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Original Research Article

Impact of esophageal myotomy length during laparoscopic Heller myotomy for achalasia type II

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ABSTRACT

Background: Type II achalasia (Ach2) is the most prevalent esophageal motility disorder. Current guidelines recommend laparoscopic Heller myotomy (LHM) as a standard treatment, but the optimal esophageal myotomy length remains under investigation.
Methods: A retrospective cohort study was conducted from 2018 to 2023 at a tertiary care center. Consecutive Ach2 patients undergoing LHM were included. Patients were grouped based on myotomy length (short vs long). Outcomes assessed included operative time, adverse events, Eckardt scores at 6 and 12 months, and treatment success.
Results: Fifty-one patients were analyzed. Eckardt score at 6 months was significantly lower in the long myotomy group compared to the short group (1.2±1.1 vs 0.6±1.2, p=0.023). At 12 months, Eckardt scores were also significantly better in the long myotomy group (1.8±1.7 vs 0.9±1.2, p=0.033). Treatment success at 12 months was 92% in the short group and 100% in the long group, but it was not statistically significant (p=0.191).
Conclusions: LHM provides high clinical success in Ach2 patients. While treatment success at 12 months was similar between groups, longer myotomy was associated with significantly lower Eckardt scores at both 6 and 12 months. These findings suggest a potential benefit in symptom control with longer myotomy, supporting further prospective studies to determine its clinical utility.

Keywords: Achalasia, Heller myotomy, Esophageal surgery, Myotomy length, Eckardt score, Esophageal motility disorder

INTRODUCTION

Achalasia is a chronic, idiopathic esophageal motility disorder resulting from the degeneration of inhibitory neurons in the myenteric (Auerbach's) plexus, which leads to failure of the lower esophageal sphincter (LES) to relax and absence of coordinated peristalsis in the esophageal body.[1,2] The condition affects between 1.8 to 27.1 individuals per 100,000 annually, with similar incidence rates across populations, and is most commonly diagnosed in patients aged 30 to 60 years.[3]

The pathogenesis of achalasia is multifactorial and not fully understood.[4] Several mechanisms have been proposed, including autoimmune responses, viral infections, and genetic predispositions. For instance, infection with herpes simplex virus type 1 (HSV-1) has been linked to oligoclonal T-cell responses, while chagas disease, caused by *Trypanosoma cruzi*, mimics idiopathic achalasia through complete neuronal destruction.[5,6] Genetic conditions such as allgrove syndrome (Triple-A syndrome) also support a hereditary component in some cases.[7] Autoimmune involvement is suggested by the

presence of circulating antibodies against myenteric neurons and associations with specific HLA subtypes.[8]

Histopathologically, the disease shows selective loss of inhibitory neurons producing nitric oxide (NO) and vasoactive intestinal peptide (VIP), both critical for LES relaxation.[1] The imbalance between excitatory acetylcholine and diminished inhibitory input results in hypertonic LES and aperistalsis. Interestingly, eosinophilic infiltration and mast cell degranulation have also been observed in biopsy specimens, indicating immune-mediated neurodegeneration.[9,10]

Achalasia presents clinically with dysphagia, regurgitation, chest pain, and weight loss. Dysphagia typically progresses from solids to liquids. Regurgitation of undigested food, retrosternal pain, and misdiagnosis as gastroesophageal reflux disease (GERD) are also common.2 The Eckardt score, which assesses these symptoms along with weight loss, is a validated tool for determining disease severity and monitoring treatment efficacy. A score of ≤3 is typically considered a marker of clinical success.[11]

Three subtypes of achalasia are identified by high-resolution manometry (HRM) using the Chicago Classification v4.0.[12] Ach2 is characterized by panesophageal pressurization and has the highest response rate to therapy.[3,13] As such, it is often the focus of surgical outcome research. Standard therapeutic options include pneumatic dilation, botulinum toxin injection, POEM, and LHM, with the latter two preferred for their durable symptom control.[2,14]

Although LHM is widely adopted, optimal surgical technique, particularly the ideal length of esophageal myotomy, remains under investigation. Studies comparing long versus short myotomy lengths in POEM have yielded comparable success rates, though data specific to LHM and its variations are limited.[15] Given the impact of surgical approach on outcomes such as postoperative reflux and symptom recurrence, especially in Ach2, more evidence is needed to refine procedural standards.[16,17]

This study aims to evaluate the clinical impact of esophageal myotomy length in patients with Ach2 undergoing LHM, focusing on symptom resolution and treatment success at 6- and 12-month follow-up.

METHODS

This was a retrospective, single-center study conducted at a tertiary care facility (Instituto Mexicano del Seguro Social, Unidad Médica de Alta Especialidad No. 25) in Monterrey, Mexico, from January 2018 to December 2023. Patients diagnosed with type II achalasia using Chicago classification v4.0 on HRM, who underwent laparoscopic Heller's myotomy (LHM) were included in the study. The exclusion criteria were age <18 years, type I/III achalasia, or other motility disorders.

Surgical technique

All procedures were performed by a single experienced surgeon. The length of the esophageal myotomy (short ≤3 cm or long ≥6 cm) was determined randomly and measured intraoperatively using a 10 cm Nelaton catheter previously cut for measurement guidance (Figure 1). The gastric myotomy length was standardized and similar across all cases (2-4 cm). Fundoplication (Dor or Toupet) was performed in a non-randomized, alternating manner without considering the assigned myotomy length. After the procedure, all the patients were kept nil per oral for 24-hours and a timed barium esophagram (TBE) was performed on the next day.

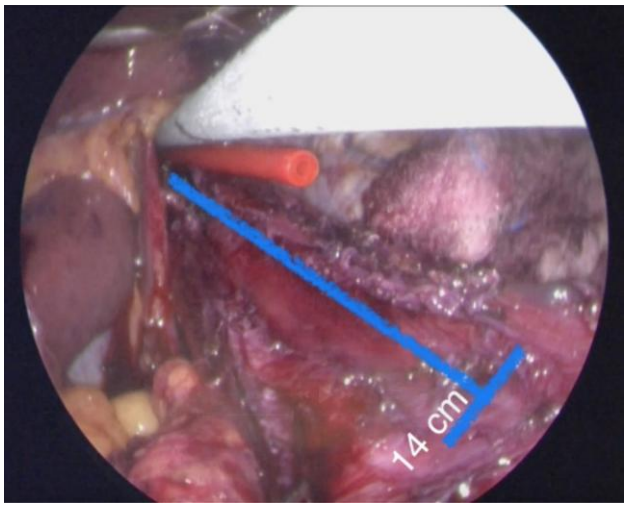

Figure 1: Esophageal myotomy measurement.
Measurement of myotomy length using a nelaton catheter (orange tube).

Follow-up

Patients were evaluated at 1, 3, 6, and 12 months after the LHM procedure. Assessment of clinical efficacy was performed using the Eckardt score.

Outcomes

The primary aim of the study was comparison of clinical success (Eckardt ≤3) of LHM procedure using short versus long esophageal myotomy techniques at 1-year follow-up. The secondary outcome measures included difference in operating times, intraoperative adverse events and reduction in Eckardt score at 6 and 12 months.

Analysis

Data expressed as mean±SD for continuous variables; % for categorical variables. Tests were-unpaired t test, Mann-Whitney U test, and Fisher's exact test. Significance set at $p<0.05$. Analysis used SPSS v20.0.

All procedures were conducted in accordance with the ethical standards of the institutional and national research committee. Ethical approval for this study was obtained from the appropriate institutional ethics committee prior to data collection.

RESULTS

A total of 51 patients with Ach2 who underwent LHM were analyzed and divided into 2 groups based on esophageal myotomy length: standard (5.7±0.5 cm, n=25) and long (7.4±0.8 cm, n=26). No significant differences in baseline characteristics between groups in terms of age, sex distribution/preop Eckardt score (Table 1).

Surgical duration was similar between groups (150±46.4 min vs 143.6±36.7 min, p=0.596), as was gastric myotomy length (3±0.6 cm vs 3±0.3 cm, p=0.805). No significant differences were observed in intraoperative mucosal injuries (16% vs 11.5%, p=0.703) (Table 2).

Table 1: Baseline characteristics of patients in standard and long myotomy groups.

Baseline characteristics	Standard myotomy	Long myotomy	P value
No. of patients	25	26	
Age (in years)	50.7±15.2	49.1±16.9	0.730
Sex			
Males	11 (44)	8 (30.8)	0.393
Females	14 (56)	18 (69.2)	
Eckardt score before surgery	10.4±1.8	9.3±2.4	0.154

*Data presented as mean±SD or n (%). SD-standard deviation.

Table 2: Peri-operative outcomes in both comparison groups.

Procedure characteristics	Std group, (n=25)	Long group, (n=26)	P value
Duration of surgery (in min)	150± 46.4	143.6± 36.7	0.596
Length of esophageal myotomy (in cm)	5.7±0.5	7.4±0.8	0.000
Length of gastric myotomy (in cm)	3±0.6	3±0.3	0.805
Funduplicature type, N (%)			
Dor	22 (88)	14 (53.8)	0.013
Toupet	3 (12)	3 (46.2)	
Mucosal injuries	4 (16)	3 (11.5)	0.703

*Data presented as mean±SD or n (%). SD-standard deviation.

Clinical outcomes favored the long myotomy group. Eckardt scores at 6 and 12 months were significantly lower (1.2±1.1 vs 0.6±1.2, p=0.023; and 1.8±1.7 vs 0.9±1.2, p=0.033, respectively). However, no significant differences were observed in clinical success rates at 12 months (92% vs 100%, p=0.191) (Table 3).

Table 3: Comparison of clinical success between short and long myotomy groups.

Success parameters	Standard myotomy, (n=25)	Long myotomy (n=26)	P value
Eckardt score at 1 month	1±1.1	0.6±0.8	0.076
Eckardt score at 6 months	1.2±1.1	0.6±1.2	0.023
Eckardt score at 12 months	1.8±1.7	0.9±1.2	0.033
Treatment success at 12 months	23 (92)	25 (100)	0.191

*Data are presented as mean±SD or n (%). SD-standard deviation.

DISCUSSION

This study aimed to determine whether the length of esophageal myotomy during LHM in patients with Ach2 has a meaningful impact on clinical outcomes. The findings demonstrated that although treatment success at 12 months was high in both groups and did not significantly differ, patients who underwent a longer esophageal myotomy reported significantly lower Eckardt scores at both 6 and 12 months. These outcomes support the notion that symptom burden may be more effectively reduced with a longer myotomy.

Our results align with those observed in studies evaluating myotomy length in peroral endoscopic myotomy (POEM), which also report similar clinical success between long and short myotomies, though symptom improvement trends have varied.[15,18,19] Unlike POEM, LHM inherently includes an antireflux procedure such as fundoplication, potentially mitigating one of the main concerns associated with longer myotomies-postoperative gastroesophageal reflux disease (GERD).[14,16]

In the present cohort, extending the esophageal myotomy length did not result in a higher rate of intraoperative complications or prolonged operative time, suggesting that the procedure is technically feasible and safe when performed by an experienced surgeon. However, given the variability in patient anatomy, disease chronicity, and esophageal compliance, tailoring the myotomy length may be beneficial in optimizing symptom control.

The non-randomized nature of fundoplication type, the modest sample size, and the single-center, retrospective design represent limitations that should be addressed in future prospective, multicenter investigations. In particular, standardized criteria for myotomy length assignment and fundoplication choice are needed to minimize potential confounding effects.

CONCLUSION

This study demonstrates that extending the length of esophageal myotomy during LHM in patients with Ach2 is associated with improved symptom control, as evidenced by significantly lower Eckardt scores at 6 and 12 months postoperatively. Although overall treatment success rates were comparable between short and long myotomy groups, the enhanced symptomatic relief observed with a longer myotomy suggests a potential clinical advantage. These findings support the notion that tailoring myotomy length may improve patient outcomes without increasing surgical risk. Future prospective, multicenter studies are warranted to validate these results and further refine the surgical approach to optimize care in Ach2.

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