An element of doubt: four divers with acute neurological problems

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Decompression illness, case report, medical conditions and problems, death

Abstract
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Decompression illness (DCI) presents in a multitude of guises, and divers may present with concomitant disease processes that can confuse diagnosis and management. The cases described are of four divers who were referred for hyperbaric oxygen therapy with an initial working diagnosis of neurological DCI, These illustrate the diagnostic and management dilemmas that may arise. One diver was diagnosed with migraine, whilst in one no final diagnosis was made but DCI was considered to be unlikely. Two patients died, one from a brainstem infarct due to severe cerebrovascular disease and the other from an acute cardiac event secondary to viral myocarditis.

Introduction

Decompression illness (DCI) presents in a multitude of guises. Often neurological lesions are multiple, making diagnosis difficult. Concomitant disease processes may be present in the individual diver to compound this. Recently, the case was reported of a patient with eosinophilic meningitis who was referred for hyperbaric treatment.[1] Here four divers are described who presented to the same regional hyperbaric unit with an initial working diagnosis of neurological DCI in order to illustrate the diagnostic and management dilemmas that may arise.

Case One

A 39-year-old, fit, professional underwater cine photographer diving in a remote area developed two episodes of transient visual disturbance each lasting five minutes about half an hour after a single nitrox (40/60 mix) dive to 22 m for 25 minutes. These were described as a hazy zigzag arc at 11 to 3 o'clock just above visual focus, progressing into a more widespread blurred patch in the temporal visual field, stronger in the right than the left eye but present bilaterally. As these episodes were brief he took no action. This phenomenon recurred the next day whilst crossing a mountain pass at above 800 m altitude. After two further episodes, each worse than the last and accompanied by headache, he presented in his home town two days post-dive, and was air evacuated to the regional hyperbaric unit.

On admission, his only symptom was a "pounding" right frontal headache unrelieved by anti-inflammatory analgesics. Previous medical history was unremarkable except for one year earlier when, after a period of intensive diving lasting several weeks, he developed intermittent neck pain and tingling in the arms which had persisted. At the time of presentation, he was awaiting MRI investigation for this.

On examination, he was fully conscious and orientated and in no distress. No visual field abnormalities were detectable, and the fundi were normal, with no evidence of focal emboli or oedema. The cranial nerves and power, tone, reflexes and sensation in the limbs were all normal. Sharpened Romberg's test was stopped at 60 sec on the first attempt.

In discussing the situation with the patient it was considered that his symptoms were unlikely to be due to DCI. However, given his high public profile, he underwent a US Navy Table 6 hyperbaric oxygen treatment, which relieved his headache. This was followed the next day by a further short oxygen treatment at 283 kPa. During this period he remained entirely asymptomatic and at no stage were there any identifiable objective neurological signs.

Two days after discharge he reported a further similar episode of transient visual changes. MRI of the neck showed no abnormality. Since then he has had further episodes associated with a "cracking" headache. He was prescribed sumatriptan succinate (Imigran), which provided rapid relief if taken at the start of an attack. During a recent intensive diving programme over several weeks in the Southern Ocean for a new film he had no further attacks. He now reports a family history of migraine, in his mother, of which he was not previously aware.

Case Two

A 29-year-old recreational diving instructor with 12 years' diving experience did two dives on the first day to maximum depths of 37 m and 30 m, and a single 25 m dive lasting 50 minutes and including a long safety stop in shallow water on the second day. Diving conditions were described as "lovely", all three dives being very relaxed and well within safety limits.

Nine hours post-dive, whilst on night shift as a baggage handler, he suffered an "explosive" onset of severe "stabbing" left retro-orbital and frontal headache, photophobia, blurred vision, slurred speech and left-calf pain. According to eyewitnesses, he "collapsed" with severe shaking and hyperventilation.

On admission at about 0200hr, his headache had eased to a score of 3 out of 10 compared with the initial severity. He denied any previous medical history. He was a smoker and indulged in binge drinking at weekends. There was a family history of polycystic kidney disease. On examination, he was fully conscious and orientated, with a mini-mental score of 30 out of 30. Neurological examination was normal apart from a subjective, patchy and inconsistent diminution of light touch and pinprick sensation in the left arm and leg. There was no neck stiffness.

It was considered unlikely his symptoms were due to DCI, and he was admitted to hospital for observation and urgent investigation, but not for hyperbaric oxygen treatment (HBO_2). In the morning, the headache had improved considerably. Physical examination was essentially unchanged with possibly diminished pinprick sensation on the left as above, and minimally reduced power in the left arm below the elbow.

Brain CT scan and lumbar puncture were normal. The case was discussed again with the Hyperbaric Unit. It was felt that a diving-related illness was extremely unlikely, but it was agreed that he be given a short HBO_2 treatment at 242 kPa. This resulted in improvement of his symptoms. A further treatment was given the following day, by which time he still had a mild headache but was otherwise well.

At this stage his previous medical records came to hand. At age 15 he had been admitted to hospital following collapsing on the beach secondary to a possible seizure. Investigations at that time, including a CT scan and EEG were normal. After this episode he continued to have headaches and episodes of clumsiness and "loss" daily for some weeks, but was not placed on any medication. At the ages of 23 and 26 he was admitted with unexplained neurological symptoms including severe headache that were attributed to (unconfirmed) viral meningitis. No final diagnosis was made.

Case Three

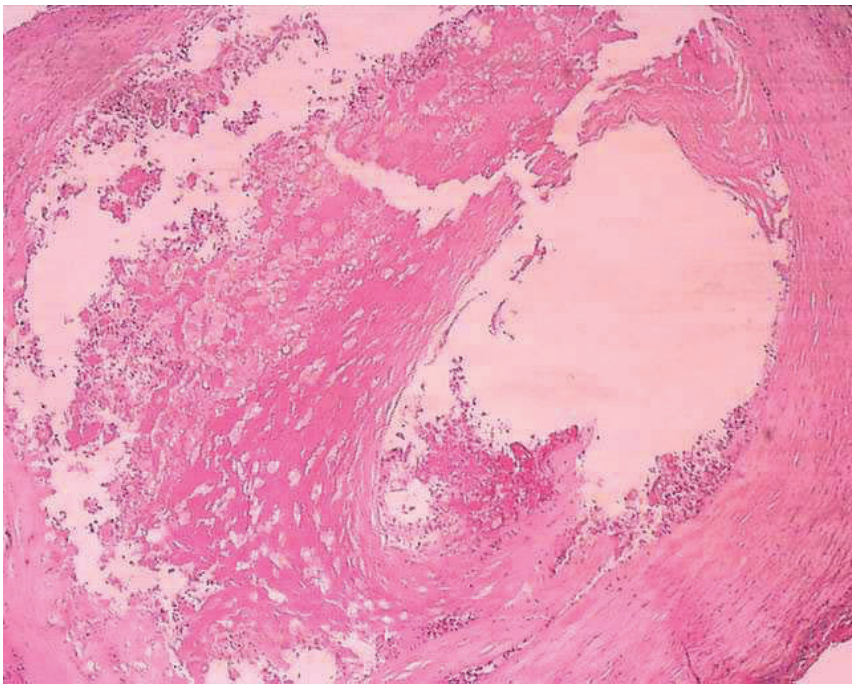
A 47-year-old experienced diver undertook an apparently uneventful solo dive for crayfish to 26 m for approximately 17 to 20 minutes. On boarding the boat a friend noted his mouth to be asymmetrical and he appeared drowsy. A quarter of an hour later he developed left-sided weakness and consciousness became impaired. When retrieved by paramedics about an hour later on shore, his Glasgow Coma Scale (GCS) score was 8 out of15.

On admission about an hour later, he was fully conscious and cooperative, but then rapidly deteriorated again in the Emergency Department with aphasia and a fluctuating level of consciousness. Examination was curtailed to commence recompression on the assumption that he had severe early-onset neurological DCI. The only known medical history at the time of admission was that he was a heavy smoker and had hypertension for which he was on some type of medication.

He underwent a US Navy Table 6 HBO_2 treatment during which his neurology continued to fluctuate markedly and bizarrely, but with a steadily worsening picture. At times he was talking and appeared rational, at others barely rousable, whilst both left and right transient hemiparesis were observed. The treatment was not extended or deepened, as there was a growing conviction that he was not suffering from DCI. After his treatment he was transferred to the ward where he rapidly became deeply unconscious with a GCS of 3 out of 15. He was intubated and transferred to the intensive care unit for life support.

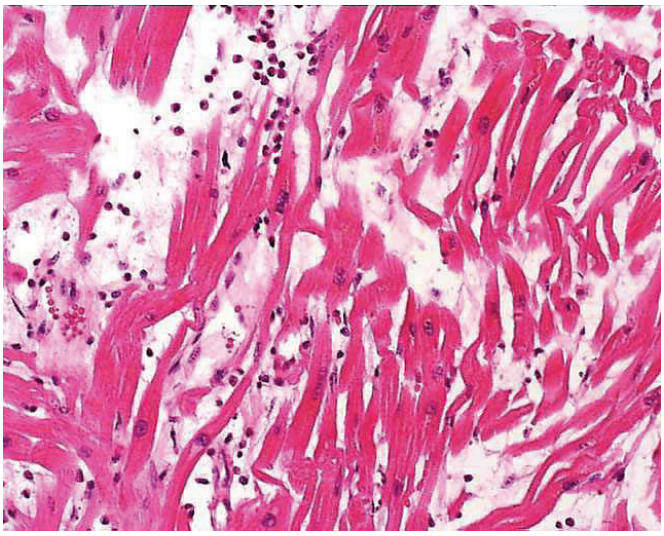
Further history became available from the family. He had been due for neurological assessment and brain CT scan that week for recurrent transient ischaemic attacks, including episodes of aphasia. A CT scan showed large areas of infarction of his brain, particularly the cerebellum and brainstem, but no gas. He was subsequently pronounced

FIGURE 1
CROSS-SECTION OF A CEREBRAL ARTERY FROM CASE THREE, SHOWING EXTENSIVE ATHEROMATOUS DEGENERATION OF THE WALL WITH RECENT THROMBUS FORMATION WITHIN THE NARROWED VESSEL LUMEN
(original magnification x 40)

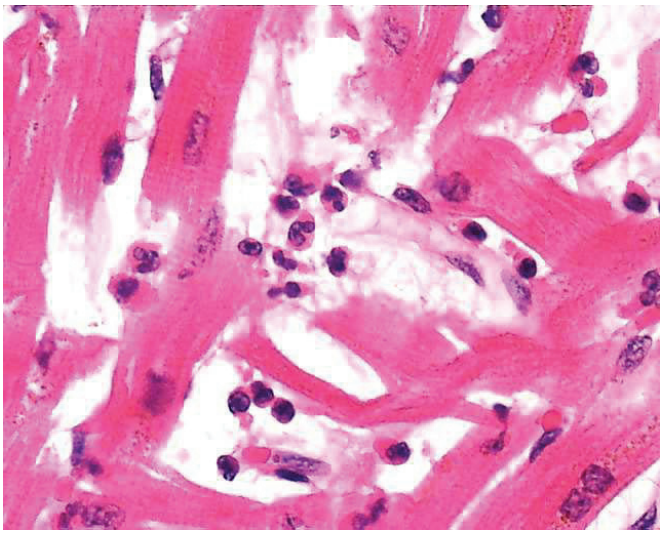


FIGURES 2a and 2b
THE ABNORMAL FOCUS OF MYOCARDIUM FROM CASE FOUR, SHOWING LOSS OF NORMAL PATTERN WITH POLYMORPH, MACROPHAGE AND LYMPHOCYTE INFILTRATION
(original magnifications: 2a x 200; 2b x 400)

2a



2b



brain dead and, following discussion with the family, life support was withdrawn. During organ donation, he was found to have widespread atheroma throughout his aorta and coronary vessels. A coroner's autopsy also showed widespread atheroma of his cerebral vasculature with recent in-situ thrombus consistent with the massive brainstem infarct. There was no evidence of vascular air embolism.

A typical atheromatous plaque with recent thrombus in one of his cerebral arteries is shown in Figure 1.

Case Four

This previously healthy, 41-year-old, experienced female diver was diving with her diving-instructor father. They

swam from the shore and then descended to the bottom in about 3 m depth. She indicated that she felt unwell and immediately surfaced. Whilst swimming to shore she collapsed and lost her mouthpiece and her father towed her to shore. On reaching shore, he confirmed that she was pulseless, and commenced basic life support. This was continued by he and bystanders for about half an hour until an advanced ambulance and medical team arrived by helicopter. She was intubated and a high-dose adrenaline infusion was commenced. Sinus rhythm was re-established with a reasonable cardiac output. An arterial gas embolism was suspected, and the regional base hospital requested an air evacuation for hyperbaric oxygen therapy. Whilst awaiting air transport, a brain CT scan revealed gross cerebral oedema, but no gas.

On arrival she was deeply unconscious; GCS score of 3 out of15. Spontaneous respiratory activity was present but no cough or gag reflex. Her pupils were fixed and dilated. Her cardiovascular status was stable on a high-dose adrenaline infusion. Core temperature was 32.5^{O}C. She had a distended abdomen with profuse, watery, blood-stained diarrhoea and haematuria. Investigations revealed severe metabolic acidosis with a base deficit of -21, and a coagulopathy.

With the possibility of a cerebral arterial gas embolism (CAGE) considered unlikely and with evidence of a gross global hypoxic brain injury, a short HBO_2 treatment was reluctantly undertaken. She underwent a US Navy Table 5, during which she also received four units of fresh frozen plasma and was continued on high-dose adrenaline. A lignocaine infusion was commenced. Towards the end of this treatment there was deterioration in her cardiac output and she became profoundly shocked. On transfer to the ICU, and following discussion with the family, active treatment was withdrawn and she died soon after.

Subsequent history from the family revealed she had recently had a flu-like illness and was not feeling well prior to the dive. Postmortem histology indicated a small focus of active myocarditis (Figures 2a and b) and no evidence of arterial gas embolism. It was thought likely that she had suffered an acute myocardial event leading to arrhythmia and cardiac arrest, and that cerebral gas embolism was not a factor in her death.

Discussion

In the first case, migraine rather than DCI was suspected as the most likely diagnosis from the outset. The patient's single dive was well within safety limits, and there was a nearly 24-hour delay between this and his drive to altitude. However, having been air evacuated from a city 400 km away, it was felt appropriate that he be managed as though he had acute neurological DCI. His subsequent course and the successful use of sumatriptan really confirmed the original suspicions. HBO_2 has been reported as an effective treatment for migraine and Wilmshurst has reported a link between migraine and DCI, so the approach to management was not unreasonable.[2,3] Investigation for patent foramen ovale (PFO) might have been useful in this man, as presence of a PFO would have implications for his professional diving activities for the future.

In the second case, the explosive onset and nature of his symptoms made acute sub-arachnoid haemorrhage by far the most likely working diagnosis initially, the diving being coincidental. Therefore, he was not considered for HBO_2 until such other diagnoses were excluded. No explanation for the improvement of his symptoms with HBO_2 is offered, unless this was indeed a very atypical presentation of DCI. No final diagnosis was made, although neurological consultation labelled him as a possible case of 'pseudo-epilepsy'. Given the subsequent information on his past medical history, he was advised to cease scuba diving, advice he declined to accept. Headache is a common presenting symptom in DCI and its differential diagnosis, as in other circumstances, may be difficult.

In the third case, the experienced forensic pathologist conducting the autopsy considered it highly unlikely that the dive contributed to his death, given the widespread, severe atherosclerotic disease with occlusion of the circle of Willis. However, it remains possible that a small intra-vascular gas phase was the final precipitator of his acute demise. HBO_2 was considered appropriate, but his presentation and subsequent course were so unusual that a diagnosis of DCI was doubted. For this reason we did not extend the USN Table 6 or switch to a deep heliox therapy (RNZN 1A), either of which is a common approach to management for severe neurological or life-threatening DCI not responding to oxygen at 283 kPa. Other vascular pathologies that have been reported in divers presenting with neurological signs include inflammatory vasculitis and carotid artery dissection.[4,5]

Acute viral infections have been reported previously in association with decompression sickness.[6] We have had one diver present with non-specific systemic symptoms and pyrexia following diving, who convulsed on oxygen at 283 kPa early during recompression therapy, which was then abandoned. A subsequent diagnosis of influenza was made. We are unaware of any reports of viral myocarditis presenting as suspected CAGE. The referral of this unfortunate woman (Case Four) for HBO_2 was appropriate given the history, although by the time of her arrival her condition was clearly terminal.

Other non-diving pathologies masquerading as DCI that have been reported include acute appendicitis and acute psychosis.[7-9] Personality and affective changes are common in divers with DCI and after successful recompression therapy it is not unusual to feel that one is dealing with a different person to the one who first presented. However, it is unlikely that psychosis in a diver is attributable to DCI in the absence of other neurological symptoms of injury.

Munchausen's syndrome has been reported presenting as DCI,[10] and we believe that we have seen one such case.

These cases and other case reports illustrate the importance of a careful history, physical examination and investigation of divers referred with DCI. Whilst more often than not a diver in whom the diagnosis of DCI is doubtful has been given hyperbaric treatment, this has usually been on a precautionary basis. Physicians are reluctant not to treat divers once they have been evacuated, often at considerable expense, to a regional hyperbaric centre. DCI may present in a myriad of ways, leading to it being called the "syphilis of diving" by one expert. That other pathologies may mimic its presentation is consequently not surprising.

Acknowledgements

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