Case reports

Recurrent dysbarism presenting with amnesia and hypoaesthesia in a professional breath-hold diver

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Abstract

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Dysbarism is a medical condition arising from change in ambient pressure which outpace the rate at which the body adapts to it. We report a case of recurrent dysbarism consistent with possible decompression illness presenting with amnesia, hypoaesthesia and other neurological manifestations in a professional breath-hold diver treated successfully with hyperbaric oxygen and fluid resuscitation.

Introduction

Dysbarism is a medical condition arising from changes in ambient pressure that outpace the rate at which the body adapts to it. This encompasses decompression sickness (DCS), nitrogen narcosis, high-pressure neurological syndrome, barotrauma, and arterial gas embolism (AGE).[1]

Two of these dysbaric conditions (DCS and AGE) involve bubble formation, and it may be difficult to distinguish between them clinically. For that reason they are sometimes referred to collectively as 'decompression illness' (DCI).[2] DCS is a multi-system condition that arises when dissolved gas molecules, primarily composed of nitrogen, emerge from solution and form bubbles within body tissues.[2] This occurs due to inadequate elimination of dissolved gas during ascent from a dive, and thus decompressing from high underwater pressure to atmospheric pressure.[3] AGE can occur when expanding gas causes pulmonary barotrauma, introducing bubbles into the arterial circulation.[2]

Professional breath-hold divers achieve great depths with fast descents and ascents with the practice of glossopharyngeal insufflation (often referred to as 'lung packing') and exsufflation; the latter being a strategy that facilitates equalising of paranasal sinuses and the middle ear. The deeper and longer breath-hold dives place them at an increased risk of DCS.[4]

We report a case of a professional breath-hold diver with recurrent dysbarism consistent with possible DCI. He presented with amnesia, hypoaesthesia and other neurological manifestations treated successfully with hyperbaric oxygen and fluid resuscitation.

Case report

The patient provided consent for publication of their case.

A 52-year-old male professional breath-hold diver carried out three recreational breath-hold dives accompanied by a diving buddy. The dives took place in calm seawater during winter season, with water temperature on the day of diving averaging 16°C. He completed the following dives without lung packing:

- Dive 1: maximum depth 33 metres of seawater (msw), total dive time (TDT) 1–2 min followed by a 5–6 min surface interval;
- Dive 2: maximum depth 42 msw, TDT 2 min 30 sec followed by an 8–9 min surface interval;
- Dive 3: maximum depth 60 msw, TDT 2 min 30 sec.

Within 10 minutes of surfacing from his last dive, he noted difficulty coordinating his right lower limb while swimming back to shore. He was offered assistance by his diving buddy which he refused. He completed a difficult water exit over uneven rocks independently and drove home unassisted. After 50 minutes, his right lower limb ataxia resolved spontaneously but was followed shortly by less severe ataxia in his left lower limb, described by the patient as "*difficulty with coordination*". This also lasted approximately 50 minutes and was associated with bilateral paraesthesia in the hands. These symptoms then resolved. He also noted non-vertiginous dizziness but denied any urinary incontinence or

urinary problems. Six hours later, he developed a right frontal headache which rapidly became bifrontal, radiating to the back of his head in a band-like distribution. He described it as a "*pressure*" sensation without throbbing and denied any preceding aura. It was associated with mild photophobia.

Approximately 24 hours later, the patient was well and he completed another three breath-hold dives uneventfully with 5-minute surface intervals:

- Dive 1: maximum depth 33 msw, TDT 1 min;
- Dive 2: maximum depth 40 msw, TDT 2 min;
- Dive 3: maximum depth 53 msw, TDT 2 min 30 sec.

Approximately 48 hours later, the patient mentioned that he had a headache and dizziness. He attributed the symptoms to caffeine intake and proceeded to dive as follows, again with 5-minute surface intervals:

- Dive 1: maximum depth 10–12msw, TDT 1 min;
- Dive 2: maximum depth 35 msw, TDT 2 min, after which the patient mentioned to his diving buddy that the headache had disappeared at the deepest point of the dive;
- Dive 3: maximum depth 45 msw, TDT of 2 min 30 sec.

Approximately 72 hours later, the patient presented to an accident and emergency department complaining of unresolved, severe, band-like headache and dizziness. He was noted to be alert and oriented to place and person but was unable to give a clear history and chronology of events. He did not recall that there was a third participating diver on two of the diving days, as well as being unsure whether he himself was diving on those days. A collateral history was therefore obtained from the diving buddy.

On examination the patient was comfortable breathing air with a Glasgow Coma Scale of 15. His vital signs were stable and a cardiorespiratory examination was within normal limits. Hamman's sign was negative and there was no subcutaneous emphysema. There was no neck stiffness, no rashes and he was afebrile.

Neurological examination did not elicit pyramidal drift or cerebellar signs. Tone, power, reflexes and gait were normal throughout. Serial sevens (subtraction) were assessed and he achieved 5/5. Hypoaesthesia was present over the lateral aspect of the foot. Visual acuity was 6/6 bilaterally and visual fields were normal. No nystagmus was found. Further cranial nerve assessment was normal apart from longstanding hearing loss on the left side present since childhood. An unenhanced CT brain was performed and no abnormalities were detected.

The case was managed jointly by a specialist in diving medicine and a consultant neurologist. Intravenous crystalloid infusion, paracetamol and aspirin were administered and the patient was transferred urgently to the hyperbaric unit where he was recompressed as per US Navy Treatment Table 6 for suspected DCI.

The patient's headache decreased in intensity during the 2nd oxygen period at 284 kPa (18 msw equivalent). At 192 kPa (9 msw equivalent), the patient was able to answer questions about the last three days correctly, with answers tallying with his diving buddy's version of events.

On completion of the US Navy Treatment Table 6, the patient mobilised with a normal gait. Sensation was found to be normal with resolution of the previous sign. He claimed to be feeling better and that his dizziness had resolved. He was transferred to a general acute hospital for an urgent magnetic resonance imaging (MRI) scan which detected no intracranial abnormalities. He remained neurologically intact and was discharged home. A follow-up transthoracic echocardiogram with agitated saline bubble contrast study showed normal cardiac function and no evidence of a right-to-left shunt, including no late passage of bubbles into the left atrium.

The patient was otherwise healthy and a non-smoker. Interestingly, he gave a history of apparent dysbarism secondary to breath-hold diving 11 years earlier experienced while training for a competition. On this occasion, he had dived to 40 msw for a TDT of 1 min 30 sec, followed by a 90 msw dive. Ten minutes after surfacing, he had experienced numbness in his left thigh and calf and was treated in a hyperbaric chamber for 2.5 hrs at 284 kPa with resolution of symptoms. These dives had been preceded by two dives to 95 msw and two dives to 40 msw in the prior 72 hours.

Discussion

We present a case of recurrent dysbarism in a professional breath-hold diver with no known risk factors. One specific form of dysbarism in breath-hold divers is DCI involving either AGE or DCS or both. Taravana syndrome is a form of DCS resulting from multiple deep breath-hold dives with short surface intervals. It was first reported in the Polynesian harvester divers of the Tuamotu archipelago, where '*Tara*' means '*to fall*', and '*vana*' means '*crazily*'.[5] The combination of significant depths and short surface intervals predisposed them to clinical manifestations of Taravana syndrome. The symptoms include headache, dizziness, hemiparesis/hemiplegia and disturbance of consciousness.[7,8]

In the present case, the temporal relation of the onset of neurological manifestations to a pattern of deep dives with short surface intervals provides reasonable indication that this was a case of DCI. However, the duration of the dives and the relatively mild symptoms somewhat contrast with classical Taravana syndrome described in the literature. A diving pattern resulting in Taravana syndrome typically involves 20 to 60 dives per hour for anywhere between 2 to 6 hours daily, unlike the presented case where a maximum of three dives were completed per day. Moreover, symptoms such as hemiparesis, speech disturbance and visual deficits typical of Taravana were absent.[9]

Taravana patients typically demonstrate ischaemic lesions on MRI that are compatible with neurological findings, before and after recompression,[10,11] yet the present case had normal MRI findings after recompression. Despite MRI being considered a relatively sensitive test, the diffuse and patchy nature of spinal and cerebral damage following DCS poses difficulty in identifying lesions definitive of neurologic involvement.[12] The case has thus been described as recurrent dysbarism, perhaps a manifestation of DCI, following breath-hold diving rather than being characterised as 'Taravana syndrome'.

One of the main presenting signs was an altered memory of events which interestingly appears to have occurred after an initial dysbaric injury and persisted in the three following days of diving until the diver sought medical assistance. Unfortunately, a formal cognitive assessment was not performed. Hence, the symptoms and response to hyperbaric oxygen treatment were subjectively reported.

Venous bubbling has been noted using echocardiography after repetitive deep breath-hold diving.[13] Our patient experienced two lifetime episodes of dysbarism, yet a right-to-left intracardiac shunt was not found. It is possible that an unidentified risk factor for dysbarism following breath-hold diving may exist. For example, it is possible that right-to-left shunting via intrapulmonary arteriovenous anastomoses (IPAVA) may have occurred in our patient despite an unremarkable follow-up bubble contrast echocardiogram. Interestingly, shunting via IPAVA may be a gas-dependent mechanism, whereby hypoxic conditions exacerbate the right-to-left shunting. Compelling evidence can be found in a study on healthy adults exposed to a gas mixture with a reduced inspired oxygen fraction of 10%. This led to opening of the IPAVA in all subjects at rest.[14,15] Furthermore, another study in which subjects were exposed to hypoxic conditions during exercise reported all participants exhibited a hypoxia-induced intrapulmonary shunt.[16] It is also possible in the present case that a PFO existed but was unidentified due to pitfalls during the transthoracic echocardiogram. For example, the Eustachian valve can divert bubble contrast away from the interatrial septum posing challenges in identification of a PFO. This phenomenon may be reduced by Valsalva, inspiratory sniff or abdominal pressure manoeuvres which provoke an increase in pressure in the right atrium.[17]

It is plausible that our patient experienced anterograde amnesia secondary to dysbarism, with symptoms persisting 72 hours later. In one study the impact of long-term training in breath-hold diving on neurocognitive function in three different groups of men stratified by apnoea performance was investigated. A negative correlation was reported between neurocognitive test performance and length of diving career, as well as between neurocognitive test performance and static apnoea duration. This indicated short-term memory loss associated with the years of apnoea training.[18] Sub-clinical or frank Taravana syndrome may be an underreported contributing factor to these findings, however more studies are needed in this regard.

An important differential diagnosis to be considered is pulmonary barotrauma leading to arterial gas embolism. Cases of arterial gas embolism following breath-hold diving are rare, but have been documented.[19,20] The onset of symptoms beyond five minutes and the absence of other signs and symptoms suggestive of pulmonary barotrauma in the history and clinical examination make this diagnosis less likely. Typically, patients with AGE present acutely with loss of consciousness, altered mental status, hemiparesis, seizures, or focal neurological deficits immediately after surfacing.[21]

Conclusion

Dysbarism in some form is a rare but well-documented complication of breath-hold diving. This phenomenon is typically associated with repetitive deep breath-hold dives interspersed by short surface intervals. Delayed presentation for medical advice may occur, in particular, if symptoms are mild. It is possible that an unidentified risk factor for dysbarism following breath-hold diving may exist especially in recurrent cases. Neurological symptoms following breath-hold diving merit prompt recompression in a hyperbaric chamber using hyperbaric oxygen, and fluid resuscitation. Further efforts are required to raise awareness in the freediving community about the nature of this disease and its treatment.

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