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Factors influencing the outcome of idiopathic sudden sensorineural hearing loss treated with hyperbaric oxygen therapy

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Abstract Idiopathic sudden sensorineural hearing loss (ISSNHL) is an otologic emergency with an incidence of about 5–20 per 100,000 of the population per year. There is no universally accepted standard protocol for the treatment of patients with ISSNHL. Hyperbaric oxygen therapy (HBOT), was first reported to improve the outcome following acute inner ear disorders during the late 1960s by both French and German authors. The increase in perilymph oxygenation produced by HBOT provides logical basis for the use of this treatment modality in ISSNHL. We reviewed the records of 97 cases that received HBOT for SSNHL to identify the factors that may affect the treatment outcomes. The effects of age, gender, affected ear, status of the contralateral ear, symptoms associated with hearing loss, presence of a cardiovascular disease, dyslipidemia, history of diabetes mellitus, seasonal factor, smoking, degree of hearing loss, audiogram type, medical treatments provided prior to HBOT, onset time, and number of HBOT sessions were evaluated. The mean hearing gain in all cases after the HBOT was 29.5 dB. The gains were statistically significant in the following cases: early onset of HBOT ($p = 0.016$), higher number of HBOT sessions ($p < 0.01$), steroid usage ($p = 0.009$), low frequency-ascending and total audiogram configuration ($p < 0.01$) and profound hearing loss ($p = 0.011$). The success rate was significantly lower in cases with high frequency-descending audiogram configuration ($p < 0.001$). The most important factor affected the prognosis favorably was found as steroid therapy. This retrospective study and our clinical experience suggest that HBOT has beneficial effects when administered in the early phase of the disease together with steroids. HBOT is a safe practice when used properly by an experienced hyperbaric team. In the treatment of ISSNHL, 20 sessions of HBOT at 2.5 ATA can be tolerated well besides some minor side effects. HBOT should be considered for the cases especially with total or profound hearing loss.

Keywords Idiopathic sudden sensorineural hearing loss · Hyperbaric oxygen treatment

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Introduction

Idiopathic sudden sensorineural hearing loss (ISSNHL) is an otologic emergency with an incidence of about 5–20 per 100,000 population per year [1]. It was first described in 1944 as a sudden, usually unilateral and greater than 30 dB

hearing loss (HL) over at least three contiguous frequencies, occurring over a period of 72 h. It is diagnosed by the lack of a definite cause of this sudden hearing loss [2]. The 32–65% reported rate of spontaneous recovery suggests the assumption that some patients may have this disease without visiting any physician [2, 3]. Therefore, the actual incidence rate of ISSNHL might be higher than that reported [4]. Various theories have been proposed for the cause of the disease. These theories are vascular disturbances (tromboembolic events, vasoconstriction, hypertension and blood hyperviscosity), viral infections, immune disorders, cochlear membrane damage, metabolic, and toxic causes [3, 5–8]. The confusion about the etiology and incidence has an influence on the treatment. Therefore, such a large number of treatment protocols have been suggested for ISSNHL. Vasodilators, vitamins, steroids, anticoagulants, histamine, tranquilizers, diuretics, prostacyclin, hypervolemic hemodilution, antiviral agents, carbogen, stellate ganglion blockage, and hyperbaric oxygen treatment are currently used alone or in different combinations for the treatment of ISSNHL in our country, as in all around the world [9–16].

Hyperbaric oxygen therapy (HBOT), was first reported to improve the outcome following acute noise-induced hearing loss, sudden deafness, and peripheral vestibular disorders, during the late 1960s by both French and German authors [10, 11]. This medical treatment is performed by intermittent 100% oxygen administration to a patient completely under pressures higher than 1 atmosphere absolute (ATA) in a pressure chamber [17]. The increase in perilymph oxygenation provided by HBOT is the logical basis for the use of this treatment modality in ISSNHL [10–15]. During the HBOT, oxygen is diffused from various terminal cochlear capillary networks into the perilymph and cortilymph, supplying the oxygen needs of the sensory and peripheral neuronal structures of the inner ear, since these structures are not directly vascularized. The increase in arterial partial oxygen pressure (pO_2) correlates with the pO_2 in the perilymph and cortilymph. In experimental models of SSNHL induced by gunshots, impulse noise and broad-band noise, the arterial pO_2 was increased up to a level of 241.5 kPa, together with 560% increase in perilymphatic pO_2 levels by HBOT at 2.6 ATA. It was also shown that high levels of perilymphatic pO_2 remained 60 min after the HBOT [18, 19]. This effect cannot be obtained by a medical treatment.

Some clinical and experimental studies about the therapeutic effect of HBOT in SSNHL have been reported since its first use in 1970. Sudden deafness has been recommended as an optional indication for HBO treatment in Europe since the First European Consensus Conference on Hyperbaric Medicine in Lille, France, in 1994 [10–15, 18, 19]. In our country, HBOT is used as an auxiliary treatment in ISSNHL, as in various conditions associated with hypoxia such as necrotizing soft tissue infections, crush injuries, compartment syndromes and other acute traumatic peripheral ischemias, radiation necrosis, and impaired wound healing. However, it is difficult to evaluate the therapeutic effectiveness of HBOT or other treatment modalities in ISSNHL since the modalities are used in combination and the spontaneous recovery rate ranged between 32 and 65% [2, 3]. When concerns about the cost for treatment of ISSNHL are considered, the data available on therapeutic effect of hyperbaric oxygenization are becoming a need for otologists and hyperbaric physicians. Therefore, determining the factors influencing the outcome of the treatment protocols used in clinical practices for ISSNHL was one of the important elements of this study.

Materials and methods

The records of 97 patients who received HBOT with the diagnosis of SSNHL between the years 2005 and 2008 were reviewed. The inclusion criteria in Table 1 were used to enroll the cases to the study. The previous treatment protocols before HBOT started, cohort characteristics and response to HBOT were evaluated in 80 patients who met the inclusion criteria. The effects of age, gender, affected ear (localization), status of the contralateral ear, symptoms

Table 1 Inclusion criteria

Sudden, unilateral sensorineural hearing loss of at least 30 dB over three frequencies developing within 72 h
Pre-treatment and at least one post-treatment audiogram was performed
No evidence of retrocochlear disease evident on magnetic resonance imaging
No history of otologic surgery
No history of Meniére's disease, trauma, autoimmune hearing loss, radiation-induced hearing loss, or other potential etiology for sensorineural hearing loss
No history of acoustic trauma or barotrauma
No history of genetic sensorineural hearing loss or known inner ear anomaly
Contraindications for HBOT [e.g., untreated pneumothorax, uncontrolled seizure disorders, severe chronic obstructive pulmonary disease (COPD), multiple pulmonary blebs, severe upper respiratory infection, malignancy, presence of cardiovascular instability]

associated with hearing loss (tinnitus, dullness of hearing, vertigo, nausea, vomiting), presence of a cardiovascular disease (hypertension, ischemic heart disease, dysrhythmia), dyslipidemia, history of diabetes mellitus, season of the onset of the disease, smoking, degree of hearing loss, audiogram type, medical treatments provided prior to HBOT (anti-inflammatory agents, blood flow promoting drugs, antiviral agents), onset time, and number of HBOT sessions were evaluated, as factors that may influence the outcome. Pretreatment and post-treatment audiometric evaluations including pure tone average (PTA) were analyzed and gain greater than 20 dB in PTA was considered significant. PTA was calculated as an average of the threshold measured at 0.5, 1.0, and 2.0 kHz. The pretreatment audiogram configurations were categorized into one of five types (low frequency-ascending, midfrequency U-shaped, high frequency-descending, flat and total deafness) using the classification scheme of Mazzoli et al. [20]. Hearing loss degree classification was made according to Goodman Scale [21]. HBOT was administered in a double lock multiplace hyperbaric chamber twice a day. The treatment pressure was 2.5 ATA and each HBOT sessions consisted of three 25 min oxygen periods with 5-min air break intervals. HBOT sessions were carried out by trained healthcare personnel under the supervision of hyperbaric medicine specialist. 5-min air breaks during treatment were used to reduce the risk of oxygen toxicity. The decision of terminating HBOT was given by the referral clinic by evaluating the audiometric follow-ups. Statistical evaluation of data was performed by using SPSS for windows 10.0 multitask statistics program. Student's t, Mann–Whitney U, paired t and Chi-square tests were used for comparisons. Multivariate analysis was done by using logistic regression analysis. $p < 0.05$ was accepted as statistically significant.

Results

Seventeen out of 97 cases were excluded since five patients had a diagnosis of Meniére's disease before or during HBOT: two patients had a history of head injury, three cases had otologic surgery, one case had acoustic trauma and six cases had missing or incomplete records. Forty-two male (52.5%) and thirty-eight female (47.5%) cases with average age of 40.9 years were included in the study. The right ear was involved in 36 cases (45%) and the left in 44 (55%) cases. While hearing loss was associated with tinnitus in 97% of the cases, vertigo (in 36% of cases) and nausea, and vomiting (in 18% of cases) were the associated vestibular symptoms. Cardiovascular risk factors were identified as hypertension (in 26% of cases), dyslipidemia (in 17% of cases), ischemic heart disease (in 7% of cases), and rhythm disorders (in 5% of cases). There was history of smoking in 56% of the cases. Three cases had Type II (non insulin dependent) and one case had Type I (insulin dependent) diabetes mellitus. All cases had systemic medical treatments prior to HBOT. The most commonly preferred drugs were steroids (in 85% of the patients), and they were prescribed in different combinations with blood flow promoting drugs (Dextran 40, pentoxyfilline, betahistine HCl, gingko biloba extract (EGb 761), piracetam) and antivirals. In more than 50% of the 68 patients, steroids were given intravenously and in high doses; in the rest of the patients oral steroids were used; intratympanic steroid injections had never been used. The mean interval between the onset of symptoms and HBOT was 11.2 (±10.8) days ranged between 3 and 67 days. The audiogram configuration before HBOT was flat in 30%, total deafness in 11.3%, high frequency, descending in 32.5%, low frequency, ascending in 25% and midfrequency U-shaped in 1.3% of cases. There was more than 70 dB hearing loss in 50.05% of cases. The cohort characteristics of the cases are shown in Table 2. The average number of total HBOT session was 18 (median 20, range 5–31 sessions). The treatment tolerated well except seven cases. One case could not complete the number of HBOT sessions planned and another case had to receive sedatives before HBOT sessions since she had confinement anxiety. In five cases the treatments were interrupted for 3–5 days because of minor ear or sinus barotraumas. The mean hearing gain was 29.5 dB in the cases after HBOT. The gains were 20 dB or greater in 51.2%, between 10 and 20 dB in 16.2%, and 10 dB or less in 23.7% of the cases. There was no response to HBOT in seven cases (8.7%). The HL during HBOT was not worsened in any case. The initial and final average hearing levels (±SD values) for six frequencies are shown Fig. 1. The hearing gains were statistically significant in the cases with early onset of HBOT ($p = 0.016$), high number of HBOT sessions ($p < 0.01$), steroid usage ($p = 0.009$), low frequency-ascending and total audiogram configuration ($p < 0.01$) and profound hearing loss ($p = 0.011$). The success rate was significantly lower in cases with high frequency-descending audiogram configuration ($p < 0.001$). No statistically significant differences were found among the groups set by age, gender, localization, contralateral hearing loss, seasonal distribution, existence of tinnitus, dullness of hearing, vertigo, nausea, vomiting, hypertension, ischemic heart disease, dysrhythmia, dyslipidemia, history of diabetes mellitus, and smoking. The recovery rates in patients who received Dextran 40 infusion or did not were 68.8 and 31.3%, respectively. The difference was not significant (χ^2 test, $p = 0.11$). The recovery rate was worse but not significant in patients who had history of smoking: 55.6 versus 44.4% (χ^2 test, $p = 0.16$). Steroid therapy by logistic regression analysis (β coefficient was 1.647; constant, −1.720) was the most important factor that affected the prognosis.

Table 2 Characteristics of patients with idiopathic sudden SNHL

Age, mean (years)	40.9 (range 13–70)
Gender (male/female)	42 (52.5%)/38 (47.5%)
Localization (right/left)	36/44
Season of the hearing loss occurence (winter/spring/summer/autumn)	15/27/22/16
Presence of associated symptoms	
Tinnitus	78 (97%)
Vertigo	29 (36%)
Nausea and vomiting	15 (18%)
Dullness of hearing	9 (11%)
Cardiovascular disease	
Hypertension	21 (26%)
Dislipidemia	14 (17%)
Coroner artery disease	6 (7%)
Dysrrhythmia	4 (5%)
Diabetes mellitus	5 (6%)
Smoking	45 (56%)
Prior systemic treatment	
Antiinflammatory agents	
Steroid	68 (85%)
Blood flow promoting drugs	
Betahistine HCl	20 (25%)
Dextran 40	16 (20%)
Pentoxyfilline	16 (20%)
Piracetam	8 (10%)
Gingko extract (EGb 761)	5 (6%)
Antiviral agents	24 (30%)
Type of pure-tone audiometry curve	
Low frequency, ascending	20 (25%)
Midfrequency, U-shaped	1 (1%)
High frequency, descending	26 (32%)
Flat	24 (30%)
Total	9 (11%)
Degree of hearing loss	
Mild	11 (14%)
Moderate	14 (17%)
Moderately severe	15 (19%)
Severe	21 (26%)
Profound	19 (24%)
Interval between onset of symptoms and HBO therapy (mean ± SD)	11.2 ± 10.8
Number of HBO treatments (mean ± SD)	18.2 ± 7.0 (range 5–31)

Discussion

Since the first use of HBOT in ISSNHL in 1970s, the unidentified etiopathogenesis and unpredicted clinical course of the disease has kept its secret. The disease has high spontaneous recovery rate and these recoveries are

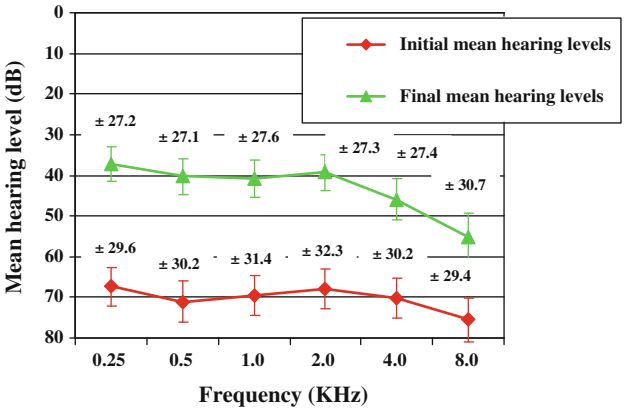

Fig. 1 Initial and final mean hearing levels (±SD values) at six frequencies in patients receiving HBOT

take place usually in the first week. Therefore, it is difficult to decide on optimal dosage, onset time, and treatment period of HBOT, as well as on selection of the cases, like in the other treatment modalities for ISSNHL. Arguments on the results of the treatment modalities up to now led us to investigate the factors that affect the outcomes. Since in the medical literature the treatment protocols were compared by evaluating age, sex, type of audiogram, degree of hearing loss, tinnitus, vertigo, duration of the hearing loss, seasonal distribution, existence of a systemic disease (diabetes mellitus, hypertension, dysfunctions of thyroid), and onset of the treatment, we investigated these factors in the medical records of our cases [1–3, 12–16, 22]. In this retrospective study we also tried to get data about the different treatment approach for SSNHL in our country, since different centers have different treatment modalities in Turkey, as in all over the world.

In our study we detected that steroid was the most preferred and effective pharmaceutical agent. We noticed that more than half of our cases had high dose of steroids during hospitalization. Most of these cases had severe and profound hearing loss. The most preferred form of steroid was methylprednisolon. All cases who did not receive steroid treatment had diabetes mellitus or hypertension, and these cases are treated as outpatient. The clinical studies showing the positive effect of the combination of high doses of steroids with HBOT in the treatment of ISSNHL support this approach in the current literature [12–15]. Experimental acoustic trauma models also point out the effectiveness of the same combination. It was noticed that fivefold increase in perilymphatic oxygen tension by HBOT at 2.6 ATA was not sufficient enough for therapeutic effect, and for the best hearing recovery on auditory brainstem and compound action potentials of auditory nerve, HBOT should be combined with steroids [18]. In the majority of the cases, steroid was used in combination with blood flow promoting agents. It was striking that antiviral

agents were added to the treatment in 30% of cases. There are studies reporting that the agents widely used in the treatment protocols of ISSNHL like dextran, piracetam, betahistine HCl, pentoxyfilline, and antivirals had no substantial effect on the treatment outcome [15, 16].

One of the proposed etiological mechanisms is viral theory and some of our cases had antiviral agents. Therefore, we investigated if there is any correlation between the prognosis and seasonal changes. Although the number of the cases is a bit higher in spring, seasonal distribution of the cases did not have an effect on the prognosis [14]. The numbers of the male and female cases were close to each other and there was no correlation between prognosis and sex. In our study the treatment outcomes in the cases with old ages did not differ from other cases, in contrast to some studies [1, 3, 12, 14].

The audiogram characteristics of the cases had effects on the prognosis. The success of the treatment in the cases with low frequency-ascending type audiogram was better than the cases with high frequency-descending type audiogram, in accordance with the literature [3, 23]. In our study the success of the treatment in the cases with total hearing loss was surprising. There was also unexpected success in the cases with profound hearing loss. The authors indicated that extensive damage in cochlea may cause severe hearing loss together with vestibular symptoms like vertigo, nausea, and vomiting [1, 3, 14]. The cases with these serious symptoms may seek for treatment without a delay. The good result in our cases may be due to early treatment. We did not find a correlation between the response to the treatment and the existence of symptoms as vertigo, nausea and vomiting, and dullness of hearing.

In majority of our cases the hearing loss had developed in a short period of time, or it was noticed in the morning which is in accordance with the current knowledge about ISSNHL. This characteristic of the disease is similar in the other end-artery pathologies as myocardial infarction, cerebral stroke, and amaurosis fugax. This similarity also underlies the basis for the efforts to find relationship between the pathologies in labyrinthic artery end functional system that supplies blood to the vestibulocochlear structures and cardiovascular risk factors [22]. In our cases there was no relation between cardiovascular risk factors and prognosis. On the other hand the healing rates in non-smoker and smokers were 60 and 44.4%, respectively.

This study also showed us there is no consensus on HBOT in ISSNHL. Some of the ENT specialists refer their patients for HBOT if there is no response to medical treatment. But there are also specialists who believe that HBOT has beneficial effects on prognosis if it is started without delay. The referring doctors might also have concerns about the cost for HBOT. When we look at the distribution of the cases in each year, there is an increase in the number of the referred cases every year. Of the total cases the percentage of the cases referred in the first year was 10.7; it was 13% in the second year and 18.8% in the third year. In our cases the time interval between the diagnosis and beginning of HBOT ranged between 3 and 67 days. Although there are some patients who applied to only one ENT center before HBOT, some patients applied to several centers (average 2 centers) before they were referred for HBOT. The response to the HBOT was better in the cases with relatively shorter time interval between the diagnosis and the onset of the treatment. But since the spontaneous recovery rate is higher in early phase of the disease, it is difficult to evaluate the effect of early HBOT on the prognosis, like the effect of other treatment modalities [3, 4]. The factors like diversity in timing of HBOT, the effects of the treatments done before HBOT, lock of consensus on the standards for patient selection, and different approaches in audiometric evaluations cause difficulties in comparing our result with studies in literature. Comparison might be easier if the cases can be grouped as the cases who receive HBOT together with the other medical therapies within the first 2 weeks after the diagnosis and the cases who received HBOT within 2–6 weeks when there is no response to medical treatment [15]. The data about the cases who received HBOT solely for the treatment of ISSNHL is very restricted in the literature. Out of 18 cases, Lamm et al. [23] observed complete recovery in 11 cases, and hearing gain greater than 20 dB in 6 cases. Nakashima et al. analyzed 1,614 cases and suggested that hearing gain was better if the HBOT was added to the conventional treatment in the first week of the diseases, when compared with the cases who received HBOT after the first week [24]. Bennett et al. made a meta-analysis and commented that HBOT was not effective in chronic phase of the disease [25]. Lamm et al. performed a literature survey about the cases who received HBOT with the diagnosis of SSNHL, acoustic trauma, and tinnitus. They reported that out of 4,109 cases, half of the cases who received the HBOT in the first 2-6 weeks had remarkable hearing gain (20 dB or greater), one-third had partial healing (10–20 dB), and 13% of cases had no response to the treatments. The complaints of tinnitus disappeared in 81.4% of these cases. They indicated that healing rate decreases when the treatment is delayed [23]. The timing of the early treatment that has beneficial effect on prognosis was described by Nakashima et al. as the first 2 weeks after the symptoms. But this period of time was longer in the large series of Lamm. In our country, the cases series are limited when compared with the studies from Germany and Japan, about HBOT for inner ear pathologies. Aslan et al. [12] administered HBOT in the first 5–6 days together with stellate ganglion blockage and medical treatment for ISSNHL, and showed that HBOT

increased the hearing gain twice as much. Topuz et al. [13] added HBOT to the other treatments in 2 weeks after the symptoms had started and they found substantial effects of HBOT on the hearing loses above 61 dB at frequencies of 250, 500, 1,000, and 4,000 Hz in 51 cases. In our study, more than half of our cases had hearing loss above 71 dB and 51.2% of the cases had 20 dB or more hearing gain. The data in the literature together with our results seem to support the efficiency of HBOT commenced in the early phase of ISSNHL.

Another important aspect of the treatment is the number of total HBOT sessions. In treatments predicted as an average of 18 sessions and completed on an average of 10.4 days, the excessive number of sessions positively affected the hearing gain. In clinical practices it is known that while some cases have beneficial effects of HBOT in a short term, other cases might need HBOT longer to achieve the good results. There are some studies reporting that the hearing gain increased by increasing the number of HBOT sessions, in the cases who started receiving HBOT after the medical treatment. But in the literature the number of the studies is limited [26, 27]. Since the data are limited, it is difficult to evaluate clinical experiences with the studies by meta-analysis [25]. The cost of HBOT is another factor influencing the choice between treatment and increasing the number of HBOT sessions. It makes sense to continue HBOT as long as there is hearing gain confirmed by audiometric controls.

Another purpose of this study was to evaluate the safety and side effects of HBOT in the treatment of ISSNHL. HBOT is regarded as a relatively benign intervention. HBOT is usually performed at about 2.5 ATA pressure as daily sessions. In some indications where the viability of the tissues is under hypoxic threat, the number of daily sessions might be increased. In general, if pressures do not exceed 3 ATA and the length of treatment is less than 120 min, HBOT is safe [17]. The central nervous system and pulmonary toxicity of oxygen are very rare side effects of HBOT. But middle ear, sinus, and dental barotraumas are relatively frequent. Visual disturbances, pareses in the fingers, confinement anxiety are the other side effects of HBOT. Visual disturbances due to conformational changes in lenses are seen after longer HBOT therapies, and it recovers spontaneously in most of the cases [17, 25, 28]. Claustrophobia, which appears to be present in about 2% of the general patient population, may cause some degree of confinement anxiety, even in a multiplace chamber. Occasionally, mild sedation is required for such individuals to continue to receive daily HBOT [17]. Barotrauma can affect any air-filled cavity in the body and occurs as a direct result of compression. Middle ear barotrauma is the most common side effect of HBOT. It can be prevented by teaching the patients how to equalize the pressure during compression. The equalization is possible with the active effort of the patients themselves. It will be difficult if there is congestion in the mucosa of the airways. Barotrauma is thus not a consequence of HBOT directly, but rather of the physical conditions required administer it [17, 25]. Fernau et al. [29] reported that complaints related to Eustachian dysfunction are seen 45% of 33 cases. In another study by Plafki et al. during 11,376 HBOT sessions employed to treat 782 patients with different indications, equalization problems were encountered in 17.8% of the cases in only one session, and usually it was the first session. They inserted a tube into the tympanic membrane in 12 cases and continued the HBOT. Thirteen cases refused to have such an invasive intervention. Sinus barotrauma was rare and there was no inner air or pulmonary barotrauma reported [28]. The low barotrauma rates in recent studies was attributed to several factors; anti-inflammatory and anti-edema effects of steroids used together with HBOT, slow compression procedures and otoscopic examination prior to HBOT sessions [15, 30]. Otoscopic examinations were applied to our cases both prior or after HBOT sessions and topical and/or systemic decongestant drugs prescribed together with slow compression if there is congestion or edema. In our study the middle-air barotrauma prevalence was lower than that of the previous studies. Attendance of an experienced hyperbaric technician, especially to the first HBOT session might be the reason for lower side effects. The patient may ignore active ear equalization because of anxiety in the first HBOT session.

Conclusion

ISSNHL is a potentially devastating condition with no definitive successful treatment regimen. HBOT has been used as an adjunctive for the treatment for about 40 years. But there is ongoing controversy about HBOT in the treatment of ISSNHL. However, ambiguous etiology and high rate of spontaneous recovery from the disease cause debates about the efficacy of other treatment modalities as well. The only valid means to demonstrate efficiency of HBOT would be to perform a placebo-controlled study; however, such a study seems to be impossible both practically and ethically. This retrospective study and our clinical experience suggest that HBOT has beneficial effects when administered in the early phase of the disease together with steroids. HBOT should be considered as a primary treatment, especially for the cases with total or profound hearing loss. HBOT is a safe practice when used properly by an experienced hyperbaric team. In the treatment of ISSNHL, 20 sessions of HBOT at 2.5 ATA can be tolerated well besides some minor side effects. In cases having good response to treatment with continuing hearing

gain, daily single sessions and frequent audiometric follow-ups may help to define the prolonged treatment protocols.

Conflict of interest statement The authors declare that they have no conflict of interest with any financial support or any commercial source that may, directly or indirectly, interfere with the results reported in the present article.

References

1. Byl FM (1984) Sudden hearing loss: eight years' experience and suggested prognostic table. Laryngoscope 94:647–662
2. Wilson WR, Byl FM, Laird N (1980) The efficacy of steroids in the treatment of idiopathic sudden hearing loss. A doubleblind clinical study. Arch Otolaryngol 106:772–776
3. Mattox DE, Simmons FB (1977) Natural history of sudden sensorineural hearing loss. Ann Otol Rhinol Laryngol 86:463–480
4. Simmons FB (1973) Sudden idiopathic sensori-neural hearing loss: some observations. Laryngoscope 83:1221–1227
5. Van Dishoeck H, Bierman T (1957) Sudden perceptive deafness and viral infection (report of the first one hundred patients). Ann Otol Rhinol Laryngol 66:963–980
6. Rasmussen H (1949) Sudden deafness. Acta Otolaryngol 37:65–70
7. Simmons FB (1968) Theory of membrane breaks in sudden hearing loss. Arch Otolarngol 88:41–48
8. Stokroos RJ, Albers FWJ (1996) The etiology of idiopathic sudden sensorineural hearing loss: a review of the literature. Acta Otorhinolaryngol Belg 50:69–76
9. Agarwal L, Pothier DD (2009) Vasodilators and vasoactive substances for idiopathic sudden sensorineural hearing loss. Cochrane Database Syst Rev 4:CD003422. doi:10.1002/14651858.CD003422.pub4
10. Lamm H (1968) Klinische Ergebnisse nach Behandlung von Innenohrschwerhörigkeiten mit hyperbaren Sauerstoff. Diskuss. Beitrag HNO-Jahreskongress der DDR, Halle
11. Appaix A, Demard F (1970) Hyperbaric oxygenotherapy and sudden perceptive deafness. Rev Laryngol Otol Rhinol (Bord) 91:951–972
12. Aslan I, Oysu C, Veyseller B, Baserer N (2002) Does the addition of hyperbaric oxygen therapy to the conventional treatment modalities influence the outcome of sudden deafness? Otolaryngol Head Neck Surg 126:121–126
13. Topuz E, Yigit O, Cinar O, Seven H (2004) Should hyperbaric oxygen be added to treatment in idiopathic sudden sensorineural hearing loss? Eur Arch Otorhinolaryngol 261:393–396
14. Narozny W, Kuczkowski J, Kot J, Stankiewicz C, Sicko Z, Mikaszewski B (2006) Prognostic factors in sudden sensorineural hearing loss: our experience and a review of the literature. Ann Otol Rhinol Laryngol 115(7):553–558
15. Horn CE, Himel HN, Selesnick SH (2005) Hyperbaric oxygen therapy for sudden sensorineural hearing loss: a prospective trial of patients failing steroid and antiviral treatment. Otol Neurotol 26(5):882–889
16. Samim E, Kilic R, Özdek A, Gocmen H, Eryilmaz A, Unlu I (2004) Combined treatment of sudden sensorineural hearing loss with steroid, dextran and piracetam: experience with 68 cases. Eur Arch Otorhinolaryngol 261:187–190
17. Feldmeier JJ (ed) (2003) Hyperbaric oxygen: indications and results; the hyperbaric oxygen therapy committee report. Undersea and Hyperbaric Medical Society, Kensington
18. Lamm K, Arnold W (1999) Successful treatment of noise-induced cochlear ischemia, hypoxia, and hearing loss. Ann N Y Acad Sci 884:233–248
19. Lamm K, Lamm C, Arnold W (1998) Effect of isobaric oxygen versus hyperbaric oxygen on the normal and noise-damaged hypoxic and ischemic guinea pig inner ear. Adv Otorhinolaryngol 54:59–85
20. Mazzoli M, Van Camp G, Newton V, Giarbini N, Declau F, Parving A (2003) Recommendations for the description of genetic and audiological data for families with nonsyndromic hereditary hearing impairment. Audiol Med 1:148–150
21. Goodman AC (1965) Reference zero levels for pure-tone audiometers. Asha 7:262–263
22. Ballesteros F, Alobid I, Tassies D, Reverter JC, Scharf RE, Guilemany JM, Bernal-Sprekelsen M (2009) Is there an overlap between sudden neurosensorial hearing loss and cardiovascular risk factors? Audiol Neurotol 14:139–145
23. Lamm K, Lamm H, Arnold W (1998) Effect of hyperbaric oxygen therapy in comparison to conventional or placebo therapy or no treatment in idiopathic sudden hearing loss, acoustic trauma, noise induced hearing and tinnitus. A literature survey. Adv Otorhinolaryngol 54:86–99
24. Nakashima T, Fukata S, Yanagita N (1998) Hyperbaric oxygen therapy for sudden deafness. Adv Otorhinolaryngol 54:100–109
25. Bennett MH, Kertesz T, Yeung P (2007) Hyperbaric oxygen for idiopathic sudden sensorineural hearing loss and tinnitus. Cochrane Database Syst Rev 1:CD004739. doi:10.1002/14651858.CD004739.pub3
26. Desloovere C, Germoupre P (1997) Sudden sensorineural deafness: treatment with hyperbaric oxygen therapy after failure of ten day course of "classical" drug therapy. In: Proceedings of the 23rd EUBS Congress. European Underwater and Baromedical Society, Bled, pp198–202
27. Welslau W, Almeling M, Lommerging A, Busch R, Trombitas G, Hesse G (1997) How many HBO treatments are necessary for the therapy of sudden deafness and acute tinnitus? In: Proceedings of the 23rd EUBS Congress. European Underwater and Baromedical Society, Bled, pp 203–209
28. Plafki C, Peters P, Almeling M, Welslau W, Busch R (2000) Complications and side effects of hyperbaric oxygen therapy. Aviat Space Environ Med 71:119–124
29. Fernau JL, Hirsch BE, Derkay C, Ramasastry S, Schaefer SE (1992) Hyperbaric oxygen therapy: effect on middle ear and eustachian tube function. Laryngoscope 102:48–52
30. Vahidova D, Sen P, Papesch M, Zein-Sanchez MP, Mueller PH (2006) Does the slow compression technique of hyperbaric oxygen therapy decrease the incidence of middle-ear barotrauma? J Laryngol Otol 120(6):446–449