Cerebral Air Embolism after Esophagogastroduodenoscopy: Insight on Pathophysiology, Epidemiology, Prevention and Treatment

Malik Ghannam, MBBCh,* Azizullah Beran, MD,† Dana Ghazaleh, MD,‡ Tanner Ferderer, MD,* Brent Berry, MD,* Mona Al Banna, MD,* Leighton Mohl, MD,* Christopher Streib, MD,* Tapan Thacker, MD,§ and Ivan Matos, MD§

Background: Air embolism is an extremely rare complication that can follow gastrointestinal endoscopy. The most accepted treatment of cerebral air embolism (CAE) is hyperbaric oxygen (HBO). Limited evidence suggests that lidocaine may have a neuroprotective effect. The exact mechanism does not appear to be well elucidated. *Methods:* We conducted a literature search using multiple combinations of keywords from PubMed and Ovid Medline databases according to the PRISMA guidelines. We included articles with cases of air embolism caused by an esophagogastroduodenoscopy (EGD). We excluded cases related to other procedures e.g. colonoscopy, endoscopic retrograde cholangiopancreatography, cholangioscopy, Kasai procedure, bronchoscopy, laparoscopy or thoracoscopy. We were able to identify 30 cases of CAE associated with EGD. We included our experience in treating one patient with CAE after elective EGD. *Results:* Given the results of our literature search and this patient's characteristics, we chose to treat our patient with HBO and lidocaine infusion. Our case series consists of 31 patients of post EGD CAE, the mean age was 63.7 ± 11.14 years, 38.7% of the patients were women (n = 12). 38.7% of the cases underwent esophageal dilatation (n = 12), while 19.35% had EGD biopsy (n = 6), 9.6% had variceal ligation (n = 3), and 3.22% had variceal banding (n = 1). In 20 out of 31 cases, echocardiography has been documented, 20% of those patients (n = 4) had patent foramen ovale. HBO was used in treatment of 48% of cases (n = 15), among the included patients, 61% survived (n = 19). Our patient showed significant neurological improvement. *Conclusions:* Despite the rare incidence of CAE during or after EGD, physicians should be aware of this potential complication. In patients who develop sudden acute neurological symptoms, early diagnosis and intervention may prevent devastating neurological injury and death. The most accepted emergent treatment for CAE includes HBO, consideration of lidocaine, and work-up of source of the air embolism.

Key Words: Cerebral air embolism—esophagogastroduodenoscopy—pathophysiology—epidemiology—prevention—treatment

From the *Department of Neurology, University of Minnesota, Minneapolis, Minnesota; †Department of Gastroenterology and Hepatology, Mayo clinic, Rochester, Minnesota; ‡University of Minnesota, Minneapolis, Minnesota; and §Department of Neurology, Hennepin County Medical Center, Minneapolis, Minnesota.

Received July 29, 2019; revision received August 25, 2019; accepted September 8, 2019.

Address correspondence to Malik Ghannam, MBBCh, University of Minnesota, MMC 295, 12-181 Phillips Wangensteen Building, 516 Delaware St. SE, Minneapolis, MN 55455. E-mail: mghannam@umn.edu.

1052-3057/$ - see front matter

https://doi.org/10.1016/j.jstrokecerebrovasdis.2019.104403

Background

Esophagogastroduodenoscopy (EGD) is an essential, routine procedure used for diagnostic and therapeutic purposes in many medical conditions. Due to recent increasing trends in the use of endoscopic procedures, the incidence of endoscopy-related complications has increased, making it crucial for physicians to acutely detect and manage procedural complications.[1]

Air embolism is an extremely rare complication that can occur during or after EGD. Moreover, it is much more uncommon in diagnostic EGD compared to the therapeutic one. Air embolism may present with a multitude of neurological and cardiopulmonary manifestations, such as acute myocardial ischemia, arrhythmia, acute respiratory failure and acute cerebral hypoperfusion. The diagnosis of acute cerebral dysfunction is complicated by procedural sedation, often resulting in the delayed diagnosis of cerebral air embolism (CAE).[2] In 2013, Donepudi et al published a review which identified only 41 reported cases of air embolism following endoscopy in the medical literature.[2]

Methods

We conducted a literature search with the keywords "air embolism" and "endoscopy" from PubMed and Ovid Medline databases according to the PRISMA guidelines (Fig. 1). We included articles with cases of air embolism caused by an EGD. We excluded cases related to other procedures e.g., colonoscopy, endoscopic retrograde cholangiopancreatography, cholangioscopy, Kasai procedure, bronchoscopy, laparoscopy or thoracoscopy. Articles were limited to those published in English language. We were able to identify 40 cases of air embolism associated with EGD. Thirty of those were cases of CAE while 10 were cases of venous air embolism e.g., cardiopulmonary or portal air embolism. To our knowledge, there have been 30 cases in the literature to date of CAE associated with EGD procedure in addition to our current case. We included our experience in treating one patient with CAE after elective EGD.

Results

Our case series consists of 31 patients with post EGD CAE (Table 1). The mean age was 63.7 ± 11.14 years and 38.7% of the patients were women (n = 12). Regarding the indications for EGD, 35.5% of the patients had esophageal stricture (n = 11), 22.5% had variceal bleeding (n = 7), 6.4% had a gastric fistula (n = 2), and 6.4% had eosinophilic esophagitis (n = 2). The remaining 5 patients had other

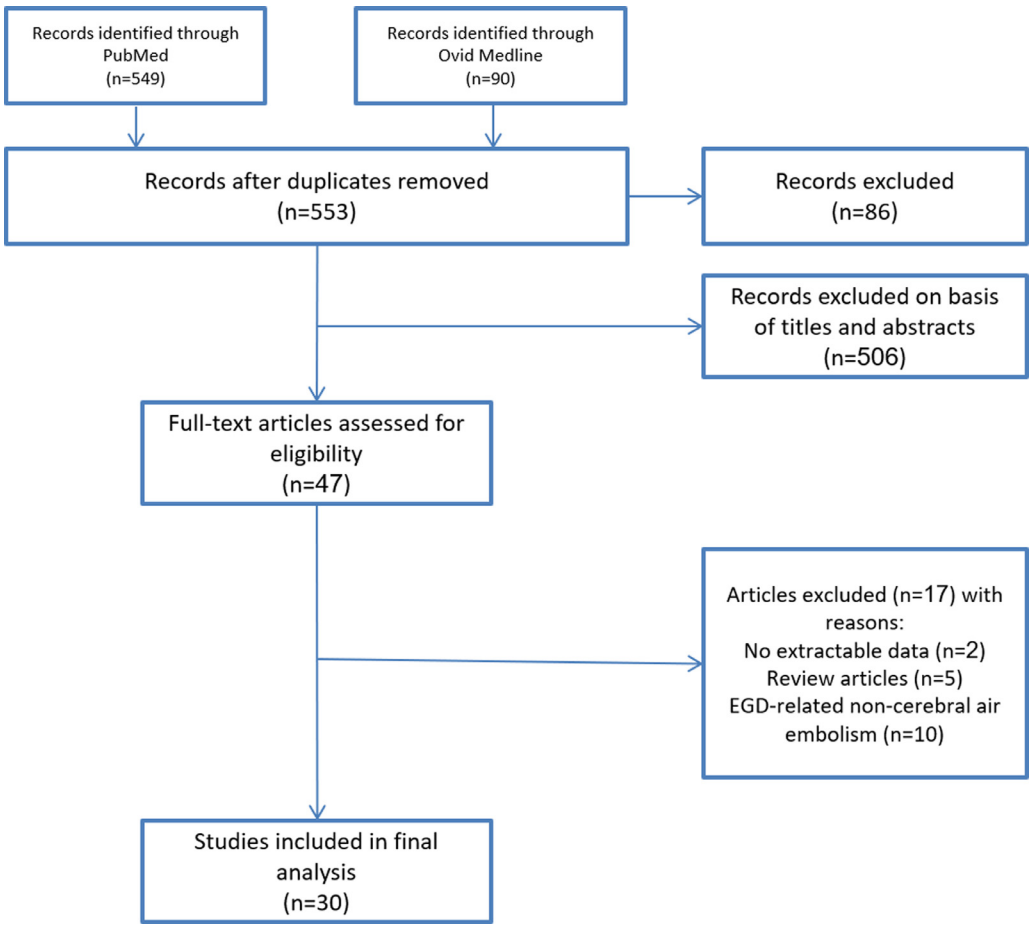

Figure 1. *Flow diagram according to PRISMA guidelines.*

Table 1. *All cases of CAE after EGD reported thus far*[1,6,26-35,8,36-44,12,20-25]

Case	Authors/year	Age/sex	Indication of EGD	Risk factor	Diagnosis	Treatment of CAE/outcome	Shunt
1	Christl et al.,1994[19]	66/F	Bleeding duodenal ulcer	Duodenocaval fistula	CAE	Supportive/dead	PFO (-)
2	Bou-Samra et al., 1997[20]	66/M	Gastric erosions	Gastric erosions	CAE	Supportive/survived	Unknown
3	Raju et al., 1998[21]	75/M	Benign esophageal stricture	Balloon dilation and esophageal biopsy	CAE	HBO/dead	PFO (+)
4	Akhtar et al., 2001[22]	80/F	Malignant esophageal stricture	Esophageal biopsy	CAE	Supportive/survived but in PVS	PFO (-)
5	Weber et al., 2003[23]	56/F	Hematemesis	Esophageal bleeding due to perforated Barret's ulcer	CAE	Supportive/survived	PFO (-)
6	Demaerel et al., 2003[24]	80/M	Red-brown sputum and Melena	Total laryngectomy	CAE	Supportive/survived	PFO (-)
7	Takeuchi et al., 2004[25]	59/M	Bleeding esophageal varices	EVL	CAE	Supportive/dead	Unknown
8	Green et al., 2005[6]	71/M	Benign esophageal stricture	Balloon dilation	CAE	HBO/survived	PFO (-)
9	Mellado et al., 2005[26]	52/F	Esophagitis	Esophagitis	CAE	Supportive/dead	Unknown
10	McAree et al., 2008[27]	69/M	Hematemesis	Esophageal biopsy of erosive esophagitis	CAE	Supportive/dead	PFO (-)
11	Ter Laan et al., 2009[28]	48/M	Gastro-mediastinal fistula	Stent placement for gastro-mediastinal fistula	CAE	HBO/survived	PFO (-)
12	Vinetti et al., 2010[29]	46/F	Gastric fistula	Gastric endoprosthesis placement	CAE	HBO/dead	PFO (+)
13	Oatis et al., 2010[30]	77/M	Odynophagia	Esophageal erosions and aphthous ulcers	CAE	HBO/survived	Unknown
14	Lopez et al., 2010[31]	61/F	Bleeding esophageal varices	Variceal banding	CAE	Supportive/survived	Unknown
15	Pandurangadu et al., 2012[7]	71/M	Duodenal AVM	Esophageal biopsy and ablation of duodenal AVM	CAE	Supportive/survived	PFO (-)
16	Truyols et al., 2012[32]	48/M	Hematemesis	Esophageal varices, chronic liver disease and portal hypertension	CAE	Supportive/dead	PFO (+)
17	Koster et al., 2012[33]	76/F	Post-radiation esophageal stricture	Balloon dilation for post-radiation esophageal stricture, esophageal cancer	CAE	Supportive/survived	Unknown
18	Zampeli et al., 2013[34]	72/M	Malignant esophageal stricture	Balloon dilation, radiation therapy for cardiac adenocarcinoma	CAE	Supportive/dead	Unknown
19	Niehaus et al., 2013[35]	64/F	Esophageal stricture	Esophageal dilation	CAE	HBO/dead	PFO (-)
20	Pee et al., 2013[36]	84/F	Benign esophageal stricture	Balloon dilation, esophagitis	CAE	HBO/dead	Unknown
21	Eoh et al., 2015[37]	64/F	GEJ anastomotic stricture	Balloon dilation	CAE	HBO/survived	PFO (-)
22	Covington et al., 2016[38]	49/M	EGD biopsy	EGD biopsy	CAE	HBO/survived	Unknown
23	Park et al., 2016[1]	59/M	Benign esophageal stricture	Balloon dilation	CAE	HBO/survived	PFO (-)

(*Continued*)

Table 1 (*Continued*)

Case	Authors/year	Age/sex	Indication of EGD	Risk factor	Diagnosis	Treatment of CAE/outcome	Shunt
24	Kim et al., 2016[39]	61/F	Hematemesis	EVL, liver cirrhosis	CAE	HBO/survived	PFO (+)
25	Cooper et al., 2017[12]	71/M	Food impaction	Dilation for food impaction	CAE	HBO/dead	Unknown
26	Cooper et al., 2017[12]	58/M	Esophageal stricture	Dilation	CAE	HBO/survived	Unknown
27	Kjellberg et al., 2018[40]	42/M	Eosinophilic esophagitis	Esophageal biopsy	CAE	HBO/survived	PFO (-)
28	Uldin et al., 2018[41]	72/M	Eosinophilic esophagitis	Dilation	CAE	Supportive/survived	PFO (-)
29	Bai et al., 2018[42]	50/F	Hematemesis	EVL, hepatitis C virus-related liver cirrhosis	CAE	Supportive/dead	PFO (-)
30	Fang et al., 2019[43]	67/M	Tarry stools	Mucosal erosion of anastomotic stoma, surgery of cardiac carcinoma and PNET	CAE	Supportive/survived	PFO (-)
31	Present case	61/M	Benign esophageal stricture (Schatzki's ring)	Balloon dilation	CAE	HBO and Lidocaine/survived	PFO (-)

Abbreviations: AVM, Arteriovenous malformation; CAE, Cerebral air embolism; EGD, Esophagogastroduodenoscopy; EVL, Endoscopic variceal ligation; GEJ, Gastroesophageal junction; HBO, Hyperbaric oxygen therapy; PFO, Patent foramen ovale; PVS, Persistent vegetative state.

indications which include a bleeding duodenal ulcer, a duodenal arteriovenous malformation (AVM), odynophagia, food impaction and tarry stool. In terms of the intervention that was undertaken, 38.7% of the cases underwent esophageal dilatation (n = 12) (8 of which had balloon dilatation), 19.4% of patients had EGD biopsy (n = 6), 13% underwent diagnostic EGD (n = 4), 9.6% of patients had variceal ligation (n = 3), 6.5% had stent placement (n = 2) and 3.2% of patients had variceal banding (n = 1). In 20 out of 31 cases, echocardiography has been documented. 20% of those patients (n = 4) had a confirmed patent foramen oval (PFO) whereas it was excluded in 80% of cases (n = 16). Hyperbaric oxygen (HBO) was used in treatment of 48% of cases (n = 15). Among the included patients, 61% survived (n = 19) (Fig. 2). Our patient showed significant neurological improvement.

Given the results of our literature search and this patient's characteristics, we chose to treat our patient with HBO and lidocaine infusion.

Case Description

Our experience includes a 61-year old Caucasian male with a past medical history of hyperlipidemia who was transferred to the Emergency Department (ED) with suspected CAE following a successful elective EGD with esophageal dilation under procedural sedation.

The procedure included a balloon dilation for a benign Schatzki's ring (size of ring opening: 16 mm) at the level of the gastroesophageal junction with a balloon size of 16 mm, 17 mm, and 18 mm. The procedure was uneventful with only routine resistance to balloon dilation and minor bleeding noted. Upon awaking from sedation that included propofol and lidocaine, the patient was dysarthric and unable to move his left arm and leg. The patient was immediately transferred to the ED where his neurologic exam demonstrated left-sided facial droop, complete left-sided hemiplegia, rightward gaze deviation, and severe dysarthria. The patient's initial National Institutes of Health Stroke Scale (NIHSS) scale was 19. Prior to the procedure, the patient was otherwise healthy without neurologic deficits.

Upon initial assessment in the ED, it was noted that his right hand was twitching, and he received 2 mg of IV lorazepam followed by 2 gm of levetiracetam as empiric treatment for partial seizures. Computed tomography (CT) scan of chest without IV contrast was performed in the ED and showed no pneumomediastinum or pneumothorax after EGD. However, a small amount of gas in the right hepatic lobe was noted. CT scan of the head showed multiple foci of air within the right frontal, parietal, and occipital regions and early loss of gray-white matter differentiation in the posterior right cerebrum, particularly in the right parietal lobe (Fig. 3). A diagnosis of CAE was made, and the patient was admitted to the neurocritical care unit.

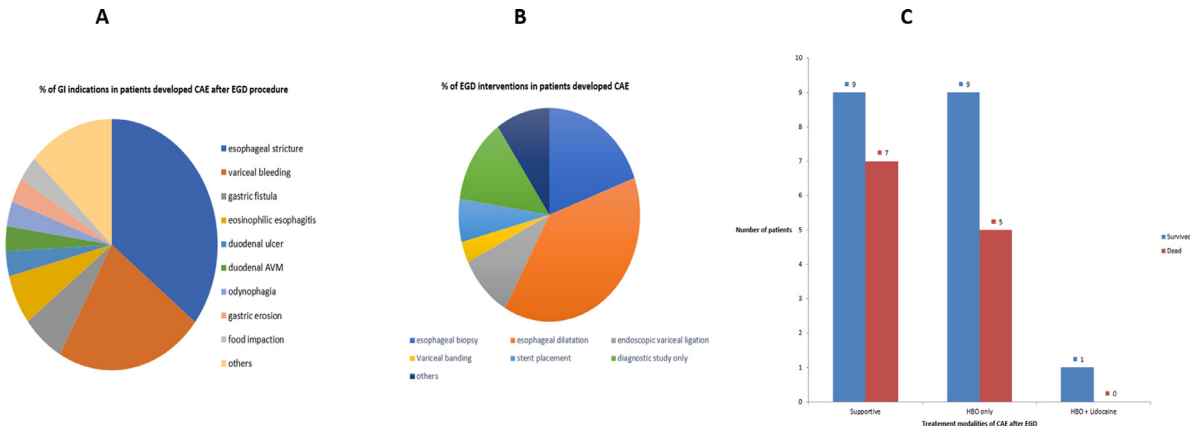

Figure 2. *GI indications (A) and EGD interventions (B) in patients who developed CAE. Treatment modalities of CAE after EGD (C).*

The patient underwent 2 dives of HBO treatment. The first dive lasted approximately 6.5 hours. The second dive occurred the following day and lasted approximately 3 hours. Please see Table 2 for further details. After the first dive of HBO, the patient was given 1 mg/kg bolus of IV lidocaine over 5 minutes, followed by 240 mg over the first hour and 120 mg over the second hour, and then 60 mg/h thereafter for 10 hours. On hospital day 2, he had echocardiography which was normal with the estimated left ventricular ejection fraction of 66 % and without evidence of any intracardiac shunting. He also underwent magnetic resonance imaging (MRI) of the head which showed diffusion restriction throughout the right cerebral hemisphere. After completing the hyperbaric oxygen treatment, the patient continued to be lethargic, but the right arm tremors subsided spontaneously. Twenty four-hour video EEG monitoring did not demonstrate any epileptiform activity. In the first 48 hours of admission, he was febrile up to 39°C . However, other vital signs were normal. Septic work up was negative except for mild leukocytosis on presentation of 11.27×10^3 / mm^3, that normalized after 2 days of treatment with broad spectrum antibiotics. Noticeably, on admission, the patient was complaining of severe, holocephalic, pounding, non-positional headache that subsided after receiving 2 gm of IV magnesium sulfate and divalproex sodium 750 mg twice daily.

From hospital day 3 until discharge, the patient had follow-up CT head which showed evolving right cerebral hemisphere infarct with increased mass effect on

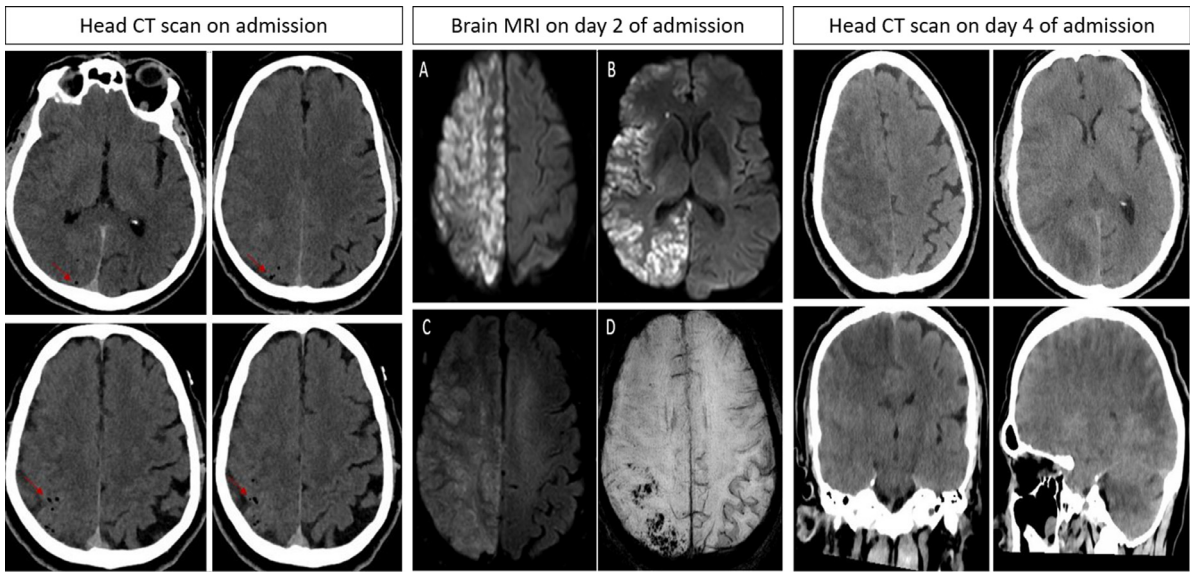

Figure 3. CT scan of the head on admission *showing multiple foci of air within the right frontal, parietal, and occipital regions and vague diminishment of the gray-white differentiation in the posterior right cerebrum, particularly of the right parietal lobe, which could represent hyper acute ischemia and/or infarction.* Brain MRI on day two of admission *showing diffusion restriction throughout the right cerebral hemisphere with a mixture vascular distribution involving the right occipital lobe, parietal lobe and posterior frontal lobe (A-C) and hemosiderin deposition within the occipital lobe and posterior parietal lobe (D).* CT scan of the head on day four of admission *showing evolving right cerebral hemisphere infarct with increased mass effect on the right lateral ventricle and multiple foci of low-attenuation in the corpus callosum. No midline shift.*

Table 2. *Patient's National Institutes of Health Stroke Scale (NIHSS) on presentation and HBO treatment course*

Patient's National Institutes of Health Stroke Scale (NIHSS) on presentation		Hyperbaric oxygen treatment course	
		Treatment day 1 started at 17:36	Treatment day 2 started at 1417
1a: LOC Not alert; requires repeated stimulation to attend or is obtunded and requires strong or painful stimulation to make movements (not stereotyped)	2	• Stop during compression due to otalgia and performed bilateral myringotomy • Tolerated remainder of compression without issue • Reached 60 feet (2.8 ATA) at 17:46 • Made decision to convert to USN Table 6A and left 60 feet at 18:02 • Reached 165 feet (6ATA) at 18:08, patient placed on Heliox 50% • Patient tolerated hyperbaric Heliox at 165 feet (6ATA) without issue • Left 165 feet at 18:38 • Reached 60 feet (2.8 ATA) at 19:13 • Patient tolerated hyperoxia at 60 feet without issues • Left 60 feet at 20:28 • Reached 30 feet (2.0 ATA) at 20:58 • Given 1mg/kg Lidocaine bolus at 21:33 • Lidocaine infusion started at 21:35 • Tolerated hyperoxia at 30 feet, became increasingly tachycardic to the 100s-120. IVF increased to 200 L/hr. Temp checked and was 100.5 axillary, later temp increased to 102.0 axillary • Left 30 feet at 23:28 • Surface reached at 23:58 End time: 23:58	• Was unable to tolerate compression beyond 2-3 feet due to left otalgia. Aborted and was brought back to surface and right myringotomy was revised • Attempted compression again and he did require a few stops to reach pressure but was able reach pressure with continuous coaching • He tolerated hyperoxia at 2.8 ATA with no problems. • During treatment, he required 2 small doses of hydromorphone because he was pulling at mask. He was also given 1 DuoNeb for expiratory wheeze throughout prior to initial staged decompression • Decompression to 2.0 ATA was uneventful • He tolerated hyperoxia at 2.0 ATA with no problems • Decompression to normobaric conditions was uneventful. • End time: 16:41
1b: Age/Month Answers neither question correctly	2		
1c: Grip/close eyes Performs both tasks correctly	0		
2: Best gaze Forced deviation, or total gaze paresis not overcome by the oculocephalic maneuver	2		
3: Visual fields No visual loss	0		
4: Facial Palsy Partial paralysis (total or near-total paralysis of the lower face)	2		
5a: Motor Arm L No movement	4		
5b: Motor Arm R No drift; limb holds 90 (or 45 degrees) for full 10 seconds	0		
6a: Motor Leg L No movement	4		
6b: Motor Leg R No drift; limb holds 90 (or 45 degrees) for full 5 seconds	0		
7: Limb Ataxia Absent	0		
8: Sensory Normal: no sensory loss	0		
9: Best Language Mild-moderate aphasia; some obvious loss of fluency or facility of comprehension, without significant limitation on ideas expressed or form of expression.	1		
10: Dysarthria Severe dysarthria; patient's speech is so slurred as to be unintelligible in the absence of or out of proportion to any dysphasia	2		
11: Extinction/Inattention None	0		
TOTAL	19		

Abbreviations: ATA, atmosphere absolute; IVF, intravenous fluid; L/hr, liter per hour; USN, US navy.

the right lateral ventricle, but without midline shift. CT chest without IV contrast 3 days after initial presentation was unremarkable, and the previously seen air in the right hepatic lobe was no longer visualized. Esophagogram was attempted twice to assess for possible esophageal tract injury as a potential source of gas but failed given the patient's mental status. On hospital day 4, the patient started to show neurological improvement in his motor symptoms, with voluntarily movement of his left arm and leg and some antigravity effort noted. He also became less somnolent and dysarthric. He was discharged after 7 days of admission into an acute rehabilitation facility with a discharge NIHSS of 8.

Discussion

Air embolism is an infrequent but serious adverse event of EGD and can be life-threatening or lead to deleterious long-term neurological deficits. Air embolism requires the presence of both a communication between the source of air and the vasculature accompanied by a sufficient pressure gradient favoring passage of air into the circulation.[3] During an EGD, air insufflation creates a pressure gradient which favors the passage of air into the vasculature.[4] It is believed that the disruption of the mucosal barrier allows the introduction of air into the esophageal venous channel.[4] However, there are reported cases without known mucosal barrier disruption.[4] In our case, mild bleeding occurred during the endoscopy supports the hypothesis of mucosal barrier disruption as the most likely etiology of air embolism.

As shown in Figure 4, air embolism can be venous, paradoxical or arterial.[5] Venous air may enter the arterial circulation by several mechanisms: a right-to-left intracardiac shunt (i.e., PFO), an intrapulmonary shunt, a gastrointestinal AVM, or insufficient filtration by the pulmonary capillaries.[5,6]

The development of systemic air embolism can cause a wide range of cardiopulmonary symptoms such as hypotension, arrhythmia, right heart failure, cardiovascular collapse, myocardial ischemia, cardiac arrest, acute dyspnea, hypoxia, respiratory failure and cyanosis. In addition, it can also cause neurological symptoms such as hemiparesis, altered mental status, hypertonicity, cerebral edema, cerebral hypoperfusion, coma and even death.[2] The most important determinants of CAE outcome are the amount of air entered, the type of gas (i.e., carbon dioxide, room air or nitrous oxide), patient position when the embolization occurs, and the rate of gas entry. Lethal volume of gas (approximately 3-5 ml/kg) and/or rapid rate of entry to the circulation will put a large strain on the right ventricle causing a reduction in left ventricular preload and hence cardiac output, eventually causing cerebral hypoperfusion. Moreover, the air bubbles can

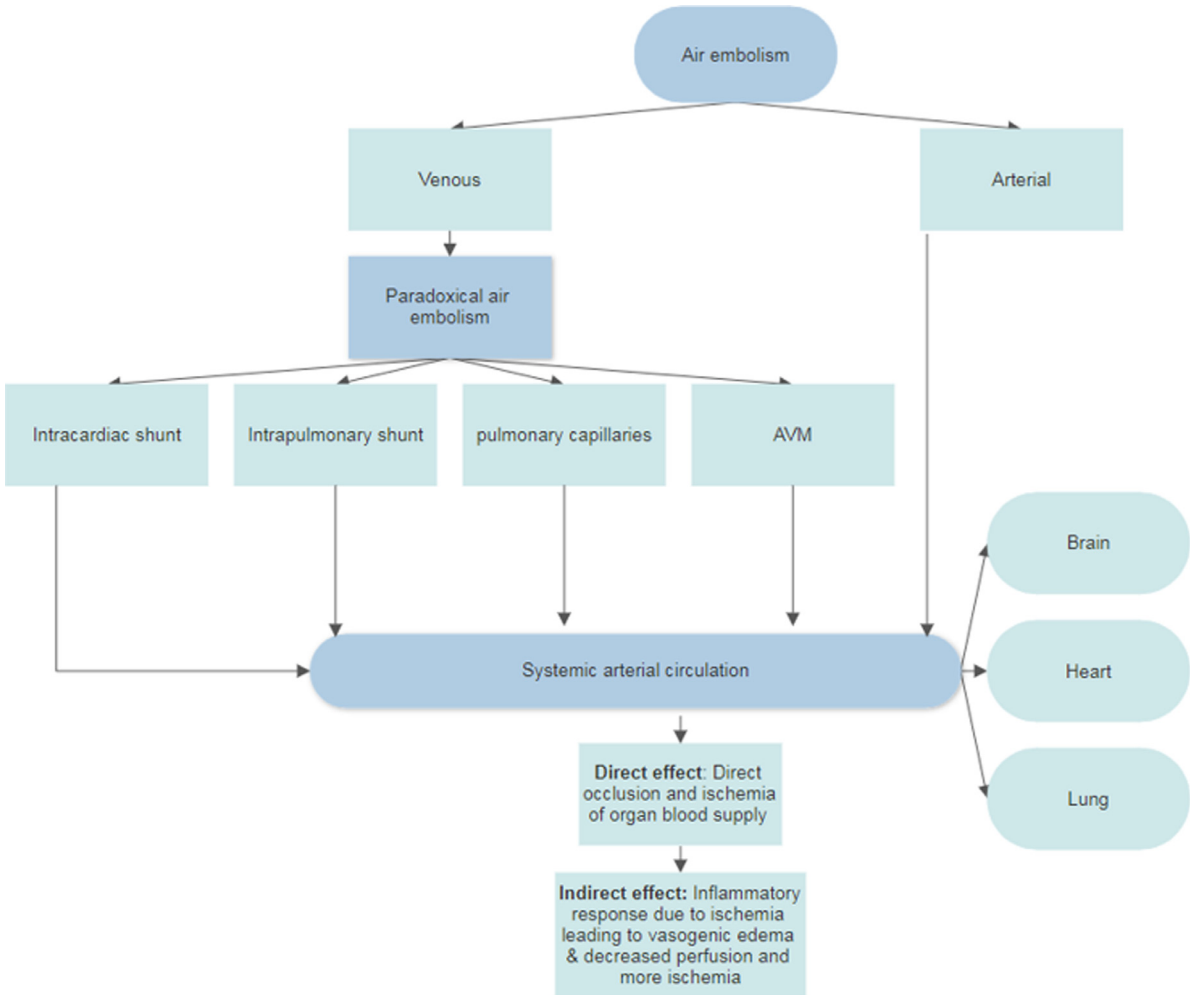

Figure 4. *Mechanism of paradoxical air embolism during EGD.*[5,6]

cause gas air-lock to the blood flow and inflammatory response that generates tissue edema, augmenting the vascular obstruction, and eventually causing a reduction in perfusion distal to the blockage leading to tissue hypoxia. Additionally, the emboli itself initiates a pulmonary inflammatory response, producing bronchospasm and ventilation-perfusion mismatch that will also lead to hypoxia and hypercapnia.[1,7]

Diagnosing CAE is often challenging due to nonspecific symptoms.[2] Delays in diagnosis can lead to permanent cerebral damage due to the intolerability of brain tissue to hypoxia.[8] Therefore, physicians must have a high index of suspicion for air embolism. CAE can also be detected early by new monitoring devices that allow early diagnosis and treatment before catastrophic cardiovascular or neurological compromise occur.[9] Measurement of end-tidal carbon dioxide by capnography in sedated patients may help to detect air embolism early, as a drop in end-tidal carbon dioxide could indicate gas in the heart.[9] However, this is a nonspecific finding, and bedside echocardiography may provide more reliable information in making the diagnosis by visualization of air within the right heart as well as excluding an intracardiac shunt.[2] CT head imaging is considered sensitive for CAE but diagnostic only if carried out instantaneously.[10] MRI of the head can also be used but is of limited value due to its unavailability in some settings.[8]

The most accepted treatment for patients with cerebral air embolism is hyperbaric oxygen treatment.[11] In theory, hyperbaric oxygenation treatment is thought to minimize the size of air bubbles, increase the diffusion gradient out of the bubbles, improve cerebral edema, accelerate the reabsorption of nitrogen, reduce platelet aggregation due to bubble induced endothelial damage, prevent O2 free radicle release and increase oxygen concentrations in the blood, all consequently decreasing the levels of ischemia.[11,12] Hyperbaric oxygen is based on the principle of Boyle's law that surface area and volume of gas bubble is inversely proportional to pressure at constant temperature.[5] Thus, time to chamber is the most important factor in treatment of CAE.[12] According to Murphy et al, when time to chamber is within 5 hours of symptom onset, full recovery is twice as likely compared to receiving HBO after 5 hours of symptom onset in CAE patients. Moreover, Tekle et al demonstrated an adjusted odds ratio of 9 for a favorable outcome in a retrospective study of 36 patients who received HBO $\leq$ 6 hours. However, it is important to mention that 2 of the 36 patients were treated with intravenous and intraarterial thrombolytics due to initial concern for thrombo-embolic events. One of these patients had a fatal intracerebral hemorrhage, which emphasizes the importance of early diagnosis and treatment of CAE with HBO.[12]

Our patient was successfully treated with hyperbaric oxygen (within 6 hours of symptom onset) and with lidocaine infusion. Limited evidence from animal studies suggests that lidocaine may have neuroprotective effects.[13-16] However, up to our knowledge, there have been no human clinical trials to date. The proposed mechanism of lidocaine is that it counters the suppressant effects of gas embolism on somatosensory evoked potentials and the elevation in intracranial pressure.[14,15] The exact mechanism does not appear to be well elucidated. Older animal models have had quite positive outcomes.[14-16] It is also important to note lidocaine's adverse effects when given excessively, which include cardiac bradyarrhythmias and central nervous system depression.[9] Given our patient's severe neurological deficits and the relatively low risks of lidocaine therapy in the absence of any cardiac history, we opted to follow the lidocaine protocol outlined by Mitchell et al. for neuro protection during cardiac surgery (1 mg/kg "bolus" over 5 minutes, followed by 240 mg over the first hour and 120 mg over the second hour, and then 60 mg/h thereafter).[13] Our plan was to continue lidocaine therapy for at least 12 hours in the absence of signs of systemic toxicity. Other studies have recommended up to 24-48 hours of lidocaine therapy.[13] We hypothesize that lidocaine might have facilitated our patient's fast recovery but emphasize the need for clinical trials and further data to support this hypothesis.

Other recommendations for CAE include placing the patient in left lateral and Trendelenburg position emergently to decrease the migration of air to the cerebral vessels, inserting a central catheter when air is detected in the right heart chamber, and decompressing with nasogastric suction.[2] A suggested algorithm for diagnosis and management of air embolism during EGD is included in Figure 5.

There are several signs that suggest poor prognosis in CAE, including the presence of Babiniski sign.[17] Other poor prognostic indicators in CAE include advanced age, cardiac arrest at the time of air embolism, acute kidney failure, prolonged mechanical ventilation for more than five days and Simplified Acute Physiology II (SAPS II) score of 33 or more at ICU admission.[17]

Previous studies suggested the use of carbon dioxide (CO_2) insufflation instead of room air, which has been found to reduce air embolism.[18,19] Therefore, it is recommended to routinely administer CO_2 instead of air in endoscopic procedures.[18,19] In addition, the reduction of maximum pressure has also been found to decrease the risk of developing cerebral air embolism.[18] The use of a precordial Doppler probe monitor during endoscopy has also been found useful to detect air in the vasculature early before the development of clinical manifestations.[2] A suggested algorithm for prevention and early detection of air embolism during EGD is included in Figure 6.

Conclusions

Despite the rare incidence of cerebral air embolism during or after esophagogastroduodenoscopy, physicians

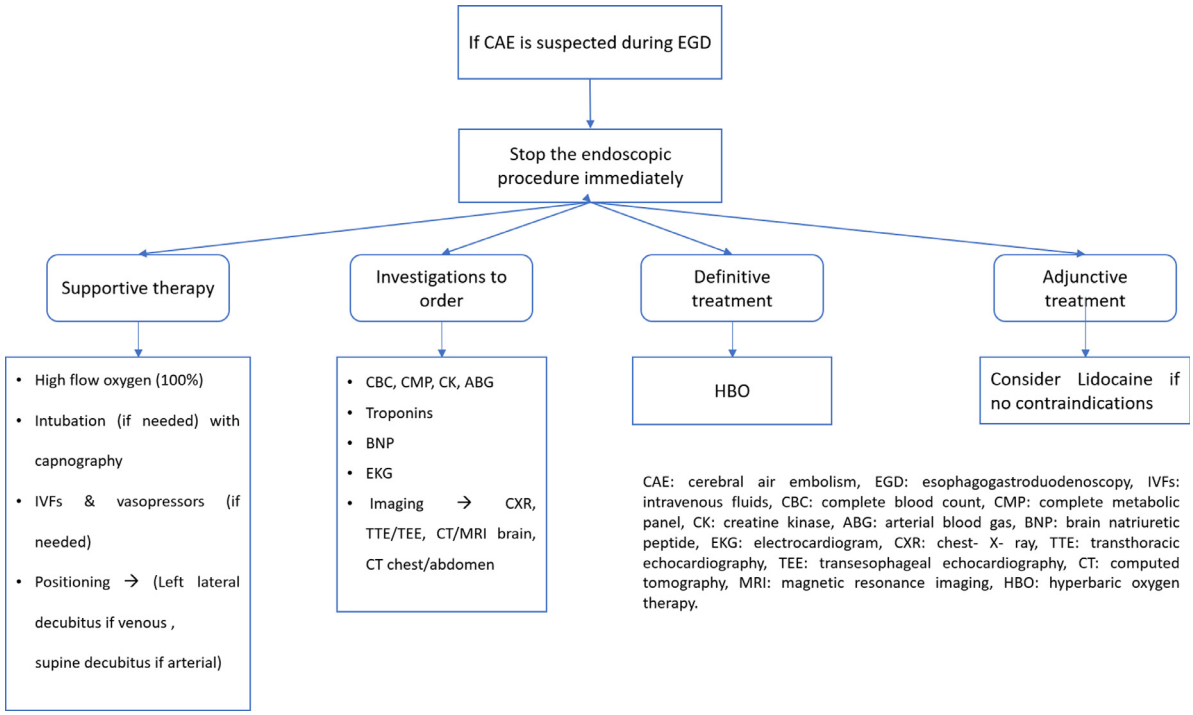

Figure 5. *Suggested algorithm for management of air embolism caused by EGD.*[9]

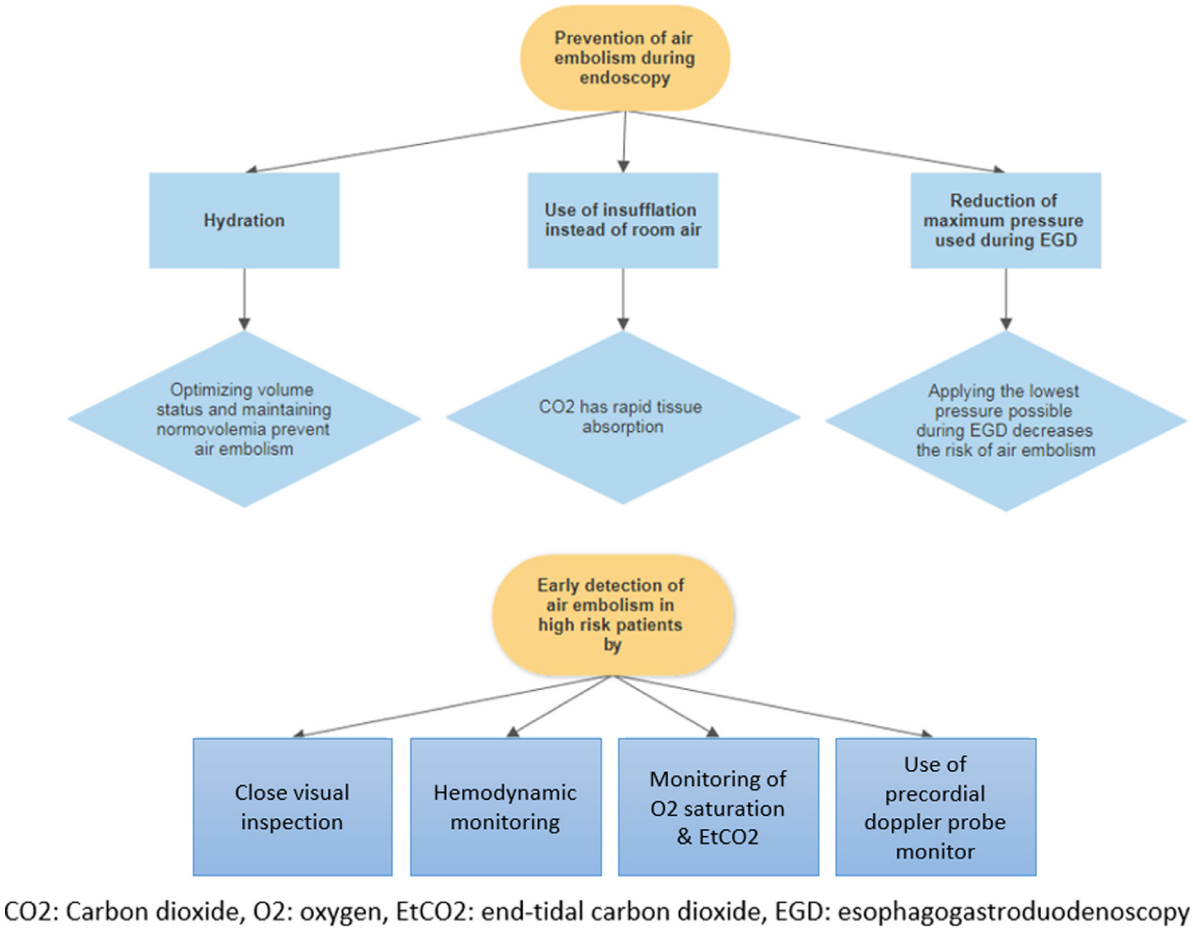

Figure 6. *Suggested algorithm for prevention and early detection of air embolism during EGD.*[2, 9,18,19]

should be aware of this potential complication. In patients who develop sudden acute neurological symptoms, early diagnosis and intervention may prevent devastating neurological injury and death. The most accepted emergent treatment for CAE includes HBO, consideration of lidocaine, and work-up of the underlying source of the CAE.

Conflict of Interest

The authors declare that they have no conflict of interest.

References

1. Jeon S-B, Park S, Kim J-H, et al. Two cases of cerebral air embolism that occurred during esophageal ballooning and endoscopic retrograde cholangiopancreatography. Clin Endosc 2016;49:191-196. https://doi.org/10.5946/ce.2015.071.
2. Donepudi S. Air embolism complicating gastrointestinal endoscopy: a systematic review. World J Gastrointest Endosc 2013;5(8):359. https://doi.org/10.4253/wjge.v5.i8.359.
3. CLAUS M. MUTH ESS. Venous gas embolism. N Engl J Med 2000;342:476-482. https://doi.org/10.1056/NEJM200002173420706.
4. Ha JF, Allanson E, Chandraratna H. Air embolism in gastroscopy. Int J Surg 2009;7:428-430. https://doi.org/10.1016/j.ijsu.2009.08.003.
5. Lanke G, Adler DG. Gas embolism during endoscopic retrograde cholangiopancreatography: diagnosis and management. Ann Gastroenterol 2019;32:156-167. https://doi.org/10.20524/aog.2018.0339.
6. Green BT, Tendler DA. Cerebral air embolism during upper endoscopy: case report and review. Gastrointest Endosc 2005;61:620-623. https://doi.org/10.1016/S0016-5107(04)02788-9.
7. Gordy S, Rowell S. Vascular air embolism. Int J Crit Illn Inj Sci 2013;3:73-76. https://doi.org/10.4103/2229-5151.109428.
8. Pandurangadu AV, Paul JAP, Barawi M, et al. A case report of cerebral air embolism after esophagogastroduodenoscopy: diagnosis and management in the emergency department. J Emerg Med 2012;43:976-979. https://doi.org/10.1016/j.jemermed.2010.11.031.
9. Mirski. Diagnosis and treatment of vascular air embolism. Anesthesiology 2007;106(1):164-177. http://www.sarasotaanesthesia.com/reading/monthlyarticles/Anes_Jan07_VenouseAir.pdf.
10. McAree BJ, Gilliland R, Campbell DM, et al. Cerebral air embolism complicating esophagogastroduodenoscopy (EGD). Endoscopy 2008:191-193. doi:10.1055/s-2007-995728.
11. Blanc P, Boussuges A, Henriette K, et al. Iatrogenic cerebral air embolism: importance of an early hyperbaric oxygenation. Intens Care Med 2002;28:559-563.
12. Cooper JS, Thomas J, Singh S, et al. Endoscopic bubble trouble: hyperbaric oxygen therapy for cerebral gas embolism during upper endoscopy. J Clin Gastroenterol 2017;51:e48-e51. https://doi.org/10.1097/MCG.0000000000000614.
13. Mitchell SJ, Pellett O, Gorman DF. Cerebral protection by lidocaine during cardiac operations. Ann Thorac Surg 1999;67:1117-1124.
14. Dutka AJ, Mink R, McDermott J, et al. Effect of lidocaine on somatosensory evoked response and cerebral blood flow after canine cerebral air embolism. Stroke 1992;23:1511-1515.
15. Evans DE, Kobrine AI, LeGrys DC, et al. Protective effect of lidocaine in acute cerebral ischemia induced by air embolism. J Neurosurg 1984;60:257-263. https://doi.org/10.3171/jns.1984.60.2.0257.
16. Evans DE, Catron PW, McDermott JJ, et al. Effect of lidocaine after experimental cerebral ischemia induced by air embolism. J Neurosurg 1989;70:97-102. https://doi.org/10.3171/jns.1989.70.1.0097.
17. Bedford RF, Marshall WK, Butler A, et al. Cardiac catheters for diagnosis and treatment of venous air embolism: a prospective study in man. J Neurosurg 1981;55:610-614. https://doi.org/10.3171/jns.1981.55.4.0610.
18. Katzgraber F, Glenewinkel F, Fischler S, et al. Mechanism of fatal air embolism after gastrointestinal endoscopy. Int J Legal Med 1998;111:154-156. https://doi.org/10.1007/s004140050137.
19. Wang WL, Wu ZH, Sun Q, et al. Meta-analysis: the use of carbon dioxide insufflation vs. room air insufflation for gastrointestinal endoscopy. Aliment Pharmacol Ther 2012;35:1145-1154. https://doi.org/10.1111/j.1365-2036.2012.05078.x.
20. Christl SU, Scheppach W, Peters U, et al. Cerebral air embolism after gastroduodenoscopy: complication of a duodenocaval fistula. Gastrointest Endosc 1994;40:376-378.
21. Bou-Samra G, Darby PJ, Christensen GD. Cerebral air embolism during endoscopy. Mo Med 1997;94:704-707.
22. Raju GS, Bendixen BH, Khan J, et al. Cerebrovascular accident during endoscopy: consider cerebral air embolism, a rapidly reversible event with hyperbaric oxygen therapy. Gastrointest Endosc 1998;47:70-73.
23. Akhtar N, Jafri W, Mozaffar T. Cerebral artery air embolism following an esophagogastroscopy: a case report. Neurology 2001;56:136-137.
24. Weber M-A, Fiebach JB, Lichy MP, et al. Bilateral cerebral air embolism. J Neurol 2003;250:1115-1117. https://doi.org/10.1007/s00415-003-0130-4.
25. Demaerel P, Gevers A-M, De Bruecker Y, et al. Stroke caused by cerebral air embolism during endoscopy. Gastrointest Endosc 2003;57:134-135. https://doi.org/10.1067/mge.2003.43.
26. Takeuchi Y, Yoshikawa M, Shirahama K, et al. Cerebral artery air embolism during endoscopic variceal ligation: case report. Gastrointest Endosc 2004;59(1):123-125.
27. Mellado TP, Constanzo PF, Miquel PJF, et al. [Ischemic brain infarction after an air embolism. Case report]. Rev Med Chil 2005;133:453-456. doi:S0034-98872005000400009.
28. McAree BJ, Gilliland R, Campbell DM, et al. Cerebral air embolism complicating esophagogastroduodenoscopy (EGD). Endoscopy 2008;40(Suppl 2):E191-E192. https://doi.org/10.1055/s-2007-995728.
29. ter Laan M, Totte E, van Hulst RA, et al. Cerebral gas embolism due to upper gastrointestinal endoscopy. Eur J Gastroenterol Hepatol 2009;21:833-835. https://doi.org/10.1097/MEG.0b013e328310aefc.
30. Vinetti M, De Roock S, Hantson P. Image of the month. Massive cerebral air embolism during esophagogastroduodenoscopy. Clin Gastroenterol Hepatol 2010;8:A20. https://doi.org/10.1016/j.cgh.2009.03.008.
31. Oatis K, Agarwal A, Bruce-Tagoe C. Acute stroke from air embolism to the middle cerebral artery following upper gastrointestinal endoscopy. Radiol case reports 2010;5:359. https://doi.org/10.2484/rcr.v5i1.359.

32. Lopez JC, Perez X, Esteve F. Cerebral air embolism during upper endoscopy. Endoscopy 2010;42(Suppl 2):E41. https://doi.org/10.1055/s-0029-1215313.
33. Truyols C. Fatal air embolism after upper endoscopy in a 48-year-old man. Endoscopy 2012;44(Suppl 2 UCTN): E282. https://doi.org/10.1055/s-0032-1310024.
34. Koster GT, Brandsma D, Boot H, et al. Neurological picture. Multiple cerebral air emboli during upper gastrointestinal endoscopy. J Neurol Neurosurg Psychiatry 2012;83:1110-1111. https://doi.org/10.1136/jnnp-2012-303377.
35. Zampeli E, Tsagalou E, Kanakakis I, et al. Fatal cerebral air embolism complicating esophageal dilation. Endoscopy 2013;45(Suppl 2 UCTN):E358. https://doi.org/10.1055/s-0033-1344409.
36. Niehaus K, Zamora E, Harkins M. Fatal central nervous system air embolism during elective esophagogastroduodenoscopy. Endoscopy 2013;45(Suppl 2):E205. https://doi.org/10.1055/s-0033-1344328.
37. Pee L, Basu S, Loganayagam A. Cerebral air embolism: a rare complication following balloon dilation. Endoscopy 2013;45(Suppl 2 UCTN):E93-E94. https://doi.org/10.1055/s-0032-1326251.
38. Eoh EJ, Derrick B, Moon R. Cerebral arterial gas embolism during upper endoscopy. A A Case Rep 2015;5: 93-94. https://doi.org/10.1213/XAA.0000000000000187.
39. Covington D, Bielawski A, Sadler C, et al. A favorable outcome despite a 39-hour treatment delay for arterial gas embolism: case report. Undersea Hyperb Med 2016;43: 457-461.
40. Kim D. Cerebral air embolism during endoscopic band ligation with liver cirrhosis. Neurol Asia 2016;21(2):195-197. www.neurology-asia.org/articles/neuroasia-2016-21(2)-195.pdf.
41. Kjellberg A, Nystrom H, Soderberg M, et al. Massive air embolism as a complication of upper gastrointestinal endoscopy: a case report illustrating a stroke mimic, literature review, and suggested management. Clin case reports 2018;6:1862-1867. https://doi.org/10.1002/ccr3.1725.
42. Uldin HHU, Shabana AAS, Raslan OOR. A case of retrograde venous air embolism causing cerebral infarction following upper gastrointestinal endoscopy: a novel pathophysiological mechanism. Radiol Case Rep 2018;13:1093-1096. https://doi.org/10.1016/j.radcr.2018.08.002.
43. Bai X-S, Yang B, Yu Y-J, et al. Cerebral air embolism following an endoscopic variceal ligation: a case report. Medicine (Baltimore) 2018;97:e10965. https://doi.org/10.1097/MD.0000000000010965.
44. Fang Y, Wu J, Wang F, et al. Air embolism during upper endoscopy: a case report. Clin Endosc 2019;52(4): 365-368. https://doi.org/10.5946/ce.2018.201. [Epub 2019 Mar 13].