Case reports

Hyperbaric oxygen therapy as salvage treatment for post-traumatic sudden sensorineural hearing loss: a case report

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Abstract
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Introduction: We report the first case of hyperbaric oxygen therapy used to treat sensorineural hearing loss in a child after head trauma.
Case report: A 13-year-old boy with no relevant past medical history presented to the emergency department with tinnitus and hypoacusia following head trauma. An ear computed tomography scan showed a right longitudinal temporal fracture sparing the otic capsule, and the audiogram identified a moderate sensorineural hearing loss in the right ear involving frequencies between 2,000 and 8,000 Hz. He was treated with corticosteroids and betahistine for an acute audiovestibular loss with resolution of the vestibular symptoms. At three months post-trauma the sensorineural hearing loss persisted. The patient started treatment with hyperbaric oxygen therapy with complete resolution of the hearing loss after 11 sessions.
Conclusions: This case identifies potential benefit from salvage hyperbaric oxygen therapy in the treatment of sudden sensorineural hearing loss of traumatic etiology.

Introduction

Post-traumatic sudden sensorineural hearing loss (SSNHL) accounts for approximately 4.2% of all SSNHL cases, with causes including head trauma, barotrauma, and acoustic trauma.[1] In cases of head trauma, SSNHL is mostly attributed to temporal bone fractures, which cause indirect damage to the cochlea or cochlear nerve. However, other mechanisms, such as labyrinthine concussion or direct damage to the central auditory pathways, have also been identified.[2] Despite advances in understanding the pathophysiology of post-traumatic SSNHL, specific treatment guidelines remain unclear, and management is often individualised.

Hyperbaric oxygen therapy (HBOT) is a well-established treatment for sudden sensorineural hearing loss (SSNHL), especially in cases refractory to corticosteroid therapy.[3] However, its role in post-traumatic SSNHL remains poorly defined, particularly when temporal bone fractures are absent or when the otic capsule is spared. This case report explores the first use of HBOT as a salvage treatment for post-traumatic SSNHL in a paediatric patient, providing valuable insights into the potential efficacy of this therapeutic modality for trauma-induced hearing loss.

Case report

The patient's legal guardian gave written consent for publication of this case history.

A 13-year-old male was admitted to the emergency department of a tertiary hospital after a right temporal head trauma with loss of consciousness during a football game. Upon regaining consciousness, he reported tinnitus and hypoacusia in the right ear, along with severe holocranial headache. He had no relevant past medical history, with no known drug allergies and up-to-date immunisations.

On admission, he was haemodynamically stable, afebrile, Glasgow Coma Score (GCS) 15, and had no neurological deficits on examination. Physical examination revealed erythema in the right periauricular area and haemotympanum

with an intact tympanic membrane on the right. Acumetric testing with 256 Hz and 1,024 Hz tuning fork revealed a Weber test lateralising to the right and an absent Rinne on the same side. Audiological testing was not performed at admission.

A computed tomography (CT) scan of the ear (Figure 1) showed a right longitudinal temporal fracture extending superiorly to the external auditory canal and temporal squama (without misalignment). The fracture crossed the mastoid cells and extended to the epitympanic lateral wall, sparing the otic capsule. A tissue opacity was observed in the ipsilateral posterior and inferior mastoid cells, as well as in the recesses of the posterior wall of the tympanic cavity and adjacent to the oval window, which was interpreted as bleeding.

The patient was admitted to the paediatric special care unit and treated with fluids, analgesics, and intravenous amoxicillin-clavulanic acid (50 $mg \cdot kg^{-1} \cdot dose^{-1}$). Forty-eight hours later, he developed rotational vertigo accompanied by nausea and vomiting, along with subjective worsening of the right-sided hypoacusia. Examination revealed a grade II horizontal nystagmus to the left and a pathological head impulse test (HIT) to the right. Follow-up CT scan showed near-complete reabsorption of blood in the middle ear and the audiogram demonstrated moderate sensorineural hearing loss (SNHL) on the right side, involving frequencies between 2,000–8,000 Hz (Figure 2). Prednisolone (1 $mg \cdot kg^{-1}$ for 15 days) and betahistine (24 mg every 12 hours for one month) were prescribed for a presumed post-traumatic inner ear irritative process. The patient was discharged three days later with resolution of the vertigo but no improvement in hearing.

At three months follow-up, the patient continued to report right-sided hypoacusia. The audiogram did not show significant changes (Figure 3) and videonystagmography testing was normal. HBOT was recommended and initiated three months post-trauma, consisting of 11 sessions at 253 kPa (2.5 atmospheres absolute) with 100% oxygen for approximately 70 minutes per session. Following HBOT, the patient reported complete symptom resolution, and an audiogram revealed complete recovery of SNHL (Figure 4).

Figure 1
Axial CT scan image demonstrating a right longitudinal temporal bone fracture (white arrow)

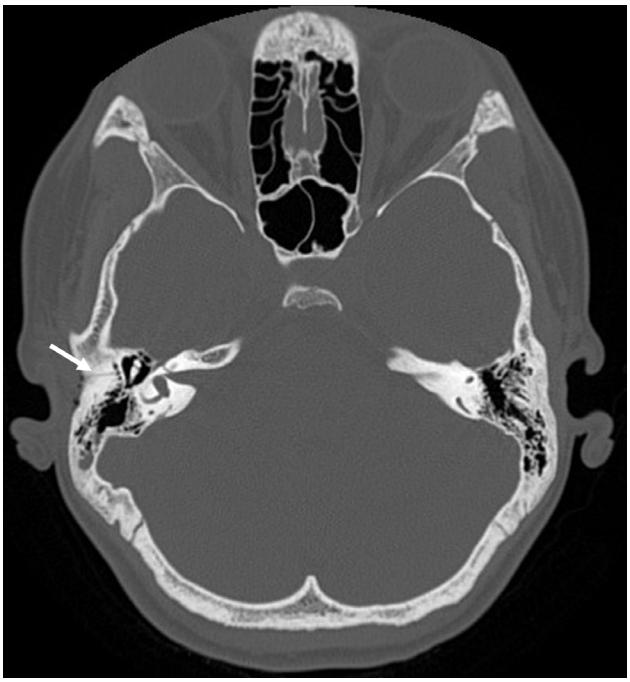
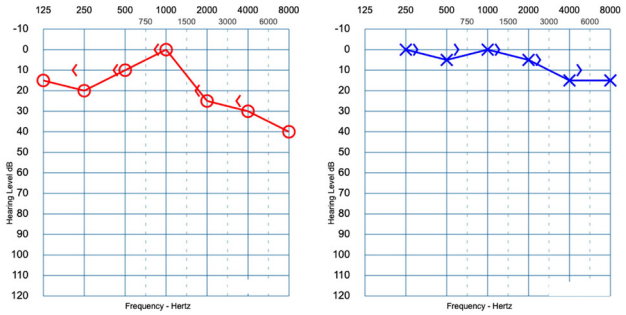


Figure 2
Pure tone audiometry showing moderate sensorineural hearing loss in the right ear (red, open circles)



Discussion

We describe a case of post-traumatic sudden sensorineural hearing loss (SSNHL) in which complete recovery after a three-month period with no improvement was associated with HBOT. To the best of our knowledge, this is the first report demonstrating potential efficacy of HBOT for sensorineural hearing loss of traumatic etiology.

Clinical studies have shown that head trauma can cause hearing loss in 15% to 66% of adults.[4] Temporal bone trauma resulting from head injury can lead to hearing loss with or without the presence of a temporal bone fracture. Since the 1940s, such fractures have been classified based on their angle relative to the petrous ridge into longitudinal, transversal or mixed (comminuted or oblique) types.[5] Longitudinal fractures tend to be associated with ossicular chain disruption and subsequent conductive hearing loss, whereas transverse fractures more commonly led to sensorineural hearing loss due to trauma to the labyrinth.[6,7] More recently, classification into otic capsule violating versus otic capsule sparing fractures has proven to be more informative in terms of prognosis, with capsule violating fractures linked to higher incidence of SSNHL, nerve disruption, cerebrospinal fluid fistula, facial nerve paralysis and intracranial complications.[8–10]

Our patient had an otic capsule sparing longitudinal fracture, which likely accounted for the immediate hearing loss (conductive, as indicated by acumetric testing), most probably caused by the haemotympanum. However, it

Figure 3
Pure tone audiometry three months after the event, showing no improvement in the moderate sensorineural hearing loss in the right ear (red, open circles)

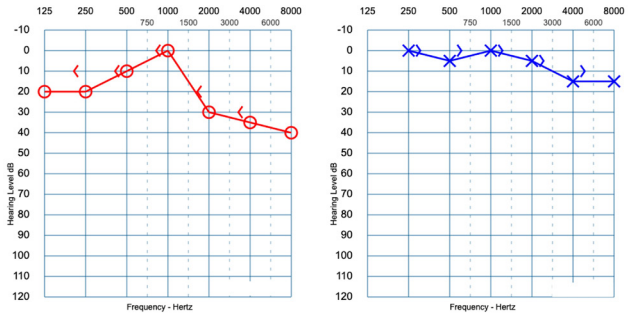
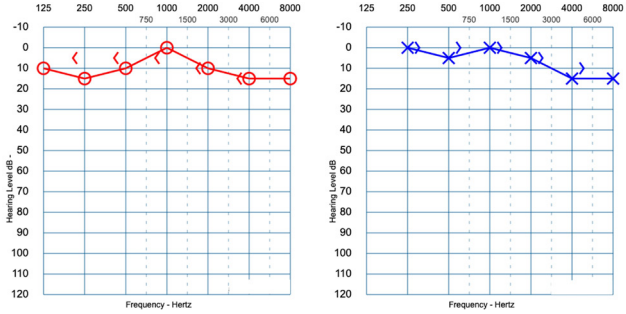


Figure 4
Pure tone audiometry showing complete recovery of sensorineural hearing loss after HBOT



is noteworthy that vertigo and worsened hearing loss (sensorineural, as confirmed on the audiogram) developed 48 hours post-injury, rather than immediately. This delayed onset can be explained by secondary audiovestibular loss, likely due to a labyrinthine concussion mechanism. Haemorrhage caused by a microfracture of the bony labyrinth could infiltrate the peri or endolymphatic spaces, leading to neuroepithelial degeneration and causing a labyrinthine concussion. In such cases, vestibular symptoms tend to cease over days as anatomic lesions heal and central balance mechanisms are reestablished, while hearing loss often persists.[11,12] Another possible explanation for this case would be a traumatic perilymphatic fistula, where tension-induced pressure transmission through the cochlear aqueduct or internal auditory canal would lead to oval window rupture. In such cases, diagnostic testing often appears normal, and a definitive diagnosis requires surgical exploration of the middle ear.[13]

Regarding treatment, initial management with corticosteroids and antibiotics followed common clinical practice. The use of HBOT as salvage therapy was proposed after persistent hearing loss at three months. The risks discussed with the patient and family included middle ear barotrauma, sinus and paranasal sinus barotrauma, ocular side effects, oxygen-induced seizures and claustrophobia.[14]

Clinical HBOT is defined as placing a patient in an increased pressure environment and having them inhale 100% oxygen for a defined period per treatment.[15] Studies have proved the efficacy of HBOT in treating idiopathic SSNHL, especially when combined with steroids, showing better outcomes in patients with severe or profound hearing loss (≥ 70 dB).[16,17] Furthermore, a 40-year review of research provided strong evidence supporting the use of HBOT for acute severe traumatic brain injury by reducing brain ischaemia protecting the neurovascular system.[18] However, its role in treating post-traumatic SSNHL remains less defined, particularly in instances where temporal bone fractures are absent or the otic capsule is spared.

Moreover, HBOT's efficacy is time-sensitive, with results diminishing as treatment is delayed, making it recommended to be started within 48 hours of diagnosis in idiopathic SSNHL.[19] Our case suggests a potentially wider therapeutic window for post-traumatic cases, possibly benefiting patients unable to receive early treatment, such as those admitted to intensive care units.

It is important to highlight that, given the natural course of sensorineural hearing loss, spontaneous recovery cannot be ruled out as the cause of hearing improvement in this case. Studies suggest that approximately 32% to 65% of patients with sudden SSNHL experience spontaneous recovery, with the majority of these improvements occurring within the first two weeks following onset.[20] Therefore, the causal relationship between HBOT and hearing recovery remains speculative and cannot be definitively proven. Only prospective randomised controlled trials with adequate sample sizes can confirm the efficacy of HBOT in post-traumatic SSNHL.

Conclusions

This case highlights the potential of HBOT as an effective salvage treatment for post-traumatic SSNHL. Additionally, it challenges the previously assumed strict time window for HBOT, suggesting that delayed intervention may still provide meaningful benefits. Future studies are necessary to establish clear treatment protocols and expand the understanding of HBOT's therapeutical potential in managing trauma-induced hearing loss.

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