Case Report

Necrotizing gastritis: a case report

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ABSTRACT

Gangrene of the stomach is a rare and often fatal disease. It occurs when there is vascular anomalies, gastric volvulus or herniation and in infectious gastritis. A 65-year-old man was wheeled into the Emergency Department with complaints of abdominal pain for 8 hours. The pain was in the epigastric region which was severe in nature continuous and associated with nausea. There was no history of hematemesis or vomiting. The past medical history was not significant. He was HIV negative. On examination, he was in agonizing pain and tachypniec. The vital signs were temperature 98.6°F, pulse 118/min, respiratory rate 28/minutes, BP 90/60 mmHg. Abdominal examination found diffuse tenderness with voluntary guarding and rigidity. Bowel sounds were sluggish. Rectal examination was normal. The Ryle’s tube showed hemorrhagic aspirate. Erect X-ray abdomen showed large gastric air fluid level and no evidence of pneumoperitoneum. Routine hemogram showed leukocytosis. Blood sugar level and serum urea, creatinine levels were within normal limits. After initial resuscitation with intravenous fluids and antibiotics, decision was taken to proceed for an emergency exploratory laparotomy. Gastric necrosis is a rare and fatal condition. Etiology includes thromboembolism and occlusion of major arterial supply, ingestion of corrosive agents, volvulus of the stomach, herniation of the stomach through the diaphragm, bulimia nervosa, iatrogenic gelfoam embolism, endoscopic hemostatic injections and infectious gastritis. So, the most probable cause of the gastric necrosis was infectious gastritis. It begins as phlegmonous (suppurative) gastritis (PG), and then it progresses to the lethal severe form: acute necrotizing gastritis. The few case reports of gastric gangrene are there in literature with the underlying etiological factors. Harvey et al, reported a rare case of multifocal infarction of stomach secondary to atheromatous emboli originating in a thoracic aortic aneurysm. Bradley et al, reported a case of extensive gastric necrosis in a patient with recurrent massive upper gastrointestinal hemorrhage. They found massive gastric gangrene secondary to therapeutic transcatheater embolization of the left gastric artery with fragments of gelatin sponge. Ovnat et al, reported three cases of acute obstruction of the celiac trunk. In all the three patients, the mucosa along the lesser curvature of the stomach was necrotic but the gross appearance of the stomach was only mildly ischemic. Organisms isolated from the gastric wall include hemolytic streptococci, proteus, E. coli and clostridium welchi. PG can be diagnosed by upper gastrointestinal endoscopy, CT scan, or endoscopic ultrasound. Its endoscopic findings can show purple colored gastric mucosa covered with dirty necrotic materials. Absolute diagnosis is made, most frequently at laparotomy. Gastric gangrene due to necrotizing gastritis is a rare and fatal disease. The diagnosis is usually made at laparotomy. Treatment consists of resection and feeding tube placement followed by intravenous antibiotics. Increased awareness of this rare entity may lead to more prompt diagnosis and an increased chance for patient survival.

Keywords: Gastric necrosis, Necrotizing gastritis, Surgery
INTRODUCTION

The stomach is very resistant to ischemia due to its rich blood supply. Of the several causes of gastric necrosis, the rarest is acute necrotizing gastritis which appears to be a variant of phlegmonous gastritis. In acute necrotizing gastritis all four major gastric vessels are patent, but gastric gangrene occurs secondary to a necrobiotic infection. We report a case of a 65-year-old man who presented with gastric gangrene without any vascular, chemical or mechanical etiological causes.

CASE REPORT

A 65-year-old man was wheeled into the Emergency Department with complaints of abdominal pain for 8 hours. The pain was in the epigastric region which was severe in nature continuous and associated with nausea. There was no history of hematemesis or vomiting. The past medical history was not significant. He was HIV negative. On examination, he was in agonizing pain and tachypneic. The vital signs were temperature 98.6°F, pulse 118/minute, respiratory rate 28/minute, BP 90/60 mm Hg. Abdominal examination found diffuse tenderness with voluntary guarding and rigidity. Bowel sounds were sluggish. Rectal examination was normal. The Ryle’s tube showed hemorrhagic aspirate. Erect X-ray abdomen showed large gastric air fluid level and no evidence of pneumoperitoneum (Figure 1).

Routine hemogram showed leukocytosis. Blood sugar level and serum urea, creatinine levels were within normal limits. After initial resuscitation with intravenous fluids and antibiotics, decision was taken to proceed for an emergency exploratory laparotomy.

On exploration, the peritoneal cavity had no free fluid demonstrable and all other quadrants of the abdomen was inspected. There was gangrene of the proximal one third of the stomach including fundus, body and greater curvature of the stomach extending up to the pylorus distally and proximally up to the lower one third of esophagus which was paper thin (Figure 2) and friable with thick cherry black color fluid oozing out (Figure 3).

Figure 1: Plain X-ray of the abdomen showing a large gastric air fluid level and no pneumoperitoneum.

Figure 2: Gangrene of the proximal one third of the stomach including fundus, body and greater curvature of the stomach extending up to the pylorus distally and proximally up to the lower one third of esophagus which was paper thin and friable.

Figure 3: Thick cherry black color fluid oozing out.

There was normal pulsation of the gastric vessels. The upper and mid third of the esophagus, small intestine, colon and rectum were pink and viable. The pyloric stump was closed in two layers. The proximal small gastric stump was closed after insertion of a tube drain and was followed by a feeding jejunostomy and the
abdomen was closed. Definitive surgery was not done at this time because the patient’s condition was unstable during surgery and secondly there was doubt whether the gangrene will spread further. Resected specimen of the gangrenous stomach (Figure 4).

Histopathological examination of the resected stomach showed hemorrhage and necrosis. After 6 weeks, the patient underwent a second stage operation. Roux en y esophagojejunostomy was done as depicted (Figure 5) Post-operative recovery was uneventful. Barium swallow was performed on the 14-postoperative day. There was no leak. The patient was discharged home and is currently under follow up.

DISCUSSION

Gastric necrosis is a rare and fatal condition. Etiology includes thromboembolism and occlusion of major arterial supply, ingestion of corrosive agents, volvulus of the stomach, herniation of the stomach through the diaphragm, bulimia nervosa, iatrogenic gelfoam embolism, endoscopic hemostatic injections and infectious gastritis. So the most probable cause of the gastric necrosis was infectious gastritis. It begins as phlegmonous (suparative) gastritis (PG), and then it progresses to the lethal severe form: acute necrotizing gastritis. The few case reports of gastric gangrene are there in literature with the underlying etiological factors. Harvey et al, reported a rare case of multifocal infarction of stomach secondary to atheromatous emboli originating in a thoracic aortic aneurysm. Bradly et al, reported a case of extensive gastric necrosis in a patient with recurrent massive upper gastrointestinal hemorrhage. They found massive gastric gangrene secondary to therapeutic transcatheter embolization of the left gastric artery with fragments of gelatin sponge. Ovnat et al, reported three cases of acute obstruction of the celiac trunk. In all the three patients the mucosa along the lesser curvature of the stomach was necrotic but the gross appearance of the stomach was only mildly ischemic. Organisms isolated from the gastric wall include hemolytic streptococi, proteus, E. coli and clostridium welchi. PG can be diagnosed by upper gastrointestinal endoscopy, CT scan, or endoscopic ultrasound. Its endoscopic findings can show purple colored gastric mucosa covered with dirty necrotic materials. Absolute diagnosis is made, most frequently at laparotomy.

CONCLUSION

Gastric gangrene due to necrotizing gastritis is a rare and fatal disease. The diagnosis is usually made at laparotomy. Treatment consists of resection and feeding tube placement followed by intravenous antibiotics. Increased awareness of this rare entity may lead to more prompt diagnosis and an increased chance for patient survival

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REFERENCES

1. Stein LB, Greenberg RE, Ildardi CF, Kurtz L, Bank S. Acute Necrotizing gastritis in a patient with peptic ulcer disease. Am J Gastroenterol. 1989;84:1552-4.
2. Strauss RJ, Friedman M, Platt N, Gassner W, Wise L. Gangrene of the stomach: a case of acute necrotizing gastritis. Am J Surg. 1978;135:253-7.
3. Richieri JP, Pol B, Payan MJ. acute necrotizing ischemic gastritis: clinical, endoscopic and
histopathologic aspects. Gastrointestinal Endoscop. 1998;48:210-2.
4. Dharap SB, Ghag G, Biswas A. Acute necrotizing gastritis. Ind J Gastroenterol. 2003;22:150-1.
5. Harvey RL, Doberneck RC, Black WC. Infarction of the stomach following atheromatous embolization. Gastroenterol. 1972;62(3):469-72.
6. Bradley 3rd EL, Goldman ML. Gastric infarction after therapeutic embolization. Surg. 1976;79(4):421-4.
7. Ovnat A, Dukhno O, Pinsk I, Shaked G, Levy I. Acute obstruction of the celiac trunk. J Clin Gastroenterol. 2005;39(7):647.
8. Soon MS, Yen HH, Soon A, Lin OS. Endoscopic ultrasonographic appearance of gastric emphysema. World J Gastroenterol. 2005;11(11):1719.