myocarditis who was treated by a conventional immuno-suppressive treatment. In particular, steroids and azathioprine were administered at the usual dose while cyclosporine, an additional component of the immunosuppressive regimen normally unable to control the disease, was not included in the treatment. Moreover, patient recovery was accompanied by the disappearance of inflammatory infiltrates including giant cells at a repeated cardiac biopsy, and finally a full cardiac recovery was maintained on a low dose of azathioprine at 16 months of follow-up. These observations suggest giant cell myocarditis might be a heterogeneous disease having either a curable or an untreatable substrate. It can be argued that a sarcoid or a granulomatous myocarditis (such as Wegener’s disease or Churg-Strauss syndrome) might have been missed. However, no systemic manifestation, such as vasculitis, renal and lung disease, nor hematologic (ie, eosinophilia for Churg-Strauss) nor immunologic (ie, antineutrophil cytoplasmatic antibody for Wegener’s disease) abnormalities, usually associated with these entities, were documented in our patient. Finally neither epithelioid histiocytes nor distinct granulomatous lesions were observed on several histologic sections of three cardiac biopsies.

On the other hand, additional reports of giant cell myocarditis responding to immunosuppression are currently available.

In conclusion, conventional doses of steroids and azathioprine may relieve in some cases the severe cardiac compromise of a giant cell myocarditis, avoiding the need for a heart transplantation.

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The Surgical Management of Severe Gastroparesis in Heart/ Lung Transplant Recipients*

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This article describes the use of gastric bypass surgery for severe gastroparesis in two lung transplant recipients. In addition to feeding intolerance, both our patients suffered from severe erosive esophagitis, transfusion-dependent upper GI hemorrhage, and recurrent aspiration pneumonia. They responded poorly to promotility agents and were eventually treated with Roux-en-Y esophagojejunostomy.

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Gastrointestinal—one patient with subtotal gastrectomy, and one with gastric bypass without distal gastric resection. Both cases were improved by surgery. Early surgical referral may be indicated in the management of lung transplant recipients with severe symptomatic gastroparesis in whom medical management has failed. On the basis of our experience, gastric bypass with esophagojejunosutony is a worthwhile option in lung transplant recipients with severe gastroparesis. (CHEST 2000; 117:907–910)

Key words: gastroparesis; gastroscope; lung transplant; surgery

Gastrointestinal complications are common in lung and heart/lung transplant recipients. Delayed gastric emptying, in particular, can lead to significant morbidity and mortality.1,2 In this article, we describe the use of gastric bypass surgery in the management of severe symptomatic gastroparesis in heart/lung transplant recipients. Our two cases suffered from erosive esophagitis, transfusion-dependent upper GI hemorrhage, and recurrent aspiration pneumonia. After demonstrating poor responses to promotility agents and jejunal tube feeds, both patients eventually underwent surgical bypass of the parietal stomach with acceptable outcomes.

CASE 1

A 31-year-old woman underwent heart/lung transplantation in March 1995 for idiopathic lung fibrosis and cardiomyopathy. She had no GI complaints preoperatively. Postoperatively, she developed hoarseness and right vocal cord paralysis. Posttransplant spirometry revealed an FEV1 of 1.27 L (43% predicted). Three months posttransplant, she developed dysphagia, early satiety, heartburn, and coughing during meals. Endoscopy revealed retained food in the stomach but no evidence of pyloric stenosis, gastritis, or peptic ulcer. A barium swallow revealed aspiration and free gastroesophageal reflux. Gastric scintigraphy demonstrated a markedly prolonged gastric half emptying time (6 h; normal, 90 min). The patient was encouraged to keep her head elevated after meals, and cisapride tablets, 20 mg qid, were prescribed. In July 1995, she developed melena. Endoscopy revealed a large gastric bezoar (successfully removed by an Ewald tube) and a 2 × 3 cm lower esophageal ulcer that was thought to be the source of bleeding. A jejunostomy tube with a gastrostomy port (allowing simultaneous jejunal feeding and venting of the stomach) was placed and omeprazole, 20 mg bid, was prescribed. She continued to have repeat episodes of nausea, vomiting, heartburn, epigastric discomfort, and melena, and required four transfusions of packed RBCs each month in order to maintain a hematocrit > 25%. In February 1996, antralrophic manometry was consistent with a marked neuropathic disturbance. Endoscopy confirmed severe gastroparesis and bezoar formation. In May 1996, because of continued blood transfusions, refractory esophagitis, and declining pulmonary function (due to repeated aspiration), she underwent a partial gastrectomy with a Toupet fundoplication, a pyloromyotomy, and a Jane-way gastrostomy. Two months postoperatively, a barium swallow revealed a stricture at the gastroesophageal junction, a dilated proximal esophagus, and obstruction at the level of the gastrojejunostomy that required repeated dilatations. Because of the patient’s continued symptoms, a completion gastrectomy with a retrocolic Roux-en-Y esophagojejunosutony and placement of a feeding jejunosutony was performed. The early postoperative course was complicated by a mechanical small bowel obstruction, requiring lysis of adhesions. For a period of 18 months, she took all her nutrition po and remained free of GI bleeding and aspiration. In early 1998, however, she again developed progressive feeding intolerance with increasing regurgitation and recurring aspiration. Spirometry revealed an FEV1 of 0.81 L (25% predicted). A small bowel enterolysis was consistent with small bowel dysmotility. A laparoscopic enteroteroenterostomy was performed without appreciable improvement in her symptoms. Currently, she receives half of her calorific requirements through central IV hyperalimentation and has gradually become tolerant of small quantities po. By September 1998, her pulmonary function and exercise tolerance had improved (spirometry revealed an FEV1 of 1.8 L [55% predicted]), and she is now able to carry out normal activities of daily living.

CASE 2

A 41-year-old man underwent a heart/lung transplant in February 1997 for idiopathic pulmonary fibrosis and cor pulmonale. He had a history of gastroesophageal reflux disease treated with omeprazole, 20 mg daily. A preoperative barium swallow was normal. Posttransplant spirometry revealed an FEV1 of 2.09 L (51% predicted). Four weeks posttransplant, endoscopy (for water brash and aspiration) revealed focal areas of gastritis and esophagitis. A percutaneous endoscopic gastrostomy tube was placed. In spite of endoscopic feeding, he continued to suffer recurrent aspiration pneumonia. Six weeks posttransplant, the gastrostomy tube was converted to an endoscopic gastrostomy/jejunostomy for simultaneous small bowel feeding and gastric drainage. Thirteen weeks posttransplant, he was readmitted with melena and anemia. He required multiple blood transfusions to maintain a hematocrit > 25%. Endoscopy revealed gastroparesis and Barrett’s esophagus. Cisapride was continued, and the dosage of omeprazole was increased to 20 mg tid. Three months later, he was readmitted because of severe epigastric pain and hematemesis. Endoscopy revealed erosive bleeding esophagitis and gastroparesis. Six units of blood were administered in order to maintain a hematocrit > 25%. Throughout the ensuing month, with nonsurgical (conservative) management, he required 18 blood transfusions for GI bleeding and melena. Spirometry revealed an FEV1 of 2.46 L (61% predicted). Because promotility agents, omeprazole, and jejunal tube feedings had failed to control his symptoms, he was referred for surgery. We chose to perform an unct Ouex-y-Y to a small gastric pouch. A gastrostomy tube (with a jejunal feeding port) was placed into the distal gastric pouch. This allowed decompression of the distal gastric pouch, and permitted feeding po. His postoperative course was uncomplicated. On the eighth postoperative day, he was able to tolerate a normal diet and his jejunal feedings were stopped. Postoperative gastric emptying studies, performed through the distal gastrostomy, demonstrated persistent markedly prolonged emptying of the stomach (not appreciably different from preoperatively). Nine months postoperatively, he has had no further blood transfusions. GI symptoms, or episodes of aspiration, and he has returned to work. Spirometry reveals an FEV1 of 3.24 L (80% predicted).

DISCUSSION

This article describes gastric bypass surgery in the management of severe gastroparesis in nondiabetic heart/lung transplant recipients. In both cases, severe symptoms persisted in spite of conservative management and jejunal tube feeding. Both underwent extensive surgery for severe gastroparesis, reflux esophagitis, internal bleeding, and repeated aspiration pneumonia. The first patient initially underwent a partial gastrectomy in order both to preserve a gastric
reservoir and to avoid the development of the Roux syndrome. The aim was to provide a good overall functional result by performing a significant gastric restrictive procedure, a partial posterior hemifundoplication to prevent reflux, and a pyloromyotomy to facilitate gastric emptying. Although a subtotal gastrectomy and esophagojejunostomy (performed because of dysphagia at the site of fundoplication and poor tolerance of gastrostomy tube feedings) produced a good outcome for approximately 30 months, she has suffered further problems, possibly due to progressive small bowel dysmotility. On the basis of this case, GI dysmotility that occurs after lung transplantation apparently may involve large areas of bowel and may be progressive. Our second patient underwent a gastric bypass procedure with an esophagojejunostomy to relieve delayed gastric emptying. He tolerated the procedure well, and is asymptomatic after 18 months of follow-up.

After lung transplantation, there is a high incidence of abdominal complications including prolonged adynamic ileus, diaphragmatic hernia after omental wrap, ischemic bowel, and giant gastric ulcers. Gastroparesis is a serious complication of heart/lung, bilateral lung, and isolated lung transplantation. One series reports the development of gastroparesis in 4 of 13 right lung, 2 of 12 left lung, and 2 of 4 heart/lung transplant recipients. In another series of 10 heart/lung transplant recipients, delayed liquid and solid gastric emptying was observed in three patients (30%) on radiographic gastric emptying studies. At Stanford, approximately 25% of heart/lung, bilateral lung, and isolated lung transplant recipients have symptoms suggestive of gastric dysmotility. Most patients respond to promotility agents and conservative management (antacids, raising the head of the bed, frequent small meals). In our experience with more than 150 heart/lung and 110 isolated lung transplants, only the two heart/lung transplant recipients in this report have required gastric bypass surgery. Interestingly, bilateral, but not unilateral, vagus nerve injury is believed to cause severe gastroparesis.

The pathophysiology of posttransplantation gastroparesis is not completely understood. The vagus nerve is at operative risk of mechanical and thermal injury during heart/lung or lung transplantation. Both vagus nerves course posteroinferiorly through the mediastinum to lie on the posterior surface of the corresponding lung root, where they give branches to the pulmonary plexuses. Both nerves then divide into branches to form the vagal plexuses that surrounds and supplies the esophagus. During thoracotomy, complete vagotomy, which can lead to gastric atony and dysphagia, is more likely to occur above the level of the lung roots, before the division of the vagi. Partial vagotomy, which can lead to enhanced liquid but delayed solid gastric emptying, may occur after injury to the vagus nerve below the level of the hilum. Vagus nerve injury, either from direct trauma or from thermal injury during cauterization of blood vessels in the posterior mediastinum, does not explain the gastric dysmotility that occurs in single-lung transplant recipients, in whom the vagus nerve bundles are generally well guarded and unexposed intraoperatively. Nonsurgical factors such as immunosuppressive therapy have also been implicated in the development of gastric dysmotility after lung transplantation. Although cyclosporine therapy may lead to neuropathy and vomiting, the use of immunosuppressive medication alone (cyclosporine, azathioprine, and prednisone) is unlikely to have caused the severe gastroparesis seen in the two cases in this report. Gastroparesis is not associated with liver and kidney transplantation. Moreover, improvements in gastric emptying have been reported in diabetic subjects with gastroparesis who undergo combined pancreas and kidney transplantation. Gastroparesis is therefore more likely due to operative injury to the vagus nerve than to an effect of medication or organ transplantation per se.

In addition to adverse effects on patient nutrition and well-being, gastroparesis appears to have a negative impact on pulmonary function in lung transplant recipients. Gastroparesis might predispose to microaspiration and pulmonary infections. Although there is no clear relation between delayed gastric emptying and bronchiectasis after heart/lung transplantation, aspiration pneumonia and deteriorating pulmonary function were prominent features in the current cases, and both were improved after
gastric bypass surgery. In a previous report, a lung transplant recipient with massive gastroesophageal reflux and obliterative bronchiolitis demonstrated improved pulmonary function after gastrojejunostomy. Early recognition, diagnosis, and effective treatment of gastroparesis might help to limit deteriorating lung function (particularly due to aspiration) in these patients.

This article is, to our knowledge, the first detailed description of gastric bypass surgery in heart/lung transplant recipients for the treatment of severe symptomatic gastroparesis. Surgical management is rarely necessary in these patients, but may be appropriate for patients with severe symptoms, in whom medical management has failed (see Fig 1). Based on our experience, the procedure that appears to result in a good functional outcome is an uncut Roux-en-Y gastric bypass to a small proximal gastric pouch. Further study of the pathophysiology of gastroparesis after lung transplantation is warranted.

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Effect of Pericardial Pressure on Human Coronary Circulation*

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A 52-year-old patient underwent percutaneous balloon pericardiotomy because of rapid fluid accumulation. During the procedure, we calculated the amount of blood flow to the nondiseased left anterior descending coronary artery at pericardial pressures of 1 mm Hg (upper boxes) and 17 mm Hg, (lower boxes) before (left boxes) and after (right boxes) adenosine vasodilation. Under increased pericardial pressure, although the hyperemic response persists, the absolute values of time-averaged peak flow velocity are substantially lower both before and after vasodilation than under normal pericardial pressure. A contributory factor to this is the increase in retrograde systolic flow that can be seen in tracings in the lower boxes (arrows).

Figure 1. Doppler recording of coronary blood flow velocity (in centimeters per second) in the left anterior descending coronary artery at pericardial pressures of 1 mm Hg (upper boxes) and 17 mm Hg, (lower boxes) before (left boxes) and after (right boxes) adenosine vasodilation. Under increased pericardial pressure, although the hyperemic response persists, the absolute values of time-averaged peak flow velocity are substantially lower both before and after vasodilation than under normal pericardial pressure. A contributory factor to this is the increase in retrograde systolic flow that can be seen in tracings in the lower boxes (arrows).

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