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MAIN ARTICLE

Is hyperbaric oxygen therapy a salvage treatment option for sudden sensorineural hearing loss?

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Abstract

Objective: To investigate of the efficacy of hyperbaric oxygen therapy as a salvage treatment after unsuccessful oral corticosteroid therapy.

Methods: Case notes of patients who were followed up because of idiopathic sudden sensorineural hearing loss between 2005 and 2011 in a tertiary care centre were examined retrospectively. Audiograms from before and after hyperbaric oxygen therapy were examined in terms of mean gains in pure tone average and at 0.25, 0.5, 1, 2, 4 and 8 kHz. In addition, recovery according to Siegel's criteria was noted.

Results: Mean gain in pure tone average was 10.55 ± 13.56 dB. Mean gains at 0.25, 0.5, 1, 2, 4 and 8 kHz were 16.66 ± 18.43 dB, 16.94 ± 19.93 dB, 12.63 ± 16.71 dB, 7.36 ± 15.28 dB, 5.27 ± 11.58 dB and 2.91 ± 12.44 dB, respectively. Three patients had complete recovery, 1 had partial recovery, 5 had slight recovery and 25 had no improvement.

Conclusion: Hyperbaric oxygen utilised as a salvage therapy after failed corticosteroid therapy may be beneficial in some patients. Studies with more patients are needed.

Key words: Hearing Loss, Sudden; Hyperbaric Oxygenation; Salvage Therapy

Introduction

Sudden sensorineural hearing loss (SSNHL) is defined as a hearing reduction of 30 dB or more, in at least three consecutive frequencies, occurring within 3 days of symptom onset.[1] The cause is evident in about 10–15 per cent of cases, with 1 per cent having retrocochlear causes such as acoustic neuroma, demyelinating disease and stroke.[2,3]

Sudden sensorineural hearing loss affects 5–20 per 100 000 people per year.[4] The spontaneous remission rate has been reported as 45–65 per cent.[2,4] Vascular occlusion, viral infections, labyrinthine membrane breaks, immune-associated mechanisms and abnormal stress response in the cochlea have been proposed as mechanisms for idiopathic SSNHL.[5]

Many therapeutic modalities have been employed to treat the condition. Oral corticosteroids and intratympanic steroids are the most commonly preferred options. Hyperbaric oxygen is utilised as an adjunct first-line treatment in Europe. This study aimed to examine the usefulness of hyperbaric oxygen treatment for SSNHL after oral corticosteroid therapy failure in a retrospective case note study.

Materials and methods

Case notes of patients who were followed up because of idiopathic SSNHL between 2005 and 2011 in a tertiary care centre were examined retrospectively.

Patients

The criteria for SSNHL diagnosis were a hearing reduction of 30 dB or more, in three consecutive frequencies, within 3 days of symptom onset.[1]

Patients were included in this study if their mean pure tone averages (PTAs) improved by less than 15 dB HL or deteriorated after a 14-day course of oral corticosteroid therapy, and if they had received hyperbaric oxygen therapy as a salvage treatment after oral corticosteroid therapy.

Patients were excluded if their oral corticosteroid treatment was started more than 10 days after the onset of symptoms, or if hyperbaric oxygen treatment was started more than 35 days after the onset of symptoms. Patients were also excluded if: a cause for the hearing loss was evident, their hearing loss was bilateral, they were aged less than 12 years, or pre-treatment and post-treatment audiograms were not available.

The oral corticosteroid treatment protocol for SSNHL in our clinic was 1 mg/kg body weight of oral prednisolone or equivalent for 7 days, with a 10 mg taper every 2 days. Oral corticosteroid therapy lasted about 14 days. Hyperbaric oxygen treatment consisted of 20–30 sessions of 120 minutes at 2.5 atmospheres absolute pressure, over a treatment period of 14–21 days.

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TABLE I
REASONS FOR EXCLUSION

Reason	Cases (*n*)
Oral corticosteroid therapy start after 10 days	11
Hyperbaric oxygen treatment start after 35 days	10
Bilateral SSNHL	2
Aetiology found (acoustic tumour)	1
Patients aged <12 years	1
Pre- &/or post-treatment audiograms absent	8

SSNHL = sudden sensorineural hearing loss

Evaluation criteria

Pure tone average was calculated as the arithmetic mean of thresholds at 0.5, 1, 2 and 4 kHz. The PTAs before oral corticosteroid therapy, and before and after hyperbaric oxygen treatment were recorded. Treatment outcome was evaluated using: Siegel's criteria[6] before and after hyperbaric oxygen therapy, and mean PTA gain and gains at 0.5, 1, 2, 4 and 8 kHz (by subtracting post-hyperbaric oxygen therapy threshold values from pre-therapy threshold values).

Treatment outcomes according to Siegel's criteria can be categorised as follows: (1) complete recovery – final threshold of more than 25 dB; (2) partial recovery – gain of more than 15 dB, with a final hearing threshold of 25–45 dB; (3) slight recovery – gain of more than 15 dB, with a final threshold of more than 45 dB; and (4) no improvement – gain of less than 15 dB, with a final hearing threshold of more than 75 dB.[6]

A Wilcoxon signed rank test was used to compare mean values before and after salvage hyperbaric oxygen therapy. Statistical analysis was performed with the use of SPSS software (SPSS, Chicago, Illinois, USA). A p value of less than 0.05 was considered significant.

The protocol of this study was approved by the institutional review board.

Results

Thirty-six patients, all unilateral cases, were found to meet the criteria. Excluded patients and reasons for exclusion are shown in Table I. Of these 36 patients who met the criteria, 12 (33.3 per cent) were male and 24 (66.3 per cent) were female. Mean age was 41.42 ± 15.90 years (range, 12–75 years). The time to onset of oral corticosteroid therapy was 2.67 ± 2.91 days (range, 0–10 days). The time to onset of hyperbaric oxygen therapy was 19.19 ± 6.86 days (range, 11–35 days).

Audiogram characteristics before and after hyperbaric oxygen treatment are shown in Tables II and III. Mean gains after hyperbaric oxygen treatment, at each frequency, and PTA gain, are shown in Table IV and in Figure 1.

Of 3 patients who recovered completely, 1 did so after 30 sessions of hyperbaric oxygen treatment (mean gain of 51.25 dB). Another patient achieved complete recovery after 20 sessions (mean gain of 27.5 dB). A further patient achieved partial recovery after 10 sessions (mean gain of 36 dB), followed by

TABLE II
MEAN THRESHOLDS BEFORE HYPERBARIC OXYGEN TREATMENT

Parameter	0.25 kHz	0.5 kHz	1 kHz	2 kHz	4 kHz	8 kHz	PTA
Mean ± SD (dB) Range	66.38 ± 26.18 15–110	69.31 ± 26.70 20–115	71.25 ± 28.17 15–115	68.61 ± 31.27 10–120	70.69 ± 32.74 5–120	72.92 ± 30.13 10–110	69.97 ± 27.09 21–117

Pure tone average represents the mean of thresholds at 0.5, 1, 2 and 4 kHz. PTA = pure tone average; SD = standard deviation

TABLE III
MEAN THRESHOLDS AFTER HYPERBARIC OXYGEN TREATMENT

Parameter	0.25 kHz	0.5 kHz	1 kHz	2 kHz	4 kHz	8 kHz	PTA
Mean ± SD (dB) Range	49.72 ± 31.07* 10–110	52.36 ± 31.83* 5–110	58.61 ± 33.2* 5–110	61.25 ± 35.38* 5–110	65.41 ± 35.07* 5–110	70 ± 31.95 5–120	59.40 ± 31.63* 8–110

Pure tone average represents the mean of thresholds at 0.5, 1, 2 and 4 kHz. *$p < 0.05$ (compared with data collected before hyperbaric oxygen salvage therapy). PTA = pure tone average; SD = standard deviation

TABLE IV
MEAN GAINS AFTER HYPERBARIC OXYGEN TREATMENT

Parameter	0.25 kHz	0.5 kHz	1 kHz	2 kHz	4 kHz	8 kHz	PTA
Mean ± SD (dB) Range	16.66 ± 18.43 −20–55	16.94 ± 19.93 −15–75	12.63 ± 16.71 −10–70	7.36 ± 15.28 −20–65	5.27 ± 11.58 −20–40	2.91 ± 12.44 −25–35	10.55 ± 13.56 −15–51.25

Pure tone average represents the mean of thresholds at 0.5, 1, 2 and 4 kHz. PTA = pure tone average; SD = standard deviation

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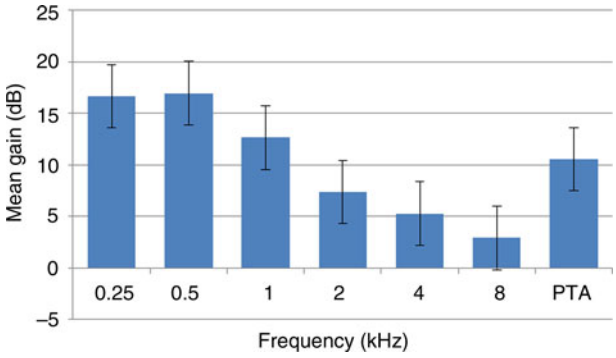

FIG. 1

Gain at each frequency and pure tone average (PTA) gain. The PTA represents the mean of thresholds at 0.5, 1, 2 and 4 kHz.

complete recovery after 20 sessions (mean gain of 45 dB).

Analysis

The thresholds at frequencies 0.25, 0.5, 1, 2, 4 and 8 kHz and the PTA values before and after hyperbaric oxygen salvage therapy were compared. The thresholds at 0.25 kHz ($Z = -4.125$, $p = 0.0$), 0.5 kHz ($Z = -4.166$, $p = 0.0$), 1 kHz ($Z = -4.090$, $p = 0.0$), 2 kHz ($Z = -2.786$, $p = 0.005$) and 4 kHz ($Z = -2.530$, $p = 0.011$), and PTAs ($Z = -3.957$, $p = 0.0$), were statistically significantly different.

Discussion

The cause of SSNHL is unknown in 85–90 per cent of cases.[2] Therefore, the treatment is empirical. The most commonly used treatments are oral corticosteroids, intratympanic corticosteroids and hyperbaric oxygen therapy. When primary treatment fails, intratympanic steroids or hyperbaric oxygen are generally offered if they were not administered as primary treatment.

Given the possibility that SSNHL is of ischaemic origin, oxygen may be a beneficial therapeutic option. Oxygen diffusion across the blood brain barrier reaching target tissues is the rationale behind hyperbaric oxygen treatment; furthermore, this treatment is well tolerated.[7,8] The purpose of hyperbaric oxygen therapy in SSNHL treatment is to increase partial oxygen pressure in blood and inner-ear fluids. Nagahara *et al.*, in 1983, measured perilymphatic oxygen tension through an electrode inserted in the perilymphatic space and found it to be decreased in some patients with SSNHL.[9] Kubo *et al.* suggested that decreased cochlear blood flow and subsequent capillary oedema may be a possible mechanism of cochlear hypoxia.[10] If 100 per cent oxygen is inhaled at 2 atmospheres absolute pressure, the amount of dissolved oxygen increases from 0.32 per cent at normal atmospheric pressure to 4.44 per cent. Hyperbaric oxygen treatment also decreases cyclo-oxygenase 2 and prostaglandin E2 production, and serves as an anti-inflammatory agent equivalent to 20 mg/kg diclofenac.[11]

Some previous studies have investigated the role of hyperbaric oxygen utilised as a salvage treatment after systemic corticosteroid therapy failure. In 2005, Horn *et al.* reported on nine patients who underwent hyperbaric oxygen salvage treatment after two weeks of corticosteroid and antiviral therapy.[12] Two patients improved dramatically and one patient improved in word recognition score only.

In 2010, Muzzi *et al.* reported on 19 patients who received 30 sessions of hyperbaric oxygen therapy after unsuccessful medical treatment.[11] Details of the medical treatment were not provided. Higher gains were observed in lower frequencies (mean gains of 13.42 dB, 11.84 dB, 8.95 dB, 6.05 dB, 8.42 dB and 3.16 dB at 0.25 kHz, 0.5 kHz, 1 kHz, 2 kHz, 4 kHz and 8 kHz, respectively). Recovery data were not presented categorically but as relative decreases in thresholds. Absolute mean gains were similar to our gain results.

In a prospective randomised study, Cvorovic *et al.* examined the effect of intratympanic steroids and hyperbaric oxygen therapy after unsuccessful medical treatment (6 days of systemic corticosteroid therapy).[13] Fifty patients received either 20 sessions of hyperbaric oxygen therapy or intratympanic dexamethasone. Both treatments were found to be effective in decreasing threshold values.

Yang *et al.* compared intratympanic steroid injection, hyperbaric oxygen treatment and both of these treatments combined in a retrospective controlled study comprising 103 patients.[14] Their pre-salvage treatment protocol consisted of systemic corticosteroid therapy for 10 days, plasma expanders and radiopaque contrast material application. Final PTA was measured two months after diagnosis. Patients received 10 sessions of hyperbaric oxygen treatment over 2 weeks. The authors reported that both individual salvage treatments and the combination therapy resulted in higher gains in PTA and increased word recognition scores than the control group. They defined gains in PTA that were higher than 15 dB as good recovery, and found that only 22.2 per cent of cases achieved good recovery in the control group compared with 45.5–68.4 per cent in the salvage treatment groups. The mean hearing gain of the hyperbaric oxygen salvage group was 17.4 dB while that for the control group was 7.4 dB.

Psillas *et al.* administered 15 sessions of hyperbaric oxygen therapy as a salvage treatment in 15 patients, and compared their outcomes to those of a control group of 30 patients.[15] The authors reported a greater ratio of improvements (46 per cent *vs* 13.3 per cent) and better PTA gains (12.1 dB *vs* 2.7 dB) in the hyperbaric oxygen therapy group compared with the control group.

Pezzoli *et al.* managed 23 patients with hyperbaric oxygen treatment and 21 patients with observation.[16] The treated patients had a better hearing gain (15.6 ± 15.3 dB *vs* 5.0 ± 11.4 dB). The authors also reported

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a better recovery rate with 15 sessions of hyperbaric oxygen treatment compared with observation only (1 of the treated patients completely healed, 5 had good recovery, 10 had fair recovery and 7 were unchanged, whereas 3 of the observed patients had good recovery, 1 had fair recovery and 17 were unchanged). Good recovery was defined as recovery in PTA within 15 dB of the unaffected ear. Fair recovery was defined as more than 10 dB recovery but out of the 15 dB range. Poor recovery was defined as a less than 10 dB increase in PTA.

Our patients had statistically significant decreases in thresholds at 0.25–4 kHz, and in PTAs, after hyperbaric oxygen treatment.

Mean gains were more prominent in lower frequencies (0.25 kHz and 0.5 kHz), approaching 17 dB. The higher gains obtained in the lower frequencies could mean that these frequencies are more responsive to hyperbaric oxygen salvage therapy. The basal turn of the cochlea has been proposed to be more vulnerable to damage and may therefore be less prone to recovery.

The gains at 1 kHz and PTA were just exceeding 10 dB (Table IV), which is the lower limit that it is suggested should be reported, as stated in the American Academy of Otolaryngology – Head and Neck Surgery clinical practice guideline for sudden hearing loss. However, using a 10 dB HL change in PTA has been considered worrisome because this magnitude of change is within test–retest reliability of measuring pure tone thresholds,[17] making the amount of change very close to 10 dB clinically unreliable.

Changes in Siegel recovery classification are another means of determining salvage treatment benefit. Three patients (8.33 per cent) had complete recovery, while one patient (2.7 per cent) had a more than 45 dB PTA gain and five patients (13.88 per cent) had a 15–45 dB PTA gain. Twenty-five patients (69.44 per cent) showed no improvement. None of the five total hearing loss patients in our group attained serviceable hearing that enabled them to use hearing aids.

Studies on salvage hyperbaric oxygen treatment administered after oral corticosteroid therapy failure report mostly mean gains, but categorical recovery is mentioned in a minority of studies.[12,14,16] We think that the latter is a more appropriate way of presenting findings.

- **Hyperbaric oxygen therapy can be used as salvage treatment for sudden sensorineural hearing loss**
- **The mean gain seems to be marginal, but some patients may have better recovery**

A study by Yeo *et al*. investigated 156 patients who received oral corticosteroids for 10 days (with a 5-day period for tapering), carbogen treatment and vasodilators.[18] The authors reported recovery of hearing (at least a 15 dB PTA gain) to their final hearing level after: 10 days in 66 cases (42.3 per cent), 10–38 days in 43 cases (27.56 per cent) and 38–120 days in 12 cases (7.69 per cent). The high rate of continuing recovery after treatment at this magnitude may have contributed to the recovery rates after salvage therapy reported in other studies. Therefore, we think that larger studies should be conducted that investigate the natural course of the SSNHL, with or without salvage treatment.

Even though the benefit reported in other studies is higher, we were able to find some significant recovery gain in the lower frequencies and some recovery in outcome according to Siegel's criteria. Thus, some patients may have benefited from hyperbaric oxygen treatment.

Conclusion

Hyperbaric oxygen salvage therapy utilised after corticosteroid therapy failure may be beneficial in some patients. Studies on the natural course of the disease after systemic corticosteroid therapy are needed, and further studies should comprise a greater number of patients.

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