Surg Endosc (2005) 19: 1188–1192
DOI: 10.1007/s00464-004-8199-0

SURGICAL ENDOSCOPY
and Other Interventional Techniques

Timing of surgical intervention does not influence return of esophageal peristalsis or outcome for patients with achalasia

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Received: 1 February 2005/Accepted: 8 February 2005/Online publication: 28 July 2005

Abstract
Background: It has been suggested that abnormal function of the lower esophageal sphincter is the primary abnormality in esophageal achalasia, and that the absence of esophageal peristalsis is secondary to the outflow obstruction caused by the lower esophageal sphincter. Furthermore, it has been proposed that early elimination of the resistance at the level of the gastroesophageal junction by surgical intervention could result in return of esophageal peristalsis. This study aimed to assess whether the timing of surgical intervention affects the return of esophageal peristalsis and the clinical outcome for patients with achalasia.
Methods: Between January 1991 and May 2003, 173 patients underwent a Heller myotomy by minimally invasive surgery for treatment of esophageal achalasia. Of these patients, 41 (24%) had pre- and postoperative esophageal manometry. These patients were divided into three groups based on the duration of symptoms: group A (10 patients; duration of symptoms 12 months group B (19 patients, duration of symptoms 12 to 60 months), and group C (12 patients; duration of symptoms longer than 60 months).
Results: The average duration of symptoms (dysphagia was present in all patients) was as follows: group A (8 ± 4 months), group B, (35 ± 16 months), and group C, (157 ± 94 months). Vigorous achalasia was present in 40%, 21%, and 17% of the groups respectively. The differences between the groups were not significant. Postoperatively, improvement in esophageal motility was seen in no patient in group A, 1 patient (5%) in group B, and 1 patient (8%) in group C. Excellent or good results were obtained for 90% of the group A patients, 95% of group B patients, and 92% of the group C patients. Again, the differences were not significant.
Conclusions: The results show that: a) the presence of vigorous achalasia is independent of symptoms duration; b) the timing of surgical intervention does not influence the return of peristalsis; and c) the results of a Heller myotomy are independent of symptoms duration.

Key words: Achalasia — Vigorous achalasia — Esophageal manometry — Esophageal peristalsis — Heller myotomy

Esophageal achalasia is the most common and well-defined primary esophageal motility disorder [3]. Although the etiology still is unknown, the physiologic abnormalities associated with the disease are well described [3]. Classically, the manometric criteria for diagnosis have been the absence of peristalsis throughout the body of the esophagus (i.e., simultaneous contractions) and the presence of a hypertensive lower esophageal sphincter (LES) that fails to relax in response to swallowing [4].

Traditionally, these abnormalities have been considered to occur simultaneously, secondary to a process involving both the lower esophageal sphincter (LES) and the esophageal body. Alternatively, however, it has been proposed that the abnormalities of esophageal peristalsis might be secondary to the obstruction caused by the hypertensive and nonrelaxing LES [8, 15]. Therefore, impaired LES function would the primary pathophysiologic event, with esophageal body dilation and dysmotility as secondary events, determined by the degree and duration of the obstruction. On the basis of this hypothesis, the contractions in the esophageal body would show high amplitude initially (vigorous achalasia), then subsequently would show low amplitude over time, particularly when esophageal dilation occurs [2, 18]. This hypothesis, supported by experimental studies in cats [6, 15] and clinical observation in patients

Presented at the Poster Session of the Society of American Gastrointestinal Endoscopic Surgeons (SAGES) meeting, Denver, CO, USA, 31 March–3 April 2004

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undergoing either pneumatic dilatation or myotomy [8, 17, 18], suggests that peristalsis can return if the obstruction at the level of the gastroesophageal junction is removed early in the course of the disease.

The goal of this study was to determine whether the preoperative manometric profile in patients with achalasia is correlated with the duration of symptoms, and whether the timing of surgical intervention affects the return of esophageal peristalsis and the clinical outcome.

Patients and methods

Between January 1991 and May 2003, 173 (83 women and 90 men) patients underwent minimally invasive surgery for the treatment of esophageal achalasia. The mean age of these patients was 46 years. Of the 173 patients, 25 underwent thoracoscopic Heller myotomy and 148 had a laparoscopic Heller myotomy and Dor fundoplication.

This study enrolled 41 of these patients (24%) who underwent pre- and postoperative esophageal function tests in the Swallowing Center of the University of California, San Francisco. These 41 patients comprised 17 women (41%) and 24 men (59%). The mean age was 42 years (range, 14–80 years). They had been symptomatic for an average of 62 months (range, 1–400 months), and 20 patients (50%) had undergone previous treatment. Specifically, 15 patients (75%) had undergone pneumatic dilations alone; 1 patient (5%) had received intrasphincteric Botulinum toxin injections; and 4 patients (20%) had experienced both types of treatment.

Study design

All the patients ($n = 41$) who had undergone the postoperative esophageal function test were divided in three groups based on their duration of symptoms: group A (duration of symptoms 12 months or less), group B (duration of symptoms 12 to 60 months, and group C (duration of symptoms longer than 60 months).

Symptomatic evaluation

Patients scored the severity of their dysphagia and regurgitation using a 5-point scale ranging from 0 (no symptoms) to 4 (disabling symptoms). The patients' ability to swallow was graded as follows: excellent (no dysphagia), good (occasional dysphagia; fewer than two times per week), fair (frequent dysphagia; two or more times per week, requiring dietary adjustments), and poor (severe dysphagia preventing ingestion of solid food). Patients were questioned before and after the operation by a fellow (not involved with the operation) who explained to them the questionnaire. All the patients reported dysphagia; 68% reported regurgitation; and 60% reported retrosternal pain. Chest pain was present in 60% of the patients with vigorous achalasia (distal esophageal amplitude of 37 mmHg or greater) and in 60% of the patients with classic achalasia (distal esophageal amplitude less than 37 mmHg).

Barium esophagogram

The barium esophagogram showed a characteristic bird beak appearance of the esophagus in 37 patients (90%). The esophageal diameter was less than 4 cm in 32% of the patients, between 4 and 6 cm in 45% of the patients, and more than 6 cm in 23% of the patients.

Upper endoscopy

Endoscopy was performed to rule out a peptic or neoplastic stricture of the distal esophagus.

Esophageal manometry

The patients were studied after an overnight fast using a technique previously described [10]. Medications that might interfere with esophageal motor function (i.e., metoclopramide, nitrates, and calcium channel-blocking agents) were discontinued at least 48 h before the study. The following variables were assessed: resting pressure of the LES and esophageal peristalsis (amplitude, duration, and velocity of the contractions measured 3, 8, 13, and 18 cm above the upper border of the LES). In 10% of the patients, the manometry catheter was placed under fluoroscopy because it could not be passed blindly through the gastroesophageal junction.

Operative technique

Of the 41 patients, 10 (24%) underwent a left thoracoscopic myotomy. The myotomy was 7 cm long and extended about 0.5 cm onto the gastric wall. No fundoplication was added [11]. A laparoscopic approach was used for 31 of the patients (76%). The operation consisted of an anterior myotomy 7 to 8 cm long, which was extended 2.0 to 2.5 cm onto the gastric wall. After division of the short gastric vessels, the fundus of the stomach was folded over the exposed mucosa, and an anterior 180° fundoplication (Dor fundoplication) was created [9].

Follow-up evaluation

All the patients were examined 2 and 6 weeks postoperatively. They subsequently were seen in the office or contacted by telephone every 3 to 4 months. The mean follow-up period was 56 ± 36 months for the group A patients, 74 ± 33 months for the group B patients, and 67 ± 18 months for the group C patients.

Statistical analysis

Statistical analysis was performed using Fisher's exact test, analysis of variance (ANOVA), and the paired t test, as indicated. We calculated the change in chest pain and dysphagia symptom scores from presurgery to postsurgery for each patient. We then applied the sign test to these changes to test the null hypothesis of no improvement on the average. This fully nonparametric assessment of matched pre–post treatment data was based only on whether each patient improved, stayed the same, or worsened. All the results are expressed as mean ± standard deviation. Differences are considered significant at a value p value less than 0.05.

Results

Hospital course

The median length of hospital stay was 72 h for the patients who underwent a thoracoscopic Heller myotomy and 24 h for the patients who underwent a laparoscopic Heller myotomy. Overall, 85% of the patients were discharged within 48 h. There were no deaths.

Symptomatic assessment

The severity of dysphagia decreased postoperatively in the three groups ($p < 0.05$). Overall, 38 (92%) of the 41 patients considered their swallowing status to be good or excellent after the myotomy. Specifically, excellent or good results were obtained in 90% of the group A patients, 95% of the group B patients, and in 92% of the group C patients. Chest pain resolved in 83% of the group A patients, 83% of the group B patients, and 86% of the group C patients (Table 1). The differences were not significant. There was no difference in clinical outcome between the patients with vigorous achalasia and the patients with classic achalasia.

Table 1. Postoperative symptom assessment

	Group A (n = 10)	Group B (n = 19)	Group C (n = 12)
Symptoms duration (months)	8 ± 4	35 ± 16	157 ± 94
Follow-up (months)	56 ± 36	74 ± 33	67 ± 18
Swallowing status: excellent/good results (% pts)[a]	90	95	92
Resolution/Improvement Chest pain (% pts) ‡	83	83	86

[a] A vs B vs C vs A (p = NS)
NS, not significant

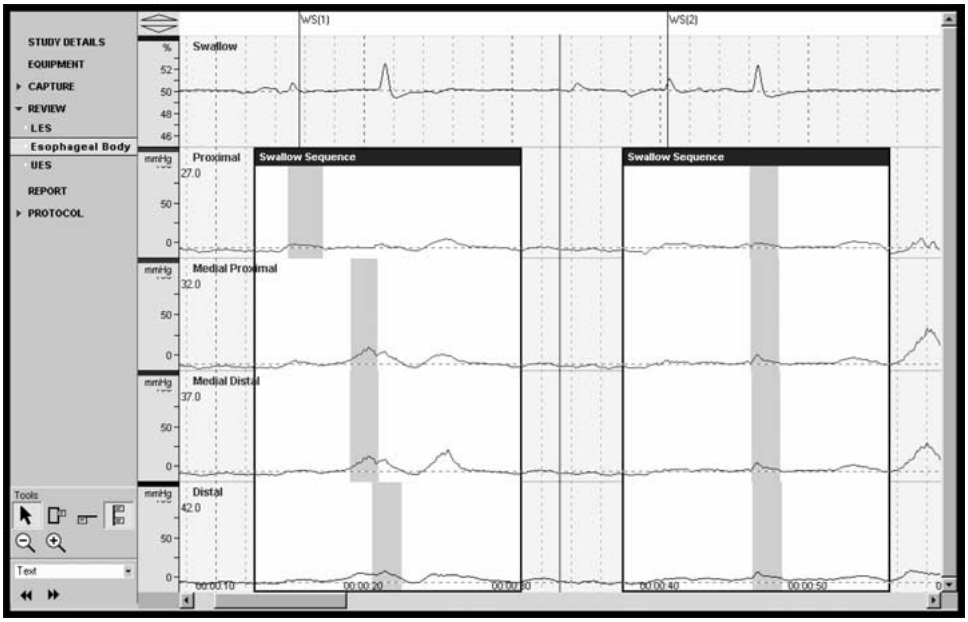

Fig. 1. Postoperative esophageal manometry for one patient in group B (duration of symptoms 24 months) showing in wet swallow 1 (WS[1]) a progression of waves 3 to 8 cm above the lower esophageal sphincter (LES). In this patient, 40% of the waves were simultaneous and 60% were segmented. Distal esophageal amplitude was of 28 mmHg preoperatively and 18 mmHg postoperatively.

Esophageal manometry

The median postoperative time to esophageal function tests was 14 months. Preoperatively, the average LES pressure was higher in the patients who had a shorter duration of symptoms (group A). However, in this group, only 20% of the patients had undergone previous treatment, as compared with 53% of the group B patients and 66% of the group C patients. Regardless of the preoperative values, LES pressure decreased postoperatively from 33 ± 14 to 11 ± 3 mmHg in group A ($p < 0.05$), from 16 ± 10 to 11 ± 9 mmHg in group B ($p < 0.05$), and from 24 ± 12 to 9 ± 3 mmHg in group C ($p < 0.05$). The postoperative LES pressure was similar among the three groups of patients, with no significant difference. No significant difference was noted in the preoperative distal esophageal amplitude among the three groups.

Overall, vigorous achalasia was present in 10 (24%) of the 41 patients. Specifically, it was present in 40% of the group A patients, 21% of the group B patients, and 17% of the group C patients. The difference were not significant. Esophageal peristalsis was absent preoperatively in 100% of the patients. Minimal changes were observed postoperatively in only two patients (5%): one in group B and one in group C. Only one of these patients had vigorous achalasia preoperatively (distal esophageal amplitude of 53 mmHg).

Patient 1 (Group B: preoperative duration of symptoms, 24 months).

Postoperative esophageal manometry showed that 40% of the waves were simultaneous and 60% were segmented, with some progression of peristalsis 3 to 8 cm above the LES. Distal esophageal amplitude was of 28 mmHg preoperatively and 18 mmHg postoperatively. (Fig. 1)

Patient 2 (group C: preoperative duration of symptoms, 68 months).

Post-operative esophageal manometry showed 80% of the waves were simultaneous and 20% were segmented, with some progression of peristalsis 3 to 8 cm above the LES. Distal esophageal amplitude was 53 mmHg preoperatively and 68 mmHg postoperatively.

Discussion

The study results show that: a) the presence of vigorous achalasia is independent of symptoms duration; b) the timing of surgical intervention does not influence the return of peristalsis; and c) the results of a Heller myotomy are independent of symptoms duration.

Clinical manifestations of esophageal achalasia

The most common clinical manifestations of esophageal achalasia are dysphagia and regurgitation, which are considered secondary to the outflow resistance caused by the LES and to the absence of peristaltic waves [4, 13]. Chest pain occurs in about 60% of patients. It has been suggested that chest pain is present early in the course of the disease, and that it is caused by high-amplitude contractions (vigorous achalasia) [2, 14]. However, our study contradicts this theory, because it showed that the incidence of chest pain was comparable when patients with vigorous achalasia and classic achalasia were evaluated (60% vs 60%). The findings also showed that chest pain was independent from the duration of symptoms because it occurred with similar frequency in the three groups of patients (60% vs 63% vs 58%). In addition, the clinical outcome was similar for the patients with vigorous achalasia and the patients with classic achalasia.

Natural history of esophageal achalasia

The physiologic manifestations observed with achalasia are thought to result from the selective loss of postganglionic inhibitory neurons in the myenteric plexus of Auerbach [3, 13]. The abnormal function of the LES and the absence of peristalsis would therefore occur simultaneously as a consequence of this neuromuscular dysfunction. In contrast, on the basis of animal experimentation and clinical observation, others have postulated that the primary event would occur at the level of the LES, and that the loss of peristalsis would be secondary to the outflow obstruction caused by a hypertensive and nonrelaxing LES. High-amplitude simultaneous contractions (vigorous achalasia) would be present early in the course of the disease, whereas low-amplitude contractions would develop later when esophageal dilation occurs [6]. Others, however, have questioned vigorous achalasia as a variant of classic achalasia [1, 4, 12].

Schneider et al. [15] demonstrated using a feline model that banding of the gastroesophageal junction results in progressive loss of amplitude and propagation of peristaltic waves similar to that seen in patients with achalasia. Additionally, they demonstrated that these abnormalities were reversible if the outflow obstruction caused by the band was removed. These findings are in part supported by clinical observation of patients treated by either pneumatic dilation or myotomy [7, 8, 17, 18]. Parrilla et al. [8] studied 45 patients with achalasia in whom a myotomy had been performed. They documented the return of peristalsis postoperativey in the upper esophagus of 47% of the patients in the mid-esophagus, of 24% and in the whole esophagus of 9%. Furthermore, peristalsis returned more frequently when the duration of symptoms was shorter (24 vs 60 months), and when vigorous achalasia was present (73% vs 33%). Some return of peristalsis also was documented by Vantrappen et al. [17] among 22 patients treated with pneumatic dilation.

Although this theory is interesting, it is indeed based on many faulty assumptions. Many studies have shown that the LES is hypertensive in only about 50% of patients [5, 16]. For instance, we found that among 109 untreated patients with achalasia, the LES was hypertensive in 45%, showed normal pressure in 27%, and was hypotensive in 28% [5]. We have no clear explanation for the difference in terms of postoperative motility between our study and the studies of Parrilla et al. [8] and Vantrappen et al. [17]. However, it is important to note that although some studies describe a ''return of peristalsis,'' in reality they are documenting only minimal changes, probably without any meaning in terms of food bolus propagation. Even Parrilla et al. [8] admitted that ''return of normal peristaltic activity was not recorded in any patient''. We did not find any return of peristalsis postoperatively. We rather observed a negligible change of the preoperative pattern in only two patients (5%), in whom the contractions went from simultaneous to segmented because of minimal progression 3 to 8 cm above the LES (Fig. 1). Moreover, these changes were not related to the duration of symptoms, the presence of vigorous achalasia, or the postoperative LES pressure.

Goal of therapy for patients with esohaeal achalasia

Although it is important to treat patients early in the course of the disease to prevent esophageal dilation, weight loss, and the risk of aspiration, the clinical outcome is not affected by the timing of intervention. In our study, good or excellent results were, in fact, obtained independently of symptoms durations, and specifically in 90% of the group A patients, 95% of the group B patients, and 92% of the group C patients (Table 1). Because we did not observe any return of peristalsis, we believe that the improvement of symptoms was attributable to elimination of the outflow resistance caused by the myotomy. This finding confirms that the treatment is palliative rather than curative.

Conclusions

In summary, motility changes after surgical treatment for achalasia cannot be predicted either by clinical history or manometric findings. These changes are clinically irrelevant and unrelated to the duration of the disease. Therefore, early elimination of the resistance at the level of the gastroesophageal junction by surgical intervention does not result in a return of peristalsis and does not affect outcome.

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