Minimally Invasive Surgery for Achalasia: A 10-Year Experience

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Minimally invasive esophagomyotomy for achalasia has become the preferred surgical treatment; the employment of a concomitant fundoplication with the myotomy is controversial. Here we report a retrospective analysis of 53 patients with achalasia treated with laparoscopic Heller myotomy; fundoplication was used in all patients except one, and 48 of the fundoplications were complete (floppy Nissen). There were no deaths or reoperations, and minor complications occurred in three patients. Good-to-excellent long-term results were obtained in 92% of the subjects (median follow-up 3 years). Two cases (4%) of persistent postoperative dysphagia were documented, one of which was treated with dilatation. Postoperative reflux occurred in five patients, four of whom did not receive a complete fundoplication; these patients were well controlled with medical therapy. We suggest that esophageal achalasia may be successfully treated with laparoscopic Heller myotomy and floppy Nissen fundoplication with an acceptable rate of postoperative dysphagia. (J GASTROINTEST SURG 2004;8:18–23)

KEY WORDS: Achalasia, esophagomyotomy, minimally invasive surgery, dysphagia, gastroesophageal reflux

Achalasia, the most common motility disorder of the esophagus, has an incidence of 0.5 to 1 per 100,000.[1–3] The etiology of achalasia may involve autoimmunity and/or viral infection, but this is controversial.[4–6] Heller described the successful surgical treatment of chronic cardiospasm in 1913.[7] His anterior and posterior cardiomyotomy technique was modified in 1923 by Zaaijer[8] to include a single anterior myotomy. Since then, standard surgical treatment for achalasia has been this modified Heller myotomy performed through the abdomen or chest.[9] Heller myotomy is successful in 85% to 90% of patients with achalasia, which compares favorably with nonoperative treatment.[10–13] A minimally invasive approach to Heller myotomy became standard in the 1990s.[9,14,15] We began treating achalasia with minimally invasive Heller myotomy in 1992; this is a 10-year retrospective report of 53 consecutive patients treated with this procedure.

PATIENTS AND METHODS

Between January 1992 and August 2002, data were collected on patients who underwent laparoscopic Heller myotomy for esophageal achalasia, under the supervision of one of us (C.T.F.). Routine preoperative evaluation included a history and physical examination, esophagogastroduodenoscopy, manometry, and barium esophagram. Manometry was used

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to confirm the diagnosis of achalasia (i.e., presence of lower esophageal dysfunction). The barium esophagram (real-time fluoroscopic) was used as a semiquantitative measure of esophageal peristalsis. Traditionally achalasia has been associated with absence of peristalsis in the esophageal body. Because the pathophysiology of achalasia involves the lower esophageal sphincter, however, peristalsis in the esophageal body can still be present, even in advanced cases of achalasia.[16,17] Mild-to-moderate dysmotility was defined as zero to one peristaltic waves and severe dysmotility was defined as two or more peristaltic waves in the esophageal body during the course of the barium study. This fluoroscopic assessment of esophageal peristalsis provided an indicator of preoperative disease severity.

The technique of laparoscopic Heller myotomy has been well described[18]; briefly, a 6 to 7 cm myotomy on the anterior cardia (extending to the distal esophagus) was performed with an insulated hook electrocautery after mobilization of the gastroesophageal junction. The insulated hook cautery device (product under development) consisted of a conventional hook cautery with insulation covering the outer edge of the hook. This arrangement limits the application of electrical energy to the tissue, which is caught within the inner edge of the hook, minimizing any collateral injury to tissue that may inadvertently contact the outer edge of the hook. The myotomy is performed over the tapered tip of a 50 F lighted esophageal bougie (Fig. 1). After completion of the myotomy, the bougie is advanced so that the 50 F circumference is appropriately placed at the level of the gastroesophageal junction. The fundoplication is then performed with the bougie in proper position.

Either a floppy Nissen[19,20] or a Toupet (posterior 270-degree)[18] fundoplication was performed after the myotomy; the pertinent details of the Nissen fundoplication are given here. Division of the short gastric vessels is routine. The distal esophagus is mobilized downward so that 4 to 5 cm is intra-abdominal without tension. The esophageal hiatus is closed around the esophagus (with the 50 F bougie in place) using interrupted 2-0 polyester sutures if necessary. The fundus is carefully identified to avoid creating a wrap with the body of the stomach. The wrap is constructed using three 2-0 polyester sutures with the 50 F bougie in place and is 2 cm long when completed. The cephalad wrap stitch anchors the wrap to the diaphragm; none of the stitches incorporate the esophagus. The completed fundoplication is tested for laxity by inserting a 10 mm instrument alongside the esophagus. If the wrap is not loose, it is taken down and reconstructed.

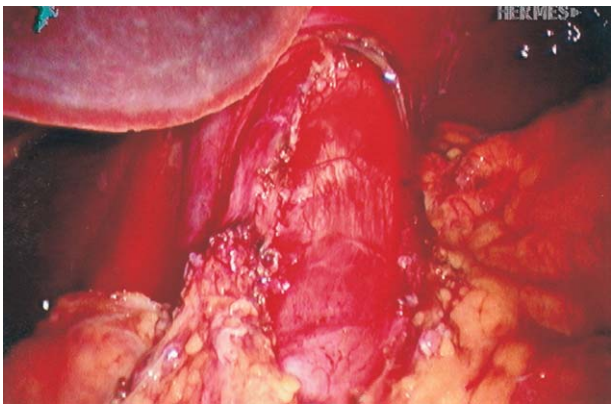

Fig. 1. Esophagomyotomy with lighted bougie in place.

Routine postoperative visits were scheduled at 1 week, 1 month, 3 months, and yearly after surgery; if a visit was not possible, the patient was contacted by phone. Each patient was questioned specifically about dysphagia, heartburn, and regurgitation. Routine postoperative testing included a barium esophagram within the first 3 months after the operation. Postoperative manometry and endoscopy were performed at the discretion of the referring gastroenterologist. Postoperative outcome was scored according to the method of Visick,[21] who described a method of classifying postgastrectomy outcome based on patients' symptoms. We have modified this scoring system to apply to our series and simply refer to the system as the modified Visick, or m-Visick. An excellent result (m-Visick I) was defined as the patient with rare (once per week or less) to no episodes of dysphagia or gastroesophageal reflux; these patients typically did not take medications for esophageal symptoms. A good outcome (m-Visick II) was defined as occasional episodes (several times per week) of dysphagia and/or reflux that may have required medication. A fair result (m-Visick III) was defined as more frequent symptoms controlled with (daily or near-daily) medical treatment. Unsatisfactory outcome and/or treatment failure (m-Visick IV) was defined as symptoms that were poorly controlled with medication and/or any patient undergoing reoperation.

RESULTS

All of the patients (n = 53; 37 men and 16 women; mean age 48 [range 21 to 75] years) in this series had preoperative dysphagia. The diagnosis, evaluation, and any endoscopic therapy for achalasia were performed by each patient's gastroenterologist prior to the surgical referral. Routine preoperative barium

esophagram revealed severe or mild-to-moderate dysmotility in 18 (34%) and 35 (66%) patients, respectively. Eighteen patients (34%) underwent preoperative pneumatic dilatation and nine patients (17%) had intrasphincteric botulinum toxin injections; 26 patients (49%) did not have any endoscopic therapy prior to esophagomyotomy.

All patients underwent a laparoscopic Heller myotomy. Forty-eight patients (90%) underwent concomitant Nissen fundoplication, four patients (8%) underwent Toupet fundoplication (patients 2, 6, 15, and 17, who all had severe esophageal body dysmotility), and one (patient 42, who had megaesophagus with severe dysmotility) did not receive a fundoplication. Average operative time was 87 ± 24 minutes (standard deviation [SD]; range 41 to 148 minutes); there were no open conversions or intraoperative complications. The median hospital stay was 2 days (range 1 to 4 days), and there were no deaths. Major postoperative complications consisted of one short-term reintubation and one case of pneumonia; the former patient was extubated prematurely and required reintubation in the recovery room, and the latter patient aspirated during induction of anesthesia. Minor complications consisted of urinary retention (n = 2), atelectasis (n = 2), and trocar site hematoma (n = 1).

Median follow-up was 3 years (range 6 months to 9 years). There were no reoperations. The m-Visick scores of postoperative outcome are presented in Table 1; the rate of the m-Visick I–II was 50/53 = 94%. Postoperative gastroesophageal reflux and dysphagia occurred in five (9%) and two (4%) patients, respectively. Four of the five patients with postoperative reflux had severe preoperative esophageal dysmotility, and one patient had moderate preoperative dysmotility; the concurrent antireflux procedure in these patients was a Nissen fundoplication (the one patient with moderate dysmotility), a Toupet fundoplication (n = 3), or no fundoplication (n = 1). These five patients opted for medical management of their postoperative reflux; four of these patients were classified as m-Visick II, and the fifth patient (the only person in this series without an antireflux procedure) had postoperative reflux severe enough to be placed in the m-Visick III category.

One of the two patients with postoperative dysphagia (see Table 1) had severe dysmotility on preoperative evaluation, and the other had mild-to-moderate dysmotility. Both of these patients had a Nissen fundoplication at the time of their myotomies, and both of their postoperative esophagrams indicated either an incomplete myotomy or an excessively tight wrap. The former patient declined any further intervention after the myotomy. This patient's postoperative dysphagia was improved compared to the preoperative state, so this patient was classified as m-Visick III. The latter patient with postoperative dysphagia (who preoperatively had mild-to-moderate dysmotility) underwent successful pneumatic dilatation of the distal esophagus. Even though his final outcome was good, he was classified as m-Visick IV because the surgical procedure was not successful.

DISCUSSION

In a total of 616 patients treated with minimally invasive esophagomyotomy culled from 12 publications on the subject, the rate of m-Visick I–II outcome was 90% (Table 2). This data summary is flawed, of course, because of the wide range of definitions, follow-up, and evaluation schemes used in these studies. Nevertheless, the results of our series are in concordance with the available results from patients undergoing minimally invasive esophagomyotomy for achalasia. This occurred despite our near-routine use of complete (Nissen) fundoplication. The addition of a fundoplication to an esophageal myotomy for achalasia is controversial. Many investigators advise selective utilization of fundoplication (especially partial) during esophagomyotomy for achalasia, because of the concern of wrap-induced dysphagia.[22,23] We attribute the low rate of dysphagia in our patients with complete wraps to our technique of floppy fundoplication, which is slightly modified from

Table 1. m-Visick scores in the follow-up period

m-Visick score	No. of patients	Severe preoperative dysmotility	Mild-to-moderate preoperative dysmotility	Fundoplication		
				N	T	None
I	46	11	35	46	0	0
II	4	3	1	1	3	0
III	2	2	0	0	1	1
IV	1	0	1	1	0	0
TOTAL	53	16	37	48	4	1

N = Nissen; T = Toupet.

Table 2. Published results of minimally invasive esophagomyotomy

Reference	No. of cases	m-Visick I–II (%)	Major complications (%)	Postoperative dysphagia (%)	Postoperative GERD (%)	Mean follow-up (mo)
Ancona et al.[30]	17	94	0	6	0	7
Rosati et al.[31]	25	96	4	4	NA	12
Delgado et al.[32]	12	83	1.6	16	0	3
Swanstrom and Penning et al.[33]	12	92	0	8	17	16
Hunter et al.[34]	40	90	7.5	10	5	12
Vogt et al.[35]	20	90	10	10	10	2
Wang et al.[36]	30	89	NA	22	54	18
Kjellin et al.[37]	21	100	9.5	29	21	22
Patti et al.[14]	133	90	9	11	17	23
Heniford et al.[38]	49	96	10	NA	NA	12
Luketich et al.[39]	57	92.5	14.5	9.4	9.4	19
Bloomston and Rosemurgy et al.[40]	100	86	9	16	14	22
Mean (total)	(516)	91.5	6.8	12.8	14.7	14

GERD = gastroesophageal reflux disease; NA = data not available.
Total number of cases = 516.

the classic description of Donahue et al.[19] (see Patients and Methods).

Early in the series we performed either a Toupet or no fundoplication in conjunction with the myotomy in five patients who had severe esophageal body dysmotility, because the convention had been to perform (at most) a partial fundoplication after the myotomy in patients with advanced disease. Four of these patients developed postoperative reflux. Subsequently we decided to perform a floppy Nissen fundoplication in conjunction with the myotomy in all patients. There have been several reports[24,25] of postmyotomy reflux being the cause of severe esophagitis, Barrett's metaplasia, and adenocarcinoma. In addition, 24-hour pH monitoring studies have revealed a decrease in the frequency of reflux when a fundoplication is performed.[26] To address these concerns, our standard approach has been to employ fundoplication routinely with myotomy for surgical treatment of achalasia. It is conceivable that there were cases of occult postoperative reflux in our series; because routine postoperative pH monitoring was not performed, however, these hypothetical cases were not diagnosed. The importance of reflux that can be detected with the pH probe, but which does not cause symptoms, is unclear.

The type of fundoplication to perform in conjunction with an esophageal myotomy is somewhat controversial. The Toupet fundoplication purportedly results in decreased postoperative reflux and dysphagia compared to a complete wrap[27]; the Dor fundoplication has been favored because it is an easily constructed partial wrap that covers the myotomy site.[28,29] Comparative data from various fundoplication methods in prospective trials are lacking. Our series suggests that a floppy Nissen fundoplication can be performed in conjunction with esophageal myotomy in patients with achalasia without incurring postoperative dysphagia.

Common causes of persistent dysphagia after Heller myotomy include underlying esophageal dysmotility, incomplete myotomy, preoperative error in diagnosis, esophageal stricture, improperly constructed fundoplication, spontaneous closure of the myotomy, or any combination of the above. The etiology of persistent postoperative dysphagia in the two patients in this series is not clear. Spontaneous closure as an etiology is unlikely because of a lack of a symptom-free interval; preoperative error in diagnosis is unlikely because of the extensive (endoscopic, manometric, and radiographic) preoperative evaluation obtained in these two patients, and there was no evidence of stricturing noted intraoperatively. An improperly constructed fundoplication or an incomplete myotomy is a more likely cause(s) of persistent dysphagia in these two patients. Because these patients did not undergo reoperation, the precise cause remains unknown.

We maintain that the technique of floppy fundoplication, as described in the Patients and Methods section, helped produce the low rate of postoperative dysphagia. In particular, we believe that our practice of anchoring the fundoplication to the crura, but not to the esophagus itself, facilitates the final product of a loose wrap. Another factor that likely contributed to our low rate of dysphagia was our use of the lighted

esophageal bougie during the myotomy. We particularly like how the bougie illuminates the muscle fibers during the myotomy, which we believe improves the precision of this procedure; there is a superb visualization of the interface between the muscularis propria and the submucosa. In addition, the surgeon can watch (via the laparoscope) the insertion of the lighted bougie past the gastroesophageal junction, and thereby advise the anesthetist on his/her progress with the insertion. We believe that this communication improves the safety of the potentially dangerous bougie insertion. In more than 600 laparoscopic esophageal procedures in which the lighted bougie was used, we have not had any insertion-related complications.

In most of the patients in this series, we treated achalasia with laparoscopic Heller myotomy and floppy Nissen fundoplication, and obtained long-term results comparable to those from other published series. Even though we used a complete wrap in the vast majority of our patients, the rate of postoperative dysphagia was at the low end of the published range. We support the concept that laparoscopic Heller myotomy with floppy Nissen fundoplication is a sound surgical option for achalasia.

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