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Treatment of Esophageal Achalasia with Emphasis on Laparoscopic Heller Cardiomyotomy

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Abstract

Background: Achalasia is an esophageal motility disorder characterized by failure of lower esophageal sphincter relaxation and absence or abnormality of esophageal peristalsis. No available treatment can reverse ganglion-cell degeneration, restore lost esophageal neurons, or normalize esophageal motility. Current therapies are therefore directed toward reducing lower esophageal sphincter pressure, relieving functional obstruction, facilitating esophageal emptying, and improving dysphagia and regurgitation.

Objective: To review the treatment options for esophageal achalasia, with particular emphasis on laparoscopic Heller cardiomyotomy, including patient selection, operative principles, the role of partial fundoplication, clinical outcomes, complications, and comparison with pneumatic dilation and peroral endoscopic myotomy.

Conclusion: Laparoscopic Heller myotomy remains a major definitive treatment for esophageal achalasia and provides effective symptomatic relief by dividing the circular muscle fibers of the distal esophagus and proximal stomach while preserving the mucosa. Addition of a partial fundoplication reduces postoperative gastroesophageal reflux. Appropriate patient selection, adequate myotomy, careful mucosal preservation, and structured follow-up are essential for favorable outcomes and early detection of recurrent dysphagia or reflux.

Keywords: Esophageal Achalasia; Laparoscopic Heller myotomy; Cardiomyotomy; Pneumatic dilation; Peroral endoscopic myotomy; Fundoplication.

Treatment of Achalasia

Unfortunately, no treatment can reverse ganglion-cell degeneration, restore lost esophageal neurons, or normalize esophageal motility. Current therapies, whether endoscopic or surgical, address the functional obstruction but do not restore normal esophageal motility. Although symptom control can be durable, treatment efficacy may decline over time, and long-term follow-up with repeated or alternative interventions may become necessary.[1]

Treatment of achalasia is aimed at reducing the resting pressure of the LES to relieve the functional obstruction and facilitate esophageal emptying.[2]

This can be accomplished by pharmacological reduction in LES pressure (e.g., injection of botulinum toxin or use of oral nitrates) or by mechanical disruption of the muscle fibers of the LES (e.g., pneumatic dilation [PD], surgical myotomy, or peroral endoscopic myotomy). For any of the invasive therapies, results are best for patients with type II achalasia.[3]

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Treatment options:

Pharmacological therapy:

Pharmacologic therapy is the least invasive but also the least effective modality for treating achalasia. It is mainly reserved for patients who are poor candidates for definitive treatments such as pneumatic dilation, laparoscopic Heller myotomy (LHM), or peroral endoscopic myotomy (POEM), or for those unwilling to undergo invasive procedures. The main objective of medical therapy is to lower the resting pressure of the LES, thereby facilitating esophageal emptying and improving dysphagia.[4]

The LES tone is primarily mediated by cholinergic excitatory and nitrergic inhibitory neural pathways. Pharmacologic agents act by enhancing smooth muscle relaxation through mechanisms such as nitric oxide release, calcium channel blockade, or cyclic guanosine monophosphate (cGMP) modulation. However, these agents do not correct the underlying neuronal degeneration of the myenteric plexus, providing only transient symptomatic improvement rather than long-term disease modification.[5]

Short-acting nitrates such as isosorbide dinitrate (5–10 mg sublingually) or nitroglycerin (0.3–0.6 mg sublingually) given 10–15 minutes before meals transiently lower LES pressure. These agents act by releasing nitric oxide, which stimulates guanylate cyclase to increase cGMP, causing smooth muscle relaxation. They can reduce LES pressure by up to 60%, providing temporary relief of dysphagia. However, their benefits are short-lived (30–60 minutes), and frequent use is limited by adverse effects such as headache, dizziness, and hypotension.[6]

Calcium channel blockers reduce LES tone by inhibiting L-type calcium channels in smooth muscle cells. Nifedipine, administered in doses of 10–30 mg orally about 30 minutes before meals, can reduce LES pressure by 30–50%. Symptom relief is variable, and side effects such as hypotension, flushing, and peripheral edema often limit long-term compliance. Despite modest benefit, calcium channel blockers remain an option for patients unable to tolerate nitrates or invasive treatments.[4]

Phosphodiesterase-5 (PDE5) inhibitors such as sildenafil (50 mg orally) have been shown to reduce LES pressure by enhancing nitric oxide–mediated smooth muscle relaxation through inhibition of cGMP degradation. Clinical studies demonstrate temporary reductions in LES and residual pressures with improved esophageal clearance, although the effect typically lasts less than 90 minutes. Adverse effects such as headache and flushing may limit their use.[7]

Although pharmacologic therapy offers a non-invasive treatment option, it provides only short-term relief. The need for frequent dosing, limited duration of action, and systemic side effects make it a poor substitute for mechanical or surgical interventions. Consequently, medical therapy is reserved for patients with high surgical risk, those unwilling to undergo intervention, or as temporary palliation until definitive treatment becomes feasible.[6]

Table (1): Pharmacologic Options in Achalasia.[6]

Drug Class	Example	Mechanism	Dose	Effect on LES Pressure	Duration	Limitations
Nitrate	Isosorbide dinitrate	↑ cGMP → smooth muscle relaxation	5–10 mg SL before meals	↓ 60%	30–60 min	Headache, hypotension, tolerance

Calcium channel blocker	Nifedipine	↓ Ca^{2+} influx in smooth muscle	10–30 mg PO before meals	↓ 40%	1–2 h	Hypotension, edema, short effect
PDE5 inhibitor	Sildenafil	Inhibits cGMP breakdown	50 mg PO	↓ 50%	30–90 min	Flushing, headache

Ca^{2+}: Calcium Ion, cGMP: Cyclic Guanosine Monophosphate, h: Hour, LES: Lower Esophageal Sphincter, mg: Milligram, PDE5: Phosphodiesterase Type 5, PO: Per Os (Oral Administration), SL: Sublingual

Botulinum toxin injection:

Botulinum toxin injection (BTI) is a minimally invasive endoscopic therapy for achalasia that acts by producing chemical denervation of the LES. The neurotoxin blocks acetylcholine release from presynaptic cholinergic nerve terminals at the neuromuscular junction, resulting in temporary paralysis of the LES smooth muscle and reduction of sphincter tone. This effect improves esophageal emptying and alleviates dysphagia, albeit transiently.[8]

The technique is performed during upper endoscopy using a sclerotherapy needle to deliver botulinum toxin type A directly into the LES region. A total of 80 to 100 units of botulinum toxin is typically diluted in 4–5 mL of saline and injected into four to five quadrants approximately 1–2 cm above the gastroesophageal junction. The entire procedure usually lasts less than 15 minutes and does not require general anesthesia, making it well tolerated even by elderly or high-risk patients.[9]

The onset of clinical improvement typically occurs within a few days after the procedure. Most patients experience a marked reduction in dysphagia and regurgitation symptoms. Studies report a short-term response rate of approximately 70–90% after the first injection. However, this benefit is temporary, with relapse occurring in about 50% of patients within 6–12 months. Repeated injections can re-establish symptom control, but their effectiveness tends to decrease with subsequent administrations, possibly due to antibody formation or degenerative changes in the myenteric plexus.[10]

Despite its efficacy in symptom relief, BTI does not modify the underlying pathophysiology of achalasia and therefore cannot halt disease progression. This limitation underscores its role as a palliative measure rather than a curative treatment.[4]

In terms of safety, botulinum toxin injection is generally well tolerated. The most common adverse effects are mild chest discomfort and transient heartburn. Rare complications include minor bleeding, mediastinitis, and allergic reactions. The incidence of gastroesophageal reflux disease (GERD) post-injection is lower compared to that observed after surgical or endoscopic myotomy, due to the partial and temporary relaxation of the LES.[11]

Botulinum toxin therapy is mainly indicated in patients who are poor candidates for surgery or endoscopic myotomy, such as those with advanced age, severe comorbidities, or limited life expectancy. It is also appropriate for patients who decline invasive procedures or in whom pneumatic dilation or POEM has failed. In some cases, BTI can be used as a bridging therapy to relieve symptoms before a definitive procedure is performed.[12]

The cost of botulinum toxin (BTX) injection for the treatment of esophageal achalasia is generally lower than that of definitive interventions such as laparoscopic Heller myotomy (LHM) or peroral endoscopic myotomy (POEM) because it is performed as an outpatient endoscopic procedure with minimal recovery time. However, its economic advantage is often offset by the need for repeated injections due to the transient nature of symptom relief. Studies evaluating the cost-effectiveness of achalasia therapies have demonstrated that although the initial procedural cost of BTX injection is relatively low, repeated treatments increase cumulative healthcare expenditures over time.

Zaninotto et al. reported that the initial direct cost of BTX therapy was approximately €1,245 compared with €3,555 for laparoscopic myotomy; however, when long-term effectiveness was considered, the difference in cost-effectiveness narrowed substantially because of the high recurrence rate after BTX treatment.[13] Similarly, Panaccione et al. found that a botulinum toxin strategy was more costly than pneumatic dilation ($5,033 versus $3,608).[14] Furthermore, O'Connor et al. concluded that pneumatic dilation was the most cost-effective strategy over a five-year period.[15] Therefore, while botulinum toxin injection remains an attractive option for elderly patients and those unfit for surgery, the need for repeated administration may increase cumulative costs and reduce its cost-effectiveness compared with more durable treatment modalities.

Pneumatic dilation:

Pneumatic dilation (PD) is one of the most established non-surgical treatments for achalasia, designed to disrupt the muscle fibers of the LES and improve esophageal emptying. The procedure achieves this by forcibly stretching and tearing the circular muscle layer of the LES using a non-compliant balloon under endoscopic and fluoroscopic guidance.[16]

The dilation is typically performed using the Rigiflex balloon system, which consists of polyethylene balloons available in diameters of 30 mm, 35 mm, and 40 mm.

The balloon is positioned across the LES under fluoroscopic control and inflated rapidly using a pneumatic pressure system until the waist of the balloon disappears, indicating disruption of the LES fibers. Inflation is maintained for 15–60 seconds before deflation and withdrawal of the catheter.[17]

Pneumatic dilation is usually performed in a graded manner, starting with a 30-mm balloon and progressing to larger diameters at intervals of 2–4 weeks, depending on clinical response and tolerance. This graded approach reduces the risk of perforation while optimizing long-term efficacy. The procedure may be repeated if symptoms recur, and the need for repeat dilations has been shown to correlate with disease subtype and patient age.[18]

Clinical success, defined by symptomatic improvement and reduced esophageal retention on timed barium swallow, is achieved in approximately 70–85% of patients after a single or multiple dilation sessions. Long-term follow-up data demonstrate that symptom relief can last 5–10 years in many patients, although relapses may occur and are often managed by repeat dilation. Type II achalasia tends to respond best to PD, while types I and III exhibit less favorable outcomes.[19]

The most serious complication of pneumatic dilation is esophageal perforation, which occurs in approximately 2–5% of cases. Early recognition and prompt management are crucial, with most cases amenable to conservative treatment or endoscopic clipping if identified early. Minor complications such as chest pain, transient fever, and gastroesophageal reflux disease (GERD) are relatively common but generally self-limiting.[20]

Pneumatic dilation is often compared to laparoscopic Heller myotomy (LHM) and peroral endoscopic myotomy (POEM) in terms of efficacy and long-term outcomes. Randomized trials and meta-analyses indicate that while PD provides slightly lower long-term success than myotomy, it offers a less invasive and repeatable alternative with similar short-term results. The choice between PD and myotomy is usually individualized based on patient factors, achalasia subtype, and local expertise.[21]

Over four to six years, nearly one-third of patients experience symptom relapse and require retreatment.[22]

Surgical myotomy:

Laparoscopic Heller myotomy (LHM) is currently considered the surgical gold standard for the definitive management of esophageal achalasia. It aims to relieve functional obstruction at the LES by dividing the circular muscle fibers of the distal esophagus and proximal stomach while preserving the mucosa. This restores esophageal emptying and improves dysphagia and regurgitation symptoms. The laparoscopic approach has

largely replaced the open technique due to lower morbidity, shorter hospital stay, and superior visualization of the gastroesophageal junction.[23]

Surgical myotomy was first described in 1913 by Ernest Heller, who performed both anterior and posterior incisions at the gastroesophageal junction. The operation was subsequently modified to a single anterior myotomy.[24]

LHM is indicated for all symptomatic patients with primary achalasia who are fit for general anesthesia and surgical intervention. It is particularly preferred in younger patients (<40 years) and those with types I and II achalasia, as these subtypes respond well to myotomy. In type III (spastic) achalasia, the need for a longer proximal myotomy may reduce LHM efficacy, and POEM may be favored. LHM is also indicated in cases of failed pneumatic dilatation or botulinum toxin therapy. Contraindications include severe comorbidities, morbid obesity, and distorted esophageal anatomy (sigmoid esophagus), where esophagectomy might be considered instead.[25]

All candidates for LHM undergo a comprehensive preoperative evaluation, including upper endoscopy to exclude pseudoachalasia, high-resolution manometry (HRM) to confirm diagnosis and subtype, and a barium esophagogram to assess esophageal diameter and emptying. Cardiac and pulmonary optimization is essential given that laparoscopy involves pneumoperitoneum and Trendelenburg positioning.[26]

The standard myotomy length is 6 cm on the esophagus and 2–3 cm on the gastric cardia. In cases of type III achalasia, the myotomy may need to extend proximally up to 10–12 cm to relieve esophageal spasm. The distal extension onto the stomach is critical, as an incomplete gastric component may lead to persistent dysphagia. Conversely, excessive gastric extension increases the risk of postoperative reflux. Adequate hemostasis should be maintained throughout to avoid obscuring the dissection plane. The procedure is typically completed with a partial fundoplication to prevent reflux.[27]

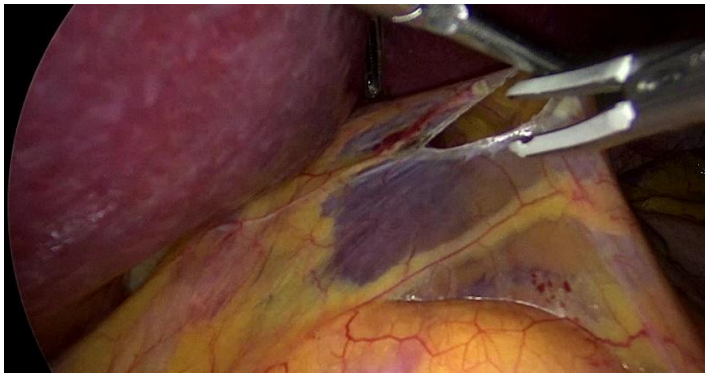

Figure (1): Opening of the gastrohepatic ligament.

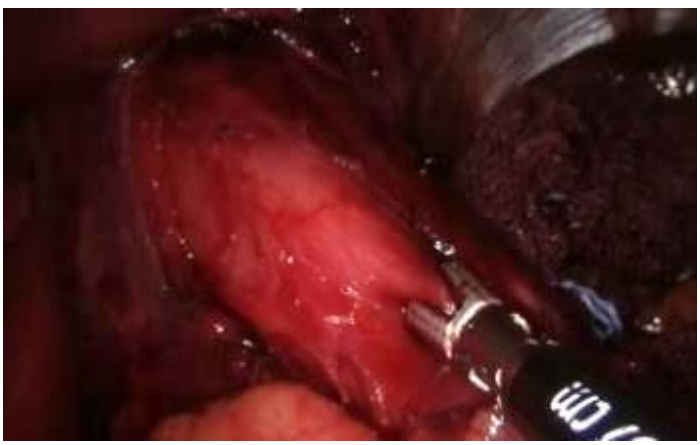

Figure (2): Division of the lower esophageal sphincter muscle fibers.

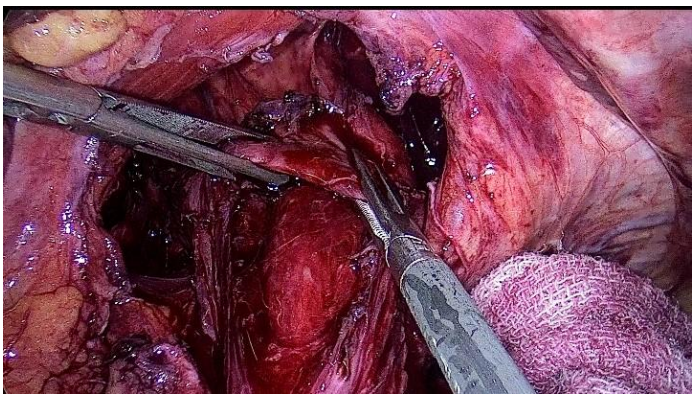

Figure (3): Mucosal bulge after myotomy.

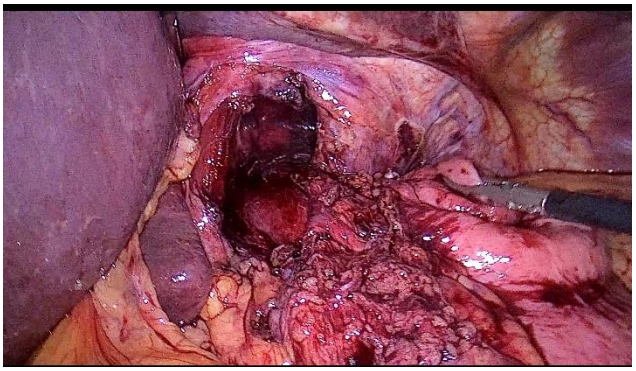

Figure (4): Endoscopic assessment of the myotomy.

The Dor fundoplication (anterior 180° wrap) is most commonly performed due to its technical simplicity and ability to cover the exposed mucosa, minimizing risk of perforation. The Toupet fundoplication (posterior 270° wrap) is also widely used and provides comparable reflux control while preserving physiological swallowing. Randomized studies have shown that both Dor and Toupet fundoplications achieve similar efficacy in reflux prevention and dysphagia relief. The addition of any partial fundoplication reduces reflux incidence from 30–40% to under 10% compared to myotomy alone.[28]

Intraoperative mucosal perforation remains the most common complication, occurring in approximately 5–10% of cases. When recognized promptly, it can be repaired laparoscopically with interrupted absorbable sutures and covered by the Dor fundoplication. Other intraoperative risks include bleeding, vagal nerve injury, and pneumothorax due to hiatal dissection. Postoperatively, transient dysphagia, reflux, and subcutaneous emphysema are possible but generally self-limiting. Routine postoperative contrast swallow studies are often performed to exclude leak before resuming oral intake.[27]

LHM achieves excellent long-term results, with 85–90% of patients reporting sustained symptomatic improvement at 5–10 years of follow-up. Eckardt scores typically drop from 7–9 preoperatively to <3 postoperatively. Manometric studies confirm normalization of LES pressure, and esophageal emptying improves significantly on timed barium swallow. Long-term follow-up is essential, as late recurrence of dysphagia may occur in up to 20% of cases due to scarring or incomplete myotomy.[29]

Long-term durability of LHM is excellent, with sustained improvement in esophageal emptying and swallowing function. The main predictors of failure are incomplete gastric myotomy, fibrosis, or twisted fundoplication. Secondary interventions such as pneumatic dilation, POEM, or redo myotomy may be required in 10–15% of cases.[23]

The advantages of surgical myotomy are high initial success rates and, compared with pneumatic dilation, lower rates of symptom recurrence. The main disadvantages of surgery are the high initial cost, the prolonged recovery period, and the frequent development of gastroesophageal reflux disease postoperatively, especially if a fundoplication is not part of the procedure. Thus, the primary goal of the operative treatment is to relieve the functional obstruction of the LES while preventing reflux.[30]

An alternative to laparoscopic surgical myotomy is robotic-assisted minimally invasive surgery. Theoretical advantages for the surgeon over standard laparoscopic surgery include increased range of motion, decreased tremors, improved visibility with respect to depth perception, and improved ability to perform finer movements in a smaller, confined space.[31]

Peroral endoscopic myotomy (POEM):

Peroral endoscopic myotomy (POEM) represents a major advancement in the management of esophageal achalasia. Introduced in 2010 by Inoue and his colleagues, POEM applies natural orifice transluminal endoscopic surgery (NOTES) principles to achieve a myotomy of the LES without external incisions.[32]

The technique provides a minimally invasive alternative to conventional laparoscopic Heller myotomy (LHM) and pneumatic dilation, offering the advantage of precise submucosal dissection and the ability to tailor the myotomy length according to the manometric subtype of achalasia.[33]

The underlying concept of POEM is to relieve functional obstruction at the LES by performing selective division of the circular muscle fibers of the distal esophagus and proximal stomach, thereby restoring esophageal emptying and reducing dysphagia. The procedure is conducted entirely through the endoscope using a submucosal tunnel approach, which allows direct visualization of the muscular layer while preserving the mucosa. This approach minimizes trauma, postoperative pain, and recovery time while maintaining high efficacy.[34]

Prior to POEM, a detailed preoperative assessment is mandatory. High-resolution manometry (HRM) confirms the diagnosis and determines the achalasia subtype according to the Chicago Classification v4.0. Timed barium esophagogram evaluates esophageal diameter, emptying, and sigmoid deformity. Upper endoscopy rules out pseudoachalasia due to malignancy or stricture. Cross-sectional imaging (CT or EUS) may be indicated when secondary causes are suspected. Patients should be counseled about the potential risk of postoperative gastroesophageal reflux disease (GERD), which is higher after POEM compared to surgical myotomy.[26]

POEM is performed under general anesthesia with endotracheal intubation. The patient is positioned supine, and carbon dioxide is used for insufflation to minimize mediastinal emphysema.

1. Mucosal Entry:

A 2-cm longitudinal mucosal incision is made 10–12 cm above the gastroesophageal junction using an endoscopic knife.

2. Submucosal Tunnel Creation:

Through this entry, the endoscope is advanced into the submucosal space, and a tunnel is created by injecting saline mixed with methylene blue to separate the mucosa from the muscularis propria. The tunnel is extended 2–3 cm beyond the LES into the gastric cardia.

3. Myotomy:

The circular muscle fibers are divided using an electrosurgical knife, while the longitudinal muscle layer is preserved. In type III (spastic) achalasia, the myotomy is extended proximally up to 10–12 cm.

4. Closure:

The mucosal entry is closed with multiple endoscopic clips to prevent leakage.

The procedure typically lasts 60–90 minutes, and intraoperative endoscopy is used to confirm adequacy of the myotomy.[34]

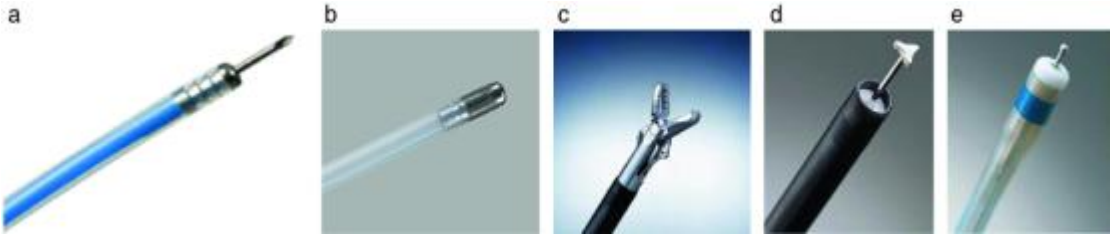

Figure (5): Endoscopic material used for peroral endoscopic myotomy (POEM): (A) injection needle to create a submucosal bleb; (B) spray catheter to apply methylene blue and identify the submucosal tissue plane; (C) Coagrasper hemostatic forceps (Olympus) for monopolar coagulation in cases of bleeding; (D) Triangle Tip knife (Olympus) for myotomy; and (E) DualKnife (ERBE) for myotomy.[35]

Figure (6): Equipment: (A) oblique cap placed and oriented to 6 o'clock position on the scope; (B) standard ERBE (Erbe Elektromedizin GmbH) machine setting for peroral endoscopic myotomy procedure.[36]

POEM is indicated for all types of primary achalasia, particularly type III (spastic) achalasia, where the myotomy can be extended proximally to relieve esophageal spasms. It is also recommended for patients with recurrent or persistent symptoms following pneumatic dilation or Heller myotomy. Other indications include esophagogastric junction outflow obstruction and spastic motility disorders such as diffuse esophageal spasm or jackhammer esophagus.[37]

Absolute contraindications include severe erosive esophagitis, portal hypertension with varices, and prior esophageal radiation or endoscopic mucosal resection that causes submucosal fibrosis, making tunneling hazardous. Relative contraindications are advanced sigmoid achalasia, coagulopathy, and inability to tolerate general anesthesia.[37]

Outcomes and Efficacy:

POEM has shown excellent short- and medium-term results, with clinical success (Eckardt score ≤ 3) in 90–95% of patients at 2–5 years follow-up. LES pressure decreases from a mean of 35 mmHg preoperatively to less than 15 mmHg postoperatively. Timed barium swallow demonstrates markedly improved emptying. These outcomes are comparable to, or better than, those achieved with laparoscopic Heller myotomy.[38]

Recent studies also confirm favorable outcomes in type III achalasia and post-myotomy recurrence, conditions traditionally difficult to manage. The flexibility of POEM in tailoring the myotomy length contributes to superior results in spastic disorders.[39]

Adverse events occur in about 3–5% of cases. Intraoperative complications include mucosal perforation, bleeding, and pneumothorax, which are generally managed conservatively or endoscopically. Delayed

complications mainly involve GERD, observed in 30–50% of patients on objective pH testing. Most cases are asymptomatic or controlled with PPIs. Endoscopic surveillance may reveal erosive esophagitis, particularly in those not adhering to acid suppression therapy.[40]

When compared with LHM, POEM provides equivalent symptom relief and lower postoperative pain, but a higher incidence of GERD due to the absence of an antireflux mechanism. The choice between POEM and LHM often depends on patient factors, achalasia subtype, and surgeon or endoscopist expertise.[29]

POEM offers several distinct advantages:

- No external incisions and minimal postoperative discomfort.
- Adjustable myotomy length tailored to disease subtype.
- Applicability in previously treated or failed surgical cases.
- Short hospitalization and rapid recovery.

These benefits make POEM a highly versatile therapeutic option, especially in centers with advanced endoscopic expertise.[33]

Despite promising results, POEM has limitations. Long-term (>10 years) efficacy data are limited, and the true incidence of post-POEM GERD and its long-term sequelae remain under investigation. The learning curve is steep, and procedural outcomes are significantly operator-dependent. Furthermore, the absence of fundoplication makes reflux management a persistent concern in follow-up.[39]

Choice of treatment:

For patients with an average surgical risk, the primary therapeutic options for achalasia include pneumatic dilation, laparoscopic Heller myotomy with partial fundoplication, and peroral endoscopic myotomy (POEM). These modalities demonstrate comparable efficacy in relieving symptoms and maintaining favorable long-term outcomes.[26]

In patients who are poor surgical candidates or those who decline invasive procedures such as POEM or laparoscopic myotomy, endoscopic injection of botulinum toxin may be considered. This approach offers temporary improvement in swallowing and symptomatic relief, though its effects are generally short-lived.[41]

For patients unable or unwilling to undergo surgery, pneumatic dilation, or POEM, and who have not responded to botulinum toxin therapy, pharmacologic treatment using nitrates or calcium channel blockers may be employed. These medications act by inducing transient relaxation of the lower esophageal sphincter, providing partial and temporary symptom improvement.[41]

Conclusion

Current evidence supports pneumatic dilation, laparoscopic Heller myotomy with partial fundoplication, and peroral endoscopic myotomy as effective definitive therapies for esophageal achalasia. Laparoscopic Heller myotomy remains an established and durable treatment that provides substantial relief of dysphagia and regurgitation when an adequate esophageal and gastric myotomy is performed. The addition of a partial fundoplication reduces postoperative gastroesophageal reflux without compromising swallowing. Treatment selection should be individualized according to achalasia subtype, patient characteristics, previous interventions, local expertise, and patient preference. Long-term comparative studies remain important, particularly to clarify the durability of POEM and the clinical consequences of post-procedural reflux.

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