Original Research Paper

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Anesthesiology

AIR EMBOLISM DURING ERCP UNDER GENERAL ANAESTHESIA EARLY DETECTION AND MANAGEMENT.

Dr Sukhvir Singh* Associate Consultant, Dept Of Anaesthesiology Max Hospital Mohali. *Corresponding Author

Dr Navreet Kaur

ABSTRACT Air embolism a rare complication during endoscopic procedures that can lead to cardiovascular collapse and neurological impairment. Cardiac arrest often occurs in air embolism as timely diagnosis is delayed. A patient, during or immediately after Endoscopic retrograde cholangiopancreatography who suffers cardiac and neurologic impairment differential diagnosis of air embolism should be kept in mind. What holds a key in diagnosis and management of air embolism is clinical deterioration. Once fatal air embolism occurs advanced cardiac life support system activation play an important role. Thus, for management of air embolism we need a team approach. We present a case report of air embolism of 83 yr old female during endoscopic retrograde cholangiopancreatography under general anaesthesia with role of end tidal carbon dioxide monitoring which was diagnosed early with good outcome.

KEYWORDS : Air embolism, Endoscopic retrograde cholangiopancreatography, general anaesthesia.

INTRODUCTION

Endoscopic retrograde cholangiopancreatography (ERCP) is a routine invasive endoscopic procedure which is used for both diagnostic and therapeutic indications.[1] Commonly reported complications of ERCP include pancreatitis (incidence of 1.6 to15.7%), haemorrhage (1.3%), cardiopulmonary(1%), air embolism (unknown) and duodenal perforation (0.1to 0.6).[2] Air embolism with a unknown incidence is usually iatrogenic which can occur in any endoscopic procedure leading to morbidity and mortality.[3,4,5,6] Air embolism is a rare complication during ERCP hence less no of cases reported , in this paper we report an interesting case of air embolism in ERCP with good recovery.

CASE REPORT

83-year-old female patient known case of diabetes and hypertension but without coronary artery disease and pulmonary disease presented with chief complaint of pain abdomen in gastroenterology opd. Complete blood profile, renal profile was normal, liver function tests shows raised liver enzymes. She was advised MR cholangiopancreato graphy which shows choledocholithiasis with extra and mild intrahepatic biliary dilatation, with cholelithiasis and was planned for ERCP under general anaesthesia. Her pre-operative work up was done with routine investigations which were normal. Electrocardiography, chest x-ray and echocardiography were normal. She was posted for ERCP next day in operation theatre. The patient was preoxygenated with 100% oxygen for three minutes and premedicated with 1 mg midazolam and 100 mcg fentanyl. Standard ASA monitors were recorded intraoperatively. The patient was then induced with 1mg/kg of propofol and with 0.5mg/kg of atracurium for muscle relaxation via a 20 G peripheral IV. Intubation was done with with a cuffed endotracheal tube. End-tidal carbon dioxide (EtCO2) was kept between 29-37 mmHg on volume control mode. After induction and intubation vitals were stable. For maintenance of anaesthesia inhalational agent sevoflurane was used with 50% FiO2. Patient was turned lateral for the procedure. ERCP procedure was done by gastroenterologist with cannulation of bile duct with sphincterotomy and removal of bile stones. Thirty minutes into the procedure, a precipitous drop in EtCO2 (30 to 12 mmHg), measured via capnography, was noted with concomitant hypotension (BP 90/60 mmHg) and narrow complex tachycardia (HR ~ 110). Within one minute, EtCO2 dropped to 5 mmHg further to 2 mmHg (figure 1) associated with severe hypoxia (Sp02 ~ 35%) and nonpalpable carotid pulse. As we know air has been traditionally used for insufflation, venous air embolism was quickly suspected. The gastroenterologist was immediately notified to abort the procedure. The patient was immediately turned supine with 100% FiO2 and positioned with head down position. Advanced cardiac life support (ACLS)was activated as the patient demonstrated pulseless electrical activity. Return of spontaneous circulation was achieved after 2 minutes of ACLS, with 1 mg of epinephrine, and the patient became normotensive without any vasopressor support. A right internal jugular central line was placed during ACLS for fluid resuscitation and 2 L of Plasmalyte was given over 15 minutes. Transthoracic echocardiography performed intraoperatively with normal findings. The patient was transferred to the intensive care unit intubated on ventilatory support with normal vitals for further monitoring and management. Cardiologist was consulted and a formal echocardiography was done showed an ejection fraction of 50% with no wall motion abnormalities. The patient was successfully extubated in the evening as the patient remained hemodynamically stable and a neurologic exam did not reveal any neurologic deficits. she was discharged from the hospital 14 days after the incident.

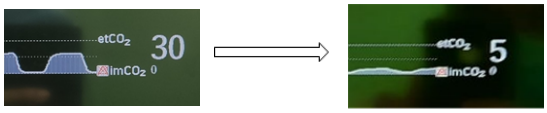

Figure 1 Monitor Showing Fall In Etco2 From 30 To 5

DISCUSSION

Endoscopic retrograde cholangiopancreatography has diagnostic as well as therapeutic indications in many diseases, such as lesions of the pancreatobiliary system and choledocholithiasis. Cardiopulmonary complications of ERCP that include arrhythmias, hypoxemia and transitory myocardial ischemia occur in 1% of cases.[7,8] Air embolism is a fatal and rare complication of ERCP leading to cardiopulmonary and neurological adverse events. Cardiac manifestations of air embolism include right heart failure, hypotension, cardiovascular collapse leading to cardiac arrest. Dyspnoea, tachypnoea,hypoxia,cyanosis, decrease in end-tidal carbon dioxide (EtCO2) concentration and respiratory failure are pulmonary manifestations of air embolism. Neurological impairment can manifest as failure to regain consciousness after completion of procedure and anaesthesia or as loss of consciousness, altered mental status, hemiparesis, cerebral oedema, dilated pupils.[9] Air embolism being a rare complication is less considered during ercp although it can manifest in any endoscopic procedure. Kennedy et al was among the first who noted air embolism as a complication of ercp in 1997.[10] In their case report, a 63-year-old female who underwent ERCP due to choledocholithiasis developed cardiorespiratory arrest due to hepatic venous air embolism. Coming on to proposed pathophysiological mechanism during ERCP leading to air embolism is that the high pressure which is used to distend the duodenum during sphincterotomy create a pressure gradient, thus leading to the passage of air into the portal venous system. Duodenal vein radicles which are cut transversely during the procedure allows the insufflated air to passes intramurally into the portal venous system. If there is presence of shunt or a biliary-venous fistula , gastrointestinal fistulas, trauma to the liver which further eases the occurrence of air embolism.[11,12] Stabile et al proposed a another possible mechanism of fatal air embolism in a patient who didn't have a patent foramen ovale or presence of shunt, other septal defects .[13] According to them pulmonary circulation is not capable of filtering all the venous gas that occurs in massive air embolism thus leading to build up of gas on the arterial side which further leads to ischemia of effected organ.

In our case, we observed sudden change in vital signs, most notably the decrease in EtCO2. We suspected air embolism that led us to respond quickly in a timely manner. The procedure was immediately stopped, the patient was quickly turned supine on so the head can be positioned

below the level of the feet to prevent air from causing right ventricular outflow obstruction. High flow 100% oxygen was administered to maintain adequate oxygenation, and helps reduce the bubble's nitrogen content and size. High volume fluid resuscitation was initiated via a central line, and cardiopulmonary resuscitation (CPR) was started immediately as per ACLS protocol. We also did not place the patient in a left lateral decubitus position to minimize air lock in the pulmonary artery because the position would interfere with our ability to perform adequate CPR. Return of spontaneous circulation was achieved and the patient remained hemodynamically stable after the event. CT imaging and neurologic examination of patient was normal. Similarly, Romberg presented a case report where the first sign of a possible air embolism was a decrease in EtCO2 detectable on capnography.[14] Prompt intervention resulted in a good outcome with no morbidity. In his review of nine patients, EtCO2-monitored patients had a 75% survival rate without permanent morbidity, compared to 0% without EtCO2 monitoring.[14] so EtCO2 monitoring during ERCP holds a key.

Any damage to the integrity of gastrointestinal structures which is caused by high insufflation pressures and by invasive procedure such as sphincterotomy as was done in our case leads to air embolism. Injured blood vessel during invasive procedure also provide access to bloodstream which act as an additional risk factor. Air embolism is difficult to diagnose(figure2) one can rely on clinical signs and hemodynamic variables such as drop in saturation, bradycardia, hypotension, or neurologically impairment if the procedure is done under monitored anaesthesia care and if it is under general anaesthesia fall in ETCO2 as it happens in our case. For finding gas in the heart transoesophageal echocardiography is very useful .[15]

As soon as air embolism is suspected patient should be positioned in trendelenburg position which helps by minimising air bubble to brain and also force out air embolism from right ventricle (figure2). Furthermore, if patient is in general anaesthesia 100% oxygen should be used to eliminate gas from air bubble and it also prevents hypoxia to organs which can cause reperfusion injury.[16] Central venous catheter should be inserted as early as possible as we can aspirate excess of air from the heart. Fluid resuscitation should be done to increase the central venous pressure so as to prohibit entry of air. Vasopressors should be used if blood pressures are on lower side .The rate of delivery of air is most important determinant of outcome after air embolism.[9] Immediate termination of procedure once air embolism is suspected helps in reducing the rate of delivery of air .Supportive treatment plays an important role in managing embolism depending upon the organ involved .If patient had seizures as occurs in cerebral embolism benzodiazepines should be used.[17] If there is pulmonary embolism anticoagulation is recommended.[18] In case of severe hemodynamic compromise and neurologic deficits hyperbaric oxygen therapy is recommended.[19]

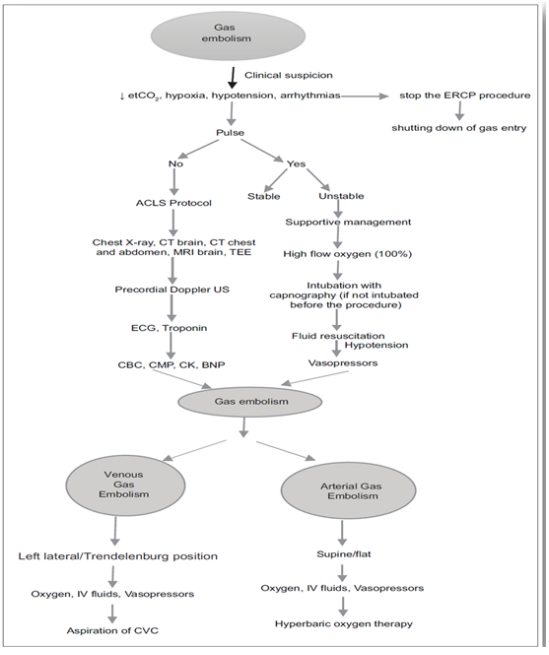

Figure 1 Diagnosis And Management Of Air Embolism (CBC, complete blood count; CMP, complete metabolic panel; CK, creatine kinase; ECG, electrocardiogram; BNP, brain natriuretic peptide;CVC,central venous catheter)

In conclusion, we present a case of systemic air embolism after ERCP with early diagnosis and management under general anaesthesia.

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