

Figure 1. Acute CT imaging demonstrates subtle edema on initial CT(a), with evolution imaging on CT 4 hours later (b) where extensive edema, effacement of the sulcal and gyral pattern, loss of gray-white matter differentiation and mild mass effect is seen. At 1-month post event, repeat CT brain is normal (c).

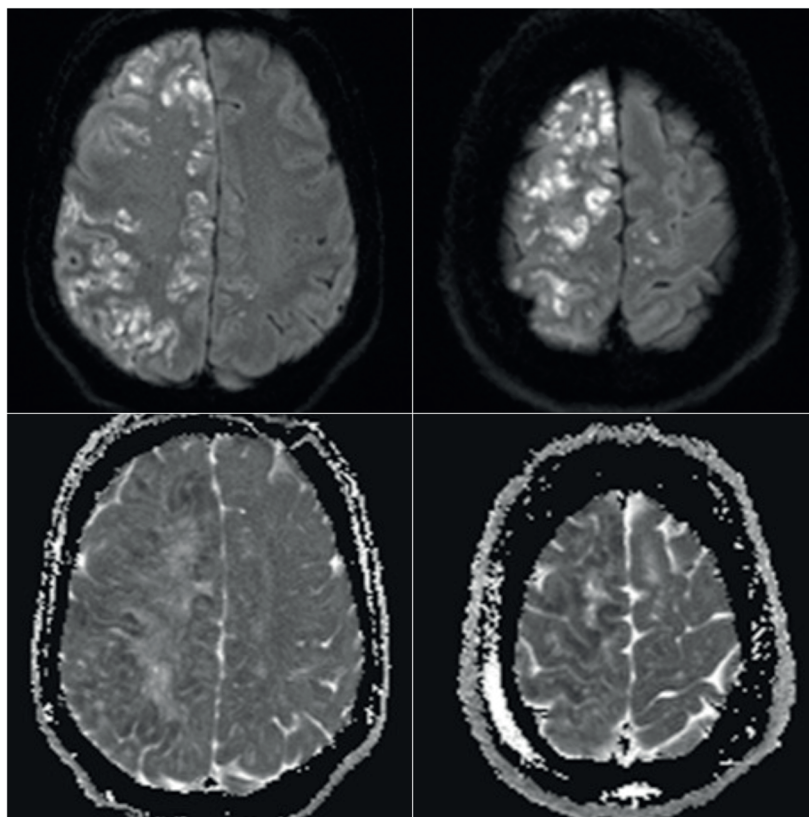


Figure 2. MR b1000 (a,c) and corresponding ADC map (b,d) demonstrate innumerable scattered small infarcts along the cortical gyri of the frontal and parietal lobes on the right, involving the anterior and middle cerebral artery territories. Occasional scattered punctate infarcts within the left anterior cerebral artery territory.

The patient received several days of conservative management, which incorporated high fraction of inspired oxygen (FiO₂) therapy. She improved enough to be discharged from the intensive care unit (ICU). She proceeded to neurological rehabilitation with improvement of right hemiparesis and gradual but progressive recovery of left hemiparesis. Repeat CT brain at 4 weeks did not show any infarct progression (Figure 1c).

Discussion

CGE is an uncommon, but potentially fatal, event that occurs as a consequence of air entry into the

vasculature. Surgery, trauma, vascular interventions, and barotrauma from mechanical ventilation are common causes.

The diagnosis of CO₂ CGE in this case study was established based on a number of factors. First, there are published cases of CO₂ emboli resulting from thoracoscopic and laparoscopic surgery where gas insufflation is utilized and there is a vessel injury. This allows a portal of entry into the circulation [5]. In this case, it is most likely that the pulmonary vein injury provided an entry point for CO₂ into the arterial circulation with subsequent embolization to the intracranial vasculature.

Second, the patient was in the left lateral position with head up 45 degrees during the operation, which was conducive to gas emboli favoring mainly the superior regions of the right cerebral hemisphere. Both hemispheres were involved, which is suggestive of a central source.

Third, gas in the brain can be reabsorbed rapidly, particularly CO₂ which dissolves readily in the blood [6]. This is consistent with literature that recognizes the poor sensitivity and specificity of acute CT findings in identifying CGE [7]. The acute phase edema may be associated with CO₂ induced vasodilation. It is likely that the patient's relatively rapid recovery was due to rapid reperfusion of the at-risk brain tissue, and relatively small volume of infarcted tissue, due to rapid CO₂ dissolution. We recognize that another potential source of CGE exists in this patient in the form of paradoxical embolism from central venous access and the presence of a PFO; this was clinically considerably less likely given the uncomplicated central venous catheter insertion, small size PFO, and stable intra-operative course.

Initial supportive measures for CGE are focused on ensuring airway protection, adequate ventilation, oxygenation, and hemodynamic stability. High FiO₂ therapy should be administered. The supplemental oxygen increases the partial pressure of oxygen and decreases the partial pressure of nitrogen in the blood. This causes diffusion of nitrogen from inside the air bubble (which has a high nitrogen content) into the blood (which has a low nitrogen concentration), which reduces bubble size and accelerates bubble resorption [8]. In severe cases of CGE with associated hemodynamic instability, HBOT is the mainstay of treatment. The mechanism is similar to high FiO₂ therapy but on a much greater magnitude. In this case, the absence of residual intracranial gas in a hemodynamically stable patient was felt to outweigh any potential benefit of a limited resource unavailable at our institution. There is also a recognized risk of death during transfer to a suitable institution, which needs to be considered in all cases.

Risk factors for death or poor prognosis include cardiac arrest, increasing age, focal neurological deficits at ICU admissions, acute kidney failure, prolonged mechanical ventilation for more than 5 days, and gyriform air on brain imaging [9,10].

Conclusion

In patients who have acute neurological manifestations following a medical procedure, an acute cerebrovascular event must be considered. Rapid access to neuro-diagnostic imaging is paramount in order to initiate correct supportive and/or definitive therapy. As acute CT findings can be non-specific, MRI with diffusion weighted sequences is essential to confirm the diagnosis of acute embolic infarction and help elucidate the pathophysiological mechanism involved [5]. Initial supportive therapy for CGE should be commenced while investigations for

more common thromboembolic sources are performed. Transfer to a center that provides HBOT should be considered in severe cases.

What is new?

CGE are a rare cause of stroke. There are some case reports of this condition in the literature; however, very few of that have occurred from gas insufflation during thoracoscopic surgery. We will be adding to the body of literature on this rare condition.

Acknowledgment

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List of Abbreviations

CGE	Cerebral gas emboli
CO ₂	Carbon dioxide
CT	Computed tomography
HBOT	Hyperbaric oxygen therapy
MRI	Magnetic resonance imaging

Consent for publication

Written informed consent was obtained from the patient to publish this case.

Ethical approval

Epworth Healthcare's Research Development & Governance Unit granted ethics approval to publish this case report on 6th July 2020 (Reference number: EH2020-603).

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Summary of the case

1	Patient (gender, age)	Female, 70-year old
2	Final diagnosis	Cerebral gas emboli
3	Symptoms	Quadriplegia
4	Medications	Nil
5	Clinical procedure	Thoracoscopic lobectomy for lung cancer
6	Specialty	Cardiothoracic surgery; neurology; radiology