

Acute stroke from air embolism to the middle cerebral artery following upper gastrointestinal endoscopy

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Acute stroke from cerebral-artery air embolism is a very rare occurrence during esophagogastroduodenoscopy (EGD). Computed tomography is a quick and reliable method of detecting cerebral air embolism if performed within an appropriate timeframe. We found 20 reported cases of air embolism with EGD in the literature; only seven of those resulted in cerebral air embolus. We report an eighth such case in which the patient suffered cerebral air embolus with neurological signs of a stroke while undergoing upper gastrointestinal endoscopy.

Case report

A 77-year-old male with a history of large B-cell lymphoma occurring in the head of the pancreas was hospitalized for neutropenic fever. During this time he complained of painful swallowing. A esophagogastroduodenoscopy (EGD) was performed for his complaints of odynophagia. During the procedure, many erosions and aphthous ulcers were noted in the esophageal mucosa. Fresh blood in the antrum of the stomach as well as in the duodenal bulb was also reported. Although EGD determined that there were no signs of perforation, tumor necrosis and exposed blood vessels leading to air entrapment could not be excluded. During intubation of the duodenal bulb, the patient's blood pressure dropped sufficiently to warrant termination of the procedure. He was immediately given an IV fluid bolus and transferred to the ICU for further observation. On arrival

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Figure 1. Axial noncontrast CT scan of the brain demonstrating a right MCA infarct along with multiple foci of air within the right MCA branches (best seen within the Sylvian fissure).

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at the ICU, the patient was observed to have left-sided weakness and hemineglect as well as slurred speech and an absence of accommodation and corneal reflexes. He also displayed a left-sided upgoing plantar response.

A multichannel axial CT of the head without contrast revealed a large hypodense area involving the cortical and subcortical temporoparietal regions of the right hemisphere. Multiple foci of air were noted within this region, which appeared to be within the right middle cerebral artery branches, and in some veins draining the region of the parenchyma fed by the right middle cerebral artery (MCA)



Figure 2. Linear collections of air are seen in the right MCA territory, likely within the MCA branches and possibly in the draining veins as well.

(Figs. 1, 2). There was no evidence of hemorrhage or mass as possible causes of the patient's mental status changes. It was thus concluded that air embolism led to an acute infarct in the right MCA territory, and the patient was immediately scheduled for transfer to another hospital for hyperbaric treatment. While in the ICU awaiting transfer, the patient's mental status improved somewhat and he was eventually transported to the hyperbaric treatment facility approximately 22 hours after cerebral insult, with a reported Glasgow Coma Score of approximately 14/15.

Discussion

Stroke can be caused by air introduced into the vascular system that becomes lodged within the vessels and impedes blood flow to brain parenchyma. Some common means of

air introduction to the vascular system include certain invasive procedures such as those in neurosurgery done with the patient in the sitting position, laparoscopic procedures, total hip arthroplasties, central line placements, and cesarean sections. Compared to the listed procedures above, gastric endoscopy carries a medium risk of introducing vascular air by allowing passage into an abnormally open negative pressure vein or forcing air via insufflation into a high pressure artery (1, 2).

Once air is introduced into the venous system, it travels the path of least resistance and is filtered out through the pulmonary vasculature, which acts as an internal sieve to protect the brain from suffering an embolic stroke (1-4). However, when there is a breakdown in the system through a patent foramen ovale, portosystemic shunt, A-V shunt, or alveolar bypass or deficit in the lungs that overrides the capillary filtration system, the air then has access to the cerebral arterial vascular system (1, 3-5). A common vessel of entry into the cerebral vasculature is the middle cerebral artery, whose primary branches feed into the lenticulostriate arteries and supply the basal ganglia and further branches, thus supplying the frontal and temporal cortices via the superior and inferior divisions, respectively [6].

Computed tomography (CT) is a fast and often first-line means of detecting air embolisms that have not yet been reabsorbed by the body. It is especially useful in detecting cerebral air emboli that have managed to escape the many protective barriers set up by the body. The detection of air in the brain, however, is limited by the amount of air introduced as well as the time from initial insult to CT scan. Air in the brain is absorbed in a time- and pressure-dependent fashion. In other words, the higher the partial pressure of oxygen in the circulation, as in a patient who is receiving 100% oxygen, the faster the clearance or reabsorption of air. In fact, Dexter and Hindman show that cerebral air emboli large enough to be detected on CT can take anywhere from 20 seconds to 40 hours to reabsorb, depending on oxygen supplementation and volume of air in the cerebral vasculature (7).

This is helpful not only when interpreting CT findings but also when determining treatment options. The most widely agreed-upon treatment for clinically significant cerebral air, whether detected with diagnostic imaging or suspected clinically with equivocal image findings, is emergent hyperbaric therapy within the first 30 hours after initial insult. Indeed, hyperbaric therapy shows best results if instituted within five hours (8). Starting the patient on 100% oxygen while waiting for transport to a facility is also a good initial therapy but should not delay patient transfer to a hyperbaric unit when possible (7, 9). However, if hyperbaric therapy is not an option (due to availability or patient condition precluding transfer), various treatment options have been reported in the literature. Alternative treatments such as administration of 100% oxygen, increasing regional cerebral blood flow, hemodilution with perfluorocarbons (all to increase clearance of nitrogen), as well as the administration of heparin to prevent further clotting complications have all shown beneficial or protective results

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in human or animal studies (1, 5, 7). However, it is widely agreed that these measures are inferior to the benefits of hyperbaric therapy, which should be considered in refractory cases (1, 3-5, 7-11).

Most of the documented cases of cerebral air embolism with EGD were confirmed with noncontrast head CT within five hours of initial symptom onset and demonstrate variable areas of parenchymal infarct as well as air in the vasculature involving MCA territory. The exception to this was a case in which Cristl et. al. reported detection of air on a repeat CT more than 42 hours after the incident (11). As with the majority of cases, our patient was sent to the radiology department within a few hours of the initial incident and, with the aid of noncontrast CT, was found to have parenchymal hypodensity and concomitant air distributed over a large portion of the right MCA territory, which correlated with reported left-sided symptoms of neurological deficit. The quick detection by radiology staff and the immediate actions of the ICU staff while awaiting transfer to an offsite hyperbaric unit led to a rapid improvement in the patient's neurological function.

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