Cutaneous decompression sickness after an air dive with oxygen breathed during decompression in a commercial diver with a persistent foramen ovale

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Abstract

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A 43-year-old commercial diver had cutaneous decompression sickness after a dive to 17 metres of seawater for 160 minutes breathing air with transfer under pressure and oxygen breathed during decompression in a dry chamber. He had worked as a commercial diver for 16 years without previous problems. A bubble contrast transthoracic echocardiogram showed a large atrial right-to-left shunt. His persistent foramen ovale (PFO) was closed using a transcatheter technique and he has returned to commercial diving. As far as we are aware, shunt-mediated decompression sickness has not been reported previously after a shallow air dive with oxygen breathed during decompression. The findings in this diver adds to the observation of occurrence of three episodes of cutaneous decompression sickness after dry hyperbaric exposure breathing air and decompression whilst breathing oxygen in two individuals with atrial right-to-left shunts.

Introduction

The incidence of some forms of decompression sickness (DCS), particularly cutaneous, neurological and cochlear-vestibular DCS, is increased in divers with a clinically significant right-to-left shunt.[1–5] After many innocuous dives, small numbers of venous bubbles are liberated but, during passage through the pulmonary capillaries, the gas diffuses from the bubbles into the pulmonary alveoli and bubbles do not reach the systemic circulation. A right-to-left shunt, which allows paradoxical bubble embolism, increases the risk of DCS after dive profiles with a theoretically low risk.[2,6] It is believed that when paradoxical bubble embolism enables bubble emboli to invade tissues supersaturated with inert gas (usually nitrogen) after a dive, the bubbles are amplified as the dissolved gas in the tissue passes down the pressure gradient from the tissue into the bubbles, which increase in size to cause DCS.[6,7]

Similar degrees of pressure reductions in dry conditions can also cause DCS, but it is rare when modern commercial decompression profiles are employed. A review of the small number of reported cases in which appropriate investigations were performed suggests that a right-to-left shunt is present in most cases when DCS occurs after low risk decompression profiles in non-divers, such as hyperbaric tunnel workers and hyperbaric chamber attendants.[8] The review also suggests that the manifestations of shunt-mediated DCS were predominantly neurological in those who decompressed whilst breathing air and always cutaneous in those who decompressed whilst breathing oxygen to reduce tissue nitrogen load.[8] As far as we are aware, there are no comparable observations of DCS when oxygen decompression is used for shallow air dives.

We describe a commercial diver who had cutaneous DCS after a dive breathing air at a depth of 17 metres of seawater (msw) for 160 minutes with transfer under pressure and oxygen breathed during decompression in a dry chamber. He was found to have a large right-to-left shunt across a persistent foramen ovale (PFO). After the PFO was closed using a transcatheter technique he has been permitted to return to commercial diving.

Case report

The diver has seen this report of his history, confirmed its accuracy and has given written consent to its publication.

A male diver age 43-year-old, height 170 cm and weight 81 kg, had occasional migraine but he never had aura. There was no other significant medical history. He had smoked intermittently since his teens, but had not smoked for some months before his DCS.

During the incident dive in July 2024, he wore a suit heated with hot water. At the end of the dive for 160 minutes at 17 msw breathing air, he returned to a diving bell at a depth of 12 msw. He transferred under pressure to a dry chamber where he decompressed breathing 100% oxygen. Decompression was with the French number 5 (air/oxygen 12 m table) decompression table for an 18 msw dive for 180 minutes, which consisted of breathing 100% oxygen for 10 minutes at 12 msw, 15 minutes at 9 msw and 15 minutes at 6 msw.[9] After the dive he did not undertake unusual or strenuous activity. It was his practise of not showering for three hours after surfacing – so he did not shower in the one-hour period between finishing decompression and onset of symptoms.

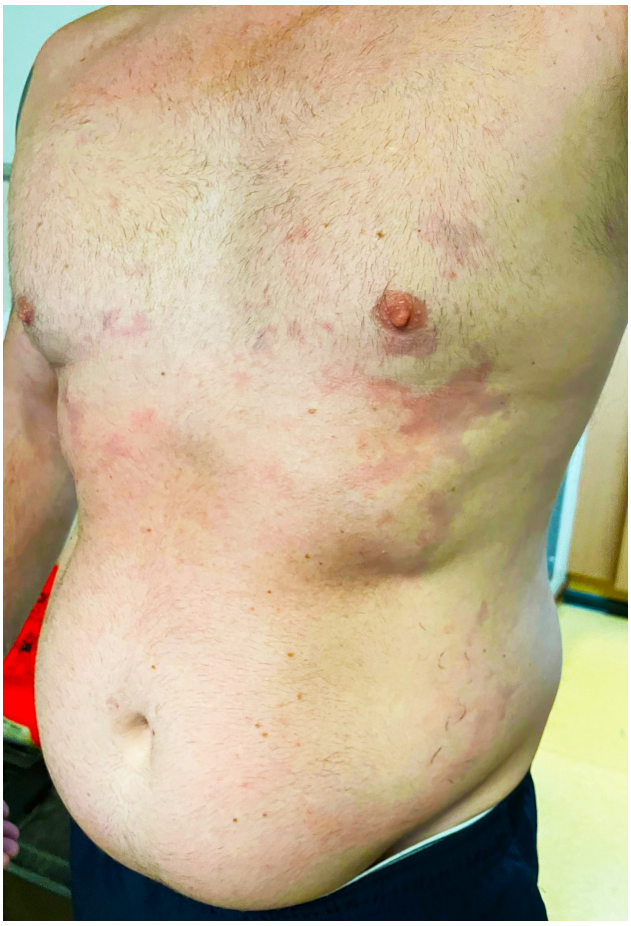
One hour after surfacing, he developed a pruritic erythematous marbled rash over the upper anterior abdomen which spread to his lower chest bilaterally associated with mild discomfort (Figure 1). There were no other abnormal findings and specifically neurological examination was normal. His rash and the associated discomfort completely resolved with a single recompression using US Navy Treatment Table 6. The other diver on the dive had no symptoms.

In the previous 16 years, the diver had performed more than 1,000 dives in the UK and abroad without any problems. During the first 10 years of his career, he had performed many relatively shallow air dives with long bottom times. During the next 18 months he performed more than 100 dives to depths of 30–40 msw with air as the breathing gas and surface decompression. The next period in his career involved dives used air or nitrox as the breathing gas, which only occasionally required in-water decompression stops, but when required the stops were performed using the same breathing gas. The exception during that period was a single 28-day trimix saturation dive with a storage depth of 60 m and maximum depth of 80 m. Thermal insulation on the dives consisted of dry suits, wet suits, water-heated suits or simple coveralls depending on the water temperature.

In 2024 he started a contract that required transfer under pressure with oxygen decompression. He did a total of ten dives. There were six days with one dive per day followed by one dive-free day. Then he did three dives in three days before the incident dive. All dives were similar to the incident dive, being at about the same depth as the incident dive with the transfer bell at 12 msw.

A transthoracic echocardiogram showed a structurally normal heart with a mobile interatrial septum. Bubble contrast injection when breathing normally showed a right-to-left atrial shunt with between 25 and 50 bubbles seen in the left heart on the stop-frame with the greatest number of bubbles. The shunt was slightly larger on a second bubble contrast injection with a sniff. On the third injection of bubble contrast with release of a Valsalva manoeuvre, the shunt was much larger with bubbles causing complete opacification of the left heart chambers. The patient was counselled about the implications of the finding and the options for management in accordance with the joint position statement on atrial shunts and diving.[10]

Figure 1
The rash of cutaneous decompression sickness in the diver



The diver had transcatheter closure of his PFO under fluoroscopy and transoesophageal echocardiography guidance using a 25 mm Amplatzer Talisman PFO Occluder (Abbott). Following the closure procedure, he took clopidogrel 75 mg daily for three months.

Ten weeks after the closure procedure transthoracic echocardiography showed that the alignment of the occlusion device was good. Six bubble contrast injections were given (one with normal breathing, two with sniffing and three with release of Valsalva manoeuvres). There was no evidence of a significant shunt. There were very occasional single bubbles seen in the left heart which might have been through pulmonary transit.

After cessation of clopidogrel, he was approved to return to diving. Since then he has performed 21 dives using air and nitrox as breathing gases. All were shallow and did not require decompression stops.

Discussion

The great majority of divers who have shunt-mediated DCS have followed a low-risk decompression profile that either required no stage decompression or required in-water decompression stops, which were performed correctly.[2,6]

The diver described in this report suffered cutaneous DCS after an uneventful dive that was within the limits of an accepted low risk decompression table. He was found to have a large right-to-left shunt across a PFO. He differed from the majority of divers who have shunt-mediated DCS because he transferred under pressure and performed the required decompression stops in dry conditions whilst breathing 100% oxygen. As far as we are aware this is the first report of shunt-mediated DCS after oxygen decompression in a diver. Though he performed a wet dive, his decompression was analogous to that of a hyperbaric tunnel worked or an attendant in a therapeutic hyperbaric chamber.

A recent case report of DCS in a hyperbaric worker that included a review of four additional cases in the literature found that shunt-mediated DCS is an important occupational risk for individuals with a large right-to-left shunt when working in hyperbaric air, but the manifestations of DCS differ in those who decompress whilst breathing oxygen compared with those who decompress whilst breathing air.[8] Prolonged oxygen breathing during decompression does not entirely eliminate venous bubbles in every decompression in every person and, in the two hyperbaric workers described, it was associated with cutaneous DCS on three occasions but not neurological DCS.[8] Decompression whilst breathing oxygen can be associated with migraine aura, which can be triggered by arterial bubbles without decompression.[11] In contrast, DCS was usually neurological when it occurred after dry decompression whilst breathing air.[8]

If further observations confirm that breathing oxygen during decompression protects against neurological DCS but not cutaneous DCS, it may provide a mechanism for reducing the more serious forms of DCS. It will also support the hypothesis that DCS resulting from paradoxical gas embolism is critically dependent on the peripheral amplification of bubble emboli in supersaturated tissues with a high nitrogen load, because the duration of oxygen breathing would have cleared nitrogen from tissues with rapid elimination half-lives such as neurological tissues, but not from tissues with slow elimination half-lives such as subcutaneous tissues.[6,7]

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