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Original Research Paper

Anaesthesiology

ERCP: PNEUMO "CRISIS"

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ABSTRACT 31 year old female with cholelithiasis with common bile duct stone with no associated comorbidity presented to surgery clinic. She underwent ERCP for removal of common bile duct stone and placement of biliary stent.Intraoperatively her vitals were stable and after the procedure she was shifted to postoperative care unit. Though her haemodynamics remained stable, she was never "fully awake", though responding and nodding to commands. After about 20 minutes in recovery room she developed bradycardia with heart rate 30 beats/minute and hypotension. Assisted ventilation was started with 100% oxygen using bains circuit and inj atropine 1 mg iv was given and noradrenaline infusion was started and titrated to effect. She responded positively within minutes and started maintaining haemodynamics and respiration without assistance/drugs. Strangely her GCS deteriorated to E1V1M2; and she developed decerebrate rigidity and ophisthotonus posture. She was intubated under cover of propofol (2.5mg/kg) and succinylcholine (2mg/kg). Her CT head, thorax and abdomen was done which showed normal study. Her MRI was done which showed early hypoxic ischaemic encephalopathy. This was surprising as these changes were caught within 4 hours of completion of procedure indicating relatively long standing hypoxic damage to brain. She was managed conservatively. After 1 week her MRI showed hypoxic encephalopathy involving bilateral fronto-parietal regions. Tracheostomy was done and managed conservatively and was discharged from intensive care unit with tracheostomy tube in situ. The patient recovered gradually and was able to perform normal daily activities over a period of one month.

KEYWORDS :

INTRODUCTION

Endoscopic retrograde cholangiopancreatography (ERCP) is an invasive procedure that is performed to diagnose and treat pancreatic and biliary disease. It is used for diagnosis of jaundice, evaluation of known or suspected pancreatic disease, and pre- or postoperative assessment of biliary tree. Applications of therapeutic ERCP include sphincterotomy, removal of common bile duct stones, lithotripsy, biliary drainage and stricture dilation. Though a seemingly non invasive procedure, incidence of general/systemic complications are similar to invasive surgeries (5%-10% of cases), excluding wound complications.[1] The complications can be secondary to biliary and pancreatic manipulation or related to endoscopy procedure, specially high pressure, high volume gas inflation of bowel. Mortality post ERCP is around 0.33%; among 41 published air embolism cases among the entire spectrum of endoscopic surgical procedures, 26 (60%) cases were during ERCP alone.[2] These ERCP related embolism cases have a noted mortality nearing 50%, re-emphasizing the gravity in cases of complications.[2]

Here we present and discuss three pneumo "crisis" disease modalities post ERCP.

Case Report

Case 1: Gas (CO_2) embolism

A 31 year old female with cholelithiasis with common bile duct stone with no associated comorbidity presented to surgery clinic. Her investigations showed slightly elevated AST 127U/L and ALT 76U/L. Bilirubin was within normal limits. CBC showed Hb 11.4g/dl, WBC count 17.10 thou/uL and neutrophils were 85%. She underwent ERCP for removal of common bile duct stone and placement of biliary stent. During the procedure she was given (along with two 10% lignocaine sprays orally) inj glycopyrrolate 0.2mg iv, inj ondensetron 4mg iv, inj propofol 20mg and inj ketamine 10mg + 10mg iv. Intraoperatively her vitals were stable and after the procedure she was shifted to postoperative care unit. Though her haemodynamics remained stable, she was never "fully awake", though responding and nodding to commands. After about 20 minutes in recovery room she developed bradycardia with heart rate 30 beats/minute and hypotension. Assisted ventilation was started with 100% oxygen using bains circuit and inj atropine 1 mg iv was given and noradrenaline infusion was started and titrated to effect. She responded positively within minutes and started maintaining haemodynamics and respiration without assistance/drugs. Patient was taken to ICU for observation as her conscious level was E4V2M4. Her heart rate was now 140 beats/ min and blood pressure was 150/80 mmhg. Ultrasound of abdomen and thorax was done which was normal. ABG done during the episode showed pH 7.2, pCO_2 45.7mmHg, pO_2 64.8mmHg & bicarbonates were 20.2mmol/L. Strangely her GCS deteriorated to E1V1M2; and she developed decerebrate rigidity and ophisthotonus posture. She was intubated under cover of propofol (2.5mg/kg) and succinylcholine (2mg/kg). Her CT head, thorax and abdomen was done which showed normal study. Her MRI was done which showed early hypoxic ischaemic encephalopathy involving head of caudate nucleus and anterior part of lentiform nucleus on left side, left insular cortex and bilateral basal ganglia. This was surprising as these changes were caught within 4 hours of completion of procedure indicating relatively long standing hypoxic damage to brain. After 24 hours in intensive care unit patient developed malena and her Hb was 6g/dl. She was managed conservatively. After 1 week her MRI showed hypoxic encephalopathy involving bilateral fronto-parietal regions. Tracheostomy was done and managed conservatively and was discharged from intensive care unit with tracheostomy tube in situ. The patient recovered gradually and was able to perform normal daily activities over a period of one month.

Case 2: Pneumoperitoneum/ Pneumothorax/ Pneumomediastinum

A 45 year old female patient was admitted for common bile duct clearance. She had underwent laparoscopic cholecystectomy 1 month ago. During ERCP a guide wire

assisted sphincterotomy was performed and common bile duct stones were removed. Immediately after the procedure, the patient developed hypotension (Systolic BP 80 mm Hg) and bradycardia (40 beats/min) bradycardia (40 beats/min) along with abdominal distention, abdominal pain, dyspnoea, chest pain and loss of consciousness.. Resuscitation was initiated by turning the patient's supine, intubating the patient after giving inj succinylcholine 75 mg and giving inj. atropine 1mg i/v and starting titrated dopamine infusion. Physical examination revealed tense rigid abdomen and provisional diagnosis of pneumoperitoneum was made. Patient was also having subcutaneous emphysema. Ultrasound was used to establish the diagnosis and tension was relieved by inserting 14 G cannula. Though haemodynamics improved, there was subsequently an increase in peak and plateau airway pressures along with diminished breath sounds on the right side of chest, with fall in saturation to around 80% with fiO2 of 100%. Chest X ray showed extensive right side pneumothorax and pneumomediastinum. Chest tube and abdominal drain was inserted to ease the tension in cavities. Oxygen saturation improved to 95%. Chest and abdominal CT scan confirmed presence of pneumoperitoneum, pneumoretroperitoneum, pneumomediastinum and right side pneumothorax. Patient was further managed conservatively in the ICU and improved subsequently.

Case 3: Pneumomediastinum / Subcutaneous emphysema

80 year male was admitted in surgery unit for common bile duct stone with history of cholangitis. He had a history of diabetes and had underwent cholecystectomy 15 years back. Procedure was uneventful and patient was shifted to postoperative recovery room. In the recovery room subcutaneous emphysema was noticed in facial, cervical and thoracic regions. Patient had episode of bradycardia (HR 30/min) and hypotension which was managed with Inj. atropine 1mg and initiation of titrated Dopamine infusion. Thoracic computed tomography showed pneumomediastinum, which was considered to be the culprit. Eventually the patient improved on conservative management.

DISCUSSION

Endoscopic retrograde cholangiopancreatography is a procedure used to diagnose and treat diseases such as choledocholithiasis or neoplastic lesions of the pancreatobiliary system. The procedure involves continuous insufflation of gas (air or preferably carbon dioxide), into the bowel at relatively high pressure around 30ml/sec till the entire duration of the procedure approximately 30 minutes and this is what causes the "Pneumo crisis" or gas related complications of ERCP.

Various mechanisms have been postulated for existence of these complications

GAS EMBOLISM ROUTES

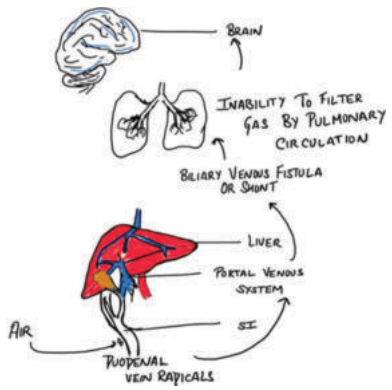

Fig 1

Finsterer et al in a study conducted concluded that "In case that patient does not wake up after the procedure, air embolism should be taken into consideration"[3]

Insufflated Air -> Duodenal vein radicles -> Portal venous system (Fig 1)

Presence of biliary venous fistula or shunt, trauma, previous surgery, mucosal injury predispose for this event and route[3] due to continuous high pressure insufflation which causes gas movement. Due to the same reason, there is transient inability to filter gas by pulmonary circulation causing cerebral air embolism. Other route suggested for cerebral gas embolism is:

Paradoxical Air Embolism due to continuous high pressure insufflation:

Insufflated Air -> Duodenal vein radicles -> Portal venous system -> IVC -> SVC -> Cerebral Vessels.[4] (Fig 2)

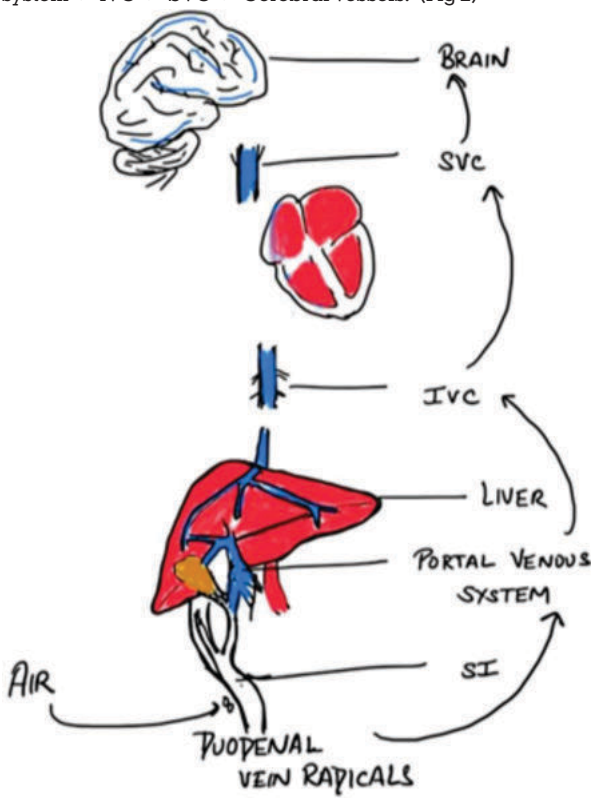

Fig 2

First case of air embolism in ERCP was noted by Kennedy et al in 1997[5]. In this case a 63 year female patient developed bradycardia and hypoxia. She suffered cardiac arrest and resuscitation was unsuccessful. Her chest x ray showed gas within the right ventricle and pulmonary arteries.

Goins et al[6] reported a case of a 72 year female who underwent ERCP for cholangiocarcinoma. She developed cardiac arrest due to massive air embolism in right heart. Pulmonary artery catheter was inserted and air was aspirated and pulses returned. MRI showed infarcts in right frontal lobe. Patient recovered fully.

A case of fatal cerebral embolism was reported by Rangappa et al of a 50 year old female who underwent ERCP for choledocholithiasis. She was unresponsive after the procedure and her CT head showed air embolism in the right hemisphere with tonsillar herniation and diffuse cerebral edema. Cause of death was found out to be brain edema due to paradoxical air embolism.[7]

Nayagam et al reported a case of a 57 year male who developed cerebral ischaemia caused by venous and arterial embolism during ERCP. Patient became comatose and had saturation of 70% as the procedure ended. He was intubated and MRI scan of brain showed multiple grey matter infarcts.[8]
Arguelles et al reported a fatal case of 59 year old female due to cerebral artery embolism during ERCP due to severe ischaemic brain injury.[9]

These cases describe mortality in patients in ERCP due to air embolism affecting heart or brain.

PNEUMOTHORAX

Routes:
1. Gut Perforation/ Gas leakage -> Retroperitoneal gas -> fascial plane -> Mediastinum -> Pneumothorax
2. Peritoneal Cavity Air (Either directly through serosa or through Retroperitoneum space) -> diaphragmatic fenestrations -> Pleural space -> Pneumothorax

PNEUMOMEDIASTINUM

Gut perforation -> Retroperitoneal gas -> Anterior pararenal space -> diaphragmatic hiatus -> pneumomediastinum [10]
In all these complications following factors predispose:
1. Gas Flow rate: Literature and Different ERCP machines indicate different flow rates ranging from 200 ml/min to really high ones of 30ml/sec [11] The higher the rate, higher are chances of these complications.
2. Gas Pressures: One study found out that pressures at distal end of endoscope was > 300 mm Hg when flows were nearly 2l/min. To extrapolate these pressures to what is used in our institution which is higher than recommended (which gastro in our institute accepts) will be naïve and unacademic but we can safely imagine that really high pressures might be generated with high flows. This explains the high rate of (around 50%) mortality in case of embolism in ERCP.
3. Duration of procedure: In case of gas leakage more the time of leak higher the complications in frequency as well as intensity. [12]
4. Mucosal breach: either intentional or iatrogenic: opens up vascular end points leading to rapid absorption of gases at high rate and high pressure. Also it aids in rapid expulsion of gas into the abdominal cavity from where it spreads to other areas.
5. Air vs CO2: Air being three times less diffusible leads to more catastrophic results. Endoscopic society also opines and suggests to use CO2 in preference to air[13] but due to high volume turn over of cases, management issues and cost air is frequently used especially in cases where mucosal breach is not suspected, as in most institutes, including ours.

Thus gas (air more than CO2) at high flow rate, at high pressures, if it starts leaking into either vascular structures or abdominal cavity for relatively long duration may lead to drastic complications.

In our data analysis we found that we had 7 cases related to pneumo crisis during or after ERCP. Of these 5 were of pneumoperitoneum progressing to pneumothorax. Out of these 5 we lost 2 patients. In our first experienced case of this complication entire OT team was unaware due to prone position and rapidly deteriorating hypotension and bradycardia leading to cardiac arrest in spite of all resuscitative measures and insertion of 14 G cannula in infra axillary (pneumothorax) as well as intra abdominal cavity (pneumoperitoneum). As this case was highlighted among all anaesthetists in order to be aware of the same we could save the next 3 patients having the same complication. Awareness about this relatively rare though high risk complication is crucial for early active management. Stoppage of procedure and gas flows and turning the patient supine should be immediate, followed by initiation of resuscitative measures including high FiO2 positive pressure ventilation through endotracheal tube and inotropes, as and when required. Early diagnosis may be made clinically with percussion (hyperresonance in abdominal and thoracic cavities), auscultation (decreased breath sounds), decrease in EtCO2 and ultrasonography. Bedside ultrasonography is of paramount importance in management of this complication. It aids in rapid diagnosis by detecting abdominal air outside bowel as well as insertion of trocar or cannula to release the same .

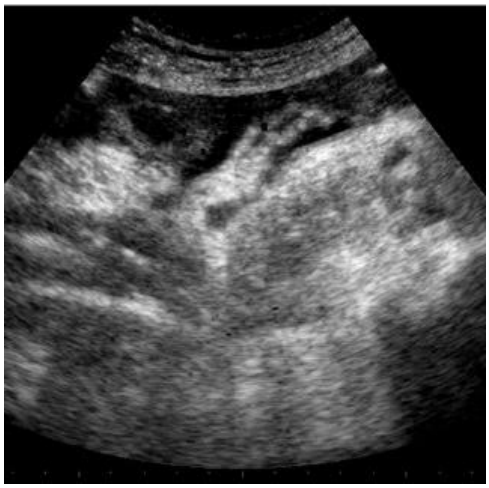

Fig-3 Ultrasound Image Showing Pneumoperitoneum

PNEUMOPERITONEUM

Ultrasonography is also important in detection and diagnosis of pneumothorax. Barcode/ stratosphere sign on M mode is a classical sign seen on ultrasound .

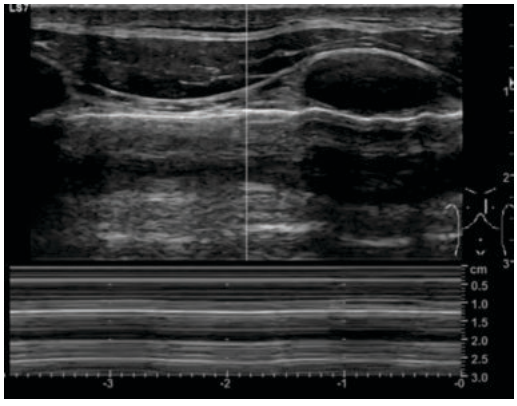

Fig 4 Ultrasound Image M Mode Showing Barcode Sign

In pneumothorax normal lung sliding and comet tail artifacts from pleura are absent.

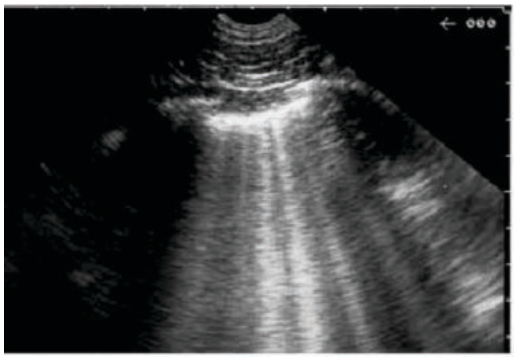

Fig 5 Ultrasound Showing B Lines

Early USG guided release of tension pneumothorax on first sign of desaturation, alongwith rising plateau and peak

pressures, is imperative in prevention of morbidity and even mortality. 5th patient having this complication deteriorated drastically due to previous heart failure and COPD. Rapid deterioration and failure of resuscitation may also be caused by pneumomediastinum which directly may impede myocardial activity or cause arrhythmias due to vagal stimulation.

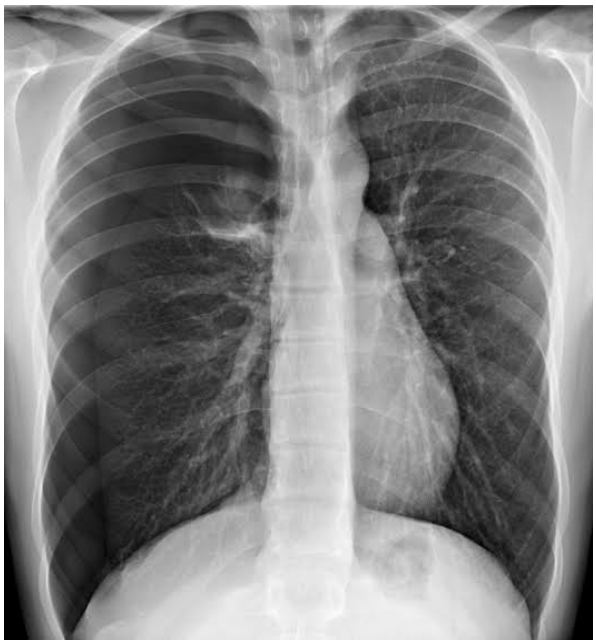

Fig .6 Chest X-ray Pa View Showing Pneumothorax

Subcutaneous emphysema occurs most commonly due to duodenal perforation during ERCP but even during absence of perforation it mostly occurs due to insufflations of pressurized air with mucosal disruption. The air may spread to fascial planes to subcutaneous tissue causing subcutaneous emphysema and can also lead to pneumothorax and pneumomediastinum.[14]

Our case of cerebral embolism is the one which made us focus into gas flows and pressures during ERCP. The rapidity of CNS depression indicated significant long duration of ischaemia in cerebral tissue which was quite unexplainable at that time due to non detection of any hypoxic event. Patient was responding, nodding purposefully and also had comprehensible speech though not very clear. On retrospective analysis the only indication could have been "not fully alert" waking up. Transient ischaemic irritation of midbrain might have led to the event of bradycardia and hypotension.

Rapidly progressing early rigidity (within 2 hours) and detection of hypoxic changes in MRI within 5 hours of insult points towards diffuse embolism[13] blocking of blood supply for a significant period of time, leading us to gas embolism. Pathophysiology of diffuse air embolism mimics fat embolism injury and hence persistence of tachycardia, absent insufflation sign, rapid onset of coma and early decerebrate rigidity all went towards diagnosis of CO2 embolism.[15] Presence of melena and subsequent endoscopic findings of upper GI mucosal injury validated the diagnosis and severity of the condition. On reviewing the literature and our case profile, we along with neurologist concluded that the most probable diagnosis must be paradoxical CO2 embolism. Mucosal injury might have occurred at early part of procedure with either scope or initial high pressure insufflations directly on the gut wall leading to opening up of potential way for gas to reach portal veins which might have continued for the entire duration of procedure. During the procedure along with carbon dioxide , air was also used. Also as must be with most ERCP machines the flow rates are high, unregulated and there was no pressure gauge to regulate pressures, only directly attached cylinders, which as already discussed can lead to significantly high pressures. All this might have caused prolonged transmission of CO2 showers to cerebral vessels leading to hypoxic cerebral injury.

Thus pneumo crisis may happen during ERCP cases and following may be undertaken to prevent its incidence and / or severity:

1. Use of CO2 and not air
2. Limit flow rate to bare minimum
3. Limit and regulate gas pressures as done in laparoscopic surgeries
4. Procedure time should be minimum
5. Mucosal injury should be avoided and sphincterotomy should be carefully and minimally done
6. Continuous awareness, observation, monitoring and early active management on the part of Anaesthetist is the key to prevention of morbidity and mortality

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