Cerebral arterial gas embolism proven by computed tomography following transthoracic echocardiography using bubble contrast

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Abstract

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A 75 year-old male developed features of an acute stroke following bubble contrast echocardiography, which was shown on emergent computed tomography scanning to be a result of cerebral arterial gas embolism (CAGE) to the left middle cerebral artery. Ischaemic stroke symptoms have previously been reported as a rare complication of bubble contrast echocardiography. Radiologically proven CAGE from bubble contrast echocardiography had not been reported at the time this case occurred. Immediate provision of 100% oxygen and administration of hyperbaric oxygen are recommended treatments for CAGE and were associated with a substantial recovery for this patient.

Introduction

Bubble contrast echocardiography (BCE) is a common investigation performed to determine the presence of a persistent (patent) foramen ovale (PFO) in patients with cryptogenic stroke, decompression sickness (cutaneous, neurological and inner ear) and platypnoea-orthodeoxia syndrome.[1] Complications are rare, and radiologically proven cerebral arterial gas embolism (CAGE) from BCE had not been reported at the time this case occurred.

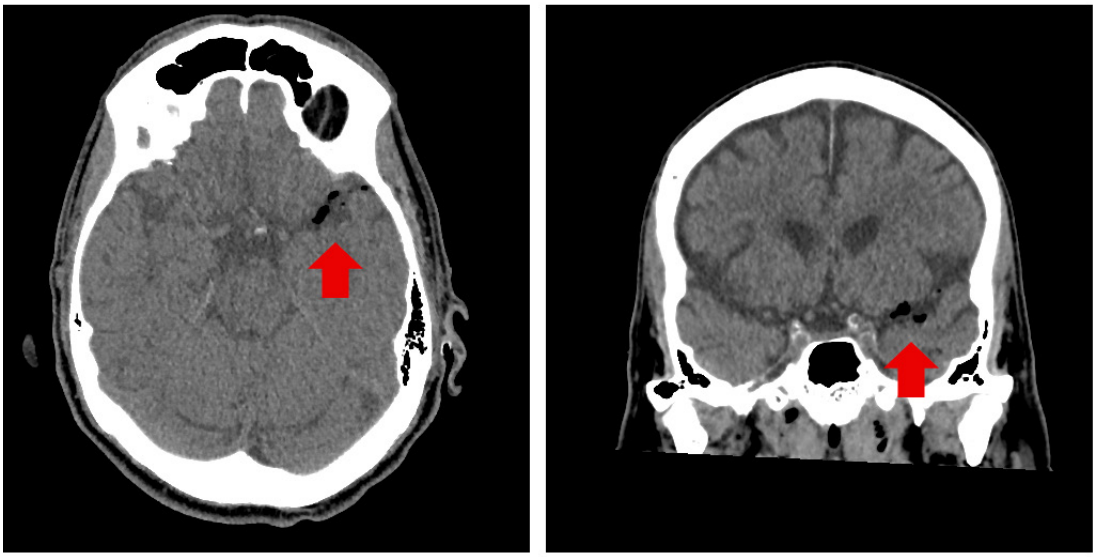
Case report

In January 2016, a 75-year-old male developed sudden onset right hemiparesis, dysarthria and dysphasia 50 minutes after a transthoracic echocardiogram (TTE) study using agitated saline contrast. The procedure had been requested by his general practitioner to investigate a small symptomatic right frontal cortical infarct. The procedure was performed as per the usual protocol in a private cardiology testing practice which had performed this procedure many thousands of times without incident over more than 30 years. Intravenous access in this case was via a vein on the dorsum of the hand rather than the antecubital fossa. The BCE confirmed a persistent (patent) foramen ovale (PFO), with trivial bubbles at rest (1–2 per frame) and minor shunting (~20 bubbles) post Valsalva release. Only four injections were administered via a 3-way tap with a total of 2 ml of air agitated into saline.

Approximately 50 minutes post procedure, the patient stood up while being connected to Holter monitoring and immediately became aphasic with right-sided weakness. An urgent computed tomography (CT) scan of the brain was performed at Sir Charles Gairdner Hospital, a nearby tertiary stroke referral centre, which demonstrated intravascular gas along the length of his left middle cerebral artery (MCA) as well as its cortical branches (Figure 1). His initial NIH Stroke scale was 18 and he had a modified Rankin score of 4. He was transferred to Fiona Stanley Hospital (FSH) for emergency hyperbaric oxygen treatment (HBOT) according to a US Navy Table 6 which was commenced within 4.5 hours of symptom onset. A repeat CT brain and CT angiogram an hour post HBOT showed resolution of air emboli with interval loss of the grey white differentiation in the left MCA territory in keeping with developing infarction. He continued to have some residual speech impairment and motor weakness.

The patient sustained moderate aural barotrauma during his first session of HBOT and hence bilateral myingotomies and grommet insertions were performed. Following this he received a further eight sessions of HBOT until there was a plateau of symptom recovery.

Figure 1
Non-contrast CT head (axial and coronal views) demonstrating extensive gas in the left MCA vessels consistent with air embolism



Following an inpatient stay in the acute stroke unit the patient was discharged to rehabilitation and recovered aside from mild language deficits.

Discussion

CAGE is an uncommon and probably under-recognised complication of invasive medical procedures. Although ischaemic neurological events have been reported post BCE, none of these cases demonstrated intravascular air on imaging.[2,3] It is well recognised that not all cases of CAGE show gas on brain imaging.[4,5]

Although microbubbles entering the arterial circulation almost inevitably pass to the cerebral circulation during BCE, they rarely cause symptoms because of their small size. This contrasts with the likely growth of similar sized arterial bubbles entering tissues supersaturated with inert gas in recently surfaced divers.[6] In this case, it is possible that usually harmless microbubbles generated in preparation of bubble contrast may have coalesced into a large bubble in forearm veins post-procedure and migrated to the central circulation, across the PFO and then to the brain when the patient stood up; possibly augmented by upper limb muscle contraction when used to assist this change in position. Presumably, aggregation of microbubbles is less likely to occur if the injection is into a proximal (antecubital) vein, which is not always accessible. This aggregation and sequestration of microbubbles, with fewer therefore available to proceed centrally and be seen during the BCE study itself, suggests that the PFO was actually larger than the contrast echocardiogram demonstrated.

Improved clinical outcomes after CAGE may correlate with reduced time to HBOT.[7–9] This patient had some delay to HBOT as ambulance transfers were required; initially to Sir Charles Gairdner Hospital, and then (when CAGE was diagnosed as the cause of his acute stroke) to Fiona Stanley Hospital, the only centre providing HBOT in Western Australia.

This case was initially presented at the World Stroke Congress in 2016 and published as an abstract in the related Proceedings.[10] Since that time a further case has been reported; that of an 89 year old woman who had a large right-sided CT-proven CAGE with symptom onset 20 minutes post procedure. This patient succumbed to her illness the day after without active treatment. HBOT was not administered.[11] The role of investigation for PFO in cryptogenic stroke in patients over 60 years of age is still to be defined.[12]

Current CAGE treatment guidelines recommend immediate cessation of further entry of gas, 100% oxygen and appropriate resuscitation of the patient as CAGE can lead to haemodynamic instability and cardiac arrest.[4] Early HBOT should be provided in a centre equipped to manage potentially unstable patients.

Conclusion

Iatrogenic CAGE causing stroke is a very rare complication of BCE. It should be considered where there is onset of neurological symptoms following this procedure, or any other where the possibility of introduction of intravascular gas exists. Emergent HBOT is indicated.

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