Case reports

Delayed hyperbaric intervention in life-threatening decompression illness

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Cerebral arterial gas embolism (CAGE); Rebreathers/closed circuit; Hyperbaric oxygen therapy; Intensive care medicine; Persistent (patent) foramen ovale; Case report

Abstract

(Perez MFM, Ongkeko-Perez JV, Serrano AR, Andal MP, Aldover MCC. Delayed hyperbaric intervention in life-threatening decompression illness. *Diving and Hyperbaric Medicine*. 2017 December; 47(4):257-259. doi10.28920/dhm47.4.257-259.) Arterial gas embolism is a catastrophic event. Bubbles in the arterial circulation may lodge in the brain and cause infarction in the affected area and/or in a coronary vessel causing acute myocardial ischaemia. There is no well-defined window of time beyond which a response to hyperbaric oxygen is not expected. Major improvement may occur if the patient is treated as soon as possible, but is less likely in divers with severe decompression illness who have delayed intervention. We report on a 51-year-old, male rebreather diver who suffered loss of consciousness and cardiovascular collapse within minutes of a 30-metre deep dive at a remote Micronesian dive site. Recompression treatment did not start for six days for reasons to be presented, during which time he remained deeply comatose, cardiovascularly unstable and intubated on ventilator support. Despite this, following aggressive hyperbaric treatment over many days he made a functional recovery. At one year post injury, he is leading a functional life but has not returned to his previous occupation as a diver and suffers from moderately severe tinnitus and impaired right ear hearing and occasional mild speech problems. He is undertaking a number of on-line courses with a view to re-employment.

Introduction

Decompression illness (DCI) is classified into decompression sickness (DCS) and arterial gas embolism (AGE).[1] Gas embolism occurs when gas bubbles enter arteries or veins. AGE was classically described during submarine escape training, in which pulmonary barotrauma occurred during free ascent after breathing compressed gas at depth.[3] The treatment of choice for DCI is recompression on oxygen (HBOT), with minimal delay. Most dive physicians believe that less therapeutic effect can be expected the longer the delay to hyperbaric treatment. In practice, the expected clinical benefit of recompression treatment administered more than a week following symptom onset is likely to be insignificant.[3,4] However, no matter the delay to treatment, HBOT is still indicated and remains the treatment of choice. We report a technical diver who suffered life-threatening DCI for which HBOT was delayed for many days.

Case report

A 51-year-old, male technical diver was diving at Chuuk Lagoon using a REVO closed-circuit rebreather (CCR). He had more than 4,000 dives, two years on CCR, and was certified on the REVO to 40 metres' depth on air diluent. He had completed four dives over two days of his trip. His first dive on the second day was to a maximum depth of 30 metres' sea water (msw) for 120 minutes, with a 5-hour surface interval. The second dive was to a maximum depth of 30 msw for an unknown duration with 23 minutes of decompression at 3 msw. According to the diver's wife, after boarding the small dive boat, he removed and stowed his gear and sat down before falling backwards unconscious about 10 to 15 minutes after the dive. He was breathing irregularly.

He was given oxygen using a scuba regulator and taken to the local hospital emergency room where he was delirious, and unresponsive to commands, afebrile and in mild respiratory distress. This was accompanied by excessive movements of all extremities. On auscultation, crepitations were heard in both lung fields and an electrocardiogram (ECG) showed sinus tachycardia (200 beats per min). Two intravenous (IV) lines were inserted and lactated ringers and normal saline solutions commenced to a total of seven litres, with heart rate now 100–110 bpm and BP 120/80 mmHg. The following medications were started: valium 5 mg every 6 hours (h), metoprolol 5 mg every 4 h, nitroglycerin transdermal patch, cefoxitin sodium 1 g every 12 h, cefazolin sodium 1 g every

6 h, furosemide 20 mg every 8 h, sodium bicarbonate (8.4%) 10 ml every 8 h and a stat IV dose of 200 mg hydrocortisone.

Two hours after admission, the patient became more restless and had progressive difficulty in breathing. The decision was made to intubate and he was admitted to the intensive care unit (ICU). On the second day at Chuuk Hospital, the patient was unresponsive and semi-comatose. The working diagnosis was DCS with severe brain injury. Although there is a stand-alone recompression chamber on Chuuk, he was judged to be too ill to transfer safely for hyperbaric treatment. Divers Alert Network – Asia Pacific (DAN-AP) was approached to procure air retrieval from Chuuk. After multiple attempts throughout the Asia-Pacific region proved unsuccessful, the US Coast Guard agreed to air evacuate him (unpressurized) to Guam for further management to include recompression treatment.

In Guam two days post-injury, computer tomography (CT) of the head was reported as showing "*multiple acute infarcts in anterior/posterior circulation*" and a CT of the chest "*pleural effusion; anasarca; ascites; pericardial effusion; atelectasis*". Quantitative Troponin-I, CK-MB, random blood glucose, creatinine, urea, C-reactive protein and B-natriuretic peptide levels were all elevated, electrolyte levels were fluctuating and the albumin level was below normal. The working diagnoses in Guam were DCI, multiple acute cerebrovascular accidents, ventilator-dependent acute respiratory failure, acute renal insufficiency, aspiration pneumonia and atrial fibrillation with rapid ventricular response. Only supportive care and medical management was given, as the hyperbaric facility in Guam was unable to accept the patient whilst ventilated. DAN-AP called several centres including Australia and Singapore in an effort to transfer him elsewhere for hyperbaric treatment without success, availability of suitable aircraft also being a problem.

On the sixth day post injury, DAN came into contact with the SPH-HH hyperbaric facility in Saint Patrick's Hospital Medical Centre (SPH) in the Philippines, who agreed to accept him. On arrival 127 hours post incident, he was admitted to the ICU still intubated and comatose, with a Glasgow coma scale (GCS) of 4/15 (E2V1M1), eye opening to deep pain only. Anasarca and crepitations in both lungs were present and his vital signs were stable. CXR on admission showed "*considerable pulmonary congestion and/or edema, bilaterally*", ECG showed "*poor R-wave progression*" and he had a metabolic alkalosis. Parenteral nutrition was started since the patient had not been fed since the initial event six days previously.

Bilateral tube myringotomies were done and he underwent a US Navy Treatment Table 6 (USN TT6) accompanied by two hyperbaric-trained nurses as attendants. Management during the treatment was physically demanding for the inside attendants. After the initial hyperbaric treatment, there was no significant change in the patient's status. On the second (now day 7) day, the patient had a GCS of 6/15 (E2V1M3). Vital signs were normal. A brain CT scan with contrast was reported as showing "*minute acute right cerebral peduncular infarct may indicate compromise to the tip of the basilar artery*". He underwent a second USN TT6. At 284 kPa pressure, he raised his eyebrows and moved the fingers of his left hand spontaneously.

The next day, his GCS was 11/15 (E4V1M6), with spontaneous eye opening and spontaneous movement of his right foot. He was able to follow simple instructions such as moving his arms or legs and raising his eyebrows. Left-sided weakness was noted. Weaning from the respirator was started as was deep vein thrombosis prophylaxis. He underwent a third USN TT6. On the fourth day at SPH (day 9 post injury), he was successfully extubated prior to his fourth USN TT6. Amiodarone was commenced pre-HBOT because of an episode of tachycardia. After this treatment he was awake, with spontaneous movement of the right extremities, predominantly the right leg, and a strong grip. He had sensory awareness over all four limbs and was able to stick out his tongue (deviated to the left) on command.

On day 10 post injury, his GCS was 12/15 with the right side of his body stronger than the left. CXR was now essentially normal. However, he had redeveloped atrial fibrillation with a rapid ventricular response so beta blockers, calcium channel blockers and digoxin were given intravenously to control the rapid rate. In the meantime, hyperbaric treatment was deferred. On day 11 post injury, the patient had an episode of supraventricular tachycardia (200 bpm) for which verapamil IV was given. Once this was controlled, he underwent a fifth USN TT6 during which his condition remained stable. He was now able to nod or shake his head when asked.

Two further daily USN TT6 were given and he was then transferred out of the ICU on the fourteenth post injury day. During an eighth USN TT6 the following day, he became restless at depth and immediately after the hyperbaric treatment appeared exhausted and would not cooperate with a post-treatment assessment, simply falling asleep. He was now moving all his extremities spontaneously, though the right side remained stronger than the left.

On day 15, a cranial CT angiogram (performed under sedation because the patient was restless and agitated) was within normal limits. A planned ninth USN TT6 was converted to a USN TT5 because he became combative and uncooperative in the chamber to a degree that it was feared he might harm himself or the two inside attendants. The following day the patient refused to undergo further hyperbaric treatment and his wife signed a waiver to that effect. That day, he started having difficulty swallowing and a nasogastric tube was reinserted and enteral feeding restarted. Digoxin and amiodarone IV medications were changed to oral administration.

After the last HBOT, his condition slowly but steadily

improved, with increasing strength and his voice and speech were clearer. By day 21 post injury, he was able to tolerate clear liquids and a soft diet, was starting to mobilise and the nasogastric tube, IV lines and urinary catheter were removed. By day 31 post injury, he was able to walk on his own and had no problems with micturition and defecation. He was repatriated home. Discharge diagnoses were hypoxic encephalopathy and coma secondary to DCI and ECG evidence of an anteroseptal wall myocardial infarct (old).

One year after the accident, a 9–11 mm diameter PFO was closed. He describes his degree of recovery is "*about 90%*" and he is on no medications. His occupation was diving but he has not returned to work since the incident. He was diagnosed with Eustachian tube dysfunction and experiences moderately severe tinnitus and a 75% right-sided hearing loss. He is currently working on some on-line courses for potential re-employment. He has no problems walking and doing mechanical tasks, but has occasional speech problems – "*I can still get easily tongue tied if I try to talk too fast*".

Discussion

The manifestations of CAGE usually begin during ascent or immediately after surfacing. When coma is the dominant manifestation, symptoms generally develop within 30 seconds to one minute of surfacing. A time lapse of more than 10 minutes between surfacing and the onset of symptoms is generally regarded as inconsistent with the diagnosis of CAGE, although there may be exceptions.[5] At the time of admission to SPH-HH, no information as to his previous medical history was available in order to provide us with a better grasp of his health status, particularly evidence of coronary artery disease, before the accident, as the transfer notes from Guam were of limited value. We considered that the patient may have had a cerebrovascular accident (CVA) either during the ascent from the dive or when he passed out on the boat. However, since the patient had been diving, the case was managed as CAGE even though the CVA issue was always at the back of our minds. A subsequent CT angiogram did not support this diagnosis and the initial CT scan showed multiple cortical lesions suggestive of CAGE.

Whilst USN TT6A was traditionally recommended for treating CAGE, clinical experience has suggested that it will respond to USN TT6.[2–4] An alternative option is the use of helium-oxygen tables such as the Comex 30 table.[6] However, SPH-HH does not have this capability. An additional management decision is how long to continue with HBOT and which treatment tables to use.[7] Given the severity of this diver's presentation, we opted for rather aggressive therapy with eight USN TT6 and one USN TT5. Indeed, were it not for the patient's refusal of further treatment, we would have planned to continue his hyperbaric course until he reached a plateau in terms of symptoms and signs.

Since there was nothing really irregular about the patient's dives, he was advised to be screened for a persistent foramen ovale (PFO), since PFO appears to be associated with an increased risk of cerebral DCS.[3] This proved to be the case, and the PFO was successfully closed.

The delay after which no benefit from hyperbaric treatments can be obtained may be many hours or even days. Whilst a proportionally less therapeutic effect can be expected the longer the delay, any patient with a diagnosis of DCS or CAGE should be considered for recompression treatment, especially in severe cases. Coma is associated with a high mortality and severer morbidity rate.[8] It is always better to give the patient a fighting chance. It is difficult to understand why other hyperbaric centres that were approached would or could not accept this diver for treatment. Certainly, his care challenged our resources and personnel to the utmost. We had not expected to obtain such a remarkable recovery in this diver given the long delay to recompression and the severity of his presentation. In this era of advanced life support and intensive care medicine, there is no substitute for good patient care by skilled and dedicated professionals.

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