Iatrogenic cerebral arterial and venous gas embolism leading to massive hemorrhage, herniation, and death

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Practical Implications

Consider cerebral gas emboli in periprocedural stroke.

Cerebral gas embolism (CGE) has been reported since 1946. Besides deep-sea diving, CGE is largely iatrogenic. The diagnosis is based on strokelike symptoms and brain imaging. Hyperbaric oxygen therapy (HBOT) is the mainstay of treatment.

There are a number of case reports of either arterial or venous cerebral gas emboli, which were at times associated with patent foramen ovale (PFO), allowing for paradoxical emboli.

This article reports a patient manifesting with both arterial and venous cerebral gas emboli (but without intracardiac shunt) ultimately leading to death, explaining the mechanism as well as reviewing current literature.

Case report

A 91-year-old man with Alzheimer disease and Parkinson disease presented with fever. He had hypertension, congestive heart failure, and asthma. He was taking carbidopa/levodopa 25/100 mg once daily and aspirin 81 mg daily. Initially he was awake, alert, and talking. Blood pressure was 140/73 mm Hg, heart rate was 92 beats/min, respiratory rate was 18 breaths/min, and temperature was 99.5°F. Laboratory data revealed a white blood cell (WBC) count of 47,300/ μ L, platelets of 43,000/ μ L, sodium of 144 mEq/L, and creatinine of 1.1 mg/dL. Urinalysis reported numerous WBCs and blood cultures revealed Gram-negative rods. He was started on 4 IV antibiotics. A right internal jugular (IJ) central venous catheter (CVC) was placed with difficulty, leading to a subcutaneous hematoma. Chest x-ray showed the CVC tip to be in the superior vena cava (figure, A). Immediately afterward he became obtunded, moaning occasionally in response to noxious stimuli. There was right arm extensor posturing with rigidity and resting tremor.

Head CT 1 hour post CVC placement revealed more than 70 cerebral gas emboli, all within the right anterior, middle, and posterior cerebral arteries and the superior sagittal sinus (figure, B). None were detected in the right IJ vein or the left hemisphere. Forty-eight hours post admission his right pupil was fixed and dilated. Vital signs remained stable. CT showed resolution of the gas but a large right hemispheric infarct, extensive edema, and a 4.9 $\times$ 4.0 cm right frontal hematoma with a 2 cm shift, causing herniation (figure, C).

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Funding information and disclosures are provided at the end of the article. Full disclosure form information provided by the author is available with the **full text of this article at Neurology.org/cp**.

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Figure Imaging

(A) Chest x-ray demonstrating the central venous catheter (CVC) tip situated in the superior vena cava. (B) CT 1 hour post CVC placement revealing more than 70 cerebral gas emboli, all within the right anterior, middle, and posterior cerebral arteries and several in the superior sagittal sinus. Note that none were detected in the left hemisphere. (C) CT 48 hours post CVC placement revealing a 4.9×4.0 cm right hemispheric stroke with extensive edema and a frontal intraparenchymal hematoma with a 2 cm shift.

He was deemed do not resuscitate/do not intubate. The family declined aggressive management, so HBOT was not performed. He died 5 days post admission.

DISCUSSION

Cerebral air embolism was first described in 1946.¹ Gas embolism complicates 2.65 per 100,000 hospitalizations, with a mortality rate of 21%.² CGE has been shown to occur in the setting of lung transplant,³ transbronchial lung biopsy,⁴ central venous catheterization,⁵ coronary artery bypass,⁶ and flexible bronchoscopy,^{4,6} among others. The diagnosis is based on classic strokelike symptoms after a precipitating event and brain imaging. HBOT^{7,e1} was first described as treatment for air embolism^{e2} and specifically for CGE.^{e3} Mechanical compression of air bubbles to a smaller size with an increase in the surface-to-volume ratio leads to delivering high-dose oxygen to ischemic brain tissue.^{e4,e5} Air bubbles may exist for up to 40 hours, and HBOT should be considered at any point within this time frame.^{e6} Bubble size does not seem to affect the severity or frequency of the stroke.^{e7}

This case report was unique as there were both venous and arterial cerebral gas emboli. Previous reports describe either venous or arterial cerebral gas emboli but not both (table e-1 at Neurology.org/cp).^{5,6,e8-e15} There was no PFO or other cardiac shunt. Animal studies have shown that cerebral arterial gas can pass through the capillaries and into the venous system in large quantities.^{e16}

It is believed that air was injected into the right internal carotid artery during the attempted IJ line insertion, passing through capillaries and ultimately reaching the superior sagittal sinus. The air could not have originated from the IJ vein because (1) there was no air in the IJ vein on the CT images (not shown), and (2) the emboli were in only the right hemisphere; had the air been injected via the IJ vein then it would travel to the heart and the pulmonary arteries and veins prior to reaching both internal carotid arteries, leading to bilateral (and not unilateral) emboli. The theory of the right carotid artery having been the source is also consistent with the right neck hematoma. The infarct was due to the numerous emboli compromising cerebral arterial flow within the right hemisphere. The hemorrhage was most likely due to a combination of age, thrombocytopenia, and hemorrhagic transformation.

Supplemental Data

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In summary, there should be a high index of suspicion for central line–related CGE in patients with subsequent strokelike symptoms. An early referral to a hyperbaric treatment facility should be considered, even if air evacuation is required.

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ACKNOWLEDGMENT

The author thanks Dr. Pieter Bothma and Dr. James Wallman for providing their expertise and knowledge on this case.

AUTHOR CONTRIBUTIONS

Drafting/revising the manuscript; Study concept or design; Analysis or interpretation of data; Acquisition of data; Statistical analysis.

STUDY FUNDING

No targeted funding reported.

DISCLOSURES

W.S. Baek serves on the editorial boards of *Journal of Neurology and Neuroscience* and *JSM Alzheimer's Disease and Related Dementia*. Full disclosure form information provided by the author is available with the **full text of this article at Neurology.org/cp**.