



Secrets for successful laparoscopic antireflux surgery: adequate follow-up

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Abstract: A laparoscopic fundoplication achieves long-term reflux symptom control in most patients. However, dysphagia, recurrence of symptoms and gas-related symptoms are experienced in 10% to 20% of patients postoperatively. To date, the adequate follow-up of patients undergoing laparoscopic fundoplication is not well established. In addition, the outcomes of patients undergoing antireflux surgery for Barrett' esophagus (BE) are controversial. This article reviews the current evidence about the most effective follow-up after antireflux surgery, focusing on the management of the symptomatic patients and the need for surveillance in Barrett's esophagus patients.

Keywords: Gastroesophageal reflux disease; heartburn; dysphagia; Barrett' esophagus (BE); fundoplication; barium esophagogram; esophageal manometry; ambulatory 24-hour pH monitoring

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Introduction

A laparoscopic fundoplication is the most successful surgical treatment modality in patients with gastroesophageal reflux disease (GERD). Long follow-up studies have shown that it is very effective in symptom relief in most patients (1-4). However, recurrence of symptoms, dysphagia, and gas-related symptoms are experienced in 10% to 20% of patients postoperatively (5,6), and the proper management of symptomatic patients is not well established.

The long-lasting exposure of the esophageal mucosa to pathological duodenal-gastric refluxate leads to the development of intestinal metaplasia (IM) in up to 15% of GERD patients, with the potential risk of progression to dysplasia and cancer (7,8). To date, the real impact of antireflux surgery on the prevention of progression from IM to dysplasia is under evaluation, and the outcomes of patients undergoing antireflux surgery for Barrett' esophagus (BE) are controversial (9).

The aim of this article is to summarize the current

evidence about the most appropriate follow-up after antireflux surgery, focusing on the management of the symptomatic patients and the need for surveillance in BE patients.

Patients with persistent or recurrent symptoms: evaluation

Failure of a laparoscopic fundoplication is secondary to one or more of the following reasons: (I) improper patient selection; (II) inappropriate preoperative work-up and (III) surgical technical errors.

The evaluation of symptomatic patients after laparoscopic fundoplication includes a thorough medical history, a meticulous comparison of preoperative and postoperative symptoms (GERD related symptoms, bloating and flatulence), the need for the use of anti-secretory medications and the patient response in terms of symptom control.

However, the diagnosis of recurrent GERD relying on symptoms evaluation only is not accurate. Even though it is usually assumed that heartburn after a fundoplication is due to the failure of the operation, several studies have shown that both sensitivity and specificity of regurgitation and heartburn for abnormal gastroesophageal reflux are low. For instance, Lord *et al.* (10) conducted a study aiming to determine the frequency of GERD in a cohort of surgically treated patients with postoperative symptoms. They included 86 patients complaining symptoms after total fundoplication. The mean postoperative follow-up period was 28 months. Acid reducing medications were prescribed in 37 patients (43%) postoperatively. All patients completed a detailed symptom questionnaire. The 24-hour pH monitoring showed that only 23% of all study population and 24% of the patients receiving medical therapy had pathological acid exposure of the esophagus. A disrupted or abnormally constructed fundoplication was the most significant factor associated with abnormal acid exposure. Similar results were reported by Galvani *et al.* (11) in a prospective study analyzing esophageal manometry and pH monitoring data of 124 patients who developed symptoms after laparoscopic fundoplication with a mean follow-up of 17 months. A total of 62 patients (50%) were receiving H₂ blocking agents and/or proton pump inhibitors (PPIs) at the time of the evaluation. They found that abnormal esophageal exposure to acid refluxate was present in 39% of the study population, while it was normal in 61% of them. Pathologic reflux was present in only 32% of patients who were medically treated. Lastly, Thompson *et al.* (12) revised 76 patients who had 24-hour pH monitoring for recurrent heartburn. The 24-hour pH monitoring tracing was pathological in only 26% of these patients. Interestingly, medications were taken to treat heartburn by 35 patients (63%) who had a normal 24-hour pH monitoring. The 24-hour pH monitoring was more likely abnormal in patients who had a previous partial fundoplication and in those who had a good response in terms of symptom relief when acid reducing medications were recommenced. These results suggest that ambulatory 24-hour pH monitoring should be part of the early assessment of patients complaining symptoms after antireflux surgery in order to prevent inappropriate medical treatment or a reoperation.

Esophageal manometry should be obtained when patients complain postoperative dysphagia, thus assessing the lower esophageal sphincter pressure and relaxation, and the quality of the peristalsis of the esophageal body, since a too tight or too long wrap can lead to an achalasia type

picture (13).

Barium esophagram and upper endoscopy should always be obtained in patients with symptoms after laparoscopic fundoplication. These tests allow to evaluate both esophageal length and breadth, the morphological characteristics of a hiatal hernia, and the presence of an esophageal narrowing. Horgan *et al.* (14) proposed in 1999 a morphologic classification of failures of fundoplication that helps the clinician understand the reason why the surgical operation failed. There are 4 types of hernia:

- (I) Type IA hernia: the fundoplication and the gastroesophageal junction (GEJ) are both in the chest above the diaphragm;
- (II) Type IB hernia: the GEJ is in the chest above the diaphragm, while the fundoplication is positioned in the abdomen below the diaphragm;
- (III) Type II hernia: redundant gastric fundus is above the fundoplication and located in the chest;
- (IV) Type III hernia: the fundoplication is performed using the gastric body and not the fundus. It is similar to type II hernia, but both GEJ and fundoplication are in the abdomen.

Barrett's esophagus: does a fundoplication prevent progression to dysplasia and cancer?

Several randomized trials and non-randomized controlled studies have compared the outcomes of medication versus antireflux surgery in BE patients, showing a lower risk to develop dysplasia or cancer in patients surgically treated than in patients treated with PPIs (15). Ortiz *et al.* (16) included 59 BE patients in a randomized controlled trial (medical therapy, n=27; fundoplication, n=32). With a median follow-up of 4 years for the medically treated patients and 5 years for the patients undergoing a fundoplication, the BE segment length decreased in a higher number of patients in the surgical group (8 *vs.* 2 patients). On the other hand, the BE segment length more likely increased in the group of patients who received medical therapy (11 *vs.* 3 patients). Histologic evaluation of endoscopic biopsies showed the presence of low grade dysplasia (LGD) or high grade dysplasia (HGD) only in patients who received medical therapy or experienced failure of the fundoplication. None of the patients with effective antireflux surgery had evidence of dysplasia during the follow-up.

Parrilla *et al.* (17) compared 43 BE patients randomized to PPIs and 58 BE patients randomized to antireflux

surgery. With a median follow-up of 5 years for the medically treated patients and 6 years for the surgically treated patients, patients with successful antireflux surgery had a significantly lower incidence of HGD than patients randomized to PPIs (2% vs. 20%). This beneficial effect of antireflux surgery in patients with non-dysplastic IM was confirmed by Öberg *et al.* (18), who found that none of the 46 patients undergoing surgery developed HGD or cancer compared to 7.4% of 94 medically treated patients after a median follow-up of almost 6 years.

Oelschlager *et al.* (19) investigated the clinical impact of antireflux surgery on the risk of developing esophageal cancer in 106 BE patients. With a median follow-up of 40 months, 1 patient with a preoperative diagnosis of LGD experienced regression to normal epithelium, while 2 other patients had progression: 1 case to HGD and 1 case to T1N0 cancer. Complete IM regression to normal esophageal mucosa was demonstrated only in patients with short-segment BE (55% vs. 0% patients with long-segment BE). Among patients with complete IM regression, the esophageal exposure to acid refluxate was normal on 24-hour pH monitoring in 89% of patients cases compared to 69% of those who did not have complete regression.

The need for objective assessment of pathological reflux by upper endoscopy and esophageal function tests even many years after antireflux surgery has been well explored by Csendes *et al.* (20). They followed up until December 1999 a total of 161 BE patients who had antireflux surgery between 1978 and 1992. Dysplastic changes were detected in 17 (10.5%) patients, while esophageal cancer developed in 4 cases (2.5%). The authors compared these 21 patients with 126 patients who did not develop dysplasia or cancer after antireflux surgery. Short-segment BE was present in 2 (12%) patients with dysplasia, while long-segment BE was detected in all patients who developed cancer. The esophageal manometry found a hypotensive LES in more than two third of the patients who developed dysplasia and of the patients complaining recurrence of symptoms without dysplasia, and in all patients who had esophageal cancer. The 24-hour pH monitoring was positive for abnormal acid or duodenal reflux in most patients with dysplasia and in those with recurrent symptoms in the absence of dysplasia.

Recurrent reflux after antireflux surgery has been suggested as a possible risk factor for the progression from IM to dysplasia and eventually to esophageal adenocarcinoma. However, there are very few data that show the link between failure of antireflux surgery and progression of BE. For instance, O’Riordan *et al.* (21)

evaluated 58 patients with BE after antireflux surgery, with a questionnaire, upper endoscopy with biopsies, and preoperative and postoperative 24-hour pH study. With a median follow-up of almost 5 years, symptom control was excellent in 52 patients (90%). Recurrent symptoms were complaint by 6 patients (10%). Postoperative pH monitoring showed abnormal reflux in 17 (41%) patients. A total of 35% of patients had regression of BE, while 2 (3.4%) patients developed dysplasia. Esophageal cancer was diagnosed in two (3.4%) more patients at 4 and 7 years after antireflux surgery. All 4 patients had positive 24-hour pH monitoring.

Similarly, Zehetner *et al.* (22) reviewed 75 patients with BE followed for almost 9 years after antireflux surgery, showing that IM was more likely to progress in patients with failure of the fundoplication.

Conclusions

Even though a laparoscopic fundoplication is successful in the vast majority of patients, dysphagia or recurrence of symptoms or dysphagia may occur in up to 20% of patients. A careful clinical evaluation and a thorough objective evaluation of these patients is highly suggested before starting with medical therapy or proposing revisional surgery.

A successful antireflux surgery seems to decrease the likelihood of IM progression to dysplasia or adenocarcinoma mainly in patients with short segment BE, but it does not eliminate the risk. Long-term endoscopic surveillance with biopsies and 24-hour pH monitoring after antireflux surgery is recommended in patients with BE, since BE patients with recurrent abnormal reflux are often asymptomatic, and BE progression may occur late during the follow-up (23).

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