Causes of Long-Term Dysphagia
After Laparoscopic Nissen Fundoplication
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ABSTRACT

Background: Laparoscopic fundoplication has revolutionized the surgical treatment of gastroesophageal reflux disease. Despite improvements in the technique of fundoplication, persistent dysphagia remains a significant cause of postoperative morbidity.

Method: Causes of persistent postoperative dysphagia were analyzed in a consecutive series of 167 patients after laparoscopic Nissen fundoplication. Short gastric vessel division and its effect on postoperative dysphagia were analyzed.

Results: Follow-up was possible in 139 patients (83%). The mean follow-up period was 27 ± 21 months. Nine patients (6%) had persistent (moderate to severe) dysphagia, and 33 patients (24%) had mild dysphagia. The satisfaction score among patients with persistent dysphagia was significantly lower than that in patients with mild dysphagia ($P < 0.0002$). On the other hand, the satisfaction rate among patients with mild dysphagia and those who are asymptomatic was similar. Manometry, performed in 7 of 9 persistent dysphagia patients revealed no difference in postoperative lower esophageal sphincter (LES) pressure and relaxation as compared with that in the control group ($n = 52$). Six of 9 patients with persistent dysphagia underwent a re-do antireflux procedure. Dysphagia as related to fundic mobilization (complete vs. partial) or bougie size ($< 58 \text{ Fr. vs.} \geq 58 \text{ Fr.}$) revealed no difference in the dysphagia ratings.

Conclusions: Laparoscopic short Nissen fundoplication with or without fundic mobilization achieved an acceptable long-term dysphagia rate. Careful patient selection, identification of the short esophagus, and accurate construction of the fundoplication can lead to a decrease in the incidence of persistent postoperative dysphagia.

INTRODUCTION

Transient dysphagia occurs in 40% to 70% of patients after Nissen fundoplication.1,2 This is thought to be secondary to edema at the gastroesophageal junction (GEJ) or transient esophageal hypomotility.3 Fortunately, dysphagia usually resolves spontaneously within 2 to 3 months.1,4 Persistent dysphagia (PD), however, occurs in 3% to 24% of patients after Nissen fundoplication.5-10 The laparoscopic approach may be associated with a higher rate of PD than its open counterpart.8

The precise etiology of PD is unclear. The construction of a tight, slipped or displaced fundoplication, peptic stricture, impaired esophageal clearance, and unrecognized achalasia are believed to be the primary causes.11-13 The role of short gastric vessel division and fundic mobilization in the avoidance of PD remains controversial.6,8,14,15

In this study, we examined the causes of PD after laparoscopic Nissen fundoplication and analyzed the benefit of complete versus incomplete gastric fundic mobilization in reducing the risk of postoperative dysphagia.

MATERIALS AND METHODS

Patients

From August 1991 to February 1999, 167 patients underwent laparoscopic Nissen fundoplication. A standardized preoperative symptom questionnaire was used for all patients. Esophagogastroduodenoscopy (EGD) was performed in all patients. Physiological testing of the esophagus (manometry and 24 hour pH monitoring) was performed selectively. The indication for surgery included refractory gastroesophageal reflux disease (GERD) in patients under 50 years of age, noncompliance with medical therapy, breakthrough symptoms, Barrett’s esophagus, peptic stricture, or associated pulmonary disorders. No patient had previous foregut surgery.

Key Words: Laparoscopy, Laparoscopic Nissen fundoplication, Dysphagia.
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requiring laparoscopic paraesophageal hiatal hernia repair, re-do surgery, or Toupet fundoplication were excluded from the study.

**Operative Technique**

From 1991 to 1996 (n = 80), gastric fundus mobilization was considered incomplete as adherence to complete ligation of all short gastric vessels (gastric vessels were divided with hemoclips and the hook cautery) was not possible in our early experience due to technical limitations. In some patients sufficient distance did not exist between the superior pole of the spleen and the fundus to safely apply hemoclips and divide tissue. Since 1997 (n = 87), all short gastric vessels including the posterior short gastrics were divided using the Harmonic scalpel (Ethicon Endosurgery, Cincinnati, OH). This together with the use of a 45-degree laparoscope has made short gastric ligation a safe part of the procedure. Fundic mobilization was considered complete when the posterior aspect of the left limb of the right crus was fully exposed. Intraoperative flexible endoscopy was not performed for assessment of esophageal length during this study period. The crura are then approximated posteriorly with nonabsorbable sutures, and a 2-cm fundoplication was constructed over a 58 Fr. or 60 Fr. bougie using a 2-0 Prolene pledgeted U-stitch. The right and left limbs were checked for continuity.\(^1\)

**Postoperative Evaluation**

The patients were interviewed by phone questionnaire by an independent observer. The severity of dysphagia (mild to severe) and the satisfaction score between 0 (unsatisfied) and 10 (very satisfied) were obtained. Dysphagia is defined as mild (occasional with coarse food lasting for a few seconds), moderate (requiring cleaning with liquids), and severe (a semi-liquid diet, a history of meat impaction, or dysphagia for liquids). Persistent dysphagia (PD) is defined as moderate or severe dysphagia lasting more than 3 months after the laparoscopic Nissen fundoplication. Patients with PD requiring dilatation or reoperation underwent EGD, manometry, and barium swallow selectively to determine the cause of dysphagia. Manometric evaluation of the LES resting pressure and relaxation was performed using the station pull-through method. Sphincter relaxation was considered complete (100%) when it dropped to the gastric baseline on swallowing and poor when it was < 85% at any level across the fundoplication. Five wet swallows were used at each 1-cm increment through the high-pressure zone. The manometric data of patients with PD was compared to that of nondysphagia patients who had undergone laparoscopic Nissen fundoplication with the same operative technique (n = 52).\(^1\)

Results were reported as the mean (± SD). Statistical comparison of continuous data was made using the Student's \(t\) test and the ANOVA test. Fisher's exact test was used to determine the significance of a 2 x 2 contingency table. A \(P\) value < 0.05 was considered significant.

**RESULTS**

The postoperative clinical outcome was obtained from 139 of 167 patients (83%) after laparoscopic Nissen fundoplication. The mean follow-up period was 27 ± 21 months. The group comprised 73 males (53%) and 66 females (47%). The mean age at the time of first operation was 51 years (range 16 to 78 years). Symptom presentation included heartburn (n = 121, 87%), regurgitation (n = 82, 59%), dysphagia (n = 46, 33%), chest and/or epigastric pain (n = 31, 22%), chronic cough and/or asthma (n = 15, 11%), and nausea (n = 2, 1.4%).

At follow-up, 9 patients (6%) complained of PD (Table 1), and 33 patients (24%) had mild dysphagia (Table 2). The satisfaction score for patients with PD was significantly lower than that in those who were asymptomatic (3.8 ± 3.6 vs. 8.9 ± 2.2, \(P < 0.0002\)). On the other hand, the satisfaction score for patients with mild dysphagia was similar to that in the asymptomatic group (8.6 ± 1.8 vs. 8.9 ± 2.2, NS). Endoscopic dilatation following the primary procedure was required in 17 patients (12%), PD (n = 9), and mild dysphagia (n = 7). One patient who was asymptomatic at follow-up underwent endoscopic dilatation for early postoperative dysphagia.

Six of 9 patients with PD required re-do antireflux surgery. The mean duration between the original operation and re-do surgery was 12 months (range, 3 to 36). These patients underwent an average of 3 endoscopic dilatations before the re-do surgery. Two patients underwent an open thoracotomy, and the other patients underwent laparoscopy. Following the re-do procedure, the dysphagia improved in all but 1 patient who was discovered to have cancer of the GEJ and died 6 months later. The outcome was considered fair in 2 patients in whom the dysphagia had improved postoperatively. In 1 patient, recurrence of reflux symptoms was due to disruption of
| No. | Age/Sex | Preop. Symptom* | Preop. LES relaxation | Procedure | Causes of Dysphagia | Re-do | Outcome/Follow-up in Months ‡ |
|-----|---------|----------------|-----------------------|-----------|--------------------|-------|-------------------------------|
| 1   | 59F     | HB/Reg         | 92                    | Nissen    | Hiatal stenosis    |       | Excellent No dysphagia 2 months |
| 2   | 55F     | HB/Reg         | 99                    | Nissen    | Possibly anterior angulation of GEJ | Nissen | Good Mild dysphagia 8 months |
| 3   | 56F     | HB/Cough       | 88                    | Nissen    | Too tight fundoplication | Nissen | Excellent No dysphagia 4 months |
| 4   | 46M     | HB/Reg         | 100                   | Nissen    | Slipped Nissen with short esophagus, Peptic stricture | Collis-Belsey | Fair: recurrent reflux symptoms due to stricture formation and fundoplication disruption. Mild dysphagia 6 months |
| 5   | 57F     | Dys/HB/Reg     | 86                    | Nissen    | Slipped Nissen with short esophagus, Severe esophagitis | Collis-Nissen | Fair: recurrent reflux symptoms. All investigations were normal. Mild dysphagia 38 months |
| 6   | 76F     | Dys            | 100                   | Nissen    | GEJ cancer         | Toupet | Death 6 months postoperative |
| 7   | 51M     | Dys/HB         | 100                   | Nissen    | Nutcracker esophagus | †       | Fair: chest pain controlled with Ca blocker. Moderate dysphagia |
| 8   | 43F     | Dys/HB/Reg     | 100                   | Nissen    | Nutcracker esophagus | †       | Fair: reflux symptoms Moderate dysphagia |
| 9   | 36M     | HB/Reg/Chest pain | 98                   | Nissen    | Unknown             | †       | Good Moderate dysphagia |

*Dys: Dysphagia, HB: Heartburn, Reg: Regurgitation
Preop. = Preoperative, LES=Lower esophageal sphincter
† = No surgery
‡ = Post re-do
Excellent: no symptoms
Good: occasional symptoms not necessitating medication
Fair: symptoms controlled with medication
Poor: worsening symptoms requiring frequent dilations or reoperation.
the Belsey repair that was constructed for esophageal dysmotility (> 30% simultaneous esophageal body contractions).

Three patients with PD did not undergo surgery. These patients underwent an average of 2 endoscopic dilatations. A nutcracker esophagus was the cause of dysphagia in 2 patients. The cause of dysphagia was unknown in the last patient who would not consent to postoperative testing.

Specific causes of dysphagia were determined either by manometry, endoscopy, or operative findings. Postoperative esophageal manometry was performed in 7 of 9 patients with PD. The mean duration between the primary operation and manometry was 15 ± 14 months. The mean postoperative resting pressure of the LES, relaxation of the LES, and esophageal body pressure among patients with PD was not significantly different compared with that in our control group (n = 52). The mean preoperative LES relaxation for the 3 groups (asymptomatic, mild, and persistent dysphagia) was 98.5 ± 4.5, 98 ± 4, and 90 ± 5, respectively (ANOVA test, \( P = 0.22 \); Bartlett’s test for homogeneity of the variance, \( P = 0.5 \), NS). In addition, the mean preoperative contraction amplitude at the lower end of the esophagus among the 3 groups was 68 mm Hg (range, 11 to 204), 102 mm Hg (range, 15 to 285), and 119 mm Hg (range, 37 to 251) respectively (ANOVA test, \( P = 0.3 \); Bartlett’s test, \( P = 0.4 \), NS). Persistent dysphagia as related to the degree of fundus mobilization or bougie size is shown (Table 3). Intraoperative complications related to complete and incomplete fundic mobilization occurred in 6 (bleeding from the short gastrics, n = 5; splenic injury, n = 1) and 3 patients (bleeding from the short gastrics, n = 3), respectively, \( P = 0.7 \), NS. Among the 46 patients with preoperative dys-

| Age/Sex       | Preop. Symptom, n | Preop. LES Relaxation | Satisfaction Rate | Outcome, n |
|---------------|-------------------|-----------------------|-------------------|------------|
| 49 ± 10.5     | Heartburn, 28     | 98 ± 4                | 8.6 ± 1.8         | Good, 33   |
| Male, n = 18  | Regurgitation, 18  |                       |                   |            |
| Female, n = 15| Dysphagia, 7      |                       |                   |            |
|               | Chest pain, 7      |                       |                   |            |
|               | Asthma, 1          |                       |                   |            |
|               | Cough, 1           |                       |                   |            |
|               | Epigastric pain, 1 |                       |                   |            |
|               | Nausea, 1          |                       |                   |            |

Table 2.

Patients (n = 33) With Mild Dysphagia.

| Persistent Dysphagia (yes) | Persistent Dysphagia (no) | \( P \) Value |
|----------------------------|---------------------------|---------------|
| Complete (n = 82)          | 5                         | 77            | 1             |
| Incomplete (n = 57)        | 4                         | 53            |               |
| Bougie Size - Fr.          |                           |               |               |
| \( \geq 58 \) (n = 133)    | 8                         | 125           | 0.33          |
| \(< 58 \) (52-56) (n = 6)*  | 1                         | 5             |               |

*Patients with small fundus
phagia, 4 developed PD postoperatively; on the other hand, 5 of 93 patients with no preoperative dysphagia developed PD postoperatively, \( P = 0.5, \text{NS} \).

**DISCUSSION**

The advent of minimally invasive surgery has revolutionized the surgical treatment of GERD. Studies have shown that the functional results of laparoscopic antireflux procedures are equal to that in open surgery with significantly less postoperative morbidity and a shorter hospital stay.\(^2\,^7\,^16\) Minimally invasive surgery, however, is not without complications, and the management of these complications may negate the benefit of surgery over the alternative long-term medication.\(^17\) One of the most troublesome complications is PD. In this report, we have established that laparoscopic Nissen fundoplication has a 6% long-term PD rate. The causes of PD were inaccurate preoperative endoscopy, hiatal stenosis secondary to severe fibrotic reaction, anterior angulation of the GEJ, missed diagnosis of the short esophagus, nutcracker esophagus, and a too tight fundoplication. The cause of PD was not clear in 1 patient who would not consent to postoperative testing. As the laparoscopic operative procedure evolved over time, the degree of fundic mobilization increased but this did not influence the results. Our study demonstrates the following: (1) preoperative dysphagia did not predict patients at risk of developing PD, (2) preoperative LES relaxation and esophageal body contractions were similar across the 3 groups (asymptomatic, mild dysphagia, and PD), and (3) postoperative manometric analysis among patients with PD was similar to that in the control group.

Anvari et al\(^18\) demonstrated that chronic PD patients had a higher postoperative LES basal and nadir pressure than patients without postoperative dysphagia. Herron et al\(^19\) found that preoperative manometric analysis was similar among patients with PD and their control group. These authors identified preoperative difficulty of swallowing as the sole risk factor for PD. Others, however, found that 68% of laparoscopic Nissen fundoplication patients with preoperative dysphagia noted an improvement in swallowing function postoperatively.\(^6\)

One of the controversial technical aspects of Nissen fundoplication is whether the short gastric vessels should be divided to minimize the risk of PD. Good results have been reported after both laparoscopic and open surgery with and without division of these vessels.\(^8\,^14\,^15\,^18\,^20\) Early reports of laparoscopic Nissen fundoplication compared their early and late experience. Because of the inherent problem of a learning curve bias associated with such an analysis, the outcome in the group is usually better, leading to the conclusion that the short gastric vessels should always be divided.\(^14\) We did not observe a significant difference in the PD rate between patients with complete and incomplete short gastric division even though the latter was performed earlier in the series. It appears that the construction of a loose fundoplication is probably more important than whether the short gastric vessels are divided or not. Nevertheless, we continue to divide the short gastric vessels and completely mobilize the fundus, as this has been an integral part of our protocol since 1997 and teaching commitments to our surgical residents.

The right posterior limb of the fundoplication may elevate and angulate the distal esophagus from its resting position just cephalad of the fundoplication. This was a possible but unproven explanation of PD in one of our patients. Tight fundoplication with achalasia like LES relaxation was the cause of dysphagia in 1 patient despite normal preoperative manometric profile and construction of the fundoplication over a 60 Fr. bougie. Hiatal stenosis is a rare complication of laparoscopic Nissen fundoplication.\(^10\) It must be differentiated from an excessively tight closure of the hiatus, which is possible if bougie calibration of the hiatus is not done.\(^21\) Hiatal stenosis results from excessive scar formation constricting the esophagus at the hiatal level. It is easily recognized at reoperation and can be corrected by incising the hiatus anteriorly. The short esophagus is a serious complication of GERD that, if not recognized, at initial surgery carries a 25% failure rate.\(^22\) Although preoperative tests often help in predicting the short esophagus,\(^23\) the only reliable way to confirm or exclude the presence of the short esophagus is to demonstrate intraoperatively that the GEJ lies at least 3 cm below the arch of the crus;\(^24\) otherwise, a lengthening procedure is needed.\(^25\)

**CONCLUSION**

We conclude that PD is usually related to a technical error. Although some cases of PD are of an unknown etiology and unavoidable, most cases are avoidable. Careful patient selection, surgeon preoperative endoscopy, intraoperative flexible endoscopic assessment of esophageal length and proper construction of the fundoplication are recommended for the successful performance of laparoscopic Nissen fundoplication.
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