Case reports

Cerebral arterial gas embolism (CAGE) during open water scuba certification training whilst practising a controlled emergency swimming ascent

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Abstract

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We report the case of a 23-year-old male novice diver who sustained cerebral arterial gas embolism (CAGE) during his open water certification training whilst practising a free ascent as part of the course. He developed immediate but transient neurological symptoms that had resolved on arrival to hospital. Radiological imaging of his chest showed small bilateral pneumothoraces, pneumopericardium and pneumomediastinum. In view of this he was treated with high flow normobaric oxygen rather than recompression, because of the risk of development of tension pneumothorax upon chamber decompression. There was no relapse of his neurological symptoms with this regimen. The utility and safety of free ascent training for recreational divers is discussed, as is whether a pneumothorax should be vented prior to recompression, as well as return to diving following pulmonary barotrauma.

Introduction

We report the case of a 23-year-old male novice diver who sustained a cerebral arterial gas embolism (CAGE) during his open water certification training whilst practising an emergency swimming ascent as part of his course.

Case report

Written informed consent was obtained from the patient for presentation of the details of his clinical history and de-identified imaging.

The adult male diver had a history of childhood asthma for which he had not used any bronchodilator inhaler since about seven years of age and was otherwise well. His other hobbies included flying, without previous issues.

He had two unremarkable pool training dives the week before his CAGE episode during personal tuition by a certified diving instructor of a recognised training agency.

The incident dive was at a well-known recreational shore dive located approximately 30 kilometres south of Fiona Stanley Hospital (FSH), near Perth, Western Australia (WA). Fiona Stanley is the state referral centre for diving and hyperbaric medicine in WA.

He reported surface swimming from the shore, then descending to a depth of about six metres of sea water (msw) to practise emergency swimming ascent training with his instructor. The instructor demonstrated ascending to the surface with the regulator out of his mouth, breathing out and then exhaling into his buoyancy control device (BCD) upon reaching the surface. The diver then repeated this but upon reaching the surface noticed blackness of his vision and then briefly lost consciousness after exhaling his remaining breath into his BCD. His BCD was further inflated by the dive instructor who towed him back to shore. At that time the diver stated that he could not see but only hear his instructor speaking. He reported that he was unable to speak and that he had right-sided weakness with 'pins and needles' sensation. He also described sharp left-sided pleuritic chest pain and mild shortness of breath that had started to improve prior to arrival of the ambulance, as well as mild headache.

On arrival of the ambulance, some 20 minutes later, his weakness and altered sensation had resolved. Paramedics

noted that he was mildly confused and vague with a Glasgow Coma Score (GCS) of 14 which rapidly improved to GCS 15 with oxygen (O_2). His vital signs were documented as peripheral oxygen saturation 92% on air, respiratory rate 16 breaths·min^{-1}, blood pressure 125/69 mmHg and heart rate 104 beats·min^{-1} regular. An electrocardiogram (ECG) performed by paramedics on the scene apparently showed ST elevation and hence aspirin 300 mg was given, as per ambulance protocol. On arrival to the emergency department some 30 minutes later, the diver reported that he was asymptomatic except for mild left-sided pleuritic chest pain. A repeat ECG was normal.

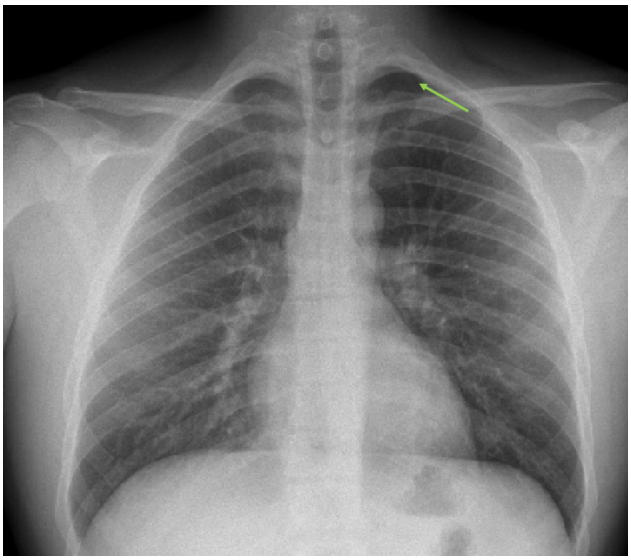
A chest X-ray (CXR) was performed soon after arrival was reported as showing "*features suspicious for a left apical pneumothorax measuring up to 11.7 mm in depth. Equivocal appearance of the right lung apex may be projectional or represent a further pneumothorax*" (Figure 1). The FSH Hyperbaric Medicine Unit (HMU) was contacted and reviewed the CXR prior to it being reported, with a similar interpretation but the possibility of an associated pneumopericardium was also noted. Our advice was to immediately obtain high resolution chest computed tomography (HRCT) and then send the patient directly to the HMU while continuing high flow oxygen via non-rebreather reservoir mask. The first author went to the CT scanner with the patient and interpreted the images in real time with the radiologist.

The HRCT was reported as showing "*bilateral pneumothoraces measuring 7.2 mm in maximal depth on the right and 4.7 mm on the left. There is soft tissue surgical emphysema seen within the pericardium and throughout the mediastinum extending along the paraesophageal, paratracheal and parabronchial regions*" (Figures 2 and 3). Interestingly, the CT scan showed a larger pneumothorax on the right side compared to the left which was poorly visible if at all on the CXR.

On examination in the HMU he had normal vital signs and was alert with GCS 15. He had coherent and fluent speech, without evidence of dysphonia. Cranial nerve and limb motor and sensory function were normal, and reflexes were symmetrically brisk. Coordination via finger-nose testing was normal. The patient was kept supine and hence gait was not tested. Cardiovascular examination was unremarkable; of note Hamman's crunch was not heard on cardiac auscultation and subcutaneous emphysema was not evident.

In view of the presence of small bilateral pneumothoraces and given that the patient was asymptomatic with a normal neurological exam, the decision as to whether or not to recompress him (with United States Navy Treatment Table 6 (USN TT6)) was discussed by three senior hyperbaric physicians. The invasive nature of inserting bilateral chest drains was considered, as was the risk of a tension pneumothorax during the decompression phases of the table should the pneumothorax(es) continue to expand without

Figure 1
Chest radiograph showing small left apical pneumothorax (green arrow); the right apical pneumothorax is occult



placement of chest drains. It was noted that untreated pneumothorax is listed as an absolute contraindication to hyperbaric oxygen treatment (HBOT) in many references (see **Discussion**) although, theoretically, if there is no longer a gas leak into the pleural space, HBOT should accelerate resolution of a pneumothorax,[1] as it does for surgical emphysema and pneumomediastinum.

Based on the principle of 'first do no harm', it was decided to observe the patient overnight with frequent neurologic observations and continuous vital signs monitoring while continuing high flow O_2 (via a non-rebreather mask) with a plan to insert bilateral chest drains and proceed to USN TT6 should neurological symptoms recur.

Upon review the next morning, he remained well. Cardiorespiratory and neurological examinations were normal. He was able to perform a Sharpened Romberg test to 60 seconds on the third attempt. Bedside ultrasound scanning showed no evidence of either pneumothorax or pneumopericardium. The patient was discharged with planned follow-up in the HMU in four weeks with advice to rest and not dive or fly until then.

Upon review at four weeks, he reported no problems except for extreme tiredness; sleeping up to 12 hours nightly instead of his usual seven hours. He had recently attended the emergency department of the adjacent private hospital for this where a repeat chest HRCT was performed. This was reported as being entirely normal.

He was advised not to scuba dive until he was reviewed again at six months post event, during which time formal pulmonary function would be performed and he had time to consider whether he wished to continue diving. Pulmonary function testing performed two months post event was

Figure 2
Coronal chest computed tomography (CT) slice showing bilateral apical pneumothoraces (green arrows) and pneumomediastinum (red arrow)

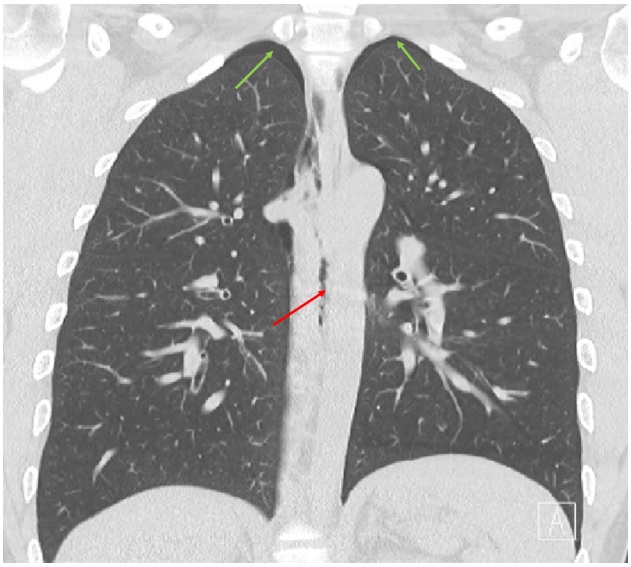
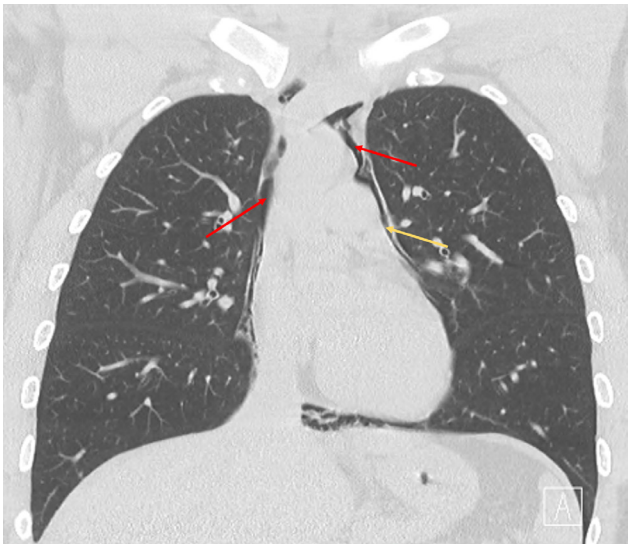


Figure 3
Coronal chest computed tomography (CT) slice showing pneumopericardium (yellow arrow) and pneumomediastinum (red arrows)



normal with no evidence of small airways disease or gas trapping.

Discussion

This case highlights several issues. The first being the utility and safety of controlled emergency swimming ascent training for recreational divers.

Controlled emergency swimming ascent (CESA) training and 'buddy breathing' were introduced in the early days of diving when out-of-air events were commonplace, reportedly contributing to half of scuba fatalities in Australia and New Zealand in the 1980s.[2] Since then, pressure gauges have become ubiquitous, training more focused and breathing gas exhaustion less common.[3,4] However, some occasionally still find themselves out of gas and distant from a buddy and have no alternative but to free ascend with the associated risk of pulmonary barotrauma (PBt), arterial gas embolism, hypoxic blackout and/or drowning.

Previous data from the Professional Association of Diving Instructors (PADI) revealed that from 1989 to 1992, there were four cases of CAGE or PBt in a calculated 1,251,568 CESAs, an incidence of 0.31 per 100,000 ascents.[5] Periodically, there is discussion about whether there is sufficient benefit in continuing to teach a training practice that can potentially cause harm. For example, a report from Belgium found that in a cohort of 34 cases of PBt treated at a particular hospital from 1995 to 2005, 16 had occurred during emergency ascent training.[6] As a result, the Belgium Underwater Federation discontinued this training and a subsequent review revealed a substantially decreased incidence of PBt.[7]

More current Australian data from PADI indicate that from 2001 to July 2023, there was one reported case (the present case) of PBt or CAGE associated with ascent training during approximately 550,000 open water certifications (each including at least one CESA) (D Dwyer, personal communication, 2023 July 13). Although there might be some shortcomings in reporting, it is obvious that the risk of such an incident is very low.

Some certification agencies have abandoned teaching free ascent, others teach it as a simulation in a horizontal orientation, while others require divers to demonstrate the procedure under strict guidelines and control. PADI requires that open water diver students perform a CESA from a depth of 6 to 9 m in open water. The instructor maintains control through contact with both a secure ascent line and the candidate. The latter keeps their regulator in their mouth, looks upwards and continuously exhales throughout the ascent while maintaining contact with their BCD deflator to control ascent, the rate of which should not exceed 18 $m.min^{-1}$. The student is advised to resume normal breathing if they stop the ascent or have any difficulty. At the surface, they orally inflate their BCD or drop their weights.[8] Scuba Schools International (SSI) training standards mandate that students perform an emergency ascent in a pool or open water with a maximum depth of 9 m.[9]

In this case, other than the free ascent itself, which was reportedly not overly fast, there appears to be no obvious precipitating or risk factors. Although the diver did not have his demand valve in his mouth as required, he believes that he did not aspirate any water and, despite feeling a bit low on breath over the last couple of metres, was not aware of breath-holding, blew bubbles throughout (confirmed by the

instructor) and had sufficient residual air on surfacing to exhale some into his BCD.

The second issue is how best to manage a diver who has confirmed CAGE with neurological symptoms that have resolved, but who has a pneumothorax. Recompression is the recommended treatment for CAGE, even where symptoms have resolved, because of the tendency of patients to deteriorate after apparent recovery.[10,11]

Pneumothorax is uncommon in diving, occurring in fewer than 10% of cases of PBt.[12] Recompression without drainage of a pneumothorax is often characterised as contraindicated, particularly in monoplace chambers,[1] although recommendations vary as shown below.

The widely accepted practice is: "*...a diver with pneumothorax should always have a chest drain inserted before any air evacuation, or before recompression if there is another problem such as CAGE or DCS that justifies recompression in the presence of a pneumothorax*".[13] However, Neuman, in Bove and Davis' Diving Medicine textbook states: "*Theoretically, recompression and then subsequent decompression can convert a simple pneumothorax to a tension pneumothorax; however, this has not been reported as a frequent problem, and standard therapy is appropriate in such cases*".[14]

Moon, in Hyperbaric Oxygen Therapy Indications, states: "*In patients with AGE caused by pulmonary barotrauma there may be a coexisting pneumothorax, which could develop into tension pneumothorax during chamber decompression. Therefore, placement of a chest tube in patients with pneumothorax prior to HBO_2 should be considered and is recommended for patients treated in a monoplace chamber. For multiplace chamber treatment, careful monitoring is a feasible option*".[10] He provides similar advice elsewhere.[11]

In Neuman and Thom's Physiology and Medicine of Hyperbaric Oxygen Therapy, it is advised that "*Although an untreated pneumothorax is almost universally considered an absolute contraindication to hyperbaric treatment, if a practitioner is so equipped, a tension pneumothorax can be treated by simple venting within a multiplace chamber*".[15]

In the US Navy Diving Manual Revision 7 it is advised: "*Divers recompressed for treatment of arterial gas embolism or decompression sickness, who also have a pneumothorax, will experience relief upon recompression. A chest tube or other device with a one-way relief valve may need to be inserted at depth to prevent expansion of the trapped gas during subsequent ascent. A tension pneumothorax should always be suspected if the diver's condition deteriorates rapidly during ascent, especially if the symptoms are respiratory. If a tension pneumothorax is found, recompress to depth of relief until the thoracic cavity can be properly vented. Pneumothorax, if present in combination with arterial gas embolism or decompression sickness, should not prevent immediate recompression therapy. However, a pneumothorax may need to be vented as described before ascent from treatment depth. In cases of tension pneumothorax, this procedure may be lifesaving*".[16]

The relatively recent availability of the expertise and equipment to perform in-chamber ultrasound allows detection of a pneumothorax under pressure, rather than other causes of deterioration, and hence confirms the need for thoracostomy.[17]

Other cases of CAGE with PBt have been reported where an expectant approach was decided and recompression withheld, after balancing the risk of recurrent CAGE upon decompression, in divers with neurological symptoms that had resolved.[18]

From the above, it would seem prudent to drain a pneumothorax prior to recompression unless there is equipment and expertise to diagnose a pneumothorax in chamber and perform tube thoracostomy where required. This option was considered by three senior hyperbaric physicians versus normobaric O_2 and careful observation for deterioration, which was the eventual management. Certainly, if this patient did not have pneumothoraces, they would have been recompressed with USN TT6. A similar conservative approach for the management of a CAGE case in a remote area with difficult and delayed access to recompression could also be considered.

A third issue is return to diving after PBt / CAGE. This has evolved from the traditional absolute contraindication to further scuba diving, to informed decision making by the diver in consultation with a diving medicine specialist, occasionally with input from a respiratory medicine specialist.[13] This risk assessment approach may not be unreasonable, particularly if HRCT and pulmonary function testing after recovery are normal, and a suitable period of time has elapsed to allow tissue healing. The evidence for the question 'when?' returning to dive after a PBT is lacking, and it can only be inferred from other recommendations for other conditions. The European Diving Technology Committee imposes an absolute contraindication for diving during the first three months after any pneumothorax. Return to diving could be considered in some situations, after 3 months, if pulmonary function testing is normal, with a FEV_1/FVC at least 0.7. The same is suggested for returning to dive after thoracic surgery.[19]

The medical manual of the U.S. Navy, in its "*Examination and Standards*", differentiates, for disqualifying purposes, spontaneous from traumatic pneumothoraces. Spontaneous events are an absolute contra-indication for diving duties, while traumatic (but non-barotrauma) related events could be accepted, even for a diver candidate, after a recovery period of six months, with favourable lung function tests, thoracic radiology and expert opinion. For diving related PBt (i.e., pneumothorax, mediastinal or subcutaneous

emphysema, or CAGE), a diver candidate will be excluded, but a "*designated diver*" may receive a waiver to return to duties after a 30-day period if predisposing respiratory conditions are excluded.[20]

From the above it is evident that there is a different approach in the case of a spontaneous compared to traumatic pneumothorax, the main difference being related to the risk of recurrence based on an underlying lung condition. Even though the risk of recurrence of a spontaneous pneumothorax is highest in the first two years, incidences as high as 18% were reported more than six years after the initial episode.[21] Reasonably, traumatic pneumothorax is not considered such a problem as it is associated with low rates of recurrence.

The Undersea and Hyperbaric Medical Society (UHMS) in its Diving Medical Guidance to the Physician and the British Thoracic Society have a similar recommendation of ceasing diving after a spontaneous pneumothorax, unless there are no lung parenchymal abnormalities on HRCT and the diver underwent bilateral surgical correction.[21,22] However, a clear discussion should take place with the diver as even after surgery the risk of another episode could be as high as 10%.[21] A recent systematic review on returning to diving following a first episode of primary spontaneous pneumothorax concluded that *"Given that a pneumothorax at depth can result in a fatality, the current practice of generally advising against further diving is probably sound"*.[23]

Returning to dive after an episode of CAGE is more consensual, with commercial and military standards allowing it provided the diver is symptom free.[19,20] The US Navy Medical Manual also requires a normal magnetic resonance imaging scan of the brain performed within seven days of the event.[20] For our case, considering that no abnormalities were found on HCRT and pulmonary function testing, return to diving after a period of six months seemed a reasonable approach, with a low risk of recurrence.

Conclusion

We report a novice diver without apparent predisposing factors or significant violation of training procedures who developed PBt with bilateral pneumothoraces and CAGE during CESA training. The utility and safety of such training for recreational divers is debateable. Patients exhibiting complete spontaneous recovery after CAGE are typically recompressed but prolonged surface oxygen and careful observation for relapse is an option if there are obvious associated risks such as in the present case with bilateral pneumothoraces.

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