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Complications of Antireflux Surgery

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Antireflux surgery anatomically restores the antireflux barrier and is a therapeutic option for proton pump inhibitor (PPI)-refractory gastroesophageal reflux disease or PPI intolerance. Laparoscopic fundoplication is the standard antireflux surgery, though its popularity has declined due to concerns regarding wrap durability and adverse events. As the esophagogastric junction is an anatomically complex and dynamic area subject to mechanical stress, wraps are susceptible to disruption, herniation or slippage. Additionally, recreating an antireflux barrier to balance bidirectional bolus flow is challenging, and wraps may be too tight or too loose. Given these complexities it is not surprising that post-fundoplication symptoms and complications are common. Perioperative mortality rates range from 0.1 to 0.2% and prolonged structural complications occur in up to 30% of cases. Upper gastrointestinal endoscopy with a comprehensive retroflexed examination of the fundoplication and barium esophagram are the primary tests to assess for structural complications. Management hinges on differentiating complications that can be managed with medical and lifestyle optimization versus those that require surgical revision. Reoperation is best reserved for severe structural abnormalities and troublesome symptoms despite medical and endoscopic therapy given its increased morbidity and mortality. Though further data are needed, magnetic sphincter augmentation may be a safer alternative to fundoplication.

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INTRODUCTION

Gastroesophageal reflux disease (GERD) is defined as the presence of troublesome symptoms and/or complications that develop due to retrograde reflux of gastric contents in the esophagus [1]. GERD affects one in five Americans and is one of the most common diagnoses managed in the outpatient gastroenterology clinic [1, 2]. Although the majority of patients with GERD derive symptom relief with proton pump inhibitors (PPIs), 30 to 40% do not respond adequately [3]. Various mechanisms can drive PPI non-response [4], one being PPI-refractory GERD. PPI-refractory GERD patients continue to experience troublesome symptoms in relation to ongoing objective GERD despite optimized PPI therapy. In the case of PPI-refractory GERD or PPI intolerance, antireflux surgery is a therapeutic option [5, 6].

The objective of antireflux surgery is to anatomically restore the antireflux barrier and thereby reduce gastroesophageal reflux episodes. This is no easy task as the esophagogastric junction and its antireflux mechanisms are complex and dynamic. The antireflux barrier is composed of the lower esophageal sphincter, the extrinsic crural diaphragm and the flap valve configuration related to supporting structures and orientation of the intrinsic and extrinsic sphincters. The current standard for antireflux surgery is laparoscopic fundoplication which seeks to repair a hernia, reposition the sphincter within the abdomen and recreate a flap valve using a posterior or anterior fundoplication [7, 8]. Success rates of laparoscopic fundoplication range from 67 to 95%, and depend highly on surgical expertise, adequate preoperative evaluation and appropriate patient selection [9–11]. A comprehensive diagnostic evaluation prior to antireflux surgery is requisite to ensure that the patient has objective evidence of GERD, to confirm that ongoing PPI-refractory GERD symptoms are due to GERD rather than non-GERD causes and to exclude contraindications to surgery. Gastroenterologists commonly evaluate patients with PPI non-response and function as the primary point of care for patients following antireflux surgery. Since laparoscopic fundoplication is an invasive and irreversible intervention associated with non-negligible morbidity and mortality rates, it is essential that gastroenterologists are familiar with the evaluation and management of complications following fundoplication [12]. This paper will focus on complications of antireflux surgery, and provide a matrix for the evaluation and management of complications following antireflux surgery.

LAPAROSCOPIC FUNDOPLICATION

Trends in utilization

Surgical Nissen fundoplication was first described in 1956 and regarded as a highly efficacious antireflux intervention, particu-

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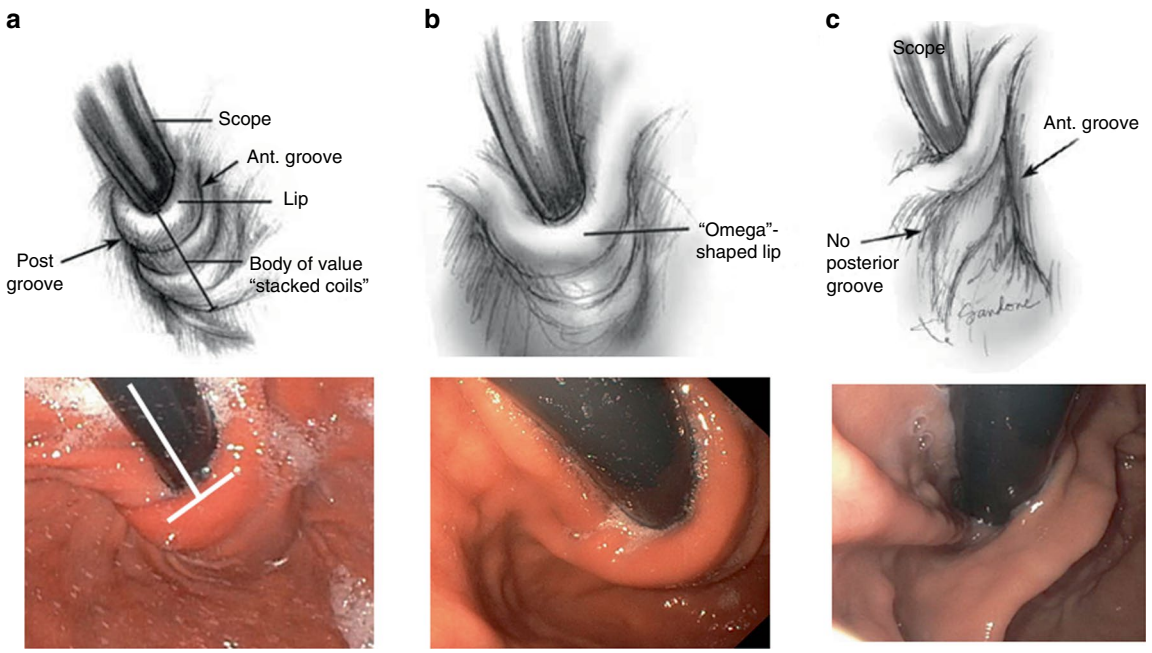

Fig. 1 Surgical fundoplication techniques. (**a**) A complete 360 degree Nissen fundoplication creates a nipple valve. On retroflexed endoscopic view the lip of the valve should be thin, the body of the valve should have a "stacked coils" appearance in alignment with the long axis of the endoscope, and the valve should adhere tightly to the scope. The posterior groove will be deep and anterior groove will be shallow. White lines depict the appropriate orientation of the gastric folds as just below the diaphragm and directed perpendicular to the endoscope and parallel to the diaphragm. (**b**) The Toupet fundoplication is a partial 270 degree posterior wrap which creates a flap valve. The lip of the valve should be thick and "omega" shaped and the valve should be moderately adherent to the scope. Both the anterior and posterior groove should be shallow. (**c**) The Dor fundoplication is a partial 180 degree anterior wrap which creates a flap valve. The lip of the valve should be wide and "S" shaped, and the valve should be moderately adherent to the scope. The anterior groove should be shallow, and there is no posterior groove. Drawings borrowed from Jobe et al. [22]. Endoscopic images courtesy of the Esophageal Center at Northwestern

larly in comparison to the pharmacologic alternative of histamine-2 receptor antagonists at that time [13, 14]. In 1991, the less invasive laparoscopic approach to fundoplication was introduced and quickly became the standard in antireflux surgery [8]. Accordingly, the volume of antireflux surgeries increased from 4.4 in 1990 to 15.7 in 1999 per 100,000 adults among the Nationwide Inpatient Sample database [15–17]. However, as concerns regarding the durability and side effects of laparoscopic fundoplication surfaced along with the increasing accessibility to PPIs, the surge in antireflux surgery declined by 30% in the ensuing years [15, 18, 19]. Between 2005 and 2010, the annual volume of elective open or laparoscopic fundoplication plateaued at approximately 5.3 cases per 100,000 adults, equating to approximately 19,000 surgical fundoplications [20, 21].

Laparoscopic fundoplication technique

The major tenants of surgical fundoplication are to return the esophagogastric junction to an intraabdominal location, close the hiatal opening and create a one-way valve. Laparoscopic fundoplication may be performed via a complete or partial technique. Laparoscopic Nissen fundoplication is a 360-degree fundoplication after crural closure, and aims to circumferentially plicate the stomach to the esophagus in order to strengthen the esophagogastric junction valve (Fig. **1a**) [22]. The two most common partial laparoscopic fundoplication techniques include a 270-degree posterior (Toupet) fundoplication sutured to the crura and esophagus which creates a flap valve while leaving the anterior esophagus exposed to allow for radial expansion (Fig. **1b**), and a 180-degree anterior (Dor) fundoplication sutured to the esophagus and right crus which restores the angle of His and creates a flap valve mechanism (Fig. **1c**) [22, 23].

PREOPERATIVE EVALUATION

Appropriate patient selection for fundoplication is integral to achieving positive postoperative outcomes (Table **1**). Objective documentation of pathologic acid reflux, either by endoscopic evidence of erosive disease or pH monitoring, is essential [5, 6, 9]. The primary purposes of preoperative esophageal manometry are to rule out contraindications to antireflux surgery and to assess esophageal contractile vigor and reserve. There is no distinct cut-off for risk of dysphagia based on peristaltic function after a fundoplication as most dysphagia is related to underlying anatomical and mechanical issues related to the wrap. That being said, absent contractility is an absolute contraindication for a fundoplication as this creates pseudo-achalasia. Thus, the degree of ineffective peristalsis that is tolerable before fundoplication should be based on a case-by-case evaluation. There are some data supporting that augmentation after multiple rapid swallows and a lack of preoperative dysphagia are indicators of a reduced likelihood of dysphagia [9]. Therefore, a surgeon may select a partial fundoplication technique in the setting of reduced

Table 1 Diagnostic evaluation prior to antireflux surgery

Diagnostic testing	Required	Reason to test	Results in support of considering antireflux surgery	Recommending groups	Comments
pH monitoring off of PPI	Yes	Document pathologic acid reflux; associate reflux events with symptoms	Pathologic acid exposure; positive symptom-reflux association in context of other abnormalities associated with pathologic reflux (e.g., large hiatal hernia)	ACG, AGA, EDAP	Not needed if previous evidence of Los Angeles Grade C or D esophagitis or long-segment Barrett's esophagus
Upper GI endoscopy	Yes	Evaluate for signs of erosive disease; assess EGJ anatomy; exclude non-GERD etiologies	Los Angeles Grade C or D esophagitis; long-segment Barrett's esophagus	ACG, AGA, EDAP	
Esophageal manometry	Yes	Exclude achalasia; assess esophageal peristaltic reserve	Defective antireflux barrier; intact esophageal peristalsis	ACG, AGA, EDAP	Absent contractility is a contraindication to antireflux surgery
Barium esophagram	No	Evaluate overall anatomy	Possibly a large hiatal hernia	EDAP	
Gastric emptying study	No	Evaluate for delayed gastric emptying	Normal gastric emptying		Should perform in the setting of bloating and dyspeptic symptoms that are not otherwise explained, and when considering surgical revision in setting of refractory GERD

ACG American College of Gastroenterology, *AGA* American Gastroenterological Association, *EDAP* Esophageal Diagnostic Advisory Panel, *GERD* gastroesophageal reflux disease, *GI* gastrointestinal, *PPI* proton pump inhibitor

peristaltic vigor [5, 6, 9]. Barium esophagram is a useful preoperative study to evaluate the foregut anatomy [9]. Additionally, a gastric emptying study should be performed if there is suspicion of gastroparesis, as this will also tailor the type of antireflux intervention [9].

COMPLICATIONS OF LAPAROSCOPIC FUNDOPLICATION

Complication following laparoscopic fundoplication can occur in both the acute and prolonged settings (Tables **2** and **3**) [24].

Acute complications of laparoscopic fundoplication

The reported 30-day surgical mortality rate of laparoscopic fundoplication ranges from 0.1% to 0.2% [24–27]. In a recent population-based cohort study of 2655 patients who underwent a primary laparoscopic fundoplication between 2005 and 2014, 4.1% of patients had a defined complication within 30 days of surgery, which included infection (1.1%), bleeding (0.9%) and esophageal perforation (0.9%) [27]. Acute-onset dysphagia is also common, affecting approximately 50% of patients, and presumed to be a consequence of edema and inflammation caused by the surgery. Acute postoperative dysphagia is managed with dietary modification and reassurance, and typically resolves within 3 months [28–30].

Prolonged complications of laparoscopic fundoplication

Beyond the acute postoperative setting, patients are susceptible to a multitude of prolonged complications which may impair quality of life [24]. Prolonged complications following fundoplication can be separated into structural complications of the fundoplication or functional abnormalities.

Structural complications of fundoplication

Structural complications following fundoplication occur in up to 30% of cases and are often related to surgical positioning or construction of the wrap [27]. The esophagogastric junction is a complex anatomical area that is subject to mechanical stress related to the gastroesophageal pressure gradient and its dynamic nature that allows it to move axially during swallowing and reflux. Thus, the durability of a fundoplication weakens over time, and is exacerbated by intermittent abdominal strain such as from nausea, vomiting, coughing, trauma, abdominal exercises, heavy lifting and weight gain/obesity. These stressors increase susceptibility to wrap disruption, herniation or slippage. Additionally, recreation of an antireflux barrier that can balance antegrade and retrograde bolus flow is difficult and can result in either a tight or loose wrap. Given these anatomical and physiologic issues, it is not surprising that patients can present with post-fundoplication symptoms and structural complications [24].

Structural laxity of fundoplication. Laxity of the fundoplication can progress to various structural dysfunctions of the fundoplication (Fig. **2**), as described by the Hinder and Horgan classifications [31, 32]. A common dysfunction is hiatal herniation where the esophagogastric junction is displaced proximally through the hiatus (Fig. **2c**). Herniation can result from partial or complete wrap disruption, or slippage of stomach proximal to the fundoplication in the setting of an intact or partially disrupted wrap (Fig. **3**). When the crural repair is disrupted the fundoplication itself may also herniate into the chest and redundance of a

Table 2 Rates of complications following antireflux surgery

Complication	Reported rates
Primary fundoplication	NIS database 2010 $n=18,780$
Acute postoperative complications (within 30 days)	4.1% [27] ($n=769$)
30-Day surgical mortality	0.1 to 0.2% [26, 27] ($n=19$ to 38)
Infection	1.1% [27] ($n=207$)
Bleeding	0.9% [27] ($n=169$)
Esophageal perforation	0.9% [27] ($n=169$)
Acute postoperative dysphagia	50% [28, 30] ($n=9390$)
Failure of fundoplication: wrap herniation, pouch formation, paraesophageal herniation	2 to 23% [23, 27] ($n=376$ to 4319)
Post-fundoplication stenosis	10% ($n=1878$)
Post-fundoplication dilation rate	2.8% [23] ($n=530$)
Gas-bloat syndrome	10 to 32% [23, 42, 43] ($n=1878$ to 6010)
Esophageal dysmotility	
Chest pain	
Diarrhea	18 to 33% [40, 44] ($n=3380$ to 6197)
Secondary fundoplication	
Acute postoperative complications (within 30 days)	23.4% [27]
30-Day surgical mortality	1% [35]
Infection	6.5% [27]
Bleeding	5.2% [27]
Esophageal perforation	6.5% [27]
Dysphagia	24.7% [27]
Magnetic sphincter augmentation	
Acute postoperative complications	0.1% [53–55]
Postoperative mortality	0% [53–55]
Device erosion	0.15% [53–55]
Rate of device removal	2.7% [56]
Dysphagia	7.0% [57]
Bloating	10.0% [57]

The *n* values reported for primary fundoplication extrapolated from Nationwide Inpatient Sample (NIS) database volume of 18,780 elective surgical fundoplications performed in 2010 [16]

fundoplication can also progress to a paraesophageal hernia component (Fig. 2b).

Patients with a failed fundoplication due to structural laxity will often present with recurrence of or persistent GERD symptoms including heartburn, regurgitation and erosive esophageal disease such as peptic stricture formation or reflux esophagitis. Recurrent GERD is the primary indication for reoperation and is more common among females, older patients and patients with a greater number of comorbidities [27, 33]. Patients with laxity of the fundoplication and herniation may also present with obstructive symptoms related to compression within the hiatus or as a consequence of the paraesophageal component causing extrinsic compression. It is not uncommon to have both GERD and obstructive symptoms with wrap herniation.

Post-fundoplication stenosis. Persistent dysphagia due to fundoplication-related stenosis without herniation may occur in 10% of cases [33] and is secondary to a very tight or long fundoplication (Fig. 4) [23, 24, 29]. This is usually the result of poor positioning of the wrap or construction of a tense fundoplication wrap even when a bougie is used during the fundoplication component of the operation [27]. Other structural sources of persistent dysphagia include excessive angulation at the esophagogastric junction and intraluminal penetration of prosthetic surgical material [23].

Evaluation. Upper gastrointestinal endoscopy and barium esophagram are the primary diagnostic modalities used to evaluate for a structural complication following fundoplication (Fig. 5). Endoscopy is useful in assessing for the presence of esophagitis and also in appraising the location, orientation and integrity of the post-fundoplication valve. When assessing the fundoplication in the retroflexed position, the gastric folds of the fundoplication should be located just below the diaphragm and directed perpendicular to the endoscope and parallel to the diaphragm (Fig. 1). The relationship between the fundoplication and crura is crucial in defining abnormalities of the fundoplication. Presence of gastric folds above the diaphragm indicates herniation and this may represent slippage of the stomach through the fundoplication or disruption of the wrap. Slippage can occur in the setting of an intact or partially disrupted wrap. Absence of fundoplication folds on retroflexion indicates total disruption of the fundoplication. Endoscopy may also be useful in assessing for a paraesophageal hernia, where the herniated pouch of stomach will be seen next to the fundoplication folds. Retention of food and fluid in the esophagus, angulation at the distal esophagus and difficulty passing the endoscope may indicate proximal slippage of the fundoplication or stenosis at the wrap.

Barium esophagram provides valuable and complimentary information to endoscopy. Barium esophagram can help to define the anatomy and location of the fundoplication, better appreciate a paraesophageal hernia and provide valuable information on esophageal emptying [29]. Further body imaging with chest computed tomography scans and magnetic resonance imaging can be helpful in defining anatomy, the degree of crural disruption and the extent and type of herniation. When persistent GERD is suspected, esophageal pH monitoring is useful to assess the pattern and burden of reflux, and to establish a congruence between reflux episodes and symptoms as recurrent symptoms may not be recurrent acid reflux. Endoscopy may not be able to determine whether the wrap is tight in the context of a normal-appearing wrap and the functional lumen imaging probe (FLIP) may be useful to document abnormal distensibility of the esophagogastric junction. In addition, esophageal manometry may be useful to assess postoperative integrated relaxation pressures (Figs. 3 and 4). While there are limited data on normal manometric values after fundoplication,

Table 3 Mechanisms of post-fundoplication symptoms

Mechanism	Post-fundoplication symptoms						
	Dysphagia	Regurgitation	Heartburn	Esophagitis stricture	Chest pain	Dyspepsia/ Gas-bloat	IBS/Diarrhea
Mechanical							
Tight fundoplication; no hernia	++	+	−	−	++	++	−
Disrupted fundoplication/laxity; no hernia	+	++	++	++	+	−	−
Slipped wrap with/without hernia	++	++	+	++	++	+	−
Paraesophageal herniation type II/III/IV (new/recurrent)	++	++	++	++	+	+	−
Esophageal dysmotility							
Primary or pseudo-achalasia	++	++	+	−	++	+	−
Hypercontractility/esophageal spasm	+	+	+	−	+	−	−
Reduced preoperative esophageal peristaltic vigor	+	+	+*	+*	+*	−	−
Functional							
Hypersensitivity/functional esophageal syndrome	+	+	+	−	+	+	+
Vagal nerve injury	−	+*	+*	+*	+*	++	+
Accelerated gastric emptying	−	−	−	−	−	+	++

Association of mechanism with symptom if present: ++ strongly associated, + may be associated, − likely not associated
*May be associated if abnormal reflux is occurring due to a disrupted wrap, but if abnormal reflux is not occurring likely not associated

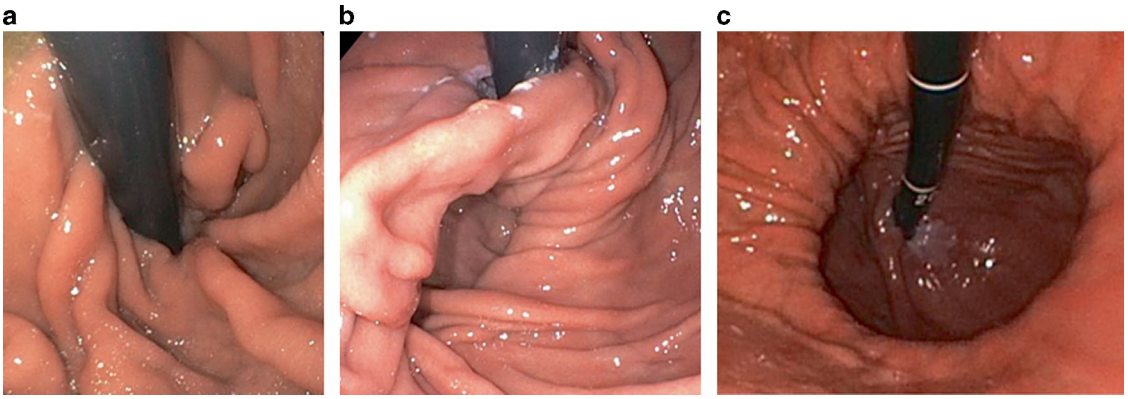

Fig. 2 Endoscopic views of fundoplication complications associated with disruption. Endoscopic Views of fundoplication complications associated with disruption. **a** shows that the wrap is disrupted and the folds are more parallel with the endoscope. In **b** the wrap is partially intact and there is disruption of the crural repair and a paraesophageal hernia tracking along side of the wrap and into the chest. **c** is a frank recurrence of the hernia with only a hint of the remnant wrap noted deep in the type III hernia. Courtesy of the Esophageal Center at Northwestern

integrated relaxation pressure values are slightly higher after a normal fundoplication without symptoms [34]. Thus, the interpretation of integrated relaxation pressure in the evaluation of post-fundoplication symptoms can be difficult and using cut-off to diagnose esophagogastric junction outflow obstruction may be problematic. However, if the values are very high with an elevated intrabolus pressure, and the elevation in integrated relaxation pressure persists despite position change, a diagnosis of obstruction can be made. Distinguishing primary achalasia from pseudo-achalasia from the fundoplication may be possible by evaluation of the preoperative and postoperative peristaltic pattern. In a report from 1986, the amyl nitrite inhalation test was able to distinguish mechanical from neurogenic obstruction [35].

Management. When post-fundoplication symptoms are due to persistent gastroesophageal reflux, the management options include medical antireflux therapy, endoscopic dilation of peptic stricture if present and surgical revision of the fundoplication [29]. Surgical revision should be reserved as a last-resort option for patients with significant symptom burden that is not controlled by PPI and/or endoscopic therapy and evidence of structural abnormality. If symptoms and complications are controlled by PPI therapy, surgical revision may not be necessary unless there is an overt surgical indication. Surgical revisions are more complex than the primary fundoplication, in part related to adhesion formation and altered anatomy, and the proportion of reoperation interventions that can be performed laparoscopically declines

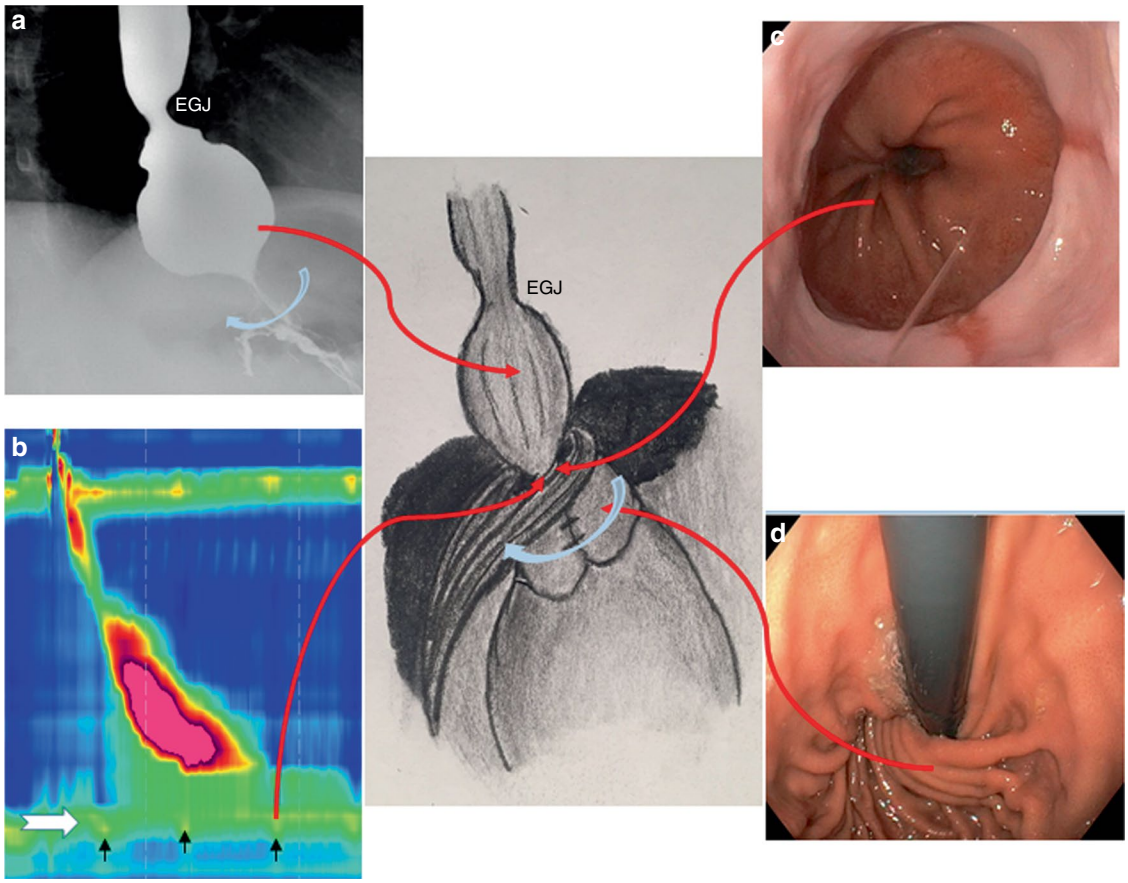

Fig. 3 Slipped Nissen with frank herniation above the diaphragm and a partially intact wrap below the diaphragm depicted on esophagram (**a**), high-resolution manometry (**b**), and Endoscopy (**c**-front view, **d**-Retrofelxed view. The red lines show the corresponding anatomic locations. **a** represents the esophagram showing the herniation and the tight wrap [rotating blue arrow] with an elevated intrabolus pressure between the end of the distal esophagus and the diaphragm. The wrap is located at the white arrow and the obstruction is at the diaphragm (black arrows showing crural contraction). **c** and **d** are the endoscopic images. Note the esophagitis in this patient who presented with food impactions and reflux symptoms. Courtesy of the Esophageal Center at Northwestern

with each subsequent reintervention. The reported success rate of subsequent revisions is lower than for the primary fundoplication, and continues to decrease with subsequent reoperations [31, 36]. The mortality rate for reoperation is approximately 1%; however, complications of all types (perforation, postoperative leak, gastrotomy, vagal nerve injury and treatment failure) is significantly increased, particularly for multiple time redos [37, 38]. In a study of 940 patients undergoing primary or redo antireflux surgery fewer patients were satisfied with subsequent reinterventions at follow-up (excellent satisfaction following: primary antireflux surgery 91%, first redo 76%, second redo 49%, and third redo 33%). Similarly, a higher proportion of patients were taking acid suppression at follow-up with subsequent reintervention (primary 24%, first redo 46%, second redo 67% and third redo 78%) [36].

The most common reoperative intervention includes takedown of the previous fundoplication, redo fundoplication and repair of a recurrent hiatal hernia if present [36, 39, 40]. Choice of reoperation will be helped by an assessment of peristaltic function and gastric emptying. If a shortened esophagus contributed to the primary failure, a redo fundoplication with Collis gastroplasty should be performed. A thoracic approach should be considered in cases with very short esophagus (for adequate mobilization) or in a multiple redo situation (approaching the hernia through "virgin" tissue planes). In cases of significantly reduced esophageal peristalsis, esophagectomy may be considered as a last resort. Roux-en-Y reconstruction may be an option after a failed primary or reoperative fundoplication (Table 4) [41]. It is also important to note that Roux-en-Y gastric bypass should be a primary antireflux intervention choice for patients with PPI-refractory GERD and a body mass index over 35 kg/m^2 or obesity with obesity-related comorbidities such as diabetes or hypertension [42].

In the case of a tight fundoplication and an intact wrap below the diaphragm, endoscopic dilation is an option. According to case series, endoscopic dilation relieves symptomatic dysphagia in up to two-thirds of cases, of which 75% of patients only require one session of dilation therapy [29]. Most of these reports describe bougie dilations to a mean diameter of 18 mm (54 French gauge) [28, 30]. Despite these favorable reports, in our personal experiences, bougie dilation and through the scope balloon dilation for tight post-fundoplication stenoses are often unsuccessful. Pneumatic dilation, similar to protocols used for achalasia, is also attempted if the anatomy is uncomplicated with variable success rates.

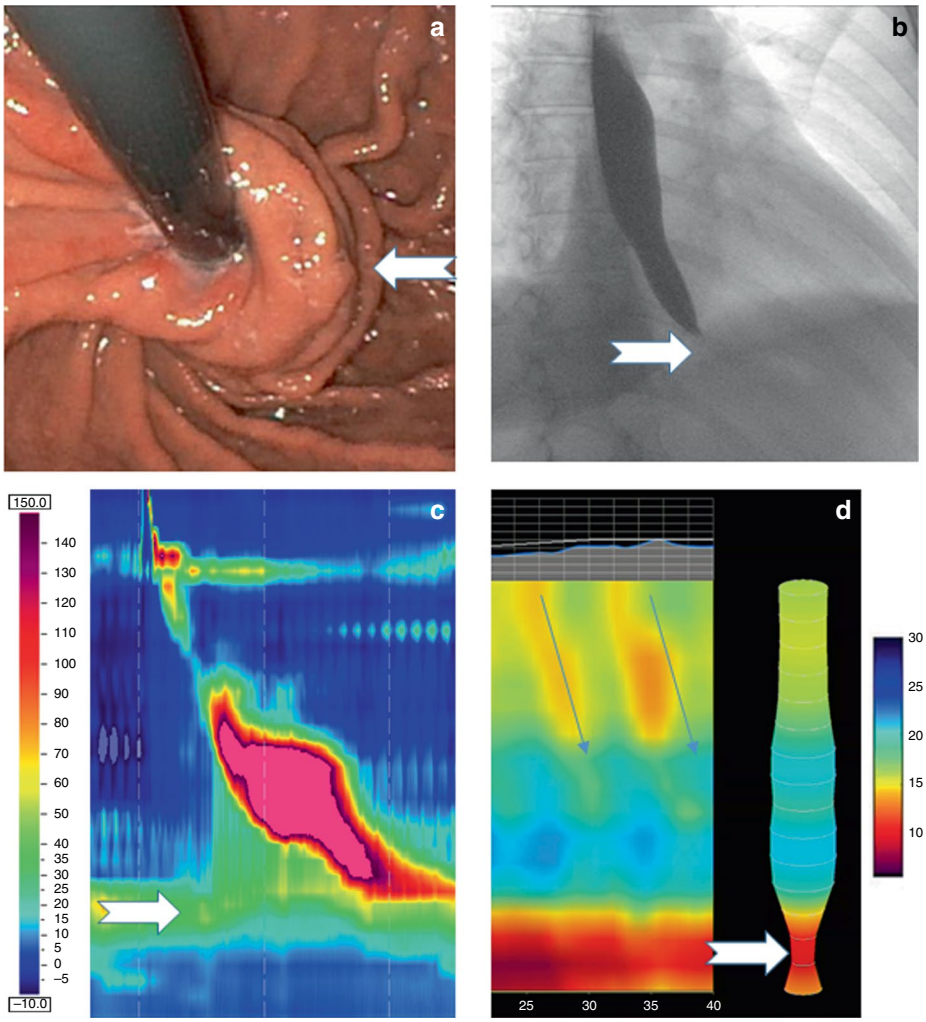

Fig. 4 A fundoplication associated with dysphagia and food impaction**.** The large white arrow is identifying the esophagogastric junction (EGJ) and location of the wrap. **a** is a tight fundoplication that appears intact and outside of being slightly long is relatively normal appearing. The esophagram in **b** supports minimal emptying and exhibits a tight EGJ at the diaphragm. **c** is a highresolution manometry with high intrabolus pressure compartmentalized between the peristaltic contraction and the wrap and this is associated with a high integrated relaxation pressure of 31.5 mmHg. **d** represents a new approach using FLIP-panometry that defines EGJ opening dimensions and also provides evidence of motor function by assessing changes in diameter as opposed to pressure. The scale in **c** is pressure (mmHg) while the scale in **d** is diameter (mm). Courtesy of the Esophageal Center at Northwestern

Functional complications following antireflux surgery

Upper gastrointestinal symptoms following fundoplication in the absence of mechanical or anatomic abnormalities are common, and unfortunately the mechanisms are often not well understood [33, 43].

Dysphagia due to esophageal dysmotility. Esophageal dysmotility is a less common source of post-fundoplication dysphagia. When a structural etiology of dysphagia is not forthcoming on endoscopy or barium esophagram, esophageal manometry should be pursued. In the setting of ineffective esophageal motility, functional dysphagia may arise from insufficient esophageal peristaltic reserve or bolus transit to overcome the obstructive effect of the fundoplication. Achalasia that was missed preoperatively, or a pseudo-achalasia pattern that developed postoperatively, is also another potential source of post-fundoplication dysphagia. Additionally, a hypercontractile pattern can be seen after fundoplication related to an esophageal response to obstruction. It can be difficult to determine whether the hypercontractility is a normal response or pathologic, and empiric trials of smooth muscle relaxants may be helpful.

Gas-bloat syndrome. Gas-bloat syndrome may present with abdominal bloating, inability to burp, postprandial fullness, nausea, flatulence, inability to vomit and abdominal pain [44]. Gas-bloat is more common with complete laparoscopic Nissen fundoplication than with partial fundoplication [45]. The mechanism of gas-bloat is thought to arise from an inability to vent gas from the stomach into the esophagus and may be related to the impaired relaxation of the recreated esophagogastric junction valve in response to gastric distension and an alteration in receptive gastric relaxation and accommodation. In addition, patients may develop aerophagia and supragastric belching in an attempt to palliate these uncomfortable symptoms [23, 24, 29]. There is a

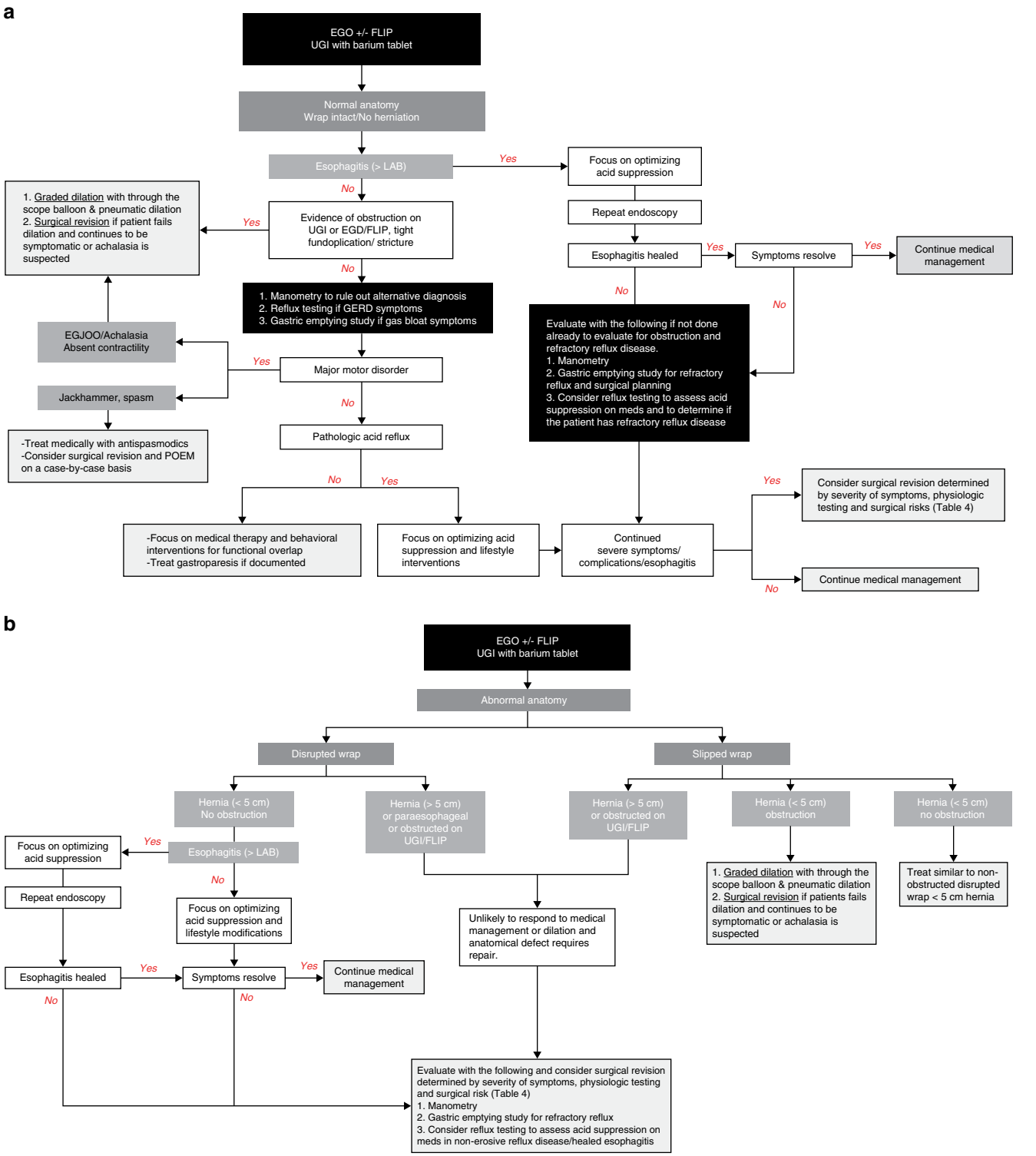

Fig. 5 Approach to patient with symptoms after an antireflux procedure. (**a**) Approach to patient with symptoms after an antireflux procedure with normal anatomy on esophagogastroduodenoscopy (EGD) and/or upper GI (UGI) series. (**b**) Approach to patient with symptoms after an antireflux procedure with abnormal anatomy on EGD and/or UGI series. Symptoms may be obstructive: dysphagia, esophageal regurgitation, gas-bloat, chest pain or food impaction. Symptoms may be associated with abnormal reflux: heartburn, chest pain, regurgitation

paucity of data to guide the management of gas-bloat syndrome, and generally lifestyle modifications are recommended. These include avoidance of gas-producing foods and carbonated beverages, eating slowly to prevent aerophagia, tobacco cessation and using gas-reducing agents (i.e., simethicone) and prokinetic drugs [23, 24, 29]. While revision surgery to convert from a complete to an incomplete fundoplication may be beneficial, supportive data are lacking. Clinicians must be mindful to differentiate

Table 4 Surgical revision options

Surgical revision	When to consider	Technical considerations
Redo fundoplication	Peristalsis intact and no evidence of gastroparesis	Must completely undo previous fundoplication and reduce hernia sac. Careful dissection to avoid vagal nerve injury
Redo fundoplication with Collis gastroplasty	Peristalsis intact and no evidence of gastroparesis, with short esophagus	Thoracic approach should be considered when very short esophagus discovered on pre-op testing or in multiple redo setting
Roux-en-Y	BMI >35 kg/m^2 and/or gastroparesis	May require partial gastric remnant resection due to ischemia
Esophagectomy	Absent peristalsis and gastroparesis	Consider pyloroplasty

gas-bloat syndrome from mechanical small bowel obstruction, peptic ulcer disease and delayed gastric emptying, and should have a low threshold to order a gastric emptying scan during the workup of the post-fundoplication patients especially those with bloating as a dominant symptom. Gastric emptying of solids may be delayed in the setting of vagal nerve injury, in which case treatments aimed at relaxing the pylorus such as pyloroplasty, botulinum toxin or gastric peroral endoscopy myotomy may be considered [29].

Chest pain. Although post-fundoplication chest pain is common, the mechanism is not well understood. Potential etiologies include mechanical stimulus from distension or contraction, or chemical stimulus from acidic or non-acidic esophageal exposure. Workup should focus on assessing recurrent GERD and evaluating for an esophageal motor disorder.

Diarrhea. Diarrhea is also a frequent complication of fundoplication affecting 18 to 33% of patients [46]. While the mechanism of post-fundoplication diarrhea is not well understood, suggested mechanisms include accelerated gastric emptying, vagal nerve injury, postoperative dietary modifications and unrecognized pre-existing irritable bowel syndrome [29, 31, 47, 48]. Concomitant cholecystectomy reportedly increases the risk for postoperative diarrhea [29].

MAGNETIC SPHINCTER AUGMENTATION

Biomechanical augmentation of the lower esophageal sphincter by use of a magnetic reinforcing appliance was first described in 2008 as a novel approach to manage GERD [49, 50]. Four years later, the LINX Reflux Management System, a magnetic sphincter augmentation device for GERD, was FDA (Food and Drug Administration) approved for refractory GERD. The magnetic sphincter augmentation device is a ring of magnets that are surgically, typically laparoscopically, placed circumferentially around the esophagogastric junction to augment the lower esophageal sphincter and function as a two-way valve to allow bolus transit into the stomach and allow for belching and vomiting [51]. Magnetic sphincter augmentation is often contrasted to laparoscopic fundoplication as a fundic-sparing reversible, reproducible and technically simple antireflux intervention that does not alter gastric anatomy [42,52–55]. While magnetic sphincter augmentation is not FDA approved in the setting of hiatal hernias larger than 3 cm, a recent prospective multicenter study of 200 patients undergoing magnetic sphincter augmentation with repair of hernias larger than 3 cm reported favorable postoperative outcomes [56].

To date, there are no reports of perioperative deaths or life-threatening complications following magnetic sphincter augmentation implantation. The most feared complication of magnetic sphincter augmentation is device migration and erosion into the esophagus. In a study of 3283 patients who underwent magnetic sphincter augmentation, 0.15% ($n=5$) had device erosion. All cases of device erosion presented non-emergently with dysphagia or odynophagia, and were removed endoscopically or laparoscopically without complication. In a few cases, patients subsequently underwent an uncomplicated laparoscopic fundoplication [57–59]. The original magnetic sphincter augmentation device was magnetic resonance conditional only up to 0.7 Tesla; however, the new version is safe up to 1.5 Tesla.

In a review of 1048 patients who underwent magnetic sphincter augmentation, the overall perioperative complication rate was 0.1% and considered to be unrelated to the device. Endoscopic dilation was performed in 5.6% of patients, the majority being within 90 days after the operation [60]. In a review of 3283 patients, the overall rate for device removal was 2.7%, most commonly for dysphagia. A prospective observational study comparing magnetic sphincter augmentation to laparoscopic fundoplication reported a higher rate of dysphagia (10.6% vs 7.0%) and bloating (31.9% vs 10.0%) among the laparoscopic fundoplication group compared to the magnetic sphincter augmentation group [61, 62]. A meta-analysis comparing magnetic sphincter augmentation to laparoscopic fundoplication reported a significantly reduced risk of gas-bloat with the magnetic sphincter augmentation (relative risk 0.71, 95% confidence interval 0.54 to 0.94) [63]. Thus, initial data support a more favorable safety profile of the magnetic sphincter augmentation; however, follow-up over a longer period is needed to understand the actual long-term outcomes (Table **2**) [61, 64].

SUMMARY

In summary, antireflux surgery is indicated for patients with PPI-refractory GERD or PPI intolerance in the context of documented pathologic gastroesophageal reflux. Although effective, complications following antireflux surgery are common, particularly following laparoscopic fundoplication. These include fundoplication failure, dysphagia related to stenosis as well as various functional gastrointestinal symptoms. Rates of complications seem to be lower with magnetic sphincter augmentation, though further outcomes data are needed.

Post-fundoplication complications are one of the most challenging entities gastroenterologists and foregut surgeons manage in the esophageal field, and should be approached from a multidisciplinary standpoint. Generally, upper gastrointestinal symptoms of dysphagia, heartburn and regurgitation following fundoplication should be

first assessed with endoscopy and barium esophagram to evaluate for anatomic disturbances such as a slipped fundoplication, herniated wrap, paraesophageal hernia, tight wrap and presence of erosive reflux disease. Ambulatory pH monitoring is useful when recurrent GERD is suspected. Generally, medical and endoscopic options are futile, as the majority of complications are post-surgical and structural in nature (Fig. 3). The key to favorable outcomes after reoperations is comprehensive preoperative assessment and appropriate patient selection, otherwise the success and prognosis may be poor.

CONFLICT OF INTEREST

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