Laparoscopic Management of Esophageal Achalasia: A Prospective Study Evaluating Laparoscopic Heller Myotomy without Dor Fundoplication

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ABSTRACT

Background*: Evaluation of laparoscopic Heller myotomy(LHM) without fundoplication in the treatment of esophageal achalasia , and assessment of its safety and effectiveness on the short and long-term results.* ***Patients and Methods****: In this prospective study, 21 patients were included in the study were reviewed including patient demography, clinical manifestations, preoperative investigations, operative procedure, intraoperative, postoperative complications, and postoperative follow up. AII patients enrolled in the study underwent laparoscopic Heller myotomy without Dor fundoplication. Mean age was 42 years, average operative time was (70-110) minutes, no patient was lost to follow up period. All patients were operated on in our surgery department.* ***Results:*** *21 patients in our study were diagnosed to have achalasia by means of patient symptomatology, full investigatory methods in the form of upper GI endoscopy, esophageal manometry, 24-h PH monitoring, upper contrast series.(Barium swallow). All the 21 patients underwent laparoscopic Heller myotomy without Dor fundoplication, 2 female patients had esophageal perforations, one of them after pneumatic dilation , the other during laparoscopic myotomy. Both patients had intraoperative suture repair during laparoscopic myotomy. All patients had satisfactory postoperative course with no recurrence of dysphagia, and no evidence of gastroesophageal reflux.19 patients were discharged on the 2nd and 3rd postoperative days, and the 2 patients who presented with esophageal tear were discharged after 1week.* ***Conclusion****: Laparoscopic Heller myotomy without fundoplication seems to be an effective surgical technique as compared to laparoscopic Heller myotomy with Dor fundoplication , with similar advantages, and efficacy. It provides both short and long-term symptomatic relief, with short hospital stay, and less complication rate.*
Key words*: Laparoscopic Heller myotomy, achalasia, fundoplication.*

INTRODUCTION

Achalasia is a primary esophageal motor disorder of unknown etiology, characterized manometrically by insufficient relaxation of lower esophageal sphincter (LES) (figure 1), and loss of esophageal peristalsis, radiographically; by aperistalsis, esophageal dilation, with minimal LES opening, "bird-beak appearance", and poor emptying of barium. Endoscopically is characterized by; dilated esophagus, with retained saliva, liquids, and undigested food particles in the absence of mucosal stricturing or tumors .**(1)**

The symptomatic consequence of this motility disorder , is the classic presentation of dysphagia to solid and liquids associated with regurgitation of blunt undigested food or saliva. Substernal chest pain during meals is present in the setting of dysphagia , weight loss, and even heart burn. **(2)**

The etiology is autoimmune, viral immune or neurodegenerative.**(3)** The pathologic consequence of the disease is degeneration of ganglion cells in the myentric plexus of the esophageal body and the LES. Although the cause of the degenerative process is unclear, the end result of the inflammatory process is loss of inhibitory neurotransmitters ,nitrous oxide and vasoactive intestinal peptide **,(4)** and consequently imbalance between the excitatory and inhibitory neurons. This results in unopposed cholinergic activity that leads to incomplete relaxation of the LES and aperistalsis due to loss of latency gradient along esophageal body. **(5,6)**

The manometric finding of aperistalsis and incomplete LES relaxation without evidence of a mechanical obstruction solidifies the diagnosis of achalasia in the appropriate setting. (7,8) The diagnosis of achalasia is supported by esophagram findings including dilated esophagus, a narrow GEJ with a bird- peak appearance, a peristalsis, and poor emptying of barium.**(9)**

The primary role of upper GI endoscopy in the work of achalasia is focused on rolling out a mechanical obstruction or pseudoachalasia as they

can mimic achalasia both clinically and manometrically. Mechanical obstruction can result in both impaired EGJ relaxation and abnormal esophageal body function (a peristalsis or spastic contraction).[10,11]

Achalasia is a chronic condition without cure. Current treatment options in achalasia are aimed at reducing the hypertonicity of the LES by pharmacological, endoscopic or surgical means.[12] The goals in treating achalasia are to relief patient symptoms, improve esophageal emptying, and prevent further dilation of the esophagus .[13]

Oral pharmacological therapies are the least effective treatment options in achalasia. Calcium channel blockers, and long acting nitrates are the two most common medications used to treat achalasia, they transiently reduce LE pressure by smooth muscle relaxation. The phosphodiestrase-5-inhibitor; sildenafil , has also been shown to lower the LES tone, and residual pressure. Botulinum toxin (Botox) is a potent pre synaptic inhibitor of acetyle choline release of nerve endings that has proven to be a useful treatment in achalasia.[14]

Pneumatic dilation (PD) is the most effective non surgical option for patients with achalasia. Bougienage or standard balloon dilators are not effective in fracturing the muscularis propria needed for symptomatic relief. All patients considered for PD must also be candidates for surgical intervention in the event of esophageal perforation needing repair. Heller myotomy remains the surgery of choice for many years. The technique evolved initially with a laparotomy approach, which was subsequently supplanted by minimally invasive technique. A thoracoscopic approach was developed and used with success, but laparoscopic myotomy has become the preferred method because of decreased morbidity, and faster recovery. [15]

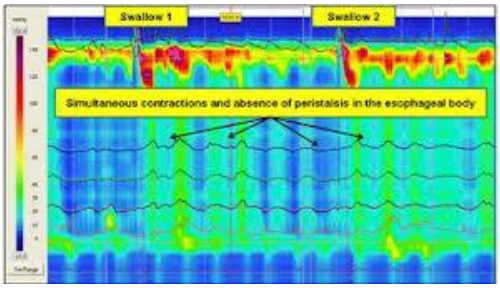

Fig.1-A: Esophageal manometry

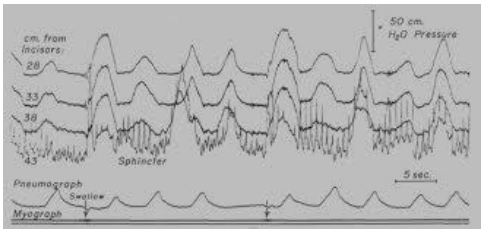

Fig.1-B: Esophageal manometry

PATIENTS AND METHODS

In this prospective study, 21 patients presented with achalasia were assigned to undergo laparoscopic Heller myotomy. Patients were investigated via esophageal manometry, upper GI endoscopy, barium swallow. All patients involved in the study after giving written informed consent, were assigned to undergo laparoscopic Heller myotomy without Dor fundoplication. All patients were studied postoperatively via 24-h ph study and manometry for 6monthes, pathological GER was defined as distal esophageal acid exposure time greater than 4.2% per 24h period. Diagnosis of achalasia was confirmed manometrically by the presence of simultaneous esophageal body contracture and non relaxing lower esophageal sphincter(LES).[15]

There were 21patients ; 12 (58.2%) of them were females ,and 9 (42.8%) were males, with a mean age of 42 years old , range (25-68 ys), mean operative time was 90 min, range (70-110min). The disease was graded according to esophageal diameter into : Stage1(<4cm): 12 patients, stage II (4-6cm): 6 patients, stage III(>6cm): 3 patients. Median LES pressure was 32.5 mm Hg, mean (27.5-37.5 mm hg), esophageal peristalsis was absent in all patients, none of them had undergone previous abdominal surgery.

Operative technique:

Briefly, under general anaesthesia , the patient was placed in supine position, with the surgeon between legs. A standard 5-mm trochar approach was used. The gastrohepatic ligament was initially divided to expose the right crus. The posterior attachments of esophagus were bluntly divided, preserving the posterior vagus nerve, the anterior phrenoesophageal ligament was incised, and the esophagus was circumferentialy dissected and encircled with a penrose drain. The esophagus was mobilized superiorly for at least 8cm in the mediastinum. The anterior vagus nerve was also

identified and bluntly dissected from the proposed myotomy site .The myotomy extended for 4-5cm above the GEJ and at least 1-2cm onto the stomach. Intraoperative endoscopy was routinely performed to ensure an adequate myotomy , and identified mucosal injury in every patient. If an inadequate relaxation was encountered by using bougies , esophageal site myotomy was extended for another 2cm . Figures (2,3,4).

Fig. 2: Exposure of right crus

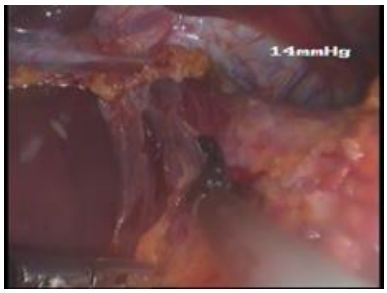

Fig. 3: Division of phrenoesophageal ligament

Fig. 4: Longitudinal myotomy is performed extending for 4-5 cm above EGJ and for 1-2 cm onto the stomach

Post operative follow-up was conducted at 1^{st},2^{nd} ,6^{th} months in each patient for symptoms of recurrent achalasia , The data was collected from routine office visits , and our staff also contacted patients who were unable to come to visits by phone. A complete follow-up questionnaire was done for each patient, and classified according to ***Vantrappen*** and ***Hellmans*** modified classification as follows**:**[16] 1-Exellent**:** Asymptomatic, 2-Good: Occasional dysphagia, thoracic pain, or heart burn requiring no therapy**.** 3-Fair: Dysphagia , thoracic pain or heart burn more than once a week, requiring therapy although patient was better than before surgery. 4-Poor: The patient worse than before surgery as regard (dysphagia, thoracic pain, or heart burn, regurgitatin , and weight loss) .

Patients were asked to return for a 24-h PH study and stationary manometry 3-5 months after surgery. After completing the questionnaire related to severity and frequency of dysphagia after surgery.[17]

Dysphagia score was calculated by combining the ***frequency*** of dysphagia: 0-never, 1<1day per wk, 2=1day per wk, 3=(2-3)days per wk, 4= (4-6)days per wk, 5=daily

As regard severity:

0=none, 1=very mild, 2=mild, 3=moderate, 4=moderately severe

5=severe. The presence of pathologic GER was established when the total time with PH <4 more than 4.2% per 24-hrs.[15,16]

RESULTS

All the 21 patients included in such study had laparoscopic Heller myotomy without Dor fundoplication. All surgeries were completed with laparoscopic technique, apart from one female patient who had intraoperative complication in the form of esophageal perforation, during laparoscopic myotomy, which was secured via primary repair. Another female patient who had esophageal perforation during PD underwent repair in conjunction with laparoscopic myotomy, repair was assessed intraoperatively by air insufflation inside esophagus and operative field was filled with saline, with the resultant no bubbling was noticed and repair was efficient, patients had satisfactory postoperative course without manifestations of leakage, also the 2 patients had upper GI contrast study (barium swallow, and barium meal) and pelvi-abdominal CT scan with no evidence of leak.

The median operative time was 90min (range70-110min). The median hospital stay was 2 days (range 1-8days), the average blood loss was 100ml. No perioperative deaths, no other concurrent operations, or additional procedures

done during laparoscopic Heller myotomy. All patients preoperative demographic data, and clinical course were evaluated in (table1).

All patients had satisfactory post operative course, with no GER symptoms. A complete follow-up questionnaire for each patient was done according to ***Vantrappen*** classification to assess relief of symptoms in postoperative period, revealed the following data:

14 patients were asymptomatic (graded as excellent), 5 patients with occasional dysphagia, and chest pain (graded as good) with no need for therapy. 2 patients graded as fair (dysphagia more than once a weak).

According to dysphagia score regarding ***frequency*** of attack:

14 patients had no attack in 6 monthes postoperative period (grade0=never), 6 patients had dysphagia attack less than 1 day per wk (grade 1), 1 patient had dysphagia attack equal to1 day per wk (grade 2).

According to ***severity*** of dysphagia: 14 patients were completely asymptomatic (non complaining of dysphagia) (grade0), 4 patients had very mild attack of dysphagia (grade1) , 2 patients had mild attack (grade2), 1 patient had moderate attack (grade3) (table2).

Table (1): Preoperative patients demographics

Number	***21***
Male	9(42.8%)
Female	12(57.1%)
Median(range)of age	42(28-65) years old
BMI(kglM^2)	25(19-48)
ASA classification	
1	8(38.1%)
2	10(47.6%)
3	3(14.2%)
Duration of symptoms(years)	1(0.13-8)
Achalasia subtype (according to esophageal dilation per cm)	
1	12(57.1%)
II	6(28.6%)
III	3(14.2%)
Median LES pressure	32.5mmhg (mean 27.5-37.5%)

Esophageal PH study and manometry performed at a median of 6 months. Median LES pressure was 14.5 mm hg, range (6.5-28.3mm hg), the median length of the LES pressure zone was 3.5cm, range (2-6cm).

Table (2): Perioperative outcome

Median(range) operative time (minute)	90(70-110)
Myotomy length(cm)	9(8-14)
Dysphagia score	
(1) frequency	
Grade 0 (never)	*14(66.6%)*
Grade 1 (attack<1day per wk)	*6(28.6%)*
Grade2 (attack=1day per wk)	*1(4.7%)*
(2)severity	
Grade 0 (none)	*14(66.6%)*
Grade 1 (very mild)	*4(19.1%)*
Grade2 (mild)	*2(9.5%)*
Grade3 (moderate)	*1(4.7%)*
Symptomatic relief according to Vantrappen:	
Exellent	*14(66.6%)*
Good	*5(23.8%)*
fair	*2(9.5%)*
Postoperative median LES pressure	*14.5 mm hg (6.5-28.3)*
Postoperative median length LES pressure zone	*3.5 cm (2-6)*
Intraoperative blood loss (average)	*100 ml*
postoperative complications	
1-major:*esophageal perforation*	*1(4.7%)*
2-minor:	
Subcutaneuos emphysema	*1(4.7%)*
Atrial fibrillatin(AF)	----
Urinary retention	*2(9.5%)*
Length of hospital stay per day	*2(1-8)*
Time(%)PH<4 while upright	*6.2+10.4*
Time(%)PH<4 while supine	*7.1+12.2*
No.of episodes with PH<4	*30+52*

DISCUSSION

Heller myotomy is the most effective treatment of dysphagia due to achalasia. The laparoscopic approach allows extending the myotomy well below GEJ. Some aspects of this procedure, however have yet to be defined. **(17)**Firstly, there is no agreement about who much the myotomy should be extended in order to be effective. Secondly, it is still not clear to what extend the myotomy causes GER.**(18)**

Another unresolved issue is the need to add an antireflux procedure to the myotomy, and what type of procedure should be performed. Finally, there is still debate about the incidence of recurrent achalasia following the anti- reflux

procedure, and what are the reasons for this. Topan, in1992; reported an incidence of 30% redo-surgery for recurrent dysphagia following Heller myotomy associated with Nissen fundoplication.[19]

Oelschlager et al. recently reported recurrence of dysphagia up to 17.3% in patients treated with myotomy extended for 1.5cm or less below GEJ, and only of 3.4% when extended for 3cm below GEJ.[20]

Richter et al. reported severe dysphagia after myotomy in16% of patients, requiring either surgical or endoscopic treatment. The myotomy was extended for 2cm below GEJ, and the anti reflux procedure was performed. [21]

Zaninotto et al. reported an incidence of recurrent dysphagia of 8.8% after laparoscopic Heller myotomy followed by anterior partial fundoplication (Dor).[22]

According to the author, this was due to either an incomplete section of the muscle fibres , or to the fibrotic scar of the myotomy edge. Hypothesis that scarring between fundoplication and the esophageal mucosa in the anterior wrap may account for the poorer result after Dor procedure is also argued by Lyass. [22]

Oelschlager, compared Dor vs Toupet fundoplication following Heller myotomy. reporting an incidence of recurrent achalasia 17.3% and 3.4% respectively (P=0.001), this could be due to the fact that, covering the myotomy site with the Dor fundoplication could lead to adhesions formation between the two surfaces and provoke achalasia. [23]

The toupet fundoplication keeps the myotomy edges apart by keeping fixed the fundus to each side, thus reducing the risk of fibrosis. Toupet fundoplication also required dissection of posterior esophageal attachments and the section of some of the short gastric vessels, which could reduce the GEJ competence with subsequent postoperative reflux.[24]

However, there are cases in which partial fundoplication may be beneficial. In patients with hiatal hernia, fundoplication may prevent stomach herniation. When unnoticed mucosal perforatin occurs during myotomy. A partial fundoplication may be used to cover the repair, clearly neither of the 2 approaches (Toupet or Dor fundoplication) resulted in a completely competent cardia and normal acid exposure.[25]

On the other hand, (Raiss, 2002); reported a 2% incidence of recurrent achalasia after laparoscopic Heller myotomy without fundoplication.

Lyass, in a review of literature from 1991 to 2001, analyzed persistent dysphagia and postoperative GER after laparoscopic Heller myotomy associated or not to anti reflux procedure, in this paper, an incidence of abnormal esophageal PH-manometry is reported in 35-36% of patients who underwent trans-abdominal open Heller myotomy without fundoplication and 10-16% of patients who had a partial fundoplication.[26]

In the present laparoscopic series, the rate of abnormal PH-manometry findings in the anti reflux vs no anti reflux procedure groups were10% and7.9% respectively. This may be explained by the different technical aspects of the procedures. The extent of esophageal dissection performed in the open surgery may be disruptive. The angle of Hiss, a natural barrier to reflux, is often destroyed by the extensive dissection performed in open surgery. The laparoscopic approach is less traumatic to this area, and more often preserves this angle. Also the length of the myotomy may be shorter especially on the gastric side.

Bloomston et al. compared the results of laparoscopic Heller myotomy with or without concomitant fundoplication (Dor), the dysphagia rate was 14% among patients who underwent myotomy alone, compared to 26% of patients received concomittant fundoplication.

The incidence of anti-acid treatment required postoperatively in the two groups was 13% vs10% respectively. [27]

Decker et al. reported 10% incidence of postoperative dysphagia, and Ackroyd et al, (2001) reported 13%. The incidence of post operative dysphagia reported in the literature varies between 17.3% and 30% after short myotomy, compared to 3.4-16% after long myotomy. [19]

Katz et al. reported a 6% incidence of endoscopic proved esophagitis after laparoscopic cardiomyotomy using the Dunhee technique, which limits the mobilization to the anterior wall of the abdominal and thoracic esophagus, stating that the routine addition of an anti reflux operation is not justified in patients undergoing laparoscopic cardiomyotomy, provided that the

lateral and posterior attachments of esophagus are kept intact.[8]

Decker, reported 17% postoperative dysphagia after myotomy and partial anterior or posterior fundoplication, and 7% of mucosal perforation.[29]

Lukttich and co-workers, reported an overall incidence of 9.6% of intra operative mucosal perforation, despite the use of epinephrine injection to lift up muscular layers. Recurrent dysphagia representing 12.9%, with 4.8% of redo-myotomy.

Even if the laparoscopic approach offers better exposure of the GEJ, injuries to the mucosa still occur especially when extending the myotomy on the gastric side, where the plane between the submucosa and the muscle layer is less evident and bleeding is profuse. [30]

In our study, we performed laparoscopic Heller myotomy alone without (Dor) anterior gastric fundic wrap in order to avoid adhesion formations, and scarring between raw mucosal surfaces of the esophagus and the overlapping gastric wall, and our results were close to those obtained by addition of Dor fundoplication as compared to the literature. [31]

In addition to the power and force added by Dor wrap through suturing the divided esophageal muscle edges to the anterior gastric fundus wrap, but still there is no evidence proved that this technique could prevent recurrence of achalasia through assumption that it will stop muscle approximation and healing again.[31]

It was found that adhesion between gastric wall (of Dor wrap) and exposed mucosal surface could be a cause for dysphagia.[31]

CONCLUSION

Laparoscopic Heller myotomy proved to be a safe and effective procedure in treating achalasia of esophagus, with minimal postoperative dysphagia or reflux symptoms.

In our study, we performed laparoscopic Heller myotomy alone in order to avoid adhesions formation and scarring between raw mucosal surfaces of the esophagus and the overlapping gastric wall with results close to those obtained by addition of Dor fundoplication as compared to the literature.

Also our patients had no postoperative dysphagia and no problem regarding achalasia symptoms recurrence.

In spite of good outcome and results; further data are required regarding Long-term symptomatic and physiological outcomes in terms of esophageal function and GER.[32]

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