Decompression sickness after a highly conservative dive in a diver with known persistent foramen ovale: Case report

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Abstract

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A diver returned to diving, 15 months after an episode of neuro-spinal decompression sickness (DCS) with relapse, after which she had been found to have a moderate to large provoked shunt across a persistent (patent) foramen ovale (PFO), which was not closed. She performed a single highly conservative dive in line with the recommendations contained in the 2015 position statement on PFO and diving published jointly by the South Pacific Underwater Medicine Society and the United Kingdom Sports Diving Medical Committee. An accidental Valsalva manoeuvre shortly after surfacing may have provoked initial symptoms which later progressed to DCS. Her symptoms and signs were milder but closely mirrored her previous episode of DCS and she required multiple hyperbaric oxygen treatments over several days, with residua on discharge. Although guidance in the joint statement was mostly followed, the outcome from this case indicates that there may be a subgroup of divers with an unclosed PFO, who have had a previous episode of serious DCS, who may not be safe to dive, even within conservative limits.

Introduction

Diving with a persistent (patent) foramen ovale (PFO) has been linked with many forms of decompression sickness (DCS).[1–3] The hypothesis is that the usual venous bubbles generated after diving can cross through the PFO to the arterial circulation. Some of these bubbles pass into tissues that are supersaturated with inert gas which then diffuses into them causing amplification and resulting in DCS.[4]

A joint position statement (JPS) from the South Pacific Underwater Medicine Society (SPUMS) and the United Kingdom Sports Diving Medical Committee (UKSDMC) provides advice on diving with a known PFO; this includes the option to continue diving but within conservative limits.[5] The example given is to dive well within no stop limits, restrict depths to less than 15 metres, perform only one dive per day, use nitrox with air planning tools, lengthen a safety stop or decompression time at shallow stops and avoid heavy exercise or unnecessary lifting or straining for at least three hours after diving. Follow-up studies have found conservative diving lowers the risk of recurrent DCS in divers, with or without a right-to-left shunt.[6,7]

We report the case of a diver with a PFO, who, 15 months after recovering from neurological DCS after a rapid ascent, returned to diving and stayed mostly within the JPS recommended limits yet developed significant DCS.

Case report

The diver provided written consent for her case to be reported. This account is constructed through direct involvement with her acute management (WB) combined with information provided by colleagues and the diver, together with case note review.

INCIDENT ONE

In October 2018, a 28-year-old female diver undertook a weekend of diving near Oban on the west coast of Scotland. Her previous diving experience was uneventful and was estimated at just over 30 dives, all of them cold water and with a maximum depth of 35 metres' sea water (msw). Following two shallow dives the previous day (both < 10 msw) she performed a wreck dive to a maximum depth of approximately 20 msw and duration of 40 min but at the end of the dive she and her dive buddy became entangled in

the line of a surface marker buoy during its deployment and both made a rapid ascent (estimated at 30 to 60 m·min^{-1}) to the surface. On surfacing at 12:55, her buddy had symptoms consistent with DCS; he was given oxygen (O_2) on the boat and was subsequently treated with a standard Royal Navy 62 hyperbaric oxygen table (RN62), the Oban hyperbaric unit's routine 283.6 kPa (2.8 atmospheres absolute, 18 msw-equivalent) treatment table.

The female diver also received O_2 on the dive boat and some oral fluids before being transferred to Oban hospital accident and emergency (A&E) department. Her initial assessment, by a physician from the hyperbaric medical team, was unremarkable; she reported a headache that was present before the dive, and some mild discomfort to the posterior neck plus mild tenderness along the line of the trapezius muscle that were attributed to mechanical injury caused by the rapid ascent. As is normal practice in Scotland for divers who have had an uncontrolled ascent but no DCS, she was not recompressed but continued to receive normobaric O_2 for 4 h in A&E, during which there was no change to the previous symptoms. As at her initial presentation, these symptoms were not judged attributable to DCS so she was discharged under supervision of friends with a review planned for the next morning. Coincidentally, she was reviewed again at 21:30, when the group collected the buddy following his treatment; she was asymptomatic.

At 01:10 she developed paraesthesia in her right arm in ulnar distribution that was spreading and worsening in severity and she re-presented. She was recompressed at 04:25 on a RN62 modified with extensions. The treatment was eventful with episodes of vomiting and diarrhoea; she surfaced at 11:20 with residual symptoms. When reassessed at 17:00 she had developed new hyperparaesthesia in buttocks, thighs and legs consistent with deteriorating spinal DCS so was recompressed at 21:30 with a second RN62 modified with extensions. She surfaced with residua and received two once daily Comex 12 msw (Cx12) treatments over the following two days. Subsequently, mild balance impairment and lower back discomfort persisted but it was assessed maximum benefit had been gained and she was discharged. She later reported that the residual symptoms resolved over a number of weeks. Standard discharge advice from the Oban Hyperbaric Unit, for all divers who have suffered neurological DCS, is not to dive again but with the caveat of being tested for a PFO if continuing to dive.

In February 2019 she underwent examination by bubble contrast transthoracic echocardiogram (TTE), performed at a non-specialist centre. She was diagnosed with a right-to-left shunt caused by a probable PFO based upon the appearance of more than 30 bubbles in the left ventricle (LV) within three 3 beats, after the release of a Valsalva manoeuvre. Beyond this, no specific comment was made about the size of the shunt, or if there was an unprovoked shunt. She consulted a cardiologist in June 2019, who reported a past medical history of mild migraine with aura and a family history of PFO. Considering the rapid ascent to have been a clear provoking factor explaining the DCS, without having to invoke embolism across a shunt, the cardiologist advised that PFO closure was not indicated. She then consulted a UKSDMC-approved medical referee, in September 2019, who cleared her to dive with care using DCIEM air tables, or computer, to 15 msw on air and on nitrox below that.

INCIDENT TWO

She returned to diving in January 2020, 15 months after the first incident. Her first dive back was a shore based cold-water dive in a sea loch on the west coast of Scotland. She dived to a maximum depth of 12 msw with a bottom time of 30 min. She breathed air from surface to depth and during a controlled ascent to 6 msw at which point she switched to 70% nitrox. She made planned 3 min stops at both 6 and 3 msw before surfacing at a controlled rate at 13:30, with a total dive time of 40 min. There were no unplanned or adverse events during the dive. Whilst de-kitting, she accidently performed a Valsalva manoeuvre when bending and straining to remove tight fins. This was followed by a sharp, sudden onset occipital headache, which passed off rapidly, but no other symptoms.

At 16:40, having driven home with only minor altitude changes to a maximum of 200 m above sea level, she developed an itchy right shoulder and upper arm, but no rash. This progressed over about 90 min to include altered sensation in her right lower arm and hand with aching elbows and fingers. A home trial of oxygen at 20:45 made no difference but she felt the symptoms worsened when discontinued. At 22:00 she contacted the Scottish Hyperbaric Helpline and was brought to the hyperbaric medical unit in Aberdeen. Here, her symptoms were confirmed together with her history of neurological DCS and subsequent diagnosis of PFO. On examination the only abnormal finding was an unsteady sharpened Romberg's test, immediately falling to right. The rest of the neurological examination, including unprovoked Romberg's test and gait, was normal.

A diagnosis of neurological DCS with possible cutaneous and joint components was made and she was treated with the Aberdeen unit's standard 283.6 kPa treatment table – an un-extended US Navy Treatment Table 6, commenced at 02:00. After surfacing at 06:55 she was asymptomatic from her DCS and her sharpened Romberg's was normal. She was admitted to hospital for observation. Later that day she relapsed; at 16:30 she reported bilateral heaviness of her legs and "*unusual sensation*" in both thighs. This was very similar to, but milder than, the relapse she experienced after her first HBO treatment in 2018. Her sharpened Romberg's test was also unsteady again but there was no other neurological abnormality on examination. These symptoms were mild and stable but, in view of the similarity to 2018, it was decided further HBO was indicated. She was suffering some

troublesome pulmonary O_2 toxicity symptoms so, in the absence of deterioration, further HBO treatment was delayed until the next morning, to give her a longer air break. She then received the first of three daily Cx12 treatments. The second and third Cx12 treatments were given because new left-hand paraesthesia developed after the first Cx12 and the lower limb and balance symptoms persisted, although they were improving. After these treatments, the mild left-hand paraesthesia and subjective poor balance persisted but it was assessed that maximum benefit had been obtained so she was discharged with advice to stop diving. The residual symptoms settled over the ensuing two weeks, without further treatment.

Most strikingly, the pattern and timings of symptoms during this incident virtually mirrored those of her previous DCS, although of milder severity in the second episode.

Discussion

The DCS that developed after the first incident is fully explainable by the diver's unplanned rapid ascent causing autochthonous bubble formation in tissues, without having to postulate shunt across a PFO. However, in our experience, it is atypical for this to present so late after four hours of prophylactic surface O_2. As part of the treating unit's standard discharge advice, she was advised to have a bubble contrast TTE, if continuing diving, and this revealed a likely PFO with shunt by provocation following a Valsalva manoeuvre. The TTE was not done in a specialist centre and it is unclear if the bubble count of > 30 bubbles in the LV, within three beats of release of the Valsalva, was from a single frame or an overall total. Also, the standard method is to count bubbles in the left atrium (LA) rather than LV. A single frame count in the LA of > 30 would be taken by most specialists to indicate a large shunt.[8,9] This indicates the diver had, at least, a moderate and, likely, a large provoked shunt but the study would have been better done in a specialist centre as recommended by the JPS. A transoesophageal echo scan (TOE) can measure the size of the defect but TTE is the investigation of choice recommended by the JPS[5] and, in the UK, TOE is only likely to be used if a decision to close a PFO was being considered.

For the second episode of DCS, we postulate this was shunt related, but tissue inert gas load was low, so it is very unlikely to be caused by the mechanism of bubble amplification within supersaturated tissues, as is normally hypothesised.[4] Her dive followed the conservative diving approach by keeping to a maximum depth that was shallower than recommended as a conservative diving profile,[5,6,10,11] with a bottom time that was well within no-decompression limits (her bottom time was 120 min less than the no-decompression limit for 12 msw (150 min) following the DCIEM air decompression tables[12]), and with intentionally performed safety stops, using 70% nitrox, that were not required. This dive would have theoretically generated some gas supersaturation in her tissues, but it would have been low and short lived, as indicated by Repetitive Group 'B' on the DCIEM tables, had the dive been conducted without the safety stops. Shortly after surfacing, however, she did breach the JPS guidelines with an accidental Valsalva whilst de-kitting. We hypothesise that the occipital headache associated with this was indicative of a shower of venous inert gas bubbles passing through the PFO to the arterial side causing transient meningeal irritation and that, simultaneously, additional bubbles impacted other tissues, initiating the pathophysiological processes leading to DCS. Symptoms began some three hours after surfacing, which is longer than expected after usual shunt-related DCS,[1,13] but we submit that a different, and apparently slower, mechanism was in play with an arterial shower of bubbles alone being sufficient to provoke DCS in those areas damaged by the first episode. The similarity of the second DCS to her more severe previous one, suggests the presence of residual sub-clinical damage with vulnerability to further insult.

A previous study demonstrated that 14 of 19 divers with a 'grade 3' PFO (defined as a Valsalva provoked shower of bubbles too numerous to count in middle cerebral artery) generated detectable venous bubbles following a chamber dive to 30 msw, and six of these had arterial bubbles detected. This compared with divers in whom the PFO has been successfully closed where, although 11 of the 15 had venous bubbles detected, none had arterial bubbles.[14] In a deeper simulated dive to 50 msw in the same study, seven out of eight divers with a PFO had detectable venous bubbles, all of whom also had arterial bubbles but, although all five divers with a closed PFO generated venous bubbles, none had arterial bubbles detected. The dives in that study were deeper than the second dive in our case where the liberation of venous bubbles after surfacing would be expected to be low because of the conservative dive profile. However, we postulate from Honěk et al.,[14] that, if venous bubbling occurs after any dive, there is likely a significant chance of arterialisation across a PFO, particularly under provocation. The timing of the accidental Valsalva in this present case presumably coincided with venous bubbling, which then shunted to the arterial circulation.

Another cohort study compared divers with unclosed right to left shunt, who had been advised to dive conservatively, against those who had a closure procedure and found a higher risk of DCS in the former group.[7] Recent correspondence from Honěk's group, describing results from their DIVE-PFO registry, reports continuing incidences of 'unprovoked' DCS in divers with unclosed PFO but not in those who have had closure.[15] Both studies have limitations, they had a low number of end points, relied upon self-reporting by divers and do not describe the dive profiles associated with each DCS. However, they do demonstrate continued diving with a PFO carries an increased risk of DCS compared to diving after PFO closure.

In the case we report, other pathologies could have been considered in the differential diagnosis, such as cervical disc herniation or spinal cord pathology, but the diagnosis of DCS on each occasion was felt secure at the time, so these were not investigated. That DCS was the diagnosis would be strongly supported by the clear precipitating cause in the first incident and, on both occasions, by the proximity to diving, the pattern of evolution, response to hyperbaric oxygen, subsequent resolution of residual symptoms and absence of symptomatology before, between or since these incidents. It is possible that the diver had existing cervical cord pathology that predisposed to DCS. Spinal canal narrowing is more common in divers who have previously had DCS than those who have not.[16] Appropriate investigations and onward referral should be considered in divers who have suffered spinal cord DCS.

The JPS provides an important package of guidance[5] and this case illustrates how ambiguity can be introduced if it is not followed as a whole. In particular, PFO testing should be undertaken by centres well practiced in the technique who can provide definitive assessment of the significance of the shunt. The diver may have been better advised if this higher quality information had been available.

The JPS is based upon the available evidence but this, inevitably, only reaches level IIa at best.[5] The recommendations for divers with unclosed PFOs returning to diving following DCS are based on level IV evidence, expert consensus, and are founded upon the hypothesis of bubble amplification in supersaturated tissues. This may well apply to the majority of cases but even a single case that indicates it is not universally applicable is important. In the present case, it is the diver's second incident that casts doubt. It appears that, despite a very conservative dive, arterial bubbles embolised into tissues with a low inert gas load and this alone was sufficient to cause DCS, probably because of previous damage from an earlier, severe episode and possibly predisposed to by undiagnosed cervical cord pathology. There may be a subgroup of divers with a similar history who are not necessarily safe to dive, even within very conservative limits, with a PFO. In any case where the PFO is not closed, and the diver chooses to continue diving, this decision should be informed by high quality information about the shunt with expert interpretation. In addition, the necessity to avoid Valsalva manoeuvres following diving should be stringently reinforced.

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