Complications of Metabolic and Bariatric Surgery for the Gastroenterologist: A Comprehensive Review

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Abstract: By 2035, more than one-half of the global population is expected to have overweight or obesity, amounting to a substantial $4 trillion toll on the global economy. The uptake of metabolic and bariatric surgery has increased worldwide, providing treatment for both obesity and associated disorders of metabolic function. Laparoscopic sleeve gastrectomy and Roux-en-Y gastric bypass are the most commonly performed metabolic and bariatric surgical procedures. Despite advances in surgical techniques, complications are common and can occur long after surgery. This article provides gastroenterologists with a comprehensive compendium for understanding and managing the complications associated with metabolic and bariatric surgery.

Keywords
Bariatric surgery, postoperative complications, endoscopic management, sleeve stenosis, anastomotic leak, marginal ulcer, gastroenterology

The escalating prevalence of obesity worldwide has led to a substantial increase in the number of bariatric and metabolic surgeries performed globally. The 2 most commonly performed metabolic and bariatric surgical procedures are laparoscopic sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB), which will be the focus of this article. In 2022, more than 160,000 SG and 62,000 RYGB operations were performed in the United States.[1] SG involves removing approximately 75% to 85% of the stomach along the greater curvature, leaving a cylindrical, or sleeve-shaped, stomach. There are 4 key components of RYGB: creation of a gastric pouch, creation of a biliopancreatic limb, jejunojejunostomy creation, and gastrojejunostomy creation.

Bariatric operations were historically classified as malabsorptive, restrictive, or both. However, the mechanisms are now understood to be far more complex.[2] Metabolic and bariatric surgeries have emerged as transformative interventions for addressing not only obesity but also associated disorders of metabolic dysfunction, such as type 2 diabetes mellitus (T2DM), with outcomes often surpassing those achieved through conventional medical therapies.[3] As the field of metabolic and bariatric surgery continues to expand, gastroenterologists are encountering a growing number of bariatric patients with surgically altered anatomy, presenting unique challenges in management. This article aims to

Table. Complications of Common Bariatric Surgeries

Complications	Timing of onset since surgery	Diagnostic tips	Diagnostic modalities	Management strategies
Sleeve gastrectomy				
Gastric sleeve stenosis	Early or late	Staple-line leak can be the initial presentation of sleeve stenosis Endoscopic clues: esophagitis, a persistently wide-open GEJ, pooling of secretions in the proximal esophagus and/or stomach, dilated upstream stomach, rotation of the staple line, inability to see through to the antrum, and upward-rightward maneuvering of the scope at the incisura to enter the antrum	Upper endoscopy, upper GI series	Hydrostatic or pneumatic balloon dilation of sleeve G-POEM is an option for helical sleeve
Sleeve leaks	Postoperative, early, or late	Evaluate for underlying downstream stenosis, which precipitates leaks	Cross-sectional imaging with contrast Upper endoscopy	Diversion therapy, internal/external drainage, balloon dilation of downstream stenosis, surgery
GERD	Late	Treat gastric sleeve stenosis Look for esophageal dysmotility	EGD/Bravo pH testing and FLIP High-resolution manometry if FLIP and Bravo do not correlate	High-dose PPI ± sucralfate Surgical conversion to RYGB
Weight regain	Early or late	Consider anatomic, genetic, dietary, psychiatric, and temporal factors (eg, gastric volume following sleeve, food urges, loss of disinhibition when eating, anxiety, time since surgery)	Varying definitions	Dietary/lifestyle modifications GLP-1 RAs Revisional - ESG Conversion to gastric bypass
RYGB				
Marginal ulcer	Early or late	One of the most common causes of abdominal pain in the post-RYGB patient; always consider Consider in the setting of GI bleeding Pouch biopsies and breath tests are unreliable for diagnosing *H pylori*, which most often resides in the inaccessible remnant stomach	CT scan of the abdomen Upper endoscopy *H pylori* stool antigen	Liquid or open-capsule high-dose PPI ± sucralfate Treat *H pylori* if positive Smoking cessation, avoid NSAIDs, optimize glycemic control in diabetes mellitus Endoscopic oversewing of ulcer or use of FCSEMS for recurrent/recalcitrant ulcers Rarely surgical intervention
Anastomotic stricture	Late	Lumen of anastomosis <6 mm in diameter Look for concomitant ulcers in the Roux limb	Upper endoscopy	Through-the-scope balloon dilation to at least 8 mm Other options: needle-knife incision, LAMS, SEMS, surgical revision
Anastomotic leak	Postoperative or early	Consider leak when there is abdominal pain, tachycardia, leukocytosis, and elevated CRP	Cross-sectional imaging	Diversion therapy, internal/external drainage, surgery
Gastrogastric fistula	Early or late	Upper GI series can miss fistulas high in the gastric pouch Need to retroflex the endoscope to fully examine the pouch	Upper GI series Upper endoscopy CT with oral contrast	Surgical closure for fistulas >1 cm Fistulas <1 cm may be managed with APC + over-the-scope clips or suturing

(Table continues on following page)

Table. *(Continued)* Complications of Common Bariatric Surgeries

Complications	Timing of onset since surgery	Diagnostic tips	Diagnostic modalities	Management strategies
RYGB				
Internal hernia	Early or late	Most often presents with chronic and intermittent cramping abdominal pain, especially localized to LUQ, but can be varied Maintain low threshold for surgical re-exploration because internal hernias are often missed on CT scans	CT scan with intravenous contrast	Surgery Endoscopy is not indicated for extraluminal causes of obstruction
Choledocholithiasis	Late	One of the most common causes of abdominal pain in the post-RYGB patient; always consider	RUQ ultrasound Cross-sectional imaging MRCP	Device-assisted enteroscopy Laparoscopic-assisted ERCP EDGE procedure
Weight regain	Late	May be owing to a dilated GJA	Varying definitions	Dietary/lifestyle modifications GLP-1 RAs TORe for dilated GJA

APC, argon plasma coagulation; CRP, C-reactive protein; CT, computed tomography; EDGE, endoscopic ultrasound–directed transgastric ERCP; EGD, esophagogastroduodenoscopy; ERCP, endoscopic retrograde cholangiopancreatography; ESG, endoscopic sleeve gastroplasty; FCSEMS, fully covered self-expanding metal stents; FLIP, functional lumen imaging probe; GEJ, gastroesophageal junction; GERD, gastroesophageal reflux disease; GI, gastrointestinal; GJA, gastrojejunal anastomosis; GLP-1 RA, glucagon-like peptide-1 receptor agonist; G-POEM, gastric peroral endoscopic myotomy; *H pylori*, *Helicobacter pylori*; LAMS, lumen-apposing metal stents; LUQ, left upper quadrant; MRCP, magnetic resonance cholangiopancreatography; NSAIDs, nonsteroidal anti-inflammatory drugs; PPI, proton pump inhibitor; RUQ, right upper quadrant; RYGB, Roux-en-Y gastric bypass; SEMS, self-expanding metal stents; TORe, transoral outlet reduction.

provide gastroenterologists with a practical compendium for understanding and managing the complications associated with metabolic and bariatric surgery (Table).

Marginal Ulcer

Marginal ulcer (MU) is a common late complication of RYGB with an incidence of 0.6% to 25%, although this is variable because of the asymptomatic nature of this condition.[4] MU usually develops at the gastrojejunal (GJ) anastomosis (Figure 1), most commonly on the intestinal side. Patients who undergo RYGB have hypochlorhydria; however, in the presence of MU, suppressing the few acid-producing parietal cells in the gastric pouch is important for healing. Although the antral G cells in the remnant stomach are excluded from the alimentary limb, gastrin-producing G cells may also still exist in the pouch (depending on size), which can contribute to increased acid production, as seen in patients with MU.[5] Equally if not more importantly, the diverted pancreaticobiliary secretions are unable to buffer this gastric acid and so undue chemical stress may be placed on the jejunal mucosa at the GJ anastomosis, resulting in MU formation. Risk factors for the development of MU include *Helicobacter pylori* infection, T2DM, and smoking. Interestingly, although nonsteroidal anti-inflammatory drug (NSAID) use, a

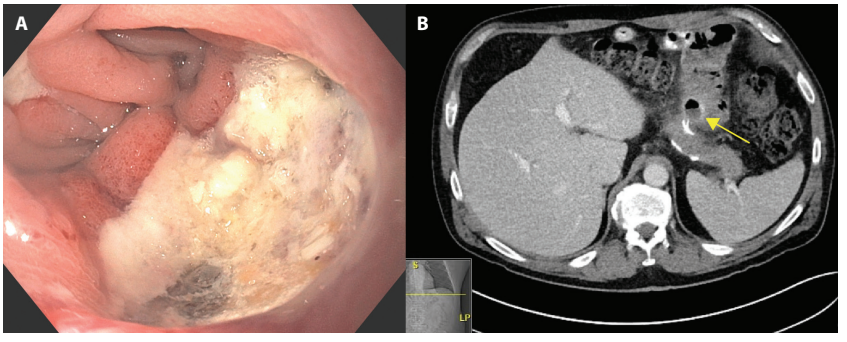

Figure 1. Marginal ulcer at the gastrojejunal anastomosis on endoscopy **(A)** and computed tomography **(B)**. The arrow indicates a ring-enhancing lesion with air-fluid level and adjacent fat stranding, suggesting ulceration.

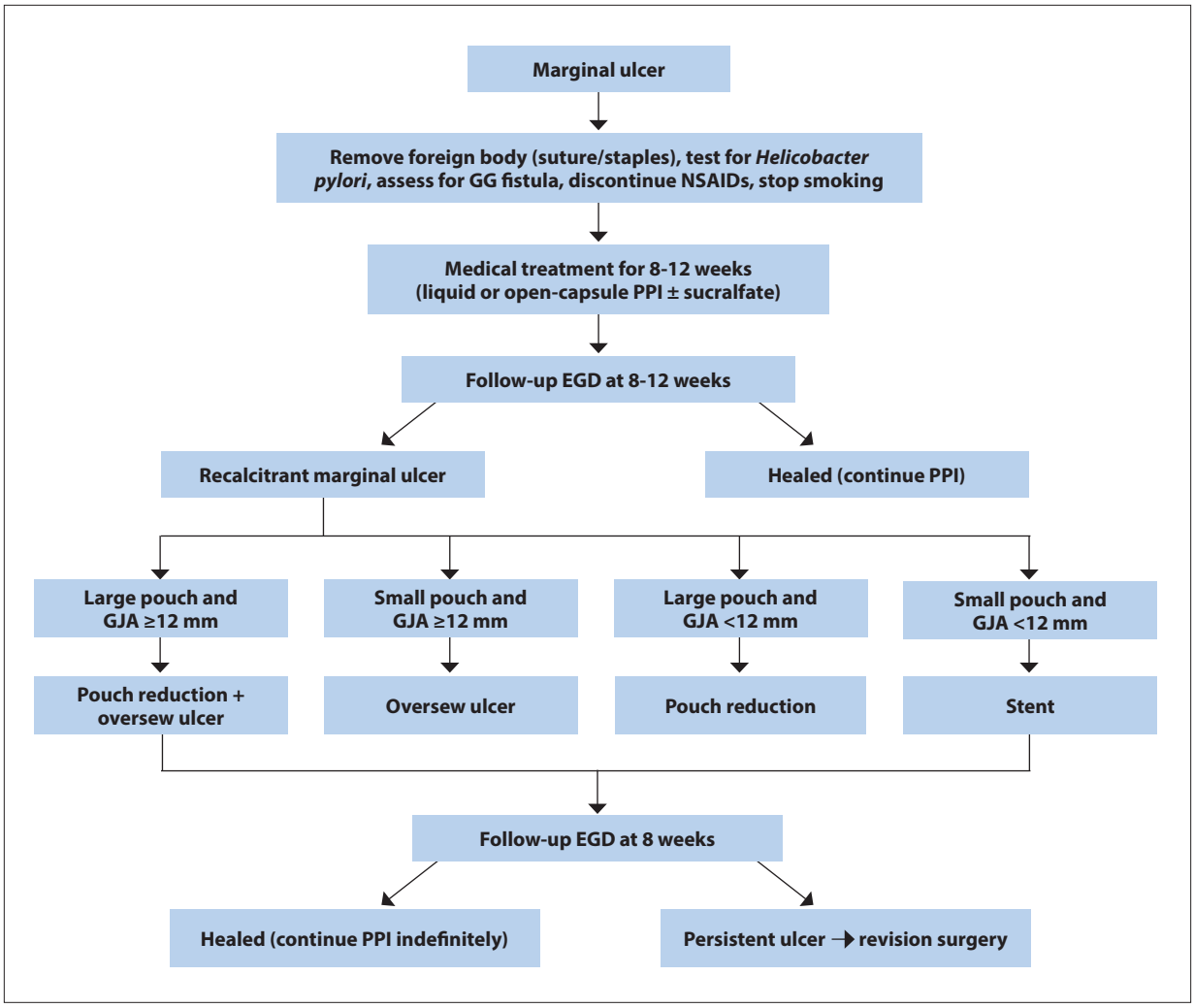

Figure 2. Algorithm outlining endoscopic management of marginal ulcer after gastric bypass.

EGD, esophagogastroduodenoscopy; GG, gastrogastric; GJA, gastrojejunal anastomosis; NSAID, nonsteroidal anti-inflammatory drug; PPI, proton pump inhibitor.

well-known risk factor for peptic ulcer disease, shows a trend toward increased risk for MU formation, it is not the most significant risk factor.[6,7] Additional factors associated with MU formation include increased acid production in an oversized gastric pouch, circular stapled anastomoses, tension at the site of anastomosis, staple-line disruption, presence of suture material within the pouch, and gastrogastric (GG) fistula.[8,9] Clinical presentation of MU ranges from asymptomatic to epigastric pain, nausea, vomiting, gastrointestinal (GI) bleeding, perforation, and stricture formation.[4] When MU is suspected, an upper endoscopy should be performed to confirm the diagnosis. Computed tomography (CT) scanning, when used to rule out other potentially serious pathologies, may detect perienteric fat stranding, focal bowel-wall thickening, and contour abnormalities, which may indicate the presence of MU.[10]

Treatment for MU typically begins with smoking and NSAID cessation and high-dose proton pump inhibitors (PPIs) with or without sucralfate, followed by repeat endoscopy in 2 or 3 months to confirm healing. Although there are currently no randomized controlled trials comparing PPI use with no PPIs in the treatment of MU, the use of liquid or open-capsule PPIs allows for enhanced absorption and should be favored over intact capsules because they significantly reduce healing time.[11] Stool antigen testing for *H pylori* should be performed and, if positive, eradication therapy should be initiated. Importantly, pouch biopsies and breath tests are less reliable in patients who underwent RYGB because the majority of the stomach where *H pylori* resides is inaccessible. If MU does not heal after high-dose PPI therapy, it is referred to as recalcitrant. Step-up endoscopic therapy for recurrent or recalcitrant MU includes oversewing the ulcer or, if suturing is technically unfeasible owing to an insufficient

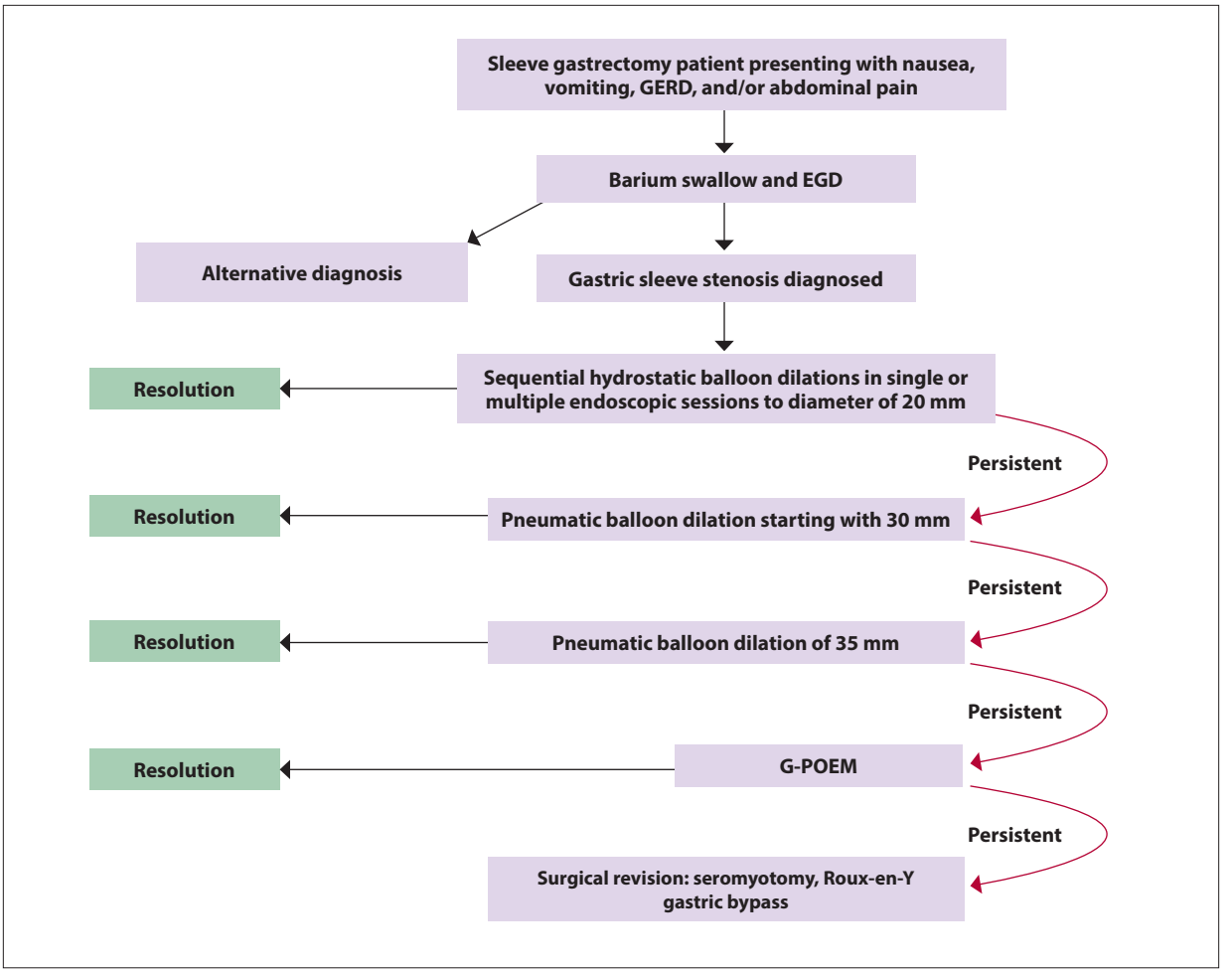

Figure 3. Treatment protocol for gastric sleeve stenosis.

EGD, esophagogastroduodenoscopy; GERD, gastroesophageal reflux disease; G-POEM, gastric peroral endoscopic myotomy.

gastric outlet caliber, then a fully covered self-expandable metal stent (FCSEMS) can be deployed to cover the ulcer bed.[12] Endoscopic suturing is particularly useful for a bleeding MU. Once the recalcitrant MU has healed, PPIs should be used indefinitely. Rarely, surgical intervention of the anastomosis and pouch reduction are required. These interventions include anastomosis revision, vagotomy to reduce acid production, subtotal/total gastrectomy, and reversal to normal anatomy.[13] The treatment algorithm for MU is summarized in Figure 2.

Stenosis

Gastric Sleeve Stenosis

Gastric sleeve stenosis (GSS) occurs most commonly at the incisura angularis but may also occur as a twisted or helical sleeve. GSS is often not a true mucosal stricture but rather an area of relative narrowing with significant angulation, requiring scope manipulation to traverse the stenosed segment. Thus, GSS may be functional or mechanical in nature. The causes of GSS include rotation of the staple line along the sleeve's long axis or a staple line placed too close to the lesser curvature. Furthermore, staple-line oversewing, postoperative hematomas, and staple-line leaks heal by scarring, which retract over time, causing strictures. The stenosed segment obstructs the flow of gastric contents, leading to elevated pressure within the lumen of the proximal stomach.

The symptoms of GSS include nausea, vomiting, regurgitation, early satiety, inability to tolerate solids, epigastric pain, gastroesophageal reflux, and esophagitis. The diagnosis of GSS is best made with upper endoscopy. Endoscopic clues for possible GSS are as follows: esophagitis, a persistently wide-open gastroesophageal junction (GEJ), pooling of secretions in the proximal esophagus and/or stomach, dilated upstream stomach, rotation of the staple line, luminal compromise at the stenosed site, inability to see through to the antrum, and upward-

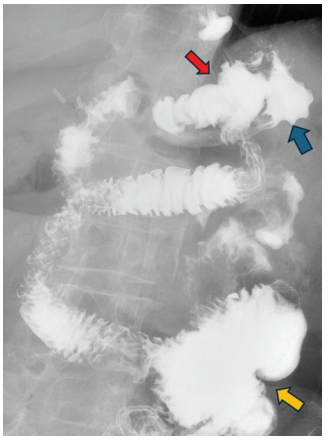

Figure 4. Gastrogastric fistula after Roux-en-Y gastric bypass seen on fluoroscopy, with the blue arrow showing excluded stomach, the red arrow showing the gastric pouch, and the yellow arrow showing the jejunojejunal anastomosis.

rightward maneuvering of the scope at the incisura (similar to the movements traversing the D1-D2 junction) to enter the antrum.[14]

Fluoroscopic injection of contrast from the proximal stomach is useful to identify downstream stenosis; if contrast fills the proximal stomach and distal esophagus before progressing to the antrum after a mild delay, this may imply an underlying stenosis.[14] Evans and colleagues recently published new benchmark values for the diagnosis of GSS using impedance planimetry, which will likely significantly improve recognition of this common complication.[15] The authors found that a diameter of at least 20 mm and a distensibility index of at least 15 mm^2/mm Hg were predictive of normal gastric sleeve anatomy, and decreasing diameter and distensibility index correlated with increasing stenosis severity.

Endoscopic balloon dilation, which includes hydrostatic and pneumatic dilation, is the primary treatment modality for GSS with an overall success rate of 76% after multiple dilations.[16] A meta-analysis of 16 studies involving 360 patients demonstrated higher clinical success with single pneumatic balloon dilation compared with hydrostatic balloon dilation (62.2% vs 36.4%; *P*=.007).[17] However, the latter is preferred by some experts owing to its more favorable safety profile.[18,19] When hydrostatic balloon dilation is employed first, the balloon should be inflated to a diameter of 20 mm. Multiple dilations may be required. If this is unsuccessful, pneumatic balloon dilation can be performed to a diameter of 30 mm, and up to a maximum of 40 mm, although the latter is rarely done.[20] If there is no improvement following a second pneumatic dilation to 35 mm, it is best to proceed to an alternative treatment modality. Our proposed treatment algorithm for GSS is summarized in Figure 3. It is important to note that proximal staple-line leaks can be the initial presentation of GSS and may occur many years after surgery.[21] In contrast to early leaks, endoscopic stenting with FCSEMS ± suture fixation is ineffective because symptoms recrudesce on stent removal.[14] Novel endoscopic modalities have emerged for the treatment of GSS, namely gastric peroral endoscopic myotomy. This technique involves full-thickness myotomy to untwist the helical stenosis and will likely gain more attention in the future.[22]

Anastomotic Stricture

Anastomotic strictures/stenosis are well-known complications following RYGB and are typically diagnosed 2 to 3 months following surgery.[23] They occur most frequently at the GJ anastomosis and less commonly at the jejunojejunal (JJ) anastomosis. The causes of stenosis are multifactorial and include ulceration, ischemia, subclinical anastomotic leaks, iatrogenic causes such as transoral gastric outlet reduction (TORe), and surgical techniques such as the use of circular staples.[24] Symptoms depend on the site of the stenosis but may include progressive dysphagia from solids to liquids, nausea/vomiting, gastroesophageal reflux disease (GERD), postprandial abdominal pain, and the sequelae of nutritional deficiencies. The diagnosis of a stricture is made when the lumen of the anastomosis is less than 6 mm in diameter, making it difficult for a standard endoscope to traverse the stoma. Upper GI series can also be used to diagnose strictures, but more subtle narrowing may be missed. The goal of treatment is to relieve symptoms by widening the stricture diameter to greater than 8 mm. Under fluoroscopic guidance, through-the-scope dilation of the stricture should be performed to allow easy passage of the endoscope. It is important to evaluate the Roux limb for causes that may have precipitated strituring, such as ulcerations. Repeat endoscopy should be performed 4 weeks later if the patient remains symptomatic. If the stricture diameter is less than 6 mm, the stricture can be incised using needle-knife electroincision, followed by repeat through-the-scope balloon dilation to at least 12 mm. Other treatment options include intralesional corticosteroid injection (eg, triamcinolone), lumen-apposing metal stents (LAMS), SEMS, and surgical revision. Caution is advised, as aggressive dilation may lead to a dilated GJ anastomosis and weight regain (WR).

Gastrogastric Fistula

GG fistula is an abnormal connection between the gastric pouch and the excluded stomach. This late complication of RYGB is rare, with an incidence of approximately 1%.[25] The etiology of GG fistula is related to chronic ischemia and inflammation, which often renders this complication refractory to advanced closure techniques such as endoscopic suturing and over-the-scope clips. Acid from the remnant stomach can traverse the fistula and precipitate marginal ulceration, GJ stricture, and GERD.

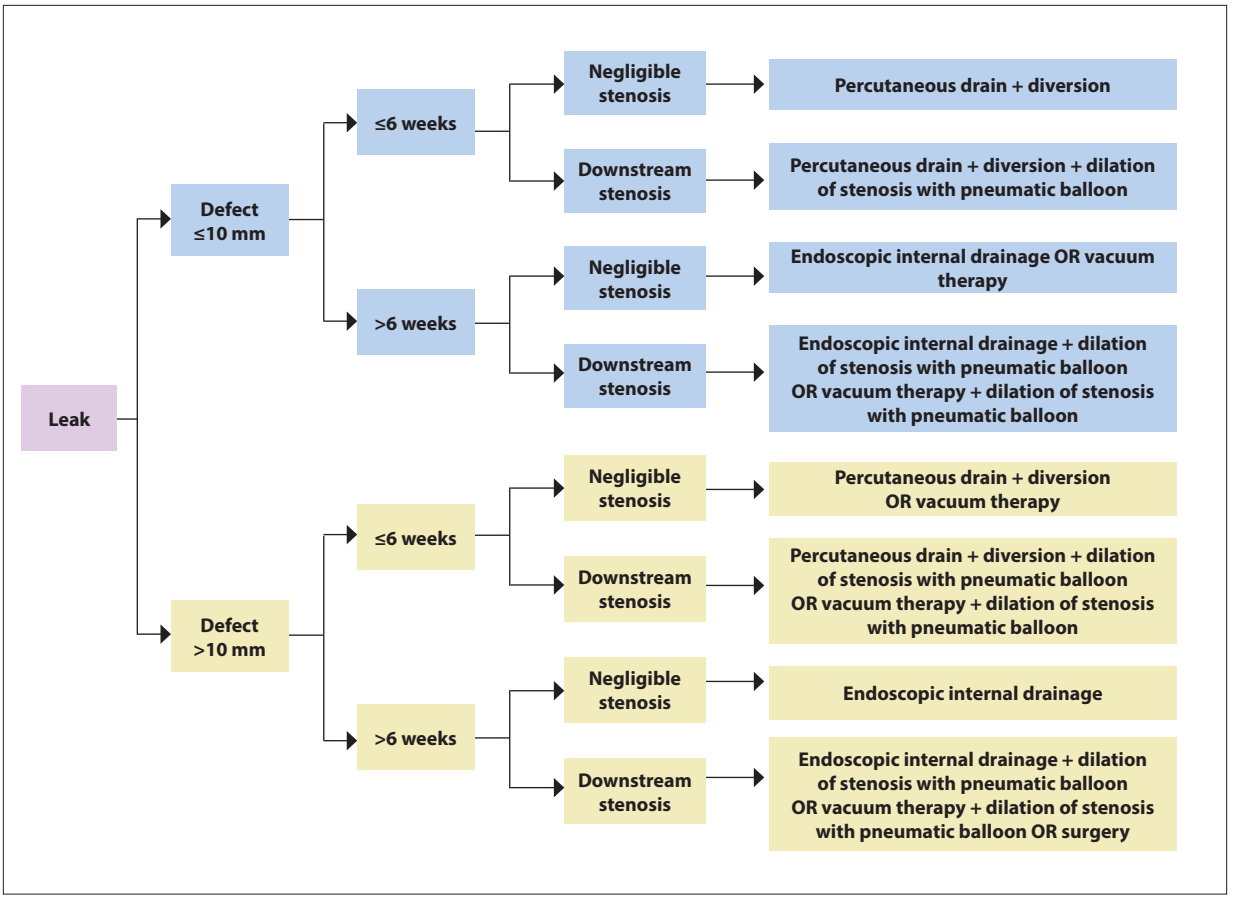

Figure 5. Treatment protocol for endoscopic management of leaks. If a leak has not closed after reasonable endoscopic attempts, then surgical revision is required.

Patients with GG fistula may present with abdominal pain, nausea, vomiting, gastroesophageal reflux, WR, or inability to lose weight. Investigations include upper GI series, esophagogastroduodenoscopy (EGD), or CT of the abdomen with oral contrast (Figure 4). Importantly, upper GI series will occasionally miss a GG fistula depending on the location, particularly if higher up in the gastric pouch. Fistulas can be missed on EGD if time is not taken to carefully examine the pouch, particularly on retroflexion. Definitive treatment of a GG fistula typically requires surgical closure if the fistula is greater than 1 cm. If the fistula is small (<1 cm), alternative options that may result in technically successful closure are endoscopic closure techniques such as over-the-scope clips or suturing after ablation around the fistula edges using argon plasma coagulation to devitalize the tissue. However, the fistulized tract may reopen, and these patients will need surgical closure. Other novel strategies, such as the use of cardiac septal defect occluders, may be options in the future,[26] particularly for patients who are not surgical candidates. However, septal occluders are very expensive and carry the risk of embolization.

Leaks

Anastomotic Leaks

Leaks are defined as the exit of intraluminal content at an anastomosis site or staple line. They most commonly arise from the GJ anastomosis but also may arise, more dangerously, from the JJ anastomosis and the remnant stomach. These are among the most feared complications of bariatric surgery. The incidence of leaks following RYGB is 1.6%.[27] The average time to presentation of anastomotic leaks following RYGB is 9.5 ± 7.4 days, which far exceeds average discharge times.[28] This underscores the need for close vigilance in the postoperative period.

Early detection of leaks may be challenging owing to the difficulty in eliciting clinical signs in patients with large body habitus. Tachycardia, leukocytosis, and elevated C-reactive protein are the most common presenting findings. Severe abdominal pain, shoulder tip pain, and hiccups in the setting of tachycardia are portentous signs that may suggest an underlying leak. When a leak is suspected, an oral contrast study is the next best step in evaluation.

Sleeve Gastrectomy Leaks

The incidence of laparoscopic SG staple-line leaks is approximately 0.8% to 4%.[27,29] Although post-RYGB leaks commonly arise within 1 month after surgery, leaks associated with SG may manifest months to years later, posing considerable diagnostic and management challenges and carrying substantial morbidity and mortality risks. Following SG, most leaks occur at the angle of His. The gastric wall is thinnest in this area, and gastric wall perfusion is also decreased at the angle of His and fundus when compared with other gastric sites.[30,31] These factors predispose the angle of His to mechanical and ischemic stressors. Staples fired too close to the GEJ can transect the segmental vascularization of this area, resulting in localized ischemia and leakage.[32] Moreover, a mismatch between staple height and tissue thickness also predisposes to leakage. When fashioning a gastric sleeve, the surgeon needs to adjust the stapler direction at the angularis, potentially leading to torsion of the sleeve and functional stenosis. If a narrower bougie is used to create a smaller-caliber sleeve, then greater weight loss is achieved, but the risks for GERD and leaks increase. Downstream stenosis results in increased intragastric pressure, which can precipitate and propagate leaks and is therefore important to address concurrently when managing leaks.[33,34] Oversewing of the staple line and application of fibrin glue may decrease these risks for leakage.

Management

Leaks can be classified based on temporality from surgery: acute (<7 days), early (1-6 weeks), late (6-12 weeks), and chronic (>12 weeks). Management of staple-line leaks following SG and RYGB depends on whether the leak occurs early or late, the size of the defect, and the presence of downstream stenosis (Figure 5). Strategies include diversion therapy, internal/external drainage, balloon dilation of downstream stenosis, and surgery. Success rates are higher for early and acute leaks, whereas chronic leaks often necessitate multiple endoscopic interventions or salvage surgical approaches.

Diversion therapy employing SEMS alongside percutaneous drainage of abdominal collections aids in early leak management, promoting early diet initiation, and addressing downstream stenosis when a long stent is used.[35,36] Both partially covered SEMS (PCSEMS) and FCSEMS can be utilized, with notable distinctions. First, it is important to suture a deployed FCSEMS to reduce the risk of migration. Second, the FCSEMS may not be able to create a watertight seal, compromising diversion effectiveness as gastric contents leak around the stent. PCSEMS are preferable to FCSEMS because tissue hyperplasia at the extremities of the stent creates a tight seal and reduces the risk of migration but results in more challenging stent removal. Patients often tolerate both stents poorly because tissue invagination and ulceration often occur at the distal end of the stent.

Alternatively, clinicians may opt for internal drainage strategies if an organized perigastric collection is present, keeping the fistula open to facilitate drainage and allow the perigastric collection to contract over time.[37,38] The rationale for internal drainage is based on the concept that contents will preferentially flow from the perigastric collection to the gastric lumen across the existing pressure gradient. Treating coexistent downstream stenosis, which can be a mechanical narrowing or torsion of the sleeve, is therefore essential to preserving this path of least resistance. Internal drainage is particularly suitable for mature, epithelialized leak sites where traditional closure methods may be less effective, typically beyond 6 weeks. Internal drainage techniques include double pigtail stent placement, septotomy, or endoscopic vacuum therapy, with necrosectomy aiding in cases of infected debris. If internal drainage fails, deployment of a cardiac septal occluder across the leak site can be attempted, but this technique requires further study.[39] If endoscopic interventions prove unsuccessful, surgical options include fistulojejunostomy, conversion from SG to RYGB, or total- or near-total gastrectomy with esophagojejunal anastomosis.[40,41] Direct surgical repair of chronic leak sites is not advised because it is rarely effective.

Gastroesophageal Reflux Disease and Barrett Esophagus

The relationship between GERD, obesity, and bariatric surgery is entwined and complex. GERD is a common comorbidity in patients with obesity and occurs because of multiple factors such as increased intra-abdominal pressure, impaired gastric emptying, decreased lower esophageal sphincter (LES) pressure, and higher frequency of transient LES relaxation. Hiatal hernias, which predispose to GERD, are also more prevalent in patients with obesity. Weight loss improves the symptoms of GERD and, therefore, bariatric surgery is a viable treatment option. Symptom improvement is considerably more frequent following RYGB than SG.[42] Apart from weight loss, improvement in GERD following RYGB is attributed to decreased acid production owing to reduced parietal cell mass in the gastric pouch, enhanced gastric emptying, and re-routed biliopancreatic secretions, resulting in diminished bile reflux. Conversely, preexisting GERD can be exacerbated or can occur de novo, particularly after SG.[43,44] This is the Achilles heel of SG, which may curtail its burgeoning popularity. A meta-analysis of 46 retrospective cohort studies totaling 10,718 patients found that 19% experienced exacerbation of preexisting GERD, whereas 23% developed de novo GERD after SG. More-

over, the long-term prevalences of esophagitis and Barrett esophagus (BE) were 28% and 8%, respectively, beyond 2 years. Four percent of all patients required conversion to RYGB owing to severe reflux.[45] Some experts believe that SG should be contraindicated in patients with preexisting GERD, but this remains a subject of ongoing debate.[46]

Pathomechanism

The pathomechanism of GERD following SG is not fully elucidated but involves anatomic and motility disturbances. Injury to any of the anatomic structures comprising the antireflux barrier can predispose to reflux. Additional factors include the shape of the sleeve (and the distance from the pylorus), an overly narrow sleeve, reduced gastric compliance, and sleeve stenosis, which all lead to elevated intragastric pressure; other factors include the presence of a de novo hiatal hernia or a preexisting hiatal hernia that was not repaired at the index surgery.[47,48] Johari and colleagues adeptly examined the physiologic paradigms underlying reflux after SG, delineating 3 distinct categories of reflux: bolus-induced deglutitive reflux, postprandial reflux, and elevated fasting esophageal acid exposure. The gastric sleeve did not demonstrate a constant elevation in basal intraluminal pressure without provocative events. Swallow-induced isobaric hyper-pressurization of the noncompliant proximal stomach was the predominant mechanism responsible for reflux events.[49] Gastric dysmotility owing to aberrant distal ectopic pacemaking and retrograde propagation following gastric resection may also be implicated.[50] A recently described achalasia-like pattern defined on high-resolution impedance manometry by normal integrated relaxation pressure, esophageal aperistalsis, and increased intragastric pressure, referred to as postobesity surgery esophageal dysmotility (POSED), may also underlie reflux in this patient population, particularly if dysphagia is present.[51] POSED has been observed in both RYGB and SG patients but is more prevalent in the former.

Management

Assessing GERD after bariatric surgery is challenging owing to a lack of standardized reporting and inconsistencies in definitions. Treatment of GERD following bariatric surgery depends on the underlying cause. We suggest initiating evaluation with EGD/Bravo pH testing and endoluminal functional lumen imaging probe (FLIP; Endoflip, Medtronic). EGD can assess the presence of esophagitis and BE, determine the Hill grade of the gastroesophageal flap valve, and identify potential underlying causes of reflux such as abnormal sleeve shape and size, sleeve stenosis, or hiatal hernia. In the post-RYGB patient, EGD can evaluate for additional structural abnormalities that may provoke the occurrence of GERD, such as a distended pouch, anastomotic stricture, and GG fistula. Bravo pH testing can confirm abnormal esophageal acid exposure, and FLIP will characterize motility patterns. If results from Bravo pH testing and FLIP do not correlate, then high-resolution manometry should be performed. If the patient cannot tolerate high-resolution manometry, then an upper GI series can be performed. Depending on the results of these investigations, patients may require referral to a motility clinic or surgery.

Screening for Barrett Esophagus

The true epidemiology of BE and esophageal adenocarcinoma in the bariatric surgery population is difficult to determine owing to the relative novelty of SG and insufficient long-term follow-up data for an already low-incident cancer in patients with BE. Although SG is a refluxogenic procedure and leads to a high incidence of GERD and esophagitis, this may not translate to a proportionally high incidence of BE.[52] A 10-year follow-up analysis of the SLEEVEPASS trial[44] demonstrated a cumulative incidence of de novo BE of only 4% following both SG and RYGB, consistent with some studies[53,54] but discordant with another study reporting a pooled prevalence of 11.6% at least 3 years after SG.[55] These differences may be explained in part by varying definitions of BE and variability of endoscopic assessment of BE. The potential risks of developing esophageal adenocarcinoma, an obesity-associated cancer, may be mitigated by weight loss after bariatric surgery.[56,57] Until more high-quality data become available, consensus among societies and experts will continue to be varied. The International Federation for the Surgery of Obesity and Metabolic Disorders recommends a single screening endoscopy at 1 year post-SG and then every 2 to 3 years, depending on its outcome.[54] In contrast to SG, 63% of patients with preoperative BE who underwent RYGB had endoscopic evidence of regression of BE at approximately 2 years of follow-up.[54] As a result, screening endoscopy recommendations to detect BE after RYGB have not been proposed.

Intestinal Obstruction

Bowel obstruction following gastric bypass in the early postoperative period occurs with comparable frequency to that observed in other abdominal surgeries. In gastroenterology practice, late intestinal obstruction is more commonly seen and occurs because of adhesions, ventral incisional and internal hernias, and intussusceptions. Internal hernia and intussusception are discussed in the following sections.

Internal Hernia

An internal hernia occurs when the small bowel herniates through one of the mesenteric defects that form during

the creation of the Roux and biliopancreatic limbs in a RYGB. Variations exist in surgical technique with respect to whether the Roux limb is positioned antecolic or retrocolic. Currently, an antecolic approach is more common and can result in 2 potential mesenteric spaces: (1) a meso-jejunal defect between the biliopancreatic limb and the common limb jejunojejunostomy (Brolin space) and (2) a defect between the Roux limb mesentery and the transverse mesocolon (Petersen space). A Roux limb placed in the retrocolic position can also herniate through a defect in the transverse mesocolon.[58] The incidence of internal hernia ranges from 4% to 17%, depending on whether these defects are closed at the time of the index surgery.[59] A high index of clinical suspicion is needed for the diagnosis of internal hernias because they can result in significant morbidity and mortality, including bowel strangulation, perforation, and massive small-bowel resections leading to short gut syndrome.[60] Internal hernias often present with chronic and intermittent crampy abdominal pain that is mild, but can also present with abdominal pain that is acute and severe with nausea and vomiting. The abdominal pain may be localized (especially in the left upper quadrant) or diffuse, radiate to the back, occur postprandially, or present with symptoms or signs of peritonitis.[61] CT of the abdomen and pelvis with intravenous contrast is the diagnostic modality of choice. Importantly, many internal hernias are missed on CT scans; therefore, the threshold for surgical re-exploration in post-RYGB patients who present with unexplained abdominal pain should be low.[59]

Intussusception

Intussusception is a rare complication following RYGB with an incidence of 0.64%.[62] In the general population with intussusception, telescoping occurs in an antegrade (peristaltic) fashion. However, in patients who have undergone RYGB, the common channel telescopes into the JJ anastomosis in a retrograde fashion for unclear reasons. The mechanism may be owing to disruptions in peristaltic pathways, including abnormal pacemaker potentials occurring in the Roux limb.[63,64] Patients can present with recurrent abdominal pain, obstruction, bowel ischemia, and necrosis. The diagnosis is made with abdominal CT, which may demonstrate the classic target sign, bowel thickening, and mesenteric edema. Surgical exploration is needed to reduce the intussusception along with either enteropexy or revision of the JJ anastomosis.[62]

Cholelithiasis

Obesity is a known risk factor for cholelithiasis, but rapid weight loss also increases the risk of gallstone formation. The incidence of gallstones following bariatric surgery may be as high as 40%.[65] Although cholecystectomy is the gold-standard treatment of symptomatic gallstones, prophylactic cholecystectomy at the time of bariatric surgery has historically been a point of contention with recent data no longer supporting this practice.[65] Ursodeoxycholic acid prevents both gallstone formation and symptomatic gallstone disease following bariatric surgery and should be prescribed at 600 mg per day for 6 months postoperatively.[66]

Endoscopic retrograde cholangiopancreatography (ERCP) is technically challenging in post-RYGB anatomy because standard duodenoscopes are unable to reach the major papilla. Currently, there are 3 endoscopic options for performing ERCP in this patient population: over-tube-assisted enteroscopy ERCP, laparoscopic-assisted transgastric ERCP, and endoscopic ultrasound–directed transgastric ERCP (EDGE).[67] EDGE is a novel technique that involves the creation of a GG fistula using a LAMS deployed under endoscopic ultrasound guidance, allowing access to the remnant stomach. EDGE is clinically successful in specialized centers, but it is not yet in mainstream practice.

Dumping Syndrome

Dumping syndrome (DS) arises from the rapid emptying of undigested gastric contents into the small bowel. DS consists of 2 subtypes: early and late DS. Early DS is thought to be caused by rapid delivery of hyperosmolar food into the small intestine, which results in osmotic fluid shifts from the intravascular compartment to the intestines. Peptide hormone release and autonomic neural responses are also implicated.[68] The pathophysiology of late DS involves an incretin-driven hyperinsulinemic response to the presence of excess glucose in the jejunum with resultant hypoglycemia.[68]

The symptoms of early DS occur within 1 hour (usually within 15 minutes) of ingestion and include GI symptoms (abdominal distention, painful abdominal cramping, borborygmi, diarrhea, and nausea) and vasomotor symptoms (flushing, palpitations, sweating, tachycardia, hypotension, and syncope). Patients often feel the need to lie down after a meal owing to generalized weakness.

Late DS, sometimes called postbariatric hypoglycemia,[69] occurs within 1 to 3 hours after a carbohydrate-rich meal. Although late DS can present with the same symptoms as early DS, it is characterized by hypoglycemia or, more seriously, manifestations of neuroglycopenia (confusion, fatigue, seizure, or coma). The diagnosis of DS is primarily made clinically. Scoring systems such as the Sigstad score and Arts questionnaire can be used to facilitate this assessment. The modified oral glucose tolerance test is the preferred diagnostic method to confirm the diagnosis of

DS. An increase in hematocrit greater than 3% or increase in pulse rate greater than 10 bpm 30 minutes after the start of the glucose intake are diagnostic of early DS, and a nadir hypoglycemia level less than 50 mg/dL is diagnostic of late DS.[70]

Dietary Modifications and Pharmacotherapy

Dietary modification is the first step in the management of DS and is usually sufficient for patients with mild to moderate symptoms. This approach includes consuming low-volume, protein-rich, and high-fiber meals; eating slowly and chewing well; eliminating rapidly absorbable carbohydrates; and delaying fluid intake until 30 minutes after meals.

If these dietary modifications fail, then the use of acarbose is recommended, particularly for late DS. Acarbose is an alpha-glucosidase inhibitor that attenuates postprandial hyperglycemia by delaying the conversion of oligosaccharides to monosaccharides. In patients who fail to respond to acarbose, somatostatin analogs such as octreotide can control symptoms of both early and late DS.[70] Somatostatin analogs function by inhibiting the secretion of incretin hormones and insulin, but the prescriber should consider that these agents also slow gallbladder emptying, increase gallstone formation, and inhibit pancreatic enzymes, leading to steatorrhea.[71]

Newer pharmacologic strategies being investigated include the use of glucagon-like peptide-1 (GLP-1) receptor agonists, sodium-glucose cotransporter-2 inhibitors, and GLP-1 receptor antagonists. Avexitide (exendin 9-39), a first-in-class GLP-1 receptor antagonist, competes with endogenous GLP-1 for its receptor, counteracting the effects of excess GLP-1 secretion. It has shown promising results for the treatment of postbariatric hypoglycemia.[72]

Endoscopic Revision of Gastrojejunal Anastomosis

Endoscopic treatment of DS is focused on delaying gastric emptying by reducing the diameter of a dilated or incompetent GJ anastomosis following RYGB.[73] This procedure is referred to as TORe or GJ anastomosis revision, and is also a treatment option for WR.

Surgical Re-intervention and Enteral Tube Feeding

In patients who continue to experience treatment-refractory DS, the value of continuous enteral feeding and surgical revision, such as gastric tube placement, gastric bypass reversal, and gastric pouch restriction, is unclear and not recommended based on limited available data.[70] Continuous enteral feeding through a jejunostomy tube has been proposed; however, its invasiveness and potential impact on quality of life warrant careful consideration. Restoring the original nutrient transit route via a gastric tube placed in the remnant stomach may also be effective.[74]

Nutritional Complications

Nutritional deficiencies often occur after RYGB owing to changes in GI anatomy, motility, pH, and enzyme profiles. It is crucial to promptly assess and address these potential deficiencies. Clinical practice guidelines endorsed by multiple societies are available to help guide nutritional supplementation after bariatric surgery.[75]

Chronic Abdominal Pain

Visits to the emergency department and readmissions occur in up to one-third of patients long term after RYGB, with abdominal pain being the most common presenting complaint.[76] Despite extensive investigations, including CT scans and diagnostic laparoscopy, many patients have no diagnosis to explain their pain.[77] A prospective study of 441 post–bariatric surgery patients with abdominal pain found that the 3 most common definitive diagnoses were cholelithiasis, ulcers, and internal hernias, followed by irritable bowel syndrome, anterior cutaneous nerve entrapment syndrome (ACNES), and constipation. Still, more than one-third of patients had unexplained abdominal pain at the end of the follow-up period.[78] Chronic abdominal pain occurs in 11% to 34% of patients after RYGB and is associated with reduced quality of life, with a subset notably experiencing chronic pain even before undergoing surgery.[79-81] Patients with irritable bowel syndrome appear to experience unexplained abdominal pain more often.[78,79] The etiology of such pain may be related to underrecognized chronic visceral neuropathic pain and complex pain syndromes, obviating the need for multidisciplinary management of these patients.

Dull and aching pain in the left upper quadrant should prompt consideration of conditions affecting the remnant stomach after RYGB, particularly in the setting of unexplained anemia. Remnant gastropathy likely results from mucosal atrophy owing to reduced nutrient contact, and chemical irritation from the pooling of bile. Diagnosis via device-assisted enteroscopy with biopsy demands specialized expertise. Cholescintigraphy (technetium-99m–hepatobiliary imaging) is useful for identifying patients at risk for bile acid gastropathy. The radiolabeled agent is absorbed by hepatocytes and excreted in bile, providing insights into bile flow and pooling in the remnant stomach.[82] Treatment with ursodiol has shown some success.[83]

In the absence of an identified structural cause for abdominal pain, the provider should consider abdominal wall pain such as ACNES. The abdominal pain from ACNES is often severe and located in the distribution of the affected sensory nerve branches of the intercostal nerves as they penetrate the rectus sheath. Pinching a skin fold in the affected area reproduces the pain, and there is often

hypo- or hyperesthesia with an altered perception of cold. Carnett sign is also a clinically useful physical examination maneuver to distinguish abdominal wall pain from visceral pain. Treatment includes trigger point injections, pulsed radiofrequency, or neurectomy of the affected nerve.[84]

Despite the various potential causes of abdominal pain in bariatric surgery patients discussed in this article, the clinician should still consider standard abdominal pain differentials and evaluations as those for non–bariatric surgery patients. Other potential causes of abdominal pain to consider are small intestinal bacterial overgrowth (especially if accompanied by nausea, vomiting, bloating, or diarrhea) and nephrolithiasis. Several studies have revealed an alarmingly high prevalence of small intestinal bacterial overgrowth following RYGB.[85] Post-RYGB patients are also at increased risk for nephrolithiasis owing to altered gut absorption and hyperoxaluria.[86]

Weight Regain

WR is a common complication of bariatric surgery with a lack of standardized definition, resulting in wide variation in reported WR outcomes.[87,88] At least 1 in 6 patients who undergo bariatric surgery experience WR of at least 10% by approximately 5 years of follow-up.[89] Risk factors are numerous and can be categorized into 5 groups: anatomic, genetic, dietary, temporal, and psychiatric factors.

Expectedly, WR results in the progression or relapse of T2DM, dyslipidemia, and hypertension; a decline in physical health–related quality of life; and dissatisfaction with surgery. Treatment options for WR include behavior intervention, medications approved for weight loss, endoscopic interventions, and revisional surgery.[90] Currently available data have failed to demonstrate a clinically meaningful efficacy of dietary, behavioral, and exercise interventions alone in reversing WR.[91] The treatment for WR and obesity is trending toward combination therapies requiring a multidisciplinary approach to care. GLP-1 receptor agonists demonstrate considerable efficacy in achieving weight loss and are promising options for the treatment of WR.[92]

Endoscopic Approaches

TORe is an endoscopic procedure that reduces the diameter of an enlarged GJ anastomosis. Most commonly, TORe is performed by ablating the stomal edges on the gastric side of the GJ anastomosis with argon plasma coagulation followed by suturing with the OverStitch Endoscopic Suturing System (Boston Scientific) to achieve a stomal diameter of 8 mm. Plication devices may also be used, such as the revisional obesity surgical endoluminal procedure via the incisionless operating platform, particularly for longer gastric pouches.[93] Other reported options include cryoablation and radiofrequency ablation.[94,95] In the setting of WR owing to a dilated GJ anastomosis, TORe plus nutritional management achieved a significantly higher percentage of excess weight loss compared with nutritional management alone at 12 months of follow-up.[96] Significant WR following SG previously resulted in conversion to RYGB. Minimally invasive options are now available, such as the sleeve-in-sleeve procedure, which involves performing an endoscopic sleeve gastroplasty on a preexisting dilated gastric sleeve.[97] Although the efficacy of endoscopic approaches in achieving significant weight loss is generally inferior to bariatric surgery, adjuvant therapy with antiobesity medications can enhance weight loss.[98,99]

Revision Surgery

Many patients require revision surgery for the treatment of WR.[100] Options for WR after SG include conversion to biliopancreatic diversion/duodenal switch, RYGB or single anastomosis gastric bypass, and revisional SG. Options for WR after RYGB include biliopancreatic diversion/duodenal switch, distalization of the RYGB, and revision of the gastric pouch and GJ anastomosis. Revision surgery for WR enhances weight loss but lacks the efficacy of the index surgery and presents a higher risk of complications.

Conclusion

Obesity is one of the most significant health problems worldwide, and its prevalence continues to rise. Bariatric and metabolic surgeries offer unparalleled weight loss and metabolic benefits. Despite surgical advancements, complications persist, emphasizing the complexity of managing the post–bariatric surgery patient. Effective management requires a multidisciplinary approach, with the expanding endoscopic tool kit providing minimally invasive options alongside traditional methods. Gastroenterologists will play an increasingly pivotal role in managing these surgical complications, particularly with the increasing adoption of primary endoscopic therapies. Evidence-based recommendations for endoscopic management remain limited, highlighting the need for more large-scale studies and randomized controlled trials to refine strategies for this challenging patient population.

Disclosures

The authors have no relevant conflicts of interest to disclose.

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