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# Efficiency of hyperbaric oxygen and steroid therapy in treatment of hearing loss following acoustic trauma

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ABSTRACT

**Introduction:** Most commonly used treatment modalities for acute acoustic trauma (AAT) include steroid and hyperbaric oxygen ($HBO_2$) therapy. The aim of this study is to investigate the effectiveness of combined steroid and $HBO_2$ therapy in patients who develop AAT during firearms training and the effect of delay to treatment on treatment success.

**Materials and methods:** Patients admitted with the complaint of hearing loss after firearms training between January 2011 and April 2013 were evaluated retrospectively. Patients were grouped according to date of admission; patients admitted within the first 10 days were included in Group A and those admitted between Days 11 and 30 in Group B.

**Results:** A total of 48 patients (73 ears) with AAT were included. There were 37 ears in Group A and 36 ears in Group B. The number of ears with complete treatment response, partial treatment response and treatment failure (unchanged) were one (2.7%), 7 (18.9%) and 29 (78.4%) in Group A and 0 (0%), 3 (8.3%) and 33 (91.7%) in Group B, respectively. There was no statistically significant difference between the groups ($p=0.095$). Late-term results (at Week 6) demonstrated Group A showed higher hearing gain on high frequencies than Group B ($p<0.05$), but this result was not consistent with clinical outcome results.

**Conclusion:** The success rate of combined $HBO_2$ and steroid therapy was very low in our study. However, early initiation of treatment results in better outcomes. Protective measures have great importance in preventing AAT.

## INTRODUCTION

Acute acoustic trauma (AAT) is a sudden sensorineural hearing loss (SSNHL) due to exposure to acoustic overstimulation. Temporary or permanent hearing loss may occur after noise exposure [1]. Temporary hearing loss below 40 decibels (dB) recovers quickly, while hearing loss above 40 dB shows a delayed improvement. Over time, repeated temporary hearing loss that fails to recover completely can lead to permanent hearing loss. Severe inner ear damage, which occurs when the critical sound level intensity is exceeded, can also cause permanent hearing loss [2-4]. Several factors such as presence of impulse noise, exposure time, peak sound pressure, age and concomitant use of ototoxic drugs are important in the development of permanent hearing loss [3,4]. Firearms cause impulse noise when fired. Impulse noise exposure levels should not exceed 140-dB sound pressure levels (SPL) in any way. However, a rifle shot can produce an impulse noise up to 157 dB SPL [5,6]. Soldiers in the military service are exposed to impulse noise during firearms training. The simplest method for prevention of acoustic overstimulation is use of hearing protection. However, laws in many countries do not regulate conditions and obligations of hearing protection use [4]. The prevalence of noise-induced hearing loss among individuals using firearms

KEYWORDS: Acute acoustic trauma, sensorineural hearing loss, hyperbaric oxygen therapy, steroid therapy

is up to 40%-60% in the literature [6]. Hearing protection can provide an absolute dB reduction from 10 dB up to 41.2 dB although they are not frequency selective. Soldiers do not regularly wear hearing protections due to various reasons. The most common reason for not wearing hearing protection is to communicate with each other more easily. There are publications that report hearing cannot be adequately protected despite the use of hearing protection. Hearing protection is considered inadequate in the presence of symptoms like tinnitus and aural fullness [1,4,7,8]. In one study, full-time hearing protection was reported in only 5.3% among officers, and these officers felt they had only moderate or fairly good hearing protection [8]. Patients diagnosed with AAT in our study were not wearing hearing protection during firearms training because the firing instructors wanted to ensure that the instructions were heard properly. The firing instructors also wanted the recruits to get accustomed to loud rifle noises, as they would experience the same in a real battlefield.

Acoustic trauma causes hearing loss by two main mechanisms, which include mechanical and metabolic pathways. Inflammatory cytokines and free oxygen radicals are produced in the inner ear after exposure to sounds of high intensity in the metabolic pathway. Inflammatory cytokines and free oxygen radicals cause a reduction in cochlear blood flow and lead to hypoxia [9-12]. Severe acoustic overstimulation can cause loss and fusion mechanically of the inner ear sterocilia, loss of adjacent supporting cells and disruption of the organ of Corti [2]. There can be significant differences in severity of acoustic trauma-induced hearing loss even if they occur in the same environmental conditions and at the same sound level intensity. These differences may be due to individual susceptibility to noise [7].

Many treatment modalities have been tried for the treatment of AAT. Most commonly used treatment modalities include steroid therapy and hyperbaric oxygen ($HBO_2$) therapy. Some antioxidants have also been found to be beneficial. The best results in response to treatment were obtained in those who were started on steroid therapy as early as possible. The effects of early or late inductions of $HBO_2$ therapy on the results of treatment are still being studied [9,13]. The aim of this study was to investigate the effectiveness of combined $HBO_2$ and steroid therapy in patients who develop AAT after firearms training. We also aimed to investigate the effect of treatment delay on the effectiveness of the treatment.

## MATERIALS AND METHODS

Patients admitted to the otolaryngology service with complaint of hearing loss after firearms training between January 2011 and April 2013 were included in the study, and their records were evaluated retrospectively. 48 patients (73 ears), ages 25.8 ± 3.9 years (range: 21-36 years) were included in the study. All were male, and none of the patients had any history of hearing impairment before the AAT. Inclusion criteria were as follows:

- having sensorineural hearing loss due to AAT;
- having a detailed otolaryngological examination records and pure-tone audiometry measurements (values for 0.5, 1, 2, 4, 6, 8, 10, 12.5, 14 and 16 kHz) before, 10 days and 6 weeks after initiation of the treatment; and
- having combined steroid therapy and $HBO_2$ therapy.

Exclusion criteria were as follows:

- age under 18 years;
- intracranial malignancy:
- hypertension
- comorbid upper respiratory disease; and
- history of hearing impairment before firearms use.

Sudden hearing loss is a clinical emergency and defined as a 30-dB hearing level (HL) loss over three consecutive frequencies occurring within three days after exposure. However, there is no accepted threshold shift for AAT in the literature. In our study, the average hearing threshold of ≤20 dB HL at high frequencies (4, 6, 8, 10, 12.5, 14 and 16 kHz) was accepted as normal, whereas >20 dB HL was abnormal (AAT).

All patients were diagnosed with AAT, and treatment was initiated on the day of diagnosis. Three of the patients were admitted on Day 1, one patient on Day 3 and the rest of the patients on Days 5 to 30 after the AAT. The patients were grouped according to time of admission to hospital:

- Patients admitted to hospital within first 10 days of inciting event were included in Group A; and
- Patients admitted to hospital between Days 11 and 30 after the inciting event were included in Group B.

The patients were treated with $HBO_2$ therapy in

addition to steroid therapy (deflazakort 30 mg, Flantadin tb®, Sanofi Aventis, Scoppito (L'Aquila), Italy). Steroid treatment was started with 90 mg deflazakort, tapered 15 mg in three-day intervals and completed in 18 days.

Pantoprazol 40 mg (Panthec tb®, Sanovel, Istanbul, Turkey) was used in order to avoid adverse gastric effects of steroid therapy. $HBO_2$ therapy was carried out in a multiplace hyperbaric chamber (Galeazzi, Livorno, Italy). Each session lasted for 90 minutes at a pressure of 2.4 atmospheres daily, and the whole therapy period included at least 10 sessions. If an incomplete improvement was observed on pure-tone audiometry after 10 sessions, $HBO_2$ therapy continued up to 20 sessions.

The following otolaryngological examination and pure-tone audiometry were performed on all patients on Day 10 and Week 6. The clinical outcomes of patients' hearing recovery were classified into three grades:

- Grade 1: a complete recovery in which hearing restored to within ≤20 dB HL;
- Grade 2: partial recovery in which the average loss at the follow-up was improved by 10dB HL or more (≥10dB HL); and
- Grade 3: unchanged if there was a difference of less than 10 dB HL (≤10 dB HL) or deteriorated after treatment [14].

Treatment results between the groups were statistically analyzed.

The institutional review board approved the study protocol (date: 02.05.2013, protocol number; 2013-43). Data were analyzed by using IBM SPSS Statistics Version 20.0.0 (Chicago Inc., Illinois, U.S.). Chi-square test was used to compare the clinical outcomes of the patients' hearing recovery in early (Group A) and delayed (Group B) treatment groups (Table 1). Repeated measures ANOVA was used to compare Group A and Group B in terms of hearing improvement in each frequency after six weeks (Table 3). Descriptive statistical methods for univariate data analysis were used. A p-value less than 0.05 was accepted as statistically significant.

**Table 1. Degree of hearing recovery for the two groups at six weeks**

| groups | ears | complete (%) | partial (%) | unchanged (%) |
|---|---|---|---|---|
| Group A | 37 | 1 (2.7) | 7 (18.9) | 29 (78.4) |
| Group B | 36 | 0 | 3 (8.3) | 33 (91.7) |
| TOTAL | 73 | 1 (1.4) | 10 (13.7) | 62 (84.9) |

There was no statistically significant difference between the two groups (P= 0.095)

## RESULTS

A total of 48 patients diagnosed with AAT were included in this study. 23 of 48 patients had unilateral hearing loss, while 25 of 48 patients had bilateral hearing loss. 35 had a hearing loss on the right ear, and 38 had hearing loss on the left ear. There were 37 ears in Group A and 36 ears in Group B. None of the participants had used hearing protection devices during military firearms training. They presented with AAT after training, which included firing a G3 rifle (caliber 7.62 mm, Heckler & Koch®, Turkey) while lying in a recumbent position in an open field.

The average periods between the onset of AAT and initiation of the treatment was 7.44 ± 1.97 (SD) days in Group A and 18.86 ± 6.95 (SD) days in Group B. This delay was mainly due to patients' conceptions that their hearing would spontaneously improve over time. Complete recovery was achieved in only one ear (1.3%) and partial recovery in 10 ears (13%) in both groups. The number of ears with complete treatment response, partial treatment response and treatment failure (unchanged) were 1 (2.7%), 7 (18.9%) and 29 (78.4%) in Group A and 0 (0%), 3 (8.3%) and 33 (91.7%) in Group B, respectively (Table 1). There

**Table 3. Comparison of Group A and Group B at six weeks**

| Frequency (Hz) | 500 | 1000 | 2000 | 4000 | 6000 | 8000 | 10000 | 12500 | 14000 | 16000 |
|---|---|---|---|---|---|---|---|---|---|---|
| Group A vs. Group B | 0.257 | 0.316 | 0.223 | 0.080 | **0.016** | **0.047** | 0.144 | **0.013** | **0.039** | **0.015** |

The values marked in **bold** are statistically significant (P<0.05)

**Table 2. Average hearing outcome (±SD) for each frequency measured at admission, after 10 days of treatment and at 6 weeks.**

| frequency (Hz) | 500 | 1000 | 2000 | 4000 | 6000 | 8000 | 10000 | 12500 | 14000 | 16000 |
|---|---|---|---|---|---|---|---|---|---|---|
| Group A | 15.95[a] (10.46) | 17.57 (10.78) | 24.46 (19.1) | 50.54 (16.06) | 57.16 (17.82) | 51.08 (25.47) | 47.16 (23.85) | 50.81 (20.87) | 50.27 (14.76) | 46.22 (15.07) |
| | 14.19[b] (8.54) | 15.81 (9.24) | 21.62 (17.36) | 45.41 (16.97) | 50.68 (16.84) | 44.73 (24.29) | 43.38 (24.04) | 46.22 (21.03) | 46.62 (15.64) | 43.51 (15.41) |
| | 14.05[c] (8.57) | 15.68 (9.51) | 21.49 (17.48) | 44.05 (17.35) | 49.19 (16.22) | 43.24 (24.33) | 41.76 (23.07) | 45.41 (21.09) | 45.54 (15.54) | 42.43 (15.21) |
| Group B | 12.92[a] (9.52) | 14.03 (10.34) | 29.17 (20.13) | 56.94 (18.91) | 67.64 (17.79) | 67.5 (20.02) | 57.78 (25.73) | 55.56 (21.27) | 51.25 (15.69) | 45.97 (14.87) |
| | 12.5[b] (9.52) | 13.47 (10.34) | 28.33 (20.18) | 54.72 (19.71) | 65.83 (17.55) | 64.86 (20.89) | 55.97 (25.49) | 54.86 (21.79) | 50 (16.56) | 45.14 (15.33) |
| | 12.22[c] (9.6) | 13.33 (10.42) | 27.92 (20.23) | 53.61 (19.63) | 64.31 (18.83) | 64.03 (21.61) | 55 (25.27) | 54.17 (21.73) | 49.58 (16.75) | 44.72 (15.9) |

[a] initial audiogram (at admission) [b] 10 days after treatment [c] final audiogram (six weeks after treatment)

was no statistically significant difference between the two groups (P=0.095).

Average hearing outcomes of pretreatment, tenth day of treatment and six weeks after treatment are presented for each frequency in Table 2. Comparison of Group A and Group B in terms of average hearing outcomes at six weeks of treatment is presented in Table 3. There was greater recovery at 6, 8, 12.5, 14 and 16 KHz frequencies in Group A compared to Group B. These recovery rates were statistical significant, but not significant according to clinical outcomes of patients' hearing recovery grading system.

Bilateral myringotomy was performed in one patient because of Eustachian tube dysfunction on the seventh day of $HBO_2$ therapy; bilateral myringotomy and ventilation tube insertion were performed in one patient because of middle ear effusion, which developed after barotrauma in the $HBO_2$ chamber on the third day of $HBO_2$ therapy. All patients completed $HBO_2$ therapy. Grommet ventilation tubes were removed after $HBO_2$ therapy. All patients' tympanic membranes were intact in the control examination six weeks after admission.

## DISCUSSION

Temporary or permanent hearing loss may occur after noise exposure. Temporary hearing loss usually recovers within 24 hours [1,2]. Hearing loss in AAT usually develops in 3 kHz and higher frequencies, while 1- to 2-kHz frequencies are minimally affected [1,4,10]. The average hearing level between 1- and 2-kHz frequencies were 19.01 dB, and 53.99 dB in 3-kHz and higher frequencies in our study, in accordance with the literature. The impact of high sound energy on the inner ear leads to a loss of function of the outer hair cells and may be associated with a loss of supporting Deiter's cells. These damaged sensory cells are kept in a transitional phase over a period between regeneration and cell death. Theoretically, it is this transitional phase where therapy can make a difference [15]. The average periods between the onset of AAT and initiation of treatment were 7.44 days in Group A and 18.86 days in Group B in our study. So, there was at the very least a possibility of temporary hearing loss and spontaneous recovery in our patients. Because of that, we think that our hearing recovery results were not affected by erroneous hearing recovery results caused by spontaneous recovery.

There are number of treatment methods used in the treatment of AAT. Fetoni, et al. demonstrated in a rat model that antioxidants can restore cortical neuronal morphology and hearing functions in noise-induced hearing loss [16]. Rhee, et al. showed that low-level laser irradiation facilitates the recovery of hearing thresholds after AAT in rats [17]. In general, effective treatment methods should focus on reduction of cellular

hyper-reaction, removal of oxygen free radicals, repair of the impaired microcirculation and elimination of hypoxia. The most accepted treatments are $HBO_2$ therapy and steroid therapy among currently used treatments [10,13]. Studies have shown that there are steroid receptors in the inner ear. It has been proposed that steroids have their effect through these receptors by maintaining ion balance in the inner ear, stabilizing the cellular membrane, enhancing perfusion and inhibiting local proinflammatory cytokines [13,18].

$HBO_2$ therapy is inhalation of 100% oxygen at a pressure greater than normal atmospheric pressure. In general, $HBO_2$ therapy is given at pressure of 2.0-2.8 atmospheres for 60-90 minutes and usually once daily for 10-15 days [19]. $HBO_2$ increases the amount of oxygen dissolved in the blood and carried to tissues. Lamm and Arnold showed reductions in parameters of partial oxygen pressure in the perilymph, amplitudes of the cochlear microphonics and cochlear nerve action potentials within the first 24 minutes after AAT in guinea pigs, and an immediate increase was observed in these parameters just after $HBO_2$ therapy [13].

Ylikoski, et al. demonstrated that the average recovery of hearing and cessation of tinnitus were significantly better in the $HBO_2$ therapy group than the normobaric oxygen therapy group. The delay periods from the exposure to the initiation of the treatment were <48 hours in their study [19]. We think that the short delay period is the main reason of the recovery rate in that study compared to our study.

Colombari, et al. used the electrophysiological brain stem auditory-evoked potential to investigate the effectiveness of $HBO_2$ alone in AAT but did not demonstrate effectiveness of $HBO_2$ alone [20]. d'Aldin, et al. proposed that $HBO_2$ can cause a dangerous situation when used alone. Cochlear damage was greater than for the controls: The inner cells and all three rows of the outer hair cells were heavily damaged when $HBO_2$ was administered within one hour. The greatest damage was observed at the first row of the outer hair cells and at the row of the inner hair cells. They concluded that $HBO_2$, when administered alone and within one hour, worsened hearing levels [21].

Liu, et al. indicated that the addition of $HBO_2$ therapy to the conventional steroid therapy in patients with sudden sensorineural hearing loss (SSNHL) provided better hearing outcomes [14]. Fakhry, et al. compared $HBO_2$ therapy alone and steroid therapy alone with a control group in adult guinea pigs after acoustic trauma exposure. They reported that hearing results of animals treated with $HBO_2$ alone or steroid therapy alone did not differ from the control group. They also compared the $HBO_2$ therapy and steroid therapy combination with the control group. An $HBO_2$-and-steroid therapy combination was administered one day and six days after acoustic trauma exposure, and the results were compared. The combination treatment provided significant protection from noise-induced loss of auditory thresholds, especially when started one day after the exposure [22].

Harada, et al. reported that the time from noise exposure to presentation was longer in patients with unchanged hearing despite treatment. Recovery of hearing was poorest at 4 kHz, followed by 8 kHz and 2 kHz. They concluded that hearing in patients with AAT is likely to return to normal when the hearing level at 4 kHz recovers gradually; partial recovery of hearing is expected when the hearing level at 4 kHz reaches an early plateau [23].

In their systemic review van der Veen, et al. studied the effect of $HBO_2$ therapy on hearing thresholds in patients who suffered a recent AAT. They were able to find only three eligible original articles for the data pool. The mean dB of hearing recovery in these studies ranged from 17 dB to 47 dB in the groups treated with $HBO_2$ therapy vs. 5 dB to 46 dB in the groups who did not receive $HBO_2$ therapy. They found that most of the studies were not randomized – especially problematic since hearing loss caused by AAT may show partial spontaneous improvement because of a temporary threshold shift and improvement over time. Second, the studies were difficult to compare. $HBO_2$ therapy protocols and follow-up differed to a great extent, and the results were not presented in the same manner. Some of the studies summed the results for all or a part of the frequencies together, thereby showing a statistical effect, without thought for the clinical relevancy. Other studies described a significant effect without presenting the data. Because of these methodological flaws, pooling of the study results was not feasible. According to original articles that van der Veen, et al. found eligible for the review, it remains uncertain

what the clinical significance is of the studies that did report a significant effect of $HBO_2$ therapy on hearing improvement in patients with AAT. The highest effect reported was 15 dB additional improvement in the $HBO_2$-treated group, but other studies did not find any additional effect. Because of the contrary results of the reported studies, van der Veen, et al. conclude that the effect of $HBO_2$ therapy on hearing thresholds in patients with hearing loss caused by a recent AAT remains unclear [15].

We found only one study similar to ours in the literature. In this study Lafere, et al. compared medical therapy (consisting of methylprednisolone and piracetam) alone with the combination of $HBO_2$ therapy and medical therapy in patients diagnosed with noise-induced hearing loss and obtained better hearing results in the combined therapy group. For these patients, the delay in transfer to the Military Hospital was less than 43 hours (six to 43 hours) [24].

$HBO_2$ therapy can cause some side effects such as middle-ear effusion due to barotrauma. When $HBO_2$ is combined with steroid therapy, steroids can also help to prevent serous otitis media as a result of $HBO_2$ therapy by reducing vascular permeability, edema and inflammation [14]. In our study, there was only the combined $HBO_2$ and steroid therapy group. This is because our study was retrospective, and our clinic's routine treatment protocol for patients with AAT is combined $HBO_2$ and steroid therapy. Because of this, we did not create such groups as an $HBO_2$-only, steroid therapy-only alone or a no-treatment group.

The question as to when to initiate $HBO_2$ therapy in AAT remains a controversial issue. It is reported that $HBO_2$ therapy, especially when applied immediately after an acoustic trauma, initially increases reactive oxygen species. At 30 minutes, reactive oxygen species reach the maximum level and cause cellular damage through lipid, protein and DNA oxidation [9,10,21,25].

d'Aldin, et al. demonstrated that $HBO_2$ therapy was very effective in improving hearing thresholds when combined with conventional steroid therapy in SSNHL [21]. Cakir, et al. studied frequency ranges most affected by AAT using distortion product otoacoustic emission (DPOAE) and identified that the range of 6-8 kHz was the most affected frequency range. They also worked to find the correct time to initiate $HBO_2$ therapy after AAT by comparing pre-exposure and post-exposure DPOAE values. Groups that received $HBO_2$ therapy within the first one hour and two hours showed no differences between pre-exposure and post-exposure DPOAE values, while there were significant DPOAE value improvements in groups that initiated $HBO_2$ therapy within the first six hours, 24 hours and 48 hours. They suggested that the best interval to start $HBO_2$ therapy for AAT is between six to 48 hours after the AAT [10].

Arslan, et al. reported that the most effective method of treatment in noise-induced hearing loss is early administration of high-dose dexamethasone. They also reported that $HBO_2$ therapy started within the first three hours could cause adverse effects such as increase of reactive oxygen metabolites, noting that the optimal time to initiate $HBO_2$ therapy was 24 hours after the exposure [9]. Psilla, et al. achieved the best results with steroid and piracetam therapy within the first hour after AAT; 65% had complete recovery, and 10% were unchanged. Success rates were decreased in the groups that initiated treatment at least 24 hours after AAT; only 13.3% had complete recovery, and 53.3% were unchanged [26].

Overall, the literature supports that steroid therapy should be started as early as possible, while $HBO_2$ therapy should not be begun earlier than the first six hours. In our study, combined $HBO_2$ and steroid therapy was initiated one day at the earliest after exposure and at the latest one month after exposure according to the patients' admission date. Group A (patients admitted to the hospital within the first 10 days) and Group B (patients admitted to the hospital between Days 11 and 30) were compared six weeks after admission: There was greater recovery at high frequencies (6, 8, 12.5, 14 and 16 KHz) in Group A compared to Group B. There was no significant difference at low frequencies. The reason for this might be that AAT has a minimal effect on hearing levels in low frequencies (0.5, 1, 2 kHz).

There was no statistically significant difference between the two groups in terms of the clinical outcomes of patients' hearing recovery. Only one ear (2.7%) fully recovered, seven ears (18.9%) partially recovered, and there was no recovery in 29 ears (78.4%) in Group A. In Group B three ears (8.3%) partially recovered, and there was no recovery in 33 ears (78.4%). When both

groups were evaluated together, only one ear (1.4%) fully recovered, 10 ears (13.7%) partially recovered, and there was no recovery in 62 ears (84.9%).

It is well known that the use of steroids carries a number of possible side effects. Based on our findings, we think that routine use of steroid therapy and $HBO_2$ therapy should be carefully reconsidered in patients who are admitted late after AAT. Further clinical studies are needed to determine the optimal initiation time for $HBO_2$ therapy, and also to compare the effectiveness of $HBO_2$ therapy alone, steroid therapy alone and no treatment in a larger number of patients.

## CONCLUSION

Early initiation of $HBO_2$ and steroid therapy leads to higher hearing gain in high frequencies. However, the clinical outcomes of patients' hearing recovery are very low in our study. Additional therapy beyond the first 10 days has demonstrated no significant contribution to the treatment. Therefore, protective measures should be taken to prevent the occurrence of AAT.

Policy-makers should make it mandatory to use hearing protection in firearms use and work develop ways to implement these laws to prevent permanent hearing loss as a result of firearms use. When individuals develop hearing loss after using firearms, they should be instructed to admit themselves to a hospital without waiting for spontaneous resolution of hearing loss.

**Conflict of interest**

The authors report no conflict of interest with this submission. ■

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