Simultaneous Fundoplication and Gastric Stimulation in a Lung Transplant Recipient With Gastroparesis and Reflux

Lori A. Filichia, BS, Maher A. Baz, MD, Juan C. Cendan, MD

ABSTRACT

Background: Gastroparesis following lung transplantation can complicate medical management leading to malnutrition, weight loss, and erratic absorption of immunosuppressive medications, which are all important factors in the success of grafts. Gastric electrical stimulation has been shown to reduce the frequency of nausea and vomiting and lead to weight gain in patients with gastroparesis refractory to standard medical treatment; however, it has not yet been reported as being used for the treatment of gastroparesis in lung transplant recipients.

Methods: We report the case of a female bilateral lung transplant recipient suffering from severe gastric reflux and severe gastroparesis, who was successfully treated with simultaneous creation of a laparoscopic Nissen fundoplication and placement of a gastric stimulator.

Results: The patient noted an immediate and sustained decrease in her symptoms of nausea and vomiting, and an increased appetite, and less variability in the serum levels of her immunosuppressive medication.

Conclusion: Lung transplant recipients with severe gastroparesis and reflux may benefit from Nissen fundoplication and gastric electrical stimulation.

Key Words: Gastroparesis, Lung transplant, Reflux, Gastric stimulation.

INTRODUCTION

Gastroparesis, or delayed gastric emptying, is a recognized complication following lung transplantation, with incidences reported between 24% and 83%. Gastroparesis can complicate medical management in these patients, leading to complications like malnutrition, weight loss, electrolyte disturbances, aspiration pneumonia, and variable absorption of their immunosuppressive medications, which may place them at increased risk of rejection. Frequent nausea, vomiting, and gastroesophageal reflux can also lead to aspiration pneumonitis. Following lung transplant, this condition may predispose the patient to obliterator bronchiolitis, a form of chronic lung transplant rejection. The inability to take scheduled medications and the variability in absorption of these medications can also be challenging in these sensitive patients, and can threaten the well being of the allograft. Gastroparesis following organ transplantation, specifically lung and combined heart-lung transplantation is a well-recognized phenomenon. The pathogenesis of gastroparesis in this group of patients is unknown; however, it is thought to be multifactorial and may involve vagal nerve injury or dysfunction or viral illness secondary to immune suppression, which can cause damage to the myenteric nervous system. The question of whether the cause is related to the toxic effects of immunosuppressive medications, specifically cyclosporine, has also been raised but is unlikely considering gastroparesis is uncommon following both renal and liver transplantation.

Gastric electrical stimulation (GES) has been shown in both controlled and uncontrolled studies to reduce the frequency of nausea and vomiting and lead to weight gain in patients with gastroparesis refractory to standard medical treatment; however, little evidence supports the use of gastric stimulation in lung transplant recipients. Accordingly, patients following lung transplantation can also experience significant gastric reflux, which can pose the same risks of aspiration pneumonia and threaten the graft. This patient was experiencing severe symptoms of both gastric reflux and gastroparesis, and this combination of simultaneous treatment with a Nissen fundoplication and implantation of a gastric stimulator has not been reported in the medical literature.
CASE REPORT

A 55-year-old female was referred to the GI surgery clinic by her pulmonologist in February 2007, five months after a successful bilateral lung transplant for end-stage pulmonary fibrosis. Her maintenance immunosuppressive regimen consisted of Prednisone 10 mg PO BID, mycophenolate mofetil 250 mg PO TID, and tacrolimus 1mg PO BID. The patient complained of severe heartburn with regurgitation and symptoms of intractable nausea and vomiting, early satiety, and loss of appetite, which began shortly after her lung transplantation and had been progressively worsening. The patient’s weight had dropped to 89 lb (BMI = 21.6) despite oral nutritional supplements. The patient had no history of gastroesophageal reflux disease (GERD) and had been on omeprazole 20 mg PO daily, lansoprazole 30 mg PO BID, promethazine 12.5 mg q 4 hours, and metoclopramide 10 mg PO daily for greater than 4 months.

Preoperative evaluation consisted of an upper gastrointestinal series that revealed extensive episodes of reflux to the level of the clavicles. An intravesophageal pH monitoring study confirmed acid (pH < 4) reflux with 444 episodes recorded in 12 hours (normal < 50). Esophageal manometry showed low-normal LES pressure of 14 mm Hg (10 to 40 mm Hg normal) and no evidence of achalasia. Her gastric emptying study revealed delayed gastric emptying with a half-time of greater than 5 hours, which is greater than 333% of normal. The patient had failed medical therapy, as her symptoms continued to worsen despite being on omeprazole 20 mg PO daily, lansoprazole 30 mg PO BID, promethazine 1.25 mg q 4 hours, and metoclopramide 10 mg PO daily for greater than 4 months.

After multiple discussions with the pulmonologist, anesthesiology team, and the patient, it was determined that simultaneous operations consisting of laparoscopic implantation of the Enterra gastric pacemaker and laparoscopic Nissen fundoplication would be performed as therapy for this patient’s gastroparesis and acid reflux. Although the patient’s LES was in the normal range, she was experiencing significant reflux and had failed maximum medical therapy with the medications listed above and had already experienced one episode of lung rejection complicated by aspiration. The possibility that all symptoms were related to the gastroparesis alone was discussed; however, gastric emptying does not improve following GES and, given such frequent and large-volume reflux, the team decided that GES without antireflux protection would be insufficient as a solitary therapy in this individual.

The patient underwent treatment with a laparoscopic Nissen fundoplication and simultaneous placement of an Enterra (Model 3116 neurostimulator, Model 4351 leads, Medtronic, MN) gastric stimulator. Prior to initiating the abdominal component of the surgery, endoscopy was performed which showed posterior erosion of the lower to middle one third of the esophagus, and a moderate-sized bezoar was seen in her stomach. The Nissen fundoplication was performed first, followed by implantation of the Enterra gastric pacemaker using the standard technique. The patient’s hiatus was without herniation, and a single stitch closed the hiatus posteriorly. The fundoplication was done over a 50 French bougie with three 0 silk interrupted sutures spanning 2 cm top-to-bottom. The 2 highest short gastric vessels were transected, and the surfaces of the fundoplication were without tension. For the gastric pacemaker portion of the procedure, the 2 electrodes were placed 1 cm apart, 9 cm to 10 cm proximal to the pylorus along the greater curvature of the stomach’s inferior surface. Simultaneous EGD was performed to exclude perforation.

The patient’s perioperative course was uncomplicated. The generator was programmed with the common initial settings of 3 Volts, with a 12 Hz cycle (5 seconds off and 0.1 seconds on). Initial follow-up at 3 weeks to 4 weeks revealed early improvements in symptoms. Postoperative studies with GES have shown unpredictable improvements in gastric emptying; therefore, postoperative gastric emptying studies are not routinely performed in these patients and was not performed in this patient. This patient had no postoperative complications. She was then contacted by telephone for follow-up 5 months later, and a survey assessing the severity of her symptoms both prior to and after the surgery was administered. The patient reported that she first noticed improvements in her symptoms 3 weeks to 4 weeks after the surgery was performed. She had marked improvements in her symptoms of nausea, vomiting, and retching, and reported a significant improvement in her appetite. She denied any symptoms of heartburn and no longer required the use of lansoprazole or omeprazole. The only antiemetic she continued to take was metoclopramide 12.5 mg one time per day, which was a significant reduction from her 6 times per day preoperatively. She has had continued weight gain since her surgery up to 142 lb, BMI = 21.6. She also reported that her quality of life went from a 0 to a 4 on a 0- to 5-point severity scale. She is now tolerating her oral medications well, which has resulted in less variation in her serum Tacrolimus levels.

DISCUSSION

The exact cause of gastroparesis following lung transplant is unknown and is thought to be multifactorial with the likely mechanism being injury to the vagus nerve during the surgery, either from direct mechanical injury or ther-
nal injury secondary to cauterization. The question of whether the cause is related to the toxic effects of immunosuppressive medications, specifically cyclosporine, has also been raised but is unlikely considering that gastroparesis is uncommon following both renal and liver transplantation. In either case, gastroparesis following lung transplantation can be the cause of numerous complications, most importantly, aspiration pneumonia leading to obstructive bronchiolitis, a form of chronic rejection. Compromised oral intake may lead to the inability to take scheduled medications as well as poor medication absorption, which could endanger graft function in these immune suppressed patients. As a result of these chronic symptoms, patients have difficulty meeting nutritional and caloric needs. They may eventually require supplemental feeding via tube feeding. Current medical treatments include administration of prokinetic and antiemetic drugs to control symptoms. Patients who do not respond to these drugs can only be managed by enteral or TPN support.

In past reports, severe gastroparesis in either lung or combined heart-lung transplants has generally been managed medically with prokinetic or antiemetic pharmacologic therapy, with variable results. The only reports of surgical management include the successful use of gastric bypass surgery in 2 lung transplant recipients, and the successful use of TENS therapy in 2 lung transplant patients. The only published report of the use of gastric pacing in this population has been one report of the successful use of a gastric pacemaker in a single patient with a combined heart-lung transplant. Other procedures were considered along with the risks and benefits of performing various procedures in this high-risk patient on immunosuppressive therapy; a pyloroplasty or gastrojejunostomy was not done to avoid possible luminal injury or leakage in this high-risk patient. Because the patient had no evidence of a pyloric scar on preoperative imaging or EGD, endoscopic dilation was not deemed the best mode of treatment.

CONCLUSION

To our knowledge, this is the first report of using gastric pacemaker therapy for the successful treatment of severe gastroparesis in a lung transplant recipient. This case is unique in that it involved the simultaneous performance of a Nissen fundoplication to relieve the symptoms of posttransplant gastric reflux as well. Since the surgery, the patient has had a marked increase in weight, a significant reduction to resolution of her gastroparesis-related symptoms, and is able to tolerate her oral immunosuppressive medications. Because of the combining of the 2 operations, the patient has been able to improve her weight, reduce her symptoms, and improve her quality of life; perhaps as important, she has reduced her risk for aspiration of stomach contents, which greatly threatened the viability and longevity of her graft. We recognize that there are limitations to analyzing and scoring patient’s symptoms both pre- and posttherapy retrospectively, and definitive prospective evaluation should follow this initial report.

References:

1. Yiannopoulos A, Shafazand S, Ziedalski T, et al. Gastric pacing for severe gastroparesis in a heart–lung transplant recipient. J Heart Lung Transplant. 2004;23(3):371–374.
2. Berkowitz N, Schulman LL, McGregor C, Markowitz D. Gastroparesis after lung transplantation: potential role in postoperative respiratory complications. Chest. 1995;108:1602–1607.
3. Morgan KG, Szurszewski JH. Mechanisms of phasic and tonic actions of pentagastrin on canine gastric smooth muscle. J Physiol. 1980;301:229–242.
4. Forster J, Sarosiek I, Delcore R, Lin Z, Raju GS, McCallum RW. Gastric pacing is a new surgical treatment for gastroparesis. Am J Surg. 2001;182:676–681.
5. Medtronic Technical Brief, MEDN-0460 version 1. July 2007; 2–3.
6. Olufemi AA, Faul IJ, Vierra M, Triadafilopoulos G, Theodore J. The surgical management of severe gastroparesis in heart/lung transplant recipients. Chest. 2000;111:907–910.
7. Weinkauf JG, Yiannopoulos A, Faul JL. Transcutaneous electrical nerve stimulation for severe gastroparesis after lung transplantation. J Heart Lung Transplant. 2005;24(9):1444.