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STOMACH GANGRENE IN A TEENAGER : A RARE SURGICAL EMERGENCY

General Surgery

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ABSTRACT

Acute gastric gangrene is rare due to rich vascularity of the stomach and the anastomotic nature of blood supply to it. Only a very few cases of gastric gangrene have been reported in the medical literature. Acute gastric dilatation can have multiple etiologies which may lead to ischemia of stomach and eventually gangrene. Without proper timely diagnosis and treatment potentially fatal events such as gastric perforation , hemorrhage and other serious complications can occur. Here we present a similar rare case of acute gastric dilatation leading to massive gangrene of stomach and perforation, due to a territorial infarct in the blood supply of stomach and how a novel surgical improvisation was done to salvage the patient. Circulatory shock is the fate of this condition if not intervened at the right time.

KEYWORDS

Stomach gangrene, Gastric necrosis, Gastric infarct, Acute gastric dilatation

INTRODUCTION:

Acute gastric gangrene is rare due to rich vascularity of the stomach and the anastomotic nature of blood supply to it.[1,2] Only a very few cases of gastric gangrene have been reported in the medical literature. The first case of major gastric infarct was reported by Bauman in 1909.[1] The stomach is a well vascularised organ with four major vascular supplies; hence, infarction of the stomach is an uncommon phenomenon compared to other parts of the gastrointestinal tract.[3]

Several possible causes of gastric gangrene have been identified. Most common of them are, herniation of stomach through diaphragm as in diaphragmatic hernia, gastric volvulus, acute necrotizing gastritis, electrolye imbalance, as a complication after recent abdominal surgery, trauma to abdomen and rarely due to diabetes mellitus. Gastric gangrene has also been observed in patients with eating disorders like anorexia nervosa and bulimia nervosa ; reason behind which is thought to be due to acute massive gastric dilatation from binge eating.

Acute gastric dilatation can have multiple etiologies which may lead to ischemia of stomach and eventually gangrene. They are lifestyle habits, underlying morbidities, acute vascular insufficiency and pathological aerophagia.[4,5,6,7,8,9,10] Acute gastric gangrene is a difficult entity to diagnose and is often confused with other causes of peritonitis. This is because of its rarity that this condition is mostly diagnosed intra-operatively.[11,12] Without proper timely diagnosis and treatment potentially fatal events such as gastric perforation , hemorrhage and other serious complications can occur. Here we present a similar rare case of acute gastric dilatation leading to massive gangrene of stomach and perforation, due to a territorial infarct in the blood supply of stomach and how a novel surgical improvisation was done to salvage the patient.

CASE DESCRIPTION:

A 15 year old teenage young boy, referred from peripheral hospital, presented to the causualty department in Sawai Mansingh Hospital, Jaipur with altered sensorium , pain abdomen, multiple episodes of vomiting and abdominal distension for 3 days. Patient was being managed conservatively at a district hospital with intravenous fluids and analgesics, but, as the condition of the patient was worsening, he was referred to Sawai Mansingh Hospital.

The father of the patient gave history that 3 days ago the boy ate a lot of raw rice grains and drank lot of alcohol, which actually was being practiced as a part of ritual to cerebrate Holi festival in their culture in India. The boy started to complaint of abdominal pain along with distension around 8 hours later , since he ingested raw rice grains. He also had multiple episodes of vomiting which contained ingested food particles. The patient had developed altered sensorium just 3 hours before reaching the hospital. His past history was not significant. He was not suffering from any psychiatric illness or any co-morbidity like diabetes and had not undergone any surgeries.

On examination, pulse rate of the patient was 140 beats per minute, blood pressure measured in right arm sitting position was recorded to be 84/58 mm Hg .The patient was disoriented in time, place and person. On examination, abdomen was grossly distended. There was diffuse tenderness, guarding and rigidity suggestive of gross peritonitis. But there was no gas under the domes of diaphragm in both the plain radiograph of chest and erect x-ray of abdomen. Patient was prepared for surgery and taken to operation theatre solely based on clinical findings and abdominal examination. Before inducing the patient for general anesthesia , there was tachycardia and hypotension. Nasogastric decompression as part of resuscitation yielded instantly 200 ml of brownish fluid. Fluid resuscitation was done adequately but urine output was less than 100 ml in over 3 hours. So, patient was taken on noradrenaline and dopamine support intra operatively. Midline exploratory laparotomy incision was given and abdomen was opened in layers. Around 2 litres of blackish fluid with undigested raw rice grains was suctioned out. The contamination fluid had no remarkable odour. The fluid also had lot of necrotic tissue which was later identified to be that of stomach tissue. The gastro esophageal junction was normal. The stomach was gangrenous and the gastric tissue was fully necrosed starting from 2cm distal to gastro esophageal junction upto 4 cm proximal to pylorus, mainly in the anterior wall of the stomach ,in the territory supplied by the left gastric artery.(Figure 1).The contamination fluid contained gangrenous and necrotic tissue of stomach. (Figure 2).

The necrotic tissue was thoroughly debrided and necrosectomy was done.(Figure 3). Lesser sac was accessed by incising through the gastro colic ligament to examine the posterior wall of stomach. The posterior wall of stomach was found normal. A thin rim of normal tissue was seen at both the lesser and greater curvature of stomach, which probably had survived due to the rich arterial anastomotic arterial arcade of the stomach. Both the edges were brought closer, approximated and repaired primarily with silk and vicryl sutures. Omental patch with good vascularity was secured diligently. This eventually resulted in a conduit primarily based on the epiploic arterial arcade supplying the greater curvature of the stomach. Now, for the repaired area to heal well, it needed to be kept bereft of gastric secretions and bile, as, if these secretions accumulated in the repaired area, they could rip through the stitches. So, for this tube gastrostomy was done through anterior wall of stomach, 2 cm proximal to the pylorus.

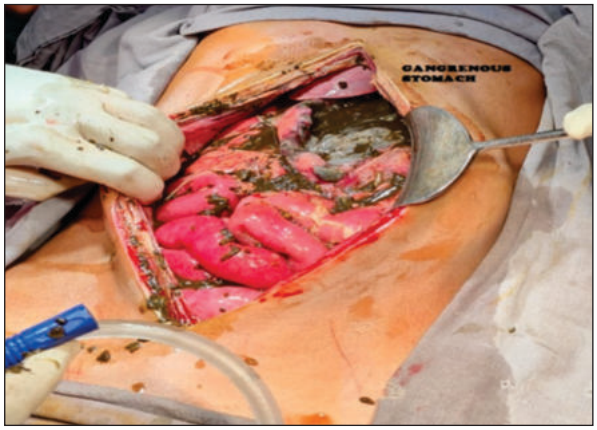

Figure1 Gangrenous Stomach With Necrotic Fluid Inside Peritoneum.

Figure 2 Necrotic Fluid Being Suctioned Out.

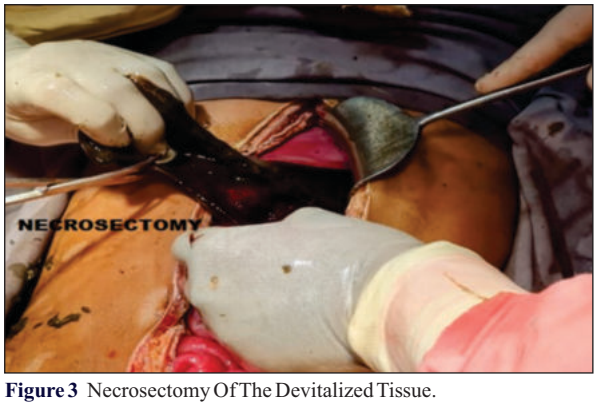

Figure 3 Necrosectomy Of The Devitalized Tissue.

The gall bladder was distended. So, to reduce the amount of bile collecting in the second part of duodenum and to reduce the biliary reflux into the stomach, cholecystostomy was done with Foley's catheter 18 French units. Ryles tube which was inserted pre operatively for naso gastric decompression was pulled further distally towards the pylorus.

It was evident that the patient will not be fed orally atleast for 2 weeks in order to endow adequate time for the repaired area and the conduit created , to heal. So, to provide enteral nutrition to the patient, feeding jejunostomy tube was also placed 25 cm distal to the duodeno jenunal junction , in the jejunum. An abdominal drain was kept near the posterior wall of stomach through the already opened lesser sac. Another rubber drain was kept in the pelvis, behind the bladder. (Figure 4). The layers of abdomen were closed en mass. Patient was managed in intensive care unit for the initial three post operative days. He was weaned off from the ventilator support on the second post operative day. Inotropic support was withdrawn after the patient recovered from hypovolemic shock and his blood pressure stabilized. Around 300 ml of bile and gastric secretions, on average, drained continuosly from the gastrostomy tube and 50 ml of bile daily in cholecystostomy tube. Minimal serosanguinous fluid drained in the lesser sac and pelvic drains. Enteral feeding for the patient was started from the feeding jejunostomy tube on the third post operative day. Patient passed motion on the 4th post operative day.

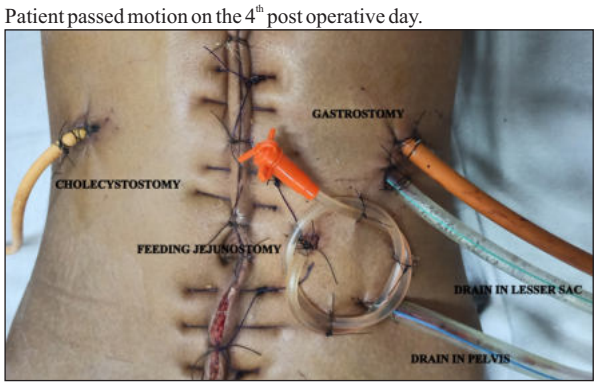

Figure 4 Gastrostomy, Cholecystostomy, Feeding Jejunostomy, Drain In Pelvis And Lesser Sac.

The total white blood cell counts reduced from 56000 cells per cubic mm pre operatively to 9600 cells per cubic mm on the 4th post operative day. His Hemoglobin also improved to 10.2 grams. Patient started to ambulate freely from the 4th post operative day. He had an uneventful post operative recovery. The drain in the pelvis was removed on the 7th day. He was discharged on the 8th post operative day with gastrostomy tube, cholecystostomy tube and drain in lesser sac ,in situ. He was kept on regular weekly follow up.

DISCUSSION :

Acute gastric necrosis is a rare surgical emergency. Because of its rarity , diagnosis is mostly delayed and virtually made only at laparotomy. Pre operative diagnosis can be supported by radiological modalities, for which computed tomography is relevant in revealing diagnosis and probable etiology. The cardinal consequence of binge eating as occurred in this case is acute gastric dilatation which has been implicated as the key pathway leading to gastric necrosis in more than half of the cases reported. In the setting of acute massive gastric dilatation a net pressure of about 14 mm Hg is sufficient enough to cause venous insufficiency leading to ischemia. Intra gastric volumes of at least 3 liters have been found to be impeachable to generate such pressures. In this case, 2 liters of fluid was drained intraoperatively. Gastric rupture can occur with pressure more than 120 mm Hg or 4 liters of fluid.[13] In majority of cases, greater curvature and gastric fundus are prone for necrosis, meanwhile, lesser curvature and pyloric regions of the stomach tend to be spared.[14] In our case too, the pyloric region remained untouched. Most of the reported cases of gastric necrosis occurred in patients with eating disorders like bulimia, but this patient had no history of such eating disorders.

Circulatory shock is the fate of this condition if not intervened at the right time. In our case too, the patient was in circulatory shock with low blood pressure and reduced urine output. So fluid resuscitation and the wise use of ionotropes like noradrenaline and dopamine is warranted , before surgical intervention is planned. The management of acute gastric necrosis involves resection of the necrotic portion of the stomach with establishment of gastrointestinal continuity in stable patients. But , in our case, as the patient was in circulatory shock and his clinical condition was precarious, a savvy surgical procedure was done to salvage the patient. A conduit relying upon the epiploic arcade was created by approximating the viable ends of stomach after necrosectomy was completed.(Figure 5).Tube gastrostomy and cholecystostomy was done to bypass the bile and gastric secretions from the stomach and duodenum, along with a feeding jejunostomy for enteral feeding of the patient.

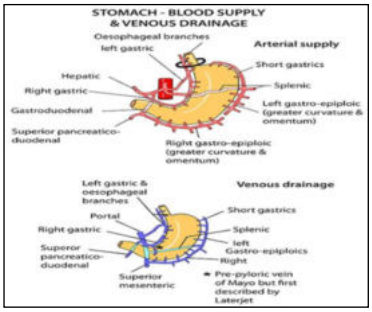

Figure 5 Blood Supply And Venous Drainage Of Stomach

The mortality associated with acute gastric necrosis ranges from 50 to 80 percent which underscores the severity or life threatening nature of the condition. The presence of circulatory collapse is a significant factor contributing to mortality. But, in our case prompt fluid resuscitation and surgical management accounts for the different picture in terms of outcome.

CONCLUSION:

Acute gastric necrosis is a rare surgical emergency that require a high index of suspicion and warrants prompts fluid resuscitation and shrewd surgical management to obviate the high mortality rate associated with the condition.

Conflicts Of Interests:

The authors declare that there is no conflict of interests regarding the publication of this article.

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