

Total Gastrectomy Performed in Emergency Conditions for Gastric Necrosis Due to Acute Gastric Dilatation

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Rezumat

Gastrectomie totală în urgență pentru necroză completă a stomacului prin dilatație acută gastrică

Dilatația acută gastrică masivă reprezintă un eveniment rar citat în literatură, a cărui patogeneză este încă discutată. Dilatația acută gastrică masivă conduce aproape invariabil la necroză gastrică cu sau fără perforație. Prezentăm cazul unei paciente de 50 ani, internată în condiții de urgență în colaps, cu distensie abdominală marcată instalată brusc în ultimele 12 ore. S-a intervenit chirurgical de urgență decelând un stomac ce ocupa întreaga cavitate peritoneală, cu necroză cvasicompletă gastrică și cu prezența unei perforații la nivel piloric anterior. S-a practicat gastrectomie totală cu anastomoză Esojejunală mecanică pe ansă în „Y” a la Roux. Evoluția postoperatorie a fost simplă, lipsită de complicații. Dilatația acută gastrică este o afecțiune severă, cu potențial letal, în a cărei etiologie sunt incriminați factori psihogeni ca anorexia nervosa sau cauze traumatice, diabet, perioada postoperatorie în chirurgia abdominală, diselectrolitemii, etc. De cele mai multe ori tratamentul chirurgical se impune de urgență, mai ales atunci când se asociază fenomene de necroză sau perforație. Tratamentul conservator poate reprezenta o opțiune dacă este instituit precoce.

Cuvinte cheie: dilatație acută gastrică, necroză gastrică, gastrectomie totală, anastomoză mecanică

Abstract

Acute massive gastric dilatation is a rare event, with a pathogenesis still debated. Acute massive gastric dilatation invariably leads to necrosis and perforation. We present the case of a 50 year-old woman, admitted in emergency conditions with circulatory collapse and abdominal distension with the onset 12 hours prior to presentation. An emergency laparotomy was performed revealing a stomach occupying the entire abdominal cavity with necrosis and anterior pyloric perforation. The operation consisted in total gastrectomy with stapled Roux-en-Y anastomosis. The postoperative evolution was simple, without complications. Acute massive gastric dilatation is a severe, lethal condition with multiple etiologic factors like anorexia nervosa, trauma, diabetes, postoperative period in abdominal surgery, electrolyte disturbances. In most cases emergency surgical treatment is necessary, dictated by gastric necrosis or perforation. Conservative treatment may represent an option if it is early instituted.

Key words: acute gastric dilatation, gastric necrosis, total gastrectomy, stapled anastomosis

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Introduction

Acute gastric dilatation was first described by S.E. Duplay in 1833 (1). Multiple factors are involved in its etiology, like:

eating disorders (polyphagia), diabetes, gastric volvulus, diselectrolytemia, trauma resuscitation, superior mesenteric artery syndrome and postoperative period in abdominal surgery (2,3,4). Although rare, the condition could have disastrous consequences through gastric necrosis or perforation leading to a reported mortality of up to 80-100%.

Case report

Female patient, 50 years old, with no medical history, 46 kg, 150 cm, admitted in the emergency room - resuscitation unit, with altered state, presenting abdominal pain started 12 hours prior to hospital presentation, associated with abdominal distension and repeated vomiting. At physical exam we detected a conscious patient, in circulatory collapse (blood pressure = 74/50 mmHg, heart rate = 105/min), with cold, mottled teguments, presenting an abdomen with important distension, with diffuse pain at palpation and abdominal rebound tenderness in the superior abdominal area.

Laboratory exams on admission revealed leucocytosis (18.500/mmc), high levels of hepatic cytolysis enzymes, high amylasemia (431 U/L), without others changes.

CT exam described a dilated stomach with air and hypersecretion, occupying the entire abdomen, with emphysematous

gastric wall, fluid in the peritoneal cavity surrounding the liver and in the pelvic area, leading to a preoperative diagnosis of acute gastric dilatation. (Fig. 1)

The patient was intensively resuscitated preoperatively with crystalloid iv fluids and plasma expanders, and a nasogastric tube which decompressed the stomach, evacuating air and approximately 2 l of liquid gastric content was mounted.

An emergency laparotomy was performed, intraoperatively visualizing a dilated stomach, occupying the entire peritoneal cavity, with gastric cvasicomplete necrosis respecting the pylorus, with gastric pneumatosis and a 3 mm perforation in the anterior wall of the pyloric region. A transudative peritonitis, without alimentary gastric content in the peritoneal cavity was also described. (Fig. 2)

Intraoperatively, the stomach was emptied through the nasogastric tube, evacuating approximately 6 l of non-alimentary hematic content.

Next, after gastric decompression, in the contextual situation of irreversible ischemic gastric lesions with normal esophagus and pylorus, but with an anterior pyloric perforation of 3 mm, we decided to perform a total gastrectomy with Roux-en-Y mechanical anastomosis with stapling devices like PP-CEEA, TA and PURSTRING. (Fig. 3)

After the resection of the necrotic stomach, the duodenal



Figure 1. CT scan showing gastric dilatation

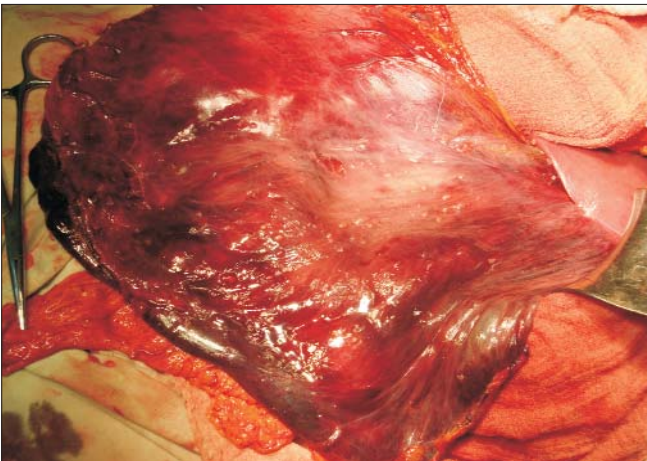


Figure 2. Intraoperative gastric necrotic aspect after decompression

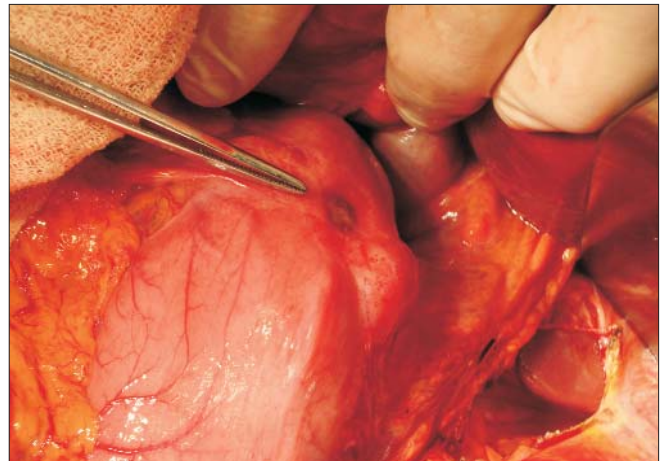


Figure 3. Intraoperative aspect of the anterior pyloric perforation

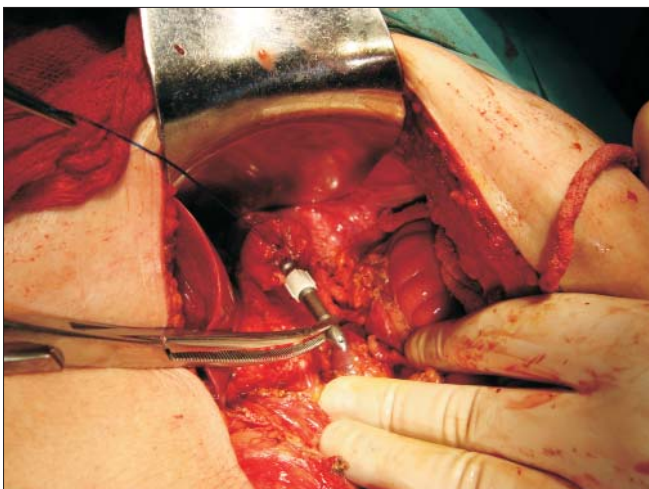


Figure 4. Intraoperative aspect of the anvil introduced in the esophageal distal stump

stump was closed by TA 55 linear stapling. The anvil of the circular stapling device was introduced into the esophagus and secured by a PURSTRING 45 suture. (Fig. 4)

Then, the Roux-en-Y esophago-jejunal anastomosis was performed using a circular stapler PP-CEEA 25, the proximal end of the jejunal loop being closed by linear stapler TA 55 and fixed to the phrenoesophageal ligament. (Fig. 5)

Afterwards, the jejuno-jejunal anastomosis was realized at 50 cm below the esojejunosomy in manual manner with closure of the mesenteric and mesocolonic defects. (Fig. 6, 7)

The postoperative evolution was simple, without complications, under sustained treatment consisting in water-electrolyte re-equilibration, parenteral nutrition and antibiotherapy, with discharge on the 9th postoperative day with normal digestive tolerance and good general condition.

Discussions

Acute gastric dilatation is a rare condition, with debated physiopathology and different theories postulated in time, many surgeons like Corneliu Adamesteanu expressing their interest towards in this problem. In 1842 Rokitanski describes the superior mesenteric artery syndrome, considering that the gastric dilatation is determined by a duodenal obstruction produced between the superior mesenteric artery and the aorta (3). Brinton in 1859 introduces the atonic theory in patients with eating disorders (3). In those cases during the starvation period the stomach suffers atony and muscular atrophy so that a sudden ingestion of food overtakes an already weakened stomach.

It is well known that the stomach is very resistant to ischemia due to its rich blood supply. Gastric ischemia is caused in acute gastric dilatation by venous insufficiency. Gastric ischemia occurs when the intraluminal pressure is higher than 14 mmHg (20 cm H₂O), exceeding parietal venous pressure. In order to create such conditions approximately 3 l of intragastric content are necessary in acute cases (3,5).

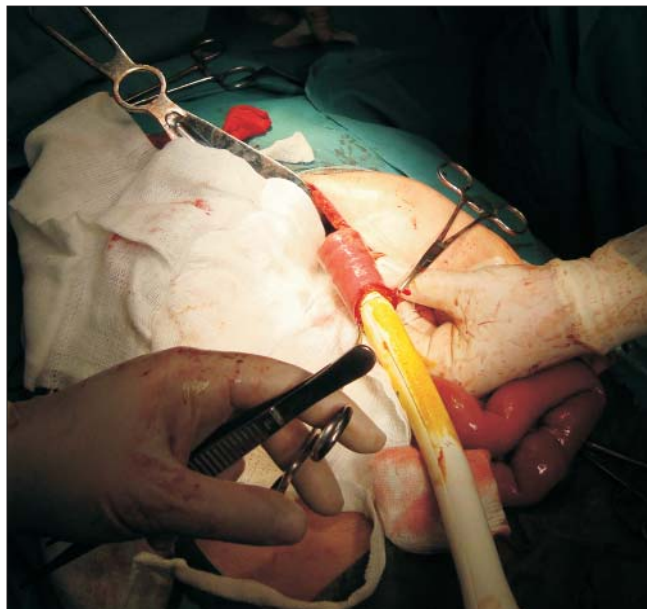


Figure 5. The preparation of esojejunosomy with circular stapling device PPCEEA 25

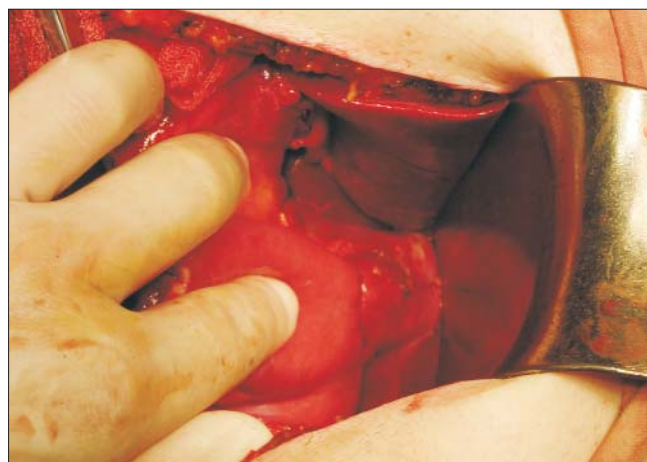


Figure 6. Final aspect of Roux-en-Y anastomosis

Perforation can appear at an intragastric pressure of 120-150 mmHg, meaning 4 l of intragastric fluid. (2)

Symptoms of acute gastric dilatation can be initially vague. Over 90% of patients may present vomiting, but there can be



Figure 7. Aspect of the resected stomach

situations in which the patient is unable to vomit. (3,6) This fact is due to the distension of the gastric fundus which closes the esocardial junction, producing an unidirectional flap-valve mechanism. Diffuse abdominal distension later appears, accompanied by abdominal pain. However, pain is sometimes mild in intensity in contrast with the massively distended abdomen.

Plain abdominal X-ray may reveal a fluid level in a markedly distended stomach or may show the presence of pneumoperitoneum when perforation occurs.

The most useful diagnostic investigation is an abdominal CT scan that can demonstrate gastric distension or can also reveal a superior mesenteric artery syndrome or other associated pathologies. (3)

The treatment of acute gastric dilatation is based on early detection and gastric decompression throughout a nasogastric tube, which may improve the ischaemic lesions up to remission. First line treatment is fluid resuscitation. Upper endoscopy can be performed on patients with hemodynamic stability and without clinical signs of perforation, in order to evaluate the lesions.

Likewise, in rare cases, conservative measures can be successfully applied, even in the presence of gastric ischemia (3,4).

In most cases, emergency surgical treatment is mandatory, especially when necrosis or perforation occurs. The surgical attitude depends on the extension of gastric necrosis. The majority of the findings reported in literature show that ischaemic modifications occur in the greater curvature, the lesser curvature and the pyloric region being spared. (2) The surgical procedures may consist in partial gastric resections up to total gastrectomy with or without feeding jejunostomy. (2,5)

The majority of authors sustain that total gastrectomy is the safest option, due to the delayed ischemia and poor healing of the gastric tissue left behind (2).

After consummation of the acute episode, a psychiatric exam of the patient is necessary.

Overall, the surgical mortality rate has been described as 50% to 65%, and without appropriate treatment it is 100% (2,6).

Our patient's case is particular considering the rarity of the condition and that we were unable to establish the etiology, the

patient denying any eating disorders, ingestion of corrosive substances or psychiatric problems. The therapeutic decision was an emergency laparotomy based on clinical presentation and exam and on imagistic description.

Also particular was the intraoperative discovery of a massive gastric dilatation associated with pyloric perforation, which imposed the performance of a total gastrectomy. The gastric decompression through nasogastric tube of approx. 8 l of gastric content explains the physiopathological context that determined gastric ischemia up to necrosis and perforation.

The possibility of using stapling devices facilitated the operation performed in emergency conditions, offering the patient all the known benefits, such as the quality of the anastomosis performed on a patient with poor nutritional status, hemodynamic unstable and with high risks of developing anastomotic leaks (7).

Postoperative care was complex, involving parenteral nutrition with simple evolution, without complications.

In the presented case we consider that the surgical decision and the performance of a total gastrectomy were the only therapeutic measures that could offer the patient the optimal conditions of surviving. The patient was followed-up at 3 and 6 months postop, presenting a good biological and psychological status, with weight gain of approx. 10 kg.

Conclusions

Acute gastric dilatation is a rare condition with lethal consequences. Early diagnosis of this pathology is essential in the management and prognosis of the patient. Conservative treatment may represent an option if it is early instituted. In most cases, emergency surgical treatment is necessary, especially when necrosis or perforation occurs.

The major benefit offered in patients admitted and operated in emergency conditions is represented by stapling devices, which leads to the idea of implementing The National Program of Mechanical Sutures.

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