

CASE REPORT | STOMACH

Gastric Electrical Stimulators Causing Erosion Through the Colonic Wall

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ABSTRACT

Gastroparesis is a chronic motor disorder of the stomach characterized by the demonstration of delayed gastric emptying without obstruction and a grouping of symptoms including nausea, vomiting, early satiety, postprandial fullness, bloating and abdominal pain. When conservative medical management is not effective, gastric electrical stimulation is an effective alternative. Gastric electrical stimulation, in general, has had a low complication rate as of yet, with the most common being infection of the implanted device. We present a complication in which the gastric electrical stimulator electrodes eroded through the colonic wall.

INTRODUCTION

Gastroparesis is a chronic motor disorder of the stomach diagnosed through demonstration of delayed gastric emptying without obstruction and symptoms such as nausea, vomiting, early satiety, bloating, and upper abdominal pain.¹ The main etiologies are diabetic, postsurgical, and idiopathic.² Gastroparesis is often difficult to treat, involving a multimodal approach including lifestyle changes, pharmacotherapy, and screening for comorbid conditions or medication that could contribute to symptoms.³ In refractory patients, the development of gastric electrical stimulators has brought success to many with gastroparesis.⁴ The gastric electrical stimulator works by activating the vagal afferent pathways of the stomach. This affects central control mechanisms for nausea and vomiting, stimulates appetite and reduces satiety, and may influence the myoelectrical effects of the stomach. The 2 electrodes, 1 cm apart, are approximately 10 cm from the pylorus on the greater curvature of the stomach and connected by leads to the neurostimulator which is placed subcutaneously in the abdominal wall. The most commonly reported complication is an infection at the pulse generator site. Patients with diabetes, systemic infections, or trauma/injuries are at the highest risk.⁷

CASE REPORT

In 2008, a 59-year-old man had nonhealing gastric ulcers and underwent surgical intervention with a Billroth II procedure, followed by a Roux-en-Y and a vagotomy. Shortly thereafter, his symptoms of gastroparesis started. After placement of his gastric electrical stimulator in July 2010, he has required up to 4 revisions, most recently in August 2018, 1 month before presentation, for battery replacement. Multiple hardware infections with device exchanges have complicated his course, including the most recent infection in 2017, resulting in the total replacement of the gastric electrical stimulator and placement of tobramycin Stimulan beads.

During the patient's hospitalization, he presented with left-sided intermittent abdominal pain for 1 week, along with erythema around the battery site with purulent drainage and associated nausea, vomiting, and diarrhea. An abdominal computed tomography scan showed gastric stimulator leads extending into the transverse colon and a small amount of fluid or soft tissue thickening around the stimulator power pack. This prompted a colonoscopy, which demonstrated 2 wires from the gastric stimulator piercing the distal

ACG Case Rep J 2020;7:e00313. doi:10.14309/crj.00000000000313. Published online: March 2, 2020 Correspondence: Thomas Abell, MD (Thomas.abell@louisville.edu). transverse colon approximately 60 cm from the anus (Figure 1). There was also a disk-like structure attached to the distal part of the wire. After 3 days' treatment with daptomycin and zosyn, the implanted gastric stimulator was surgically removed, and he recovered without complications. Chronic suppressive antibiotics were discontinued.

DISCUSSION

Management of gastroparesis involves 4 modes of treatment: nutrition, glycemic control in diabetics, pharmacologic, and invasive treatment (ie, GES and surgery). Dietary modifications are small meals that are low in fat and fiber. When dietary changes are ineffective, prokinetics are indicated, including metoclopramide, macrolide antibiotics, and domperidone, supplemented with symptomatic treatment for nausea and pain. In refractory patients, GES is considered as a next step. There is no guideline for the appropriate time to consider GES, but patients should have failed noninvasive methods.^{8,9} Efficacy has been variable: In one cohort study of 151 patients, 75% had symptom improvement. A meta-analysis of 13 studies, 12 of which were uncontrolled, showed overall improvement with GES. Among the 5 studies reporting on gastric emptying, significant improvement at 2 and 4 hours were 23% and 12.6%, respectively. Seventy-eight percent of patients requiring parenteral and enteral feeding was no longer dependent. The total symptom severity score showed significant, although variable, improvement.¹⁰ In one randomized, controlled study of 55 patients, weekly vomiting decreased 67% at 1 year.¹¹

Few complications have been reported but will occur more frequently because gastric stimulators become more common. Long-term studies of GES show a complication rate of 7%–10%. The main complication is an infection of the subcutaneous pocket in the abdominal wall where the stimulator resides. Less common is the erosion of the abdominal wall by the stimulator, stimulation of abdominal rectus muscles, penetration of the leads through gastric mucosa, tangling of wires in the generator pocket and formation of adhesions, and development of volvulus around the wires.^{12,13,15} Per a meta-analysis, the infection rate is about 1%–2% and gastric body perforation rate 0.8%.¹⁰ In considering GES, risks and benefits should be weighed depending on a patient's preference and current quality of life.

The first sign of erosion is an infection of the subcutaneous port or failure of the device's intended purpose. It is thought that inflammation or erosion of gastric mucosa interferes with electrode impedance.¹⁴ This can occur anytime but is most likely in the immediate postoperative period. In our patient, it occurred about 7 weeks after his most recent stimulator replacement. He presented with worsening gastroparesis symptoms, consistent with 2 other reported presentations of gastric electrical stimulator erosion, which penetrated the gastric mucosa. Interestingly, these 2 cases had no changes in gastric electrical stimulator settings on interrogation.¹⁵ Previous or current infection is a major risk factor because both cases of gastric erosion presented in Liu et al had hardware infections as well.¹⁶ Our patient had multiple device exchanges related to pocket

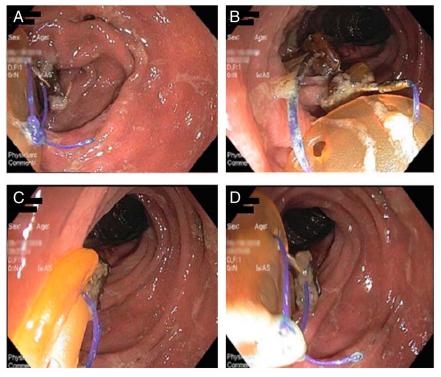


Figure 1. (A–D) Endoscopic images from colonoscopy showing 2 wires from gastric stimulator seen piercing the distal transverse colon about 60 cm from the anus, with a disk-like structure attached to the distal part of the wire.

infections. Revisions, as well as multiple infections, likely contributed to the weakening of the tissue surrounding the device and his risk for the perforation that occurred.

If there are signs of infection or a decrease in efficacy, a workup should be started to rule-out electrode erosion, beginning with an abdominal x-ray or computed tomography scan. The gastric stimulator should be tested with device interrogation, followed by endoscopic evaluation. Electrodes should be removed and replaced laparoscopically.14 In our patient, the stimulator was not replaced. However, it appears to be safe to replace the gastric electrical stimulator immediately after repair if there are no signs of infection.¹² In a longitudinal study of 55 patients with gastric electrical stimulator 11 were removed because of infection or technical problems. Ten were successfully replaced after proper treatment of infection if applicable.¹⁷ In conclusion, erosion through the gastric mucosa and other parts of the bowel is a rare but important complication of GES. It should be suspected in patients with signs of infection or ineffective hardware. Diagnosis is through imaging and endoscopy, and treatment is surgical.

DISCLOSURES

Author contributions: All authors contributed equally to this manuscript. T. Abell is article guarantor.

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