ACG CASE REPORTS JOURNAL

CASE REPORT | STOMACH

Post-Fundoplication Dumping Syndrome: A Frequent "Rare" Complication

Rahul Kataria, MD[1], Sandar Linn, MD[2], Zubair Malik, MD[1], Abbas E. Abbas, MD[3], Henry Parkman, MD[1], and Ron Schey, MD[1]

[1]*Section of Gastroenterology, Department of Internal Medicine, Temple University Hospital, Philadelphia, PA*
[2]*Section of Gastroenterology, Department of Internal Medicine, Brooklyn Hospital Center, Brooklyn, NY*
[3]*Department of Thoracic Surgery and Medicine, Temple University Hospital, Philadelphia, PA*

ABSTRACT

Gastroparesis, caused by delayed emptying of the stomach, has been shown to be associated with Nissen fundoplication. However, symptomatic rapid emptying of the stomach is rare after Nissen fundoplication, and its treatment is often challenging. We report 2 patients with dumping-like syndrome post-fundoplication with marked improvement of symptoms after dietary management and medical treatment.

INTRODUCTION

Nissen fundoplication is an anti-reflux surgery originally described by Nissen in 1956 to treat patients with refractory gastroesophageal reflux disease (GERD).[1] Some studies have demonstrated impaired relaxation of the proximal stomach in response to meal ingestion after this surgical procedure.[2] Postsurgical gastroparesis may be the overt appearance of an unrecognized preoperative disorder or may suggest injury to vagal nerves that innervate the antrum.[3] Nissen fundoplication is still frequently used to treat refractory symptoms for GERD, and it was the most common cause of postsurgical gastroparesis in the audit performed by the National Institute of Diabetes and Digestive and Kidney Diseases Gastroparesis Consortium.[4] Dumping syndrome, caused by rapid emptying of the stomach, is rare after Nissen fundoplication. Thus far, data is scarce on the subject with a limited number of cases reported.[5-10]

CASE REPORT

Case 1: A 44-year-old woman was referred to the gastroenterology practice for evaluation of long-standing heartburn refractory to proton pump inhibitor (PPI) therapy. Her 24-hour multichannel intraluminal impedance-pH study on dexlansoprazole 60 mg daily revealed increased esophageal acid exposure (5.7% total time spent with esophageal pH <4) and a total of 15 impedance reflux episodes (10 acidic, 5 weakly acidic). High-resolution esophageal manometry with impedance (HREMI) showed normal esophageal function with 100% bolus clearance. HREMI revealed a sliding hiatal hernia of approximately 2 cm, which was confirmed on endoscopy. Biopsies were negative for eosinophilic esophagitis. Due to her persistent symptoms and high daily dosage of antacids, she underwent robotic-assisted laparoscopic hiatal hernia repair and Nissen fundoplication, during which the anterior and posterior vagal nerves were identified and preserved. Her postprocedure recovery was unremarkable, and she was discharged on the second day after the procedure. She was readmitted on 7 days later due to dehydration with symptoms of nausea, vomiting, and coughing. During this admission, she was hydrated and her symptoms improved. However, she gradually developed postprandial weakness and was eventually admitted 5 months postsurgery with diaphoresis, dyspnea, and palpitations occurring 2 hours after meals. Upper

ACG Case Rep J 2018;5:e1. doi:10.14309/crj.2018.1. Published online: January 3, 2018.

Correspondence: Ron Schey, Section of Gastroenterology, Department of Internal Medicine, Parkinson Pavilion, 8th Floor, Temple University School of Medicine, 3401 N. Broad St, Philadelphia, PA, 19140 (ron.schey@tuhs.temple.edu).

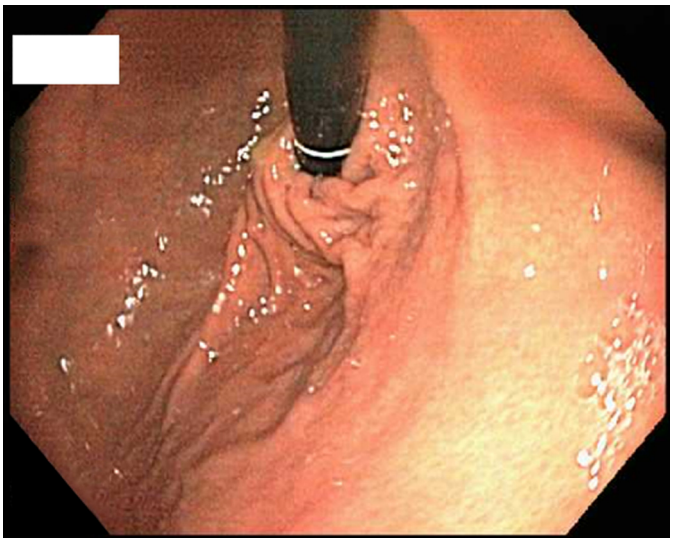

Figure 1. Upper endoscopy revealing intact fundoplication.

endoscopy demonstrated an intact wrap (Figure 1). A barium swallow revealed a normal post-fundoplication appearance. Interestingly, gastric emptying scintigraphy revealed rapid gastric emptying (Figure 2) with only 7% solid meal left in the stomach after 1 hour, 3% after 2 hours, and 1% after 4 hours (<30% after 1 hour is considered abnormal).[11] Small bowel transit was normal, with 60–70% accumulation of the radiotracer bolus in the ileocecal region at 6 hours. As a result of her symptoms and objective rapid gastric emptying, the patient was diagnosed with dumping syndrome. Dietary modification with a low-carbohydrate diet and separating solids from liquid ingestion was instituted. The patient reported improvement of symptoms with these dietary modifications during her next follow up clinic visit 2 months later.

Case 2: A 70-year-old man presented with a 10-year history of heartburn, acid brash, and hoarseness. He was placed on oral pantoprazole 40 mg twice daily and oral ranitidine 150 mg at bedtime with some improvement of symptoms. In addition, he had been experiencing episodes of chest pain for 1 year that were attributed to acid reflux. His barium esophagram demonstrated esophageal dysmotility and a moderate hiatal hernia with evidence of gastroesophageal reflux. This was verified with a 24-hour multichannel intraluminal impedance-pH study performed while the patient was taking oral pantoprazole 40 mg twice daily and oral ranitidine 150 mg at bedtime, demonstrating increased esophageal acid and non-acid exposure (16 acidic episodes, 132 weakly acidic) and a Johnson-DeMeester score of 35.6 (normal <14.7). HREMI revealed a decreased lower esophageal sphincter pressure of 7.5 mm Hg (normal 13–43 mm Hg) with evidence of a hiatal hernia. Esophagogastroduodenoscopy showed a 4-cm hiatal hernia and esophagitis without evidence of Barrett's esophagus on distal esophageal biopsy. The patient underwent a robotic-assisted hiatal hernia repair with Toupet fundoplication without major complications. During his second postoperative visit, approximately 4 weeks after his surgery, he noted 3 episodes of sudden-onset lightheadedness, hunger, tachycardia, perspiration, and diarrhea shortly after eating. Due to his history of diabetes mellitus type 2, he was able to check his blood glucose and found himself to be hypoglycemic after these episodes. He was given recommendations to eat small, frequent meals with complex carbohydrates (e.g., oatmeal, brown rice) and to avoid sugar and caffeine. Stool studies were also negative. Despite keeping a strict log of his diet, he continued to experience his previously described episodes. He was prescribed oral dicyclomine 20 mg 3 times daily with mild improvement of his diarrhea; however, he continued to have similar postprandial episodes. After a few weeks, his diarrhea returned, and he discontinued the dicyclomine. He was started on oral acarbose 25 mg 3 times daily before meals with significant improvement in his symptoms. At 1 year postsurgery, his symptoms remain controlled.

DISCUSSION

Dumping syndrome is defined by a constellation of symptoms due to rapid emptying of nutrients from the stomach into the small intestine, resulting in the release of vasoactive agents and glucose-modulating hormones. The incidence of rapid gastric emptying is clinically significant in patients with a spectrum of symptoms such as nausea, vomiting, abdominal pain, diarrhea, weakness, faintness, sweating, and hypoglycemia after a meal. The etiology of dumping syndrome is poorly understood and is multifactorial.[12] It is typically seen after surgery for peptic ulcer disease, gastrectomy, or gastric bypass due to the reduced reservoir of the stomach or disruption of the pyloric sphincter.[3] In most cases, these symptoms decrease 1–1.5 years after surgery, which is attributed to gut adaptation and patient education.[13]

Dumping syndrome is a rare presentation after Nissen fundoplication and is predominantly reported in the pediatric population. Many of these reports suggest that there may be an association with vagal nerve damage, although the exact etiology is unclear.[14] We propose that the loss of fundal accommodation after the fundoplication leads to rapid transit of food through the stomach, resulting in accelerated gastric emptying. This could be caused by the fundoplication mechanically preventing accommodation or injury to the vagal nerve to the fundus governing accommodation. The incidence of dumping syndrome in patients with Nissen versus Toupet fundoplication has not been reported, although we believe it would be more commonly seen with Nissen fundoplication due to the tighter lower esophageal sphincter wrap. This could be more extensively studied as a prospective study.

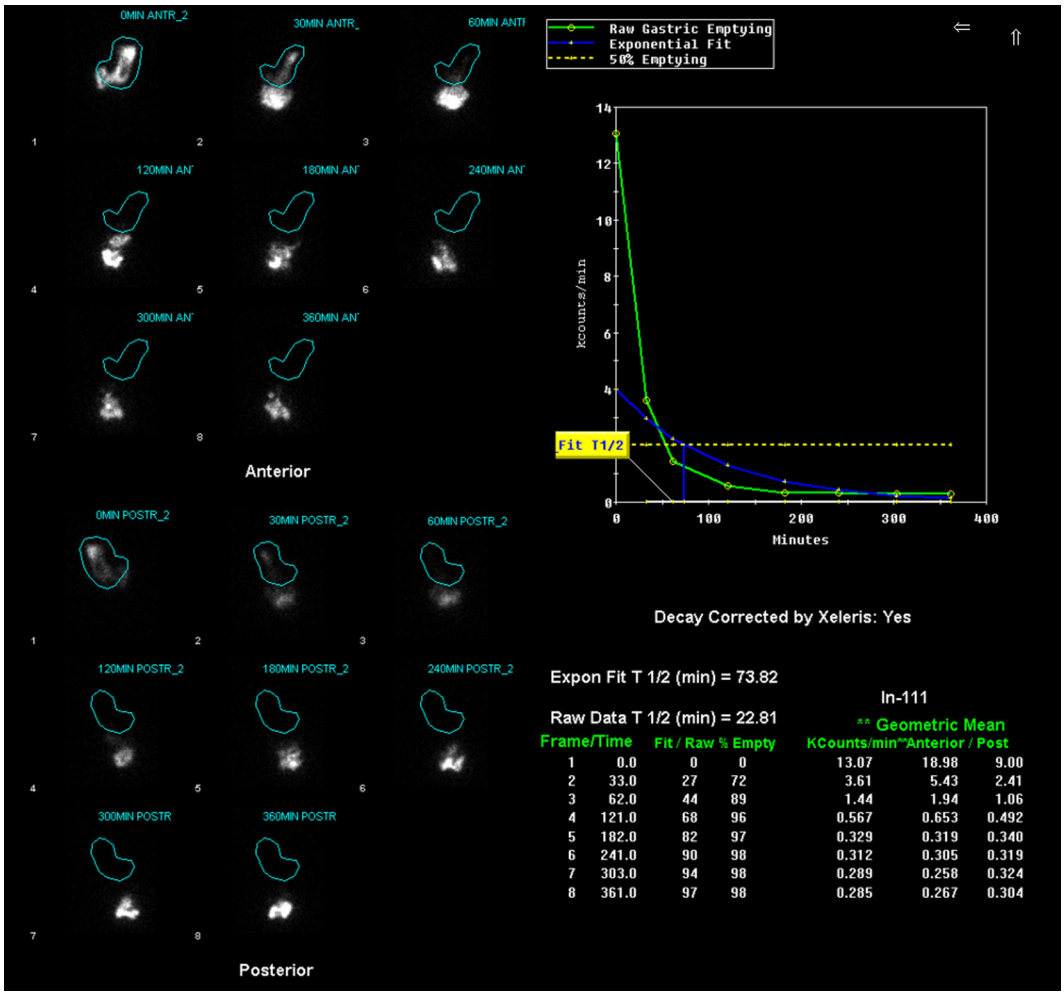

Figure 2. Gastric emptying scintigraphy showing rapid emptying with 7% of radiolabeled solid meal remaining in the stomach after 1 hour.

Dumping syndrome is often classified into 2 types: early and late. Both types are caused by a large amount of gastric contents emptying into the duodenum, resulting in vasomotor symptoms from changes in blood pressure, pulse, and glucose levels, as well as gastrointestinal symptoms (e.g., abdominal cramping, nausea, diarrhea). Early dumping syndrome usually presents within 45 minutes of a meal and results in vasomotor symptoms such as tachycardia, sweating, and flushing. This is thought to be caused by a shift in intravascular fluid to the small bowel due to the osmotic changes caused by a large amount of gastric contents entering the duodenum. Late dumping syndrome presents 2–4 hours after a meal and results may include abdominal cramping, diarrhea, and episodes of hypoglycemia.[3] These symptoms may be a result of an insulin spike caused by the rapid delivery of sugar into the small bowel.[15] Gastric emptying scintigraphy and 13C-octanoic acid breath tests are also helpful in diagnosing dumping syndrome.[3,5] The gastric emptying test in Case 1 revealed rapid gastric emptying, where rapid gastric emptying was defined as <30% retention, i.e., >70% emptying at 1 hour.

An oral glucose challenge test can also be used to diagnose dumping syndrome. For this, the patient ingests 50 g glucose after an overnight fast. Pulse and blood pressure are measured before, during, and after the ingestion. If the pulse increases by 10 beats per minute or more, the test is positive. Additionally, hematocrit can also be measured at the same time intervals. An increase of 3% in hematocrit in the first 30 minutes suggests early dumping syndrome. Hypoglycemia 2–3 hours after ingestion suggests late dumping syndrome, as in Case 2.[16]

In the majority of cases, symptoms can be resolved with dietary management, particularly with a reduction of carbohydrate intake and supplementation of dietary fibers (bran, methycellulose) with meals.[3] Only 3–5% of patients require

medical therapy such as octreotide, a somatostatin analog.[3] Another medical treatment is acarbose, an α-glycosidase hydrolase inhibitor that slows carbohydrate digestion in the small intestine. This slows postprandial hyperglycemia and subsequent hypoglycemia.[16] Surgical management is occasionally performed for dumping syndrome, primarily for dumping syndrome after gastric surgery. For instance, stoma revision, pyloric reconstruction, or Roux-en-Y conversion can be considered after failing a 1-year trial of medical and dietary management.[17]

Although the exact mechanism of dumping syndrome after fundoplication is unclear, we believe the loss of fundal accommodation plays a role in rapid gastric emptying. It is important to recognize that most symptoms improve from both gut adaptation and patient education. During this transition period, it is recommended to stabilize symptoms in order to avoid additional surgical procedures.

DISCLOSURES

Author contributions: R. Kataria wrote the manuscript. S. Linn wrote and edited the manuscript. Z. Malik, AE Abbas, H. Parkman, and R. Schey edited the manuscript. R. Schey is the article guarantor.

Financial disclosure: None to report.

Informed consent was obtained for this case report.

Received June 28, 2017; Accepted November 8, 2017

REFERENCES

1. Nissen R. A simple operation for control of reflux esophagitis. *Schweiz Med Wochenschr*. 1956;86:590–2.
2. Vu MK, Ringers J, Arndt JW, Lamers CB, Masclee AA. Prospective study of the effect of laparoscopic hemifundoplication on motor and sensory function of the proximal stomach. *Br J Surg*. 2000;87(3):338–43.
3. Ukleja A. Dumping syndrome: Pathophysiology and treatment. *Nutr Clin Pract*. 2005;20(5):517–25.
4. Sarosiek I, Yates KP, Abell TL, et al. Interpreting symptoms suggesting gastroparesis in patients after gastric and esophageal surgeries. *Gastroenterology*. 2011;140(5):S-813.
5. Mizrahi M, Almogy G, Adar T, Lysy J. Dumping syndrome following Nissen fundoplication in an adult patient diagnosed by continuous online 13C/12C monitoring of 13 C-octanoic acid breath test: A case report. *BMC Gastroenterology*. 2011;11(1):98.
6. Kreckler S, Dowson H, Willson P. Dumping syndrome as a complication of laparoscopic Nissen fundoplication in an adult. *JSLS*. 2006;10(1):94–96.
7. Bernard B, Kline GA. Hypoglycaemia following upper gastrointestinal surgery: Case report and review of the literature. *BMC Gastroenterology*. 2010;10:77.
8. Miholic J, Hoffmann M, Holst JJ, et al. Gastric emptying of glucose solution and associated plasma concentrations of GLP-1, GIP, and PYY before and after fundoplication. *Surg Endosc*. 2007;21(2):309–14.
9. Lindeboom MY, Ringers J, van Rijn PJ, Neijenhuis P, Stokkel MP, Masclee AA. Gastric emptying and vagus nerve function after laparoscopic partial fundoplication. *Ann Surg*. 2004;240(5):785–90.
10. Roldán Baños S, Ruiz de Angulo Martín D, Munítiz Ruiz V, Martínez de Haro L, Parrilla Paricio P. Dumping syndrome with severe hipoglycemia after Nissen fundoplication in adults. Case report and literature review. *Endocrinol Nutr*. 2014;61(10):550–1.
11. Abell TL, Camilleri M, Donohoe K, et al. Consensus recommendations for gastric emptying scintigraphy: A joint report of the American Neurogastroenterology and Motility Society and the Society of Nuclear Medicine. *Am J Gastroenterol*. 2008;103(3):753–63.
12. Hejazi RA, Patil H, McCallum RW. Dumping syndrome: Establishing criteria for diagnosis and identifying new etiologies. *Dig Dis Sci*. 2010;55 (1):117.
13. Ukleja A. Dumping syndrome. *Practical Gastroenterology*. 2006;30 (2):32.
14. Bufler P, Ehringhaus C, Koletzko S. Dumping syndrome: A common problem following Nissen fundoplication in young children. *Pediatr Surg Int*. 2001;17(5):351–5.
15. Hasler W. Physiology of gastric motility and gastric emptying. In: Yamada T, Alpers DH, Laine L, Owyang C, Powell DW,eds. *Textbook of Gastroenterology*. 4th ed. Philadelphia, PA:Lippincott Williams & Wilkins; 2003.
16. Beek A, Emous M, Laville M, Tack J. Dumping syndrome after esophageal, gastric or bariatric surgery: Pathophysiology, diagnosis, and management. *Obes Rev*. 2017;18(1):68–85.
17. Berg P, McCallum R. Dumping syndrome: A review of the current concepts of pathophysiology, diagnosis, and treatment. *Dig Dis Sci*. 2016;61 (1):11–8.