Chapter 2
Laparoscopic Heller Myotomy and Dor Fundoplication

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2.1 Introduction

Esophageal achalasia affects between 1 and 3 in 100,000 people, with no noticeable difference regarding gender or race. The risk of developing achalasia increases with the age of patients. Interestingly, it appears that the increasing incidence of the disease is probably due to improvements in diagnostic methods.

Under physiological conditions the lower esophageal sphincter (LES) relaxes in response to swallowing. This mechanism is dependent on esophageal and LES neurogenic control through the myenteric plexus, involving excitatory acetylcholine neurons and inhibitory nitric oxide and VIP neurons. Idiopathic achalasia is caused by the degeneration of the myenteric plexus. As a result, esophageal peristalsis is absent, the LES does not relax properly in response to swallowing and it is often hypertensive.

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2.2 Clinical Presentation

Absent peristalsis and decreased relaxation of the LES hinder the passage of a food bolus. Dysphagia occurs in almost every patient. Consequently, difficulties in swallowing often lead to weight loss. Regurgitation of retained food is also common and may result in aspiration with complications such as pneumonia, wheezing, cough and hoarseness. About half of patients with achalasia experience heartburn which is caused by stasis and fermentation of undigested food in the esophagus. Chest discomfort or pain caused by esophageal distention my also occur. The severity of achalasia symptoms is assessed by the Eckardt score [1].

2.3 Preoperative Evaluation

A comprehensive evaluation should be carried out in every patient and should include a symptomatic evaluation, esophagogastroduodenoscopy (EGD), barium swallow, and esophageal manometry.

The Eckardt score is the grading system most frequently used for the evaluation of symptoms and efficacy of treatment [1]. It attributes points (0–3 points) to 4 symptoms of the disease (dysphagia, regurgitation, chest pain, and weight loss), ranging from 0 to 12.

After the symptomatic evaluation, the work-up usually begins with an EGD to exclude other causes of dysphagia such as a peptic stricture or a tumor. Typical findings are esophageal dilation and presence of retained food. Sometimes candidiasis of the esophageal mucosa is present. It is worth mentioning that gastroesophageal junction cancer infiltrating the LES may mimic achalasia. This misleading condition, called pseudo-achalasia, should be ruled out, especially in elderly patients with short duration of symptoms and marked weight loss [2].

The barium swallow often shows the characteristic ‘bird beak’ sign (narrowing at the level of the gastroesophageal junction), delayed passage of the contrast into the stomach, an air-fluid level, and tertiary contractions of the esophagus.

The gold standard for the diagnosis of achalasia is the high-resolution esophageal manometry (HRM). It enables the measurement of the pressure, length and relaxation of the lower and upper esophageal sphincters, and assessment of esophageal peristalsis. To confirm the diagnosis of achalasia, it is necessary to document lack of esophageal peristalsis and partial or absent LES relaxation. The Chicago classification introduced by Pandolfino and his colleagues, distinguishes three types of achalasia [3]. Type I involves aperistalsis and absence of esophageal pressurization; type II is associated with aperistalsis and pan-esophageal pressurization in at least 20% of swallows; and in type III there are premature spastic contractions (distal latency < 4.5 seconds) in at least 20% of swallows. The Chicago classification can also help predicting treatment outcome, as many studies have shown higher success rates in patients with type II achalasia [4, 5]. An ambulatory pH monitoring study is rarely performed, mostly in patients in whom heartburn is present.

2.4 Technique

2.4.1 Position of the Patient

After induction of general endotracheal anesthesia, the patient is positioned supine on the operating table and the lower extremities are extended on stirrups with the knees flexed to 20°. Pneumatic compression stockings are applied to the lower extremities and subcutaneous heparin is administered. The surgeon stands between the patient's legs, with the first and second assistant standing on the right and left side of the table (Fig. 2.1).

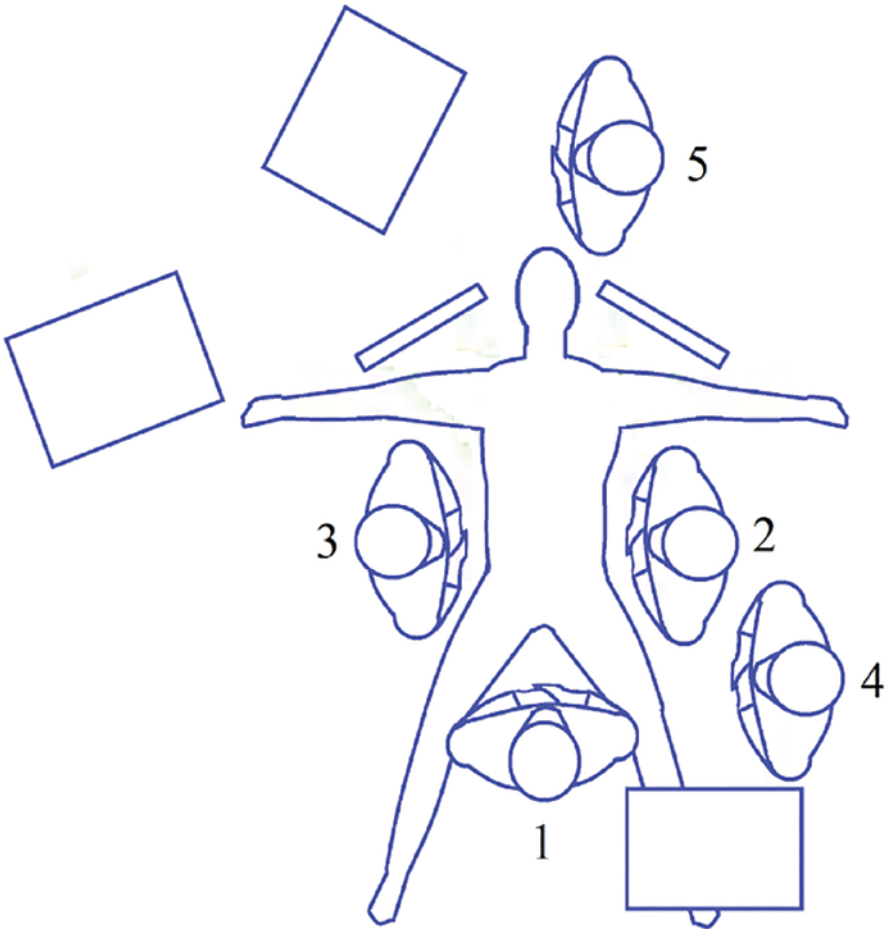


Fig. 2.1 Positions of patient and team: 1—surgeon, 2—first assistant, 3—second assistant, 4—scrub nurse, 5—anesthesiologist

2.4.2 Placement of the Trocars

Five trocars are used for the procedure (Fig. 2.2). The camera port is placed in the midline, 14 cm distal to the xiphoid process. Two additional ports are placed at the same level on the right (for the liver retractor) and left mid-clavicular line (for a bipolar instrument to take down the short gastric vessels and for a Babcock used for traction and exposure). The final two ports are placed below the right and left costal margins, forming a 120° angle, and are used for the dissection, the hook cautery for the myotomy, and suturing for the fundoplication. It is important to make sure that these trocars are not placed too low as this would make the transection of the proximal short gastric vessels and the retraction of the gastroesophageal junction more difficult.

2.4.3 Division of the Gastro-hepatic Ligament and Identification of the Right Crus and Posterior Vagus Nerve

The left segment of the liver is retracted using a laparoscopic retractor to expose the gastroesophageal junction. We begin the dissection of the gastrohepatic ligament above the caudate lobe of the liver and continue proximally until the right crus is

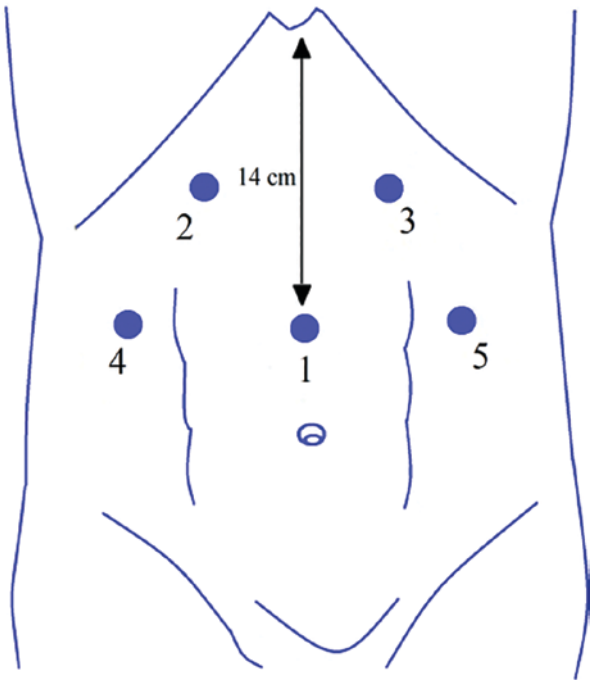


Fig. 2.2 Position of trocars: 1–30° scope camera port, 2—assisting port, 3—dissecting/suturing port, 4—liver retractor port, 5—Babcock clamp port

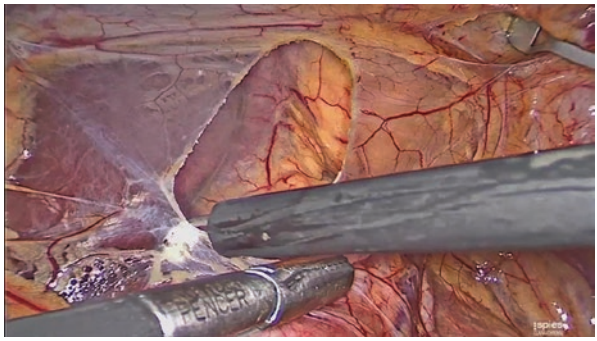


Fig. 2.3 Opening of gastrohepatic ligament

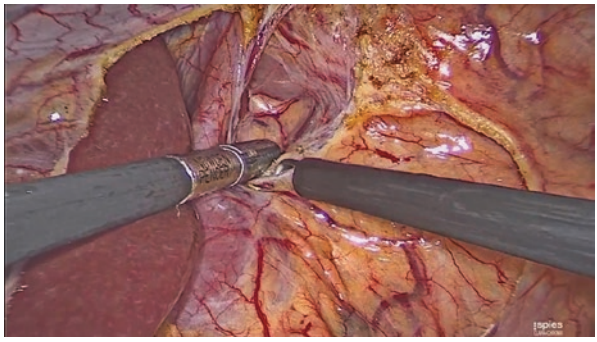


Fig. 2.4 Dissection of right pillar of the crus

identified (Fig. 2.3). The crus is then separated from the esophagus by blunt dissection, and the posterior vagus nerve is identified (Fig. 2.4). We avoid the use of the monopolar cautery during the dissection to prevent injury to the posterior vagus nerve.

2.4.4 Division of Peritoneum and Phreno-esophageal Membrane Above the Esophagus and Identification of the Left Crus of the Diaphragm and Anterior Vagus Nerve

The peritoneum and the phreno-esophageal membrane above the esophagus are divided and the anterior vagus nerve is identified (Fig. 2.5). The left pillar of the crus is separated from the esophagus. Dissection is limited to the anterior and lateral aspects of the esophagus, and no posterior dissection is needed if a Dor fundoplication is planned. Care is given to avoid any injury to the anterior vagus nerve.

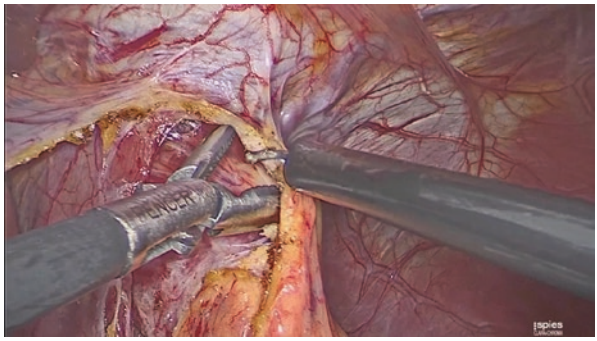


Fig. 2.5 Transection of peritoneum and phreno-esophageal membrane overlying esophagus

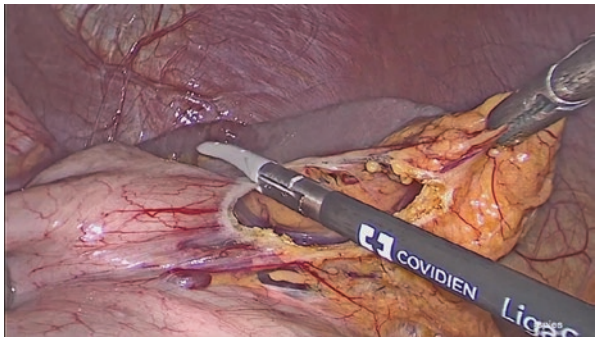


Fig. 2.6 Division of short gastric vessels

2.4.5 Division of the Short Gastric Vessels

The short gastric vessels are divided starting from a point midway along the greater curvature of the stomach all the way to the left pillar of the crus using a bipolar instrument (Fig. 2.6). It is important to avoid too much traction to prevent bleeding from the short gastric vessels or injuring the spleen. In addition, even when using a bipolar instrument, the dissection should be kept about 5 mm away from the gastric wall to avoid electrical damage. The dissection is continued in the posterior mediastinum, lateral and anterior to the esophagus, to expose 6 to 7 cm of the esophagus (Fig. 2.7).

2.4.6 Esophageal Myotomy

The fat pad over the esophageal and gastric wall is removed in order to expose the gastroesophageal junction (Fig. 2.8). A Babcock clamp is then applied below the proximal gastric wall to pull the esophagus downward and to the left in order to expose the right side of the esophagus. We perform the myotomy at the 11 o' clock position using a monopolar electrocautery with a 90° hook as it allows careful

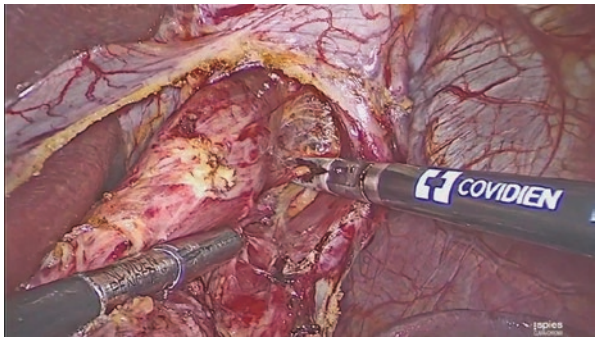


Fig. 2.7 Mediastinal dissection

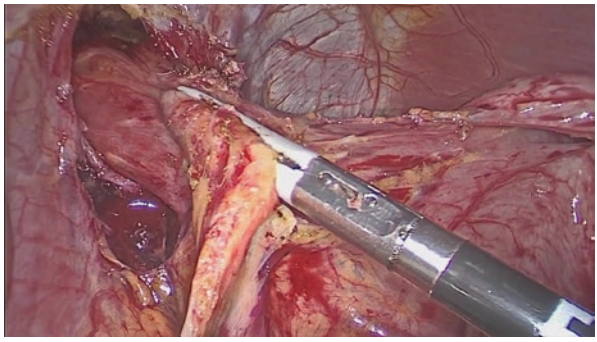


Fig. 2.8 Removal of the fat pad

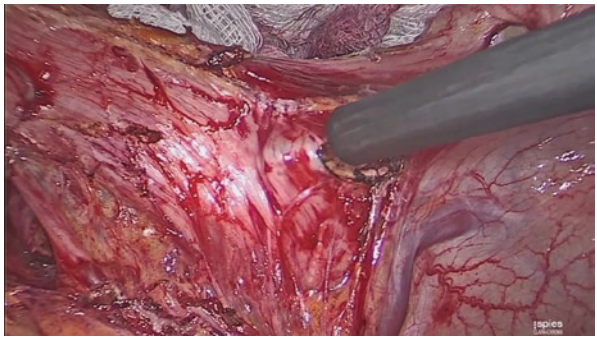


Fig. 2.9 Beginning of myotomy at the gastroesophageal junction

lifting and division of the circular fibers. We usually start the myotomy about 2 cm above the gastroesophageal junction with the goal of reaching the proper submucosal plane (Fig. 2.9). The myotomy is then extended proximally for about 6 cm above the esophago-gastric junction, and distally for about 2.5 cm onto the gastric wall (Figs. 2.10, 2.11, and 2.12). Thus, the total length of the myotomy is typically about 8.5 cm (Fig. 2.13). The edges of the myotomy are then separated so that about 40% of the mucosa is exposed. Sometimes it is quite difficult to identify the proper plane when fibrotic tissue is present due to prior injections of botulinum toxin and/or pneumatic dilatations. If bleeding occurs from the cut muscle fibers, gentle

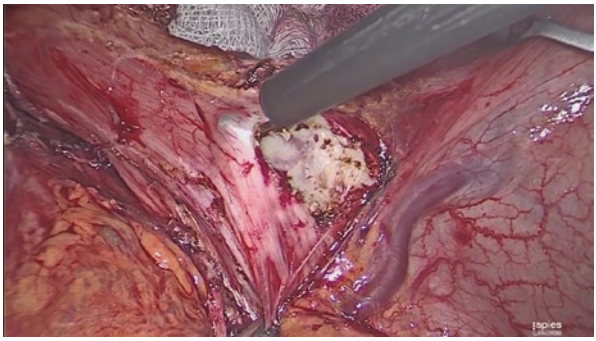
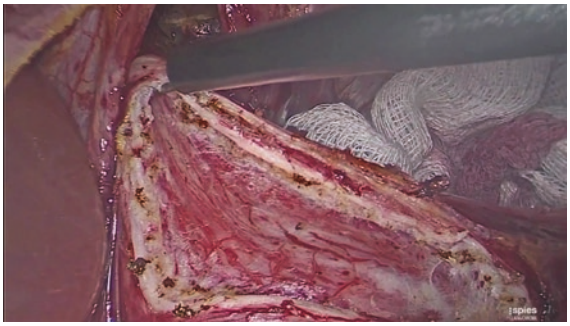


Fig. 2.10 Proximal extension of the myotomy



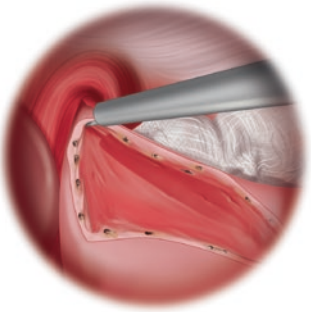


Fig. 2.11 Upper limit of the myotomy

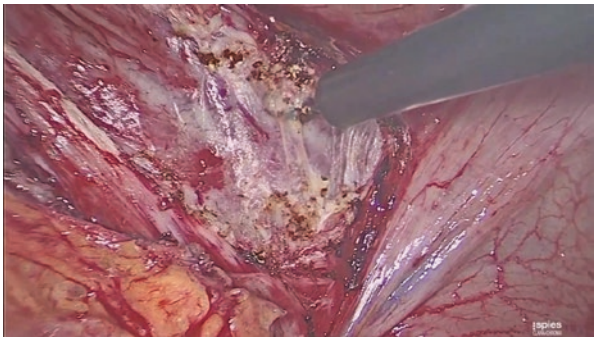


Fig. 2.12 Distal extension of the myotomy onto the gastric wall

compression should be applied avoiding the use of the electrocautery. If a perforation occurs, it is repaired using fine absorbable suture material (4-0 or 5-0).

2.4.7 Dor Fundoplication

The Dor anterior 180° fundoplication has two rows of sutures, one left and one right. The left row comprises three stitches: the uppermost stitch incorporates the fundus of the stomach, the esophageal wall, and the left pillar of the crus; the other two incorporate the stomach and the esophageal wall (Fig. 2.14). The gastric fundus is then folded over the exposed mucosa, so that the greater curvature is next to the right pillar of the crus. The second row of stitches comprises three stitches between the fundus and the right pillar of the crus, and one or two additional stitches between the superior aspect of the fundoplication and the rim of the esophageal hiatus (Fig. 2.15). These last stitches remove any tension from the second row of sutures.

The choice between a Dor fundoplication (180° anterior) and a Toupet fundoplication (220° posterior) is usually based on surgeon's preference. The advantages of

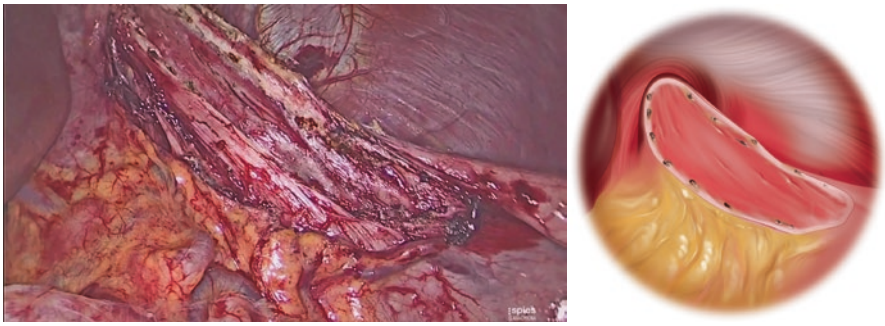


Fig. 2.13 Completed myotomy

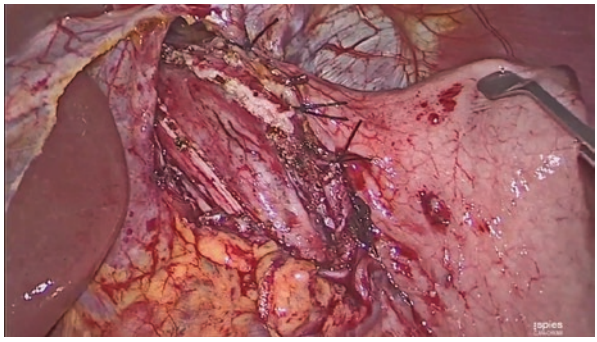


Fig. 2.14 Dor fundoplication: left row of sutures

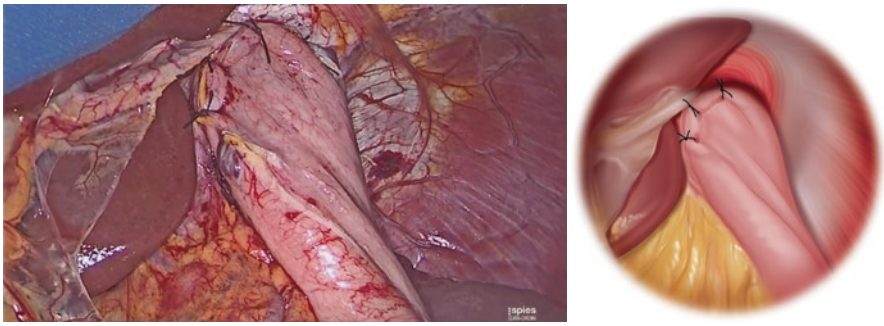


Fig. 2.15 Completed fundoplication with right row and apical sutures

a Dor fundoplication are that it does not require posterior dissection (avoiding a possible injury to the posterior vagus nerve), and that it covers the exposed esophageal mucosa. The advantages of a Toupet fundoplication are that it keeps the edges of the myotomy separated and may provide better reflux control [6].

2.5 Postoperative Course

We do not routinely obtain a contrast study on postoperative day one. This is done only if a mucosal perforation occurs during the myotomy. Otherwise we start with a clear liquid diet for breakfast and advance to a full liquid diet for lunch. Patients are usually discharged on day one. We prescribe oral pain medications for a couple of days, and proton pump inhibitors for 4 weeks. If the patient is asymptomatic, we stop these medications at the second postoperative visit. Endoscopy is recommended every 3 years or in case of persistent or recurrent symptoms.

Overall, we feel that a laparoscopic myotomy with partial fundoplication should be the initial procedure for patients with achalasia, particularly if they are young. While many studies have shown that POEM is at least as effective as a surgical myotomy, it is associated with pathologic reflux in 50% to 60% of patients, therefore repeating the experience of the thoracoscopic myotomy in the 1990s [7, 8].

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