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Acute necrotising gastritis with gangrenous perforation : a case report

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ABSTRACT

Gangrene of the stomach is a rare, often fatal disease which may be due to vascular, chemical, mechanical, or infectious etiologies. We report a case of gastric gangrene due to acute necrotizing gastritis in a 58 -year-old male. Review of the literature on suppurative gastritis emphasizes the rarity and high morbidity of acute necrotizing gastritis. The patient reported on in this study, however, survived after sleeve gastrectomy and antibiotic therapy. It is our opinion that surgical removal of involved non viable gastric tissue with healthy margin is inevitable for successful treatment.

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.

Cases of acute necrotizing gastritis being the rarest[1] have been linked to peptic ulcer disease, gastric outlet obstruction and vagotomy[2] .We report on a man with stomach gangrene that appeared to be caused by a severe necrotizing infection.

CASE REPORT

A 58 years old male patient was admitted to emergency with history of pain abdomen followed by abdominal distension and vomiting. On examination patient was having tachypnoea with acidotic breathing and tachycardia. Per abdomen examination revealed features of peritonitis. Erect abdominal X- ray revealed gas under diaphragm. Emergency CT abdomen showed multiple air fluid collections with enlarged mesenteric and retroperitoneal lymph node. CT chest showed destruction of left lung (cavitation in lower lobe) with irregular fibro nodular infiltrates in right lung and superior mediastinal adenopathy.

Patient was adequately resuscitated in ICU and after surgical consultation he was taken up for exploratory laporotomy. During surgery two liters of foul smelling yellow coloured turbid ascitic fluid was aspirated. There was patchy gangrenous greater curvature of stomach with large perforations [Fig-1 and Fig-2]. Normal pulsation of gastric vessels were present. Diaphragmatic domes appeared normal. Other structures in the vicinity were normal. Small bowel appeared inflamed. Sleeve gastrectomy with adequate peritoneal lavage was done. Abdomen closed in layers with closed drain in situ.

Resected specimen of stomach was showing gangrene with perforation and necrotic membrane at places. Microscopic examination was showing gangrene and destruction of all layers of stomach. No vasculitis or atypia was seen. Culture of peritoneal fluid was non specific.

In post operative period a leak was detected from the stappled line of stomach which was managed endoscopically. Patient was kept on antibiotics, IV fluid and TPN. Four units of blood transfusion was given. After ten days feeding was started which he tolerated well. During the post operative staying he had fever with respiratory difficulty for which he was evaluated and found to have significant right side pleural effusion for which he was given intra thoracic chest tube drainage. He was evaluated for tuberculosis and found to have sputum positive for AFB. The patient was discharged on oral ATT.

DISCUSSION

The abundant and anastomotic nature of the stomach's

vascular supply makes gangrene very rare. In 1943, Babkin et al [3] 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 anastomosis between the left gastric artery and branches of the phrenic and esophageal arteries. However, Harvey et al[4] reported a case of multifocal gastric infarction secondary to atheromatous emboli originating in a thoracic aortic aneurysm. In another report[5], 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.

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 causing 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 culture was non specific. The stomach contents was 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

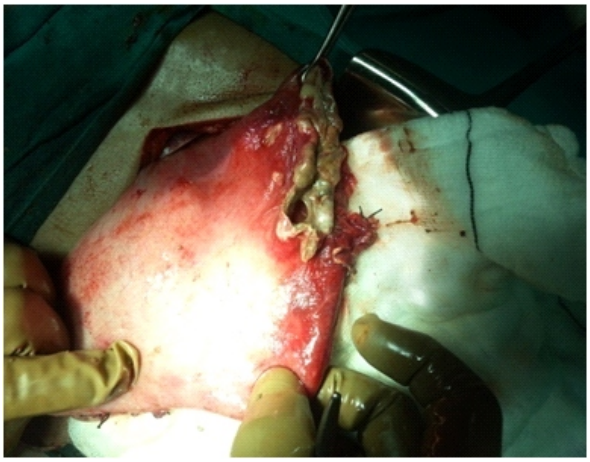

Figure 1: Gangrenous perforations on greater curvature of stomach

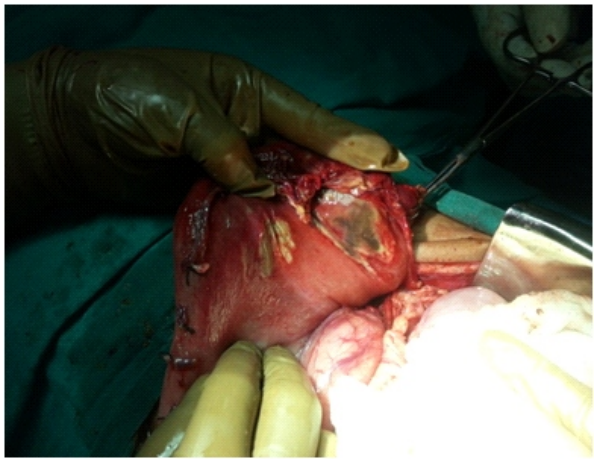

Figure 2: Necrotic membranes on stomach wall

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 immuno suppression [6-8].

In our case the patient was a prominent case of pulmonary tuberculosis evidenced by cavitation in base of left lung with irregular fibronodular infiltrates in right lung and superior mediastinal adenopathy and more over sputum positive for AFB. In our case biopsy of enlarged mesenteric and retroperitoneal lymph nodes showed reactive hyperplasia non consistent with tuberculosis and peritoneal fluid culture showed no evidence of tuberculosis. As such tuberculosis of stomach is rare, because of sparsity of lymphoid tissue in upper GI tract, high acidity of peptic secretion and rapid passage of ingested organisms[9].

Phlegmonous gastritis may arise from local or disseminated hematogenous infection causing localised or diffuse involvement of stomach. The most frequent organisms are Streptococcus, Staphylococcus, E Coli, Proteus and Clostridia. Mixed bacterial infections have also been reported[10]. So here in our case mixed bacterial infection along side tubercular infection might be the culprit.

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[6-8]. 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.

Diagnosis of acute necrotizing or gangrenous gastritis is usually made at laparotomy, although endoscopy, endosonography, 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.

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. The mortality rate of patients treated by surgical resection is far lower than for those treated by medical therapy alone (20% vs. 50%) [10]

CONCLUSION

Acute phlegmonous or necrotizing gastritis is a rare condition and clinical diagnosis may be difficult. It is usually diagnosed by laparotomy. It may be monomicrobial or poly microbial. Treatment consists of resection of the gangrenous portion, 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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