Does previous endoscopic treatment affect the outcome of laparoscopic Heller myotomy?

L. Bonavina, R. Incarbone, M. Reitano, L. Antoniazzi, A. Peracchia

Clinica Chirurgica, Ospedale Maggiore Policlinico, Via F. Sforza 35, 20122 Milan, Italy

ABSTRACT

Study aim: Aim of this study was to assess symptomatic and objective outcome in patients undergoing laparoscopic Heller myotomy after unsuccessful endoscopic treatment, compared to patients having primary surgery.

Patients and method: Between November 1992 and December 1998, 92 patients with esophageal achalasia underwent laparoscopic Heller myotomy and Dor fundoplication. Intraoperative endoscopy was routinely performed. Sixty patients had primary surgery (PS); 32 patients had surgery after unsuccessful pneumatic dilatation (PD) (n=22), or botulinum toxin (Botox) injection (n=10).

Results: The mean operative time and the incidence of postoperative dysphagia were similar in the two groups. The incidence of intraoperative mucosal tears was 5% in the PS group and 12.5% in the PD/Botox group (P=NS). Mucosal tears occurred more frequently during the first 30 operations (17% vs 3.2%, P < 0.05). Median follow-up was 28 months (range 4–76). An abnormal esophageal acid exposure was documented in 2 patients in the PS group (7.7%), and in two patients in the PD/Botox group (13.3%) (P=NS). Lower esophageal sphincter pressure significantly decreased in both groups (P < 0.01). The mean percentage of radionuclide residual activity in the esophagus at 1 and 10 minutes significantly decreased in both groups (P < 0.01).

Conclusion: There is only a trend, although not statistically significant, towards an increased risk of complications and adverse effects in patients previously treated by PD and/or Botox. The higher incidence of mucosal tears during the first 30 operations suggests the effect of the learning curve. © 2000 Éditions scientifiques et médicales Elsevier SAS
Symptomatic relief of achalasia may be achieved by surgery [1, 2], endoscopic pneumatic dilatation [3, 4], or intrasphincteric botulinum toxin injection [5, 6]. Although laparoscopic myotomy is emerging as optimal initial therapy [7], still many patients are referred to the surgeon only after an unsuccessful endoscopic approach.

It is unknown whether previous treatment may cause histopathological changes at the esophagogastric junction resulting in a more difficult surgical procedure and a less satisfactory outcome. The purpose of this study was to assess the results of laparoscopic Heller myotomy performed in patients with achalasia previously treated by pneumatic dilation or botulinum toxin injection, compared to patients undergoing surgery as a primary treatment.

**PATIENTS AND METHOD**

Between November 1992 and December 1998, 92 patients referred to our Department for esophageal achalasia, underwent laparoscopic Heller myotomy with anterior fundoplication. There were 51 females and 41 males with a mean age of 38 years (range 9–66). Dysphagia was graded as: 0 = absent, 1 = to solid food, 2 = to liquids, 3 = to both solids and liquids; and according to the frequency as: 1 = occasional, 2 = once a month, 3 = once a week, 4 = twice a week, 5 = daily. Sixty patients with a mean dysphagia score of 2.5 ± 0.6 as grade and 4.5 ± 0.8 as frequency underwent primary surgery (PS); 32 patients with a mean dysphagia score of 2.7 ± 0.5 as grade and 4.7 ± 0.7 as frequency had surgery after unsuccessful pneumatic dilation (PD) (n = 22), or intrasphincteric botulinum toxin injection (Botox) (n = 10) performed elsewhere. Six patients of the Botox group were also previously treated with balloon dilatation. The number of previous dilations ranged from 1 to 4, and the number of Botox injections from 1 to 2. None of these patients had sustained a perforation during the endoscopic procedure. The mean time elapsed between endoscopic treatment and surgery was 8 months (range 2–17). Age and sex distribution, and preoperative radiological, manometric, and scintigraphic data were similar in both groups.

The operation was performed laparoscopically with a five-port access. The myotomy was extended up to 6 cm on the esophagus and 2 cm on the stomach, and a partial anterior fundoplication according to Dor was routinely added [1, 2]. In patients without a sigmoid esophagus, the dissection was limited to the anterior esophageal wall, in an attempt to prevent postoperative reflux by preserving the anatomical relationships of the cardia. An upper gastrointestinal endoscopy was carried out intraoperatively in all patients to aid identification of the esophagogastric junction. At the beginning of the experience, a balloon dilator (Rigiflex) was used to distend the lower esophagus and cardia at 1 PSI in order to facilitate division of all residual muscle fibers; subsequently, inflation and transillumination provided by the endoscope was considered appropriate to assist the myotomy.

Operative time, intraoperative complications, and symptomatic follow-up were recorded for each patient. A barium swallow study was routinely performed 1 to 3 months after surgery. Postoperative manometry and 24-hour esophageal pH monitoring could be performed in 62 and 41 patients, respectively. In addition, 13 patients underwent scintigraphic assessment of esophageal transit before and after surgery.

The χ² test and the Student’s t test were used for statistical analysis. Significance was established at the 0.05 level. Figures were reported as mean ± standard deviation.

**RESULTS**

There were three conversions to open procedure in the PS group. The reason was a mucosal tear in two patients and a difficult exposure in one. All these conversions occurred during the first two years of the experience. Subsequently, one additional tear
Laparoscopic Heller myotomy

Table I. Intraoperative and postoperative data of patients undergoing Heller myotomy after failed pneumatic dilation (PD) or botulinum toxin injection (Botox) compared to patients having primary surgery (PS).

<table>
<thead>
<tr>
<th></th>
<th>PD/Botox (n=32)</th>
<th>PS (n=60)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean operative time</td>
<td>110 ± 34 min</td>
<td>108 ± 44 min</td>
<td>0.89</td>
</tr>
<tr>
<td>Intraoperative tears</td>
<td>4 (12.5 %)</td>
<td>3 (5 %)</td>
<td>0.38</td>
</tr>
<tr>
<td>Postoperative dysphagia</td>
<td>6 (18.7 %)</td>
<td>*6 (10 %)</td>
<td>0.39</td>
</tr>
<tr>
<td>Dysphagia score</td>
<td>2.7 ± 1.5</td>
<td>2.5 ± 1.3</td>
<td>0.87</td>
</tr>
<tr>
<td>Abnormal pH score</td>
<td>**2/15</td>
<td>2/26 (7.7 %)</td>
<td>0.97</td>
</tr>
</tbody>
</table>

*1 pneumatic dilation; **both patients had previous PD.

was repaired laparoscopically. Conversely, three tears occurred in the PD group and one in the Botox group. All were treated laparoscopically, and the postoperative course was uneventful. The difference between the rate of perforation in the PS and in the PD/Botox group was not statistically significant (5% vs 12.5%). However, the incidence of mucosal perforation was significantly higher in the first 30 patients of the series (5/30, 17% vs 2/62, 3.2%, P < 0.05).

Postoperative morbidity rate was 6.7%: subcutaneous emphysema (3 pts), bleeding from the port site (2 pts), bleeding from acute gastric ulcer (1 pt). The median hospital stay was 4 days (range 3–8 days).

At a median follow-up of 28 months (range 4–76), 80 patients were completely relieved of symptoms, 11 complained of mild dysphagia requiring only dietary adjustments, and one individual required a pneumatic dilatation of the cardia for relief. The incidence of postoperative dysphagia and/or abnormal esophageal acid exposure was higher in the PD/Botox group, but this was not statistically significant (table I). None of the patients with a positive 24-hour pH test complained of reflux symptoms or had evidence of esophagitis at endoscopy. The mean esophageal diameter on standard barium swallow study significantly decreased compared to preoperative values from 4.4 ± 2 to 2.5 ± 0.5 (P < 0.05) in the PD group, and from 4.2 ± 1.5 to 2.9 ± 1.7 in the PD/Botox group (P < 0.05). Manometric and scintigraphic data are reported in table II and III, respectively. The lower esophageal sphincter pressure and the residual radionuclide activity significantly decreased after surgery in both groups, indicating reduced outflow resistance at the esophagogastric junction.

**DISCUSSION**

Treatment of achalasia is aimed to decrease the resistance to esophageal outflow by reducing sphincter pressure. This may be achieved by primary surgery,
endoscopic pneumatic dilation, or intrasphincteric botulinum toxin injection.

It has been shown that Heller myotomy leads to better long-term symptomatic results compared to endoscopic dilation [8]. However, balloon dilation proved to have a high success rate, and offers some advantages over surgery, ie, no need of general anesthesia and the possibility to be performed on an outpatient basis [9]. Recent uncontrolled studies show that dilation can achieve success rates similar to myotomy when performed by expert operators [10]. Patients who do not respond well to the first two dilations are unlikely to benefit from subsequent sessions, which may increase the risk of perforation [11].

The recent introduction in the clinical practice of intrasphincteric botulinum toxin injection has lessened the drawbacks of endoscopic dilation, ie, forceful disruption of the muscle fibers and risk of perforation. However, after a median follow-up of 2.4 years, only one third of the patients are still in remission despite multiple injections [5].

The advent of laparoscopic surgery has provided a minimally invasive approach for the treatment of patients with achalasia, making surgery a more attractive option as the initial therapy [2, 12]. Yet, a proportion of patients are often referred for surgery after being unsuccessfully treated by endoscopic dilatation or intrasphincteric botulinum injection. Transient tissue damage in the mucosa-submucosa layer has been documented by high-resolution endoscopic ultrasonography [13], but it is still unknown whether previous endoscopic treatments may cause histopathological changes of the esophagogastric junction leading to periesophageal inflammation, difficult identification of the circular or sling fibers, or difficult dissection of the submucosal plane. As a result, there may be a higher risk of intraoperative perforation [14, 15] and postoperative dysphagia [16].

It has recently been reported that in patients undergoing myotomy after intrasphincteric injection of botulinum toxin, the identification of the anatomic planes at surgery may be more difficult; moreover, patients who previously responded to Botox show a fibrotic reaction at the gastroesophageal junction leading to a higher rate of intraoperative mucosal tears [16-18].

In our series, a trend toward a higher rate of intraoperative mucosal tears was noted in the group of patients having myotomy after previous unsuccessful endoscopic treatment. Since the majority of the mucosal tears occurred in the first 30 patients, it can also be speculated that this is the effect of the learning curve. It has to be emphasized that all mucosal perforations were detected immediately; as experience was gained, the repair was achieved laparoscopically and the postoperative course was uneventful in these patients. Keeping this in mind, the surgeon should attempt to perform a complete myotomy without an excessive fear to enter the lumen even in patients with marked fibrotic changes at the esophagogastric junction. An incomplete myotomy on the gastric side has long been recognized as the most common technical fault associated with the Heller operation in the era of open surgery [19, 20]; this problem may be even greater in the laparoscopic era due to the effect of the learning curve of the procedure.

It has been emphasized that intraoperative endoscopy is helpful to identify precisely the esophagogastric junction and to verify the completeness of the myotomy minimizing the risk of occult perforations [21]. Alves et al. [22] have shown that endoscopic and laparoscopic criteria for the identification of the esophagogastric junction were discordant in 58% of the patients; in all these cases, the cardia was at a more distal site with endoscopic criteria. Since laparoscopic criteria underestimate the length of the myotomy over the stomach, intraoperative endoscopy should be recommended to improve surgical outcome.

CONCLUSION

Laparoscopic Heller myotomy is feasible with a high success rate also in patients who had previously undergone endoscopic treatment; the increased level of technical difficulties encountered at operation in these patients can be offset by routine use of intraoperative endoscopy and improved expertise in laparoscopic surgery.

REFERENCES


