Treatment of Endoscopy Associated Cerebral Gas Embolism

Jeffrey Cooper¹, MD

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INTRODUCTION

Arterial gas embolisms, albeit rare [1], can have devastating outcomes. Iatrogenic cases have mortality of 16% at hospital discharge and 21% at one year [2]. Embolization of gas during endoscopy is a rare complication with only a few cases having been reported [3] including three of our own [4]. The inciting pathophysiology remains unclear. It is likely that gas is entrained first in the venous system and then shunts to the arterial system via an arteriovenous malformation or cardiac septal defect such as a patent foramen ovale (PFO). PFO with shunting can occur in up to 25% of the general population [5] making screening for PFOs impractical [6].

Endoscopy requires insufflation for better visualization. Such insufflation creates pressure gradients that have been cited as a factor in creating gas embolisms [7]. Apart from occlusion, vascular irritation from large boluses of air cause higher neutrophil and cytokine release leading to further inflammation [8] (Fig. 1).

PREVENTION

Carbon dioxide (CO₂) is recommended over air for insufflation. CO₂ can be more readily absorbed and should not cause an occlusion in the vasculature. Several studies have been performed showed superiority of CO₂ over air in endoscopic procedures [9]. No trials or series have looked at this problem so incidence is a gross estimate based on case reports. These reports are suggestive of contributing factors. Care should be taken to avoid over insufflation as an increased pressure gradient is clearly a factor [10]. Patient risk factors include mucosal defects such as ulcers and atrophy, radiation strictures, inflammatory bowel disease and patent foramen ovale. Kasai procedure, varices, vasculocenteric fistula and Roux-en-Y limb all increase susceptibility. Certain procedures have a higher risk as well, such as stent placement, sphincterotomy, dilation, and biopsy with endoscopic retrograde cholangiopancreatography (ERCP) in general being the most highly associated with gas emboli. However, Gas emboli have occurred with all types of endoscopic procedures including observation only. The use of air is noted to be a strong risk factor for emboli as opposed to CO₂ or NO₂ (Table 1).

PAST REPORTS

A number of reviews exist in the literature on this topic but no major patterns appear and pooling the data is difficult. Donepudi reports on 26 cases of gas emboli associated with ERCP [3]. In total 6 of 12 of these dying out of those with cerebral arterial gas embolism (CAGE). Others succumbed to pulmonary, cardiac or venous gas embolism. Another report included 12 cases of EGD related CAGE [7]. In total 4 of these were ERCP with sphincterotomy related, 4 with simple EGD, 2 with biopsy and 2 endoscopy of Kasai pouches. Four of these were fatal. Neither of the above reports commented on outcome beyond survival which is concerning as the sequelae of CAGE can be quite debilitating. For example, Laan [6] reported that of 10 such cases of EGD related CAGE, 6 died but of the 4 survivors 3 were left hemiparetic and the fourth was in a persistent vegetative state. The bottom line is that although CAGE complicating upper endoscopy is rare, it can and does occur regardless of risk factors. Prevention, early detection and prompt treatment are imperative to provide the best outcomes.

DETECTION

Diagnosis can be difficult because other causes must be ruled out, and the gas bubbles might be absorbed before the necessary test is completed. An echocardiogram or chest computed tomography (CT) can be useful [3, 7, 11]. Head CT has low sensitivity of about 30%. However, no test, including CT, has adequate sensitivity to rule out gas emboli as bubbles may be reabsorbed quickly [3]. The best strategy may be prompt referral for hyperbaric oxygen therapy (HBO) based on clinical suspicion alone, given the low risk of HBO and the rapid increase of morbidity and mortality with delaying treatment [12]. Sudden cardiovascular collapse is suggestive of venous gas embolism to the right heart and should be treated by resuscitative efforts and evacuation of the gas from the right ventricle by central line insertion and aspiration [11]. Any stroke like symptoms, during the procedure or within minutes after, should raise the suspicion of cerebral gas embolism (Figs. 2 and 3).

¹University of Nebraska Medical Center, Omaha, NE, 68198-1150, USA. **Correspondence:** J.C. (email: jeffrey.cooper@unmc.edu)

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Fig. 1 Demonstrating right to left shunting, allowing venous gas to embolize to the arterial system

Table 1 Risk factors for endoscopy related gas embolism

Patient factors	Procedural risks
Mucosal defects	ERCP
Ulcers	Stent placement
Atrophy	Sphincterotomy
Radiation strictures	Dilation
Inflammatory bowel disease	Biopsy
Anatomic issues	Air insufflation
Vasculointestinal fistula	Over insufflation
Kasai procedure	
Varices	
Roux-En-Y limb	
Patent foramen ovale

Fig. 2 CT scan demonstrating cerebral gas emboli from a 71 year old male with esophageal food bolus impaction. EGD was performed with the obstruction advanced into the stomach. While being prepared for subsequent dilation, he suddenly became bradycardic, hypoxic, and unresponsive. A CT after the initial HBO treatment showed gas resolution but extensive cerebral swelling

TREATMENT

As for treatment, initially, the patient should be given 100% O₂ to help dissolve the gas bubbles [3]. Nitrous oxide (N₂O), if used, must be stopped immediately due its likelihood of entering the gas emboli. Gas used to insufflate should be suctioned out. The use of the Trendelenburg position to reduce the likelihood of bubbles going to the brain, has been disproved and may cause harm secondary to increased intracranial pressure and cerebral edema may result [4, 13, 14]. Positioning in the left lateral decubitus position may help remove gas from the right ventricle outflow tract [11]. Central venous line placement has been shown to be helpful in removing gas bubbles from the right heart [11].

The most beneficial therapy in eliminating the gas bubbles is hyperbaric oxygen [7, 8]. HBO acts by a variety of mechanisms leading to its use in many adjunctive indications. One of its primary indications is in the treatment of gas embolism [15]. Its beneficial effects in the setting of gas emboli include: Reduced bubble volume, increased diffusion gradient out of the bubbles,

oxygenation of hypoxic tissues, amelioration of cerebral edema, reduced platelet aggregation and activation of the coagulation cascade due to bubble induced endothelial trauma, decreased endothelial binding of leukocytes and prevention of oxygen free radical release [4]. In a retrospective study of 16 patients with gas emboli, 50% of the patients made complete recoveries after HBO [8].

Time to chamber is a crucial factor. Patients who reach the chamber within five hours are twice as likely to show a full recovery as those receiving HBO after 5 hours [8]. There are few adjunctive therapies for cerebral gas embolism. These include IV fluids to maintain preload, inotropic support to maintain cerebral

Fig. 3 CT scan demonstrating aortic gas. We detected a similar case using echocardiography in a patient undergoing stent placement during ERCP. During the procedure he became pulseless with ventricular tachycardia. He had complete resolution with HBO with no sequelae

perfusion pressure and lidocaine [14]. However, rapid initiation of hyperbaric oxygen therapy cannot be overstressed.

SUMMARY

Gas emboli are a rare complication in endoscopic procedures that can have dire outcomes. Due to the inescapability of the factors that may cause them, one must be vigilant when performing an endoscopic procedure for the possibility of gas emboli, especially in someone with advanced pathology, in long procedures requiring interventions and much insufflation, and when using air insufflation instead of carbon dioxide. While there are few adjunctive treatments, the definitive therapy for gas embolisms is prompt hyperbaric oxygen.

CONFLICT OF INTEREST

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