

## CASE REPORT

# Laparoscopic management of a late gastric perforation post-fundoplication

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## Abstract

Nissen fundoplication is a safe and effective procedure for gastroesophageal reflux. Gastric ulceration leading to perforation is an uncommon late complication with a mechanism of action that is poorly understood.

A 40-year-old male presented 3 years following his fundoplication with a perforated ulcer in the upper body of the stomach. The patient admitted ibuprofen use leading up to the perforation. The perforation was successfully repaired via laparoscopy, with good postoperative outcome.

Gastric ulcer perforation post-fundoplication is a rare late outcome. Gastric perforation may have been precipitated by the use of non-steroidal anti-inflammatory drugs (NSAIDs) in concert with the vascular mechanical changes caused by this procedure. A review of the literature reveals only a handful of accounts of gastric ulcers or perforations as a late outcome. Of note, many of these perforations occur in the upper stomach and in tandem with NSAID use.

## INTRODUCTION

Laparoscopic Nissen fundoplication is recognized as a safe and effective procedure for treating gastroesophageal reflux. Major improvement in GERD symptoms is reported in up to 89% of patients. [1] Mortality is low, between 0.2 and 1% [1–3]. Perioperative and immediate morbidity rates are between 8 and 17%. Early postoperative complications include gastroesophageal perforation (0–4%), splenic injury (<1%), pneumothorax (0–10%), and nausea and vomiting (0–10%) [3, 4].

The most common late postoperative complications include postprandial abdominal bloating (1–85%), dysphagia lasting less than 3 months (10–50%), dysphagia lasting more than 3 months (3–24%) diarrhea (18–33%), and heartburn (10–62%) [3, 4]. 94% of late postoperative complication symptoms resolved within 12 months [5]. Gastric ulceration and perforation is a potential late postoperative complication, although rare in the literature. We present a case of perforation occurring 3 years after fundoplication.

## CASE REPORT

Our patient is a 40-year-old man with chronic GERD. He experienced worsening symptoms including nocturnal awakenings despite diet modification and double-dose PPI medication compliance. Endoscopy showed esophageal ulcers. DeMeester 2-day score was 29.8. Standard Nissen type 360 degree fundoplication was performed, with hiatal repair. Over the course of the next year, endoscopy performed for abdominal discomfort and dyspepsia showed proximal gastric ulceration.

The patient presented to the emergency room three years postoperatively with complaints of sharp epigastric pain, in the setting of ibuprofen use. Abdominal exam showed tenderness with guarding in the upper abdomen but without generalized peritonitis. CT scan revealed a moderate pneumoperitoneum consistent with a perforated viscus. Exploratory laparoscopy was initiated after creating pneumoperitoneum via a Verses needle and placement of four upper abdominal laparoscopic

Received: May 7, 2019. Accepted: June 29, 2019

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ports. The distal stomach and duodenal bulb were healthy without any perforation or contamination. The proximal stomach was examined after elevating the left lobe of the liver with a Nathanson retractor. On the posterior wall of the proximal stomach there was a 5–7 mm perforation located above the level where the greater curve had been mobilized. Tissues surrounding the perforation appeared relatively healthy with no gross evidence of neoplasia. Primary repair was performed with three interrupted 2–0 Surgidac sutures. Omentopexy was performed by tacking the omentum down to the stomach using the upper and lower sutures. The patient did well postoperatively, was started on a liquid diet on postoperative Day 1, and discharged home on Day 3.

Twice daily PPIs and sucralfate were continued for 2 weeks, followed by maintenance PPI therapy. Serum gastrin level was normal. Endoscopy performed 6 weeks postoperatively revealed a healed ulcer on the posterior gastric wall approximately 5 cm from the gastroesophageal junction, consistent with the perforation site. Mild antral gastritis was noted, without any ulceration. *Helicobacter pylori* was negative by histopathology and CLO test.

## DISCUSSION

Nissen fundoplication has seen marked success in treating the symptoms of GERD. While the commonly cited late complications can be a nuisance for patients and impact quality of life, gastric ulceration and perforation are potentially far more serious, albeit rare. Delayed diagnosis of a perforation in the post-fundoplication setting can result in death and therefore ought to be better understood [6].

Perforation is cited more commonly as an intraoperative complication. Schauer *et al.* reported 15 perforations out of 713, 12 of which were diagnosed intraoperatively and 3 postoperatively [6]. For delayed ulcers, Huget *et al.* recorded a total of six perforations out of 1 600 procedures. These were located on the anterior fundal wall [2]. Tores *et al.* described a gastric ulcer perforation, four months post-fundoplication, located near the cardia [7]. Braungart *et al.* reported an ulcer 5 months post-fundoplication, at the gastroesophageal junction [8]. The patient in the case of Tores was on high dose ibuprofen and the patients highlighted by Huget were all on celecoxib. It is interesting to note that all of the late perforations occurred in the proximal stomach.

When perforation does occur, management can be conservative or operative. Our experience was that a laparoscopic approach with omentopexy was satisfactory. The three reported by Huget were all repaired via laparotomy [2]. Braungart cites management by intravenous antibiotics and PPIs in the case of a stable patient with perforation, while Toros reported open gastrectomy [7, 8].

The mechanism of delayed perforation post fundoplication is poorly understood. It may begin with the iatrogenic interruption to the vascular supply during the procedure, resulting in mucosal ischemia or underperfusion. Several authors have discussed this possibility, although there is a paucity in actual data [9, 10]. Despite the rich collateral blood supply of the stomach, mechanical alteration as a result of the procedure might set the stage for greater susceptibility to ulceration in the long-term.

Other possible mechanisms of post-fundoplication ulcer formation that have been described in the literature include, gastric stasis, gastric irritant medications, or foreign body [2]. In our experience, however, endoscopy revealed no foreign body or evidence of stasis; nor was this an actual finding by any of the aforementioned authors we reviewed.

*Helicobacter pylori* and NSAIDs are well established as the two leading causes of gastric ulceration [10]. However, our patient and others in the literature we reviewed, revealed absence of *H. pylori* colonization. NSAID involvement, on the other hand, was a common theme. Anatomically, post-fundoplication ulcers appear to occur in the proximal stomach. We theorize that gastric ulceration occurs in this patient population, as a result of NSAID action on relatively underperfused proximal stomach post-fundoplication.

## CONCLUSION

Gastric ulceration and perforation after laparoscopic Nissen fundoplication is a rare outcome. NSAID action on relatively underperfused stomach likely accounts for proximal gastric ulceration and perforation. With this in mind, we recommend preserving as much of the vasculature as feasible in preparation of the fundal wrap. Patients should be counseled on the avoidance of NSAIDs when possible. If not, augmentation with PPIs may be warranted to counteract the ulcerative predilection of NSAIDs. If signs and symptoms of peptic ulcer disease present, a low threshold for upper endoscopy may be prudent. Further research is needed to establish the mechanism of the ulcer formation post-fundoplication and whether NSAIDs increase this risk.

## CONFLICT OF INTEREST STATEMENT

None declared.

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