Cerebral arterial gas embolism in a diver using closed-circuit rebreather diving apparatus

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Key words
Cerebral arterial gas embolism (CAGE), closed-circuit underwater breathing system, recompression therapy

Abstract

(Chong SJ, Tan TW. Cerebral arterial gas embolism in a diver using closed-circuit rebreather diving apparatus. *Diving and Hyperbaric Medicine*. 2008; 38: 46-7.)
A Singaporean naval diver suffered symptoms and signs of cerebral arterial gas embolism (CAGE) following a panicked ascent from a depth of 5–6 metres' sea water while using a Dräger LAR V closed-circuit oxygen rebreather system. He presented with altered mental status and paresis. CAGE due to gas mixtures with high oxygen content has seldom been reported. The diver had no sequelae following prompt recompression therapy. This positive clinical outcome may be attributable to the high oxygen content in the diver's inspired gas and/or the promptness of recompression.

Introduction

Pulmonary barotrauma following ascent from depth whilst diving is one of the most serious forms of all barotraumas. Gas in the lungs expands during ascent and may rupture into adjacent lung tissues, resulting in mediastinal emphysema, pneumothorax or arterial gas embolism.[1] Cerebral arterial gas embolism (CAGE) is of particular concern as the vascular damage, hypoxia and the triggering of the inflammatory cascade in cerebral vessels may result in high rates of relapse, neurological deficits and even death.[2] CAGE has been widely reported with the use of open-circuit compressed gases, but there have been few reports of pulmonary barotrauma associated with closed-circuit rebreather (CCR) diving systems.[3,4] This may be due to several factors, including the experience of the divers using the CCRs and the high oxygen (O_2) content in the gases used in these diving systems.

We present the case of a young, healthy Royal Singapore Navy (RSN) diver who presented with symptoms and signs of CAGE after an uncontrolled ascent while using a CCR system. He responded clinically to recompression therapy and did not demonstrate any sequelae at follow up.

Case report

A 21-year-old RSN diver with nearly two years' diving experience was performing a routine compass dive for 60 minutes just outside the naval camp, with a maximum depth of 5–6 metres' sea water (msw) and using the Dräger LAR V oxygen CCR system. The O_2 concentration of the breathing gas in the LAR V CCR has been shown to be up to 74%.[5] The diver had completed about 35 minutes of his training dive when he developed difficulties with his mouthpiece and subsequently panicked and ascended uncontrollably to the surface as he seemed to be "choking".

At the surface, the dive supervisor noted that the diver had altered sensorium as he could not recognise his supervisor. He was thrashing about wildly but was relatively weak. The dive supervisor rapidly rescued him and administered 100% oxygen while evacuating him to the recompression facility within the nearby naval camp.

At the recompression facility, intravenous hydration was commenced. Blood pressure and heart rate were normal but there was tachypnoea of 22 breaths/min and pulse oximetry showed a decrease in oxygen saturation (92%) on 40% oxygen. There was altered mental status with a GCS score of 11 (E3 M4 V4) and the patient was not orientated to time, place or person. Neurological examination revealed generalised weakness with Medical Research Council grade of 4/5. An urgent chest X-ray did not demonstrate emphysema or pneumothorax.

A diagnosis of CAGE was made and recompression therapy was initiated within 35 minutes of the accident using the USN Treatment Table 6 (USN T6). The patient's symptoms began to improve at depth and achieved complete resolution by the first air break. A neurological examination done at depth demonstrated complete mental acuity and full power. The entire treatment table was completed and the patient monitored overnight with no relapse or sequelae. The patient underwent a repeat USN T6 the following day.

The blood investigations done showed a normal full blood count and urea, glucose and electrolyte levels within normal limits. A computed tomography (CT) scan of the patient's chest and a spirometry performed three months later did not reveal any abnormality. He returned to active diving six months after the incident.

Discussion

The expert opinion is that serious symptoms that develop immediately after ascent must be regarded as AGE and treated accordingly until a definitive diagnosis can be made.[1] Experimentally, cadaveric lungs have been shown to burst at pressures as low as 70 mmHg. In addition, there have been reports of intensive-care patients with lung ruptures following positive pressure of more than 70 mmHg.[1] This is approximately equivalent to an ascent of one metre in water. There have also been reports of CAGE occurring following ascents of one metre depth of water.[6]

The diver in this case suffered a CAGE following a rapid uncontrolled ascent from a depth of about 6 msw while using the LAR V CCR system. The rapid manifestation of his clinical symptoms on surfacing and signs of altered mental status with paresis were classical for CAGE. However, early recompression following CAGE carries a good prognosis, as seen in our patient.

The interesting aspect of this case was that the diver suffered a CAGE while using a Dräger LAR V oxygen CCR system. Using the single fill/empty cycle purge procedure developed by Thalmann and Butler, a diver can effectively increase the oxygen concentration in his breathing gas to 74%, in contrast to the 20.9% found in normal air.[5] This was the most likely oxygen concentration in the breathing gas of our patient at the time of the incident. With this oxygen-rich gas mixture, the gas bubbles responsible for the pathogenesis of this case of CAGE should theoretically be more easily metabolised by the surrounding tissues, reducing in size rapidly, and the oxygen content should mitigate the hypoxic effects of air embolism to tissues. The rapid and complete resolution of the symptoms may thus be contributed to by the high oxygen content. In a similar report by Carstairs the patient also achieved complete resolution.[4] The rapid initiation of treatment (within 35 minutes of the incident), thanks to the proximity of the recompression facility to the dive site, also would have contributed to the positive outcome.

Mediastinal emphysema, pneumothorax and local pulmonary damage may be associated with CAGE. However the subsequent CT scan of this patient's thorax did not reveal any pulmonary changes or pathologies. There were also no changes in the spirometry results of this patient. These follow-up medical investigations help to ascertain the future risk factors for diving, as well as shed light on the origins of pulmonary barotrauma. Even if there has been a history of rapid ascent, the presence of pulmonary bullae or other abnormalities must be sought.

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