ABSTRACT

Persistent postoperative dysphagia (PPD) is one of the most troublesome complications of laparoscopic antireflux surgery. Hiatal stenosis, although rare, is a serious complication and is one of the causes of PPD after antireflux procedures.

In the 2 presented patients, progressive dysphagia started immediately after the antireflux procedure and did not respond to esophageal dilations. The cause of dysphagia in both patients was hiatal stenosis and was corrected laparoscopically.

Key Words: Esophagus, Hiatus, Stenosis, Dysphagia.

INTRODUCTION

The advent of minimally invasive surgery (MIS) has revolutionized the surgical management of gastroesophageal reflux disease (GERD) leading to an explosion in the number of antireflux procedures performed. Laparoscopic antireflux surgery, however, is not without complications, and the management of these complications may negate the proposed cost benefit for surgery over long-term medication. One of the most troublesome complications is persistent postoperative dysphagia (PPD).1 Although some patients experience preoperative dysphagia and may experience some degree of early postoperative dysphagia, persistent difficulties affect 10% to 15% of patients and require surgical revision or dilation in 1% to 3% of cases.2,3 The construction of a tight, slipped, or displaced fundoplication, peptic stricture, impaired esophageal clearance, and unrecognized achalasia may be the cause of PPD.4-6 Esophageal motility and lower esophageal sphincter dynamic studies have not defined a specific cause for PPD in some patients.7 Hiatal stenosis is another cause of PPD that has been reported previously.8

We report herein the cause and treatment for 2 cases of hiatal stenosis after laparoscopic Nissen fundoplication.

CASE REPORT 1

A 59-year-old female developed progressive dysphagia immediately following laparoscopic Nissen fundoplication performed for long-standing GERD. Her medical history was unremarkable apart from fibromyalgia that was controlled with analgesics. Esophagogastroduodenoscopy (EGD) showed an intact fundoplication. A barium esophagram demonstrated narrowing at the gastroesophageal junction (GEJ). Esophageal manometry showed a normal mean relaxation of the lower esophageal sphincter (LES) at 89%, and ineffective esophageal motility with 60% interrupted and 30% dropped waves (esophageal manometry done prior to the primary procedure showed a hypertensive intrathoracic LES with good relaxation and normal esophageal body function).

The dysphagia did not improve despite 3 dilations. Two months later, the patient underwent a laparoscopic reop-
operative procedure. The fundoplication was taken down and a flexible endoscope passed transorally could not traverse the hiatus; abundant scar formation was present, tightening the anterior and posterior hiatus. An anterior stricturoplasty of the diaphragmatic hiatus plus Toupet fundoplication was performed. At 2-year follow-up, the patient has grade II dysphagia (requiring clearing with warm liquids for solid food), and she considers the outcome satisfactory.

**CASE REPORT 2**

A 38-year-old male patient developed increasing dysphagia immediately after laparoscopic Nissen fundoplication. The patient had fibromyalgia and a history of a left orchiectomy for testicular seminoma for which radiation therapy was administered to the periaortic region (3000 rads).

An upper endoscopy showed narrowing at the GEJ and a barium esophagram revealed hold-up of the barium tablet at the GEJ. Esophageal manometry showed a hypertensive LES with poor mean relaxation at 64%, an immotile esophagus with 100% simultaneous waves, and low amplitude contractions (<30 mm Hg), features consistent with pseudoachalasia (esophageal manometry done prior to the primary procedure showed an incompetent LES with normal esophageal body function). The dysphagia did not improve despite 2 dilations. Three months later, the patient underwent laparoscopic reoperative surgery, after the fundoplication had been dismantled, intraesophageal endoscopy showed an occlusion at the hiatus. An anterior stricturoplasty plus Toupet fundoplication was done. At 2-year follow-up, the patient had no dysphagia.

**DISCUSSION**

Hiatal stenosis is a rare complication of laparoscopic Nissen fundoplication. It results from excessive perihiatal scar tissue formation constricting the esophagus at the hiatal level, even in patients who did not undergo crural repair. It must be differentiated from overtightening of the crural closure, which is a possible sequela if bougie calibration of the hiatus is not performed. In both patients, progressive dysphagia started immediately after the antireflux procedure and did not respond to esophageal dilations. In addition, the cause of dysphagia in both patients was diagnosed and corrected laparoscopically.

The cause of hiatal stenosis is unclear. In some patients, it may be a response to diathermy dissection. In one patient, radiation to the periaortic region was a significant risk factor for hiatal stenosis, but in the other patient no obvious risk factors could be identified. Both of our patients had fibromyalgia, but it is unknown whether this has any bearing on the development of hiatal stenosis.

Skinner and Low reported that esophageal manometry in patients with esophageal clearance failure frequently shows motor disorders that may be secondary to a partial esophageal obstruction following the antireflux repair. A study of 163 patients who underwent laparoscopic Nissen fundoplication for GERD showed that patients with high or normal LES pressure are at a higher risk of developing postoperative dysphagia. Esophageal preoperative manometry showed hypertensive LES in 1 patient, while a postoperative manometry profile showed impaired esophageal body motility in both patients, and in one of them defective relaxation of the LES was present. In a Watson et al study, esophageal manometry was performed in 2 out of 3 patients with hiatal stenosis. It was normal in both, but in an Orsoni et al study, esophageal manometry was done in 1 of 2 patients, revealing impaired LES relaxation.

Both of our patients underwent esophageal dilatations after the primary procedure, which did not improve dysphagia, so a reoperative Toupet fundoplication was done in both patients. Khaitan et al indicated that repeat dilations may be inadequate therapy and that complete take-down of the fundoplication may be a durable option in treating patients with achalasia after fundoplication.

The diagnosis of hiatal stenosis is usually made intraoperatively as preoperative testing (EGD, barium esophagram, or esophageal manometry) cannot discern with certainty the cause of dysphagia in this subset of patients. Passage of a transoral endoscope after the fundoplication is dismantled will determine whether the cause of dysphagia is at the hiatus or the fundoplication. Laparoscopic hiatal dissection will demonstrate excessive scarring and fibrous tissue formation especially at the anterior and lateral aspects of the hiatus. Overtightening of the crural closure is not associated with excessive scar formation, and the patient never experiences normal swallowing in the postoperative period.
CONCLUSION

Hiatal stenosis is a rare but serious complication of laparoscopic antireflux surgery. Patients with previous radiation to the upper abdomen may be at increased risk of developing hiatal stenosis. The association between hiatal stenosis and fibromyalgia needs further assessment. The laparoscopic approach with intraoperative endoscopy is effective in diagnosing hiatal stenosis.

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