

Acute massive gastric dilatation: a surgical emergency

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Abstract

A 40 year old patient presents with acute pain abdomen with abdominal distension. History of unusual heavy meal one day before and following which symptoms appear. Few episodes of vomiting were associated. Resuscitation done. Straight x ray shows dilated gastric shadow. Patient posted for laparotomy after failing of conservative measures. On opening abdomen, hugely distended stomach seen with thinned out gastric wall and patchy areas of discolouration. A side to side gastrojejunostomy done after decompression on the dependent stomach. Post-operative recovery was uneventful. Psychological evaluation did not reveal any abnormality. Acute gastric dilatation can cause mucosal necrosis and gastric perforation therefore early diagnosis and gastric decompression is the key. Though most patients respond to conservative measures, failing which surgical decompression is needed.

Keywords: Acute gastric dilatation; eating disorders; gastric decompression; gastrojejunostomy; acute massive gastric dilatation

INTRODUCTION

Acute gastric dilatation [AGD] is a rare disorder with most of the references in the literature as case reports. AGD is encountered most often as a postoperative complication in abdominal surgery and in a multitude of disorders, such as anorexia and bulimia nervosa, psychogenic polyphagia, trauma, diabetes mellitus etc. [1-5]. Acute massive gastric dilatation [AMGD] is the extreme form of AGD. In literature the demarcation of AGD and AMGD is not clearly mentioned. When the stomach is extremely distended occupying the abdomen from diaphragm to pelvis and from left to right, the AGD is referred to as AMGD. Most frequently AMGD requires surgical intervention to prevent or to treat gastric necrosis [3].

We present a case of acute massive gastric dilatation with thinned out gastric wall but without perforation. After failure of conservative measures, the patient was treated surgically.

CASE REPORT

A 40 year old male patient came to the emergency department at night with complaints of acute abdominal pain and distension, multiple episodes of vomiting and obstipation for 2 days.

The patient was apparently well until 2 days ago when after having an unaccustomed heavy lunch he started complaining of abdominal distension which was not relieved by any means but aggravated with food and fluid intake. The distension progressed gradually with abdominal pain and multiple episodes of vomiting. The vomitus comprised of food materials, non-bilious, non-projectile and vomiting did not relieve the distension. There was no history of hematemesis or fever. There was history of obstipation for which he received enema at the primary health centre, referred thereafter to our hospital.

The patient had no history of alcohol/tobacco addiction. He had normal bowel and bladder habits with one episode of binge eating two days ago. There was no history of any chronic illness like diabetes or hypertension. He was not taking any medication and did not undergo any surgery before.

On general examination, the patient had no signs of anaemia, oedema, jaundice, clubbing or cyanosis. He was of average built and nutritional status. At the time of admission his pulse was 110 per minute and blood pressure was 110/70 mmHg.

On local examination, the abdomen was hugely distended with no dilated veins, scar mark and the umbilicus everted. There was generalised tenderness. There was no ascites or any abdominal lump. There was moderate

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Received: 10 September 2019 Accepted: 25 October 2019

amount of guarding and rigidity but no rebound tenderness.

After admission the patient was resuscitated with normal saline and urgent upright abdominal x-ray was done. The x-ray shows grossly dilated stomach (Figure 1) but there was no evidence of free gas under the diaphragm. A large bore nasogastric tube was inserted and careful suctioning done. Scanty food materials and fluid came out but then the material stopped coming out of the stomach. The nasogastric tube repositioned and changed but still without any yield. Then the NG

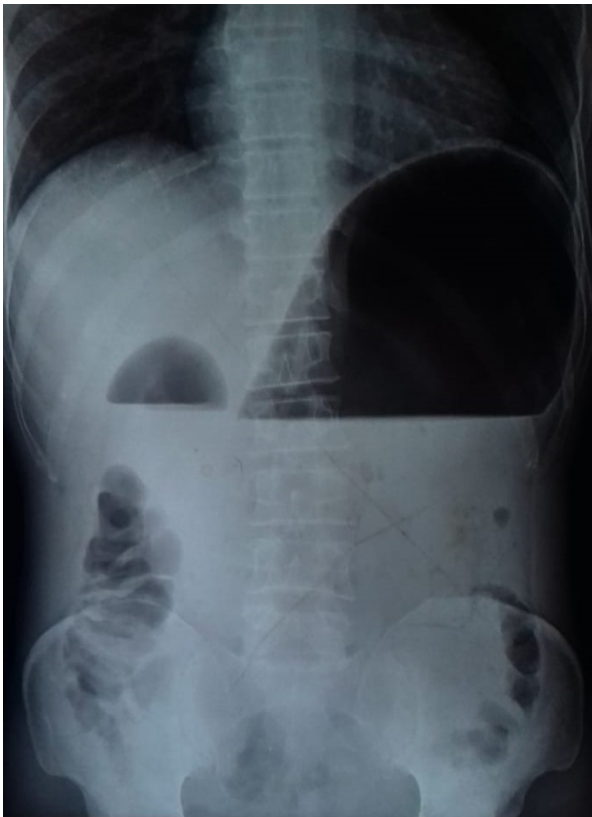


Figure 1. Plain radiograph in erect posture shows hugely dilated stomach.

tube was subjected to continuous vacuum suction pump but still nothing evacuated. The abdominal distension did not improve and the patient's condition gradually worsened. The abdominal pain was not relieved either. At this stage conservative measures abandoned and the patient was posted for emergency laparotomy after taking high risk consent. Standard midline vertical incision given. As soon as the peritoneum opened, hugely distended stomach was seen extending from xiphisternum to below umbilicus going towards the pelvis. The stomach wall was thinned out but there was no perforation or obvious necrosis but a few patchy areas of slightly dusky discoloration seen with one area of serosal tear



Figure 2. Operative picture shows massively dilated stomach with thinned out wall and an area of serosal tear.

(Figure 2). There was no rotation of the stomach and the rest of the bowel was normal. Liver, spleen was normal, there was no fluid inside the peritoneal cavity and no other lump noted.

Again NG tube suctioning was tried intraoperatively but it failed to relieve the massively dilated stomach. Fearing impending necrosis or perforation of the stomach we decided to go for surgical decompression by doing dependent small gastrotomy near the greater curvature. On opening the stomach more than 2.5 litres of food materials and fluid evacuated along with few large apparent chunks of food. The mucosa around the gastrotomy site was normal.

Posterior, retrocolic, isoperistaltic side to side gastro-jejuno-stomy done in two layers incorporating the gastrotomy. Primary repair done for the serosal tear with absorbable suture.

Full psychological evaluation of the patient done in the post-operative period but no abnormality including any eating disorders was found.

The recovery was uneventful with the patient passing flatus on third day and discharged on satisfactory condition after 7 days. Upper GI endoscopy was performed after 6 weeks and it shows normal gastric mucosa and with good and healthy anastomosis and no other abnormal finding.

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% blockage of the collaterals is required^[2]. The important causes are postoperative complications^[3,4], anorexia nervosa and bulimia, psychogenic polyphagia, diabetes mellitus, trauma, electrolyte disturbances, gastric volvulus, and spinal conditions^[1, 5-10].

Our patient was of average built and nutrition. He was not anorexic and psychological evaluation did not reveal any abnormality. He was non diabetic and there was no history of any other chronic illness or any prior surgery. He had one binge eating episode about 48 hours ago. Afterwards he developed the symptoms and was admitted in a primary health centre from where the patient was referred to our institution.

Ischemia is caused presumably due to venous insufficiency when massive dilatation occurs^[11, 12]. To impair venous return, either 14mmHg 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 120mmHg or 4 litres of fluid. In the majority of the cases, greater curvature and gastric fundus are more prone for necrosis and require emergent treatment^[13]. Lesser curvature and pyloric regions of the stomach tend to be spared^[1].

A consequence of 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, the stomach was hugely dilated and the greater curvature was below the umbilicus going towards the pelvis. The gastric wall was thinned out and few patchy areas of discoloration seen. No definite area of full thickness gastric wall necrosis seen. There was one area of serosal damage with impending perforation seen on the body of the stomach. Patient received naso gastric suction at the primary health centre which we believe although failed to relieve the patient, but prevented the rise of intra-gastric pressure to very high level and causing full thickness necrosis of the stomach.

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 Rokitsansky 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 sign 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 oesophagus 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 radiograph and CT scan can demonstrate gastric distension and free air if present. In this patient plain abdominal radiographs revealed grossly distended stomach but no free air. As the patient was not improving with conservative measures and he was clinically deteriorating, we opted for emergency laparotomy fearing imminent perforation or necrosis. 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 non-productive. If conservative measures fail or gastric infarction with or without perforation is suspected, immediate surgical intervention is mandatory^[10].

We performed a gastrotomy for proper decompression of the stomach near the greater curvature at the most dependent part and after decompressing, we performed a side to side, two layered gastrojejunostomy. Primary repair done of the small area of serosal damage. We believe that, in this condition a drainage procedure was better than simple closure of the gastrotomy wound in view of preventing recurrence. It has been reported to perform partial or even total gastrectomy depending on the area of the necrosis and general condition of 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 unaccustomed 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.

DECLARATIONS

Conflicts of interest

The authors declare that there is no conflict of interest among the authors regarding publication of this paper.

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