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Selected Topics: Neurological Emergencies

IATROGENIC ARTERIAL GAS EMBOLISM FROM ESOPHAGOGASTRODUODENOSCOPY

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☐ **Abstract—Background: Arterial gas embolus (AGE) is a rare complication of esophagoduodenoscopy (EGD) that has been described in only a few case reports in the literature. The exact etiology remains unknown, but many of the cases share some common characteristics. Case Report: We report the case of a 52-year-old otherwise healthy man who underwent outpatient EGD for a sensation of retained food in his esophagus. During the procedure, he suffered a tonic–clonic seizure, bradycardia, and hypoxia. Subsequent emergency department workup showed pneumocephalus on computed tomography brain imaging, and he was diagnosed with a cerebral AGE (CAGE). He was transferred to our facility for treatment of CAGE with hyperbaric oxygen therapy (HBOT). After multiple hyperbaric treatments, he was discharged with a residual left hemiparesis, which represented a significant improvement in his overall neurologic status. We also present a review of similar EGD CAGE cases from the literature and discuss their outcomes and the need for HBOT. Why Should an Emergency Physician Be Aware of This?: Although CAGE from EGD is rare, these patients will often be transferred to the ED from gastrointestinal procedural suites and an emergency physician should understand that an iatrogenic CAGE can result from this procedure and that CAGE is a clinical diagnosis. Definitive care at a critical care–capable hyperbaric chamber will provide the patient with the best chance of meaningful recovery, and transport should be arranged as expeditiously as possible.** © **2019 Elsevier Inc. All rights reserved.**

☐ **Keywords—arterial gas embolism; esophagogastroduodenoscopy; hyperbaric oxygen therapy**

INTRODUCTION

Esophagogastroduodenoscopy (EGD) is a common procedure done routinely in outpatient clinics and on an emergent basis in the emergency department (ED) or hospital setting. During the procedure, patients are typically sedated, placed in the left lateral decubitus position, and then have an endoscope introduced in the esophagus, often using compressed air to dilate the esophagus for better visualization. The most common serious complications include cardiopulmonary adverse events, such as hypoxia and hypotension; tissue bleeding; perforation; and infection (1). Less commonly, an arterial gas embolism (AGE) can occur, often with devastating consequences. Here we present the case of a patient who underwent outpatient EGD and suffered a cerebral AGE (CAGE). He underwent resuscitation and was transferred to our facility for hyperbaric oxygen therapy (HBOT). We also discuss the ED management of the patient, as well as review the available case reports of CAGE during EGD to demonstrate the need for HBOT in these cases.

CASE REPORT

The patient was a 52-year-old otherwise healthy man who underwent routine EGD for progressive dysphagia with

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occasional vomiting after meals. The procedure was performed at an outpatient clinic and started at approximately 11:00 AM. During the procedure, the patient had a seizure and remained obtunded with a Glasgow Coma Scale score of 4–5 after medical staff noted cessation of seizure activity. His vital signs demonstrated oxygen saturation levels between 80% and 89% and bradycardia as low as 40 beats/min.

The clinic activated emergency medical services who transported the patient to a nearby ED. The patient arrived at that facility at approximately 1:00 PM and was obtunded with decerebrate posturing as well as rigidity and clonus in his lower extremities. His vital signs on presentation were heart rate of 92 beats/min, blood pressure of 151/94 mm Hg, and oxygen saturation of 95% on supplemental oxygen. Given the earlier seizure activity, providers administered lorazepam 2 mg and fosphenytoin 1000 mg i.v. The physician then intubated the patient and started fentanyl and propofol infusions for sedation. With the airway secured, the providers obtained a noncontrast head computed tomography (CT) scan, which showed multiple, bilateral areas of pneumocephalus consistent with extensive air bubble embolization (often referred to as a "shower of bubbles" in the hyperbaric community), which can be seen with CAGE (Figure 1). After the head CT, the patient remained hemodynamically stable and the emergency physician suspected an iatrogenic CAGE.

Recognizing the need for definitive care, the emergency physician initiated helicopter transfer to our facility for emergent HBOT. Our facility is the regional hyperbaric referral center and has a 24/7 on-call, critical care–capable chamber, equipped to deal with an intubated and sedated patient.

The patient arrived at our facility 6 h after the start of the EGD. His neurologic examination on arrival showed the patient intubated, sedated on 40 μg propofol, with decerebrate posturing to pain, and making occasional spontaneous non-purposeful movements of his bilateral hands and feet. His pupils were pinpoint, round, and reactive to light (he had been given opiates). His reflexes were as follows: biceps/triceps/brachioradialis 3 + bilaterally,

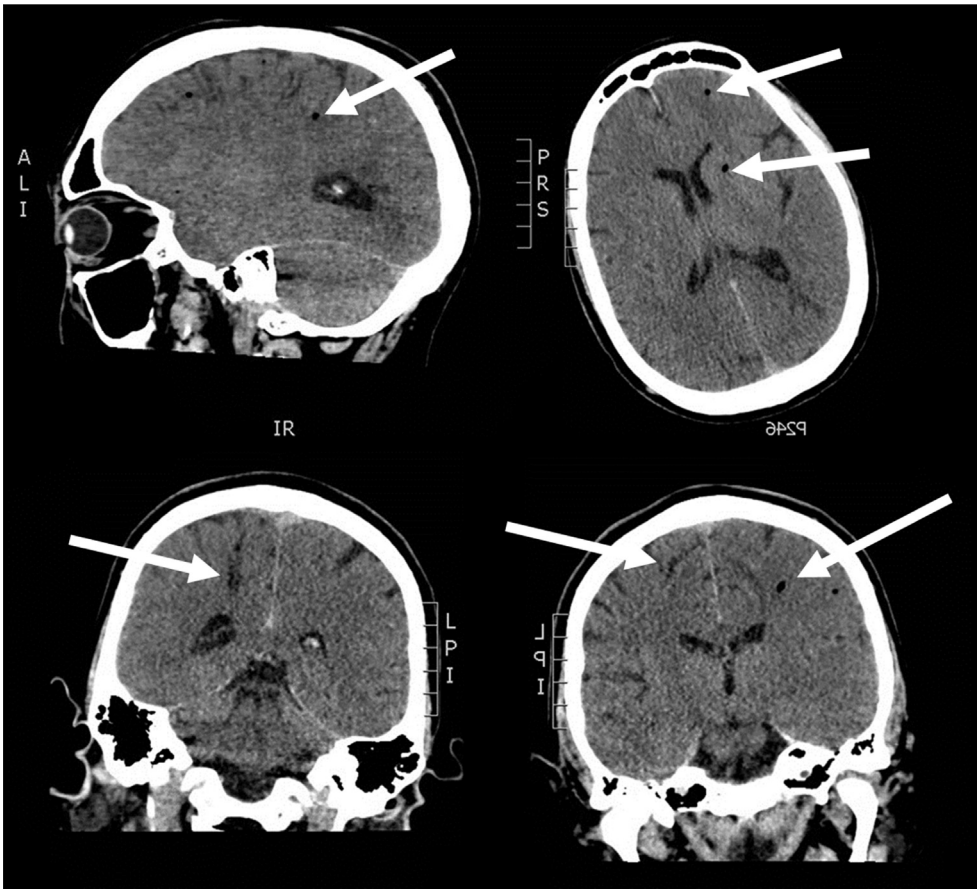

Figure 1. Non-contrast head computed tomography (CT) scan at the initial emergency department visit showing multiple foci of pneumocephalus (white arrows). Although present in both hemispheres, the gas had a greater effect on the right hemisphere (based on CT and subsequent magnetic resonance imaging).

patellar 2 + bilaterally, and Achilles 2 + bilaterally. He exhibited bilateral ankle clonus of 4–5 beats.

Our team made the decision to treat emergently with HBOT, starting with a U.S. Navy Treatment Table 6 (USN TT6) with extensions (2). Nine hours and 45 min later, he completed the USN TT6 with full extensions, tolerating the treatment well. His post-treatment neurologic examination, while still intubated and lightly sedated with propofol, showed localization of pain to his right arm, withdrawal from pain with his left arm and left leg, and only slight movement of the right leg to deep stimuli. Babinski reflexes were upgoing bilaterally. His pupils were 3 mm and reactive bilaterally.

Approximately 16 h after completing the first HBOT, he received another HBOT, this time a U.S. Navy Treatment Table 9 (USN TT9) (2). In total, the patient underwent four HBOT treatments over 4 days, one USN TT6 on the first day and three subsequent USN TT9 each day. He exhibited stepwise improvement of neurologic function after each of the first three treatments. After the third HBOT, his examination while lightly sedated and still intubated by the neurological intensivist showed him able to open his eyes to voice commands, have 2-mm and reactive bilateral pupils, to move his right upper and lower extremities to voice commands, and have normal sensation on his right side. He "reacted" with his left upper extremity to noxious stimulation but did not move his left lower extremity according to the neurological intensivist. An electroencephalogram showed "moderate diffuse nonspecific abnormalities" and no epileptiform discharges. His neurologic status demonstrated no interval improvement between the third and fourth treatments and remained unchanged after the fourth HBOT. Therefore, we felt that the patient would not benefit from further HBOT.

After the fourth HBOT, the neurological intensivists weaned sedation, trialed him on spontaneous ventilation, felt he could protect his airway, and successfully extubated him according to protocol. Following extubation, his neurologic examination revealed that he was awake, alert, and fully oriented. He followed commands but with left-sided hemineglect. His cranial nerve examination showed rightward deviated gaze with an inability to cross the midline as well as 2–3 beats of horizontal nystagmus with right to left eye movements. His motor examination displayed 5/5 strength in the right upper and lower extremities. He withdrew his left lower extremity to pain and showed a flicker of movement with voluntary toe wiggle. Unfortunately, his left upper extremity was completely without movement. He was able to feel only a hard pinch on the left upper extremity, but his right upper extremity had normal sensation.

Magnetic resonance imaging that day showed "diffuse right hemispheric ischemic injury most notably affecting the posterior frontal and parietal lobes. There is also evidence of ischemic injury in the left posterior frontal and parietal lobes although significantly less compared to the contralateral side." An echocardiogram, performed as part of the evaluation to determine why the patient had suffered a CAGE, showed no patent foramen ovale. With the completion of his HBOT and extubation, his insurer initiated a transfer back to his original hospital system, where he continued to undergo rehabilitation. At the time of his discharge, his neurological examination remained unchanged compared to his examination after extubation.

DISCUSSION

AGEs occur most commonly among SCUBA divers when taking a breath of compressed gas and ascending rapidly with a closed glottis leading to pulmonary overinflation and subsequent barotrauma. AGE may also occur iatrogenically among patients undergoing procedures where gas inadvertently enters the arterial circulation. This patient's EGD results did not reveal an obvious cause for his air embolism, but the gastroenterologists hypothesized the presence of an arteriovenous malformation (AVM) that could have allowed the insufflated gas to arterialize and then travel to the central nervous system. Note, however, that no evidence of an AVM was found and the mechanism that produced the CAGE remains unclear. CAGEs can result from gas bubbles occurring directly in the arterial circulation but also from venous bubbles entering the arterial circulation via intracardiac right to left shunting or intrapulmonary shunting. Once formed, the bubbles are directed by vascular flow characteristics and can affect any tissue or organ system; but in clinical practice, the organs most sensitive to tissue perfusion may be more seriously compromised, such as the brain and heart. Identifying the mechanism for a gas embolism is less important than recognizing that a CAGE has occurred and initiating definitive treatment in a timely manner.

CAGE is a clinical diagnosis and no test establishes the presence of a clinically significant AGE. Imaging studies are supportive when gas is seen, such as in the case presented here, however, the absence of gas does not exclude the diagnosis. The hallmark of CAGE is the rapid onset of new neurologic symptoms in the context of SCUBA diving or a procedure with the potential for gas to enter the circulation. Typically, patients who suffer a CAGE will develop sudden mental status changes, sometimes with an accompanying seizure, and then do not return to their neurologic baseline. They may have a new focal neurologic deficit or remain obtunded. The patient's presentation with persistent unresponsiveness, however, is typical of CAGE after a procedure requiring sedation and where gas may enter the circulation.

Once CAGE is suspected, emergency stabilization includes airway management if necessary, maintenance of blood pressure, and placing the patient on 100% oxygen, which creates a gradient for gas exchange within the bubbles. To the extent that the gas in the bubbles is replaced by oxygen, the bubbles may then dissipate more easily. Oxygen alone at normobaric pressures is not the definitive treatment, however, as only HBOT has been shown to mitigate the inflammatory cascade resulting from the effect of bubbles in the circulation (3). Patients may be placed in the supine position, however, reverse Trendelenberg or head-down position is not recommended, as it can increase intracranial pressure and worsen cerebral edema.

We reviewed the literature and found 13 adult case reports of CAGEs from routine EGD (Table 1) (4–16). To our knowledge, no one has published a review of EGD CAGE cases to demonstrate the benefit of HBOT. HBOT for CAGE is the gold standard for U.S. military personnel and is an American Heart Association class I recommendation with Level C evidence (2,3). We excluded pediatric cases, procedures involving Kasai limbs, and cases involving endoscopic retrograde cholangiopancreatography from our review, as these were not directly comparable with our patient. Some patients in the literature we reviewed underwent HBOT while others did not. We found that the overwhelming majority of patients who received HBOT, even if delayed, survived; whereas most patients who did not receive HBOT, died (see Tables 1 and 2). It is generally accepted that the sooner a patient receives HBOT, the better the potential outcome. However, because these events are rare, there is no high-level evidence to establish the maximum time to initiate HBOT beyond which there is no benefit. A case has been reported in which the patient benefitted from HBOT with a delay of 39 h (16). In reviewing these case reports, we were unable to identify a unifying gastrointestinal anatomic abnormality that would predict gas embolism from this common procedure. Interestingly, when bubbles were detected on CT, they tended to exhibit a preference for the right cerebral hemisphere. In our patient for example, the "shower of bubbles" was sufficient to affect both cerebral hemispheres, but more gas was seen in the right hemisphere on the CT and MRI, which correlated with the patient's clinical findings. Possibly, the right cerebral vasculature represents the path of least resistance for the bubbles to travel due to the convention of laying the patient in the left lateral decubitus position to perform the EGD.

Although a rare complication of EGD, a CAGE is a significant event that may result in permanent neurologic deficits and even death. HBOT has demonstrated a significant clinical benefit in CAGE cases regardless of etiology (17). Arterial gas bubbles may travel to the brain

Table 1. Published Cases of Iatrogenic Cerebral Arterial Gas Embolus From Esophagoduodenoscopy Treated Without Hyperbaric Oxygen Therapy

Case Report, First Author, Year	History	Procedure	Event	Imaging	PFO	Treatment	Outcome
Christl, 1994 (4)	66 yo, female, GI bleeding	EGD: duodenal-caval fistula	ALOC, spastic paresis in bilateral lower extremities	CT: normal. CT at 42 h: + air and massive cerebral edema	No PFO	No HBOT	Death
Katzgraber, 1995 (5)	56 yo male, esophageal stricture	EGD with dilation	Cardiac arrest	Echo: + air bubbles. Autopsy: massive air embolism in right atrium and right ventricle.	No PFO	No HBOT	Death
Bou-Samra, 1997 (6)	66 yo male	EGD: gastric erosions	ALOC	CT: cerebral air	Unknown	No HBOT	Death
Akhtar, 2001 (7)	80 yo female, esophageal cancer with stricture	EGD with biopsies	ALOC, left hemiparesis	CT: right hemispheric air	No PFO	No HBOT	Persistent vegetative state
Demaerel, 2003 (8)	80 yo female, GI bleeding	EGD: normal	ALOC, left hemiparesis	CT: right frontal lobe air	No PFO	No HBOT	Residual left hemiparesis
Mellado, 2005 (9)	52 yo female, 2 weeks of vomiting	EGD with biopsies: esophagitis	ALOC	CT: right hemispheric air	Unknown	No HBOT	Death
McAree, 2008 (10)	69 yo male, erosive esophagitis	EGD with biopsies	ALOC, seizure, left hemiparesis	CT: right hemispheric air	No PFO	No HBOT	Death

Only 1 of the patients who did not receive HBOT had a meaningful recovery.
ALOC = altered level of consciousness; CT = computed tomography; EGD = esophagoduodenoscopy; GI = gastrointestinal; HBOT = hyperbaric oxygen therapy; PFO = patent foramen ovale; s/p = status post; yo = years old.

Table 2. Published Cases of Iatrogenic Cerebral Arterial Gas Embolus From Esophagoduodenoscopy Treated With Hyperbaric Oxygen Therapy

Case Report	History	Procedure	Event	Imaging	PFO	Treatment	Outcome
Raju, 1998 (11)	75 yo female, esophageal stricture	EGD with balloon dilation	ALOC, left hemiparesis, aphasia	CT: air in right MCA distribution	+ PFO	+ HBOT Time to HBOT: 2 h	Normal
Green, 2005 (12)	71 yo male, GERD with esophageal stricture	EGD with balloon dilation	Hypotension, seizure, apnea	CT: right hemispheric air	No PFO	+ HBOT Time to HBOT: 5 h	Normal mental status, residual left upper extremity paresis
ter Laan, 2009 (13)	48 yo male, esophageal cancer s/p subtotal esophagectomy	EGD with stent	ALOC, left hemiparesis	CT: right hemispheric air	No PFO	+ HBOT Time to HBOT: within 24 h	Normal mental status, mild residual left hemiparesis
Niehaus, 2013 (14)	64 yo female, esophageal stricture	EGD with balloon dilation	ALOC, left gaze deviation, decerebrate posturing, myoclonus	CT: right hemispheric air	No PFO	+ HBOT Time to HBOT: "emergent"	Death
Eoh, 2015 (15)	64 yo female, esophageal stenosis	EGD with dilation	ALOC, left facial droop, left gaze deviation, seizure	CT: right hemispheric air in arterial distribution	No PFO	+ HBOT Time to HBOT: unknown	Occasional spontaneous eye opening, left hemiparesis
Covington, 2016 (16)	49 yo male, GERD	EGD with biopsies	ALOC, seizure	CT: right hemispheric air	No PFO	+ HBOT Time to HBOT: 39 h	Normal mental status, mild left upper extremity hemiparesis

All but 2 of the patients treated with HBOT had meaningful improvement in neurologic symptoms.
ALOC = altered level of consciousness; CT = computed tomography; EGD = esophagoduodenoscopy; GERD, gastroesophageal reflux disease; GI = gastrointestinal; HBOT = hyperbaric oxygen therapy; MCA = middle cerebral artery; PFO = patent foramen ovale; s/p = status post; yo = years old.

where they function as emboli leading to tissue ischemia and infarction. At the cellular level, bubbles from an AGE can cause shear stress on vascular endothelial cells and initiate an inflammatory cascade that can lead to cellular death (18–20). HBOT can be beneficial in AGE through hyperoxygenation of ischemic tissue, pressure effects on the bubbles (shrinking the bubbles until they dissipate), and attenuation of the inflammatory cascade (21–23). Early HBOT is likely to have a greater benefit than delayed treatment, although case reports of AGE patients undergoing delayed treatments have demonstrated excellent recovery (16,24–27). The hyperbaric environment serves to diminish bubble size, although very often these bubbles are not detected on brain imaging either because they are exceedingly small or have diffused into the surrounding tissue by the time of imaging. Even without bubbles seen on imaging, a patient who has suffered a CAGE should undergo HBOT for the best possible chance at recovery.

Initiation of an emergent evaluation for stroke including brain imaging is necessary in these cases to rule out other intracranial pathology. Typically, emergent evaluation for stroke will include a non-contrast CT scan of the head; blood tests including finger-stick glucose, cardiac markers, chemistry, creatine phosphokinase, complete blood count, and coagulation studies, as well as electrocardiogram. A consultation with a neurologist or, if possible, a stroke neurologist would be ideal but may not be possible in resource-limited settings. Of note, investigators have described elevated creatinine kinase values in diving AGE patients (28). Whether this elevation is a feature of iatrogenic CAGE remains to be established, but clinicians may find elevated creatinine kinase in the absence of other cardiac marker changes increases their suspicion of CAGE.

Once an emergency physician suspects CAGE and determines that transfer to a facility with a hyperbaric chamber is necessary, finding an accepting facility may prove challenging. Some hyperbaric chambers are available for emergency cases and able to treat critically ill patients, but the majority are not. Therefore, finding a chamber able to treat severely ill CAGE patients who are intubated and receiving infusions may be difficult for the emergency physician (29). If unsure about the nearest critical care–capable chamber, an emergency physician can contact the Diver's Alert Network emergency line to seek further information. Transfer of these patients often requires critical care transport by either ground or air, depending on the distance involved. If transferring the patient requires air travel, a discussion with the hyperbaric physician is recommended to mitigate high altitude exposure if possible.

WHY SHOULD AN EMERGENCY PHYSICIAN BE AWARE OF THIS?

Emergency physicians should be aware of this because they will be responding to these code events in their hospitals or receiving these patients from outpatient facilities. We seek to increase familiarity among emergency physicians of CAGE complicating an EGD and reviewing key points in the recognition, stabilization, and referral of CAGE patients for HBOT.

Ultimately, CAGE is a clinical diagnosis and regardless of the etiology (diving, iatrogenic) requires an understanding of the pathophysiology. In a patient who has undergone a procedure where a gas has the chance to enter the circulatory system and the patient has had a sudden mental status change or new neurologic deficit, iatrogenic CAGE should be a part of the clinician's differential diagnosis. HBOT is considered the treatment of choice for patients suspected to have CAGE (2,3). If unable to determine an alternative etiology for the patient's new neurologic findings, one should strongly suspect CAGE and consider consultation with a hyperbaric physician to determine if the patient would be a candidate for HBOT even if treatment will be delayed.

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