



(CASE REPORT)



CARDIAC ARREST ASSOCIATED WITH AIR EMBOLISM DURING ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY

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ABSTRACT

Air embolism is an uncommon but potentially fatal complication of therapeutic endoscopic retrograde cholangiopancreatography (ERCP). We report the case of a 78-year-old woman with arterial hypertension who underwent ERCP for suspected choledocholithiasis under general anesthesia with tracheal intubation and air insufflation. Preprocedural magnetic resonance cholangiopancreatography showed cholelithiasis with dilatation of the common bile duct. Approximately 20 minutes after anesthetic induction, during attempted sphincterotomy, she developed abrupt hemodynamic collapse with an end-tidal carbon dioxide (ETCO₂) decrease to approximately 15 mmHg, followed by asystolic cardiac arrest. Cardiopulmonary resuscitation included chest compressions, intravenous epinephrine, ventilatory adjustment, central venous access, and norepinephrine. Intra-arrest transesophageal echocardiography was interpreted by the treating team as demonstrating intracardiac air, supporting the diagnosis of air embolism. Return of spontaneous circulation was achieved after approximately 40 minutes. After initial stabilization and extubation, the patient developed massive upper gastrointestinal bleeding with refractory hemorrhagic shock and died. This case highlights the importance of considering air embolism when sudden hypotension and a marked ETCO₂ decrease occur during therapeutic ERCP, and illustrates the diagnostic value and reporting limitations of intra-arrest echocardiography.

Keywords: Air embolism, Endoscopic retrograde cholangiopancreatography (ERCP), Cardiac arrest, Capnography, Transesophageal echocardiography

1 INTRODUCTION

Endoscopic retrograde cholangiopancreatography (ERCP) is an established intervention for biliary obstruction and choledocholithiasis. Although pancreatitis, bleeding, perforation, and infection dominate discussions of procedural complications, intravascular gas embolism can produce abrupt cardiovascular collapse. Its recognition is particularly difficult during general anesthesia, when neurological symptoms cannot be elicited and circulatory deterioration may initially be attributed to more common anesthetic or procedural causes [1,2].

The frequency of embolism depends on how it is detected. In a prospective study of 843 ERCP procedures, Afreen et al. identified venous air embolism in 20 procedures (2.4%) using precordial Doppler monitoring; 10 events were associated with hemodynamic changes. All detected events occurred during procedures involving therapeutic interventions. These results describe Doppler-detected embolization in a monitored cohort and must not be interpreted as the incidence of massive embolism or cardiac arrest [1].

We report abrupt circulatory collapse during attempted sphincterotomy with air insufflation. A marked fall in end-tidal carbon dioxide preceded cardiac arrest, and intra-arrest transesophageal echocardiography (TEE) was interpreted as showing intracardiac air. The case illustrates recognition under general anesthesia, prolonged resuscitation, and a subsequent fatal hemorrhagic event.

2 CASE PRESENTATION

A 78-year-old woman with arterial hypertension treated with an angiotensin-converting enzyme inhibitor was admitted for ERCP for suspected common bile duct lithiasis. Preprocedural magnetic resonance cholangiopancreatography (MRCP) showed cholelithiasis with dilation of the common bile duct. Preanesthetic laboratory testing demonstrated hepatic cytolysis, with aminotransferase levels approximately twice the upper limit of normal, associated with biochemical cholestasis; the remainder of the preanesthetic evaluation was unremarkable. General anesthesia was induced with fentanyl 200 µg, propofol 140 mg, and rocuronium 50 mg, followed by tracheal intubation. Air, rather than carbon dioxide, was used for endoscopic insufflation.

Approximately 20 minutes after anesthetic induction, during attempted sphincterotomy, abrupt cardiovascular instability developed with an ETCO₂ decrease to approximately 15 mmHg. Periprocedural monitoring showed stable values from 12:15 to 12:30, with heart rate 61–69 beats/min, SpO₂ 99%, and a noninvasive blood pressure of 114/75 mmHg at 12:27. Hypotension was then documented at 69/51 mmHg (mean 57 mmHg) at 12:33. At 12:35, heart rate was 150 beats/min and SpO₂ 79%; by 12:40, the treating team also reported a transient blood pressure of approximately 60/40 mmHg and heart rate of approximately 150 beats/min, and jugular venous distension (Figure 1) followed about five minutes later by asystolic cardiac arrest. The lowest displayed SpO₂ was 25% at 12:50.



Figure 1: Two views of a prominent superficial cervical vein in the intubated patient, compatible with the clinically observed jugular venous distension during the hemodynamic event

External chest compressions were initiated immediately after recognition of asystolic cardiac arrest. Epinephrine 1 mg was administered intravenously every 5 minutes during resuscitation; ventilatory settings were adjusted, a central

jugular venous catheter was inserted, and norepinephrine infusion was initiated. TEE performed during resuscitation was interpreted by the treating team as demonstrating intracardiac air, supporting the diagnosis of air embolism. No still image or video from the examination was archived. Return of spontaneous circulation was achieved after approximately 40 minutes of resuscitation. At 13:30, monitoring showed an organized ECG rhythm, heart rate 86 beats/min, SpO₂ 92%, and invasive arterial pressure 175/102 mmHg (mean 126 mmHg), together with a capnogram and a low-ETCO₂ alarm (Figure 2).

Figure 2. Later Monitor and Capnography Snapshot



Figure 2: The bedside monitor clock displays 13:30, with heart rate 86 beats/min, SpO₂ 92%, and invasive arterial pressure 175/102 mmHg (mean 126 mmHg). An organized ECG tracing and arterial waveform are visible. The anesthesia workstation displays a capnogram and a low-ETCO₂ alarm.

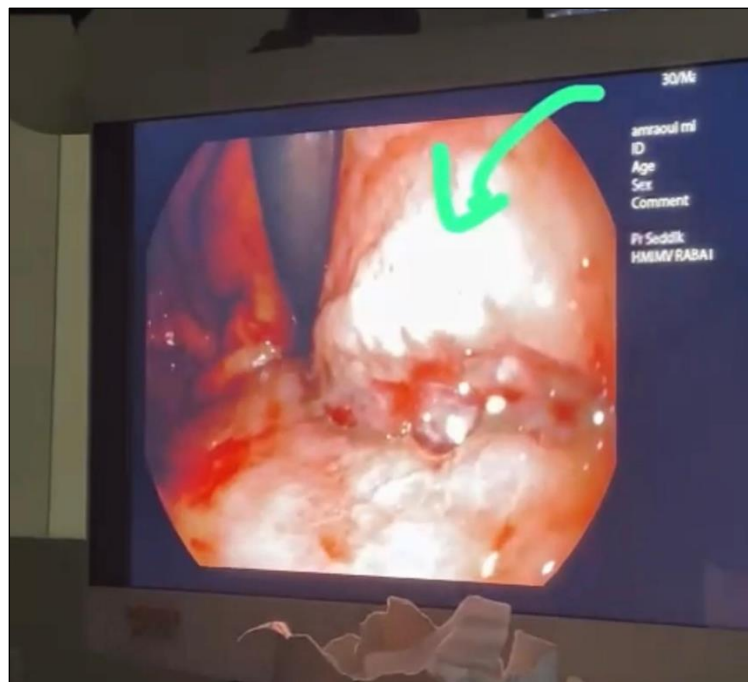


Figure 3: Emergency upper gastrointestinal endoscopy during hemorrhagic deterioration.

After return of spontaneous circulation, vasoactive requirements decreased and the patient was transferred to the intensive care unit. Extubation was recorded as H12. Massive upper gastrointestinal bleeding occurred at H17,

prompting transfusion and emergency endoscopy, which demonstrated active gastric bleeding (Figure 3). Durable hemostasis was not achieved.

Refractory hemorrhagic shock developed despite transfusion, vasoactive support, endoscopic intervention, and further resuscitation. Death was recorded as H2O.

3 DISCUSSION

The reported incidence of air embolism during ERCP varies markedly according to how events are detected. Clinically recognized events are exceptionally uncommon. In an analysis of the U.S. National Inpatient Sample covering 1998–2013, Olaiya and Adler reported air embolism after ERCP at a rate of 3.32 per 100,000 procedures. This estimate was based on coded inpatient diagnoses and therefore reflects clinically apparent events rather than all episodes of intravascular air entry [10].

By contrast, active surveillance identifies a substantially higher frequency of subclinical or less severe embolization. In the prospective study by Afreen et al., precordial Doppler monitoring detected venous air embolism in 20 of 843 ERCP procedures (2.4%); 10 of these events were associated with hemodynamic changes, and all occurred during therapeutic interventions [1]. The large difference between administrative data and prospective Doppler surveillance illustrates the dependence of measured incidence on the sensitivity of the detection method. Small or transient embolic events may therefore go unrecognized in routine practice, whereas massive clinically significant air embolism leading to cardiovascular collapse or cardiac arrest remains rare and its precise incidence is not established [1,3,10].

For insufflated air to enter the circulation, an abnormal communication with a vessel and a favorable pressure gradient are required. Biliary or periampullary instrumentation may disrupt small venous channels; inflammatory injury, fistulous communications, and previous biliary interventions are other reported settings. Potential routes include direct venous entry and pathways involving the portal or hepatic circulation. None of these anatomical routes was demonstrated in this patient [3].

The close temporal relationship between attempted sphincterotomy and collapse supports a procedure-related mechanism, but does not prove that the sphincterotomy created a fistula. Air insufflation provided a plausible gas source. Whether air entered at the papillary, ductal, or another vascular interface remains uncertain. Similarly, the description of an attempted sphincterotomy does not establish the extent of tissue incision or guidewire manipulation.

Experimental measurements by Wardhan et al. showed that endoscopy equipment can generate pressures exceeding typical venous pressures in a model system. This supports the plausibility of pressure-driven intravasation, but bench measurements cannot establish the pressure present in an individual patient. No intravascular gas volume was available in this case. The term “massive” therefore describes the clinical severity, including profound shock and arrest, rather than a measured volume of air [4].

A large venous gas burden may impair right ventricular ejection and obstruct the pulmonary circulation, increasing right ventricular afterload and reducing left ventricular filling. Pulmonary perfusion and transport of metabolically produced CO₂ to the alveoli then decrease. A sudden fall in ETCO₂ can consequently accompany profound hypotension even without an initial change in minute ventilation. This mechanism offers a coherent explanation for the reported combination of hypotension and reduced ETCO₂ before arrest [2,3].

Capnography is nevertheless a warning signal, not a gas-specific diagnostic test. Acute pulmonary thromboembolism, severe blood loss, anaphylaxis with circulatory failure, tension pneumothorax, and major cardiac dysfunction may produce a similar pattern. Airway displacement, circuit disconnection, and sampling problems must also be considered [5]. In this case, ETCO₂ decreased to approximately 15 mmHg during the initial hemodynamic collapse. The later monitor and workstation image (Figure 2) also documents a low-ETCO₂ alarm after restoration of an organized circulation.

Sisk et al. described two ERCP-associated cardiac arrests in which TEE demonstrated substantial gas in the right heart, illustrating the potential value of intra-arrest echocardiography in this setting [6]. TEE can also assess ventricular filling and function and identify alternative causes of circulatory collapse. When imaging is archived, clinically useful reporting should specify the chambers containing gas, right ventricular size and systolic function, septal position, left ventricular filling, and any evidence of left-sided gas or a right-to-left shunt [6].

The clinical TEE interpretation supports air embolism, but the absence of archived images prevents independent confirmation of the chamber distribution or ventricular response. We therefore avoid classifying the event as

exclusively venous or arterial or inferring a patent foramen ovale. Procedural timing, pulmonary hypoperfusion reflected by the ETCO₂ decrease, and the clinical TEE finding form the diagnostic argument.

Air is predominantly nitrogen and is less readily absorbed than CO₂. The faster absorption of CO₂ provides a physiological rationale for its use in endoscopy and may limit persistence of intravascular gas. The ASGE technology review describes potential benefit in procedures carrying a risk of gas embolism. These considerations support CO₂ as a risk-reduction strategy, but do not quantify the reduction in catastrophic embolism or establish that its use would have prevented this patient's arrest [7].

Chuang et al. reported a fatal gas embolism during ERCP performed with CO₂ insufflation. Gas solubility therefore does not remove the danger when entry into the vasculature is substantial. Gas choice should be considered alongside avoidance of unnecessary insufflation and recognition of tissue or vascular injury. In the present case, air was the insufflation gas; its independent contribution to severity cannot be separated from the unmeasured amount and rate of intravascular entry [4,8].

Suspected endoscopic gas embolism calls for immediate interruption of insufflation and the procedure while airway, breathing, circulation, and reversible causes of arrest are addressed. Echocardiography should not compromise chest compressions. Current adult advanced life support guidance supports epinephrine 1 mg every 3–5 minutes during cardiac arrest [3,5]. In this case, epinephrine 1 mg was administered every 5 minutes during resuscitation.

The 2025 American Heart Association guidance recommends 100% oxygen for life-threatening gas embolism and considers hyperbaric oxygen reasonable in affected adults; consideration is not restricted to documented cerebral arterial embolism. Early specialist discussion should account for clinical severity, stability, transport, and availability. Aspiration may be considered when a life-threatening right ventricular gas collection is localized and accessible. Positioning on a level surface is advised; during CPR, the supine position permits effective compressions. Routine head-down positioning has uncertain benefit and should not interfere with resuscitation [9].

The prolonged resuscitation in this case was followed by return of spontaneous circulation after approximately 40 minutes, demonstrating the potential reversibility of severe air-embolism-associated cardiovascular collapse when effective cardiopulmonary resuscitation and circulatory support are maintained.

The patient was extubated at H12 after initial stabilization. The terminal event was refractory hemorrhagic shock after massive upper gastrointestinal bleeding. Emergency upper gastrointestinal endoscopy directly documented active gastric bleeding (Figure 3), supporting hemorrhage as the terminal clinical event. The available endoscopic image does not, however, establish the precise pathological nature of the gastric lesion or a causal relationship with the preceding ERCP, prolonged resuscitation, ischemic injury, TEE, or a pre-existing gastric lesion. In particular, the bleeding should not be relabeled as post-sphincterotomy hemorrhage without evidence of a papillary source which was inaccessible.

Important limitations include the absence of archived TEE images or video, which prevents retrospective assessment of chamber-specific gas distribution. The monitor trend provides intermittent rather than continuous values during the acute event. Although the later endoscopic image documents gastric bleeding, it does not establish the exact pathological nature or cause of the lesion.

4 CONCLUSIONS

During therapeutic ERCP, abrupt hypotension accompanied by a marked ETCO₂ decrease should prompt immediate consideration of air embolism and other causes of acute pulmonary hypoperfusion. In this patient, TEE was documented by the treating team as showing intracardiac air during resuscitation and supported the clinical diagnosis, although no echocardiographic image or video was archived for retrospective review. Initial return of circulation after prolonged CPR was followed by a distinct fatal hemorrhagic complication.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Disclosure of conflict of interest

The authors declare no conflicts of interest regarding this manuscript.

Statement of ethical approval

This case report describes routine clinical care and involved no research-specific intervention. No formal ethics committee approval is reported.

Statement of informed consent

Written informed consent for publication has not been documented. As the patient is deceased.

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