The diving doctor's diary

Cerebral arterial gas embolism with delayed treatment and a fatal outcome in a 14-year-old diver

John Lippmann, Andrew Fock and Shalini Arulanandam

Key words
Case reports, cerebral arterial gas embolism, deaths, children, recreational diving, fitness to dive, medical conditions and problems

Abstract

(Lippmann J, Fock A, Arulanandam S. Cerebral arterial gas embolism with delayed treatment and a fatal outcome in a 14-year-old diver. *Diving and Hyperbaric Medicine*. 2011;41(1):31-4.)
In today's recreational diving climate, diving fitness examinations are not mandatory, and even divers who go for these examinations may not have routine chest X-rays (CXR) done in the absence of respiratory symptoms or a past history of respiratory problems. We present a case of an ultimately fatal cerebral arterial gas embolism in a 14-year-old boy with an undiagnosed lung cyst, the contribution of which to his death is uncertain. Various factors such as lack of oxygen first aid at the remote dive site; poor communication; lack of diving medicine expertise, poor oxygen administration and management in a local hospital and long delay to recompression therapy contributed to the poor outcome. It is imperative that dive operators and physicians working in close proximity to popular dive sites be educated on how to recognise and treat diving emergencies and be well-acquainted, as should divers, with the contact numbers of diving medical hotlines that offer timely and appropriate advice in case of emergency.

Introduction

Dive operators need to be adequately prepared for diving emergencies, with quality first-aid and oxygen-provider training, suitable and readily accessible oxygen (O_2) equipment and an appropriate accident management plan. This is especially important in areas, such as parts of the Asia-Pacific region, lacking ready access to recompression facilities and diving medical expertise.

It is also important that both prospective and existing divers are adequately screened for medical contraindications to diving. In most places, a short self-reporting medical questionnaire is used, with only those people with positive responses advised to seek medical assessment. However, in some cases, medical conditions may not be detected even by a relatively thorough pre-dive medical examination by a trained physician, with serious and sometimes fatal consequences. There remains ongoing debate about the appropriate level of medical investigation required prior to or during continuing diving activities. The following case serves as a reminder of the importance of these issues.

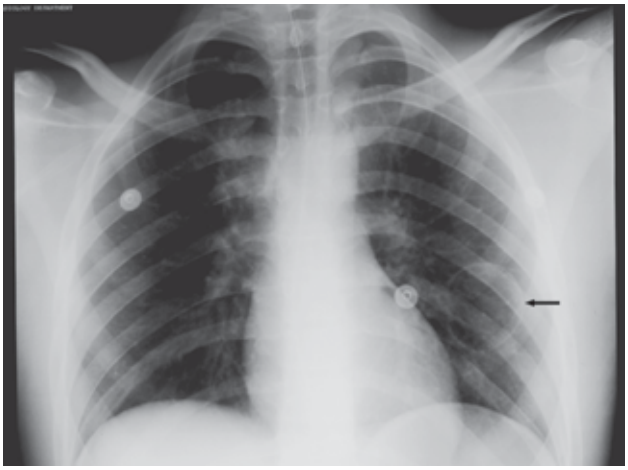
Case report

AB (not his real initials) was a 14-year-old Australian male. Permission to report his case was given in writing by his father. His past medical history was unremarkable with no serious illnesses, including respiratory problems. He was 176 cm tall, weighed about 73 kg and played sport regularly. Prior to scuba training, AB underwent a diving medical assessment in accordance with Australian Standard AS 4005.1 by a physician trained in diving medicine. This found no obvious contraindications to diving. A chest X-ray was not considered to be indicated.

AB had conducted two resort dives prior to his open-water diver training and a further four incident-free dives to a maximum depth of 15 metres' sea water (msw) during his course. He had done no further diving between certification and the day of the accident. AB and his father, an experienced diver, were diving in South East Asia. On the first dive, AB, his father and the dive guide dived to a maximum depth of 18 msw for a total of 45 minutes, following the contour of the reef and ending with a safety stop. He was described as "*very active*" during the dive, "*swimming with his arms and legs all over the place just like a newly certified diver*". However, it did not appear that he was exerting himself or changing depth levels rapidly. After surfacing, AB mentioned that he had been sneezing underwater and that he had an "*itchy feeling*" in his chest, but this did not appear to be bothering him.

After lunch and a surface interval of one and a quarter hours, they descended to a maximum depth of 18 msw, again following the reef into shallower water throughout the dive. The 40-minute dive appeared to be free of problems and the group ascended "*normally*" to a 5 msw safety stop, when AB swam to the other divers and began to squeeze his right arm as though it was numb. Soon after this, he appeared to float towards the surface. The divemaster noticed that he appeared to be "*passing out*" and brought him to the surface.

Figure 1
Initial chest X-ray showing cyst on left lung



He was quickly brought aboard the boat which departed immediately for the resort, some 10–15 minutes away. There was no O_2 equipment on board. He was initially unconscious and convulsing, with froth coming from his mouth, but breathing spontaneously. The convulsions continued throughout the boat trip to shore, although he was intermittently responsive between seizures. On arrival at the resort, O_2 was administered. The resort instructor reported that the O_2 was delivered via a rebreather mask with an O_2 flow rate of 15 litres per minute (lpm). AB's father is adamant the mask did not have a reservoir.

Shortly after, he was taken by ambulance to a nearby public hospital, where he was attended by several doctors, none with training or knowledge in the assessment and management of diving accidents. A chest X-ray revealed a large (3 cm) air-filled 'bullus' on the left lung, although there was no evidence of pneumothorax or mediastinal emphysema (Figure 1). He was then transferred to a private hospital and examined by several other doctors, again without knowledge or training in diving medicine. On examination, his Glasgow coma score (GCS) was assessed as 8, but his conscious state continued to fluctuate. There were marks on his abdomen, which were described by his father as "*bruises which resembled randomly and widely-spaced burst blood vessels*". A provisional diagnosis of cerebral vascular accident was made and computerised axial tomography (CT), a magnetic resonance image scan (MRI) and magnetic resonance angiography (MRA) were performed. The MRI demonstrated bilateral occipital lobe oedema without clear evidence of infarcts. Throughout this time, AB's conscious state continued to fluctuate, with intermittent seizures, some of prolonged duration. His vital signs remained stable, but he was unable to move his right arm or leg and appeared to have cortical blindness. O_2 was reportedly delivered in hospital via a simple face mask with a flow rate of 6 lpm.

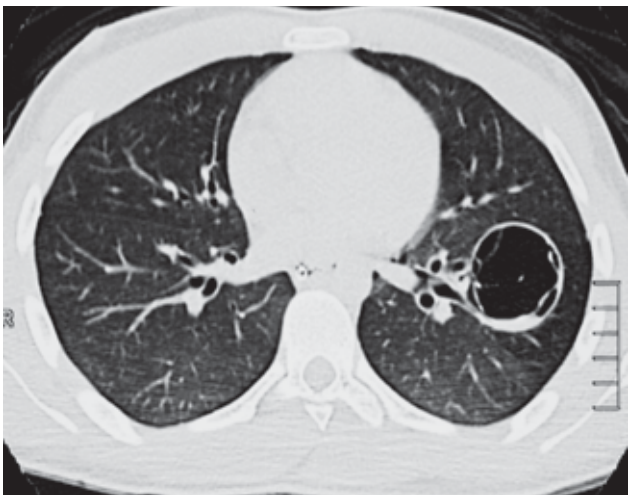
Some six hours after the accident, a local naval doctor arrived and diagnosed that AB was suffering from decompression illness. He believed the 'bruises' on AB's lower abdomen to be a decompression-related rash (*cutis marmorata*). He recommended recompression and investigated whether AB could be transferred to the nearest naval chamber, some three hours away by road and boat. Although, after several hours, permission to use the chamber was granted, the doctor was concerned that the relatively basic chamber lacked adequate facilities to manage such an ill patient, especially one with a lung bullus.

The father then contacted their travel insurer's assistance company which, after several hours' further delay, called the Diver Alert Network (DAN) America hotline for advice. The DAN physician recommended urgent evacuation to a chamber for recompression. Approximately 10 hours later, Singapore General Hospital was contacted, and permission was promptly given to transfer AB to its hyperbaric unit. The evacuation team arrived at the hospital five hours later. AB was intubated in the ambulance at the airport just prior to boarding the aircraft for the two-hour flight to Singapore, and he arrived at the hospital 32 hours post-dive.

On admission, he was unresponsive, with a GCS of 4 with doll's eyes negative, heart rate 100 per min, blood pressure 190/90 mmHg and respiratory and abdominal examination unremarkable. He was sedated and paralysed (propofol and atracurium), bilateral myringotomy performed and a left intercostal catheter inserted. The initial hyperbaric treatment was a US Navy Treatment Table 6 (USN T6).

When sedation and paralysis were reversed the next morning, he remained deeply comatose with a right hemiplegia. An MRI taken at this time showed large acute territorial infarcts involving the occipital and posterior parietal lobes, and watershed territories of the middle and anterior cerebral arteries (left more than right), cerebral oedema with mild

Figure 2
Axial view CT showing cyst; the image is reversed so the left lung appears on the right



compression of the fourth ventricle and no hydrocephalus or venous thrombosis. A further USN T6 was given, with no improvement. Following this, another brain CT confirmed the extensive watershed infarcts with cerebral oedema, now with tonsillar herniation. The decision was made to discontinue recompression therapy in view of his poor response, and the family was advised of the poor prognosis. AB was repatriated to Australia on ventilatory support, where assessment two days later in intensive care confirmed brain death and life support was withdrawn.

Discussion

CAUSATION

AB's history and symptoms make CAGE the most likely diagnosis. Gas entering the arterial system may come from one of two major sources, either entering the pulmonary veins as a result of pulmonary barotrauma (PBT) or from venous gas emboli transiting to the arterial circulation via a right-to-left shunt (e.g., patent foramen ovale (PFO) or across the pulmonary vascular tree).[1] Most commonly, CAGE is associated with a lung over-inflation injury resulting from breath-holding while using compressed gas and/or rapid ascent. In this case, the ascents on both dives were reportedly well-controlled and slow. Whether or not the CAGE resulted from a PBT, the presence of the 'bullus' must be taken into consideration. This 'bullus', actually a large lung cyst (Figure 2), was thick-walled and appeared to communicate with terminal airways. Such cysts are usually stable and may be ventilated or non-ventilated. Generally they have no effect on lung function or respiratory performance in the non-hyperbaric environment. However, it is believed that air-trapping can sometimes occur either on the basis of gas diffusion, a one-way valve mechanism, or by a volume increase upon ascent due to a narrow inlet-outlet opening.[2] While the presence of the cyst, if detected, would almost certainly have resulted in AB not being granted medical clearance to dive, there is conflicting opinion as to whether such a cyst actually poses an increased risk of PBT.[2,3] The lack of radiological evidence of PBT in this case does not preclude its presence.

An alternative is that AB had a PFO or atrial septal defect (ASD), but without the benefit of an autopsy this remains speculative. Both dive profiles involved only a few minutes at the maximum depth followed by a slow ascent up the reef. Such a dive profile would usually not be expected to result in significant tissue supersaturation. The presence of what was described as *cutis marmorata* and AB's report of an itching feeling in his chest between dives may have been due to undetected skin decompression sickness, which has been associated with the presence of a PFO.[4]

RETRIEVAL AND CLINICAL MANAGEMENT

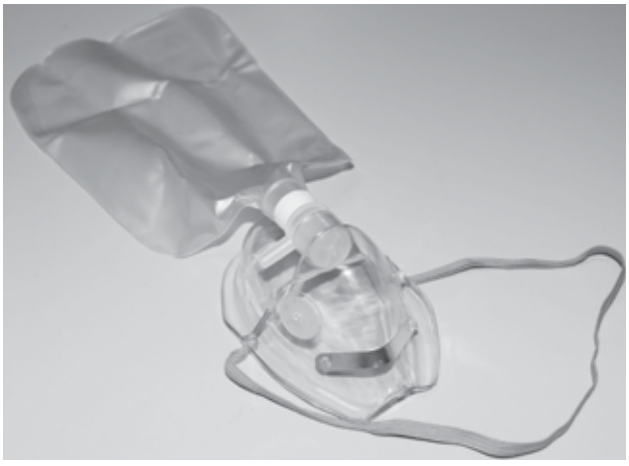
It is disturbing that no O_2 was available on the dive boat. Prompt and effective first-aid delivery of high-concentration O_2 can often result in symptom resolution or improvement and a reduced number of recompression treatments.[5] DAN Asia-Pacific records contain numerous reports of rapid improvement of CAGE symptoms when near-100% O_2 is provided promptly. There is an expectation and, in many places, a 'standard of care' to have appropriate O_2 equipment and a trained provider present wherever diving is undertaken. The dive operator did provide O_2 back at its base but only via a mask without a reservoir bag, which would not have provided the near-100% O_2 needed.

Poor communication between various parties led to long delays before appropriate medical advice was obtained and acted upon. It would have been better to call a divers' emergency hotline (such as the DAN Diver Emergency Service in Australia). The on-call doctor would certainly have recognised the seriousness of the situation and provided appropriate management advice, as was done when a DAN hotline was finally called some 10.5 hours after the accident.

Medical professionals without knowledge or training of diving medicine often fail to recognise decompression illness, its potential severity and the urgent need for high-concentration O_2 and rapid recompression. A simple face mask with an O_2 flow rate of around 6 lpm in the hospital would have likely provided an inspired O_2 concentration of less than 40%.[6] The correct choice and management of O_2 delivery devices is well covered in several publications, and a suitably designed mask is illustrated in Figure 3.[7,8] Unfortunately, in many under-developed countries in the Asia-Pacific region, O_2 supplies are often quite limited.

Given AB's critical neurological status, it is unfortunate that he was not intubated and ventilated with high-concentration O_2 and normocapnea long before this was finally done.

Figure 3
A non-rebreather mask with reservoir; two one-way valves on the sides minimise air entrainment and one prevents exhalation of expired gas into the reservoir; the fresh gas inlet is seen on the left side



Indeed, basic management strategies for a patient with severe cerebral injury would not appear to have been instigated until his transfer to Singapore.

The decision to recompress in a local chamber or to transfer to a comprehensive chamber with ICU support can be a difficult one to make, especially where local resources are limited. While there is little doubt that immediate recompression can be life-saving, evidence would indicate that local recompression in small chambers where the medical staff are unable to deal with complex patients may result in worse outcomes.[9] In this case, consultation with a respiratory physician or suitably-trained radiologist should have indicated that the presence of the cyst posed minimal risk for recompression compared to the negative outcome from significant gas embolism. Furthermore, despite AB's fluctuating conscious state, he was haemodynamically stable during his time in the local hospital. Given the delay before being assessed by the naval diving physician, it must remain speculative whether by the time a diagnosis of DCI was made there would have been any change in outcome.

FITNESS-TO-DIVE CONSIDERATIONS

The value of a formal medical remains a matter of considerable debate.[10,11] AB underwent a medical in accordance with AS 4005.1, which does not mandate a CXR in the absence of respiratory symptoms. Indeed surface bullae, which are probably more likely to be pathological, will not usually be picked up on plain CXR. Had AB had a CXR and subsequent CT scan, it is likely he would not have been issued a fitness-to-dive certificate. However, as previously discussed, it is debatable to what extent a stable thick-walled cyst of this nature poses a real threat of PBT, or if it was the culprit in this case. Similarly, the value of screening for ASD/PFO in the absence of symptoms has been discussed extensively in the literature.[12–14] Any link between PFO and AB's injury remains purely speculative.

Conclusion

This case highlights the need for divers, especially those travelling to remote locations, to be educated about diving accidents and to carry contact numbers for diving medical hotlines so that prompt expert advice can be obtained should symptoms appear post-diving. It also indicates the need for better education of the general medical community about the management of dive accidents and the referral options available to them. Finally, it highlights the reality that some divers, who perhaps should not dive on medical grounds, will pass even a thorough dive medical assessment, sometimes with tragic consequences.

Conflict of interest: John Lippmann is the Executive Director of DAN Asia-Pacific which sells O_2 equipment, including non-rebreather masks and provides diving injury insurance. Andrew Fock is a member of the Board of Directors of DAN AP.

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John Lippmann, OAM, BSc, DipEd, MAppSc, is Executive Director, Divers Alert Network (DAN) Asia-Pacific.
Andrew Fock, MB,BS, FANZCA, Dip DHM, is a consultant to the Hyperbaric Medicine Unit, The Alfred Hospital, Melbourne.
Shalini Arulanandam, MBBS, MRCS, MMed, is a diving medical officer in the Republic of Singapore Navy and consultant to the Hyperbaric Medicine Unit, Singapore General Hospital.
Address for correspondence:
John Lippmann
P.O. Box 384, Ashburton, VIC 3147, Australia
Phone: *+61-(0)3-9886-9166*
Fax: *+61-(0)3-9886-9155*
E-mail: *<johnl@danasiapacific.org>*