Acute gastric necrosis after routine oesophagogastroduodenoscopy with therapeutic argon plasma coagulation

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ABSTRACT

A 56-year-old woman presented to the accident and emergency department with peritonitis 2 days after a routine oesophagogastroduodenoscopy. She was taken to theatre with the finding of gastric necrosis. Blood and peritoneal cultures grew group A haemolytic *Streptococcus*. Histology revealed normal vasculature, no volvulus but marked neutrophilia in the submucosa with an intact mucosa. The stomach was resected and the patient recovered in the intensive care unit but overwhelming acidosis progressed to multiorgan failure and treatment was eventually withdrawn.

Acute phlegmonous gastritis has been well described in the literature but mainly before the advent of antibiotics. The most common organism is group A haemolytic *Streptococcus* (commonly found in throat infections) and predisposing factors include instrumentation. Should antibiotics be given at the start of an oesophagogastroduodenoscopy and should routine procedures be delayed if active upper respiratory tract infections are present?

KEYWORDS

Gastric necrosis – Endoscopy – Gastrectomy

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Case history

A 56-year-old Caucasian woman presented to the accident and emergency department with a 1-day history of severe generalised abdominal pain radiating through to her back. She was tachycardic at 125 beats per minute with a blood pressure of 51/35mmHg and a temperature of 37.6°C. On examination, her abdomen was grossly tender with guarding and rebound tenderness over the epigastrium. Digital rectal examination was unremarkable. Initial blood tests revealed a haemoglobin level of 12.1g/dl, leucocytes of 3,000/mm³ and a platelet count of 222,000/mm³. Other laboratory studies revealed raised urea of 8.1µmol/l, a creatinine level of 214µmol/l, a C-reactive protein level of 123mg/l, with normal liver function tests and an amylase level of 42u/l. The clotting function was deranged with an international normalised ratio of 2.4 and an activated partial thromboplastin time ratio of 2.8.

Two days prior to admission, the patient underwent an uneventful elective oesophagogastroduodenoscopy and argon plasma coagulation (APC) of known gastric antral vas-

cular ectasia causing anaemia (Fig 1). She had had three previous APC treatments without complication. Her past medical history included longstanding rheumatoid arthritis (controlled with 2.5mg of methotrexate four times a week), Sjögren's syndrome and a hiatus hernia.

The patient deteriorated and was transferred immediately to theatre. A laparotomy revealed three litres of turbid ascites and complete gastric necrosis with no perforation or acute dilatatory changes. The necrosis was confined solely to the stomach and extended from the fundus to the antrum. A total gastrectomy was performed with transection of the stomach just below the gastro-oesophageal junction and at the pylorus. A Foley catheter was inserted into the lower oesophagus for drainage and a feeding jejunostomy was inserted into the proximal jejunum. Broad spectrum antibiotics (piperacillin/tazobactam, metronidazole and gentamicin) were given.

The patient was transferred to the intensive care unit postoperatively with the intention of a second-look laparotomy and gastrointestinal reconstruction at a later stage

Figure 1 Oesophagogastrroduodenoscopy of gastric antral vascular ectasia: characteristic prominence of vasculature (taken at the time of the argon plasma coagulation, two days before admission)

Figure 2 Haematoxylin and eosin stain illustrating dense inflammatory changes in all layers (with neutrophilia particularly noted in the submucosa)

Figure 3 Gram stain illustrating Gram-positive cocci in the submucosa

when she was more stable. Microbiology cultures of the peritoneal fluid and blood grew a group A haemolytic *Streptococcus* sensitive to penicillin. Despite appropriate antibiotics and aggressive resuscitation, the patient deteriorated and died.

Histological examination of the gastrectomy specimen revealed a thickened and oedematous gastric wall (Fig 2) with a heavy infiltration of neutrophils throughout the submucosa and widespread necrosis of the muscularis propria, in keeping with acute phlegmonous gastritis. Gram-positive cocci were identified in the submucosa (Fig 3) and no granulomas were seen. There was no evidence of thrombosis or vasculitis to account for the ischaemia.

Discussion

Gastric ischaemia occurs rarely due to the vascularity of the organ and the vascular reserve of intramural anastomosis.¹ The possible causes of gastric ischaemia include vascular, chemical, mechanical and septic. Infection is the rarest cause of gastric ischaemia but a life threatening form is acute phlegmonous gastritis,² which can best be described as a condition of diffuse cellulitis confined to the stomach. Patients usually present with abdominal pain, fever, nausea, vomiting and, occasionally, pus in the vomitus, which is said to be pathognomonic.⁵ The condition is usually fatal.

Various routes of infection have been implicated, including haematogenic with/without direct contamination of the gastric lumen due to ingestion of infected secretions

from the throat and lung.⁴ The most common pathogens are *Streptococcus* spp, *Staphylococcus* spp, *Escherichia coli*, *Haemophilus influenzae*, *Proteus* and *Clostridia*.⁵ While the pathogenesis of the disease remains unclear, there is often evidence of preceding mucosal injury with cases reported after endoscopic intervention, chronic gastritis, peptic ulcer disease and in association with general debility.⁶

Examination of histopathological specimens following APC show that the heat induced changes are confined to the submucosa, making it safe to coagulate tissues even with high outputs and prolonged application.⁷ Any mucosal injury, however, provides a potential route for bacterial translocation. A possible explanation for the series of events in this case is that the group A haemolytic *Streptococcus* isolated in the blood cultures was introduced into the gastric mucosa by endoscopic mucosal injury caused by the APC. The immunosuppression caused by the combination of chronic ill health (rheumatoid arthritis and Sjögren's syndrome) and long-term use of methotrexate may have contributed to the subsequent fulminating bacterial infection.

Conclusions

Acute phlegmonous gastritis is a modern day rarity with most cases described before the advent of antibiotics. Nevertheless, over recent years, there has been a rise in incidence in the Western world.⁸ With ever increasing numbers of endoscopies performed and more therapeutic interventions undertaken, it is important to be aware of this potentially devastating complication of endoscopy.

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