupture, vascular dysfunction, and vascular embolization or thrombosis[4-6]. The labyrinthine artery is the sole terminal artery supplying blood to the inner ear, with 3 tiny branches of the anterior vestibular artery, the main cochlear artery, and the vestibulocochlear artery[20]. The main cochlear artery supplies the apical three-fourths of the cochlear (primarily at the apical and middle turn of the cochlear), while the vestibulocochlear artery supplies one-fourth of the cochlear (basal turn of the cochlear) and the posterior semicircular canal[20]. From this, we can see that the occlusion of the vestibulocochlear artery can lead to high-frequency SSNHL associated with basal turn of cochlear dysfunction, as well as vertigo associated with posterior semicircular canal dysfunction. Recent studies demonstrated that vascular impairment is linked with the idiopathic etiology of SSNHL[2,5,6]. Moreover, there are 3 pieces of evidence that migraines (vascular disease) might be linked with SSNHL. Firstly, there is a higher incidence of migraines in patients with SSNHL. Regarding a systematic review and meta-analysis conducted on three observational studies[7], a migraine is associated with a 1.84-fold increased risk of developing SSNHL. However, the increase may be due to the vascular etiopathogenesis (vasospasm) shared between the two conditions[8]. Secondly, migraine aura (disturbance in the visual, olfactory, or auditory sense) can present as low-frequency hearing loss periodically and sometimes persistently, mimicking the symptoms of SSNHL[9]. Lastly, there is histopathological support that migraine-associated vasospasm might result in SSNHL. There is a postmortem examination of the cochlea of a man with a long-standing migraine who developed SSNHL at age 50, showing prominent fibrosis in the stria vascularis and spiral ligament[10].

Management

Clinicians should be able to make the diagnosis of SSNHL within 2 weeks from symptom onset in order to offer systemic corticosteroid as a treatment[1]. The proposed mechanism for steroids is that they can modify the inflammatory cell-death cascade in SSNHL, as demonstrated in the laboratory, according to the standard guideline[1]. In animal studies, steroids can mediate immune suppression, erythrocyte proliferation, anti-inflammation, and mediate Na+ and K+ transport to restore hearing[21]. The dose for oral prednisolone is 1 mg/kg/day in a single dose with a maximum dose of 60 mg daily. The equivalent dose of prednisolone (60 mg) is 48 mg for methylprednisolone and 10 mg for dexamethasone. The treatment duration is 10 to 14 days. Patients with certain

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medical conditions, such as poorly controlled diabetes, peptic ulcer, labile hypertension, glaucoma, and tuberculosis, will receive intratympanic steroids instead of systemic steroids as their initial treatment. Research demonstrates that intratympanic steroids are not inferior to systemic steroids in terms of efficacy in the initial treatment[22,23]. The dose for an intratympanic steroid is methylprednisolone 30-40 mg/mL or dexamethasone 10-24 mg/mL injected 0.4-0.8 mL into the middle ear space for 15-30 minutes with a protocol of 4 injections over 2 weeks. If a patient presented late (more than 2 weeks from the onset of symptoms), studies have shown that systemic steroids provide little benefit after 4 to 6 weeks from the onset of symptoms[1].

However, the effectiveness of systemic steroid treatment is still unclear due to potential adverse effects from steroids and contradictory treatment outcomes. A systematic review and meta-analysis in 2013 showed that only 1 out of 3 RCTs had improved hearing outcomes in the steroid group, while others showed no difference in hearing outcomes compared to the placebo group. Moreover, the sample size in this review is relatively small (a total of 267 patients)[24]. There is still no research that directly compares the efficacy of oral steroids to intravenous steroids. The role of intravenous steroids in SSNHL is very limited, as their efficacy has been demonstrated in only 2 retrospective cohorts[25,26]. Even with the addition of the intravenous (IV) form of steroids, hearing outcome still shows no difference compared to oral steroids alone, as demonstrated by the RCT in 2016[27]. According to Cochrane's review in 2022, intratympanic steroid therapy in primary therapy results in little to no difference in hearing but increases the risk of complications from injection, compared to systemic steroids. For the combined treatment of systemic and intratympanic steroids, there is a small effect on change in hearing when compared to systemic steroids alone, but the evidence is very uncertain[28]. Thus, shared decision-making with the patient about the use of systemic steroids is needed. Further research about the effectiveness is also needed.

In case the patient has an incomplete recovery from SSNHL 2-6 weeks after onset, the clinician should offer intratympanic steroid therapy to the patient as a salvage therapy[1]. Intratympanic steroids diffuse into the inner ear through the round window membrane. Intratympanic injections raise higher concentrations locally at the inner ear, minimizing the systemic side effects of steroids[29]. From the current data, 6 of 7 RCTs[30-36] showed significant improvement in hearing outcome after intratympanic steroid therapy. Although there is still no

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consensus on a definite timeline for salvage therapy in the current guideline, 4 of the 7 RCTs initiated their salvage treatment within 7 days after completion of the initial treatment. The other 3 RCTs didn't mention the time between the initial and the salvage treatment. Also, there is still no consensus about the cutoff for hearing improvement. Most of the RCTs set their cutoff at a 10-decibel improvement in pure tone average (PTA). One of the popular cutoffs in sudden sensorineural hearing loss is Siegel's criteria, which is defined as complete recovery (final PTA less than 25 decibels), partial improvement (more than a 15-decibel improvement and final PTA between 25-45 decibels), slight improvement (more than a 15-decibel improvement and final PTA more than 45 decibels), and no recovery (less than 15 decibels and final PTA more than 75 decibels).

Hyperbaric oxygen therapy (HBOT) is also considered a potential option of treatment combined with steroid therapy. It can be used as an adjunct to the primary treatment within 2 weeks after onset, along with a systemic steroid, or as an adjunct to salvage treatment within 4 weeks after onset, along with an intratympanic steroid[1]. The recommended setting for hyperbaric oxygen therapy is 100% oxygen at a pressure level of 1.5 to 2 atmospheres absolute in a specially designed chamber. This enables the delivery of the increased partial pressure of oxygen to tissue, specifically at the cochlear, which is sensitive to ischemia[29]. Hyperbaric oxygen therapy is thought to have positive effects on immunity, oxygen support, and hemodynamics[37]. For this reason, hyperbaric oxygen therapy might solve ischemia, infection, and immunity issues, which are all possible etiologies of sudden sensorineural hearing loss, as mentioned above.

In the setting of adding hyperbaric oxygen therapy to conventional systemic steroids as a primary treatment, 5 of the 6 RCTs published between 2015 and 2024 showed significant improvement in hearing outcome[38-43]. The hearing outcome is even more favorable, especially in cases of severe to profound hearing loss[44]. In the setting of adding hyperbaric oxygen therapy to intratympanic steroids as a salvage treatment, only 1 of the 5 RCTs showed significant improvement in hearing outcome[45-49]. However, there is a lot of heterogeneity in the study design and a limited sample size in both settings. Further large-sample or multi-center studies are necessary to develop a standardized treatment protocol and determine the proper therapeutic timeline[50,51].

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Other pharmacologic therapies, such as antivirals and vasoactive agents, were strongly discouraged[1]. The most recent Cochrane review, in 2012, which included 4 RCTs, concluded that there is no evidence to support the use of antivirals in the treatment of sudden sensorineural hearing loss. The treatment protocol (dose and duration) is very different in each study, and the hearing outcome is not significant in all the studies[52]. There is also a Cochrane review from 2009 regarding the use of vasodilators and vasoactive agents in the treatment of sudden sensorineural hearing loss, which includes 3 RCTs[53]. It demonstrated that the effectiveness of treatment remains unproven, as the degree of heterogeneity is high. The methodology of RCT in both Cochrane reviews was relatively poor in quality, and the sample sizes were small. Other therapies, such as vitamins (high-dose vitamin C)[54-56], minerals (zinc)[57-59], supplements (N-acetyl cysteine)[60,61], or alternative medicine (Ginkgo biloba)[62,63], still had insufficient evidence to make a recommendation.

Counseling and rehabilitation are strongly recommended for patients with SSNHL who have residual hearing loss[1]. Unilateral SSNHL patients have handicapping effects that may impact function and quality of life. A retrospective study showed that the majority of unilateral SSNHL patients (86%) reported hearing handicap[64]. Thus, honest counseling about disease information and personal adjustment is beneficial and essential. A variety of amplification options are available for unilateral SSNHL, such as hearing aids, Osseo-integrated bone-conductive devices, and cochlear implants. A systematic review showed that amplification improves quality of life for SSNHL patients by reducing psychosocial and emotional manifestations[1]. Some patients may require rehabilitation, such as speech reading, auditory training, and support groups, to reduce any self-perceived hearing handicap[1].

Although no best modality is established, migraine medications have shed light on novel adjuvant treatment for SSNHL. There is a retrospective study about adjuvant prophylactic migraine medications on SSNHL patients (without a diagnosis of migraine or any previous use of migraine medications), showing that patients with adjuvant treatment had significantly greater improvements in PTA and hearing thresholds at lower frequencies. These patients also received a lower number of intratympanic steroid injections[65]. Another retrospective study with a similar methodology, including both with and without migraine diagnoses, also showed significant improvements in speech recognition threshold and concluded that SSNHL may be an otologic migraine phenomenon[66]. Regarding the mechanism of how migraine medications, particularly topiramate (a GABA-A receptor agonist, glutamate receptor antagonist, carbonic anhydrase

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inhibitor, voltage-gated sodium channel antagonist, and voltage-gated K+ channel modulator), can improve hearing levels, glutamate is a major neurotransmitter of inner hair cell ribbon synapses in the cochlea; therefore, it is the most important afferent neurotransmitter in the human auditory nervous system. One of the main etiologies of idiopathic SSNHL is vascular impairment to the cochlea[4-6]. This is because the blood supply to the cochlea emanates from 2 tiny terminal arteries, resulting in its susceptibility to ischemia through injury from a wide range of vascular issues, including migraine disease[67]. It has been shown that cochlear ischemia can cause a rise in the concentration of glutamate in the perilymph[68]. However, too much glutamate can also be toxic to inner hair cells, as shown in an animal study with gerbils, in which hearing loss developed after glutamate efflux in perilymph[68]. Furthermore, the animal study shows that a glutamate receptor antagonist can significantly diminish loss of inner hair cells in the cochlea of gerbils[69]. Adjuvant migraine medication or topiramate showed promising hearing results, but these studies are limited by their retrospective design[65,66]. Although the link between migraine and SSNHL is still emerging, it is not yet definite. This might guide us toward new treatments for SSNHL and further RCT studies.

Prognosis

According to the latest meta-analysis, the rate of spontaneous recovery was 30–65%[70,71]. However, this reported rate may be overestimated due to varying definitions of recovery (high heterogeneity of data), the potential for delayed or missed cases, and a lack of standardized measurement (Siegel's criteria). Therefore, early evaluation and appropriate treatment are essential for maximizing the chance of hearing recovery.

The current standard guideline states that the prognosis of this disease depends on several factors, such as age, presence of vertigo at onset, degree of hearing loss, audiometric configuration, and interval between onset and treatment[1]. A recent study showed that the recovery rate was statistically significantly worse in those over 70 years[72], while the cutoff is more than 60 years in most of the older literature[73-75]. Age under 15 years is also reported as a negative prognostic factor[73]. However, recent studies showed that children have a higher rate of recovery compared to adults[76-80]. Vertigo or abnormal caloric tests/video head impulse tests are known to be negative prognostic factors for recovery[5,81]. The precise relationship between vertigo and hearing loss is still an ongoing discussion[29]. Audiogram patterns with poor prognosis had profound hearing loss,

high-frequency hearing loss, and down-sloping or flat configuration[5,82,83]. Even though there is no clear-cut duration for early treatment, recent research showed that hearing improvement is significantly better in patients who received their first treatment within 2–4 weeks after the onset of sudden sensorineural hearing loss[84,85].

As mentioned above, sudden sensorineural hearing loss is related to vascular impairment. Inflammatory and metabolic biomarkers are potential factors linked with prognosis. The neutrophil-to-lymphocyte ratio (NLR) might become a useful biomarker to determine prognosis. However, there is a limitation of evidence in terms of cutoff, heterogeneity, and reporting bias due to retrospective studies[86-89]. Other inflammatory markers, such as platelet-to-lymphocyte ratio (PLR), tumor necrosis factor Alpha (TNF-α), and C-reactive protein (CRP), still require further research regarding their role in the prognosis of loss[89-92].

Vascular impairment is directly linked to metabolic syndrome (hypertension, diabetes mellitus, hyperlipidemia, and obesity). The risk for SSNHL was up to 4 times higher, and the prognosis was poorer in patients with metabolic syndrome[93-96]. But the association's clinical significance is unclear[1]. There is still insufficient evidence to demonstrate the relationship between SSNHL and each of the risk factors. For hyperglycemia, 2 retrospective studies demonstrate that hyperglycemia may be a negative prognostic factor for SSNHL[97,98]. For hyperlipidemia, there are 4 studies showing a potential relationship between total cholesterol and the prognosis of SSNHL[91-94]. Other parameters of hyperlipidemia, such as LDL, HDL, and triglycerides, did not demonstrate a relationship with the prognosis of SSNHL. LDL/HDL ratio might have a potential link to the prognosis of SSNHL, as demonstrated in 2 studies[99,100].

Conclusion

Seventy percent of SSNHL cases are idiopathic but potentially linked to vascular impairment. There is also evidence that migraines (vascular disease) might be linked with SSNHL. Etiologies must be identified because some require specific management. Bilateral or fluctuating sensorineural hearing loss should raise suspicion for infection and autoimmune disease, while focal neurologic deficit indicates central nervous system involvement. MRI is recommended for the workup of retrocochlear pathology in the setting of SSNHL, but routine CT of the head/neck and laboratory testing are strongly recommended against.

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Proposed options of treatment are systemic (oral) steroids within 2 weeks as an initial treatment and intratympanic (IT) steroids as a salvage treatment. Hyperbaric oxygen therapy (HBOT) is also considered a potential adjunct treatment combined with steroid therapy. The effectiveness of treatment with IT steroids is proven, but not for oral steroids or HBOT. Thus, shared decision-making with the patient is needed. Other pharmacologic therapies are strongly discouraged. Migraine medications have shed light on novel adjuvant treatment for SSNHL.

The proven prognostic factors are age, presence of vertigo at onset, degree of hearing loss, audiometric configuration, and interval between onset and treatment. The rate of spontaneous recovery is 30–65%. Potential prognostic factors are inflammatory biomarkers and metabolic syndrome. However, there is still insufficient evidence and knowledge gaps in many aspects of SSNHL. Thus, further research is needed.

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Conflict of interest

None.

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