REVIEW
J Audiol Otol 2024;28(1):10-17
pISSN 2384-1621 / eISSN 2384-1710
https://doi.org/10.7874/jao.2024.00024

# Contemporary Review of Idiopathic Sudden Sensorineural Hearing Loss: Management and Prognosis

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**Received** January 3, 2024
**Accepted** January 7, 2024

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Sudden sensorineural hearing loss (SSNHL) is a rapid decline in auditory function that needs urgent medical management. Although etiologic factors, including viral infections, autoimmune diseases, and vascular issues, contribute to the understanding of SSNHL, the condition remains unclear in most cases. Systemic steroids are often used as the first-line treatment because they reduce inner ear inflammation; however, there remains numerous discussions about the effectiveness of alternative treatments. To predict hearing recovery is crucial in patients' counseling with factors, including delayed treatment, vertigo, and other health conditions, which indicate poor prognosis. Herein, we review contemporary research on the treatment approaches and outcome predictions of SSNHL to establish important guidelines for physicians in evaluating and treating patients with SSNHL. **J Audiol Otol 2024;28(1):10-17**

**Keywords:** Sudden hearing loss; Sensorineural hearing loss; Treatment; Prognosis.

## Introduction

Idiopathic sudden sensorineural hearing loss (ISSNHL) is regarded as one of the medical conditions that necessitates urgent medical intervention. It is defined by the occurrence of sensorineural hearing loss of 30 dB or more in three consecutive frequencies within three days [1]. Often, it is accompanied by tinnitus, vertigo, aural fullness, nausea, and vomiting.

It is reported to affect 5 to 20 individuals per 100,000 in the United States [2] and over 10 individuals per 100,000 in Korea. Furthermore, there is a noticeable upward trend in incidence rates with increasing age [3]. Given that a natural recovery rate of approximately 32% to 65% has been documented even without specific treatment [4,5], the actual incidence rate is believed to be higher than these figures suggest. Although sudden sensorineural hearing loss (SSNHL) can occur across all age groups, it predominantly affects individuals in their 30s to 50s. Notably, there is no significant gender-based difference observed in its occurrence. The etiological factors of ISSNHL encompass various categories such as infectious diseases, otologic conditions, traumatic events, vascular and hematological disorders, and neoplastic conditions. Despite extensive research, a significant proportion of cases are categorized as idiopathic due to the absence of identifiable specific causes for hearing loss.

Factors influencing auditory recovery include the age at onset of hearing loss, severity of hearing loss, frequency of impact, presence of vertigo, and the time interval between the onset of hearing loss and seeking medical treatment.

With over 3,000 articles accessible on PubMed, along with a considerable number of articles predating the PubMed era, the literature surrounding SSNHL is extensive. Given the potentially overwhelming volume of papers for clinicians, there is a need to summarize available literature that is relevant to clinical practice. This summary aims to provide guidance regarding the treatment and prognosis of SSNHL offering assistance in making informed clinical decisions.

## Treatment

Table 1 presents a summary of treatments for SSNHL. The use of systemic and intratympanic steroids, hyperbaric oxygen therapy, and other potential therapies, along with their clinical effectiveness, treatment protocols, and debated utility



 

**Table 1.** Proposed treatment for idiopathic sudden sensorineural hearing loss

| Treatment methods | Remarks (administration route, drug name, dose, etc.) |
|---|---|
| Steroid | |
| Systemic steroid | - Oral, prednisolone 1 mg/kg/day (60 mg/day) |
| (full dose for 7 to 14 days, then taper) | - Oral/IV*, methylprednisolone 0.8 mg/kg/day (48 mg/day) |
| | - IV, dexamethasone 9–10 mg/day |
| Intratympanic steroid† | - Dexamethasone‡ (4–10 mg/mL), dose: 0.4–0.8 mL |
| (up to 4–6 injections over a 2-week period) | - Methylprednisolone (30–40 mg/mL), dose: 0.4–0.8 mL |
| Hyperbaric oxygen therapy§ | - 100% $O_2$ at 2.0–2.5 atmospheres for 90 minutes daily for 10 to 20 sessions |
| Vasoactive/hemodilution treatment | - Prostaglandin E1 (PGE1) (IV 20–60 μg/day, continuous infusion) |
| | - Pentoxifylline (IV 300 mg/day) |
| | - Dextran |
| | - Carbogen (a mixture of 95% $O_2$ and 5% $CO_2$) inhalation |
| | - Calcium channel blocker (nifedipine) |
| Antioxidants | - Vitamin: high-dose vitamin C (200 mg/kg/day intravenously for 10 days, then orally 2,000 mg for 30 days), vitamin E+C combination (tocopherol nicotinate, 600 mg/day + vitamin C, 1,200 mg/day, orally) |
| | - Magnesium (Oral/IV) |
| Other supplements | - Zinc gluconate (20 mg twice a day, orally [40 mg/day]) |
| | - EGb761 (*Ginkgo bioloba* extract) (70–200 mg/day, IV or oral) |
| | - N-acetyl-cysteine (600 mg twice a day, orally [1,200 mg/day]) |

*if the patient has gastrointestinal problems or oral administration is difficult, the intravenous route can be used. Superiority of route of steroid administration has not been established; †the choice of systemic and intratympanic use of steroids may depend on the degree of hearing loss, tolerance to high-dose systemic steroids, and patient preference. Combination therapy with systemic steroids (administered concurrently or sequentially, in either order) can be offered; ‡dexamethasone is recommended over methylprednisolone as salvage therapy; §depending on initial hearing threshold, it is optionally used in combination with systemic steroid therapy or as salvage therapy. In Korea, insurance benefits are provided to patients with an initial hearing threshold of 80 dB or higher. IV, intravenous

in medical practice, will be discussed below.

## Systemic corticosteroids

Steroid therapy is currently a widely used and standard approach for the initial treatment of SSNHL. Steroids are believed to reduce inflammation and immune responses, which includes the benefits of addressing infectious, inflammatory, and other immunological conditions affecting the cochlea and auditory nerve. The foundation for such steroid treatment is based on the randomized, double-blind, placebo-controlled study conducted by Wilson, et al. [1] in 1980. This study demonstrated a recovery rate of 61% in the oral steroid treatment group, compared to only 32% in the placebo group [1]. However, due to the high rate of natural recovery in SSNHL cases and conflicting studies regarding the effectiveness of steroids, there remains ongoing debate about their necessity. The Cochrane review also concluded that the value of oral steroids in the treatment of ISSNHL has not been convincingly demonstrated [4]. As mentioned in the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) practice guidelines, the recommendation for steroid use is presented as optional: "Clinicians may offer corticosteroids as initial therapy to patients with ISSNHL"[6].

The dosing regimen for oral steroid treatment varies, but for prednisone, a commonly recommended protocol involves initiating treatment with a single dose of up to 60 mg per day (1 mg/kg/day) [7]. This starting dose is then gradually tapered over a period of 10 to 14 days. A common tapering schedule involves using the maximum dose for 4 days, followed by a reduction of 10 mg every 2 days. The selection of this dose is based on the maximum adrenal output of hydrocortisone (cortisol), which ranges from 200 to 300 mg/day during periods of stress. Steroids used for the treatment of SSNHL include prednisone, methylprednisolone, and dexamethasone. These can be administered via systemic (oral, intravenous or intramuscular) and/or intratympanic (IT) routes. The equivalent dose of prednisone 60 mg is 48 mg for methylprednisolone and 10 mg for dexamethasone. These steroid treatments exhibit their most significant therapeutic effects within the first 2 weeks of administration, and there is a tendency for the efficacy to decrease after the onset of SSNHL, around 4 to 6 weeks. The updated 2019 AAO-HNS guidelines suggest an early treatment with steroids within 2 weeks [8].

## Intratympanic steroids

To minimize systemic side effects of steroids and maintain

higher concentrations locally within the inner ear, a method involves direct injection of the medication through the eardrum into the middle ear, known as intratympanic administration. Steroids administered intratympanically diffuse into the inner ear through the round window membrane.

IT corticosteroids administration can be employed in various scenarios. It can be utilized when there is no improvement following systemic steroid use (salvage therapy), as an adjunct treatment alongside systemic steroids, or as a primary treatment without the use of systemic steroids.

The AAO-HNS guidelines suggest immediate primary treatment or salvage therapy [6,8]. When used alongside systemic steroids, conflicting results exist regarding the effectiveness and superiority of IT corticosteroids, leaving room for ongoing debate. Assessing IT corticosteroids as an initial therapy is challenging due to variations in concentration, dosing frequency, and concurrent use with systemic steroids. Concentrations of corticosteroids can vary widely between studies, with most referring to dexamethasone at concentrations of 10 mg/mL and methylprednisolone at 30 mg/mL and higher. While IT corticosteroid treatment carries lower potential toxicity compared to systemic corticosteroid therapy, it is important to note that IT corticosteroids can still entail possible side effects. Although rare, adverse effects might encompass pain, transient dizziness, infections, persistent tympanic membrane perforation, and, during injection, the remote possibility of injuring the tympanic nerve or causing fainting.

### Hyperbaric oxygen therapy

Based on the results of randomized controlled trials suggesting that hyperbaric oxygen therapy (HBOT) could contribute to the recovery of hearing loss, the AAO guidelines present it as an option. The guidelines recommend considering HBOT alongside steroid treatment within 2 weeks of SSNHL onset, or performing HBOT within 1 month of onset [8]. Particularly noteworthy is its enhanced effectiveness in patients with moderate to severe (60 dB or more) hearing loss compared to those with mild hearing loss [6].

HBOT involves subjecting a patient to 100% oxygen at a pressure level higher than 1 atmosphere absolute within a specially designed sealed chamber. This method enables the delivery of significantly increased partial pressure of oxygen to tissues, particularly the cochlea, which is highly susceptible to ischemia. Due to the evidence of compromised blood supply or ischemic symptoms in the cochlea being potential causes of SSNHL, supplying oxygen to the cochlea has been proposed as a means of achieving therapeutic effects. Additionally, there are reports suggesting that HBOT may have immunomodulatory effects, facilitate oxygen transport, and impact hemodynamics, while also potentially mitigating damage caused by hypoxia or edema.

Since the utilization of HBOT for SSNHL in the 1960s, numerous studies have highlighted its positive effects. More recently, it has been reported that a combination therapy involving both HBOT and steroids shows more favorable outcomes than monotherapy. In recent systematic review and meta-analysis comparing HBOT combined with medical therapy (MT) to MT alone, the combination therapy demonstrated significant benefits in terms of hearing improvement [9,10]. Notably, this combined approach showed particularly favorable outcomes in cases of severe to profound hearing loss [10].

### Other treatment modalities

A wide range of agents have been examined as potential treatments for idiopathic SSNHL. These include anti-inflammatory agents, antimicrobials, calcium antagonists, vitamins (high dose vitamin C), essential minerals (such as zinc or magnesium), supplements (such as N-acetyl-cysteine, alpha lipoic acid), vasodilators, and vasoactive substances (like carbogen therapy, calcium channel blockers, pentoxifylline, prostaglandin E1, naftidrofuryl), volume expanders, defibrinogenators, diuretics, antioxidants, intravenous diatrizoate, herbal remedies, and bed rest. The majority of these drugs have been studied in combination with high-dose steroid therapy to evaluate their effectiveness. Recent research has focused on antioxidants such as vitamins, vasoactive agents like prostaglandin E1, as well as substances like zinc, EGb761(extract of *Ginkgo biloba*), and N-acetyl-cysteine [11-15]. These are currently undergoing a process of validation for their therapeutic efficacy through several retrospective studies and systematic reviews. However, there is a notable lack of recent studies on the others. There is currently insufficient evidence supporting the therapeutic efficacy of various medications other than steroids, and large-scale randomized controlled trials are needed in the future for a comprehensive assessment. Due to insufficient evidence demonstrating sufficient benefits, the AAO-HHS guidelines does not recommend the use of pharmacological treatments other than steroids, including antiviral agents, thrombolytics, vasodilators, or vasoactive substances [8].

### Combination therapy

Efforts are underway to enhance efficacy through various combinations of systemic steroids, IT dexamethasone, and HBOT. Theoretically, each treatment modality contributes to the recovery of cochlear function in different ways. Combining these therapies may optimize the potential for auditory restoration through synergistic effects. Concurrent treatment with systemic steroids and IT dexamethasone has been at-

tempted to increase the concentration of steroids within the cochlear. Additionally, given the distinct mechanisms of action, using HBOT in conjunction with steroids may enhance therapeutic effects.

## Prognosis

The likelihood of a successful recovery varies based on several factors, such as the patient's age, the presence of vertigo when symptoms first appear, the severity of hearing impairment, the initial audiogram pattern, and the duration between the onset of hearing problems and the initiation of treatment (Table 2).

The relationship between prompt treatment and auditory recovery has been well established since its initial report by Byl [2] in 1984. Byl [2] reported a complete recovery rate of 56% when patients started treatment within 7 days, but only 27% showed a complete recovery rate when patients started treatment after more than 30 days. Shaia and Sheehy [16] reported that the prognosis was better when patients received treatment within 1 month. The rationale for early intervention lies in the fact that the pathological changes responsible for causing hearing loss can progress to irreversible states over time. Although there is variation among studies regarding the exact timeframe reported, it appears that the most significant hearing recovery occurs within the first 2 weeks after onset, emphasizing the importance of initiating treatment within this window. However, it should be noted that there is no clear-cut evidence proving the benefits of early treatment, considering that approximately two-thirds of those who do not receive treatment experience spontaneous recovery within the first 2 weeks. Additionally, negative prognostic outcomes associated with longer time between onset of SSNHL and treatment initiation may simply reflect the natural course of SSNHL.

Among demographic factors, aging (typically defined as 60 years and older in most studies) is universally correlated with a decrease in the rate of hearing recovery, as well as an increase in absolute hearing thresholds.

The severity of hearing loss and the audiometric type are well-known prognostic factors, with severe to profound hearing loss and a down-sloping audiometric configuration associated with poorer recovery outcomes. Typically, patients with higher initial hearing thresholds at the onset of symptoms exhibit a slower rate of recovery compared to those with milder losses [1,2,5,17]. Although some studies did not find a significant relationship between the prognosis of hearing recovery and audiometric configuration, there are also studies suggesting a significant correlation between audiometric patterns and recovery. In most cases, it has been proposed that individuals with low-frequency hearing losses or upward-sloping audiograms may have a more favorable prognosis compared to those with high-frequency losses or downward-sloping audiograms [2,5,17,18]. Mattox and Simmons [5] considered a low-frequency or mid-frequency audiogram contour to be a valuable predictor of hearing recovery improvement. They also confirmed that, regardless of the initial hearing thresholds or the type of audiogram, hearing loss at the apex of the cochlea tends to have better recovery outcomes than at the base of the cochlea [5]. They viewed high-frequency thresholds (at 8 kHz) as an indicator of the preservation of cochlear function at the basal turn, potentially serving as a marker for potential hearing recovery [5].

**Table 2.** Factors thought to be related to the prognosis of hearing recovery in idiopathic sudden sensorineural hearing loss

| Factor | Impact on prognosis | |
|---|---|---|
| | Positive | Negative |
| Age | Young | Old (typically defined as 60 years and older) |
| Duration from the onset to treatment | Early (typically within 2 weeks or sooner) | Later |
| Pattern of audiogram (frequency loss) | Upward-sloping audiogram (low-frequency loss) | Down-sloping audiogram (high-frequency loss) |
| Severity of initial hearing thresholds | Severe to profound hearing loss | Mild to moderate hearing loss |
| Presence of vertigo | Absent | Present |
| Factors under ongoing debate | | |
| Inflammatory marker | WBC, NLR, PLR, etc. elevation | |
| Other laboratory findings | High blood glucose, high lipid profiles (TG, total cholesterol, LDL/HDL) | |
| Comorbidities | DM, HTN, dyslipidemia, metabolic syndrome | |
| Previous history of hearing loss | Hearing levels of the unaffected ear | |
| Other accompanying ear symptoms | Ear-fullness, tinnitus | |

WBC, white blood cell; NLR, neutrophil to lymphocyte ratio; PLR, platelet to lymphocyte ratio; TG, triglyceride; LDL, low-density lipoprotein; HDL, high-density lipoprotein; DM, diabetes mellitus; HTN, hypertension

Dizziness in SSNHL is reported in approximately 30% of cases and is considered to be associated with poorer prognosis. However, the precise relationship between vertigo and hearing loss, as well as the exact role of vestibular dysfunction and the location of lesions, continues to be a subject of ongoing discussion. In theory, various types of vestibular involvement can be expected in SSNHL. Anatomically, the closest vestibular organ to the cochlear is the saccule. The anterior vestibular artery, branching from the labyrinthine artery, supplies blood to all vestibular organs except parts of the posterior semicircular canal (PSCC) and the saccule. Since the PSCC receives blood from the posterior vestibular artery without collateral circulation, disruptions in blood flow can lead to ischemic damage. From an inflammatory perspective, the proximity of the inner ear's lymphatic drainage and the vestibulocochlear nerve can explain involvement of adjacent vestibular organs, which may vary among patients.

Numerous studies have explored the clinical significance of vestibular function testing in SSNHL, and recent meta-analyses indicate that vestibular organs are involved in SSNHL regardless of dizziness presence [19]. This suggests that dizziness may not independently determine SSNHL, and vertigo symptoms could result from the anatomical proximity of the cochlear duct to vestibular organs. Considering this context, studies have investigated the prognostic significance of benign paroxysmal positional vertigo (BPPV) [20,21]. In SSNHL patients with concurrent BPPV, hearing levels before and after treatment were notably less favorable than in patients without BPPV [20]. It has also been theorized that abnormal PSCC functioning, possibly due to its anatomical and vascular proximity to the cochlear and PSCC, is linked to poor hearing recovery [22]. Evaluating vestibular function, including each semicircular canal, might offer a novel prognostic indicator for hearing recovery.

Cochlear microcirculation disorders, viral, immunological, and inflammatory causes have been suggested as potential etiologies for SSNHL, and research has extensively explored the roles of blood and coagulation parameters, as well as glucose and lipid levels, as potential prognostic biomarkers.

Inflammation can induce endothelial dysfunction, leading to the acceleration of a pro-thrombotic state through vascular wall thickening, even within the ear. Against this backdrop, various inflammatory biomarkers such as white blood cell (WBC) count, WBC subtypes counts (neutrophils and lymphocytes), C-reactive protein, tumor necrosis factor-α, neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio have been studied to determine their associations with SSNHL. It has been found that when categorizing SSNHL patients into "recovered" and "unrecovered" groups, the "unrecovered" group exhibits significantly higher NLR [23], and meta-analysis have indicated associations between other markers and the prognosis of SSNHL [24,25].

Several case-control studies have illuminated a significant relationship between a higher risk of developing diabetes mellitus (DM) in SSNHL patients compared to the control group [26,27]. Furthermore, Ryu, et al. [28] observed a significantly lower hearing recovery rate in the group with impaired glucose regulation when they combined the normal glucose tolerance, prediabetes, and DM into two categories: impaired glucose regulation and normal glucose tolerance group. This suggests a hypothesis that hyperglycemia may have a negative impact on both microvascular function and neuropathy associated with high blood sugar levels, potentially influencing the prognosis of SSNHL.

Dyslipidemia, characterized by hypercholesterolemia and/or triglyceridemia, is another risk factor for SSNHL. The cochlear is highly sensitive to changes in blood circulation due to its reliance on terminal artery blood supply, and elevated blood viscosity resulting from hyperlipidemia can impede cochlear microcirculation. Hence, lipid profiles in the blood are being recognized not only as significant risk factors for cardiovascular diseases but also as major contributors to SSNHL [29,30]. Some studies have suggested that elevated total cholesterol levels and a high LDL/HDL ratio (low-density lipoprotein/high-density lipoprotein) are associated with poorer recovery outcomes [31-33], but there is still debate regarding the relationship between dyslipidemia and hearing improvement in SNHL patients.

In addition to DM and dyslipidemia, other accompanying systemic conditions related to the incidence of ISSNHL include hypertension (HTN), coronary artery disease, cerebrovascular disease, chronic kidney disease, and anemia. However, there is ongoing debate regarding the evidence supporting the association between these comorbidities and hearing recovery rate in SSNHL. Nevertheless, Jung, et al. [34] demonstrated that the rate of recovery from SSNHL was lower among patients with metabolic syndrome, particularly those with 4 or more diagnostic criteria (hyperglycemia or type 2 DM, hypertriglyceridemia, HTN, obesity, HDL reduction).

There is little evidence that routine blood testing can significantly change diagnosis, treatment, or prognosis, and although guidelines do not recommend routine laboratory testing [8], it may help identify underlying conditions that may be being missed.

Due to these various factors, efforts have been made to develop prognostic prediction algorithms for patients' chances of hearing recovery [35-37], and recently, research is underway utilizing artificial intelligence for this purpose [38].

### Pediatrics

Little is known about the prevalence, treatment outcomes, and prognostic factors of SSNHL in children. Etiologies of SNHL in children include congenital *Cytomegalovirus*, Epstein-Barr virus infection, enlarged vestibular aqueduct, Mondini's dysplasia, ototoxicity, trauma, noise-induced hearing loss, meningitis, and parotitis. Due to the scarcity of pediatric cases, it is difficult to determine the precise contribution of each etiology. Previous literature has reported a poor prognosis for pediatric SSNHL, with age (typically under 15 years) mentioned as a negative prognostic factor [2]. However, the number of pediatric patients and their recovery rates have not been clearly reported [2]. In retrospective studies targeting pediatric patients confirmed as having idiopathic SNHL, the recovery rates (complete+partial recovery) in children varied from 26.4% to 92.8% [39]. Two studies comparing the outcomes of SNHL in adults and children have found that children exhibit a higher rate of complete recovery compared to adults [40,41]. Kim, et al. [42] conducted a study in which pediatric SNHL patients were divided into two groups, taking into consideration the language development period in children (childhood; 4–12 years, adolescence; >12 years). The recovery rate in the childhood group was 36.4%, lower than the 64.4% in the adolescence group, and multivariate analysis confirmed that older age and lower initial pure-tone average thresholds were positively related to hearing recovery [42].

In a meta-analysis, increasing age, age over 12 years, an upward-sloping audiogram, and the presence of tinnitus were associated with a positive correlation with recovery [39]. On the other hand, longer treatment delays, delays of more than 6 days, greater initial hearing loss, initial hearing loss >80 dB, and profound audiograms were associated with no improvement [39]. Diagnosing SSNHL in children, especially at a young age, can be challenging, and hearing loss at a young age can impact speech and language development, academic performance, and social outcomes. While there is no clear guidance yet on the most suitable diagnostic tests and treatments for pediatric SSNHL, similar to adults, systemic steroid treatment and consideration of IT steroids as a secondary treatment may be necessary.

### Geriatrics

As individuals age, the likelihood of accompanying systemic conditions such as DM, HTN, and dyslipidemia increases, and these conditions can have a negative impact on the prognosis of SSNHL. However, there is limited research on the prognosis of SSNHL specifically in elderly patients. In recent studies involving elderly patients (typically 65 years and older) with SSNHL, the recovery rate was relatively low, which is presumed to be due to the presence of pre-existing hearing loss (presbycusis) [43]. Interestingly, these studies did not find any significant factors associated with hearing recovery [43,44]. However, other studies, which have compared elderly patients to control groups (younger individuals), have reported significantly lower hearing recovery rates among the elderly population [45,46]. Additionally, these studies have indicated that the timing of treatment initiation and the hearing status of the contralateral ear play a role in the prognosis of SSNHL in elderly patients [45,46]. In a study comparing 40 patients aged 65 years or older with 40 relatively younger patients (aged 55–64 yeasrs), there were no significant differences in hearing status or underlying conditions between the elderly and control groups [47]. Considering these findings, it remains unclear what the appropriate age cut-off should be for defining elderly patients in the context of SSNHL, and whether the prognosis of SSNHL in elderly patients is actually worse. Contrary to what was commonly believed, the treatment outcomes for SSNHL in elderly patients can be relatively favorable. However, it is crucial to consider that most elderly patients already have some degree of hearing loss. In cases where there is additional hearing impairment due to presbycusis, the possibility of hearing rehabilitation using assistive hearing devices should be early mentioned and discussed.

## Conclusion

This study reviewed numerous literature sources related to SSNHL, and a majority of researchers are progressively advancing the understanding of its treatment and prognosis prediction. Beyond steroids, the effectiveness of various treatment approaches remains limited and subject to debate, as conflicting results persist. Thus, there is a need for a clear and comprehensive understanding of the treatment of SSNHL, given the ongoing controversy and contrasting findings associated with different therapeutic methods.

#### Acknowledgments

This research was supported by a grant from the Korea Health Technology R&D Project through the Korea Health Industry Development Institute (KHIDI), funded by the Ministry of Health & Welfare, Republic of Korea (grant number: HI21C1574). This research was supported by grants from National Research Foundation (NRF) (2021R1F1A1054810).

#### Conflicts of Interest

The authors have no financial conflicts of interest.

#### Author Contributions

Conceptualization: Jae Ho Chung. Funding acquisition: Jae Ho Chung. Project administration: Jae Ho Chung. Visualization: Hyeon A Lee. Writing—original draft: Hyeon A Lee. Writing—review & editing: Jae

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