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An Unusual Presentation of Gas Embolism during ERCP

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Abstract

Gas embolism is a potentially life-threatening rare complication during endoscopy procedures. It is difficult to diagnose and clinical suspicion remains the key for diagnosis and management of gas embolism. During ERCP a patient developed cardiovascular and neurological adverse events, with paradoxical gas embolism being the most likely cause.

Keywords: Clinical Suspicion, Endoscopic Retrograde Cholangio-Pancreatography (ERCP), Gas Embolism

1. Introduction

Gas embolism defined as the passage of air into the systemic circulation, is a rare cause of fatal cardiopulmonary compromise. It is usually iatrogenic and can occur during any endoscopic procedure with Endoscopic Retrograde Cholangio-Pancreatography (ERCP) being the most frequent one. A list of 51 reported cases of air embolism associated with ERCP has been summarized in a recent systematic review[1].

We report a non-fatal highly suspicious case of gas embolism during ERCP with Common Bile Duct (CBD) stone extraction and biliary stent removal under general anaesthesia. Patient developed acute myocardial ischemia and stroke secondary to gas embolism.With prompt recognition and intervention for possible gas embolism, the patient recovered with mild residual left sided weakness.

2. Case Report

A 45 year oldASA grade- 1 female with no comorbidities presented to us in February 2021 with upper abdominal pain and cholestatic jaundice. Serum amylase and lipase was elevated and the diagnosis of acute pancreatitis was confirmed by a Magnetic Resonance Cholangio-Pancreatography (MRCP) which also revealed a large stoneinthe bile duct. An ERCP was attempted, but the stone seemed impacted, so a biliary stent was deployed. After resolution of pancreatitis and jaundice, she was taken up for ERCP for stent removal and CBD stone extraction under general anesthesia.

General physical examination was unremarkable. The patient was afebrile, with baseline blood pressure 130/70 mm hg, heart rate 84/minute maintaining oxygen saturation of 99-100 percent on room air. Patient was 150 cm in height and 60 kg in weight. In the operating room, all standard ASA monitors were attached.Patient waspre-oxygenated for 3 minutes. Anaesthesia was induced with midazolam 1mg, fentanyl 100 mcg, propofol 120mg and atracurium 30 mgvia a 20 G i/v cannula followed by direct laryngoscopy and endotracheal intubation with 6.5 mm ET cuffed tube. Patient was then put on volume controlled mode of ventilation. Vitals remained stable during and after induction of anaesthesia. Anaesthesia

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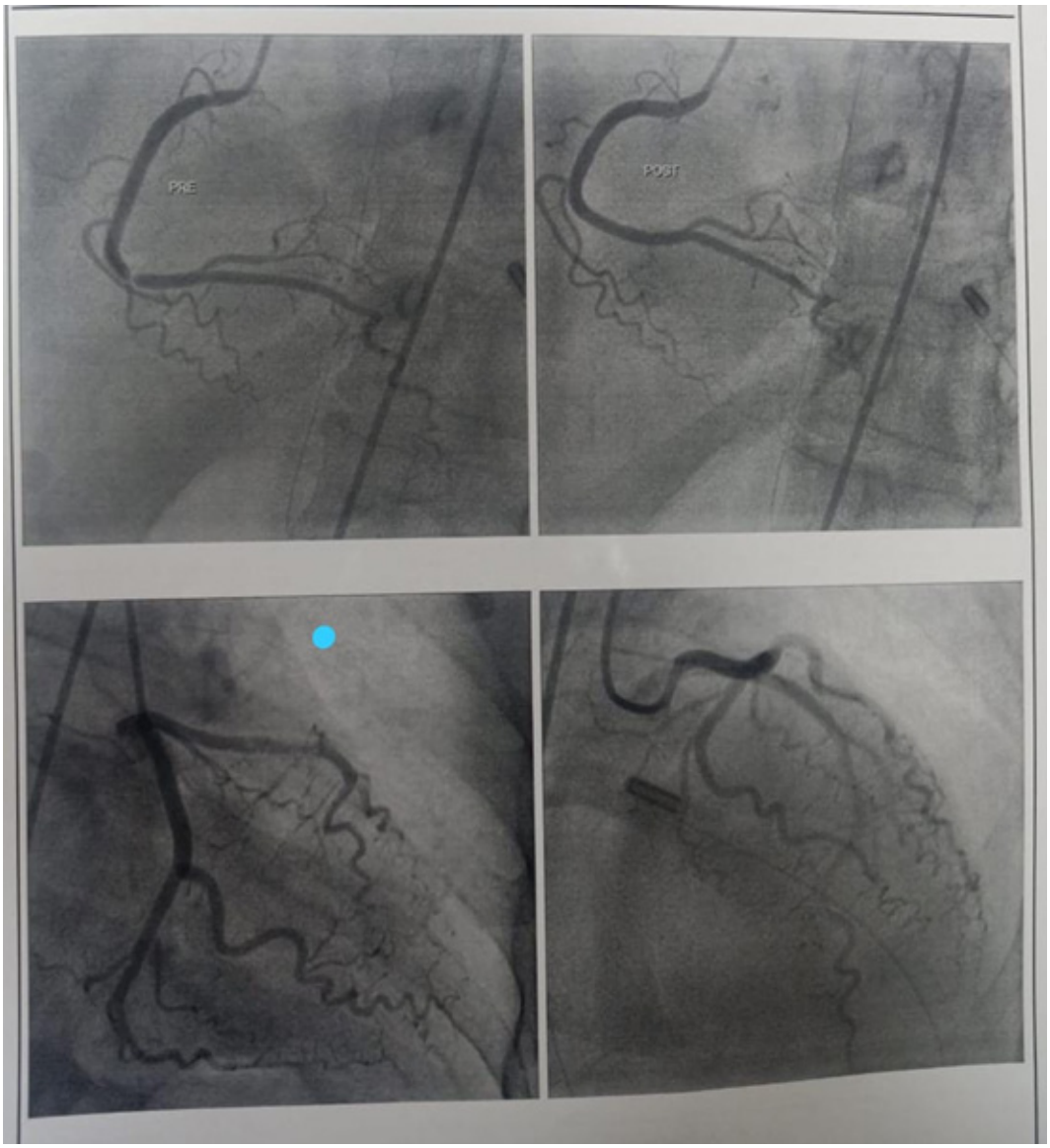

Figure 1. Coronary angiography image showing blockage and vasospasm in right coronary artery.

was maintained with 0.6 % isoflurane, 50% N_2O and 50% O_2. For the ERCP, the patient was positioned in a semi-prone position. The endoscope was introduced and stomach and duodenum were insufflated with carbon dioxide to optimize visualization. During the procedure, the previous stent was removed, wire guided selective biliary cannulation done, cholangiogram obtained and in view of the large stone, balloon dilation of ampulla was done till 15mm with a CRE™ balloon. The stone was then removed under guidance with a biliary basket. An occlusion cholangiogram was obtained, which confirmed that the bile duct was clear. At the end of the procedure, which took nearly 30 minutes, a precipitous fall in $EtCO_2$ from 34 mmhg to 14 mmhg with concomitant bradycardia (HR - 45 per min), hypotension (BP- 80/50 mmhg) and ST- segment elevation in lead II on ECG monitor was noted. Carotid pulse was palpable but peripheral pulses were feeble. Patient was immediately taken on 100 percent

Figure 2. MRI image showing multifocal acute infarct in both cerebrii likely to be embolic.

oxygen. Inj atropine 1.2 mg and ephedrine 15mg i/v stat were administered, 500ml NS rushed. Patient was turned supine and left lateral trendelenberg position was made. Despite stabilizing the blood pressure with vasopressors, $EtCO_2$ persistently remained low. 12 lead ECG done was suggestive of ST- segment elevation in leads II, III, aVF,V_1-V_3. Arterial blood gases werenormal.Immediate cardiology consultation was taken and primary PTCA was advised. During coronary angiography, 75 percent blockage and distal vasospasm was noted in right coronary artery which got relieved with intracoronary nitroglycerine (Figure 1).

Transthoracic echocardiography showed normal cardiac wall contractility. Patient was kept on ventilatory support for a day and ashaemodynamics were stable without any support, so trachea was extubated. However, post extubation, she remained drowsy, had no verbal response and left sided hemiparesis. NCCT head was done which revealed hypodense lesion in right high parietal region suggestive of anterior cereberal artery infarct. A subsequent MRI showed multifocal acute infarct in both cerebrii (right > left) likely to be embolic (Figure 2). After 3 days, patient's verbal response recovered but residual left sided weakness was present in upper and lower limb. Patient was managed conservatively and discharged after 11 days. On follow up after 1 month, she had minimal residual deficit and was able to carry out her day-to-day activities.

3. Discussion

Gas embolism is defined as undesired entry of air into the vascular structures[1]. It is usually observed with air but can occur with carbon dioxide. Gas embolism can occur via venous or paradoxical route during endoscopy procedure[2-4]. It is often catastrophic with significant high risk of perioperative morbidity and mortality. The various proposed mechanisms for air embolism from ERCP include endoscopy induced mechanical irritation of bile duct wall, air entry into biliary venous shunt,

transgression of air into adjacent veins from inflammation of mucosa and muscular wall[5,6]. Venous embolism can progress to paradoxical air embolism via intracardiac and intrapulmonary right to left shunt, retrograde flow to cerebral veins through superior vena cava or passage into left atrium through pulmonary veins[7]. Air embolism remains under-recognised and difficult to diagnose due to its variable presentation[8]. It can lead to variety of cardiopulmonary manifestations which includes arrhythmias, hypotension, dyspnea, respiratory failure and cardiac arrest. Neurological symptoms include unconsciousness, paraparesis, hemiparesis, dilated pupils, cerebral edema and coma[7,9].

We presented here a non-fatal highly suspicious case of gas embolism during ERCP. During endoscopy, high pressure CO_2 was used to distend the duodenum and to ensure adequate visualization by the camera and manipulation of instruments in the duodenum. The patient had an impacted stone which required sphincteroplasty, basketing and removal, which may have led to injury of bile duct wall, promoting the development of a biliary-venous shunt which could have been the gateway for gas entry. Usually CO_2 is rapidly absorbed from bowel, delivered directly to lungs by the circulation and excreted by the lungs during respiratory exchange. In our case, we observed an abrupt and dramatic change in hemodynamics, most notably a sudden decrease in $EtCO_2$, which is pathognomonic for gas embolism. We initiated resuscitation with high index of suspicion for gas embolism. Coronary artery embolization results in ischaemia, infarction, dysrhythmias, and myocardial suppression. In our case, embolization of gas into the right coronary artery is evident by ST elevation in the inferior leads and vasospasm in right coronary artery on coronary angiography. Our patient developed acute stroke with aphasia and left sided hemiparesis due to cerebral arterial gas embolization, as evident by MRI brain showing multifocal infarct in cerebrum.

This sequelae of cardiovascular and neurological complicationsled to high suspicion of paradoxical gas embolism. Paradoxical gas embolism is said to occur when there is evidence of gas in the arterial circulation which have originated from venous circulation. The arterialization of gaseous products may occur by passage of gas through an anatomical right-to-left shunt such as an open foramen ovale or through pulmonary Arterio-Venous (AV) malformations or by passage of gas through pulmonary veins into left atrium[10]. Though transthoracic echocardiography did not reveal any right-to-left shunt, the definitive perioperative diagnosis could not be made due to non-availability of trans-esophageal echocardiogram.

4. Conclusion

An increased awareness of possibility of gas embolism during ERCP with early recognition can help in definitive intervention to reduce the morbidity and mortality associated with this rare event.

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