

Air embolism complicating gastrointestinal endoscopy: A systematic review

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Therefore, we wanted to review the risk factors, the clinical presentation, and the therapy of an air embolism from the perspective of the practicing endoscopist.

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Key words: Air embolism; Endoscopy; Endoscopic retrograde cholangiopancreatography; Complications; Therapy

Core tip: Air embolism at the time of endoscopy can cause cardiovascular, pulmonary, and neurological symptoms. Symptom onset during the position change from prone to supine is characteristic and should trigger immediate suspicion for air embolism. Potentially lifesaving therapeutic measures should be promptly initiated, including placing the patient in Trendelenburg and left lateral decubitus position, high-flow oxygen, volume expansion and urgent hyperbaric oxygenation therapy.

Abstract

Gastrointestinal endoscopy has become an important modality for the diagnosis and treatment of various gastrointestinal disorders. One of its major advantages is that it is minimally invasive and has an excellent safety record. Nevertheless, some complications do occur, and endoscopists are well aware and prepared to deal with the commonly recognized ones including bleeding, perforation, infection, and adverse effects from the sedative medications. Air embolism is a very rare endoscopic complication but possesses the potential to be severe and fatal. It can present with cardiopulmonary instability and neurologic symptoms. The diagnosis may be difficult because of its clinical presentation, which can overlap with sedation-related cardiopulmonary problems or neurologic symptoms possibly attributed to an ischemic or hemorrhagic central nervous system event. Increased awareness is essential for prompt recognition of the air embolism, which can allow potentially life-saving therapy to be provided.

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INTRODUCTION

Air embolism is a consequence of direct communication between a source of air and the vasculature and a pressure gradient favoring the passage of air into the circulation. The effect of an air embolus depends upon both the rate and the volume of air introduced into the circulation. A venous air embolism occurs when air enters the systemic venous circulation. An arterial air embolism results from introduction of air into the arterial system and can produce ischemia of any organ. An air embolism

is an uncommon but potentially catastrophic event. Many cases are subclinical with no adverse outcome. However, severe cases are characterized by hemodynamic collapse and/or acute vascular insufficiency of specific organs, such as the brain or the spinal cord. Symptoms may be nonspecific, and therefore, a high index of clinical suspicion for a possible air embolism is required to prompt investigations and initiate appropriate therapy.

RESEARCH

We conducted a systematic review by searching the PubMed database on reported air embolisms complicating all endoscopic procedures. Medical subject headings “endoscopy, complications, air embolism, cerebrovascular accident, cardiovascular abnormalities, esophago-gastroduodenoscopy (EGD), enteroscopy, colonoscopy, sigmoidoscopy, endoscopic ultrasound (EUS), and endoscopic retrograde cholangiopancreatography (ERCP)” were used in the title, the abstract, or the index term fields. Manual searches were then conducted using the reference lists from identified articles.

RISK FACTORS FOR AIR EMBOLISM

Air embolism is most commonly associated with an ERCP, but it can result from any endoscopic procedure including an EGD, an enteroscopy, an EUS, a colonoscopy, and a sigmoidoscopy.

Risk factors for an air embolism that have been reported are previous interventions or surgeries of the bile duct system, transhepatic portosystemic shunt^[1-3], blunt or penetrating trauma to the liver^[4], inflammation of the digestive system, post-surgical gastrointestinal fistula^[5-7], and particular interventional techniques.

The inflammatory conditions associated with an increased risk for an air embolism include inflammation of the bile duct or surrounding veins (pylephlebitis), hepatic abscesses, inflammatory bowel diseases, necrotizing enterocolitis, and mesenteric ischemia^[2,7,8]. In addition, gastrointestinal tumors and biliary atresia have been described as risk factors^[9,10].

Interventional techniques include cholangioscopy, biliary sphincterotomy, metal stent placement, liver biopsy, insufflation of air with high pressure, excessive amount and/or increased rate of air infusion, procedural site located higher than the level of the heart, and the use of nitrous oxide (N₂O)^[5,11-18].

MECHANISMS AND CLINICAL SIGNS AND SYMPTOMS

A number of potential mechanisms for air entry into the venous system have been described. These include intramural dissection of insufflated air into the portal vein, transection of duodenal vein radicles, biliary-venous fistulas/shunts, portocaval collaterals, air flow directly into the hepatic veins or inferior vena cava, retrograde

flow into cerebral veins *via* superior vena cava^[7], inability of the pulmonary circulation to filter out gas emboli^[19,20], or entry into the vertebral venous plexus^[21]. Rapid entry or large volumes of air entering the systemic venous circulation causes a substantial strain on the right ventricle, especially if this results in a significant rise in pulmonary artery pressures. This increase in pulmonary artery pressure can lead to right ventricular outflow obstruction and further compromise pulmonary venous return to the left heart. Consequently, the diminished pulmonary venous return will lead to decreased left ventricular preload with resultant decreased cardiac output, and eventually, systemic cardiovascular collapse.

Importantly, a venous air embolism can be limited to the portal venous system or can evolve into a systemic air embolism through intracardiac shunts, intrapulmonary right to left shunts, retrograde flow into cerebral veins *via* the superior vena cava, or air passage into the left atrium *via* the pulmonary veins^[18,22]. The most common cause of an intracardiac shunt is a patent foramen ovale^[11,23]. Atrial septal defect, arterio-venous shunts, and intrapulmonary shunts are also reported mechanisms^[24-26].

The systemic air embolism can cause cardiovascular, pulmonary, and neurological symptoms^[15]. Cardiovascular signs, symptoms, and findings include arrhythmia, hypotension, myocardial ischemia, right heart failure, cardiovascular collapse, and cardiac arrest. Pulmonary signs, symptoms, and findings include acute dyspnea, tachypnea, breathlessness, rales, wheezing, decrease in end tidal carbon dioxide concentration, hypoxia, cyanosis, and respiratory failure. Neurological signs, symptoms, and findings include eye deviation, dilated pupil(s), failure to regain consciousness after anesthesia, hypertonicity, altered mental status, loss of consciousness, hemiparesis, cerebral hypoperfusion, cerebral edema, and coma.

In the case of an ERCP-related air embolism, typically the symptoms appear or get significantly worse when the patient is repositioned from prone to supine position at the end of the procedure. This patient deterioration with position change should immediately raise a red flag and trigger suspicion for an air embolism as the underlying cause of the patient symptoms.

REPORTED CASES OF AIR EMBOLISM

We were able to identify 41 cases of air embolism in the published literature following various endoscopic procedures.

Air embolism cases following EGD and intraoperative small bowel endoscopy

The first case of air embolism following an EGD was reported by Lowdon *et al*^[18] in 1988. A 5-wk old infant with biliary atresia status post Kasai procedure (hepatoporojejunostomy) died during endoscopy, and the autopsy revealed air in both the right atrium and right ventricle and in the large hepatic vein in the area of the porta hepatis. The patient was also found to have a patent fora-

men ovale and air in the coronary arteries. The authors proposed that air under pressure dissected across the diseased hepatic tissue into the large hepatic vein lying just below the denuded liver surface. This combined with her patent foramen ovale resulted in the systemic embolism.

Christl *et al*^[3] described the first incident of a cerebral air embolism following endoscopy in a patient with a duodenal ulcer and a duodenocaval fistula. It was believed that the air emboli exceeding the absorptive rate of the pulmonary capillary bed might be the cause in this patient, especially with the total amount of air entering the inferior vena cava.

A case report by Katzgraber *et al*^[14] identified an embolism risk when air insufflation occurs in the presence of damaged vessels. The patient, whom had a history of a perforated gastric ulcer surgically treated 13 years prior, underwent an upper endoscopy for the evaluation of epigastric pain. High-volume air insufflation was noted during the procedure. As the study was continued, the patient suddenly went into cardiac arrest and resuscitation was unsuccessful. On forensics, the right gastric vein was found to be eroded most likely due to his history of multiple ulcerations. The presence of this lesion in combination with the amount of insufflation required allowed air to enter the venous supply, and eventually, enter the heart causing death.

McAree *et al*^[27] reported a cerebral air embolism in a patient being evaluated for metastatic adenocarcinoma of an unknown origin. Abdominal computed tomography (CT) scan showed ascites and a thickened cecal wall; cytology study of the ascites determined the presence of an adenocarcinoma. Soon after, the patient began vomiting blood and an EGD showed erosive esophagitis. As the procedure was ending, the patient became unresponsive and displayed neurological symptoms. An emergency cerebral CT confirmed air in the brain, specifically the right frontotemporal area. The esophagitis mucosal breakdown is to be considered as the leading cause of the embolism.

Meier *et al*^[28] reported a patient with an air embolism during an EGD. The patient had a history of a pancreaticoduodenectomy for pancreatic adenocarcinoma and a percutaneous transhepatic cholangiography (PTC) for recent ascending cholangitis. An EGD was done due to the patient developing melena. As the scope was maneuvered towards the hepaticojejunostomy site, the patient's condition became unstable; unfortunately, the patient was unable to be successfully resuscitated and passed away. The investigators believed the PTC catheter may have created a fistula between the vasculature and the biliary tract. This abnormality allowed the air to enter the venous supply, specifically the hepatic veins and inferior vena cava, upon air insufflation during endoscopy.

Pandurangadu *et al*^[24] reported about an incident of a cerebral embolism in 2010. The patient received an outpatient EGD, which required an esophageal biopsy and ablation of duodenal arteriovenous malformations. Shortly after the EGD procedure, he presented to the emergency room with neurological symptoms of sudden

onset lethargy and left-sided weakness. CT scan of the brain showed multiple gas emboli in two areas, the right frontal lobe and frontoparietal region. A transesophageal echocardiogram (TEE) was also done, which ruled out a patent foramen ovale. Therefore, the study proposed the most likely cause of the embolism was the duodenal arteriovenous malformations.

Additionally in 2010, a case reported by Park *et al*^[23] described a paradoxical air embolism during an intraoperative small bowel endoscopy. An adolescent female, with a history of a Kasai operation for biliary atresia, presented with gastrointestinal bleeding. The plan was for an exploratory laparotomy and intraoperative endoscopy for further evaluation. It must also be noted she had a previous exploratory laparotomy three months prior for hematochezia, which was unremarkable with the exception of some bluish edema on the small bowel wall. All preoperative protocols were performed, including an abdominal CT scan; the scan revealed a highly, irregular liver architecture showing massive fibrosis. As the endoscopy procedure ensued, excessive air insufflation was needed to facilitate visualization. An ulcerative lesion was found near the site of the previous Kasai procedure. The patient thereafter became unstable systemically, and immediate stabilization methods were started. TEE confirmed air bubbles entered the heart and the systemic vasculature. In this case, there were multiple risk factors present. The high amount of air insufflation administered and the ulcerative lesion are two of the immediate possibilities. However, two other risk factors can be added. Hopkins *et al*^[29] found that 47% of patients with chronic liver disease possess an intrapulmonary right to left shunt. The other possibility is the patient's history of biliary atresia. A previous study showed that 18 of 88 biliary atresia patients, a total of 9.1%, between the ages of 8 mo and 16 years old possessed an intrapulmonary shunt; this shunt can cause fatal complications in previously operated biliary atresia patients^[9].

Reported air embolism cases following colonoscopy and sigmoidoscopy

Chorost *et al*^[21] reported on a case of a routine screening colonoscopy. Three days after the procedure, the patient presented with severe, lower back pain. After a CT scan of the abdomen, it became evident there was air anterior to the lumbar vertebrae. Batson^[30], in 1940, suggested that an increase in intraabdominal pressure could allow venous effluent from the pelvis to enter into unimpeded valveless venous channels, such as in this example, the vertebrae. Therefore in this case, it was proposed the combination of the high intraabdominal pressure along with the low intraluminal pressure system of the vertebral venous system provided an optimum pressure gradient for air to seep, causing an air embolism.

Mittnacht *et al*^[31] reported the only sigmoidoscopy case known to be complicated by an air embolism. The patient had a history of long-standing Crohn's disease and 2 years status post left partial colectomy with de-

Table 1 Reported cases on air embolism complicated endoscopic retrograde cholangiopancreatography

Case	Ref.	Age/sex	Risk factor(s)	Diagnosis	Outcome
1	Bisceglia <i>et al</i> ^[7]	78/male	Surgical gastroduodenal resection	Pulmonary air embolism	Dead
2	Rabe <i>et al</i> ^[12]	87/male	Metal stent placement	Cerebral air embolism	Survived
3	Rabe <i>et al</i> ^[12]	54/male	Billroth II operation, Metal stent placement	Cardiac air embolism	Dead
4	Jow <i>et al</i> ^[38]	65/male	Biliary duct stones/inflammation	Cardiac air embolism	Dead
5	Maccarone <i>et al</i> ^[11]	45/male	Percutaneous transhepatic biliary drainage	Cerebral air embolism	Survived
6	Siddiqui <i>et al</i> ^[37]	43/female	Biliary sphincterotomy, liver biopsy	Venous air embolism	Dead
7	Nayagam <i>et al</i> ^[39]	57/male	-	Cerebral air embolism	Dead
8	Kennedy <i>et al</i> ^[8]	63/female	Biliary sphincterotomy	Venous air embolism	Dead
9	Stabile <i>et al</i> ^[6]	65/male	Biliary sphincterotomy, PTC	Cerebral air embolism	Dead
10	Mohammedi <i>et al</i> ^[4]	27/male	Biliary sphincterotomy, blunt hepatic trauma	Cardiac air embolism	Survived
11	Romberg ^[40]	53/male	Biliary duct stones	Cardiac air embolism	Survived
12	Rangappa <i>et al</i> ^[41]	50/female	Biliary duct stones	Cerebral air embolism	Dead
13	Bechi <i>et al</i> ^[33]	79/female	Biliary sphincterotomy	Cerebral air embolism	Survived
14	Goins <i>et al</i> ^[16]	72/female	Cholangiocarcinoma	Cerebral air embolism	Survived
15	Cha <i>et al</i> ^[42]	50/female	Biliary duct stones, liver abscesses, choledochoduodenostomy	Cardiac air embolism	Dead
16	Di Pisa <i>et al</i> ^[13]	8/male	Splenoenteric portal shunt	Venous air embolism	Survived
17	Giuly <i>et al</i> ^[43]	60/female	Biliary sphincterotomy, choledochal varices	Venous air embolism	Survived
18	van Boxel <i>et al</i> ^[44]	82/male	-	Cerebral air embolism	Survived
19	Tan <i>et al</i> ^[45]	82/female	Metal stent placement	Cerebral air embolism	Dead
20	Nern <i>et al</i> ^[46]	58/female	Cholangiocarcinoma	Cerebral air embolism	Dead
21	Simmons ^[47]	Not available	Biliary sphincterotomy	Venous air embolism	Survived
22	Merine <i>et al</i> ^[48]	39/female	Biliary sphincterotomy	Venous air embolism	Survived
23	Barthet <i>et al</i> ^[49]	31/male	Biliary sphincterotomy	Venous air embolism	Survived
24	Efthymiou <i>et al</i> ^[11]	62/female	Cholangioscopy	Cerebral air embolism	Survived
25	Our case ^[50]	66/male	Metal stent placement	Cerebral air embolism	Dead
26	Our case ^[50]	51/female	Status post Whipple's operation	Spinal air embolism	Survived

PTC: Percutaneous transhepatic cholangiography.

scending colostomy, which was complicated by poor wound healing and fistula formations. The patient required the sigmoidoscopy before a revision of her previous abdominal bowel surgery. During the procedure, the patient went into cardiac arrest as a result of an air embolism. The history of Crohn's disease, which led to inflamed and deteriorated mucosa, was proposed to allow air entry. Also proposed, there was possible injury to hemorrhoidal veins during the biopsy of the sigmoid. An additional risk was the patient being in Trendelenburg position, allowing for the surgery site to be above the heart.

Reported air embolism cases following EUS

Pfaffenbach *et al*^[32] reported about a patient with severe upper abdominal pain requiring an EUS for evaluation of a pancreatic head lesion. EUS-guided fine needle aspiration was performed. Hepatic portal venous gas was found on a follow up abdominal ultrasonography.

Reported air embolism cases following ERCP

Most described cases of endoscopy-related air embolism have been related to an ERCP. We recently reported two air embolism cases following an ERCP, one with an intracranial air embolism and one with a spinal air embolism. To date, a total of 26 cases of systemic air embolism complicating ERCP have been reported (Table 1). Described risk factors for an air embolism following an ERCP are previous interventions or surgeries of the bile duct system, transhepatic portosystemic shunts, per-

cutaneous transhepatic biliary drains, blunt or penetrating trauma to the liver, sphincterotomy, metal stent placement, the inflammation of the bile duct or surrounding veins, hepatic abscesses or tumors, liver biopsy, and insufflation of air with high pressure. Cholangioscopy with air insufflation directly into the bile duct appears to be a particularly strong risk factor for an air embolism. Reported clinical presentations are cardiovascular, pulmonary, and neurological symptoms. Again, we want to emphasize the onset of symptoms or symptom escalation with change of patient position from prone to supine should immediately trigger suspicion for an air embolism.

DIAGNOSIS

The diagnosis of an air embolism is often difficult and is complicated by the fact that air may be rapidly absorbed from the circulation while diagnostic tests are being arranged. Exclusion of other life-threatening processes is generally required.

Transthoracic and transesophageal echocardiography have been used to document the presence of air and may show evidence of acute right ventricular dilation and pulmonary artery hypertension consistent with air embolism. An echocardiography also aids in the diagnosis of cardiac anomalies, assessment of volume status, and cardiac contractility; this allows exclusion of other causes of hypotension, dyspnea, and aiding in further patient management. End-tidal CO₂ monitoring may show a fall in end-tidal CO₂; however, this finding is nonspecific and also

occurs with pulmonary embolism, massive blood loss, circulatory arrest, and disconnection from the anesthesia circuit. The pulmonary artery catheter may show a rise in pulmonary artery pressure in venous air embolism, but this is a nonspecific finding. Ventilation-perfusion scan abnormalities may be seen in the setting of a massive air embolism, but this is also a nonspecific finding and the perfusion defects resolve rapidly. The chest CT can detect air with higher sensitivity for massive air emboli. A pulmonary angiography could also be useful but may be normal often times in patients who have suffered an air embolism because of rapid resorption of air.

MANAGEMENT

The most crucial step in patient management is to maintain a high index of suspicion for an air embolism. An air embolism should be included in the differential diagnosis of procedural or periprocedural cardiopulmonary instability and neurologic symptoms, particularly in patients with recognized risk factors. Since the clinical presentation of an air embolism can significantly overlap with sedation-related problems and ischemic or hemorrhagic cerebrovascular events, some simple maneuvers to decrease the impact of a potential air embolism should be promptly initiated while the definitive diagnosis is established. These maneuvers include: (1) immediately stop the procedure if at all possible; (2) administer high flow 100% oxygen, which can reduce air bubbles expansion; (3) initiate high volume normal saline infusion; (4) place the patient in Trendelenburg (feet higher than the head) and left lateral decubitus position in order to minimize air migration to the brain and to force-out air from the right ventricular outflow tract^[33], thereby increasing venous return^[23]; and (5) if N₂O is being used, it must be discontinued because of its ability to rapidly diffuse into the trapped air bubbles, causing an additive effect on the embolism^[23].

After these initial stabilizing measures are implemented, which should take no more than a few minutes, a decision has to be made regarding the type of evaluation needed to secure the diagnosis. This is a crucial branching point in the management of these patients. Since cerebrovascular accident is most commonly suspected in patients with neurologic symptoms, arrangements for an urgent head CT scan are typically made. If the underlying problem is an air embolism, the patient being sent for a CT scan can have some serious, adverse consequences, because it will delay the diagnosis and the application of specific targeted therapy. Therefore if an air embolism is suspected, a bedside echocardiogram should be promptly performed to quickly secure the diagnosis with visualization of air within the right heart. This can have immediate therapeutic implications. An air aspiration *via* a central venous catheter can be done, and arrangements for urgent hyperbaric oxygenation therapy can be carried out. Hyperbaric oxygenation therapy may reduce air bubble size, accelerate nitrogen reabsorption, and increase the

oxygen content of arterial blood; this potentially reduces the ischemia. In the event of circulatory collapse, cardiopulmonary resuscitation (CPR) should be initiated in order to maintain the cardiac output. CPR may also serve to break large air bubbles into smaller ones and force air out of the right ventricle into the pulmonary vessels.

PROPHYLACTIC MEASURES TO DECREASE THE RISK OF AIR EMBOLISM

Using CO₂ for insufflation instead of air can eliminate the risk of an air embolism, because CO₂ can be easily absorbed^[34]. The use of CO₂ for insufflation during gastrointestinal endoscopy has been shown superiority than using room air by multiple randomized controlled trials and a meta-analysis^[35,36]. It was associated with a decreased postprocedural pain, flatus, and bowel distension. CO₂ insufflation also appears to be safe in patients without severe underlying pulmonary disease. This finding supports the use of CO₂ in most cases if available. In our unit, we perform all endoscopies with CO₂; if CO₂ is not available for routine use, we believe it must be used in all cholangioscopy cases or when other risk factors are present.

Another option for patients at risk is to use a precordial Doppler probe monitor during the procedure; it can quickly detect air within the heart and pulmonary vasculature before clinical symptoms may appear^[37].

CONCLUSION

In summary, endoscopists should be aware of the signs and symptoms of an air embolism. In patients with risk factors, prophylactic measures can be applied. A high index of suspicion for an air embolism should be maintained, because prompt recognition can allow timely administration of specific, potential life-saving therapy.

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