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Case Report

Unexpected cardiovascular collapse from massive air embolism during endoscopic retrograde cholangiopancreatography

K. M. Goins[1], J. M. May[1], C. Hucklenbruch[1,2], K. E. Littlewood[1] and D. S. Groves[1]
[1]*Department of Anaesthesiology, University of Virginia, Charlottesville, VA and* [2]*Department of Anaesthesiology and Intensive Care Medicine, University Muenster, Muenster, Germany*

A 72 year-old woman with cholangiocarcinoma presented for endoscopic retrograde cholangio pancreatography (ERCP) for diagnostic intraductal endoscopy under GETA. During the technically difficult procedure the patient became suddenly hypoxic, hypotensive, bradycardic, and progressed to PEA code (ETCO2 5 mmHg). ACLS was initiated. Transesophageal echo demonstrated massive right heart air accumulation; abdominal X-Ray showed air filled bile ducts. Central access was obtained, a pulmonary artery catheter floated, and 30 ml of air aspirated from the RV. Within 5 minutes pulses returned; the patient was transferred to the ICU. MRI revealed two watershed infarcts in the right frontal lobe. The patient fully recovered and returned a month later for an uneventful ERCP.

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Case report

Air embolism is a rare and potentially fatal complication of endoscopic retrograde cholangiopancreatography (ERCP) only noted in a few case reports in the literature. We present a case of a 72-year-old woman with unresectable cholangiocarcinoma undergoing outpatient ERCP for diagnostic intraductal endoscopy using the SpyScope (Boston Scientific Corp., Natick, MA) under general anesthesia. Induction of anesthesia and intubation were uneventful (150 mg propofol, 75 mg lidocaine, 5 mg rocuronium, 60 mg succinylcholine). The patient was breathing spontaneously and was hemodynamically stable (blood pressure 130/60 mmHg, heart rate 75 b.p.m.). Approximately 20 min later, during a technically difficult period of the procedure, end tidal carbon dioxide level was noted to decline rapidly from 50 to 5 mm Hg during spontaneous ventilation. Subsequently, the patient became hypoxic (SpO_2 ~ 50%), followed by hypotension, bradycardia, and finally a pulseless electrical cardiac arrest. Airway pressures during manual and then controlled ventilation were increased (peak pressures 40 mmHg). The procedure was aborted and cardiopulmonary resuscitation (CPR) was initiated using the ACLS algorithm. Simultaneously, a radial arterial line was placed for invasive blood pressure monitoring. Because all clinical signs suggested a massive pulmonary embolism, a transesophageal echo (TEE) probe was placed. Surprisingly, it showed a large amount of trapped air within the right heart consistent with massive air embolism. Central access (MAC™ introducer sheath, Arrow, Reading, PA) was obtained via the femoral vein under continued CPR, and a pulmonary artery catheter (PAC) (Swan-Ganz VIP, Baxter, Irvine, CA) was placed into the right ventricle. It was possible to aspirate approximately 30 ml of air under echo guidance. After approximately 5 min, the patient regained a spontaneous circulation and was transferred to the medical ICU. Weaning from mechanical ventilation was uneventful and the patient was extubated the following day. Afterwards, the patient presented with short-term memory problems and deficits in gross and fine motor function. A follow-up brain MRI 3 days later showed two small foci of

acute infarct in the right superior frontal lobe most likely consistent with a watershed infarct due to decreased cerebral perfusion during her cardiac arrest. However, the patient fully recovered without neurological sequelae and was discharged to an acute rehabilitation facility 6 days later, where she stayed for another week. Since then, she returned twice in a stable condition for completely uneventful follow-up ERCP laser treatments as an outpatient.

Discussion

The risk of venous air embolism is increased in procedures above the heart level, but it can also occur during procedures using high-pressure insufflation of air. Although venous air embolism is an extremely rare complication of ERCP, it can potentially have fatal consequences. It is such a rare event that it is often not suspected and recognized until it is too late for successful treatment.

Lowdon and Tidmore[1] first described a fatal air embolism in 1988 in a case report of a 4-month-old infant undergoing gastroscopy after a Kasai procedure. During the procedure it was thought that air was introduced into the hepatic veins just below the enterostomy site. The air embolism was later confirmed on autopsy. Katzgraber et al. describe a case of a 56-year-old man also undergoing gastroscopy for epigastric pain secondary to a gastric ulcer. During the procedure, he suffered from a cardiac arrest, and on autopsy, there was evidence of a massive air embolism in the right ventricle and an open blood vessel was noted at the base of the ulcer. They then performed tests with a commercial gastroscope to show that overpressures of up to 45 kPa can be generated within seconds without the ability to drain the air. They suggest that, if an open vessel is also present, this is enough pressure to produce an air embolism.[2]

There are several case reports of air embolism during ERCP. In 1997, Kennedy et al.[3] reported a case of a 63-year-old woman undergoing ERCP for choledocholithiasis. During the endoscopic sphincterotomy, the patient developed a rapid and fatal cardiac arrest secondary to a hepatic venous air embolism. Mohammedi et al.[4] presented a case of a 27-year-old man who suffered from a hepatic injury after a blunt abdominal trauma, which required ERCP to identify the site of a bile leak. A massive air embolism complicated the sphincterotomy. This was thought to be the result of gas entering the lacerated hepatic veins during continued insufflation. Early identification and management of the problem prevented any serious sequelae and the patient survived. Siddiqui et al.[5] report another case of fatal air and bile pulmonary embolism in a 43-year-old woman undergoing percutaneous liver biopsy and ERCP. Finally, Stabile et al.[6] discuss a case of fatal paradoxical cerebral air embolism in a 65-year-old man after ERCP in the absence of a patent foramen ovale or a septal defect. They hypothesize that in the presence of massive air embolism, the pulmonary circulation may be unable to filter out all the venous gas such that it may mass to the arterial side.

In the case we present here, the patient survived without serious neurological or cardiopulmonary sequelae due to prompt diagnosis and management of the problem. The procedure had been technically difficult and it is possible that a small vessel injury occurred. Probably, due to the insufflation pressure, the source of bleeding was never visible. It is likely that air was introduced into the bile ducts and into the venous system (portal venous system, hepatic veins, inferior vena cava, and ultimately into the right heart). X-ray images taken during the procedure show contrast initially filling the bile ducts, followed by air in the bile ducts (Fig. 1).

Because our patient presented with classic symptoms of pulmonary embolism, we initiated CPR and additionally placed a TEE probe, which quickly confirmed the diagnosis (Fig. 2). Because of technical obstacles to store the images during the resuscitation we were only able to retrieve a few images afterwards. These images were taken in the middle of the resuscitation, and do not appreciate the full amount of entrapped air. Basically, the complete right heart was full of air and appeared white on echo. A few bubbles were seen in the left atrium and ventricle after the successful resuscitation (images also not available). We also used the TEE to guide the PAC into the right ventricle and close to the air pockets because the traditional floating technique was very limited under CPR. Using this technique, we were able to aspirate about 30 ml of air through the distal lumen of the PAC. The treatment of venous air embolism by aspiration from central venous catheters is well established, and the effectiveness of air aspiration through central lines using different catheters (single-orifice vs. multi-orifice) and techniques (stationary vs. pulling through) and positioning of the catheter (right atrium vs. right ventricle vs. pulmonary artery) has been studied in the literature.

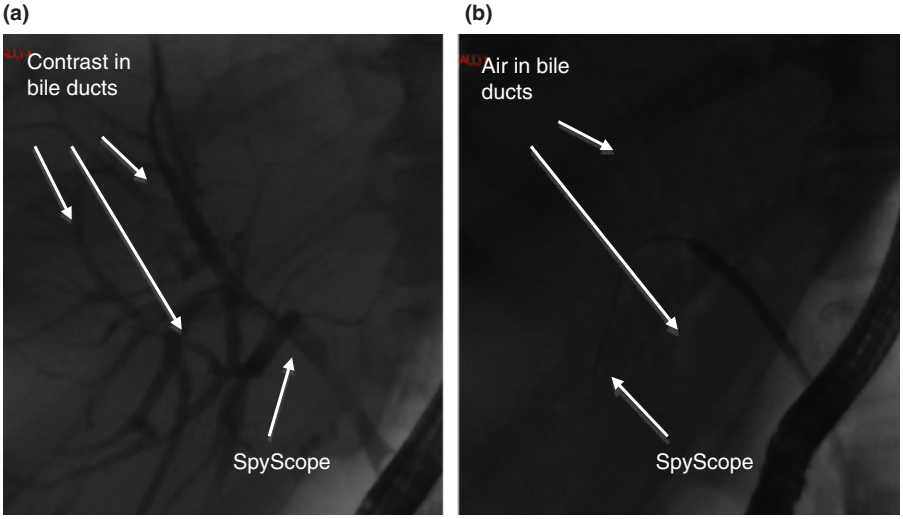
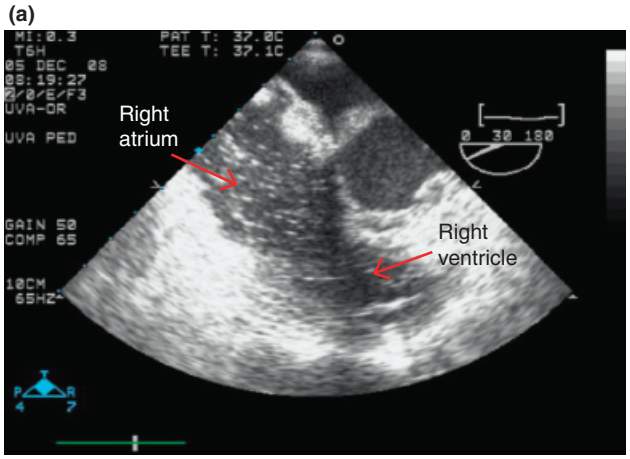
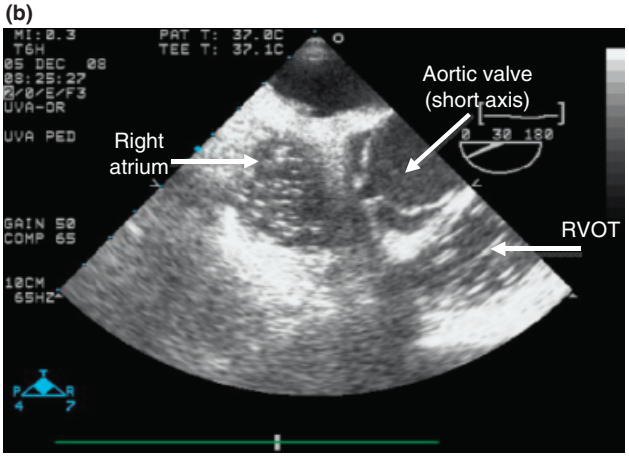


Fig. 1. Bile ducts filled with contrast medium (A) and with air (B); SpyScope in situ.



Entrapped air shown in right atrium and right ventricle



Entrapped air shown in right atrium
and right ventricular outflow tract (RVOT)

Fig. 2. (A) Entrapped air shown in the right atrium and the right ventricle (B) Entrapped air shown in the right atrium and the right ventricular outflow tract (RVOT).

In addition, the procedure table can be placed in the Trendelenberg position to reduce the risk of the cerebral embolism. This was, however, not possible in our case. Ultimately, if necessary, an open cardiac massage or cardiopulmonary bypass may be required to support these patients; however, it can be difficult to set up emergently in off-site procedure areas away from the main operating room.

Because the patient fully recovered after this event and the tumor was not surgically resectable, the patient decided to return for further palliative laser treatment under general anesthesia. It was initially not clear whether potentially an anatomical alteration and/or fragile vessels supplying the tumor played a major role in the occurrence of this event. In preparation for these upcoming visits, we discussed options to be prepared for a potential recurrent air embolism. However, after extensive discussion with the gastroenterologists we came to the conclusion that the technically difficult nature of the procedure led to the air embolism. Because this specific-endoscopic procedure was not necessary for the laser treatment, we decided to proceed without pre-procedural placement of invasive monitoring.

Venous air embolism may be an unsuspected and rare complication of many gastroenterology procedures including ERCP. It is important to recognize that it may rapidly lead to a fatal cardiac arrest if not diagnosed and treated quickly. During a technically complicated procedure, communication between the anesthesiologist and the gastroenterologist is essential. Warning signs of impending cardiovascular collapse may be subtle, but sudden decrease in end tidal carbon dioxide, hypoxia, bradycardia,

hypotension, and arrhythmia should be warning signs that the patient may be suffering from an air embolism and that fatal consequences may occur if not managed appropriately.

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Address:
Danja S. Groves
Department of Anaesthesiology
University of Virginia
PO Box 800710
Charlottesville, VA 22908-0710
USA
e-mail: dgroves@virginia.edu