# A dreaded procedural complication during endoscopic retrograde cholangiopancreatography

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## ABSTRACT

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Endoscopic retrograde cholangiopancreatography (ERCP) procedure has gained popularity and has become the method of choice for diagnostic and therapeutic modalities. But there is a concern about albeit concerns about the dreaded serious complications that can be a threat to the life of subjects. We, herein, report a case of air embolism. A 30-year-old lactating woman presented with nausea, vomiting, and epigastric pain for 2 weeks with deranged liver function tests. Computed tomography abdomen was suggestive of gallbladder calculus with the prominent common bile duct. She was electively posted for the ERCP procedure. During the course of procedure, untoward complication was encountered, and with transthoracic echocardiography and fluoroscopy, diagnosis of air embolism was more or less confirmed. In spite of our tremendous resuscitative maneuvers, the condition deteriorated and later succumbed to death.

Key words: Air embolism, AV block, Complications, Endoscopic retrograde cholangiopancreatography, Temporary pacing

Ithough a commonly performed intervention, endoscopic retrograde cholangiopancreatography (ERCP) poses a greater challenge to the surgeon as well as the anesthesiologist, and it is associated with a substantially increased risk of morbidity and mortality [1]. Incidence of air embolism is very rare, only 51 cases of air embolism reported following the ERCP, according to the literature search [2]. It can present as cardiopulmonary instability and neurological symptoms [3]. Hence, it is important to understand and watch for potential complications during such procedures.

In the context of rarity and significant mortality of air embolism, we, hereby, report a case of common bile duct (CBD) calculus, for which ERCP was performed with all precautions and standard protocols but still resulted in air embolism causing death in spite of all possible supportive measures. Considering the rapid worsening of the patient's status, it is very painful and difficult to accept the reality from the patient's relative aspect also.

## CASE REPORT

A 30-year-old lactating female presented with complaints of nausea, vomiting, and epigastric pain for 2 weeks duration. There was no history of any comorbidity in the past. On examination, her vitals were stable with a pulse rate of 80/min regular and blood pressure (BP) of 130/70 mmHg. Physical examination revealed tenderness in the right hypochondrium and epigastrium.

Investigation revealed elevated total bilirubin of 3 mg% with direct bilirubin 1.8 mg% with Serum Glutamic Oxaloacetic Transaminase and Serum Glutamic Pyruvic Transaminase within normal limits. Her serum amylase and lipase were normal. Ultrasonography abdomen showed evidence of fatty hepatomegaly. Computed tomography scan abdomen showed dilated CBD with gallbladder calculus and hepatomegaly with fatty infiltration. Electrocardiography (ECG) showed sinus bradycardia.

On the very next day, she was posted electively for the ERCP procedure. After explaining about the procedure, she was premedicated with intravenous midazolam 1 mg, glycopyrrolate 0.2 mg, and ondansetron. Then, she was taken into the endoscopy room, and pulse oximeter, ECG, and non-invasive BP were attached. Intravenously, fentanyl 100 mcg was administered and pre-oxygenated with 100%  $O_2$  for 3 min. A priming dose of atracurium was given followed by intravenous propofol 150 mg and 30 mg atracurium. Patient was ventilated for 2 min and trachea was intubated with a high volume low pressure 7 mm cuffed endotracheal (ET) tube. She was maintained with 50%  $O_2$  in N<sub>2</sub>O (nitrous oxide) and sevoflurane 1.5%. The patient was shifted to a prone position after induction. Through side view endoscopy, endoscopist commenced the procedure.

After ½ h of commencement, the patient recovered from the muscle relaxant effect. Patient's heart rate increased to 120/min, and BP twas 180/110 mmHg, then a dose of muscle relaxant was given; Sevoflurane dial concentration was increased from 1.5%

to 2%. Within 10 min, BP came down to 100/60 mmHg and we noticed a drastic fall in end tidal carbon dioxide (CO<sub>2</sub>) amplitude from 36 mmHg to 21 mmHg; ET tube position was confirmed. BP further dropped to 90/60 mmHg. There was no fall in SpO<sub>2</sub>, it remained at 98%. Intravenous mephentermine, 12 mg was administered at that time. ECG monitor showed sinus arrhythmia, heart rate suddenly dropped to 40/min, and intravenous atropine 0.6 mg was given, notified the surgeon to curtail the procedure.

The patient was shifted from prone to a supine position, chest compression initiated according to the American Heart Association protocol. We called the help of senior anesthesiologist, ventilated the patient with 100%  $O_2$ . Injection adrenaline, 1 mg diluted was given intravenously in increments. Peripheral pulses reappeared, heart rate suddenly rose to 150/min. Following that, the monitor showed ventricular tachycardia, defibrillated twice with 200 joules of shock, and reverted to sinus rhythm but with severe bradycardia with second-degree atrioventricular (AV) block. Inotropic support with noradrenaline and dopamine was started. Cardiologist summoned for help, with the advice from him injection Deriphyllin IV and injection sodium bicarbonate 7.5% slow IV was given. Vitals were stable with inotropic support; intra-arterial BP and the right internal jugular vein were cannulated in the meanwhile, shifted to cath lab on the ventilator.

Bedside, echocardiography (ECHO) and fluoroscopy were done and showed air in the main pulmonary artery with severe left ventricular dysfunction with no evidence of the right-toleft shunt, intermittent high-grade AV block requiring pacing [Video 1]. Arterial blood gas (ABG) analysis noted severe acidosis with hypokalemia, sodium bicarbonate with potassium supplementation was given, and temporary pacing was done. The patient was shifted to post-operative intensive care unit with inotropic support on intermittent positive pressure ventilation (IPPV). Serial ABG and central venous pressure (CVP) monitoring were executed. Despite all these painstaking efforts, the patient's life could not be served.

#### DISCUSSION

Air embolism is a rare and dreaded complication of ERCP [1]. Anesthesiologists often get confused to clinch the diagnosis under the effect of IPPV. Detection of air embolism is perplexing; monitors used for detection should provide a high level of sensitivity and specificity. The conglomerate of a precordial Doppler and expired concentration of  $CO_2$  monitoring is the current standard of care. Transthoracic ECHO is more sensitive than the precordial Doppler and it offers the advantage of detecting right-to-left shunting of air.

The symptoms of air embolism associated with ERCP occur or worsen when the patient is repositioned from prone to supine [1]. The diagnosis is confirmed after visualizing air in superior vena cava, portal vein, hepatic vein, right atrium, right ventricle, left atrium, left ventricle, or brain.

The mechanism of the air embolism occurs due to direct communication intervening air and circulation. Different mechanisms have been elucidated for the cause of air embolism: (1) Intramural dissection of air blown into the portal vein, (2) transection of duodenal veins, (3) fistula, (4) retrograde flow through the superior vena cava, (5) inability of pulmonary circulation to filter gaseous emboli, and (6) portocaval collateral circulation [3]. Other factors that may contribute include through the peripheral or central venous line, prolonged exposure to highpressure blowing air, and damage during biliary stent deployment of biliary dilatation. Rapid egress of air that enters the systemic circulation led to the strain on the right ventricle. This, in turn, leads to cardiovascular compromise that, unfortunately, does not respond to resuscitative maneuvers [4].

The use of CO<sub>2</sub> in place of air for insufflation during ERCP can eliminate the risk of an air embolism. However, there is a possibility for CO<sub>2</sub> embolism, which is not as severe as air as it is easily absorbed [5]. In the aforementioned case, the air was used. The use of N<sub>2</sub>O must be curtailed promptly after even apparently minor air embolism due to the possibility of that air becoming larger in size [6]. Ideally, the presence of the right-to-left shunt should be ruled out in all patients scheduled for ERCP [7]. Insertion of a pulmonary artery catheter with the help of expertise is needed to treat intravascular air [8]. Risk of air embolism during ERCP is high in following conditions such as- previous interventions or surgeries of the bile duct system, transhepatic portosystemic shunts, percutaneous 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, and liver biopsy [3].

One should notify notify the surgeon contiguously to flood or pack the surgical field. Place the patient in lateral decubitus or Trendelenburg position to improve the venous return. Jugular compression and decompression of the stomach with nasogastric suction catheter might help. Supportive treatment with vasopressors and inotropes must be initiated. Hyperbaric oxygen therapy is a method of choice but is not available in all places [9]. Differential diagnoses to be considered during air embolism are acute coronary syndrome, pulmonary embolism, and cardiogenic shock [10].

Wills-Sanin et al. have described a case of a 55-year-old female, with a history of liver transplantation due to Budd-Chiari syndrome, developed air embolism causing cardiopulmonary collapse following ERCP for multiple biliary calculi. Following cardiopulmonary resuscitation, the return of spontaneous circulation was obtained and put on a ventilator and inotropic supports. Later, the patient was recovered [4]. Hauser et al. described another case of sudden death following ERCP in a patient of non-Hodgkin lymphoma with cholestatic jaundice due to gall stones [10]. Lanke and Adler identified 51 cases of air embolism following ERCP, which includes systemic air embolism, venous air embolism, and cerebral air embolism. According to this data out of 51 patients, 25 patients succumbed to the complication, which are 49.02% mortality [2]. A systemic review done by Donepudi et al. shows the data of 26 patients who underwent ERCP developed air embolism and 15 patients died, which accounts for 57.7% [3]. In our case also, all supportive

measures including a temporary pacemaker that was also tried, but the patient succumbed to illness.

#### CONCLUSION

These days more and more ERCP procedures are being performed. This must involve a close-loop communication between surgeon and anesthesiologist. A proper review of the procedure and the complications that may encounter during it must be well explained to the relatives. We, anesthesiologists, must be aware of the morbidity and mortality associated with the procedure, especially in the endoscopy room with the lack of infrastructure, adequate equipment, and supportive staff.

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