Esophagotomy During Laparoscopic Heller Myotomy Cannot Be Predicted by Preoperative Therapies and Does Not Influence Long-term Outcome

Steven Rakita, M.D., Mark Bloomston, M.D., Desiree Villadolid, B.S., Donald Thometz, B.S., Emmanuel Zervos, M.D., Alexander Rosemurgy, M.D.

The conventional wisdom is that inadvertent esophagotomy complicates laparoscopic Heller myotomy. This study was undertaken to determine if esophagotomy at myotomy can be predicted by preoperative therapy, and if esophagotomy and/or its repair jeopardizes outcomes. Of 222 laparoscopic Heller myotomies undertaken since 1992, inadvertent esophagotomy occurred in 16 patients (7%); 60 patients who underwent myotomy without esophagotomy were utilized for comparison. Dysphagia and reflux before/after myotomy were scored by patients on a Likert scale (0–5). The median (mean ± SD) follow-up after myotomy with esophagotomy was 38.8 months (31.6 ± 21.9 months) versus 46.3 months (51.0 ± 21.2 months) after myotomy alone. All esophagotomies were immediately recognized and repaired. Patients who experienced esophagotomy were similar to those who did not in application of Botox (56% vs. 77%) or dilation (44% vs. 65%), years of dysphagia (7.3 ± 5.4 vs. 7.4 ± 6.0), and mean preoperative dysphagia score (4.9 ± 0.4 vs. 4.8 ± 0.4). Esophagotomy led to longer hospitalizations (5.2 days ± 2.5 days vs. 1.5 days ± 0.7 days, $P < 0.05$) but not different postoperative dysphagia scores (1.5 ± 1.7 vs. 2.1 ± 1.4), reflux scores (1.4 ± 1.7 vs. 2.3 ± 1.3), or good or excellent outcomes (86% vs 84%). Esophagotomy during laparoscopic Heller myotomy is infrequent and cannot be predicted by preoperative therapy or duration or severity of dysphagia. Furthermore, complications after esophagotomy are infrequent and outcomes are indistinguishable from those of patients undergoing uneventful myotomy. (J GASTROINTEST SURG 2005;9:159–164)

KEY WORDS: Achalasia, Heller myotomy, esophagotomy, fundoplication

INTRODUCTION

Achalasia presents as dysphagia with regurgitation, heartburn, chest pain, and weight loss.[1] It is a rare primary motor disorder characterized by increased resting pressure with incomplete relaxation of the lower esophageal sphincter (LES) as well as uncoordinated contractions of the esophagus and, classically, aperistalsis. This is thought to be caused by a lack of nonadrenergic, noncholinergic inhibitory ganglion cells resulting in an imbalance of excitatory and inhibitory neurotransmission.[2,3]

Treatment focuses on weakening, dividing, or disrupting the constricting circular muscle fibers of the distal esophagus to incapacitate the LES. Modalities include medical therapy, endoscopic interventions including pneumatic dilation or botulinum injections, and surgery. Medical treatment, most often employing calcium channel blockers or nitrates, offers only limited temporary relief.[4–6] Endoscopic botulinum toxin (Botox) injected into the LES has been found to provide improved LES relaxation with

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From the Department of Surgery, University of South Florida College of Medicine, Tampa, Florida.
Reprint requests: Alexander Rosemurgy, M.D., P.O. Box 1289, Tampa, FL 33601. e-mail: arosemur@hsc.usf.edu

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relatively good efficacy; however, the results are uniformly short-lived, with only about 30% remaining palliated at 1 year.[7–10] Forceful pneumatic dilation of the LES results in good outcomes in 60% to 72% of patients but with a high recurrence rate, a definable perforation rate of 2% to 13%, and a risk of significant gastroesophageal reflux due to an incompetent LES in 30%.[1,10–12]

Surgery provides well-documented relief of dysphagia in over 90% of patients.[12–16] With the advent of the laparoscopic approach, the risk of complications is low and patients are treated with relatively little postoperative pain, short hospital stays, and rapid return to normal daily activities. With increasing frequency, surgery is being offered as first-line therapy for achalasia,[17] although most patients have been treated with endoscopic therapies at the time of referral. We, like others, have suggested a trend toward increased esophagotomy rates in patients with multiple preoperative endoscopic interventions.[17]

The long-term effect of esophagotomy during Heller myotomy remains unknown. The purpose of this study was to determine if esophagotomy during laparoscopic Heller myotomy can be predicted by the duration or severity of dysphagia or preoperative therapy with Botox or pneumatic dilation, and if esophagotomy and/or its repair affects long-term outcome following myotomy. Our hypotheses were that the duration or severity of dysphagia would not increase the incidence of esophagotomy and that preoperative therapy would increase the incidence of esophagotomy, but that the long-term outcome after myotomy would not be negatively impacted by myotomy.

MATERIALS AND METHODS

Preoperative Assessment

All patients underwent barium esophagram and esophageal manometry prior to laparoscopic Heller myotomy. The diagnosis of achalasia was confirmed in all patients. For all patients, barium esophagram demonstrated a dilated, aperistaltic proximal esophagus with a "bird's beak" narrowing distally, and esophageal manometry confirmed incomplete relaxation of the lower esophageal sphincter mechanism with an absence of esophageal peristalsis.

Operation

Our technique for laparoscopic Heller myotomy with concomitant endoscopy has been previously described.[16] Briefly, laparoscopic Heller myotomy was undertaken utilizing three 10 mm ports and two 5 mm ports. The first port, a Hasson cannula, was placed through the umbilicus and pneumoperitoneum was established. A second port was placed along the right anterior axillary line cephalad to the umbilicus. A fan retractor was utilized through this port to lift the liver off the anterior surface of the esophagus and stomach. The third port was placed in the subxiphoid position just below the liver edge. This port functioned as the videoscopic port for the remainder of the operation. The fourth and fifth ports, both 5 mm ports, were placed in the right and left mid-clavicular lines along the subcostal margins.

The epigastric fat pad was mobilized and the peritoneum and phrenoesophageal membrane directly over the esophagus were dissected sufficiently to identify and split the longitudinal muscle fibers of the lower esophagus. The anterior vagus nerve was identified and preserved. The circular muscle fibers were identified and myotomy undertaken using the 90°-angle hook cautery, extending 6 to 8 cm into the mediastinum and 0.5 to 1.0 cm onto the stomach. The trocar along the right mid-clavicular line was upsized to a 10 mm port and concomitant fundoplication was undertaken if there was a large hiatal hernia, if there was a patulous esophageal hiatus, or as part of the repair of an esophagotomy.

Concomitant per oral endoscopy was undertaken at the time of operation.[18] Gentle endoscopic esophageal insufflation was utilized to document adequacy of myotomy. After myotomy, the esophagus was tested for esophagotomy. This was done by instilling saline around the esophageal hiatus and intraesophageal insufflation. Esophagotomy was noted by the presence of air bubbles. Esophagotomies noted intraoperatively were closed with fine monofilament sutures. Anterior fundoplication (Dor fundoplication) was undertaken to buttress the esophageal repair.

Postoperative Management

On the day of operation, all patients undergoing uneventful myotomy underwent postoperative gastrografin esophagram followed by barium esophagram to detect occult esophageal perforations and to assess esophageal emptying. If no leak was present and esophageal emptying was swift, patients were started on clear liquid diets and discharged the following day on a full liquid diet with instructions to slowly advance their diet over the next 2 weeks. If slow emptying was seen during the esophagram, then patients were maintained on clear liquids in the hospital until the edema of the lower esophagus resolved, as determined by progressive improvement in their swallowing and/or repeat esophagram. Patients were discharged with instructions to advance their diets over the ensuing weeks, as tolerated. Patients were