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Hyperbaric oxygen therapy for sudden sensorineural hearing loss (Review)

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Abstract. Sudden sensorineural hearing loss (SSNHL) is an acute hearing disorder that develops rapidly and is an otolaryngology emergency. Hyperbaric oxygen therapy (HBOT), a non-pharmacological treatment, has gained increasing attention for SSNHL management. HBOT exerts therapeutic effects by increasing inner ear oxygen partial pressure, improving the microcirculation and reducing inflammation, as its main mechanisms. When combined with glucocorticoids, HBOT can significantly improve treatment outcomes, particularly when initiated as an early intervention. However, its optimal clinical application has not yet been determined, mainly due to the lack of standardized treatment parameters, such as pressure settings, the duration of treatment and the therapeutic time window. In addition, clinical studies have yielded inconsistent results. The purpose of the present review is to explore the biological mechanisms, clinical efficacy and existing controversies of HBOT in the treatment of SSNHL, review and summarize the latest research progress, and discuss potential directions for future development.

Contents

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1. Introduction

Sudden sensorineural hearing loss (SSNHL) is defined as sudden, unexplained sensorineural hearing loss occurring within a 72-h period, involving at least three consecutive frequencies with a hearing loss of ≥30 dB. It typically affects one ear and may be accompanied by symptoms including tinnitus, vertigo, ear swelling and blockage. The possible mechanisms underlying SSNHL include viral infections, disturbances of inner ear microcirculation and autoimmune disorders. In cases of mild hearing loss, the cochlea may exhibit self-repair capacity, and spontaneous recovery may occur. Approximately 36% of untreated patients experience a hearing improvement of ≥30 dB within 3 months, with an average gain of 24 dB (1,2). However, in severe cases, the likelihood of a full recovery is markedly lower. At present, corticosteroids, hyperbaric oxygen therapy (HBOT) and vasoactive drugs are the main treatments for SSNHL. Among these, the combination of HBOT and glucocorticoids is widely used clinically. HBOT can rapidly increase the partial pressure of oxygen in the blood and tissue, extend the diffusion distance of oxygen in the tissue, correct cochlear tissue hypoxia, improve inner ear circulation and reduce blood viscosity (3). It has also been shown to inhibit inflammatory responses and regulate immune function (4,5). With the continuous advancement of clinical research, in 2019, the American Academy of Otolaryngology-Head and Neck Surgery Foundation (AAO-HNSF) updated its 2012 guidelines by incorporating HBOT into the recommended treatment plan. The 2019 AAO-HNSF guidelines recommended (6) the combination of HBOT with corticosteroids as a first-line treatment within 2 weeks of symptom onset, or as well as salvage therapy within 1 month. The strength of this recommendation was rated as ‘optional’ and the level of evidence as ‘moderate’, indicating growing recognition of the clinical role of HBOT in SSNHL treatment. The purpose of the present review is to explore the role of HBOT in SSNHL, focusing on its mechanisms of action, clinical efficacy and existing controversies in its use.

Although the use of HBOT in SSNHL has been recommended as a first-line or salvage therapy in combination with glucocorticoids, its clinical value is unclear. Several key issues regarding the use of HBOT remain unclarified. First, the precise mechanisms underlying the therapeutic effects of HBOT are not yet fully understood, and the molecular pathways by which it improves inner ear microcirculation, reduces inflammation and modulates the immune system require further experimental verification. Secondly, there is a serious lack of standardization of treatment parameters, including the optimal pressure, the number of sessions and the mode of oxygen delivery. Thirdly, the ideal treatment time window remains unclear. While some studies suggest that this intervention is most effective when initiated within 2 weeks of symptom onset (*vide infra*), the biological basis for the difference in outcomes for patients whose treatment begins within 72 h and those whose treatment begins after 1 month has not been fully elucidated. Finally, the cost-effectiveness of HBOT and the lack of predictive markers to identify individuals who are likely to benefit from this treatment are unclear. These knowledge gaps highlight the challenges faced when attempting to develop HBOT as a precision medicine rather than as an empirical therapy for SSNHL treatment.

2. Etiological mechanism of SSNHL

SSNHL encompasses a variety of internal ear diseases. Its etiology and pathophysiological mechanisms are not yet fully understood; however, studies suggest that vascular lesions, autoimmunity and viral infections (7,8) may be associated with its development.

Vascular lesions. Vascular lesions, such as cochlear ischemia or cochlear infarction, are among the most likely causes of SSNHL. The blood vessels in the human ear are terminal blood vessels without collateral circulation (9). Once vasospasm or embolism occurs, blood supply to the nerve cells of the inner ear is compromised, resulting in nutritional depletion. Furthermore, the hair cells of the cochlea have a high oxygen requirement and poor tolerance to hypoxia, making them particularly vulnerable to damage when the microcirculation of the inner ear is impaired. Underlying conditions including hypertension, diabetes and hyperlipidemia can accelerate vascular sclerosis and microcirculatory disorders (10), further increasing disease risk. In addition, studies have shown that hyperfibrinogenemia, antithrombin deficiency and high-factor VIII plasma levels are associated with SSNHL (11-15). These changes lead to hypercoagulability and microthrombosis, which may cause cochlear ischemia and lead to SSNHL.

Autoimmunity. Certain patients with autoimmune diseases produce specific antibodies against their own inner ear tissues, including anti-inner ear antibodies and anti-heat shock protein (HSP) antibodies (16). In such cases, the immune system mistakenly attacks the inner ear tissues. These antibodies can directly bind to hair cells, the stria vascularis or auditory nerves within the cochlea, leading to cell dysfunction or death. In addition, circulating antigen-antibody complexes may deposit in the blood vessels or basement membrane of the inner ear, activate the complement system and trigger a local inflammatory response. This can lead to microcirculation disorders or thrombosis, ultimately resulting in inner ear ischemia (10,11).

Patients with SSNHL have been found to exhibit elevated levels of circulating immune complexes and HSP70, as well as IgG antibodies against the inner ear-specific proteins cochlin and b-tectorin (15). Upregulated mean erythrocyte sedimentation rate, antinuclear antibody levels, C3, C4 and monocyte counts have also been reported (17). Furthermore, steroid therapy greatly improves hearing recovery in SSNHL (18), likely due to its immunosuppressive and anti-inflammatory effects, which supports the hypothesis that immune-mediated mechanisms contribute to SSNHL.

Viral infection. Common viruses, such as mumps, measles, varicella zoster and influenza, can directly invade the inner ear or auditory nerve, leading to inflammation. Following viral infection, the immune system releases large amounts of pro-inflammatory factors, including TNF-α and IL-6, which can trigger local inflammation in the inner ear, resulting in increased vascular permeability, tissue edema and hair cell dysfunction (4,5). In one case series, sudden hearing loss was reported in a patient with a confirmed severe acute respiratory syndrome-coronavirus-2 infection, suggesting that the infection may have contributed to SSNHL through mechanisms associated with direct damage to the inner ear structure, immune-mediated inflammation and thrombosis (19). In an experimental model, Zhuang *et al* (20) established cytomegalovirus infection in neonatal mice, and monitored auditory brainstem response tests for 3 consecutive weeks. The study revealed hearing loss, a significant increase in reactive oxygen species (ROS) levels in the inner ear, and activation of the neutrophil/lymphocyte ratio (NLR) family pyrin domain containing 3 inflammasome in the infected mice. These findings suggest that cytomegalovirus infection can induce immune or inflammatory reactions in the inner ear, which are important contributors to hearing loss.

3. Introducing HBOT

HBOT was first reported for the treatment of acute noise-induced deafness and sudden deafness by European doctors in the late 1960s. HBOT is the inhalation of 100% pure oxygen in a pressurized environment of >1 atmosphere, typically 2.0-3.0 absolute atmospheres (ATA) (21). This approach is known to elevate the oxygen content in the inner ear, rapidly raise the partial pressure of oxygen in blood and tissues and increase the diffusion of oxygen into tissues. In SSNHL HBOT attenuates cochlear tissue hypoxia, improves inner ear circulation, reduces blood viscosity, inhibits inflammatory responses and modulates immune function (22).

HBOT action mechanism. The pathological mechanisms underlying SSNHL may involve ischemia, hypoxia and microcirculatory dysfunction within the inner ear, while HBOT can ameliorate hypoxia in the inner ear, sustain normal metabolic activity in the inner ear microcirculation, induce vasoconstriction and reduce edema by increasing the oxygen partial pressure of the perilymph. Consequently, HBOT improves the

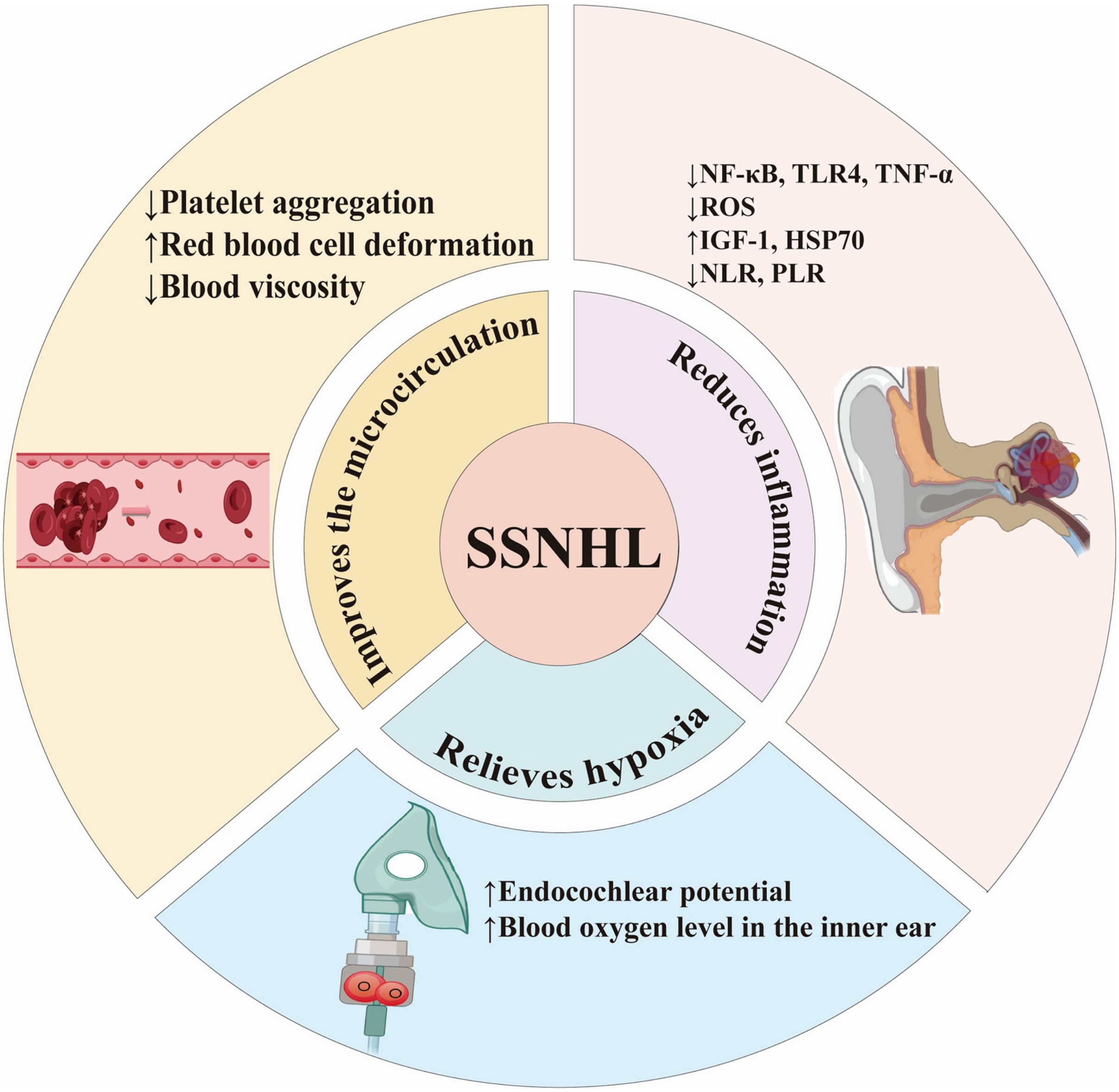

Figure 1. Mechanism of hyperbaric oxygen therapy in the treatment of SSNHL. SSNHL, sudden sensorineural hearing loss; TLR4, Toll-like receptor 4; ROS, reactive oxygen species; IGF-1, insulin-like growth factor-1; HSP70, heat shock protein 70; NLR, neutrophil/leukocyte ratio; PLR, platelet/leukocyte ratio.

permeability of the inner ear and reduces ischemia-related injury, thereby providing therapeutic effects in the treatment SSNHL. The main mechanism of HBOT in SSNHL is shown in Fig. 1.

Correction of hypoxia. The cochlea, located within the temporal bone, has a rather limited blood supply, which is mainly provided by the labyrinthine artery. Cochlear potentials play a crucial role in auditory signal conduction, as the cochlea mediates the conversion of sound waves into nerve impulses. This process is dependent on the magnitude of the cochlear potential, which relies on an adequate supply of oxygen. Elevation of the blood oxygen content leads to an increase in the amplitude of the cochlear potential and improves auditory sensitivity, whereas hypoxia results in the loss of intravascular potential, leading to SSNHL (23). Therefore, the correction of inner ear hypoxia is an important component in the treatment of SSNHL. At normal pressure at sea level, ~98% of the oxygen in the blood is complexed with hemoglobin in blood cells, and only a small amount of oxygen is dissolved in the plasma (24). However, inhalation of 100% oxygen at pressures >1.4 ATA significantly increases the diffusion radius of oxygen from capillaries to surrounding tissues (25). Breathing pure oxygen under high pressure increases the partial pressure of oxygen in the lungs and significantly increases the plasma oxygen concentration, enabling the rapid correction of ischemic and anoxic states in the inner ear. This increase in the blood oxygen tension within the inner ear capillaries ensures that sufficient oxygen reaches the auditory cells, thereby maintaining their normal metabolic function (26).

Alleviation of inflammatory reactions. Inflammation of the inner ear has been implicated in the development of sudden deafness (27,28). Toll-like receptor 4 (TLR4), NF-κB and TNF-α levels have been demonstrated to be upregulated in peripheral blood of patients with ISSHL, and to decrease after HBOT. This therapy inhibits the activation of TLR4 and NF-κB, which reduces the release of pro-inflammatory cytokines (4,29). These findings suggest that HBOT may alleviate hearing loss in patients with SSNHL by suppressing TLR4/NF-κB-mediated inflammatory responses (30). In addition, HBOT has been shown to inhibit the expression of TLR4 mRNA, thereby further blocking the transmission of inflammatory signals.

Oxidative stress is a known risk factor for microcirculatory damage (31) and plays a key role in the pathogenesis of SSNHL. It is characterized by alterations in the structure and function of erythrocyte membranes, along with increased levels of membrane lipid peroxidation and intracellular ROS production. HBOT can improve oxygen delivery and reduce the production of inflammatory mediators, including ROS (32), thereby inhibiting oxidative stress and the inflammatory cascade.

The upregulation of insulin-like growth factor-1 (IGF-1) and HSP70 is closely associated with hearing recovery. In particular, IGF-1 has been indicated to alleviate neuroinflammation by inhibiting inflammatory pathways such as TLR4/NF-κB and promoting the polarization of M2-type microglia (33), while the upregulation of HSP70 has been demonstrated to improve the inner ear microenvironment by reducing oxidative stress and inflammatory responses (6). HBOT increases the concentration of IGF-1 and HSP70 in the serum of patients (34), thereby enhancing cellular tolerance to hypoxia and injury, and contributing to improved hearing outcomes in patients with SSNHL.

The NLR is a valuable indicator of inflammatory processes, while the platelet/lymphocyte ratio (PLR) is an important indicator of microcirculatory dysfunction associated with ischemia (35). An elevated NLR may indicate inner ear microvascular inflammation, while an elevated PLR may indicate platelet activation, vascular endothelial injury or atherosclerosis. The NLR and PLR are both significantly upregulated in patients with SSNHL, suggesting the presence of underlying inflammation and vascular ischemia. In addition, a higher NLR and PLR prior to treatment are associated with a poor patient prognosis. Notably, HBOT has been demonstrated to reduce the NLR and PLR (4,36) thereby mitigating local inflammatory responses and improving clinical outcomes (14).

Overall, these findings indicate that HBOT inhibits the inflammatory response through multiple targets and pathways, including suppression of the TLR4/NF-κB signaling pathway, reduction of ROS production, upregulation of IGF-1 and HSP70, downregulation of NLR and PLR, and the enhancement of antioxidant capacity. Together, these mechanisms alleviate inner ear inflammation, protect auditory cells and promote hearing recovery.

Amelioration of ischemia and microcirculation disturbance. HBOT has been shown to significantly reduce whole blood viscosity, plasma viscosity and the blood cell aggregation index by enhancing erythrocyte deformability and reducing platelet adhesion and aggregation (3). These effects improve coagulation function and reduce blood viscosity, thereby helping to alleviate blood hypercoagulability. This promotes the restoration of blood flow and microcirculation in the inner ear, promotes local vasoconstriction, reduces capillary permeability and exudation, diminishes inner ear edema and effectively mitigates cochlear damage caused by microcirculation disorders (24-26).

Treatment parameters. HBOT can be administered in single- or multi-patient chambers (37). Single-patient chambers accommodate only one patient, and the entire chamber is usually pressurized with 100% oxygen, allowing the patient to breathe directly from the chamber atmosphere. By contrast, multi-patient chambers accommodate several individuals, where each patient breathes ~100% oxygen through a mask, hood or endotracheal tube. For HBOT to be effective in the treatment of SSNHL, the pressure in the hyperbaric chamber should be ≥1.4 ATA to provide an adequate increase in the diffusion radius of oxygen from the capillaries to the surrounding tissues (38). According to European Committee for Hyperbaric Medicine (ECHM) guidelines (39), HBOT is recommended to last for ~90 min and consist of three main phases: i) The first phase lasting ~10 min, with air compressed to 2.0-2.5 ATA and maintained for 70 min; ii) the second phase where the patient breathes pure oxygen through a mask three times for 20 min, interspersed with two 5-min air breaks without the mask; and iii) the final decompression stage. The intermittent oxygen breathing protocol is designed to minimize symptoms of oxygen toxicity.

The optimal treatment strategy for SSNHL varies according to the severity and duration of symptoms and the response to treatment. Most treatments involve an average of 10-20 sessions, with a total HBOT time of ≥1,200 min, and have the greatest effectiveness at a pressure of 2.2-2.5 ATA (40,41).

Factors influencing the curative effect of HBOT. Age, diabetes, smoking and a history of heart disease have been reported as factors affecting the severity of SSNHL (10,42). An association between metabolic syndrome and SSNHL has also been identified, and the prognosis of SSNHL in patients with metabolic syndrome is poor (43). Early intervention is critical, and studies have emphasized that the optimal effects of HBOT are achieved when this therapy is initiated within 2 weeks of symptom onset (44,45). Generally, earlier treatment initiation leads to improved outcomes (46). Notably, patients with more severe hearing loss tend to experience greater benefits from HBO (47), which may be due to the extent of improvement being more apparent in cases of greater loss. However, the recovery rate of patients with mild hearing loss is higher, whereas the prognosis of patients with severe or total deafness remains poor (48).

Side effects. The treatment parameters of HBOT can be adjusted based on factors including the age of the patient, the degree of hearing loss and underlying conditions. HBOT is generally considered safe when administered under the guidance of professional doctors (49). The main side effects are barotrauma and oxygen toxicity, which tend to be mild and reversible (50). However, it is necessary to strictly adhere to treatment indications and operating protocols, proactively prevent and manage side effects, assess the potential risks and benefits, and closely monitor patient responses to optimize treatment strategies and maximize treatment safety.

4. SSNHL therapy

The treatment of SSNHL comprises early combined intervention, with corticosteroids such as dexamethasone or prednisone as the primary treatment, supplemented with HBOT or vasoactive drugs. The treatment strategy may be adjusted according to prognostic factors.

Steroid treatment. Wilson *et al* (51) published the findings of a double-blind controlled trial in 1980, which demonstrated that oral steroids effectively improve hearing in patients with SSNHL. Since then, corticosteroids have become the first-line treatment for SSNHL and remain the main treatment option. Although the specific mechanism of steroid action in the inner ear remains unclear, the basic therapeutic effect is the reduction of edema and inflammation (52). Multinational guidelines recommend systemic or local corticosteroid therapy as the initial treatment choice (6,53), which should be initiated within 2 weeks of symptom onset. Systemic administration includes both intravenous and oral administration, while local administration includes intratympanic or retroauricular injections. No difference in efficacy has been detected between the two types of administration (54). Systemic administration is more convenient and does not involve invasive procedures. For patients without contraindications, systemic medication rapidly achieves therapeutic drug concentrations, potentially inhibiting the immune response and improving inner ear microcirculation (55). Conversely, local glucocorticoid therapy offers advantages in safety and targeting, particularly in patients with contraindications to systemic hormone use (18).

Combined application of HBOT and glucocorticoids. HBOT increases the oxygen concentration in the inner ear, elevates the perilymphatic oxygen partial pressure, and promotes the recovery of cochlear function, particularly in patients with severe hearing loss (≥70 dB) (34). The 2019 AAO-HNSF guidelines recommend initiating HBOT and steroids within 2 weeks from symptom onset, or as salvage therapy within 1 month. The efficacy of HBOT is time-dependent, with an optimum benefit within 24-48 h, or at latest within 2 weeks (45,56). Although the recommended timing of treatment varies in different studies, the expert consensus is that earlier intervention achieves improved outcomes (46). The mainstream theory holds that the effect of combined HBOT and glucocorticoids is improved compared with that of drug therapy alone. However, factors including high cost, a dependence on specialized equipment, strict treatment windows and potential delays to treatment may reduce the beneficial effect.

Vasoactive drugs. Depending on the condition of the patient, such as elevated initial fibrinogen levels, vasoactive drugs including prostaglandin E1 (PGE1), batroxase and *Ginkgo biloba* extract may be added to standard corticosteroid therapy. These drugs help to reduce fibrinogen levels and blood viscosity, increase local blood flow, and improve hemorheology (57). Combining vasoactive drugs with conventional treatment has been shown to be more effective in the treatment of SSNHL than conventional treatment alone (58).

Other treatments. Due to insufficient evidence supporting their efficacy, the 2019 AAO-HNSF guidelines explicitly recommend against the routine use of antiviral drugs, thrombolytics, vasodilators and antioxidants (6). Therefore, in clinical practice, the decision to incorporate these into the conventional treatment plan recommended by the guidelines requires careful consideration. Further research is necessary to explore the mechanisms of action of these drugs to establish an accurate and reliable clinical basis for their use.

5. Clinical application of HBOT in SSNHL

According to existing studies, HBOT is effective in treating SSNHL, with few reported side effects, and may serve as a useful supplementary regimen to corticosteroid therapy (Table I) (3-5,29,34,46-47,59-86). However, the current research evidence is limited by low quality and high heterogeneity, underscoring that the efficacy of HBOT requires further verification in further high-quality studies.

6. Controversies in HBOT for SSNHL

As mentioned above, although HBOT is recommended for the treatment of SSNHL in current guidelines, its clinical value remains debated, mainly due to the following aspects: i) Inconsistent clinical evidence for efficacy. While several studies report that HBOT combined with glucocorticoids significantly improves the overall response rate (34,59-61), other studies have indicated that HBOT combination therapy does not significantly improve SSNHL (34,65,69), or exhibits reduced efficacy compared with steroids alone (71). These discrepancies may be attributable to heterogeneity in study design, insufficient statistical power due to small sample sizes, and variations in treatment timings and combination medications. In addition, HBOT was reported to be more effective in the treatment of low-frequency SSNHL in one study (84), but more effective against high-frequency SSNHL in another (77). This could be associated with differences in the oxygen metabolism sensitivity of different areas of the inner ear. However, the mechanism underlying the differences in HBOT efficacy according to hearing loss frequencies remains unclear, and there is no quantitative evidence supporting a differential sensitivity of inner ear hair cell regions to hypoxia. ii) Lack of standardized treatment parameters. Standardized treatment parameters for HBOT lack standardization, particularly regarding the optimal pressure and treatment course. Although current guidelines recommend a pressure range of 1.4-3.0 ATA (6), the optimal pressure has not been defined. The recommended number of treatments is 10-20; however, some patients require up to 3 treatment courses, which is not supported by robust evidence. Furthermore, it is unclear whether intermittent oxygen administration is superior to continuous oxygen inhalation, and the differences in efficacy between single- and multiple-occupancy chambers have not been adequately studied. The associations of the number of treatments, pressure and duration with the degree of hearing recovery have not been established. Therefore, the standardization of treatment is challenging. iii) Uncertainty regarding the treatment window. The guidelines recommend that HBOT should be initiated within 2 weeks of symptom onset (6); however, certain studies suggest that the optimum effect is achieved within 72 h, with a marked reduction in efficacy

Table I. Summary of clinical trials evaluating HBOT for SSNHL, published 2015-2024.

First author/s, year	Study type	Groups	HBO intervention	Results	(Refs.)
Feng *et al*, 2022	Randomized controlled trial	HBO + alprostadil (n=52), alprostadil (n=52)	2.0 ATA 100% O_2 for 60 min, 10 sessions	↑Tinnitus; aural fullness and vertigo amelioration; ↑coagulation function	(3)
Çiçek *et al*, 2021	Retrospective	HBOT (n=30), healthy control (n=30)	2.5 ATA 100% O_2 for 120 min, 10 sessions	↓NLR; ↓PLR	(4)
Li *et al*, 2015	Retrospective	HBOT (n=41), control (n=45), healthy control (n=14)	2.0 ATA 100% O_2 for 90 min, 10 sessions	↓NLR	(5)
Liu *et al*, 2020	Randomized controlled trial	HBO (n=60), healthy control (n=20), control (n=60)	2.0 ATA 100% O_2 for 60 min, 15 sessions	↓TLR4, NF-κB and TNF-α	(29)
Wang *et al*, 2023	Retrospective	HBOT (n=79), control (NA)	2.5 ATA 100% O_2 for 92 min, 10-20 sessions	↑Hearing improvement (vs. initial value)	(46)
Choi *et al*, 2020	Retrospective	HBOT (n=37), control (n=45)	1.5-3.0 ATA 100% O_2 for 50 min, 14 sessions	↑Hearing improvement	(47)
Caragli *et al*, 2024	Retrospective	HBOT (n=86), control (NA)	2.2 ATA 100% O_2 for 40 min, 20 sessions	↑Hearing improvement (vs. initial value)	(59)
Sanda *et al*, 2024	Retrospective	HBOT (n=67), control (n=68)	2 ATA 100% O_2 for 60 min, 10 sessions	↑Hearing improvement	(60)
Lee *et al*, 2024	Retrospective	HBOT (n=18), control (n=66)	2 ATA 100% O_2 for 60 min, 10 sessions	↑Hearing improvement	(61)
Choi *et al*, 2024	Retrospective	HBOT (n=54), control (n=59)	2.4 ATA 100% O_2 for 90 min, 14 sessions	↑Hearing improvement (among diabetic patients)	(34)
Celik and Akil, 2024	Retrospective	HBOT (n=50), control (NA)	2.5 ATA 100% O_2 for 60 min, 20 sessions	↑Hearing improvement (vs. initial value)	(70)
Mariani *et al*, 2023	Retrospective	SS (n=34), SS + ITS (n=12), SS + HBOT (n=16)	2.5 ATA 100% O_2 for 90 min, 10 sessions	↑Hearing improvement	(71)
Skarzynski *et al*, 2023	Retrospective	HBOT (n=36), control (n=27)	2.5 ATA 100% O_2 for 60 min, 10-15 sessions	↔Hearing improvement	(62)
Včeva *et al*, 2022	Retrospective	HBOT (n=59), control (NA)	2.0 ATA 100% O_2 for 90 min, 20 sessions	↑Hearing improvement (vs. initial value)	(72)
Dova *et al*, 2022	Randomized controlled trial	Control (n=25), HBOT (n=25)	2.2 ATA 100% O_2 for 80 min, 15 sessions	↑Hearing improvement	(73)
Huo *et al*, 2022	Retrospective	Control (n=20), HBOT (n=72)	2.2 ATA 100% O_2 for 60 min, 10 sessions	↑Hearing improvement; ↑HGB, HCT and SOD	(74)
Huang *et al*, 2021	Randomized controlled trial	HBOT (n=60), control (n=45)	2.4 ATA 100% O_2 for 120 min, ≤10 or >11 sessions	↑Hearing improvement	(75)
Kayalı Dinç *et al*, 2020	Retrospective	HBOT (n=22), control (n=28)	2.4 ATA 100% O_2 for 120 min, 20 sessions	↔Hearing improvement (as salvage therapy)	(63)
Keseroğlu *et al*, 2020	Retrospective	SS (n=32), SS + ITS (n=32), SS + HBOT (n=32)	2.5 ATA 100% O_2 for 120 min, 20 sessions	↔Hearing improvement	(76)
Suzuki *et al*, 2019	Retrospective	HBOT (n=174), control (n=127)	2.5 ATA 100% O_2 for 60 min, 10 sessions	↓Hearing improvement	(65)
Cho *et al*, 2018	Prospective	HBOT (n=30), control (n=30)	2.5 ATA 100% O_2 for 60 min, 10 sessions	↑Hearing improvement	(77)
Hosokawa *et al*, 2018	Retrospective	SS (n=160), ITS + SS (n=35), HBOT + SS (n=161)	2.0 ATA 100% O_2 for 60 min, 10 sessions	↑Hearing improvement	(78)
Krajcovicova *et al*, 2018	Prospective	HBOT (n=47), control (n=21)	2.0 ATA 100% O_2 for 90 min, 10 sessions	↑Hearing improvement	(79)

Table I. Continued.

First author/s, year	Study type	Groups	HBO intervention	Results	(Refs.)
Almosnino *et al*, 2018	Retrospective	HBOT (n=18), control (n=18)	2.36 ATA 100% O_2 for 90 min, 10-20 sessions	↔Hearing improvement	(64)
Khater *et al*, 2018	Retrospective	HBOT (n=11), control (n=11)	2.0 ATA 100% O_2 for 60 min, 20 sessions	↑Hearing improvement	(80)
Sun *et al*, 2018	Retrospective	Control (n=41), ITS (n=31), HBOT (n=32)	2.0 ATA 100% O_2 for 90 min, 21 sessions	↑Hearing improvement	(67)
Chi *et al*, 2018	Prospective	HBOT (n=30), control (n=30)	2.5 ATA 100% O_2 for 90 min, 10 sessions	↑Hearing improvement	(81)
Ajduk *et al*, 2017	Retrospective	HBOT (n=43), control (n=50)	2.5 ATA 100% O_2 for 60 min, 20 sessions	↑Hearing improvement (particularly severe hearing loss)	(82)
Hosokawa *et al*, 2017	Retrospective	HBOT (n=167), control (n=160)	2.0 ATA 100% O_2 for 60 min, 10 sessions	↑Hearing improvement (as salvage therapy)	(83)
Gülüstan *et al*, 2016	Retrospective	HBOT (n=27), control (n=30)	2.5 ATA 100% O_2 for 120 min, 21 sessions	↔Hearing improvement (as salvage therapy)	(84)
Callioglu *et al*, 2015	Retrospective	HBOT (n=21), control (n=23)	2.5 ATA 100% O_2 for 90 min, 20 sessions	↔Hearing improvement	(85)
Capuano *et al*, 2015	Retrospective	SS (n=100), IVS + HBOT (n=100), HBOT (n=100)	2.5 ATA 100% O_2 for 90 min, 16 sessions	↑Hearing improvement	(86)

Control groups were treated with steroids, and HBOT groups were treated with HBOT + steroids. HBOT, hyperbaric oxygen; SSHNL, sudden sensorineural hearing loss; ATA, absolute atmospheres; NA, the number of participants was not recorded; SS, systemic steroids; ITS, intratympanic steroids; IVS, intravenous steroids; HGB, hemoglobin; HCT, hemocrit; SOD, superoxide dismutase; NLR, neutrophil/lymphocyte ratio; PLR, platelet/lymphocyte ratio; TLR4, Toll-like receptor 4; ↑increased in the HBOT group; ↓decreased in the HBOT group; ↔ similar in the HBOT and comparator group(s).

after 1 month (46). For patients receiving delayed treatment (>30 days), HBOT has been suggested to improve long-term prognosis by promoting collateral circulation (85), although this is not supported by high-quality evidence. The critical time point at which hair cell damage and auditory nerve injury become irreversible has not been established, which complicates the definition of salvage therapy. iv) Controversy regarding the necessity of vasoactive drugs. Although current guidelines clearly oppose the routine use of vasodilators or anticoagulants, clinical regimens combining HBOT with batroxobin and PGE1 have been used (57,86). However, the efficacy and safety of these combinations lack evidence-based support. v) Divergence in the strength of guideline recommendations. The 2019 AAO-HNSF guidelines (6) list HBOT as a recommended regimen with a moderate level of evidence, whereas some European guidelines, such as those from the ECHM (39), classify it as a second-line treatment. vi) Questionable economic value. HBOT is highly dependent on specialized equipment and incurs a high cost. Whether its long-term efficacy exceeds that of conventional treatment requires confirmation based on long-term follow-up data from multiple centers with large sample sizes.

In the future, treatment parameters such as pressure, course duration and oxygen supply mode should be standardized based on evidence-based medicine. In addition, the optimal timing of early intervention and salvage therapy should be determined through prospective studies to clarify the treatment window period. Furthermore, the cost-benefit ratio of HBOT requires evaluation, to optimize the allocation of medical resources.

7. Discussion

As an adjuvant therapy for SSNHL, the principal value of HBOT is its ability to increase the oxygen partial pressure of the inner ear, improve microcirculation and suppress inflammatory responses, thereby exerting therapeutic effects in coordination with corticosteroids. The etiology of SSNHL is complex and multifactorial, and may involve vascular lesions, autoimmune reactions and viral infections. HBOT increases the partial pressure of oxygen in the inner ear, which improves the cochlear blood supply and reduces tissue damage by enhancing the deformability of red blood cells and reducing blood viscosity. In addition, HBOT inhibits the TLR4/NF-κB inflammatory pathway, reduces the release of proinflammatory mediators, and enhances the tolerance of cells to hypoxia by upregulating protective proteins such as IGF-1 and HSP70. Collectively, these mechanisms provide a physiological basis for the use of HBOT in the treatment of SSNHL.

The early combined use of HBOT has been shown to significantly increase the hearing recovery rate, with greater effects being observed for severe hearing loss, refractory cases and young patients (46-48). However, its clinical application remains challenging due to several inconsistencies. First, evidence of its efficacy is contradictory, with certain studies reporting that HBOT combined with corticosteroids improves outcomes while others have not observed any significant benefit; this may be attributable to heterogeneity in the study design and limited sample sizes. Secondly, the treatment parameters are not standardized, with no consensus on the optimal pressure, number of sessions or mode of oxygen delivery. Thirdly, the definition of the therapeutic time window is unclear; although guidelines recommend starting within 2 weeks of symptom onset, high-quality evidence supporting the feasibility of salvage therapy beyond 1 month is lacking. Finally, the evidence base for combining HBOT with vasoactive drugs is weak and conflicts with the conservative stance of current guidelines.

Follow-up studies involving multidisciplinary collaboration are necessary to conduct high-quality clinical trials, combine basic and clinical research, optimize treatment parameters and define the therapeutic time window. In addition, it would be beneficial to establish a biomarker prediction system to identify the patients most likely to benefit from HBOT, as well as to systematically evaluate the long-term efficacy and cost-effectiveness of HBOT.

8. Conclusion

As an adjunctive therapy, HBOT combined with steroids can improve hearing capacity and alleviate symptoms of tinnitus and vertigo in patients with SSNHL. Although the application of HBOT in SSNHL is supported by some clinical evidence, standardized parameters, treatment timing and the underlying mechanism require further elucidation. Individualized treatment plans that consider the physical condition of each patient are important to maximize efficacy and reduce side effects. In addition, the optimization of combination treatment strategies is essential to improve the hearing recovery rate.

Future large-sample, multi-center studies are necessary to develop standardized treatment protocols, identify patients most likely to benefit from HBOT via molecular marker testing, and evaluate cost-effectiveness. Such research will help to promote the transformation of HBOT from empirical use to an individualized evidence-based treatment by addressing existing controversies and clarifying unresolved issues.

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Availability of data and materials

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Authors' contributions

SMP and XLG conceived the idea for the study and obtained funding. YTS contributed to study design and wrote the manuscript. SYW and HMF were responsible for structuring the manuscript and designing the narrative to enhance its coherence. Data authentication is not applicable. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

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Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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