Surgical Management for Gastroesophageal Reflux Disease

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INTRODUCTION

Gastroesophageal reflux disease (GERD) is one of the most prevalent gastrointestinal disorders. In a sample US adult population, 20% reported symptoms of GERD once per week. Quality of life for patients with GERD is severely affected and is lower than the quality of life for patients with congestive heart failure or angina.

GERD represents a derangement of the physiologic and anatomic components of the antireflux barrier. The exact cause is unknown. In healthy individuals, gastric distension can cause transient relaxation of the lower esophageal sphincter (LES). In up to 80% of patients, inappropriate transient LES relaxation is the principal mechanism for pathologic reflux of gastric contents into the esophagus. In severe GERD, other contributing factors have been identified, including hypotensive LES, diaphragmatic crural dysfunction, and hiatal hernia.

GERD is defined by the presence of (1) reflux esophagitis (based on Los Angeles classification system), and/or (2) reflux symptoms that are sufficient to impair quality of life, and/or (3) risk of long-term complications (eg, esophageal stricture, Barrett’s esophagus). Common presenting symptoms include heartburn and regurgitation. Atypical laryngeal-pulmonary symptoms, such as cough and hoarseness, often require a multidisciplinary approach to reach the correct diagnosis.

Different patterns of clinical behavior are observed among patients with GERD. In most patients, symptoms play a central role in clinical presentation and endoscopic evidence of pathologic reflux is lacking. This clinical presentation is referred to as endoscopy-negative reflux disease or nonerosive reflux disease. Approximately 20% to 30% of patients present with reflux esophagitis and have endoscopic findings such as erosions, ulcers, and stricture formation. Barrett’s esophagus is present in 5% to 15% of patients and predisposes to adenocarcinoma.

REFLUX ESOPHAGITIS

CASE 1 PRESENTATION

A 52-year-old man with an 8-year history of GERD is referred to you by a gastroenterologist. Endoscopy at the time of diagnosis revealed esophageal erosions. Since then, the patient has been compliant with lifestyle changes and medical therapy; the patient’s current medication is omeprazole 80 mg/day. Although his symptoms have improved with medical therapy, the patient has continued to experience heartburn at least twice weekly. In addition, he has experienced dysphagia for solid food for the past 3 months.

• Are this patient’s symptoms typical for GERD?

CLINICAL FEATURES OF REFLUX ESOPHAGITIS

This patient’s clinical description is consistent with reflux esophagitis, a clinical pattern of GERD characterized by mucosal erosions evident on esophagogastroscopy. Reflux esophagitis can range from mild disease with superficial erosions to severe disease complicated by ulcers and strictures.

Heartburn, as defined by a burning feeling originating from the lower chest or stomach and radiating toward the lower neck, is the most common presenting symptom in GERD. Symptoms of dysphagia occur less commonly than heartburn in patients with GERD. Dysphagia has a point prevalence of 14% in the community and 37% in patients with reflux esophagitis. The main concern with dysphagia is whether the symptom is related to esophageal adenocarcinoma. Dysphagia has a low predictive value for cancer. Thus, not all patients with this symptom require endoscopic evaluation. However, new symptom onset, increasing intensity of symptoms, and a lack of response to medical therapy warrant further investigation to rule out malignant etiology.

• How is failure of medical therapy for GERD defined?
FAILURE OF MEDICAL THERAPY

Failure of medical therapy is defined by the outcome measured. Control of symptoms is the outcome most frequently used in clinical practice. However, symptom frequency, severity, and duration are all important characteristics that must be determined to assess failure of therapy. It is generally agreed that 2 or more symptoms per week are associated with impaired quality of life.7

Medical therapy with a proton pump inhibitor (PPI) is effective for control of symptoms in approximately 90% of patients with reflux esophagitis;19,20 however, complete elimination of symptoms does not guarantee normalization of intraesophageal pH. In a recent study by Milkes et al,21 50 patients with GERD who were asymptomatic while taking a standard-dose PPI underwent 24-hour pH studies; 25 patients (50%) had abnormal pH profiles (DeMeester score > 14.7). In patients with reflux esophagitis, control of esophageal acid exposure or endoscopic response, as defined by complete healing of erosions after 6 weeks of therapy, is a more desirable outcome. PPI therapy can produce healing of erosions in more than 90% of patients with Los Angeles grade A or B GERD, but failure rates as high as 40% are reported with severe erosions (Los Angeles grades C and D).20

• What is the next step in this patient’s management?

It appears that this patient’s heartburn is not fully responsive to maximal acid suppressant therapy with omeprazole, a PPI, and he now is experiencing dysphagia. New-onset dysphagia unresponsive to medical therapy in the setting of chronic GERD may signify esophageal stricture or malignancy and requires further investigation. Thus, the first step in management of this patient is to repeat endoscopy.

CASE 1 CONTINUED

Repeat endoscopy reveals no gross evidence of malignancy. Multiple erosions extending to half of the circumference of the esophagus are identified. A 4-cm hiatal hernia is present. The distal esophageal lumen appears narrow, but an adult endoscope can traverse the area in question. Multiple biopsies are obtained. Examination of the stomach reveals no mucosal abnormality; however, 600 mL of bilious fluid is seen. The proximal duodenum is normal. Endoscopic dilatation of the distal esophagus is performed. Biopsies are negative for Barrett’s metaplasia and malignancy.

• How would this patient’s esophagitis be graded?
• What is the clinical significance of his hiatal hernia?

GRADING OF ESOPHAGITIS AND IMPLICATIONS OF HIATAL HERNIA

Esophageal erosions have been described by a variety of grading systems. The Savary–Miller classification was the first grading system for reflux esophagitis. Classification by this system requires endoscopic evaluation of the depth of the erosions; however, variability in the interpretation of results is a limitation of this system. For this reason, use of the Savary–Miller classification has been surpassed by the Los Angeles classification,22 first described in 199723 and modified in 1999 (Table 1).6 The Los Angeles classification has good inter- and intraobserver agreement,24 regardless of the experience level of the endoscopist,6,23,24 and the grades correlate with severity of disease. The endoscopic description for case patient 1 is consistent with Los Angeles grade C esophagitis.

Hiatal hernia is closely associated with GERD and is a likely contributing factor in this patient. Not every patient with hiatal hernia experiences GERD, and the mere presence of this condition is not an indication for intervention. In patients with proven GERD, however, the size of the hernia is directly proportional to LES dysfunction, esophageal acid exposure, and grade of esophagitis.25 This relationship is in part explained by altered mechanics of the gastroesophageal junction, where an increase in axial length of herniation is associated with shortening of the high pressure zone of the hiatal hernia.

Table 1. The Los Angeles Classification of Esophagitis by Endoscopic Assessment

<table>
<thead>
<tr>
<th>Grade</th>
<th>Lesion Appearance on Endoscopic Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>≥ 1 mucosal break ≤ 5 mm that does not extend between the tops of 2 mucosal folds</td>
</tr>
<tr>
<td>B</td>
<td>≥ 1 mucosal break &gt; 5 mm long that does not extend between the tops of 2 mucosal folds</td>
</tr>
<tr>
<td>C</td>
<td>≥ 1 mucosal break that is continuous between the tops of ≥ 2 mucosal folds but that involves &lt; 75% of the circumference</td>
</tr>
<tr>
<td>D</td>
<td>≥ 1 mucosal break that involves ≥ 75% of the esophageal circumference</td>
</tr>
</tbody>
</table>

LES and progressive impairment of the diaphragmatic crural component of the gastroesophageal junction. In the case of a nonreducing hernia, esophageal acid clearance is also affected, further contributing to pathologic reflux.

• **Is surgical intervention an appropriate option for managing this patient’s GERD?**

This patient has Los Angeles grade C esophagitis as well as early evidence of esophageal stricture, a known complication of long-standing erosive disease. Failure of medical therapy in this patient is an indication for surgical treatment with fundoplication. New endoluminal modalities are still under investigation and are currently not offered to patients with Los Angeles grade C or D esophagitis or a large hiatal hernia. Current indications for fundoplication include (1) failure of medical therapy to control symptoms or prevent complications and (2) patient preference for an intervention that offers an alternative to life-long medical treatment.

• **What should the preoperative work-up for this patient entail?**

**PREOPERATIVE WORK-UP**

Given the large quantity of residual fluid in this patient’s stomach, gastric dysmotility must be ruled out. The work-up should also include an assessment of esophageal motility, as surgical therapy is the likely next step.

The diagnosis of GERD can be reached based on appropriate symptoms and endoscopic evidence of reflux esophagitis. Gastroparesis or delayed gastric emptying can be a contributing factor in GERD. An analysis of a large body of data indicated delayed gastric emptying as a causative factor in GERD in only a small fraction of patients. However, in patients with GERD refractory to medical therapy and with endoscopic evidence of increased residual gastric fluid, scintigraphic assessment of gastric motor function may be warranted. For this assessment, solid and liquid meals labeled with radioactive isotopes are ingested and the residual meal is measured at fixed time intervals. Using a technetium 99-labeled low-fat meal (egg substitute), assessment of emptying at 1 hour, 2 hours, and 4 hours is 10% or more complete, 40% complete, and 90% complete, respectively.

Preoperative assessment should also include manometric evaluation of the esophagus to ensure adequate motor function and to rule out motility disorders. Four variables are noted: (1) esophageal peristaltic activity (percentage of swallows with progressive contraction sequences, with a normal value > 80%); (2) amplitude of contraction (normal, 30–180 mm Hg); (3) LES resting pressure (normal, < 26 mm Hg); and (4) LES relaxation pressure with swallowing (normal, < 3 mm Hg).

**CASE 1 CONTINUED**

The results of the patient’s gastric emptying study are within normal range (5% retention at 4 hours). Esophageal manometry reveals 90% peristalsis; the esophageal amplitude ranges from 26 to 55 mm Hg, with normal LES resting and relaxation pressures.

• **What surgical procedure would you offer this patient?**

**SURGERY FOR GERD**

The most commonly performed surgery for GERD is Nissen fundoplication. The German surgeon Rudolf Nissen performed the first fundoplication in 1937 for a nonreflux indication, and his procedure remains conceptually the most effective surgical therapy for GERD. This surgery involves “wrapping” the fundus of the stomach around the intra-abdominal esophagus. Many modifications of Nissen’s original procedure have been described in terms of the surgical approach and configuration of the wrap.

**The Ideal Wrap: What is the Evidence?**

**Rossetti modification.** The Rossetti modification of the original Nissen fundoplication involves division of short gastric vessels of fundus to remove tension from the wrap. Although this modification makes theoretical sense and is widely used, available data do not indicate better outcomes with this modification.

**Laparoscopic versus open approach.** When comparing laparoscopic versus open fundoplication for the surgical treatment of GERD, the laparoscopic approach appears to be equally safe and effective for control of symptoms and esophageal acid exposure, is associated with a lower incidence of perioperative complications, and allows for quicker patient recovery time.

**Partial fundoplication.** Many surgeons advocate partial fundoplication in the face of low-amplitude esophageal peristalsis to avoid postoperative dysphagia; however, the results of numerous randomized controlled trials do not support this approach. Individual studies have found a higher incidence of dysphagia with complete fundoplication in the early postoperative period, but these differences seem to dissipate over time. Chrysos et al compared laparoscopic complete fundoplication (Nissen–Rossetti technique, n = 14) with partial fundoplication (Toupet technique, n = 19) in patients with distal esophageal peristaltic amplitude less than 30 mm Hg, a group most likely to experience postoperative dysphagia. Although the partial wrap resulted in a lower incidence of
dysphagia and gas bloat at 3 months, this difference disappeared at 1-year follow-up. This finding might be explained by significant improvement in amplitude of contraction in the distal esophagus in both groups.

Partial fundoplication can be created anterior or posterior to the esophagus. Hagedorn et al\(^{19}\) compared laparoscopic posterior partial fundoplication (Toupet technique, \(n = 48\)) with anterior partial wrap (Watson technique, \(n = 47\)) in a randomized controlled trial and found better control of esophageal acid exposure with posterior fundoplication. Routine calibration with esophageal bougie, routine primary closure of the hiatus, and the use of prosthetic mesh for very large hiatal or paraesophageal hernias are all practices for which data from randomized controlled trials are insufficient but that are supported by most retrospective data.\(^{54-57}\)

**Technique: Laparoscopic Nissen–Rossetti Fundoplication**

In the laparoscopic Nissen–Rossetti fundoplication, the patient is placed in either a supine split-leg or lithotomy position. The operating surgeon stands between the patient’s legs with an assistant surgeon on the left side of the patient. Standardized trocar placement is a useful practice. The abdomen is first insufflated to 15 mm Hg using a Verres needle. With the pneumoperitoneum in place, the camera port is placed 15 cm from the xiphoid process, to the left of the midline through the rectus muscle. A robotic arm can be used if available to drive a 30 degree–angled laparoscope. Operating ports include an 11-mm port placed 11 cm from the xiphoid process along the left costal margin and a 5-mm port placed at approximately the same distance on the right. The assistant surgeon places a 5-mm port 15 to 20 cm from the xiphoid process along the left costal margin; the liver retractor is placed at the same distance on the right. The 4 essential steps in a laparoscopic total fundoplication are: (1) mobilization of the intra-abdominal esophagus, (2) division of the short gastric vessels and gastroepiploic ligament, (3) closure of the hiatus, and (4) creation of the fundoplication.

**Mobilization of the intra-abdominal esophagus.** The first step is division of the proximal gastrohepatic ligament for a short distance to allow visualization of the right limb of the right crus (hereafter referred to as the right crus) of the diaphragm. The avascular plane between the right crus and the adjacent tissues on the left, including the esophagus, are developed by lateral opposing sweeps of the blunt dissecting instruments. Early performance of this step allows the surgeon to divide the tissues superficial to the esophagus, including the phrenoesophageal ligament concomitantly in the next step, carrying the dissection toward the midline and then to the left side in a circumferential manner. The space posterior to the esophagus is then developed. The posterior vagus nerve is identified and retracted anteriorly along with the esophagus. The left limb of the right crus (hereafter referred to as the left crus) of the diaphragm is identified. The plane immediately superficial to the left crus is developed for passage of a Penrose drain. Next, by applying caudal traction on the Penrose drain, mediastinal attachments of the lower esophagus are released to provide a 3-cm length of intrabdominal esophagus.

**Division of the short gastric vessels and gastroepiploic ligament.** With the assistant surgeon lifting the gastroepiploic ligament, the short gastric vessels of fundus are visualized and divided using ultrasonic shears. This division is carried to the angle of His. In the lesser sac, posterior attachments of the proximal stomach to anterior surface of the pancreas are divided to allow creation of a tension-free fundoplication.

**Closure of the hiatus.** Interrupted nonabsorbable sutures are used to approximate the right and left crura of the diaphragm (Figure 1). Pledgets may be used. Care must be taken to avoid a tight closure of the hiatus by visual confirmation of at least a 1- to 1.5-cm gap posterior to the esophagus.

**Creation of the fundoplication.** To ensure proper configuration of the wrap and to avoid twisting the wrap, a point on the posterior fundus approximately 5 cm distal to the angle of His and 2 cm from the greater curvature is identified. This point may be marked by a suture or grasped by the assistant surgeon and passed posterior to the esophagus and retrieved by the surgeon from the right side. A 56-Fr bougie is passed orally into the stomach. The marked point on the posterior fundus is then sutured to the opposing tissue of the anterior fundus (Figure 2). A total of 3 interrupted nonabsorbable sutures are usually required to create a short and floppy wrap. The first 2 sutures include the anterior esophagus. When the wrap is complete, the bougie is withdrawn under direct vision. The Penrose drain and trocars are then removed.

**CASE 1 CONCLUSION**

The patient undergoes laparoscopic Nissen fundoplication without any complication. After surgery, he is allowed to take liquids orally and is advanced to a soft diet prior to discharge on postoperative day 1. His diet is gradually advanced to solids after 3 weeks. The patient experiences mild dysphagia, which may occur in up to 30% of patients in the early postoperative period, but this symptom subsides after 1 month. At 1 year, the patient is free of all symptoms.
NONEROSIVE REFLUX DISEASE

CASE 2 PRESENTATION

A 42-year-old woman with a 4-year history of heartburn and regurgitation is referred to you by a gastroenterologist. Her initial work-up included an upper endoscopy and upper gastrointestinal study that revealed normal esophageal mucosa and a 2-cm hiatal hernia. The patient was diagnosed with GERD based on her clinical presentation. Her heartburn has been controlled with maximal-dose esomeprazole; however, she has persistent regurgitation and seeks nonmedical therapy for control of her symptoms.

• What is the next step in this patient’s management?

This patient’s presentation is consistent with nonerosive reflux disease (endoscopy-negative reflux disease), which affects 55% to 81% of patients with GERD. Once considered a mild form of GERD, nonerosive reflux disease is increasingly viewed as a separate entity with distinct clinical features. Persistent regurgitation on maximal medical therapy is an indication for more invasive treatments such as endoluminal therapies or surgical fundoplication. As a first step for this patient, GERD must be confirmed by a 24-hour pH study.

DIAGNOSIS OF NONEROSIVE REFLUX DISEASE

Approximately 75% of patients who experience heartburn as a major or sole symptom have GERD as the cause. In such patients, an initial trial of medical therapy with acid-suppressing agents can be used as both a diagnostic and therapeutic tool. This approach does not distinguish nonerosive reflux disease from reflux esophagitis, however, because neither the clinical presentation nor the pattern of response to medication differentiates these 2 conditions.

As described by Johnson and DeMeester, 24-hour pH measurement has been the gold standard for diagnosis of GERD for more than 2 decades in patients who have no endoscopic evidence of reflux disease. Six components of physiologic relevance are measured in this assessment (Table 2). For each measured component, a score is calculated, using the following formula (based on mean and standard deviation values obtained from 50 healthy controls):

\[
\text{Component score} = \frac{\text{Patient value} - \text{Mean}}{\text{Standard deviation}} + 1
\]

The formula weighs each component according to dependability and reliability of the measurement, which are inversely proportional to the standard deviation for
the measurement. The 6 component scores are then added to obtain a 24-hour pH composite score, called the DeMeester score. A score greater than 14.72 (ie, the 95th percentile of 50 healthy controls) is considered abnormal.

Intraluminal impedance monitoring is a new modality that measures reflux based on the changes in resistance to electrical flow between 2 electrodes when a bolus of food passes through them. This modality has the advantage of detecting nonacid and superimposed reflux episodes and quantifying the extent of refluxate. With accrual of data from clinical trials and further validity, a combination of pH and impedance measurement will likely be used in the future to increase the sensitivity of diagnosis of all reflux episodes.

CASE 2 CONTINUED

The patient undergoes a 24-hour pH study after discontinuing esomeprazole. The study reveals a DeMeester score of 37 (normal, ≤ 14.72).

• What nonmedical therapeutic options would be appropriate for this patient?

Given the diagnosis of GERD and failure of medical therapy to control regurgitation, this patient may be considered for new endoluminal therapies or surgical fundoplication. Endoluminal therapies are currently not offered to patients with large hiatal hernias (> 3 cm), active Los Angeles grade C or D esophagitis, or Barrett’s esophagus. For case patient 2, a discussion of new endoluminal therapies as alternatives to surgical fundoplication is appropriate if local expertise is available, especially under a research protocol.

ROLE OF ENDOLUMINAL THERAPIES

The US Food and Drug Administration has approved 3 endoluminal procedures for the treatment of GERD: the Stretta® (Curon Medical, Inc., Fremont, CA), EndoCinch™ (Bard Endoscopic Technologies, Billerica, MA), and Enteryx® (Boston Scientific, Natick, MA) procedures. Other procedures are currently being evaluated.

The Stretta procedure uses radiofrequency energy to cause a controlled thermal burn at 85°C at the gastroesophageal junction. The contraction and healing that follow result in narrowing of the gastroesophageal junction. Additionally, this procedure may interfere with vagal afferent fibers originating from the gastric fundus, which are responsible for transient LES relaxations. Several studies have evaluated the short-term outcomes of the Stretta procedure; available data indicate that this procedure improves symptoms, improves quality of life, and decreases medication use. This procedure also decreases esophageal acid exposure, although not as effectively as surgical fundoplication. These data must be interpreted in light of the lack of long-term follow-up and the relatively mild to moderate intensity of GERD symptoms in the patients enrolled in these trials.

EndoCinch is a procedure aimed at internal plication of the stomach by endoscopic application of sutures below the gastroesophageal junction. The 2-year data for this procedure indicate a high failure rate, with 75% to 80% of patients returning to use of medication as therapy. Technical modifications in endoscopic plication are currently underway. Enteryx is a relatively new procedure, which involves intramural esophageal injection of a bulking agent. Long-term data are required before recommending this procedure in the management algorithm of GERD.

CASE 2 CONCLUSION

After discussing the endoscopic options and surgery, the patient chooses to undergo the Stretta procedure. The procedure is uneventful, and the patient is discharged home the same day. At 3-month follow-up, she is free of regurgitation, and the results of her quality-of-life survey have improved. At 1-year follow-up, she continues to be symptom free and takes half the preoperative dose of omeprazole.

BARRETT’S ESOPHAGUS

CASE 3 PRESENTATION

A 50-year-old man with a long-term history of GERD and Barrett’s esophagus is referred to you for surgical evaluation. The patient has received acid suppressant...
therapy since diagnosis and has undergone surveillance endoscopy every 3 years, with multiple biopsies. On his most recent endoscopy (Figure 3), the staff pathologist in your hospital diagnosed low-grade dysplasia.

- **What is the next step in this patient’s management?**

Barrett’s esophagus is a premalignant condition in which a specialized intestinal type of metaplastic columnar epithelium with goblet cells replaces the squamous epithelium of the lower esophagus. Approximately 10% of patients with a long-term history of GERD have Barrett’s esophagus. These patients are at increased risk for developing adenocarcinoma of the esophagus. The absolute risk of esophageal adenocarcinoma in patients with Barrett’s esophagus is estimated at 0.5% per patient-year. Dysplasia is seen in approximately 20% of cases of long-segment Barrett’s (> 3 cm in length) and 12% of cases of short-segment Barrett’s (< 3 cm in length). Dysplasia increases the risk of cancer significantly and may be either low grade or high grade. Histologic diagnosis by 2 separate expert pathologists is recommended because of the relatively high interobserver disagreement and the potential therapeutic implications of the diagnosis of dysplasia. Thus, confirmation by a second pathologist is the next step in the management of this patient.

**CASE 3 CONTINUED**

A second expert pathologist confirms the diagnosis of low-grade dysplasia in the patient.

- **How should this patient be managed?**

**MANAGEMENT OF LOW-GRADE DYSPLASIA**

Management of Barrett’s esophagus involves control of symptoms and esophageal acid exposure as well as prevention of progression to cancer. Management of low-grade dysplasia in the setting of Barrett’s involves the same principles along with frequent endoscopic surveillance.

**Control of Symptoms and Acid Exposure**

Control of symptoms is achieved with the same approach as that for patients with GERD and no evidence of Barrett’s esophagus, that is, with a standard acid-suppressing agent (eg, a PPI) or with surgical fundoplication, if medical therapy is ineffective.

Control of acid exposure in patients with Barrett’s esophagus often requires a higher dosage of medications. In a study by Yeh et al., 62% of patients with Barrett’s esophagus who were taking esomeprazole had abnormal nocturnal intraesophageal pH profiles. These authors suggest using a higher PPI dosage with pH monitoring for confirmation. In patients with Barrett’s esophagus unresponsive to medical therapy, surgical fundoplication is indicated to create an antireflux barrier. Compared with GERD patients without Barrett’s esophagus, fundoplication is equally successful (approximately 90% of cases) in controlling symptoms and preventing esophageal acid exposure in GERD patients with Barrett’s esophagus.

**Prevention of Cancer**

To date, no therapy has been effective for preventing the progression of Barrett’s metaplasia to cancer. Medical therapy has consistently failed to induce regression of dysplasia or prevent progression to cancer. Therefore, the role of medical therapy in the treatment of Barrett’s esophagus is limited to control of symptoms and, with optimal dosing, control of esophageal acid exposure. The role of surgical therapy in cancer prevention in Barrett’s esophagus is also unclear. No study has been able to show a reduction in the incidence of cancer in patients with Barrett’s esophagus following surgical fundoplication. The failure may be in part a result of the limitations of research trials, given the relatively low risk of cancer in this setting. A randomized controlled trial conducted at Veterans Administration institutions with 10-year follow-up comparing medical with surgical therapy failed to provide conclusive evidence, given the lack of adequate power to address this question. To study the effect of surgery on regression of dysplasia, a recent prospective study of 91 patients with Barrett’s esophagus with various degrees of dysplasia compared surgical therapy with medical therapy. The surgical group had a significantly higher rate of histologic regression of dysplasia (36% versus 7%; P < 0.05), although the incidence of cancer was similar in the 2 groups.

**Endoscopic Surveillance**

The current recommendation for cancer surveillance in patients with Barrett’s esophagus and without evidence of dysplasia is performance of an upper endoscopy every 3 years. This recommendation may be revised to every 5 years, given that the guideline was based on earlier data suggesting a higher risk of cancer. With low-grade dysplasia, as in the case patient, annual surveillance endoscopy is recommended.

**CASE 3 CONTINUED**

The patient is maintained on PPI therapy and followed by annual endoscopy with biopsies. After 3 years, he is noted to have high-grade dysplasia confirmed by 2 pathologists (Figure 4). He is referred for surgical consultation.
Is there a role for nonsurgical management in this patient?

MANAGEMENT OF HIGH-GRADE DYSPLASIA

Management of Barrett’s esophagus with high-grade dysplasia is controversial. Given that the prevalence of occult malignancy in Barrett’s esophagus with high-grade dysplasia is approximately 30% to 57%, esophagectomy has been the gold standard of therapy. However, because of the potential morbidity associated with esophagectomy, investigators have examined the role of aggressive endoscopic surveillance, ablative procedures, and more recently endoscopic mucosal resection in the setting of high-grade dysplasia.

The advantage of a surgical approach is early and definitive intervention. It is well documented that survival at 2 years after surgery is significantly reduced when the disease has advanced to full thickness of the wall or spread to surrounding lymph nodes. In a prospective trial of 33 patients with Barrett’s esophagus and high-grade dysplasia, Romagnoli et al demonstrated improvement in cancer-related survival with early surgical treatment compared with expectant nonsurgical treatment (100% versus 53%; P < 0.05).

Proponents of aggressive endoscopic surveillance argue that the nonsurgical approach does not affect survival or lead to delay in diagnosis of cancer. Data on endoscopic surveillance of high-grade dysplasia are mostly derived from nonrandomized studies. In 1 study, Schnell et al followed 75 patients with high-grade dysplasia up to 20 years. Of 75 patients, 12 developed cancer; of these 12 patients, 11 (92%) were considered cured with surgical or ablation therapy.

Ablative procedures use laser or photodynamic therapy to destroy the diseased segment of mucosa. These procedures are associated with up to 30% stricture rate and a risk of adenocarcinoma in untreated areas of Barrett’s mucosa. Randomized controlled trials are required to fully evaluate and better define the role of these modalities in treatment of Barrett’s esophagus.

Circumferential endoscopic mucosal resection is increasingly reported for treatment of Barrett’s esophagus with dysplasia. Advances in technical aspects of the procedure using a dual-channel endoscope and sequential resections have decreased the incidence of incomplete resection and stricture formation associated with this technique. Larger studies with long-term follow-up are required to fully assess the place for these modalities in management of Barrett’s esophagus. Until better data become available, esophagectomy remains the gold standard of therapy for Barrett’s esophagus with high-grade dysplasia.

CASE 3 CONCLUSION

Given his good functional status and lack of significant comorbidities, the patient is deemed a good candidate for esophagectomy. He undergoes a transthoracic esophagectomy, using his stomach as a conduit with a cervical anastomosis. On postoperative day 5, the patient develops an anastomotic leak as is evident by his cervical drain output. He is managed conservatively, and, at 3-month follow-up, his anastomosis has healed. On pathology report, high-grade dysplasia with no evidence of malignancy is found.
SUMMARY

Many aspects of the diagnosis and management of GERD are the subject of ongoing debate. Refinements in 24-hour pH measurement and the addition of newer diagnostic modalities (eg, intraluminal impedance monitoring) will likely improve the sensitivity of the diagnostic work-up. Traditionally, treatment options for GERD were medical or surgical therapies. As data accumulate on the safety, efficacy, and long-term outcomes of newer endoluminal therapies, the role of these modalities in GERD management will be better defined.

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