The diving doctor's diary

Two case reports of epileptic seizures related to probable cerebral arterial gas embolism

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Abstract

(Wilson CM, Sayer MDJ. Two case reports of epileptic seizures related to probable cerebral arterial gas embolism. *Diving and Hyperbaric Medicine*. 2008; 38: 43-6.)
Two cases are presented of divers suffering epileptic seizure and loss of consciousness as a result of probable cerebral arterial gas embolism (CAGE). Both cases had apparently problem-free dives with no obvious provocation for CAGE, though one case may have been having repeated embolisms for some time in their diving career. Demonstrated also is the Type III form of decompression sickness, where spinal cord disease follows CAGE in a biphasic manner.

Introduction

Cerebral arterial gas embolism (CAGE) is a serious, potentially fatal complication of scuba diving. It is associated with rapid or poorly controlled ascents or breath-holding on ascent, but may occur with apparently normal ascent profiles even in individuals without pre-existing pulmonary pathology. It may also occur in decompression sickness (DCS) where a clinically significant right-to-left heart communication, such as a large patent foramen ovale (PFO), is present. We report here two cases of divers with probable CAGE complicated by tonic-clonic seizures following open-circuit scuba dives.

Case 1

A 41-year-old male, with three-and-a-half years of diving experience, was making his first dive of the weekend on a shallow wreck a short distance from Oban on the west coast of Scotland. He entered the water at 1030 hr, diving to a maximum depth of 16 metres' sea water (msw), using a dive computer for decompression management. He surfaced after an uneventful dive of a total duration of 43 minutes having stopped for a precautionary one minute at 6 msw. He got back aboard the rigid-hulled inflatable boat (RIB) quickly, but then developed a visual disturbance described as "all going dark". This was followed by severe spasms in his arms and legs that progressed to a tonic-clonic seizure, all within 10 minutes of surfacing.

The dive buddies in the RIB understandably panicked and made their way to shore while radioing the coastguard; as a result of their panic their message was incoherent, incomplete and confusing. However, an ambulance was quickly on the scene, oxygen was commenced, and the diver was transferred to the Dunstaffnage Hyperbaric Unit (DHU) a short distance away. The diver arrived at 1145 hr (32 minutes after surfacing) and was found to be confused, agitated and disorientated. Cooperation with examination was limited. He was lying with his hips flexed and his arms, hands and legs were rigid with muscular spasm. His hands were white and cold with poor capillary refill. There were no respiratory symptoms or abnormal physical findings in the chest. Full neurological examination was difficult at this time; he had been incontinent of urine. He had improved since the ambulance paramedics had attended him, probably because of oxygen administration, though the passage of time post-seizure might also have been a factor. Radiological facilities are not available at the DHU, but a chest X-ray at Oban Hospital after recompression was normal.

He was recompressed to 282 kPa (18 msw) at 1200 hr, less than an hour after surfacing, using the Royal Navy Treatment Table 62 (RN 62). Within ten minutes of being on 100% oxygen he claimed to be symptom free. The treating doctor (CMW) locked into the chamber to re-examine him at the first air break. Peripherally, he was now warm and pink with normal capillary refill. His vision was normal and he had normal muscular power that enabled him to walk normally. A Romberg test was negative and he was fully orientated in time and space and capable of delivering normal 'serial sevens'. However, it was noted that he had brisk reflexes in his arms and legs and had disdiadochokinesis of his right arm. He had no recollection of events from just after surfacing until going on oxygen under pressure. Having had a rapid and complete resolution of his signs and symptoms following the first cycle of 20 minutes of oxygen, he continued on an RN 62 treatment without modification.

Following treatment, the patient reported some discomfort in his calves and slight soreness in his right upper arm; both symptoms had not been mentioned previously. He felt considerably better but remarked on feeling tired. It was noted that he had slight impairment in heel-toe walking and

some difficulty in writing his address and signing his name. He was admitted to Oban Hospital for monitoring.

The following morning, there was a reduction in the discomfort in his calves and arms and his heel-toe walking was improved. A vast improvement in his writing was noted when compared with the efforts made the previous evening immediately post-treatment. He was returned to the DHU and was treated later that morning with a Comex 12 (222 kPa) hyperbaric oxygen table. All his residual symptoms and signs had settled following this treatment.

He admitted to some past diving-related medical history. Three years before he had had a similar seizure-like event while diving. After some delays he had been flown to a recompression facility. On arrival he was asymptomatic with a normal examination; he was given a precautionary treatment of an unmodified RN 62. Following that incident he had been assessed for a PFO and found to be negative, though he had no information on the examination method used. He continued to dive and, over the subsequent years, he had four further episodes following diving. These occurred typically within five minutes of surfacing and followed a pattern of visual upset, coordination problems, blue lips and some variable degree of loss of consciousness. He tended to be "sat in the corner" by his buddies as his symptoms resolved over the next hour. The 'diving doctor' associated with this dive club had put these episodes down to "carotid stimulation from a tight neck seal".

Following his treatment at DHU, he was diagnosed as having had a CAGE. It was thought probable that he had had repeated embolisms during his diving career. He was advised not to dive again and discharged home the day after his second treatment.

Case 2

A 22-year-old male scaffolder was learning to dive with shellfish divers on the west coast of Scotland. He had not undergone any dive training course or medical assessment and, in fact, had a history of using inhaled salbutamol when he had respiratory tract infections. The incident dive was only his sixth dive in total. Initially, there was very little information relating to the incident and even on arrival at the DHU he was unable to provide a full story of what had happened. During his recompression treatment, numerous telephone calls eventually collected the required information to piece together the chain of events.

He had entered the water with a seasoned shellfish diver as his buddy to what was initially reported to the DHU as a depth of 50 msw. In the fullness of time, this was corrected to 50 feet (16 msw) for 18 minutes. On the surface, at about 1230 hr, he had difficulty in swimming back to the boat and had a marked weakness of his left arm. He was unable to climb the ladder back on board and required help from the other divers. On the boat he suffered a tonic-clonic seizure, becoming rigid for 30 seconds with mouth clenching followed by rhythmic contractions of all his limbs. He remained deeply unconscious for 5–10 minutes, and thereafter his conscious level improved slowly. An ambulance was alerted as they returned the short distance to the harbour. During this time, the diver reported visual loss, being unable to see his hand in front of his face. He also reported a right temporal headache, paraesthesia and weakness in his left arm and leg. On meeting the vessel on the quayside, the ambulance paramedics commenced him on high-flow oxygen and found it necessary to remove him from the boat by stretcher.

In Scotland, there is a national emergency telephone support service run from Aberdeen Royal Infirmary, where the National Health Service has the only funded registration service in the UK. The Aberdeen consultant on call advised that the diver should be transferred to a recompression facility for urgent assessment and treatment. Air transportation was not available at that time but, as the diver's condition was improving, the ambulance continued toward the DHU at Oban. Transfer took approximately two hours and during this time his conscious level continued to improve. On arrival at DHU at 1500 hr, examination demonstrated a fit, muscular individual with no abnormal physical signs and he passed urine without difficulty. With a diagnosis of a probable CAGE, intravenous fluids were commenced and he was recompressed on an RN 62 at 1515 hr.

During the second oxygen cycle at 282 kPa (18 msw) he reported paraesthesia with altered sensation in the anterior aspects of both his thighs running to his feet and also paraesthesia and numbness in his left wrist. The treatment table was extended at 282 kPa, with the symptoms in his left hand completely settling, before decompressing to 191 kPa, though his legs remained little changed. On surfacing, he had persisting altered sensation in L3 to L5 dermatome distribution in both his legs, with intermittent paraesthesia in both his feet. He was transferred to Oban Hospital for monitoring, where a chest X-ray was normal.

The following morning, his muscle power remained normal but he was found to have altered sensation from dermatome T9 on the right and T10 on the left to L5. He had noticed difficulty in micturition both in initiating and maintaining his flow. He was diagnosed as having had a relapse and was transferred back to the DHU for further recompression therapy on an RN 62 table.

During the second treatment he had some improvement with the level of altered sensation slowly moving distally; he was able to pass urine more easily. The treatment table was extended by two additional oxygen:air cycles at 282 kPa and one at 191 kPa. On surfacing, the altered sensation showed little improvement, with a level of T10 on the right and T12 on the left. He was returned to the hospital for further post-treatment monitoring.

The diver's girlfriend had driven the 200 miles to be with him the next morning. His behaviour was reported by the nursing staff as being "odd" in her presence; in fact, she accompanied him to the shower. His altered sensory level remained unchanged and he reported his micturition as being normal. However, his girlfriend confided in nursing staff that he still had some problems. He was reluctant to undergo further recompression, claiming claustrophobia, but eventually agreed to further treatment. He was treated with a Comex 12 (222 kPa) hyperbaric oxygen table on the morning of the third day of treatments and returned to hospital afterwards with no apparent benefit.

With the imminent departure of his girlfriend for home two hours after surfacing from the third treatment, he decided he was going to accompany her and was not persuaded to stay, discharging himself against medical advice. However, he said he had no intention of continuing to dive; there has been no further contact with this man.

Discussion

The onset of symptoms resulting from CAGE will occur within 10 minutes, as happened in both cases reported here. Neither of them had a deep dive or any provocative ascent problems that could result in pulmonary barotrauma (PBT), though this cannot be completely excluded. There is also the possibility of a right-to-left shunt as a contributing factor. However, we know in Case 1 that investigations had excluded a PFO before his treatment at the DHU, although he gives a history of probable repeated, though possibly less severe, gas embolisms over his diving career. Both these men suffered a tonic-clonic seizure soon after surfacing.

Seizure associated with DCS is uncommon, but in association with CAGE is more common. The involvement of epilepsy in the two cases of seizure reported here is not discounted although neither diver had any past medical history suggestive of epilepsy. There may be a group of people for whom there is a lower threshold for seizure activity and a gas embolism could be a precipitating event. With the push to consider individuals with epilepsy as fit to dive, we may see more patients having seizures with less provocation, and in a hostile environment they may enter the fatality statistics.[1,2]

The most common reason for divers to succumb to an arterial gas embolism (AGE) is gas entering the circulation from PBT. This is the result of damage to the lungs from the over-expansion of pulmonary gas during ascent. The common symptoms of PBT of cough, haemoptysis, retrosternal chest pain and dyspnoea were all absent in the cases reported above. In the majority of divers with AGE, PBT often cannot be diagnosed radiographically.[3] These cases, of course, may have had asymptomatic PBT. It is also possible that venous gas bubbles could have been transferred to the arterial tree by right-to-left shunting, as a result of cardiac or respiratory anomalies.

Case 2 demonstrates the Type III form of DCS with a biphasic pattern where patients, initially with symptoms of AGE, then exhibit signs of spinal DCS.[4–6] Individuals who suffer CAGE when associated with a gas load, even if this load is thought to be trivial when applying standard decompression schedules, may suffer a severe form of spinal DCS that is often resistant to therapy.[3] It is postulated that the second phase of this DCS is caused by the presence of bubbles from an embolism precipitating further bubble formation in saturated spinal tissues. Greer reported a case where a scientific diver had a gas embolism with epileptic seizure and then developed severe spinal involvement although, in that case, there was a delay of over nine hours before recompression.[4] It is also observed that some divers suffering CAGE have a relapsing disease and, despite additional recompression, little benefit is achieved.[3]

A number of studies on CAGE have compared the results of submarine escape tower (SET) training with sport diving.[3,7] These noted differences in the respective presentations and results of treatment. These differences were not related solely to the speed of treatment but were also considered to be caused by the differences in inert gas loading, which was assumed to be virtually absent in the SET cases.

In a review of 300 consecutive cases of decompression illness (DCI) (84% of whom were sport divers) treated at DHU, 10.3% were diagnosed as having suffered CAGE, and of those 10% presented with a history of having had a seizure (i.e., 1% of 300, or three cases; Wilson CM, Sayer MDJ, unpublished observations). All of those 300 cases would be assumed to have had gas loadings greater than SET trainees. The incidence of CAGE in the sport-diver population has been falling over recent years. Reports of DCI collected by the Divers Alert Network (DAN) indicate that those cases attributed to CAGE have fallen from 18% of the total in the 1980s to less than 7% in 2001.[8] DAN have speculated that this decrease may be attributable to the use of modern dive computers indicating ascent rates that, in turn, may contribute to divers better controlling their ascents.

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