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Conservative management of gastric pneumatosis following left gastric artery embolisation

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Accepted 31 July 2022

SUMMARY

Gastric pneumatosis, the presence of air within the stomach wall, is a very rare occurrence with poor outcomes. One of the most common mechanisms for gastric pneumatosis is gastric ischaemia, also a rare entity. Although patients with gastric ischaemia may require surgical intervention, they can often be treated with conservative measures such as a proton pump inhibitor, broad-spectrum antibiotics, nasogastric tube decompression, fluid resuscitation and total parenteral nutrition. We report a rare case of gastric ischaemia and pneumatosis following therapeutic left gastric artery argon plasma coagulation that was treated with conservative measures.

BACKGROUND

Gastric pneumatosis, the presence of air within the stomach wall, is a very rare occurrence with poor outcomes. In one recent review, there was 111 publications over the last 25 years, mostly single case reports.[1] Some of the most common mechanisms for gastric pneumatosis include gastric ischaemia or infarction, bacterial infection and gastric mucosal disruption facilitating air penetration, such as in forceful vomiting or gastric outlet obstruction.[1 2]

Due to the rich collateral blood supply to the stomach, gastric ischaemia itself is a rare entity. Aetiologies of gastric ischaemia can be divided into systemic hypoperfusion, such as in shock, or local causes of hypoperfusion, such as in gastric volvulus, coeliac axis occlusion or in superior mesenteric artery syndrome.[1]

The treatment of gastric pneumatosis is often conservative; however, surgery may be required depending on the aetiology.[1] In one recent study, patients with elevated serum lactic acid, elevated serum creatinine, or concomitant small bowel or colonic pneumatosis were found to have higher mortalities. It has been suggested that more aggressive treatment may be warranted in these patients.[3]

We report a rare case of gastric ischaemia and pneumatosis following therapeutic left gastric artery gelfoam and coil embolisation that was treated with conservative measures.

CASE PRESENTATION

A man in his 70s with a medical history significant for recurrent upper gastrointestinal (GI) bleeding secondary to a gastric dieulafoy lesion, erosive gastritis, coronary artery disease with a drug-eluting stent (on aspirin 81 mg daily), chronic kidney disease stage III, hypertension and non-insulin dependent type 2 diabetes presented with a 1-week history of worsening fatigue and shortness of breath.

The patient was recently hospitalised three times in the past few months for severe symptomatic acute blood loss anaemia secondary to a bleeding gastric dieulafoy lesion. This was treated on the first admission with argon plasma coagulation and clipping and the third admission with epinephrine and clipping. No intervention was necessary during the second admission.

On presentation, he was found to have a haemoglobin of 57 g/L and was transfused two units of packed red blood cells (pRBCs). He began having multiple episodes of melena associated with a brief hypotensive episode and was subsequently transfused three additional units of pRBCs. He underwent oesophagogastroduodenoscopy (OGD) revealing active bleeding of the gastric dieulafoy lesion that was treated with Injection Gold Probe.

Next day, the patient had a drop in his haemoglobin, which required transfusion of an additional unit of pRBC. This drop in haemoglobin was attributed to continued bleeding of his dieulafoy lesion. Since endoscopic therapeutic approaches had failed three times and the recurrent bleeding was significant enough to warrant multiple hospitalisations the decision was made to consult interventional radiology (IR) for embolisation. The patient underwent gelfoam and coil embolisation of the left gastric artery with successful haemostasis.

He had no further drops in haemoglobin or evidence of melena or haematochezia after the embolisation. Over the next 72 hours after the procedure, he developed persistent diffuse abdominal pain and vomiting. He then became septic with fever of 38.9°C, tachycardia to 120 s and leucocytosis with white cell count of 20.8 10^9/L.

INVESTIGATIONS

A CT abdomen pelvis with intravenous contrast showed pneumatosis and mural thickening along the wall of the lesser curvature of the stomach (figure 1). An OGD showed diffuse ischaemic changes, characterised by discolouration and deep ulceration in the proximal stomach involving the anterior and posterior wall extending down to the lesser curvature of the stomach (figure 2). Acute care surgery was consulted and recommended conservative management with strict bowel rest, serial abdominal exams and trending of serum lactic acid.

To cite: Bloom MD, Ladna M. *BMJ Case Rep* 2022;**15**:e249773. doi:10.1136/bcr-2022-249773

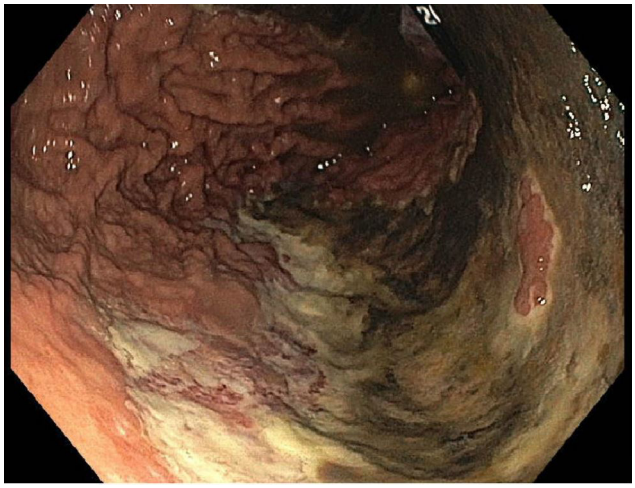

Figure 1 OGD showing discolouration and deep ulceration in the proximal stomach involving the anterior and posterior wall extending down to the lesser curvature of the stomach. OGD, oesophagogastroduodenoscopy.

TREATMENT

Intravenous broad spectrum antibiotics vancomycin, cefepime, metronidazole were started. Blood cultures were drawn which did not yield growth of any organisms. An OGD was performed 2 days after diagnosis of pneumatosis to assess extent of ischaemia and found diffuse ischaemic changes characterised by discolouration and deep ulceration of the proximal stomach involving the anterior and posterior wall with extension to lesser curvature of stomach. Nasogastric (NG) was placed to suction to alleviate the large volume of gastric secretions (1–2 L per day). He was treated with high-dose proton pump inhibitor (PPI) therapy with intravenous pantoprazole 40 mg three times a day. He was unable to tolerate postpyloric feeding via NJ tube due to abdominal pain and was initiated on total parenteral nutrition (TPN). A repeat CT abdomen done 5 days after initial diagnosis showed improvement in gastric pneumatosis. He completed a 10-day course of intravenous antibiotics. He was on TPN for a total of 21 and then transitioned to enteral tube feeds that were well tolerated. He was able to slowly restart eating by mouth and tolerated advancements of diet from clear liquid to solids. Enteral tube feeds were stopped day before discharge.

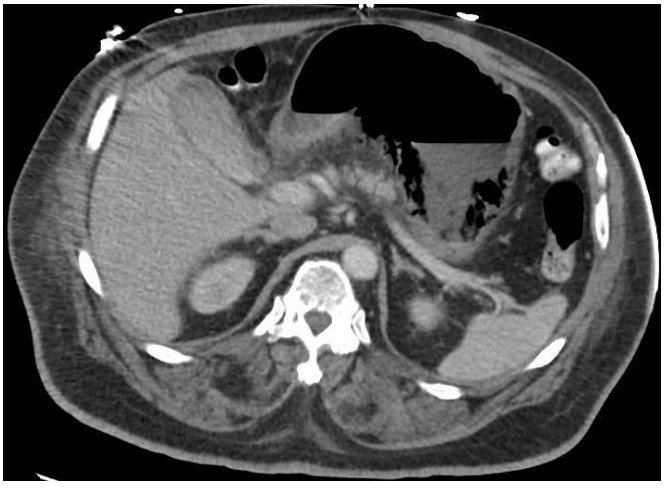

Figure 2 CT showing pneumatosis and mural thickening along the wall of the lesser curvature of the stomach.

OUTCOME AND FOLLOW-UP

The patient was hospitalised for a total of 33 days. On discharge, he was tolerating a low fat and low fibre diet. A repeat CT abdomen pelvis non-contrast prior to discharge showed persistent and similar appearing gastric pneumatosis. The patient was seen in the GI clinic 1.5 months after discharge and was noted to be feeling well overall with occasional bloating during meals. He continues to follow up with his primary care physician and gastroenterologist over a year after discharge.

DISCUSSION

Gastric pneumatosis has a variety of causes, including gastric ischaemia and infarction. Gastric ischaemia is a rare condition on its own, that may be under-reported, under-recognised and underappreciated.[2]

The aetiologies of gastric ischaemia are many and there are numerous risk factors that have been identified. One retrospective study separated aetiologies into local vascular (n=8; 47%), mechanical obstruction (n=5; 29%) and systemic hypoperfusion from shock (n=4; 24%). Local vascular causes include coeliac artery ostial stenosis, postsurgical complications, portal vein thrombosis and hepatic artery embolisation. Causes of mechanical obstruction include paraoesophageal hernias and gastric outlet obstruction.[4] Other rarer causes of gastric ischaemia that have been reported in case reports include disseminated intravascular coagulation,[5] severe mesenteric ischaemia,[6] antiphospholipid syndrome complicated by adrenal insufficiency due to bilateral adrenal gland infarction,[7] complication of endoscopic submucosal dissection for early gastric cancer[8] and following intra-arterial infusion of vasopressin into the left gastric artery.[9]

Known risk factors for gastric ischaemia include advanced age, smoking, atherosclerosis, hyperlipidaemia, diabetes, hypertension and portal hypertension.[10] Ageing and diabetes decrease gastric motility and delay gastric emptying which leads to gastric distention which then results in increased intragastric pressure.[11] This mechanism has been shown to decrease gastric blood flow in animal models.[12] Smoking increases acid and pepsin secretion and levels of oxygen-free radicals as well as decreasing prostaglandin synthesis, gastric blood flow and mucus secretion.[13] Portal hypertension decreases gastric blood flow due to vascular congestion.[10] Our patient had multiple risk factors for the development of gastric ischaemia after left gastric artery embolisation, including advanced age, atherosclerosis, diabetes and hypertension.

The most common presenting symptoms of gastric ischaemia are abdominal pain, GI bleeding and acute toxic metabolic encephalopathy. CT imaging may show evidence of gastric pneumatosis or portal vein air.[4] Diagnostic endoscopy is recommended for suspected gastric ischaemia because it can confirm the diagnosis, estimate severity of the ischaemia and rule out other pathologies.[2] The most common endoscopic findings are mucosal congestion and haemorrhagic (purplish hue) mucosa with or without ulceration, usually on the greater curvature of the body.[2]

Management options for gastric ischaemia are divided into surgical and conservative. Conservative approaches involve gastric acid suppression with PPIs, broad-spectrum antibiotics, NG tube decompression, fluid resuscitation and TPN.[4] Broad-spectrum antibiotic use is selective with some authors using antibiotics if there is presence of gastric pneumatosis on imaging.[2] Intervention can be surgical via gastric resection or with IR. For example, in the latter case, if the aetiology is hepatic artery ostial stenosis then a stent can be placed with IR.[4] Surgical

intervention is recommended for gastric perforation and gangrenous or necrotising gastritis that has not responded to conservative management as well as all cases of ischaemia secondary to gastric volvulus.[2] In one study, the median duration of hospitalisation was 15 days and the mortality was 24% within 6 months of diagnosis.[4]

Gastric ischaemia following embolisation is rare due to rich collateral supply to the stomach. If ischaemia does occur, it tends to occur after what is called 'blind embolisation', which involves the occlusion of several main gastric pedicles such as left gastric and right gastroepiploic arteries.[14] Even in these procedures' ischaemia tends to be temporary. Widespread necrosis and subsequent gastric pneumatosis from left gastric artery embolisation is rare and limited to case reports. One case reported transient and focal gastric infarction in a patient without underlying risk factors.[15] Another case reported full thickness necrosis of the gastric wall on postmortem examination, however, being the patient had died the ischaemia could have been due in large part to systemic hypotension rather than left gastric artery embolisation.[16]

Learning points

- Gastric ischaemia after embolisation of left gastric artery is rare due to rich collateral vascular supply.
- Endoscopy is required to confirm diagnosis, determine severity and rule out other pathologies.
- Conservative therapeutic approach involves gastric acid suppression via high-dose proton pump inhibitor, broad-spectrum antibiotics, nasogastric tube for decompression, fluid resuscitation and total parenteral nutrition.

Contributors MDB and ML contributed equally to the writing of this paper. MDB wrote the case segment and ML wrote the introduction and discussion. Both authors edited the entire abstract.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s)

Provenance and peer review Not commissioned; externally peer reviewed.

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