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A Rare Case of Acute Gastric Dilatation With Gastric Wall Necrosis Without Perforation - Case Report

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ABSTRACT

Acute gastric dilatation can have multiple etiologies which may lead to ischemia and necrosis of the stomach. Without proper timely diagnosis and treatment, potentially fatal events such as gastric perforation, haemorrhage, and other serious complications can occur. Here we present a 50-year-old man who came to the casualty with pain abdomen and distension for 3 days. Clinically, abdomen was distended with diffuse tenderness and rigidity. X-ray abdomen showed fundal air shadow without any Air under Diaphragm. Ultrasound Abdomen revealed mild free fluid in Abdomen and Pelvis with internal echoes and two hyperechoeic foci (? Air pockets) with an impression of ? Perforative Peritonitis. Emergency Exploratory Laparotomy revealed grossly dilated stomach with multiple patchy necrotic sloughed out lesions in Anterior stomach wall over greater curvature and fundus involving serosa with impending perforation. About 50ml of serosanguinous fluid was noted in pelvis. Thorough peritoneal lavage with Feeding jejunostomy was done. The patient recovered well and was discharged on the tenth postoperative day.

KEY WORDS: Acute gastric dilation, Gastric wall necrosis, Perforation, Feeding Jejunostomy.

INTRODUCTION

Acute gastric dilatation can have multiple etiologies which may lead to ischemia and necrosis of the stomach. The etiologies are lifestyle habits, underlying morbidities, malnutrition, acute necrotizing inflammation, acute vascular insufficiency, and postoperative complications. Without proper timely diagnosis and treatment, potentially fatal events such as gastric perforation, haemorrhage, and other serious complications can occur. We here present a rare case of gastric dilatation leading to multiple patchy sloughed out necrotic lesions on the surface of stomach without perforation and Feeding Jejunostomy was carried out.

CASE HISTORY

A 50-year-old male patient, a manual labourer, presented to the casualty with complaints of Abdomen pain and Abdominal distension for three days with associated vomiting since past two days. Three days ago he had binge eating which occurred after two days of starvation. His past history was not significant. He was not suffering from any psychiatric illness or any co-morbidity like diabetes, malnutrition, trauma, immuno suppression or any spinal disorders and had not undergone any previous surgeries. He presented with poor general condition, thin built and malnourished, dehydrated with tachycardia. Abdominal examination showed diffuse tenderness with abdominal distension and rigidity with signs of peritonitis. Straight X-ray abdomen showed fundal gas shadow without any air under Diaphragm (Figure 1). Ultrasound Abdomen revealed mild free fluid in Abdomen and Pelvis with internal echoes and two hyperechoeic foci (? Air pockets) with an impression of ? Perforative Peritonitis (Figure 2). Since the patient presented with features of Perforative peritonitis, the patient was planned for Emergency Exploratory Laparotomy. Ryle's tube aspiration was done and the patient was put on Nil per Oral (NPO) and started on intravenous Antibiotics and analgesics. On opening the abdomen through upper midline laparotomy incision, it was found that stomach was grossly dilated with patchy necrotic sloughed out lesions at multiple areas in anterior wall of greater curvature (Figure 4_) and the the fundus of stomach (Figure 3). On palpation, the lesion was found to be involving the serosal layer. Since the patient had propensity for ischaemia, gastrotomy and biopsy was not done for fear of perforation. About 50ml of serosanguinous fluid was noted in pelvis. Rest of the solid and hollow visceral

organs appeared normal. Thorough Peritoneal lavage with Feeding jejunostomy was done (Figure 5). Postoperatively, patient was started with feeds through feeding jejunostomy tube on the 3rd day (Figure 6). He passed flatus and stools on 4th day. He was given liquids orally on the 7th day (Figure 7) and semisolid diet on the 8th day and was discharged on the 10th postoperative day.

Fig.1 : Xray Abdomen Erect showing Fundal gas shadow without any Air under Diaphragm.

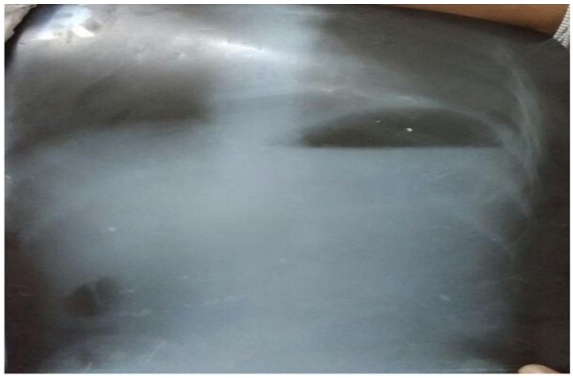


Fig.2: USG report showing ? Perforative Peritonitis

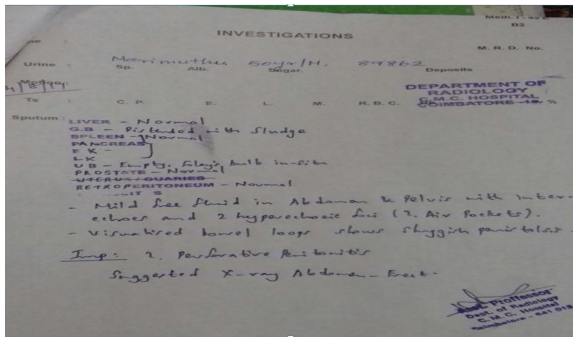


Fig.3: Dilated Stomach with necrotic sloughed out lesion at Fundus

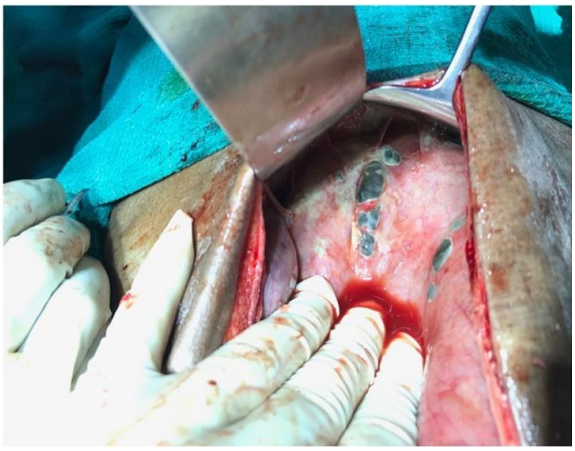


Fig.4: Dilated Stomach with necrotic sloughed out lesion at Greater Curvature

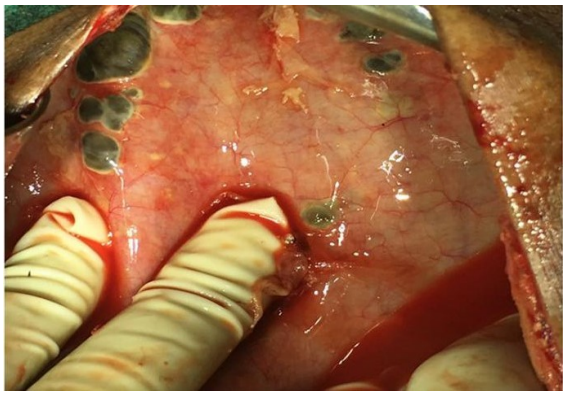


Fig.5: Feeding Jejunostomy

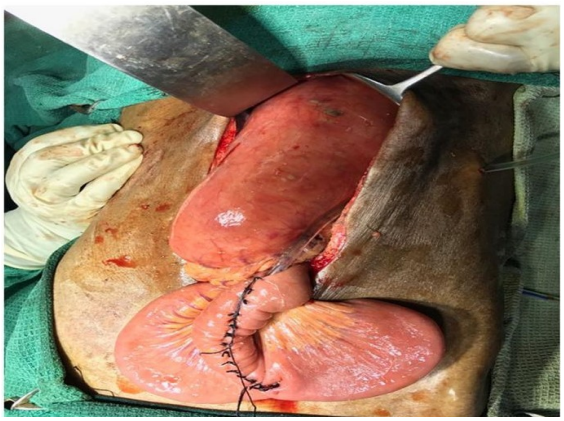
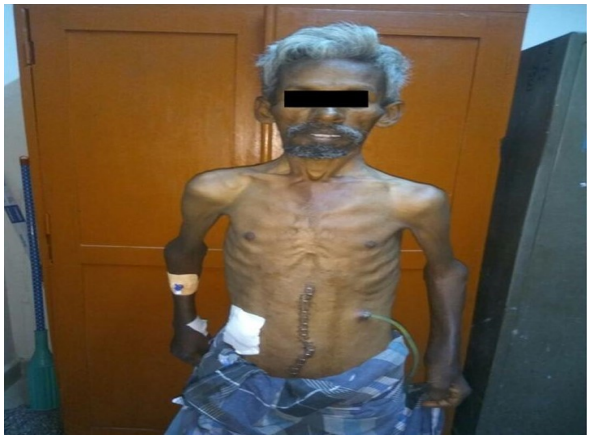


Fig.6: Starting feeds through Feeding Jejunostomy on post- operative Day 3.

Fig.7: Patient after recovery on post operative Day 6.



DISCUSSION

In 1833, Duplay first described acute gastric dilatation [1]. Acute ischemic necrosis of stomach is a very rare disease due to its abundant vascular supply. In experimental animals, in order to produce ischemic necrosis, closure of the right and left gastric and gastroepiploic arteries together with at least 80% of the collaterals is required [2]. The important causes are postoperative complications [3, 4], anorexia nervosa and bulimia, psychogenic polyphagia, diabetes mellitus, malnutrition, trauma, electrolyte disturbances, gastric volvulus, and spinal conditions [1,5–10].

This patient was diagnosed as having atypical eating disorder of an episode of binge eating following two days of Starvation. He did not have any past history of psychiatric illness, diabetic gastropathy, any other comorbid illness, trauma, or any previous surgery on stomach.

A thorough search of PubMed revealed only less than 50 cases of acute gastric dilatation, ischemia, and necrosis recorded till now in the literature. Ischemia is caused presumably due to venous insufficiency when massive dilatation occurs [11, 12]. To impair venous outflow, either 14 mmHg of pressure or more than 3 litres of fluid is sufficient, although more than 15 litres has been described in eating disorders in chronic distension. Rupture can occur with intragastric pressures of more than 120 mmHg or 4 litres of fluid. In the majority of the cases, greater curvature and gastric fundus are more prone for necrosis and require emergency treatment [13]. Lesser curvature and pyloric regions of the stomach tend to be spared [1]. A consequence in events as postulated by Abdu et al. is mucosal necrosis, followed by full-thickness involvement of the gastric wall and perforation [10–12]. Surgery may be avoided if the diagnosis is established in an early stage. A mortality rate of 80% to 100% has been reported due to gastric ischemia and perforation as a result of dilation [14]. In our case, there was impending perforation at the area of necrosis on greater curvature and fundus of Stomach which was tackled before it could perforate and lead to devastating complication.

Several theories have been postulated to explain the pathogenesis of acute gastric dilatation. Morris et al. claimed that anaesthesia and debilitation may be predisposing factor as it is a very frequent postoperative complication. Relaxation of the upper oesophageal sphincter with aerophagia may be a factor leading to gastric distention [3, 4, 10]. In 1859, Brinton introduced the atonic theory [10]. The stomach undergoes atony and muscular atrophy during a period of starvation, so that a sudden ingestion of food overtaxes an already weakened stomach in patients with eating disorders. In 1861, von Rokitansky proposed superior mesenteric artery syndrome (mechanical theory) in which vascular compression of the third segment of the duodenum, between superior mesenteric artery, aorta, and vertebral column, causes acute gastric dilatation [5]. Other authors suggest that pancreatitis, peptic ulcer, gallbladder disease, and appendicitis also cause acute gastric dilatation [15, 16] and infectious causes like necrotizing gastritis generally involving immunocompromised patients like Diabetes, AIDS, and Neoplasia are also reported [17, 18].

In more than 90% of cases of acute gastric dilatation, vomiting is an important and common symptom [19]. Another symptom reported in the literature is the inability to vomit which is not fully understood. This may be due to the occlusion of the gastroesophageal junction by the distended fundus, which angulates the esophagus against the right crus of the diaphragm, producing a one-way valve [20]. Significant, diffuse abdominal distension accompanied by abdominal pain is common.

Plain abdominal films and CT scan can demonstrate gastric distension and free air and are useful in the diagnosis. In our case, since the distension was increasing and huge and clinically presenting with features of Perforative peritonitis, we planned to explore the patient with just abdominal X-ray which showed fundal air shadow without any evidence of Perforation and USG which revealed mild free fluid in Abdomen and Pelvis with internal echoes and two hyperechoeic foci (? Air pockets) with an impression of ? Perforative Peritonitis. Treatment focuses on early diagnosis and decompression of the stomach, thus halting the vascular congestion and thus ischemia [21]. Decompression with nasogastric tube should be the first step in the management, followed by immediate surgery in case of perforation. A normal size nasogastric tube may prove to be inefficient in decompressing stomach. Sometimes, when semisolid material is present in the stomach, even a large tube may be inefficient. In our case too since the contents were semisolid nasogastric tube was nonproductive. If conservative measures fail or gastric infarction with or without perforation is suspected, immediate surgical intervention is mandatory [10]. Necrosis might be partial or involving the full organ. Total gastrectomy is the procedure of choice [14], but it requires time and stable hemodynamic conditions. Partial resections have already been described in case of patchy necrosis and gangrene [22]. Here, we did thorough Peritoneal lavage with feeding jejunostomy with minimal morbidity to the patient. Surgeons should be aware that acute gastric dilatation may occur even in patients who are not diagnosed as having a typical eating disorder after an

episode of binge eating. A high index of suspicion is necessary to diagnose this condition in order to avoid fatal complications. First line of treatment should be conservative with nasogastric decompression. If it fails, necessary timely surgery would prevent unnecessary morbidity. Even though total gastrectomy is the treatment of choice, Feeding Jejunostomy can be done safely in limited patchy necrosis and gangrene.

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