CASE REPORT

A 16-year-old boy with emphysematous gastritis and oesophageal candidiasis

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SUMMARY

Emphysematous gastritis is a rare and frequently fatal condition caused by invasion of gas-forming bacteria into the gastric wall. There have only been a handful of reported cases in the paediatric population, and none of these have evidence of candidal infection or mucormycosis. Patients typically present with abdominal pain, vomiting, melaena and haematemesis. Risk factors for emphysematous gastritis are those that interfere with the natural barriers to infection in the stomach. Diagnosis is made on the basis of typical appearances on abdominal CT. Treatment is generally conservative with surgery reserved for failed medical management or later complications. Antimicrobial cover should be broad with a low threshold for antifungals. It is important to look for predisposing factors for this condition, perhaps including an assessment of the patient's immunocompetency. We present a 16-year-old boy with global developmental delay who presented with this condition associated with candidal infection.

CASE PRESENTATION

Our patient was a 16-year-old boy with a history of global developmental delay, spastic quadriplegia and a ventriculoperitoneal shunt for hydrocephalus in situ. A gastrostomy had been inserted several years previously due to gastro-oesophageal reflux and feeding difficulties. The patient had had a right orchidectomy 7 days prior to presentation for an infarcted testis. He had received 5 days of oral co-amoxiclav following this. His only other medication was a regular laxative for constipation. The patient presented to our department with a short history of haematemesis, melaena and abdominal pain. The HR was 96, he was warm and well perfused, his respiratory rate was 20, he was afebrile and had oxygen saturations of 98%. On examination, he was found to be distressed and pale with a distended, generally tender abdomen but with no signs of peritonism. The site of his previous surgical incision looked healthy and not infected. Over the course of the next few hours the percutaneous endoscopic gastrostomy tube drained 'coffee-ground' fluid, he developed signs of peritonism and he became increasingly tachycardic (HR=155).

INVESTIGATIONS

A laboratory workup showed a haemoglobin of 8.4 g/dL, white cell count $27.2 \times 10^9/L$, with a neutrophil count $19.2 \times 10^9/L$. The international normalised ratio was 1.2 and activated partial thromboplastin time was 1.1. The C reactive protein was 56 but rose to 397 by the end of the

same day. Renal function tests revealed urea of $14.1 \mu\text{mol/L}$, but other parameters were within normal range. Liver function tests were normal including transaminases.

A CT of the abdomen and pelvis was performed, which showed the walls of the distal oesophagus and stomach were thickened with diffuse areas of intramural gas.

An oesophagogastroduodenoscopy (OGD) was undertaken to further evaluate these findings. This showed severe oesophageal candidiasis and inflammation around the gastro-oesophageal junction. There was no evidence of perforation. Biopsies were not taken for fear of causing iatrogenic perforation.

Blood cultures and swabs taken from the orchidectomy site were sterile. These were taken before antibiotics or antifungals.

Immunodeficiency screening tests: total protein 73 g/L (60–80), albumin 44 g/L (35–50), IgG 6.53 g/L (6–16), IgM 1.35 g/L (0.5–1.9), IgA 3.83 g/L (0.8–2.8), IgG1 2.96 g/L (3.2–10.2), IgG2 3 g/L (1.2–6.6), IgG3 1.25 g/L (0.2–1.9), IgG4 0.77 g/L (0.1–1.3), CD4 35% (25–48), CD4 total $1900 \times 10^6/L$ (400–2100), CD8 50% (9–35), CD8 total $2670 \times 10^6/L$ (200–1200), CD4:CD8 ratio 0.7 (0.9–3.4), response to vaccines Hib/DPT normal.

DIFFERENTIAL DIAGNOSIS

- Emphysematous gastritis
- Gastric emphysema

OUTCOME AND FOLLOW-UP

Our patient was managed with intravenous fluid boluses, omeprazole and a 15mL/kg packed red cell transfusion. Following this he remained cardiovascularly stable. He was initially started on teicoplanin, gentamicin and tazocin (tazobactam and piperacillin). Following the results of the OGD he was also started on fluconazole intravenously. The antibiotics and antifungals were continued for a total of 14 days. Feeds were initially withheld but were gradually restarted on day 2 of the illness and he was fully enterally fed by day 7 of the admission. Blood parameters gradually returned to normal, and there was no further evidence of enteral bleeding.

DISCUSSION

Emphysematous gastritis is a rare and frequently fatal condition caused by invasion of gas-forming bacteria into the gastric wall. There have only been a handful of reported cases in the paediatric population, and none of these have evidence of candidal

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infection or mucormycosis.^{1 2} The first description of emphysematous gastritis was in 1889.³ The condition has a poor prognosis with mortality of 55–61%.^{4 5} Patients with emphysematous gastritis will present with acute abdominal pain often accompanied by nausea, vomiting and diarrhoea as well as haematemesis and melaena.⁶ Physical examination reveals a toxic appearance, an acute 'surgical' abdomen with distension, guarding and absent or decreased bowel sounds. Laboratory investigations typically reveal a leucocytosis and a metabolic acidosis. Liver enzymes and alkaline phosphatases may also be raised.⁷ Diagnosis is typically made on the basis of radiological imaging. The most useful medium is CT, which typically shows gastric wall thickening and intramural gas.^{6 7} The gas in the wall of the stomach typically collects around the fundus and the greater curvature and forms irregular bubbles⁶ (figures 1 and 2). Endoscopy can be helpful and may show bubbles beneath the gastric mucosa or features of necrosis.⁷

Emphysematous gastritis should be distinguished from gastric emphysema. Gastric emphysema is a much more benign condition that in itself normally requires no treatment. It is due to mechanical redistribution of air from the lumen of the stomach into the gastric wall. Typically this occurs following interventions such as nasogastric tube insertion or instrumentation. It can also occur in situations where there is raised intraluminal pressure such as distal obstruction or forceful vomiting.⁷

Figure 1 The abdominal CT showing the typical appearances of emphysematous gastritis. There is prominent gastric intramural gas seen. A gastrostomy tube is also in situ.

Figure 2 the abdominal CT showing the typical appearances of emphysematous gastritis. There is prominent gastric intramural gas seen. A gastrostomy tube is also in situ.

There have been a number of predisposing factors for emphysematous gastritis identified in the literature. Risk factors for emphysematous gastritis are those that interfere with the natural barriers to infection in the stomach. Factors that have been identified include: ingestion of corrosives, alcohol abuse, recent abdominal surgery and any condition affecting the immunocompetency of the host.^{6 7} In our case no predisposing factors were identified. The causative organisms can be Gram positive, Gram negative, anaerobic bacteria or sometimes fungal.^{6 7} Although rare, fungal mucormycosis in the context of emphysematous gastritis has been described in the literature, although never in children. In these patients *Candida* is the most common culprit.⁶ Since *Candida* is not gas forming it is likely that in these cases there is concomitant bacterial invasion of the stomach wall. In our case, we cannot prove invasive candidal disease but we presumed it on the basis of the macroscopic endoscopic findings. Current consensus is conservative management with broad-spectrum antibiotics and antifungals along with aggressive fluid management; surgery is reserved for those cases resistant to medical treatment.^{6 7} In the longer term there is the risk of gastric strictures, which may require surgical intervention.

Learning points

- ▶ Diagnosis is made on the basis of a typical history and appearances on CT.
- ▶ Treatment of emphysematous gastritis is normally supportive with surgery reserved for failed medical management or later complications.
- ▶ Antimicrobial cover should be broad with a low threshold for antifungals.
- ▶ The cause of this condition should be considered and may include work up for an immunodeficiency.

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Competing interests None.

Patient consent Obtained.

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