

Gastric Gangrene - A Unusual Case in Young Girl Causing Stomach Perforation

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Abstract: *Gangrene of the stomach is a rare and a catastrophic occurrence as stomach is a highly vascularised organ. We present here a case of gastric gangrene in a 25 year female lady presenting with acute tender and guarded abdomen. Exploratory laparotomy with resection of gangrenous stomach (proximal gastrectomy) with esophago-gastric anastomosis followed by pyloroplasty and feeding jejunostomy. Patient is critically unstable and required ventilatory and inotropic support post operatively.*

Keywords: Gastric gangrene

1. Introduction

Stomach is one of main organ of body which is highly vascularized. Multiple primary arteries and other various collaterals prevents the stomach from any deleterious ischaemic effect. Hence stomach is the toughest organ to undergoes gangrenous changes. Gangrene in GIT are mostly seen in small and large bowel due to various pathology in mesenteric blood flow. Gangrene of stomach is a very rare event and very few case reports are present till date. It is highly critical condition and associated severe morbidity and high mortality.

2. Case Report

A 25 year lady came with chief complaints of severe generalised colicky abdominal pain since 3 days. Patient also complaint of multiple episodes of vomiting since 3 days and also constipation with obstipation since 3 days. Patient is afebrile and hemodynamically unstable. Her pulse was 120bpm and BP was 80/60 mm of Hg. Abdomen was generalized tender and guarding present all over abdomen. Abdominal distension also present. Ryles tube and foleys inserted. Initial resuscitation by IV fluids and minimal dose of noradrenalin started. Jugular central line placement done and CVP monitoring maintained. Patients primary investigation suggestive of Hb of 14 with TLC of 14600 and Platelets of 197000. Renal function test and serum

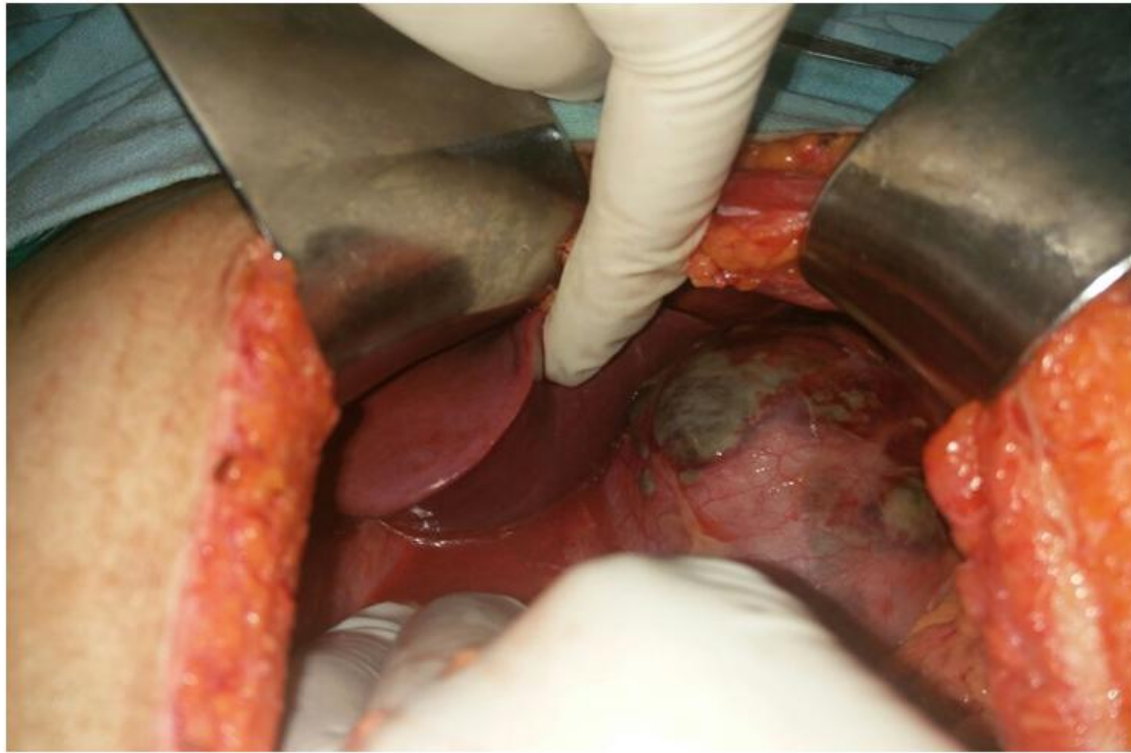
electrolyte level was normal. Arterial blood gas analysis was also normal. There is significant elevation on Sr AMYLASE 305 and LIPASE 1218 levels. After stabilization X ray chest and abdomen done which suggestive of multiple air fluid levels.

USG (A+P)- s/o subacute intestinal obstruction with dilated bowel loops, max diameter being 4.1cm, and shows sluggish to and fro peristalsis.

CECT (A+P)-Stomach, D1, D2 and D3 segments of duodenum grossly overdistended with food particles within, with Ryle's tube in situ and is seen occupying the upper abdomen upto the pelvic brim. Compression of the D3-D4 segments of duodenum seen between the SMA anteriorly and the aorta posteriorly with transition zone noted in the D3-D4 region. SMA angle- 19 degrees and Aortomesenteric distance -7.3mm

Features s/o SMA syndrome with duodenal obstruction. No decrease in the size of obstructed duodenal loop, hence associated duodenal stricture cannot be ruled out entirely. Moderate ascites. No obvious leak on oral contrast administration.

Resuscitation of fluids and inotropic support continued and patient is taken for exploratory laparotomy for intestinal obstruction.



3. Operative Findings

Multiple patches of Ischaemic necrosis of proximal anterior and posterior stomach of maximum size of 5cm. Diagnosis of gastric gangrene was done intraoperatively. No necrosis of liver/ spleen/ duodenum. Traced small and large bowel

loops was normal. Pulsations of hepatic artery, splenic artery, IMA, and SMA well felt. No oesophageal perforation appreciated at GE junction. No thickening of stomach appreciated. Purulent collection of 200 cc with pus flakes. Sigmoid and descending colon filled with pellets of stools.



Resection of proximal gangrenous stomach done with esophago gastric anastomosis along with pyloroplasty and feeding jejunostomy. Patient was not hemodynamically unstable and kept under ionotropic and ventilatory support. On post operative day 1 patient regains consciousness with

good respiratory efforts but still on minimal dose of ionotropic supports. On post operative day 3 patient was extubated and without any ionotropic supports. One unit of blood transfused in view of low Hb and hematocrit. On post op day 5 patient is vitally stable, abdomen is soft, passing

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flatus, one drain is removed and Feeding Jejunostomy started. On post op day 9 **esophagogram** done which is normal without any leak. On post op day 11 soft diet started and all drains removed. patient tolerated diet very well. On post op day 15 patient was discharged.

In between ward course blood investigations were done to find out the probable cause of gastric gangrene. All investigation including Rheumatoid factor, Anti- SMA, ANA, Anti DS DNA reported negative. The histopathology report s/o ischaemic necrosis of entire wall of stomach areas of mixed inflammation predominantly polymorphs and markedly dilated blood vessels. Mucosa completely ulcerated showing gangrenous changes. Occasionally blood vessels show presence of fibrin thrombi. No evidence of any viral or fungal infection.

4. Discussion

Gangrene of the stomach is a rare and associated with high mortality. Causes includes thromboembolism and occlusion of major arterial supply, ingestion of corrosive agents, Gastric outlet obstruction, retroperitoneal tumor involving duodenum, post abdominal surgery, trauma, diabeticketoacidosis, anorexianervosa, cerebral palsy / muscle dystrophy, stomach volvulus, herniation of the stomach through the diaphragm, bulimia nervosa, gelfoam embolism, endoscopic haemostatic injections and infectious gastritis [4, 5], recent large intake of alcohol, 'gastritis' and upper respiratory tract infections [2]. The acute necrotizing gastritis is the most rare cause of gastric gangrene. It begins as suppurative gastritis, then it progresses to the lethal severe form: acute necrotizing gastritis. Organisms isolated from the gastric wall include hemolytic streptococci, proteus, E. coli and clostridium welchii [2, 3]. The sequence of events that takes place after acute gastric dilatation include increasing intra-gastric pressure leading to gastric wall ischemia and perforation as well as pressure on the inferior venacava causing reduced venous return and hypotension.

In the present case there was no history suggestive of atherosclerosis. Neither volvulus nor herniation of the stomach was observed intraoperatively. The main vessels were intact, ruling out a thromboembolic event. There was no history of ingestion of caustic substances. There was no evidence of generalized peritonitis.

Diagnosis of gastric ischaemia is often delayed because of its rarity and non specific symptoms [7]. Initially, patients may have symptoms of mild epigastric tenderness, vomiting or diarrhoea that rapidly progresses to acute peritonitis, septic shock and death. All radiological tests are non specific. Gastroscopy may show purplish or blackish mucosa covered by exudates [3]. Absolute diagnosis is made, most frequently, at laparotomy [8, 9].

If a diagnosis of ischaemia is being considered, resuscitation and intravenous antibiotics should be initiated immediately, followed by an emergency exploratory laparotomy. Resection of a necrotic stomach is required, with total gastrectomy if necessary. One stage resection with esophago-jejunostomy has been reported [7]. Alternative includes resection with cervical esophagostomy for proximal

diversion or resection and placement of an esophageal drain. A jejunal feeding tube should always be placed [7]. Diagnosis and treatment must be expeditious, because mortality rates for gastrectomy due to acute ischaemia are high.

5. 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 [7].

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