Case report

Cerebral arterial gas embolism in a scuba diver with a primary lung bulla

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Abstract

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Primary lung bullae have been reported to cause pulmonary barotrauma and lead to cerebral arterial gas embolism (CAGE) in the context of diving; however, a lack of symptoms and often minimal radiographic findings often preclude a diagnosis of lung bullae prior to undertaking diving activity. We present the case of a healthy 27-year-old Caucasian male who presented following the second of two introductory resort dives with neurological symptoms attributable to CAGE. Investigations revealed a previously undiagnosed large primary lung bulla. This case highlights the clinical sequelae of primary lung bullae in the context of pulmonary barotrauma related to recreational diving activity.

Introduction

Primary lung bullae are air-filled, thin-walled spaces occurring within the lung in a patient free of airway obstruction or disease.[1] They are characteristically asymptomatic, and occur more frequently in patients with Marfan's syndrome and Ehlers-Danlos syndrome.[2,3] Bullous lung disease is an absolute contraindication to diving, however a lack of symptoms and often minimal radiographic findings often preclude a diagnosis of lung bullae prior to undertaking diving activity. We describe the case of a previously healthy 27-year-old Caucasian male who developed cerebral arterial gas embolism (CAGE) as a result of pulmonary barotrauma secondary to a previously undiagnosed primary lung bulla.

Case report

A 27-year-old Caucasian male tourist presented to the emergency department following an episode of transient neurological symptoms that developed after a dive ascent. His medical history was significant for a multinodular goitre requiring total thyroidectomy, inguinal hernia repair and testicular hydrocoele, with no history of respiratory disease. He was a non-smoker and had never had any chest imaging or lung function testing performed. This was his first dive experience and was a 'resort' dive with a reef company which did not require a dive medical to be performed. His first dive of the day was to 3–5 metres depth for 10 minutes, and was uneventful. His second dive occurred one hour later to 10 metres for 30 minutes, with a one-minute safety stop. He ascended slowly following the directions of the instructor, and there was no panic or breath holding. He began to feel dizzy on the last 1–2 metres of the ascent and began coughing one metre from the surface with a sensation of fullness of the chest. After reaching the surface he began to feel unwell, and on approaching the boat he was unable to lift himself unaided due to a left-sided hemiparesis involving his arm and leg. He also noted left-sided paraesthesiae and an expressive aphasia. He was helped into the boat and given high-flow oxygen, noticing an improvement in his symptoms within 10 minutes and complete resolution after 30 minutes.

His observations in the Emergency Department demonstrated an oxygen saturation of 98% on high-flow oxygen, blood pressure of 134/90 mmHg, heart rate of 77 beats per minute, and respiratory rate of 20 breaths per minute. Neurological examination was normal and cardiorespiratory examination was unremarkable. A chest X-ray was performed which revealed a large bulla in the right upper zone containing an air-fluid level (Figure 1). Computed tomography scanning of the chest confirmed the presence of a large thin-walled fluid-containing bulla in the right upper lobe with surrounding alveolar shadowing, likely representing pulmonary haemorrhage (Figure 2). There was also a smaller subpleural bulla in the left lower lobe (Figure 3). The diagnosis was of pulmonary barotrauma secondary to a primary lung bulla leading to CAGE. The patient was discussed with the

Figure 1
Chest radiograph of 27 year-old diver presenting with cerebral arterial gas embolism showing a large bulla in right upper zone containing an air-fluid level

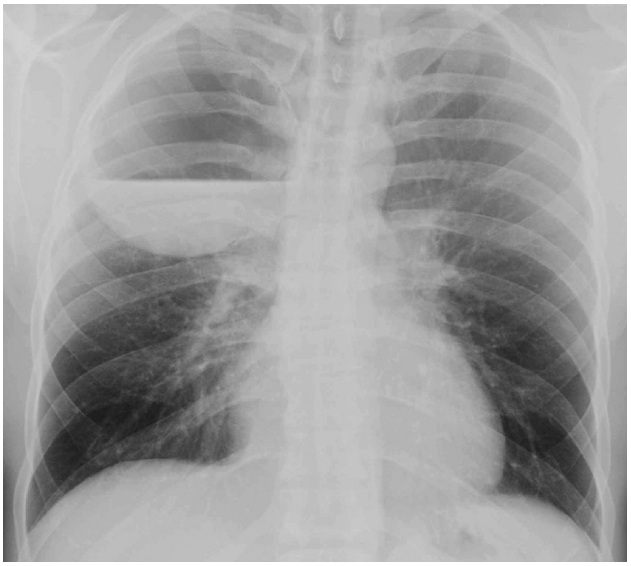


Figure 2
Computed tomography scan of the chest showing a large thin-walled fluid-containing bulla in the right upper lobe of a diver presenting with cerebral arterial gas embolism

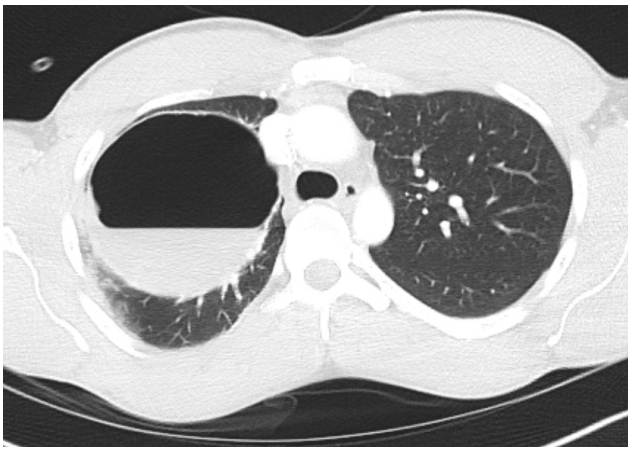
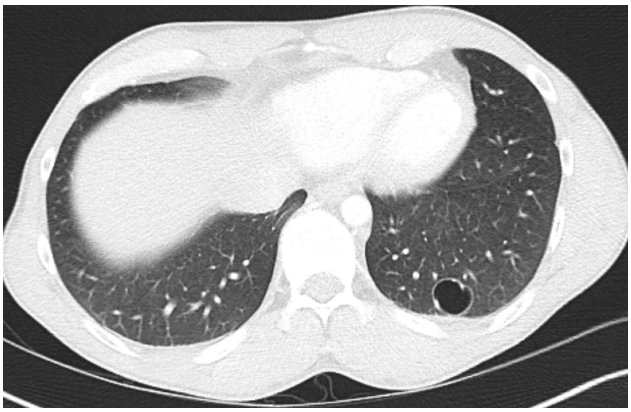


Figure 3
Computed tomography scan of the chest showing a smaller bulla in the left lower lobe of the same diver



nearest hyperbaric medicine unit, and recompression was not performed – partly because his symptoms had resolved, and partly due to concerns regarding possible further barotrauma on decompression. The patient fully recovered and returned home to be followed up in another centre. An MRI brain was performed several weeks later and was reportedly normal. He subsequently underwent a bullectomy. He was counselled never to dive again.

Discussion

Lung bullae are an absolute contraindication to diving. Since pressure is inversely related to volume (Boyle's Law), dangers arise when a poorly ventilated area of lung (such as that inside a primary lung bulla or cyst) takes up some gas during the time at depth, and is then subject to reduced pressure on ascent. If it is unable to be vented, possibly due to a one-way valve effect or intermittent plugging or obstruction, the gas that has accumulated within the bulla expands and may cause pulmonary barotrauma. The rate of change of pressure over the ascent will also affect the ability of the bulla to equilibrate the pressures. If the pulmonary parenchyma is damaged such that air enters the pulmonary vasculature, there may be systemic arterial gas embolism leading to stroke-like symptoms as bubbles reach the brain and/or myocardial ischaemia from coronary arterial air embolism.

It has been demonstrated previously that divers with pre-existing small lung cysts or bullae may be at risk of pulmonary barotrauma;[4] however as in our patient, owing to the frequent absence of chest imaging prior to the event, there is often uncertainty as to whether the bullae caused, or were the result of, the pulmonary barotrauma event. There are a few reports in the literature of pulmonary barotrauma related to primary lung bullae. A similar case to our patient has been described, wherein a 33-year-old, previously well female developed neurological symptoms attributable to CAGE shortly after ascending from an 18-metre dive, which was associated with a sensation of dyspnoea and pleuritic chest pain.[5] Subsequent investigations identified a giant emphysematous bulla in the left hemithorax. Her symptoms fully resolved, and recompression was not performed, as in our patient. Interestingly, the patient's previous chest imaging from five and three years prior was reviewed and demonstrated an area of hypertranslucency in the left lung, though the films were reported as normal at the time. This suggests that the bulla was indeed present prior to the dive.

In an interesting case series of three patients in whom a primary bulla was demonstrated radiographically, an increase in diameter of the bulla was attributed to diving activity, ultimately leading to a barotraumatic diving accident including CAGE.[6] Two of the cases demonstrated a marked increase in the size of the bulla immediately following the episode of CAGE when compared to previous imaging, which the authors supposed may have been a

consequence of the dive and contributory to the sequelae. Another series of three cases has been reported where unsuspected congenital lung bullae led to diving accidents without CAGE.[7] In two cases, the bullae ruptured causing spontaneous pneumothorax and in the third a tension bulla led to symptoms.

There are also case reports of pulmonary barotrauma and CAGE resulting from lung bullae during commercial air flight.[8–10] Our patient, in retrospect, recalled experiencing similar upper chest discomfort and dyspnoea four years previously following skydiving, which self-resolved after several minutes, as well as occasional upper chest discomfort during commercial air flights during the preceding several years. These symptoms were never investigated and were never associated with any neurological symptoms. That his respiratory symptoms developed most convincingly after skydiving, wherein he would have experienced a more sudden change in air pressure, is notable, and supports the notion that the bullae were pre-existing and not a consequence of the dive.

Thankfully the patient's symptoms resolved with supportive cares; however, this case raises the important question of whether recompression using hyperbaric oxygen treatment (HBOT) should be administered for persistent symptoms in cases of CAGE attributable to pulmonary barotrauma with underlying lung pathology. The dilemma is that while HBOT is the treatment of choice for CAGE,[11] it necessitates further recompression and, more importantly, decompression, which is the presumed mechanism of injury causing the pulmonary barotrauma in the first instance. There are two case reports of CAGE attributable to HBOT in the context of underlying lung pathology, such as bullous lung disease in a smoker[12] and diffuse interstitial pulmonary fibrosis with severe emphysema.[13] It is likely that recompression would have been offered to our patient if his neurological symptoms had persisted, on the basis that decompression from HBOT occurs at a much slower rate than the insult from the dive, and would therefore be less likely to cause further injury. The risks of recompression would, however, need to be openly and frankly discussed with the patient and/or next-of-kin, if possible, given the uncertainties.

This case also highlights the difficulties in screening an otherwise healthy population for underlying lung disease. Although our patient had experienced possible respiratory symptoms during previous flights and sky diving, he never underwent lung imaging and the screening medical questionnaire that he completed – the only form of assessment required for a leisure resort dive in Queensland – would not identify underlying bullous lung disease based on symptoms alone. Given the normal cardiorespiratory examination after the event, there is no certainty that a face-to-face diving medical would have identified the problem either. Further, as illustrated in several cases discussed above,[5,6] plain chest imaging is frequently inadequate for diagnosing primary lung bullae or cysts. The question of whether computed tomography of the chest should be incorporated into screening practices has been raised; however, this would unlikely be practical or cost effective.[14] Optimal screening practices for primary lung bullae is an area that warrants on-going study.

In summary, we have described the case of a healthy young male who developed CAGE secondary to pulmonary barotrauma owing to a previously undiagnosed primary lung bulla. This case highlights the need to maintain a degree of suspicion for pulmonary barotrauma in any patient who develops neurological symptoms following a dive.

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Dr John Knight FANZCA, Dip DHM, Captain RANR

John Knight *was* the South Pacific Underwater Medicine Society. Having joined the Society in 1972, he went on to fill the roles at various times of Committee Member, Secretary, President, Public Officer (the predecessor of the Education Officer), Assistant Editor of the SPUMS Journal (Doug Walker as Editor) 1985–89, and then Editor of the Journal from 1990 to 2002. He was elected a Life Member of SPUMS in recognition of his enormous contributions. Our Journal would not exist today if not for John's efforts.

John passed away on 09 May, aged 89. A full obituary will appear in the September issue of *Diving and Hyperbaric Medicine*.

A Celebration of John's life will be held on Friday 7 June 2019 at 1.30 pm in the Newcomb Library Meeting Room, Geelong. RSVP to liz@legalpm.com.au