Pulmonary barotrauma with cerebral arterial gas embolism from a depth of 0.75–1.2 metres of fresh water or less: A case report

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Key words

Diving medicine; Decompression illness; Hyperbaric oxygen therapy; Military diving; Underwater escape training

Abstract

(Lindblom U, Tosterud C. Pulmonary barotrauma with cerebral arterial gas embolism from a depth of 0.75–1.2 metres of fresh water or less: A case report. Diving and Hyperbaric Medicine. 2021 June 30;51(2):224–226. doi: 10.28920/ dhm51.2.224-226. PMID: 34157741.)

During underwater vehicle escape training with compressed air, a fit 26-year-old soldier suffered pulmonary barotrauma with cerebral arterial gas embolism after surfacing from a depth of 0.75–1.2 metres of freshwater or less. She presented with an altered level of consciousness. Rapid neurological examination noted slurred speech, a sensory deficit and right hemiparesis. Eleven hours after the accident, hyperbaric oxygen treatment was initiated using US Navy Treatment Table 6. The soldier almost completely recovered after repeated hyperbaric oxygen treatment. Given the very shallow depth this is an unusual case with only two similar case reports published previously.

Introduction

Cerebral arterial gas embolism (CAGE) is a serious medical condition where air enters the central circulation and reaches the brain, disrupting circulation, initiating inflammation and causing stroke-like symptoms. CAGE secondary to pulmonary barotrauma was originally described during free ascent training for submarine rescue¹ and diving with compressed air,² but it has also been described after breathhold diving^{3,4} and as a complication of various invasive medical procedures.⁵ Hyperbaric oxygen treatment (HBOT) is the preferred treatment for CAGE⁶ and should be initiated as soon as possible, but can have significant positive effect even if the treatment is delayed.7 The minimum depth from which surfacing can provoke pulmonary barotrauma and CAGE has not previously been defined. A case is presented which occurred after ascent from a very shallow depth, between 0.75–1.20 metres of freshwater (mfw), or less.

Case description

In September 2017, a fit 26-year-old female Combat Vehicle 90 (CV90) crew member was undertaking submerged vehicle escape training with a short term air supply system (STASS) in a swimming pool. STASS is a breathing device consisting of a small cylinder with 70 L of compressed air at 200 kPa (Figure 1) and has been used by helicopter crews for many years. During the training, a shallow water egress trainer (SWET) (Figure 2) is submerged, from which the trainees escape, breathing compressed gas from the STASS. Due to some serious accidents where a CV90 was accidentally submerged, the Swedish Army introduced this training for CV90 crews in 2017.

The SWET is first positioned upside down and then tilted slightly sideways in the water (Figure 2). The soldier, becoming submerged while strapped to the chair gradually learns to grab the STASS and escape through the 'roof', which symbolises the hatch of the CV90, during five exercises with increasing levels of difficulty. In the present case the soldier was uncomfortable breathing from the STASS underwater and had to repeat two of the five exercises, making the total number of exercises seven. After the sixth exercise, the soldier complained about a sharp headache focused on a point to the left in the back of her head, but continued. After escaping from the SWET the seventh and final time, the soldier stood up in the water without the mouthpiece, then fell towards the instructors. She was placed in a supine position beside the pool with 100% oxygen administered immediately. The officers present performed a rapid medical assessment, finding bilaterally dilated pupils, loss of sensation in the right part of the face and a reduced motor function in the right arm and foot. The soldier was responsive and could follow instructions, but talked slowly, incoherently, with slurred speech. Afterwards the soldier could remember the last exit from the SWET and how she felt her shoulder hitting the bottom of the pool. After that she had partial amnesia for 20 minutes.

An ambulance and the on-call military diving physician arrived in 10 minutes, and the neurological findings were confirmed. The soldier was helped to a sitting position

Figure 1 Short-term air supply system (STASS) (reproduced with the



twice within the first 20 minutes to remove wet clothing and lost consciousness both times. She was taken to the emergency department at the local hospital and was initially managed as a drowning accident. At the emergency department a computed tomography (CT) scan of head and thorax was performed approximately 90 minutes after the accident. It showed no cerebral hemorrhage, no evidence of intracerebral gas, no air trapped in the mediastinum and no pneumothorax. The physician at the emergency department intended to admit the patient with a diagnosis of stroke, but after discussion with the military diving physician the soldier was transported in a fixed wing aircraft with normalpressure cabin 1,400 km (870 miles) to the nearest available recompression chamber.

US Navy Treatment Table 6 was initiated 11 hours after the accident. One hour into the treatment, the soldier had almost completely recovered. The treatment table was not extended, and after its completion the only recidual symptom was a minimal reduction of motor function in the right arm and thigh, and minor sensory loss in the upper right extremity. During the following two days she was re-treated three times for 90 minutes at 243 kPa (2.4 atmospheres absolute). Two magnetic resonance imaging (MRI) scans of the brain were performed two and four days after the accident, without

Figure 2 The shallow water egress trainer (SWET) used during the accident (reproduced with the permission of the Swedish Armed Forces)



any pathological findings. The soldier was released from hospital four days after the accident in good condition with only a negligible sensory loss in the upper right extremity. After the accident, additional medical examination of the soldier, including spirometry, coagulation markers, vasculitis markers, transthoracic echocardiogram for persistent foramen ovale (PFO) and a CT-angiogram of the carotid arteries all showed no pathology.

The technical investigation showed no malfunction of the STASS equipment used. Measurements after the accident revealed that the distance from the water surface to the soldier's mouth was 0.75 mfw if she was turned completely upside down. The maximum depth of the pool was 1.2 mfw.

Discussion

CAGE following a pulmonary barotrauma is a well described complication in compressed air diving. Air in a distensible space will expand as the surrounding pressure decreases. This means that divers holding their breath during ascent are at risk for barotrauma of the lung. If alveoli and adjacent blood vessels are simultaneously damaged air may enter the pulmonary vessels, pass to the left atrium, and distribute in the systemic circulation. Given the brain receives 20–25% of the cardiac output, some of the bubbles will inevitably enter the cerebral circulation. Large bubbles may become trapped in cerebral arteries and cause ischaemia. Bubbles that redistribute through the cerebral circulation can initiate a secondary inflammatory response. Manifestations of CAGE include loss of consciousness, confusion, focal neurological deficits and ischaemia.⁵

In the present case the soldier is thought to have experienced pulmonary barotrauma when surfacing from a depth of 1.2 mfw or less. Such an event at such shallow depth has, to our knowledge, been described only twice before.^{8,9}

Nevertheless, it has been shown that a transpulmonary pressure between 73–90 mmHg can induce pulmonary barotrauma.^{1,10} The equivalent pressure of 1 mfw is 75 mmHg and it follows that pulmonary barotrauma in such shallow depths is certainly possible, if for example, the diver were to breathhold during ascent after a maximal inspiration. In the present case it is impossible to know exactly at what depth the injuring breath was taken, but it cannot have been deeper than 1.2 m and it seems clear that there was significant barotrauma with CAGE. It is notable that investigations did not find any obvious pulmonary predisposition to barotrauma. The sharp posterior headache after the second to last exercise might be an indication that pulmonary barotrauma had already occurred prior to the final exercise, but that is difficult to prove.

The instructors described clearly how the soldier was very motivated to complete the training but did have trouble breathing from the STASS and was uncomfourtable during the exercises. There was less time for the CV90 crews to do water exercises prior to the actual STASS training, in comparison to helicopter crews. This may have contributed to the accident. If the breatholding is initiated after the person has taken a very large breath the elasticity of the lung is already almost completely engaged and the added pressure required for pulmonary barotrauma can be quite small.

In this case the soldier exhibited altered consciousness for approximately 20 minutes after the initial CAGE. Each time she sat upright she lost consciousness completely, which might be due to re-embolisation when more air ascends to the brain or to lowered blood pressure in the affected vessels.

Patients presenting with initial neurological symptoms can improve without immediate recompression but might deteriorate clinically after a few hours.¹¹ It is important to treat patients even after a delay, since treatment initiated hours after the CAGE can still be beneficial.⁷ The mechanism is uncertain, but resolution of residual bubbles or amelioration of inflammatory effects are possible. This patient had a delay to treatment of 11 hours and still improved in temporal relation to HBOT.

This case is illustrative of how important it is for divers and dive medical specialists to enter a dialogue with medical professionals at a receiving local hospital. Otherwise there is a great risk that HBOT will be delayed or completely withheld with possible serious consequences for a patient with barotrauma-induced CAGE.

Conclusions

Pulmonary barotrauma with CAGE is rare but possible during compressed gas diving in very shallow waters. A diver with symptoms such as loss of consciousness, confusion, focal neurological deficits, cardiac arrhythmias, or ischaemia, occurring immediately or within minutes of surfacing should be considered as a possible AGE and treated with HBOT. The risk of re-embolisation should be considered and the patient should not have the head or torso elevated during first aid management. Although this seems to be a rare occurrence with only two previous case-reports published,^{8,9} it is important for divers and medical professionals to have sufficient knowledge concerning this possible injury even at shallow depths.

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Conflicts of interest and funding: nil

Submitted: 04 October 2020 Accepted after revision: 19 November 2020

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