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Distal Esophageal Perforation Repair During Laparoscopic Esophagomyotomy: Evaluation of Outcomes and Review of Surgical Technique

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ABSTRACT

***Objective*: To describe the technique employed and our experience with primary laparoscopic repair of distal esophageal perforations produced during laparoscopic esophagomyotomy, as well as to evaluate the outcomes.**

***Materials and Methods*: We analyzed six cases of patients with primary achalasia in whom distal esophageal mucosal perforations were caused during laparoscopic esophagomyotomy. A primary repair and fundoplication was performed in five cases; in the sixth patient, the perforation could not be recognized during the surgical procedure. The postoperative follow-up included clinical evaluation, upper gastrointestinal endoscopy, esophageal manometry, and ambulatory 24-h esophageal pH monitoring.**

***Results*: Five patients reported dysphagia relief and were highly satisfied with the final surgical outcome. In one case we observed an altered postoperative 24-h esophageal pH. Two patients developed esophageal leakage, one with a fatal outcome.**

***Conclusion*: The primary repair of distal esophageal perforations during laparoscopic esophagomyotomy is a valid therapeutic option and does not alter the surgical purpose. However, if the perforation is not recognized early on, the prognosis can change.**

INTRODUCTION

ESOPHAGEAL PERFORATION IS ONE of the most serious lesions that may occur during laparoscopic esophagomyotomy (LEM), carrying the risk of fistulas, mediastinitis, and sepsis, among other complications, as well as also limiting the achievement of the surgical goal.[1–3]

Options for the management of esophageal perforations include conservative treatment with total parenteral nutrition (TPN), intravenous antibiotic, and gastric drainage;[4,5] esophageal covered metal stents;[6] endoscopic closure;[7] open,[4,8] thoracoscopic, or laparoscopic esophageal primary closure;[9,10,11] or esophageal resection.[4,9]

One of the therapeutic alternatives during the LEM is primary closure of the distal esophageal mucosa. Various authors have agreed on the possibility of laparoscopic repair through the same surgical procedure.[1,2,10–13] Nevertheless, in several published series, neither the clinical evolution nor postoperative monitoring with esophageal tests are sufficiently detailed, which may have led to misleading figures for postsurgical failure and complications.

We describe our experience with distal esophageal perforation management during LEM. In addition, the surgical technique employed is explained together with an analysis of long-term outcomes.

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MATERIALS AND METHODS

Our surgical team has carried out 64 LEM from February 1993 to August 2003 to treat primary achalasia at our institution. We detected esophageal perforations in six patients.

The pre- and postoperative protocol studies for the patients with primary achalasia included clinical evaluation, upper gastrointestinal endoscopy (UGE), esophagogastric biopsies, and esophageal manometry (lower esophageal sphincter [LES] normal pressure, 14.3–34.5 mm Hg). The 24-h ambulatory esophageal pH monitoring (normal value, $<$14.72 on the Johnson-DeMeester score) was performed as a postoperative test.

Surgical technique

Four trocars were placed (Fig. 1). The phrenoesophageal and gastrohepatic ligaments were dissected, exposing the hiatus area, and a retroesophageal window was created. The myotomy was performed on the longitudinal esophageal axis until the mucosa protrusion was achieved in 50% of the esophageal perimeter (Fig. 2). In the case of an esophageal perforation, the mucosa was carefully repaired with intracorporeal running suture, employing 4–0 polyglactin 910 or 4–0 polypropylene sutures (Fig. 3).

Antireflux procedure

Our group has been using the posterior esophagogastropexy[14,15] as an antireflux procedure and a nonabsorbable suture (0-polypropylene) and the Gea extracorporeal knot have been employed for the fundoplication (Figs. 4, 5).[16,17]

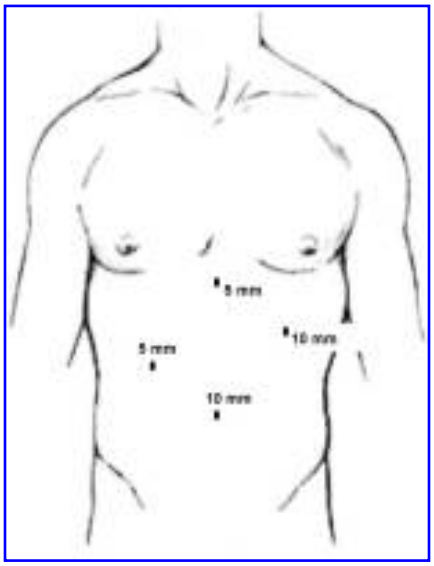

FIG. 1. Port placement for the laparoscopic esophagomyotomy.

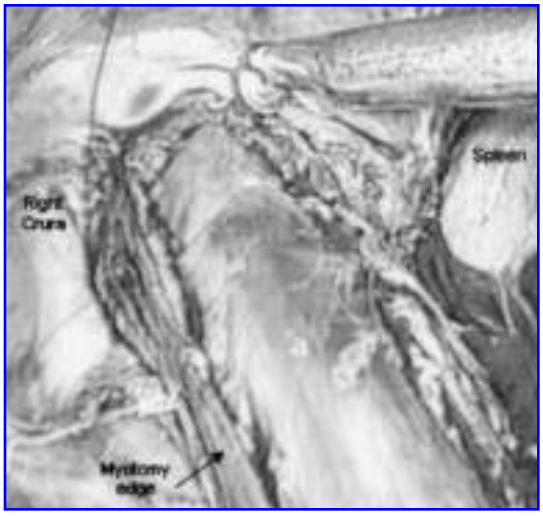

FIG. 2. The esophageal mucosa (a) is protruded through one half of the esophageal perimeter.

RESULTS

Three of six patients (cases 1, 2, and 3) had no significant clinical history. Case 4 had a history of laparotomy for gastrostomy two years earlier, due to food intake intolerance, as well as endoscopic dilations over the past 7 years, with an unsatisfactory response. The physical examination revealed a chronic malnutrition. Case 5 had a history of medical treatment with nitrates and endoscopic dilations five years earlier. Case 6 had a failed open Heller myotomy 11 months previously.

All six patients complained of progressive dysphagia from solids to liquids, associated with regurgitation, postprandial vomiting, and weight loss (Table 1).

A dilated esophagus was seen by UGE in all patients and one had a sliding hiatus hernia (case 3). All six individuals showed retained food and a distal esophageal concentric stricture that partially impeded passage of the videoendoscope to the gastric chambers, particularly in cases 1 and 6, this latter patient with a proximal gastric "hourglass" appearance.

Esophagogastric biopsies were performed in all patients in order to rule out a malignant stenosis. The esophagogram reports were in accordance with these UGE findings. The esophageal manometries reported the absence of primary esophageal peristalsis and impaired LES relaxation in response to swallowing. The primary achalasia was diagnosed and the LEM was suggested.

In all cases and especially in case 6, we observed difficulties during the dissection of the submucosal plane close to the gastroesophageal junction (GEJ), caused by the dense fibrosis between the muscular and the submucosal plane.

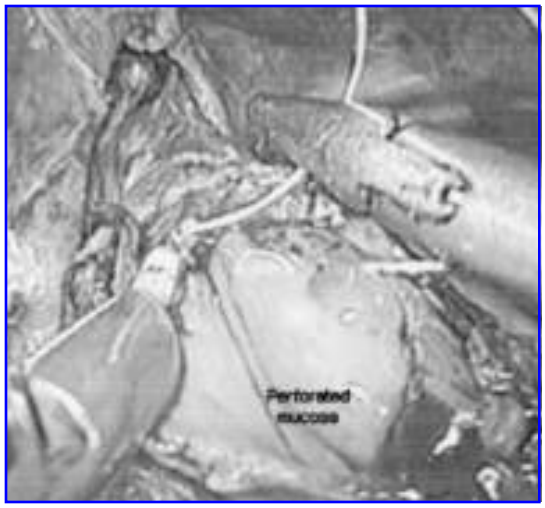

FIG. 3. The esophageal mucosa is repaired with an intracorporeal suture.

In cases 1 and 2, due to this esophageal fibrosis, it was not possible to separate the muscular plane from the submucosal plane using the conventional surgical technique that we used in the other 62 cases of the total series. This led us to perform a controlled esophageal wall opening, on the anterior surface (about 1.5 to 2 cm in length), 1 cm above the GEJ, and to repair it transversally, like a pyloroplasty, to obtain an adequate esophageal diameter at the GEJ level.

In case 6, two incidental esophageal perforations occurred. We also observed a partial gastric volvulus caused by some gastric adhesions, attributable to their prior open Heller myotomy, that was attempted to correct.

In cases 3 and 4, 1-cm incidental perforations above the GEJ occurred. In case 5, no perforation was identified perioperatively.

After the LES was completed, the esophageal walls were repaired (except in case 5) and a 360-degree fundoplication[14,15] was performed in cases 1, 2, 3, 5, and 6. In case 4, an anterior partial fundoplication (180-degree) was done to diminish the surgical time, because of a right pneumothorax after attempting subclavian vein access.

In all patients the diaphragmatic crura were closed, intra-abdominal drainage was placed, and nasogastric tubes were inserted. The surgical procedures lasted for 90 to 240 minutes.

Postoperative course

The postoperative course was satisfactory for cases 1, 2, 3, and 4. All patients were maintained with nasogastric tubes, intravenous antibiotics, and restricted oral intake. An abdominal computed tomography scan was carried out on case 3 in order to rule out a clinical suspicion of an intra-abdominal collection.

Prior to starting oral intake, an esophagogram with water-soluble contrast was indicated between postoperative days 2 and 5 to rule out the possibility of leakage; after this the patients were discharged. Oral intake was initiated earlier in patients in better nutritional condition (cases 1 and 2), while TPN was administrated in cases 3 and 4 until they were ready for oral feeding.

After the first postoperative month, a control UGE was done for case 3 as a result of the recurrence of dysphagia. The endoscopic findings were compatible with esophageal candidiasis and the patient had a satisfactory response to the antifungal treatment. The patient's clinical condition and weight increase are shown in Table 1.

However, the postoperative picture was unsatisfactory for two patients: case 6 developed a controlled esophagothoracic fistula and empyema: the cardiothoracic surgeon performed a thoracotomy for their debridement and drainage. During the following three months this patient required endoscopic esophageal dilation because of persistent dysphagia. Afterwards, the patient's condition improved.

The patient in case 5 had chest pain and tachycardia 48 hours postoperatively. The postoperative esophagogram showed with a fine esophageal leakage and the CT scan demonstrated a subhepatic collection. An exploratory laparoscopy and a transoperative intraesophageal methylene blue injection were done, with no evidences of esophageal leaks. In this surgical procedure, a 5-cc collection was drained and two closed drainages were placed; however, the esophageal perforation could not be identified. Over the following two weeks her clinical condition grew worse, and the patient was admitted to the ICU as a result of an acute ventilation failure in addition to a systemic inflamma-

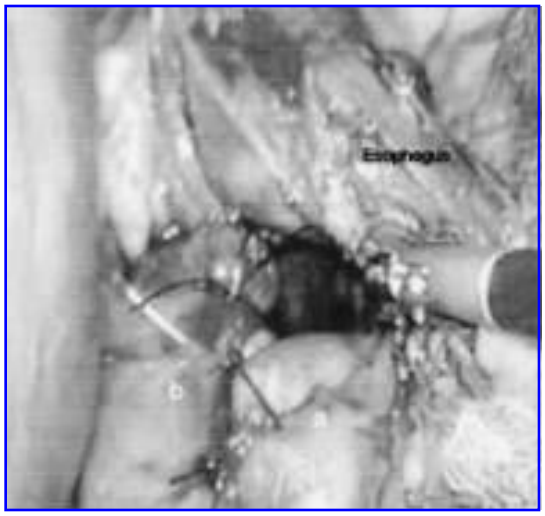

FIG. 4. The gastric fundus (a) is sutured to the anterior edge of the right crura (b), and then the left edge of the myotomy is fixed to the left crura and to the left side of the anterior gastric fundus.

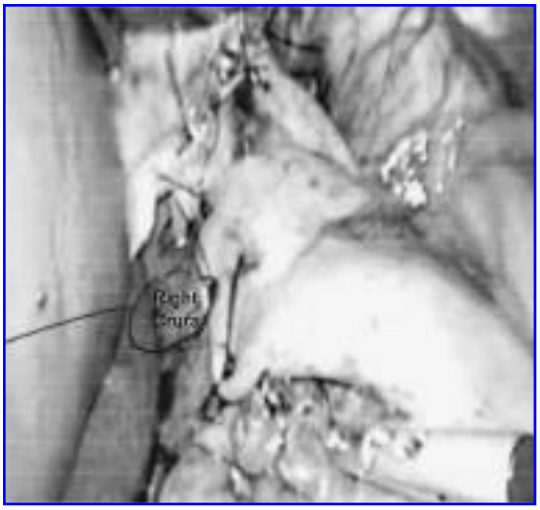

FIG. 5. The distal end of the esophagus is totally covered with the anterior surface of the gastric fundus.

tory response syndrome and sepsis. This patient died. An autopsy could not be performed because of family refusal.

In the rest of the five cases, a UGE was made at 78, 58, 13, 12, and 20 months for patients 1, 2, 3, 4, and 6, respectively. These showed a functional funduplication and data compatible with chronic gastritis, except for patient 1 who presented a grade A esophagitis (Los Angeles classification).[18]

DISCUSSION

Predisposing factors for esophageal perforations during LEM in patients with achalasia include a dilated wall, lack of serosa, and some therapeutic procedures, such as endoscopic dilations and botulinum toxin injection.[2,3] In our study, two patients (cases 4 and 5) had a history of endoscopic dilations and one (case 6) had an open Heller myotomy; nevertheless, all cases had a dense adherence between the muscular esophageal planes.

Perioperative esophagoscopy and methylene blue injection are two recommended diagnostic methods when an esophageal perforation is suspected during LEM.[19] Esophagoscopy was employed for cases 3 and 4, and methylene blue injection was used for case 5. In this patient there was no evidence of intraoperative leakage.

When the esophageal perforation is evident, esophagoscopy allows the characterization of lesions and assures maximal visualization. Likewise, esophagogastric videoendoscopy turns out to be very useful for evaluating the esophageal wall closure and the final anatomical result of the fundoplication.

Several authors have described various therapeutic procedures for dealing with esophageal perforations.[9,20,21] We used a laparoscopic primary closure of the perforation and a fundoplication, avoiding the morbidity associated with the conventional surgical approach (laparotomy or thoracotomy) and keeping the advantages offered by the intra-abdominal fundoplication.[10,11]

In two patients who developed postoperative esophageal leakages, we think that these complications resulted from the gastric volvulus previously described for the patient in case 6, which may have increased the intraesophageal pressure, and from a delayed esophageal perforation caused by an electrocautery burn in case 5.

In spite of esophageal wall scaring and the healing process secondary to the mucosa repair, we observed dys-

TABLE 1. PATIENT DEMOGRAPHICS AND CLINICAL FOLLOW-UP IN PATIENTS UNDERGOING LEM AND PRIMARY ESOPHAGEAL PERFORATION REPAIR

	Preoperative			*Postoperative*			
N/age/sex	*Length of symptoms (years)*	*LES pressure (mm Hg)*	*Follow-up (months)*	*LES pressure (mm Hg)*	*LES total/intra-abdominal length (cm)*	*24-h esophageal pH monitoring*	*Symptoms/weight gain*
1/53/M	8	40.0	78	16.5	3.0/2.8	2.8	None/10 kg
2/29/F	5	31.8	58	7.9	4.0/3.5	4.0	Isolated epigastralgia/5 kg
3/64/F	20	35.0	13	12.0	3.0/2.0	3.0	None/6 kg
4/67/F	16	Not evaluated	12	5.6	2.3/1.3	124.6	Isolated heartburn/15 kg
5/29/F	5	48.1	NA	NA	NA	NA	NA
6/23/F	2	30.3	20	11.7	3.2/1.4	3.5	Mild dysphagia to solids/9 kg

LES, lower esophageal sphincter.

phagia relief and a decrease of the preoperative LES pressure in all patients. Interestingly, the patient in case 4 with a 180-degree fundoplication had an abnormal postoperative intraesophagal pH measurement, in spite of having occasional heartburn (1–2 times/month) and no evident esophageal mucosal injuries at the UGE control. Similarly, heartburn has also been reported in patients with emergency esophageal repair and Dor fundoplication.[10] The abnormal esophageal acid exposure in case 4 may be explained by an asymptomatic, nonerosive, gastroesophageal reflux disease;[22] nevertheless, esophageal aperistalsis favors food retention, which acidifies the esophageal lumen.[23]

In conclusion, we recommend: an adequate esophageal dissection (at least 6–8 cm of the distal esophagus) and a gentle downward esophageal traction that allows intraabdominal location of the injured segment, in order to diminish the morbidity of the intrathoracic esophageal perforation; a complete esophagomyotomy (5 cm above the GEJ and at least 1 cm below it); careful closure of the perforation and an aligned distal esophagus, attempting to diminish the distal pressure that could contribute to fistula development; and regular use of intraesophageal methylene blue, as a simple and low-cost method for the early detection of perforations, or esophagoscopy, when feasible.

Since the fundoplication acts as an antireflux procedure, this protects the repaired zone; in this way, the posterior esophagogastropexy is a 360-degree antireflux procedure with 5 cm of length that provides coverage of the distal end of the esophageal surface and the proximal gastric surface. This fundoplication minimizes the intrathoracic displacement risks and muscular scarring, thanks to the fixation of the gastric fundus to the myotomy edges and the diaphragmatic crura.

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