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A RARE CASE OF GASTRIC GANGRENE IN A YOUNG PATIENT

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ABSTRACT

Gastric necrosis is rare. Without proper diagnosis and treatment, potentially fatal events such as gastric haemorrhage, perforation, and other serious complications can occur. We report a case of idiopathic gastric gangrene in 16 years old male patient, admitted with complaints of abdominal pain since one day.

KEY WORDS: gastric haemorrhage, perforation.

INTRODUCTION

The stomach is resistant to gangrene and ischemia, as it is highly vascular organ due to numerous vascular anastomosis. The etiological causes of gangrene of stomach can be vascular, chemical, mechanical or infectious agents. We report a case of a young boy who presented with gastric gangrene without any vascular, chemical or mechanical etiological causes.

CASE REPORT

A 16 year old boy presented in the emergency, with abdominal pain for 24 hours. The pain was in epigastrium, severe, continuous in nature and was associated with nausea and vomiting. There was no history of ingestion of any corrosive liquid. There was no previous history of any acute abdominal pain of similar nature. Family history was insignificant. On examination, the patient was febrile with tachycardia (110 beats/min), tachypnea (26 breaths/min) with a systolic blood pressure of 90 mm of Hg.

His abdomen was distended with generalized guarding and rigidity. Rebound tenderness and absent bowel sounds were noted on auscultation. On laboratory investigation, white blood cell count was 22,000/mm³, while the serum creatinine and urea levels were within the normal limits. Chest and abdomen X-rays showed no gas under the diaphragm and no air fluid levels.

An exploratory laparotomy was performed after initial resuscitation. The brown hemorrhagic fluid was drained from the peritoneal cavity. The proximal one-third of stomach was black in color, consistent with proximal gastric necrosis and affected part of stomach appeared paper thin, distended and friable. The pulsation of major gastric arteries were present. All other organs, liver, spleen, duodenum, esophagus, gall bladder, pancreas, small bowel and colon appeared normal. No volvulus or the diaphragmatic hernia was noted. The excision of proximal part of stomach was done after ensuring bleed from cut ends of the stomach. The distal stomach was tabularized and anastomosed with the divided end of the abdominal esophagus.

We received a partial gastrectomy specimen. On gross examination, the partial gastrectomy specimen measured 12x10x4cm (fig 1). The distal resected end measured 12 cm in diameter while the proximal resected end measured 8 cm in diameter. Gangrenous part measured 8x6 cm, was papery thin, friable with blackish discoloration. Focally rugal folds were flattened.

Microscopic examination showed transmural gangrene with focal areas of patchy mucosal gangrene along with focal transmural acute inflammation and serositis. No vascular thrombi were noted.

The patient improved and was discharged on 8th postoperative day. The patient performed well on 3 months follow up.

DISCUSSION

Gastric gangrene is a very rare and fatal condition. The etiology of gastric necrosis includes vascular thromboembolism and occlusion of major arterial supply, volvulus of stomach, ingestion of corrosive agents, bulimia nervosa, iatrogenic gel foam embolism, endoscopic hemostatic injections, infectious gastritis, herniation of the stomach through the diaphragm, and infectious gastritis.^[1, 2]

Gangrene of stomach is very rare due to abundant and anastomotic nature of the vascular supply. The few case reports of gastric gangrene are there in literature with the underlying etiological factors. Harvey et al^[3] reported a rare case of multifocal infarction of stomach secondary to atheromatous emboli originating in a thoracic aortic aneurysm. Bradly et al^[4], reported a case of extensive gastric necrosis in a patient with recurrent massive upper gastrointestinal hemorrhage. They found massive gastric gangrene secondary to therapeutic trans catheter embolization of the left gastric artery with fragments of gelatin sponge. Ovnat et al^[5] 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. In both the patients, exploratory laparotomy was followed by total gastrectomy and splenectomy. Two cases of gastric gangrene were reported in female patients with primary antiphospholipid antibody syndrome.^[10]

The present case is unique as the gangrene of stomach was a coincidental finding especially with pulsatile arteries which made the possibility of vascular accident unlikely. The dome of the diaphragm was normal, ruling out the possibility of a diaphragmatic hernia. No volvulus, no history of intake of a corrosive agent or any systemic disease was found. No obvious thromboembolic phenomena were found. No such history in past or any other family member was elicited.

The diagnosis of gastric ischemia is often not suspected in an emergency room because of its rarity.^[7] Initially, patients may present with symptoms of mild epigastric tenderness, vomiting or diarrhea that can rapidly progress to acute peritonitis, septic shock, and death. All radiological investigations are non-specific. Gastroscopy in rarely performed in such conditions, it may show purplish or blackish mucosa covered by exudates.^[6] Accurate diagnosis is made, most frequently, at laparotomy.^[8, 9]

Management of gastric gangrene requires resuscitation and appropriate intravenous antibiotics, followed by an emergency exploratory laparotomy. Resection of the necrotic part of the stomach is required. Massive gastric gangrene may necessitate the total gastrectomy. Onestage resection along with esophago-jejunostomy has also been reported which then followed by total gastrectomy.^[7] The alternative procedure includes cervical esophagostomy for the proximal diversion with resection of gangrenous stomach or resection with the placement of an esophageal drain. A jejunal feeding tube should always be placed.^[7] Mortality rates for gastrectomy due to acute ischemia are high so the diagnosis and timely treatment must be expedited.

Our case was unique in its presentation as proximal onethird of stomach showed gangrene and could be managed by proximal partial proximal gastrectomy and maintaining the gastrointestinal continuity by esophagogastric anastomosis. The possible causative factor would be acute gastric dilatation.

CONCLUSION

Gastric gangrene is a rare and fatal disease requiring early diagnosis and prompt treatment. Treatment requires resection of the gangrenous segment and antibiotic treatment. Increased awareness of this rare condition may lead to timely diagnosis and better survival of the patient. The present case is rare as in literature the reported cases of gastric gangrene had one or other causative agent. In this case, no causative agent was found thus making it idiopathic gastric gangrene.

REFRENCES

- 1. Richieri, J.P., Pol, B. and Payan, M.J.Acute Necrotizing Ischemic Gastritis: Clinical, Endoscopic and Histopathologic Aspects. Gastrointestinal Endoscopy. 1998; 48: 210-212.
- Dharap, S.B., Ghag, G. and Biswas, A. Acute Necrotizing Gastritis. Indian Journal of Gastroenterology. 2003; 22: 150-151.
- Harvey RL, Doberneck RC, Black WC. Infarction of the stomach following atheromatous embolization. Report of a case and literature review. Gastroenterology. 1972; 62: 469-72.
- 4. Bradley EL 3rd, Goldman ML. Gastric infarction after therapeutic embolization. Surgery. 1976; 79: 421-4.
- Ovnat A, Dukhno O, Pinsk I, Shaked G, Levy I. Acute obstruction of the celiac trunk. J Clin Gastroenterol. 2005; 39: 647.
- 6. Hisham A R Hamad Post vagotomy gastric gangrene due to necrotizing gastritis. The Middle East Journal of Emergency Medicine, 2003; 3(2). Sept 2003.
- 7. Ammori GB, McHugh J, Cimmino VM (2007) Acute gastric necrosis: assessing the risk factors. Surgical Rounds. March 2007.
- 8. Harvey RL, Doberneck RC, Black WC. Infarction of the stomach following atheromatous embolization. Gastroenterology. 1972; 62: 469.
- Vilardel F (1985) Gastritis. Inc: Berk JE, Haubrich WS, Kalser MH, (eds.). Bockus gastroenterology. Philadelphia: WB Saunders, 943–4.
- 10. Srivastava V, Basu S, Ansari M, Gupta S, Kumar A. Massive gangrene of the stomach due to primary antiphospholipid syndrome: report of two cases. Surg Today. 2010; 40(2): 167-70.