Acute Necrotizing Gastritis with Gangrene: A Case Report

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An 80-year-old man presented with signs of peritonitis. Laparotomy revealed gangrene of the stomach and abdominal esophagus without obvious cause. The patient underwent total gastrectomy with partial esophagectomy. Bacterial culture of the peritoneal fluid grew Morganella morganii and Proteus mirabilis. The patient died of irreversible sepsis on the second postoperative day.

Key words: necrotizing gastritis, gangrene, stomach

Introduction

Gangrene of the stomach is a rare and fulminating catastrophic event that is often fatal. Its cause has been attributed to embolization of atherosclerotic plaque, thrombosis of major arterial supply, occlusion of gastric vessels by therapeutically injected foreign bodies, psychogenic polyphagia resulting in massive gastric dilatation, ingestion of corrosive materials, intrathoracic herniation of the stomach through the diaphragm, gastric volvulus, and necrotizing gastritis caused by organisms.

We report on a man with stomach gangrene that appeared to be caused by a severe necrotizing infection with no known portal of entry.

Case Report

An 80-year-old, previously healthy man, was sent to our emergency room with the chief complaint of abdominal distension and pain for one day. On examination, the patient was afebrile, tachycardic (110 beats/min), tachypneic (26 breaths/min), with a systolic blood pressure of 90 mmHg. His abdomen was distended, guarded and rigid, with diffuse rebound tenderness and absent bowel sounds. White blood cell count was 18,700/mm³, chest and abdomen x-rays showed no gas under the diaphragm and no air-fluid levels.

With a clinical diagnosis of peritonitis, an exploratory laparotomy was performed after initial resuscitation. When the peritoneum was opened, about 1000 ml of brown hemorrhagic, foul-smelling fluid could be seen in the peritoneal cavity. The whole of the stomach and abdominal esophagus were black in color, the walls appearing paper-thin and friable. Pulsations of major gastric arteries were present. The liver, spleen, duodenum, gallbladder, pancreas, small bowel and colon all appeared normal. The diaphragmatic domes were normal and there was no evidence of gastric volvulus. En-bloc resection of the entire stomach, omentum and distal esophagus was performed; cervical esophagostomy and tube jejunostomy were created. Immediate reconstruction for alimentary continuity was not performed due to the critical condition of the patient. The patient died of septic
shock on the second postoperative day.

Gross examination of the specimens revealed gangrenous, black discoloration, thinning and absent rugal folds in the entire stomach and abdominal esophagus; the proximal esophagus and omentum appeared uninvolved. (Fig. 1). Microscopic examination of the stomach showed destruction of the wall by a diffuse necrotic process extending through all layers. An extensive purulent process had completely destroyed the mucosa and had invaded the submucosa, with massive accumulation of inflammatory cells and necrosis of the underlying muscularis. The esophagus revealed detachment of the squamous epithelium and acute inflammation of the mucosa and muscle layers. The omentum was unremarkable.

Cultures of the peritoneal fluid taken at the time of surgery grew *Morganella morganii* and *Proteus mirabilis* that were sensitive to cefuroxime and gentamicin.

**Discussion**

The abundant and anastomotic nature of the stomach’s vascular supply makes gangrene very rare. In 1943, Babkin et al (1) found that tying all the gastric arteries did not cause gastric infarction in the dog. The lack of infarction was explained by the presence of multiple anastomoses between the left gastric artery and branches of the phrenic and esophageal arteries. However, Harvey et al (2) reported a case of multifocal gastric infarction secondary to atheromatous emboli originating in a thoracic aortic aneurysm. In another report (3), a
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A patient was described with extensive gastric necrosis after therapeutic transcatheter embolization of the left gastric artery with fragments of gelatin sponge for recurrent massive upper gastrointestinal hemorrhage. Ovnat et al. (4) reported three cases of acute obstruction of the celiac trunk. The first patient was diagnosed by abdominal computed tomography (CT) and angiography, and was treated successfully with thrombolytic therapy. The other two patients underwent explorative laparotomy to establish the diagnosis: the most impressive finding was that all of the gastric mucosa along the lesser curvature was necrotic but the appearance of the stomach was only mildly ischemic. Both these patients required total gastrectomy and splenectomy.

In our case, the finding of gangrene of the stomach was an operative surprise. Pulsatile gastric arteries made vascular accident unlikely. Both the domes of the diaphragm were normal, which ruled out a diaphragmatic hernia content strangulation as the cause of the gastric gangrene. No twisting of the stomach was noted, and so volvulus was ruled out. There was no history or evidence of swallowing any corrosive substance.

The peritoneal cavity fluid grew *Morganella morganii* and *Proteus mirabilis*. Transmigration of organisms into the peritoneal cavity across the gangrenous stomach was considered but, normally, the contents of the stomach are sterile. Therefore, we suspect that the stomach wall was involved as a result of some necrotizing infection.

Abscess or spreading cellulitis of the stomach wall caused by microorganisms, known as phlegmonous gastritis, is a rare condition, with only about 500 cases having been reported in the world literature. The pathogenesis is unclear, although predisposing factors include chronic gastritis, increased age, alcoholism, hypoacidity, protein-energy malnutrition and immunosuppression (5-7). Phlegmonous gastritis may arise from a local or disseminated hematogenous infection, and may involve a portion of the stomach (localized type) or the entire stomach (diffuse type). The most frequent causative agents, in order of frequency, are *Streptococcus, Staphylococcus, Escherichia coli, Haemophilus influenza*, Proteus and Clostridia. Mixed bacterial infections have also been reported (8).

Patients with acute phlegmonous gastritis have severe upper abdominal pain with associated fever, nausea and vomiting. The pain usually increases in severity as the abscess enlarges, does not radiate and is non-colicky in nature. Physical findings include fever, signs of peritoneal irritation and, occasionally, a palpable mass (5, 8). Diagnosis may be delayed due to the lack of typical signs and this, combined with the rapid progression to peritonitis, often results in a fatal outcome. Surgical intervention with gastrectomy is thought to be the most effective form of treatment. At laparotomy, the stomach is usually found to have intact extrinsic blood supply, with thrombosis of the microscopic intrinsic vascular plexus producing the appearance of extensive infarction. There is dark discoloration of the stomach wall and sloughing of the mucosa. The submucosa is the layer most characteristically involved by contiguity but necrosis is rare and, if it occurs, is usually focal.

Acute necrotizing gastritis is a variant of phlegmonous gastritis, with organisms producing necrosis and gangrene of the stomach wall rather than just an intramural abscess. Etiologically, *Streptococci*, fusiform and spirochetal organisms (commonly found in the mouth), or combinations of various organisms have been reported.

Diagnosis of acute necrotizing or gangrenous gastritis is usually made at laparotomy, although endoscopy, endosonography (9) and endoscopic snare biopsy have also been used to reach a diagnosis. However, in a patient with frank signs of peritonitis, as in the present case, these are not feasible. Phlegmonous involvement of segments of the gas-
trointestinal tract other than the stomach is extremely rare: involvement of the esophagus\textsuperscript{(10)}, small intestine\textsuperscript{(11-13)} and colon\textsuperscript{(12,13)} have been reported. Our case had diffuse involvement of the stomach and the abdominal esophagus.

During the last 50 years, there have been reports of the successful treatment of patients with phlegmonous gastritis by medical therapy alone\textsuperscript{(14,15)}. Overall, the mortality rate is 17\% for patients with a medically treated localized disease and 60\% for the diffuse disease. Depending on the clinical situation, patients with the localized disease may respond to prompt and aggressive treatment. Polymicrobial infection occurs in nearly a third of cases, so initial treatment should include broad-spectrum antibiotic coverage (i.e. ampicillin/subbactam and ciprofloxacin). Nevertheless, the combination of antibiotic therapy and early gastric resection offers the best prospect for patient survival. The mortality rate of patients treated by surgical resection is far lower than for those treated by medical therapy alone (20\% vs. 50\%)\textsuperscript{(10)}.

In conclusion, acute phlegmonous or necrotizing gastritis is a rare condition and clinical diagnosis may be difficult. Streptococcus is the most frequent organism identified but polymicrobial infection has also been found. Early diagnosis and prompt treatment are essential to reduce the mortality rate.

References

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急性壞死性胃炎合併胃壁壞疽：病例報告

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胃壁壞疽是一種非常罕見而致命的疾病。其原因包括：動脈粥狀硬化造成的栓塞，胃部主要血管的血栓，治療性的胃血管栓塞，心因性嗜食症造成的胃極度擴張，腐蝕劑吞食傷，橫膈膜疝氣，胃扭轉，以及細菌感染引起的壞死性胃炎。

本病例報告一名八十歲男性因腹膜炎接受緊急手術，術中發現全部胃及腹部食道呈現嚴重壞疽，雖作廣泛切除，病患仍於術後二日死於敗血症，茲就此罕見病例提出報告並作相關文獻之回顧。

關鍵詞：壞死性胃炎，胃，壁壞疽