Acute massive gastric dilatation: A rare cause of gangrene of stomach

Acute gastric dilatation (AGD) followed by necrosis is a rare and potentially fatal event; it is usually seen in patients with eating disorders such as anorexia nervosa or bulimia. A 13-year-old girl with no remarkable medical history was brought to the Emergency Department suffering acute abdominal symptoms. Emergency laparotomy revealed massive gastric dilatation and necrosis of most of the stomach. We performed subtotal gastrectomy and the patient recovered uneventfully. We report this case to demonstrate that AGD and subsequent gastric gangrene can occur in patients without any underlying disorders.

Key words: Gastric dilatation, gangrene, stomach

INTRODUCTION

Acute gastric dilatation (AGD) is a rare entity, with varying aetiologies, the majority of which are benign. Prompt recognition and appropriate management is essential to prevent sequelae such as gastric emphysema (pneumatosis), emphysematous gastritis, gangrene, and perforation. It is encountered often as a postoperative complication in abdominal surgery and also in a multitude of disorders, such as anorexia and bulimia nervosa, psychogenic polyphagia, trauma, most common of these being anorexia nervosa.[1‑4] The pathogenesis of AGD is still unclear, with different theories being postulated. The gastric reservoir is well known for its rich vascular network which generally protects it from ischemia when significant gastric distension occurs, so gastric necrosis is a very rare event.[5‑7] But in situations where the stomach is extremely distended occupying the abdomen from diaphragm to pelvis, intragastric pressure from gastric distension exceeds 20 cm H₂O, intramural blood flow is impaired, and results in gastric ischemia and necrosis.[8,9] It can have devastating consequences and has a reported mortality rate of 80% to 100% as a consequence of gastric necrosis and perforation. Early diagnosis with a prompt gastric decompression in the phase of parietal ischemia and mucosal necrosis is critical since delay may result in gastric necrosis, perforation, shock, and death. We present here a case of acute massive gastric dilatation (AMGD) resulting in gastric gangrene and the patient could be salvaged by urgent laparotomy and partial gastrectomy.

CASE REPORT

A 13-year-old female patient presented to the emergency department with complaints of acute-onset abdominal pain and progressively distended abdomen for the past 2 days. She had persistent nausea with multiple episodes of vomiting at the start of symptoms but afterward she was unable to vomit although she had attempted several times. She was taken to a local practitioner for the complaints where she received symptomatic treatment and was then referred to our institute. On examination, the patient was conscious and her vitals were stable, pulse rate was 110/min feeble, blood pressure was 90 mmHg systolic. On abdominal examination, the abdomen was massively distended, with diffuse tenderness with no guarding or rigidity on palpation. On investigation, a complete blood count revealed a leucocytosis of 12.5×10³/mm³, hemoglobin 15.8 g/dL, and a normal platelet count. Intravenous fluid replacement was immediately started and a nasogastric tube was placed without difficulty and about 100 cc of dark colored hemorrhagic fluid could be aspirated. A plain abdominal film showed a hugely dilated gastric shadow occupying the abdominal cavity entirely from diaphragm to pelvis and a large intragastric air fluid level without any free air in the peritoneal cavity [Figure 1]. The nasogastric tube, which was left in place, continued to be unproductive. After resuscitation she was taken to the
operative theatre and exploratory laparotomy was done and the operative findings were: The stomach was massively dilated with most of it gangrenous, only some viable appearing part around cardiac area and portion of body near pyloric area [Figure 2]. There was no defect in hiatal area and the stomach was normally placed without any volvulus. Resection of whole of the gangrenous part was done with repair of a viable-appearing stomach wall. A drain was put around the repaired stomach. Postoperatively the patient was kept nil per orally and a nasogastric tube was left in place for gastric decompression for about 10 days. Her condition gradually improved but there was pus discharge from the drain site which continued for 2 weeks. A postoperative CT scan of the abdomen was done which revealed collection in the pelvis which resolved on conservative management. The patient was discharged home in third week in good condition with advice of liquid and semisolid diet. The patient is well after 2 months of follow up.

**DISCUSSION**

AGD was first described by Powell *et al.* in 2003 and it can be as a result of eating disorders, hemorrhage/truma resuscitation, volvulus, medications, electrolyte abnormalities, infections, superior mesenteric artery syndrome, diabetes mellitus, and slow gut transits causing chronic constipation. It can have devastating consequences and has a reported mortality rate of 80% to 100% as a consequence of gastric necrosis and perforation. The pathogenesis of AGD is still debated as several theories have been postulated. Due to its frequency as a postoperative complication, Morris *et al.* claimed that anesthesia and debilitation may be predisposing factors. These factors can cause relaxation of the upper esophageal sphincter with aerophagia leading to gastric distention. In patients with eating disorders, the stomach undergoes atony and muscular atrophy during a period of starvation, so that a sudden ingestion of food overtaxes an already weakened stomach. A mechanical theory, commonly known as superior mesenteric artery syndrome (SMAS), was proposed by von Rokitansky in 1861 in which AGD follows vascular compression of the third segment of the duodenum, between superior mesenteric artery, aorta, and vertebral column. Other authors suggest that the AGD may be a functional entity secondary to regional diseases, such as pancreatitis, peptic ulcer, gallbladder disease, appendicitis, etc. Patients with anorexia nervosa and bulimia nervosa are at increased risk for AGD due to decreased gastric motility, increased gastric capacity, and decreased gastric emptying.

Even years after the cessation of anorexia nervosa, patients may still have gastric dysmotility, predisposing any patient with a history of an eating disorder to AGD. Studies into normal gastric capacity using intra-gastric balloon expansion demonstrate that healthy individuals tolerate volumes of up to 800 mL and it has been demonstrated that 4 L is the capacity of the stomach before perforation occurs. Normally stomach is very resistant to ischemia due to its rich blood supply and its extensive intramural anastomoses. Experimental studies have shown that both arterial and venous circulation must be interrupted before gastric ischemia and necrosis can occur. Increased intragastric pressure is usually the result of a closed loop, secondary to mechanical compression of the cardio-esophageal and pyloro-duodenal or duodeno-jejunal junctions as seen in volvulus. However, in this patient there was no evidence of any volvulus because the Ryle’s tube was easily placed which is difficult in patients of gastric volvulus. In 1970, Edlich demonstrated that a massive gastric dilatation results in a decrease intramural blood flow when the intragastric pressure exceeds 30 cm H₂O. At this pressure regimen the consequent occlusion of the venous drainage due to the high intraluminal tension has been surely a highly significant factor in our case too. Moreover, we believe the abnormal gastric dilatation with compression of the inferior vena cava caused a decreased venous return, with consequent marked hypotension. Thus, the associated splanchnic vessel sequestration worsened and the increased intragastric pressure over 20 cm H₂O results in mucosal necrosis. As the intragastric pressure rises above 30 cm H₂O, a decrease in venous outflow may result in ischemia and infarction of the gastric wall which can rupture. In cases of AMGD, intragastric pressure usually exceeds 30 cm H₂O and produces a dramatic decrease of intramural blood flow, with necrosis and perforation. As there is no other pathology like volvulus or SMA syndrome observed in this patient, sudden massive gastric dilatation might have resulted the infarction and gangrene of the gastric wall.
Patients with AGD typically present with sudden onset abdominal pain, distention, and inability to vomit. Why these patients are not able to vomit is not fully understood. It has been suggested that 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. The abdomen will be tympanic on percussion and a succussion splash may be elicited, but only if the gastric contents are liquid. Plain abdominal radiography will review the grossly enlarged gastric shadow, and nasogastric insertion can be both diagnostic and therapeutic. The mortality rates associated with gastric wall necrosis and rupture have been reported to be 37.5% and 55.6%, respectively. Initial treatment of AGD includes nasogastric decompression and fluid resuscitation. However, when nasogastric decompression is impossible due to the large retained food items, urgent laparotomy and gastrostomy are required to evacuate the stomach contents. In stable patients, a CT scan or other imaging may be useful to assess the feasibility of NGT decompression and to determine the etiology of the gastric outlet obstruction. However, unstable patients with suspected AGD should undergo immediate diagnostic and therapeutic laparotomy without delay for imaging to prevent fatal complications.

REFERENCES


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