673-014

CLINICAL CASE REPORT

# Inner ear decompression sickness in a hyperbaric chamber inside tender: a case report

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## ABSTRACT

Inner ear decompression sickness (IEDCS) is a rare diving complication that presents with vestibular dysfunction, cochlear dysfunction, or a combination of both. While scuba diving is a known cause, no cases have been reported in the occupational hyperbaric setting.

We present the case of a 55-year-old man who developed IEDCS after working as a hyperbaric multiplace chamber inside tender. The patient was treated with seven sessions of hyperbaric oxygen therapy, resulting in resolution of the majority of his symptoms. This case illustrates a potential occupational hazard of working in a hyperbaric chamber and demonstrates successful treatment with hyperbaric oxygen therapy.

## BACKGROUND

Inner ear decompression sickness (IEDCS) is a rare complication of scuba diving. Clinical manifestations of IEDCS include vertigo, nausea, nystagmus, tinnitus and hearing loss [1,2]. The etiology is not completely understood but is thought to involve precipitation of gas bubbles in endolymphatic and perilymphatic spaces. Rapid identification of IEDCS is important, as early hyperbaric oxygen ($HBO_2$) therapy can help prevent permanent injury [3,4]. We present a case of IEDCS involving a hyperbaric multiplace chamber inside tender at an academic tertiary care hospital.

Hyperbaric oxygen therapy takes place in either a monoplace hyperbaric chamber or a multiplace chamber. Multiplace chambers can hold multiple patients and an accompanying inside tender, while monoplace chambers host a single patient with the tender assisting from outside. Inside tenders of multiplace chambers provide patient care and technical support. At our institution, inside tenders apply head tents, assist and coach middle ear equalization, administer medications, and provide for patient comfort needs. When accompanying critical care patients in the chamber, tenders take on additional roles including ventilator management and administration of intravenous infusions such as crystalloids, sedatives and vasopressors.

Inside tenders breathe compressed room air while patients receive 100% oxygen through a mask. As a result, the attendants are exposed to the risks of hyperbaric pressure experienced by recreational divers. Several safety measures exist in order to protect tenders. At our institution inside tenders are screened pre-employment for contraindications to hyperbaric exposure, including history of obstructive lung disease, seizure disorder, and inability to equalize middle ear cavities, among others.

Tenders are typically compressed once a day, with the occasional second compression after an appropriate surface interval. The U.S. Navy Diving Manual is used to guide the duration of any given surface interval. Report of an upper respiratory or severe sinus infection temporarily excludes an inside tender from being compressed. Requiring 100% oxygen breathing for tenders at the end of an $HBO_2$ treatment session additionally mitigates the risk of complications [5,6]. Despite these various safety precautions, known risks for chamber inside tenders include decompression illness and barotrauma [7]. To our knowledge, IEDCS has not been previously reported in the setting of a therapeutic hyperbaric multiplace chamber.

**KEYWORDS:** hyperbaric complication; hyperbaric medicine; hyperbaric occupational hazard; hyperbaric oxygen therapy

## CASE PRESENTATION

A 55-year-old man who worked as a respiratory therapist and hyperbaric multichamber operator was evaluated for changes in hearing minutes after completing a shift as an inside chamber tender. He had completed many previous dives as a part of his job, with no prior complications.

On the day of presentation he was the inside tender for a dive profile of 2.4 atmospheres absolute (ATA; 243.2 kilopascals) for 90 minutes plus compression and decompression time. The tender breathed compressed air for the majority of the dive, with five minutes of oxygen breathing at 1.6 ATA (162.1 kilopascals) prior to the end of the session. The session was uneventful, and he experienced no symptoms throughout its duration, including no difficulty clearing his ears. He worked only one dive that day, and his most recent previous dive had taken place three days prior. He was not sick with an upper respiratory or other illness on the day of presentation.

Approximately five minutes after the hyperbaric chamber session the tender began experiencing aural fullness and tinnitus in his left ear, without otalgia. The attending hyperbaric physician performed an otoscopic evaluation that revealed no abnormalities, and the tender was directed to employee health services. While walking there approximately 45-60 minutes after exiting the hyperbaric chamber he developed nausea, vertigo and ataxia. Upon arrival to the employee health office, he was directed to the emergency department (ED) for expedited evaluation. He denied dysarthria, aphasia, syncope, headaches, numbness or tingling, otalgia, otorrhea, prior history of ear infection, facial pain, dental pain, chest pain, palpitations, shortness of breath, fever or chills. A vague history of previously unquantified chronic right-sided hearing loss and well-controlled hypertension was noted.

Vital signs taken in the ED were unremarkable. A physical exam revealed the tender showed right-beating horizontal nystagmus on lateral gaze to the right, and a right and rotary down-beating nystagmus on left lateral gaze. Extraocular movements were otherwise normal. His cranial nerves were intact, with the exception of baseline decreased hearing with finger rub on the right side. Gait was mildly ataxic with a wide base. Romberg test was negative. An otoscopic exam revealed pearly, mobile tympanic membranes bilaterally with no middle ear effusion. No signs of middle ear barotrauma (MEBT) were observed. The Weber test showed midline localization. The Rinne test was negative in the right ear (bone conduction > air conduction) and positive in the left (air conduction > bone conduction), consistent with the tender's baseline chronic right-sided hearing loss. The Dix-Hallpike maneuver did not elicit nystagmus or vertigo in either direction.

Further testing, which included complete blood count, blood chemistry, urine and blood drug screens, electrocardiogram and troponin, were normal. ED providers consulted Otolaryngology and Hyperbaric Medicine, who reached the consensus diagnosis of IEDCS in our patient. Diagnosis of inner ear barotrauma (IEBT) was deemed less likely, as symptoms started five minutes after the $HBO_2$ therapy session ended and were predominantly vestibular in nature. Additionally, otoscopic exam revealed no evidence of tympanic membrane injury, effusion or fistula, and the tender did not have difficulty clearing his ears during the compression [1]. Magnetic resonance imaging (MRI) was completed the next day and showed no acute intracranial pathology. Additionally, transthoracic echocardiogram (TTE) with bubble study was normal, with no evidence of right-to-left shunt.

Upon the diagnosis of IEDCS, the patient was treated with a U.S. Navy (USN) Table 6 protocol $HBO_2$. The interval between symptom onset and start of recompression was approximately five hours. The tender was admitted to the hospital for additional $HBO_2$ sessions and initiation of a prednisone taper. While hospitalized, he received 50 mg of prednisone for a total of three days. He then underwent three $HBO_2$ therapy sessions as an inpatient. The first treatment session adhered to the USN Table 6 protocol. This protocol begins with compression to 2.8 ATA followed by at least three 20-minute cycles of oxygen breathing alternating with five minutes of air breathing. This is followed by a slow ascent at 1 foot per minute to 1.9 ATA. Two 60-minute oxygen breathing periods ensue at 1.9 ATA, with an intervening 15-minute air breathing interval between the two. The treatment concludes with the remaining ascent to the surface at a rate of 1 foot per minute.

Our patient completed a second $HBO_2$ treatment session consisting of 120 minutes at 2.4 ATA 16 hours after the first session. An additional USN Table 6 session was completed 19 hours later. Following these sessions he reported resolution of his ataxia, nausea and vertigo. His tinnitus did not improve, and physical exam revealed residual nystagmus. His most marked symptomatic improvement occurred after the first $HBO_2$ session, with more modest additional improvement in the following two sessions.

**Table 1**

| | middle ear barotrauma | inner ear barotrauma | inner ear decompression sickness |
|---|---|---|---|
| symptoms | ear pain on descent<br>hearing loss,<br>possible transient vertigo | ear pain on descent<br>hearing loss, tinnitus,<br>severe vertigo and nausea | ear pain on or after ascent<br>hearing loss, tinnitus<br>vertigo, nausea |
| signs | conductive hearing loss<br>TM injury<br>unilateral facial paralysis | sensorineural hearing loss<br>nystagmus, vomiting<br>ataxia, Romberg sign | sensorineural hearing loss<br>nystagmus, vomiting<br>ataxia, Romberg sign<br>other neurological findings<br>dermatological findings |
| treatment | medical management followed<br>by surgery if needed | medical management followed<br>by surgery if needed | $HBO_2$ therapy |

Comparison of typical signs, symptoms, and treatment for middle ear barotrauma, inner ear barotrauma, and inner ear decompression sickness. Modified from Byyny and Shockley (22).
Abbreviations: $HBO_2$, hyperbaric oxygen; TM, tympanic membrane.

Upon hospital discharge, the tender was able to ambulate independently and reported baseline hearing. He was sent home with the remainder of the prednisone taper (40 mg for three days, 30 mg for three days, 20 mg for three days, 10 mg for three days) and underwent four additional $HBO_2$ treatment sessions as an outpatient (2-2.4 ATA) for a total of seven sessions. Additionally he completed outpatient vestibular rehabilitation. He was scheduled for outpatient audiometry, but declined to keep the appointment. At the three-month follow-up he reported that other than the persistent and unchanged tinnitus, all other symptoms had completely resolved, and he had returned to baseline function and activity.

## DISCUSSION

This is a case of IEDCS induced by working as an inside tender during $HBO_2$ therapy at an academic tertiary care hospital. To our knowledge, this is the first case report of an employee developing IEDCS in a therapeutic hyperbaric multiplace chamber.

IEDCS is a subset of decompression sickness, and as such the underlying mechanism is thought to be the same. Decompression sickness is caused by the generation of nitrogen gas bubbles in the vasculature and organ tissue. These gas bubbles form during or after diving ascent, when dissolved nitrogen is released from tissues and allowed to rapidly re-expand. In IEDCS, gas bubbles form in the endolymphatic and perilymphatic spaces, resulting in vestibular symptoms (i.e., vertigo, nystagmus, nausea), cochlear symptoms (hearing loss, tinnitus), or a combination of both [1,2].

IEDCS is a rare complication of scuba diving. A review published in JAMA Otolaryngology reported the incidence of IEDCS as 0.2-0.3% per recreational dive [8]. We were unable to find any studies regarding the prevalence of IEDCS secondary to $HBO_2$ therapy or hyperbaric chamber use. The most common complication of $HBO_2$ therapy is middle ear barotrauma, occurring in up to 9.2% of patients [9]. MEBT results from failure to equalize pressure between the middle ear and the external environment, which can lead to effusion, vessel rupture, and tympanic membrane perforation. The incidence of IEBT (injury to the vestibule, cochlea, and/or semicircular canal) as a consequence of hyperbaric chamber use is unknown.

IEBT and IEDCS present similarly, with overlapping symptoms of nystagmus, vertigo, tinnitus and hearing loss (Table 1). Distinguishing between the two is important for management but can be difficult without surgical exploration. For this reason, a diagnostic schema termed the HOOYAH tool was created to assist in differentiating between IEBT and IEDCS [2]. HOOYAH stands for the dive and symptom characteristics that help to discern the correct diagnosis, including:

- H: hard to clear
- O: onset of symptoms
- O: otoscopic examination
- Y: your dive profile
- A: additional symptoms
- H: hearing (Table 2).

**Table 2**

| HOOYAH criteria | typical IEBT characteristics | typical IEDCS characteristics | case patient characteristics |
|---|---|---|---|
| H: Hard to clear ears | present on descent or ascent; forceful Valsalva | not associated with difficulty clearing | no difficulty clearing |
| O: Onset of symptoms | may occur on descent, ascent, or after surfacing | may occur on ascent or after surfacing | occurred 5 minutes after surfacing |
| O: Otoscopic exam | Findings of MEBT | normal | normal |
| Y: Your dive profile | profile with no/low risk of DCS; fast ascent or descent | decompression diving; missed decompression stops; any dive profile with risks for DCS | low-risk profile, no missed decompression stops |
| A: Additional symptoms | none; symptoms isolated to innerear | may be associated with other decompression sickness symptoms (e.g., neurologic, dermatologic findings) | no skin or CNS findings beyond ataxia |
| H: Hearing loss | very common; fluctuating hearing loss, often high-frequency loss | vestibular symptoms more common; often right-sided | vestibular symptoms (vertigo, nystagmus, nausea) symptoms + tinnitus |

HOOYAH Criteria adapted from Rozycki, Brown, and Camacho [2]. Abbreviations: CNS, central nervous system; DCS, decompression sickness; IEBT, inner ear barotrauma; IEDCS, inner ear decompression sickness; MEBT middle ear barotrauma

Our patient's presentation fulfilled most but not all of the HOOYAH criteria for IEDCS (Table 2). Notably, our patient's dive profile was not high-risk, although such profiles are more typical among cases of IEDCS. He had no difficulty clearing his ears during the dive and no findings of middle ear barotrauma on otoscopic exam, however, consistent with the diagnosis of IEDCS. Though the presence of additional symptoms of decompression sickness is a criterion that helps to identify IEDCS, another study found that only 17% of IEDCS cases present with other decompression sickness-related symptoms [10]. Therefore, the lack of additional neurologic or skin findings did not diminish the suspicion for IEDCS in this case.

IEBT typically occurs during dive descent as a result of difficulty equalizing middle ear pressure, which may be noted as difficulty clearing the ears with forceful autoinflation [2,11]. Resulting vestibular membrane rupture leads to effusion or perilymphatic fistula formation [11,12]. Treatment for IEBT generally starts conservatively, with bed rest and precautions to decrease middle ear pressures, and surgery following if symptoms persist [9,13]. In contrast, IEDCS management ideally requires early $HBO_2$ therapy [2-4]. The USN Table 6 treatment profile is commonly used for this purpose, and is highly effective in resolving symptoms of decompression sickness [14]. USN Table 6 is also used in the treatment of arterial gas embolism.

Isolated IEDCS has been observed during deep dives, especially those with incomplete or missed decompression stops [2]. Gas dissolves into liquid in proportion to pressure [15], and this gas can precipitate as injurious bubbles in body tissues upon ascent [3]. The mechanism of increased vulnerability of the inner ear is not completely understood but may result from longer gas washout times. Gas molecules are released from the ear at a slower rate than other tissues, thus allowing for supersaturation and increased risk of bubble formation, even at ambient pressures [16,17].

Some isolated cases of IEDCS are associated with right-to-left blood shunting, which is found in approximately eight in 10 patients [1,18,19]. This association has led to the theory that shunted gas emboli induce injury by occluding the labyrinthine artery [18]. Our patient underwent a bubble transthoracic echocardiogram (TTE) study which did not demonstrate a shunt. The lack of this common predisposing factor, along with the absence of prior issues working in the hyperbaric chamber, makes his case particularly interesting.

The patient's unexpected tuning fork test results provided additional interesting findings. The Weber test showed midline localization, while the Rinne test was negative in the right ear (bone conduction > air conduction) and positive in the left (air conduction > bone conduction). These findings were inconsistent with our expected results. We presume that the symmetric Weber result derived from acute sensorineural hearing loss in the left ear and chronic hearing loss in the right ear. Bilateral hearing loss would produce a symmetric test result as was found. With regard to the Rinne test, we can only presume that these screening tests are imperfect and vary with exact technique. Given these unexpected results, we hoped to obtain formal audiometry, but the patient elected not to pursue testing.

Optimal treatment of IEDCS includes $HBO_2$ therapy [20]. This should ideally be initiated as soon as possible after the inciting dive, as prompt treatment is associated with improved resolution of symptoms and recovery [21]. Delaying the initiation of $HBO_2$ therapy beyond six hours results in a high incidence of permanent damage [3]. Recovery is incomplete in the majority of IEDCS cases, with most patients sustaining residual damage [1,19].

## CONCLUSION

Working as an inside tender during multichamber $HBO_2$ therapy is relatively safe as long as safety protocols are followed. This line of work is not without risks, however. Potential complications include barotrauma and decompression sickness. To our knowledge, there are no published cases of IEDCS developing in hyperbaric chamber tenders. Though extremely rare, this complication is important for hyperbaric providers to be aware of, as rapid identification and treatment initiation are crucial. The possibility of IEDCS should additionally be considered in risk-benefit discussions with employees of multiplace chamber hyperbaric centers.

### *Conflict of interest statement*

*The authors have declared no conflicts of interest.*

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