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Review Article

Acute necrotizing gastritis with gangrenous stomach

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ABSTRACT

Acute necrotizing gastritis with gangrene of the stomach wall is a rarely encountered condition. This often a fatal condition if is attributed to vascular occlusion, chemical and mechanical injuries or infectious etiologies. We review the available literature with a case of acute necrotising gastritis with unknown etiology. Case was managed successfully with emergency partial gastrectomy.

Keywords: Acute necrotizing gastritis, Blood supply of stomach, Gastritis, Gangrenous stomach, Perforation peritonitis, Partial gastrectomy, Septic shock, Sleeve gastrectomy

INTRODUCTION

Gangrene of the stomach is an extremely rare clinical finding, as the stomach receives a very rich vascular supply from branches of the celiac trunk and also from various collaterals. Various causes like arterial embolism, gastric herniation, gastric volvulus, acute dilatation, bulimia nervosa, trauma, exposure to chemicals, iatrogenic gelfoam embolism, endoscopic haemostatic injections and infectious gastritis have been published in literature.[1-5] An author described that the complete occlusion of arteries of the stomach does not cause gastric gangrene in human as well as in animals, because of rich collateral arterial supply. However, they opined that complete occlusion of venous flow can result to the gastric gangrene.[3] Richieri J et al brought out endoscopic aspects of gangrenous gastritis.[4] The endoscopic features that may suggest the diagnosis includes irregular thickening of mucosa with purplish discoloration accompanied by diffuse exudate and erosions. An author reported that the histology of endoscopic lesions can confirm the diagnosis.[6] Selective celiomesenteric arteriography may be diagnostic. It is however emphasised that stenosis of celiomesenteric axis only along with diseased collateral network is sufficient enough to cause gastric ischemia.

VASCULAR SUPPLY OF THE STOMACH

Celiac artery with its branches is the major vascular supply of the stomach (Figure 1). The celiac artery is the first major branch of the abdominal aorta amongst the anterior divisions, and it further gives rise to the left gastric artery, splenic artery, and the common hepatic artery. A minor esophageal branch arises from the left gastric artery close to the gastric cardia, and the main left gastric artery curves over the proximal lesser curvature. The splenic artery runs towards the splenic hilum. The terminal branches of the splenic artery give rise to many short gastric arteries that supply parts of the gastric fundus. The splenic artery then proceeds around the greater curvature, forming the left gastroepiploic artery. The common hepatic artery divides into proper hepatic artery and gastroduodenal artery. The right gastric artery arises from the hepatic artery to supply the antrum and distal lesser curvature. The gastroduodenal artery forms

the right gastroepiploic artery and loops over the antrum and distal greater curvature. The left gastric artery and a right gastric artery forms the arterial arch on the lesser curvature. The arterial arcade over greater curvature is formed by the right and left gastro-epiploic arteries. The anastomoses between the two arterial arches occur in the submucosal layer at a distance of about two-thirds from the lesser to greater curvature. The gastric cardia is receiving its blood supply from the left gastric artery, and also from connections with short gastric arteries, the right gastric, and the left inferior phrenic arteries. The first branch of the superior mesenteric artery namely the inferior pancreaticoduodenal artery, at occasions provides collateral blood supply to the stomach. The collaterals from esophageal arteries and the left inferior phrenic artery also contributes to the gastric vascular network. On endoscopy of patients with gastric atrophy, the mucosal vascular network of the stomach sometimes can be seen.[7]

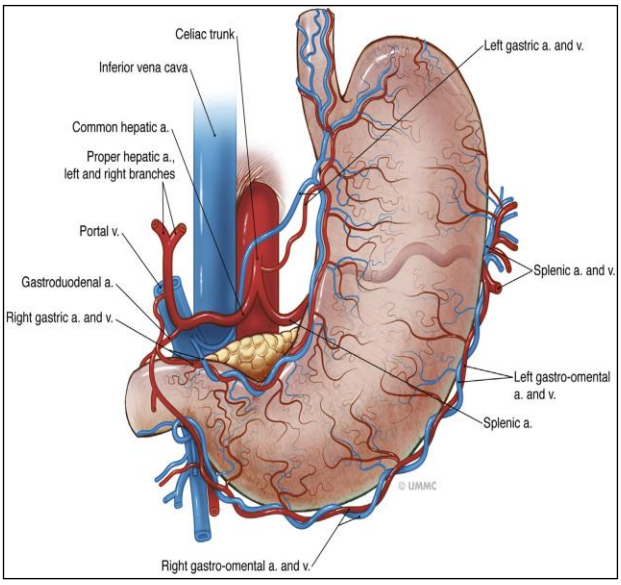

Figure 1: Vascular supply of stomach.

Etiopathogenesis

Acute gastric ischemia has been reported in sundry conditions as disseminated intravascular coagulation, severe mesenteric ischemia, antiphospholipid antibody syndrome complicated by adrenal insufficiency, gastric volvulus, after intra-arterial infusion of vasopressin in the left gastric artery, after endoscopic submucosal dissection and injection sclerotherapy. Gastric distention often induces and also exacerbates gastric ischemia. Acute gastric distention after ingestion of a large meal or binge eating as may occur in patients with anorexia nervosa or bulimia, may lead to gastric ischemia.

At tissue level, early gastric ischemic changes include capillary dilatation, mucosal edema, vascular congestion, and superficial necrosis[7]. These findings progress to mucosal coagulative necrosis, with surface erosions and necro inflammatory (fibrinopurulent) exudates. Persistent ischemia produces full-thickness hemorrhagic necrosis and deep ulceration of the gastric wall. Peripheral epithelial gets involved in reactive changes such as mucin depletion, nuclear enlargement, and hyperchromasia and increased mitotic activity. Histopathological findings of chronic ischemia include hyalinization and fibrosis of the lamina propria and withered atrophic glandular epithelium.

Diagnosis

The diagnostic work-up of gastric ischemia includes endoscopy, imaging studies (including CT scan and CT angiography), and selective mesenteric angiography. Endoscopy, though an invasive procedure, offers early diagnosis and an estimation of the severity and extent of gastric ischemia.

Table 1: Endoscopic grades of stomach ischemia.[7]

Mild	Mottled and pale gastric mucosa
Moderate	Mottled and pale gastric mucosa, numerous stellate gastric erosions or small ulcerations
Severe	Mottled and pale gastric mucosa, numerous stellate erosions or small ulcerations, large and confluent gastric ulcerations

Imaging studies may detect intramural gastric air and help rule out other intra-abdominal causes for the clinical presentation. Emphysematous or phlegmonous gastritis, gastric emphysema, and pneumatosis are associated with intramural gastric air, seen sometimes on plain X-ray film and confirmed by a CT scan.

Case explanation of the review topic

A 35-year male patient presented in the surgery emergency with complaints of pain abdomen, vomiting and obstipation since 48 hrs. On examination: the patient was afebrile, had tachycardia of 110/min, was tachypnoeic and had a systolic blood pressure of 80 mmHg. On per abdomen examination, he had generalised peritonitis. Blood investigation showed leucocytosis while rest of haematological and biochemical investigations were all within normal limits. Abdomen x-rays showed no free gas under the diaphragm and no signs suggestive of intestinal obstruction were seen. USG abdomen showed presence of free fluid was present with internal echoes, in the peritoneal cavity. Patient was diagnosed as a case of generalized peritonitis with hypovolemic shock. Patients was resuscitated and taken up for exploratory laparotomy.

Intra operative findings included:

- One-liter foul smelling haemorrhagic fluid with in peritoneal cavity.

- The stomach wall was gangrenous along the greater curvature involving around 1/3rd of the body of stomach (involvement of both anterior and posterior aspects) with status impending perforation (Figure 2 and 3).
- On palpation major gastric vessels were normal pulsatile.
- Rest solid and hollow viscous appeared normal. The diaphragmatic domes were normal and there was no evidence of gastric volvulus or hernia.

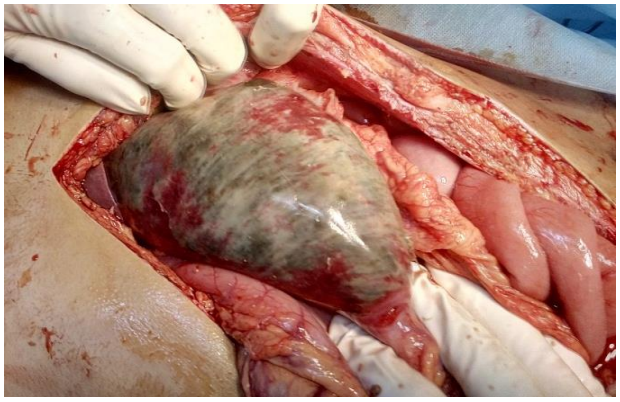

Figure 2: Gangrenous wall of stomach (anterior wall).

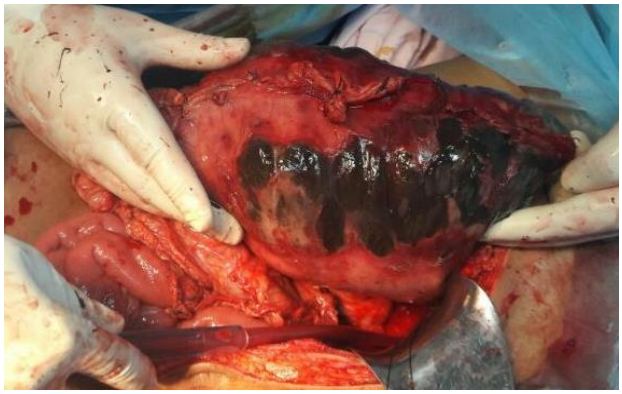

Figure 3: Gangrenous wall of stomach (lateral wall).

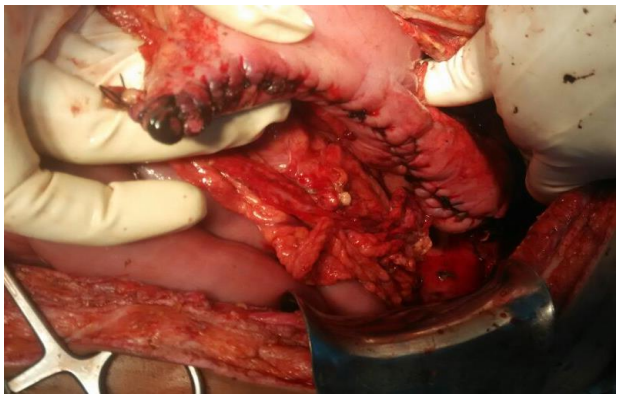

Figure 4: Repaired after partial gastrectomy.

We performed longitudinal partial gastrectomy (vis a vis sleeve gastrectomy) with primary hand sewn repair in two layers (Figure 4). Feeding jejunostomy was fashioned to start early enteral feeding. Postoperative period went uneventful. FJ feed started after 48 hours and patient accepted orally on post-operative day 9. He was discharged well on post-operative day 11. Feeding jejunostomy was removed after 3 weeks. On regular follow-up of 6 months, patient is doing well.

DISCUSSION

Acute necrotizing gastritis with gangrene of the wall is a rare clinical condition. The abundant vascular supply of stomach makes its gangrene very rare. A study in dogs by an author brought out that even tying of all the gastric arteries did not cause gastric gangrene.[8] The underlying reason for this is multiple collaterals between the left gastric artery and branches of the phrenic and oesophageal arteries. The literature however still shows gangrene or necrosis after arterial occlusion or thrombosis. An author published a case of multifocal gastric infarction due to emboli originating from thoracic aortic aneurysm.[9] Extensive gastric necrosis after therapeutic trans catheter embolization in upper gastrointestinal bleed reported.

Our case was operated under emergency conditions and diagnosis was established intra operatively, we could not subject the patient to extensive investigations. However, the patient was subjected to contrast enhanced CT scan of abdomen with CT angiography that did not show any underlying pathology. Similarly, the post-operative blood profile including coagulation studies were essentially normal. Patient managed with partial gastrectomy and thorough lavage of peritoneal cavity. Histopathological examination report was suggestive of acute necrotizing gastritis. Cause of necrotising gastritis, in our case could not be found out.

CONCLUSION

It is concluded that acute necrotizing gastritis is a rare finding and it is very difficult to diagnose preoperatively, but early and judicious management can save the life of a patient. The causes of gangrene are multitude; however, a large proportion is idiopathic.

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