

Gastric necrosis: A late complication of nissen fundoplication

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Abstract

Gastric necrosis is a rare condition because of the rich blood supply and the extensive submucosal vascular network of the stomach. "Gas-bloat" syndrome is a well known Nissen fundoplication postoperative complication. It may cause severe gastric dilatation, but very rarely an ischemic compromise of the organ. Other factors, such as gastric outlet obstruction, may concur to cause an intraluminal pressure enough to blockade venous return and ultimately arterial blood supply and oxygen deliver, leading to ischaemia. We report a case of a 63-year-old women, who presented a total gastric necrosis following laparoscopic Nissen fundoplication and a pyloric phytobezoar which was the trigger event. No preexisting gastric motility disorders were present by the time of surgery, as demonstrated in the preoperative barium swallow, thus a poor mastication (patient needed no dentures) of a high fiber meal (cabbage) may have been predisposing factors for the development of a bezoar in an otherwise healthy women at the onset of old age. A total gastrectomy with esophagoje-

junostomy was performed and patient was discharged home after a 7-d hospital stay with no immediate complications. We also discuss some technical aspects of the procedure that might be important to reduce the incidence of this complication.

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Key words: Gastric dilatation; Gastric outlet obstruction; Necrosis; Fundoplication; Nissen operation

Core tip: Gastric necrosis is a rare condition because of the rich blood supply and the extensive submucosal vascular network of the stomach. "Gas-bloat" syndrome is a Nissen fundoplication postoperative complication that causes gastric dilatation, but very rarely an ischemic compromise of the organ. We report a case of a 63-year-old women, who presented a total gastric necrosis following laparoscopic Nissen fundoplication and we discuss technical aspects of the procedure that are important to prevent this complication.

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INTRODUCTION

The rich blood supply of the stomach preserves this viscera from ischemic events, even after the ligation of all the major vessels^[1] (a. gastrica dextra, a. gastrica sinistra, a. gastro-omentalis dextra, a. gastro-omentalis sinistra and aa. gastricae breves). Notwithstanding this fact, acute gastric dilatation accompanied with or without gastric outlet obstruction^[2-7], eating disorders^[8-10] or gas-bloat syndrome are recognized causes of ischemic gastric necrosis.

The gas-bloat syndrome is defined as a variable group



Figure 1 Plain abdominal radiography.

of symptoms resulting from the inability to relieve gas from the stomach after fundoplication. Its incidence may vary from 1% to 85%^[11]. Gastrointestinal gas may proceed either from an excessive production (carbohydrate or fat rich food, small intestinal bacterial overgrowth) or from an excess of swallowed air (dysphagia secondary to oropharyngeal or esophageal motility disorders or anxiety disorders with inefficient chewing, gastroesophageal reflux disease, *etc.*). The predominant complaint is bloating, but other symptoms include abdominal distention, early satiety, nausea, upper abdominal pain, flatulence, inability to belch, and inability to vomit^[11]. Antireflux surgery may contribute to the obstruction of gas flow into the esophagus by means of different mechanisms^[12] (surgically altered physiology of the gastroesophageal junction, surgical injury to the vagus nerve, mechanical compression of the wrap), specially when associated to previous gastroesophageal motility disorders, such as delayed gastric emptying.

Delayed gastric emptying is a preexisting condition in many of the patients undergoing antireflux surgery. It is associated with gastroesophageal reflux disease (GERD) in up to 40% of patients, but Nissen fundoplication is known to accelerate gastric emptying and has a high rate of success controlling GERD-related symptoms^[13], thus delayed gastric emptying is not a contraindication for antireflux surgery^[14]. Nevertheless, a thoughtful preoperative assessment of esophagogastric motility with barium swallow is mandatory and may identify a subset of patients that will still have symptoms related to motility disorders postoperative.

Very few cases of near-total or total gastric necrosis following Nissen fundoplication have been reported. We present a case of gastric necrosis following laparoscopic Nissen fundoplication and pyloric obstruction by a phytobezoar.

CASE REPORT

A 63-year-old woman was admitted to our Emergency Room with a history of sudden abdominal pain, without nausea or vomiting. No other symptoms were reported. Past medical history revealed acetylsalicylic acid intoler-



Figure 2 Gastric mucosal necrosis, endoscopic view.

ance, osteoporosis, supraspinatus rupture and gastroesophageal reflux disease secondary to a type I hiatal hernia, with a normal barium swallow esophagogastric motility pattern, a DeMeester of 33.1 and pathologic acid-clearance in pH-metry and a mild hypotonic lower esophageal sphincter in manometry, for which she underwent laparoscopic Nissen fundoplication 7 mo ago, with no postoperative complications and a 3-d hospital stay. The surgical record described a Rossetti-Nissen fundoplication without diversion of short gastric vessels (SGV), with a short wrap (3 cm) and suturing of the valve to the right crura. Treatment history revealed no medication that could interfere with upper digestive tract motility.

On physical examination, she was conscious, alert and oriented. Blood pressure was 123/75 mmHg with a pulse rate of 76/min. The abdomen was distended, painful to palpation with generalized peritonism and involuntary guarding in the epigastrium. Bowel sounds were diminished. Laboratory findings revealed: hemoglobin, 14.6 g/dL; hematocrit, 44.7%; white blood cell count, 18900/ μ L (with 15500 neutrophils); platelet count, 372000/ μ L; lactic dehydrogenase and amylase were elevated to 251 U/L (normal, 87-246 U/L) and 564 U/L (normal, 25-115 U/L), respectively. Arterial blood gases showed a metabolic acidosis with a blood pH of 7.23.

The patient was taken to the radiology department and plain abdominal films were performed (Figure 1). A gastroscopy was performed after X-rays, since a nasogastric tube couldn't be placed, and a wide area of mucosal necrosis was found in the posterior wall of the lesser curvature (Figure 2). In the operating room an extreme gastric dilatation was found with ischemic changes. The lesser sac was opened and dissection of the posterior gastric surface confirmed endoscopic findings. An anterior longitudinal gastrotomy was performed and trapped air was released. Suction of the gastric chamber through the gastrotomy revealed a phytobezoar in the pyloric channel. Total gastrectomy (Figure 3) with esophagojejunostomy and Roux-en-Y reconstruction was performed. The pathology (Figure 4) revealed a transmural submassive ischemic necrosis with intravascular thrombi. After a postoperative period without complications, the patient was successfully discharged home.

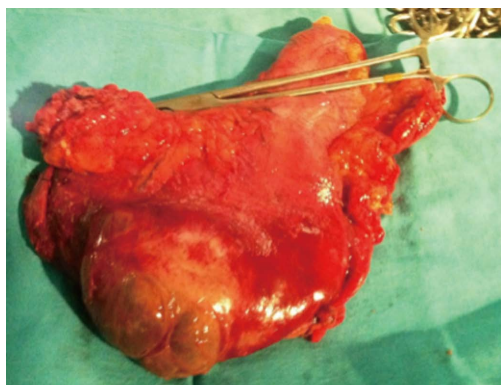


Figure 3 Transmural gastric necrosis, total gastrectomy surgical specimen.

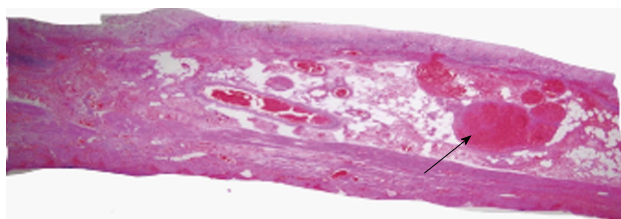


Figure 4 Submassive transmural gastric necrosis with intravascular thrombi (arrow); Hematoxylin-Eosin stain.

DISCUSSION

Gastric dilatation is a minor postoperative complication that can eventually occur after major surgery. This condition usually resolves spontaneously or with the insertion of a nasogastric tube for gastric decompression. On the contrary, ischemic necrosis following extreme gastric dilatation is a very rare phenomenon and requires urgent diagnosis and surgical treatment.

Clinical manifestations of the “gas-bloat” syndrome (abdominal pain, distension, tympanic percussion, *etc.*) are common to many other abdominal pathologies, that without a full comprehension of the surgical history, may delay the diagnosis with the increased morbidity and mortality that this entails. Early surgical consult for the evaluation of patients with abdominal pain and surgical history is vital in the emergency room context and decisive for patient outcome.

In the Nissen antireflux procedure, compression of the distal oesophagus by the gastric wrap is required to ensure the one-way valve effect that prevents gastric reflux. Expelling trapped air and belching is therefore sometimes hindered, resulting in a progressive gastric dilatation. Thanks to the antegrade propulsion, which works as an exhaust valve, this condition is intermittent. When antegrade propulsion is totally or partially blocked, gastric distension progressively tightens the periesophageal wrap, thus increasing the intragastric pressure and compromising blood flow. Some studies have shown that an intragastric pressure greater than 20–30 cm H₂O is necessary to cause the occlusion of the gastric luminal blood circulation^[15]. Cases of gastric necrosis following acute small bowel obstruction based on adhesions and

gastric outlet obstruction based on antral gastric cancer and trichophytobezoar after an antireflux technique^[3–5] have been reported. In infants, this rare post Nissen complication has also been described^[16]. In the case we present, the trigger event was a phytobezoar causing pyloric obstruction.

Since 1955, when Rudolph Nissen performed his first fundoplication, many other procedures that try to reduce the incidence of postoperative complications, including the “gas-bloat” syndrome, have been developed, without a loss of effectiveness in preventing gastroesophageal reflux disease, as demonstrated in several randomized controlled trials and long case series^[17–20].

Nevertheless, due to the lack of long-term effectiveness data, it is hard to recommend one type of fundoplication over another, and Rossetti-Nissen laparoscopic fundoplication still a valid and widespread procedure with a low rate of postoperative complications^[17], when performed by trained surgeons. Some technical aspects during a Nissen fundoplication are important, specially for the novice surgeon, and should be considered to avoid surgery-related complications, including the “gas-bloat” syndrome. This may include a short wrap (typically no more than 2 cm), instead of a long one, or the systematic division of the SGV to facilitate a tension-free wrapping^[21,22]. The latter is controversial and some prospective randomized trials have proven the contrary: a higher incidence of gas-bloat syndrome when the SGV are divided^[23]. In the case presented, no division of the gastric short vessels was performed, but a floppy fundoplication was achieved, and the patient referred no complaints in follow-ups. So the authors do not consider that the preservation of the gastric short vessels was a contributing factor in this particular case.

An important issue to be also remarked is the importance of identifying patients with anxiety and comfort or binge eating conducts, which could have been disregarded in the preoperative consult, and that could potentially have higher risk of complications in the long term. Anti-depressant medication and early psychiatric consult might be necessary.

COMMENTS

Case characteristics

The only symptom referred by the patient was sudden and intense pain in epigastrium.

Clinical diagnosis

Main clinical findings were upper abdominal distension, tympanic percussion and peritoneal irritation.

Differential diagnosis

Differential diagnosis was mainly based on past surgical history: acute gastric dilatation vs small bowel obstruction.

Laboratory diagnosis

Laboratory findings were congruent with an acute inflammatory process with poor splanchnic perfusion: high leukocyte count and high lactic dehydrogenase and amylase.

Imaging diagnosis

Plain abdominal X-ray is more than adequate to diagnose gastric distension, but a computed tomography-scan may be also helpful to note ischemic changes

and involvement.

Pathological diagnosis

Routine Hematoxylin-Eosin stain was performed to find out ischemic tissue injuries.

Treatment

Surgery was mandatory in this case based on clinical and analytical findings, and a total gastrectomy was performed.

Experiences and lessons

Antireflux surgery, although it might not be of extreme complexity, it is not free of severe complications and common pitfalls such as long or tightened wraps should be avoided. Also, patients with antireflux surgery and anxiety symptoms or eating disorders should be recognized and properly treated with psychiatric consult if necessary.

Peer review

The case report is useful as it serves to highlight that fundoplication can have serious complications.

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