CASE REPORT

Fatal cerebral air embolism following endoscopic evaluation of rectal stump

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SUMMARY

A 63-year-old man underwent endoscopic evaluation of the rectal stump for rectal bleeding and suffered a massive cerebral air embolism with severe neurological impairment and subsequent death. The patient underwent a Hartmann's procedure 9 month previously for ischaemic bowel and was noted to have portal hypertension at laparotomy. We hypothesise that air entered the venous plexus around rectum and entered the azygos vein via a porto-systemic shunt and travelled retrogradely via the superior vena cava to the venous sinuses of the brain.

BACKGROUND

Endoscopy is one of the most common procedures performed around the world with known complications of bleeding and perforation. Cerebral air embolism is a well-documented but rare complication of several medical procedures including trauma, central venous access, iatrogenic and pulmonary barotrauma. Cerebral air embolism is associated with severe neurological injury and poor outcomes.¹ We describe an unusual case of fatal cerebral air embolism following a flexible endoscopy of the rectal stump.

CASE PRESENTATION

A 63-year-old man underwent an endoscopic evaluation of the rectal stump for rectal bleeding. He had an emergency Hartmann's procedure 9 month previously for ischaemic bowel. He had a medical history of heavy alcohol intake, diabetes mellitus, chronic pancreatitis (secondary to alcohol abuse) and hypertension. At laparotomy, he was noted to have portal hypertension and liver cirrhosis. He had a stormy postoperative course due to renal impairment and an expected dehiscence of the rectal stump which was managed conservatively by drains placed at the time of surgery and the patient discharged home well after.

He presented with intermittent episodes of profuse rectal bleeding and was booked for endoscopic evaluation of rectal stump. On admission for the procedure, his vital signs were stable and his full blood count and biochemistry were unremarkable. Standard monitoring was applied and 5 mg of midazolam was titrated as sedation for the procedure which lasted under 10 min. During the procedure, vitals stayed within the normal range. Of note there was no change in oxygen saturation. Flexible endoscopy was performed from the anal canal to investigate the source of bleeding and the rectum was insufflated and apart from some areas of

Figure 1 Air in cortical veins.

erythema and ecchymosis, an overt source of bleeding was not identified. Postcolonoscopy, the patient remained drowsy with a decreased level of consciousness despite the administration of flumazenil.

INVESTIGATIONS

An urgent CT scan of the brain was performed. This revealed a significant volume of air in a cortical venous distribution (figure 1). CT scan of the abdomen and the pelvis also displayed air in the portal system (figure 2). Air was also noted in the bone marrow and right common femoral vein (figure 3). The patient was intubated for a persistently low Glasgow Coma Scale and ventilated with 100% oxygen. Trans-thoracic Echo was performed, which revealed no evidence of a structural or functional lesion explaining a right to left shunt. Advice

Figure 2 CT abdomen and pelvis air in peripheral portal veins ascites gastric and splenic varices.

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Figure 3 Air in right common femoral vein.

was sought from the national hyper-baric therapy unit. Transfer of the patient for hyperbaric therapy was deemed unlikely to be beneficial owing to the severity of the insult, the time-critical nature of therapy required and geographical logistical issues. Repeat CT brain scan at 24 h revealed infarction with massive cerebral oedema (figure 4) and following discussion with the family, care was redirected towards palliation and the patient died the subsequent day.

DISCUSSION

Colonoscopy is one of the safest procedures performed with only rare complications including bleeding and perforation. Cerebral air embolism is rare. However, it has been reported in association with several different medical procedures.^{1–6} Although upper gastrointestinal (GI) endoscopy has been documented to cause cerebral air embolism, we are unable to find any report in the literature of a massive air embolism associated with lower GI endoscopy or endoscopy of the rectal stump. Upper GI endoscopy has been documented as a cause of cerebral air embolism usually in association with erosive oesophageal and gastric tumours or endoscopic retrograde cholangiopancreatography.⁷

Aetiologically, cerebral air embolism requires two predisposing factors: entry of air into the circulation and transport of the embolus to the cerebral vasculature. Diagnostic colonoscopy has been reported to enable entrainment of air into the circulation

in the setting of vessel trauma and parenchymal defects.² Studies have shown that the mean rate of air-insufflation at colonoscopy varies from 0.24 to 0.26 l/min for both air and carbon dioxide, respectively. Per colonoscopy a mean of 8.3 l of carbon dioxide (range 1.2–19.8) and 8.2 l of air (range 1.8–18 l) were insufflated.⁸

In our case, we hypothesise that the insufflation of the rectal stump with pressure led air to enter the venous plexus because of rectal varices. Air then entered the azygos vein via a porto-systemic shunt secondary to the associated portal hypertension. From here it would have entered the superior vena cava and we postulate that air may have travelled retrogradely via the superior vena cava to the venous sinuses in the brain. While retrograde venous flow of air obviously represents an unorthodox view, air insufflation during colonoscopy is pressurised. Also this patient was lying in lateral decubitus position presenting minimal gravitational venous resistance and was also known to have portal hypertension which is associated with global venodilation. We believe the predominantly right-sided cerebral injury was a result of the patient being positioned on his left side at the time of the insult, making that side of the brain more susceptible to air.

Cerebral air embolism has been described following systemic venous air entrainment despite extensive searches for potential intracardiac shunt defects.⁵ The exact mechanism whereby such systemic venous air reaches the cerebral vasculature in patients without intracardiac defects is unknown. One possible explanation is that the pulmonary venous microcirculation is overwhelmed by the volume of air and eventually traversed due to the ability of the air emboli to deform.¹ Another possibility is that an undiagnosed patent foramen ovale temporarily opens when pulmonary arterial pressure rises. This is considered analogous to cerebral fat embolisation that occurs during hip surgery.⁶

We believe that continuous insufflation of small air volumes during the procedure into a closed rectal stump with microscopic rectal vasculature tissue injury, was the source of intravascular air in our patient. The venous plexus may have been particularly susceptible to air entrainment because of the portal hypertension.

Figure 4 CT brain 24 h postcolonoscopy demonstrating extensive infarction and cerebral oedema.

Learning points

- ▶ Treatment of cerebral air embolism is predominantly supportive in nature.
- ▶ Logically, some authors advocate hyperbaric therapy. However, there is no consensus on the efficacy of this modality.¹ Logistical difficulties and patient instability prevented the administration of hyperbaric therapy in our case.
- ▶ We conclude that cerebral air embolism is a rare but potentially fatal complication of endoscopy due to air insufflation. It might be considered in patients with unexplained neurological deterioration after endoscopic evaluation.

Competing interests None.

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