Laparoscopic Redo Nissen Fundoplication

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ABSTRACT

Nine patients underwent redo laparoscopic Nissen fundoplication because of failed primary laparoscopic antireflux procedure. Symptoms prior to reoperation included heartburn (n = 5), dysphagia (n = 2), dysphagia and heartburn (n = 1), and early satiety and epigastric pain (n = 1). Endoscopic and radiologic findings prior to reoperation included esophagitis (n = 6), reflux (n = 6), stenosis (n = 2), and hiatal hernia (n = 1). Findings at reoperation included fundoplication positioned on the stomach (n = 5); a disrupted cruroplasty (n = 1); gastric volvulus (n = 1); and an excessively tight wrap (n = 1) or cruroplasty (n = 1). Reconstruction of the fundoplication was performed according to accepted principles for this procedure. All patients were discharged within 2 days after the redo procedure. Follow-up time is 4–14 months. Preoperative symptoms were relieved in all patients and all antireflux medication have been discontinued. Routine postoperative esophagram and endoscopy demonstrated intact repair and without gastroesophageal reflux or stenosis. Reoperative laparoscopic Nissen fundoplication is feasible and effective.

INTRODUCTION

Ligastroesophageal reflux disease (GERD). The failure rate for laparoscopic Nissen in published series (intermediate follow-up) appears similar to the failure rate for open Nissen fundoplication. Some authors have noted that 4% of laparoscopic antireflux procedures will have reoperation for failure of the primary operation; the reoperative rate for open antireflux procedures has been quoted as 3%-6%. The principles of performing an open or laparoscopic Nissen fundoplication and of performing a redo antireflux procedure have been described. We have applied these principles to the performance of reoperative laparoscopic Nissen fundoplication, and here report our results in 9 patients.

PATIENTS AND METHODS

Nine consecutive patients (5 men: 4 women, average age = 41, range 23-67) were referred for evaluation of disabling symptoms after laparoscopic anti-reflux procedure (performed 2-23 months prior to re-

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ferral). Eight patients had symptoms within the first month after surgery; I patient experienced recurrence of heartburn about I year after surgery. All patients had undergone laparoscopic Nissen (the primary operation) for symptoms of heartburn and/or regurgitation, and all had mild or moderate esophagitis on esophagogastroduodenoscopy (EGD) before the primary operation. None of the patients complained of dysphagia. Esophageal manometry and pH monitoring were not performed routinely before the primary operation. Each patient had a different surgeon for the primary operation. All of the surgeons but one were inexperienced with laparoscopic fundoplications (<10 laparoscopic fundoplications).

Five patients suffered heartburn after the primary operation; EGD demonstrated persistent esophagitis and upper gastrointestinal barium radiography (UGI) revealed a patulus gastroesophageal junction with reflux. Two patients had dysphagia after the primary operation and UGI revealed stenosis at the fundoplication site. Another patient had early satiety, epigastric pain, and a large hiatal hernia on UGI after the primary operation.

One patient experienced dysphagia after the primary operation, which the surgeon believed was secondary to a tight wrap. This patient underwent laparoscopic revision of her Nissen to a 270° fundoplication (Toupet procedure) by the primary surgeon. After the second laparoscopic antireflux procedure, this patient suffered from both heartburn and dysphagia. Persistent esophagitis and near obstruction (below the gastroesophageal junction) was present on EGD and UGI, respectively. Manometry documented good esophageal motility. Dilatation only made this patient's heartburn worse.

A detailed history was taken in our clinic after referral for failed laparoscopic Nissen. EGD and UGl were obtained if none had been performed within I month. Our practice is to obtain esophageal manometry if, upon careful questioning, the patient describes difficult swallowing and/or if abnormal peristalsis is observed on UGI. The dysphagia present as sole symptom in 2 patients was of new onset after the primary operation. This dysphagia was explainable after viewing the UGI: therefore, manometry was not performed in these patients. The symptoms of all patients were disabling and not medically manageable. None of the patients had a shortened esophagus or esophageal dysmotility, either of which would have made a redo Nissen unadvisable.

After review of findings and a discussion of therapeutic options and risks, reoperation was desired by all patients. Redo laparoscopic Nissen fundoplication was performed following tenants described for both open and laparoscopic operation. 11-20 Adhesions invariably present between the inferior surface of the left lobe of the liver and stomach were lysed, the fundoplication was exposed, and all plication stitches were cut. Identification of the gastroesophageal junction was aided with transillumination from an intraesophageal lighted bougie or endoscope. In two cases the identification of the gastroesophageal junction (Z line) was facilitated by transillumenation of this area with the use of a gastroscope.

The wrap and fundus were mobilized and the short gastric vessels were divided if not done already (in 1 patient). The right and left bundles of the right crus were defined. The lower 5 cm of esophagus was mobilized to lie intraabdominally without tension. A posterior cruroplasty was done with interrupted nonabsorbable sutures and with a 50–60 Fr Maloney dilator within the esophagus. The patient with the large hiatal hernia was given a polytetrafluoroethylene (PTFE) mesh onlay to the cruroplasty to reinforce this repair, as previously described. Finally, a floppy 2–3 cm long 360° fundus-to-fundus wrap was performed with three interrupted nonabsorbable sutures; the superior suture included a portion of the crus.

RESULTS

The 5 patients with heartburn were found only at operation to have the fundus sewn to the greater curvature of the stomach (i.e., too "low"), resulting in an ineffective antireflux mechanism. The 2 patients with esophageal stenosis only had either a constrictive wrap (the short gastric vessels had not been divided) or a constrictive cruroplasty. The patient with the large hiatal hernia had a disrupted cruroplasty. The patient with both dysphagia and heartburn who had two previous laparoscopic antireflux procedures by the same surgeon was found to have the fundus sutured to the upper third of the lesser curvature of the stomach, which produced gastric volvulus.

Average operative time was 3.5 hr (range 2.5-4.5 hr). All patients had a gastrografin esophagogram the

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first post-operative day and were given a soft diet. Hospitalization time was ≤2 days, and all patients resumed a reasonable activity level after 1 week. There were no intraoperative or postoperative complications. Follow-up time is 4–14 months. All patients have resolution of preoperative symptoms and are off antireflux medication. Postoperative EGD and UGI were obtained routinely, and each fundoplication was intact without reflux, stenosis, or hiatal hernia, and resolution of esophagitis. Bloating was invariably present in all patients the first post-operative month with gradual resolution thereafter. Patient satisfaction with the redo procedure has been good to excellent.

DISCUSSION

Nine patients who underwent 10 previous laparoscopic antireflux procedures (9 Nissens, 1 Toupet) were referred for failure of operation. After evaluation, redo laparoscopic Nissen was elected in each. The most common cause (n = 5) of failure was suturing the fundus to the greater curvature of the stomach, with subsequent persistent reflux. Other causes were constriction of the gastroesophageal junction by the wrap or cruroplasty, and disrupted cruroplasty. Redo laparoscopic Nissen fundoplication was completed in all patients without complication, and after short-term follow-up results are good to excellent.

The various causes of antireflux operation failure are well described and include disruption of fundoplication or hiatal hernia repair (loss of sphincter mechanism), creation of fundoplication low on the stomach (inadequate sphincter mechanism), an overly tight wrap or cruroplasty (too vigorous a sphincter mechanism), esophageal motor disorder (failure of esophageal clearance), a combination of sphincter mechanism and esophageal clearance failure, alkaline reflux, or incorrect primary diagnosis of GERD. ^{5,8} The relative incidence for each cause varies from report to report. It has been stressed repeatedly in the literature that successful management of the failed antireflux operation requires thorough preoperative evaluation including careful history, knowledge of the procedure that was done which failed, endoscopy, radiography, ambulatory pH monitoring, and esophageal manometry. ³⁻¹⁰ We may be criticized for our selective use of manometry, but this practice has not produced an untoward result at our institution.

The management of antireflux operation failure also is well described and is distinct for each failure type. 5-8 The failed sphincter needs to be redone, poor esophageal clearance may be handled with dilatation or a partial fundoplication, a failed sphincter combined with poor esophageal clearance may require more complex reconstruction (e.g., Collis-Nissen or transhiatal esophagectomy), and alkaline reflux can be treated with Roux-en-Y diversion. There are proponents for both the abdominal and thoracic approach to reoperative antireflux surgery; generally speaking, dissection through fresh planes is advisable when confronted with a complicated patient.

The patients in this report represent uncomplicated failure of laparoscopic Nissen fundoplication secondary to isolated failure of sphincter mechanism, and all patients were treated with reconstruction of the sphincter. Creating a fundus-to-stomach wrap (as opposed to fundus-to-fundus) was the most common cause of sphincter failure. This finding may indicate a tendency for malplacement of the wrap when Nissen fundoplication is performed laparoscopically; the surgeon should be alert to this potential trap. We also advocate ligation of the short gastric vessels to produce a mobile fundus, which permits a floppy fundoplication. The cruroplasty and floppy fundoplication should be performed with a large (50–60 Fr) esophageal bougie in place. These maneuvers should prevent constriction at the gastroesophageal junction. Cruroplasty disruption may be minimized by taking wide bites of the crura with a nonabsorbable suture. We currently are engaged in a trial of cruroplasty reinforcement with PTFE onlay for a hiatal defect ≥8 cm.²¹

DePaula et al.²² performed laparoscopic and thoracoscopic reoperation on 19 patients with failed antire-flux procedure. There was one conversion. Results were good to excellent in 16 patients (84%) after a mean follow-up of 13 months. Alexander and Hendler²³ reported two cases of laparoscopic reoperation on failed antire-flux procedure with excellent results. These authors have emphasized that redo laparoscopic antire-flux surgery best is performed by those with extensive experience with both laparoscopic surgery and reoperative antire-flux surgery (a similar plea has been made concerning open reoperative antire-flux surgery), and we agree.

Selection of the patient for redo laparoscopic antireflux operation must be made carefully, and not all pa-

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tients will benefit by a redo of the original operation. The cause of failure of the primary procedure in the aforementioned 9 cases was identified preoperatively as defective sphincter mechanism, which made all of these patients manageable with a redo of the primary procedure. Redo laparoscopic Nissen fundoplication is feasible and effective if performed for the proper indication.

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