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CASE REPORT

Hyperbaric oxygen in the treatment of sudden deafness

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Abstract Currently, the treatment of sudden deafness (SD) is based mainly on complete bed rest and the administration of corticosteroids. Hyperbaric oxygen therapy (HBOT) has previously been suggested as adjunctive treatment. We describe two cases of successful HBOT for SD. The first patient presented with moderate mid-frequency hearing loss without accompanying symptoms, whereas the second patient had moderate low-frequency hearing loss with persistent tinnitus and a single episode of vertigo. HBOT in addition to conventional treatment soon after diagnosis resulted in full recovery of hearing in both patients. The pathogenesis of SD may involve a reduction in cochlear blood flow and perilymph oxygenation, making early HBOT a reasonable treatment modality for this condition.

Keywords Hyperbaric oxygen therapy · Sudden deafness

Introduction

The pathophysiology of sudden deafness (SD) is still obscure, although reduced cochlear blood flow and a decrease in perilymph oxygenation have both been documented [5, 11]. The improvement in perilymph oxygenation produced by HBOT provides a logical basis for the use of this treatment modality in SD. Despite this, if HBOT is provided at all, it is administered as adjuvant therapy only when other treatment modalities have failed. We report two cases of SD for which HBOT was given in parallel with conventional treatment soon after diagnosis was reached.

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Case reports

Case no. 1

A previously healthy, 49-year-old Caucasian man was admitted to the hospital due to acute hearing loss and a complaint of increased pressure in his left ear. The symptoms had appeared on arousal that day, 3.5 h before presentation. A complete otolaryngologic physical examination revealed no abnormalities. Audiometry demonstrated a left sensorineural hearing loss of 35 dB at 750 Hz, 40 dB at 1,000 Hz, 55 dB at 1,500 Hz, 50 dB at 2,000 Hz, and 40 dB at 3,000 Hz. Speech reception threshold (SRT) was 35 dB and discrimination score 100%. A diagnosis of SD was made and 30 mg of prednisone twice daily was commenced. HBOT was initiated 3.5 h after the patient's symptoms had first appeared. The HBOT protocol consisted of 100% oxygen breathing at 2.5 atmospheres absolute (ATA) for 90 min. After the treatment, the patient reported improved hearing and relief of the sensation of pressure in his left ear. A follow-up audiogram showed pure tone thresholds of 15 dB at 750 Hz, 15 dB at 1,000 Hz, 30 dB at 1,500 Hz, 50 dB at 2,000 Hz, and 30 dB at 3,000 Hz in the left ear. SRT was 20 dB and discrimination was 100%. Due to the residual hearing loss, a second HBOT session was given 6 h later using the same protocol, with further subjective improvement in hearing. Audiometry demonstrated complete resolution of the hearing loss.

Case no. 2

A 17-year-old Caucasian girl presented with a 4-day history of decreased hearing and a sensation of increased pressure in her right ear accompanied by tinnitus. A single episode of true vertigo was reported. The patient had a history of

bronchial asthma and allergic rhinitis, treated with fluticasone propionate inhalation 500 mcg twice daily. A complete otolaryngologic physical examination revealed no abnormalities. Audiometry showed a sensorineural hearing loss of 60 dB at 250 Hz, 55 dB at 500 Hz, and 25 dB at 750 Hz. SRT was 15 dB and discrimination was 100%. A diagnosis of SD was made and 30 mg of prednisone twice daily was prescribed. HBOT was recommended after chest auscultation, X-ray and spirometry were normal. The patient was treated with 100% oxygen at 2.5 ATA for 90 min. After the first treatment, the sensation of increased pressure disappeared, whereas the tinnitus persisted albeit to a lesser degree. Audiometry showed bilateral normal hearing.

Discussion

Sudden deafness is defined as a hearing loss greater than 30 dB at three contiguous frequencies, occurring over a period of less than 3 days [5]. It has an incidence of 5 to 20 per 100,000 persons per year [11, 15]. Tinnitus and a sensation of increased pressure are often present, whereas vertigo is less commonly associated with this syndrome. The possible aetiology includes viral and bacterial infections, vascular occlusion, cochlear membrane breaks, autoimmune neurologic disorders, neoplasms, ototoxic drugs, and psychogenic causes. However, the majority of cases elude precise aetiologic determination. Spontaneous recovery varies from 30 to 60%, usually taking place within the first weeks [2, 3, 6, 13]. Treatment is based mainly on corticosteroids, which appear to produce better results compared with the natural history of the disease [6, 9]. The reduction in cochlear blood flow and perilymph oxygenation plays an important role in the pathophysiology of SD [10, 16].

HBOT is a treatment modality in which the patient breathes oxygen at an ambient pressure greater than one atmosphere. Animal studies have shown that breathing 100% oxygen at 2.5 ATA increases the PO_2 in the perilymph by more than 450% [7]. Considering the central role of hypoxia in the proposed pathophysiology of SD on the one hand, and on the other, the effects of HBOT on perilymph oxygenation, there would appear to be a logical basis for using this treatment modality in SD. Nevertheless, HBOT is still not generally recommended for the treatment of SD. When provided, it is considered mainly for refractory cases when conventional treatment has failed [8, 12]. A study by Aslan et al. [1] showed that the addition of HBOT to conventional treatment modalities significantly improved final hearing levels in SD, especially in patients younger than 50 years. The delay from diagnosis to the commencement of HBOT is of great importance. Kau et al. [4] showed that the effectiveness of HBOT in SD decreases with increasing duration of the illness. Another study [14] recommended 10–15 HBOT sessions according to the response. The authors noted hearing improvement in 25.6 and 32.6% of patients after the tenth and fifteenth treatments, respectively.

The two patients reported here, both under 50 years of age, presented with moderate hearing loss, at mid to high frequencies in one patient and low frequencies in the other. HBOT was given soon after the diagnosis was made, in parallel with the commencement of corticosteroid treatment. The HBOT administered to our patients differs from the accepted mode of therapy. HBOT was started early, and was not kept in reserve in case of failure of the primary treatment. The reasoning behind this was the assumption that hypoxia is of great importance in the pathophysiology of SD, and should be resolved as early as possible if further damage to inner ear organs is to be avoided. The decision to stop treatment sessions after the patients regained normal hearing is based on our policy, which states that HBOT is to be ended whenever full recovery has been achieved or when two consecutive treatments show no improvement. Definite conclusions about the place of HBOT in the treatment of SD cannot be drawn on the basis of these two case reports. However, the logical basis for early HBOT and the favorable results described in this paper warrant further investigation.

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