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Case Report

What to do in case of an endoscopic image of gastric necrosis or ischaemia post-funduplication? A case report

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ABSTRACT

The association of gastric ischemia with a fundoplication is very rare and its management is not always surgical. The present paper describes the mechanism of post-fundoplication gastric ischemia that occurred in a patient diagnosed with gastroesophageal reflux disease treated with a Nissen-type gastric fundoplication. A clinical case of the Foregut Clinic of the Hospital General de Mexico (HGM) is presented. This is a 24-year-old patient undergoing a Nissen-type fundoplication who was discharged without eventualities and who presented intestinal occlusion, acute gastric dilation and gastric ischemia.

Keywords: Fundoplication, Gastric ischemia, Gastric necrosis, Gas blunt syndrome

INTRODUCTION

Ischemia and gastric necrosis are infrequently encountered pathologies, and it is unusual for them to occur following a fundoplication, although their association with acute gastric dilation has been described as part of its pathophysiology. The objective of Nissen fundoplication is to prevent gastroesophageal reflux by increasing the pressure at the esophagogastric junction (EGJ) secondary to an increase in intragastric pressure.[1] The most frequently reported complications of Nissen fundoplication are dysphagia (25%), gas blunt syndrome (20%), and heartburn sensation (10%). Gas blunt syndrome (also called post-Nissen syndrome or gastric bubble syndrome) occurs due to significant tension in the fundoplication, leading to chronic accumulation of swallowed air, causing distension in the stomach and small intestine. Symptoms include difficulty belching and vomiting, early satiety, distension, flatulence, and weight loss due to early satiety.[2] In a patient with intestinal obstruction and a history of fundoplication, acute gastric dilation may occur due to the inability to vomit, creating a vicious circle where an increase in intragastric pressure enhances lower esophageal sphincter incompetence, resulting in increased gastric pressure due to the inability to vomit[1]. Persistent elevation of intragastric pressure causes vascular compromise of the gastric wall.

CASE REPORT

A 24-year-old female, native and resident of Mexico City, a pastry chef. She denies chronic-degenerative, allergic, traumatic, or transfusion-related history. The only surgical history is lacrimal duct permeabilization in 2000. On 28 November 2022, she underwent laparoscopic Nissen floppy fundoplication for gastroesophageal reflux disease and esophageal stenosis, seemingly without complications. She was discharged 48 hours post-procedure without complaints (Table 1).

On 13 December 2022, she developed severe abdominal pain (intensity 10/10), colicky, without radiation, migration, alleviating or exacerbating factors. Nausea, inability to vomit or belch, and generalized abdominal distension were subsequently added. Consequently, she sought evaluation at the emergency department of the

General Hospital. On arrival, laboratory tests were ordered, and an abdominal X-ray was performed, leading to evaluation by the upper digestive tract clinic due to suspected intestinal obstruction.

Physical examination

Upon admission, vital signs were blood pressure 90/60, heart rate 110, respiratory rate 20, temperature 37 degrees Celsius. The patient appeared in pain, with dehydrated mucous membranes. Cardiopulmonary examination was unremarkable. The abdomen showed distension, predominantly in the upper quadrants, with scars from previous laparoscopic port incisions. Superficial palpation revealed no hyperesthesia but demonstrated hyperalgesia and pain in the upper quadrants. The abdomen was tense, with a negative rebound sign in all four quadrants. Negative findings for Murphy's, McBurney's, Lanz's, pancreatic, and ureteral points. No signs of peritoneal irritation were present. Percussion indicated tympany in the epigastrium and upper quadrants. The rectal ampulla was empty, and peristalsis was decreased in frequency and intensity.

Laboratory test

Initial laboratory tests upon admission are summarized in Table 1.

Table 1: Laboratory tests.

Date	Values
14 December 2022 03.56 H	Leukocytes: 18 cells/mm^3, neutrophils: 88.7%, lymphocytes: 5.6%, hemoglobin: 12.9 g/dl, hematocrit: 39.9%, platelets: 418 cells/mm^3, glucose: 244 mg/dl, urea: 42.1 mg/dl, creatinine: 1.17 mg/dl, sodium: 136.5 mEq/l, potassium: 3.4 mEq/l, chloride: 98 mEq/l, calcium: 10.6 mg/dl, phosphorus: 5.6 mEq/l, magnesium: 1.9 mEq/l Venous blood gas: pH: 7.26, carbon dioxide pressure (PCO_2): 33 mmHg, oxygen pressure (PO_2): 85 mmHg, bicarbonate: 21.1 mmol/l, base excess: -6 mmol/l, lactate: 5.7 mmol/l
14 December 2022 06.56 H	Leukocytes: 13.4 cells/mm^3, neutrophils: 94.7%, lymphocytes: 1.8%, hemoglobin: 13 g/dl, hematocrit: 39.2%, C-reactive protein: 3.13, glucose: 208 mg/dl, urea: 49.2 mg/dl, creatinine: 0.93 mg/dl, sodium: 137 mEq/l, potassium: 3.9 mEq/l, chloride: 99 mEq/L, calcium: 10.9 mg/dl
Post-endoscopy values	
14 December 2022 10.36 H:	Arterial blood gas: pH: 7.43, carbon dioxide pressure (PCO_2): 33 mmHg, oxygen pressure (PO_2): 85 mmHg, bicarbonate: 21.9 mmol/l, base excess: -2.4 mmol/l, lactate: 2.6 mmol/l
15 December 2023	Arterial blood gas: pH: 7.42, carbon dioxide pressure (PCO_2): 36 mmHg, oxygen pressure (PO_2): 85 mmHg, bicarbonate: 23.4 mmol/l, base excess: -1.1 mmol/l, lactate: 0.7 mmol/l

Radiology

Before any invasive procedures, an emergency anteroposterior chest and abdominal X-ray was performed (Figure 1). A nasogastric tube was identified in the esophagus, revealing an image consistent with gastric distension and no subdiaphragmatic free air.

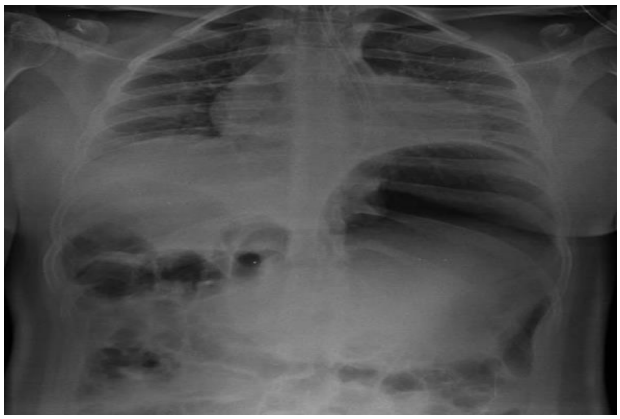

Figure 1: Chest X-ray showing gastric dilation. The passage of the nasogastric tube through the esophageal hiatus is not observed.

Initial management

Initial management in the emergency department included oxygen supplementation with nasal cannulas at 3 L/min to maintain saturation above 92%, intravenous fluid resuscitation with crystalloid solutions, transurethral catheter insertion, and unsuccessful nasogastric tube placement, as confirmed by the aforementioned X-ray.

Diagnosis integration

The clinical presentation, the aforementioned X-ray, and laboratory results led to the diagnosis of intestinal obstruction and acute gastric dilation. Endoscopic gastric decompression was considered the optimal treatment and presented as an urgent case.

Endoscopic management

Urgent panendoscopy was performed, achieving gastric decompression without complications. The procedure revealed erosive gastropathy in the cardias, discoloration consistent with vascular compromise of the gastric fundus initially diagnosed as acute gastric necrosis (Figure 2). A nasogastric tube was placed under guidance.

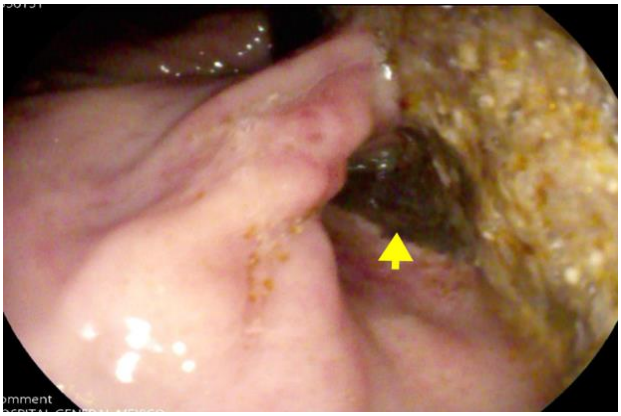

Figure 2: During gastric decompression by endoscopy, the gastric fundus is observed with a necrotic patch, possibly indicating vascular compromise (yellow arrow).

Progression in the general surgery ward

The patient showed normalization of vital signs and pain improvement. A follow-up gasometry demonstrated decreased lactate levels and increased urinary output. Regarding the apparent gastric necrosis found on endoscopy, medical management was decided upon due to clinical improvement and post-gastric decompression laboratory results. Medical management of intestinal obstruction continued with fluid and electrolyte replacement, nasogastric tube diversion, and intestinal rest. The patient remained stable, without signs of inflammatory response or acute abdomen. A follow-up endoscopy 24 hours later reported an ulcer in the gastric fundus with fibrin plaques (Figure 3). Active peristalsis was auscultated at 48 hours, and an oral diet was initiated with liquids, progressing to a soft diet with adequate tolerance throughout the day. The patient progressed appropriately without complications. She was discharged after 72 hours, asymptomatic, tolerating a normal diet and regular bowel movements.

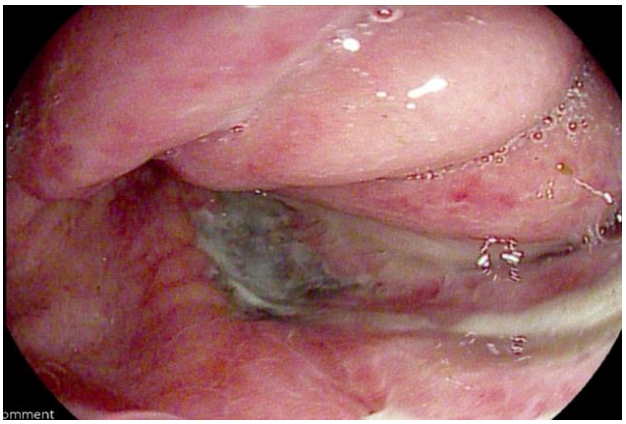

Figure 3: Control endoscopy at 48 hours after gastric decompression. An ulcer with remnants of fibrin on its surface is observed.

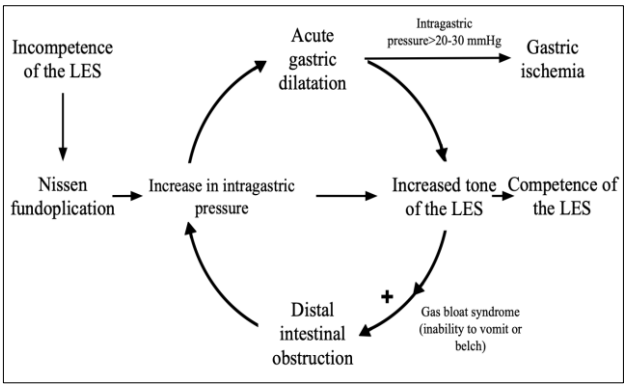

Figure 4: Pathophysiology of acute gastric dilation and acute gastric ischemia in a fundoplication. LES: lower esophageal sphincter.

DISCUSSION

Acute gastric dilation was first described in 1983, with the first reports of gastric necrosis associated with acute gastric dilation occurring in two pediatric cases of intestinal occlusion due to adhesive disease.[3] This case presents a patient with acute gastric dilation causing gastric ischemia due to the combination of gas bloat syndrome and intestinal occlusion, which is noteworthy for its pathophysiology, being a rarely studied complication of the Nissen procedure, and the patient's favorable outcome.

Gas bloat syndrome occurs in postoperative patients with excessively narrow gastric fundoplication.[4] It is characterized by abdominal discomfort, early satiety, distension, flatulence, and weight loss (secondary to satiety) due to the inability to vomit or belch, resulting from a too narrow fundoplication, increasing the risk of acute gastric dilation.[2,5,6] In this case, the symptoms of gas bloat syndrome were exacerbated by the underlying intestinal occlusion. Two weeks before the current illness, the patient underwent a floppy Nissen fundoplication, involving the division of short vessels and shortening the length of the fundoplication.[7] Although the floppy Nissen fundoplication was initially described as a procedure preventing reflux and allowing the patient to vomit or belch when necessary, subsequent cohorts reported a highly variable frequency of gas bloat syndrome post-Nissen fundoplication, ranging from 1% to 85%, with no significant difference in presentation frequency when short vessels were or were not sectioned.[8-12]

The most common presentation of acute gastric dilation in over 90% of patients is vomiting, progressive abdominal distension, and pain.[2] The frequency of acute gastric dilation, ischemia, and gastric necrosis post-fundoplication are not well determined. In our patient, acute gastric dilation presented as an early complication of the surgical procedure, which is unusual since the average time to develop a post-Nissen fundoplication complication is 1.5 years.[1,3] Cases have been reported in both adults and

children.[5] Although no reported mortality is directly attributed to gastric necrosis associated with gastric fundoplication, necrosis of any segment of the gastrointestinal tract is recognized as a precursor to gastrointestinal perforation, necessitating urgent surgical intervention due to life-threatening implications.

The most common symptoms of gas bloat syndrome are difficulty belching, vomiting, early satiety, distension, flatulence, and weight loss (secondary to satiety).[2] When ischemia occurs, it most commonly presents as excruciating pain disproportionate to physical examination findings, given that ischemia initiates in the mucosa and progresses to the serosa.[1,6] The patient in our case did not exhibit the typical manifestations of gastric ischemia, a fact considered when deciding the management.

In a patient post-fundoplication, there is increased competence of the lower esophageal sphincter. The increased intragastric pressure due to distal obstruction increases the pressure in the fundoplication, preventing emesis and creating a closed-loop obstruction that exacerbates distension, leading to acute gastric dilation (Figure 4).[3,5] For acute gastric dilation to occur, the presence of distal occlusion is a sine qua non condition. The incidence of adhesions associated with Nissen fundoplication ranges from 3.4% to 6.2%.[5] Our patient presented with intestinal occlusion 15 days after surgery due to adhesive disease, and it is noted that intra-abdominal adhesions typically form within 10 days post-surgery.[14]

Gastric necrosis after fundoplication can result from short vessel section or acute gastric dilation.[15] In this case, during the decompressive endoscopy, an area of necrotic appearance in the gastric fundus was reported. This finding was cautiously interpreted by the surgical team, considering the absence of peritoneal irritation and the absence of intestinal compromise in laboratory results. It was determined to be an ischemic lesion, and a follow-up endoscopy was requested in 24 hours, revealing an ulcer with a fibrin base. The patient's favorable evolution is supported by the gastric arterial system, which has abundant collateral flow and intramural anastomoses that protect the stomach from ischemia, except in extreme situations. Reports indicate that ligating 4 major arteries and 80% of small vessels does not lead to gastric ischemia.[2,6] The primary factor in gastric necrosis post-fundoplication is vascular insufficiency secondary to increased intragastric pressure exceeding intramural venous flow, so when intragastric pressure exceeds 20-30 mmHg, venous congestion coincides with mucosal arterial insufficiency, inducing ischemia and eventually transmural necrosis and rupture.[2,3,5,6] In our patient, in addition to endoscopic decompression, a nasogastric tube with a hydrophilic guide was placed. A gasometry control was conducted an hour after the procedure, reporting a decrease in lactate levels from 5.7 to 2.6 mmol/l.

The most severe complication of acute gastric dilation is gastric necrosis (mortality 73%).[3] Its mortality is 50-60% with surgical management and 100% without it, making the identification of a patient eligible for surgical management the most crucial point in assessing suspected gastric necrosis. Due to the high morbidity of the procedure and the patient's adequate evolution in this case, conservative management was deemed appropriate for the finding of an ischemic ulcer in the gastric fundus.

According to current guidelines, the management of gastric ischemia is based on the following guidelines: fluid resuscitation to improve visceral perfusion, correction of electrolyte imbalances; administration of broad-spectrum antibiotics, as there is a risk of loss of the mucosal barrier facilitating bacterial translocation; correction of the non-occlusive cause of ischemia, i.e., gastric decompression; if the patient is hemodynamically unstable with signs of peritonitis, laparotomy is indicated to perform damage control; and if the patient is stable without signs of peritonitis, diagnostic laparoscopy is suggested or monitoring for the subsequent appearance of peritonitis, which would warrant surgical exploration.[16]

Our case followed the aforementioned guidelines as soon as it arrived in the emergency department, and the endoscopic report of gastric necrosis was evaluated according to the patient's clinical and laboratory findings. In the absence of peritonitis and hemodynamic stability, medical management was decided. Both intestinal occlusion and gastric ischemia have very similar management pillars, so the vicious cycle of gastric ischemia associated with gas bloat syndrome was broken after a few hours.

CONCLUSION

Acute gastric dilation is a complication of gastric fundoplication that occurs in the context of distal intestinal occlusion. Acute gastric dilation is managed conservatively with gastric decompression (using a nasogastric tube or endoscopy). Gastric ischemia and necrosis post-fundoplication are not complications per se of the surgical procedure; gas bloat syndrome and distal occlusion are sine qua non conditions for their presentation. A case of gastric ischemia associated with acute gastric dilation with successful conservative management is reported.

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REFERENCES

1. Barker JA, Burnett H, Carlson GL. Gastric necrosis complicating acute gastric dilatation after Nissen fundoplication BMJ Case Rep. 2011;1-4.
2. Patuto N, Acklin Y, Oertli D, Langer I. Gastric necrosis complicating lately a Nissen fundoplication. Report of a case. Langenbecks Arch Surg. 2008;393(1):45-7.
3. Cloyd JM, Moskovitz A, Stevenson J. Acute gastric dilation and necrosis as a late complication following laparoscopic nissen fundoplication. Dig Dis Sci. 2015;60(1):32-4.
4. Hazan TB, Gamarra FN, Stawick L, Maas LC. Nissen fundoplication and gastrointestinal-related complications: a guide for the primary care physician, South Med J. 2009;102(10):1041-5.
5. Glick PL, Harrison MR, Adzick NS, Webb HW, DeLorimier AA. Gastric infarction secondary to small bowel obstruction: a preventable complication after Nissen fundoplication. J Pediatr Surg. 1987;22(10):941-3.
6. Merrill JR. Gastric necrosis and perforation after fundoplication. South Med J. 1981;74(12):1543-5.
7. Ipek T, Eyuboglu E, Ozben V. Partial splenic infarction as a complication of laparoscopic floppy Nissen fundoplication. J Laparoendosc Adv Surg Tech A. 2010;20(4):333-7.
8. Donahue P, Salmelson S, Nyhus L, Bombeck C. The floppy Nissen fundoplication. Effective long-term control of pathologic reflux. Arch Surg. 1985;120(6):663-8.
9. Salinas J, Georgiev T, González-Sánchez JA, López-Ruiz E, Rodríguez-Montes JA. Gastric necrosis: a late complication of Nissen fundoplication. World J Gastrointest Surg. 2014;6(9):183-6.
10. Tian Z, Wang B, Shan C, Zhang W, Jiang D, Qiu M. A meta-analysis of randomized controlled trials to compare long-term outcomes of nissen and toupet fundoplication for gastroesophageal reflux disease. PLoS One. 2015;10(6):e0127627.
11. Nikolic M, Schwameis K, Semmler G, Asari R, Semmler L, Steindl A. Persistent dysphagia is a rare problem after laparoscopic Nissen fundoplication. Surg Endosc. 2019;33(4):1196-205.
12. Khatri K, Sajid MS, Brodrick R, Baig MK, Sayegh M, Singh KK. Laparoscopic Nissen fundoplication with or without short gastric vessel division. Surg Endosc. 2012;26(4):970-8.
13. Bass KD, Meagher DP, G M Haase. Gastric necrosis after fundoplication: A novel approach for esophageal preservation. J Pediatr Surg. 1998;33(11):1720-2.
14. Latifi R. Surgery of complex abdominal wall defects. Springer. Suiza. 2017.
15. Kennedy T, Magill P, Johnston GW, Parks TG. Proximal gastric vagotomy, fundoplication, and lesser-curve necrosis. Br Med J. 1979;1(6176):1455-6.
16. Bala M, Catena F, Kashuk J, Simone B, Gomes C, Weber D. Acute mesenteric ischemia: updated guidelines of the World Society of Emergency Surgery. World J Emerg Surg. 2022;17(1):54.

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