Original articles

Diagnostic dilemmas in inner ear decompression sickness

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Decompression sickness, decompression illness, ear barotrauma, inner ear, labyrinth, case reports

Abstract

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Inner ear symptoms associated with diving illness may result from barotrauma or decompression sickness. Differentiation of the underlying pathology presents a diagnostic dilemma for the diving clinician, especially if remote from otological laboratories. We present a series of such cases, some of which were likely to be due to inner ear (vestibular) decompression sickness, and others which could be confused with this diagnosis. These cases highlight the diagnostic and management dilemmas involved.
Authors' note: The term decompression sickness (DCS) was chosen in preference to decompression illness (DCI), as the latter term may lead to confusion. Inner ear DCI could mean barotrauma or DCS.

Introduction

The symptom complex of vertigo, nystagmus and nausea occurring in decompression sickness (DCS) may be caused by injury to the inner ear, the cerebellum or their connections. Cerebellar vertigo is almost always associated with other signs of cerebellar dysfunction that are obvious on clinical examination, and nausea is not usually a prominent symptom. However, DCS is an acute disease affecting vestibulo-cerebellar connections and may also have associated raised intracranial pressure.

Injury to the inner ear, however, may be the result of barotrauma (Bt) or decompression sickness (DCS), and it may be difficult to differentiate between these two pathological processes. Barotrauma-induced injury may be associated with a history of difficulty in equalising the middle ear during the dive, and with clinical signs of tympanic membrane injury and hearing loss.

Isolated inner ear decompression sickness (IEDCS) occurs more commonly in mixed-gas divers, especially at times of change in gas mixture. It has therefore become a general perception that IEDCS is rare in compressed-air diving and that most inner ear symptoms that occur are due to inner ear barotrauma. Nevertheless, Reissman et al alerted medical personnel to be aware of the possible occurrence of IEDCS among the wider population of scuba divers.[1] Subsequently, Nachum et al reported their twelve years' experience with 24 divers and concluded that IEDCS in sport diving is not as rare as previously thought.[2] IEDCS is usually associated with other signs of DCS, and in their series as many as a quarter of the divers suffering from 'serious' or 'neurological' DCS had inner ear involvement.[2] Differentiation of IEDCS and inner ear barotrauma (IEBt) is important from a therapeutic point of view because, whereas early recompression is indicated for IEDCS, recompression is relatively contra-indicated in IEBt and may worsen the condition. This paper presents eight divers from Western Australia, Tasmania and Queensland with a referral diagnosis of DCS, all arising from air diving, and in whom vertigo was a predominant symptom. The diagnostic difficulties and their management are discussed.

Case One

A 44-year-old male with 30 years' diving experience was diving alone using hookah equipment. He dived as follows:
Dive 1: 15-17 metres of seawater (msw) for 60 mins, a surface interval (SI) of 15 mins
Dive 2: 17 msw for 30 mins, SI of 5 mins
Dive 3: 17 msw for 15 mins. He surfaced with no decompression stops.

Twenty minutes after surfacing, he experienced dizziness, nausea and vomiting, especially on looking upwards. He had a tendency to fall to the right, deafness in his right ear, a generalised headache and right facial paraesthesia. He was taken to hospital where he was given oxygen, intravenous (IV) fluids, antibiotics, prochlorperazine and bed rest with head-up tilt for two days. The provisional diagnosis was IEBt, although he claimed that he had not experienced any difficulty in equalising his ears. Tympanic membranes were normal and there was no nystagmus.

The day after his dive, he developed pain in his left elbow, right shoulder, right knee, both thumbs and his back. There was little change in his symptoms over the next 24 hours. By the third day, his paraesthesia and headache had resolved. However, his dizziness, nausea and limb pains persisted and his right ear remained deaf, with no tinnitus. On the fifth day, he saw his local doctor and on his own

insistence flew to Perth on a commercial flight (lasting two hours). He claimed that his symptoms did not deteriorate en route, but later told the nurses he felt worse in transit.

On examination, there was no nystagmus nor any signs of cerebellar abnormalities. Unsteadiness prevented an assessment of the sharpened Romberg test (SRT), but he walked with a broad-based gait. Tympanic membranes were intact and mobile, and tympanogram was normal. Pre-treatment audiometry revealed a high-frequency hearing loss of greater than 50 dB at 6000 and 8000 Hz. His previous annual medical from his employment had shown a near-normal audiogram in the right ear.

He was recompressed on Royal Navy Table 62 (RN62), and had two subsequent daily hyperbaric oxygen treatments (HBOT), after which his symptoms resolved except for SRT <20 secs and subjective hearing loss in the right ear.

He was then referred to and assessed by the same ENT surgeon who had operated on him in 1997 for an 'inner ear fistula' following a diving incident. His pure tone audiogram showed 60 dB loss at 8000 Hz. A surgical exploration found no evidence of a perilymph fistula on this second occasion.

On review three months later, he still had some dizziness when he moved his head quickly, but his balance was normal and he was able to ride a motorcycle. Despite advice to the contrary, he had returned to diving.

DIAGNOSIS: Decompression sickness; neurological, vestibular and musculoskeletal

COMMENT: The presence of other symptoms of DCS (paraesthesiae, headache and limb pains) in the absence of any history of barotrauma point toward a diagnosis of IEDCS. Recompression therapy would have been indicated anyway due to the presence of the other symptoms.

Case Two

A 51-year-old, experienced diver who had been diving since he was 15, was diving off the Abrolhos Island. He dived as follows:
Dive 1: 40 msw for 30 mins, stopping at 6 msw for 10 mins to decompress, SI of 1-1.5 hours
Dive 2: 27 msw for 40 mins, surfacing without a decompression stop.

On surfacing, he felt nauseated, and claimed that his abdomen felt "bloated". This progressed to vertigo and vomiting over the next two hours. He felt weak and was unable to stand. He informed the divemaster of his symptoms and was told that he must have gastroenteritis. The following day, still feeling unwell, he was transferred from the island to Geraldton via a light aircraft. He felt worse during this air transfer. In Geraldton, a medical officer noted marked nystagmus to the right, vertigo, nausea and vomiting. Oxygen was administered and IV fluids, prochlorperazine, ondansetron, dexamethasone and salbutamol. A CT scan of his head was normal. He was transferred via the Royal Flying Doctors Service to Fremantle.

On arrival at the hyperbaric unit 30 hours after surfacing from his last dive, he was very tired and complained of vertigo especially when he attempted to sit up. He was ataxic and had nystagmus with fast beat to the right. Muscle power and tone were normal, as were sensation and reflexes. There were no cerebellar signs detected. Tympanic membranes were normal. His audiogram prior to recompression showed high-frequency loss in his right ear worse than the left. Previous audiograms had demonstrated symmetrical high-frequency hearing loss bilaterally.

He was recompressed according to RN62. After treatment, he still had vertigo, especially when his head was turned to the side. He was able to stand, but walked with a wide-based gait. Pre-treatment on the third day, he was able to do a standard Romberg test, and still had nystagmus. He received a total of nine HBOT.

Two weeks later, an ENT surgeon confirmed his pre-existing hearing abnormality. A bilateral high tone loss consistent with noise-induced injury was observed. He was ataxic with a wide-based gait. Caloric testing was said to be difficult because of a latent nystagmus that was right beating, although this changed direction when he was gazing to the left. Unfortunately, due to his disability, he was declared 'unfit' to work at his place of employment. He was referred to a neurologist who referred him to attend physiotherapy for vestibular rehabilitation.

Despite advice not to, he resumed diving seven months later, and has dived to 45 msw in Bali on numerous occasions. He says initially he had difficulty telling which way was up or down, but he claims that he is now 'OK'.

DIAGNOSIS: Vestibular decompression sickness, inner ear barotrauma

COMMENT: Although caloric testing was unsuccessful in determining the origin of this man's nystagmus, the absence of other symptoms of DCS would ordinarily point to IEBt as the cause of his symptoms. He had no history of difficult equalisation, and tympanic membranes were normal. His symptoms improved with recompression, although he retained some disability, possibly due to the delay in treatment.

Case Three

A 38-year-old diver with 10 years' experience of over 5000 dives dived to 35 msw for 45 mins on hookah. He stopped at 9 msw for 5 mins on ascent with the intention of also breathing 100% oxygen on the surface.

About 15 mins after surfacing, he felt unsteady and had difficulty standing. He went to lie down and noted he had low back pain, headache, a numb right forearm and hand, and a tingling sensation in his feet and calves. He was given oxygen to breathe from a demand regulator, but he became nauseated when he opened his eyes and vomited. He was transferred to hospital by helicopter for treatment. In transit, he was given 100% oxygen, IV fluids and metoclopramide.

On examination, he was fully conscious, clear headed and well hydrated, and although he felt generally weak he was able to move his legs freely against gravity. However, he was unable to move his head without nausea and vomiting. He did not notice any hearing loss and had no tinnitus. Neurological examination showed that all tendon jerks were brisk but equal bilaterally. Muscle tone was normal but muscle power was observed to be reduced in all groups. Light touch, position and vibration sense were all normal. Tympanic membranes appeared normal, but he appeared to be slightly deaf clinically and had unsustained lateral nystagmus. A diagnosis of IEDCS was made.

Following treatment with an RN62 his symptoms were greatly improved, with some residual dizziness when he moved his head. He was still unsteady on his feet.

The following day he felt a lot better, but still had vertigo on sitting up or with rapid eye movement. He was able to walk with assistance and was again noted to have unsustained lateral nystagmus. An audiogram revealed bilateral high-frequency hearing loss and was identical to one performed for his last diving medical examination.

By the third day he was able to eat and to walk normally, although sudden head movement still caused dizziness. An opinion was sought from an ENT specialist, who agreed with the diagnosis of IEDCS. After a total of four HBOT his nystagmus had resolved, his reflexes were normal and SRT was 60 secs.

DIAGNOSIS: Neurological decompression sickness with vestibular involvement

COMMENT: The presence of other symptoms of DCS (paraesthesia, back pain, muscular weakness) and the absence of signs of barotrauma in a provocative dive point towards IEDCS as a cause for his vestibular symptoms.

Case Four

A 43-year-old man with 28 years' diving experience dived with hookah equipment as follows:
Dive 1: 13 msw for 2.5 hr, stopping at 3 msw for 5 mins, SI approximately 45 mins
Dive 2: 10 msw for 1.5 hr, stopping at 3 msw for 5 mins, SI 1 hr
Dive 3: 10 msw for 1 hr, stopping at 3 msw for 5 mins.

He did not use a depth gauge, and admitted that his dives could have been deeper than he estimated. He had no difficulty equalising his ears at any stage of his dives.

About an hour after the last dive, as he leaned over the side of the boat to scoop up crabs, he became dizzy, unable to balance, and noticed pain and deafness in the right ear. On reaching shore, he was unable to walk due to lack of balance. He felt very tired and nauseated, and vomited once. He did not seek treatment until 24 hours later, when he presented to the regional hospital with persistent nausea and vertigo.

On examination, he was found to be unsteady with a simple Romberg test of less than 10 secs. Weber test was stated to be positive on the left. The diagnosis of 'IEBt and DCI' was made, despite his tympanic membranes being normal. He was transferred to a hyperbaric facility, arriving 29 hours after his last dive.

He was recompressed according to RN62, following which his vertigo was largely resolved and his SRT was 30 secs. Tuning fork tests were normal. His audiogram the following day showed a hearing deficit of 35 dB at 4000, 6000 and 8000 Hz of the left ear similar to his previous audiogram, due to previous noise exposure in his occupation as a mechanical fitter. He was given a follow-up treatment with Royal Navy Table 61 (RN61). Following treatment, his SRT was 60 secs. There was no nystagmus, or vertigo even when moving his head. The next day, he noticed a mild frontal headache, and some dizziness, which cleared following another HBOT. On discharge, he still had mild dizziness on rapid head movement.

DIAGNOSIS: Vestibular and neurological decompression sickness, inner ear barotrauma

COMMENT: IEBt is less likely in this case due to the lack of history of difficult equalisation, and normal tympanic membranes and normal tuning fork tests. His symptoms are predominantly vestibular in origin, and IEDCS would be a likely diagnosis.

Case Five

A 40-year-old, qualified diver with several years' experience had recommenced diving after a five-year break. He had been unwell during the preceding week with gastroenteritis, which had resolved three days earlier. He dived on a surface air supply, with no depth gauge, as follows:
Dive 1: 15 msw for 40 mins approximately, SI 90 mins
Dive 2: 28 msw for 30 mins.
Although the ascent from the second dive was controlled, he did not perform any decompression stops. He had no difficulty with equalisation during the dives.

On reaching the surface, he noticed an immediate headache, began to cough, and had pain in his chest and lower abdomen. He swam back to the boat, and boarded it unaided. He then recalls saying "You blokes better look after me because I'm really crook", then felt his face begin to twitch

and lost consciousness. He remained unconscious for the entire trip back to shore (approximately 90 mins) and was left sitting up in the back of the boat by his dive buddies.

On arrival at the jetty they were met by the local doctor, who noted him to be grossly cyanosed and making little respiratory effort. Blood pressure was 70 mmHg systolic. He was placed supine and was administered oxygen and IV fluid. Within about 20 minutes, he became responsive and was opening his eyes to command. He was retrieved to the nearest hyperbaric facility by air ambulance, but was troubled by vomiting during the transfer. Metoclopramide 10mg IV was given with no effect.

On arrival, he was conscious and cooperative, but constantly vomiting and severely nauseated. Pulse 108 per min, BP 123/80 mmHg, chest clear, equal air entry. There was a mottled rash over the left lower chest and abdomen, which blanched with pressure. His mental state was assessed with difficulty due to his constant vomiting and severe nausea, but appeared normal. He had nystagmus on central gaze, worse on lateral gaze to the right. Tone, power, and reflexes were normal. Sensation was grossly normal, but difficult to assess due to limited cooperation as a result of constant vomiting. Examination of the fundi showed no evidence of papilloedema. A Romberg test was not performed due to his inability to stand up. He had no joint pains. An audiogram showed no change from a previously recorded test at his workplace.

He was commenced on IV lignocaine infusion and recompressed according to RN62. His nausea ceased at 18 m but recurred after arrival at 9 m, and remained to the end of the treatment. He was commenced on prochlorperazine 10 mg orally as required for nausea. The lignocaine infusion was continued for 48 hours. He underwent a further thirteen daily HBOT. His nausea and vertigo gradually improved, and he was able to walk with assistance by the fifth day. By the seventh day his SRT was greater than 60 secs. On the ninth day, he reported altered sensation to temperature in his legs. Review by a neurologist showed a reduction in temperature sensation below dermatome level T11 and reduced pain sensation below T10. There was a gradual improvement in his sensory loss over the following weeks.

DIAGNOSIS: Neurological decompression sickness with vestibular involvement, cerebral arterial gas embolism (CAGE).

COMMENT: The hyper-acute onset of symptoms may suggest CAGE, but the dive was extremely provocative for bubble formation. Acute DCS is also likely, especially given the diverse nature of symptoms that evolved over subsequent days. The predominant symptoms at presentation were vertigo and nausea, and IEBt could have been a possible consideration. Given the history of unconsciousness there was never any doubt about the need for recompression therapy.

Case Six

A 21-year-old male with no formal diving training was diving with Case Five on hookah surface air supply, with no dive plan, depth gauge or dive computer as follows:
Dive 1: 15 msw for 40 mins, SI 40 mins
Dive 2: 28 msw for 30 mins followed by a controlled ascent to the surface, without a 'safety' or decompression stop.

His dive buddy lost consciousness on surfacing and was evacuated for urgent recompression. Despite this, and despite being personally contacted and advised to seek medical attention if he developed any unusual symptoms, he did not seek medical attention until six days later.

At presentation he complained of constant vertigo and nausea, worse in the mornings, and generalised aches and pains worst in his neck, shoulders and hips. The symptoms had developed within six hours of his dive, and had been sufficiently severe that he had been unable to attend work that week. Examination was normal except for some coarse nystagmus on far lateral gaze bilaterally, and an impaired SRT of 5 secs. There were no signs of cerebellar dysfunction. An audiogram was normal, and ENT opinion suggested IEDCS was the most likely cause of his nausea and vertigo.

He was recompressed according to RN62, and had a further five hyperbaric oxygen treatments. All symptoms completely resolved with this course of treatment.

DIAGNOSIS: Neurological decompression sickness with vestibular involvement

COMMENT: This diver had also undertaken an extremely provocative dive, and subsequently developed DCS. Vestibular symptoms were prominent, and were the reason for presentation. The complete response to recompression should be noted, especially considering the long delay to treatment.

Case Seven

This 33-year-old man with 18 months' diving experience had been diving for abalone as follows:
Dive 1: 15 msw for 120 mins, SI 15 mins
Dive 2: 10 msw for 90 mins, SI 30 mins
Dive 3: 6 msw for 120 mins.

The following day he dived to 16 m for 90 mins, with no decompression stops. About 20 minutes after surfacing, he developed dizziness, unsteadiness, vomiting, and abdominal pain. He waited until his dive buddy returned to the boat about 30 mins later, and they returned to the marina, arriving about two hours later. He was transferred by ambulance to the nearest hyperbaric unit.

On arrival, he had marked horizontal and rotatory nystagmus, worse on gaze to the right. Cranial nerve examination was otherwise normal. Muscle tone, power

and reflexes were normal, as was respiratory, cardiovascular and abdominal examination. Gait and SRT were not assessed due to severe nausea and vomiting on movement. Audiogram was unchanged from a pre-diving test.

He was treated with IV fluids and lignocaine infusion. A Doppler assessment detected high-grade intravascular bubbles (Grade 4) five hours after ascent from depth. He was recompressed on RN62, and all symptoms initially resolved at 18 m (280 kPa). Following ascent to 9 m (190 kPa), there was a gradual return of symptoms, and following treatment he reported a return of his dizziness and nausea. He was commenced on prochlorperazine and had eight further daily HBOT. His nausea and vertigo were severe for three days, necessitating inpatient treatment, then gradually resolved, and he was well at discharge.

DIAGNOSIS: Vestibular decompression sickness

COMMENT: The normal audiogram, absence of barotrauma, lack of neurological abnormalities, and the presence of severe vestibular symptoms all point to IEDCS.

Case Eight

A 25-year-old, recreational diver dived as follows:
Dive 1: 12.3 msw for 60 mins, stopping at 5 msw for 5 mins during ascent, SI 90 mins
Dive 2: 18 msw for 62 mins. During this dive, he did multiple ascents and descents between 6 msw and 18 msw. At the end of the dive, he did another safety stop, a '5 at 5'.

He had no difficulty equalising his ears during or after the dive. After his dive, he went to the gym and did a 'work-out' for about two hours and experienced some dizziness. At work the next day, he experienced true vertigo and also noticed some visual blurring and clumsiness of his hands. He also noticed that he was staggering a bit and had a 'twinge' of pain in his left shoulder, which did not persist.

He went to see his local medical practitioner, who found him to have marked diplopia on left lateral gaze, but no nystagmus. He had mild staccato dysarthria and moderately severe Rombergism with a tendency to fall to the right and backwards. He had a stamping, wide-based gait, dysdiadokinesis worse on the left, past pointing with intention tremor, and a positive heel-shin test. Muscle power and tone were normal, and there was no change in sensation or proprioception. Hearing was normal. He was recompressed on RN62 with complete recovery.

DIAGNOSIS: Cerebellar decompression sickness

COMMENT: This diver suffered one episode of vertigo, but had no nausea or nystagmus. His symptoms were predominantly cerebellar in nature, easily detected at clinical examination.

Discussion

The aetiology of inner ear DCS is not well understood. Doolette and Mitchell have proposed a three compartment model of the inner ear, comprising membranous labyrinth (vascular compartment), perilymph and endolymph.[3] In circumstances where the tissues become saturated with inert gas, such as prolonged air diving, decompressing too rapidly will exceed the ability of the labyrinthine vessels to remove inert gas as it diffuses from the two fluid compartments of the inner ear, leaving them susceptible to bubble formation. The unique fluid make-up of the inner ear therefore makes it a prime site for DCS under provocative circumstances.

IEDCS is an uncommon occurrence in air diving, but this should not lead to the assumption that IEDCS occurs only in heliox or hydrox diving and that, therefore, most inner ear problems in air diving are due to IEBt. The differential diagnosis between IEBT and IEDCS may be difficult.[4]

Farmer[5] and Nachum et al[2] suggested some distinguishing features that might be of assistance in making a diagnosis:

- In IEBt, a diver will normally report difficulty in clearing his ears at some stage.
- Symptoms of IEBt appear during the dive, whereas those of IEDCS appear after the dive.
- Other symptoms of DCS may accompany IEDCS.
- Signs of middle ear Bt may accompany IEBt but do not necessarily occur with IEDCS.
- Hyperbaric therapy will generally improve the symptoms of IEDCS, but may aggravate those of IEBt.

The distinction is not straightforward. Edmonds indicated that the delay of onset of symptoms is not necessarily a diagnostic feature in DCS, as it is not uncommon to find this in cases with perilymph fistulae.[6] Vertigo may also occur without hearing loss in both diseases. Also DCS may cause cerebellar manifestations (staggers) as well as vestibular damage, as illustrated in Case Eight.

When doubt exists, Edmonds recommends electronystagmography and iced-water caloric tests in the recompression chamber. Other useful tests include dynamic posturography, vestibulospinal response reactions to stress, and electrocochleography. Temporal bone polytomography and CT might be of value.[7] Molvaer, however, indicated that there is no conclusive test to tell whether or not a perilymph fistula is present.[8] If in doubt, recompression may be diagnostic.

Usually these cases present to the diving physician outside normal working hours, when complex neurophysiological diagnostic testing is not available. In some centres, there is a significant waiting period before neurophysiological testing can be performed, and in most cases it is not in the best interests of the diver to wait for confirmatory testing prior to recompression. Clinicians therefore have to rely on details gleaned from the history of the dive, and from

physical signs elicited on examination to make their diagnosis. As in all types of DCS, conservative treatment with bed rest, IV fluids and oxygen may result in some improvement without recompression, but the chance of long-term sequelae is greatly increased.

Classical teaching has told us not to recompress divers who may be suffering from inner ear barotrauma, but in some cases recompression may produce improvement in symptoms. Molvaer hypothesised that in IEBt, especially if there is dysfunction of the Eustachian tube, some gas from the middle ear might be forced through the perilymph fistula and enter the inner ear, either into scala tympani or scala vestibuli, depending on which window is damaged.[8] During further ascent, the inner-ear gas will expand and may damage cochlear or vestibular structures. If this hypothesis is correct, recompression may be therapeutic.

In most cases of IEDCS, divers have violated recommended dive tables. In the series reported by Nachum et al, 79% (23 cases) violated the tables.[2] In the same series, the onset of symptoms varied from immediate onset to five hours following the dive. Interestingly, one diver had five episodes of IEDCS. Ten cases (34%) had pure vestibular symptoms and all but one with vestibular symptoms had nystagmus. Four cases (14%) had cochlear symptoms alone and 15 cases (52%) had a combination of symptoms. Fifteen (52%) had isolated IEDCS, whereas 14 had additional symptoms of DCS. Thus, absence of other symptoms of DCS does not confirm that the diagnosis is IEBt. Of the 17 patients treated within six hours of symptom appearance, nine (53%) were cured. Of the 25 cases with vestibular injury and the 19 with cochlear damage, only seven (28%) and six (32%) respectively made a full recovery. Prompt treatment is therefore prudent to increase the chance of recovery, and delays in recompression whilst awaiting neurophysiological testing may not be warranted.

A recent study by Cantais et al found that a major degree of right-to-left shunt, as detected by transcranial Doppler, was associated with an increased incidence of cerebral and IEDCS, suggesting paradoxical venous gas embolism as a possible aetiology.[9] This may explain the occurrence of IEDCS in divers with minimally provocative dive profiles, and raises the question of whether we should seek a right-to-left shunt in divers presenting with DCS.

This series helps to illustrate the diagnostic dilemma faced by physicians when confronted with a diver with acute vertigo. The main decision, to recompress or not to recompress, must be carefully considered on the weight of history and symptoms and physical signs at presentation.

Conclusions

Although IEDCS is uncommon in air diving, it must be considered as a differential diagnosis for vertigo, as failure to treat IEDCS with recompression may result in permanent disability for the diver. It is sometimes difficult to distinguish between IEDCS and IEBt, but a careful history and clinical examination will often allow a distinction to be made between these two clinical entities.

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