Original articles

Functional outcome and quality of life after iatrogenic cerebral air embolism treated with hyperbaric oxygen: a prospective cohort study

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Abstract
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Introduction: Iatrogenic cerebral air embolism (CAE) is an uncommon, potentially fatal condition characterised by accidental introduction of air into the circulation during invasive procedures. Prompt recognition and treatment with hyperbaric oxygen therapy (HBOT) are required. Data on long-term functional outcome and specifically quality of life (QoL) in patients experiencing CAE are limited.

Methods: This prospective, single-centre, observational cohort study examined patients with iatrogenic CAE who were treated with HBOT. Patient characteristics, clinical severity scores and treatment details were recorded. The primary outcomes of the study were the Glasgow Outcome Scale (GOS) score at discharge and six months, and QoL measured by the World Health Organization quality of life brief version at six months.

Results: A total of 22 patients were included, with 14 patients (64%) having arterial CAE, five (23%) retrograde venous CAE, and the remaining three having either both (n = 1) or unknown (n = 2) forms of CAE. Median time-to-HBOT was seven hours [IQR 5–10]. The overall mortality rate was 23% (n = 5), eight of 22 patients achieving full recovery (GOS 5) at six months, and another six patients having moderate disability (GOS 4) at six months. Nine of 17 survivors (53%) reported a decline in QoL compared to their pre-incident status. Outcome in patients with retrograde venous CAE seemed to be better, and outcome in patients with CAE following neuroangiographic procedures for stroke or subarachnoid haemorrhage seemed to be worse, compared to the remainder of patients.

Conclusions: Iatrogenic CAE is associated with substantial morbidity and mortality, with only a third of patients in our cohort achieving good functional recovery. Over half of survivors in this cohort self-reported reduced QoL as compared to their situation before the CAE incident.

Introduction

Inadvertent introduction of gas into the vasculature is a known risk of diving, because reduction of ambient pressure during ascent may lead to pulmonary barotrauma and consequent introduction of gas into the pulmonary veins. The most feared destination of these air bubbles is the cerebral and coronary arteries, requiring urgent application of hyperbaric oxygen therapy (HBOT). While having totally different causes, the iatrogenic introduction of air into the cerebral vasculature bears many similarities with diving related arterial gas embolism.[1] Iatrogenic cerebral air embolism (CAE) can occur during a wide variety of medical interventions, such as cardiac surgery, neurosurgery, vascular catheterisation, and lung biopsies.[2] The outcome of CAE ranges from asymptomatic to fatal.[3] Even though it is a rare condition that affects approximately 2.65/100,000 hospitalised patients, its high morbidity and mortality makes it of critical concern to have a high index of suspicion.[4]

Besides prevention, which is reliant on stringent procedural protocols and awareness during at-risk interventions,[5] rapid diagnosis and intervention are key to reduce morbidity and mortality associated with CAE. Prompt application of an

inspired oxygen concentration as close to 100% as possible, followed by HBOT, is the gold standard treatment.[6] HBOT reduces air bubble size according to Boyle's law and by expediting denitrogenation, thereby aiming to restore blood flow. Additionally it enhances oxygen delivery to affected tissues and has anti-inflammatory properties.[7] Its proven efficacy in mitigating neurological injury has been well documented, and there is an inverse relationship between time to HBOT initiation and the probability of a favourable outcome.[8]

Despite the well-documented risks associated with CAE there is still limited research exploring the long-term impact on patients' functional outcome, and quality of life (QoL) specifically.[4] While acute management focusses on survival and immediate complications, patients may experience long-term deficits.[4] It is not unreasonable to assume that these persistent issues can significantly impact their functional abilities and overall well-being. This study reports on the six-month functional outcome and QoL of prospectively followed patients with CAE who were treated with HBOT.

Methods

STUDY DESIGN AND PATIENTS

This prospective, single-centre, observational cohort study was conducted at the department of Hyperbaric Medicine of the Amsterdam University Medical Center. The local ethics committee reviewed the study and waived the need for formal approval (review number W22_455). Written informed consent was obtained from all patients or their lawful representatives. We included patients from inception of the database in 2020 until the end of 2023 who met the following criteria: (1) at least 18 years of age at the time of diagnosis; (2) diagnosed with CAE; (3) able to communicate in Dutch. Diagnosis of CAE was made clinically (intravascular air entry followed by neurological deterioration) and/or radiologically (presence of intravascular air on computed tomography [CT]).

CLINICAL EVALUATION

Collected data included: general patient characteristics; type and source of the CAE; Glasgow Coma Scale (GCS) score at presentation; neurological, circulatory, and respiratory symptoms; use of high-flow supplemental oxygen before initiation of HBOT; details of HBOT. Type of the CAE (i.e., the cerebral vasculature affected by the air bubbles) was determined to be arterial, retrograde venous, both or unknown, based on a combination of causative procedure, symptomatology and location of intravascular air on imaging. To evaluate illness severity the Acute Physiology and Chronic Health Evaluation II (APACHE-II) and Simplified Acute Physiology Score II (SAPS-II) scoring systems were used.

TREATMENT

All patients were provided with supportive care, including sedation, respiratory management and mechanical ventilation when clinically required. HBOT was initiated as soon as possible after presentation, with the first session adhering to the U.S. Navy Treatment Table 6 (starting at 284 kPa, 2.8 atmospheres absolute [atm abs]) with the possibility of extension if required.[9] Subsequent treatments were conducted daily at 243 or 253 kPa (2.4 or 2.5 atm abs) with a session duration of two hours. Sessions could be repeated to a maximum number of 10 if the patients demonstrated ongoing clinical improvement. In 2022, after consultation with international experts, the protocol was updated to mandate a minimum of three HBOT sessions for all patients, unless the neurological situation was deemed so severe that continuation of HBOT was considered futile.

FOLLOW-UP

Glasgow Outcome Scale (GOS) score was determined at discharge and six months after the event in a telephone call with the patient (or relative, if deceased or unable) by one of the authors (RPW). At six-month follow-up patients were sent the Dutch version of the World Health Organization quality of life brief version WHOQOL-BREF[10] by email or postal mail and requested to fill it out and return it. This questionnaire is a shortened version of the commonly used WHOQOL-100 which investigates QoL on four domains: physical, psychological, social, and environmental. Questions are answered on a 1–5 scale and scores of the four domains are transformed into a 0-100 score according to the official instructions from the WHOQOL user manual (WHO/HIS/HSI Rev.2012.03). In addition to the WHOQOL-BREF questionnaire we asked two supplementary questions regarding self-reported change in QoL between the situation before the CAE and the current situation, and the perceived relationship of this change (if any) with the CAE.

STATISTICS

Data for this study were prospectively entered in Castor EDC (Ciwit BV, Amsterdam, the Netherlands). For patients referred to our institution, prospective data collection started on arrival in our hospital. All efforts were made to retrospectively collect data from the referring hospital. Only descriptive statistics are presented, using GraphPad Prism (version 10.2; GraphPad Software, San Diego, CA, USA), and no inferential statistical analysis was performed.

Results

CLINICAL EVALUATION

Over the four-year period 22 patients were entered into the database, all of whom were eligible for inclusion in this study (Table 1 and Table 2). Most common causative

Table 1
Patient characteristics, severity of illness at admission, and presenting symptoms; for each variable, *n* indicates the number of patients with available data. Continuous data are shown as median (interquartile range). APACHE-II – Acute Physiology and Chronic Health Evaluation II; SAPS-II – Simplified Acute Physiology Score II

Characteristic	*n* = 22
Age (years), median (range)	69 (60–76)
Female sex, *n* (%)	11 (50%)
Referred patient, *n* (%)	16 (73%)
Severity scores at admission, median (range)	
Glasgow Coma Scale score, *n* = 21	10 (8–13)
SAPS-II, *n* = 17	31 (30–42)
APACHE-II, *n* = 18	20 (15–22)
Presenting symptoms, *n* (%)	
Neurological, *n* = 21	21 (100%)
Lateralization, *n* = 20	15 (75%)
Impaired consciousness, *n* = 19	14 (74%)
Confusion / agitation	6 (29%)
Seizure	3 (14%)
Circulatory, *n* = 21	12 (57%)
Cardiopulmonary resuscitation	2 (9.5%)
Hypotension	3 (14%)
Hypertension	4 (19%)
ST-segment abnormalities	4 (19%)
Arrhythmias	2 (9.5%)
Respiratory, *n* = 21	7 (33%)
Respiratory insufficiency	5 (24%)
Haemoptysis	2 (9.5%)

procedures were either radiological or thoracic in nature. In all cases air was the gas involved. Neurological status prior to HBOT was unknown for one patient who was diagnosed intraoperatively based on witnessed air entry into a peripheral vein and air bubbles in the carotid arteries on ultrasound. This patient remained intubated until HBOT. In the remaining 21 patients median GCS score at presentation was 10 [interquartile range (IQR) 8–14]. Fifteen patients (68%) exhibited signs of lateralisation, and three (14%) presented with epileptic seizures. Circulatory symptoms developed in 12 patients (57%), including two who required cardiopulmonary resuscitation. Respiratory symptoms were present in seven patients (33%).

TREATMENT

Nine patients (41%) received full treatment with high flow supplemental oxygen, defined as initiation soon after diagnosis of CAE and continuation until start of HBOT (Table 3). HBOT was initiated a median of seven hours from symptom onset, which includes two outliers who received their initial session at 22 and 23 hours post embolism. All

Table 2
Air embolism characteristics, imaging and causative medical procedures

Air embolism characteristics	*n* (%)
Vascular bed of entry	
Cerebral arterial	5 (23%)
Systemic arterial	2 (9.1%)
Pulmonary venous	6 (27%)
Systemic venous	8 (36%)
Left heart	1 (4.5%)
Type of cerebral air embolism	
Arterial	14 (64%)
Retrograde venous	5 (23%)
Both	1 (4.5%)
Unknown	2 (9.1%)
Air on imaging, *n* = 21	
Yes, intracerebral air	10 (48%)
Yes, air in other vasculature	4 (19%)
No air	7 (33%)
Causative medical procedure	
Interventional radiology	
Neuro angiography	5 (24%)
Cardiac angiography	2 (9.1%)
Thoracic procedures	
Cardiac valve surgery	1 (4.5%)
Bronchoscopy	1 (4.5%)
Lung or pleural biopsy/puncture	4 (18%)
Chest drain manipulation	1 (4.5%)
Other	
Haemodialysis	3 (14%)
Arthroscopy	1 (4.5%)
Endoscopy	1 (4.5%)
Central venous catheter	2 (9.1%)
Peripheral venous catheter	1 (4.5%)

initial sessions adhered to the treatment protocol, and for two patients an extension at 284 kPa (2.8 atm abs) was applied. Both these patients were not intubated, and extension was performed due to persistence of neurological symptoms. A total of 82 HBOT sessions were conducted, with 19 (86%) patients receiving at least two sessions and 12 (55%) patients receiving at least three sessions. The majority of abnormalities (10 of 19) in the hyperbaric chamber occurred during the first session (Table 3).

FOLLOW-UP

The median length of hospital stay was six days [IQR 2–9], with one patient who died on the second day post-embolism after withdrawal of life support because of the extent of

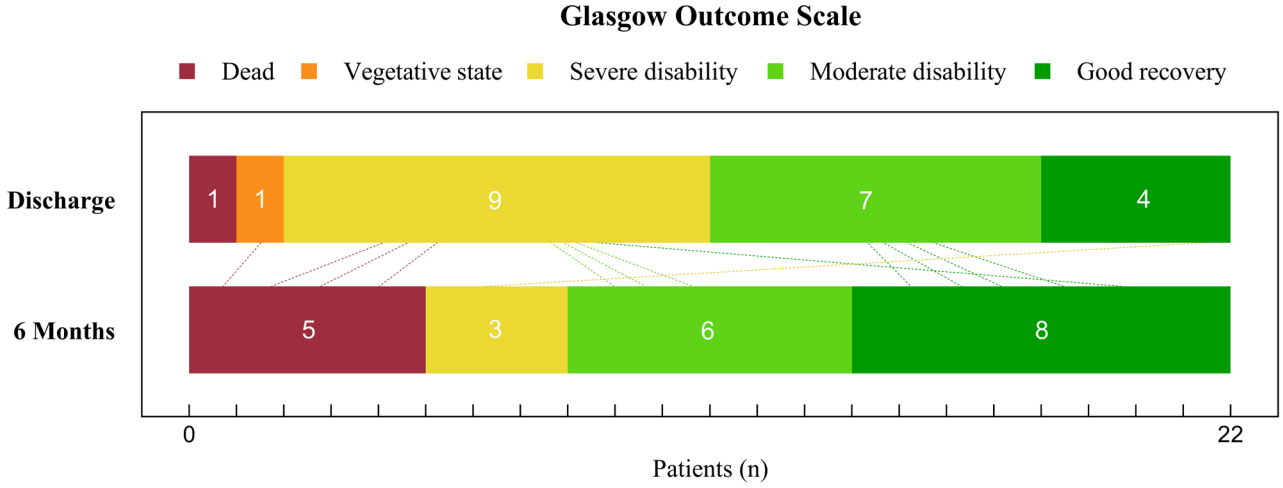
Table 3

Treatment characteristics and abnormalities during hyperbaric oxygen therapy (HBOT); continuous data are shown as median (interquartile range). Regarding high-flow supplemental oxygen prior to HBOT, 'completely' means initiation of (close to) 100% inspired oxygen concentration immediately after diagnosis until HBOT, 'partly' means that supplemental oxygenation was performed, but not from diagnosis until HBOT and/or not with adequately high flow

Treatment characteristics		**Abnormalities during HBOT (*n* = 82 sessions)**	
Time from incident to diagnosis (hours)	1 (0–2)	Psychomotor agitation	6 (7.3%)
Time from incident to HBOT (hours)	7 (5–10)	Seizure without indication for O_2 toxicity	5 (6.1%)
Intubated prior to HBOT	13 (59%)	Respiratory deficits requiring supportive care	3 (3.7%)
Normobaric oxygen prior to HBOT		Vomiting	2 (2.4%)
Completely	9 (41%)	Neurological deterioration	1 (1.2%)
Partly	4 (18%)	Seizure suggestive of O_2 intoxication	1 (1.2%)
None	9 (41%)	Respiratory deficits requiring intubation	1 (1.2%)
HBOT sessions per patient	**3 (2–5)**		
With anaesthetist present	3 (1–4)		

Figure 1

Stacked bar chart illustrating the Glasgow Outcome Scale (GOS) score at hospital discharge and six-month follow-up; each bar is subdivided into GOS scores 1 (left) to 5 (right). Dotted lines between bars show changes in individual patient outcomes from discharge to six-month follow up



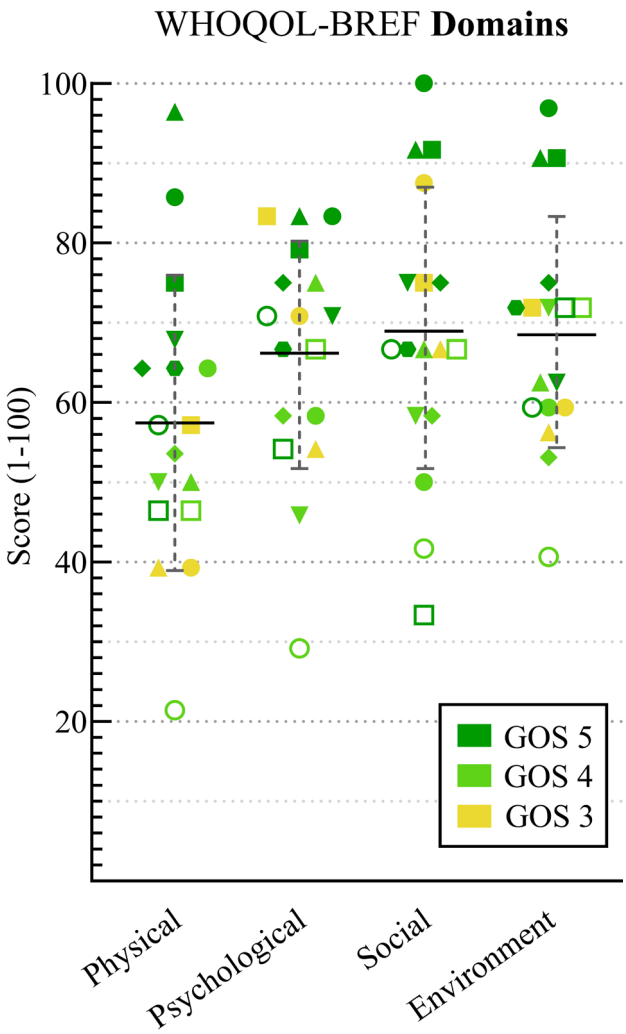
cerebral injury. The overall mortality was 23% (five patients) at six months. GOS data were received from all surviving patients at discharge, GOS and QoL data were received from all surviving patients at six months. At the time of discharge the median GOS score was 4 [IQR 3–4], and at six-month follow-up the median GOS score was 4 [IQR 3–5] (Figure 1).

To further assess QoL after sustaining CAE, QoL was measured using the WHOQOL-BREF questionnaire at six-month follow-up. Median overall perception of QoL and health satisfaction were both 3 out of 5 [IQR 2–4]. The mean (SD) scores for the WHOQOL-BREF in the four domains were as follows: physical health 58 (18), psychological health 66 (15), social relationships 69 (18), and environment 69 (15) (Figure 2).

SUBGROUPS

Retrograde venous CAE has a distinct aetiology that differs from arterial CAE. Likewise, patients with arterial CAE as a complication of a neuroangiographic procedure for stroke or subarachnoid haemorrhage are an interesting subpopulation, because they suffered two serial episodes of cerebral injury. Although no formal statistical testing was performed, it seems from Table 4 that patients with retrograde venous CAE had better outcomes than the remainder of patients. All five patients survived and reported no deterioration of their health as compared to before the CAE. However, they were also younger and had shorter time from incident until start of HBOT than the remainder of patients. Conversely, only two of five patients with CAE after neuroangiographic procedures survived, and both survivors self-reported

Figure 2
World Health Organsiation quality of life brief version (WHOQOL-BREF) domain scores at six month follow-up in the 17 survivors, color-coded by Glasgow Outcome Scale (GOS) score; symbols represent individual patients and the black and grey lines show the mean (standard deviation) for each domain



deterioration of their health compared to before the event. They were, however, older and more likely to be intubated before HBOT.

Discussion

In this prospective cohort study we evaluated the outcome and QoL of 22 patients who suffered iatrogenic CAE and were treated with HBOT. To our knowledge, this is only the second prospective study focusing on iatrogenic CAE. Crude mortality in this study was 23%, which underscores the severity of this condition. At six-month follow-up, eight of 22 patients achieved complete recovery (GOS 5) and another six of 22 patients had moderate disability (GOS 4). Despite HBOT and supportive care, the remaining eight of 22 patients either died (n = 5, GOS 1) or were left with severe disabilities (n = 3, GOS 3).

In our cohort, neurological dysfunction most frequently manifested as signs of lateralisation (75%) and/or decreased consciousness (74%), with circulatory (57%) and respiratory (33%) disabilities also frequently observed. In general, the severity of illness was somewhat less pronounced than that reported by the only previously published prospective cohort study, in which median GCS score was eight [IQR 3–15] and SAPS-II was 33 [IQR 21–55], in contrast to our median GCS score of 10 [IQR 8–14] and SAPS-II of 31 [IQR 30–42].[4] Furthermore, more patients in the previously published cohort experienced cardiac arrest and shock compared to our study.

The profile of causative procedures appears to be different in our study than in previously reported series. In earlier studies, cardiac surgery and central venous catheterisation were identified as the primary cause, in contrast to our cohort with predominantly radiological and lung procedures.[4,11,12] One explanation may be the general increase in the use of interventional radiology, for instance for mechanical thrombectomy in acute stroke, which by its nature carries a high risk of CAE.[13] Similarly, the inherent nature of lung surgery and lung biopsies continues to pose a procedural risk.[14]

Interestingly, although CAE is regarded a primarily clinical diagnosis, CT imaging was available in all but one of our cases, showing intravascular air in 67% of them. In diving medicine, a diver who surfaces with acute neurological injury may be treated with HBOT without prior cerebral imaging. The fact that in our clinical cases almost all patients undergo CT imaging is probably a result of the clinical reality in which the incident occurs. The rarity of iatrogenic CAE, suboptimal clinical awareness among non-hyperbaric physicians and the broad differential diagnosis of acute neurological symptoms in the hospitalised population most likely explain why most patients in our series underwent CT imaging. It must be reiterated, however, that intravascular air is not a prerequisite for the diagnosis of CAE, although its presence does support the diagnosis.

Previous CAE studies have largely focused on neurological sequelae and functional outcomes (e.g., GOS), rather than patient-reported QoL. We applied the WHOQOL-BREF at six months, providing new insights into self-perceived well-being. It must be noted that all our patients were, by definition, already exposed to a medical procedure before they suffered CAE. Compared with previously hospitalised elderly patients (mean age 80 years) who reported QoL six months after hospital admission, our CAE survivors reported comparable scores in the physical (58 vs. 58), psychological (66 vs. 67) and social (69 vs. 65) domains, and better scores in the environmental (69 vs. 55) domain.[15]

Table 4

Outcome for all patients, patients with retrograde venous cerebral arterial embolism (CAE), patients with CAE that occurred during neuroangiography, and all patients with exception of these two categories; continuous data are shown as median (interquartile range). *as only two observations were made the unique values of both patients are shown; CAE – cerebral air embolism; GCS – Glasgow Coma Scale; HBOT – hyperbaric oxygen therapy

Characteristic	All patients *n* = 22	Retrograde venous CAE *n* = 5	CAE during neuroangiography *n* = 5	Remainder
General				
Age	69 (60–76)	59 (50–73)	75 (69–77)	68 (61–76)
Female sex	11 (50%)	2 (40%)	2 (40%)	7 (58%)
GCS score at admission	10 (8–13) (*n* = 21)	12 (9–15)	9 (8–9)	11 (8–14) (*n* = 11)
Intubated prior to HBOT	13 (59%)	2 (40%)	4 (80%)	7 (58%)
Time from incident to HBOT	7 (5–10)	5 (5–5)	6 (3–11)	8 (5–11)
Overall mortality	5 (23%)	0 (0%)	3 (60%)	2 (17%)
Discharge				
n survivors	21	5	4	12
Glasgow outcome score	4 (3–4)	5 (4–5)	3 (3–3)	4 (3–4)
Six months				
n survivors	17	5	2	10
Glasgow outcome score	4 (3–5)	5 (3–5)	1 (1–4)	4 (4–5)
Quality of life	3 (2–4)	3 (2–4)	3 and 4*	4 (2–4)
Health satisfaction	3 (2-4)	3 (2–4)	3 and 4*	4 (2–4)
Health deterioration	9 (53%)	0 (0%)	2 (100%)	7 (70%)

When comparing functional recovery in our cohort to previous CAE research, our outcomes appear less favourable. The prospective study by Bessereau et al. notes that 75% of patients achieved a good recovery by six months, while only 33% of our patients reached this level of functional independence.[4] It must be noted, however, that Bessereau et al. included not only CAE, but also cases of gas embolism with only circulatory abnormalities. It cannot be calculated from their data how many patients actually had CAE and how many had gas embolism without cerebral involvement. In this regard, the retrospective study by Blanc et al. is more informative, since they only included patients experiencing cerebral gas embolism.[12] Although GCS is not reported, their 70% incidence of impaired consciousness at presentation is comparable to our data. They report that 58% of their patients had full recovery on discharge, compared to 14% in our data (increasing to 36% at six months). Another retrospective study, by Beevor and Frawley, that only included patients with cerebral gas embolism, reported a mean moderate disability at discharge (6.5 on the extended GOS that ranges from 1 to 8),[11] whereas our median GOS score at discharge was only three, reflecting severe disability. However, median GOS score in our cohort increased to four (moderate disability) at six-month follow up, and these data are not known for the Beevor and Frawley cohort.[11] The comparatively worse condition of our patients at discharge may be partly explained by the fact that the percentage of patients with hemiplegia was much higher in our cohort (75% as opposed to 20% in the Beevor and Frawley study) and the fact that hemiplegia was associated with poor outcome in the Beevor and Frawley study.

An important point to make when comparing studies, is that distinction between cerebral arterial and cerebral retrograde venous air embolism is usually not made. Although the abovementioned studies do report on arterial versus venous embolism, from the text it can be deduced that this refers to the type of vessel where the air was introduced, not to the cerebral vasculature in which the bubbles lodged. Despite absence of studies investigating the clinical outcome of arterial versus retrograde venous CAE, mechanistically it can be hypothesised that retrograde venous embolism has a more benign course, given the fact that these bubbles have ascended retrogradely to the cerebral veins and therefore did not obstruct arterial flow. The small size of our cohort precludes meaningful quantitative analysis, but it is interesting to note that the patients with retrograde venous CAE seemed to have higher GOS score at six-month follow up and none of them self-reported a deterioration in their health compared to baseline. On the other hand, however, these patients were younger and their median time to HBOT was shorter than in the remainder of the study population, which may confound the suggested improved outcome.

Another relevant subgroup in our cohort is formed by the five patients who suffered CAE as a complication of either mechanical thrombectomy for stroke (n = 4) or coiling after subarachnoid haemorrhage (n = 1). These patients suffered two serial episodes of cerebral injury. Only two of these patients survived and both reported reduced QoL compared to before the events. The increasing use of neuro-interventional procedures warrants further study into the incidence of CAE and the role of HBOT in these patients.

Strengths of our study are its prospective nature and complete availability of outcome data. Several limitations should however be acknowledged. Firstly, the relatively small size of our cohort, reflective of the rarity of CAE, constrains the generalisability of our findings and precludes statistical analysis to identify prognostic factors. This is compounded by the heterogenic background of patients suffering CAE, which is indicative of the wide variety of medical procedures that can cause this complication. Secondly, the functional status of our patients before the CAE incident is unknown. All patients at least required a medical intervention that caused the CAE. It cannot be determined with certainty to what extent the functional outcome as determined at six months is attributable to the CAE, or to any underlying disease. Specifically, when CAE occurs as a complication of a neurological intervention such as thrombectomy after stroke, analysis of outcome is troubled by the fact that patients already suffered from neurological injury before they sustained CAE. Lastly, the follow-up period was limited to six months, which in neurological disorders may not adequately reflect final outcome. Nevertheless, evidence from stroke literature suggests that the six-month timepoint can be predictive of longer-term results.[16]

Conclusions

We have shown that in our cohort of 22 patients who suffered iatrogenic CAE, six-month mortality was 23%, while 36% obtained good functional outcome. The fact that only 41% of our patients received continuous high-flow supplemental oxygen prior to HBOT indicates a potential for improving care, by continuing emphasis on this important initial treatment. Also, the median time until initiation of HBOT of seven hours, despite only a median of one hour between onset of symptoms and diagnosis, suggests that familiarity with HBOT and logistical factors such as referral and transportation can still be optimised. Given the recently reported[8] clear inverse relationship between time-to-HBOT and probability of favourable outcome, all efforts should be pursued to initiate HBOT as early as possible. The suggestion of improved outcome in retrograde venous CAE as compared to arterial CAE warrants further investigation in future studies. The specific subpopulation of CAE after neuroangiographic procedures also requires additional study.

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