

Gastric necrosis and perforation caused by acute gastric dilatation

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ABSTRACT

Acute gastric dilatation was first defined by Duplay in 1833. We herein present the case of a 55-year-old male patient diagnosed with gastric necrosis and perforation caused by acute gastric dilatation. Since the stomach has a rich blood circulation, necrosis and perforation are rarely seen. Clinically, more than 90% of cases have complaints of vomiting. The most useful method in revealing the diagnosis and aetiology is computerized tomography. Medical treatment is appropriate for cases where no necrosis and peritonitis findings are detected through endoscopy. Delays in surgical treatment increase the risk of mortality.

Key Words: Gastric perforation, gastric necrosis, gastric dilatation

INTRODUCTION

Gastric dilatation leading to necrosis and perforation is very rare (1, 2). It often occurs as a postoperative complication. Failure to diagnose the condition or presentation of the patient to the hospital during the late period may cause necrosis and rupture that may lead to death (2). The pathogenesis is still controversial (3). The most frequent complaints are abdominal pain and vomiting, and are usually accompanied by abdominal bloating (4). Emergency surgery is required in patients with necrosis and perforation. Herein we presented a patient who presented to our emergency department with complaints of vomiting and abdominal pain, and was diagnosed with necrosis and perforation of the stomach due to acute gastric dilatation.

CASE PRESENTATION

A 55-year-old male patient was admitted to our emergency department with complaints of nausea, vomiting and abdominal pain that started about 4 days ago. His past medical history revealed diabetes mellitus for 10 years, and he was also receiving dialysis for chronic renal failure since 3 years. On physical examination, the patient was anxious, tachycardic and hypotensive. He did not have fever. The abdomen was distended and there were no bowel movements on auscultation. He had guarding and rebound tenderness. The leukocyte count was higher than 14,000/mm³. The plain abdominal X-ray in the lateral decubitus view showed presence of free air within the abdomen (Figure 1). The abdominal computed tomography (CT) revealed free air and fluid in the abdominal cavity, and intramural air bubbles within the gastric wall (Figure 2).

The patient was taken to an emergent laparotomy with a diagnosis of acute surgical abdomen. On exploration, the entire greater curvature of the stomach was found to be completely necrotic and perforated (Figure 3). Although there were excessive gastric contents in the abdomen, the perforated stomach was also dilated and filled with undigested food. There were no volvulus or adhesions. The celiac trunk and the superior mesenteric artery were patent. Based on these findings, the preliminary diagnosis was acute gastric dilatation induced ischemia and perforation. Total gastrectomy and Roux-Y esophagojejunostomy was performed. When the specimen was dissected, it was observed that the entire stomach, except the cardia, was necrotic (Figure 4). The pathological examination was interpreted as ischemic gastropathy.

After the operation, the patient was admitted to the intensive care unit. He died on the third postoperative day due to sepsis and pneumonia.

DISCUSSION

Duplay first described acute gastric dilatation in 1833 (1). Necrosis and perforation of the stomach is very rare, because the stomach has a rich circulatory system. The majority of reported cases are from postoperative patients. Other reasons include gastric outlet obstruction, retroperitoneal tumors, anorexia

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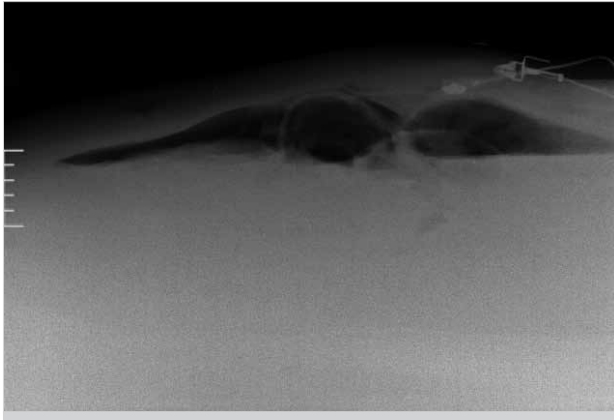


Figure 1. Free intraabdominal air on lateral abdominal X-ray



Figure 2. Free intraabdominal air and intramural air within gastric wall on computed tomography

nervosa and bulimia, psychogenic reasons, diabetes mellitus, trauma, electrolyte disorders, cerebral palsy, diaphragmatic hernia, and gastric volvulus (2, 3). The pathogenesis is still controversial (2, 4). Our patient had a history of diabetes mellitus for 10 years and he was on dialysis for chronic renal failure since 3 years.

Clinically, more than 90% of cases present with vomiting. Epigastric fullness, abdominal distention and pain may also be present (5-8). Our patient was admitted to the emergency department with complaints of nausea, vomiting and severe abdominal pain.

On physical examination, distension, epigastric tenderness, guarding and rebound tenderness can be determined. The abdominal X-ray may reveal a large air-fluid level that belongs to the fundus of the stomach, and subdiaphragmatic free air in cases with perforation. The most useful imaging modality is abdominal computed tomography, which is not only useful in diagnosis but also in revealing the etiology (6). Endoscopy is important for visualization of the gastric mucosa (7). On examination, our patient had guarding and rebound tenderness. Intra-abdominal free air was detected on lateral view abdominal X-ray. The abdominal CT revealed free air and fluid in the abdominal cavity, and intramural air bubbles within the gastric wall.

The excessive increase in stomach contents by acute gastric dilatation compresses the gastric wall and leads to necrosis



Figure 3. Complete necrosis of the greater curvature and site of perforation

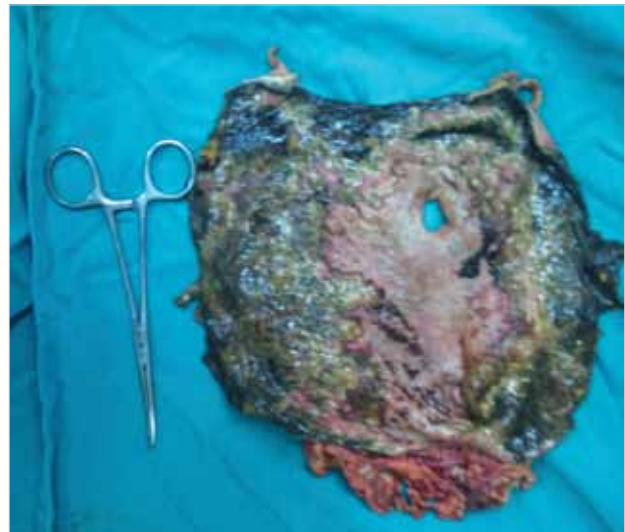


Figure 4. The gastric specimen

and perforation of the stomach. It also leads to compression on the inferior vena cava, and causes hypotension due to the decrease in venous return. Acute gastric dilatation can be controlled by nasogastric decompression, and appropriate fluid-electrolyte therapy. Upper gastrointestinal endoscopy can help decompression. Medical treatment is suitable in cases without signs of peritonitis on examination and without signs of necrosis on endoscopy (1, 2).

Advanced cases with necrosis and perforation require surgical intervention (2). In the literature, the majority of acute massive gastric dilatations have been surgically treated (1, 2, 9). Mortality rates after delayed surgery in patients with necrosis and perforation are reported to be as high as 80% (9). Surgical treatment options include surgical decompression, partial gastrectomy, total gastrectomy and esophago-jejunostomy, total gastrectomy with cervical esophagostomy and feeding jejunostomy (1, 2, 4, 8). On laparotomy, the entire greater curvature of the stomach was ischemic and perforated. Therefore, total gastrectomy and Roux-Y esophago-jejunostomy was

performed. The patient died on the third postoperative day due to sepsis and pneumonia.

CONCLUSION

Acute massive gastric dilatation is rare. Early diagnosis is important. CT is important in revealing the diagnosis and etiology. Medical treatment can be attempted in patients without signs of peritonitis on examination and without signs of necrosis on endoscopy. Mortality rate is high if surgical treatment is delayed.

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