# Cerebral arterial oxygen embolism as a complication of hyperbaric oxygen treatment: a case report

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**Abstract**

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Cerebral arterial gas embolism (CAGE) is a recognised complication of diving-related barotrauma and of medical procedures whereby gas enters the vascular system. CAGE is a principal indication for hyperbaric oxygen treatment (HBOT). In contrast, CAGE resulting from HBOT itself is exceedingly rare. We describe the case of a 73-year-old man undergoing his first session of HBOT for a chronic lower limb wound, who developed acute focal neurological deficits during decompression. He had no known respiratory disease or smoking history. Immediate assessment demonstrated stable physiology. Initial computed tomography (CT) scan of the brain and neck vessels and perfusion imaging did not show any evidence of thrombus or intravascular gas, and partial neurological recovery occurred over several hours. Subsequent magnetic resonance imaging of the brain demonstrated acute infarction involving the left precentral and postcentral gyri. High-resolution CT chest revealed multiple bilateral thin-walled pulmonary cysts, including a left upper lobe cyst with an air–fluid level consistent with recent barotrauma. A transient broncho-venous fistula allowing arterial oxygen embolisation during decompression was considered the most plausible mechanism. The patient improved with supportive management and was discharged with minimal residuae. This case highlights an exceptionally rare but serious complication of HBOT and underscores the importance of vigilance during decompression and careful consideration of occult pulmonary pathology.

## Introduction

Changes in pressure can lead to pulmonary barotrauma, particularly when gas becomes trapped within the lungs. This is well recognised in diving and aviation, and the same principle applies to hyperbaric oxygen treatment (HBOT). However, decompression during HBOT is slow and carefully controlled, making clinically significant barotrauma extremely uncommon. Cerebral arterial gas embolism (CAGE) arising during HBOT is therefore exceptionally rare. We report what is, to our knowledge, only the third known case of CAGE occurring during decompression from a hyperbaric treatment.

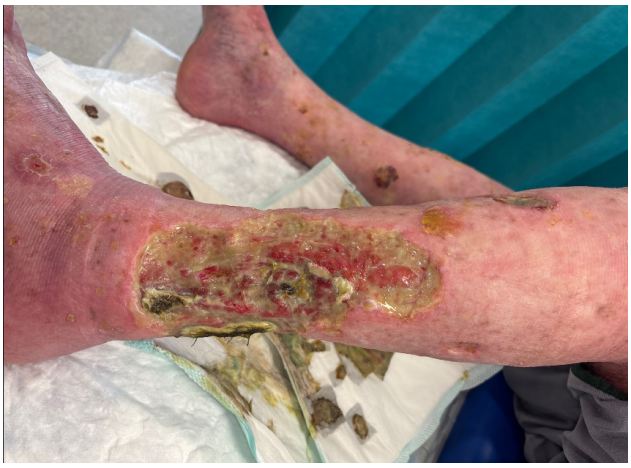
## Case report

Written consent was given by the patient for the publication of the case details and images.

A 73-year-old male was referred to our hyperbaric centre for extensive non-healing bilateral lower limb wounds (Figure 1). His history was notable for basal and squamous cell carcinomas, psoriasis, hypertension and bilateral carpal tunnel decompressions. He had no history of lung disease and had never smoked.

The patient had received radiotherapy to the lower extremities three months prior for squamous cell carcinoma,

**Figure 1**
The wounds for which the patient was referred for hyperbaric oxygen treatment



and psoralen and ultra violet A light treatment for psoriasis. His wounds had been debrided but were not amenable to grafting until further optimised. Macrovascular perfusion was intact. No chest imaging was performed as part of the assessment and no prior imaging was available for review. There were no contraindications for HBOT evident, and a decision was made to offer treatment. Some transport issues required coordination as his home was 80 km from our centre, and he commenced treatment 73 days after his assessment.

No issues were identified on his first treatment day, and a treatment table AH 14 (243 kPa [2.4 atmospheres absolute, 14 metres of sea water equivalent]), with decompression time of 14 minutes and total duration one hour 53 minutes was commenced in an air-filled multiplace chamber (Figure 2). The patient received oxygen via a head hood tent system.

The patient reported no equalisation issues and remained asymptomatic during the compression, treatment and decompression phases of HBOT. On removal of the hood immediately after decompression to atmospheric pressure, he reported being unable to move his right arm and was noted to have right upper limb paralysis and evolving left upper limb weakness. The patient was alert, responsive, and speaking full sentences. Sensation was intact in both upper limbs. There were no other neurological symptoms or signs. His vital signs were normal. Intravenous access was secured and a blood sample sent for venous blood gas which showed pH 7.39, $PCO_2$ 46 mmHg, $PO_2$ 41 mmHg, HCO3 28 mmol·L$^{-1}$, lactate 2.2 mmol·L$^{-1}$, sodium 142 mmol·L$^{-1}$, potassium 4.1 mmol·L$^{-1}$ and blood sugar 7.2 mmol·L$^{-1}$. His oxygen saturations were 97% on room air. Cardiorespiratory examination was normal, with no increased work of breathing, equal air entry bilaterally, and no signs of pneumothorax. An emergency response and code stroke were activated whilst the patient was transferred to the treatment room for monitoring, from where he was transported supine to the emergency department via the radiology department for imaging, and subsequently admitted under the care of the stroke team.

A computed tomography (CT) perfusion scan of the brain and carotids 35 minutes after onset of symptoms was reported as normal. No pneumothorax was seen on the included imaging of the lung apices. A chest X-ray was reported to be normal. As there was no radiological evidence of stroke on CT and the neurological examination was improving, he was not thrombolysed.

Significant improvement in symptoms occurred rapidly over two to three hours and continued over the course of his admission. CAGE was considered the presumed diagnosis at the time. Further recompression was decided against in view of clinical improvement and risk of further CAGE with a potential deleterious outcome. The day after the event, a magnetic resonance imaging (MRI) scan of the brain and spine was performed and demonstrated left pre-central and post-central gyri infarcts (Figure 3). High-resolution CT (HRCT) chest demonstrated multiple bilateral thin-walled lung cysts involving all lobes, with mid- to lower-zone predominance and subpleural distribution, and no evidence of emphysema (indicated with arrows in Figure 4). A dominant left upper lobe cyst measured 15 × 24 mm and contained an air–fluid level with adjacent ground-glass change (marked with an X in Figure 4). A communicating bronchus was identified along the cyst margin. No intravascular gas was seen. Two additional smaller cysts in the right middle lobe demonstrate air-fluid levels. The pleural spaces were clear. Pulmonary arteries were of normal size. No air was visualised within the cardiac chambers.

In summary, the HRCT demonstrated cystic lung disease, with imaging features suspicious for Birt-Hogg-Dubé syndrome, and with left upper lobe cyst rupture as a result of occult barotrauma. The impression of the reporting radiologist was that perhaps a transient broncho-venous fistula had allowed a gas embolus to the brain.

Following his rapid improvement, the patient was commenced on aspirin and atorvastatin and discharged home within 24 hours, with just some remaining numbness in the palm of his right hand. On follow up by telephone the day after discharge, he reported a small area of numbness on the palm of the left hand. Respiratory physician and GP follow up were advised for further investigation of potential Birt-Hogg-Dubé Syndrome.

## Discussion

### DIAGNOSIS

Cerebral arterial gas embolism should be strongly considered when focal or global neurological symptoms occur during, or within seconds to minutes of completion of decompression from HBOT or compressed gas diving. The differential

**Figure 2**
Treatment table AH 14

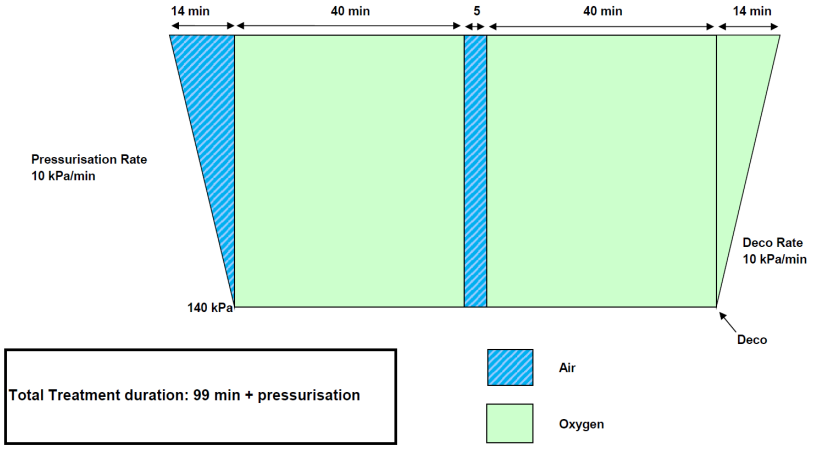



**Figure 3**
Magnetic resonance imaging of the brain demonstrating acute infarcts of the left precentral and postcentral gyri

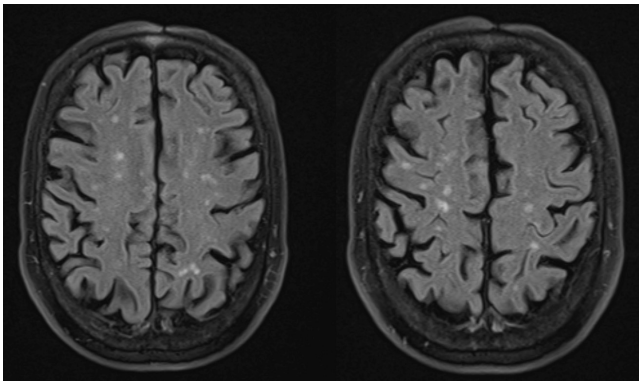
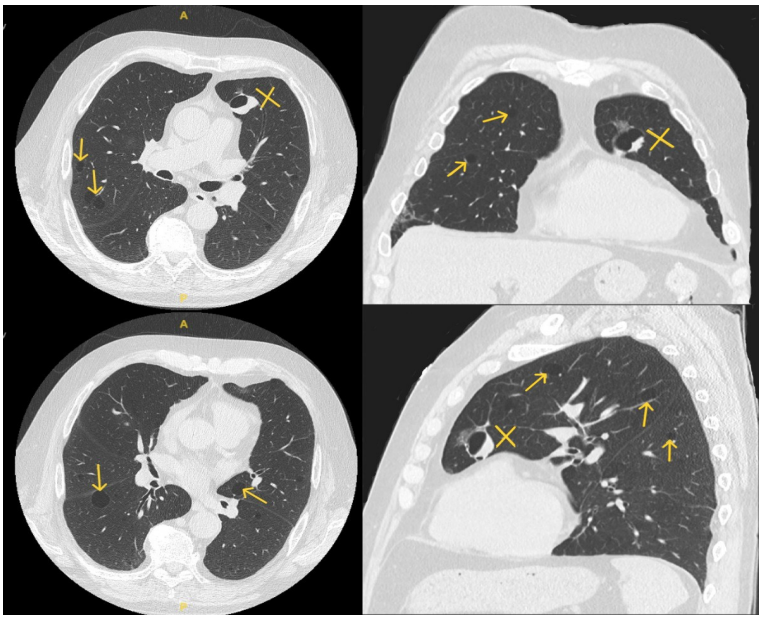


**Figure 4**
High resolution computed tomography images demonstrating multiple pulmonary cysts



diagnosis in this setting includes acute ischaemic stroke, intracranial haemorrhage, seizure including oxygen toxicity seizure, hypoglycaemia, and hypoxic events; however, the abrupt onset with decompression, rapid partial neurological recovery, and subsequent radiological findings in this case are most consistent with arterial gas embolism.

The proposed mechanism of embolism in this case is pulmonary barotrauma with cyst rupture during decompression, resulting in transient communication between the bronchial and pulmonary venous systems and subsequent cerebral arterial embolisation.

Underlying pulmonary pathology was identified in this patient, with imaging features suggestive of Birt-Hogg-Dubé syndrome. This autosomal dominant condition, caused by mutations in the folliculin (FLCN) tumour suppressor gene, is characterised by pulmonary cysts, spontaneous pneumothorax, characteristic benign cutaneous papules, and an increased risk of renal malignancy. While lung cysts are relatively common incidental findings in thoracic imaging, the incidence of arterial gas embolism in patients with cystic lung disease undergoing HBOT remains extremely low.[1]

Cerebral arterial gas embolism is well described in association with rapid ascent and breath-hold during ascent from compressed gas diving,[2,3] as well as following decompression during commercial air travel.[4–6] In these settings, the entrained gas is typically air, reflecting the gas being breathed at the time. Likewise, CAGE occurring during pressure tolerance training of military divers in an air-filled hyperbaric chamber are likely to have had air as the offending gas rather than oxygen.[7]

In contrast, after prolonged exposure to hyperbaric oxygen treatment, it is likely that gas entering the vascular system following cyst rupture would be predominantly oxygen. Gas composition, solubility and metabolic uptake influence the behaviour of gaseous emboli, endothelial injury, downstream ischaemia and resultant clinical consequences. Early experimental work by Kunkler and King indicated that an embolus made up of oxygen is about twice as well tolerated as one consisting of air.[8] A report from the early 1900s, describes the injection of oxygen gas as a therapeutic measure.[12]

Whilst this case occurred during a 14-minute decompression from 243 kPa, the previously reported cases in the HBOT setting occurred with even slower decompression over 30 minutes – highlighting that this complication can occur even with very slow rates of decompression.

## MANAGEMENT

Management of CAGE occurring during HBOT presents a unique challenge. While recompression is the standard treatment for arterial gas embolism resulting from diving or iatrogenic entrainment,[2] there is uncertainty regarding the management of CAGE when the embolism itself occurs as a complication of HBOT. Two previously published cases demonstrate this uncertainty.[9,10] In one, HBOT was readministered, but progressive deterioration with ventricular arrhythmias, haemodynamic instability, and death followed.[9] In the other, supportive management without recompression was associated with partial neurological recovery but severe permanent disability.[10]

One of the reported oxygen embolism cases, as well as our present case, occurred during the first HBOT session, suggesting a potentially high risk of recurrence should HBOT be re-administered.[10] This raises important questions regarding the balance of potential benefit versus harm when considering readministering HBOT in this context.

The solubility characteristics of oxygen differ substantially from nitrogen, with oxygen being more rapidly resorbed and potentially less harmful than air bubbles.[8,11,12] This has led to interest in alternative management strategies. The use of normobaric heliox may theoretically accelerate off-gassing and reduce bubble size by increasing the oxygen diffusion gradient, although a counter argument exists that due to increased solubility, helium might diffuse into a bubble faster than oxygen may diffuse out, and limit the rate of reduction, or even increase, the size of the bubble; clinical evidence to support either argument remains limited, and for this reason the authors currently suggest keeping the patient breathing air only, provided the patient's oxygen saturations are normal. If a patient has been decompressed on any other gas than oxygen, supplemental oxygen would of course still be indicated.

Another cornerstone of treatment of an air embolism is the use of a high fraction of inspired oxygen to denitrogenate the tissues and increase the gradient of intra-bubble to intra-vascular or tissue nitrogen. The authors postulate that in the case of an oxygen embolism, increasing the inspired oxygen fraction may offer less benefit than in nitrogen-containing emboli, and may theoretically reduce the diffusion gradient for bubble resolution; however, clinical evidence to guide optimal gas management in this context is lacking.

## RECOMMENDATIONS

Oxygen gas embolism is an extremely rare complication of HBOT, with only two prior published case reports to the best of our knowledge.[9,10] In low risk populations, the use of routine imaging prior to HBOT does not provide a cost-effective way to reduce the risk of barotrauma.[1,13–15] Given the extremely low incidence of either barotrauma or oxygen gas embolism resulting from HBOT, we do not advocate for increased screening practices for underlying pulmonary disease.

Clinicians assessing suitability of patients for HBOT should pay close attention to respiratory pathology at the time of assessment; emphasis should be on thorough history,

examination and review of any prior imaging, and, given its greater sensitivity over chest X-ray, HRCT chest should be the preferred imaging modality for those in whom concerns are identified. Lung function testing is unlikely to reveal significant abnormality in the majority of patients with Birt-Hogg-Dubé syndrome.[16]

Heightened vigilance is paramount during the decompression phase of HBOT and any onset of neurological change during or immediately after decompression should raise suspicion for arterial oxygen embolism, in the same way immediate collapse on surfacing from a dive is strongly suggestive of CAGE. Clinicians should consider performing HRCT chest at the same time as a stroke protocol CT.

The optimal management of cerebral arterial oxygen embolism from pulmonary barotrauma during HBOT likely differs in some ways from other CAGE events. With the goal of '*primum non nocere*' we elected to not recompress the patient, and whilst we welcome input on the best management of this rare condition from the wider diving and hyperbaric medicine community, we postulate that the optimal inspired gas to speed the off-gassing of oxygen from a cerebral arterial oxygen embolus, may in fact, be air.

## Conclusions

We report an extremely rare case of cerebral arterial oxygen embolism associated with decompression in a hyperbaric chamber in a patient with multiple, previously undiagnosed lung cysts. Given the rarity of this complication, we do not recommend change in screening practices, however we encourage careful consideration of possible respiratory pathology during patient assessment and in particular, vigilance during the decompression phase of HBOT is strongly advised.

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