Original articles

Potential missed cerebral arterial gas embolism in patients with in-hospital ischaemic stroke

Afshin Khazei, David Harrison, Riyad B Abu-Laban and Angeli Mitra

Key words
Cerebral arterial gas embolism (CAGE), air embolism, brain injury, hyperbaric oxygen therapy

Abstract

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Cerebral arterial gas embolism (CAGE) has been characterised as under-diagnosed, under-treated, and under-reported. It is associated with a number of commonly performed diagnostic and therapeutic procedures, and penetrating trauma to the neck or chest. This study was carried out to determine the frequency with which physicians at a university-based, tertiary-care teaching hospital with a hyperbaric facility considered the diagnosis of CAGE in patients with a stroke occurring during, or shortly after, a procedure known to be high risk for CAGE. A retrospective chart review was undertaken from April 2002 to March 2003 to identify all patients who had suffered an in-hospital ischaemic stroke (IHIS) within four hours of a high-risk procedure or penetrating trauma, and presented with symptoms consistent with the diagnosis of CAGE. Of the 150 cases of "stroke as a post-admit morbidity", 46 were classified as an IHIS. In 15 of these 46, a diagnosis of CAGE could not be excluded. Symptom onset occurred during the procedure in three cases and within one hour in five cases. Procedures most commonly associated with IHIS were percutaneous coronary angioplasty (6) and cardiopulmonary bypass (6). In only one of the 15 cases was CAGE considered in the differential diagnosis. Only two patients were functioning independently at the time of discharge, whilst two died. This study suggests that in our hospital the diagnosis of CAGE is rarely considered in patients presenting with symptoms of IHIS within four hours of a high-risk procedure. These findings are important because untreated CAGE is associated with major mortality and morbidity.

Introduction

Cerebral arterial gas embolism (CAGE) is the introduction of gaseous emboli into the cerebral arterial circulation. It is associated with pulmonary barotrauma from scuba diving, penetrating trauma of the neck and thorax, and with a wide range of surgical and medical procedures.[1]

Vancouver General Hospital (VGH) is a tertiary-care teaching centre affiliated with the University of British Columbia (UBC) in Canada. Although numerous procedures known to be associated with CAGE are performed each year at our institution, a search of the hospital's computer database over the past 21 years (1983–2004) identified only six cases in which the diagnosis of iatrogenic CAGE was made. Features that led to the diagnosis being made in these cases included: intravascular air detected by intra-operative transcranial Doppler, documented injection of air instead of contrast during angiography, and air seen on CT scan.

It has been suggested that clinically significant cases of iatrogenic CAGE are largely under-diagnosed, under-treated, and under-reported.[2] This underlies the difficulty in establishing the true incidence of CAGE and, given the major morbidity and mortality associated with CAGE, it is an important area to address. Hyperbaric oxygen therapy (HBOT) is the current standard of care for clinically significant CAGE,[2–7] and there is some evidence that patients with suspected iatrogenic CAGE who are treated with early HBOT have better outcomes than patients in whom this treatment is delayed.[8]

The primary objective of this study was to determine the frequency with which physicians at a university-based, tertiary-care teaching hospital with an on-site hyperbaric facility consider the diagnosis of CAGE in patients presenting with compatible symptoms after a high-risk procedure or penetrating trauma. A secondary objective was to determine in what proportion of possible CAGE cases HBOT was administered or considered.

Methods

After obtaining ethical approval from UBC and VGH, we undertook a retrospective chart review from April 2002 to March 2003 to identify all patients who had suffered an in-hospital ischaemic stroke (IHIS). The charts were identified through a manual and computer-generated search looking for "stroke as a post-admit morbidity" in the hospital's computer database of diagnostic and procedure codes. The search was conducted by a database analyst specialising in neurology. One hundred and fifty charts were found and reviewed by one of the investigators or research assistants using explicit criteria. The two research assistants were medical students

in their final year of school who had received training in chart review and the use of the standardised data collection form. Prior to commencing data collection, a preliminary set of medical records was analysed by the research assistants and the results reviewed by the principal investigator to ensure quality. All patients had been seen by a neurologist and the neurologist's consultation note was used to confirm the diagnosis of stroke.

We defined patients at risk for CAGE as those who were diagnosed as having an ischaemic stroke with onset of signs or symptoms either during a procedure associated with CAGE, within four hours of the end of such a procedure, or within four hours of awakening from anaesthesia after such a procedure. We employed this definition because it represents the criteria by which our hyperbaric oxygen facility at VGH would consider treatment for "possible CAGE". A list of procedures associated with CAGE was defined *a priori* (Table 1). Additionally, we reviewed all identified charts for any evidence of penetrating trauma to the head, neck, or thorax preceding the stroke. One case of IHIS that occurred during a bone marrow biopsy was excluded from our study because the procedure (even though it has been reported as a cause of CAGE) was not on our *a priori* list.

In addition, mortality and morbidity measures were collected for patients in the study group in order to provide some insight into the natural history of untreated procedure-associated IHIS. The same outcome measures are also reported for possible CAGE patients who were treated with HBOT at VGH in 2006. Descriptive statistics were used to present the data generated from this study.

Table 1
Procedures identified *a priori* as associated with risk of cerebral arterial gas embolism (CAGE)

Coronary artery bypass graft (CABG)
Heart valve surgery
Carotid endarterectomy
Central or arterial line insertion/manipulation
Cardiopulmonary bypass
Carotid artery injection/angiogram
Percutaneous coronary angioplasty (PTCA)
Liver transplantation
Haemodialysis
Brain surgery
Hysteroscopy
Thoracotomy
Transthoracic needle
Intra-aortic balloon pump
Laparoscopy
Endoscopy
Arthrography
Hip replacement
Transurethral prostatic resection (TUPR)

Results

The search for "stroke as a post-admit morbidity" identified 150 patients. After direct chart review by the investigators, and using a neurologist's consultation note as our gold standard, 85 patients were excluded because the final diagnosis was a disorder other than stroke (for example, delirium) or was pre-hospital stroke. These 85 charts had been miscoded in the database, leaving 65 patients with a true diagnosis of an in-hospital stroke. The 65 in-hospital stroke patients were further classified as: ischaemic stroke more than four hours after the procedure (19), ischaemic stroke within four hours of procedure (15), haemorrhagic stroke (15), no associated procedure (10), diagnosis unclear from the chart (4), procedure not listed in our *a priori* list (1), or other cause of stroke identified (1). There were no cases of IHIS after penetrating trauma.

Of the 46 cases of IHIS, 15 (32.6%) occurred during or within four hours of a high-risk procedure associated with CAGE. Symptom onset was during the procedure in three cases, within one hour in five cases, between one to four hours in four cases, and within four hours of awakening from post-operative anaesthesia or sedation in three cases. The procedures most commonly associated with CAGE were percutaneous coronary angioplasty (PTCA) (6 cases), and cardiopulmonary bypass for coronary artery bypass graft (CABG) or other cardiac surgery (6 cases) (Table 2). A search of the hospital database revealed that 1,622 PTCA and 556 CABG procedures were carried out during the one-year time period of our study.

In only one of the 15 cases was CAGE documented to be part of the differential diagnosis, and no patients were administered HBOT or referred for consideration of HBOT. The outcomes for these 15 patients are summarised in Table 3 (left-hand column). Of the 13 patients who survived, the median hospital stay was 18 days, and only two patients were independent at the time of discharge, the majority going to long-term care and all requiring rehabilitation.

Discussion

This study suggests that in our tertiary-care teaching hospital where HBOT is readily available, the diagnosis of CAGE is rarely considered in patients presenting with symptoms of IHIS within four hours of a procedure known to be associated with CAGE. Our results are limited by the use of a retrospective chart review methodology; however, they provide compelling evidence that the diagnosis of CAGE is rarely considered in at-risk patients.

A major limitation of our retrospective study is that some cases of IHIS may have been missed by our search methodology due to improper diagnostic coding or under-reporting of peri-operative complications. This would tend to cause an under-estimation of the number of "possible CAGE" cases.

Table 2. Clinical data for IHIS patients having symptom onset during

Age	Sex	Procedure	Time to onset	Clinical presentation
78	M	PTCA	During procedure	R hemiplegia, arm worse than leg, aphasia
77	F	PTCA	During procedure	Hypotension during procedure, L homonymous hemianopsia
82	F	CPB, CABG, CL, AL, valve surgery	During procedure	L arm weakness, LoC
55	M	PTCA	13 min	R hemiplegia, R facial weakness, aphasia
75	M	PTCA	15 min	LoC during procedure, L arm flaccid
80	M	PTCA	25 min	R hemiplegia
68	F	Thoracotomy, CPB, aortic valve replacement, CL, AL, laparotomy	25 min	LoC, R hemiplegia, aphasia
83	M	CPB, Aortic arch dissection repair	30 min	Hypotension, aphasia, R sided weakness
73	F	CPB, CL, AL, valve surgery, IABP	75 min	L hemiplegia (arm worse than leg)
57	F	Craniotomy	138 min	Seizure, L arm weakness
70	M	CABG	150 min	Unable to follow commands
69	F	Bronchoscopic laser debridement of thoracic tumour	150 min	Aphasia, L sided weakness, dyspnoea
73	M	CPB, CABG, valve surgery	8 h (SED)	R hand weakness, aphasia
69	F	PTCA	10 h 40 min (SED)	Frontal headache, L hemiparesis, L facial weakness
63	F	CL, AL, thoracotomy	19 h (SED)	R sided weakness, aphasia, dysarthria, R sided neglect

ACA, MCA, PCA – anterior, middle and posterior cerebral artery; AF – atrial fibrillation; AL – arterial line insertion; CABG – coronary artery bypass hypertension; IABP – intra-aortic balloon pump; ICA – internal carotid artery; LoC – loss of consciousness; PTCA – percutaneous coronary

It could be argued that some of the 19 patients who had IHIS after a procedure associated with CAGE but with onset of symptoms more than four hours after the procedure may still have benefited from HBOT. We agree that the four-hour cut-off is arbitrary and HBOT may prove beneficial in these cases; however, an *a priori* threshold had to be chosen for this study. It was felt, after surveying all the hyperbaric physicians at our institution regarding which patients they would elect to treat and the likelihood of a temporal cause/effect relationship, that a four-hour cut-off was reasonable. As a result, our methodology may be under-representing the extent of this clinical problem.

HBOT influences the physiologic effects of CAGE by a number of mechanisms, including reduction in the size of intravascular gas bubbles by the direct physical effect of increased ambient pressure, enhanced nitrogen diffusion out of the bubble created by a steep diffusion gradient, improved oxygenation of ischaemic tissues, and decreased cerebral oedema.[9]

The consequences of bubble passage and vascular occlusion by air bubbles include endothelial damage, platelet activation, and alteration of white blood cells, leading to sludging and further vessel occlusion.[10] Several animal studies collectively suggest that HBOT blocks the leukocyte-mediated no-reflow phenomenon seen following tissue ischaemia.[11–13]

or within four hours of a procedure associated with CAGE (N = 15)

CT/MRI	Baseline risk factors
Normal	HT, HC, AF
Acute to subacute R PCA infarct; bilateral patchy low densities in frontal lobes in keeping with cardioembolic origin	HT, HC, carotid US: R ICA 40–60%, L ICA 0–40%
Multiple nonhaemorrhagic subacute infarctions in both cerebellar hemispheres and the left occipital lobe	HT, HC
Acute large L MCA and ACA ischaemic infarcts	HT, HC, smoker; R ICA stenosis 80%, L ICA 50%
Acute large R MCA ischaemic infarct	DM, HT, HC
Acute R thalamic and L posterior frontal ischaemic infarct	HT, HC, DM, R ICA 60%, L ICA 50%
Bilateral multifocal acute MCA territory infarcts	CT-angiogram Circle of Willis: normal vessels
Acute L parietal infarct	No risk factors
Acute multifocal infarcts R MCA distribution, embolic	Mitral regurgitation, HT
Pneumocephalus, venous infarct bilateral R > L	None
Acute mutifocal infarcts R and L cerebellum	DM
No acute changes	Carotid US normal
No acute changes	Smoker, carotid US normal
Acute multifocal infarcts L parietal, L frontal, L temporal	DM, HC, prior bilateral carotid endarterectomy
Acute multifocal infarcts L frontal, L parietal. Acute infarct L PCA watershed area	HC carotid US: L ICA 70–80%, R ICA 0–40%

graft; CL – central line insertion or manipulation; CPB – cardiopulmonary bypass; DM – diabetes mellitus; HC – hypercholesterolaemia; HT – angioplasty; SED – onset within 4 hours of awakening from post-operative sedation or anaesthesia; US – ultrasound

A study of 2,108 patients undergoing CABG found that 6.1% had adverse cerebral events.[14] Another study of 2,972 patients undergoing CABG, with or without valve surgery, reported a stroke incidence of 1.6%.[15] The risk factors for peri-operative stroke overlap with those for stroke in the general population.[14,15] These studies suggest that neurological dysfunction after these procedures is not a rare occurrence.

It is difficult to determine in which cases symptoms are due to CAGE as opposed to solid emboli. However, it is known that arterial gas embolism is a common phenomenon in open-heart surgery and left-heart catheterisation for percutaneous coronary angioplasty. A study using transcranial Doppler (TCD) provided evidence of cerebral arterial gas embolus in 10 of 10 patients undergoing open-heart valve procedures.[16] In 72 patients undergoing left-heart catheterisation, of whom 29 underwent coronary intervention, all had evidence of microembolic signals on TCD.[17] Of these events, 67.5% occurred during contrast or saline injection (mean rate of occurrence: 95 +/- 45 microembolic signals per patient), and 32% occurred during wire or catheter manipulations. A further study of 127 patients undergoing CABG documented microembolic signals related to either specific cardiopulmonary bypass (CPB) events (air emboli) or surgical manipulations (solid emboli).[18] It was concluded

Table 3
Summary outcome data for patients with procedure-associated IHIS not treated with HBOT and patients with suspected CAGE treated with HBOT

	Patients with procedure-associated IHIS not treated with HBOT (N = 15)	Patients with suspected CAGE treated with HBOT (N = 6)
Acute length of stay (days)		
Mean, Median	20.5, 18	6, 2
(SD, IQR)	(13.78, 11)	(7.10, 14)
1-year mortality	2/15 (13%)	0/6 (0%)
Discharge to long-term care	9/13 (69%)	0/6 (0%)
Discharge home	4/13 (31%)	6/6 (100%)
Chronic length of stay (days)		
Mean, Median	43, 32	0, 0
(SD, IQR)	(49.26, 50)	(N/A, 0)
Neurological status on discharge*	1 = 2/15 (13.3%) 2 = 0/15 (0%) 3 = 11/15 (73.3%) 4 = 2/15 (13.3%) 5 = 0/15 (0%)	1 = 0/6 (0%) 2 = 0/6 (0%) 3 = 0/6 (0%) 4 = 2/6 (33.3%) 5 = 4/6 (66.7%)
Rehabilitation needed	13/13 (100%)	2/6 (33.3%)

CAGE – cerebral arterial gas embolism; IHIS – in-hospital ischaemic stroke; HBOT – hyperbaric oxygen therapy
SD – standard deviation; IQR – interquartile range
*neurological status as per the Glascow Outcome Scale[32]:
1 – death; 2 – persistent vegetative state; 3 – severe disability (conscious but disabled and dependent on others for care); 4 – moderate disability (some disabilities but independent); 5 – good recovery

that although CPB-induced embolic events are more common than surgically-induced embolic events they are better tolerated by the patient.

There is no definitive test for CAGE. CT scan has been reported to identify cerebral bubbles in only 25% to 75% of patients with gas embolism[19] and MRI has not been documented to be reliable in identifying gas embolism.[20]

Although microemboli in the cerebral circulation may be identified by intra-operative TCD, conventional TCD does not distinguish between solid and gaseous emboli.[21] A technique for distinguishing between gaseous and solid emboli has been described using bilateral dual depth, dual frequency TCD.[22] It was able to distinguish between solid and gaseous emboli with 80% accuracy. Although this technique shows promise, it is not widely used during procedures that may cause CAGE.

The effect of hyperbaric oxygen on an undifferentiated population of patients presenting with stroke (CAGE and thromboembolic) with onset shortly after a high-risk procedure capable of causing CAGE is unknown, due to the lack of prospective studies. However, HBOT is considered the standard of care for known cases of CAGE. The consensus guidelines from the Hyperbaric Oxygen Committee Report (HOCR) recommend HBOT as the definitive treatment for CAGE.[1] These recommendations are based on the physiologic mode of action of HBOT on CAGE and a large number of human case series, human case reports and animal studies showing reversal of neurological deficits and cardiovascular compromise by HBOT. There are also reports of excellent clinical outcomes in patients treated with HBOT up to 60 hours after the event.[23–31] Based on this body of evidence, it has been suggested that a prospective randomised trial comparing HBOT to no HBOT for patients with CAGE would be unethical because the control group would be deprived of the benefits of HBOT.[8]

The lack of a reliable diagnostic test to identify or exclude CAGE has important implications for the management of patients such as the 15 identified in our study. If it is assumed that none of these patients have CAGE, then routine stroke management is appropriate. However, it is likely that some of these patients may have in fact suffered CAGE, in which case failing to consider the diagnosis as a cause of their symptoms could deny them access to a therapy that has the possibility of completely ameliorating an otherwise devastating injury.

This represents a potentially important area of preventable morbidity and mortality.[30]

The data we have gathered in this study for potential CAGE patients provide some insight into the natural history of IHIS occurring within four hours of a high-risk procedure. From the 15 patients observed, prognosis for untreated IHIS is seen to be poor.

Following this study, our hospital saw an increase in diagnosed cases of CAGE as well as awareness of HBOT as a treatment for CAGE. In 2006, six cases of CAGE were treated with HBOT at VGH. We have provided a brief summary of outcome measures for these patients, in addition to those of the 15 study patients who did not receive HBOT (Table 3, right-hand column). From this it is apparent that patients treated with HBOT for CAGE had consistently more favourable outcomes than procedure-associated IHIS patients who did not receive HBOT.[8,19,23,24,31] Although this study was not designed for comparison of these two groups, limiting interpretation, we feel that the results are of interest and are hypothesis generating. Many questions arise from these observations pointing to the need for future research.

Conclusions

This study suggests that in our tertiary teaching hospital, where HBOT is readily available, the diagnosis of CAGE is rarely considered in patients presenting with symptoms of IHIS within four hours of a high-risk procedure. This may represent an important area of preventable morbidity and mortality, which could be influenced by simply increasing awareness of CAGE and the role of HBOT in treating diagnosed CAGE cases. Further study is needed to determine if our results arise from an institutional anomaly or if they indicate a more widespread deficiency in the diagnosis and management of this potentially treatable condition.

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Afshin Khazei, MD, FRCPC-EM, ABEM, CCFP, AAFP, ABPM-HB, is Attending Physician, Department of Emergency Medicine, and Hyperbaric Oxygen and Diving Medicine Consultant, Vancouver General Hospital, and Clinical Associate Professor, Department of Surgery, University of British Columbia
David Harrison, MD, CCFP(EM), FRCPC, is Medical Director, Vancouver General Hospital Hyperbaric Unit and Clinical Associate Professor, Department of Surgery, University of British Columbia
Riyad B Abu-Laban, MD, FRCPC, is Attending Physician and Research Director, Vancouver General Hospital Department of Emergency Medicine, and Scientist, Centre for Clinical Epidemiology & Evaluation
Angeli Mitra, BScN, MSc, Department of Surgery, University of British Columbia, and Research Coordinator, Hyperbaric Unit, Vancouver General Hospital

Address for correspondence:
Angeli Mitra, Research Coordinator
Vancouver General Hospital Hyperbaric Unit
85 West 12th Avenue
Vancouver, BC V5Z 1M9, Canada
Phone: *+1-604-875-4033*
Fax: *+1-604-875-5294*
E-mail: *<Angeli.Mitra@vch.ca>*