Recurrent cutaneous decompression sickness in a hyperbaric chamber attendant with a large persistent foramen ovale

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Abstract

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A 41-year-old female nurse had cutaneous decompression sickness on two occasions after acting as an inside chamber attendant for patients receiving hyperbaric oxygen. She breathed air during the treatments at pressures equivalent to 14 and 18 metres of seawater, but each time she decompressed whilst breathing oxygen. Latency was 2.5 hours and one hour. She was found to have an 11 mm diameter persistent foramen ovale. It was closed and she returned to work without recurrence of decompression sickness. Review of the literature suggests that shunt mediated decompression sickness is an important occupational risk for individuals with a large right-to-left shunt when working in hyperbaric air, but the manifestations of decompression sickness differ in those who decompress whilst breathing oxygen compared with those who decompress whilst breathing air.

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] It is believed that a right-to-left shunt permits paradoxical embolism of venous bubbles that form after decompression from some dives and, if those bubble emboli invade tissues supersaturated with inert gas (usually nitrogen), the bubbles are amplified as the dissolved gas in the tissue passes down the concentration gradient from the tissue into the bubble.[6,7]

When there is shunt-mediated DCS, a large persistent foramen ovale (PFO) is responsible in about 88% of cases, an atrial septal defect in about 5% of cases and pulmonary shunts in about 7% of cases.[8]

A PFO is present in 27% of the population, but only individuals with a PFO that is large enough to permit significant numbers of venous bubbles to shunt right-to-left are at risk of shunt-mediated DCS.[3,4] The median diameter of atrial shunts that cause shunt-mediated DCS is 10 mm.[8] In contrast, only 1.3% of the population have an atrial shunt that is 10 mm diameter or greater.[8]

Shunt-mediated DCS commonly occurs after a dive profile that is considered low risk and that rarely cause DCS in divers who have no right-to-left shunt, but the dive profile has to be one that liberates venous bubbles. [2,6,7] In contrast, in amateur divers who have decompression sickness but have no right-to-left shunt, the preceding dives are usually provocative and / or deep.[2–4]

There are also case reports that describe shunt-mediated decompression sickness after hyperbaric exposure in dry conditions when working as an inside attendant in a therapeutic hyperbaric chamber, in compressed air tunnel work and in hyperbaric factory work.[9–12]

This report describes recurrent cutaneous DCS after acting as an inside chamber attendant for patients receiving hyperbaric oxygen treatment.

Case report

This patient has consented to publication of this case report. She has reviewed the description below and agreed to its accuracy.

A female nurse aged 41 years, height 162 cm and weight 76 kg, was referred because she had two episodes of cutaneous DCS in early 2002. Each episode occurred after she had been an attendant in a hyperbaric chamber for patients receiving hyperbaric oxygen. She had worked at a hyperbaric unit for 18 months and she usually acted as an

attendant in a chamber twice each week. Her first episode of DCS occurred after one of the standard hyperbaric treatments given regularly in that institution to patients having hyperbaric oxygen therapy. The second episode occurred after a treatment used less routinely.

Her first episode of DCS occurred after a hyperbaric treatment at 243 kPa pressure (equivalent to 2.4 bar, 14 m of seawater [msw] pressure) for 90 minutes. Whilst the patients breathed oxygen at 'depth', she breathed air, but she switched to 100% oxygen during decompression. Decompression took eight minutes from 243 kPa (14 msw) to 132 kPa (3 msw), with a five-minute stop at 132 kPa (3 msw) and then one minute for the ascent to surface pressure. About 2.5 hours after decompression, she developed an itchy and sore mottled rash between her shoulder blades. A colleague who had seen divers with cutaneous DCS told her that the rash looked like cutaneous DCS, but she did not report it to senior chamber staff and she was not recompressed. The rash resolved in eight hours.

The second episode occurred after she acted as the chamber attendant for a single patient with carbon monoxide poisoning who was treated with Royal Navy Treatment Table 60. She breathed air during the hour at 284 kPa (18 msw) and until 15 minutes into the decompression when, at 192 kPa (9 msw), she switched to 100% oxygen for the remaining 15 minutes of decompression. One hour after decompression she developed itching over the right side of her body, which progressed to a florid mottled purple and pink rash typical of cutaneous DCS from her right hip to her right shoulder. Cutaneous DCS was diagnosed by a senior hyperbaric physician. There were no other symptoms or signs. She was rapidly recompressed using Royal Navy Treatment Table 61 (US Navy Treatment Table 5). There was rapid and almost complete resolution of itching and rash. After the treatment she had minimal residual skin discolouration over her hip.

She had a history of infrequent attacks of migraine with aura, but no family history of migraine. There was no other relevant medical history. She smoked an occasional cigarette (less than four per week).

Transthoracic echocardiography with bubble contrast showed a very large right-to-left shunt without provocative manoeuvres, with shunting seen during the inspiratory phase of normal respiration consistent with an atrial shunt. After counselling about options, she elected to have trans-catheter closure of her atrial shunt.

An 11 mm diameter PFO was closed using a 25 mm Amplatzer PFO device in September 2002. She was treated with aspirin for six months and clopidogrel for one month. Following the procedure she complained of palpitations which were the result of atrial ectopic beats. They resolved within two months after a short course of treatment with bisoprolol. Two months after the closure procedure, she had transthoracic echocardiography with six bubble contrast injections and provocative manoeuvres and there was no evidence of any residual shunt.

She returned to work as a chamber attendant for hyperbaric treatment and had no recurrence of DCS. In 21 years following the closure procedure, she has not had any attack of migraine but has had infrequent and minor visual aura.

Discussion

In the past, high incidences of DCS were described following occupational hyperbaric exposures which cannot be justified these days. For example, as late as 1971, Ghawabi and colleagues reported a DCS rate of 0.97% after caisson workers were exposed to air pressures equivalent to 28 msw for up to six hours and 25 msw for up to eight hours.[13] The authors reported that only seven of the 55 workers had no episode of DCS during the project, whereas 37 of the 55 (67%) of the workers experienced cardiopulmonary DCS ('the chokes') and 44% had radiological evidence of bone infarction.[13] The high incidence rates are consistent with unsafe profiles and, because nearly every worker had DCS at least once, there is no need to postulate the role of physical predisposition to DCS, such as right-to-left shunts.

Not surprisingly, occupational hyperbaric exposures have become more conservative, but DCS has not been entirely eliminated.

More recent publications report small numbers of episodes of DCS in workers in hyperbaric chambers, but most reports fail to provide detailed information about the pressure exposure profile or gases breathed by the affected employee or the clinical manifestations of DCS.[14] Very few reports provide the results of tests to detect whether those affected had a right-to-left shunt.

There are three reports of DCS after dry hyperbaric exposure when the individual breathed air during the hyperbaric exposure and also during decompression, and the affected individual had a right-to-left atrial shunt, either a PFO or an atrial septal defect.[9–11]

Johnson-Arbor reported a 50-year-old man who had numerous uneventful decompressions (sub-atmospheric and after diving during military service), but he had two episodes of DCS when working as an inside hyperbaric chamber attendant.[9] One was cutaneous DCS after treatment of a patient at 608 kPa (50 msw), but details of the profile and gases breathed are not provided. A second episode of DCS occurred after the chamber attendant breathed air at 223 kPa (12 msw) for two hours and also breathed air during decompression: he did not breathe oxygen at any time during the treatment. Within 10 minutes of surfacing, he became irritable and then had progressive ascending weakness and paraesthesia of both legs with a sensory level at T7. Definite spinal and probable cerebral DCS was diagnosed. There was recovery following treatment with US Navy Treatment

Table 6. Subsequently transthoracic bubble echocardiography showed a large atrial shunt.

Diederich and colleagues reported a 32-year-old man who worked packaging materials in a tank pressurised to 223 to 243 kPa (12 to 14 msw) for three to four hours, four times each week.[10] The decompression procedure used is not stated. There is no comment about oxygen breathing and that seems to be unlikely. The report said "*When brought back to atmospheric pressure, he developed headache, chest tightness, nausea, arthralgias, and vision changes, which he described as 'looking through a kaleidoscope'*." This visual disturbance is consistent with a migraine visual aura. Unfortunately, the intervals between surfacing and onset of different symptoms are not stated but it appears to have been soon after decompression. He found himself stumbling due to acute right-sided weakness, which spontaneously resolved. Upon returning home, he noticed an extensive rash overlying his torso consistent with cutaneous DCS. An echocardiogram showed the presence of a PFO. The precise type of echocardiogram is not stated and no information is provided about the size of the shunt.

Kütting and colleagues reported that in 2002 a 44-year-old tunnel worker had neurological DCS with onset 10 minutes after 42 minutes at a pressure equivalent to 375 kPa (27 msw).[11] None of his colleagues had DCS. It is also reported that he had recurrent episodes of DCS in the previous 15 years as well as episodes of 'blurred vision' after hyperbaric exposures. The visual disturbances may have been migraine aura. All these episodes of DCS occurred in years when it was very unlikely that oxygen was breathed during decompression. He was found to have an atrial septal defect.

The prominent manifestations of DCS in the three individuals, who decompressed whilst breathing air, were neurological though some also had cutaneous DCS. Where stated the onset of neurological DCS was soon after surfacing: in two cases onset was about ten minutes after surfacing. This is consistent with the peak latency of shunt-mediated neurological DCS in divers.[4]

In contrast, the patient described in this report is one of two where the casualty suffered DCS after decompression whilst breathing oxygen. In both cases, the casualty had cutaneous DCS. Colvin and colleagues reported a 32-year-old male tunnel worker who had DCS after oxygen decompression from only his third pressure exposure.[12] He worked in air at pressure equivalent to 355 kPa (25 msw) for 2.5 hours followed by one hour and 19 minutes of oxygen decompression using the Swanscombe Table. Approximately two hours after decompression he started to develop extensive cutaneous DCS with visual disturbance consistent with a migraine visual aura and pain in his left shoulder: the rash was present in the skin over the back of the left shoulder. Joint pain is not a feature of shunt-mediated DCS except when there is shoulder pain with a rash over the painful shoulder.[3] A transthoracic echocardiogram with bubble contrast showed a very large atrial right-to-left shunt at rest. He was found to have a 9 mm diameter atrial septal defect, which was closed.

Colvin and colleagues also reported that field testing with Doppler ultrasound showed that use of the Swanscombe Table liberates small numbers of venous bubbles in some workers.[12] Evidence supporting paradoxical gas embolism in the case described by Colvin and colleagues was that he had a visual aura consistent with migraine aura after his hyperbaric exposure at a time when the brain would not be supersaturated because it is a fast tissue.[12,15] Migraine visual aura can be precipitated by bubbles passing across a right-to-left shunt and it does not require supersaturation of neurological tissues, because it sometimes occurs after bubble contrast echocardiography when there is no supersaturation.[15]

The patient described in this report had two episodes of cutaneous DCS after acting as an inside attendant breathing air during hyperbaric treatments of patients at 243 and 284 kPa (14 and 18 msw). She breathed 100% oxygen during decompression on each occasion. Onset of symptoms was 2.5 hours and one hour after finishing oxygen decompression. Her bubble contrast echocardiography showed a large atrial right-to-left shunt that was found to be across an 11 mm diameter PFO. It was closed. She had a history of migraine with aura, which is associated with large right-to-left shunts.[15]

The pressure-time profiles of the two chamber dives that resulted in cutaneous DCS in the patient described in this case report were comparable to profiles demonstrated to liberate venous bubbles in some hyperbaric chamber attendants even when there was a longer period of oxygen breathing during decompression.[16,17] For example, Cooper and colleagues reported that after breathing air for 90 minutes at 243 kPa (14 msw) with 20 minutes decompressing whilst breathing oxygen, 32% of subjects had moderate to high numbers of venous bubbles on Doppler.[16] Walker and colleagues reported that 44% of exposures liberated venous bubbles after subjects breathed air at 203 kPa (10 msw) for 90 mins followed by 30 mins breathing oxygen during ascent to the surface.[17] Sixty-eight percent of exposures liberated venous bubbles after subjects breathed air at 283 kPa (18 msw) for 60 mins followed by 30 mins breathing oxygen during ascent to the surface.[17]

As far as we are aware, the patient described in this report and the patient in the paper by Colvin and colleagues are the only cases in which DCS occurred in individuals that had dry occupational hyperbaric exposure with oxygen decompression. The information available suggests that despite oxygen decompression, the profiles would liberate venous bubbles in some individuals.[12,16,17] Both individuals had large atrial defects (an 11 mm diameter PFO and a 9 mm diameter atrial septal defect). They each had cutaneous

DCS, which is commonly shunt-mediated, with onset times between one and 2.5 hours after surfacing. One had migraine visual aura at the time of their cutaneous DCS which is suggestive of paradoxical gas embolism.[15] However, in contrast to the individuals with atrial shunts who had DCS after dry hyperbaric exposures but who decompressed whilst breathing air, the two individuals who decompressed breathing oxygen did not suffer neurological DCS.

We cannot draw firm conclusions from small numbers of observations, but the data from these five case reports are consistent with the hypothesis that shunt-mediated DCS requires more than paradoxical gas embolism. It has been hypothesised that the additional requirement is amplification of embolic bubbles in supersaturated tissues as dissolved gas in the tissue diffuses into the bubbles.[6,7]

An alternative hypothesis is that shunt-mediated cutaneous DCS is not the result of amplification of bubble emboli in subcutaneous tissue, but is caused by paradoxical gas embolism to the brain that results in alterations in vasomotor control to produce the mottled skin rash of cutis marmorata, which has visual similarities to livido reticularis.[18,19]

Kemper and colleagues claimed that this hypothesis is supported by the incidental observation during experiments to investigate the effects of cerebral air embolism in which anaesthetised pigs developed a mottled skin rash, which bore a resemblance to the rash of cutaneous DCS in divers.[18,20] The development of the rash in the pigs was not reported in the original paper by Weenink and colleagues.[20] Later, Kemper and colleagues reported that in the experiments, each of the 22 pigs developed the rash within minutes of introducing air into the cerebral circulation.[18] However, the circumstances and findings in the pig experiments differed in many ways from those in divers with cutaneous DCS.[20–22]

The pigs (weights approximately 40 kg) were anaesthetised with ketamine and midazolam, paralysed with pancuronium and given atropine.[20] The experiment involved injection of 5.6 $\pm$ 1.3 ml of air directly into the ascending pharyngeal artery (equivalent to an internal carotid artery in humans) with the artery occluded by means of an inflated balloon.[20] The pigs had not been exposed to high ambient pressures before the air was injected. Therefore, their tissues were not supersaturated with gas. They were ventilated with a F_iO_2 of 0.4 during the experiments, including a stabilisation period of at least one hour.[20] Therefore the tissue partial pressures of nitrogen in the pigs in the experiments would have been lower than in a person or pig breathing air and lower than in a diver soon after a dive. The tissue partial pressure of nitrogen would also have been lower than the partial pressure of nitrogen in the air injected into the animals' cerebral vessels. As a result, the experimental model was more in keeping with cerebral arterial gas embolism in a non-diver occurring during medical interventions (for example, during cardiac surgery). In these clinical situations, a rash similar to cutaneous DCS is not a characteristic finding. Nor is the rash of cutaneous DCS a characteristic feature of cerebral arterial gas embolism in divers.

In the pigs, the rash had a wide distribution over the cheeks, neck, thorax, abdomen and thighs.[22] In divers, cutaneous DCS is usually localised to areas of the body with significant amounts of subcutaneous adipose tissue, such as over the trunk and/or thighs. In individual divers, who have recurrent episodes of cutaneous DCS, there is often a similar distribution of the rash on each occasion.

In the pigs, the rapid development of the widespread rash when air was injected coincided with large and rapid increases in intracranial pressure and a severe deterioration in cerebral metabolism.[18,20] Some pigs died immediately and the rest were euthanized. Kemper and colleagues have confirmed that if any of the animals had survived the experiments, they could have had severe neurological deficits.[22] We believe that the magnitude of the effects on intracranial pressure and metabolic derangement make it certain that if any pig survived they would have had severe neurological injury.

It is agreed that the associated rapid increases in heart rate and blood pressure in the pigs could have been the result of a catecholamine surge caused by the severe cerebral injury.[21,22] Livido reticularis is described in patients with phaeochromocytoma.[23,24] Therefore, we believe that the widespread rash observed in the pigs during the experiments reported by Weenink and colleagues was the result of the severity of the neurological injury they suffered.[20] In contrast, most divers who have cutaneous DCS do not have even mild neurological manifestations, not even when they have multiple episodes of cutaneous DCS.

There have been some attempts to demonstrate bubbles in skin rashes after diving.

Garcia and Mitchell reported ultrasound examination of the skin of four divers 4–5.5 hours after surfacing from relatively innocuous dives and 2–4.5 hours after the onset of cutis marmorata.[25] In each case, bubbles were detected passing through the microvasculature of the affected subcutaneous tissue, but not through adjacent normal skin. Each diver was later found to have a right-to-left shunt. These observations do not provide conclusive evidence about causation, because the rash was present before the bubbles were detected. Therefore, it is possible that the detection of the passage of bubbles through the affected subcutaneous tissue but not in unaffected skin could have been the result of differences in cutaneous blood flow in affected and unaffected tissues. In addition, each diver had neurological DCS at the same time as they had cutis marmorata. However, it is also possible that in affected subcutaneous tissues, bubbles were more easily detected because their size was increased by bubble amplification, but was not in unaffected skin.

It might appear that the failure of Qing and colleagues to detect bubbles in skin lesions of pigs after simulated dives in hyperbaric chambers is at variance with the report by Garcia and Mitchell, but the dive profiles were much more provocative.[26] Thirteen pigs were compressed to 507 kPa (40 msw) for 35 minutes followed by 11 minutes decompression. All animals developed widespread skin lesions and two died suddenly from what appears to have been cardiorespiratory DCS, which is consistent with a highly provocative dive profile. Transthoracic echocardiography was performed at various times from 30 minutes until six hours after surfacing. The bubble grade was greatest on the 30-minute images, when there was 'white-out' of right heart chambers in most pigs. That was also in keeping with a highly provocative dive profile. No bubbles were seen in the left heart chambers at any time. So it is unlikely that there was a right-to-left shunt, which makes it unlikely that the rashes in these pigs were the result of either paradoxical gas embolism to either the skin or the brain. As far as the authors could determine, the pigs that survived the experiment had no neurological injury. The rashes in the pigs may have had the same pathogenesis as cutaneous DCS after provocative dives in amateur divers who do not have a right-to-left shunt.

Additional observations support the hypothesis that paradoxical gas embolism with bubble amplification in subcutaneous adipose tissue can cause DCS and cannot be explained by a neurological mechanism secondary to cerebral gas embolism. Breast pain and a painful lipoma are described as manifestations of shunt-mediated DCS, but it is difficult to explain those as a result of cerebral gas embolism.[3,27]

Of 39 amateur divers that had lymphatic DCS, 30 had a significant right-to-left shunt and their dives were generally unprovocative.[28] In contrast, the remaining nine divers with lymphatic DCS either had no shunt or had only a small shunt but had performed deeper dives on trimix. Clearly lymphatic DCS cannot be explained by a cerebral insult.

The observations in individuals who had DCS after hyperbaric exposure in dry conditions may aid understanding of the role of tissue supersaturation in shunt-mediated DCS. A period of oxygen breathing during decompression allows tissues with rapid nitrogen elimination half-lives, specifically neurological tissues, to desaturate before venous bubble formation and paradoxical gas embolism occur. That means those tissues will not amplify bubble emboli. In contrast, tissues with a slow nitrogen elimination half-life, such as skin and subcutaneous tissue, remain supersaturated and able to amplify bubble emboli after decompression whilst breathing oxygen. In fact, prolonged oxygen breathing during decompression, as described by Colvin and colleagues, may actually slow elimination of dissolved nitrogen from some tissues, such as subcutaneous fat because of the vasoconstrictor effects of high partial pressures of oxygen.[29]

Although these are only a small number of cases, they add to the evidence refuting the hypothesis that cutaneous DCS is the result of a neurological mechanism caused by gas embolism to the brain.

It is difficult to draw conclusions from a small number of case reports, but these limited data suggest that individuals, who have a large atrial right-to-left shunt, either a PFO or an atrial septal defect, make up the majority of people who have DCS as a result of working in modern hyperbaric facilities. In each case, their manifestations of DCS were similar to manifestations of shunt-mediated DCS commonly observed in scuba divers.

Therefore, the guidance produced by SPUMS and UKDMC for assessment of divers who might have a PFO is also applicable to other hyperbaric workers such as inside chamber attendants and hyperbaric tunnel workers.[30]

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