

The diving doctor's diary

Isolated inner ear decompression illness following a nitrogen/oxygen dive: the difficulty in differentiating inner ear decompression illness and inner ear barotrauma

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Key words

Decompression illness, diving, technical diving, inner ear decompression illness, barotrauma

Abstract

Inner ear decompression illness is typically associated with deep dives involving helium/oxygen mixtures, usually when gas switching to air during ascent. It is most commonly associated with other symptoms of decompression illness. We present the unusual case of apparently isolated inner ear decompression illness in a diver not employing helium or gas switching. Our case illustrates the difficulty and importance in differentiating inner ear decompression illness and inner ear barotrauma. Issues regarding the immediate treatment of cases such as these are addressed.

Introduction

Inner ear decompression illness (IEDCI) is a relatively uncommon form of decompression illness (DCI) and a case review suggested the incidence of inner ear related symptoms is low.^{1,2} IEDCI is most commonly reported during and after helium/oxygen deep diving, particularly following a switch to air breathing during ascent. Bubbles probably form due to the differential solubility and diffusion properties of helium and nitrogen.³ IEDCI is usually associated with other symptoms of DCI, but can occur as an isolated clinical entity. We present a case of apparently isolated IEDCI following a nitrogen/oxygen dive and discuss the difficulty in differentiating IEDCI from inner ear barotrauma (IEBT).

Case history

A 47-year-old self-employed builder presented to hospital, having developed left-sided hearing loss with tinnitus, vertigo and nausea following a single dive to a maximum depth of 50 m.

He was an experienced diver, with over 4,000 dives and in good health. He had substantial experience in helium/oxygen/nitrogen (Trimix) mixtures, closed circuit rebreathers and planned decompression staging. On this occasion, he dived to 50 m maximum depth, (bottom time 30 minutes, total dive time 67 minutes) using a Buddy Inspiration™ closed circuit rebreather system (AP Valves, Helston, Cornwall, UK). Air was used as the diluent gas and the rebreather was set to deliver a constant P_{iO_2} of 1.35 atmospheres absolute (ATA, 136 kPa). The dive profile had been obtained using software based on the 'variable permeability model' available over the Internet (<www.v-planner.com>). He had used this planner previously without incident. On this occasion he planned for a 33 minute bottom time, an ascent time of 32

minutes and eight decompression stages. His actual bottom time was slightly shorter (30 minutes), and he had not violated the profile planner during the dive. His portable depth and time recorder confirmed this when later examined. There were no problems during the dive. In particular, he did not have difficulty with middle ear equalisation, nor did he have any upper respiratory tract infection.

Ten minutes after surfacing, while on board the dive boat, he complained of fullness and deafness in the left ear that could not be cleared by repeated gentle Valsalva manoeuvres. Symptoms gradually worsened and he began to experience right to left vertigo, nausea without vomiting, loss of balance and left-sided tinnitus. By the time he regained the shore 30 minutes after surfacing, his hearing loss was profound and he required assistance to walk from the boat. He had received no oxygen or other therapy at this time.

One and a half hours after his symptoms had started, he was assessed at a local hospital emergency department. He was placed on high flow oxygen via a non-rebreather Hudson mask and intravenous hydration was commenced. He was kept strictly supine and transferred to the Prince of Wales Hospital (POWH), where he was assessed 4 1/2 hours after his first symptoms. He continued to complain of vertigo, nausea and loss of hearing in his left ear, although he reported approximately 10% improvement since beginning oxygen administration.

On specific questioning the patient admitted to two previous episodes of DCI. The first was in 1994, when he developed shoulder pain following an uncomplicated 78 m Trimix dive. The second was in 1996, following another deep dive, complicated on that occasion by omitted decompression, strong currents and buoyancy difficulties. This dive resulted in progressive neurological and musculoskeletal DCI. He

was treated in a military facility and received extended recompression therapy and intravenous lignocaine. He made a slow but complete recovery. Although advised that he was permanently medically unfit to dive, he had logged in excess of 2,000 dives since.

On examination, nystagmus was not present. Otoscopy revealed normal tympanic membranes with no evidence of middle ear barotrauma. A fistula test (performed by application of pressure to the external ear in an attempt to elicit nystagmus), was negative suggesting there was no perilymph fistula. Rinne and Weber's tests suggested a sensorineural hearing loss on the left. As the patient had been sitting and walking until transfer to POWH, he was permitted to stand. His sharpened Romberg's test, using the method described by Fitzgerald, was only 3 seconds.⁴ There were no other neurological deficits evident, and Mini Mental State Examination scored 29/30. Other systems review was normal. No laboratory investigations were undertaken.

On the basis of assessment by the diving and ENT teams, a presumptive diagnosis of IEDCI associated with acute sensorineural hearing loss was reached. He was given methylprednisolone (50 mg) with ranitidine (150 mg) and recompressed using a Royal Navy 2.8 ATA (284 kPa) oxygen treatment table (RN62) four hours after arriving at POWH. At 2.8 ATA, hearing and tinnitus improved substantially. Vertigo improved only slightly. Once out of the chamber, tinnitus returned, but to a lesser degree and balance remained poor with a sharpened Romberg's test of 5 seconds. He continued to complain of vertigo on sudden head movement.

The patient received six daily recompression treatments involving oxygen administration at 2.4 ATA (243 kPa) for 90 minutes and a reducing dose of prednisolone for 10 days. On this regimen he reported gradual improvement. Four days after admission, he rotated to the left on the Unterberger step test, suggesting injury to the left labyrinth.⁵ Audiometry demonstrated a left-sided sensorineural hearing loss, with a dip at 3–4 kHz down to a level of 50 dB. Sensorineural loss in low tones was about 20 dB.

Over a period of six days, his symptoms continued to improve and his sharpened Romberg's test normalised to >60 seconds. In retrospect, he also reported other symptoms that had now resolved, including fatigue, malaise and an inability to concentrate and multi-task. He was discharged following the final compression.

Two weeks after hyperbaric therapy was ceased, he remained symptom free and his balance was normal on clinical assessment. A repeat audiogram showed an improvement in all thresholds except 3 and 4 kHz. This was presumed to be due to previous noise exposure. Four weeks after his injury he was completely well, and felt that his hearing had returned to its premorbid state. He was again strongly advised against continuing his diving career.

Discussion

Our patient presented with unilateral audiovestibular symptoms and signs that may have been due to IEBT or IEDCI. Indeed, the two pathologies may occasionally co-exist.⁶ While isolated IEDCI is a relatively rare presentation,² it is of considerable importance to accurately distinguish between the two where possible, given that the appropriate therapy for each may be mutually exclusive.⁷ IEDCI requires immediate recompression, but further efforts to equalise pressures in the middle ear during the compression phase could worsen IEBT.⁸ On the other hand, avoiding compression in favour of bed rest might allow rapid progression of the bubble injury, perhaps including further central neurological injury. Indeed, some centres favour early operative intervention for IEBT and general anaesthesia may be significantly hazardous in the face of evolving DCI with the inadvertent administration of nitrous oxide.⁹ In our case, differentiation between the two pathologies was difficult and making the diagnosis delayed the initiation of recompression, as seen by the four-hour delay at POWH before recompression.

The diagnosis is usually indicated by careful history taking.^{7,8} In an experienced diver, IEBT is likely to be associated with unusual difficulty equalising the middle ear, and the dive profile and gas mixtures used will contribute to an appreciation of the possibility of DCI. The symptoms of IEBT are typically of acute onset following a forceful Valsalva manoeuvre during descent or on straining after the dive. By definition, symptoms of DCI never arise prior to ascent and often appear more gradually without specific provocation. With IEBT, there may be evidence of middle ear barotrauma on examination. Other symptoms of DCI, or a previous history of DCI, are suggestive of IEDCI. IEDCI is also said to be more frequently associated with deep dives on helium/oxygen mixtures and a number of explanations have been proposed for this.^{2,3,10}

Some aspects of this case made IEDCI less likely. He had neither used helium/oxygen mixtures nor violated his decompression requirements. Isolated IEDCI is uncommon and our patient initially complained of inner ear symptoms only. Interestingly, he later revealed other mild inflammatory symptoms of DCI as they receded over time.

On the other hand, most evidence available on admission did suggest IEDCI to be the more likely diagnosis. He had suffered DCI twice previously. He had no difficulty in equalising on descent or ascent, nor had he complained of pain in his ears. His symptoms had started several minutes after the dive, gradually worsened over half an hour, and had improved with oxygen administration during transfer to POWH. On examination, there was no evidence of middle ear barotrauma. The negative fistula test was less reassuring; in ours and others' experience the fistula test is rarely positive in the presence of a perilymph fistula.¹¹

Although often associated with helium/oxygen gas mixtures, a retrospective study of 29 cases over 12 years suggested IEDCI after compressed-air recreational diving is more common than previously thought, and has certainly been reported in the absence of decompression schedule violations.⁸

In addition to oxygen, rehydration and recompression, our patient was given steroids as is our practice for the diagnosis of sudden sensorineural hearing loss. In a randomized double-blind trial, steroids have been indicated for sudden sensorineural deafness of any cause.¹²

Is DCI more likely on this profile than for a conventional air dive within the usual recreational limits? This was a technical dive based on a relatively new experimental set of dive schedules outside the limits of normal recreational diving. At 50 m the PO_2 of air alone is 1.26 ATA (21% of 6 ATA, 606 kPa). With a target $P_{I}O_2$ of 1.35 ATA (136 kPa) for this dive, the rebreather set would have added very little oxygen at 50m depth. Thus, during the deeper part of this dive there was little benefit in terms of reduced nitrogen load, although nitrogen elimination was enhanced by the use of supplemental oxygen on ascent.

Our patient gradually improved with hyperbaric oxygen. In a previous report using a similar treatment protocol, IEDCI was similarly less responsive than other forms of DCI.¹³ A positive response to HBOT does not establish a diagnosis of DCI. If our patient in fact suffered from IEBT, his tinnitus and deafness may still have improved with hyperoxia provided further barotrauma during treatment was avoided. Such improvements in idiopathic hearing loss have been suggested by a number of previous reports.¹⁴

This case also raises some issues concerning the first aid of diving injuries. Surface oxygen therapy should have been administered much sooner, preferably on the dive boat, as is the policy of the South Pacific Underwater Medicine Society.¹⁵ All dive boats should carry oxygen for this purpose. In practice, he did not receive oxygen or fluids until two hours after the onset of symptoms.

Further, such an early presentation of symptoms suggests the possibility of arterial gas embolism and should have resulted in a decision to keep him supine or in the left lateral position until expert medical advice was obtained. That such experienced divers should omit these basic first-aid measures is a matter of concern and should result in a re-examination of the way in which these potentially life-saving, first-aid interventions are promoted to the diving community.

Conclusion

IEDCI and IEBT may be difficult to differentiate in practice. This should not preclude standard first-aid measures such as maintenance of the supine position, oxygen and fluid therapy. IEDCI occurs in divers not using helium/oxygen

mixtures and in divers who do not switch gases. It may appear in isolation. Our case illustrates the value of a thorough medical history and otoneurological assessment in the context of possible IEDCI.^{8,16}

Acknowledgements

The authors gratefully acknowledge Drs Michael Bennett and Simon Mitchell for their valuable assistance.

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