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DECEIVING FATALITY IN A CASE OF ACUTE NECROTISING GASTRITIS

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ABSTRACT

With only 500 cases reported in the world, Acute Necrotizing Gastritis, a variant of Phlegmonous Gastritis is a rare, yet a potentially fatal condition. Phlegmonous Gastritis means abscess or spreading cellulitis of the stomach wall caused by microorganisms and the condition when these organisms cause necrosis and gangrene of the stomach wall is called Acute Necrotizing Gastritis.

We report a case of a 17year old boy who presented with acute abdominal pain and vomiting since 1 day and with early features of shock. Abdominal guarding and rigidity and free air under diaphragm on erect abdominal X-ray called for an emergency exploratory laparotomy, where a 5x6cm, greater curvature stomach gangrene with full thickness posterior wall perforation totally surprised the surgeons. All proposed etiological factors for stomach gangrene like embolization of atherosclerotic plaque, thrombosis of major artery, 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 or gastric volvulus were ruled out and the possibility of severe necrotizing gastritis caused by organisms was considered and confirmed by isolation of streprococcus in the peritoneal cavity.

Wide excision of the gangrenous segment with primary closure of the stomach was done. With prompt and aggressive medical management, supportive and nutritional care we were able to rescue the patient from the fatal condition and the patient lives to tell the tale.

KEYWORDS: Phlegmonous Gastritis, Acute Necrotizing Gastritis, Stomach gangrene, Stomach perforation.

INTRODUCTION

With only 500 cases reported in the world, Gangrene of the stomach is a rare, yet a potentially fatal condition [1]. The proposed etiology is 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 [1].

We report a 17year old boy with stomach gangrene possibly due to severe necrotising infection.

CASE REPORT

17 year old male presented with intractable pain in the upper abdomen with vomiting since one day. No history of altered bowel habits or abdominal trauma or any previous surgery or any significant drug intake history. Patient was thin built, hypotensive (90/60mmHg) with tachycardia (140bpm) and tachypnoea. Abdomen was scaphoid, non-distended with no visible peristalsis/swelling/pulsation/any scars or sinuses. Flanks were not full. On palpation abdomen was tense tender with guarding and rigidity present. A dull percussion note was present over iliac fossa on either side and bowel sounds were absent. Cardiovascular and respiratory examinations were normal.

USG abdomen showed moderate free fluid (approximately 2lt) with low level floating air echoes in the peritoneal cavity suggestive of hollow viscus perforation.

Erect X-ray abdomen showed free air under both hemi-diaphragms confirming the hollow viscus perforation (Fig I). Chest X-ray was normal.

Raised Total Leucocyte Counts of 12400/cumm, Blood Urea of 56.1 mg/dl, a Creatinine level of 2.0 mg/dl, Total Bilirubin of 2.5mg/dl and Direct Bilirubin of 1.1mg/dl with normal liver enzymes and protein levels. Serum Amylase was 342.1U/l and Lipase was 416U/l. Patient was tested negative for WIDAL and Mantaux test.

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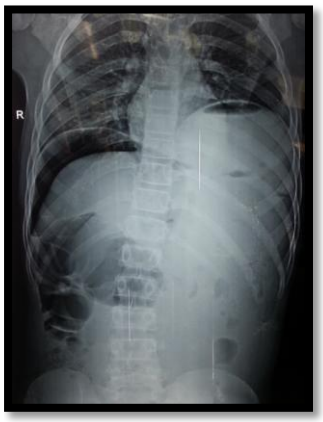

Fig I Erect abdominal X-ray showing Gas under diaphragm

The patient was managed hemodynamically and was taken for emergency exploratory laparotomy. Intraoperatively, there was generalized peritoneal thickening and 2 litres bilious fluid was drained from peritoneal cavity. There was a full thickness gangrenous perforation of posterior wall of greater curvature of stomach measuring approximately 5x6 cm with partially digested food particles around the perforated site and in the peritoneum. The stomach wall was flabby with unhealthy gastric mucosa around 2cm adjacent to the gangrenous segment. There was no other abnormal finding and rest of gastric mucosa, gastro-oesophageal junction, pyloric part of stomach and duodenum were normal. No distal obstruction was found. The rest of gut was normal (Fig II).The peritoneal cavity fluid culture showed growth of streptococcus.

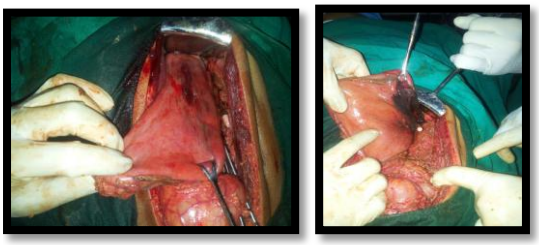

Fig II Intraoperative finding of a greater curvature posterior surface stomach gangrene with perforation

The gangrenous segment with the unhealthy segment was resected, margins were freshened and the remaining segment was sutured in 2 layers to recreate the stomach. After a thorough abdominal wash the abdomen was closed with two corrugated drains on either sides along the paracolic gutters.

Postoperatively the renal function and the liver function improved. The patient developed respiratory distress on Post-Operative Day 5 and chest X-ray showed massive left sided pleural effusion. Pleural fluid analysis showed inflammatory cells with predominant neutrophils with raised proteins and sugars, with normal ADA levels. Pleural fluid culture showed no microbial growth. An Intercostal drain was placed in the 5th intercostal space which was kept for 5 days after which the pleural collections reduced (Fig III).

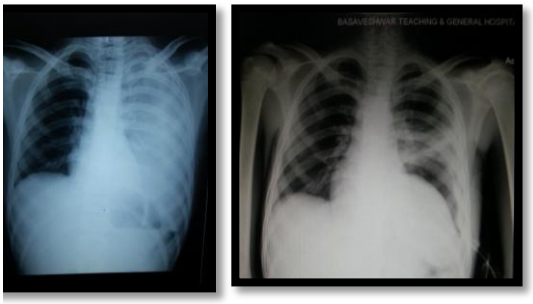

Fig III Chest X-ray, before and after ICD placement

The corrugated abdominal drain output maintained about 300ml-serous fluid each day, until on Post-Operative Day 11 the output became bile stained. The patient started losing weight and the drain output increased to 500-700ml containing partially digested food particles. Patient was managed conservatively with high protein diet, amino acid and multi-vitamin supplements, octreotide injections, blood transfusions, adequate intravenous fluids and local skin care. The output gradually reduced and stopped around Post-Operative Day 22 (Fig IV).

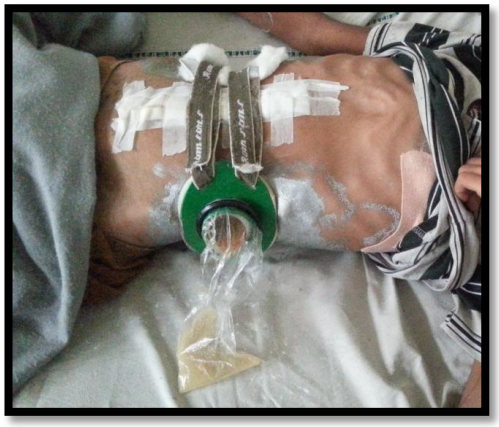

Fig IV Collecting bag around corrugated abdominal drain showing bilious contents

The renal function, liver function and serum electrolytes remained within normal limits. The patient was discharged on Post-Operative Day 30 after a repeat normal chest X-ray and a normal USG abdomen (Fig V).

Fig V Recovering patient in regular follow ups.

A Follow-up Upper GI endoscopy after 6 months showed healthy stomach walls and repair site. (Fig VI)

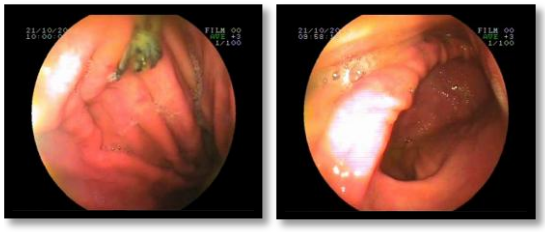

Fig VI Post-operative Endoscopy showing healthy stomach wall and repair site.

DISCUSSION

The rarity of stomach gangrene is attributed to its rich blood supply [1]. In view of the presence of multiple anastomoses between the left gastric artery and branches of the phrenic and esophageal arteries, Babkin et al [3], in 1943 found that even tying up of all the gastric arteries did not cause gastric infarction in the dog. However, Harvey et al [4] reported a case of multifocal gastric infarction secondary to atheromatous emboli originating in a thoracic aortic aneurysm.

Bradley EL et al [5], in their report, described a case with extensive gastric necrosis after therapeutic transcatheter embolization of the left gastric artery with fragments of gelatin

sponge for recurrent massive upper gastrointestinal hemorrhage. Ovnat et al [6] diagnosed acute obstruction of the celiac trunk by abdominal computed tomography (CT) and angiography which they could treat successfully with thrombolytic therapy.

Abscess or spreading cellulitis of the 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 most frequent causative agents, in order of frequency, are Streptococcus, Staphylococcus, Escherichia coli, Haemophilus influenza, Proteus, Clostridia or Mixed bacterial infections [1]. Hematogenous spread from an infective foci involves either a portion of the stomach (Localized Type) or the entire stomach (Diffuse Type).

Acute Necrotizing Gastritis, is a variant of Phlegmonous Gastritis, with organisms producing necrosis and gangrene of the stomach wall rather than just an intramural abscess. If suspected early, diagnosis of acute necrotizing or gangrenous gastritis can be made by endoscopy, endosonography [7] and endoscopic snare biopsy.

However, in our study the gangrene was an intraoperative surprise. With no known history or evidence of corrosive substance ingestion, polyphagia, or any past therapeutic intervention, a definitive etiological diagnosis could not be made. Age of the patient, absence of any predisposing factors and on exploration, pulsatile gastric arteries made vascular accident unlikely. No intra-operative gastric volvulus and bilateral normal diaphragmtic domes, ruled out a strangulated diaphragmatic hernia.

Presence of streptococcus in the peritoneal fluid culture suggests transmigration of the otherwise sterile stomach contents across the gangrenous stomach. Therefore, a diagnosis of Acute Necrotizing Gastritis with Gangrene of the Stomach Wall was considered.

Medical management alone, with broad spectrum antibiotics has also been previously tried for Plegmonous gastritis, but the reported mortality rate is 17% for patients with a Localized Disease and 60% for the Diffuse Disease [8,9]. The mortality rate even after surgical resection is reported to be 20% [10]

In our study, although postoperative complications like pleural effusion and repair site leakage prolonged the treatment, with an adequate resection and good supportive and nutritional therapy we were successful in reviving the patient.

CONCLUSION

Acute Phlegmonous or Necrotizing Gastritis leading to gangrene of stomach is a rare yet fatal condition. An early diagnosis may be difficult but once diagnosed prompt and adequate surgical resection with aggressive medical management,

supportive and nutritional care can help to achieve favourable results.

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