Complications in Bariatric Surgery

Diego Camacho
Natan Zundel
Editors

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Introduction

1

Diego Camacho and Dina Podolsky

Introduction

Over the past 60 years, the field of bariatric surgery has experienced an unprecedented growth in popularity as it has proven to be the most effective treatment of obesity and its associated comorbidities. It is estimated that nearly 200,000 bariatric procedures are performed annually in this country, a volume that may be satisfying less than 1% of the population's need [1, 2]. As weight loss surgery is being offered to increasingly complex patients with ever-rising BMIs, the impetus remains on the surgical community to provide this service in a safe and responsible manner. This textbook aims to define frequently encountered postoperative complications following weight loss surgery (WLS), as well as the current standards of care for treating them.

Over the past several decades, multiple factors have come together to decrease morbidity and mortality following WLS. From a technical standpoint, the widespread adoption of laparoscopy has greatly increased the safety profile of WLS; currently, over 90% of all bariatric surgery procedures are completed using minimally invasive techniques [3]. As the popularity of WLS increased, both the American College of Surgeons (ACS) and the American Society Metabolic and Bariatric Surgery (ASMBS) helped define standards and benchmarks for safe practice at high-volume, accredited hospitals, known as Centers of Excellence (COE) [4, 5]. The majority of bariatric surgery procedures are now being done at COEs, with various studies confirming that rates of postoperative complications are lower at accredited centers as compared to community hospitals [1, 6, 7]. Furthermore,

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bariatric surgery outcomes are now being monitored via the Metabolic and Bariatric Surgery Accreditation and Quality Improvement Program (MBSAQIP), which grants accreditation to these centers and tracks outcomes on a national level [1].

According to the most recent ASBMS data, sleeve gastrectomy is the most frequently performed bariatric procedure (54%), followed by gastric bypass (23%), revisional surgery (14%), and gastric banding (6%) [8]. All-cause mortality following bariatric surgery, regardless of procedure, has been estimated to be between 0.05% and 2% [9]. Postoperative complications can be divided by both pathophysiology and temporality. Short-term complications, defined as occurring within 30 days of the index procedure, have been estimated to occur at a rate of 4.8–10% [1, 10]. Early complications include, but are not limited to, leaks, bleeding, dvt/pe, cardiovascular and respiratory complications, and death [4]. Maintaining a high degree of suspicion in the postoperative period is imperative, as the majority of these complications can be managed effectively when diagnosed early. In less stable patients, frequently surgical re-exploration is required, a fact that any surgeon engaging in WLS should be prepared for.

Late postoperative complications, or those occurring after 30 days following the index procedure, include anastomotic stenosis, gallstone formation, bowel obstruction, intussusception, marginal ulcers, and fistula formation [4]. Some of these issues, such as stenosis or biliary disease, can be worked up in an outpatient setting and treated with either medication or endoscopic techniques. Others, such as complications from marginal ulcers and bowel obstructions, may present as surgical emergencies. Internal hernias, the most feared complication following RYGB, occur between 2.5% and 11.7% of the time, depending on technique used [11]. The use of advanced imaging techniques such as CT scan combined with a high index of suspicion can help turn these once deadly events into manageable complications. In many instances, surgical re-exploration remains the standard of care.

The purpose of this textbook is to provide a comprehensive and up-to-date reference for the management of complications stemming from bariatric surgery procedures, written by and for bariatric surgeons. Each chapter delves into common problems associated with the most frequently performed bariatric procedures, spanning the spectrum from acute to chronic presentations with a focus on both diagnosis and treatment. Our hope is that the words written in this book will provide guidance to those taking care of patients in need, as well as the tools necessary for the next generation of bariatric surgeons to continue this great public service in a safe and effective manner.

References

1. Ibrahim AM, Ghaferi AA, Thumma JR, Dimick JB. Variation in outcomes at bariatric surgery centers of excellence. JAMA Surg. Published online April 26, 2017. https://doi.org/10.1001/jamasurg.2017.0542.
2. O'Neill KN, Finucane FM, le Roux CW, Fitzgerald AP, Kearney PM. Unmet need for bariatric surgery. Surg Obes Relat Dis. 2016 pii: S1550-7289(16)30879-6. https://doi.org/10.1016/j.soard.2016.12.015.

3. American Society for Metabolic and Bariatric Surgery. Metabolic and bariatric surgery. http://asmbs.org/resources/metabolic-and-bariatric-surgery. Accessed 31 May 2016.
4. Lim RB. Complications of gastric bypass and repair. In: Fischer JE, editor. Fischer's mastery of surgery. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2012.
5. American Society for Metabolic and Bariatric Surgery. The Metabolic and Bariatric Surgery Accreditation and Quality Improvement Program (MBSAQIP). https://asmbs.org/about/mbsaqip. Accessed May 31, 2016.
6. Gebhart A, Young M, Phelan M, Nguyen NT. Impact of accreditation in bariatric surgery. Surg Obes Relat Dis. 2014;10(5):767–73.
7. Telem DA, et al. Rates and risk factors for unplanned emergency department utilization and hospital readmission following bariatric surgery. Ann Surg. 2016;263(5):956–60.
8. American Society for Metabolic and Bariatric Surgery. Estimate of bariatric surgery numbers. 2011–2015. http://asmbs.org/resources/estimate-of-bariatric-surgery-numbers. Accessed 31 May 2016.
9. DeMaria, et al. Baseline data from the American Society for Metabolic and Bariatric Surgery – designated bariatric surgery centers of excellence using bariatric outcomes longitudinal database. Surg Obese Relat Dis. 2010;6(4):347–55.
10. Coblijn UK, et al. Predicting postoperative complications after bariatric surgery: the Bariatric Surgery Index for Complications, BASIC Surg Endosc 2017. https://doi.org/10.1007/s00464-017-5494-0. [Epub ahead of print].
11. Aghajani E, Nergaard BJ, Leifson BG, et al. The mesenteric defects in laparoscopic roux-en-Y gastric bypass: 5 years follow-up of non-closure versus closure using the stapler technique. Surg Endosc. 2017. Published online February 15, 2017. https://doi.org/10.1007/s00464-017-5415-2.

2 Metabolic Complications, Nutritional Deficiencies, and Medication Management Following Metabolic Surgery

Christopher D. Still, Peter Benotti, Daniela Hangan, and Fahad Zubair

Introduction

Surgical procedures for weight management have been a part of the standard of care for patients with severe obesity since 1991. The rise in the prevalence of severe obesity and significant improvements in surgical quality and outcomes have enhanced patient and physician awareness of the health-protective and health-restorative benefits of surgical treatment for obesity and a rapid increase in the number of surgical weight loss procedures performed. The emergence of multidisciplinary care for patients with severe obesity in collaboration with metabolic surgeons has led to improved perioperative patient management and has contributed to the discovery of metabolic and nutritional complications which will be discussed in detail in this chapter.

Current Operative Procedures

The laparoscopic Roux-en-Y gastric bypass (Fig. 2.1) involves the creation of a small (15–20 ml) gastric reservoir, which is separated from the remaining stomach. The gastric reservoir is connected by a small, calibrated anastomosis to a

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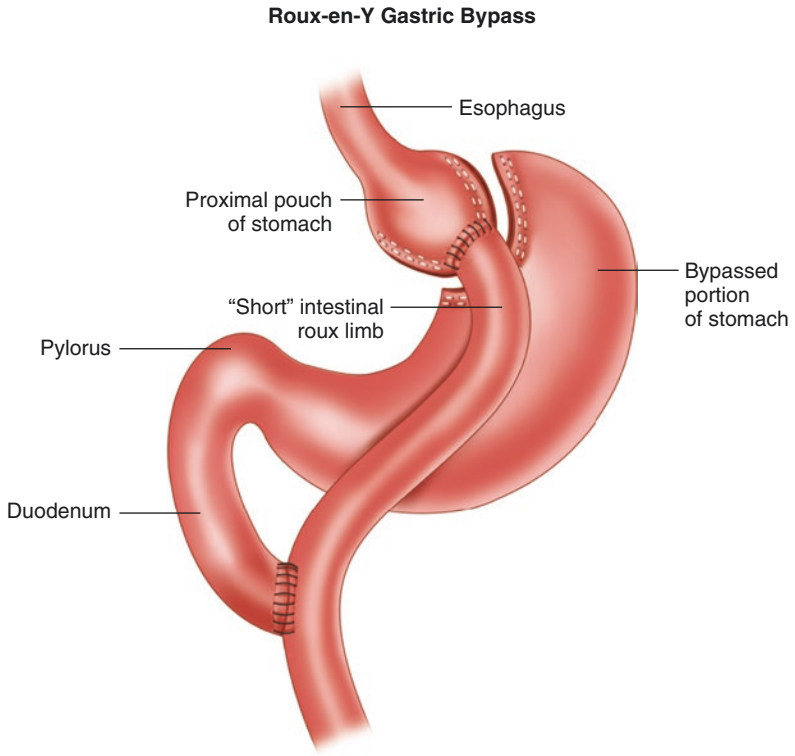

Fig. 2.1 Roux-en-Y gastric bypass

Roux-en-Y limb of jejunum, thus bypassing the duodenum and proximal jejunum. Until this past year, this has been the most popular procedure performed in the USA.

The sleeve gastrectomy (Fig. 2.2) is the most recent surgical procedure to be introduced and consists of a 70% vertical resection of the stomach which leaves a longitudinal narrow tubular gastric reservoir. The flow of nutrients via the duodenum and small intestine remains intact. This is now the most commonly performed procedure for surgical weight management in the USA.

The biliopancreatic diversion with duodenal switch (Fig. 2.3) is a more complex procedure involving a reduction in gastric capacity and a more extreme duodenal and small intestinal bypass leaving a relatively short common small intestinal channel for food absorption.

The simplest and safest procedure is the laparoscopic placement of an adjustable gastric band (Fig. 2.4). The adjustable band is a silicone collar with an inflatable component, which encircles the upper stomach and is connected to a subcutaneous port for adjustment of band size. Because of suboptimal results in long-term follow-up, this procedure has declined in popularity (Fig. 2.5).

In general, as the complexity of the surgical foregut anatomic alterations increase, the weight loss efficacy and durability increase, as does the potential for long-term

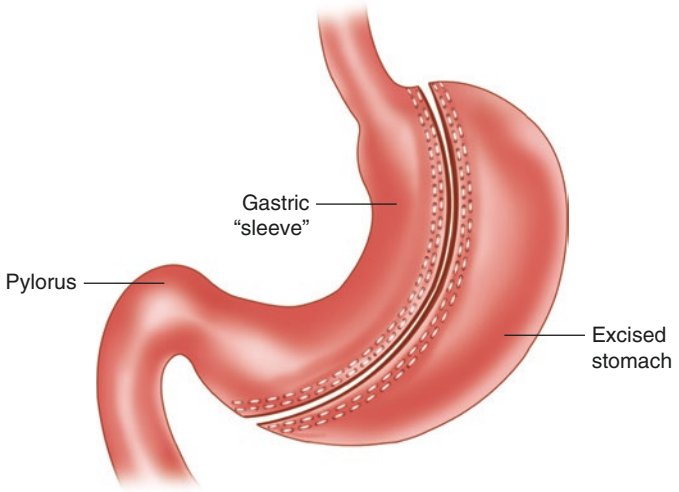

Fig. 2.2 Sleeve gastrectomy

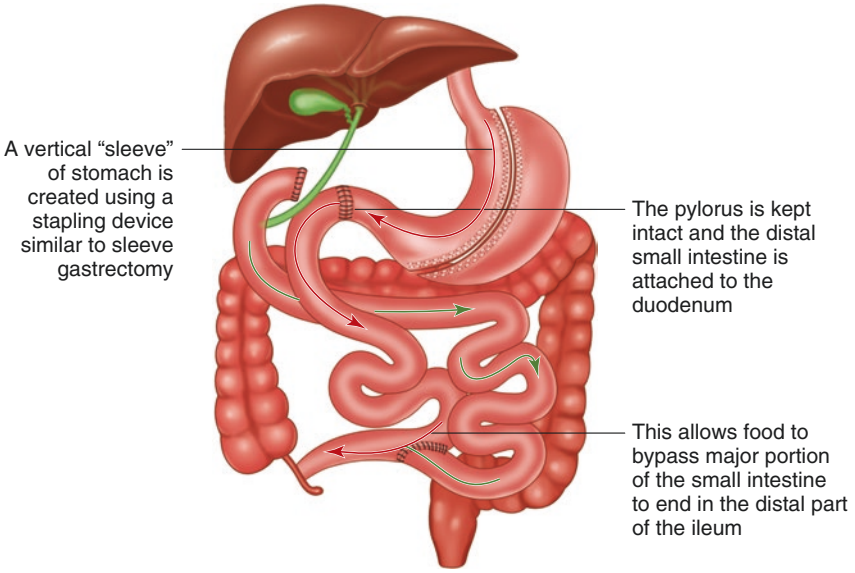

Fig. 2.3 Biliopancreatic diversion with duodenal switch

metabolic improvement. However, the more complex procedures are also associated with an increased risk of long-term nutrition and metabolic complications, which mandate close long-term follow-up in a multidisciplinary setting involving expertise in bariatric medicine, clinical nutrition, behavioral science, and metabolic surgery. Another potentially very important consideration in procedure selection is the

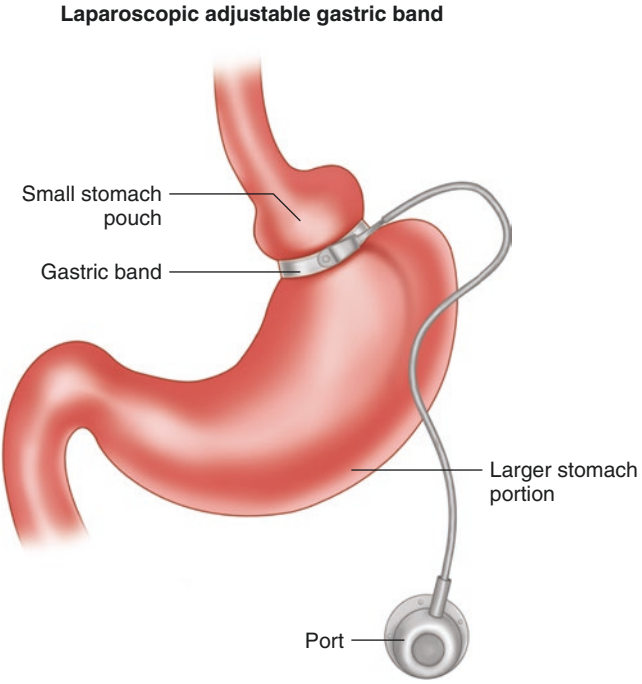

Fig. 2.4 Adjustable gastric band

emerging evidence that patient commuting distance may be an important risk factor for metabolic and nutritional complications perhaps by rendering additional challenges to close long-term follow-up [1].

Metabolic Complications

Metabolic Bone Disease

Introduction

Obesity has been thought of as protective against bone disease, with higher BMI associated with increased bone density. However, there is also a higher prevalence of vitamin D deficiency and increased parathyroid hormone (PTH) levels in obese individuals. The prevalence of vitamin D deficiency in obese individuals varies from 20% to 85%. Possible explanations include lack of sufficient sun exposure and sequestration of vitamin D in adipose tissue [2–4]. The number of bariatric procedures continues to rise in the USA with the most commonly performed procedures being sleeve gastrectomy and Roux-en-Y gastric bypass (RYGB). Animal studies have suggested greater bone loss after RYGB compared to sleeve gastrectomy, i.e., surgeries resulting in greater rates of malabsorption have higher bone loss [5, 6], whereas the limited number of human studies comparing bone loss in RYGB and

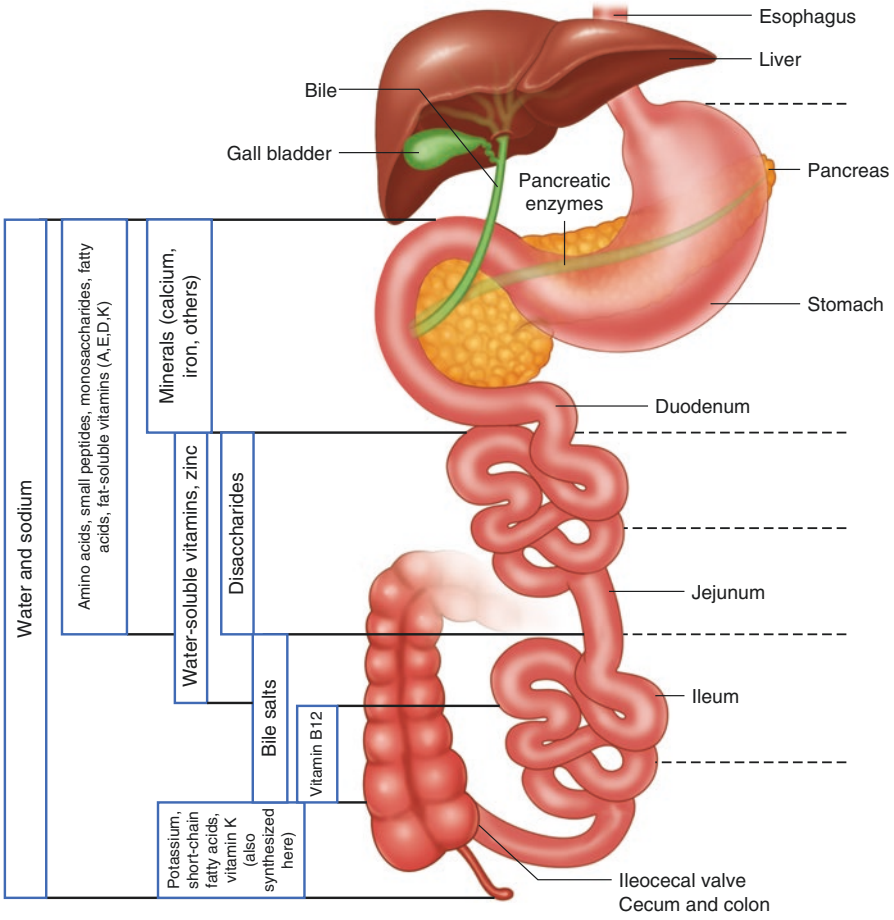

Fig. 2.5 Nutrients absorption site

sleeve gastrectomy has displayed conflicting results. Some of these studies have shown greater bone loss after RYGB and biliopancreatic diversion (BPD), whereas others have shown similar results in both surgeries [5, 7–12].

Pathophysiology

The mechanism for bone loss after bariatric surgery is multifactorial. Surgery can lead to decreased absorption of calcium and vitamin D as well as decrease production of gastric acid which can further decrease calcium absorption. This can result in hypocalcemia, a stimulus for the release of PTH, enhancing further bone loss. Evidence also suggests that bone loss after bariatric surgery correlates with the

amount of weight loss and the rate at which it occurs [13]. This is related to increased activation of the calcium-PTH axis with more and a higher rate of weight loss [13].

Sclerostin is produced in osteocytes and its main function is to inhibit bone formation. Mechanical unloading of bone after weight loss has shown an increased level of the hormone sclerostin resulting in significant loss in bone mineral density (BMD) [2, 10].

Ghrelin, a gut hormone that is known to stimulate growth hormone, promotes bone formation and has been shown in in vitro studies to have a direct effect on bone formation by having an enhanced effect on osteoblastic proliferation [14, 15]. Another gut hormone glucose-dependent insulinotropic polypeptide (GIP) has been shown to have an inhibitory effect on osteoclastic activity as well as an antiapoptotic effect on osteoblasts. Studies have generally shown decrease GIP levels after gastric bypass surgery, but influence on bone metabolism is not well studied [1, 16, 17].

Studies on GLP-1, peptide YY, amylin, and insulin have shown conflicting results and need further investigation [2].

Recent studies have also shown relationship between adipokines (adiponectin and leptin) and bone metabolism. Adiponectin has not been strongly correlated with decreasing BMD, whereas leptin has been shown to promote osteoblast differentiation and inhibit osteoclast differentiation [18–20].

Osteoprotegerin (OPG) and receptor activator of nuclear factor-κB ligand (RANKL) system has been shown to be associated with bone markers and bone mineral density as well after gastric bypass surgery. OPD (a decoy receptor for RANKL) decreases osteoclastogenesis by binding to RANKL. RANKL has shown to be increased after RYGB [21, 22].

Monitoring

Evidence-based guidelines recommend checking serum calcium, phosphorus, magnesium, 25(OH)D (and 1,25(OH)D if renal function is compromised), bone-specific alkaline phosphatase/osteocalcin, PTH, N-telopeptide (a marker of bone resorption), 24-h calcium, excretion, vitamin A and K_1 level, albumin, and prealbumin. The American Society for Metabolic and Bariatric Surgery (ASMBS) and TOS guidelines recommend checking 25 (OH)D and serum vitamin B_{12} every 3–6 months for the first year and annually thereafter in patients who underwent RYGB. Pt who had BPD with or without duodenal switch should have these checked every 3–6 months for the first year and every 3–6 months thereafter. BPD patients should also have intact PTH and 24-h urine calcium every 6–12 months after surgery [13, 23, 24].

"Dual-energy x-ray absorptiometry (DXA) is the gold standard for measuring bone density, results of which are reported in T and Z score. This score is the patients BMD in standard deviations (SD) from the mean in an age- and sex-matched reference population" [13]. World Health Organization (WHO) classifies a T score above −1 SD as normal, between −1 and −2.5 SD as osteopenia, and below −2.5 SD as osteoporosis. The endocrine society recommends performance of DXA preoperatively to establish a baseline and annually after gastric bypass [13]. Evidence supporting the benefit routine preoperative testing is lacking as stated by the The

Obesity Society (TOS) [13]. ASMBS and TOS guidelines recommend DXA 2 years postoperatively after any type of bariatric surgery [13].

Management

In patients who have undergone bariatric surgery, the primary focus should be nutritional deficiencies leading to metabolic bone disease. Patients should be screened for vitamin D deficiency, hypocalcemia, hypophosphatemia, hypomagnesemia, elevated alkaline phosphatase, secondary hyperparathyroidism, protein, and vitamin B_{12} deficiency, and appropriate treatment should be initiated if required [12].

Recommendation on Vitamin D Replacement

Per ASMBS/TOS vitamin D deficiency in patient with bariatric surgery should be treated more aggressively, especially after malabsorptive procedures [13]. They have recommended a dose of 50,000 IU one to three times a week [25, 26]. Resistant cases may require concurrent oral administration of calcitriol [27]. For prevention it is recommended that patients be supplemented with vitamin D 3000 units/day (titrate to more than 30 ng/ml) and calcium citrate 1200–1500 mg/day and 1800–2400 mg/day after biliopancreatic diversion with or without duodenal switch (BPD-DS) [27]. Biochemical markers should be repeated in 6–12 weeks, and adjustments should be made based on response to initial treatment [13, 26].

Other treatment options should be considered in patients with persistently abnormal DXA with clinical and biochemical resolution of bone disease. Due to a higher risk of anastomotic ulceration and concern for drug absorption after bariatric surgery, ASMBS/TOS and the American Association of Clinical Endocrinologist (AACE) recommend using intravenous therapy with zoledronic acid, 5 mg once a year, or ibandronate, 3 mg every 3 months [13, 27]. Patients without concerns for risk of ulceration or lack of absorption can be supplemented by mouth using alendronate 70 mg/week, risedronate 35 mg/week (or 150 mg/month), or ibandronate 150 mg/month [13, 27].

Patients should have labs checked every 6 months, and DXA should be done every 1–2 years for monitoring purpose and to look at the response to treatment interventions [13].

Nephrolithiasis After Bariatric Surgery

Mechanism

Recent studies have associated kidney stone development to bariatric surgery, particularly RYGB, which indicates up to a threefold increase in calcium oxalate stone risk compared with age-matched, obese controls. Stone development after malabsorptive (RYGB) and restrictive (sleeve gastrectomy) bariatric procedures is largely caused by changes in 24-h urine profiles, such as increased urinary oxalate, decreased urine volume, and reduced urinary citrate levels leading to increased risk of kidney stones [28]. RYGB creates an enteric hyperoxaluric state caused by

increased fatty acid, bile salt, and oxalate delivery to the intact colon. Six to 12 months after RYGB, fecal fat excretion increases, and this is thought to result in hyperoxaluria by increasing formation of calcium fatty acid salts, leading to decreased binding of calcium to oxalate and then increased oxalate absorption [29]. Alternatively, the anatomic reconfiguration also leads to alteration of the gut microflora and oxalate homeostasis. *Oxalobacter formigenes* is present in the normal human gut and metabolizes oxalate as an energy source. It is important in regulating oxalate metabolism, and its absence increases the risk of hyperoxaluria and recurrent kidney stones. Recent studies have revealed that rodents colonized by *Oxalobacter* have reduced urinary oxalate excretion [30, 31].

Management

Even though most of the oxalate excretion is from endogenous sources, about 10–20% is related to daily oxalate consumption. For this reason, a low-oxalate diet can be used as an initial step in management but is often difficult to achieve by patients. Supplemental calcium is also recommended as it binds oxalate, leading to excretion in feces, but due to increase in intestinal free fatty acids, saponification of calcium can occur rendering less calcium that is available to bind with oxalate. The most important factor in preventing stone formation is increasing fluid intake as increase urinary volume provides a dilutional effect leading to decreased supersaturation ratios [30, 32]. In severe symptomatic cases, surgical options may be explored.

The most common procedure is extracorporeal shock wave lithotripsy, but some studies have shown a negative impact of this procedure with increasing BMI [33]. It is recommended starting in the early postoperative period that patients be instructed to maintain a daily urine production of at least 2 L by increasing fluid intake, limit dietary oxalate and fat intake, and consume the recommended daily allowance of calcium (1000–1200 mg/day) [30].

Neurological Complications After Bariatric Surgery

The central and peripheral nervous system is dependent on nutrients such as B-group vitamins, vitamin E, copper, and vitamin D for optimal functioning [13]. After bariatric surgery, approximately 5–16% of patients can develop neurological complications [13, 34]. The more common complications include encephalopathy, polyneuropathy, mononeuropathy, and myeloneuropathy [13]. Here we will discuss some of the common neurological complications related to bariatric surgery.

Encephalopathy

Encephalopathy after gastric bypass is usually associated with vitamin B_1 and B_{12} deficiency and uncommonly related to folate and niacin deficiency. In one study the incidence of thiamine deficiency 2 years following the surgery was approximately

18% [35]. Due to a short half-life and lack of substantial stores of thiamine in the body, it takes approximately 4–6 weeks for these stores to be depleted [36]. In contrast vitamin B_{12} stores in the body are relatively abundant and the daily loses are minute. For this reason it may take 2–5 years, even after malabsorptive surgery, before B_{12} deficiency develops [13, 37]. Studies have reported that somewhere between 30% and 40% of the patients after gastric bypass develop B_{12} deficiency despite oral supplementation [38, 39]. Another rare cause of encephalopathy after gastric bypass is hyperammonemia [40]. The mechanisms for the hyperammonemic state after gastric bypass may be multifactorial. X-linked partial ornithine transcarbamylase (OTC) deficiency has been implicated leading to urea cycle dysfunction. Previously asymptomatic heterozygous OTC-deficient women are at risk. Other mechanisms include overgrowth of intestinal flora or a profound catabolic state which may lead to protein breakdown and accumulation of nitrogenous waste products [40]. In catabolic states, hyperammonemia may be treated conservatively with lactulose, rifaximin, and repletion of the deficient amino acids, zinc, and glucose [40]. Surgical revision of gastric bypass resulted in some clinical improvement in one case as well [41].

Management of Nutritional Deficiencies

Vitamin B_1 (Thiamine)

Thiamin facilitates intracellular energy production from carbohydrates, plays a role in muscle contraction, and facilitates nerve conduction. It is also essential for the metabolism of pyruvate and is indirectly involved in the synthesis of high-energy phosphates.

Clinical manifestations of thiamine deficiency include high-output or low-output heart failure along with neuropathy (beriberi). Dry beriberi is characterized by nerve damage leading to sensorimotor, distal, and axonal peripheral neuropathy and may lead to decreased muscle strength and eventually paralysis. Symptoms of dry beriberi include decreased muscle function, particularly in the lower extremities, tingling sensation, mental confusion, involuntary eye movement, and paralysis. Wet beriberi presents as heart failure with symptoms such as dyspnea on exertion, paroxysmal nocturnal dyspnea, and lower extremity edema. The most severe manifestation of thiamine deficiency is Wernicke's encephalopathy and Korsakoff's psychosis. Wernicke's encephalopathy is usually related to alcoholism and is more common in men, but when related to bariatric surgery, it has been more commonly reported in women. The classic clinical trial of Wernicke's encephalopathy is ocular abnormalities, gait ataxia, and mental status changes [13, 42]. Korsakoff's syndrome usually follows Wernicke's encephalopathy and is characterized by severe retrograde and anterograde amnesia.

The diagnosis of Wernicke's encephalopathy is usually clinical. Lab test includes a serum thiamine level (normal does not exclude the disease), but whole blood thiamine is more sensitive. Other tests include erythrocyte transketolase activation assay or measurement of thiamine diphosphate in red blood cells [13]. Blood samples

should be drawn before commencement of treatment. The imaging modality of choice is the MRI. Typical MRI findings include increased FLAIR signal in the paraventricular region. Other affected areas include the thalamus, hypothalamus, mammillary body, pons, and medulla, among others [43].

All post-weight loss surgery patients should take at least 12 mg thiamine daily and preferably a 50 mg dose of thiamine from a B-complex supplement or multivitamin once or twice daily to maintain blood levels of thiamine and prevent deficiency [26].

Patients with suspicion of Wernicke's usually require higher doses of thiamine (500 mg IV thiamine three times a day for 2–3 days, followed by 250 mg a day for 3–5 days, followed by daily long-term maintenance of 50–100 mg). Thiamine should be given before any administration of intravenous glucose or nutrition. The usual dose for thiamine supplementation in other cases (beriberi) is 100 mg IV every 8 h. Magnesium levels should be checked and normalized as well since magnesium deficiency may make a patient resistant to thiamine replacement [13, 26]. For more detail please refer to nutritional deficiencies section of this chapter.

Vitamin B_{12} and Folate

Vitamin B_{12}, also called cobalamin, is essential for maintaining healthy nerve cells, and it helps in the production of DNA and RNA, the body's genetic material. Vitamin B_{12} works closely with vitamin B_9, also called folic acid, to help make red blood cells as well. Folate and B_{12} work together to produce S-adenosylmethionine, a compound involved in immune function and mood. Decreased S-adenosylmethionine production may lead to reduced myelin basic protein methylation and white matter vacuolization in B_{12} deficiency.

Clinical manifestations of B_{12} deficiency may lead to neurological and hematological complications. The common neurological manifestations are myelopathy with or without neuropathy, optic neuropathy, and paresthesias. A well-known complication is subacute combined degeneration, a myelopathy, which can present as spastic paraparesis, extensor plantar response, and impaired perception of position and vibration. Other manifestations of B_{12} deficiency include impaired memory, emotional liability, psychosis and rarely delirium, and coma [13].

Vitamin B_{12} deficiency can lead to megaloblastic anemia. A rise in mean corpuscular volume may be seen as well as the presence of hypersegmented neutrophils on microscopy. The serum B_{12} level lacks sensitivity and specificity [37, 44, 45]. Levels of serum methylmalonic acid and homocysteine rise when vitamin B_{12} is deficient and can assist in establishing the diagnosis and monitoring replacement [13]. Nerve conduction studies suggest a sensorimotor axonopathy and abnormalities on somatosensory-evoked potentials, visual-evoked potentials, and motor-evoked potentials [13, 46].On imaging (MRI) a signal change or contrast enhancement in the posterior and lateral columns and less commonly subcortical white matter is seen. Increase T2 signal involving the cerebellum may be seen, and rarely white matter abnormalities have been reported suggestive of leukoencephalopathy [13].

The clinical manifestations of folate deficiency are like those of B_{12} except the neurological complications, which are rarely seen. For diagnosis serum folate

should be checked but is not a good indicator of folate stores in the body. For this reason RBC folate should be preferred diagnostic test as its levels are less affected by fluctuations in folate intake. Plasma homocysteine levels can be elevated in clinically significant deficiency [47].

Vitamin B_{12} supplementation should be initiated soon following gastric bypass surgery. The supplement dose for vitamin B_{12} in post-weight loss surgery patients varies based on the route of administration. Orally by disintegrating tablet, sublingual or liquid dosage is 350–500 μg daily, whereas the parenteral *(IM or SQ)* dose is 1000 μg monthly [26].The role of oral therapy in patients with severe neurologic disease has not been well studied [13]. For more detail on replacement, please refer to the nutritional deficiencies sections of this chapter. For more detail please refer to the nutritional deficiencies section of this chapter.

The recommended dose for folic acid supplementation after gastric bypass surgery is 400–800 μg oral folate daily from a multivitamin. Women of childbearing age should take 800–1000 μg oral folate daily [26].

Neuropathy

Neuropathy after bariatric surgery is usually as a result of deficiency of vitamin $B_{12,}$ thiamine, or copper but can also result from deficiencies in vitamin B_6 (pyridoxine), folate, niacin, and vitamin E. Neuropathy can present either as peripheral neuropathy, mononeuropathy, optic neuropathy, polyradiculopathy (may mimic Guillain-Barre syndrome) or myeloneuropathy (subacute combined degeneration) [13]. For replacement of these deficient nutrients, please refer to the nutritional deficiencies section of this chapter.

Hypoglycemia

An important metabolic complication which is attracting increasing interest is postprandial hyperinsulinemic hypoglycemia (PHH), characterized by hypoglycemic symptoms developing 1–3 h after a meal accompanied by a low blood glucose level. This condition should be distinguished from early dumping syndrome where symptoms develop within minutes to 1 h after a meal of caloric dense food, caused by the rapid and unregulated emptying of food into the jejunum, which induces rapid fluid entry into the small bowel. Early dumping often occurs early in the postoperative period, most commonly after Roux-en-Y gastric bypass, whereas PHH may develop months to years after surgery.

PHH was originally described as “late dumping” and is a well-recognized but uncommon complication of gastric resection. This condition was first described as a complication of gastric bypass in 2005 when refractory hypoglycemia, elevated insulin levels, and enlargement of pancreatic beta cells were described in six patients [48]. However, subsequent studies have not confirmed that the entity is associated with focal abnormalities in beta cell morphology [49], and the exact cause of the increased insulin secretion remains in question.

Since these early reports, additional epidemiologic studies in larger cohorts have confirmed that this condition has been most commonly associated with Roux-en-Y gastric bypass, but has been described after duodenal switch and sleeve gastrectomy [50–52]. The prevalence of this condition is wide ranging and varies per the diagnostic criteria utilized. The early studies, based on hospitalization or self-reported severe hypoglycemia symptoms, suggested a prevalence of less than 1% with symptoms occurring 1–3 years after surgery [53, 54]. More recent studies utilizing patient surveys or continuous glucose monitoring report prevalence approaching one third of patients, with much lower rates of symptomatology [51, 52]. A more recent study utilizing the electronic medical record and a clinical registry identified a large cohort of nondiabetic gastric bypass patients with a 5-year incidence of 13% with risk factors identified as a lower preoperative HbA1c and higher 6-month weight loss [55].

Symptoms related to post-PHH usually develop late after surgery in contrast to early dumping. Symptoms are wide ranging, but are usually related to Whipple's triad: symptomatic hypoglycemia, a low plasma glucose level, and resolution of symptoms after the administration of glucose. Symptoms of hypoglycemia may include anxiety, sweating, tremors, palpitations, confusion, weakness, lightheadedness, dizziness, blurred vision, disorientation, and possibly loss of consciousness.

The American Diabetes Association definition of hypoglycemia is ≤70 mg/dL (≤3.9 mmol/L) [56]. However, much lower glucose levels are commonly encountered in postoperative gastric bypass patients even in the absence of symptoms. There are no absolute diagnostic criteria for PHH, but patients must have both symptoms of hypoglycemia and documented low glucose levels, usually less than 50–60 mg/dl [57]. As a general rule, the diagnosis should involve symptoms of hypoglycemia, the documentation of a low glucose level during symptoms, and the resolution of symptoms with carbohydrate intake [58, 59].

Provocative testing can also be considered. Because of risks of over diagnosis with the oral glucose tolerance test, the optimal provocative test is the mixed-meal tolerance test [60]. For clinical screening of patients where PHH is a consideration, continuous glucose monitoring looking for increased glycemic excursions in symptomatic patients should be considered [51]. Once the diagnosis is confirmed, excess use of sulfonylureas and insulinoma must be ruled out by measuring fasting glucose and insulin levels and screen for sulfonylureas in blood samples [58, 59].

The exact mechanism for the development of the development of PHH has not been established. However, metabolic findings in symptomatic PHH patients' vs non-symptomatic post-gastric bypass controls include heightened postprandial glucose peak levels and nadirs [61]; increased levels of C-peptide, insulin, and GLP-1 [61, 62]; and restoration of more normal meal responses with feeding via gastrostomy placed in the gastric remnant [63].

Because of variability in degree of symptoms and the absence of a clear pathophysiology, management of this condition can be challenging. Fortunately, a significant percentage of patients with milder forms of the condition can be managed with dietary modifications consisting of frequent small meals with a low glycemic index [57]. This requires supervision by a dietitian and long-term patient compliance.

Additional benefit has been obtained by the addition of acarbose, an α glucosidase inhibitor in doses 100–300 mg [64].

Successful management has been also reported in case reports or small case series with diazoxide [65], calcium channel blockers [59, 66], and somatostatin analogues [67]. The role of GLP-1 in the pathogenesis of this condition is supported by the observation that infusions of GLP-1 antagonists corrected hypoglycemia in these patients [68]. These agents are investigational at present, but provide opportunity for additional future treatment approaches. For patients with persistent symptoms despite medical treatment, reversal of the bariatric procedure should be considered. Partial pancreatectomy, although used in the past, is now not recommended because of the significant morbidity and poor long-term symptom control [69].

Postprandial hyperinsulinemic hypoglycemia is an important, potentially dangerous late complication of metabolic surgery. Successful diagnosis and management of this condition requires multidisciplinary specialty resources and essential long-term follow-up capabilities. Readers are encouraged to read several excellent current reviews of this topic [70, 71].

Macronutrient Deficiencies After Metabolic Surgery

The major macronutrient deficiency after bariatric surgery is protein malnutrition [72]. Causes include intolerance to foods like red meat, a decrease amount of food ingested, major alterations in small intestinal anatomy, bypass of the duodenum, loss of gastric acidity, dumping syndrome, diarrhea, and bacterial overgrowth [73, 74]. Nearly all protein absorption (95–98%) takes place in the small intestine. Bacteria in the colon can digest some of the remaining protein [75].

Clinical signs of protein deficiency include generalized weakness, leg edema, decreased handgrip and leg extension strength, decubitus ulcers, depression, and fatigue. In severe cases anasarca, hair loss, sarcopenia, and dementia can be present. When protein malnutrition occurs after weight loss surgery, it is frequently associated with a massive weight loss trajectory.

Daily requirements for protein in humans in a healthy state are 0.8 g per kg of ideal body weight. For example, for a target weight of 70 kg (or a weight based on a BMI of 21.7), the protein requirements are minimum 56 g. In general, men require more protein than women. After bariatric surgery, the recommended protein intake per day is approximately 1.0–1.5 g/kg of ideal body weight. There is evidence that a protein intake of ≥1 g/kg/day during the first year after RYGB will result in healthier weight loss with preservation of lean body mass [76].

In the absence of an acute phase response and liver disease, the serum levels of albumin and prealbumin can be good markers of protein nutrition. However, albumin and prealbumin become rapidly consumed in an active illness where synthesis is reduced. Albumin half-life is 20 days and prealbumin half-life is 2 days [77]. C-reactive protein is elevated in acute inflammatory states and can be used in conjunction with albumin and prealbumin to determine the validity of these markers.

Therefore, these markers should not be solely used to determine the degree of malnutrition but could be of value if there is no other factor contributing to the rapid weight loss and muscle loss other than bariatric surgery.

Optimal management of protein nutrition after bariatric surgery requires the involvement of therapeutic dietitians for patient education, close postoperative nutritional surveillance, and monitoring of patient compliance. Inadequate protein intake after bariatric surgery constitutes an indication for the use of modular protein supplements. If conservative treatment cannot restore normal protein nutrition, surgical revision is indicated.

Micronutrient Deficiencies After Bariatric Surgery

Because of major alterations in foregut anatomy, duodenal bypass, and loss of gastric acid, absorption of micronutrients is affected after bariatric surgery.

The slide below illustrates the sites of absorption of the main micronutrients and will help us understand better how these deficiencies occur.

Despite the setting of overnutrition, recent evidence suggests that micronutrient deficiency is not uncommon among candidates for bariatric surgery. Since nutrient deficiencies may affect the severity of comorbid disease, the risks of surgery, and the potential for postoperative deficiency, a full nutritional assessment including detailed assessment of micronutrient status should be conducted for all candidates for bariatric surgery [78].

Patients who undergo SG or RYGB procedures are at risk for B_{12}, iron, vitamin D, calcium, zinc, and copper deficiencies. Less common deficiencies include thiamine and folate. BPDDS procedure creates a shorter common intestinal channel with the potential for greater malabsorption. This mandates additional monitoring for the fat-soluble vitamins (A, D, E, and K). Proper supplementation of all nutrients at risk and monitoring of patient compliance with supplementation are mandatory after bariatric surgery.

Vitamin B_{12}

Dietary vitamin B_{12} is released from ingested proteins by action of hydrochloric acid and pepsin in the gastric secretion. Complex R is formed and travels to the small intestine where pancreatic enzymes hydrolyze R protein and release free vitamin B_{12}. Intrinsic factor binds vitamin B_{12} and reaches the terminal ileum where the complex that attaches to a receptor facilitates the absorption and transport of this complex into the portal circulation. Therefore, any abnormality affecting this pathway can produce vitamin B_{12} deficiency [73].

Manifestations of vitamin B_{12} deficiency include nervous system abnormalities and megaloblastic anemia. Neurologic manifestations include distal sensory neuropathy, hyporeflexia, or a more severe myelopathy called subacute combined degeneration of the spinal cord. This manifests with weakness, loss of postural

sense, and walking difficulties. Psychiatric disturbances including dementia and depression may occur as well as visual loss from optic atrophy [79]. In addition, hyperpigmentation of the skin of the forearms, atrophic glossitis, and diarrhea may be present [80].

Deficiency of B_{12} after bariatric surgery is common with prevalence recorded as 26–70% [81]. The liver stores of this vitamin allow for a long interval between the onset of deficient intake and the development of symptoms. Factors contributing to deficiency include limited postoperative intake of animal protein, impaired gastric cleavage of B_{12} from dietary protein, and diminished production of intrinsic factor. The initial test to monitor for deficiency is to measure the B_{12} level. Normal values are 200–900 ng/ml. Levels less than 200 suggest deficiency and levels less than 400 should cause concern [80]. Confirmation of deficiency usually involves the measurement of serum levels of methylmalonic acid or total homocysteine as these levels will rise in the presence of B_{12} deficiency and they will fall promptly with replacement [82].

Supplementation of this vitamin after bariatric surgery is of vital importance. Use of only a multivitamin preparation does not usually contain sufficient amounts of vitamin B_{12} to prevent deficiency. The recommended replacement after surgery is 350–500 mcg daily administered sublingually, intranasal, or as a disintegrating tablet [80]. Management of vitamin B_{12} deficiency includes administering 1000 mcg intramuscular vitamin B_{12} weekly for 8 weeks. For patients who develop neurological symptoms, parenteral treatment with 1000 mcg vitamin B_{12} daily for 5 days is recommended, followed by monthly injections for life [74].

Thiamine B_1

Absorption takes place in the proximal small intestine, especially the jejunum via an active carrier-mediated process. The vitamin can also be passively absorbed if the dosage is high. Because of a short half-life and limited stores, a continuous supply of this vitamin is essential for normal metabolism.

About 50% of thiamine is stored as a coenzyme in skeletal muscle. In the blood, thiamine is bound to albumin and is taken up by the liver to produce its biological active coenzyme TDP 9(thiamine diphosphate). Malabsorptive bariatric surgical procedures can limit thiamine absorption [73].

Clinical manifestations of deficiency involve the nervous and cardiovascular systems. Wernicke's encephalopathy involves ocular abnormalities, ataxic gait, and changes in mental status. Congestive heart failure can also occur with deficiency states. Physical findings include ophthalmoplegia, nystagmus, loss of reflexes and confabulation, as well as sensory and vibratory sense abnormalities [79].

Thiamine deficiency should be suspected in any patient with recurrent vomiting and reduce food intake after bariatric surgery. It should also be considered in any patient with neurologic disturbance after bariatric surgery. The development of muscle weakness, loss of reflexes, or Wernicke's encephalopathy should be considered a neurological emergency, and immediate replacement is indicated.

Thiamine nutritional status is assessed by measurement of levels of thiamine in serum or red blood cells. The normal range for thiamine levels is 4–15 nmol/L. Routine supplementation of thiamine at 12 mg/day is recommended. Patients at risk should receive 50–100 mg daily [80]. Management of deficiency in the absence of neurologic manifestations is 100 mg twice or three times daily. In severe cases with neurologic involvement, intravenous replacement is indicated at 200 mg three times daily or 500 mg twice daily for 3–5 days or until symptoms resolve. An alternative parenteral therapy is intramuscular therapy: 250 mg once daily for 3–5 days then 100–250 mg monthly.

Dosing can be switched to oral as oral intake improves. Continued oral dosing at 100 mg daily is required until risk factors are resolved [80].

Folic Acid

Folic acid is absorbed in the small intestine [73, 83]. Humans have the capability to store about 5 mg of this vitamin. As the daily requirement is 100 mcg per day, stored folic acid will provide a supply for several months.

Clinical manifestation associated with deficiency includes megaloblastic anemia which is similar to B_{12} deficiency. Other findings include glossitis, angular stomatitis, and neurologic symptoms which are also like those associated with vitamin B_{12} deficiency.

Folate deficiency is uncommon after bariatric surgery because it is absorbed throughout the small intestine and a daily multivitamin tablet contains 400 mcg per dose which is usually sufficient to correct low levels [84]. The normal range for serum folate levels for adults is 2–20 ng/mL or 4.5–45.3 nmol/L. Deficiency is best documented by measuring red blood cell folate levels where the normal range is 40–628 ng/mL or 317–1422 nmol/L. Patient compliance with routine multivitamin supplementation should be stressed. Requirements for folate are increased in pregnancy, and daily supplementation in doses of 800–1000 mcg is indicated in women of childbearing age.

In suspected deficiency states, B_{12} levels must be checked and verified as adequate before attributing megaloblastic anemia to folate deficiency. Treatment of deficiency should include 1 mg folate replacement daily until deficiency is resolved [80].

Vitamin A

Vitamin A is a fat-soluble complex of hydrocarbons which is absorbed in small intestine after micellar solubilization by pancreatobiliary secretions. The absorbed vitamin is transported from enterocytes as chylomicrons to the liver which is the primary storage site [73, 84].

Monitoring of vitamin A status requires measurement of plasma retinol levels where normal levels are 20–80 mcg/dL [85]. A diminished retinol level under

10 mcg/dL indicates deficiency [80]. Clinical manifestations of deficiency are primarily related to the eye. Xerophthalmia, night blindness, is an early symptom. In addition, dryness of conjunctiva with occurrence of Bitot spots (desquamated cells most commonly seen in interpalpebral fissure on the temporal aspect of the conjunctiva) can be seen. Corneal superficial punctate keratopathy, ulcerations, and liquefaction are later signs [79].

Vitamin A deficiency is rare after bariatric surgery. However, low levels have been observed in later follow-up after BPDDS [86]. Routine replacement is recommended after bariatric surgery: 5000 IU daily after AGB and 5000–10,000 IU daily after BPDDS. Toxicity may occur in dosages over 10,000 IU daily [80].

Treatment of deficiency in the absence of corneal findings requires a dose of 10,000–250,000 IU daily until clinical improvement is documented. In the presence off corneal findings, dosage of 50,000–100,000 IU daily after BPDDS daily for 2 weeks is recommended. Evaluation of iron, copper, and zinc status should be carried out as deficiencies of these will impair resolution of vitamin A deficiency [73, 80].

Vitamin D

Vitamin D is an important nutrient in bariatric surgery because depletion is prevalent in patients with severe obesity. It is involved in many important metabolic functions including regulation of calcium and phosphorous homeostasis, bone health, immunocompetence, and cancer protection [78]. The majority of vitamin D is synthesized in the skin from cholesterol metabolites after exposure to ultraviolet rays in sunlight. Dietary vitamin D is absorbed in the distal jejunum and ileum in association with fats and bile salts [73, 83].

Assessment of vitamin D nutritional status is accomplished by measurement of the level of 25 hydroxy D in the serum. The normal range is 30–70 ng/mL, depletion level is 20–29 ng/ml, and deficient level is 10–19 ng/ml [80, 87].

Clinical manifestation of deficiency includes osteomalacia with bone pain and tenderness and weakness of proximal muscles which may mimic paraplegia in the elderly and muscular dystrophy in younger patients [79]. Treatment of deficiency requires the administration of oral calcium as well as vitamin D in a dose of 50,000 IU two to three times per week until levels exceed 30 ng/ml [80]. Supplementation of calcium and vitamin D at a dose of 3000 IU daily is indicated after bariatric surgery [80]. Lower supplementation doses may be acceptable for patients who undergo procedures leaving the small intestinal continuity intact (AGB and LSG).

The parathyroid hormone (PTH) level may be used as a biomarker for calcium and vitamin D replacement. In this situation, our current practice is to increase dosage of calcium and vitamin D in the setting of restoration to a vitamin D level of 30 ng/ml if PTH remains elevated.

Calcium

Calcium is absorbed in the duodenum and upper jejunum by an active vitamin D-mediated transport process and throughout the entire intestine by a passive process [88]. Therefore, bariatric surgery patients, especially those who undergo anatomic procedures which bypass the duodenum, require maintenance of proper calcium and vitamin D nutrition to prevent bone resorption and secondary hyperparathyroidism.

Calcium should be supplemented as the citrate salt because it can be absorbed in the absence of gastric acid. Calcium dosing should be fractionated in divided doses of 500 mg throughout the day to maximize absorption [81]. In addition, calcium should not be supplemented at the same time as iron to maximize absorption and minimize side effects. Calcium citrate should be supplemented in a dosage of 1200–1400 mg daily after AGB, LSG, and RYGB and 1800–2400 mg for BPDDS [85].

Vitamin E

Vitamin E is absorbed with dietary fat in the small intestine via passive diffusion and transported with chylomicrons [73, 83]. Information about vitamin E deficiency after bariatric surgery is limited, but deficiency with other fat-soluble vitamins as defined by serum level below normal is not uncommon after BPDDS [89]. The normal range for blood levels is 0.5–2 mg/dl [73].

Clinical manifestations of vitamin E deficiency may manifest as ataxia, weakness, and visual abnormalities [73]. Major deficiency manifestations have not yet been documented after bariatric surgery. Because vitamin E blood levels can fall after malabsorptive surgery, replacement of 15 mg per day is recommended [80]. Definitive recommendations for management of deficiency are lacking, but there is some evidence for providing 100–400 IU daily (more than in one multivitamin) for antioxidant benefit [80].

Vitamin K

Vitamin K is a fat-soluble vitamin which is absorbed with lipids in the jejunum and ileum. The vitamin is necessary for the synthesis of coagulation factors II, VII, IX, and X. Clinical manifestations of vitamin K deficiency include gum bleeding, mild bruising, and hemorrhage with bone abnormalities occurring with more advanced deficiency [73, 90]. Deficiencies of vitamin K are not infrequent after BPDDS because of the significant fat malabsorption [90].

Vitamin K nutritional status is monitored with the prothrombin time. Normal is 10–15 s with INR 0.8–1.2. Replacement after bariatric surgery is recommended in dosage of 90–120 mcg daily for AGB, LSG, and RYGB and 300 mg daily for BPDDS [80]. When deficiency is acute with manifestations of bleeding, parenteral replacement with 10 mg vitamin K is recommended. When deficiency is not acute,

recommended treatment is oral vitamin K at a dose of 1–2 mg daily or parenteral treatment with 1–2 mg per week [80].

Iron

Abnormalities of iron nutrition are among the most common nutritional complications after bariatric surgery. Dietary iron is available in two forms, elemental and heme iron. Elemental iron is ingested in the oxidized (Fe^{+++}) form. Gastric acid facilitates its solubilization and absorption takes place in the duodenum. Heme iron is also absorbed in the duodenum where pancreatic enzymes are necessary to release the heme complex from ingested myoglobin. Iron is stored in the liver, spleen, and bone marrow. The recommended dietary requirement for iron is 8–18 mg per day [80].

Factors predisposing to the development of iron deficiency include pregnancy, menstrual bleeding, and limitation in dietary intake. Deficiency does not occur until iron stores are exhausted. When this occurs, hypochromic, microcytic anemia develops. In the absence of anemia, a progressive fall in ferritin levels is a clue to iron depletion. Additional symptoms and signs of iron deficiency may include increasing fatigue, pallor, palpitations, headache, tinnitus, cold sensitivity, and anorexia.

It appears that negative iron balance is almost inevitable after bariatric surgery and that progression to deficiency is a major risk [91]. Mechanisms for diminished absorption include the absence of gastric acid, bypass of the duodenum, and alterations in food exposure to pancreaticobiliary secretions [92].

Iron deficiency is diagnosed by the presence of low levels of serum iron, low ferritin levels, and the presence of hypochromic, microcytic anemia. Normal values for ferritin include 24–336 ng/ml for males and 11–307 ng/ml for females. The normal for iron binding capacity is 11–307 mcg/L and for transferrin 170–340 mg/dL.

Routine iron supplementation is necessary after bariatric surgery recommended in a dose of 18 mg daily in a multivitamin tablet for individuals who undergo RYGB, LSG, and BPDDS and in a dose of 45–60 mg daily in menstruating females and those with a history of anemia [80]. Long-term surveillance of iron nutrition is mandatory as the incidence of significant iron deficiency has been shown to increase with time after surgery [93].

The recommended treatment for deficiency after bariatric surgery includes oral elemental iron doses ranging from 150 to 200 mg up to 300 mg two to three times daily. Oral supplementation should be taken separately from calcium supplements and antacid medications. Patients who fail to respond to oral iron will require iron infusion [80].

Zinc

Zinc is absorbed in the duodenum and proximal jejunum and to a lesser extent in the ileum through a carrier-mediated process. Pancreatic enzymes are necessary for the release of dietary zinc. Circulating zinc is bound to albumin, and conditions such as liver disease, protein malnutrition, or an inflammatory state which reduces albumin levels will reduce zinc levels [94]. Some nutritional supplements decrease zinc absorption like calcium and phytic acid. Large amounts of zinc compete with copper and iron for absorption producing deficiencies of these micronutrients. Zinc deficiency can precipitate vitamin A deficiency [73].

Currently, body zinc status is assessed by the plasma level. However, plasma levels do not correlate with tissue levels and may not be the optimal test for assessing zinc status. Measurement of red blood cell zinc levels may prove to be a better test, but is not commonly performed at present. Clinical manifestations of zinc deficiency include impaired taste and smell, poor wound healing, skin lesions, immune deficiencies, hypogonadism, anemia, photophobia, lack of dark adaptation, glossitis, diarrhea, hair loss, and paronychia [73, 79].

Normal level for plasma zinc is 70–120 mcg/dl, and for RBC zinc is 1000–1600 mcg/dl. Recommendations for replacement after bariatric surgery include 8–11 mg elemental zinc daily for AGB and 16–22 mg elemental zinc daily for RYGB, LSG, and BPDDS [80] Current definitive recommendations for treatment of zinc deficiency are lacking, but oral replacement at 60 mg twice daily has been recommended [80]. Administration should be separate from iron and calcium supplements.

Copper

Copper absorption takes place primarily in the duodenum, but also occurs in the stomach and ileum. Gastric acid and pepsin assist in the separation of bound copper from food. Calcium gluconate, phytates, zinc, iron, dietary fiber, molybdenum, and vitamin C can decrease copper absorption [73].

Copper nutritional status is assessed by the measurement of plasma copper level and the level of ceruloplasmin, a protein which transports copper. Normal values for copper are 0.75–1.45 mcg/ml and for ceruloplasmin 20–35 mg/dl. Low levels for both are diagnostic of copper deficiency.

Clinical manifestations of copper deficiency include hypochromic anemia in the presence of adequate iron stores and neurologic manifestations which include sensory ataxia, lower extremity spasticity, paresthesia of extremities, ataxia, and myeloneuropathy [73].Copper deficiency should be considered in the evaluation of patients who develop anemia after bariatric surgery.

After bariatric surgery, replacement of copper daily at a dose of 1–2 mg daily is recommended [80]. Guidelines for treatment of mild-to-moderate deficiency include provision of 3–8 mg daily as copper gluconate or sulfate until indices return to normal and, for severe deficiency, intravenous copper at a dose of 2–4 mg daily for

6 days or until neurologic manifestations resolve. Once copper levels normalize, surveillance is recommended every 3 months [80].

Summary

Bariatric surgery programs must provide nutritional expertise for expanded nutritional assessment for bariatric surgery candidates to establish both patient and procedure-specific nutrition education and monitoring programs. Factors predisposing to nutritional complications include a nutritionally poor high-energy diet among bariatric surgery candidates, limited dietary intake after surgery, avoidance of healthy foods, and surgically created malabsorption. Evidence indicates that individuals who undergo bariatric surgery procedures which involve major foregut bypass or malabsorption are at increased risk for nutritional complications.

Improved long-term follow-up, lifetime nutritional surveillance, and improved compliance with nutritional supplements will extend the health and quality of life benefits of bariatric surgery.

Medication Management Following Bariatric Surgery

Introduction

As discussed previously, bariatric surgery remains the most safe and effective long-term treatment modality for the chronic and relapsing disease of obesity. Although the sustained weight loss is important, the surgery's ability to resolve chronic comorbid medical problems such as diabetes mellitus, obstructive sleep apnea, fatty liver disease, hypertension, and lipid dyscrasias arguably makes the weight loss secondary to the profound medical benefit. It stands to reason then that bariatric patients presenting for surgery have a high incidence of these comorbid medical problems. In a published large cohort of bariatric surgery comorbidities, approximately 36% of patients were diabetic, 30% had obstructive sleep apnea, 25% had fatty liver disease, and 12% had cardiovascular disease [95].

This section will review optimal medication management following bariatric surgery.

General considerations: Following bariatric surgery, medication adjustments are often required to adequately and appropriately dose patients based on their new altered gastrointestinal anatomy. Which surgical procedures will dictate to what extent, if any, medication adjustment is required.

Despite the increased number of bariatric surgeries being performed worldwide, there remains a paucity of pharmacokinetic studies of medication absorption following bariatric surgery [96]. Similarly, there were no citations found on prescribing recommendations of disease-specific medications following bariatric surgery. To that end, the following discussed specific recommendations are class III-expert recommendations and not necessarily consensus unless otherwise referenced.

Diabetes Medications

Bariatric surgery has been shown to be a safe and effective treatment option for patients suffering from type 2 diabetes mellitus. For reasons not entirely clear, improvements in insulin sensitivity can occur immediately after bariatric surgery and even before any appreciable weight loss has occurred. Without altering preoperative diabetic medications early after surgery, there is a significant risk of unwarranted hypoglycemia. Especially following Roux-en-Y gastric bypass (RYGB) and pancreaticobiliary diversion (BPD), most insulin-required diabetic's daily insulin regimen can be held and replaced by sliding scale coverage as needed. Moreover, since PO intake is reduced both in volume and time of consumption, longer-acting insulin like glargine is usually better tolerated if post-op insulin is warranted. Similarly to insulin requirements, reduction in the dose of oral medications should be considered to prevent hypoglycemia. Depending on preoperative glycemic control, generally, patients adequately controlled with only oral agents preoperatively may require little if any medication administration, especially in the immediate postoperative period (7–10 days). If the patient does require resumption of oral medications, regular release and crush or liquid rather than sustained release/extended release formulation are recommended to maximize absorption. The newly created 15–30 mL pouch in RYGB patients has reduced surface area and reduced parietal cell mass and change in pH among other factors [97]. Metformin, both regular and extended release, may not be well tolerated due to its gastrointestinal (GI) intolerance. The thioglitazones may be better tolerated than metformin, but because of their propensity to cause weight gain or retard weight loss, they should be prescribed judiciously. Sulfonylureas, although better tolerated from the gastrointestinal standpoint, can produce significant hypoglycemia, and therefore lower doses and frequent monitoring are required.

Recently, newer classes of medications have been approved for the treatment of type 2 diabetes mellitus. These medications typically do not cause hypoglycemia, are relatively well tolerated from a gastrointestinal standpoint, and may also augment weight loss. The GLP-1 agonists and the SGLT2 inhibitors are both approved for glycemic control, and some agents have been shown to reduce cardiovascular events [98–100].

Antihypertensive Medications

For a variety of reasons, blood pressure routinely is decreased in the immediate postoperative period in patients undergoing RYGB. This, in turn, necessitates reduced doses of antihypertensive medications. Usually, medication dosages can be cut in half, and ACE inhibitors that are held 48 h prior to surgery can be restarted postoperatively at a reduced dose. Similarly, calcium channel blockers, due to their profound systemic effect on blood pressure, commonly need to be reduced in order to prevent postoperative hypotension. Diuretics for blood pressure control can usually be held. As discussed previously, medications that are prescribed should be in

the regular release and crushed/liquid formulation to ensure maximum absorption in the immediate postoperative period. Moreover, the fact that blood pressure can change rapidly in the early postoperative phase related to restricted intake and potential dehydration alone, orthostatic symptoms may be the signal that medications need to be adjusted.

Antidepressant/Mood Altering Medications

Due to the potential withdrawal effect with abruptly stopping these medications, they should be continued postoperatively. For the reasons already discussed, medications should be prescribed in the regular release, crushed/liquid form. Due to the propensity to cause weight gain, alternative medications to tricyclic antidepressants and mirtazapine specifically may be warranted if possible.

Dyslipidemic Medications

Although not as immediate as the improvement in insulin sensitivity/glycemic control, lipid profiles have been shown to significantly improve from 3 to 12 months after bariatric surgery [101]. Coupled with propensity of statins causing nausea in the immediate postoperative period and the fact that with rapid weight loss elevation in transaminase can occur, consideration for holding dyslipidemic medications for the first 12 weeks after surgery and reevaluating their need may outweigh the risk of restarting in the immediate postoperative period. If, however, a patient with known cardiovascular disease is prescribed statins for primary or secondary prevention of acute coronary events, the statins should be prescribed in the regular release formulation upon discharge.

Aspirin and Ibuprofen Products

Due to the increased risk of ulcers, strictures, and bleeding, the chronic use of aspirin and anti-inflammatory products should be avoided. Numerous studies have demonstrated increased complications, especially with concomitant tobacco use. If chronic anti-inflammatory use cannot be avoided, consideration for a bariatric procedure without the risk of marginal ulceration, such as the sleeve gastrectomy, may be in the patient's best interest. Short courses of anti-inflammatory therapy (3–10 days) for acute issue like gout attacks, migraine headaches, and acute musculoskeletal strain are usually well tolerated but should be taken with food to lessen the effect of direct mucosal irritation. Patients requiring aspirin therapy for antiplatelet cardioprotective should chew the 81 mg doses up to the prescribed amount.

Warfarin and Antiplatelet Medication

Warfarin and antiplatelet therapy such as clopidogrel bisulfate is usually held 7–10 days prior to surgery. Surgeon preferences should dictate when these medications should be restarted. If there are no complicating surgical issues, warfarin can usually be restarted safely on the evening postoperatively day 1 and bridged with low molecular weight heparin therapy until therapeutic levels of warfarin are achieved. Preoperative warfarin doses may need to be adjusted due to the altered anatomy and changes in diet postoperatively affecting vitamin K levels. Similarly, if no surgical issues occur, antiplatelet therapy can usually be safely restarted on the day of discharge from the hospital. If patients are discharged home on prolonged thromboprophylaxis, providers should be cognizant of their potential additive effects.

Oral Contraceptive Agents

For reasons probably similar to improvement in insulin sensitivity immediately after bariatric surgery, ovulatory rates improve soon after bariatric surgery despite little, if any, weight loss. However, for many reasons, pregnancy is not recommended for at least 12 months after surgery [102]. Like with other medications, oral contraception absorption is inconsistent, and therefore an alternative barrier method of birth control should be recommended. This becomes an important issue to discuss with patients, for many of them suffer from polycystic ovarian syndrome or amenorrhea with resultant infertility for many years prior to surgery and are under the impression that they will not be able to conceive. Since this is contrary to the truth, patient education and adherence to alternative barrier method of birth control is important in addition to their regular formulation of their oral contraceptive [102].

Medications such as Depo-Provera injections may be effective to prevent pregnancy, but also can inhibit weight loss after surgery and should be avoided if possible. Intrauterine devices may be a consideration as an alternative. Also, some patches in barrier methods for contraception are not recommended in women with a body mass index greater than 40 kg/m [2].

References

1. Mehaffey J, Michaels A, Mullen M, Meneveau M, Pender J, Hallowell P. Patient travel for bariatric surgery: does distance matter? Surg Obes Rel Dis. 2016. (in press) Dec 28. pii: S1550-7289(16)30889-9. https://doi.org/10.1016/j.soard.2016.12.025.
2. Gregory NS. The effects of bariatric surgery on bone metabolism. Endocrinol Metab Clin N Am. 2017;46(1):105–16.
3. Wortsman J, et al. Decreased bioavailability of vitamin D in obesity. Am J Clin Nutr. 2000;72(3):690–3.
4. Bell NH, et al. Evidence for alteration of the vitamin D-endocrine system in obese subjects. J Clin Investig. 1985;76(1):370.

5. Bredella MA, et al. Effects of Roux-en-Y gastric bypass and sleeve gastrectomy on bone mineral density and marrow adipose tissue. Bone. 2017;95:85–90.
6. Olbers T, et al. Body composition, dietary intake, and energy expenditure after laparoscopic Roux-en-Y gastric bypass and laparoscopic vertical banded gastroplasty: a randomized clinical trial. Ann Surg. 2006;244(5):715–22.
7. Carrasco F, et al. Changes in bone mineral density after sleeve gastrectomy or gastric bypass: relationships with variations in vitamin D, ghrelin, and adiponectin levels. Obes Surg. 2014;24(6):877–84.
8. Hsin M-C, et al. A case-matched study of the differences in bone mineral density 1 year after 3 different bariatric procedures. Surg Obes Relat Dis. 2015;11(1):181–5.
9. Maghrabi AH, et al. Two-year outcomes on bone density and fracture incidence in patients with T2DM randomized to bariatric surgery versus intensive medical therapy. Obesity. 2015;23(12):2344–8.
10. Muschitz C, et al. Sclerostin levels and changes in bone metabolism after bariatric surgery. J Clin Endocrinol Metabol. 2014;100(3):891–901.
11. Nogués X, et al. Bone mass loss after sleeve gastrectomy: a prospective comparative study with gastric bypass. Cirugía Española (English Edition). 2010;88(2):103–9.
12. Vilarrasa N, et al. Effect of bariatric surgery on bone mineral density: comparison of gastric bypass and sleeve gastrectomy. Obes Surg. 2013;23(12):2086–91.
13. Kushner RF, Still CD. Nutrition and bariatric surgery. CRC Press, Boca Raton, London, New York; 2014.
14. Ohlsson C, et al. Growth hormone and bone 1. Endocr Rev. 1998;19(1):55–79.
15. Fukushima N, et al. Ghrelin directly regulates bone formation. J Bone Miner Res. 2005;20(5):790–8.
16. Tsukiyama K, et al. Gastric inhibitory polypeptide as an endogenous factor promoting new bone formation after food ingestion. Mol Endocrinol. 2006;20(7):1644–51.
17. Rao RS, Kini S. GIP and bariatric surgery. Obes Surg. 2011;21(2):244–52.
18. Dirksen C, et al. Mechanisms of improved glycaemic control after Roux-en-Y gastric bypass. Diabetologia. 2012;55(7):1890–901.
19. Carrasco F, et al. Changes in bone mineral density, body composition and adiponectin levels in morbidly obese patients after bariatric surgery. Obes Surg. 2009;19(1):41–6.
20. Karsenty G. Convergence between bone and energy homeostases: leptin regulation of bone mass. Cell Metab. 2006;4(5):341–8.
21. Biagioni MFG, et al. Bariatric Roux-En-Y gastric bypass surgery: adipocyte proteins involved in increased bone remodeling in humans. Obes Surg. 2017;27(7):1789–96.
22. Balsa JA, et al. The role of serum osteoprotegerin and receptor–activator of nuclear factor-κB ligand in metabolic bone disease of women after obesity surgery. J Bone Miner Metab. 2016;34(6):655–61.
23. Organization, W.H. Assessment of fracture risk and its application to screening for postmenopausal osteoporosis: report of a WHO study group [meeting held in Rome from 22 to 25 June 1992]. 1994.
24. Cosman F, et al. Clinician's guide to prevention and treatment of osteoporosis. Osteoporos Int. 2014;25(10):2359–81.
25. Mechanick JI, et al. American Association of Clinical Endocrinologists, the Obesity Society, and American Society for Metabolic & bariatric surgery medical guidelines for clinical practice for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient. Surg Obes Relat Dis. 2008;4(5):S109–84.
26. Parrott J, et al. American Society for Metabolic and Bariatric Surgery Integrated Health Nutritional Guidelines for the Surgical Weight Loss Patient 2016 Update: micronutrients. Surgery for Obesity and Related Diseases; 2017.
27. Mechanick JI, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient—2013 update: cosponsored by American Association of Clinical Endocrinologists, the Obesity Society, and American Society for Metabolic & Bariatric Surgery. Obesity. 2013;21:S1.

28. Espino-Grosso PM, Canales BK. Kidney stones after bariatric surgery: risk assessment and mitigation. Bariatric Surg Pract Patient Care. 2017;12(1):3–9.
29. Chang AR, Grams ME, Navaneethan SD. Bariatric surgery and kidney-related outcomes. Kidney Int Rep. 2017;2(2):261–70.
30. Duffey BG, et al. Roux-en-Y gastric bypass is associated with early increased risk factors for development of calcium oxalate nephrolithiasis. J Am Coll Surg. 2008;206(6):1145–53.
31. Hatch M, et al. Oxalobacter sp. reduces urinary oxalate excretion by promoting enteric oxalate secretion. Kidney Int. 2006;69(4):691–8.
32. Borghi L, et al. Comparison of two diets for the prevention of recurrent stones in idiopathic hypercalciuria. N Engl J Med. 2002;346(2):77–84.
33. El-Nahas AR, et al. A prospective multivariate analysis of factors predicting stone disintegration by extracorporeal shock wave lithotripsy: the value of high-resolution noncontrast computed tomography. Eur Urol. 2007;51(6):1688–94.
34. Kazemi A, Frazier T, Cave M. Micronutrient-related neurologic complications following bariatric surgery. Curr Gastroenterol Rep. 2010;12(4):288–95.
35. Clements RH, et al. Incidence of vitamin deficiency after laparoscopic Roux-en-Y gastric bypass in a university hospital setting. Am Surg. 2006;72(12):1196–204.
36. Thomson AD, Marshall EJ. The natural history and pathophysiology of Wernicke's encephalopathy and Korsakoff's psychosis. Alcohol Alcohol. 2006;41(2):151–8.
37. Green R, Kinsella LJ. Current concepts in the diagnosis of cobalamin deficiency. Neurology. 1995;45(8):1435–40.
38. Amaral JF, et al. Prospective hematologic evaluation of gastric exclusion surgery for morbid obesity. Ann Surg. 1985;201(2):186.
39. Provenzale D, et al. Evidence for diminished B12 absorption after gastric bypass: oral supplementation does not prevent low plasma B12 levels in bypass patients. J Am Coll Nutr. 1992;11(1):29–35.
40. Nagarur A, Fenves AZ. Late presentation of fatal hyperammonemic encephalopathy after Roux-en-Y gastric bypass. Proc (Bayl Univ Med Cent). 2017;30(1):41.
41. Estrella J, et al. Hyperammonemic encephalopathy complicating bariatric surgery: a case study and review of the literature. Surg Obes Relat Dis. 2014;10(3):e35–8.
42. Longmuir R, Lee AG, Rouleau J. Visual loss due to Wernicke syndrome following gastric bypass. in Seminars in ophthalmology. Taylor & Francis, Boca Raton, London, New York; 2007.
43. Zuccoli G, Motti L. Atypical Wernicke's encephalopathy showing lesions in the cranial nerve nuclei and cerebellum. J Neuroimaging. 2008;18(2):194–7.
44. Carmel R. Current concepts in cobalamin deficiency. Annu Rev Med. 2000;51(1):357–75.
45. Solomon LR. Cobalamin-responsive disorders in the ambulatory care setting: unreliability of cobalamin, methylmalonic acid, and homocysteine testing. Blood. 2005;105(3):978–85.
46. Saperstein DS, et al. Challenges in the identification of cobalamin-deficiency polyneuropathy. Arch Neurol. 2003;60(9):1296–301.
47. Savage DG, et al. Sensitivity of serum methylmalonic acid and total homocysteine determinations for diagnosing cobalamin and folate deficiencies. Am J Med. 1994;96(3):239–46.
48. Service G, Thompson G, Service F, Andrews J, Collazo-Clavell M, Lloyd R. Hyperinsulinemic hypoglycemia with nesidioblastosis after gastric bypass surgery. New Engl J Med. 2005;353:249–54.
49. Meier J, Butler A, Galasso R, Butler P. Hyperinsulinemic hypoglycemia after gastric bypass surgery is not accompanied by islet hyperplasia or increased β-cell turnover. Diabetes Care. 2006;29:1554–9.
50. Shantavasinkul P, Torquati A, Corsino L. Post gastric bypass hypoglycaemia: a review. Clin Endocrinol. 2016;85:3–9.
51. Abrahamsson N, Engstrom B, Sundbom M, Karlsson F. Hypoglycemia in everyday life after gastric bypass and duodenal switch. Eur J Endocrinol Eur Fed Endocr Soc. 2014;173:91–100.

52. Lee C, Clark J, Schweitzer M, Magnuson T, Steele K, Koerner O, Brown T. Prevalence of and risk factors for hypoglycemia symptoms after gastric bypass and sleeve gastrectomy. Obesity. 2015;23:1079–84.
53. Marsk R, Jonas E, Rasmussen F, Naslund E. Nationwide cohort study of post-gastric bypass Hypoglycemia including 5040 patients undergoing surgery for obesity in 1986–2006 in Sweden. Diabetologia. 2010;53:2307–11.
54. Sarwar H, Chapman W, Pender J, Ivanescu A, Drake A, Pories W, Dar M. Hypoglycemia after Roux-en-Y gastric bypass: the BOLD experience. Obes Surg. 2014;24:1120–4.
55. Lee C, Wood G, Lazo M, Brown T, Clark J, Still C, Benotti P. Risk of post-gastric bypass hypoglycemia in nondiabetic individuals: a single center experience. Obesity. 2016;24:1342–8.
56. Seaquist E, Anderson J, Childs B, Cryer P, Dagogo-Jack S, Fish L, Heller S, Rodriguez H, Rosenzweig J, Vigersky R. Hypoglycemia and diabetes: a report of a workgroup of the American Diabetes Association and the Endocrine Society. Diabetes Care. 2013;36:1384–95.
57. Kellog T, Bantle J, Leslie D, Redmond J, Slusarek B, Swan T, Buchwald H, Ikramuddin S. Postgastric bypass hyperinsulinemic hypoglycemia syndrome: characterization and response to a modified diet. Surg Obese Rel Dis. 2008;4:492–9.
58. Vella A, Service J. Incretin hypersecretion in post-gastric bypass hypoglycemia-primary problem or red herring. J Clin Endocrinol Metab. 2007;92:4563–5.
59. Mordes J, Alonso L. Evaluation, medical therapy, and course of adult persistent hyperinsulinemic hypoglycemia after Roux-en-Y gastric bypass surgery: a case series. Encocrin Pract. 2015;21:237–46.
60. Pigeyre M, Vaurs C, Raverdy V, Hnaire H, Ritz P, Pattou F. Increased risk of OGTT-induced hypoglycemia after gastric bypass in severely obese patients with normal glucose tolerance. Surg Obes Rel Dis. 2015;11:573–7.
61. Goldfine A, Mun E, Devine E, Bernier R, Baz-Hect M, Jones D, Schneider B, Holst J, Patti M. Patients with Neuroglycopenia after gastric bypass surgery have exaggerated incretin and insulin secretory Response to a mixed meal. J Clin Endocrinol Metab. 2007;92:4678–85.
62. Salehi M, Gastaldelli A, D'Alessio D. Altered islet function and insulin clearance cause hyperinsulinemia in gastric bypass patients with symptoms of postprandial hypoglycemia. J Clin Endocrinol Metab. 2008;99:2008–17.
63. McLaughlin T, Peck M, Deacon C. Reversible hyperinsulinemic hypoglycemia after gastric bypass: a consequence of altered nutrient delivery. J Clin Endocrinol Metab. 2010;95:1851–5.
64. Frnkhouser S, Ahmad A, Perilli G, Quintana B, Vengrove M. Post-gastric bypass hypoglycemia successfully treated with alpha-glucosidase inhibitor therapy. Endocr Pract. 2013;19:511–4.
65. Spanakis E, Gragnoli C. Successful medical management of status post-Roux-En-Y Gastric bypass Hyperinsulinemic hypoglycemia. Obes Surg. 2009;19:1333–4.
66. Moreira R, Moreira R, Machado N, Goncalves T, Coutinho W. Post-prandial hypoglycemia after bariatric surgery: pharmacological treatment with verapamil and acarbose. Obes Surg. 2008;18:1618–21.
67. Myint K, Greenfield J, Farooqi I, Henning E, Holst J, Finer N. Prolonged Suddessful therapy for hyperinsulinemic hypoglycemia after gastric bypass: the pathophysical role of GLP-1 and its response to a somatostatin analogue. Eur J Endocrinol. 2012;166:951–5.
68. Salehi M, Gastaldelli A, D'Alessio D. Blockade of glucagon-like peptide 1 receptor corrects postprandial hypoglycemia after gastric bypass. Gastroenterology. 2014;46:669–80.
69. Vanderveen K, Grant C, Thompson G, Farley D, Richards M, Vella A, Vollrath B, Service J. Outcomes and quality of life after partial pancreatectomy for noninsulinoma pancreatogenous hypoglycemia from diffuse islet cell disease. Surgery. 2010;148:1237–46.
70. Ohrstrom C, Worm D, Hansen D. Postprandial hyperinsulinemic hypoglycemia after Roux-en-Y gastric bypass: An Update. Surg Obes Rel Dis. https://doi.org/10.1016/j.soard.2016.09.025.
71. Eisenberg D, Azagury D, Ghiassi S, Grover B, Kim J. ASMBS position statement on postprandial hyperinsulinemic hypoglycemia after bariatric surgery. Surg Obes Rel Dis. 2017;13:371–378. Service G, Thompson G, Service F, Andrews J, Collazo-Clavell M, Lloyd

R. Hyperinsulinemic Hypoglycemia with Nesidioblastosis after Gastric Bypass Surgery. New Engl J Med. 2005;353:249–54.
72. Bal B, Finelli F, Shope T, Koch T. Nutritional deficiencies after bariatric surgery. Nat Rev Endocrinol. 2012;8:544–56.
73. Mueller C. The ASPEN adult nutrition support core curriculum. 2nd edn. In: Clark SF, editor. Vitamins and trace elements. American Society for Parenteral and Enteral Nutrition. 2012; 8. p. 121–48.
74. Levinson R, Silverman JB, Catella JG, Rybak I, Jolin H. Isom K pharmacotherapy prevention and management of nutritional deficiencies post roux-en-Y gastric bypass. Obesity Surg. 2013;23:992.
75. Mueller C. The ASPEN adult nutrition support core curriculum, 2nd edn. In: Colaizzo-Anas T, editor. CDN nutrient intake, digestion, absorption, and excretion. American Society for Parenteral and Enteral Nutrition. 2012;1. p. 16.
76. Moize V, Andreu A, Rodriguez L, Flores L, Ibarzabal A, Lacey A, et al. Protein intake and lean tissue mass retention following bariatric surgery. Clin Nut. 2013;32:550–5.
77. Mueller C. The ASPEN adult nutrition support core curriculum, 2nd edn. In: Jensen GL, Hsiao PY, Wheeler D, editors. Nutrition screening and assessment. American Society for Parenteral and Enteral Nutrition. 2012; 9. p. 158,163.
78. Benotti P. Patient preparation for bariatric surgery New York: Springer; 2014. Chapter 7.
79. Ross AC, Caballero B, Cousins RJ, Ticker KL, Ziegler TR. **Modern nutrition in health and disease. 11th edn. In: Heimburger DC, editor. Clinical manifestations of nutrient deficiencies and toxicities. Lipppincot, Williams and Wilkins. 2014; 57. p. 757–66.
80. Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom K, Greiman L. American Society for Metabolic and Bariatric Surgery Integrated Health Nutritional Guidelines for the Surgical Weight Loss Patient 2016 Update: Micronutrients. Surg Obes Rel Dis. 2017;13:727–41.
81. Heber D, Greenway F, Kaplan L, Livingston E, Salvador J, Still C. Endocrine and nutritional management of the post-bariatric surgery patient: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2010;95:4823–43.
82. Stabler S. Vitamin B12 deficiency. New Engl J Med. 2013;368:149–60.
83. Hwang C, Ross V, Mahadevan U. Inflammatory bowel disease, micronutrient deficiencies. Inflamm Bowel Dis A Zinc. 2012;18(10):1966.
84. Toh S, Zaarshenas N, Jorgensen J. Prevalence of nutrient deficiencies in bariatric patients. Surg Obes Rel Dis. 2009;25:1150–6.
85. Valentino D, Sriram K, Shankar P. Update on micronutrients in bariatric surgery. Curr Opin Clin Nutr Metab Care. 2011;14:635–41.
86. Dolan K, Hatzifotis M, Newbury L, Lowe N, Fielding G. A clinical and nutritional comparison of biliopancreatic diversion with and without duodenal switch. Ann Surg. 2004;240:51–6.
87. Bacci V, Silecchia G. Vitamin D status and supplementation before and after bariatric surgery. Expert Rev Gastroenteiol Hepatol. 2010;4:781–94.
88. Bronner F. Mechanisms of intestinal calcium absorption. J Cell Biol. 2003;88:387–93.
89. Homan J, Betzel B, Aarts E, Dogan K, van Laarhoven K, Janssen I, Berends R. Vitamin and mineral deficiencies after biliopancreatic diversion and biliopancreatic diversion with duodenal switch- the rule rather than the exception. Obes Surg. 2015;25:1626–32.
90. Slater G, Ren C, Siegel N, Williams T, Barr D, Wolfe B, Dolan K, Fielding G. Serum fat-soluble vitamin deficiency and abnormal calcium metabolism after malabsorptive bariatric surgery. J Gastrointest Surg. 2004;8:48–55.
91. Ruz M, Carrasco F, Rojas P, Codosceo J, Inostroza J, Basfi-fer K, Valencia A, Csendes A, Papapietro K, Pizarro F, Olivares M, Westcott J, Hambridge M, Krebs N. Heme- and Nonheme-iron absorption nd iron status 12 mo after sleeve gastrectomy and roux-en-Y gastric bypass in morbidly obese women. Am J Clin Nut. 2012;96:810–7.
92. Love A, Billet H. Obesity, bariatric surgery and iron deficiency: true, true. True and Related Am J Hematol. 2008;88:403–9.
93. Monaco-Ferreira DV, Leandro-Merhi V. Status of iron metabolism 10 years after Roux-en-Y gastric bypass. Obes Surg. 2017. https://doi.org/10.1007/xs11695-017-2582-0.

94. Salle A, Demarsy D, Poirier A, Lelievre B, Top O, Guilloteau G, et al. Zinc deficiency: a frequent and underestimated complication after bariatric surgery. Obes Surg. 2010;20:1660–70.
95. Wood GC, Chu X, Manney C, Sgtrodel W, Petrick A, Gabrielsen J, et al. An electronic health record-enabled obesity database. BMC Med Inform Decis Mak. 2012;12:45. https://doi.org/10.1186/1472-6947-12-45.
96. Padwal R, Brocks D, Sharma AM. A systemic review of drug absorption following bariatric surgery and its theoretical implications. Obes Rev. 2010;11(1):41–50.
97. Miller AD, Smith KM. Medication and nutrient administration considerations after bariatric surgery. Am J Health Syst Pharm. 2006;63(19):1852–7.
98. Marso SP, Daniels GH, Brown-Frandsen K, Kristensen P, Mann JFE, et al. Liraglutide and cardiovascular outcomes in type 2 diabetes. N Engl J Med. 2016;375:311–22.
99. Zinman B, Wanner C, Lachin JM, Fitchett D, Bluhmki E, Hantel S, Mattheus M, Devins T, Johansen OE, Woerle HJ, Broedl UC, Inzucchi SE, EMPA-REG OUTCOME Investigators. Empagliflozin, cardiovascular outcomes, and mortality in type 2 diabetes. N Engl J Med. 2015;373:2117–28.
100. Neal B, Perkovic V, Mahaffey KW, on behalf of the CANVAS Program Collaborative Group, et al. Canagliflozin and cardiovascular and renal events in type 2 diabetes. N Engl J Med 2017;377:644–57. [Epub ahead of print].
101. Milone M, Lupoli R, Maietta P, Di Minno A, et al. Lipid profile changes in patients undergoing bariatric surgery: a comparative study between sleeve gastrectomy and mini-gastric bypass. IJOS. 2015;14:28–32.
102. Monson M, Jackson M. Pregnancy after bariatric surgery. Clin Obstet Gynecol. 2016;59(1):158–71.

Emergencies in Bariatric Surgery

3

John-Paul Bellistri and Erin Moran-Atkin

Introduction

Any provider performing bariatric surgery needs to have a high index of suspicion for complications and a low threshold to intervene in the postoperative bariatric patient. In patients that have undergone bariatric surgery, there is a very fine line between presenting complaints with subtle clinical findings and catastrophic hemodynamic collapse. When evaluating a postoperative bariatric patient, the clinician needs to consider the type of surgery performed, timing of presentation relative to the index surgery, and preexisting comorbidities in order to complete the requisite investigations and implement the appropriate treatment plan.

Multiple studies have suggested improved patient outcomes in high volume centers and in the hands of high volume surgeons at American Society of Metabolic and Bariatric Surgery (ASMBS) accredited centers [1–3]. However, this has been called into question by studies produced by the Center for Outcomes and Policy at the University of Michigan, which have shown that risk-adjusted morbidity may be a better predictor of outcomes than hospital and surgeon volume. In addition, there is a considerable degree of variation in outcomes among accredited bariatric surgery centers in regard to hospital volume and serious postoperative complications [4].

Irrespective of ASMBS accreditation, hospital volume, and surgeon volume, it is imperative that hospital systems are organized so that surgery personnel are notified

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immediately to evaluate bariatric patients on presentation to the emergency department or to evaluate any change in clinical status on the wards. Ability to escalate care in order to prevent failure to rescue is the key to preventing the evolution of a complication from a treatable outcome to a life-threatening morbidity [5, 6].

Bariatric Emergencies: Presentation, Workup, and Treatment

Venous Thromboembolism

Venous thromboembolic events (VTE), which include deep vein thrombosis (DVT) and pulmonary embolism (PE), are considered the most preventable causes of death in the surgical patient. The Caprini score [7] (Table 3.1) is a validated scoring system for assessing risk of VTE. Each risk factor is assigned a point value, and patients

Table 3.1 Caprini score stratifies patients based upon risk of VTE within 30 days of surgery

1 point	2 points	3 points	4 points
Age 41–60 years	Age 61–74 years	Age >75 years	Stroke (<1 month)
Swollen legs (current)	Arthroscopic surgery	History of DVT/PE	Elective lower extremity arthroplasty
Varicose veins	Malignancy	Factor V Leiden	Hip, pelvis, or leg fracture (<1 month)
BMI >25	Laparoscopic surgery (>45 Min)	Elevated homocysteine	Acute spinal cord injury (<1 month)
Minor surgery	Patients confined to bed rest (>72 h)	Heparin-induced thrombocytopenia	Multiple trauma (<1 month)
Sepsis (within last month)	Immobilizing plaster cast (>1 month)	Elevated anticardiolipin	
Serious lung disease	Central venous access	Lupus anticoagulant	
Oral contraceptives	Major surgery (>45 min)	Prothrombin 202110A	
Pregnancy or postpartum <1 month		Family history of thrombosis	
History of stillborn infant, recurrent spontaneous abortion, premature birth			
Acute myocardial infarction			
History of inflammatory bowel disease			
History of prior major surgery (<1 month)			
Abnormal pulmonary function			

are risk stratified as follows: very low (no risk factors), low (1–2 points), moderate (3–4 points), and high (5 points and greater). All patients undergoing bariatric surgery are at a high risk of VTE by merit of their obesity and the nature of the surgery. Majority of the bariatric population also has other risk factors including higher age, immobility, venous stasis disease, obesity-related hypoventilation syndrome, hypercoagulable states, or prior hormonal therapy, which add to the risk of VTE [8, 9].

Most VTE take place within 30 days of surgery, but risk can persist up to a year postoperatively. DVT can present as leg pain or unilateral leg swelling and can be diagnosed easily on lower extremity venous duplex. Treatment is therapeutic anticoagulation, which should be continued as an outpatient on either a vitamin K antagonist or new oral anticoagulant. PE can present with tachycardia, hypoxemia, or hypotension. When suspected, contrast-enhanced computerized tomography (CT) is the study of choice to investigate for a PE. In addition, chest x-ray, EKG, troponins, basic metabolic panel, and complete blood counts should be sent off to rule out other etiologies. If the treatment with anticoagulation is to be initiated, it is important to rule out bleeding as an etiology of the patient's presentation.

Portomesenteric vein thrombosis is another type of VTE that is encountered in the bariatric population [10]. This clinical entity is usually associated with laparoscopic sleeve gastrectomy but is also seen after gastric bypass. Patients usually present with abdominal pain, nausea, and vomiting. Diagnosis is made on contrast-enhanced CT scan. CT also enables identification of infarcted bowel and the extent of thrombosis. In addition, liver function tests should be trended in order to identify and monitor for signs of liver injury. Portal vein, superior mesenteric vein, and splenic vein thromboses have all been described. The treatment is therapeutic anticoagulation for at least 6 months. Follow-up imaging should be performed at 6 months and 1 year.

Any patient diagnosed with a VTE should undergo a full hematologic evaluation to investigate for Factor V Leiden, prothrombin G20210A mutation, protein S deficiency, antithrombin-III deficiency, activated protein C resistance, antiphospholipid syndrome, and JAK-2 V617F mutation.

Cardiac Complications

Myocardial infarction and sudden cardiac death are possible causes of death in patients undergoing bariatric surgery. Any patient with abnormal heart rate, blood pressure, respiratory rate, or oxygen saturation should be worked up for a cardiac etiology. This includes an EKG, troponin levels, and CK levels. Patients with known coronary artery disease are at an increased risk of perioperative cardiac event. Patients on aspirin and clopidogrel preoperatively usually stop 5 days and 3 days before surgery, respectively. These should be restarted as soon as possible after surgery when bleeding is no longer a risk. It is also important to ensure that patients on aspirin understand the importance of adherence to a proton pump inhibitor regimen. In addition, obesity is a risk factor for obesity-related cardiomyopathy, which

increases risk of cardiac arrhythmia. Any findings of ST elevation on EKG, elevated troponins, or cardiac arrhythmia on EKG should warrant immediate consultation by a cardiologist.

Nutritional Deficiencies

Nutritional deficiencies and metabolic derangements are known complications of bariatric surgery, especially in those that require rerouting of the alimentary tract. However, thiamine and vitamin B12 deficiencies require immediate attention, as neglect may result in irreversible neurologic sequelae. Thiamine is a water-soluble vitamin and is not stored in the body. Therefore, consistent nutritional supplementation is required for normal physiological functioning. Bariatric patients presenting to the emergency room with intractable nausea and vomiting in the early postoperative period should be empirically supplemented with thiamine intravenously. These patients may have undergone any type of bariatric surgery, not just those with alimentary tract rerouting. Failure to identify and treat patients with thiamine deficiency may result in heart failure or Wernicke's encephalopathy (confusion, ataxia, nystagmus). Wernicke's encephalopathy may be irreversible [11].

Vitamin B12 deficiency is a risk for patients who have undergone gastric bypass because of the small gastric pouch with little intrinsic factor production. The liver has an approximately 12-month storage of vitamin B12, and therefore, the risk of deficiency usually manifests at least 12 months after surgery. Deficiency may results in ataxia and loss of lower extremity proprioception due to damage to the posterior spinal columns, which can be irreversible. Patients with any signs of vitamin B12 deficiency should be supplemented by intramuscular injection. Sublingual tablets are rapidly absorbed into the bloodstream obviating the need for intrinsic factor and alimentary tract absorption and can be used by patients at home.

Staple Line Leak

Staple line leak, whether at an anastomosis or in the setting of a sleeve gastrectomy, is one of the most common and most feared complications of bariatric surgery. Staple line disruption occurs at a rate of approximately 0–7% after a sleeve gastrectomy and 0.1–8.3% after a gastric bypass. Leaks can be categorized as early or late. Early leaks are usually detected within the first few days postoperatively and are resultant of technical error. Technical considerations to avoid staple line leak include careful handling of the tissue, utilizing appropriate staple heights to accommodate varying tissue thickness, avoiding narrowing of the alimentary tract (e.g., at the incisura angularis for sleeve gastrectomy or jejunojejunal anastomosis of gastric bypass), avoiding twisting or kinking of tissue, and ensuring appropriate blood supply. Late leaks can occur up to weeks postoperatively and are usually the result of tissue ischemia. These patients may present with isolated tachycardia. Because of body habitus and timing of leakage, these patients usually do not have

an impressive abdominal exam. Therefore, isolated tachycardia, especially above 120 beats per minute, should warrant investigation with either an upper GI series or CT scan with oral contrast. Any patient with staple line disruption needs early and aggressive fluid resuscitation with monitoring of urinary output and central venous pressure, preferably in the ICU setting. Early antibiotic therapy should be implemented with broad spectrum coverage. All of this is done in preparation for emergent invasive intervention [12].

Staple line disruption in the setting of a sleeve gastrectomy is usually the result of narrowing at the incisura angularis with subsequent increased pressure and perforation at the top of the staple line near the angle of His. The first step in treatment of this complication is drainage and source control. If there is a discrete collection, it should be drained either by interventional radiology or laparoscopically. Microbiologic analysis should be sent off for any fluid drained. There are times when a leak has sealed off before any investigation, and the only invasive measure necessary is drainage of a collection. In the setting of persistent staple line disruption, the endoscopic placement of an expandable covered stent over the area of staple line disruption can be performed under fluoroscopic guidance. This is kept in place for 4 weeks, at which time it is removed, and fluoroscopic investigation is performed to ensure healing of staple line. If staple line is still not healed, then another stent is reinserted to allow for further healing. Providers must consider whether there is narrowing at the incisura angularis that is contributing to an increased sleeve pressure. If there is radiographic evidence of narrowing on fluoroscopy, then the incisura angularis must be dilated prior to removing the stent. In cases of early staple line disruption (within 48–72 h of index surgery), some advocate for primary oversewing of staple line. The authors favor drainage of the area and placement of an omental buttress to aid in healing, in addition to stent placement, in early leaks.

The gastric bypass has numerous staple lines that may leak: gastrojejunal anastomosis, jejunojejunal anastomosis, gastric pouch staple line, and excluded stomach staple line. The most common of these is a leak from the gastrojejunal anastomosis. These leaks require prompt return to the operating room for washout and wide drainage. If a discrete area of staple line disruption is identified, then this can be oversewn with an omental patch or oversewn with a serosal patch from the excluded stomach. If the area of staple line disruption is not identified, then endoscopy should be performed to help aid with identification. If there is still no defect identified endoscopically, then wide drainage is sufficient. A nasogastric tube is left with the tip above the level of the anastomosis. A study is then performed on postoperative day 4 before removing the nasogastric tube. Decision should be made intraoperatively whether to place a feeding access in the excluded stomach. This is dependent upon the hemodynamic status of the patient and the degree of anastomotic disruption. In patients who are hemodynamically unstable, it is not possible to attempt any type of repair on initial exploration. Therefore, it may be advisable to stage the operative intervention by resecting the anastomosis, leaving the patient in discontinuity, and planning for definitive reconstruction in 24–48 h after resuscitation has been completed.

Perforated Marginal Ulcer

Marginal ulcers after gastric bypass surgery usually occur at a mean of 1 year postoperatively. Risk factors include tobacco use, alcohol use, nonsteroidal anti-inflammatory drug use, *H. pylori* infection, ischemia, and foreign body (staple or nonabsorbable suture). Patients with perforated marginal ulceration usually present with an acute abdomen and abdominal sepsis. They may have lost a considerable amount of weight, and therefore, physical exam findings may be more prominent compared with early postoperative patients. These patients should be started on broad spectrum antibiotics with fungal coverage and aggressively resuscitated in preparation for the operating room. Patients should also be placed on a proton pump inhibitor (PPI) drip. These perforations are usually amenable to omental patch repair using 2–0 absorbable suture material. Intraperitoneal fluid should be sent for microbiologic analysis to allow for narrowing of antibiotic coverage. A nasogastric tube is left in place, and a drain should be left adjacent to repair. Upper GI fluoroscopy should be performed postoperative day 4 before removing the nasogastric tube. Antibiotics are usually continued for 5 days postoperatively, or until signs of sepsis have resolved. The intraperitoneal drain is left in place until 2 week postoperative visit, and the patient is maintained on a liquid diet during this time.

Bleeding

Typical signs of postoperative bleeding include tachycardia, hypotension, oliguria, and decrease in hematocrit. Bleeding may occur from any number of anatomic locations including the short gastric vessels, greater omentum, any staple line or anastomosis, spleen, and liver. Bleeding that requires greater than two units of packed red blood cells should prompt invasive intervention. Bleeding can be intraluminal or intraperitoneal. Most intraperitoneal bleeding can be dealt with laparoscopically.

Intraluminal bleeding can be diagnostically and therapeutically challenging. It can occur from the gastrojejunal (GJ) anastomosis, jejunojejunal (JJ) anastomosis, or the staple line of the remnant stomach. All patients with suspected intraluminal bleeding should immediately be placed on a proton pump inhibitor drip. Acid secretion in this setting serves to inhibit coagulation. Use of intraoperative endoscopy is essential for addressing anastomotic bleeding. If bleeding is identified at the GJ anastomosis, the staple line can be oversewn laparoscopically. Endoscopic intraoperative assistance can ensure that the bleeding area of the GJ anastomosis is properly addressed. If there is concern of bleeding from the JJ anastomosis, then the bowel can pushed over an endoscope to allow for passage down to the anastomosis. Endoscopic clipping or injection should be attempted. If these are not successful, the JJ anastomosis can be oversewn. Otherwise, the anastomosis may need to be revised. Bleeding from the remnant stomach may be apparent by a distention on laparoscopic examination. In such cases, a gastrotomy is created, blood clots are evacuated, and the staple line is oversewn with absorbable monofilament suture. The gastrotomy is then stapled closed.

Small Bowel Obstruction

Small bowel obstruction is a challenging entity in patients who have undergone bariatric surgery and may be attributed to adhesions, intraluminal blood clot, intussusception, or internal hernia. Intraluminal blood clot may be a source of bowel obstruction in the early postoperative patient. In particular, patients that have undergone gastric bypass are at high risk of complications due to the inability to decompress the excluded stomach. Obstruction of the roux limb leading to staple line disruption of the excluded stomach is catastrophic with a high risk of mortality. Patients will often experience postoperative nausea, vomiting, and abdominal pain. When suspected, chemical DVT prophylaxis is held, and CT scan should be performed, which will show intraluminal blood clot and a distended remnant stomach. The patient is made NPO, and the remnant stomach is decompressed either by interventional radiology with a pigtail catheter or by laparoscopic gastrostomy creation in the operating room [13–15].

Small bowel intussusception can occur at any jejunojejunal anastomosis. This occurs in less than 1% of gastric bypass patients and usually presents as a small bowel obstruction. Diagnosis is made on CT scan. Treatment requires a return to the operating room with resection and revision of the anastomosis [16].

An internal hernia can occur at any number of mesenteric defects in bariatric surgery: jejunojejunal anastomosis, Petersen's defect, and transverse mesocolon defect of retrocolic roux limb. Internal herniation of bowel is usually a late complication. As patients lose weight with a subsequent reduction in mesenteric adipose tissue, these defects become more prominent and allow for internal hernia formation. Closure of the mesenteric defect at the jejunojejunal anastomosis has led to considerable decrease in internal hernia at this site. Antecolic orientation of roux reconstructions has reduced the incidence of internal herniation at Petersen's defect compared with the retrocolic orientation. An antecolic orientation also eliminates the risk of herniation through a transverse mesocolic defect. The diagnostic modality of choice is a CT scan, which will demonstrate a "mesenteric swirl" (Fig. 3.1).

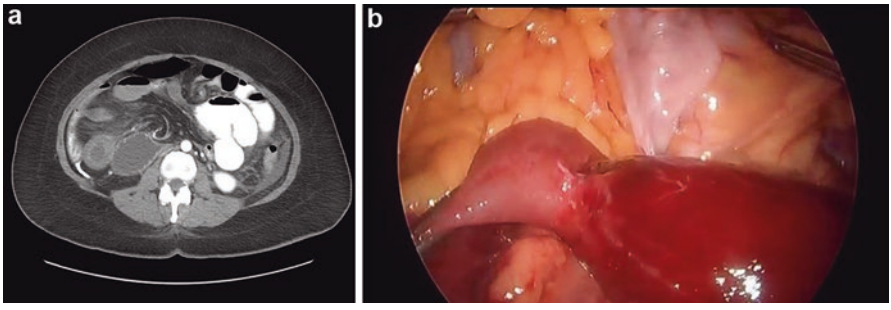

Fig. 3.1 CT scan and intraoperative findings of a patient with an internal hernia after gastric bypass. (**a**) CT scan of patient with internal hernia demonstrating mesenteric swirling. (**b**) Intraoperative findings of patient with internal hernia and impending bowel infarction

However, even in the absence of radiographic findings, laparoscopic exploration is indicated in the presence of any clinical suspicion [13, 14].

Any concern for internal hernia should prompt immediate return to the operating to avoid mesenteric ischemia and infarction of the midgut. Most internal hernias can be approached laparoscopically. The best approach is to begin running the small bowel from the terminal ileum. At this location the bowel is relatively decompressed, which reduces the risk of iatrogenic bowel injury. In addition, running the bowel from this location reduces the possibility of running the small bowel in the wrong direction, which may worsen strangulation of the bowel. Mesenteric defects should be closed with 2–0 nonabsorbable suture.

Gastric Band Complications

The two major complications of gastric band placement are band slippage and band erosion. Band slippage is an emergency. Band erosion although an urgent matter is not an emergency and, therefore, will not be discussed in this chapter. Band slippage occurs when the gastric fundus slips above the band causing a gastric obstruction and raising the risk of gastric ischemia and necrosis. The diagnosis can be made on CT scan or plain abdominal film (Fig. 3.2). Normally the gastric band is oriented in a 1–7 o'clock position. With slippage, the band is oriented in a 4–10 o'clock position, which can be appreciated on plain abdominal x-ray. The band should be immediately decompressed with emergent take back to the operating room. If there is no necrosis of bowel, then simple removal of the band is sufficient. If there is gastric necrosis, resection of necrotic stomach is necessary with creation of venting gastrostomy in the proximal stomach and feeding gastrostomy in the distal stomach. Reconstruction should be reserved for a later date as these patients are usually in extremis with severe metabolic derangements precluding safe reconstruction on initial exploration. After approximately 3–4 months, a reconstruction can be attempted with either a gastrogastrostomy or a conversion to a Roux-en-Y gastric bypass.

Critical Care in the Obese Patient

Obese patients do not tolerate physiologic insults in the same manner as nonobese patients. Bariatric patients admitted to the intensive care unit with sepsis have up to a 30% mortality rate [17].

The cardiovascular system undergoes a number of profound changes in the obese patient resultant of the increase in adipose tissue with subsequent neurohormonal and metabolic alterations on the heart. For every kg of adipose tissue, the body requires and extra 30 mL of circulating blood. This leads to a subsequent increase in stroke volume and cardiac output. Left and right ventricular hypertrophy and enlargement ensue. In addition, patients with metabolic syndrome have blunted

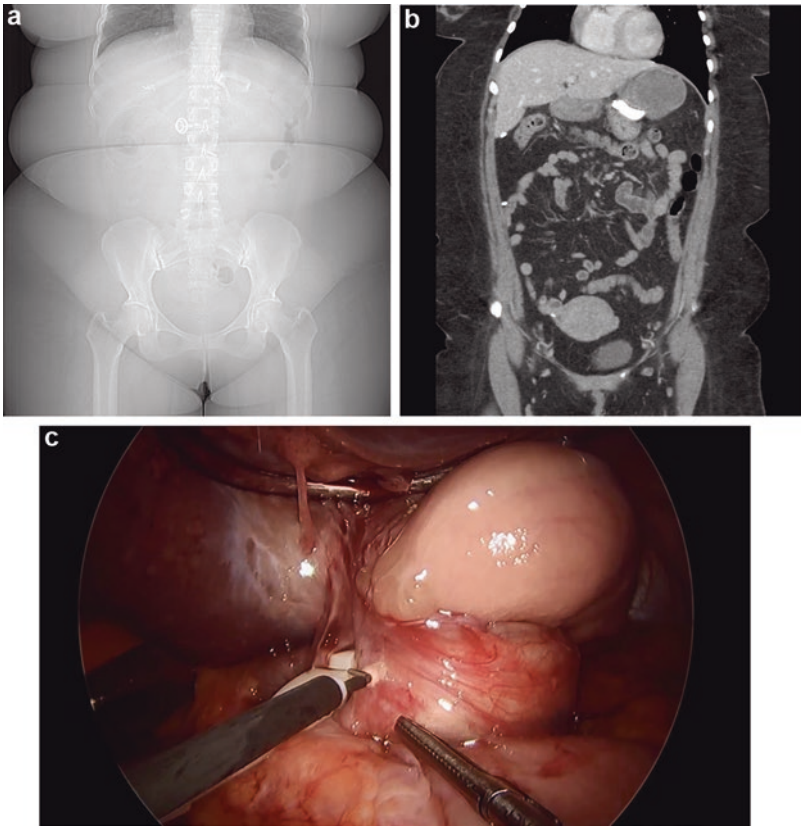

Fig. 3.2 CT scan and intraoperative findings of a patient with a slipped gastric band. (**a**) Scout film from CT scan demonstrating 4–10 o'clock positioning of slipped gastric band. (**b**) CT scan demonstrating slipped band with fundus herniating above the band. (**c**) Intraoperative findings demonstrating slipped band with herniated fundus above the band. A combination of sharp and monopolar energy dissection is used to remove the band

coronary vasodilator responsiveness to stress. All of these effects of obesity on the heart lead to increased sensitivity of the heart to physiologic insult and a decrease in sensitivity to cardiovascular medications, which further complicates their management [18].

Obesity has a profound impact on pulmonary functioning. The obese patient has a decrease in chest wall compliance and functional residual capacity. Therefore, calculations for tidal volumes should be based upon ideal body weight in order to avoid ventilator-induced lung injury. In addition, obstructive sleep apnea and obesity hypoventilation syndrome both contribute to hypoxia, acidosis, and pulmonary hypertension in obese patients [19].

References

1. Nguyen NT, et al. Outcomes of bariatric surgery performed at accredited vs nonaccredited centers. J Am Coll Surg. 2012;215:467–74.
2. Young MT, et al. A decade of analysis of trends and outcomes in baraitric surgery in medicare beneficiaries. J Am Coll Surg. 2014;219:480–8.
3. Telem DA, et al. The effect of national hospital accreditation in bariatric surgery on perioperative outcomes and long-term mortality. Surg Obes Relat Dis. 2015;11:749–57.
4. Dimick JB, et al. Identifying high-quality baraitric surgery centers: hospital volume or risk-adjusted outcomes? J Am Coll Surg. 2009;209:702–6.
5. Johnston, et al. A systematic review to identify the factors that affect failure to rescue and escalation of care in surgery. Surgery. 2015;157:752–63.
6. Tao W, et al. Casues and risk factors for mortality within 1 year after obesity surgery in a population-based cohort study. Surg Obes Relat Dis. 2015;11:399–405.
7. Bahl V, et al. A validation study of retrospetive venous thromboembolsm risk scoring method. Ann Surg. 2010;251:344–50.
8. Rowland SP, et al. Inferior vena cava filters for prevention of venous thromboembolism in obese patients undergoing bariatric surgery. Ann Surg. 2015;261:35–45.
9. The American Society for Metabolic and Bariatric Surgery Clinical Issues Committee. ASMBS updated position statement on prophylactic measures to reduce the risk of venous thromboembolism in bariatric surgery patients. Surg Obes Relat Dis. 2013;9:493–7.
10. Lakis MA, et al. Portomesenteric vein thrombosis after laparoscopic sleeve gastrectomy and laparoscopic roux-en-y gastric bypass: a 36-case series. Surg Endosc. 2017;31:1005–11.
11. Campanile FC, et al. Acute complications after laparoscopic bariatric procedures: update for the general surgeon. Langenbeck's Arch Surg. 2013;398:669–86.
12. Ghosh S, et al. A narrative of intraoperative staple line leaks and bleeds during bariatric surgery. Obes Surg. 2016;26:1601–6.
13. Brolin RE, et al. Impact of complete mesenteric closure on small bowel obstruction and internal mesenteric hernia after laparoscopic roux-en-y gastric bypass. Surg Obes Relat Dis. 2013;9:850–5.
14. Facchiano E, et al. Laparoscopic management of internal Hernia after Roux-En-y Gastric Bypass. Obes Surg. 2016;26:1363–5.
15. Green J, et al. Acute small bowel obstruction due to a large intraluminal clot after laparoscopic Roux-en-Y gastric bypass. JSCR. 2016;8:1–3.
16. Birnbaum DJ, et al. Intussusception involving Roux-en-Y limb following gastric bypass. J Gastroinest Surg. 2017;21:415–6.
17. Finks JS, et al. Predicting risk for serious complications with bariatric surgery. Ann Surg. 2011;254:633–40.
18. Alpert MA, et al. Effects of obesity on cardiovascular hemodynamics, cardiac morphology, and ventricular function. Curr Obes Rep. 2016;5:424–34.
19. Ortiz VE, et al. Obesity: physiologic changes and implications for preoperative management. BMC Anesth. 2015;15:97.

Management of Marginal Ulceration

4

Jenny Choi and Caitlin Polistena

Gastric bypass surgery was developed in the 1960s after observations of sustained weight loss in patients undergoing partial stomach removal for peptic ulcer disease [1]. Since that time, marginal ulcers (MU) have haunted gastric bypass procedures as a cause of significant morbidity. Marginal, or stomal, ulcers are defined as ulcerations on the jejunal aspect of the gastrojejunal anastomosis of a Roux-en-Y gastric bypass (RYGB), though any ulcer near the gastrojejunostomy is often labeled a marginal ulcer [2]. The majority of marginal ulcers (MUs) are located at the anastomosis (50%) or in the jejunum (40%), with the remainder occurring in the gastric pouch [3].

Incidence

Most studies place incidence of MU in the RYGB population between 0.6% and 25% [4]. However, there have been reports in the literature with incidence as high as 34% [2]. The true incidence is likely on the higher end of these estimates given asymptomatic patients are generally not evaluated endoscopically [5, 6]. While MUs may occur at any point in time after RYGB, with literature reporting MU occurrence as early as 1 month postoperatively and as late as 8 years, most seem to occur within 1 year of surgery [2, 7]. Csendes et al. [8] prospectively studied 315 RYGB patients, performing endoscopy at 1 month and 17 months postoperatively regardless of symptoms. MUs were detected in 25 patients (6%) at 1 month. Of these, 28% (7/25) were asymptomatic. Repeat endoscopy identified only one new ulcer and one recurrent ulcer despite PPI treatment. This led to the concept of early versus late MU, with incidence rates of 6% and 0.6%, respectively. A follow-up study of 550 patients undergoing serial endoscopy to assess for late MU revealed a

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1% incidence, with 4 MUs identified greater than 4 years postoperatively [7]. The exact definitions of early and late MU have shifted over time, but the generally accepted version is <12 months versus >12 months, respectively. Early MUs are more common than late, and it is believed that the etiology of ulcer formation differs for each group [7, 8].

Etiology

The etiology of MU development has been widely discussed over many decades and remains controversial. Mason and colleagues in 1976 described high gastric pouch acidity as the culprit. In addition, it has been well recognized from research on peptic ulcer disease and the Billroth procedures that an association exists between presence of parietal-cell-rich portions of the stomach and gastrojejunostomy ulcer formation [9]. The literature on gastric bypass has since supported this relationship [10, 11, 12]. For example, Hedberg et al. (2005) studied pH probe readings in post-RYGB patients and found that those with MUs were exposed to pH <4 for a greater percentage of time over a 4 h period compared to non-MU controls (69% vs. 20%). All of the patients examined in these studies of gastric pouch pH had MUs associated with gastrogastric fistulas (GGF), and the strong association between MU and GGF has subsequently been well described [3, 13–15]. Thus gastric bypass evolved to include separation of the remnant stomach from the gastric pouch to minimize the incidence of GGF [16].

Another significant modification in RYGB technique to come about in an effort to decrease incidence of MU formation is the reduction of gastric pouch size. It was theorized that creating a smaller pouch would exclude more acid secreting parietal cells, minimize acid release, and hence reduce stomal ulceration rate [17–20]. However, while dilated pouches or those >6 cm in size have been associated with MU formation, there have been studies suggesting pouch size is noncontributory [2, 3]. Behrns et al. [21], for example, provided evidence that little gastric acid is produced in the pouch, and Maclean et al. [11] showed high acid levels in the pouch only when a GGF was present.

Despite the trends of creating smaller gastric pouches and separation of the remnant stomach from the pouch to reduce the incidence of GGF, MUs continue to be problematic after RYGB, suggesting that other potential etiologies and risk factors for their development exist. Additional technical aspects of RYGB have been extensively studied for their association with MU development, including type of suture used, type of gastrojejunal anastomosis performed, and positioning of the Roux limb. While the use of absorbable instead of nonabsorbable suture significantly reduces the incidence of MU, the type of anastomosis and Roux limb position do not appear to influence ulcer formation.

Nonabsorbable suture or staples have been seen at the base of a MU in up to 35% of cases [3, 22, 23]. It is posited that the inflammatory response to a foreign body results in mucosal breakdown and hence ulceration. Frezza et al. [23] showed that endoscopic removal of remnant suture material could be performed safely and

resulted in resolution of symptoms and a normal endoscopy 6 months later. Many have shown that the use of absorbable suture at the index surgery decreases the rate of MU [13, 24, 25]. Sacks et al. [24] retrospectively reviewed 3285 patients, approximately 1/3 of which underwent RYGB with nonabsorbable suture, and found that the rate of MU decreased from 2.6% to 1.3% with the switch to absorbable suture. The authors also noted reduced incidence of visible suture adjacent to the ulcer when absorbable suture was used (64.3% versus 3.4%). While Rasmussen et al. [26] did not find a difference in ulceration rates between absorbable and nonabsorbable suture material, they did note that when suture was visible in their MUs, it was more frequently nonabsorbable (44%) than absorbable (20%). Thus most data support the use of absorbable material if suturing is to be performed at the gastrojejunostomy.

Various methods of gastrojejunostomy creation have been implemented; thus the question of whether anastomotic technique contributes to MU development has been posed. Some have advocated that a hand-sewn anastomosis results in less ischemia of the tissue and therefore may decrease MU formation; however this has not been clearly demonstrated. Single technique studies have exhibited similar MU rates among the different techniques: 1.3–7% with linear stapled anastomosis, [24, 26–28] 2.3–3% with circular stapled anastomosis [25, 29], and 1.2–1.3% with hand-sewn anastomosis [30, 31]. In a series of 882 consecutive patients with a 2-month follow-up, Bendewald et al. [32] compared the three techniques and found no difference in MU formation. These findings were mirrored in a recent meta-analysis by Jiang et al. [33], which confirmed that there is no significant difference in MU rate when hand-sewn, linear stapled, or circular stapled gastrojejunal anastomosis are performed.

The tension placed on the gastrojejunal anastomosis by an antecolic versus retrocolic Roux limb has also been evaluated as a possible risk factor for MU. Lublin et al. [34] noted zero MU perforations when a retrocolic gastrojejunostomy was performed in their first 403 patients and eight perforations in the successive 499 patients who underwent antecolic anastomosis – a difference that proved statistically significant. In a subsequent study by Felix et al. [35], no significant difference in rate of MU perforation was identified when 405 patients with a retrocolic anastomosis were compared to 3025 patients with an antecolic anastomosis. Though the data regarding Roux limb positioning is inconclusive, the antecolic approach has significant benefits over the retrocolic approach that are unrelated to MU, making a change in this aspect of surgical technique inadvisable.

While factors that may cause anastomotic inflammation, ischemia, or tension, such as those described above related to surgical technique, are felt to be the culprit in early MU, late MU may be secondary to chronic comorbid disease related to vasculopathy or other forms of mucosal damage [36]. Smoking was found to be a risk factor for MU across many studies [3, 15, 35, 37, 38]. One of the potential ulcerogenic mechanisms of smoking is the influence of nicotine on mucosal blood flow [39]. Nicotine administration has been demonstrated to markedly reduce gastric mucosal blood flow in animal models by creating a local vasoconstrictive environment [40, 41]. As such, El-Hayek et al. [2] found current or previous smoking to be the only significant risk factor for MU and recurrent MU in 112 of 328

symptomatic patients. Azagury et al. [3] also showed a significant association between smoking and MU formation in a study of 103 patients with MU. Wilson et al. [37] retrospectively studied 226 patients who underwent endoscopy for upper gastrointestinal symptoms, 81 of whom had MUs, and found that smoking and NSAID use independently predicted MU presence.

Though the relationship between MUs and gastritis has not been directly analyzed, the primary causes of gastritis – i.e., use of nonsteroidal anti-inflammatory drugs (NSAIDs) and *Helicobacter pylori* (HP) infection – have been extensively examined. Use of NSAIDs may cause mucosal damage due to inhibition of cyclooxygenase, causing decreased prostaglandin E2 levels and subsequent disruption of the gastric barrier [43]. NSAIDs are a well-known cause of peptic ulcer disease, and multiple studies have cited the use of NSAIDs as a risk factor leading to MU and perforation [35, 37, 44, 45]. Coblijn et al. [4] evaluated 19 articles in a systematic review that scored the use of NSAIDs in patients with MU and found a significant relationship. Of the 365 patients with MU, 98 used NSAIDs at the time of presentation.

The association between *Helicobacter pylori* (HP) and MU has been a topic of significant debate. Theories range from increased risk of MU with history of HP even after documented eradication, to increased risk in individuals not screened for HP, to no relationship at all. For example, Rasmussen et al. [26] found that despite preoperative treatment, HP infection was more than twice as common in patients with MUs as compared to those without (32% versus 12%), suggesting that preoperative HP infection predisposes patients to MU. Notably, active infection was not detected in any of the patients who developed MU postoperatively. It is possible that the slow regression of chronic gastritis and intestinal metaplasia after eradication of HP, which can take more than a year to resolve, could explain the occurrence of MU in the post-HP treatment period. In support of this theory, D'Hondt et al. [5] found that patients who were treated for HP preoperatively and continued to receive prophylactic proton pump inhibitor (PPI) therapy postoperatively had a significantly lower incidence of MU (0% versus 15.6%), a finding that was not present in patients who tested negative for HP preoperatively.

Even within the studies that suggest reduced MU rates with preoperative screening and eradication of HP, the relationship between HP and MU is not quite clear [22, 46, 47]. Hartin et al. [48], for example, found that in an area of high HP prevalence, 5% (6/125) of non-screened patients presented with perforated viscus compared to 0/58 of those tested and eradicated, suggesting the importance of screening. However, only two of the six untreated patients with perforation actually tested positive for HP, which brings into question the role HP plays in MU formation. A systematic review of MU after RYGB came to a similar conclusion, noting that while 22–67% of patients screened for bariatric surgery tested positive for HP, only 10.5% of patients developing MUs after surgery tested positive [4].

Ultimately, the majority of the literature indicates that there is no definitive association between HP and MU formation [35, 44, 49, 50]. Papasavas et al. [51] studied 16 MU patients in a series of 442 patients who underwent RYGB and determined that HP was not a risk factor for MU. Kang et al. [6] found no difference in the

incidence of MU in patients diagnosed with and treated for HP preoperatively compared to those without. Kelly et al. [52] identified 66 untreated HP-positive patients at the time of RYGB and found that only 5 (7.6%) went on to develop MUs, whereas 17.2% (108/628) of the HP-negative patients developed MUs. The authors went so far as to suggest that HP could be protective, hypothesizing that HP-promoted atrophic gastritis within the pouch may protect against MU. They also pointed out that the vast majority of MU complications are located on the jejunal side of the anastomosis, where it is extremely rare to find HP organisms as they preferentially congregate along mucosa of the stomach (primarily antrum and body). Thus, there is no definitive evidence that HP increases the rate of MU, and preoperative screening remains a subject of controversy.

Other factors not consistently shown to be associated with development of MUs include age, sex, body mass index, history of hypertension, and history of gastroesophageal reflux disease (GERD) [2, 4]. The use of daily low-dose aspirin following RYGB has also been studied; however, it does not appear to be a risk factor for MU formation [42]. Finally, despite the well-established detrimental effect of diabetes mellitus on peripheral vasculature, the data in support of its role in MU formation is weak and inconclusive. Overall the only well-established risk factors for MU formation are smoking and NSAID use, while most technical aspects of RYGB, presence of HP infection, and the above-described patient factors remain controversial.

Presentation

It is not surprising that the most frequent symptom encountered by individuals suffering from MU is abdominal pain (59% of cases). Other common presentations include nausea and vomiting (15%), anemia (8%), hematemesis (8%), and dysphagia (8%) [53]. Azagury et al. [3] reported that 63% of patients presented with pain and 24% with bleeding. These findings were echoed in a recent systematic review which noted 56.8% of patients experience epigastric burn and 15.1% present with bleeding [4]. Patients with perforated MU, on the other hand, will present with signs of acute abdomen at the emergency room. The positive predictive value of any individual symptom, however, is low (40%) and a poor predictor of endoscopic pathology [54, 55]. Thus evaluation of clinical symptoms alone is not sufficient to make a definitive diagnosis.

Diagnosis

Endoscopy remains the gold standard for diagnosis of MUs and even allows for therapeutic intervention at the time of diagnosis if indicated [56]. The benefits of endoscopy include the ability to evaluate size and depth of ulcer penetration and to identify foreign bodies or GGFs (though the latter can sometimes be missed). Carr et al. [57] evaluated 47 articles to create an evidence-based algorithm for the

management of MUs and based on their findings recommended early investigation of upper gastrointestinal symptoms with endoscopy. They also suggested that breath test or serology for HP be pursued, especially in situations in which a MU is not identified at endoscopy.

Prevention

Because MU continues to be a cause of significant morbidity after RYGB, many have advocated for the use of PPI prophylactically in the postoperative period. Recently a meta-analysis was published by Ying et al. [58] describing the prophylactic effect of PPIs in reduction of MU after RYGB. They found that patients receiving prophylactic PPI treatment experienced half the occurrence of ulceration compared with the non-PPI group. Coblijn et al. [59] subsequently published a historic cohort study comparing 6 months of PPI postoperatively to none and uncovered similar results (1.2% versus 7.3% occurrence of MU with PPI and without, respectively). Kang et al. [6] examined the impact of employing a 30-day versus 90-day postoperative PPI regimen on the development of MU and found a decreased incidence with the longer regimen (12.4% vs. 6.5%, respectively). Alternatively, Felix et al. [35] suggested that because only 7 (0.2%) of 3430 patients, or 1/5 of the recorded perforations, presented without warning, long-term ulcer prophylaxis should only be considered for high-risk individuals. Based on all of the above findings, it has been suggested that low-risk patients receive 6–12 months of empirical PPI therapy postoperatively to cover the period of highest incidence of MU and that only patients with multiple risk factors for MU be considered for longer postoperative therapy [57].

Treatment

The treatment of a MU depends somewhat on its etiology and presentation. As discussed above, any visible sutures or staples at the site of MU should be removed endoscopically if possible. Patient-related risk factors, such as smoking and NSAID use, must be identified and corrected. If HP is found to be present on MU workup, it should be treated with triple therapy as is standard of care. Although treatment of all-comer MUs with PPI is accepted internationally and advised in a variety of different guidelines, there is little consensus on ideal dosing or formulation [60]. Gumbs et al. [28], for example, treated 16 MUs from a cohort of 347 RYGB patients and accomplished 100% resolution with 8 weeks of PPI therapy. This is the standard advocated in the treatment algorithm by Carr et al. [57]. Schulman et al. [61] recommend administering open capsule PPI for the treatment of MU as they believe this technique allows for better medication absorption in the altered post-RYGB anatomy. They found a median 342 versus 91 days to healing with intact versus open capsules, respectively. The addition of a cytoprotective barrier medication if the patient is already on a PPI may also be considered according to Carr et al. [57],

though there is little data to support this recommendation. For example, when compared to PPI monotherapy, the combination of PPI and cytoprotective barrier medication does not significantly change healing rates (67% versus 68%, respectively) [3].

Repeat endoscopy should be performed after 8 weeks of therapy to assess for healing, and lifelong PPI therapy should be considered in those experiencing success with medical management. For those not responding to 8 weeks of therapy, most advocate for continued PPI treatment with serial endoscopic evaluation, even up to 2 years out from initial diagnosis [60]. While there are no official guidelines for appropriate length of treatment to attempt before considering surgical intervention, we may begin to see shorter treatment periods or alternative use of H2 blockers given the recent findings on long-term adverse effects of PPI use [62]. PPI use has been tied to increased infection rate, electrolyte disturbances, vitamin deficiencies, metabolic bone disease, kidney disease, and even increased risk of death. While current guidelines do not support any additional screening in patients on PPIs, the growing body of data showing detrimental long-term consequences has led to recommendations for PPI use of the shortest duration possible [62, 63].

The alternative to medical management of MU is revisional surgery, and this route is sometimes unavoidable. In a systematic review article, Coblijn et al. [4] reported a reoperation rate of approximately 23% of all MU patients for complete healing. El-Hayek et al. [2] found that despite optimal medical therapy, 12 of 328 patients went on to require revisional surgery, with 7 out of 12 exhibiting underlying anatomical abnormalities that necessitated operative intervention [2]. This highlights an important consideration in the treatment of MU: persistent ulcer after trial of medical management may indicate a more complicated etiology including a GGF or malignancy [64]. Indeed it has been shown that up to 72% of patients with persistent MUs requiring surgical intervention have GGFs [15].

Elective revision is generally reserved for those with GGF, MUs not responsive to maximum medical therapy, chronic anemia, or significant UGI hemorrhage [56]. The surgery essentially involves excision of all affected tissue of the gastrojejunostomy and creation of a new gastrojejunal anastomosis [56]. Much of the literature describing reoperation for MU discusses surgical management of a GGF, as this was a common cause of MU in the age of nondivided staple lines. Patel et al. [15] performed 39 open revisions in MU patients – 4 in patients with prior laparoscopic surgery and 35 in those with prior open surgery – and reported an 87% success rate (34 of 39 patients remained asymptomatic after revision). However, they noted some significant complications including one mortality, four early postoperative complications (two leaks, two wound infections), and three recurrences (all of which occurred in smokers). Chau et al. [36] described a similar reoperation, though performed two thirds of their procedures laparoscopically. They had a 100% success rate, with 0 MU recurrences over a median 35-month follow-up period, but also noted a high incidence of chronic symptoms and postoperative complications. Both studies emphasized an important point about revisional surgery: while it is highly effective, it can be associated with serious complications. Hence a thorough

examination of the risks and benefits must be undertaken prior to pursuing surgical revision for MUs, regardless of planned technique.

Other surgical techniques, such as truncal vagotomy, have been explored for treatment of MU but results have varied [65]. Patel et al. [15] performed truncal vagotomy in addition to ulcer excision and gastrojejunostomy revision on a few patients with refractory MU but quickly abandoned the vagotomy component due to difficulty with the dissection without evidence of benefit in terms of decreased MU recurrence. Chau et al. [36] described one patient with persistent UGI bleeding despite endoscopic interventions who was effectively treated with video-assisted thoracoscopic surgery truncal vagotomy. This procedure was selected over intra-abdominal approaches in order to avoid a lengthy revisional surgery in a patient with multiple comorbidities and polypharmacy. For those with recurrent or refractory MU after initial revision, some surgeons have performed a second revision of the gastrojejunal anastomosis; others have described total gastrectomy with esophagojejunostomy [36, 66, 67]. Unfortunately there are not any real guidelines or good data to outline the treatment of complicated MU, and therefore referral to a specialist is advisable.

Perforated Marginal Ulcer

The incidence of perforated MU after RYGB is around 1–2% in the total population, which means that around 20% of patients with MU present with perforation [4]. Like most MUs, those that perforate usually present between 3 and 24 months postoperatively. They often occur in patients with known MUs; however, up to 20% have been shown to present with no warning signs [35]. Risk factors for perforation are similar to those for MU and include NSAIDs, smoking, and steroids [35, 45]. Sasse et al. [38] found that six out of seven patients from a series of 1690 RYGB procedures were taking NSAIDs prior to perforation. Smoking has been repeatedly identified as a significant risk factor, with 50–60% of those presenting with perforated MUs reported to be smokers [35, 44, 45, 68].

The presentation of perforated MU is generally more acute than that of non-perforated MU. Patients develop acute onset abdominal pain with peritonitis, tachycardia, tachypnea, and fever [4, 64]. If suspicions are high, a simple upright chest radiograph revealing air under diaphragm confirms the diagnosis. A computed tomography scan may also be performed and could show free air, fluid collections adjacent to the gastrojejunostomy, ascites, or contrast extravasation, for example. Treatment of perforated MU, similar to any perforated viscus, begins with resuscitation and administration of broad-spectrum antibiotics and is almost always followed soon after by surgical intervention.

The operative approach for perforated MU may be laparoscopic or open, and generally involves irrigation and omental patch of the defect, with wide drainage of the upper abdomen [45]. If there is difficulty identifying the location of the perforation, intraoperative endoscopy may be employed to facilitate localization. Laparoscopic patch repair of perforated MU has been reported to be safe and

effective, though is more likely to be the treatment of choice if patients present to a bariatric surgeon [34, 44, 45, 68, 69]. Binenbaum et al. [69] suggest that a laparoscopic approach is safe within 24–48 h of the onset of symptoms, noting that the hemodynamic stability of the patient and their ability to tolerate abdominal insufflation must be considered. Moon et al. [52] also found laparoscopic oversewing with omental patch repair to be safe and effective, but additionally cited the need for resection and revision of the gastrojejunal anastomosis when the perforation was too large. Whether laparoscopic or open repair is pursued, morbidity and mortality rates of 30% and 10%, respectively, have been reported [44].

Bleeding Marginal Ulcer

Another unique presentation of MU is significant UGI bleeding. Five percent of MUs present with UGI bleeding or chronic blood loss anemia [2]. Acute massive hemorrhage is uncommon (1.1–4%) but can occur [56]. Bleeding MUs are most often located at anastomosis (64%) [70]. The major risk factor for developing a bleeding MU is the use of antiplatelet or anticoagulation medications in the setting of a known MU [64]. Presentation is the same as any surgical patient with UGI bleeding, and management consists of airway protection (when necessary), large bore intravenous access, resuscitation, active type and screen, and transfusions as needed. NGT placement is generally avoided unless absolutely required. Immediate upper endoscopy should be performed once the patient is stable and often proves to be therapeutic with no further intervention needed thereafter. Hemoclipping or dual therapy with epinephrine and heater probe can be effective in controlling bleeding, with 22% rebleeding rate and only 4% of patients requiring surgery [64]. Angiographic embolization has also been described, as has revision of gastrojejunostomy in rare circumstances [66].

Summary

Marginal ulcers (MU) are a significant cause of morbidity in patients who have undergone gastric bypass procedures. Incidence has been reported between 0.6% and 25%, though may in fact be higher. MUs may occur at any point in time after RYGB, though are frequently seen in the first year after surgery. The etiology of MU development has been widely discussed and remains controversial. It is likely multifactorial and may differ for each individual. Well-established risk factors include smoking and NSAID use. There also appears to be a decreased incidence of MU with the move toward divided staple lines and the use of absorbable suture for gastrojejunal anastomoses. The most frequent symptom encountered by individuals suffering from MU is abdominal pain (59% of cases), with other common presentations including nausea and vomiting (15%), anemia (8%), hematemesis (8%), and dysphagia (8%). Endoscopy remains the gold standard for diagnosis of

MUs and even allows for therapeutic intervention at the time of diagnosis if indicated.

Given the relatively high incidence of MU, current practice is to prophylactically administer 6–12 months of empirical PPI therapy postoperatively to low-risk patients. Patients with multiple risk factors for MU may be considered for longer postoperative therapy. When MU is diagnosed, medical management consists of 8 weeks of high-dose PPI therapy, plus or minus the addition of a cytoprotective barrier medication. Repeat endoscopy should be performed after 8 weeks of therapy to assess for healing, and lifelong PPI therapy should be considered in those experiencing success with medical management. For those not responding to 8 weeks of therapy, most advocate for continued PPI treatment with serial endoscopic evaluation, even up to 2 years out from initial diagnosis. However, we may begin to see shorter treatment periods or alternative use of H2 blockers given the recent findings on long-term adverse effects of PPI use.

In addition to the above-described medical management, any reversible causes of MU should be identified and corrected. This includes smoking cessation and discontinuation of NSAIDs. If HP is found to be present on MU workup, it should be treated with triple therapy as is standard of care. Visible sutures or staples at the site of MU should be removed endoscopically if possible. Persistent ulcer after trial of medical management may indicate a more complicated etiology including a GGF or malignancy, and further workup is warranted. There are no official guidelines for appropriate length of medical treatment to attempt before considering revisional surgery, but given the significant morbidity associated with such procedures, a careful assessment of risks and benefits should be undertaken. Various techniques for RYGB revision in the setting of MU have been described, but the most widely accepted surgery essentially involves excision of all affected tissue of the gastrojejunostomy and creation of a new gastrojejunal anastomosis. In more complicated MU cases or recurrence after revision, referral to a specialist is advised.

Perforated and bleeding MUs present a slightly different dilemma and thus must be considered separately. The incidence of perforated MU after RYGB is around 1–2%, and they often occur in patients with known MUs; however, up to 20% have been shown to present with no warning signs. Risk factors for perforation are similar to those for MU and include NSAIDs, smoking, and steroids. The presentation of perforated MU is generally more acute than that of non-perforated MU. Patients develop acute onset abdominal pain with peritonitis, tachycardia, tachypnea, and fever. If suspicions are high, a simple upright chest radiograph revealing air under diaphragm confirms the diagnosis. A computed tomography scan may also be performed. Treatment of perforated MU, similar to any perforated viscus, begins with resuscitation and administration of broad-spectrum antibiotics and is almost always followed soon after by surgical intervention. The operative approach may be laparoscopic or open, and generally involves irrigation and omental patch of the defect, with wide drainage of the upper abdomen.

Bleeding MUs more often present with mild UGI bleeding or chronic blood loss anemia, though acute massive hemorrhage can occur (1.1–4% of cases of MU). The major risk factor for developing a bleeding MU is the use of antiplatelet or

anticoagulation medications in the setting of a known MU. Presentation is the same as any surgical patient with UGI bleeding, and management consists of airway protection (when necessary), large bore intravenous access, resuscitation, active type and screen, and transfusions as needed. NGT placement is generally avoided unless absolutely required. Immediate upper endoscopy should be performed once the patient is stable and often proves to be therapeutic with no further intervention needed thereafter. Angiographic embolization has also been described, as has revision of gastrojejunostomy in rare circumstances.

References

1. Mason EE, Ito C. Gastric bypass in obesity. Surg Clin North Am. 1967;47:1345–51.
2. El-Hayek K, Timratana P, Shimizu H, Chand B. Marginal ulcer after Roux-en-Y gastric bypass: what have we really learned? Surg Endosc. 2012;26(10):2789–96.
3. Azagury DE, Abu Dayyeh BK, Greenwalt IT, Thompson CC. Marginal ulceration after Roux-en-Y gastric bypass surgery: characteristics, risk factors, treatment, and outcomes. Endoscopy. 2011;43(11):950–4.
4. Coblijn UK, Goucham AB, Lagarde SM, Kuiken SD, van Wagensveld BA. Development of ulcer disease after Roux-en-Y gastric bypass, incidence, risk factors, and patient presentation: a systematic review. Obes Surg. 2014;24(2):299–309.
5. D'Hondt MA, Pottel H, Devriendt D, Van Rooy F, Vansteenkiste F. Can a short course of prophylactic low-dose proton pump inhibitor therapy prevent stomal ulceration after laparoscopic Roux-en-Y gastric bypass? Obes Surg. 2010;20(5):595–9.
6. Kang X, Zurita-Macias L, Hong D, Cadeddu M, Anvari M, Gmora S. A comparison of 30-day versus 90-day proton pump inhibitor therapy in prevention of marginal ulcers after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2016;12(5):1003–7.
7. Csendes A, Torres J, Burgos AM. Late marginal ulcers after gastric bypass for morbid obesity. Clinical and endoscopic findings and response to treatment. Obes Surg. 2011;21(9):1319–22.
8. Csendes A, Burgos AM, Altuve J, Bonacic S. Incidence of marginal ulcer 1 month and 1 to 2 years after gastric bypass: a prospective consecutive endoscopic evaluation of 442 patients with morbid obesity. Obes Surg. 2009;19(2):135–8.
9. Schirmer BD, Meyers WC, Hanks JB, Kortz WJ, Jones RS, Postlethwait RW. Marginal ulcer. A difficult surgical problem. Ann Surg. 1982;195:653–61.
10. Mason EE, Munns JR, Kealey GP, Wangler R, Clarke WR, Cheng HF, et al. Effect of gastric bypass on gastric secretion. Am J Surg. 1976;131:162–8.
11. MacLean LD, Rhode BM, Nohr C, Katz S, McLean APH. Stomal ulcer after gastric bypass. J Am Coll Surg. 1997;185:1–6.
12. Hedberg J, Hedenström H, Nilsson S, Sundbom M, Gustavsson S. Role of gastric acid in stomal ulcer after gastric bypass. Obes Surg. 2005;15(10):1375–8.
13. Capella JF, Capella RF. Gastro-gastric fistulas and marginal ulcers in gastric bypass procedures for weight reduction. Obes Surg. 1999;9:22–7.
14. Carrodeguas L, Szomstein S, Soto F. Management of gastro-gastric fistulas after divided Roux-en-Y gastric bypass surgery for morbid obesity: analysis of 1,292 consecutive patients and review of literature. Surg Obes Relat Dis. 2005;1:467–74.
15. Patel R, Brolin R, Gandhi A. Revisional operations for marginal ulcer after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5:317–22.
16. Capella JF, Capella RF. Staple disruption and marginal ulceration in gastric bypass procedures for weight reduction. Obes Surg. 1996;6:44–9.
17. Printen KJ, Scott DME. Stomal ulcers after gastric bypass. Arch Surg. 1980;115:525–7.

18. Jordan J, Hocking M. Marginal ulcer following gastric bypass for morbid obesity. Am Surg. 1991;57:286–8.
19. Sapala J, Wood MH, Sapala M, Flake TM Jr. Marginal ulcer after gastric bypass: a prospective 3-year study of 173 patients. Obes Surg. 1998;8(5):505–16.
20. Siilin H, Wanders A, Gustavsson S, Sundbom M. The proximal gastric pouch invariably contains acid-producing parietal cells in Roux-en-Y gastric bypass. Obes Surg. 2005;15:771–7.
21. Behrns KE, Smith CD, Sarr MG. Prospective evaluation of gastric acid secretion and cobalamin absorption following gastric bypass for morbid obesity. Dig Dis Sci. 1994;39:315–20.
22. Lee JK, Van Dam J, Morton JM, Curet M, Banerjee S. Endoscopy is accurate, safe, and effective in the assessment and management of complications following gastric bypass surgery. Am J Gastroenterol. 2009;104(3):575–82.
23. Frezza E, Herbert H, Ford R, Wachtel MS. Endoscopic suture removal at gastrojejunal anastomosis after Roux-en-Y gastric bypass to prevent marginal ulceration. Surg Obes Relat Dis. 2007;3:619–22.
24. Sacks BC, Mattar SG, Qureshi FG, Eid GM, Collins JL, Barinas-Mitchell EJ, et al. Incidence of marginal ulcers and the use of absorbable anastomotic sutures in laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2006;2(1):11–6.
25. Vasquez JC, Wayne Overby D, Farrell TM. Fewer gastrojejunostomy strictures and marginal ulcers with absorbable suture. Surg Endosc. 2009;23(9):2011–5.
26. Rasmussen JJ, Fuller W, Ali MR. Marginal ulceration after laparoscopic gastric bypass: an analysis of predisposing factors in 260 patients. Surg Endosc. 2007;21(7):1090–4.
27. Dallal RM, Bailey LA. Ulcer disease after gastric bypass surgery. Surg Obes Relat Dis. 2006;2(4):455–9.
28. Gumbs A, Duffy A, Bell R. Incidence and management of marginal ulceration after laparoscopic Roux-Y gastric bypass. Surg Obes Relat Dis. 2006;2:460–3.
29. Gould JC, Garren M, Boll V, Starling J. The impact of circular stapler diameter on the incidence of gastrojejunostomy stenosis and weight loss following laparoscopic Roux-en-Y gastric bypass. Surg Endosc. 2006;20(7):1017–20.
30. Ballesta-Lopez C, Poves I, Cabrera M, Almeida JA, Macias G. Learning curve for laparoscopic Roux-en-Y gastric bypass with totally hand-sewn anastomosis: analysis of first 600 consecutive patients. Surg Endosc. 2005;19:519–24.
31. Ruiz-de-Adana JC, López-Herrero J, Hernández-Matías A, Colao-Garcia L, Muros-Bayo JM, Bertomeu-Garcia A, et al. Laparoscopic hand-sewn gastrojejunal anastomoses. Obes Surg. 2008;18(9):1074–6.
32. Bendewald FP, Choi JN, Blythe LS, Selzer DJ, Ditslear JH, Mattar SG. Comparison of hand-sewn, linear-stapled, and circular-stapled gastrojejunostomy in laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2011;21(11):1671–5.
33. Jiang HP, Lin LL, Jiang X, Qiao HQ. Meta-analysis of hand-sewn versus mechanical gastrojejunal anastomosis during laparoscopic Roux-en-Y gastric bypass for morbid obesity. Int J Surg. 2016;32:150–7.
34. Lublin M, McCoy M, Waldrep J. Perforating marginal ulcers after laparoscopic gastric bypass. Surg Endosc. 2006;20:51–4.
35. Felix EL, Kettelle J, Mobley E, Swartz D. Perforated marginal ulcers after laparoscopic gastric bypass. Surg Endosc. 2008;22(10):2128–32.
36. Chau E, Youn H, Ren-Fielding CJ, Fielding GA, Schwack BF, Kurian MS. Surgical management and outcomes of patients with marginal ulcer after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2015;11(5):1071–5.
37. Wilson J, Romagnuolo J, Byrne TK, Morgan K, Wilson F. Predictors of endoscopic findings after Roux-en-Y gastric bypass. Am J Gastroenterol. 2006;101(10):2194–9.
38. Sasse K, Ganser J, Kozar M, Watson RW, McGinley L, Lim D, et al. Seven cases of gastric perforation in Roux-en-Y gastric bypass patients: what lessons can we learn? Obes Surg. 2008;18:530–4.
39. Wu WK, Cho CH. The pharmacological actions of nicotine on the gastrointestinal tract. J Pharmacol Sci. 2004;94:348–58.

40. Nagata M, Okuma Y, Osumi Y. Effects of intracerebroventricularly applied nicotine on enhanced gastric acid secretion and mucosal blood flow in rats. Eur J Pharmacol. 1984;101:185–91.
41. Eastwood GL. Is smoking still important in the pathogenesis of pepticulcer disease? J Clin Gastroenterol. 1997;25(Suppl.1):S1–7.
42. Kang X, Hong D, Anvari M, Tiboni M, Amin N, Gmora S. Is daily low-dose aspirin safe to take following laparoscopic Roux-en-Y gastric bypass for obesity surgery? Obes Surg. 2017;27(5):1261–5.
43. Konturek SJ, Kwiecień N, Obtułowicz W, Oleksy J, Sito E, Kopp B. Prostaglandins in peptic ulcer disease: effect of nonsteroidal anti-inflammatory compounds (NOSAC). Scand J Gastroenterol Suppl. 1984;92:250–4.
44. Kalaiselvan R, Exarchos G, Hamza N, Ammori BJ. Incidence of perforated gastrojejunal anastomotic ulcers after laparoscopic gastric bypass for morbid obesity and role of laparoscopy in their management. Surg Obes Relat Dis. 2012;8:423–8.
45. Wendling MR, Linn JG, Keplinger KM, Mikami DJ, Perry KA, Melvin WS, et al. Omental patch repair effectively treats perforated marginal ulcer following Roux-en-Y gastric bypass. Surg Endosc. 2013;27(2):384–9.
46. Schirmer B, Erenoglu C, Miller A. Flexible endoscopy in the management of patients undergoing Roux-en-Y gastric bypass. Obes Surg. 2002;12(5):634–8.
47. Scheffel O, Daskalakis M, Weiner R. Two important criteria for reducing the risk of postoperative ulcers at the gastrojejunostomy site after gastric bypass: patient compliance and type of gastric bypass. Obes Facts. 2011;4(Suppl 1):39–41.
48. Hartin CW, ReMine DS, Lucktong TA. Preoperative bariatric screening and treatment of Helicobacter pylori. Surg Endosc. 2009;23(11):2531–4.
49. Yang CS, Lee WJ, Wang HH, Huang SP, Lin JT, Wu MS. The influence of Helicobacter pylori infection on the development of gastric ulcer in symptomatic patients after bariatric surgery. Obes Surg. 2006;16(6):735–9.
50. Loewen M, Giovanni J, Barba C. Screening endoscopy before bariatric surgery: a series of 448 patients. Surg Obes Relat Dis. 2008;4(6):713–4.
51. Papasavas P, Gagne D, Donnelly P. Prevalence of Helicobacter pylori infection and value of preoperative testing and treatment in patients undergoing laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2008;4:383–8.
52. Kelly JJ, Perugini RA, Wang QL, Czerniach DR, Flahive J, Cohen PA. The presence of Helicobacter pylori is not associated with long-term anastomotic complications in gastric bypass patients. Surg Endosc. 2015;29(10):2885–90.
53. Moon RC, Teixeira AF, Goldbach M, Jawad MA. Management and treatment outcomes of marginal ulcers after Roux-en-Y gastric bypass at a single high volume bariatric center. Surg Obes Relat Dis. 2014;10(2):229–34.
54. Garrido AB, Rossi M. Early marginal ulcer following Roux-en-Y gastric bypass under proton pump inhibitor treatment—prospective multicentric study. Arq Gastroenterol. 2010;47(2):130–4.
55. Huang CS, Forse RA, Jacobson BC, Farraye FA. Endoscopic findings and their clinical correlations in patients with symptoms after gastric bypass surgery. Gastrointest Endosc. 2003;58:859–66.
56. Nguyen NT, Hinojosa MW, Gray J, Fayad C. Reoperation for marginal ulceration. Surg Endosc. 2007;21(11):1919–21.
57. Carr WR, Mahawar KK, Balupuri S, Small PK. An evidence-based algorithm for the management of marginal ulcers following Roux-en-Y gastric bypass. Obes Surg. 2014;24(9):1520–7.
58. Ying WC, Kim SH, Khan KJ, Farrokhyar F, D'Souza J, Gmora S, et al. Prophylactic PPI help reduce marginal ulcers after gastric bypass surgery: a systematic review and meta-analysis of cohort studies. Surg Endosc. 2015;29(5):1018–23.
59. Coblijn UK, Lagarde SM, de Castro SM, Kuiken SD, van Tets WF, van Wagensveld BA. The influence of prophylactic proton pump inhibitor treatment on the development of symptomatic marginal ulceration in Roux-en-Y gastric bypass patients: a historic cohort study. Surg Obes Relat Dis. 2016;12(2):246–52.

60. Steinemann DC, Bueter M, Schiesser M, Amygdalos I, Clavien PA, Nocito A. Management of anastomotic ulcers after Roux-en-Y gastric bypass: results of an international survey. Obes Surg. 2014;24(5):741–6.
61. Schulman AR, Chan WW, Devery A, Ryan MB, Thompson CC. Opened proton pump inhibitor capsules reduce time to healing compared with intact capsules for marginal ulceration following Roux-en-Y gastric bypass. Clin Gastroenterol Hepatol. 2017;15(4):494–500.
62. Xie Y, Bowe B, Li T, Xian H, Yan Y, Al-Aly Z. Risk of death among users of proton pump inhibitors: a longitudinal observational cohort study of United States veterans. BMJ Open. 2017;7(6):e015735.
63. Metz DC. Long term use of proton-pump inhibitor therapy. Gastroenterol Hepatol. 2008;4(5):322–5.
64. Wernick B, Jansen M, Noria S, Stawicki SP, El Chaar M. Essential bariatric emergencies for the acute care surgeon. Eur J Trauma Emerg Surg. 2016;42(5):571–84.
65. Datta TS, Steele K, Schweitzer M. Laparoscopic revision of gastrojejunostomy revision with truncal vagotomy for persistent marginal ulcer after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2010;6(5):561–2.
66. Madan AK, DeArmond G, Ternovits CA, Beech DJ, Tichansky DS. Laparoscopic revision of the gastrojejunostomy for recurrent bleeding ulcers after past open revision gastric bypass. Obes Surg. 2006;16:1662–8.
67. Steinemann DC, Schiesser M, Clavien PA, Nocito A. Laparoscopic gastric pouch and remnant resection: a novel approach to refractory anastomotic ulcers after Roux-en-Y gastric bypass: case report. BMC Surg. 2011;11:33.
68. Wheeler AA, de la Torre RA, Fearing NM. Laparoscopic repair of perforated marginal ulcer following Roux-en-Y gastric bypass: a case series. J Laparoendosc Adv Surg Tech A. 2011;21:57–60.
69. Binenbaum SJ, Dressner RM, Borao FJ. Laparoscopic repair of a free perforation of a marginal ulcer after Roux-en-Y gastric bypass: a safe alternative to open exploration. JSLS. 2007;11:383–8.
70. Lee YC, Wang HP, Yang CS, Yang TH, Chen JH, Lin CC, et al. Endoscopic hemostasis of a bleeding marginal ulcer: hemoclipping or dual therapy with epinephrine injection and heater probe thermocoagulation. J Gastroenterol Hepatol. 2002;17(11):1220–5.

Staple Line Leak Following Laparoscopic Sleeve Gastrectomy

5

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Laparoscopic sleeve gastrectomy (LSG) was discovered by serendipity after the non-completion of the intestinal part of a laparoscopic duodenal switch in high-risk patients, super-super obese patients ((body mass index) BMI >60 kg/m^2), at Mount Sinai Hospital in New York in 2000 [100]. After observance that rapid weight loss was associated with this half of the procedure, it was proposed as a two-stage procedure for patients with higher BMI in order to decrease the risk of the complete procedure in these sicker patients [62, 99].

Eventually, the literature regarding LSG as a primary bariatric procedure has grown exponentially and has confirmed durable weight loss at 5–10 years with substantial improvement and/or resolution in obesity-related comorbidities [26, 35, 43, 48, 54, 55, 65, 70, 82, 95, 103, 118].

Techniques of Laparoscopic Sleeve Gastrectomy-Buttress Versus Suture Reinforcement

Trocar Placement: I use five trocars – a 12 mm trocar at the umbilicus (open technique to access the peritoneal cavity; this will be our extraction site), a 10 mm trocar (or 5 mm) in the midline between the epigastria region and the umbilicus for the optics, a 5 mm trocar in the right, another 5 mm trocar in the left, and a 12 mm trocar in the left midclavicular line.

The patient is placed in steep reverse Trendelenburg position, and the table is tilted right-side down to optimize visualization of the gastroesophageal junction. First, dissection begins along the distal greater curvature by dividing the branches of the gastroepiploic vessels near the gastric wall with ultrasonic shears. The second

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assistant retracts the omentum laterally with an atraumatic grasper through the 5 mm left lateral port. The greater curvature is devascularized in this manner proximally to the level of the left crus (including division of the short gastric vessels). All posterior attachments to the anterior pancreas must be freed, taking care not to injure the splenic artery, but it is important to leave the peritoneal attachments of the posterior lesser curvature vessels. This may prevent twisting and kinking (and has less chance of vessel injury or revascularization/ischemia of the sleeve).

The left crus and gastroesophageal junction must be completely exposed; helpful maneuvers may include placing the second assistant's grasper on the lateral fold of the mid-gastrosplenic ligament and retract this laterally toward the spleen. I sometimes increase the pneumoperitoneum to 20 mm Hg and ask the anesthesiologist to give an additional dose of paralytics. Place the patient in maximal reverse Trendelenburg position with an additional tilt of the patient more toward the right side. The counter-exposure maneuver consists of positioning the second assistant on the posterior fundus and retracting this toward the patient's right side. And finally, insert an additional 5 mm trocar to retract the perigastric fat and adequately expose the gastroesophageal junction.

I routinely clear the anterior perigastric fat just to the left of the GE junction to minimize tissue thickness during stapling. If there is laxity or dimpling of the phrenoesophageal ligament indicating potential hiatal hernia, the hiatus should be opened, the esophagus should be mobilized into the abdominal cavity, and the crural defect should be repaired with permanent sutures. Failure to recognize and repair a hiatal hernia at the time of initial operation may lead to weight loss failure and reflux after LSG [40].

Next the remainder of the greater curvature is liberated distally to 2 cm beyond the pylorus. The remainder of the gastrocolic ligament between the antrum and gastroepiploic arcade is divided with the ultrasonic shears [83].

Instrument palpation is used to confirm the anatomic position of the pylorus. There is significant debate regarding optimal distance from the pylorus to initiate the sleeve gastrectomy. We prefer to initiate the sleeve at least 4 cm proximal to the pylorus (at the level of the "crow's foot") to preserve the distal antrum, as there is some evidence that the closer the sleeve is initiated toward the pylorus, the higher the leak rate at this first centimeter of stapled line. Usually, the first two firings of the stapler are via the umbilical trocar. I routinely use black cartridges with closed height of 2.3 mm and buttressed with bioabsorbable Seamguard (Gore, Flagstaff AZ) for all firings. For mid-body and fundus, it may be downsized to green cartridges or gold for thinner fundic tissue. The buttressing material is sandwiched between, over, and below the anterior and posterior gastric wall and reduces staple line hemorrhage and the leakage rate.

The anesthesiologist inserts a bougie of 40 Fr, and I align the bougie along the lesser curvature. The absence of bougie results in weight regain [129]. The remainder of the sleeve gastrectomy is completed by sequential firings of the linear stapler along the bougie toward the angle of His. The differences in hemostasis are remarkable with the routine use of the buttressing Seamguard material. A total of five to six staple firings are typically required to complete the sleeve. The anesthesiologist

must pay careful attention that the bougie does not retract during stapling to prevent the tip of the bougie from being incorporated into the staple line.

Next, the anesthesiologist removes the bougie. I routinely place figure-of-eight 3-0 monofilament absorbable sutures at the apex of the sleeve gastrectomy with a fat pad (the area most prone to developing leak) and at the most distal end of the staple line (thickest part of stomach).

I routinely perform methylene blue test, not necessarily to assess the integrity of the staple line, but to have an estimate of its volume and to assess strictures, kinking, and twisting; further the expanded sleeve assesses bleeding potential at the staple line, not tested in resting state. The anesthesiologist inserts an 18 Fr orogastric tube. The surgeon clamps near the pylorus, and the anesthesiologist instills methylene blue mixed with saline through the tube. Approximately 60 ml is required to distend the sleeve. Another option is to insert a gastroscope and check for leak (and intraluminal bleeding) via air insufflation; this latter option is used less often because of the tendency of air to pass through the pylorus and distend the small bowel. The umbilical site is enlarged, the abdominal wall is dilated with an atraumatic clamp, and the specimen is extracted. Grasping the end of the sleeve and pulling it out progressively may make extraction easier and require less abdominal wall dilation. When absorbable buttressing is not available, oversewing with an absorbable suture (2-0 or 3-0) is ideal, and there has been no consensus on invagination versus simple baseball stitching (which I prefer) [80, 81]. Invagination is potentially more ischemic, but more hemostatic; it also serves some surgeons in tightening a loose sleeve or inversely increases the potential for strictures [125]. There is consensus that one has to have a bougie in place when doing so and that buttering and oversewing increase the risk of leaks.

Concerning Factors Associated with Staple Line Leaks

Leak after LSG used to occur in 1–2% of cases, and more recently this percentage has dramatically decreased to below 0,5%, the vast majority of leakage occurring near the GE junction (90%) and on the first centimeter of the staple line on the antrum (10%) [59]. They are expensive to treat, and hence any maneuvers to decrease their incidence make cost-effectiveness sense [78, 109]. For example, I tried wrapping the sleeve, but the material caused erosions [105]. Contributing factors like narrowing the sleeve at the incisura angularis may contribute to leaks, but the true pathophysiology remains unknown [21]. Possible factors include patient factors (BMI, gender, smoking), technical factors (bougie size, stapler height, distance from pylorus, use of buttressing, and/or suturing), and inadequate tissue oxygenation with subsequent ischemia [11, 12, 57, 127, 131].

There may be up to two- to threefold increased risk of leakage in patients with previous bariatric surgery, such as adjustable gastric banding or vertical banded gastroplasty [5, 13, 14, 20, 46, 84, 91, 128]. However some retrospective series of band to sleeve conversions have found comparable leak rate in one-stage vs. two-stage conversion [87]. In a recent retrospective review, of 103 leaks in 5400 LSG

cases, body mass index (BMI), male gender, sleep apnea, conversion to laparotomy, longer operative time, and intraoperative complications significantly increased leak rate [24].

Superobese patients (BMI >50 kg/m^2) may have a higher incidence of leak, like it has been shown in gastric bypass. A systematic review of 4888 LSG found the leak rate to be 2.9% among the superobese versus 2.2% in those with a preoperative BMI <50 kg/m^2, but this was not statistically significant. For these patients in particular, the 2-week liquid protein diet preoperatively may be worthwhile. The stomach has different tissue thickness throughout with the male antrum as thick as 5.5 mm (when both the anterior and posterior stomach is compressed) to the fundus being the thinner (1.7 mm). I have called for a thickness calibration device that can determine the appropriate staple height during construction of the LSG.

H. pylori may affect the thickness of the stomach, most studies to date evaluating the effect of *H. pylori* on LSG complications have shown no effect of *H. pylori* infection on LSG complication rate, but I continue to detect these bacteria preoperatively and treat them aggressively [18, 92].

Experience is playing a role [1]. Patrick Noel from France has recently published his experience to demonstrate that surgeon's experience may play an important role in decreasing the leak rate [52], especially after a prolonged learning curve of over 1000 cases, a higher number of leaks were observed within the first years of practice, decreasing over time [85]. In his first 7 years of practice (1000 cases), 18 leaks were encountered (1.8%), which decreased to two leaks or 0.2% in the subsequent 1000 cases with the use of buttress materials routinely. In general, the use of larger bougies decreases leaks, perhaps by avoidance of strictures or avoidance of a bigger mismatch in staple height to tissue ratio; bougie ≥40 Fr had a leak rate of 0.6% vs. 2.8% ($p < 0.05$) [90].

Although most studies have not found an association between leaks and distance from the pylorus, it has been my personal experience that the closer to the pylorus, the more difficult it is to staple with the current height we have, needing additional suturing in this staple line. This may be a higher risk for leakage, as <10% of leaks do occur in the most distal staple line.

Concerning staple height, there is quite some confusion as previous studies reported a combination of staple cartridges – green (4.8 mm) at the antrum and blue (3.5 mm) applied at the gastric corpus and fundus – and both companies changed their staplers, and now we see more black cartridges on the antrum and the new stapler from Medtronic having three different rows with various staple heights. Most surgeons have now replaced the green with the black (2.3 closed height) cartridge, with or without buttressing material when they initiate the sleeve on the antrum. I prefer to buttress the staple line, as it readily decreases bleeding (I was the first one to prove this in a randomized study), and it also decreases leaks, although this is still a controversial point in the literature, mainly because a randomized study is almost not feasible [28–30]. Since most surgeons believe that buttressing

decreases bleeding along the staple line, they should, by extension, agree that it may decrease leaks because a hematoma on the staple line is a known risk factor for leaks in itself [7, 33, 34, 36, 132]. The German registry data found that the risk of leak was decreased from 2.0% to 1.1% by using sutures and to 1.3% by using buttressing material, and there was no significant difference between oversewing and buttressing, perhaps because the numbers were too small for this comparison [119]. Also, using both sutures and buttressing caused higher leakage, presumably from ischemia (3.6%); hence utilizing both should be avoided. In a systematic review comparing the effect of no reinforcement, oversewing only, nonabsorbable bovine pericardial strips (BPS), and absorbable polymer membrane (APM) on LSG leak rate.

I found leak rates ranging from 1.09% (APM) to 3.3% (BPS); the APM leak rate was significantly lower than the other groups ($p < 0.05$) [47, 49]. Shikora et al. conducted a similar review of the effect of no reinforcement, oversewing, biocompatible glycolide copolymer (absorbable), and bovine pericardium after sleeve and gastric bypass. They also looked at leak and bleeding. They found that reinforcing with bovine pericardium had the lowest leak (1.28%) and bleed (1.23%) rates; buttressing with absorbable material had the second highest leak (2.61%) and bleed (2.48%) but significantly lower bleed rates than no reinforcement [113]. It should be noted that this meta-analysis included gastric bypass data in the results, and since gastric bypass was known to have lesser leak at this time interval, this skewed the results to give bovine pericardium a lower leak rates, not a fair comparison [50, 112]. The vascular supply, which arises from the left and right gastric arteries and left and right gastroepiploic arteries, may greatly vary. Too much posterior dissection may divide the terminal branch of the left gastric artery and may devascularize the upper portion of the sleeve and contribute to leak, especially if a triangular piece with a small distance from the GE junction is left in place, something that I have advocated to avoid and staple closer to the GE junction [94]. I also recommend leaving in place the peritoneal folds and attachments to the pancreas, which avoids twists and kinks and also provides a scaffold. In a retrospective review of 529 cases with 0% leak rate, Bellanger et al. discussed the technical principles for decreasing leak after LSG; a key point mentioned is to position the tip of the stapler to give a distance of one and a half times the width of the bougie at the area of the incisura angularis [10]. Other technical principles included allowing adequate compression of the gastric tissue with the stapling device and thorough visual inspection of the staple line after procedure completion. Sakran et al. proposed that heat-producing instruments might cause thermal injury to the sleeve, leading to leak [102]. The German Registry data found that conversion to laparotomy, intraoperative bleeding and/or hypotension, and prolonged operative time have been shown to impact leak rate [119].

Bleeding intraoperatively or postoperatively increases the chances of an ICU stay and also poor weight loss outcomes [44].

Surgical Management of Acute Staple Line Disruption

Even if multiple techniques are described for sleeve gastrectomy, most reports pertaining to leaks are implicating these factors: narrowing at the incisura angularis and stapling near the angle of His [77]. In my technique it is best to staple the stomach wider at the incisura, on purpose, and for the upper part near the gastroesophageal junction, I clear the fat pad and identify well the U junction between the esophagus and gastric fundus and staple just left of it. Also, use of the appropriate staple height is probably the most important and avoids burning the tissue that is left [15, 39, 56].

A Florida sleeve consensus has established the following classification: early gastric leak is defined as one which occurred before postoperative day 7 [101]. This is a leak with high output, requiring immediate surgical treatment followed by endoscopic approach in some cases.

Late gastric leak is defined as the leak which occurs later than 1 week after the initial procedure, often with a less severe clinical picture due to a smaller output. Symptoms are fewer, sometimes with episodes of back or shoulder pain and fever. For hemodynamically stable patients, percutaneous abscess drainage followed with an endoscopic intervention, added nutrition, antibiotics, and acid suppression is recommended.

Chronic gastric leak is considered after 6 weeks of diagnosis. When all endoscopic approaches have failed to close the fistula, beyond 12 weeks, a definitive reconstructive surgical treatment is proposed [101].

Others have proposed another classification with algorithm [2, 76]. Surgical management of early leaks consists mostly of a diagnostic laparoscopy, closed suction drainage, and feeding jejunostomy [32]. I have been an advocate of early stenting, even doing it on the operating table right after those laparoscopic maneuvers [3]. The chance to do a direct suture repair of the leak site depends on how many days after leakage we are. If it is 24–48 h, an attempt can be made to suture and patch with omental or perigastric fat (left fat pad often present near the GE junction if it hasn't been removed at the initial surgery). Adhesive glue has been tried in cases where the tissues are extremely fragile or even to patch absorbable membranes. It is quite difficult in some cases to create an adequate pneumoperitoneum and one as to be prepared for a laparotomy. After this period, an internal or external drainage of the fistula is recommended [8, 9, 16] and can be accomplished with the insertion of a T-tube exteriorizing the gastric content to the skin or suction on sponge apparatus trans-orally, if a stent is not used. If the perforation is quite small, an over-the-scope bear claw clip (Ovesco TM) can be used in less than 10 mm holes. One can expect about 70–80% success with these approaches [60]. Vacuum therapy is used in a few centers and has to be homemade at the present time [31, 66]. After 12 weeks, if the fistula is still present, the surgical options are very few and involve the connection with a piece of jejunum, either a conversion to Roux-en-Y gastric bypass (with or without gastrectomy) or a fistula-jejunostomy preferably also in a Roux-en-Y reconstruction to derive the bile flow away from the leakage site [25, 106]. A gastrectomy adds considerable degree of complexity, morbidity, and mortality risks

and is best avoided, reserving this in cases of associated lower strictures, where leaving a bad sleeve below is not optimal [19].

Management of Gastrocutaneous, Gastropleural, and Gastrocolic Fistulas

Eventually, if leaks are not successfully treated, they will progress to fistulas, and a surgical treatment using an open or laparoscopic approach is being advocated.

Gastrocolic fistulas are uncommon, and they may follow surgical procedures in the upper abdomen such as gastrectomies [123]. I encountered it once, in a patient with a recurrent intra-abdominal abscess caused by a leak in the gastric staple line that complicated a laparoscopic re-SG performed for weight regain following laparoscopic DS [74]. An upper GI series showed contrast in the gastric sleeve communicating with the colon, confirmed by colonoscopy. I first drained the abscess percutaneous with a pigtail catheter. Then, in a second stage, an esophageal stent was positioned from the third inferior segment of the esophagus into the gastric pouch using upper endoscopy.

The stent was removed without incident 19 days after insertion. Unfortunately 3 weeks later the patient presented with fever and abdominal pain and her work-up revealed a recurrent fistula. The patient was returned to the operating room where the fistula was repaired laparoscopically. The post-op course was uneventful. After more than 18 months of follow-up, the patient has not had any recurrence of symptoms, with a BMI of 24 kg/m^2. So, it is likely that most of these complex fistulas will require a laparoscopic reoperation, in which the tract is removed by stapling and fat, like greater omentum, is interposed by hand-sewn techniques.

For gastrocutaneous fistula, Baltasar and colleagues reported satisfactory results after creation of a Roux-en-Y limb to treat leaks following SG [6]. Stable patients can be managed surgically after drainage has been established, and appropriate nutritional supplementation has been given, so we can trust the tissues for reconstruction [4]. Enteral or, if necessary, parenteral feeding is important because an adequate nutrition appears to promote fistula closure. Now, Chouillard et al. have accumulated one of the largest experiences with fistula-jejunostomy Roux-en-Y [25, 58, 124]. Effectively, for a period of 6 years, they treated 75 patients with post-SG fistula mainly from other centers. Immediate management principles included computerized tomography (CT) scan-guided drainage of collections or surgical peritoneal lavage, nutritional support, and endoscopic stenting. Ultimately, this approach achieved fistula control in nearly two-thirds of the patients. In the remaining one-third, Fistula-jejunostomy was proposed. Thirty patients (22 women and 8 men) had RYFJ for post-SG fistula. Procedures were performed laparoscopically in all but three cases (90%). With a mean follow-up of 22 months, assessments revealed no persistent fistula and no residual collections.

Concerning gastropleural fistulas, the abdominal management is the same, but a thoracic (thoracoscopic) approach has to be added with some decortication of the

lung, in the cases I have seen presented at several meetings. Rarely, endoscopic treatment is successful [22].

After multiple abdominal surgeries, exploration of the abdominal cavity is more difficult. Also, the current clinical status of the patient may contraindicate a surgical approach. Esophageal stent placement (endoluminal technique) is an effective strategy for treatment of upper gastrointestinal enteric fistula before 12 weeks and may be performed safely to treat complications after bariatric surgery [17]. Septotomy is used in a few centers, especially in Brazil [23, 53, 68, 114]. Every attempt should be made to prevent development of leaks, often by buttressing and/or oversewing of the staple line, but also by early identification of leaks in the intraoperative and early postoperative periods.

Endoscopic Stenting and Drainage

There are a growing number of reports of small case series using endoluminal procedures for leaks after SG [37, 38, 41, 45, 67, 71–73, 86, 107, 108, 110, 116, 117, 120, 121, 122]. Among the earliest, Eubanks and co-workers reported significant results in their experience with the use of stents for treatment of leaks after bariatric surgery [42]. Although the short-term results are promising, with a primary closure rate of 84% and immediate resumption of oral feeding after stenting, the mean follow-up was only 3.6 months, in 20/34 (58%) of patients, and a stent migration was noted; however, it did not affect the effectiveness of the endoscopic treatment for most patients. Nonetheless, the most important cause of failure was related to migration of the stents. Newer longer stents may reduce this risk, as well as sewing them in place temporarily [51, 79, 111, 126, 130]. In three patients, the stents were removed surgically. Nowadays, several clinicians add suturing the upper part of the stent to the esophagus temporarily to prevent migration.

Papavramidis and colleagues used fibrin sealant for closure of leaks [88, 89]. The effectiveness was satisfactory in all patients after several applications to obtain a complete closure of the leak [63]. A group from San Paulo University, Brazil, described the endoscopic application for repair of gastric leak after bariatric surgery with an acellular biomaterial (produced with small intestine of porcine) [69]. This seems to be effective in closure of the fistulas, because it produces an inflammatory reaction. Their conclusion is that the procedure is safe and effective. However, two or three endoscopic sessions are usually needed. This initial experience seems to be promising, with a closure success of 80% (20/25 patients).

The use of the coated self-expanding stents, described by Serra et al., can bypass the site of the leak and allows the patient to maintain oral nutrition until the fistula closes. They use fluoroscopy to place the stents and endoscopy for positioning difficulties. The incidence of migration has been reported to be as high as 33% [104]. Serra et al. published a report on six patients, of whom five used coated, self-expanding stents and one had an uncovered Wallstent [104]. Unfortunately, the latter had a stenosis and mucosal hypertrophy, and a total gastrectomy was needed; therefore coated stents should be used preferentially.

Kauer et al. in a study of ten patients, using self-expanding metal stent (Choosten™, M.I. Tech, Seoul, Korea) fully covered by silicon had a closure of the leak in 90% [61]. Recently, new longer stents for colorectal complications have been used for endoscopic management of leaks after LSG. However, they seem to be poorly tolerated, as it needs to be in place 4–6 weeks [64, 96].

Lately, the use of pigtail drains [16] (Zimmon® Biliary Stent from Cook Ireland Ltd., Limerick, Ireland) [93, 97, 98] and Over-The-Scope-Clip (OTSC®) system (Ovesco Endoscopy AG, Tübingen, Germany) has increased dramatically [111, 115]. Surace et al. reported with the use of the OTSC® system in a heterogeneous group of patients with gastrointestinal fistula [122]. In their analysis of 19 patients, there were 11 cases with gastric fistulas following sleeve gastrectomy with a successful closure rate of 91%. Another successful experience was reported by Conio et al., but the difficulties are the quality of the tissue to be approximated, how deep it goes, and the need to be perpendicular in a tight sleeve [27].

Noel et al. have described a new algorithm for the treatment of leaks using endoscopic methods entirely [75]. A total of 19 patients received endoscopic treatment of leak after LSG between May 2007 and June 2013 in Bouchard Clinic and La Casamance Private Hospital (Marseille, France).

The leaks were classified according with the primary orifice's size: group A (<10 mm) and group B (leak size >10 mm) with and without presence of gastric sleeve stenosis. During the initial evaluation, a double-channel endoscope was used for all the cases, and the leak size was estimated by direct view. A new algorithm was developed and followed to reduce endoscopic procedures. For patients with a primary leak, orifice of less than 10 mm without midgastric stenosis ($n = 6$), insertion of a double pigtail drain (7 Fr in diameter and 4 or 5 cm in length) was used to facilitate direct healing process [75].

Endoscopic treatment is combined with parenteral nutrition for 2 weeks with repetitive studies between 6 and 8 weeks. During the control upper endoscopy in four cases, the double pigtail drain was removed, and in two other cases, an additional OTSC® system (Ovesco) was used in order to manage the persistence of a blind orifice.

In the presence of stenosis ($n = 3$), in addition to the pigtail drain, the insertion of a covered prosthesis, 20–23 cm in length and 24 Fr in diameter (HANAROSTENT, M.I. Tech, Seoul, Korea) was performed in order to expand the stenosis and reduce pressure inside the gastric sleeve. The endoscopic treatment includes parenteral feeding for a minimum of ~~4~~2 weeks. A new endoscopic exam was performed for stent removal with pigtail drain left in place for another 4 weeks [75].

For the ten patients with a primary leak, orifice greater than 10 mm with or without midgastric stenosis, deployment of a prosthetic covered stent was performed. The treatment was combined with parenteral nutrition for several weeks. After usually 4 weeks, an endoscopic control was performed, and if the orifice was less than 10 mm, a double pigtail drain was inserted. If the diameter of the fistulous site was greater than 10 mm, the pigtail was replaced with a new one for another 4 weeks [75].

Therefore an average number of 2.8 endoscopic procedures were required in group A and 4 in group B. Two out of 13 patients had prosthetic migration (15.4%) and necessitated an additional procedure, one in group A and one in group B. One patient in group A required additional surgery after initial drainage for uncontrolled sepsis on postoperative day 8 after the first laparoscopic drainage was performed. For group B patients, it was never necessary to perform additional surgery for uncontrolled sepsis or nonhealing leak.

Using the proposed algorithm, all the leaks achieved complete healing after an average duration of 3.4 months (range, 2–14 months), 2.8 months for group A, and 3.9 months for group B. No additional reconstructive surgery was required for persistent chronic gastric leak.

Comparing with stents, pigtail drains present few advantages (no stent migration, less pain, and more patient tolerance) that can be offered to some patients with leak size inferior to 10 mm and without gastric torsions. According to Noel et al. gastric leak management after LSG must be guided by the size of the fistulous site. There are special indications for endoscopic stents, pigtails, or clips. The decision to use one specific endoscopic approach must be made based on endoscopic findings, especially the diameter and the presence of a gastric twist or stenosis. This new algorithm based on the size of the fistula and the presence of the gastric stenosis represents an additional tool for the standardization of the endoscopic management of leaks after LSG; it was possible to achieve closure of the leak for all 19 patients (100%) with LSG using appropriate stents, pigtail catheters, or clips, without mortality or additional surgery [75].

References

1. Ali MR, Tichansky DS, Kothari SN, McBride CL, Fernandez AZ Jr, Sugerman HJ, Kellum JM, Wolfe LG, DeMaria EJ. Validation that a 1-year fellowship in minimally invasive and bariatric surgery can eliminate the learning curve for laparoscopic gastric bypass. Surg Endosc. 2010;24(1):138–44.
2. Al Hajj G, Chemaly R. Fistula following laparoscopic sleeve gastrectomy: a proposed classification and algorithm for optimal management. Obes Surg. 2018;28(3):656–64.
3. Aryaie AH, Singer JL, Fayezizadeh M, Lash J, Marks JM. Efficacy of endoscopic management of leak after foregut surgery with endoscopic covered self-expanding metal stents (SEMS). Surg Endosc. 2017;31(2):612–7.
4. Baltasar A, Bou R, Miro J, Bengochea M, Serra C, Pérez N. Laparoscopic biliopancreatic diversion with duodenal switch: technique and initial experience. Obes Surg. 2002;12:245–8.
5. Baltasar A, Serra C, Pérez N, Bou R, Bengoechea M. Re-sleeve gastrectomy. Obes Surg. 2006;16:1535–8.
6. Baltasar A, Bou R, Bengochea M, Serra C, Cipagauta L. Use of a Roux limb to correct esophagogastric junction fistulas after sleeve gastrectomy. Obes Surg. 2007;17:1408–10.
7. Barreto TW, Kemmeter PR, Paletta MP, Davis AT. A comparison of a single center's experience with three staple line reinforcement techniques in 1,502 laparoscopic sleeve gastrectomy patients. Obes Surg. 2015;25(3):418–22
8. Baretta G, Campos J, Correia S, Alhinho H, Marchesini JB, Lima JH, Neto MG. Bariatric postoperative fistula: a life-saving endoscopic procedure. Surg Endosc. 2015;29(7):1714–20.

9. Bège T, Emungania O, Vitton V, Ah-Soune P, Nocca D, Noël P, Bradjanian S, Berdah SV, Brunet C, Grimaud JC, Barthet M. An endoscopic strategy for management of anastomotic complications from bariatric surgery: a prospective study. Gastrointest Endosc. 2011;73(2):238–44.
10. Bellanger DE, Greenway FL. Laparoscopic sleeve gastrectomy, 529 cases without a leak: short-term results and technical considerations. Obes Surg. 2011;21(2):146–50.
11. Benedix F, Benedix DD, Knoll C, Weiner R, Bruns C, Manger T, et al. Are there risk factors that increase the rate of staple line leakage in patients undergoing primary sleeve gastrectomy for morbid obesity? Obes Surg. 2014;24:1610–6.
12. Benedix F, Poranzke O, Adolf D, Wolff S, Lippert H, Arend J, Manger T, Stroh C. Obesity surgery working group competence network obesity. Staple line leak after primary sleeve gastrectomy-risk factors and mid-term results: do patients still benefit from the weight loss procedure? Obes Surg. 2017;27(7):1780–8.
13. Benotti P, Forse RA. Safety and long-term efficacy of revisional surgery in severe obesity. Am J Surg. 1996;172:232–5.
14. Berger ER, Huffman KM, Fraker T, Petrick AT, Brethauer SA, Hall BL, Ko CY, Morton JM. Prevalence and risk factors for bariatric surgery readmissions: findings from 130,007 admissions in the metabolic and bariatric surgery accreditation and quality improvement program. Ann Surg. 2018;267(1):122–31.
15. Boeker C, Mall J, Reetz C, Yamac K, Wilkens L, Stroh C, Koehler H. Laparoscopic sleeve gastrectomy: investigation of fundus wall thickness and staple height-an observational cohort study : fundus wall thickness and leaks. Obes Surg. 2017. [Epub ahead of print].
16. Bouchard S, Eisendrath P, Toussaint E, Le Moine O, Lemmers A, Arvanitakis M, Devière J. Trans-fistulary endoscopic drainage for post-bariatric abdominal collections communicating with the upper gastrointestinal tract. Endoscopy. 2016;48(9):809–16.
17. Brethauer SA, Pryor AD, Chand B, Schauer P, Rosenthal R, Richards W et al. Endoluminal procedures for bariatric patients: expectations among bariatric surgery. Surg Obes Relat Dis. 2009;5(2):231–6.
18. Brownlee A, Bromberg E, Roslin M. Outcomes in patients with helicobacter pylori undergoing laparoscopic sleeve gastrectomy. Obes Surg. 2015;25:2276–9.
19. Bruzzi M, Douard R, Voron T, Berger A, Zinzindohoue F, Chevallier JM. Open total gastrectomy with Roux-en-Y reconstruction for a chronic fistula after sleeve gastrectomy. Surg Obes Relat Dis. 2016;12(10):1803–8.
20. Buchwald H, Oien DM. Metabolic/bariatric surgery worldwide 2011. Obes Surg. 2013;23(4):427–36.
21. Burgos AM, Braghetto I, Csendes A, Maluenda F, Korn O, Yarmuch J, et al. Gastric leak after laparoscopic-sleeve gastrectomy for obesity. Obes Surg. 2009;19(12):1672–7.
22. Campos JM, Pereira EF, Evangelista LF, et al. Gastrobronchial fistula after sleeve gastrectomy and gastric bypass: endoscopic management and prevention. Obes Surg. 2011;21(10):1520–9.
23. Campos JM, Ferreira FC, Teixeira AF, Lima JS, Moon RC, D'Assunção MA, Neto MG. Septotomy and balloon dilation to treat chronic leak after sleeve gastrectomy: technical principles. Obes Surg. 2016;26(8):1992–3.
24. Cesana G, Cioffi S, Giorgi R, Villa R, Uccelli M, Ciccarese F, Castello G, Scotto B, Olmi S. Proximal leakage after laparoscopic sleeve gastrectomy: an analysis of preoperative and operative predictors on 1738 consecutive procedures. Obes Surg. 2018;28(3):627–35.
25. Chouillard E, Younan A, Alkandari M, Daher R, Dejonghe B, Alsabah S, Biagini J. Roux-en-Y fistulo-jejunostomy as a salvage procedure in patients with post-sleeve gastrectomy fistula: mid-term results. Surg Endosc. 2016;30(10):4200–4.
26. Clinical Issues Committee of American Society for Metabolic and Bariatric Surgery. Sleeve gastrectomy as a bariatric procedure. Surg Obes Relat Dis. 2007;3:573–6.
27. Conio M, Blanchi S, Repici A, Bastardini R, Marinari GM. Use of an over-the scope clip for endoscopic sealing of a gastric fistula after sleeve gastrectomy. Endoscopy. 2010;42(Suppl 2):E71–2.

28. Consten EC, Dakin GF, Gagner M. Intraluminal migration of bovine pericardial strips used to reinforced the gastric staple-line in laparoscopic bariatric surgery. Obes Surg. 2004;14:549–54.
29. Consten EC, Gagner M, Pomp A, Inabnet W. Decreased bleeding after laparoscopic sleeve gastrectomy with or without duodenal switch for morbid obesity using a stapled buttressed absorbable polymer membrane. Obes Surg. 2004;14:1360–6.
30. Consten EC, Gagner M. Staple-line reinforcement techniques with different buttressing materials used for laparoscopic gastrointestinal surgery: a new strategy to diminish perioperative complications. Surg Technol Int. 2004;13:59–63.
31. Cuadrado Ayuso M Franco Herrera R, Lago Oliver J. Successful management of laparoscopic sleeve gastrectomy leak with negative pressure therapy. Obes Surg. 2017. [Epub ahead of print].
32. Csendes A, Braghetto I, Leon P, et al. Management of leaks after laparoscopic sleeve gastrectomy in patients with obesity. J Gastrointest Surg. 2010;14(9):1343–8.
33. Daskalakis M, Berdan Y, Theodoridou S, et al. Impact of surgeon experience and buttress material on postoperative complications after laparoscopic sleeve gastrectomy. Surg Endosc. 2011;25(1):88–97.
34. De Angelis F, Abdelgawad M, Rizzello M, Mattia C, Silecchia G. Perioperative hemorrhagic complications after laparoscopic sleeve gastrectomy: four-year experience of a bariatric center of excellence. Surg Endosc. 2016. [Epub ahead of print].
35. Dolan K, Hatzifotis M, Newbury L, Lowe N, Fielding G. A clinical and nutritional comparison of biliopancreatic diversion with and without duodenal switch. Ann Surg. 2004;240:51–6.
36. D'Ugo S, Gentileschi P, Benavoli D, Cerci M, Gaspari A, Berta RD, et al. Comparative use of different techniques for leak and bleeding prevention during laparoscopic sleeve gastrectomy: a multicenter study. Surg Obes Relat Dis. 2014;10(3):450–4.
37. Edwards CA, Bui TP, Astudillo JA, et al. Management of anastomotic leaks after Roux-en-Y bypass using self-expanding polyester stents. Surg Obes Relat Dis. 2008;4:594–9. discussion 599–600
38. Eisendrath P, Cremer M, Himpens J, Cadière GB, Le Moine O, Devière J. Endotherapy including temporary stenting of fistulas of the upper gastrointestinal tract after laparoscopic bariatric surgery. Endoscopy. 2007;39:625–30.
39. Elariny H, Gonzalez H, Wang B. Tissue thickness of human stomach measured on excised gastric specimens from obese patients. Surg Technol Int. 2005;14:119–24.
40. Ellens NR, Simon JE, Kemmeter KD, Barreto TW, Kemmeter PR. Evaluating the feasibility of phrenoesophagopexy during hiatal hernia repair in sleeve gastrectomy patients. Surg Obes Relat Dis. 2017;13(12):1952–6.
41. El Mourad H, Himpens J, Verhofstadt J. Stent treatment for fistula after obesity surgery: results in 47 consecutive patients. Surg Endosc. 2013;27(3):808–16.
42. Eubanks S, Edwards CA, Fearing NM, Ramaswamy A, de la Torre RA, Thaler KJ, et al. Use of endoscopic stents to treat anastomotic complications after bariatric surgery. J Am Coll Surg. 2008;206:935–8.
43. Fezzi M, Kolotkin RL, Nedelcu M, et al. Improvement in quality of life after laparoscopic sleeve gastrectomy. Obes Surg. 2011;21(8):1161–7.
44. Froylich D, Corcelles R, Davis M, Boules M, Daigle CR, Schauer PR, Brethauer SA. Factors associated with length of stay in intensive care after bariatric surgery. Surg Obes Relat Dis. 2016;12(7):1391–6.
45. Fukumoto R, Orlina J, McGinty J, Teixeira J. Use of polyflex stents in treatment of acute esophageal and gastric leaks after bariatric surgery. Surg Obes Relat Dis. 2007;3:68–72.
46. Gagner M, Rogula T. Laparoscopic reoperative sleeve gastrectomy for poor weight loss after biliopancreatic diversion with duodenal switch. Obes Surg. 2003;13:649–54.
47. Gagner M. Meta-analysis of leaks following laparoscopic vertical sleeve gastrectomy. Obes Surg. 2011;21(8):958. Abstract PL02-05; presented at the XVI World Congress of International Federation of the Surgery of Obesity (IFSO), Aug 31-Sept 3, 2011, Hamburg, Germany

48. Gagner M, Deitel M, Erickson AL, et al. Survey on laparoscopic sleeve gastrectomy (LSG) at the fourth international consensus summit on sleeve gastrectomy. Obes Surg. 2013;23:2013–7.
49. Gagner M, Buchwald J. Comparison of laparoscopic sleeve gastrectomy leak rates in four staple-line reinforcement options: a systematic review. Surg Obes Relat Dis. 2014;10(4):713–23.
50. Gagner M, Brown M. Update on sleeve gastrectomy leak rate with the use of reinforcement. Obes Surg. 2016;26:146–50.
51. Garofalo F, Noreau-Nguyen M, Denis R, Atlas H, Garneau P, Pescarus R. Evolution of endoscopic treatment of sleeve gastrectomy leaks: from partially covered to long, fully covered stents. Surg Obes Relat Dis. 2017;13(6):925–32.
52. Geubbels N, de Brauw LM, Acherman YI, van de Laar AW, Wouters MW, Bruin SC. The preceding surgeon factor in bariatric Surgery: a positive influence on the learning curve of subsequent surgeons. Obes Surg. 2014. [Epub ahead of print].
53. Guerron AD, Ortega CB, Portenier D. Endoscopic abscess septotomy for management of sleeve gastrectomy leak. Obes Surg. 2017. https://doi.org/10.1007/s11695-017-2809-0. [Epub ahead of print]
54. Hess DS, Hess DW. Biliopancreatic diversion with a duodenal switch. Obes Surg. 1998;8:267–82.
55. Hess DS, Hess DW, Oakley R. The biliopancreatic diversion with the duodenal switch: results beyond 10 years. Obes Surg. 2005;15:408–16.
56. Huang R, Gagner M. A thickness calibration device is needed to determine staple height and avoid leaks in laparoscopic sleeve gastrectomy. Obes Surg. 2015;25:2360–7.
57. Hussain A, Vasas P, Kirk K, Finney J, Balchandra S. Etiology of leaks following sleeve gastrectomy: current evidence. Surg Laparosc Endosc Percutan Tech. 2017;27(3):119–22.
58. Iannelli A, Tavana R, Martini F, Noel P, Gugenheim J. Laparoscopic roux limb placement over a fistula defect without mucosa-to-mucosa anastomosis: a modified technique for surgical management of chronic proximal fistulas after laparoscopic sleeve gastrectomy. Obes Surg. 2014;24(5):825–8.
59. Iossa A, Abdelgawad M, Watkins BM, Silecchia G. Leaks after laparoscopic sleeve gastrectomy: overview of pathogenesis and risk factors. Langenbeck's Arch Surg. 2016;401(6):757–66.
60. Jacobsen GR, Coker AM, Acosta G, Talamini MA, Savides TJ, Horgan S. Initial experience with an innovative endoscopic clipping system. Surg Technol Int. 2012;22:39–43.
61. Kauer WK, Stein HJ, Dittler HJ, Siewert JR. Stent implantation as a treatment option in patients with thoracic anastomotic leaks after esophagectomy. Surg Endosc. 2008;22:50–3.
62. Kim WW, Gagner M, Kini S, Inabnet WB, Quinn T, Herron D, Pomp A. Laparoscopic vs. open biliopancreatic diversion with duodenal switch: a comparative study. J Gastrointest Surg. 2003;7:552–7.
63. Kowalski C, Kastuar S, Mehta V, Brolin R. Endoscopic injection of fibrin sealant in repair of gastrojejunostomy leak after laparoscopic Roux-en Y gastric bypass. Surg Obes Relat Dis. 2007;3:438–42.
64. Kriwanek S, Ott N, Ali-Abdullah S, Pulgram T, Tscherney R, Reiter M, Roka R. Treatment of gastro-jejunal leakage and fistulization after gastric bypass with coated self-expanding stents. Obes Surg. 2006;16:1669–74.
65. Lazzati A, Guy-Lachuer R, Delaunay V, Szwarcensztein K, Azoulay D. Bariatric surgery trends in France: 2005–2011. Surg Obes Relat Dis. 2014;10(2):328–34.
66. Leeds SG, Burdick JS. Management of gastric leaks after sleeve gastrectomy with endoluminal vacuum (E-Vac) therapy. Surg Obes Relat Dis. 2016;12(7):1278–85.
67. Lindenmann J, Matzi V, Porubsky C, Anegg U, Sankin O, Gabor S, et al. Self-expandable covered metal tracheal type stent for sealing cervical anastomotic leak after esophagectomy and gastric pull-up: pitfalls and possibilities. Ann Thorac Surg. 2008;85:354–6.
68. Mahadev S, Kumbhari V, Campos JM, Galvao Neto M, Khashab MA, Chavez YH, Bessler M, Gonda TA. Endoscopic septotomy: an effective approach for internal drainage of sleeve gastrectomy-associated collections. Endoscopy. 2017;49(5):504–8.

69. Maluf-Filho F, Lima MS, Hondo F. Endoscopic placement of a "plug" made of acellular biomaterial: a new technique for the repair of gastric leak after Roux-en-Y gastric bypass. Arq Gastroenterol. 2008;45:208–1.
70. Marceau P, Hould FS, Simard S, Lebel S, Bourque RA, Potvin M, Biron. Biliopancreatic diversion with duodenal switch. World J Surg. 1998;22:947–54.
71. Marquez M, Ayza Ferrer M, Belda LR, del Mar Rico Morales M, Diez JM, Belda Poujoulet R. Gastric leak after laparoscopic sleeve gastrectomy. Obes Surg. 2010;20:1306–11.
72. Mejía AF, Bolaños E, Chaux CF, Unigarro I. Endoscopic treatment of gastrocutaneous fistula following gastric bypass for obesity. Obes Surg. 2007;17:544–6.
73. Montuori M, Benavoli D, D'Ugo S, Di Benedetto L, Bianciardi E, Gaspari AL, Gentileschi P. Integrated approaches for the management of staple line leaks following sleeve gastrectomy. J Obes. 2017;2017:4703236. Epub 2017 Feb 2
74. Trelles N, Gagner M, Palermo M, Pomp A, Dakin G, Parikh M. Gastrocolic fistula after re-sleeve gastrectomy: outcomes after esophageal stent implantation. Surg Obes Relat Dis. 2010;6(3):308–12.
75. Nedelcu M, Manos T, Cotirlet A, Noel P, Gagner M. Outcome of leaks after sleeve gastrectomy based on a new algorithm addressing leak size and gastric stenosis. Obes Surg. 2015;25(3):559–63.
76. Nedelcu M, Skalli M, Delhom E, Fabre JM, Nocca D. New CT scan classification of leak after sleeve gastrectomy. Obes Surg. 2013;23(8):1341–3.
77. Nedelcu AM, Skalli M, Deneve E, Fabre JM, Nocca D. Surgical management of chronic fistula after sleeve gastrectomy. Surg Obes Relat Dis. 2013;9(6):879–84.
78. Nedelcu M, Manos T, Gagner M, Eddbali I, Ahmed A, Noel P. Cost analysis of leak after sleeve gastrectomy. Surg Endosc. 2017. https://doi.org/10.1007/s00464-017-5495-z. [Epub ahead of print]
79. Nedelcu M, Noel P. Paired editorial: evolution of endoscopic treatment of sleeve gastrectomy leaks: from partially covered to long fully covered stents. Surg Obes Relat Dis. 2017;13(6):933.
80. Nguyen NT, Longoria M, Chalifoux S, Wilson SE. Bioabsorbable staple line reinforcement for laparoscopic gastrointestinal surgery. Surg Technol Int. 2005;14:107–11.
81. Nguyen NT, Longoria M, Welbourne S, Savio A, Wilson S. Glycolide copolymer staple line reinforcement reduces staple site bleeding during laparoscopic gastric bypass. A prospective randomized trial. Arch Surg. 2005;140:773–8.
82. Nguyen NT, Vu S, Kim E, Bodunova N, et al. Trends in utilization of bariatric surgery, 2009–2012. Surg Endosc. 2016;30:3723–7.
83. Noel P, Iannelli A, Sejor E, Schneck AS, Gugenheim J. Laparoscopic sleeve gastrectomy: how I do it. Surg Laparosc Endosc Percutan Tech. 2013;23(1):e14–6.
84. Noel P, Schneck A, Nedelcu M, Lee J, et al. Laparoscopic sleeve gastrectomy as a revisional procedure for failed gastric banding: lessons from 300 consecutive cases. Surg Obes Relat Dis. 2014;10:1166–22.
85. Noel P, Nedelcu M, Gagner M. Impact of the surgical experience on leak rate after laparoscopic sleeve gastrectomy. Obes Surg. 2016;26(8):1782–7.
86. Nowakowski P, Ziaja K, Ludyga T, Kuczmik W, Biolik G, Cwik P, et al. Self-expandable metallic stents in the treatment of post-esophagogastrostomy/post-esophagoenterostomy fistula. Dis Esophagus. 2007;20:358–60.
87. Obeid N, Schwack B, Kurian M, Ren-Fielding C, et al. Single-stage versus 2-stage sleeve gastrectomy as a conversion after failed adjustable gastric banding: 30-day outcomes. Surg Endosc. 2014;28:3186–92.
88. Papavramidis ST, Eleftheriadis EE, Papavramidis TS, Kotzampassi KE, Gamvros OG. Endoscopic management of gastrocutaneous fistula after bariatric surgery by using fibrin sealant. Gastrointest Endosc. 2004;59:296–300.
89. Papavramidis TS, Kotzampassi K, Kotidis E, Eleftheriadis EE, Papavramidis ST. Endoscopic fibrin sealing of gastrocutaneous fistulas after sleeve gastrectomy and biliopancreatic diversion with duodenal switch. J Gastroenterol Hepatol. 2008;23:1802–5.

90. Parikh M, Issa R, McCrillis A, Saunders JK, Ude-Welcome A, Gagner M. Surgical strategies that may decrease leak after laparoscopic sleeve gastrectomy: a systematic review and meta-analysis of 9991 cases. Ann Surg. 2013;257(2):231–7.
91. Patel RA, Brolin RE, Gandhi A. Revisional operations for marginal ulcer after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5(3):317–22.
92. Peker KD, Sahbaz NA, Seyit H, Kones O, Gumusoglu AY, Alis H. An alternative view on the necessity of EGD before sleeve gastrectomy. Surg Obes Relat Dis. 2017;13(12):1959–64.
93. Pequignot A, Fuks D, Verhaeghe P, Dhahri A, Brehant O, Bartoli E, Delcenserie R, Yzet T, Regimbeau JM. Is there a place for pigtail drains in the management of gastric leaks after laparoscopic sleeve gastrectomy? Obes Surg. 2012;22(5):712–20.
94. Perez M, Brunaud L, Kedaifa S, Guillotin C, et al. Does anatomy explain the origin of a leak after sleeve gastrectomy? Obes Surg. 2014;24:1717–23.
95. Prachand VN, Davee RT, Alverdy JC. Duodenal switch provides superior weight loss in the super-obese (BMI > or = 50 kg/m^2) compared with gastric bypass. Ann Surg. 2006;244:611–9.
96. Puli SR, Spofford IS, Thompson CC. Use of self-expandable stents in the treatment of bariatric surgery leaks: a systematic review and meta-analysis. Gastrointest Endosc. 2012;75(2):287–93.
97. Rebibo L, Delcenserie R, Brazier F, Yzet T, Regimbeau JM. Treatment of gastric leaks after sleeve gastrectomy. Endoscopy. 2016;48(6):590.
98. Rebibo L, Hakim S, Brazier F, Dhahri A, Cosse C, Regimbeau JM. New endoscopic technique for the treatment of large gastric fistula or gastric stenosis associated with gastric leaks after sleeve gastrectomy. Surg Obes Relat Dis. 2016;12(8):1577–84.
99. Regan JP, Inabnet WB, Gagner M, Pomp A. Early experience with two-stage laparoscopic Roux-en-Y gastric bypass as an alternative in the super-super obese patient. Obes Surg. 2003;13:861–4.
100. Ren CJ, Patterson E, Gagner M. Early results of laparoscopic biliopancreatic diversion with duodenal switch: a case series of 40 consecutive patients. Obes Surg. 2000;10:514–24.
101. Rosenthal RJ, International Sleeve Gastrectomy Expert Panel, Diaz AA, Arvidsson D, Baker RS, Basso N, Bellanger D, Boza C, El Mourad H, France M, Gagner M, Galvao-Neto M, Higa KD, Himpens J, Hutchinson CM, Jacobs M, Jorgensen JO, Jossart G, Lakdawala M, Nguyen NT, Nocca D, Prager G, Pomp A, Ramos AC, Rosenthal RJ, Shah S, Vix M, Wittgrove A, Zundel N. International sleeve gastrectomy expert panel consensus statement: best practice guidelines based on experience of >12,000 cases. Surg Obes Relat Dis. 2012;8(1):8–19.
102. Sakran N, Goitein D, Raziel A, Keidar A, Beglaibter N, Grinbaum R, Matter I, Alfici R, Mahajna A, Waksman I, Shimonov M, Assalia A. Gastric leaks after sleeve gastrectomy: a multicenter experience with 2,834 patients. Surg Endosc. 2013;27(1):240–5.
103. Scopinaro N, Adami GF, Marinari GM. Biliopancreatic diversion: two decades of experience. In: Deitel M, editor. Update: surgery for the morbidly obese patient. Toronto: FD-Communications; 2000. p. 227–58.
104. Serra C, Baltasar A, Andreo L, Pérez N, Bou R, Bengochea M, Chisbert J. Treatment of gastric leaks with self-expanding stents after sleeve gastrectomy. Obes Surg. 2007;17:866–72.
105. Ueda K, Gagner M, Milone L, Bardaro S, Gong K. Sleeve gastrectomy with wrapping using polytetrafluoroethylene to prevent gastric enlargement in a porcine model. Surg Obes Relat Dis. 2008;4:84–90.
106. Saglam K, Aktas A, Gundogan E, Ertugrul I, Tardu A, Karagul S, Kirmizi S, Sumer F, Ersan V, Kayaalp C. Management of acute sleeve gastrectomy leaks by conversion to Roux-en-Y gastric bypass: a small case series. Obes Surg. 2017;27(11):3061–3.
107. Salinas A, Baptista A, Santiago E, Antor M, Salinas H. Self-expandable metal stents to treat gastric leaks. Surg Obes Relat Dis. 2006;2(5):570–2.
108. Sanchez-Santos R, Masdevall C, Baltasar A, et al. Short and mid-term outcomes of sleeve gastrectomy for morbid obesity: the experience of the Spanish National Registry. Obes Surg. 2009;19:1203–10.

109. Ser KH, Lee WJ, Lee YC, et al. Experience in laparoscopic sleeve gastrectomy for morbidly obese Taiwanese: staple-line reinforcement is important for preventing leakage. Surg Endosc. 2010;24(9):2253–9.
110. Serra C, Baltasar A, Andreo L. Treatment of gastric leaks with coated self-expanding stents after sleeve gastrectomy. Obes Surg. 2007;17:866–72.
111. Shehab H, Abdallah E, Gawdat K, Elattar I. Large bariatric-specific stents and over-the-scope clips in the Management of Post-Bariatric Surgery Leaks. Obes Surg. 2018;28(1):15–24.
112. Shikora SA, Kim JJ, Tarnoff ME. Reinforcing gastric staple lines with bovine pericardial strips may decrease the likelihood of gastric leak after laparoscopic roux-en-Y gastric bypass. Obes Surg. 2003;13:37–44.
113. Shikora S, Mahoney C. Clinical benefit of gastric staple line reinforcement (SLR) in gastrointestinal surgery: a meta-analysis. Obes Surg. 2015;25:1133–41.
114. Shnell M, Gluck N, Abu-Abeid S, Santo E, Fishman S. Use of endoscopic septotomy for the treatment of late staple-line leaks after laparoscopic sleeve gastrectomy. Endoscopy. 2017;49(1):59–63.
115. Shoar S, Poliakin L, Khorgami Z, Rubenstein R, El-Matbouly M, Levin JL, Saber AA. Efficacy and safety of the over-the-scope clip (OTSC) system in the Management of Leak and Fistula after laparoscopic sleeve gastrectomy: a systematic review. Obes Surg. 2017;27(9):2410–18.
116. Simon F, Siciliano I, Gillet A, Castel B, Coffin B, Msika S. Gastric leak after laparoscopic sleeve gastrectomy: early covered self-expandable stent reduces healing time. Obes Surg. 2013;23(5):687–92.
117. Sporn E, Miedema BW, Astudillo JA, Thaler K. Lessons learned establishing an animal model for endoscopic stent placement to treat gastrojejunal anastomotic leaks after Gastric bypass. Obes Surg. 2009;19(8):1163–9.
118. Strain GW, Gagner M, Inabnet WB, Dakin G, Pomp A. Comparison of effects of gastric bypass and biliopancreatic diversion with duodenal switch on weight loss and body composition 1–2 years after surgery. Surg Obes Relat Dis. 2007;3:31–6.
119. Stroh C, Kockerling F, Volker L, Frank B, et al. Results of more than 11,800 sleeve gastrectomies: data analysis of the German Bariatric Surgery Registry. Ann Surg. 2016;263:949–55.
120. Surace M, Mercky P, Demarquay JF, Gonzalez JM, Dumas R, Ah-Soune P, et al. Endoscopic management of GI fistulae with over-the-scope clip system. Gastrointest Endosc. 2011;74(6):1416–9.
121. Swinnen J, Eisendrath P, Rigaux J, Kahegeshe L, Lemmers A, Le Moine O, Devière J. Self-expandable metal stents for the treatment of benign upper GI leaks and perforations. Gastrointest Endosc. 2011;73(5):890–9.
122. Tan TJ, Kariyawasam S, Wijeratne T, Chandraratna S. Diagnosis and management of gastric leaks after laparoscopic sleeve gastrectomy for morbid obesity. Obes Surg. 2010;20:403–9.
123. Tavenor T, Smith S, Sullivan S. Gastrocolic fistula – a review of 15 cases and an update of the literature. J Clin Gastroenterol. 1993;16:189–91.
124. Thomopoulos T, Thoma M, Navez B. Roux-En-Y Fistulojejunostomy: a new therapeutic option for complicated post-sleeve gastric fistulas, video-report. Obes Surg. 2017;27(6):1638–9.
125. Trelles N, Gagner M. Sleeve gastrectomy. In: Koltun WA, editor. Operatives techniques in general surgery, vol. 9. Hershey: Elsevier; 2007. p. 123–31.
126. Tringali A, Bove V, Perri V, Landi R, Familiari P, Boškoski I, Costamagna G. Endoscopic treatment of post-laparoscopic sleeve gastrectomy leaks using a specifically designed metal stent. Endoscopy. 2017;49(1):64–8.
127. Varban OA, Sheetz KH, Cassidy RB, Stricklen A, Carlin AM, Dimick JB, Finks JF. Evaluating the effect of operative technique on leaks after laparoscopic sleeve gastrectomy: a case-control study. Surg Obes Relat Dis. 2017;13(4):560–7.
128. Villalba MR, Villalba MR. Development of a gastric pouch-aorto-colic fistula as a complication of a revisionary open Roux-en-Y gastric bypass. Obes Surg. 2009;19:265–8. Epub 2008

129. Weiner RA, Weiner S, Pomhoff I, Jacobi C, Makarewicz W, Weigand G. Laparoscopic sleeve gastrectomy-influence of sleeve size and resected gastric volume. Obes Surg. 2007;17:1297–305.
130. Wright A, Chang A, Bedi AO, Wansteker EJ, Elta G, Kwon RS, Carrott P, Elmunzer BJ, Law R. Endoscopic suture fixation is associated with reduced migration of esophageal fully covered self-expandable. Surg Endosc. 2017;31:3489–94.
131. Yehoshua RT, Eidelman LA, Stein M, Fichman S, Mazor A, Chen J, et al. Laparoscopic sleeve gastrectomy – volume and pressure assessment. Obes Surg. 2008;18:1083–8.
132. Yo LS, Consten EC, Quarles van Ufford HM, Gooszen HG, Gagner M. Buttressing of the staple line in gastrointestinal anastomoses: overview of new technology designed to reduce perioperative complications. Dig Surg. 2006;23:283–91.

Anastomotic Leak Following Gastric Bypass

6

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Introduction

Obesity represents a major public health-care problem. The WHO estimates that 13% of the world's adult population suffers with obesity [1]. Bariatric surgery is considered to be the most effective option for its treatment and related comorbidities [2, 3]. Over the past decades, the Roux-en-Y gastric bypass (RYGB) has been proven to be very effective due to the favorable metabolic effect provided [3]. Currently, RYGB is the second most common bariatric procedure performed worldwide (36.9%) following sleeve gastrectomy (45.9%) [2]. Nonetheless, this procedure may be technically challenging because it requires operating in two different abdominal quadrants within the characteristic body habitus of a patient with morbid obesity, where advanced surgical skills are crucial for appropriate intestinal reconstruction [4].

Despite the low morbidity and mortality rates associated with RYGB, several perioperative complications may arise including bleeding, infection, port site herniation, marginal ulceration, anastomotic leaks, and anastomotic strictures [3]. Among these conditions, anastomotic leak is the most serious and feared complication following the procedure.

Postoperative anastomotic leak incidence is variable, ranging from 1% to 5.6% [5, 6], but is associated with high morbidity and mortality rates (30%) [7]. Moreover, a leak rate of 1.5% at a high-volume bariatric center would be considered at the higher end of leak rates [8]. The presence of anastomotic leaks represents a devastating complication for the patient, leading to significant morbidity and mortality, extended length of hospital stay, additional diagnostic studies, and potential reoperation. The clinical presentation may be subtle or even delayed, requiring a high

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index of suspicion and attention during the postoperative course [9]. An early diagnosis is crucial for the management of leaks and can significantly reduce the risk of further complications [10]. The most effective strategy consists in adapting the therapeutic options to the time of presentation and to the patient's hemodynamic status. The key features of the management are based on medical support, appropriate drainage of the leak, and repairing the underlying defect.

Etiology and Classification

Anastomotic leaks are defined by Brethauer et al. [11] as "the egress of gastrointestinal contents through a suture or staple line into a cavity." The pathogenesis of leaks depends on mechanical and/or ischemic factors disrupting the normal acute healing process [12], in addition to an increasing intraluminal pressure that exceeds the strength of the anastomotic staple line [13]. These factors are dived into technique-related factors, such as tissue tension or types of anastomosis reinforcement [14], and patient-related factors. Nguyen et al. [4] found that factors associated with higher rates of complications following gastric bypass were age greater than 50 and male gender. Likewise, Livingston et al. [15] reported in their study that BMI $\geq$50 kg/m^2, male gender, and previous bariatric operations were independent factors for the development of leaks after RYGB.

The post-RYGB anastomotic leak classification [6, 13, 16] depends on the time of presentation of the leak, its severity (Table 6.1), and its location (Fig. 6.1). Leaks developing within 5 days are mostly related to technical properties of the procedure [9], whereas leaks occurring after 5 days post-procedure are usually resulting from localized ischemia or infection [7, 16]. Csendes et al. [6] found intermediate leaks to be the most frequent type (46.7%) followed by early leaks (28.3%) and late leaks (25%). Additionally, they reported 80% of the anastomotic leaks as clinically severe.

Regarding the location of a leak, the most common site is at the gastrojejunal anastomosis (GJ) [6, 13], followed by leaks located at the gastric pouch staple line; jejunal stump; jejunojejunostomy (JJ), which is associated with greater mortality rates; and gastric remnant staple line (Table 6.2) [6].

Table 6.1 Classification of anastomotic leaks following RYGB

According to *time*	
Early	Occurring 1–4 days after procedure
Intermediate	Occurring 5 to 9 days after procedure
Late	Occurring 10 or more days after procedure
According to *severity*	
Type I	Small localized leak, with none or minimal systemic symptoms
Type II	Large leak with systemic repercussion ± air fluid collections

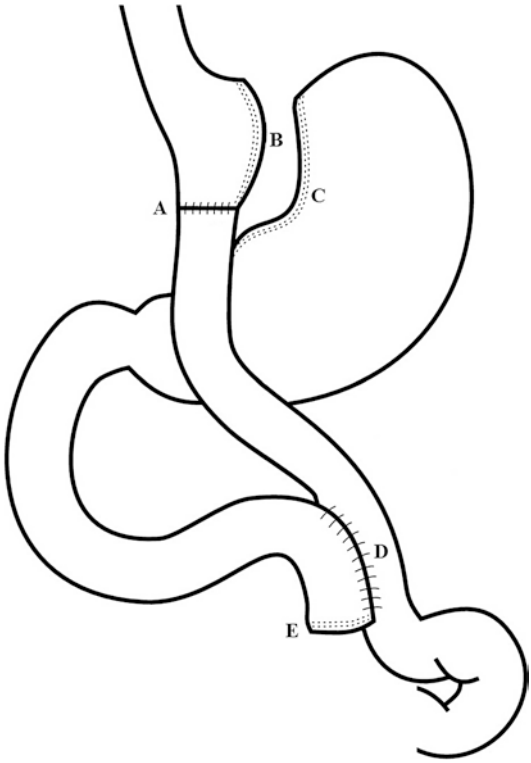

Fig. 6.1 Location of leaks following gastric bypass. (**a**) Gastrojejunal anastomosis. (**b**) Gastric pouch staple line. **c** Gastric remnant staple line. (**d**) Jejunojejunal anastomosis. (**e**) Jejunal stump

Table 6.2 Location of anastomotic leaks following RYGB

Site	Percentage
Gastrojejunostomy	53.3%
Gastric pouch staple line	18.3%
Jejunal stump	15%
Jejunojejunostomy	5.5%
Gastric remnant staple line	1.7%

Created with data from Csendes et al. [6]

Prevention

The best way to manage anastomotic leaks is to prevent its development. Multiple intraoperative methods are designed to decrease the incidence of leaks including, but not limited to, staple-line reinforcement with synthetic materials such as fibrin glue or other tissue sealants; however, studies report variable outcomes regarding the efficacy of these approaches. Varban et al. [14], in their multicentric study of anastomotic leaks following RYGB, found no association between the type of anastomosis (hand-sewn, circular or linear stapler) and the development of leaks.

Conversely, they found a significant relationship between the use of a fibrin sealant and lower leaks rate, whereas the use of buttressing material was found to be significantly related with higher rates.

Diagnostic tests such as the methylene blue test or the air-leak test may be beneficial in identifying leaks intraoperatively and allowing them to be repaired immediately during the procedure. However, these tests will not predict the future development of a leak [13].

Another important factor related to decreasing anastomotic leak rates is the surgeon's experience [17]. Schauer et al. [18] studied the learning curve of a single surgeon over 100 laparoscopic RYGB cases concluding that there is a significant relationship between greater operative performance and lower overall complication rate. Although the incidence of staple-line leaks decreased with greater surgeon's experience, the reduction did not reach statistical significance. Conversely, DeMaria et al. [19] studied 281 cases of laparoscopic RYGB and found a reduction in the rate of leaks on the latter phase of their series. They concluded that the learning curve is clearly associated with the rates of complications.

Early Diagnosis and Intervention

An early diagnosis is essential to significantly reduce the morbidity and mortality rates associated with postoperative leaks [10]. This condition may be challenging to diagnose and may rapidly progress to systemic illness; therefore, surgeons need to suspect and treat leaks in a timely manner. An appropriate evaluation of clinical signs and symptoms during the postoperative course is the key to an early diagnosis. Even minimal symptoms should be investigated for leaks, since its early identification is vital to achieving an optimal outcome. Post-procedural tachycardia, abdominal pain, fever, or persistent hiccups are the most common symptoms [20]. Tachycardia is the earliest indicator of hemodynamically instability. A heart rate greater than 120 should prompt an investigation, even if the patient looks and feels well. A pulse rate over 90 beats per minute on postoperative day 1 distinguishes between patient with and without leaks with a sensitivity of 100% and a specificity of 87% [12]. Furthermore, the combination of tachycardia, tachypnea, and fever has a high positive predictive value for the presence of leaks [10, 21]. A delay in diagnosis (>24 h) is associated with unfavorable outcomes [9].

Imaging studies are useful for the early detection of leaks [10, 16, 22]. Upper gastrointestinal series (UGIS) with soluble contrast assists evaluating the integrity of the gastrojejunostomy and jejunojejunostomy [13]. A routine UGIS has a positive predictive value of 67% and a negative predictive value of 99% for anastomotic leak detection [3]. Additionally, this study provides useful information about the intestinal anatomy or alterations such as abnormal dilation of the remnant or other causes of bowel obstruction. A contrast-enhanced abdominal CT scan has higher sensitivity and specificity than UGSI and is superior at detecting whether a leak appears contained or is communicating with the abdominal cavity. A CT scan is also helpful at detecting abscess, collections, hernias, or any other pathological

conditions after RYGB [13]. Although typically accurate, radiologic studies have limitations and can sometimes delay accurate diagnosis and therapy. Gonzalez et al. [23] reported a false-negative rate of 30% in patients with leaks undergoing combined diagnostic UGIS and CT scan.

Management

The management of anastomotic leaks needs a multidisciplinary approach based on the severity and location of the leak, as well as the hemodynamic status of the patient. Surgical management is the keystone of the treatment and should be considered in the early postoperative period and in every unstable patient. Operative management consists in adequate drainage of the leak to decontaminating the abdominal cavity and preventing future complications, followed by addressing the defect responsible for the leakage [5]. Nonoperative management may be considered in selected patient based on hemodynamic stability.

Endoscopy plays a valuable role in the diagnosis and treatment of gastrointestinal leaks after bariatric surgery. It is commonly used to delineate the gastric anatomy and to rule out the presence of distal strictures that may be contributing to the development of a leak and its failure to heal. It is also an excellent tool for multiple therapeutic interventions aimed to treating a leak (Table 6.3). Schiesser et al. [24] compared the outcomes of a group of patients treated with reoperation and drain placement versus the outcomes of a group of patients treated with endoscopy using different approaches such as stent placing, over-the-scope clip application, and percutaneous drains. The rate of leak resolution among the groups was 88% versus 100%, respectively.

Recently developed techniques, such as the use of vacuum-assisted endoscopic drainage, demonstrated promising rates of leak resolution ranging from 85% to 100% [25].

Several endoluminal therapies can be used for the management of a leak. Early drainage, early endoscopic intervention, and early correction of distal strictures are of great significance [20]. This process often includes placement of endoscopic clips, fibrin glue, absorbable fistula plugs, and endoluminal stenting. Chang et al. [26] studied the outcomes of endoscopically placed stents for the management of

Table 6.3 Outcomes of post-RYGB managed endoscopically

Study	No. of subjects	Resolution rate (%)	Therapeutic option used
Kowalski et al.	5	100%	Fibrin sealant injection
Victorzon et al.	6	100%	Fibrin sealant injection
Shehab et al.	12	100%	OTSC
Salinas et al.	17	94%	SEMS
Freedman et al.	35	86%	Sent (not specified)
Maluf-Filho et al.	25	80%	Fibrin sealant injection

Created with data from Joo [25]
OTSC over-the-scope clip, *SEMS* self-expandable metal stent

postoperative anastomotic complications after foregut surgery. Regarding the management of leaks following RYGB, they achieved 100% resolution of GJ and gastric pouch staple-line leaks, finally suggesting that managing these complications by endoscopy is very effective.

Medical support or conservative management includes nil per os status, broad-spectrum antibiotics, and percutaneous access to the gastric remnant for decompression and feeding, as well as percutaneous drainage of collections [13]. Sepsis control revolves around defining the leak and managing potential collections, either through a percutaneous approach or operative intervention. Nutritional support is essential and can be achieved in several different ways. Patients undergoing endoscopic intervention can either restore oral intake after exclusion of the leak or have reliable enteral access placed at the time of the endoscopy, either by nasojejunal route or endoscopic tube placement. Csendes et al. [6] reported in their study that 65% of anastomotic leaks were successfully managed by conservative treatment. Surgical intervention was performed in 9% of the localized leaks (type I) and in 42% of the clinically severe/disseminated leaks (type II). They concluded that early surgical intervention is necessary when dealing with type II leaks localized at the JJ or GJ anastomosis. On the other hand, when a leak develops several-day post-procedure, even if it is a type II anastomotic leak, it can be managed conservatively. Jacobsen et al. [9] studied 6000 patients post-laparoscopic RYGB and reported 64 patients complicated with anastomotic leaks (corresponding to 1.1% of the population). Two thirds of those patients were considered to have leaks categorized as IIIB or more (according to the Clavien-Dindo classification) and were managed successfully with reoperation. An interesting fact of this study was that 62% of those patients were diagnosed based only their clinical status (tachycardia, fever, abdominal pain).

The management of anastomotic leaks must be tailored to the severity and the time of presentation. Follow-up imaging studies are necessary to assessing the progress or resolution of leaks.

Conclusion

Anastomotic leaks following Roux-en-Y gastric bypass represent a serious complication in bariatric patients leading to significant morbidity and mortality. Early diagnosis is fundamental in their management. Furthermore, anastomotic leaks are predictable based on clinical symptoms in the postoperative period. Therefore, physicians should evaluate any postoperative tachycardia, tachypnea, or fever which can be the only parameters leading to an accurate diagnosis. If diagnostic tests are inconclusive but clinical suspicion is high, the patient should return to the operating room early. A delay in diagnosis and treatment is associated with adverse outcomes. If a patient is diagnosed with a contained leak on UGIS or CT scan and is hemodynamically stable, conservative management with bowel rest, antibiotics, and percutaneous drainage may be appropriate. Endoscopy must be considered as a therapeutic approach, since less invasive interventions with optimal outcomes are currently being highly demanded by the bariatric population.

References

1. Obesity and Overweight: fact sheet. World Health Organization; 2016.
2. Angrisani L, et al. Bariatric surgery and endoluminal procedures: IFSO Worldwide Survey 2014. Obes Surg. 2017;27:2279–89.
3. Berbiglia L, Zografakis JG, Dan AG. Laparoscopic Roux-en-Y gastric bypass: surgical technique and perioperative care. Surg Clin North Am. 2016;96(4):773–94.
4. Nguyen NT, Rivers R, Wolfe BM. Factors associated with operative outcomes in laparoscopic gastric bypass. J Am Coll Surg. 2003;197(4):548–55.
5. Afaneh C, Dakin GF. Enteric leaks after gastric bypass: prevention and management, in bariatric surgery complications and emergencies. In: Herron DM, editor. Springer, Switzerland 2016. p. 81–9.
6. Csendes A, Burgos AM, Braghetto I. Classification and management of leaks after gastric bypass for patients with morbid obesity: a prospective study of 60 patients. Obes Surg. 2012;22(6):855–62.
7. Brethauer SC, Chand B, Schauer P. Risks and benefits of bariatric surgery: current evidence. Cleve Clin J Med. 2006;73:993.
8. Genser L, et al. Presentation and surgical management of leaks after mini-gastric bypass for morbid obesity. Surg Obes Relat Dis. 2016;12(2):305–12.
9. Jacobsen HJ, et al. Management of suspected anastomotic leak after bariatric laparoscopic Roux-en-y gastric bypass. Br J Surg. 2014;101(4):417–23.
10. Quartararo G, et al. Upper gastrointestinal series after Roux-en-Y gastric bypass for morbid obesity: effectiveness in leakage detection. A systematic review of the literature. Obes Surg. 2014;24(7):1096–101.
11. Brethauer SA, Hammel JP, Schauer PR. Systematic review of sleeve gastrectomy as staging and primary bariatric procedure. Surg Obes Relat Dis. 2009;5(4):469–75.
12. Mickevicius A, Sufi P, Heath D. Factors predicting the occurrence of a gastrojejunal anastomosis leak following gastric bypass. Wideochir Inne Tech Maloinwazyjne. 2014;9(3):436–40.
13. Kim J, et al. ASMBS position statement on prevention, detection, and treatment of gastrointestinal leak after gastric bypass and sleeve gastrectomy, including the roles of imaging, surgical exploration, and nonoperative management. Surg Obes Relat Dis. 2015;11(4):739–48.
14. Varban OA, et al. Technique or technology? Evaluating leaks after gastric bypass. Surg Obes Relat Dis. 2016;12(2):264–72.
15. Livingston EH, Huerta S, Arthur D, Lee S, De Shields S, Heber D. Male gender is a predictor of morbidity and age a predictor of mortality for patients undergoing gastric bypass surgery. Ann Surg. 2002;236:576.
16. Arteaga-Gonzalez I, et al. Usefulness of clinical signs and diagnostic tests for suspected leaks in bariatric surgery. Obes Surg. 2015;25(9):1680–4.
17. El-Kadre L, et al. Overcoming the learning curve of laparoscopic Roux-en-Y gastric bypass: a 12-year experience. Surg Obes Relat Dis. 2013;9(6):867–72.
18. Schauer P, et al. The learning curve for laparoscopic Roux-en-Y gastric bypass is 100 cases. Surg Endosc. 2003;17(2):212–5.
19. DeMaria EJ, et al. Results of 281 consecutive total laparoscopic Roux-en-Y gastric bypasses to treat morbid obesity. Ann Surg. 2002;235(5):640–5. discussion 645–7
20. Brethauer SA. Sleeve gastrectomy. Surg Clin North Am. 2011;91(6):1265–79. ix
21. Kolakowski S, Kirkland M, Schuricht A. Routine postoperative upper gastrointestinal series after Roux-en-Y gastric bypass: determination of whether it is necessary. Arch Surg. 2007;142(10):930–4.
22. Guerron AD, Portenier DD. Patient selection and surgical management of high-risk patients with morbid obesity. Surg Clin North Am. 2016;96(4):743–62.
23. Gonzalez R, et al. Diagnosis and contemporary management of anastomotic leaks after gastric bypass for obesity. J Am Coll Surg. 2007;204(1):47–55.

24. Schiesser M, et al. Successful endoscopic management of gastrointestinal leakages after laparoscopic Roux-en-Y gastric bypass surgery. Dig Surg. 2014;31(1):67–70.
25. Joo MK. Endoscopic approach for major complications of bariatric surgery. Clin Endosc. 2017;50(1):31–41.
26. Chang J, et al. Endoscopic stents in the management of anastomotic complications after foregut surgery: new applications and techniques. Surg Obes Relat Dis. 2016;12(7):1373–81.

Gastro-Gastric Fistula Following Gastric Bypass

7

Giulio Giambartolomei, Emanuele Lo Menzo, Samuel Szomstein, and Raul Rosenthal

Introduction

Morbid obesity is a worldwide epidemic. It is well established that surgery is the most effective cure for obesity and its comorbidity. Consequently the number of such procedures has exponentially increased, as has surgeons' familiarity with bariatric procedures and with their related complications commensurately. The increased familiarity with such complications has actually led to their progressive reduction or to changes in techniques in order to avoid complications. In specific instances, procedures that led to high numbers of such complications have been completely abandoned, such as the vertical banded gastroplasty (VBG), or nearly abandoned, such as the laparoscopic adjustable gastric banding (LAGB). In fact, the VBG resulted in 20–65% of complications, or long-term failure rates, with complications including gastric distention, uncontrollable vomiting, leaks, obstruction, staple line disruption, GERD, and insufficient weight loss needing revisional intervention [1]. The LAGB, on the other hand, experienced a quick peak in popularity due to its relatively technical simplicity and potential reversibility; however, over time, it became evident that up to 50% of patients might require reoperation or removal of the band due to either complications, insufficient weight loss, or weight regain [2].

The described background explains how procedures like laparoscopic Roux-en-Y gastric bypass (LRYGB) and laparoscopic sleeve gastrectomy (LSG) gained popularity, both for their more favorable short- and long-term outcomes and their relatively low rates of complications. Obviously, complications remain an inevitable fact of any surgical intervention, and with the exponential increase in number of such procedures, surgeons had to increase their expertise in facing such complications, as well as improving their techniques, in order to avoid them.

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The purpose of this chapter is to define and discuss a potential complication that might occur after gastric bypass, specifically the gastro-gastric fistula, and scope here includes its management from diagnosis to treatment.

Definition and Pathogenesis

A gastro-gastric fistula (GGF) is an abnormal communication between the excluded gastric pouch and the gastric remnant after LRYGB. Several hypotheses exist regarding the formation of GGF. The more obvious of the determining factors of a GGF is the incomplete separation of the gastric pouch from the gastric remnant. This factor can be secondary to surgeon's inexperience, difficult anatomy, or technical problems (staple misfires). Other technical aspects of the procedure can also lead to a higher likelihood of GGF. In fact, Capella et al. in 1999 demonstrated a significantly higher incidence of GGF after nondivided gastric bypasses. In particular, they showed an incidence of 49% of GGF in the subgroup of nondivided or partially divided LRYGBs, compared with an incidence of 2.6% in the divided technique ($p < 0.0001$). Furthermore, they proposed that interposing a jejunal limb between the pouch and the remnant resulted in additional protection against the formation of a fistula, as the incidence after 492 cases was 0% [3].

Patients with particularly problematic habitus, such as male gender and higher body mass index (BMI), are known to pose challenges of visualization of the gastroesophageal (GE) junction, resulting in higher risk to develop this kind of fistula from incomplete division of the fundus of the stomach. In addition, the accidental presence of adipose tissue within the closed stapler could result in an incomplete resection as well.

Based on the abovementioned reports, the technique for LRYGB evolved from a stapled but nondivided to a stapled and divided pouch. However, a study from Cucchi et al. showed that the incidence of GGF remained substantial (6%) in spite of the divided technique, suggesting additional etiologies for GGF, such as abscess formation after a leak at the gastrojejunal anastomosis [4].

In order to understand the pathophysiology of a fistula, we need to review some basic concepts of general surgery that could explain its development. A fistula could originate from a chronic evolution of an abscess, as the inflammatory capsule of the abscess can erode into adjacent tissues and finally drains itself into a space other than the original, whether being the peritoneal cavity, another hollow organ, or outside the skin. This abnormal connection can be classified as blind, complete, or complex, according to its extension, complexity of tract through the organs, or whether it extends to the skin. Usually the chronic process moves toward loose tissues or follows the direction of existing forces, like gravity or peristaltic movements. Consequently, it has been hypothesized that a leak through a surgical suture, like the gastrojejunal anastomosis or the remnant stump, could evolve into an abscess secondary to the presence of acidic fluid, the inflammatory response, and

the superinfection by common bacteria. If the fluid collection reaches and erodes through the gastric serosa, it can evacuate inside the gastric remnant, resulting in a fistula. The clinical manifestation depends on the degree of infection and varies from completely asymptomatic to frank sepsis. This scenario is described by Cucchi et al. who reported signs and symptoms of early localized sepsis with postoperative fever and tachycardia in all of their six patients with a GGF, in spite of negative routine contrast studies [4].

However, the clinical presentation is not always abrupt and can develop over weeks with moderate symptoms. This can be explained by an internal decompression via the gastric remnant of an abscess that has caused a breakdown in staple lines without irritating the peritoneal serosa [5].

Based on this principle of GGF formation, all patient-related risk factors for poor blood supply and tissue healing at the staple lines could increase the incidence of leaks at the anastomosis despite a thorough technique. These commonly known risk factors, such as diabetes, smoking status, steroid use, and hepatic disease with hypoalbuminemia, often coexist in the bariatric population and could have a considerable impact on outcomes.

More rarely, technical aspects related to the surgical staplers can also play a role in the formation of GGF. These includes staple misfires, wrong choice of staple height with failure of staples to penetrate the gastric tissue, which can potentially result in a leak and consequently to a fistula.

Among other hypothesized etiologies of GGF, there is also the formation of a marginal ulceration. The incidence of marginal ulcers after LRYGB has been reported, ranging between 0.6% and 16% [6], and the coexistence between a marginal ulcer and a GGF has been described in up to 52% [7]. This close association sparked some controversy regarding whether a marginal ulcer is a risk factor for a fistula formation or vice versa. A break in gastric mucosa's continuity due to prolonged exposition to an acidic environment could predispose to a fistula formation, but conversely the presence of a fistula surely exposes the gastric pouch to low pH fluids originating from the gastric remnant.

Other proposed risk factors in the development of a fistula are gastric tissue migration, foreign body erosion, and anastomotic ischemia.

The ability of the gastric tissue to migrate and attach to other surfaces is well described; thus it is possible that gastric mucosa cells could spill out of the anastomosis and reach the gastric remnant through serosal attachments leading to a fistula formation.

Foreign bodies like preanastomotic rings and buttress material are potentially capable of eroding into the gastric wall and facilitating a pathological communication between the pouch and the remnant.

Finally, a crucial role is played by excessive tension in performing the anastomosis, as an under-tension suture is susceptible to ischemia and consequently to rupture and leak.

The discussed etiologic factors for GGF formation are summarized in Table 7.1.

Table 7.1 Etiology of gastro-gastric fistula (From Carrodeguas et al.)

Iatrogenic
Failure of complete gastric resection
Inadequate visualization during apical transection of stomach
Presence of perigastric fat included in transected tissue
Anastomotic leaks
Failure of staples to penetrate and anchor to gastric tissue properly despite gastric wall division
Technical nature of procedure
Failure of staple line to divide the stomach completely
Gastric wall tissue migration
Ability of gastric wall to heal and reattach to excluded stomach
Marginal ulceration and perforation
Presence of acid-secreting cells in pouch – secondary ulceration/perforation
Tissue injury
Anastomotic ischemia due to excessive tension during suturing anastomosis
Foreign body erosion

Diagnosis

Clinical

The diagnosis of GGF could be challenging because of the clinical presentation that varies depending on the etiology, the patient's objective and subjective response, the time of onset, the medications administered, and the imaging sensitivity.

In the series from Cucchi et al. the typical symptoms of a leak and abscess formation are reported in six out of six patients. In fact, in the early postoperative period, fever, tachycardia, and abdominal pain were present and supported the pathogenic hypothesis of a leak [4].

On the contrary, Carrodeguas et al. reported non-specific symptoms including nausea, vomiting, and epigastric pain, presenting after a variable time from 3 to 384 days (average 80 days) in 80% of patients [8]. The same author also showed that in 26.7% of the patients, a gastrojejunal anastomosis leak was previously diagnosed and treated, and consequently the diagnosis of GGF was achieved earlier (average 25 days) [8].

Yao et al. reported pain in 42% of the patients, nausea/vomiting in 21%, and the presence of an ulcer in 21% of the patients later diagnosed with GGF [9].

Nevertheless, for a considerable number of patients, the principal sign leading to the diagnosis of GGF was insufficient weight loss or weight regain. This percentage varies from 26.7% to 64%.

In the latter situation, since no additional symptoms were present, the diagnosis was achieved later (range from 17.1 to 19.8 months) [8, 9].

In another series the major complaint was epigastric pain (78%) followed by weight regain (44%) and gastrointestinal bleeding (11%) [10].

As demonstrated by O'Brien et al., another possible presentation of GGF could be the relapse of diabetes after its initial remission. It is well described, in fact, that the bypass of the duodenum and an accelerated transit of nutrients through the distal intestine enhance the release of peptide YY and GLP-1, improving insulin release and decreasing insulin resistance early in the postoperative course. Therefore, in the absence of other previously described symptoms, a relapse of diabetes, often concurrent with weight regain, reveals a restored transit of nutrients through the physiological route [11].

Following this concept, also a loss of food-intake restriction could be a hint of the presence of a GGF from a mechanical standpoint. Indeed, a communication between the gastric pouch and the gastric remnant enlarge the volume capacity available for food intake leading to weight regain.

As previously described, a marginal ulceration is often concurrent, and it will clinically result in epigastric pain.

Finally, a significant number of patients can be asymptomatic, and the true incidence of GGF could be underestimated, as suggested by case reports in which patients were incidentally diagnosed in spite of achieving a satisfying and sustained weight loss [12].

Imaging Studies

As discussed, the clinical presentation can be extremely variable, and further imaging investigations are often attained to confirm the clinical suspicion. Routine postoperative upper GI series (UGI) are often performed in many centers, but the sensitivity of this exam is hard to define given the large variability of the condition to examine.

It is however the first choice to investigate a symptomatic patient with a history of LRYGB.

Corcelles et al. described a sensitivity of 70% in detecting a GGF in patients readmitted for suspicious symptoms; however, the results were confirmed by an endoscopy [13].

Lee et al. discussed the impact of a selective use of upper GI studies after LRYGB and showed that morbidity and mortality were not adversely affected when a radiologic analysis is ordered after clinical suspicion driven by symptoms and surgeon's experience. They also advocated a routine sample of drain amylase as an adjunct tool to detect the presence of a leak with a sensitivity of 100% [14].

In a review from Quartararo et al. comprising 15,022 postoperative routine upper GIs after LRYGB, a sensitivity of 78% for leakage was found [15].

However, as a GGF may have other etiology than a leak, we cannot assume the sensitivity of an upper GI study being equal in detecting a leak and a GGF.

Another important consideration is timing, since the period of development of a GGF can vary depending on the mechanism of formation. This can partially explain

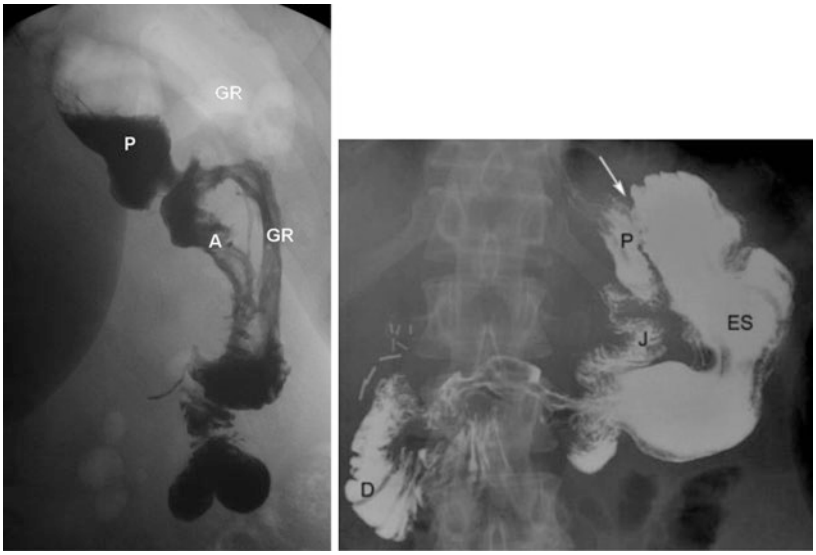

Fig. 7.1 Upper GI study demonstrating oral contrast in the gastric remnant through a gastro-gastric fistula; *P* pouch; *A/J* alimentary limb, *GR/ES* gastric remnant

the immediate positive postoperative finding occurring after an incompletely divided remnant stomach, compared to a late onset following a chronic ulceration. A different sensitivity is also found depending on the type of contrast used for the upper GI study. In fact, in the immediate postoperative period, a water-soluble oral contrast (Gastrografin®) is usually used as a potential leak will result in barium-related peritonitis. However, barium sulfate presents a slightly higher sensitivity for small leaks as compared to Gastrografin®, justifying its use in a later clinical scenario in which a GGF is most likely to present.

Ribeiro-Parenti et al. described a 100% rate of confirmation of diagnosis by UGI studies in their nine-case series [10].

Carrodeguas et al. finally showed that up to 80% of GGF may be diagnosed with an UGI study and endoscopy together, suggesting that a single study will not ensure a thorough evaluation [8] (Fig. 7.1). It is paramount to perform the UGI series in various patient positions, including supine. In fact, some of these fistulae are posterior and a standard upright UGI might miss the GGF.

Furthermore, as already discussed, GGF may be asymptomatic in patients with good weight loss results, thus discouraging the physician pursuing UGI. Consequently, the actual incidence of GGF might be underestimated and the radiological sensitivity biased.

Nevertheless, as advised by Huang et al. a barium UGI study is useful when performed in conjunction to an upper endoscopy, in the evaluation of patients with symptoms after LRYGB. They also stated that UGI provides important anatomical information that may be helpful to endoscopists not familiar with this patient population [16].

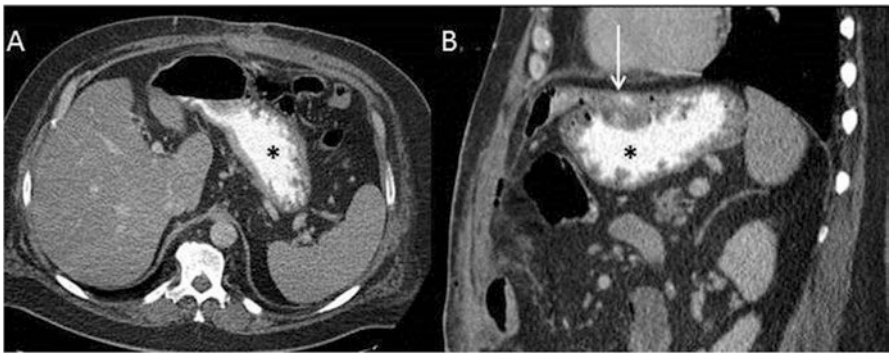

Fig. 7.2 Axial (**a**) and sagittal (**b**) CT scans demonstrating oral contrast and air-fluid levels within the gastric remnant (*asterisk*). The *arrow points* the fistulous tract

It is advisable then to associate this first level study with another, either being an endoscopy or a computed tomography (CT).

The literature is sparse regarding the use of a CT scan for the diagnosis of GGF. In general, a CT scan is used as a confirmatory tool with a high rate of success after a suspected diagnosis. The key elements of a CT scan in the diagnosis of GGF are either the presence of oral contrast into the excluded stomach or the direct demonstration of the fistulous tract, which can also be useful in defining the anatomy for an eventual surgical treatment (Fig. 7.2).

What appears to be mandatory is an esophagogastroduodenoscopy (EGD) evaluation, which is usually performed whenever a bariatric patient presents with upper GI symptoms. EGD allows for direct visualization of the defect, defines the position and the extent of the defect, and establishes the potential for successful endoscopic treatment, as discussed later (Fig. 7.3).

Although some small hidden fistulae could be missed if a thorough inspection is not performed, an experienced endoscopist may take advantage of probing all the mucosal folds with a sphincterotome or add fluoroscopy to indirectly visualize the presence of air or radiologic contrast in the excluded stomach [5]. Additionally, an upper endoscopy can usually detect a concomitant marginal ulcer, which is often not visible with an upper GI series, and subsequently guides the medical and surgical treatment.

The sensitivity of upper GI series and endoscopy together in diagnosis of a GGF is reported by Carrodeguas et al. to be 80%, whereas Corcelles et al. reported a 72.2% for endoscopy alone [8, 13].

In conclusion, the diagnosis of a GGF after LRYGB might be challenging, due to the extreme variability of the symptoms, the heterogeneity of etiologies, the time of presentation, and the relatively low sensitivity of diagnostic tools. Therefore, the diagnosis of GGF should be driven by a solid clinical judgment.

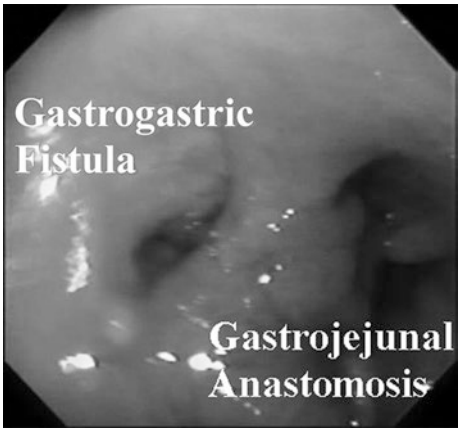

Fig. 7.3 Endoscopic view of a gastro-gastric fistula

Management

The management of GGF has been described in the several few case series published in the literature, due to its relatively low incidence. However, some algorithms have been proposed, and different interventional approaches have been described.

The management of GGF can be observational, medical, surgical, or endoscopic.

Whenever the GGF is completely asymptomatic and incidentally found, observation is considered to be the best option.

Medical

Medical treatment is basically dependent on symptoms and on endoscopic findings. The clinical presentation should dictate the generic therapy in order to stabilize the patient and make him/her suitable for a possible surgery.

As already discussed, the presenting symptoms are unspecific and can vary from sepsis to mild abdominal pain. Medical therapy should be instituted based on main symptoms, comprising pain control, antiemetic, antibiotics if there is a suspected infection, and intravenous (IV) resuscitation or nutrition whenever an indication is present.

The endoscopic findings are then crucial for the decision to institute gastroprotective therapy. Given the close association between a GGF and a marginal ulcer, the latter must be investigated by endoscopy in order to reinforce the gastroprotective therapy. As proposed by Carrodeguas et al., if a GGF fistula is found, medical therapy with full-dose protein pump inhibitors (PPI) should be initiated, in order to reduce the acid production from G cells of the remnant stomach that could

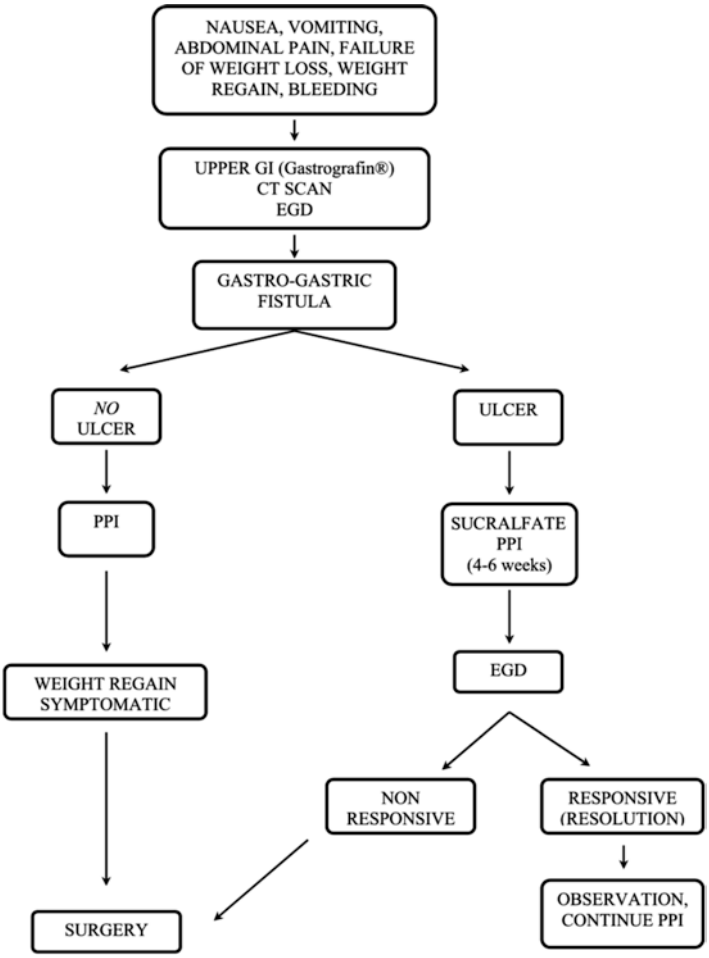

Fig. 7.4 Management algorithm for treatment of gastro-gastric fistula (permission not requested)

lead to development of a marginal ulcer through the fistula. If an ulcer is diagnosed simultaneously, sucralfate should be administered in order to create a protective layer to the pouch and the small bowel mucosa. Patients should be then reevaluated after 4–6 weeks to be reassessed and eventually elected to surgery (Fig. 7.4) [8].

Gumbs et al. reported a case of complete resolution of a GGF associated with a marginal ulcer after 6 weeks of medical therapy alone [17].

It is also advisable to test patients who tested positive for a marginal ulcer for the presence of *Helicobacter pylori* (*H. pylori*), in order to initiate proper antibiotic and gastro-protective therapy. After 3–4 weeks, patients should be reevaluated with endoscopy to assess for resolution of the ulcer and to discuss further treatment, whether observational or surgical.

Table 7.2 Indications for surgery

Indications for surgery
Medical therapy failure in resolution of defect and/or symptoms
Persistence of symptoms
Weight regain or insufficient weight loss
Gastrointestinal bleeding if associated with a marginal ulcer
Defect is deemed too significant to heal with medical therapy

Although in some cases medical therapy could lead to a resolution of small fistulae and to remission of symptoms with an acceptable weight loss, chronic PPI treatment is associated with important adverse effects, like vitamin B12 deficiency, which might be exacerbated by a malabsorptive mechanism following LRYGB [18] (Table 7.2).

Surgical

There is no standardized surgical procedure to treat a GGF, since the clinical scenario may vary depending on the time of diagnosis, the etiology, the association to marginal ulcer or a foreign body, and the clinical conditions of the patient. Also, factors such as the technique initially utilized (open or laparoscopic) or the experience of the surgeon may play a role in the timing and type of intervention. The decision to perform this kind of revisional operation laparoscopically or not is based on the individual surgeon's skills and experience. The case series published report a preference of the laparoscopic approach, except for patients who underwent the primary intervention in an open fashion. Also, Filho et al. stated that a laparoscopic approach is easier when performed in the acute postoperative course, but still reasonable in chronic GGF, even when a disrupted anatomy is due to the expected inflammation [19].

Ribeiro-Parenti et al. accomplished 87.5% of the revisions ($n = 9$) laparoscopically and reported no deaths. They also reported one postoperative leak (12.5%) that required reoperation.

The authors also proposed a simple classification of GGF based on the location and consequent involvement of the gastrojejunal anastomosis that might be helpful in guiding the surgical approach. They classified a type 1 fistula if it is found >2 cm above the anastomosis and type 2 if <2 cm from the anastomosis. The 2 cm cutoff was chosen because it is a reasonable distance that allows firing of the stapler vertically to the pouch and transection of the fistulous tract without involving or narrowing the anastomosis. In their technique, the remnant was also resected laterally to the fistula. The type 2 fistula was instead approached with a complete resection of the previous anastomosis and a subsequent gastrojejunal anastomosis [10].

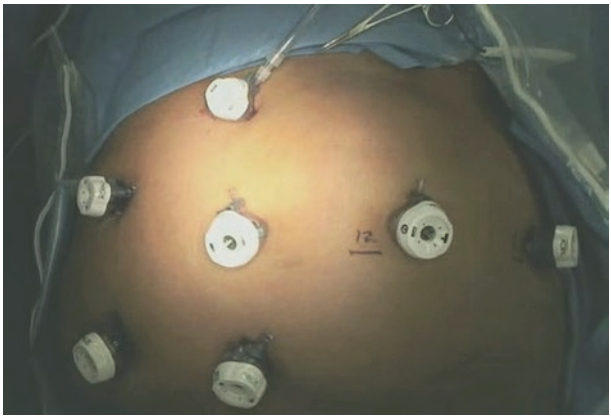

Fig. 7.5 Trocar placement (permission not requested)

Salimath et al. converted to open surgery 2 patients out of 22 that primarily underwent laparoscopic GGF repair with remnant gastrectomy.

Another option is to proceed with a remnant gastrectomy. The laparoscopic remnant gastrectomy technique is described as follows.

With the patient placed in a standard supine position and a seven-trocar approach (Fig. 7.5), the first step is to define the anatomy by lysing the adhesions between the liver, gastric pouch, gastric remnant, and alimentary limb. Then the short gastric vessels are dissected to mobilize the gastric remnant at the level of the GE junction. Intraoperative endoscopy is used to better identify the fistula, and a 32 Fr gastric lavage tube is placed to identify the GE junction and the anastomosis. A window is created between the gastric pouch and the gastric remnant to allow positioning of a linear stapler and transecting transversally the remnant at the level of the antrum (Fig. 7.6). If the pouch is sufficiently enlarged, it could be directly trimmed by means of a linear stapler in order to complete the remnant gastrectomy (Fig. 7.7). If this maneuver is too risky for the anastomosis, the gastric remnant is excised, leaving a margin of remnant tissue attached to the GGF side, and secondarily oversewn (Fig. 7.8). Finally, the gastric remnant is extracted, endoscopic and methylene blue tests are performed, and drains are left closed to both pouch and antrum. Outcomes were comparable to those shown after bariatric revision interventions, but neither mortalities nor recurrence of the fistula was reported [20].

In the series reported from Corcelles et al. 19.5% out of 36 patients required a conversion to open surgery, and 80.5% underwent revision of the gastrojejunal anastomosis, leading to a significant increase of overall postoperative complications compared to those who received a remnant gastrectomy (19.5%, $p = 0.01$) [13].

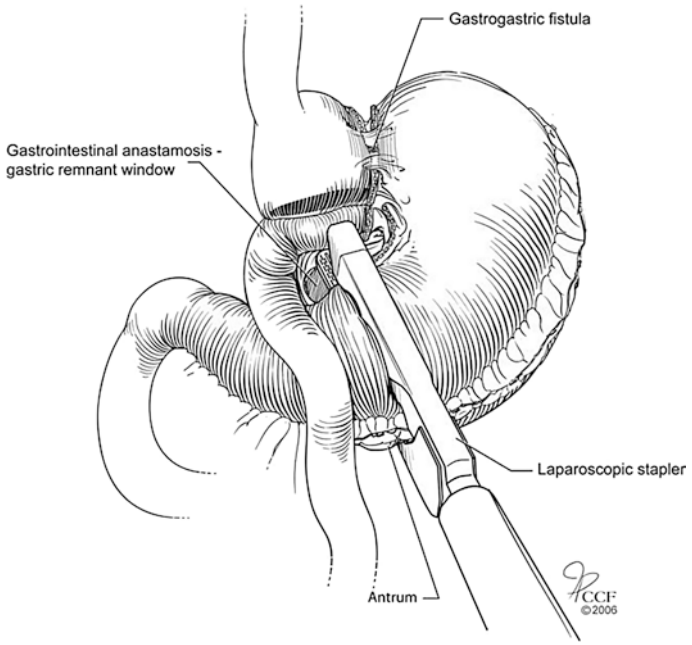

Fig. 7.6 Transection of the remnant stomach at the level of the antrum (permission not requested)

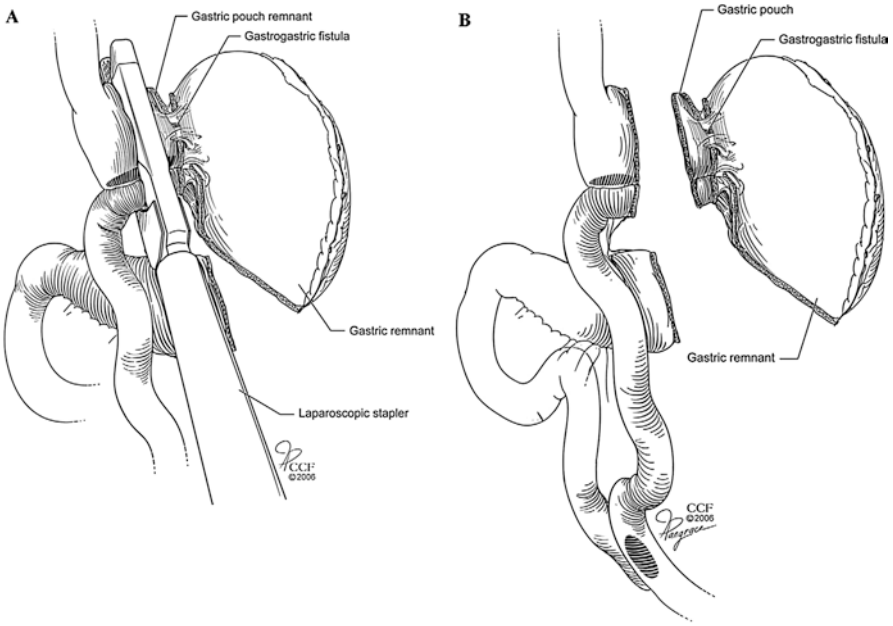

Fig. 7.7 (**a**) Trimming of the pouch (**b**) resection of the gastric remnant (permission not requested)

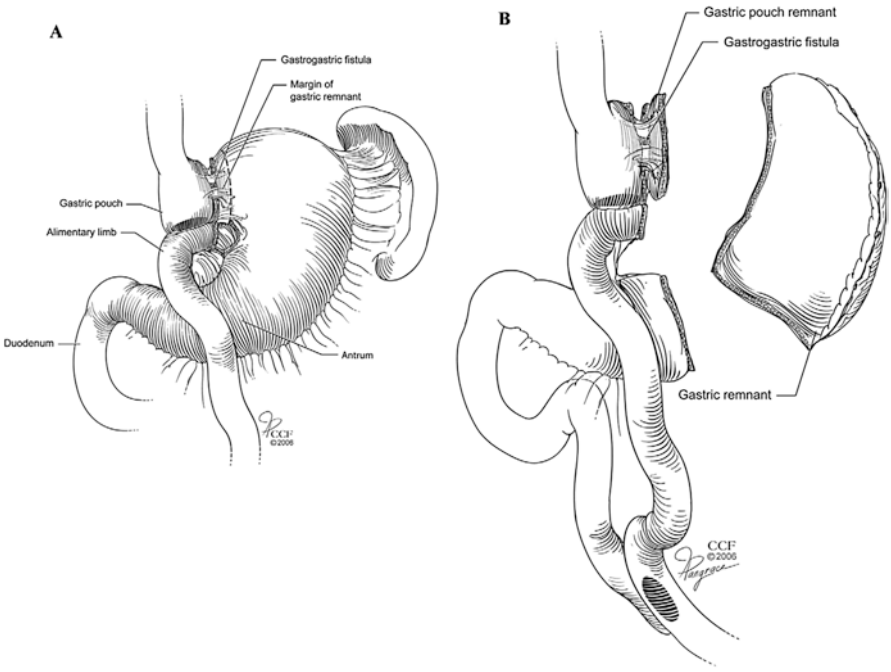

Fig. 7.8 (**a**, **b**) Transection of the gastric remnant leaving a small margin of remnant tissue attached to the gastro-gastric fistula side (permission not requested)

Endoscopic

Recently, the endoscopic approach for bariatric surgery complications has gained increasing interest, probably due to a sum of factors like increasing endoscopic expertise, technological development, and increasing number of bariatric surgeries and related complications. Nonetheless, there are still some concerns regarding this approach because of its relatively poor long-term outcomes, despite promising low complication rates having been described.

Fernandez-Esparrach et al. described a case series of 95 patients diagnosed with post-LRYGB GGF. They highlighted a 95% initial success rate in complete closure of the fistula, but also a 65% rate of reopening after a mean interval of 177 days. Some patients were endoscopically treated again, and after a median time of 217 days, 81% of patients still presented with a GGF. They advocated that the size of the fistula may play an important role in foreseeing endoscopic failure, as they found a higher rate of success in fistulae <10 mm, namely, 32%, at the end of the follow-up period. On the other hand, they encountered an acceptable 2.1% rate of complications that did not require surgical intervention [21].

Bhardwaj et al. reported a small case series of eight patients with a success rate of 50% after 8–46 months of follow-up with no complications [22].

Flicker et al. reported a group of 22 patients who underwent at least one endoscopic attempt of fistula closure before going to surgery and compared it with 13 patients who directly underwent revisional surgery for GGF. They showed no significant differences in minor and major complications after endoscopy or surgery, but endoscopy revealed a non-encouraging 9.1% of minor and 31.8% of major complications. All the patients treated endoscopically underwent surgery anyway, with no underlying fistula closure by endoscopic treatment alone [23].

Finally Niland et al. published their results with 14 patients treated with an over the scope clip technique and showed a 50% initial success rate and a 33% success rate after 6 months, again with no complications reported [24].

Based on these results, it appears that endoscopic treatment should be considered in carefully selected patients, with minor symptoms and with small defects (<10 mm) that are likely to close and in addition to medical therapy. Surgical treatment, although undermined by a considerable risk of complication, has shown to be more definitive and therefore remains the preferred approach in the treatment of success rate. The technologic drive will surely make more effective instruments available for either surgical or endoscopic approach.

References

1. Switzer NJ, Karmali S, Gill RS, Sherman V. Revisional bariatric surgery. Surg Clin North Am. 2016;96(4):827–42.
2. Elnahas A, Graybiel K, Farrokhyar F, Gmora S, Anvari M, Hong D. Revisional surgery after failed laparoscopic adjustable gastric banding: a systematic review. Surg Endosc. 2013;27(3):740–5.
3. Capella JF, Capella RF. Gastro-gastric fistulas and marginal ulcers in gastric bypass procedures for weight reduction. Obes Surg. 1999;9(1):22–7. discussion 8
4. Cucchi SG, Pories WJ, MacDonald KG, Morgan EJ. Gastrogastric fistulas. A complication of divided gastric bypass surgery. Ann Surg. 1995;221(4):387–91.
5. Pauli EM, Beshir H, Mathew A. Gastrogastric fistulae following gastric bypass surgery-clinical recognition and treatment. Curr Gastroenterol Rep. 2014;16(9):405.
6. Coblijn UK, Lagarde SM, de Castro SMM, Kuiken SD, van Wagensveld BA. Symptomatic marginal ulcer disease after Roux-en-Y gastric bypass: incidence, risk factors and management. Obes Surg. 2015;25(5):805–11.
7. Cho M, Kaidar-Person O, Szomstein S, Rosenthal RJ. Laparoscopic remnant gastrectomy: a novel approach to gastrogastric fistula after Roux-en-Y gastric bypass for morbid obesity. J Am Coll Surg. 2007;204(4):617–24.
8. Carrodeguas L, Szomstein S, Soto F, et al. Management of gastrogastric fistulas after divided Roux-en-Y gastric bypass surgery for morbid obesity: analysis of 1,292 consecutive patients and review of literature. Surg Obes Relat Dis. 2005;1(5):467–74.
9. Yao DC, Stellato TA, Schuster MM, Graf KN, Hallowell PT. Gastrogastric fistula following Roux-en-Y bypass is attributed to both surgical technique and experience. Am J Surg. 2010;199(3):382–5. discussion 5–6
10. Ribeiro-Parenti L, De Courville G, Daikha A, Arapis K, Chosidow D, Marmuse JP. Classification, surgical management and outcomes of patients with gastrogastric fistula after Roux-En-Y gastric bypass. Surg Obes Relat Dis. 2016;
11. O'Brien CS, Wang G, McGinty J, et al. Effects of gastrogastric fistula repair on weight loss and gut hormone levels. Obes Surg. 2013;23(8):1294–301.

12. Gustavsson S, Sundbom M. Excellent weight result after Roux-en-Y gastric bypass in spite of gastro-gastric fistula. Obes Surg. 2003;13(3):457–9.
13. Corcelles R, Jamal MH, Daigle CR, Rogula T, Brethauer SA, Schauer PR. Surgical management of gastrogastric fistula. Surg Obes Relat Dis. 2015;11(6):1227–32.
14. Lee SD, Khouzam MN, Kellum JM, et al. Selective, versus routine, upper gastrointestinal series leads to equal morbidity and reduced hospital stay in laparoscopic gastric bypass patients. Surg Obes Relat Dis. 2007;3(4):413–6.
15. Quartararo G, Facchiano E, Scaringi S, Liscia G, Lucchese M. Upper gastrointestinal series after Roux-en-Y gastric bypass for morbid obesity: effectiveness in leakage detection. A systematic review of the literature. Obes Surg. 2014;24(7):1096–101.
16. Huang CS, Forse RA, Jacobson BC, Farraye FA. Endoscopic findings and their clinical correlations in patients with symptoms after gastric bypass surgery. Gastrointest Endosc. 2003;58(6):859–66.
17. Gumbs AA, Duffy AJ, Bell RL. Management of gastrogastric fistula after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2006;2(2):117–21.
18. Sheen E, Triadafilopoulos G. Adverse effects of long-term proton pump inhibitor therapy. Dig Dis Sci. 2011;56(4):931–50.
19. Filho AJ, Kondo W, Nassif LS, Garcia MJ, Tirapelle Rde A, Dotti CM. Gastrogastric fistula: a possible complication of Roux-en-Y gastric bypass. JSLS. 2006;10(3):326–31.
20. Salimath J, Rosenthal RJ, Szomstein S. Laparoscopic remnant gastrectomy as a novel approach for treatment of gastrogastric fistula. Surg Endosc. 2009;23(11):2591–5.
21. Fernandez-Esparrach G, Lautz DB, Thompson CC. Endoscopic repair of gastrogastric fistula after Roux-en-Y gastric bypass: a less-invasive approach. Surg Obes Relat Dis. 2010;6(3):282–8.
22. Bhardwaj A, Cooney RN, Wehrman A, Rogers AM, Mathew A. Endoscopic repair of small symptomatic gastrogastric fistulas after gastric bypass surgery: a single center experience. Obes Surg. 2010;20(8):1090–5.
23. Flicker MS, Lautz DB, Thompson CC. Endoscopic management of gastrogastric fistulae does not increase complications at bariatric revision surgery. J Gastrointest Surg. 2011;15(10):1736–42.
24. Niland B, Brock A. Over-the-scope clip for endoscopic closure of gastrogastric fistulae. Surg Obes Relat Dis. 2017;13(1):15–20.

Hiatal Hernia and Reflux Following Bariatric Surgery

8

Patrick J. McLaren and Samer G. Mattar

Obesity in the United States is rising at alarming rates. More than one out of every three adults in the United States are now classified as obese [1]. Along with this rise in obesity, the field of bariatric surgery has rapidly expanded in recent years. From 1994 to 2004, the annual number of gastric bypass operations performed in the United States increased 20-fold [2]. Advances in laparoscopy have contributed to a major boom in popularity and changed the entire landscape of the field. In addition to laparoscopic Roux-en-Y gastric bypass (LRYGB), which has been the cornerstone of weight loss surgery for nearly two decades, newer operations are being performed with excellent outcomes. Laparoscopic adjustable gastric banding (LAGB) enjoyed popularity through the beginning of the 2000s but has been almost entirely supplanted in recent years by the laparoscopic sleeve gastrectomy (LSG, Fig. 8.1) [3]. LSG involves resection along the greater curvature removing the majority of the body and entire fundus of the stomach to create a smaller tubular stomach. LSG was initially introduced as a staged operation in patients undergoing a biliopancreatic diversion and duodenal switch operation but rapidly became a stand-alone bariatric procedure [4]. Recent analyses have demonstrated that LSG has weight loss and metabolic outcomes better than LAGB and approaching those of LRYGB [5]. Due to a lower complication and reoperation rate and the relative simplicity of the procedure compared to gastric bypass, LSG now accounts for the largest proportion of bariatric operations performed in the United States and worldwide [3, 5]. With changes in surgical technique come different morbidity and mortality profiles and perioperative management strategies. For the bariatric patient with gastroesophageal reflux disease (GERD), LRYGB can cure symptoms in the

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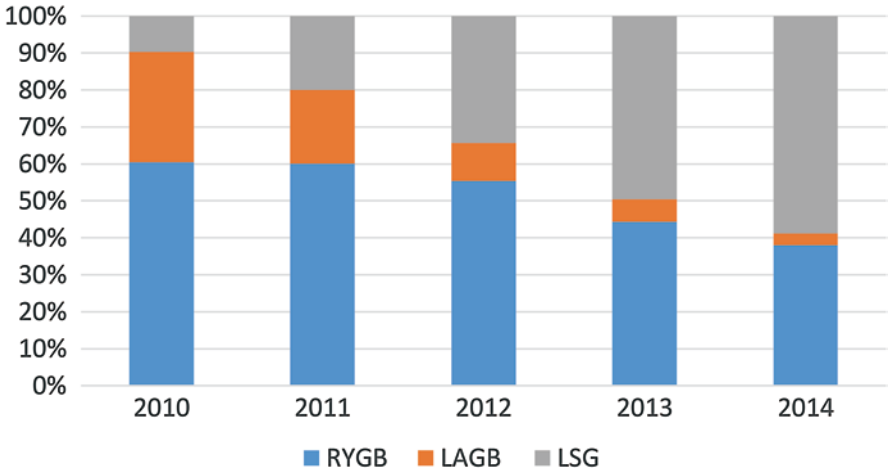

Fig. 8.1 Trends in utilization of different surgical approaches to bariatric surgery in the United States, 2010–2014 (Data from the American College of Surgeons National Surgical Quality Improvement Program database [3]). RYGB Roux-en-Y gastric bypass, LAGB laparoscopic gastric band, LSG laparoscopic sleeve gastrectomy

vast majority of patients [6]. LSG on the other hand, while improving pre-existing GERD in many patients, can be aggravated by postoperative reflux, even in patients who had not experienced symptoms prior to bariatric surgery, so-called "de novo" GERD. Additionally, hiatal hernias occur in as many as 50% of patients after LSG and are a significant contributor to postoperative reflux [7]. In this chapter, we will discuss the assessment and management options for patients with GERD and hiatal hernia in the pre- and postoperative setting.

Preoperative Assessment

The most important aspect of minimizing complications following bariatric surgery is identifying and mitigating patient risk factors preoperatively. In addition to bariatric-specific considerations, patients considering weight loss surgery should have an assessment the same as any major surgery. Patient selection for weight loss surgery should be based on a multidisciplinary team approach, which should include assessment by the patient's primary care provider, a nutritionist, a psychologist, and surgeon. The National Institutes of Health (NIH) criteria present a very broad framework of indications for surgery, so surgeons must use their judgment to select the best surgical option for each patient. Often time patients present to their surgeon having some knowledge or conducted independent research and have a preference for one weight loss operation versus another. For these patients, the surgeon must educate the patient about the expected outcomes, potential complications, and side effects associated with each specific procedure [8]. A review of medical

comorbidities should be undertaken, and two comorbidities that warrant specific considerations are GERD and hiatal hernia. These conditions may require further work-up in order to choose the right operation for each patient.

GERD is symptomatically present in approximately half of patients with severe obesity [9]. The bariatric preoperative assessment should include a thorough history and physical exam with direct questioning regarding GERD symptoms and history of antacid medication use. All bariatric patients with a known diagnosis of GERD, especially when severe, should undergo a preoperative screening endoscopy and be considered for other investigations such as esophogram, manometry, and 24 h pH monitoring. Some clinicians perform routine endoscopy on all patients being considered for bariatric surgery [10]. Upper endoscopy is a valuable tool to rule out gastritis, ulceration, Barrett's esophagus, dysplasia, and malignant lesions. Abnormal findings on endoscopy that change the surgical approach or delay the operation have been shown to occur in 12–60% of patients [11–13]. Since LRYGB excludes the majority of the stomach and all of the duodenum, postoperative endoscopic surveillance of the distal stomach and duodenum is significantly complicated and best performed prior to surgery.

The presence of a hiatal hernia is another valuable finding that can be identified on endoscopy that may alter the surgical approach. Hiatal hernia requiring concurrent repair has been shown to be present in 5–50% of patients undergoing preoperative endoscopy for bariatric surgery [14]. Several factors contribute to the development of hiatal hernias in obese patients, including increased intra-abdominal pressure which results in increased stress at the crura and esophageal shortening due to chronic GERD. There appears to be significant correlation between BMI, waist circumference, and axial separation of the lower esophageal sphincter (LES) and crura, resulting in decreased LES pressure, impaired esophageal clearance, and increased sensitivity to transient lower esophageal sphincter relaxation (TLESR) [15, 16].

In patients with questionable symptoms of GERD, consideration should be given to obtaining other tests like esophageal manometry and 24 h pH monitoring. While no definitive consensus exists on the role of manometry and pH monitoring prior to bariatric surgery, such studies can help delineate esophageal pathology that may be mistaken for GERD symptoms. In addition, 24 h pH monitoring provides clinicians with an objective measurement to rule in or out true gastric reflux.

The general consensus among experts is that LRYGB is the best surgical option for bariatric patients with GERD. Reflux symptoms are resolved in over 90% of patients after LRYGB [6]. Compared to LSG, LRYGB has been shown to be significantly better at improving symptoms of GERD postoperatively [6]. Furthermore, avoiding LSG in patients with preoperative severe GERD has been shown to reduce the need for future conversion to LRYGB [17]. It is important to note that there have been a number of series that have demonstrated improved rates of symptomatic GERD following LSG although not to the degree of improvement seen with LRYGB [18]. With newer treatment options for postoperative GERD, LSG may be a viable option in many patients.

Reflux Following Adjustable Gastric Band

Placement of the laparoscopic adjustable gastric band (LAGB) was a common weight loss operation until its gradual decline in recent years. There are numerous reasons for its demise, but the most important factor is probably related to patient and physician dissatisfaction with its need for frequent maintenance, modest weight loss profiles, and high incidence of long-term complications, resulting in the requirement for additional surgical procedures and/or device removals.

GERD is a common presenting symptom with most LAGB-related complications, which include prolapse or slippage, erosion, port or tubing complications, overfilling of the band, esophageal dilation, and weight loss failure.

Prolapse or slippage is the most common complication requiring reoperation. The underlying mechanism for this complication is advancement of gastric tissue from below the band lumen pushes up through the band circumference (prolapse) or, similarly, the band slips down on the stomach further than desired, resulting excessive stomach above the circumference of the band (slippage). The effect of both is similar in both instances: the excess tissue causes progressive or immediate food intolerance associated with heartburn. New onset of GERD symptoms in an LAGB patient strongly suggests prolapse, which should be ruled out.

Diagnosis of prolapse begins with the above clinical picture. A plain radiograph will usually show the band in an abnormally horizontal position. Barium swallow will show a significantly greater amount of stomach above the band than would be expected, confirming the prolapse. The prolapse can be anterior or posterior.

Esophageal dilation is perhaps one of the most severe complications that may result from an over-tight or malpositioned gastric band. This complication arises when the band position is too high, restricting the distal esophagus instead of the proximal stomach. The incidence is in the 1–2% range in most series. Reflux, dysphagia, pain, and food intolerance may be presenting symptoms. Resultant dilation of the esophagus occurs. Esophageal motor dysfunction may occur if the condition becomes long-standing. Treatment for the problem, once discovered, is to remove all fluid from the band, minimizing the restriction and obstruction. Hopefully this will reverse the dilation of the esophagus and restore function. Band repositioning may be needed to prevent recurrence of the problem.

Surgical Options for Malfunctioning Adjustable Gastric Bands

In view of the abovementioned unfavorable outcomes, many patients have undergone revisional surgery to address problems related to their adjustable gastric bands. The three main surgical options available for these patients are band repositioning, band replacement, or removal of the band with concurrent, or staged, performance of another metabolic operation.

It has become increasingly evident that displaced bands that are creating symptoms of dysphagia or GERD are best managed with their removal, rather than repositioning or replacement with another band. In an important study from the Netherlands, the authors were able to achieve an impressive follow-up time of 14 years on 99% of their patients. They reported that, of their 201 patients, 53% of them underwent band removal and/or were converted to a gastric bypass. Of the patients with retained bands, only 43 patients still had a functioning band. In general, there were 204 operations performed on 133 patients. Of the patients who still had their band in place, 51% were considered non-responders. Many of the patients who had band repositioning or replacement ultimately required conversion to gastric bypass. Based on this study, it appears that the most effective therapy for a malfunctioning band is conversion to a metabolic operation rather than retention or replacement of the band.

Reflux Following Roux-en-Y Gastric Bypass

Roux-en-Y gastric bypass is widely considered to the "ideal" anti-reflux procedure, especially in patients with a high BMI, and several studies have reported benefits on GERD, including symptom improvement and reduction in the use of anti-secretory medications. Underlying factors for these improvements include weight loss, reduction of parietal cell mass, and diversion of biliopancreatic and gastric secretions. However, although mostly efficacious, the RYGB has seldom been associated with 100% resolution of GERD. Most studies report 70–80% GERD resolution rates, implying that 20–30% of patients either continue to experience varying degrees of GERD or develop de novo GERD. This is particularly troubling when GERD symptoms rise to levels that are refractory to maximum-dose medical therapy, creating a therapeutic dilemma due to the inability to perform traditional anti-reflux fundoplication operations due to the unavailability of a contigious fundus. Potential reasons for post-RYGB GERD include an enlarged pouch, inhibition of G-cells, vagally mediated increased acid production, gastric outlet obstruction caused by an anastomotic stricture, or altered flow rates within the Roux limb due to kinks or adhesions. Other proposed mechanisms may be related to ineffective peristaltic waves in the Roux limb due to motor abnormalities of the Roux limb, such as inversion of slow-wave frequency gradient, retrograde slow-wave propagation, or the increased occurrence of ectopic migrating motor complexes [19].

In view of the unavailability of traditional anti-reflux operations for the treatment of refractory GERD after RYGB, several endoscopic therapeutic options have been used, with varying degrees of success and durability. Radio-frequency energy delivery to the LES has shown to be efficacious in improvement of both objective and subjective parameters in these patients, while other procedures, such as magnetic augmentation and electrical stimulation of the LES, are under investigation [20].

Gastrogastric Fistula

An important cause of progressively increasing acid reflux after gastric bypass, especially when associated with a current or past history of epigastric pain, is gastrogastric fistula. This lesion was particularly common in the era of undivided gastric bypass (usually due to a ruptured staple line), and although its incidence has markedly diminished with the adoption of the divided gastric bypass, it still remains occasionally encountered. Gastrogastric fistula (GGF), in the modern era, usually occurs due to a non-healing marginal or gastric ulcer that has penetrated into the excluded stomach. It is more common in patients with a smoking history or those who are predisposed to ulcer formation due to the ingestion of nonsteroidal anti-inflammatory agents. The incidence of GGF is 1.5–6%. Patients with GGF may present with non-specific symptoms including vomiting, pain, and hematemesis. They will also often complain of heartburn, regurgitation (especially when recumbent), halitosis, and occasional aspiration episodes. Less common presentation will be weight regain if the fistula enlarges enough to allow the food conduit to preferentially channel through the fistula rather than the gastrojejunostomy. In a recent review, the authors reported that the range of interval time of diagnosis of GGF after gastric bypass extended from 1 to 75 months, with epigastric pain being the most common presenting symptom. Upper GI contrast studies and/or CT scan with oral contrast confirmed the diagnosis in most cases, whereas endoscopy can be falsely negative in a certain number of cases. Initial treatment of GGF can be attempted with proton pump inhibitors, but most symptomatic lesions will require surgical resection, either in isolation or, if the GGF is in proximity to the gastrojejunostomy, with a resection/revision of the gastrojejunostomy. Smaller GGF lesions may be repaired endoscopically with the use of the Overstitch device [21, 22].

Bile Reflux

Single-anastomosis gastric bypass, which is also named “mini-gastric bypass” and “omega loop gastric bypass” (OLGB), was originally conceived by Rutledge and has been gaining popularity, especially outside the United States [23]. One of the barriers to its wider adoption in the United States has been the concern for the potential for bile reflux which may develop after this operation. Patients with bile reflux, whether after this operation or even after the standard RYGB, will present with dyspepsia, esophagitis, anastomotic ulcer, or stricture formation. Additionally, there is the serious concern that exposed esophageal epithelium may undergo metaplasia or dysplasia as precursors of esophageal adenocarcinoma. The treatment of choice in these situations has been, in the case of OLGB, to undergo conversion to RYGB [23]. In cases of bile reflux after RYGB, the fact that the Roux limb may be foreshortened should be contemplated and confirmed with either endoscopy or upper GI contrast radiography (or both). Once diagnosed, patient relief of biliary symptoms can be achieved by translocation of the biliopancreatic limb further downstream along the common alimentary channel.

Reflux Following Sleeve Gastrectomy

Since its introduction, LSG has quickly gained popularity among surgeons due to its high safety profile and short operative time. Additionally, the operation delivers excellent weight loss outcomes. Most estimates place the excess weight loss at 50–70% at 1-year follow-up [24, 25]. Resolution of obesity-related comorbidities has also been favorable in mid- and now long-term follow-up. *However, the operation has not been widely used in practice for long enough to determine the exact long-term complications*. A 2017 review of studies with more than 12 months follow-up identified 18 studies comparing GERD symptoms before and after LSG [18]. Of those studies, five demonstrated improvement, three showed no change, and ten studies demonstrated worsening of GERD symptoms after LSG [18]. Based on the available data, we are unable to confidently predict the effect of LSG on patients with existing GERD. Similarly, de novo GERD following LSG has also been shown to be highly variable ranging from 0% to 45% [26]. Rebecchi et al. conducted the only study to use pH monitoring to objectively measure acid exposure prior to and following LSG [26]. This study demonstrated a significant improvement in both DeMeester score and %pH<4 in patients who had pathologic reflux before surgery [27]. However, the same study noted a 5.4% increase in de novo pathologic esophageal acid exposure in patients without GERD before LSG [27].

A number of anatomic and physiologic mechanisms have been proposed to explain new onset GERD symptoms following LSG as well as explanations for improvement in GERD symptoms (Table 8.1) [28]. Proposed mechanisms for the increase in GERD after LSG include anatomic factors like blunting of the angle of His, stenosis of the gastric outlet, mid-gastric stenosis with proximal dilation, resection of gastric sling fibers of Helvetius, and hiatal hernia [27, 28]. Possible physiologic mechanisms to explain post-LSG reflux include reduced gastric compliance, increased intragastric pressure, decreased gastric emptying, and hypotensive lower esophageal sphincter [27, 28]. Opposing explanations for improved GERD symptoms following LSG include restoration of the angle of His, resection of acid-producing parietal cells, improved gastric emptying, and weight loss [27].

The effect that LSG has on the lower esophageal sphincter is also an area of controversy, with some studies showing decreased LES pressures following LSG, others showing no differences. Braghetto et al. conducted pre- and post-op manometry measures on 20 LSG patients, which demonstrated decrease in LES pressures in 85% of the cohort [29]. They concluded that LSG produces an important decrease in LES pressure, which can in turn cause the appearance of reflux symptoms and esophagitis after the operation [29]. Subsequent studies have been contradictory and found that increases in LES pressure were correlated with worsening symptoms of GERD [30]. Yet other studies showed no difference in pre- and postoperative LES pressures [27].

It is evident that the development of postoperative GERD is likely multifactorial, but most agree that surgical technique plays a significant role in the prevalence of postoperative GERD [26, 31–33]. The sleeve gastrectomy, essentially a longitudinal resection of the stomach along the lesser curvature, is an

Table 8.1 Proposed anatomic and physiologic factors affecting GERD following LSG [18, 27, 28]

Proposed anatomic and physiologic factors affecting GERD following LSG
Worsening GERD
Decreased gastric emptying
Lower LES pressure
Decreased gastric compliance and volume
Increased gastric pressure
Resection of the sling fibers of Helvetius
Blunting angle of His
Stenosis of the gastric outlet
Mid gastric stenosis with proximal distension
Increased gastric pressure
Improving GERD
Accelerated gastric emptying
Weight loss
Restoration of the angle of His
Reduced acid production due to resection of parietal cells
Removal of fundus (source of relaxation waves to lower esophageal sphincter)
Reduced wall tension

operation that is relatively simple in concept but intricate in technique. A variety of subtle maneuvers must be proficiently completed, in order to avoid a lifelong series of complications and misery to the patient. A consensus statement on best practices for surgical technique was proposed by the International Sleeve Gastrectomy Expert Panel in 2011 [26]. These guidelines are summarized in Table 8.2**.** Two specific technical aspects that have been proposed to reduce postoperative GERD are avoidance of mid-stomach stenosis and careful dissection and division of the stomach at gastroesophageal junction to avoid disruption of the angle of His [27].

The exact long-term incidence of GERD and other complications following LSG is not yet clearly defined, which is a current limitation of the operation. At the present time, LRYGB is the most effective method for both weight loss and GERD and should be the operation of choice given our current knowledge. However, as improvements in operative technique are made and new data becomes available, LSG may prove superior due to lower morbidity and mortality compared to LRYGB, even in patients with a known diagnosis of GERD. A preoperative diagnosis of GERD is not an absolute contraindication to performing LSG, and many series have shown excellent outcomes. However, surgeons must be aware of the potential of this postoperative complication and be prepared to manage this complication both medically and surgically.

Table 8.2 Consensus guidelines on surgical technique when performing LSG from the International Sleeve Gastrectomy Expert Panel. Coral Gables, FL 2011 [26]

Consensus guidelines for surgical technique when performing LSG	
Sleeve sizing	Optimal bougie size is 32–36 French
	Invagination of the staple line reduces lumen size
Stapling	It is not appropriate to use staples with a closed height less than 1.5 mm on any part of sleeve gastrectomy
	When using buttressing materials, surgeons should never use any staple with a closed height less than 2.0 mm
	When resecting the antrum, surgeons should never use any staple with closed height less than 2.0 mm
	Transection should begin 2–6 cm from the pylorus
	It is important to stay away from the GE junction with the last staple firing
Mobilization	It is important to completely mobilize the fundus before transection
Reinforcement	Staple line reinforcement will reduce bleeding along the staple line

Hiatal Hernia and Reflux

Hiatal hernia is a condition that warrants specific consideration for the bariatric surgeon. Symptomatic hiatal hernia is present in 15% of patients with a BMI greater than 35 [34] and is a known contributor to GERD symptoms in the preoperative setting. Increased intra-abdominal pressure from excess body mass is thought to be a major contributor to hiatal hernias seen in the bariatric population. The decision to repair a hiatal hernia at the time of bariatric surgery is a subject of some debate. Comparing LSG with hiatal hernia repair versus without, most studies fail to show a significant difference in the degree of GERD symptoms postoperatively [35, 36]. While the evidence is mixed on the topic, most surgeons would recommend concomitant repair of moderate to large hiatal hernias at the time of bariatric surgery [35]. If left unrepaired, symptomatic hiatal hernias may obscure more concerning postoperative conditions like staple line leak, ulceration, bowel obstruction, and internal or incisional hernias. Particularly in the post-LSG patient, postoperative hiatal hernias can pose a clinical dilemma, and for this reason, it is recommended that repair be undertaken at the time of bariatric surgery.

Interventions for Reflux After Sleeve Gastrectomy

The initial treatment for GERD symptoms following bariatric surgery should be medical. Proton pump inhibitors (PPIs) are effective in controlling symptoms in the majority of patients. However, GERD symptoms that are not controlled with PPIs pose a particular challenge in the LSG patient. Due to the altered anatomy of the GI tract following bariatric surgery, traditional surgical options like fundoplication are not an option. The most well-established method for treating GERD in this population is conversion to a LRYGB. There are however inherent risks with revision

bariatric surgery. Newer procedures like radio-frequency energy delivery to the LES, the "Stretta procedure" (Mederi Therapeutics, Greenwich, CT, United States) and magnetic augmentation of LES LINX® Reflux Management System (Torax Medical, Inc., Shoreview, MN, USA) offer additional options for treating these patients. Electrical stimulation therapy of the LES (EndoStim) is a new modality that involves implantation of electrodes to the sphincter, with their leads connecting to a stimulation device. This technology is not currently available in the United States, but Latin-American studies have shown its use has resulted in significant improvement in GERD-HRQL scores and median 24-h acid exposure with no adverse events. As with all endoscopic therapies, it is contraindicated in patients with Barrett's esophagus and in patients with hiatal hernias >3 cm [37].

Stretta was approved by the Food and Drug Administration in 2000 and is an endoscopic procedure that utilizes temperature-controlled radio-frequency energy that is endoscopically delivered to the GE junction through electrodes at the end of a catheter to the lower esophageal sphincter (LES) and the gastric cardia [38]. The procedure prevents transient relaxations of the LES and reduces compliance of the GE junction, helping to prevent GERD [38]. The energy heats the tissue, causing it to swell and stiffen and prevent reflux of gastric contents. The exact mechanism in which this procedure affects GERD is not well understood. It is thought that the heat causes local inflammation, collagen deposition, and muscular thickening of the LES. Furthermore, the procedure may disrupt the nerves at the GE junction contributing to the anti-reflux effect [38].

Stretta has been extensively studied in non-bariatric patients and has demonstrated to be safe and effective. Morbidity from the procedure has been shown to be less than 0.6% and can be performed as an outpatient procedure [38]. Furthermore, outcomes have been excellent, with 91.5% of patients demonstrating improvement in GERD symptoms [38]. Results from systematic review of the efficacy of Stretta showed improved quality of life measures and reduced the heartburn standardized score [39]. Stretta also reduced the need for PPI use post-procedurally and decreased incidence of erosive esophagitis by 24% [39]. Objective measures of LES pressure were increased, and the mean acid exposure decreased after the Stretta procedure [39]. No studies have yet investigated the use of Stretta in the LSG population for management of postoperative reflux, and there is no evidence that Stretta results in improved outcomes as compared to surgical intervention. However, the procedure is an endoscopic option in the post-bariatric surgery population for patients who wish to avoid additional surgery.

The LINX® Reflux Management System was approved for the treatment of GERD in March of 2012. The LINX device is a small flexible ring of linked titanium beads with magnetic cores. When placed around the LES, the magnetic attraction between the beads provides resistance to gastric pressures and is intended to prevent reflux from the stomach into the esophagus [40]. When swallowing, contractile forces break the magnetic bond, allowing food and liquid to pass normally into the stomach [40]. The LES is immediately closed after swallowing by the magnetic attraction of the beads [40]. The existing literature supports this device as a safe and effective option for the treatment of medically refractory GERD [41, 42]. The LINX

system offers potential treatment of GERD in postoperative LSG patients without the inherent malabsorptive effects of LRYGB. The LINX device has the advantages over conversion to LRYGB in that it is relatively simple to perform and does not alter the native configuration of the gastrointestinal track. However, until more studies are done with the device, it is still not determined if the benefits outweigh the potential long-term complications. The most common complication following LINX implantation is dysphagia occurring in as many as 43% of patients. However, the vast majority of dysphagia is self-limited, with only 5.6% of patients with requiring endoscopic dilation, and 2.2% of patients required device removal for dysphagia [42]. More serious and feared complications are far less common with an operative mortality and erosion of the device reportedly at less than 1% [42].

Literature is limited on the use of the LINX device after LSG, but small case series have shown favorable outcomes. A 2015 series of seven patients treated with the LINX device after LSG demonstrated that all patients had self-reported greatly improved gastroesophageal reflux symptoms 2–4 weeks after their procedure [43]. They were all noted to have statistically significant improved severity and frequency of their reflux, regurgitation, epigastric pain, sensation of fullness, dysphagia, and cough symptoms in their postoperative GERD symptoms compared with their preoperative evaluation [43]. It must be noted that follow-up in this series was short; however, a singular case report with one patient demonstrated similar findings of improved symptoms after LINX implantation with 12-month follow-up [44]. The LINX system shows promise for control of GERD in the LSG patient and can be considered a viable option in patients wishing to avoid LRYGB.

Finally, the most well-studied and effective option for intractable reflux following LSG is conversion to RYGB. GERD along with inadequate weight loss are the two most common indications for conversion from LSG to RYGB. For patients undergoing revision to RYGB for persistent GERD following LSG, symptomatic improvement has been reported in 96–100% of patients [7, 45, 46], while, even in the most experienced hands, revision bariatric surgery is associated with higher morbidity and mortality that alternative non-surgical modalities.

Conversion of Sleeve to Bypass in Case of Acid Reflux

Although, as described above, the causes of GERD after sleeve gastrectomy are multifactorial, the treatment of choice for patients with GERD that is refractory to medical therapy, or endoscopic therapy, is conversion to gastric bypass. This is a fairly straightforward operation in concept, but which may be rendered technically challenging in the presence of abundant or exuberant adhesions and tissue hypertrophy. Patients must be counseled accordingly as to the heightened risks of surgery, including the increased potential for anastomotic leaks and/or failure of staple line integrity. Having said that, this operation can be very rewarding as it does deliver durable and significant relief from GERD symptoms. The majority of these operations can be completed laparoscopically, but the surgical team must be prepared to convert to laparotomy, if deemed necessary [47, 48].

Conclusion

In conclusion, although bariatric surgery has delivered truly astonishing results for treating metabolic dysfunction and its presenting comorbidities, it remains a therapy that demands astute patient selection, multidisciplinary evaluation, and optimization. Operation choice plays an undeniable influence on postoperative outcomes, as does the stringent adherence to technical details. All clinicians who care for patients after bariatric surgery should be aware of the potential for complications, including reflux disease. In this chapter, we have presented a variety of treatment options that range from the medical through the endoscopic to the surgical.

References

1. Ogden CL, Carroll MD, Fryar CD, Flegal KM. Prevalence of obesity among adults and youth: United States, 2011–2014. NCHS Data Brief. 2015;219:1–8.
2. Schauer PR, Schirmer B. The surgical management of obesity. In: Brunicardi FC, Andersen DK, Billiar TR, et al., editors. Schwartz's principles of surgery. 10e ed. New York: McGraw-Hill Education; 2014.
3. Khorgami Z, Shoar S, Andalib A, Aminian A, Brethauer SA, Schauer PR. Trends in utilization of bariatric surgery, 2010–2014: sleeve gastrectomy dominates. Surg Obes Relat Dis. 2017;
4. Oor JE, Roks DJ, Unlu C, Hazebroek EJ. Laparoscopic sleeve gastrectomy and gastroesophageal reflux disease: a systematic review and meta-analysis. Am J Surg. 2016;211(1):250–67.
5. Hutter MM, Schirmer BD, Jones DB, et al. First report from the American College of Surgeons bariatric surgery center network: laparoscopic sleeve gastrectomy has morbidity and effectiveness positioned between the band and the bypass. Ann Surg. 2011;254(3):410–20. discussion 420-412
6. Frezza EE, Ikramuddin S, Gourash W, et al. Symptomatic improvement in gastroesophageal reflux disease (GERD) following laparoscopic Roux-en-Y gastric bypass. Surg Endosc. 2002;16(7):1027–31.
7. Casillas RA, Um SS, Zelada Getty JL, Sachs S, Kim BB. Revision of primary sleeve gastrectomy to Roux-en-Y gastric bypass: indications and outcomes from a high-volume center. Surg Obes Relat Dis. 2016;12(10):1817–25.
8. Schirmer B, Hallowell P. Chapter 27. Morbid obesity and its surgical treatment. In: Zinner MJ, Ashley SW, editors. Maingot's abdominal operations. 12e ed. New York: The McGraw-Hill Companies; 2013.
9. Bennett JC, Wang H, Schirmer BD, Northup CJ. Quality of life and resolution of co-morbidities in super-obese patients remaining morbidly obese after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2007;3(3):387–91.
10. D'Hondt M, Steverlynck M, Pottel H, et al. Value of preoperative esophagogastroduodenoscopy in morbidly obese patients undergoing laparoscopic Roux-en-Y gastric bypass. Acta Chir Belg. 2013;113(4):249–53.
11. Loewen M, Giovanni J, Barba C. Screening endoscopy before bariatric surgery: a series of 448 patients. Surg Obes Relat Dis. 2008;4(6):709–12.
12. Mong C, Van Dam J, Morton J, Gerson L, Curet M, Banerjee S. Preoperative endoscopic screening for laparoscopic Roux-en-Y gastric bypass has a low yield for anatomic findings. Obes Surg. 2008;18(9):1067–73.
13. Sharaf RN, Weinshel EH, Bini EJ, Rosenberg J, Sherman A, Ren CJ. Endoscopy plays an important preoperative role in bariatric surgery. Obes Surg. 2004;14(10):1367–72.
14. Zeni TM, Frantzides CT, Mahr C, et al. Value of preoperative upper endoscopy in patients undergoing laparoscopic gastric bypass. Obes Surg. 2006;16(2):142–6.

15. Emerenziani S, Rescio MP, Guarino MP, Cicala M. Gastro-esophageal reflux disease and obesity, where is the link? World J Gastroenterol. 2013;19(39):6536–9.
16. Boules M, Corcelles R, Guerron AD, et al. The incidence of hiatal hernia and technical feasibility of repair during bariatric surgery. Surgery. 2015;158(4):911–6. discussion 916–918
17. Madhok BM, Carr WR, McCormack C, et al. Preoperative endoscopy may reduce the need for revisional surgery for gastro-oesophageal reflux disease following laparoscopic sleeve gastrectomy. Clin Obes. 2016;6(4):268–72.
18. Rebecchi F, Allaix ME, Patti MG, Schlottmann F, Morino M. Gastroesophageal reflux disease and morbid obesity: to sleeve or not to sleeve? World J Gastroenterol. 2017;23(13):2269–75.
19. Rebecchi F, Allaix ME, Ugliono E, Giaccone C, Toppino M, Morino M. Increased esophageal exposure to weakly acidic reflux 5 years after laparoscopic Roux-en-Y gastric bypass. Ann Surg. 2016;264(5):871–7.
20. Mattar SG, Qureshi F, Taylor D, Schauer PR. Treatment of refractory gastroesophageal reflux disease with radiofrequency energy (Stretta) in patients after Roux-en-Y gastric bypass. Surg Endosc. 2006;20(6):850–4.
21. Pauli EM, Beshir H, Mathew A. Gastrogastric fistulae following gastric bypass surgery-clinical recognition and treatment. Curr Gastroenterol Rep. 2014;16(9):405.
22. Ribeiro-Parenti L, De Courville G, Daikha A, Arapis K, Chosidow D, Marmuse JP. Classification, surgical management and outcomes of patients with gastrogastric fistula after Roux-En-Y gastric bypass. Surg Obes Relat Dis. 2017;13(2):243–8.
23. Poghosyan T, Caille C, Moszkowicz D, Hanachi M, Carette C, Bouillot JL. Roux-en-Y gastric bypass for the treatment of severe complications after omega-loop gastric bypass. Surg Obes Relat Dis. 2017;13(6):988–94.
24. Himpens J, Dobbeleir J, Peeters G. Long-term results of laparoscopic sleeve gastrectomy for obesity. Ann Surg. 2010;252(2):319–24.
25. Menenakos E, Stamou KM, Albanopoulos K, Papailiou J, Theodorou D, Leandros E. Laparoscopic sleeve gastrectomy performed with intent to treat morbid obesity: a prospective single-center study of 261 patients with a median follow-up of 1 year. Obes Surg. 2010;20(3):276–82.
26. Rosenthal RJ, International Sleeve Gastrectomy Expert P, Diaz AA, et al. International sleeve gastrectomy expert panel consensus statement: best practice guidelines based on experience of >12,000 cases. Surg Obes Relat Dis. 2012;8(1):8–19.
27. Rebecchi F, Allaix ME, Giaccone C, Ugliono E, Scozzari G, Morino M. Gastroesophageal reflux disease and laparoscopic sleeve gastrectomy: a physiopathologic evaluation. Ann Surg. 2014;260(5):909–14. discussion 914–905
28. Chiu S, Birch DW, Shi X, Sharma AM, Karmali S. Effect of sleeve gastrectomy on gastroesophageal reflux disease: a systematic review. Surg Obes Relat Dis. 2011;7(4):510–5.
29. Braghetto I, Lanzarini E, Korn O, Valladares H, Molina JC, Henriquez A. Manometric changes of the lower esophageal sphincter after sleeve gastrectomy in obese patients. Obes Surg. 2010;20(3):357–62.
30. Kleidi E, Theodorou D, Albanopoulos K, et al. The effect of laparoscopic sleeve gastrectomy on the antireflux mechanism: can it be minimized? Surg Endosc. 2013;27(12):4625–30.
31. Daes J, Jimenez ME, Said N, Daza JC, Dennis R. Laparoscopic sleeve gastrectomy: symptoms of gastroesophageal reflux can be reduced by changes in surgical technique. Obes Surg. 2012;22(12):1874–9.
32. Del Genio G, Tolone S, Limongelli P, et al. Sleeve gastrectomy and development of “de novo” gastroesophageal reflux. Obes Surg. 2014;24(1):71–7.
33. Petersen WV, Meile T, Kuper MA, Zdichavsky M, Konigsrainer A, Schneider JH. Functional importance of laparoscopic sleeve gastrectomy for the lower esophageal sphincter in patients with morbid obesity. Obes Surg. 2012;22(3):360–6.
34. Soricelli E, Casella G, Rizzello M, Cali B, Alessandri G, Basso N. Initial experience with laparoscopic crural closure in the management of hiatal hernia in obese patients undergoing sleeve gastrectomy. Obes Surg. 2010;20(8):1149–53.

35. Che F, Nguyen B, Cohen A, Nguyen NT. Prevalence of hiatal hernia in the morbidly obese. Surg Obes Relat Dis. 2013;9(6):920–4.
36. Snyder B, Wilson E, Wilson T, Mehta S, Bajwa K, Klein C. A randomized trial comparing reflux symptoms in sleeve gastrectomy patients with or without hiatal hernia repair. Surg Obes Relat Dis. 2016;12(9):1681–8.
37. Rodriguez L, Rodriguez P, Gomez B, et al. Two-year results of intermittent electrical stimulation of the lower esophageal sphincter treatment of gastroesophageal reflux disease. Surgery. 2015;157(3):556–67.
38. Crawford C, Gibbens K, Lomelin D, Krause C, Simorov A, Oleynikov D. Sleeve gastrectomy and anti-reflux procedures. Surg Endosc. 2017;31(3):1012–21.
39. Fass R, Cahn F, Scotti DJ, Gregory DA. Systematic review and meta-analysis of controlled and prospective cohort efficacy studies of endoscopic radiofrequency for treatment of gastroesophageal reflux disease. Surg Endosc. 2017;31:4865.
40. Medical T. The Linx Reflux Management System. 2017.
41. Bonavina L, DeMeester T, Fockens P, et al. Laparoscopic sphincter augmentation device eliminates reflux symptoms and normalizes esophageal acid exposure: one- and 2-year results of a feasibility trial. Ann Surg. 2010;252(5):857–62.
42. Lipham JC, Taiganides PA, Louie BE, Ganz RA, DeMeester TR. Safety analysis of first 1000 patients treated with magnetic sphincter augmentation for gastroesophageal reflux disease. Dis Esophagus. 2015;28(4):305–11.
43. Desart K, Rossidis G, Michel M, Lux T, Ben-David K. Gastroesophageal reflux management with the LINX(R) system for gastroesophageal reflux disease following laparoscopic sleeve gastrectomy. J Gastrointest Surg. 2015;19(10):1782–6.
44. Hawasli A, Tarakji M, Tarboush M. Laparoscopic management of severe reflux after sleeve gastrectomy using the LINX(R) system: technique and one year follow up case report. Int J Surg Case Rep. 2017;30:148–51.
45. Iannelli A, Debs T, Martini F, Benichou B, Ben Amor I, Gugenheim J. Laparoscopic conversion of sleeve gastrectomy to Roux-en-Y gastric bypass: indications and preliminary results. Surg Obes Relat Dis. 2016;12(8):1533–8.
46. Parmar CD, Mahawar KK, Boyle M, Schroeder N, Balupuri S, Small PK. Conversion of sleeve gastrectomy to Roux-en-Y gastric bypass is effective for gastro-oesophageal reflux disease but not for further weight loss. Obes Surg. 2017;27(7):1651–8.
47. Poghosyan T, Lazzati A, Moszkowicz D, et al. Conversion of sleeve gastrectomy to Roux-en-Y gastric bypass: an audit of 34 patients. Surg Obes Relat Dis. 2016;12(9):1646–51.
48. Felsenreich DM, Kefurt R, Schermann M, et al. Reflux, sleeve dilation, and Barrett's esophagus after laparoscopic sleeve gastrectomy: long-term follow-up. Obes Surg. 2017;

Gastric Band Erosion

9

Melissa Felinski, Maamoun A. Harmouch, Erik B. Wilson, and Shinil K. Shah

Introduction

Surgical approaches for the management of morbid obesity and associated medical comorbidities have arisen in part secondary to poor long-term outcomes with conservative approaches alone [1, 2]. Among the first weight loss surgeries performed were the Roux-en-Y gastric bypass and the biliopancreatic diversion. These initial techniques attained substantial weight loss results and helped improve obesity-related comorbidities in patients with morbid obesity [2, 3]. Early enthusiasm, however, was tempered by the reality of perioperative mortality, early postoperative complications, and long-term sequela that often required re-intervention [4]. Additionally, these techniques were often viewed by the public as invasive and risky [5]. This led the surgical community to seek alternative options, which in part led to the development of initially fixed and subsequently adjustable gastric bands (AGB).

Initial reports describing the gastric band experience (primarily laparoscopic AGBs) conveyed a great deal of optimism. It was viewed to be a simple, safe procedure that led to significant weight loss with very low perioperative complications and near-zero mortality rates. It was also easily performed with minimally invasive techniques, even for surgeons who did not have significant laparoscopic experience. This led surgeons worldwide to rapidly adopt this new procedure and technology [6, 7].

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The initial excitement has increasingly been called into question as more and more patients with AGBs present with myriad of complications, including many that require re-intervention to explant the device or conversion to another bariatric procedure to help with weight loss or deal with a complication of the device [8–10]. In a recent review that included nearly 19,000 patients, the rates of revision ranged from 20.22% to 34.2% over 7 years [8]. Another systemic review that included nearly 9700 patients showed a median complication rate of 42.7% and a median reoperation rate of 36.5% [9]. Lazzati et al. noted in a study including nearly 53,000 patients that the rate of gastric band explantation was 6% annually and 40% at 7 years, with more than two-thirds of patients requiring revisional surgery at some timepoint [11]. Carandina et al. noted in a series of approximately 300 patients that the band survival rate at 15 years was 53.3% [12].

Complications associated with gastric banding include poor weight loss, band slip, pouch dilation, worsening reflux, esophageal motility issues, and band erosions. The reported rates of the individual complication vary widely. Multiple factors account for this variation and some of these complications, including erosion, have decreased markedly secondary to improvements in surgical technique, enhancements to the design of the band prosthesis, and increasing surgeon experience [11].

Types of Gastric Bands

While the underlying principal of applying a prosthetic device around the stomach to facilitate weight loss has remained largely unchanged, the materials used to construct the band and adjustability of the band have evolved over the last several decades. The first fixed or nonadjustable gastric band (NAGB) was introduced in 1978 by Wilkinson and Pelso in which they used a 2 cm Marlex mesh to encircle the upper portion of the stomach [13]. Since then, Dacron vascular graft, polytetrafluoroethylene (PTFE) (Gortex), polymeric silicone (silastic), and other similar materials have been utilized with varying results [14, 15]. Mason was credited for being the first to incorporate the use of a fixed band to restrict the gastric outlet after partitioning the stomach (vertical banded gastroplasty (VBG)) [16]. The silicone gastric band was later modified to include an adjustable balloon which gave rise to the AGB [14]. Since its introduction in 1993, the AGB has seen multiple phases of development due to the high complication rates associated with the first-generation band [17]. The design of the band, including its shape, diameter, and balloon volume, has been modified to allow for pressure to be more evenly distributed along the stomach while still providing adequate restriction [17]. Additionally, the surgical technique of laparoscopic AGB placement has evolved from an initial peri-gastric approach to a pars flaccida technique [18]. In contrary, the sole use of fixed bands has been widely abandoned; however, their role in conjunction with other bariatric procedures such as the banded gastric bypass is still being utilized today.

Gastric Band Erosion

Band erosion is commonly described as intragastric migration of all or part of the band. The reported incidence of gastric band erosion in the literature varies widely. Most authors report an incidence that ranges approximately from 0% to 4% [19–21]. However, rates as high as 32% have been reported [20]. In a recent systemic review that included 25 studies and 15,775 patients, band erosion occurred at a rate of 1.47% [20]. Similar to other complications seen with AGBs, the risk of erosion is directly proportional to the indwelling time of the device. It is important to note, however, that the majority of the cases occur within the first 12 months after placement [19].

Risk factors that contribute to erosion include unrecognized intraoperative injury that leads to micro-perforation, gastric plication sutures placed directly over the buckle of the AGB device resulting in pressure and ischemia, and over-tightening of the band. There is a correlation between band slip and band erosion [21]. The perigastric technique of placement of AGBs was thought to also contribute to the higher erosion rates seen in earlier experiences. In addition, patient-related factors such as smoking, NSAIDS, and alcohol have all been reported to contribute to gastric band erosion [19–21].

Clinically, band erosion rarely presents as an acute surgical problem. Symptoms are often subtle and include loss of satiety, weight regain, and epigastric pain. In addition, it can also present as an isolated port site infection that fails to resolve despite drainage and antibiotics. Rarely, patients can present with evidence of systemic sepsis and peritonitis [22]. An unexplained leak in the band system should prompt an evaluation for possible band erosion. Diagnosis is often made with upper endoscopy. The degree of erosion can be further staged on endoscopy according to the Nocca classification. Stage 1 entails the ability to visualize the band endoscopically. Stage 2 describes partial migration where more than half of the device is intraluminal. Stage 3 entails complete intragastric migration of the band. If pus is visualized in the absence of clearly visualized erosion, one can inflate the reservoir and recheck the flow of pus through the stoma upon retroflexion of the endoscope. Augmentation of pus flow upon inflating the reservoir is pathognomonic for band erosion [19–21, 23].

Management of Eroded Bands

Removal of the gastric band is often warranted once erosion occurs. Although controversy exists regarding the optimal approach, it is imperative that a gastrointestinal or bariatric surgeon be familiar with the different modalities available and be able to individualize treatment options when managing a patient with an eroded prosthesis. A variety of factors should be taken into consideration when choosing an approach, including the overall clinical picture of the patient (symptomology, previous surgeries), type of band (NAGB versus AGB), material of the band, degree of migration (complete versus partial), and timing of erosion (acute perforation versus

chronic penetration). The principal methods to be discussed are traditional surgical intervention/removal, endoscopic removal, combined laparoscopic/endoscopic technique, and expectant management. Patients should be informed of the associated risks as each method is associated with its own set of potential complications.

Surgical Approach

Surgical removal of an eroded band is feasible but can be a technically challenging endeavor with high morbidity. While minimally invasive techniques are preferred, laparoscopic approaches should be attempted by experienced surgeons. As previously discussed, a significant inflammatory response occurs around the banded portion of the stomach and left lobe of the liver. In addition, a thick fibrous capsule forms around the device as it migrates and eventually erodes into the gastric lumen. In order to minimize dissection and remove the band safely, the port tubing of an AGB should be identified and followed back toward the band. Further dissection occurs along the band until the buckle is exposed and transected. The location of a NAGB may not be as readily evident. The band itself may be densely adherent to the surrounding tissues and can be extremely difficult to remove. This is more commonly seen with fixed bands made of Marlex, Dacron, or other non-silicone materials that allow tissue ingrowth. Silastic bands, on the other hand, do not incorporate into the gastric tissue. After the band is explanted, the resulting gastrostomy defect may be closed primarily, with or without omental patch reinforcement and evaluated for leaks. This area is often very inflamed with friable tissue, making primary closure of the defect difficult and more prone to breakdown and leakage [23]. If a considerable portion of the eroded band, including buckle of an AGB is located in the gastric lumen, an alternative approach is to create a separate gastrostomy away from the site of erosion. The band is then removed from inside the stomach and subsequent closure of the gastric wall is performed with healthier tissue. Placement of intra-abdominal drains near the area of repair should be considered in high-risk settings. In the case of acute gastric perforation, emergent removal of the band and wide drainage via a laparoscopic or open approach is necessary.

Endoscopic Removal

Endoscopic removal of an eroded gastric band is an alternative approach for surgeons with an advanced endoscopic skillset or in conjunction with a skilled therapeutic endoscopist. Not only are the inherent pitfalls and potential complications of re-operative surgery avoided, several studies [24–28] have demonstrated it to be a safe option and treatment of choice for the management of a prosthesis with >50% intraluminal erosion. In the instance of an AGB, it is also crucial that the buckle of the band is positioned within the gastric lumen to avoid serious complications during explantation, such as bleeding or intra-abdominal leak. In addition, the

subcutaneous port and associated tubing should be removed under local anesthesia after it is determined that the band is amenable to endoscopic removal.

Various endoscopic instruments have been used to transect the eroded gastric band and facilitate endoscopic explantation. Some groups were able to incise the band using Nd:YAG laser ablation [25, 27] or electrosurgical devices such as argon plasma coagulation [29]. However, gastric bands made of Gortex or other synthetic materials are not electrically conductive and therefore cannot be removed with these thermal methods [26]. Other groups successfully utilized endoscopic scissors [24, 26] or a gastric band cutter (Agency for Medical Innovations GmbH, Feldkirch, Austria) [30, 31] to cut the band. At our institution, we prefer to pass a cutting JAGwire around the band after creating a space between the prosthesis and mucosa by grasping the band and bring the wire back out through the mouth. A manual Soehendra lithotripter is then used to cut the band. Once the band is successfully cut, it is grasped with a snare or grasping forceps and removed endoscopically. Successful endoscopic removal of an eroded gastric band is reported to be >92% in two separate retrospectives studies [32, 33].

Fixed gastric bands can present a particular problem when encountered endoscopically. Bands such as those that are made of Dacron can be more difficult to remove because of tissue ingrowth and may require multiple endoscopic attempts or removal in a piecemeal fashion.

Combined Approach

There are a number of cases in the literature that also describe combined approaches to facilitate removal of an eroded gastric band when purely endoscopic means fail or are not available. A laparoscopic/endoscopic approach was reported by Karmali et al. in which they transected the migrated band with laparoscopic scissors through a transgastric trocar while simultaneously viewing the band endoscopically and then removing the prosthesis transorally with an endoscopic snare [34]. A similar approach was described by Basa et al. [35].

Expectant Management

While a band with sufficient intragastric erosion should be intervened upon, a partially eroded band can be treated conservatively in some circumstances [36]. Watchful waiting can be considered in the asymptomatic patient found to have a band erosion of less than 50% of the band circumference [31]. It is anticipated that further erosion of the prosthesis will occur with time. There are several cases reported in the literature in which asymptomatic patients had spontaneous extrusion of the band during this time of surveillance [25]. Nonetheless, expectant management requires close follow-up with frequent endoscopic evaluations to avoid complications related to a band migration, including small bowel obstruction and bleeding [37–39].

To accelerate or even induce the process of band erosion, temporary placement of an endoscopic stent may be attempted. Wilson et al. reported 15 cases in which 87% of the patients had successful stent-induced erosion with 2–3 weeks of stent therapy, followed by complete endoscopic removal of their fixed bands. The remaining patients had partial removal with this technique [40]. Similar outcomes were achieved by Dugan et al. and Campos et al. [41, 42]. Approximately 22% of patients developed a fibrotic stricture requiring additional endoscopy with balloon dilation [42].

Although the stent-induced erosion technique has been primarily described with fixed gastric bands, it can be applied to AGBs in select cases. Stent-induced erosion may be performed to erode the buckle of a partially eroded AGB followed by endoscopic retrieval. This approach offers a potentially less invasive technique of band removal. It is important to maximally inflate the AGB after stent placement to encourage mucosal ischemia between the stent and the band and encourage further intra luminal erosion of the band.

Conclusion

Gastric band erosion rarely requires emergent surgical intervention. Although laparoscopic removal can be accomplished, it is often accompanied by the perils of reoperative surgery. Endoscopic techniques can facilitate removal of an eroded gastric prosthesis while avoiding a challenging operative field. Once a significant portion of the band has eroded into the stomach, the majority of bands can be safely removed electively by an endoscopic approach. Gastric stent placement to force erode NAGB has recently been reported and, in selected cases, can be applied to partially eroded AGBs.

References

1. Athyros VG, Tziomalos K, Karagiannis A, Mikhailidis DP. Cardiovascular benefits of bariatric surgery in morbidly obese patients. Obes Rev. 2011;12(7):515–24.
2. Olshansky SJ, Passaro DJ, Hershow RC, et al. A potential decline in life expectancy in the United States in the 21st century. N Engl J Med. 2005;352(11):1138–45.
3. Christou NV, Sampalis JS, Liberman M, et al. Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients. Ann Surg. 2004;240(3):416–23. discussion 423–414
4. Flum DR, Dellinger EP. Impact of gastric bypass operation on survival: a population-based analysis. J Am Coll Surg. 2004;199(4):543–51.
5. Nguyen NT, Paya M, Stevens CM, Mavandadi S, Zainabadi K, Wilson SE. The relationship between hospital volume and outcome in bariatric surgery at academic medical centers. Ann Surg. 2004;240(4):586–93. discussion 593–584
6. Nguyen NT, Slone JA, Nguyen XM, Hartman JS, Hoyt DB. A prospective randomized trial of laparoscopic gastric bypass versus laparoscopic adjustable gastric banding for the treatment of morbid obesity: outcomes, quality of life, and costs. Ann Surg. 2009;250(4):631–41.

7. Busetto L, Angrisani L, Basso N, et al. Safety and efficacy of laparoscopic adjustable gastric banding in the elderly. Obesity (Silver Spring). 2008;16(2):334–8.
8. Altieri MS, Yang J, Telem DA, et al. Lap band outcomes from 19,221 patients across centers and over a decade within the state of New York. Surg Endosc. 2016;30(5):1725–32.
9. Shen X, Zhang X, Bi J, Yin K. Long-term complications requiring reoperations after laparoscopic adjustable gastric banding: a systematic review. Surg Obes Relat Dis: Off J Am Soc Bariatric Surg. 2015;11(4):956–64.
10. Arapis K, Tammaro P, Parenti LR, et al. Long-term results after laparoscopic adjustable gastric banding for morbid obesity: 18-year follow-up in a single university unit. Obes Surg. 2017;27(3):630–40.
11. Lazzati A, De Antonio M, Paolino L, et al. Natural history of adjustable gastric banding: lifespan and revisional rate: a Nationwide study on administrative data on 53,000 patients. Ann Surg. 2017;265(3):439–45.
12. Carandina S, Tabbara M, Galiay L, et al. Long-term outcomes of the laparoscopic adjustable gastric banding: weight loss and removal rate. A single center experience on 301 patients with a minimum follow-up of 10 years. Obes Surg. 2017;27(4):889–95.
13. Wilkinson LH, Peloso OA. Gastric (reservoir) reduction for morbid obesity. Arch Surg. 1981;116(5):602–5.
14. Kuzmak LI. Silicone gastric banding: a simple and effective operation for morbid obesity. Contemp Surg. 1986;28(1):13–8.
15. Molina M, Oria HE. Gastric banding. Proceedings of the Sixth Annual Bariatric Surgery Colloquim. 1983:15.
16. Mason EE. Vertical banded gastroplasty for obesity. Arch Surg. 1982;117(5):701–6.
17. Beitner MM, Ren-Fielding CJ, Fielding GA. Reducing complications with improving gastric band design. Surg Obes Relat Dis: Off J Am Soc Bariatric Surg. 2016;12(1):150–6.
18. McBride CL, Kothari V. Evolution of laparoscopic adjustable gastric banding. Surg Clin North Am. 2011;91(6):1239–47. viii–ix
19. Cherian PT, Goussous G, Ashori F, Sigurdsson A. Band erosion after laparoscopic gastric banding: a retrospective analysis of 865 patients over 5 years. Surg Endosc. 2010;24(8):2031–8.
20. Egberts K, Brown WA, O'Brien PE. Systematic review of erosion after laparoscopic adjustable gastric banding. Obes Surg. 2011;21(8):1272–9.
21. Singhal R, Bryant C, Kitchen M, et al. Band slippage and erosion after laparoscopic gastric banding: a meta-analysis. Surg Endosc. 2010;24(12):2980–6.
22. Quadri P, Gonzalez-Heredia R, Masrur M, Sanchez-Johnsen L, Elli EF. Management of laparoscopic adjustable gastric band erosion. Surg Endosc. 2017;31(4):1505–12.
23. Yoon CI, Pak KH, Kim SM. Early experience with diagnosis and management of eroded gastric bands. J Korean Surg Soc. 2012;82(1):18–27.
24. Evans JA, Williams NN, Chan EP, Kochman ML. Endoscopic removal of eroded bands in vertical banded gastroplasty: a novel use of endoscopic scissors (with video). Gastrointest Endosc. 2006;64(5):801–4.
25. Fobi M, Lee H, Igwe D, et al. Band erosion: incidence, etiology, management and outcome after banded vertical gastric bypass. Obes Surg. 2001;11(6):699–707.
26. Karmali S, Snyder B, Wilson EB, Timberlake MD, Sherman V. Endoscopic management of eroded prosthesis in vertical banded gastroplasty patients. Surg Endosc. 2010;24(1):98–102.
27. Lunde OC. Endoscopic laser therapy for band penetration of the gastric wall after gastric banding for morbid obesity. Endoscopy. 1991;23(2):100–1.
28. Weiss H, Nehoda H, Labeck B, Peer R, Aigner F. Gastroscopic band removal after intragastric migration of adjustable gastric band: a new minimal invasive technique. Obes Surg. 2000;10(2):167–70.
29. Meyenberger C, Gubler C, Hengstler PM. Endoscopic management of a penetrated gastric band. Gastrointest Endosc. 2004;60(3):480–1.
30. Lattuada E, Zappa MA, Mozzi E, et al. Band erosion following gastric banding: how to treat it. Obes Surg. 2007;17(3):329–33.

31. Regusci L, Groebli Y, Meyer JL, Walder J, Margalith D, Schneider R. Gastroscopic removal of an adjustable gastric band after partial intragastric migration. Obes Surg. 2003;13(2):281–4.
32. Chisholm J, Kitan N, Toouli J, Kow L. Gastric band erosion in 63 cases: endoscopic removal and rebanding evaluated. Obes Surg. 2011;21(11):1676–81.
33. Neto MP, Ramos AC, Campos JM, et al. Endoscopic removal of eroded adjustable gastric band: lessons learned after 5 years and 78 cases. Surg Obes Relat Dis: Off J Am Soc Bariatric Surg. 2010;6(4):423–7.
34. Karmali S, Sweeney JF, Yee K, Brunicardi FC, Sherman V. Transgastric endoscopic rendezvous technique for removal of eroded Molina gastric band. Surg Obes Relat Dis: Off J Am Soc Bariatric Surg. 2008;4(4):559–62.
35. Basa NR, Dutson E, Lewis C, Derezin M, Han S, Mehran A. Laparoscopic transgastric removal of eroded adjustable band: a novel approach. Surg Obes Relat Dis: Off J Am Soc Bariatric Surg. 2008;4(2):194–7.
36. Zehetner J, Holzinger F, Triaca H, Klaiber C. A 6-year experience with the Swedish adjustable gastric band prospective long-term audit of laparoscopic gastric banding. Surg Endosc. 2005;19(1):21–8.
37. Abeysekera A, Lee J, Ghosh S, Hacking C. Migration of eroded laparoscopic adjustable gastric band causing small bowel obstruction and perforation. BMJ Case Rep. 2017;2017.
38. Campos J, Ramos A, Galvao Neto M, et al. Hypovolemic shock due to intragastric migration of an adjustable gastric band. Obes Surg. 2007;17(4):562–4.
39. Lemaire J, Dewit O, Navez B. Management of a jejunal obstruction caused by the migration of a laparoscopic adjustable gastric banding. A case report. Int J Surg Case Rep. 2017;30:6–8.
40. Wilson TD, Miller N, Brown N, Snyder BE, Wilson EB. Stent induced gastric wall erosion and endoscopic retrieval of nonadjustable gastric band: a new technique. Surg Endosc. 2013;27(5):1617–21.
41. Dugan J, Bajwa K, Singhal S. Endoscopic removal of gastric band by use of a stent-induced erosion technique. Gastrointest Endosc. 2016;83(3):654–5.
42. Marins Campos J, Moon RC, Magalhaes Neto GE, et al. Endoscopic treatment of food intolerance after a banded gastric bypass: inducing band erosion for removal using a plastic stent. Endoscopy. 2016;48(6):516–20.

Chronic Abdominal Pain After Roux-en-Y Gastric Bypass

10

Pearl Ma and Kelvin Higa

Introduction

Roux-en-Y gastric bypass (RYGB) is a frequently performed bariatric procedure in the United States and worldwide, second only to the sleeve gastrectomy. Its effectiveness in long-term control of morbid obesity and obesity-related diseases such as diabetes and heart disease is undisputed [1]. Despite the success of the operation, a small number of RYGB patients can develop late complications from the operation. As discussed in other chapters, complications such as development of marginal ulcers or significant protein calorie malnutrition, unexplained intractable nausea and emesis, ongoing substance abuse, severe vitamin deficiencies, or even reactive hypoglycemia can be difficult to manage [2–9]. However, patients may present months to years after gastric bypass with chronic abdominal pain. This can result in a burden to the health-care system as well as frustrate health-care providers ill equipped to diagnose and treat this issue. Abdominal pain is the most common symptom reported to health-care providers after follow-up in RYGB patients [10]. One survey conducted of patients 5 years post-RYGB discovered 33.8% incidence of mild to severe chronic abdominal pain lasting more than 3 months [11]. Therefore, this problem is not trivial.

The anatomic and physiologic changes that occur as a result of the surgical construct pose a unique set of potential complications for the RYGB patient. Because of the isolated gastric remnant and new configuration, chronic abdominal pain after RYGB requires further investigation as some conditions can lead to life-threatening illnesses. For instance, partial bowel obstructions from an internal hernia could not only result in volvulus and bowel ischemia but also blowout of the gastric remnant. Other conditions such as suture bezoars, marginal ulcers, small bowel

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intussusception, and biliary tract disease can present with intermittent abdominal pain. If the surgeon leaves an excessively long "afferent" limb when making the gastrojejunostomy, this will elongate over time, trapping food and causing pain with regurgitation immediately postprandial. In addition, pathology of the inaccessible gastric remnant such as gastroparesis and ulceration presents a challenge to diagnose, often requiring surgical intervention and a high degree of clinical suspicion.

Initial evaluation of RYGB patient with chronic abdominal pain requires a detailed history and physical examination. Although self-evident and universally agreed upon, a good history elucidating the timing, nature, and location of the abdominal pain is often substituted instead by reflexive order of imaging to develop the diagnosis. Symptoms can lead the clinician to develop a focused differential diagnosis and a more thoughtful workup and treatment plan. Further evaluation may include laboratory and imaging studies such as upper gastrointestinal (UGI) study, computed tomography (CT) scan, abdominal ultrasound, motility studies, and/or endoscopies. Although, now thought to be archaic, a barium meal will often elucidate the location of abdominal pain that ordinary contrast studies will miss. Few radiologists are willing to work with the surgical team in this regard; they may need some encouragement, especially with these challenging patients. With a reliable history and physical exam, imaging workup can be bypassed directly for surgical exploration. For example, a patient with a classic history for internal hernia consisting of severe epigastric abdominal pain associated with distension after RYGB requires exploration regardless of any radiographic study because of a defined false-negative rate and does not require a CT scan prior to exploration. In one retrospective study, 57% of RYGB patients with abdominal pain had significant intraoperative findings on diagnostic laparoscopy despite having negative findings on CT scan and upper endoscopy [12]. Another case series found 4 out of 15 patients with internal hernia on exploration for chronic abdominal pain with again negative preoperative workup [13]. Ultimately after RYGB, the work-up of chronic abdominal pain is never complete without a thorough surgical exploration despite negative noninvasive studies. Because of the risk of internal herniation, exploration is necessary to rule out and correct this possibility. This chapter will review the most common and uncommon causes for chronic abdominal pain after RYGB, including workup, management, and treatment.

Marginal Ulcers

Description

Marginal ulcers (MU) are one of the most common causes of chronic abdominal pain after RYGB. They occur on the jejunal side of gastrojejunostomy anastomosis, often associated with tobacco or NSAID use. The mechanism for MU formation is unknown but is clearly associated with acid exposure of the unprotected jejunal mucosa (Fig. 10.1). There appears to be correlation with the size of the gastric pouch and incidence of ulceration as well as the presence of gastrogastric fistulas.

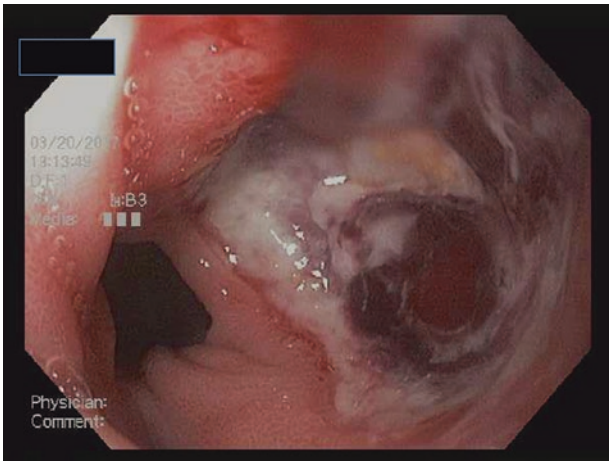

Fig. 10.1 Endoscopic view of large marginal ulcer at gastrojejunostomy

Ulcerations can also occur without any clear risk factors sporadically throughout the life of the patient. On average, MU are found around 15 months after surgery with a reported incidence of 0.6–25% of all RYGB [4, 14–18]. With more recent reports, a 2.6% incidence appears more realistic [19].

MU characteristically present with postprandial epigastric pain. When chronic, the constant inflammation can lead to strictures and dysphagia. Moon and Azagury et al. found the most common complaints of MU are abdominal pain (59–66%), nausea and vomiting (15%), and dysphagia (8%) [4, 20]. Acutely, MU can also present with perforation and upper gastrointestinal bleeding. Patel et al. found bleeding from MU presented in 20% of patients where 2.5% of patients with MU presented with acute perforation [17].

Risk Factors

Multiple factors have been implicated as potential causes for MU. Tobacco use, nonsteroidal anti-inflammatory drugs (NSAIDs), *Helicobacter pylori* (*H. pylori*) infection, use of nonabsorbable sutures, preoperative hypertension, increased gastric acid production, and presence of gastrogastric fistula have all been reported contributing to MU development [4, 6, 15, 16, 18–25]. Tobacco has known association with gastric ulcer formation, likely due to stimulation of increased acid production and oxidative damage of the mucosa [26]. Rasmussen et al. found that MU formation had a higher association with *H. pylori* infections even if adequately treated preoperatively [6]. It is still unclear if *H. pylori* infection needs to be treated prior to RYGB, although it is agreed that treatment is recommended in the presence of existing MU.

The surgical construct of the gastrojejunostomy is also important to consider in potential causes for MU. Large pouch sizes have been suggested to correlate with higher acid production as they may contain more parietal cell mass therefore creating more gastric acid to cause ulceration [20]. Mason et al. recommended a small fundic pouch and smaller stomal size to reduce acid secretion within gastric pouch [24]. Recently, Edholm et al. performed a long-term study on RYGB patients and found that each additional centimeter used to create the gastric pouch created a 14% increased relative risk of MU formation [27].

The creation of the gastrojejunostomy anastomosis is also important in considering MU formation. After switching to absorbable sutures instead of nonabsorbable sutures to create the gastrojejunostomy, Sachs et al. found the rate of MU occurrence decreased in half [19].

Gastrogastric fistulas can also be a source of recalcitrant MU. These can occur if the gastric pouch is not divided fully from the gastric remnant and staple line breakdown occurs leading to increased gastric acid exposure [28]. Gastrogastric fistulas are often difficult to diagnose, present even with a negative UGI and endoscopic examinations. Recalcitrant MU, without usual risk factors along with weight regain, is suggestive of a gastrogastric fistula and can only be dismissed by surgically separating the gastric pouch from the remnant at the time of exploration. Asymptomatic gastrogastric fistulas can be managed expectantly.

Clearly, the majority of GBP patients will never develop a marginal ulcer, despite risk factors, and up to 31% of patients will have no identifiable risk factors [29]. However, this complication can occur sporadically throughout the life of a patient and, therefore, is probably underreported, often presenting and being cared for by individuals other than the operative surgeon.

Evaluation

Workup of the marginal ulcer includes evaluation of the gastric pouch and anastomosis. UGI can evaluate subtle mucosal irregularities that signify ulcer as well as examine pouch length and presence of gastrogastric fistula. However, upper endoscopy is the standard by which to diagnose and categorize the severity of marginal ulceration.

Management

High-dose proton pump inhibitor (PPI) and Carafate remain the mainstay of initial treatment along with the elimination of risk factors such as tobacco and NSAIDs. Relief of symptoms can be seen as early as days to months. Gumbs et al. found complete resolution of MU in patients within 8 weeks after medical therapy [16]. Generally, patients can be managed as an outpatient depending on severity of symptoms. However, Moon et al. found 41% of patients with acute and chronic MU required hospitalization with 44% ultimately requiring an operation for perforation

or revision [4]. Severe ulcers seen at endoscopy which are often full-thickness perforations contained by the liver anteriorly, or pancreas posteriorly, will never yield to non-operative management; therefore, preparation for surgical intervention and optimization of nutrition is paramount to initial management upon presentation.

At our institution, we institute twice-a-day dosing of PPI and Carafate for 1 month after diagnosis of MU and then once-a-day dosing of PPI for the subsequent 2 months with cessation of offending risk factors. Upper endoscopy surveillance of MU is performed 2–3 months after treatment to ensure adequate healing.

If ulcers are refractory or in situations where the patient is unable to quit precipitating factors, then a revision operation is mandatory. Moon and Patel et al. found 16–32% of patients with MU required a revision [4, 17]. Operative strategy includes excision of the ulcer with reduction in the size of the gastric pouch, elimination of a gastrogastric fistula when present, complete excision of the gastric pouch with a subsequent esophagojejunostomy, or reversal of the RYGB. Reversal to normal anatomy or conversion to sleeve gastrectomy may be the optimal treatment in a patient with noncompliance to follow up, inability to avoid precipitating factors, or even in cases with chronic MU leading to severe malnutrition [30, 31]. At our institution, we found 68% of patients with chronic MU also had tobacco, alcohol, or substance abuse that contributed to reversal rather than revision.

Revisional operations are technically challenging and should be performed only by experienced bariatric surgeons and in institutions with the ability support for this level of care. Complications such as anastomotic leak, sepsis, bleeding, and re-interventions are much higher after revisional operations than primary bariatric operations. Chau et al. reported 25% of patients undergoing revisional operations required another operation for postoperative complications [32]. Therefore, surgeons must have adequate resources and skill to deal with these potential complications (Fig. 10.2a, b).

Bowel Obstructions

Intermittent small bowel obstruction (SBO) can present as chronic abdominal pain. Internal hernias, adhesive disease, and intussusception are some of the most common conditions and are often misdiagnosed as cholelithiasis or irritable bowel syndrome. Elms et al. reported their rates for reoperation for SBO after primary RYGB at 3.9% [33]. Because of the potential for total intestinal necrosis or gastric remnant blowout after GBP, it is imperative that surgeons have a low threshold for exploration and a high index of suspicion when evaluating these patients with chronic abdominal pain.

Patients presenting with acute SBO likely have complained of prior symptoms. In Brolin's study, almost all the patients with acute SBO had some prior complaint of nausea, bloating, and intermittent abdominal pain [34]. Karila-Cohen et al. found only 40% of CT readings were accurate for diagnosis for abdominal pain [35]. At the Cleveland Clinic, RYGB patients with abdominal pain underwent diagnostic laparoscopy and found pathologies in 57% of the patients with negative imaging

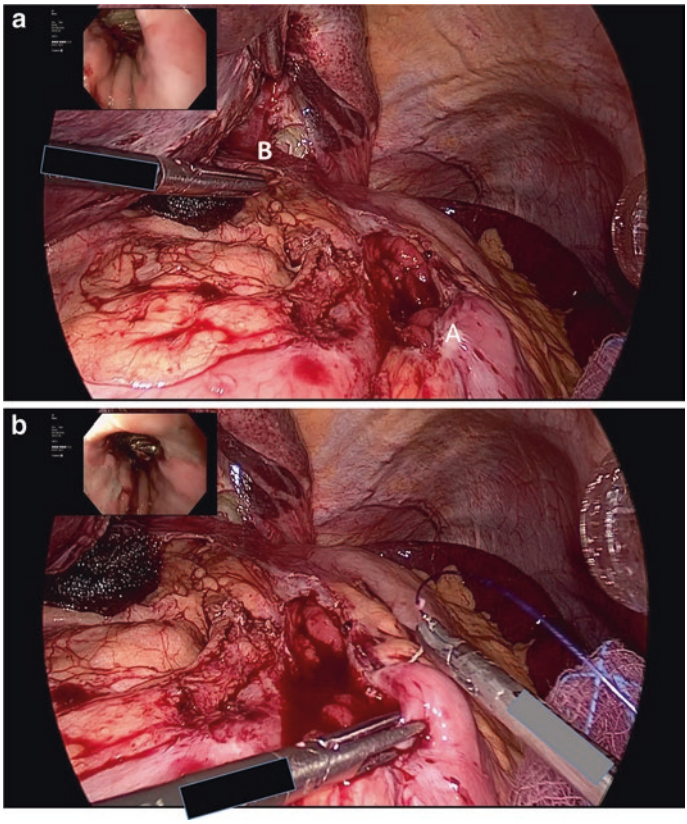

Fig. 10.2 (**a**) Complex marginal ulcer with free perforation at gastrojejunostomy (**A**) and ulceration eroding into pericardium. Endoscopy light can be seen illuminating the perforation site in mediastinum (**B**). (**b**) Suture repair of large marginal ulcer

studies and upper endoscopies. Alternatively, some patients with negative findings on exploration continued to have chronic abdominal pain without a clear diagnosis [12]. Diagnostic laparoscopy is not only safe as a diagnostic tool; it can also be therapeutic.

Internal Hernias

Description

Internal hernias are a common cause of chronic abdominal pain after laparoscopic techniques evolved with the RYGB. Considerably less postoperative adhesions formed allowing for mobility and volvulus of the small bowel to occur. Not always presenting with as an acute SBO, internal hernias can result in intermittent obstruction or vascular ischemia depending on location and severity. CT scans have a

definable false-negative rate in identifying internal hernias; radiologists and surgeons must have a high index of suspicion in looking for secondary signs such as an air/fluid level in the gastric remnant. The true incidence of internal hernias is probably underreported. Our initial report indicated a 3.1% incidence but, in the same cohort, was 16% at 10 years [36]. All mesenteric defects created by the anatomic construct are at risk for internal hernia, Petersen's space (retro-Roux space), mesocolic window (retro-colic Roux limb), and jejunojejunostomy defect. Therefore, it is plausible that the overall incidence can be less with an antecolic routing of the Roux limb. Clearly, the risk of internal hernias is lifelong, and closure with nonabsorbable sutures, although not curative, is now the standard of care.

Evaluation

The typical symptoms of epigastric abdominal pain, postprandial bloating, nausea, and/or vomiting may suggest symptoms of internal hernia, but its presentation can be highly variable. CT imaging showing a whirling of the mesentery has a high sensitivity and specificity for internal hernia. Other indicative findings may include dilated gastric remnant, transition zone of obstruction, and clustering of small bowel [35]. Patients with a clear history of obstruction may not require further imaging workup, and rather surgical exploration is warranted. As many hernia defects and internal hernias, themselves, can be asymptomatic and quiescent, other causes of abdominal pain should also be investigated such as biliary tract disease, ulceration, and functional bowel disorders.

Management

Reducing small bowel from internal hernias can be easily done or a struggle depending on the chronicity and extent of dilation of the small bowel. If there is significant bowel distension and necrosis, there should be a low threshold to convert to open or even initiating the operation open, especially when there is hemodynamic compromise. An enterotomy to decompress the small bowel can be done laparoscopically in order to increase the workable space, but it is often times difficult to avoid significant spillage of intestinal contents. A sterile Salem sump tube can be inserted through a small defect and advanced as necessary to avoid contamination. Severity of bowel ischemia may be difficult to assess laparoscopically by color; the temperature scale of the light source and the digital interpretation makes visual assessment less accurate than by direct visualization with standard operative lights. What may look to be end-stage ischemia may in fact be reversible. Injection of indocyanine green imaging may help but is not definitive in this setting. A planned second-look operation may prevent unnecessary and debilitating bowel resections (Fig. 10.3).

Closure of the mesenteric defect should be performed with nonabsorbable sutures such as 2–0 polyester sutures (Fig. 10.4a, b). Reduction of the small bowel is best performed starting retrograde from ileocecal valve to avoid confusion. Bowel

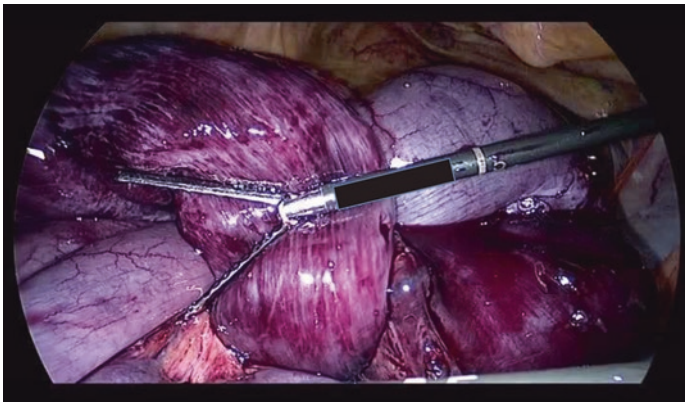

Fig. 10.3 Internal hernia reduced retrograde. Assessment of bowel viability may be impaired with laparoscopy

must be reduced gently without causing trauma and inspected afterward for serosal tears. Upper endoscopy decompression of the Roux limb may be of some use. If gastric remnant is dilated, a gastrotomy or gastrostomy tube for decompression is recommended. Postoperative management is similar to the management of standard SBO, resuming oral diet when appropriate and postoperative ileus resolves.

Intussusception

Intussusception is another potential cause of chronic abdominal pain after RYGB. Like internal hernias, this is likely underreported and misdiagnosed as functional bowel disorders. The intermittent and limited symptomatology makes diagnosis difficult. Often times, the patient presents to the emergency room with acute pain only to resolve by the time the patient is seen and the CT ordered. Most commonly, retrograde invagination of the common channel into the jejunojejunostomy occurs without a specific lead point, with spontaneous resolution. The etiology is likely due to a change in bowel motility with an alteration migratory motor complexes or development of ectopic pacemaker cells causing the small bowel at the common channel to telescope back into the anastomosis [37, 38]. Anterograde intussusception has also been described of the Roux limb into the jejunojejunostomy but likely due to adhesions as a lead point [39].

Simper et al. reports the largest case series of 23 patients with intussusception. This is probably due to the unique demographics of their practice: patients remain local for a long period of time, so any chronic disorder is captured by their follow-up. Along with case reports, there is an estimated 0.1–0.2% incidence of developing intussusception after RYGB. Like other pathologies, it generally presents years after RYGB surgery [40, 41]. In severe situations, patients present with ischemia of

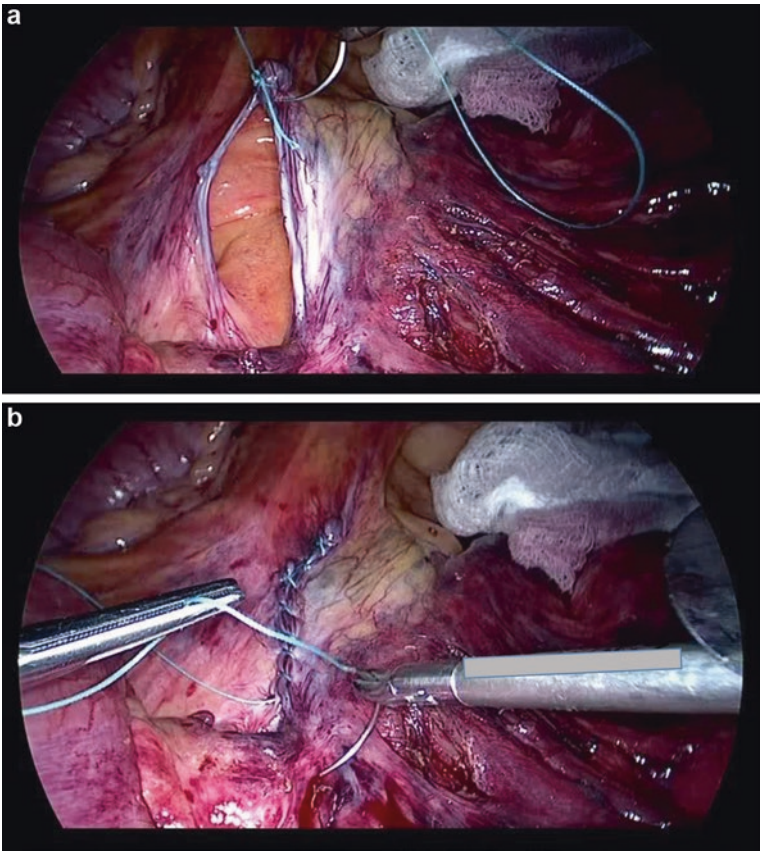

Fig. 10.4 (**a**) Large defect at the jejunojejunostomy. (**b**) Closure of mesenteric defect with nonabsorbable sutures

the intussuscepted bowel, easily seen on CT scan, often requiring small bowel resection.

Evaluation

Patients can present acutely with abdominal pain and obstruction or as chronic intermittent abdominal pain. Chronic pain is much more difficult to diagnose and difficult to pinpoint a consistent history as symptoms may be intermittent due to transient telescoping of the bowel. Pain generally occurs in left upper quadrant and can be exacerbated by food. Associated symptoms may include bloating and nausea. Imaging may be helpful, but unless not actively telescoping, CT imaging may not capture the event. In a series by Jawad et al., only 1 of the 12 patients with intussusception was diagnosed by CT imaging alone. The remaining patients were

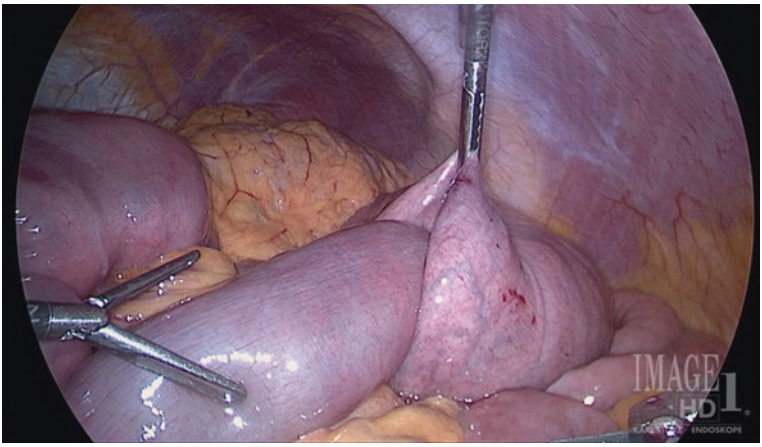

Fig. 10.5 Intussusception with common channel reduced retrograde from jejunojejunostomy

diagnosed during intraoperative laparoscopy [42]. CT findings may show a classic "target sign" of invaginated bowel within the jejunojejunostomy. Alternatively, incidental CT findings of intussusception are often found and are clinically irrelevant in asymptomatic patients. Basically, not all intussusceptions found on CT require an operation but require clinical correlation.

Management

Laparoscopic approach is performed if the patient is hemodynamically stable. Conversion to laparotomy may be required to manually reduce the common channel safely. The common channel is gently pulled distal from the jejunojejunostomy, and once reduced, viability of the small bowel needs to be examined (Fig. 10.5). Afterward, management of the jejunojejunostomy remains a debate. Options to leave the reduced bowel alone, plicate the bowel, perform an enteropexy, or resect and revise the jejunojejunostomy have all been described in the literature as potential management strategies. Nevertheless, there remains a paucity of literature to describe the optimal treatment. Recurrence of intussusception can still occur after all these strategies. Simper et al. examined patients that underwent reduction alone and found that all had recurrence. Those that underwent plication had a 40% recurrence [40]. In our opinion, resection and revision of the jejunojejunostomy leads to more definitive repair as the anastomosis tends to be dilated and an irregular configuration. Our preference is to recreate the jejunojejunostomy with two separate ends to side anastomoses with Roux limb recreated to 100 cm. Postoperative management is similar to postoperative management of SBO and bowel resection.

Biliary Pathologies

Biliary disease after RYGB is a common pathology as rapid weight loss can promote gallstone formation. Symptomatic cholelithiasis and cholecystitis can occur soon after RYGB. The Cleveland Clinic reported a 3.6% rate of developing biliary disease after bariatric surgery. Of those, 5.7% of patients had choledocholithiasis [43]. Tsirline et al. found 10.6% incidence of cholecystectomy after RYGB. Most of these occurred in the first 3 months after an excess weight loss of greater than 25% [44]. The use of ursodeoxycholic acid or ursodiol for the first 6 months after RYGB has been proposed to decrease gallstone formation. Although short-term randomized, multicenter trials indicate a significant reduction in the formation of gallstones postoperatively, the question remains whether or not this is clinically significant. Coupaye et al. reported a 32.5% incidence of gallstones on routine postoperative ultrasound. Those that were treated with ursodeoxycholic acid reduced the incidence to 5.4% [45]. However, it was not known whether this led to clinically relevant symptoms that eventually required a cholecystectomy. At our institution, we institute an optional twice-a-day ursodiol treatment for 6 months. This medication is not often covered by insurance and side effects lead to compliance issues. Patients with symptomatic cholelithiasis prior to RYGB may have concomitant cholecystectomy at primary RYGB if technically safe. In our experience, intraoperative cholangiogram at time of cholecystectomy is recommended as undiagnosed choledocholithiasis can be problematic for after the creation of RYGB.

Choledocholithiasis and other less common biliary pathologies, such as gallstone pancreatitis and sphincter of Oddi dysfunction, remain a challenge as they require an experienced gastroenterologist comfortable with transgastric endoscopic retrograde cholangiopancreatography (ERCP) or the skill set to perform this procedure retrograde through the oral route. Transgastric ERCP is the preferred method as it allows the surgeon to evaluate for other pathology, including internal hernias and alternatives if the ERCP is not possible or successful. The surgeon should be familiar with common bile duct exploration or biliary tract bypass procedures such as choledochoduodenostomy or Roux-en-Y. The technique of transgastric ERCP is not difficult: the surgeon inserts a 15 mm trocar into the stomach remnant with placement of anchoring sutures, places a bowel clamp at the ligament of Treitz to prevent bowel distension, and closes the gastrotomy following the procedure. At times, placement of a hydrophilic wire through the cystic duct and into the duodenum assists the gastroenterologist, especially when there is a duodenal diverticulum. Post-procedure pancreatitis is common but limited. Patients presenting with signs of acute cholecystitis, transaminitis, or other signs of choledocholithiasis should have a planned cholecystectomy with intraoperative cholangiogram with a gastroenterologist on standby for potential ERCP. Alternatively, interventional radiologists have been successful in transhepatic pushing of the common bile duct stones into the duodenum, and spontaneous passage of CBD stones can occur. Expectant management of CBD stones is not recommended except in unusual and high-risk situations.

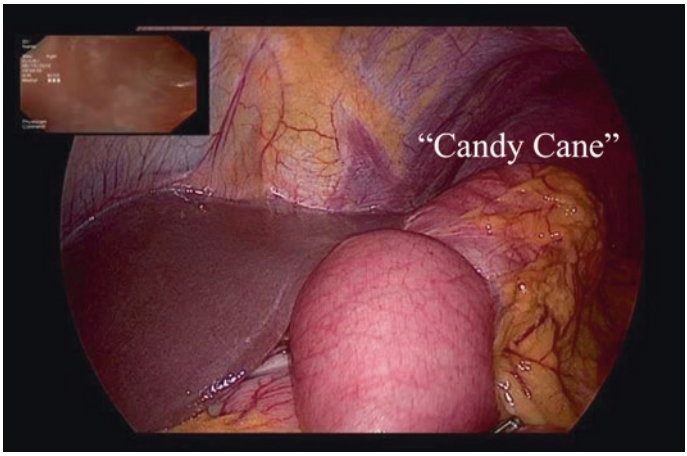

Fig. 10.6 Distended candy cane roux limb causing food trapping and abdominal pain

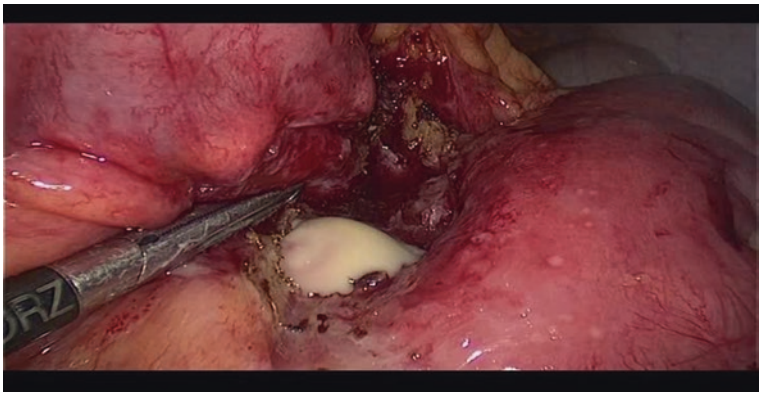

Fig. 10.7 Abscess cavity at mesentery base from migrated permanent suture

Other Less Common Causes for Chronic Abdominal Pain

RYGB patients may present with other less common etiologies for chronic pain. These conditions are important to consider in formulating a differential diagnosis for abdominal pain.

"Candy cane" Roux syndrome or also known as excessive Roux limb deformity can cause chronic abdominal pain. This condition presents insidiously years after the procedure with characteristic postprandial left upper quadrant distension and abdominal pain, relieved by regurgitation of undigested food (Figs. 10.6 and 10.7). This presentation is pathognomonic for this condition and is easily confirmed by UGI series. The etiology is gradual lengthening of the isolated segment over time; prevention is in the initial construct of the GJ – do not leave any redundancy [46, 47]. Resection of the candy cane deformity will lead to resolution of symptoms.

Picture

Suture bezoars can be another uncommon source of abdominal pain months to years after RYGB. Permanent sutures used to close anastomosis or mesenteric spaces can migrate into bowel wall causing a tethering for food to collect and obstruct bowel lumen. This can cause enough food impaction and pressure necrosis of bowel wall that an abscess can form at the mesentery. Symptoms can be vague but usually described as postprandial colicky supraumbilical abdominal pain in the upper abdomen or present as a bowel obstruction. Endoscopy can show the tethered suture that can be removed with endoshears and endoscopic graspers [48, 49]. CT imaging can determine the degree of inflammation at the mesentery. Upper endoscopy is performed to remove the offending suture. Antibiotics and conservative management can generally heal the abscess.

Gastric Remnant Pathologies

The excluded stomach also resides as potential source for chronic abdominal pain. Remnant stomach gastritis, ulcers, and gastroparesis are rare causes of abdominal pain. Diagnosis can be difficult as it may require retrograde upper endoscopy or transgastric endoscopy. However, a trial of PPI and treatment for positive *H. pylori* infections will generally suffice for diagnosis.

Lastly, a subset of patients may continue to have chronic abdominal pain without an anatomical cause after negative workup and surgical exploration. Pain may be attributable to a motility issue such as irritable bowel or psychosomatic pain. In a study of 35 patients at the Cleveland Clinic, 57% of patients with negative findings on exploration required long-term management of chronic pain [12]. Hogestol et al. found that 33.8% of patients reported some form of chronic abdominal pain with majority of complaints similar to irritable bowel syndrome [11]. Multidisciplinary support with dietary and psychologists may help. Nevertheless, these patients can be frustrating to manage, as an anatomic cause cannot explain their symptoms and the bariatric/metabolic surgeon often takes the lead responsibility or blame because of the alteration of anatomy. Ultimately, in severe cases, reversal of the GBP is the only alternative. Not surprisingly, this often does not resolve the pre-existing condition.

Conclusion

Abdominal pain after RYGB is a challenge to health-care providers. The anatomic and physiologic changes that occur after constructing RYGB can lead to a diversity of benign or life-threatening conditions. A thorough history and physical exam can often lead the provider to a correct diagnosis. Surgeons should have a low threshold of safe surgical exploration even with negative workup as pathologies can still be found. A small subset of patients may have unexplained chronic abdominal pain that require long-term management. It is still incumbent on the surgeon and the surgical team to help resolve these issues rather than relegate to other specialists.

References

1. Buchwald H, et al. Bariatric surgery: a systematic review and meta-analysis. JAMA. 2004;292(14):1724–37.
2. Kothari SN, et al. Long-term (>10-year) outcomes after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2017;13:972–8.
3. Higa KD, Boone KB, Ho T. Complications of the laparoscopic Roux-en-Y gastric bypass: 1,040 patients – what have we learned? Obes Surg. 2000;10(6):509–13.
4. Moon RC, et al. Management and treatment outcomes of marginal ulcers after Roux-en-Y gastric bypass at a single high volume bariatric center. Surg Obes Relat Dis. 2014;10(2):229–34.
5. Faintuch J, et al. Severe protein-calorie malnutrition after bariatric procedures. Obes Surg. 2004;14(2):175–81.
6. Rasmussen JJ, Fuller W, Ali MR. Marginal ulceration after laparoscopic gastric bypass: an analysis of predisposing factors in 260 patients. Surg Endosc. 2007;21(7):1090–4.
7. Campos GM, et al. Laparoscopic reversal of Roux-en-Y gastric bypass: technique and utility for treatment of endocrine complications. Surg Obes Relat Dis. 2014;10(1):36–43.
8. Lee CJ, et al. Hormonal response to a mixed-meal challenge after reversal of gastric bypass for hypoglycemia. J Clin Endocrinol Metab. 2013;98(7):E1208–12.
9. Mala T. Postprandial hyperinsulinemic hypoglycemia after gastric bypass surgical treatment. Surg Obes Relat Dis. 2014;10(6):1220–5.
10. Gribsholt SB, et al. Prevalence of self-reported symptoms after gastric bypass surgery for obesity. JAMA Surg. 2016;151(6):504–11.
11. Hogestol IK, et al. Chronic abdominal pain and symptoms 5 years after gastric bypass for morbid obesity. Obes Surg. 2016;27:1438–45.
12. Alsulaimy M, et al. The utility of diagnostic laparoscopy in post-bariatric surgery patients with chronic abdominal pain of unknown etiology. Obes Surg. 2017;27:1924–8.
13. Pitt T, et al. Diagnostic laparoscopy for chronic abdominal pain after gastric bypass. Surg Obes Relat Dis. 2008;4(3):394–8. discussion 398
14. Csendes A, Torres J, Burgos AM. Late marginal ulcers after gastric bypass for morbid obesity. Clinical and endoscopic findings and response to treatment. Obes Surg. 2011;21(9):1319–22.
15. Dallal RM, Bailey LA. Ulcer disease after gastric bypass surgery. Surg Obes Relat Dis. 2006;2(4):455–9.
16. Gumbs AA, Duffy AJ, Bell RL. Incidence and management of marginal ulceration after laparoscopic Roux-Y gastric bypass. Surg Obes Relat Dis. 2006;2(4):460–3.
17. Patel RA, Brolin RE, Gandhi A. Revisional operations for marginal ulcer after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5(3):317–22.
18. Coblijn UK, et al. Development of ulcer disease after Roux-en-Y gastric bypass, incidence, risk factors, and patient presentation: a systematic review. Obes Surg. 2014;24(2):299–309.
19. Sacks BC, et al. Incidence of marginal ulcers and the use of absorbable anastomotic sutures in laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2006;2(1):11–6.
20. Azagury DE, et al. Marginal ulceration after Roux-en-Y gastric bypass surgery: characteristics, risk factors, treatment, and outcomes. Endoscopy. 2011;43(11):950–4.
21. Bhayani NH, et al. Predictors of marginal ulcers after laparoscopic Roux-en-Y gastric bypass. J Surg Res. 2012;177(2):224–7.
22. Bhutta HY, Ashley SW. Hypertension and marginal ulceration after gastric bypass. J Surg Res. 2013;185(2):509–10.
23. Carrodeguas L, et al. Management of gastrogastric fistulas after divided Roux-en-Y gastric bypass surgery for morbid obesity: analysis of 1,292 consecutive patients and review of literature. Surg Obes Relat Dis. 2005;1(5):467–74.
24. Mason EE, et al. Effect of gastric bypass on gastric secretion. Am J Surg. 1976;131(2):162–8.
25. Wilson JA, et al. Predictors of endoscopic findings after Roux-en-Y gastric bypass. Am J Gastroenterol. 2006;101(10):2194–9.

26. Maity P, et al. Smoking and the pathogenesis of gastroduodenal ulcer – recent mechanistic update. Mol Cell Biochem. 2003;253(1–2):329–38.
27. Edholm D, Ottosson J, Sundbom M. Importance of pouch size in laparoscopic Roux-en-Y gastric bypass: a cohort study of 14,168 patients. Surg Endosc. 2016;30(5):2011–5.
28. Capella JF, Capella RF. Gastro-gastric fistulas and marginal ulcers in gastric bypass procedures for weight reduction. Obes Surg. 1999;9(1):22–7. discussion 28
29. Felix EL, et al. Perforated marginal ulcers after laparoscopic gastric bypass. Surg Endosc. 2008;22(10):2128–32.
30. Moon RC, et al. Indications and outcomes of reversal of Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2015;11(4):821–6.
31. Zaveri H, et al. Indications and operative outcomes of gastric bypass reversal. Obes Surg. 2016;26:2285–90.
32. Chau E, et al. Surgical management and outcomes of patients with marginal ulcer after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2015;11(5):1071–5.
33. Elms L, et al. Causes of small bowel obstruction after Roux-en-Y gastric bypass: a review of 2,395 cases at a single institution. Surg Endosc. 2014;28(5):1624–8.
34. Gandhi AD, Patel RA, Brolin RE. Elective laparoscopy for herald symptoms of mesenteric/internal hernia after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5(2):144–9. discussion 149
35. Karila-Cohen P, et al. Contribution of computed tomographic imaging to the management of acute abdominal pain after gastric bypass: correlation between radiological and surgical findings. Obes Surg. 2017;27:1961–72.
36. Higa K, et al. Laparoscopic Roux-en-Y gastric bypass: 10-year follow-up. Surg Obes Relat Dis. 2011;7(4):516–25.
37. Simper SC, et al. Laparoscopic reversal of gastric bypass with sleeve gastrectomy for treatment of recurrent retrograde intussusception and Roux stasis syndrome. Surg Obes Relat Dis. 2010;6(6):684–8.
38. Simper SC, et al. Retrograde (reverse) jejunal intussusception might not be such a rare problem: a single group's experience of 23 cases. Surg Obes Relat Dis. 2008;4(2):77–83.
39. Shaw D, Huddleston S, Beilman G. Anterograde intussusception following laparoscopic Roux-en-Y gastric bypass: a case report and review of the literature. Obes Surg. 2010;20(8):1191–4.
40. Simper SC, et al. Retrograde (reverse) jejunal intussusception might not be such a rare problem: a single group's experience of 23 cases. Surg Obes Relat Dis. 2008;4(2):77–83.
41. Varban O, et al. Resection or reduction? The dilemma of managing retrograde intussusception after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2013;9(5):725–30.
42. Stephenson D, et al. Intussusception after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 10(4):666–70.
43. Chang J, et al. Predictive factors of biliary complications after bariatric surgery. Surg Obes Relat Dis. 2016;12(9):1706–10.
44. Tsirline VB, et al. How frequently and when do patients undergo cholecystectomy after bariatric surgery? Surg Obes Relat Dis. 2014;10(2):313–21.
45. Coupaye M, et al. Evaluation of incidence of cholelithiasis after bariatric surgery in subjects treated or not treated with ursodeoxycholic acid. Surg Obes Relat Dis. 2016;13:681–5.
46. Dallal RM, Cottam D. "Candy cane" Roux syndrome – a possible complication after gastric bypass surgery. Surg Obes Relat Dis. 2007;3(3):408–10.
47. Romero-Mejia C, Camacho-Aguilera JF, Paipilla-Monroy O. "Candy cane" Roux syndrome in laparoscopic gastric by-pass. Cir Cir. 2010;78(4):347–51.
48. Patel C, et al. Use of flexible endoscopic scissors to cut obstructing suture material in gastric bypass patients. Obes Surg. 2008;18(3):336–9.
49. Pratt JS, Van Noord M, Christison-Lagay E. The tethered bezoar as a delayed complication of laparoscopic Roux-en-Y gastric bypass: a case report. J Gastrointest Surg. 2007;11(5):690–2.

Stricture Following Gastric Bypass and Vertical Sleeve Gastrectomy

11

Jacques Himpens

According to the official definition [1], a stenosis is an abnormal narrowing of a passage or orifice in the body, whereas a stricture is an abnormal narrowing of the lumen of a tube, duct, or hollow organ. Consequently, the narrowing of the gastro-jejunostomy of a Roux-en-Y construction represents a stenosis, and the narrowing of the stomach after a sleeve gastrectomy should be named a stricture, because of the absence of a true anastomosis. In "real life," however, both terms (stenosis and stricture) appear to be interchangeable.

Stenosis or Stricture After Roux-en-Y Gastric Bypass

Stenosis after Roux-en-Y gastric bypass (RYGB) most often addresses the gastrojejunostomy (GJ). The jejuno-jejunostomy (JJ) is only seldom affected, except in the acute postoperative setting, but stenosis at that level may be life-threatening and requires prompt operative treatment [2]. The stenosis at the GJ most often occurs around the third to fourth week, and up to 20% of the patients may eventually be affected [3]. Stenosis may also seldom occur quite late, up to several years after the initial operation, mostly in smokers and in connection with a marginal ulcer or the history of a leak or a gastro-gastric fistula [4]. Treatment of late strictures is usually surgical.

Conversely, early stenosis is more frequent but mostly straightforward in terms of diagnosis and treatment [5]. The patient usually notices a clear regression of his/her ability to eat. The indication for treatment is usually set when the patient no longer tolerates a liquid diet and cannot ingest a sufficient amount of protein [6].

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The diagnosis is obtained by esophagogastroscopy (EGS) alone or in conjunction with a barium swallow X-ray [7].

In terms of treatment, mostly, one to two sessions of endoscopic balloon (through-the-scope or TTS) or bougie dilation is all that is required [8], but sometimes more (up to seven or eight) sessions may be needed. The risks of this therapeutic regimen are minimal [9], provided the first dilation session is withheld until 3 weeks postoperatively. Some endoscopists still prefer the Savary-Gilliard dilators because of the haptic feedback dilators provide when the stenotic obstacle is crossed [8]. The failure rate of endoscopic treatment is fairly low, some 5–6.5% [9]. In case of failures, the laparoscopic reconstruction of the anastomosis is the treatment of choice [6].

Recently, self-expanding metal stents (SEMS) have been used in balloon dilation unresponsive cases [10], but success rate is quite poor and patient tolerance limited because since the gastric pouch in bypass is limited in size, the stents usually have to cross the gastroesophageal junction, a situation that is very symptomatic [11]. To address this very issue, very short stents have been designed [12]. Besides poor patient tolerance, SEMS have well-known drawbacks including stent migration (rare when the stents are partly covered) and difficulties at removal because of ingrowth in the mucosa [13]. To alleviate this latter problem, we suggest plastic stents be placed inside the SEMS at the near conclusion of the endoscopic treatment (usually 4–6 weeks) to dislodge the metallic stent with minimal trauma to the hyperplastic mucosal overgrowth [14].

In terms of prevention, of the three usually described techniques of gastrojejunostomy in Roux-en-Y gastric bypass (i.e., the linear-stapled, the circular-stapled, and the manual anastomosis), the linear-stapled anastomosis appears to be the least prone to stenosis [15, 16]. Conversely, our initial technique, the transoral circular-stapled anastomosis with a 25 mm circular stapler (the "Gagner technique") in our hands, was fraught with a 40% stenosis rate Himpens J, unpublished results), which made us abandon the technique. We must mention, however, that in the literature, the circular-stapled anastomosis most commonly is reported as inducing a low incidence of stenosis [17], even with the use of a 21 mm circular stapler [18]. Noteworthy, if one decides to use the 21 mm circular stapler technique, it may be better to use the transoral route that, unlike the transgastric route, appears to achieve the same low stenosis rate (around 5%) as the 25 mm circular stapler technique, introduced either by mouth or via the stomach [19]). If one elects the manual anastomotic technique, monofilament rather than multifilament resorbable suture material should be used [20].

Besides the anastomotic technique, the placement of the Roux limb in a retrocolic rather than an antecolic position appears to be important to avoid postoperative stenosis at the gastrojejunostomy [21].

Interestingly, the *Helicobacter* status does not seem to influence the incidence of stenosis at the gastrojejunostomy [22].

Sleeve Gastrectomy Stenosis

In recent years, the laparoscopic sleeve gastrectomy (SG) has become the most popular weight loss procedure across the world [23]. Despite its apparent technical ease, the sleeve gastrectomy technique continues to be fraught with booby traps, and nowadays complications continue to occur, even in the most experienced hands. One of these complications is stenosis of the gastric body, a condition that is quite rare (between 0% and 9.3% [24]) but the effects are significant. Stenosis most usually occurs at the level of the incisura and is the most frequent cause of postoperative leak, a condition that is usually confined to the proximal staple line [25]. Even in the absence of a leak, a stenosis at the incisura may be highly symptomatic and be accompanied by significant dysphagia and vomiting of thick, white slime [26].

Unlike for Roux-en-Y gastric bypass, the diagnosis of stricture is often quite difficult to achieve because the stenosis may be functional rather than organic and, while highly symptomatic, still may allow unhindered passage of the gastroscope (which is often the most important diagnostic sign of stenosis for endoscopists [24]). Recently, however, new X-ray techniques have emerged to objectively demonstrate the presence of a stenosis of the gastric lumen after sleeve gastrectomy [27]. These techniques allow for three-dimensional reconstruction of the radiological CT scan slices of the stomach. The new imaging techniques help confirm that there appear to be two types of stenosis of the sleeved stomach: first, the well-known and well-described "organic" narrowing of the lumen and, second, the spiral or corkscrew deformity that is characterized by the existence of two distinct axial directions of the gastric tube, which results in a "functional" stenosis [28].

There are several therapeutic options to address the stenosis of the gastric body after sleeve gastrectomy. Conversion to Roux-en-Y gastric bypass is the most frequently cited and probably the most effective and safest option. Laparoscopic conversion of the stenotic sleeve to Roux-en-Y gastric bypass should imply transection of the stomach proximal to the stenosis [29]. However, patients often are reluctant to undergo this treatment mode because the implications of bypassing stomach and duodenum are significant, including the well-known specific dietary restrictions linked with the dumping syndrome and the necessity to take vitamins and minerals. Of note, recent evidence indicates that nutritional supplements are mandatory after sleeve gastrectomy as well [30]. The outcomes of conversion from SG to RYGB are good in the literature [31].

Another quite different therapeutic option is to address the stenosis itself.

One way to achieve this is to incise the stenosis longitudinally through all the stomach layers except for the mucosa, thus creating a seromyotomy (Fig. 11.1a, b). We published this technique on a few occasions [32, 33] and reported acceptable outcomes in terms of reflux cure, but at the cost of a substantial number of complications, such as leaks (occurring in 36% of the cases). All complications could be managed conservatively or laparoscopically, but a number of patients still required conversion to Roux-en-Y gastric bypass because of recurrent stenosis [33].

Another way to address the stenosis itself is to segment the stomach that harbors the stenosis. This technique is particularly seductive in case of corkscrew deformity

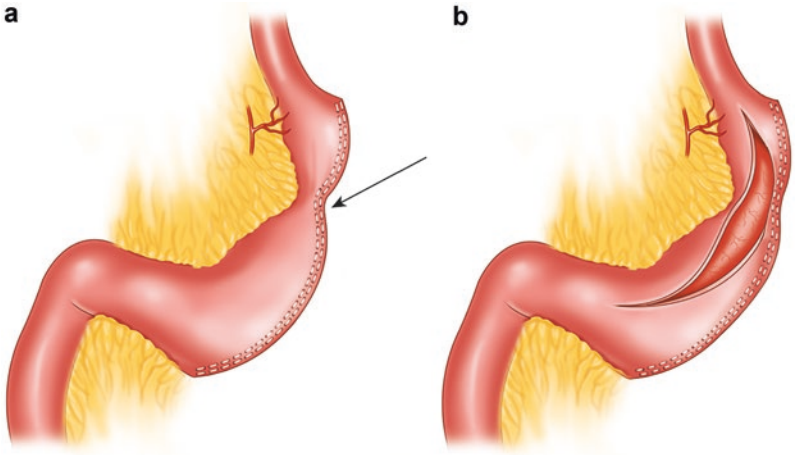

Fig. 11.1 (**a**) Artist impression of a stenosis after vertical sleeve gastrectomy. The *arrow* points to the stenosis. (**b**) The anterior stomach body has been longitudinally incised through serosa and musculosa, sparing the mucosa that now bulges through the incision. The result is a net increase of the diameter of the stomach

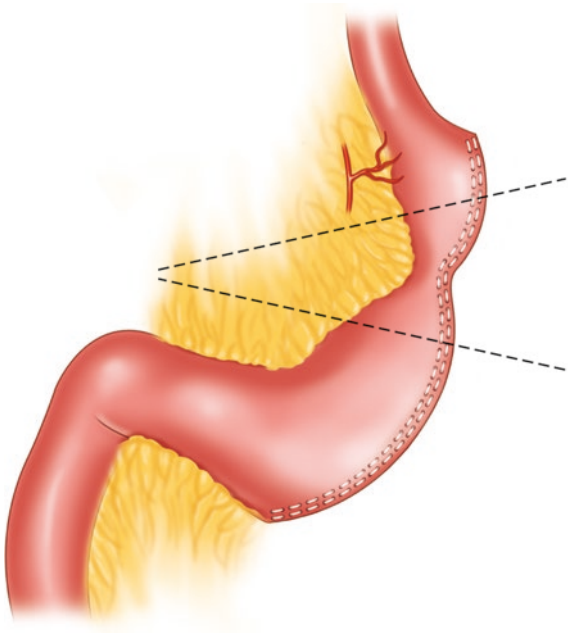

Fig. 11.2 Artist impression of the stenotic part of the vertical sleeve. The *dotted line* shows the level of transection. Note that the left gastric artery is preserved

because it deals with the acute axial angulation. After devascularizing the stenotic part of the stomach and resecting it, a one layer, end-to-end manual anastomosis is performed (Figs. 11.2, 11.3, and 11.4). We called this technique a wedge resection or wedge gastrectomy [33], but others later renamed the technique a median

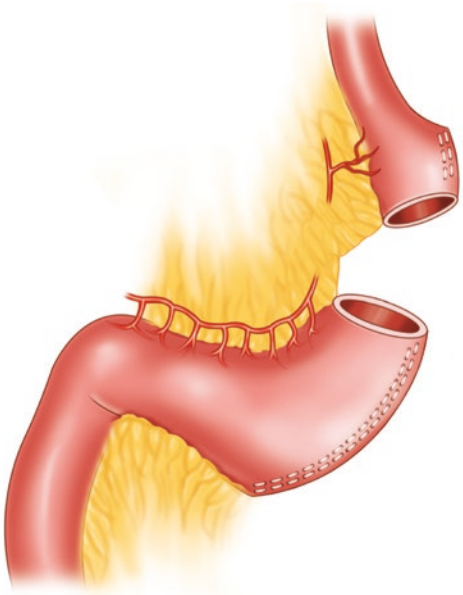

Fig. 11.3 The "wedge resection" has been performed

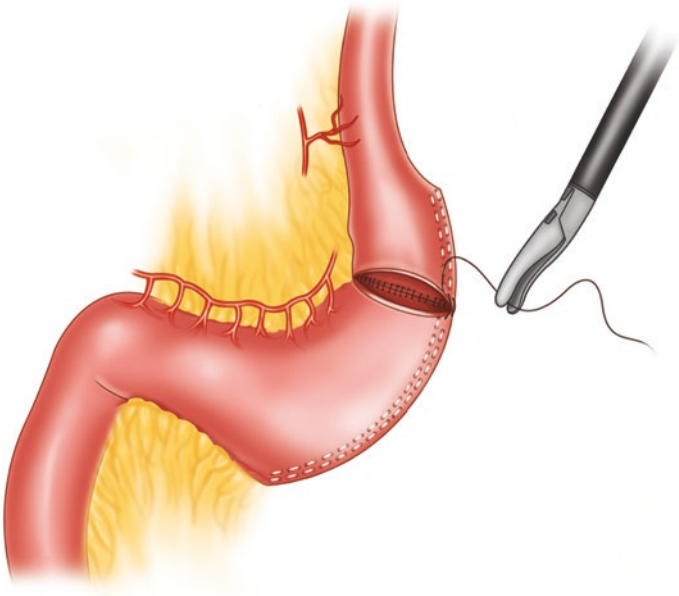

Fig. 11.4 The reanastomosis is being performed: the posterior part has been completed, and the anterior part is being initiated

gastrectomy [34]. The abovementioned technique however does not seem to be foolproof, and recurrence of the stenosis may appear. Recently, endoscopic dilation techniques (with or without a complementary treatment with SEMS) have emerged as the preferred treatment mode in case of sleeve gastrectomy stenosis [35]. With this strategy, Chang et al. [11] reported a success rate of 37%, but 50% of the patients still required conversion to Roux-en-Y gastric bypass. In their recent publication, Nath et al. [24] found that, while close to 10% of the individuals submitted to sleeve gastrectomy had developed stenosis or symptomatic angulation, 69% of those were successfully treated by one or more sessions of balloon dilation. Along the same lines, Burgos et al. [36] reported a success rate of 80% in a small group of patients, the remaining failures being addressed by conversion to Roux-en-Y gastric bypass. In order to improve outcomes, high-pressure balloons [37] are preferable to disrupt the stenosis.

In the face of the recalcitrant nature of stenosis after SG, prevention obviously is of the utmost importance. It appears that intraoperative endoscopy may favorably interfere with the development of postoperative stenosis. Nimeri et al. [38] recently demonstrated that the use of an intraluminal gastroscopy during the confection of the sleeve gastrectomy helped reduce the incidence of stenosis from 3.2% to 0%. Thanks to the intraoperative gastroscopy, the surgeon was able to detect (and, indeed, remove) narrowing oversewing sutures. The danger for stenosis by oversewing had already been stressed years ago by an Italian study [39].

Another significant factor believed to affect complications after SG is the size of the bougie used, the smaller size being linked with a higher leak and stenosis rate [40]. Recent literature data however seems to indicate that a bougie size smaller than 36 French does not appear to cause more complications/stenosis than larger bougie size [41].

Conclusion

Stenosis may occur both after Roux-en-Y gastric bypass and after sleeve gastrectomy. Whereas the condition is rather benign after bypass, at least when occurring rather early after the procedure, stenosis is a quite severe issue after sleeve gastrectomy; it may constitute a facilitating factor for leaks and demand sophisticated endoscopic means to successfully address it.

References

1. Taber's Cyclopedic Medical Dictionary, 23rd edition, ISBN-13:9787-0-8036-5904-9.
2. Cho M, Carrodeguas L, Pinto D, Lascano C, Soto F, Whipple O, Gordon R, Simpfendorfer C, Gonzalvo JP, Szomstein S, Rosenthal RJ. Diagnosis and management of partial small bowel obstruction after laparoscopic antecolic antegastric Roux-en-Y gastric bypass for morbid obesity. J Am Coll Surg. 2006;202(2):262–8.
3. Schneider BE, Villegas L, Blackburn GL, et al. Laparoscopic gastric bypass surgery: outcomes. J Laparoendosc Adv Surg Tech. 2003;13:247–55.

4. Cusati D, Sarr M, Kendrick M, Que F, Swain JM. Refractory strictures after Roux-en-Y gastric bypass: operative management. Surg Obes Relat Dis. 2011;7(2):165–9.
5. Sanyal AJ, Sugerman HJ, Kellum JM, Engle KM, Wolfe L. Stomal complications of gastric bypass: incidence and outcome of therapy. Am J Gastroenterol. 1992;87:1165–9.
6. Rosenthal R. Dilating the stenotic gastrojejunostomy after laparoscopic Roux-en-Y gastric bypass for morbid obesity: when things go wrong. J Gastrointest Surg. 2009;13(9):1561–3.
7. Lee JK, Van Dam J, Morton JM, Curet M, Banerjee S. Endoscopy is accurate, safe, and effective in the assessment and management of complications following gastric bypass surgery. Am J Gastroenterol. 2009;104:575–82.
8. Valli PV, Gubler C. Review article including treatment algorithm: endoscopic treatment of luminal complications after bariatric surgery. Clin Obes. 2017;7(2):115–22.
9. De Moura EG, Orso IR, Aurélio EF, de Moura ET, de Moura DT, Santo MA. Factors associated with complications or failure of endoscopic balloon dilation of anastomotic stricture secondary to Roux-en-Y gastric bypass surgery. Surg Obes Relat Dis. 2016;12(3):582–6.
10. Puig CA, Waked TM, Baron TH Sr, Wong Kee Song LM, Gutierrez J, Sarr MG. The role of endoscopic stents in the management of chronic anastomotic and staple line leaks and chronic strictures after bariatric surgery. Surg Obes Relat Dis. 2014;10(4):613–7.
11. Chang J, Sharma G, Boulis M, Brethauer S, Rodriguez J, Kroh M. Endoscopic stents in the management of anastomotic complications after foregut surgery: new applications and techniques. Surg Obes Relat Dis. 2016;12(7):1373–81.
12. Uchima H, Abu-Suboh M, Mata A, Cruz M, Espinos J. Lumen-apposing metal stent for the treatment of refractory gastrojejunal anastomotic stricture after laparoscopic gastric bypass. Gastrointest Endosc. 2016;83(1):251.
13. Wei W, Ramaswamy A, de la Torre R, Miedema BW. Partially covered esophageal stents cause bowel injury when used to treat complications of bariatric surgery. Surg Endosc. 2013;27(1):56–60.
14. ElMourad H, Himpens J, Verhofstadt J. Stent treatment for fistula after obesity surgery: results in 47 consecutive patients. Surg Endosc. 2013;27(3):808–16.
15. Awad S, Aguilo R, Agrawal S, Ahmed J. Outcomes of linear-stapled versus hand-sewn gastrojejunal anastomosis in laparoscopic Roux-en-Y gastric bypass. Surg Endosc. 2015;29(8):1278–83.
16. Lee S, Davies AR, Bahal S, Cocker DM, Bonanomi G, Thompson J, Efthimiou E. Comparison of gastrojejunal anastomosis techniques in laparoscopic Roux-en-Y gastric bypass: gastrojejunal stricture rate and effect on subsequent weight loss. Obes Surg. 2014;24(9):1425–9.
17. Qureshi A, Podolsky D, Cumella L, Abbas M, Choi J, Vemulapalli P, Camacho D. Comparison of stricture rates using three different gastrojejunostomy anastomotic techniques in laparoscopic Roux-en-Y gastric bypass. Surg Endosc. 2015;29(7):1737–40.
18. Rondan A, Nijhawan S, Majid S, Martinez T, Wittgrove A. Low anastomotic stricture rate after Roux-en-Y gastric bypass using a 21-mm circular stapling device. Obes Surg. 2012;22(9):1491–5.
19. Khoraki J, Funk LM, Greenberg JA, Leverson G, Campos GM. The effect of route of anvil insertion on stricture rates with circular stapled gastrojejunostomy during laparoscopic gastric bypass. Obes Surg. 2016;26(3):517–24.
20. Ruiz de Adana JC, Hernández Matías A, Hernández Bartolomé M, Manzanedo Romero I, Leon Ledesma R, Valle Rubio A, López Herrero J, Limones Esteban M. Risk of gastrojejunal anastomotic stricture with multifilament and monofilament sutures after hand-sewn laparoscopic gastric bypass: a prospective cohort study. Obes Surg. 2009;19(9):1274–7.
21. Ribeiro-Parenti L, Arapis K, Chosidow D, Dumont JL, Demetriou M, Marmuse JP. Gastrojejunostomy stricture rate: comparison between antecolic and retrocolic laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2015;11(5):1076–84.
22. Rawlins L, Rawlins M, Brown C, Schumacher D. Effect of helicobacter pylori on marginal ulcer and stomal stenosis after Roux-en-Y gastric bypass. SOARD. 2013;9(5):760–4.
23. Ponce J, Nguyen NT, Hutter M, Sudan R, Morton JM. American Society for Metabolic and Bariatric Surgery estimation of bariatric surgery procedures in the United States, 2011–2014. Surg Obes Relat Dis. 2015;11(6):1199–2000.

24. Nath A, Yewale S, Tran T, Brebbia JS, Shope TR, Koch TR. Dysphagia after vertical sleeve gastrectomy: evaluation of risk factors and assessment of endoscopic intervention. World J Gastroenterol. 2016;22(47):10371–9.
25. Rosenthal RJ, International Sleeve Gastrectomy Expert Panel, Diaz AA, Arvidsson D, Baker RS, Basso N, Bellanger D, Boza C, El Mourad H, France M, Gagner M, Galvao-Neto M, Higa KD, Himpens J, Hutchinson CM, Jacobs M, Jorgensen JO, Jossart G, Lakdawala M, Nguyen NT, Nocca D, Prager G, Pomp A, Ramos AC, Rosenthal RJ, Shah S, Vix M, Wittgrove A, Zundel N. International Sleeve Gastrectomy Expert Panel Consensus Statement: best practice guidelines based on experience of >12,000 cases. Surg Obes Relat Dis. 2012;8(1):8–19.
26. Rebibo L, Hakim S, Dhahri A, Yzet T, Delcenserie R, Regimbeau JM. Gastric stenosis after laparoscopic sleeve gastrectomy: diagnosis and management. Obes Surg. 2016;26(5):995–1001.
27. Blanchet MC, Mesmann C, Yanes M, Lepage S, Marion D, Galas P, Gouillat C. 3D gastric computed tomography as a new imaging in patients with failure or complication after bariatric surgery. Obes Surg. 2010;20:1727–33.
28. Donatelli G, Dumont JL, Pourcher G, Tranchart H, Tuszynski T, Dagher I, Catheline JM, Chiche R, Marmuse JP, Dritsas S, Vergeau BM, Meduri B. Pneumatic dilation for functional helix stenosis after sleeve gastrectomy: long-term follow-up (with videos). Surg Obes Relat Dis. 2017;13(6):943–50.
29. Lacy A, Ibarzabal A, Pando E, Adelsdorfer C, Delitala A, Corcelles R, Delgado S, Vidal J. Revisional surgery after sleeve gastrectomy. Surg Laparosc Endosc Percutan Tech. 2010; 20(5):351–6.
30. Ben-Porat T, Elazary R, Goldenshluger A, Sherf Dagan S, Mintz Y, Weiss R. Nutritional deficiencies four years after laparoscopic sleeve gastrectomy-are supplements required for a lifetime? Surg Obes Relat Dis. 2017;13:1138–44.
31. Felsenreich DM, Kefurt R, Schermann M, Beckerhinn P, Kristo I, Krebs M, Prager G, Langer FB. Reflux, sleeve dilation, and Barrett's esophagus after laparoscopic sleeve gastrectomy: long-term follow-up. Obes Surg. 2017;27:3092–101.
32. Dapri G, Cadière GB, Himpens J. Laparoscopic seromyotomy for long stenosis after sleeve gastrectomy with or without duodenal switch. Obes Surg. 2009;19(4):495–932.
33. Vilallonga R, Himpens J, Van de Vrande S. Laparoscopic management of persistent structures after laparoscopic sleeve gastrectomy. Obes Surg. 2013;23(10):1655–61.
34. Kalaiselvan R, Ammori BJ. Laparoscopic median gastrectomy for stenosis following sleeve gastrectomy. Surg Obes Relat Dis. 2015;11(2):474–722.
35. Zundel N, Hernandez JD, Galvao Neto M, et al. Strictures after laparoscopic sleeve gastrectomy. Surg Laparosc Endosc Percutan Tech. 2010;20(3):154–8.
36. Burgos AM, Csendes A, Braghetto I. Gastric stenosis after laparoscopic sleeve gastrectomy in morbidly obese patients. Obes Surg. 2013;23(9):1481–6.
37. Zundel N, Neto M. Comment on: pneumatic dilation for functional helix stenosis after sleeve gastrectomy: long-term follow-up (with videos). Surg Obes Relat Dis. 2017;13(6):950.
38. Nimeri A, Maasher A, Salim E, Ibrahim M, El Hadad M. The use of intraoperative endoscopy may decrease postoperative stenosis in laparoscopic sleeve gastrectomy. Obes Surg. 2016;26(7):1398–401.
39. Musella M, Milone M, Bellini M, Leongito M, Guarino R, Milone F. Laparoscopic sleeve gastrectomy. Do we need to oversew the staple line? Ann Ital Chir. 2011;82(4):273–7.
40. Gagner M, Hutchinson C, Rosenthal R. Fifth International Consensus Conference: current status of sleeve gastrectomy. Surg Obes Relat Dis. 2016;12(4):750–6.
41. Guetta O, Ovnat A, Czeiger D, Vakhrushev A, Tsaban G, Sebbag G. The impact of technical surgical aspects on morbidity of 984 patients after sleeve gastrectomy for morbid obesity. Obes Surg. 2017;27:2785–91.

Weight Regain Following Bariatric Surgery and Revisional Surgery

12

Cynthia Weber and Bipan Chand

Introduction

Bariatric surgery has emerged as the most successful treatment modality or "gold standard" available for morbid obesity and its associated comorbidities, especially type 2 diabetes. However, weight recidivism can occur after any intervention, including surgery. Treatment options for weight regain are also seldom mentioned in consensus statements and practice guidelines. It can even be argued that significant weight regain post-bariatric surgery should be regarded as the most important outcome when examining the lasting effects of surgery as it has medical, societal, and economical implications. The exact incidence of weight regain is poorly understood and known and also contributes to the philosophy that obesity is a chronic disease. Weight regain often correlates with negative consequences, for example, the recurrence of obesity-related comorbidities such as type 2 diabetes [1–3], impairment in quality of life and overall patient experience, and significant cost burden that threatens to offset the cost-benefit of bariatric surgery. Thus, in order to prevent this detrimental complication, providers need to increase their focus and attention to the predictors of weight regain following surgery and develop effective therapies targeting those individuals who suffer from this.

After surgery, such as the Roux-en-Y gastric bypass (RYGB) or the vertical sleeve gastrectomy (VSG), patients describe a "honeymoon period" that lasts approximately 18–24 months. During this time frame, patients experience significant weight loss and reach a nadir weight [4]. A large proportion of patients will subsequently stop losing weight or have the propensity to gain some weight back. In the literature, this weight regain usually occurs between years 2 and 5

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postoperatively. When this weight gain becomes detrimental to health, physical functioning, or medical comorbidities, then it can be considered significant. This period has been described as "back to reality" [4]. In addition, insufficient patient follow-up, as reported in many studies, hinders the accuracy of gauging the true prevalence of long-term weight regain. It has been hypothesized that patients who begin to suffer from weight regain will experience embarrassment and thus are potentially less likely to keep scheduled follow-up visits. Successful interventions are needed to target this population of patients after weight regain occurs or, ideally, to identify patients at risk perioperatively to prevent significant weight regain altogether.

How to Define Weight Regain?

One of the main obstacles to both prevention and recognition of weight regain is the lack of a standard definition of weight regain itself. This ambiguity also limits effective comparison among studies and, in some ways, potentially hinders surgeons', obesity medicine specialists', and dieticians' ability to intervene. Among descriptions, weight regain can be defined as number of kilograms gained after nadir postoperative weight, increase in BMI units, percent excess weight loss (%EWL) gained after lowest postoperative weight, or percent total weight loss regained [5–10]. Some authors define weight regain by comparing the numerical amount or percent of weight gained to the amount of weight lost at a specific time interval after surgery, most commonly 1–2 years postoperatively [11, 12]. Common quantifications used are gain of more than 10 kg, increase in BMI of 5 points or more, or regain of greater than 10%, 15%, 20%, or 25% of excess or total weight lost [5, 7, 11, 13–17]. A social media poll of bariatric surgeons found that there was no consensus on the definition of weight regain, which has been born out in the literature [18]. Felsenreich et al. argue for using an absolute value of weight regain as opposed to a percentage of weight lost to avoid creating an individual threshold for each patient [9].

Other terms used for weight regain include weight reacquisition, secondary weight gain, and weight recidivism. In addition, a distinction must be made between weight regain, which is an increase in weight after an initial period of successful postsurgical weight loss, and insufficient weight loss or surgical failure, which is generally defined as a weight loss of <50% EWL after gastric bypass. However, it is probable that initial suboptimal weight loss and weight regain postoperatively share some common features that are likely either within the domain of physiologic mechanisms that regulate appetite and metabolic rate or due to patient-specific behaviors.

Weight Regain After RYGB

The RYGB is a very effective and relatively well-tolerated weight loss operation. Postoperatively the majority of patients experience resolution or significant improvement of obesity-related comorbidities, such as type 2 diabetes, hypertension,

and hypertriglyceridemia [19]. Perioperatively, patients are generally quoted an average of 57–70% EWL [20–22]. Not only does this degree of weight loss positively affect measurable medical outcomes but it also improves patients' mobility, psychological well-being, and overall quality of life. Bariatric medical and surgical providers counseling patients on realistic expectations after surgery should provide long-term outcomes regarding the likelihood of at least some degree of weight regain in the years following surgery after a weight nadir around 18 months after gastric bypass. Thus, most patients accept a small percentage of weight gain after lowest recorded weight. However, one study that instructed patients to write down potential outcomes (including complications) after RYGB reported that only 10% of participants remembered to include weight regain [23]. This finding is somewhat alarming and reflects either a deficit in preoperative patient education or a lack of understanding by the patients' part on the chronicity of the disease. Finally, as the exact mechanisms for weight loss after RYGB are relatively unknown or least controversial, it is harder to accurately determine the predictive factors for significant weight regain after bariatric surgery.

The prevalence of weight regain after RYGB is generally quoted as averaging around 20% [22, 24–26], but in the gastric bypass literature, there is a large range (17–64%) [8, 11–13, 16, 20, 24, 27, 28] based on measurement criteria (how weight regain is defined) and length of time after surgery (2–10 years). For example, Cooper et al. report a 23% mean weight regain from nadir weight over an average of 7 years of follow-up among 276 respondents, assessed via self-administered questionnaires. They also quote a 37% rate of excessive weight regain (defined as ≥25% weight gain from postoperative year 1). These authors further categorized the patients into four groups based on mean weight loss at 1 year (<25% ($n = 39$), 25–30% ($n = 51$), 30–35% ($n = 73$), >35% ($n = 113$)). They found no statistically significant difference between groups in either the mean percent of weight regained or the percent of patients with excessive weight regain, thus implying that degree of initial weight loss does not necessarily predict weight regain [13]. A study by Shantavasinkul et al. over a mean follow-up period of 6 years reported that 244 out of 1426 patients (17.1%) experienced significant weight regain, defined as gaining ≥15% of their weight at 1 year postoperatively. Among the individuals in the weight regain group, the mean %EWL at 6 years follow-up was 47% and the mean percent weight regain was 23% [11].

In another study, conducted retrospectively, approximately 24%, or 19 out of 80 patients, had weight regain (regain of 10% or more of lowest postoperative weight), with a mean weight regain of 20% [24]. A smaller study of ten patients noted weight regain from 12% to 56% [20]. A study done in 2007 of 782 patients observed weight regain in 46% of patients at 2 years and 64% at 4 years. The mean increase in weight at 5 years was 8.8 kg (8% of nadir weight at 18 months) among patients with significant weight regain [12]. Finally, another study found that weight regain (defined as >5% increase in weight between years 1 and 2 postoperatively) occurred in 33% of patients [28].

As expected, predictors of weight regain following RYGB vary in the literature but, in general, encompass either patient-specific factors or surgery-specific factors

Etiology of Weight Regain after Bariatric Surgery

Patient-Specific Factors	Surgery-Specific Factors
Amount of physical activity	Dilation of gastrojejunal stoma
Mental health issues	Gastro-gastric fistula
Nutritional compliance	Gastric pouch length
Follow-Up	Greater residual gastric volume
Preoperative variables	Dilation of gastric sleeve
Hormonal imbalance	Retained fundus
Support group attendance	
Control of food urges/ emotional eating	

Fig. 12.1 Etiology of weight regain after bariatric surgery

(Fig. 12.1). Many of these prognosticators can also be applied to other bariatric operations including the VSG. Patient-specific factors for weight regain after bariatric surgery include lifestyle factors such as compliance with nutritional recommendations and follow-up; frequency and amount of physical activity; mental health issues including mood disorders, self-esteem issues, and substance abuse; eating behaviors such as binge eating, grazing, control of food urges, and emotional eating; preoperative variables such as BMI before surgery, age, race, patient genetic predispositions, and baseline physiologic or metabolic set points; or hormonal/metabolic imbalances [4–7, 10, 11, 13, 14, 16, 17, 20, 24, 27, 29]. Many of the above patient-specific factors are the same issues that patients struggle within programs that focus on nonoperative weight loss, whereas the formation of a gastro-gastric fistula, dilation of the gastrojejunal stoma, pouch dimensions (length and width), and time since surgery are classified as surgery-specific factors [17, 30–44]. Numerous studies have examined possible predictive or etiologic factors, and there is a general lack of consensus and even controversy in the literature. In addition, there is likely a component of weight regain or surgical failure that is related to patient selection or the inappropriate choice of surgical technique. The reality is that weight regain is multifactorial and thus difficult to prevent and treat. Also, some patients tend to experience weight regain despite strict compliance with advised behavioral changes and finding no attributable postsurgical anatomic issues.

A well-quoted systematic review of 16 articles classified the etiology of weight recidivism into 5 domains: nutritional noncompliance, mental health issues, endocrine or metabolic imbalance, physical inactivity, and anatomic surgical factors [10]. Some of the nutritional reasons in these articles were increased caloric intake over time or overall poor diet quality, grazing, uncontrolled eating, food indiscretion, and inappropriate or insufficient nutritional counseling and follow-up. Hormonal

factors identified were abnormally high ghrelin levels and low peptide YY levels as well as a reactive hypoglycemia. Mental health issues such as attention deficit hyperactivity disorder (ADHD), the presence of an eating disorder such as binge eating disorder perioperatively, impulsive behavioral traits, and two or more diagnosed psychiatric conditions were all reported as being contributors to weight regain. Physical inactivity, often defined as less than 30 min for 3–4 days per week or as lack of a baseline physical activity regimen, was also an associated factor [10]. While the American Society for Metabolic and Bariatric Surgery (ASMBS) recommends 30 min of daily physical activity [20], no definitive data exists regarding the exact amount of exercise required to prevent significant weight regain. Finally, some of the surgical factors described included a dilated gastric stoma, dilated gastric pouch, presence of a gastro-gastric fistula, and, in VSG patients, dilation of the sleeve [10].

When looking at individual studies, it is difficult to compare conclusions due to differences in study design and a lack of standard definitions. However, it is still worthwhile to consider the results reported. One study found that at both 96 and 120 months of follow-up, younger patients were more likely to experience significant weight regain. Other variables such as amount of preoperative excess weight, preoperative BMI, gender, time since surgery, and nutritional follow-up were not identified as predictive factors by these authors [16]. Whereas another study corroborated that younger age was a predictive factor for weight regain, it found that the longer the duration after surgery, the more the weight gain [11].

A study of 23 variables found the following to be risk factors for weight regain on multivariate analysis: preoperative BMI, time since surgery, first year change in physical activity, control of both portion size and food urges, and emotional eating. Current or preoperative smoking or drinking status, age, gender, and race were not statistically significant [13]. Data from the Swedish Obese Subjects (SOS) study was used to test whether or not single-nucleotide polymorphisms (SNPs) in 11 obesity candidate genes were linked with weight regain at 6 years of follow-up. This research failed to find any statistical significance between the examined SNPs and weight recidivism [45]. Despite this negative result, it is entirely possible that genetic predisposition does exist and will be born out in future research. Finally, a retrospective study identified both length of time since surgery and poor diet quality (defined as a low healthy eating index, e.g., eating less fruit and less grains) as predictive of weight regain. Other variables such as education level, age, gender, nutritional counseling attendance, preoperative BMI, maximum %EWL, and percentage of weight lost preoperatively were not statistically significant [24].

Another seldom-surveyed aspect of weight regain is the patients' perspective on causes, emotional ramifications, and their overall experience with this potential outcome. A qualitative interview-based study by Jones et al. was one of the first studies to elucidate a multitude of informative comments from patients. An overarching theme was that after years of struggling with their weight and maladaptive eating behaviors, these patients experienced a relief after RYGB as they started to lose weight and thus adopted a passive role. This passivity garnered them helpless when they started to regain significant weight. The patient responses encompassed

a wide spectrum of issues. Patients reported that they were unprepared for weight regain because either they were unaware that it could occur after RYGB, they felt it occurred at an unnoticeable rate, or they spent a shorter amount of time at their weight nadir than they anticipated. Many of these participants, especially those with higher weight regain, reported feeling worthless; recounted guilt and shame; thought they were at fault; described believing they were abandoned by the healthcare providers; reported feeling disempowered, judged, and vulnerable; or described experiencing social isolation. Some went so far as to say that surgery was not worth it. Others identified that perhaps more regular access to psychological support would have prevented their weight regain [20].

Intuitively, the presence of a gastro-gastric fistula between the pouch and the retained fundus of the stomach is an easily explained etiology for weight regain after RYGB. This complication is often an indication for revisional bariatric surgery. On the other hand, until recently, gradual enlargement of the gastric pouch or dilatation of the gastrojejunal anastomosis has been proposed as a mechanism of weight regain without substantial evidence for this theory. The thought is that a dilation of the anastomosis results in a loss of restriction, whereas pouch enlargement increases the capacity of the stomach and therefore a perceived lack of fullness. In the age of vertical banded gastroplasty, the distal gastric pouch was banded to potentially prevent these outcomes. These nonadjustable bands or foreign bodies have also been placed during gastric bypass around the gastric pouch but have failed to become adopted into common practice secondary to lack of efficacy as well as increased complications.

A study out of Harvard published in 2011 measured the diameter of the gastrojejunal stoma endoscopically on 165 consecutive patients referred to their center after RYGB for a variety of symptoms [17]. Fifty-nine percent (or 97 patients) experienced significant weight regain, defined as ≥20% of maximal weight lost after surgery. Using a multivariate linear regression model, they reported that, after 5 years, for every 10 mm increase in the gastrojejunal stomal diameter, there was an associated 8% increase in the percentage of maximal weight lost that was regained. Other factors found to be predictive in their model include time since surgery and the presence of a marginal ulcer, with time since surgery being positively associated with weight regain and the presence of a marginal ulcer being a protective factor from weight regain. On the other hand, the length of the gastric pouch was not significantly associated with postoperative weight regain. Another aspect of their study was the development of a prediction model for weight regain based on a 7-point scoring system quantified using the variables: diameter of gastrojejunal stoma, patient race, and percentage of maximal weight lost. The positive predictive value of significant weight regain was 75% in patients with a total score of ≥4 points [17].

The above authors and others offer multiple hypotheses of how a dilated gastrojejunal anastomosis contributes to weight regain after RYGB. It is possible that earlier emptying of the gastric pouch counteracts the feeling of early satiety induced by the gastric bypass, and thus these patients experience hunger more rapidly after

eating. In addition, it is thought that perhaps the dilatation of the gastrojejunal stoma is associated with generalized increased compliance of the stomach; thus, a larger quantity of food can be consumed before the mechanoreceptors of the gastric wall are stimulated. Also, the hunger hormone ghrelin is left unopposed for a longer period of time. Finally, if nutrients are more promptly emptied from the gastric pouch, jejunal enteroendocrine cells that regulate energy expenditure and the secretion of insulin are activated sooner and alter physiologic responses to hunger and satiety [6, 17].

Weight Regain After VSG

The VSG, also referred as laparoscopic sleeve gastrectomy or LSG, is quickly becoming the most popular bariatric surgery performed. A recent approximation from the ASMBS (https://asmbs.org/resources/estimate-of-bariatric-surgery-numbers) estimates that of the 196,000 bariatric operations performed in 2015, VSG represented more than half (54%) compared to RYGB that constituted only 23%. Abraham et al. found that the proportion of RYGBs performed between 2008 and 2014 decreased from 52% to 34%, whereas the proportion of VSGs performed increased from 3% to 54% [46]. Many patients are opting for sleeve gastrectomy because it is perceived as "less invasive," does not involve rearrangement of the gastrointestinal tract, and produces fewer vitamin and mineral deficiencies than the RYGB, which, for some patients, makes it easier to comprehend and live with over the long term.

Originally described as the first stage in a two-stage procedure such as the biliopancreatic diversion (BPD) or, later, the biliopancreatic diversion with duodenal switch (BPD-DS), the VSG has proven to be effective as a sole operation. While the weight loss produced from the VSG is less than that induced by the RYGB, it is still significant, with estimates of about 50–60% EWL at 5 years, and can also be associated with resolution of obesity-related comorbidities [5, 47, 48]. The long-term outcomes following VSG are not as robust as the data that exists for RYGB, as the VSG has only been performed with considerable frequency since about 2009. The mechanisms of weight loss are similar and include gastric restriction, change in gastric emptying, hormonal factors, and modification of eating behaviors.

It is thought that the prevalence of weight regain after VSG is comparable to the numbers seen after RYGB [49]. A large proportion of the predictors of weight regain following RYGB, though, are also applicable to patients undergoing VSG. Many of these, as detailed above, fall into the category of patient-specific factors such as noncompliance with nutritional guidelines and follow-up, lack of appropriate physical activity, mental health issues, and the inability to sustain the necessary behavior changes needed for maintenance of long-term weight loss [4, 5]. Furthermore, as there is a relative lack of standardization of the surgical technique, there are a handful of surgery-specific factors that have been researched as potential contributors to weight regain following VSG. These include sleeve dilation, retained

gastric fundus, greater residual gastric volume, and other technical factors specific to this operation such as the size of bougie used and the amount of antrum resected [5, 9, 14, 15, 49] (Fig. 12.1).

A study of 5-year outcomes after VSG reported the prevalence of weight gain among these 168 participants at 44%, using the definition of significant weight regain as the regaining of >10 kg from nadir weight [50]. A systematic review of 21 papers found the range of weight regain at 2 years postoperatively to be very broad, with estimates from 5.7% to 75.6% depending on multiple factors, including the definition of weight regain used [5]. Furthermore, another study reported a range of weight regain from 3% to 130% postoperatively [14]. These varying results further confirm that there is no standard way to report the incidence of weight regain.

A retrospective study of prospectively collected data determined that the magnitude of gastric antral resection is associated with weight loss and weight regain after VSG. These authors divided their sample into two groups: Group A, antral remnant length of 6 cm from the pylorus, and Group B, antral remnant length of 2 cm from the pylorus. They found that patients in Group B experienced superior weight loss compared to patients in Group A at all time points measured. In addition, patients in Group A suffered more often (22% vs 4%) from weight regain (defined as increase in body weight of more than 10 kg from weight loss nadir) [15]. Deguines et al. conducted a study in which two separate radiologists measured postsurgical residual gastric volumes using computed tomographic (CT) volumetry. A residual gastric volume of 225 cc represented the threshold above which there was an associated failure rate of approximately 80% [49]. A retrospective study performed at a median of 38 months of follow-up found that while there was no difference between those with and without significant weight regain (defined as regain of 25% or more of weight lost) with regard to preoperative BMI, there were multiple factors that did reach statistical significance. A greater residual gastric volume was one such factor associated with a higher percentage of weight regain [14]. Finally, when examining 10-year results of some of the earliest performed VSGs, no correlation was observed between the size of bougie used to create the sleeve and weight regain [9].

Based on the observation that discharge from bariatric follow-up at 18 months postoperatively was associated with the onset of weight regain, one set of authors utilized exploratory focus group discussions of patients who had experienced weight regain to define patient-perceived causes of this long-term finding. Qualitatively, the responses generated from these discussions fell into four different domains: psychological (stress, eating disorders, emotional eating), health (pregnancy, injury, dentition, and joint problems), cost (good food and access to exercise facilities is expensive), and support (availability of individualized help to provide motivation and information). Overall, the participants indicated that they felt 18 months was not a long-enough follow-up period. They desired additional support either in the form of good rapport with a healthcare provider or more peer-directed support such as the creation of a buddy system, formation of support groups that could even be text-based/Internet-based [4].

Influence of Gut Hormones on Weight Loss and Weight Regain

Enteroendocrine cells (EECs) of the proximal gastrointestinal (GI) tract release peptide hormones in response to luminal nutrients. Complex interplay between the 28 amino acid orexigenic peptide ghrelin and the 167 amino acid anorexigenic peptide leptin regulates the sensations of hunger and satiety, respectively [28]. Other gut hormones such as glucagon-like polypeptide (GLP-1), peptide YY (PYY), and gastric inhibitory peptide (GIP) as well as the pancreatic exocrine hormones insulin and glucagon are also implicated in glucose homeostasis or food intake [22, 25]. GLP-1, an incretin, and PYY induce satiety, whereas studies have shown that GIP does not play a substantial role in hunger or satiety [51]. Multiple studies have examined the response of these hormones to either medically induced weight loss or weight loss that occurs after bariatric surgery [22, 25, 28, 51–54]. In addition, attempts have been made to determine the involvement of these peptides in weight regain. Despite preliminary research, however, there still exists controversy regarding the definitive role of many of these peptide hormones.

For example, one study has shown that both the diurnal pattern and the pre- and post-prandial responses of plasma ghrelin levels are undermined after bariatric surgery, specifically after the RYGB. They demonstrated that plasma ghrelin levels were paradoxically significantly decreased in patients who underwent RYGB. These authors hypothesize that a possible etiology for this phenomenon is because the gastric bypass isolates the majority of the stomach and the entire duodenum from luminal nutrients [53]. Therefore, the stimulation of ghrelin-producing cells does not occur. Similarly, results from 5 years of follow-up after VSG corroborated the above data as plasma ghrelin levels decreased and remained low after surgery [54].

Santo et al. divided post-RYGB patients into three groups: Group A1, patients who had a maximum %EWL >50% and a weight regain of <10% of weight lost; Group A2, patients who had a maximum %EWL >50% and a weight regain of 10–50% of weight lost; and Group B, patients with unsuccessful postoperative weight loss and a weight regain of >50% of weight lost. Serum levels of gut-derived peptide hormones were measured at baseline and at several time points after a meal in all participants. Interestingly, no difference was seen in baseline and post-prandial levels of ghrelin between groups. On the other hand, both GIP and GLP-1 levels were increased in Group A at 30 min after a meal compared to Group B. Lastly, leptin levels were higher in Group B compared to Group A [22]. Overall, this data suggests the response of gut hormones to weight loss, and weight regain is extremely complex.

The data from Santo et al. favor the theory that ghrelin is not associated with postsurgical weight regain [22]. In contrast, data from 5-year follow-up after VSG found slightly higher levels of ghrelin in patients with weight regain, although it did not reach statistical significance [54]. Another study demonstrated that plasma ghrelin levels were increased after diet-induced weight loss. To add to the uncertainty, in patients who had undergone diet-induced weight loss, ghrelin levels were actually lower in patients who regained weight at baseline and at all time points studied [52].

Thus, at this time, it remains difficult to determine the exact role of ghrelin in weight regain, making it hard to develop targeted therapies.

When assessing preoperative levels of the main hunger and satiety hormones, one study found a difference in preoperative ghrelin levels among patients who underwent RYGB. They found higher levels of preoperative ghrelin in patients who regained ≥5% of weight lost compared to those who maintained or lost weight between years 1 and 2. No difference was seen in preoperative leptin levels, however [28]. This data indicates that perhaps targeted interventions to prevent weight regain should be aimed at patients with significantly higher levels of ghrelin preoperatively.

The involvement of leptin and PPY in weight loss and weight regain has also been further examined. For example, in one study, higher baseline levels of leptin were associated with weight regain after diet-induced weight loss. Perhaps this is because obesity itself is associated with elevated leptin due to the development of leptin resistance. In an experiment utilizing a rat model, in which diet-induced obese rats underwent a RYGB, the authors demonstrated that rats that sustained weight loss had elevated plasma PYY levels. Thus weight regain might be associated with the inability to maintain appropriately high PYY levels. They suggest that perhaps the pharmacologic stimulation of PYY might help prevent weight regain in patients after RYGB [25].

The Argument for Targeting Patient Behaviors

Psychological and behavioral interventions that are extremely successful for the treatment of mood and eating disorders can also be applied to bariatric patients experiencing weight regain after surgery (Fig. 12.2). The success of these treatment modalities in bariatric surgery patients is likely owed in part to the co-occurrence or emergence of disorders such as depression or binge eating in this patient population. Patients with weight regain often report low self-esteem, stressful family or work situations, and having fewer friends [8]. In addition, postoperatively, some patients may experience “addiction transfer” in which alternative substances such as alcohol replace food as a coping strategy [27]. This substitution can be particularly risky especially in patients who have undergone a RYGB, as they experience more rapid absorption of alcohol into the bloodstream due to the altered anatomy of the GI tract [27]. Two very specific examples of patient-specific behaviors worth discussing are amount of physical activity and compliance with follow-up appointments.

One of the known patient-specific risk factors for weight regain is lack of regular physical activity. Friere et al. reported that the lowest incidence of weight regain was seen in patients who developed positive physical exercise habits. Not all patients, however, are able to comply with the recommendation for increasing and maintaining daily movement [14]. Targeting obstacles to developing a successful exercise regimen could represent a useful intervention. A qualitative cross-sectional study assessed patient-perceived barriers to exercise. Utilizing responses to an online survey, they found that 78% of the participants described at least one internal motivational barrier (difficulty making exercise a priority, general motivational

Possible Interventions for Weight Regain

Behavioral	Endoscopic	Revisional Surgery
Project HELP	Sclerotherapy	Laparoscopic reduction of gastric pouch
Acceptance-based treatment (ABT)	Apollo OverStitch	Conversion to different procedure
Dialectical Behavioral Therapy (DBT)	OTSC-Clip	Redo of gastrojejunal anastomosis
Cognitive behavioral therapy (CBT)	*EndoCinch	Addition of gastric band (LAGB)
	*StomaphyX	
	*ROSE	

*No longer commerically available

Fig. 12.2 Possible interventions for weight regain

difficulties, lack of enjoyment or energy and difficulty maintaining consistency). At least one physical barrier (chronic pain or illness and postoperative issues such as lack of stamina or diet problems) was expressed by approximately 21% of respondents. Finally, 37% communicated an external barrier to exercise such as time, weather, or resources [55].

Continued follow-up after surgery is needed for maintaining successful weight loss and preventing weight recidivism. A survey-based study found that one of the behavioral predictors of weight regain was the extent of follow-up. They reported that patients with no follow-up visits were almost five times more likely to regain weight compared to those who attended four or five visits per year [27]. While the exact amount of follow-up that is needed to prevent weight regain is relatively unknown, it is likely that behavioral support can also take on other forms besides the traditional healthcare provider and patient encounter. Several studies have demonstrated that regular support group attendance is associated with better weight loss after bariatric surgery [56, 57]. It is believed that patients who attend support groups receive continual education that assists with adherence to healthy food choices and other long-term behavioral modifications. The repetitiveness and the sense of connection among peers that develops is an integral component of maintaining weight loss. In one study, participants who attended five or more monthly support groups per year achieved better short-term weight loss compared to those who attended fewer meetings [56]. Finally, a potential barrier to the success of support groups is the potential that patients who struggle with weight loss or experience weight regain may feel too embarrassed to attend [57].

One example of behavioral treatment recently tailored for obesity that could potentially be applied to post-bariatric surgery patients experiencing weight regain is Acceptance-Based Behavioral Treatment (ABBT or ABT) [58–60]. The principles of ABT are based on other well-substantiated therapy models such as Acceptance

and Commitment Therapy (ACT) and Dialectical Behavioral Therapy (DBT) that teach patients to behave according to their life values despite experiencing unpleasant thoughts, feelings, urges, and cravings. The model of ABT defined by Forman et al. in the Mind Your Health Project is based on "willingness" and "mindful decision-making" that instructs patients to accept a decrease in short-term pleasure that might occur by making healthy decisions and how to override the body's messages that produce a drive to eat [59]. ABT also focuses on making mindful behavioral decisions in order to prevent behaviors from just being automatic. Skills such as distress tolerance, present-moment awareness of internal states, clarity of one's personal values, linking values to in-the-moment decision-making, and psychological distancing or "defusion" are integral parts of ABT [58–60]. When these skills are combined with behavioral elements such as self-monitoring, stimulus, and portion control and psychoeducation, this form of treatment can be very effective for enacting change. The study conducted by Forman et al. was a randomized controlled trial of 128 obese participants, where half were assigned to 40 weeks of Standard Behavioral Treatment (SBT) or ABT. The data from the Mind Your Health Project was promising, as those who underwent ABT experienced significantly higher weight loss and 64% maintained at least 10% weight loss by follow-up. ABT was especially helpful for individuals with greater baseline depression, those more prone to emotional eating, patients with a higher responsivity to food cues, and those with greater disinhibition [59].

Another example of a targeted behavioral intervention strategy is described in a pilot study of 28 patients who experienced an average weight regain of 37% (17 kg) of initial weight lost after bariatric surgery. The intervention lasted for 6 weeks and consisted of a 1-h group per week where patients were instructed utilizing element from DBT and Cognitive Behavioral Therapy (CBT). Patients were educated on the common factors of weight regain, how to increase their accountability for their behaviors through self-monitoring, stress management skills, how to prevent or even counteract the emotional dysregulation that often leads to disordered eating, surgical diet skills, and a stepped care approach to weight maintenance. Participants experienced a decline in BMI and percentage of weight regained as well as significant improvement in their mood and a decrease in subjective binge eating behaviors [29]. Thus, although this was a low-powered study, it offers ideas for future behavioral interventions targeting weight regain. Odom et al. found that engagement in self-monitoring, which can consist of tracking food intake, exercise, weight, setting goals, developing cognitive and environmental control strategies, recognizing social stimuli, developing positive social supports, practicing visualization techniques, and celebrating success, was an independent predictor of weight maintenance after bariatric surgery. Other independent predictors were control over food urges and eating habits, whereas they found that independent predictors of weight regain were lack of control over food urges, low self-reported well-being scores, and concerns expressed by others regarding their alcohol or drug use [27].

Finally, data published from Project HELP: Healthy Eating and Lifestyle Post-surgery further substantiate the effectiveness of behavioral interventions for weight regain. This program consisted of ten online weekly modules created from ABT,

which aimed to communicate to patients acceptance-based skills in order to promote the engagement in healthy behaviors that are in line with their values over the long term despite how uncomfortable or unpleasant they might feel in the moment. Of the 11 patients who completed the program, 10 individuals experienced weight stabilization or weight loss at 3 months of follow-up. They also reported a decrease in grazing behaviors, loss of control eating, average daily caloric intake, and increased use of cognitive defusion techniques. Survey of the participants determined that this remotely delivered ABT was feasible, acceptable, and effective [58]. More research on these behavioral interventions is needed before widespread application; however, the preliminary results are promising.

Revisional Surgery and Endoscopic Therapies

Given the detrimental physical and emotional effects of significant weight regain, an option that can be considered is revisional surgery (Fig. 12.2). Often the deciding factor to pursue this choice is the recurrence of severe obesity-related comorbidities and suboptimal anatomy. However, the morbidity associated with a second operation in this patient population is higher than the morbidity of the primary bariatric surgery [33, 61]. Length of stay, cost, complication rate, and mortality are all increased with reoperative surgery [26]. As a result, the benefit of a revisional surgery must be carefully weighed against the risks. In addition, there is no standardized threshold for when weight recidivism is significant enough to warrant revisional surgery [61]. Thus this decision is left up to the individual provider and patient. Attitudes among surgeons regarding revisional surgery vary, and often because the operation can be more technically challenging, these patients are referred to tertiary centers with expertise. In addition, revisional surgery can also take the form of endoluminal therapies, which carry less risk, are more cost effective, and are often repeatable [30, 33, 36, 38–40, 42–44, 62–65]. However, the technology for these endoscopic procedures is still being refined and has limited long-term follow-up. Revisional surgery can also be applied to patients who experienced insufficient weight loss and never achieved significant weight loss postoperatively.

Ma et al. classify the types of revisional surgery into five broad categories [66]. The first category is restorative surgery, which means returning the anatomy to the intended state in patients with postoperative complications such as dilated gastric pouch after a RYGB or excess fundus after a VSG who initially had a good response to surgery. The second is corrective surgery, which focuses on other complications that have failed medical management such as marginal ulcers or anastomotic stenosis. Augmentation surgery has the goal of boosting the effects of the initial operation – examples include lengthening of the roux limb or the addition of a laparoscopic adjustable gastric band after a RYGB. Conversion surgery includes converting from one type of surgery to another such as band to bypass or sleeve. Finally, reversal surgery is done for serious long-term complications such as severe protein calorie malnutrition, reactive hypoglycemia that is refractory to medical management, drug or alcohol addiction, intractable nausea, and others. Another way

to describe revisional surgery is based on whether or not the reoperative procedure promotes further restriction, such as the addition of a band to a patient with RYGB anatomy, or if it furthers malabsorption, such as conversion to a duodenal switch or roux limb lengthening [66].

A systematic review conducted by a taskforce of the ASMBS looked at 175 articles and derived some basic conclusions regarding revisional bariatric surgery. For example, they do support the general concept of reoperative intervention to address weight regain, insufficient weight loss, postoperative complications, inadequate resolution of comorbidities, or recurrence of these comorbidities. They also discuss the notion of prejudice that exists today against obese patients as evidenced by the "one lifetime bariatric surgery" clause in some insurance policies and the pushback received when treating patients with additional surgery or procedures. They argue that it is dramatically different than the attitude toward other disciplines such as orthopedic or cancer treatment, which routinely cover reoperations. The task force also comments on the fact that the majority of the literature about revisional surgery comes from single-institution case series done retrospectively and only reports short- or medium-term outcomes. Finally, although the reported outcomes are generally favorable in terms of weight loss and improvement in comorbidities, they point out that reoperative surgery requires prudent patient selection and often the surgical experience of a bariatric center [61].

Another systematic review that included 24 articles sought to elucidate options for revisional surgery for weight regain after RYGB. They compiled the mean percentage of excess BMI loss (%EBMIL) at 1 and 3 years after each type of conversion or revision. The average %BMIL after the conversion to a distal RYGB (either creating a very long roux limb or a very long biliopancreatic limb) was 54% at 1 year and 52.2% at 3 years. For revision of the gastric pouch or the gastrojejunal anastomosis, the means were 43.3% and 14%, respectively, which the authors hypothesize indicates that this particular method of revisional surgery may have real short-term benefits but the effect does not translate into long-term success. Results after conversion to a biliopancreatic diversion or duodenal switch were excellent, with 63.7% %EBMIL at 1 year and 76% at 3 years; however, the resulting vitamin and nutrient deficiencies that develop can be very difficult for patients to manage. Augmenting the effects of the RYGB by adding a LAGB results in about 47% %EBMIL at both time periods. Finally, endoluminal therapies were reported to offer 32.1% at 1 year, with no available data yet at 3 years of follow-up [62]. These results reinforce the beneficial effect of revisional surgery for weight recidivism.

A single-institution study that looked at the results in 26 patients who underwent reshaping of the gastric pouch for >30% of weight regain from their nadir weight after RYGB also demonstrates favorable results. Comorbidities resolved in 81% of the population and weight loss back to initial postoperative nadir and weight stabilization over 4 years of follow-up frequently occurs. Major complications were observed 27% of the time, and minor complications were observed 15% of the time. Inclusion criteria for this study were relatively strict as the patients had to have failed intensive nutritional, physiotherapeutic, and

psychological interventions, report a loss of satiety, and on upper GI series had a gastric pouch measuring >6 cm in width [34].

Endoluminal therapies described to treat weight regain after RYGB could represent excellent options, especially as the technology continues to improve at a rapid pace (Fig. 12.2). Current available endoscopic procedures include the Apollo OverStitch (Apollo Endosurgery, Austin, Texas) endoscopic suturing system (also referred to as Transoral Outlet Reduction endoscopy – TORe), sclerotherapy, and the OTSC (over the scope) clip [30, 33, 37, 38, 43, 44, 62, 67]. The OverStitch system requires a double channel scope through which a curved needle is deployed and full-thickness tissue bites are taken in interrupted or continuous fashion on the gastric pouch to plicate or reduce the diameter of the gastrointestinal stoma [33, 43, 44, 62]. Several studies have demonstrated the efficacy of the OverStitch. For example, Jirapinyo et al. reported loss of 10.8 kg at 1 year out of the 24 kg regained after RYGB, and Kumar et al. reported loss of about 25% EWL at 1 year [38, 67]. Sclerotherapy involves injecting a sclerosant, usually sodium morrhuate, into the tissue around the stoma to increase the restrictive effect. Multiple studies have also established that sclerotherapy can be successful, although the effects are time-limited [30, 37, 43]. One study reported a 91.6% probability of either weight loss or weight stabilization at 1 year after sclerotherapy [37]. A study utilizing both the OverStitch and sclerotherapy demonstrated even greater success when combining these two endoscopic therapies [33]. Finally, the OTSC clip (Ovesco AG, Tubingen, Germany) is a device that is mounted on a transparent applicator cap that gets placed on the tip of an endoscope. Using endoscopic forceps the tissue is pulled into the cap, and a string is pulled to release the clip thus clamping the tissue in place [43, 65]. ASMBS members were surveyed to assess the degree of risk accepted with these endoscopic procedures. A large proportion (81%) of respondents indicated that the degree of risk tolerated when utilizing these novel endoscopic therapies to achieve a 10–20% EWL must be equal to the degree of risk encountered of any other therapeutic endoscopic procedure [68].

Other systems that are no longer commercially available include the Bard EndoCinch (C.R. Bard, Inc., Murray Hill, NJ), the StomaphyX, and the ROSE (Restorative Obesity Surgery Endoscopic) incisionless operating platform. The EndoCinch was a device that used suction to pull tissue into a hollow capsule mounted on the end of an endoscope [40]. A needle was then passed through the tissue, and the suture was cinched with a knot pusher [43]. The concern with this system was the superficial nature of the tissue bites achieved thus questioning the durability of the tissue plication achieved. The StomaphyX (Endogastric Solutions, Inc., Redmond, WA) was a device that used H fasteners to create full-thickness endoluminal tissue plications on the gastric pouch. It could not be used on the anastomosis. Data regarding the efficacy of the StomaphyX was controversial, and it was eventually taken off the market [41, 69, 70]. The ROSE platform had four channels, one for the endoscope and three operating channels. This tissue-grasping device allowed for full-thickness bites to decrease the diameter of the stoma. Data from studies utilizing the ROSE platform demonstrated that it was safe and technically resulted in a decrease of stomal diameter [39, 42, 64].

Conclusion

While bariatric surgery often results in substantial weight loss, significant weight regain is a frequent long-term adverse consequence. Weight regain can result in the return of obesity-related comorbidities and can be very psychologically damaging. The etiology of weight regain is multifactorial and thus presents a challenge to both provider and patients. Those prone to weight regain postoperatively must be identified and risk factors targeted early. Finally, revisional surgery and endoluminal interventions represent possible options for this population.

References

1. Chikunguwo SM, Wolfe LG, Dodson P, Meador JG, Baugh N, Clore JN, et al. Analysis of factors associated with durable remission of diabetes after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2010;6(3):254–9.
2. Jimenez A, Casamitjana R, Flores L, Viaplana J, Corcelles R, Lacy A, et al. Long-term effects of sleeve gastrectomy and Roux-en-Y gastric bypass surgery on type 2 diabetes mellitus in morbidly obese subjects. Ann Surg. 2012;256(6):1023–9.
3. Laurino Neto RM, Herbella FA, Tauil RM, Silva FS, de Lima SE Jr. Comorbidities remission after Roux-en-Y gastric bypass for morbid obesity is sustained in a long-term follow-up and correlates with weight regain. Obes Surg. 2012;22(10):1580–5.
4. Lauti M, Stevenson S, Hill AG, MacCormick AD. Patient perspectives about follow-up care and weight regain following sleeve gastrectomy. Obes Surg. 2016;26(11):2724–31.
5. Lauti M, Kularatna M, Hill AG, MacCormick AD. Weight regain following sleeve gastrectomy-a systematic review. Obes Surg. 2016;26(6):1326–34.
6. Maleckas A, Gudaityte R, Petereit R, Venclauskas L, Velickiene D. Weight regain after gastric bypass: etiology and treatment options. Gland Surg. 2016;5(6):617–24.
7. Yanos BR, Saules KK, Schuh LM, Sogg S. Predictors of lowest weight and long-term weight regain among Roux-en-Y gastric bypass patients. Obes Surg. 2015;25(8):1364–70.
8. Livhits M, Mercado C, Yermilov I, Parikh JA, Dutson E, Mehran A, et al. Patient behaviors associated with weight regain after laparoscopic gastric bypass. Obes Res Clin Pract. 2011;5(3):e169–266.
9. Felsenreich DM, Langer FB, Kefurt R, Panhofer P, Schermann M, Beckerhinn P, et al. Weight loss, weight regain, and conversions to Roux-en-Y gastric bypass: 10-year results of laparoscopic sleeve gastrectomy. Surg Obes Relat Dis. 2016;12(9):1655–62.
10. Karmali S, Brar B, Shi X, Sharma AM, de Gara C, Birch DW. Weight recidivism post-bariatric surgery: a systematic review. Obes Surg. 2013;23(11):1922–33.
11. Shantavasinkul PC, Omotosho P, Corsino L, Portenier D, Torquati A. Predictors of weight regain in patients who underwent Roux-en-Y gastric bypass surgery. Surg Obes Relat Dis. 2016;12(9):1640–5.
12. Magro DO, Geloneze B, Delfini R, Pareja BC, Callejas F, Pareja JC. Long-term weight regain after gastric bypass: a 5-year prospective study. Obes Surg. 2008;18(6):648–51.
13. Cooper TC, Simmons EB, Webb K, Burns JL, Kushner RF. Trends in weight regain following Roux-en-Y gastric bypass (RYGB) bariatric surgery. Obes Surg. 2015;25(8):1474–81.
14. Freire RH, Borges MC, Alvarez-Leite JI, Toulson Davisson Correia MI. Food quality, physical activity, and nutritional follow-up as determinant of weight regain after Roux-en-Y gastric bypass. Nutrition. 2012;28(1):53–8.
15. Obeidat F, Shanti H, Mismar A, Albsoul N, Al-Qudah M. The magnitude of antral resection in laparoscopic sleeve gastrectomy and its relationship to excess weight loss. Obes Surg. 2015;25(10):1928–32.

16. Monaco-Ferreira DV, Leandro-Merhi VA. Weight regain 10 years after Roux-en-Y gastric bypass. Obes Surg. 2017;27(5):1137–44.
17. Abu Dayyeh BK, Lautz DB, Thompson CC. Gastrojejunal stoma diameter predicts weight regain after Roux-en-Y gastric bypass. Clin Gastroenterol Hepatol. 2011;9(3):228–33.
18. Nedelcu M, Khwaja HA, Rogula TG. Weight regain after bariatric surgery-how should it be defined? Surg Obes Relat Dis. 2016;12(5):1129–30.
19. Sjostrom L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004;351(26):2683–93.
20. Jones L, Cleator J, Yorke J. Maintaining weight loss after bariatric surgery: when the spectator role is no longer enough. Clin Obes. 2016;6(4):249–58.
21. Buchwald H, Avidor Y, Braunwald E, Jensen MD, Pories W, Fahrbach K, et al. Bariatric surgery: a systematic review and meta-analysis. JAMA. 2004;292(14):1724–37.
22. Santo MA, Riccioppo D, Pajecki D, Kawamoto F, de Cleva R, Antonangelo L, et al. Weight regain after gastric bypass: influence of gut hormones. Obes Surg. 2016;26(5):919–25.
23. Madan AK, Tichansky DS, Taddeucci RJ. Postoperative laparoscopic bariatric surgery patients do not remember potential complications. Obes Surg. 2007;17(7):885–8.
24. da Silva FB, Gomes DL, de Carvalho KM. Poor diet quality and postoperative time are independent risk factors for weight regain after Roux-en-Y gastric bypass. Nutrition. 2016;32(11–12):1250–3.
25. Meguid MM, Glade MJ, Middleton FA. Weight regain after Roux-en-Y: a significant 20% complication related to PYY. Nutrition. 2008;24(9):832–42.
26. Sheppard CE, Lester EL, Chuck AW, Birch DW, Karmali S, de Gara CJ. The economic impact of weight regain. Gastroenterol Res Pract. 2013;2013:379564.
27. Odom J, Zalesin KC, Washington TL, Miller WW, Hakmeh B, Zaremba DL, et al. Behavioral predictors of weight regain after bariatric surgery. Obes Surg. 2010;20(3):349–56.
28. Tamboli RA, Breitman I, Marks-Shulman PA, Jabbour K, Melvin W, Williams B, et al. Early weight regain after gastric bypass does not affect insulin sensitivity but is associated with elevated ghrelin. Obesity (Silver Spring). 2014;22(7):1617–22.
29. Himes SM, Grothe KB, Clark MM, Swain JM, Collazo-Clavell ML, Sarr MG. Stop regain: a pilot psychological intervention for bariatric patients experiencing weight regain. Obes Surg. 2015;25(5):922–7.
30. Abu Dayyeh BK, Jirapinyo P, Weitzner Z, Barker C, Flicker MS, Lautz DB, et al. Endoscopic sclerotherapy for the treatment of weight regain after Roux-en-Y gastric bypass: outcomes, complications, and predictors of response in 575 procedures. Gastrointest Endosc. 2012;76(2):275–82.
31. Heneghan HM, Yimcharoen P, Brethauer SA, Kroh M, Chand B. Influence of pouch and stoma size on weight loss after gastric bypass. Surg Obes Relat Dis. 2012;8(4):408–15.
32. Muller MK, Wildi S, Scholz T, Clavien PA, Weber M. Laparoscopic pouch resizing and redo of gastro-jejunal anastomosis for pouch dilatation following gastric bypass. Obes Surg. 2005;15(8):1089–95.
33. Riva P, Perretta S, Swanstrom L. Weight regain following RYGB can be effectively treated using a combination of endoscopic suturing and sclerotherapy. Surg Endosc. 2017;31(4):1891–5.
34. Borbely Y, Winkler C, Kroll D, Nett P. Pouch reshaping for significant weight regain after Roux-en-Y gastric bypass. Obes Surg. 2017;27(2):439–44.
35. Al-Bader I, Khoursheed M, Al Sharaf K, Mouzannar DA, Ashraf A, Fingerhut A. Revisional laparoscopic gastric pouch resizing for inadequate weight loss after Roux-en-Y gastric bypass. Obes Surg. 2015;25(7):1103–8.
36. Yimcharoen P, Heneghan HM, Singh M, Brethauer S, Schauer P, Rogula T, et al. Endoscopic findings and outcomes of revisional procedures for patients with weight recidivism after gastric bypass. Surg Endosc. 2011;25(10):3345–52.
37. Spaulding L, Osler T, Patlak J. Long-term results of sclerotherapy for dilated gastrojejunostomy after gastric bypass. Surg Obes Relat Dis. 2007;3(6):623–6.

38. Kumar N, Thompson CC. Transoral outlet reduction for weight regain after gastric bypass: long-term follow-up. Gastrointest Endosc. 2016;83(4):776–9.
39. Horgan S, Jacobsen G, Weiss GD, Oldham JS Jr, Denk PM, Borao F, et al. Incisionless revision of post-Roux-en-Y bypass stomal and pouch dilation: multicenter registry results. Surg Obes Relat Dis. 2010;6(3):290–5.
40. Thompson CC, Slattery J, Bundga ME, Lautz DB. Peroral endoscopic reduction of dilated gastrojejunal anastomosis after Roux-en-Y gastric bypass: a possible new option for patients with weight regain. Surg Endosc. 2006;20(11):1744–8.
41. Goyal V, Holover S, Garber S. Gastric pouch reduction using StomaphyX in post Roux-en-Y gastric bypass patients does not result in sustained weight loss: a retrospective analysis. Surg Endosc. 2013;27(9):3417–20.
42. Raman SR, Holover S, Garber S. Endolumenal revision obesity surgery results in weight loss and closure of gastric-gastric fistula. Surg Obes Relat Dis. 2011;7(3):304–8.
43. Dakin GF, Eid G, Mikami D, Pryor A, Chand B, American Society for M, et al. Endoluminal revision of gastric bypass for weight regain – a systematic review. Surg Obes Relat Dis. 2013;9(3):335–42.
44. Patel LY, Lapin B, Brown CS, Stringer T, Gitelis ME, Linn JG, et al. Outcomes following 50 consecutive endoscopic gastrojejunal revisions for weight gain following Roux-en-Y gastric bypass: a comparison of endoscopic suturing techniques for stoma reduction. Surg Endosc. 2016;31:2667–77.
45. Sarzynski MA, Jacobson P, Rankinen T, Carlsson B, Sjostrom L, Bouchard C, et al. Associations of markers in 11 obesity candidate genes with maximal weight loss and weight regain in the SOS bariatric surgery cases. Int J Obes. 2011;35(5):676–83.
46. Abraham A, Ikramuddin S, Jahansouz C, Arafat F, Hevelone N, Leslie D. Trends in bariatric surgery: procedure selection, revisional surgeries, and readmissions. Obes Surg. 2016;26(7):1371–7.
47. Diamantis T, Apostolou KG, Alexandrou A, Griniatsos J, Felekouras E, Tsigris C. Review of long-term weight loss results after laparoscopic sleeve gastrectomy. Surg Obes Relat Dis. 2014;10(1):177–83.
48. Juodeikis Z, Brimas G. Long-term results after sleeve gastrectomy: a systematic review. Surg Obes Relat Dis. 2016;13:693–9.
49. Deguines JB, Verhaeghe P, Yzet T, Robert B, Cosse C, Regimbeau JM. Is the residual gastric volume after laparoscopic sleeve gastrectomy an objective criterion for adapting the treatment strategy after failure? Surg Obes Relat Dis. 2013;9(5):660–6.
50. Flolo TN, Andersen JR, Kolotkin RL, Aasprang A, Natvig GK, Hufthammer KO, et al. Five-year outcomes after vertical sleeve gastrectomy for severe obesity: a prospective cohort study. Obes Surg. 2017;27:1944–51.
51. Edholm T, Degerblad M, Gryback P, Hilsted L, Holst JJ, Jacobsson H, et al. Differential incretin effects of GIP and GLP-1 on gastric emptying, appetite, and insulin-glucose homeostasis. Neurogastroenterol Motil. 2010;22(11):1191–200. e315
52. Crujeiras AB, Goyenechea E, Abete I, Lage M, Carreira MC, Martinez JA, et al. Weight regain after a diet-induced loss is predicted by higher baseline leptin and lower ghrelin plasma levels. J Clin Endocrinol Metab. 2010;95(11):5037–44.
53. Cummings DE, Weigle DS, Frayo RS, Breen PA, Ma MK, Dellinger EP, et al. Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. N Engl J Med. 2002;346(21):1623–30.
54. Bohdjalian A, Langer FB, Shakeri-Leidenmuhler S, Gfrerer L, Ludvik B, Zacherl J, et al. Sleeve gastrectomy as sole and definitive bariatric procedure: 5-year results for weight loss and ghrelin. Obes Surg. 2010;20(5):535–40.
55. Peacock JC, Sloan SS, Cripps B. A qualitative analysis of bariatric patients' post-surgical barriers to exercise. Obes Surg. 2014;24(2):292–8.
56. Song Z, Reinhardt K, Buzdon M, Liao P. Association between support group attendance and weight loss after Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2008;4(2):100–3.

57. Orth WS, Madan AK, Taddeucci RJ, Coday M, Tichansky DS. Support group meeting attendance is associated with better weight loss. Obes Surg. 2008;18(4):391–4.
58. Bradley LE, Forman EM, Kerrigan SG, Goldstein SP, Butryn ML, Thomas JG, et al. Project HELP: a remotely delivered behavioral intervention for weight regain after bariatric surgery. Obes Surg. 2017;27(3):586–98.
59. Forman EM, Butryn ML, Juarascio AS, Bradley LE, Lowe MR, Herbert JD, et al. The mind your health project: a randomized controlled trial of an innovative behavioral treatment for obesity. Obesity (Silver Spring). 2013;21(6):1119–26.
60. Forman EM, Butryn ML, Manasse SM, Bradley LE. Acceptance-based behavioral treatment for weight control: a review and future directions. Curr Opin Psychol. 2015;2:87–90.
61. Brethauer SA, Kothari S, Sudan R, Williams B, English WJ, Brengman M, et al. Systematic review on reoperative bariatric surgery: American Society for Metabolic and Bariatric Surgery Revision Task Force. Surg Obes Relat Dis. 2014;10(5):952–72.
62. Tran DD, Nwokeabia ID, Purnell S, Zafar SN, Ortega G, Hughes K, et al. Revision of Roux-En-Y gastric bypass for weight regain: a systematic review of techniques and outcomes. Obes Surg. 2016;26(7):1627–34.
63. Baretta GA, Alhinho HC, Matias JE, Marchesini JB, de Lima JH, Empinotti C, et al. Argon plasma coagulation of gastrojejunal anastomosis for weight regain after gastric bypass. Obes Surg. 2015;25(1):72–9.
64. Ryou M, Mullady DK, Lautz DB, Thompson CC. Pilot study evaluating technical feasibility and early outcomes of second-generation endosurgical platform for treatment of weight regain after gastric bypass surgery. Surg Obes Relat Dis. 2009;5(4):450–4.
65. Heylen AM, Jacobs A, Lybeer M, Prosst RL. The OTSC(R)-clip in revisional endoscopy against weight gain after bariatric gastric bypass surgery. Obes Surg. 2011;21(10):1629–33.
66. Ma P, Reddy S, Higa KD. Revisional bariatric/metabolic surgery: what dictates its indications? Curr Atheroscler Rep. 2016;18(7):42.
67. Jirapinyo P, Slattery J, Ryan MB, Abu Dayyeh BK, Lautz DB, Thompson CC. Evaluation of an endoscopic suturing device for transoral outlet reduction in patients with weight regain following Roux-en-Y gastric bypass. Endoscopy. 2013;45(7):532–6.
68. Brethauer SA, Pryor AD, Chand B, Schauer P, Rosenthal R, Richards W, et al. Endoluminal procedures for bariatric patients: expectations among bariatric surgeons. Surg Obes Relat Dis. 2009;5(2):231–6.
69. Eid GM, McCloskey CA, Eagleton JK, Lee LB, Courcoulas AP. StomaphyX vs a sham procedure for revisional surgery to reduce regained weight in Roux-en-Y gastric bypass patients: a randomized clinical trial. JAMA Surg. 2014;149(4):372–9.
70. Ong'uti SK, Ortega G, Onwugbufor MT, Ivey GD, Fullum TM, Tran DD. Effective weight loss management with endoscopic gastric plication using StomaphyX device: is it achievable? Surg Obes Relat Dis. 2013;9(1):113–7.

13 Internal Hernia and Small Bowel Obstruction After Roux-en-Y Gastric Bypass

Adel Alhaj Saleh and Mujjahid Abbas

Introduction

Although laparoscopic sleeve gastrectomy (LSG) is currently the most common performed weight loss procedure, in the United States, Roux-en-Y gastric bypass (RYGB) is still the gold standard procedure [1]. However, one of the complications related to RYGB is small bowel obstruction (SBO) (5%), with internal hernia being the most common cause (42–61%) of all cases [2, 3]. Other causes included adhesions (13.7%) [4], intussusception (0.07–0.6%), phytobezoar (2–3% of all causes of SBO), and intraluminal blood clot (<0.2%) [5–8].

Interestingly, some studies have shown that the incidence of adhesive small bowel obstruction after RYGB has decreased, after the introduction of laparoscopic technique, while internal hernia incidence increased [9, 10].

Multiple techniques were described throughout the evolution of RYGB, starting from creating the gastric pouch, different anastomosis techniques of the gastrojejunostomy and jejuno-jejunostomy (hand sewn, circular, or linear staplers), and construction and positioning of the Roux limb (retrocolic-retrogastric, retrocolic-antegastric, or antecolic-antigastric) to also closure vs. non-closure of mesenteric and peritoneal defects [11, 12]. The last two factors believed to be the most important in prevention or occurrence of internal hernias after RYGB [13, 14].

Another important factor in developing internal hernia after RYGB is rapid excess weight loss (EWL), as Schneider et al. found in a large study of 934 patients

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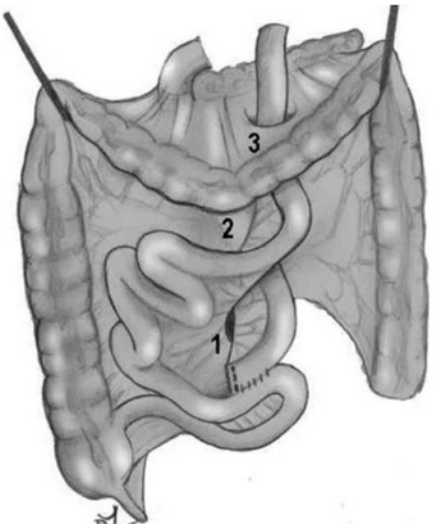

Fig. 13.1 Potential hernia sites in laparoscopic RYGBP. (*1*) entero-enterostomy mesenteric defect; (*2*) space between mesentery of Roux and transverse mesocolon (Petersen's space); (*3*) transverse mesocolon defect. All the procedures fashioning a Roux-en-Y limb result in mesenteric defects that may potentially cause IH (Facchiano et al. Internal Hernia After Bariatric Procedures. Minimally Invasive Bariatric and Metabolic Surgery. Springer, 2015)

post RYGB that patients who were above the 90th percentile of EWL (33.2%) had nearly twice the risk of developing internal hernia as the rest of RYGB population [15]. Although there was no clear mechanism why the risk of internal hernia increases after rapid EWL, one possible explanation is that quick mesenteric fat loss may not allow adequate time for potential hernia spaces to completely close [15].

Blachar and Federle defined internal hernia as protrusion of any viscus, most likely small bowel through an opening in mesenteric or peritoneal surfaces, resulting in encapsulation inside another compartment [16].

Usually, there are two to three potential hernia spaces after RYGB, and that depends on the technique used. In retrocolic Roux limb, a tunnel is created in the transvers mesocolon to the left of middle colic artery and then passed in retrogastric position to be anastomosed to the cardiac gastric pouch. In this case, this mesocolon defect might enlarge over time and allow for more room of small bowel to herniate into the retrocolic space. The transvers mesocolon defect space is eliminated while performing antegastric approach. Other two possible locations for internal hernias are the jejuno-jejunal mesenteric defect and the Petersen's space. The jejuno-jejunal mesenteric defect lies where the mesenteries of the biliopancreatic limb and the Roux limb meet at the jejuno-jejunostomy site. The Petersen's space, which was described first by the German surgeon Dr. Walther Petersen in 1900, is located between the mesentery of the Roux limb and the transvers mesocolon. It is created while bringing up the Roux limb to the cardiac gastric pouch. It happens in both retro and antecolic approaches [17, 18] (Fig. 13.1).

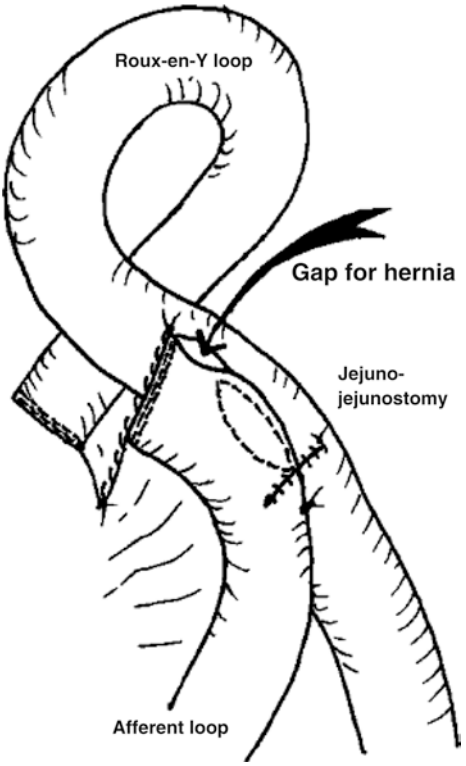

Fig. 13.2 Potential site for internal hernia between two jejunal loops Paroz (Paroz et al. A New Type of Internal Hernia After Laparoscopic Roux-en-Y Gastric Bypass. *Obesity Surgery* 2008, 19 (5))

Another potential space was described in a case report by Paroz et al. that doesn't include mesenteric defects, but a space between two jejunal loops at the jejuno-jejunostomy [19] (Fig. 13.2).

On the other hand, intussusception which is defined as telescoping of a proximal segment of gastrointestinal tract within the lumen of an adjacent segment [20] is far less common than internal hernia after RYGB. In a review study done by Daellenbach et al., they found that a variety of intussusceptions after gastric bypass were detected intraoperatively, intussusception involving jejuno-jejunostomy including retrograde intussusception of the common channel into the biliopancreatic limb or the Roux limb, and also intussusceptions distal to jejuno-jejunostomy or involving the Roux Limb [6, 7].

Diagnosis

Internal hernia can be a potentially life-threatening condition if it is not diagnosed and managed early. However, it is one of the most challenging clinical scenarios in terms of diagnosis. Internal hernia can occur any time since surgery up to many years after (1–3 years in patients with rapid EWL), while leaks, stenosis, and adhesions tend to present earlier in patients who have recently had RYGB [15, 16]. In a

retrospective chart review of 914 RGYB patients done by Obeid et al., 5% of patients (45/914) developed internal hernia. The symptoms varied from vague postprandial abdominal pain in majority of the patients (53.4%), followed by vague abdominal pain with nausea ± vomiting in 16%, while 8.8% of cases presented with acute abdominal pain ± nausea and vomiting, and one case (2.2%) presented with acute abdominal pain and peritonitis [2].

In many scenarios, abdominal examination and lab tests may not be as helpful in establishing the diagnosis [18, 21]. Nonetheless, in a retrospective chart review done by Spector et al. analyzing the findings of 4014 RYGB patients, among which 99 developed small bowel obstruction, most of these patients had internal hernia. Amylase and lipase were measured at the time of operation for obstruction in 58 patients, and in 48% were moderately high. They concluded that moderately elevated amylase/lipase in bowel obstruction after RYGB can increase the likelihood for diagnosis of internal hernia, especially if the biliopancreatic limb was obstructed [22].

Imaging studies are important for diagnosis of internal hernia, especially if they are used during the presence of symptoms. Since many internal hernias may reduce and recur, patients can present with intermittent symptoms [23]. However, it is even more important to keep in mind that in 20% of cases, imaging studies are negative [18].

Diagnostic Studies

Upper GI series and CT imaging are frequently used in diagnosing internal hernia after RYGB, but which one of them is superior to the other is a matter of controversy [24]. Some studies found overlapping in the findings of internal hernia vs. adhesions in bowel obstruction after RYGB; this made the authors conclude that upper GI cannot figure out the cause of bowel obstruction after RYGB [16]. However, in some other studies, Ahmed et al. found that upper GI can be 65% predictive of internal hernia. Although small bowel was dilated >2.5 cm in diameter in 100% of cases in both internal hernia and adhesions, there were some other findings on upper GI series suggestive of internal hernia. For example, dilated loops of small bowel clustered in the left upper or middle abdomen that remained high in the abdomen on erect position [24]. The four most frequent findings include fluid-filled dilated small bowel loops, redundant Roux limb in the lesser sac, majority of small bowel loops going to the left upper quadrant, and prolonged transit time of the contrast. Nowadays, CT scan is emerging as the preferred imaging tool in diagnosing internal hernia after RYGB for multiple reasons. It is less technically difficult than upper GI series, especially in positioning obese patients, and also CT scan has better quality of images [24, 25].

Some other reasons include the feasibility of CT scan use in acute setting, as not all centers have available technicians and radiologists especially over weekends and nights. CT scan can easily be interpreted by a general surgeon, while upper GI is

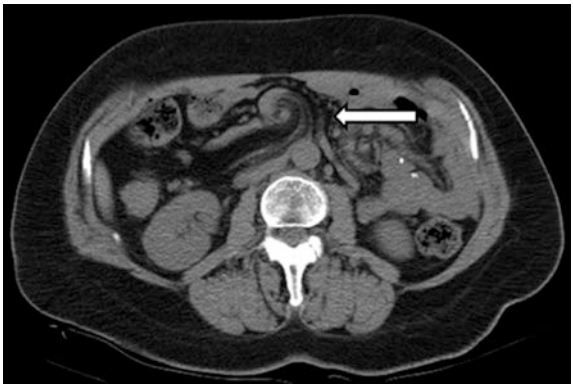

Fig. 13.3 The swirly appearance of the fat and vessels around the mesenteric root (B. Corey and J. Grams. Internal Hernias: Prevention, Diagnosis, and Management. Bariatric Surgery Complications and Emergencies. Springer, 2016)

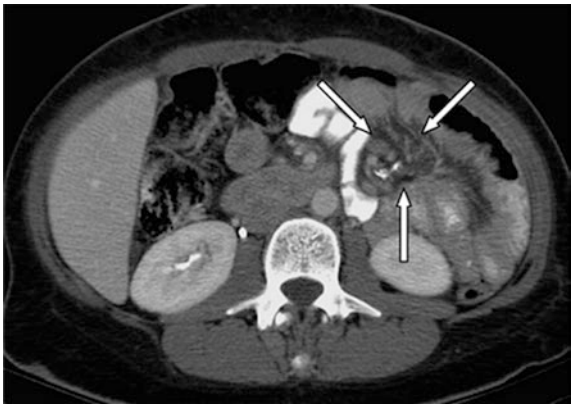

Fig. 13.4 The "hurricane eye" sign refers to the tubular shape of the distal mesenteric fat with surrounding bowel (*arrows*) (Reprinted with permission from Iannuccilli et al. [27]; [34]. © Elsevier B. Corey and J. Grams (Springer))

dynamic and needs a radiologist to report. Finally, CT is more sensitive and specific than any other imaging modalities [26].

Many CT findings of internal hernia were described in the literature: the swirly appearance of the fat and vessels around the mesenteric root (Fig. 13.3), the "hurricane eye" which is a tubular or round shape of distal mesenteric fat surrounded closely by small bowel loops (Fig. 13.4), "mushroom" shape of herniated mesenteric root (Fig. 13.5), clustered small bowel loops (Fig. 13.6), and small bowel other than the duodenum behind the superior mesenteric artery (Fig. 13.7); other findings include dilated gastric remnant, widening of the jejuno-jejunostomy, and swelling of mesenteric lymph nodes [23, 25, 27].

In two studies, conducted by Iannuccilli et al. and Lockhart et al., authors agreed that swirled mesentery is the best single predictive sign with a sensitivity of 61–100% and specificity of 67–94% [23, 27]. Lockhart et al. had even a further look, noting that the degree of swirl was important; for example, patients who had swirl <90° had negative findings of internal hernia on exploration, while patients with >270° were all positive on exploration [23].

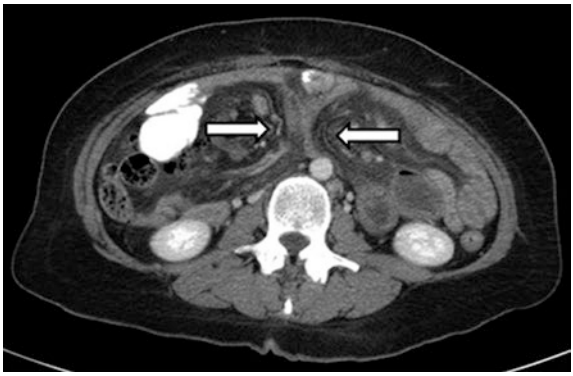

Fig. 13.5 *Arrows* point to the "mushroom sign," a typical appearance of an internal hernia on CT scan due to crowding or stretching of the vessels of the mesenteric root as they travel through the hernia (B. Corey and J. Grams. Internal Hernias: Prevention, Diagnosis, and Management. *Bariatric Surgery Complications and Emergencies*. Springer, 2016)

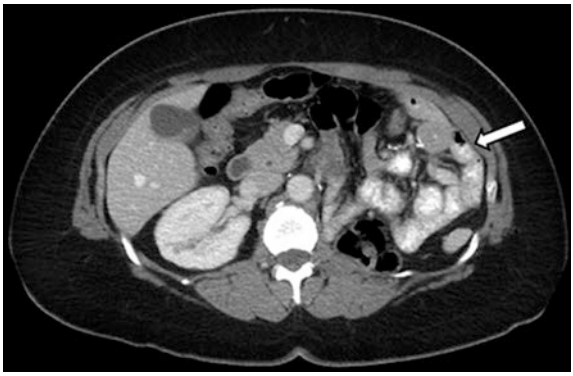

Fig. 13.6 Clustered loops of small bowel marked with an *arrow* on CT scan. An internal hernia was confirmed at the time of operative exploration (B. Corey and J. Grams. Internal Hernias: Prevention, Diagnosis, and Management. *Bariatric Surgery Complications and Emergencies*. Springer, 2016)

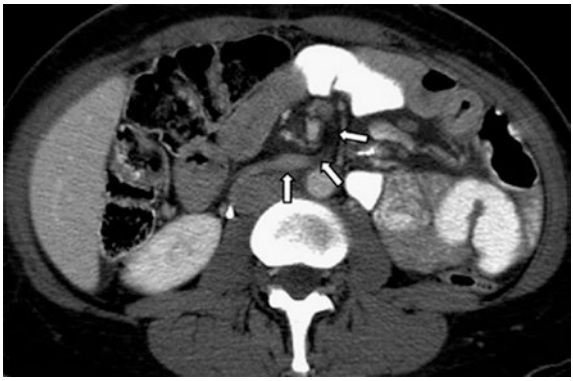

Fig. 13.7 A CT scan demonstrating small bowel behind the superior mesenteric artery. The *arrows* point to a segment of bowel which is thin and stretched. (Reprinted with permission from Iannuccilli et al. [27] (springer))

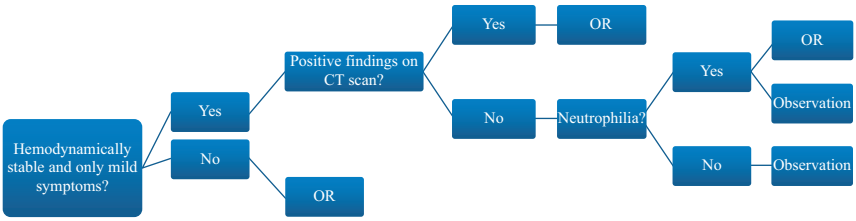

Fig. 13.8 Algorithm for treatment of the bariatric patient presenting with abdominal pain following Roux-en-Y gastric bypass/biliopancreatic diversion (M. S. Altieri et al. *Surgery for Obesity and Related Diseases* 11 (2015) 1207–1211 (Elsevier))

The "mushroom" and "hurricane" signs had low sensitivity but higher specificity, while the swollen mesenteric lymph nodes had moderate sensitivity (44–89%) and high specificity (90–100%) [27].

In another study conducted by Marchini et al., the most consistent CT finding was the clusters of small bowel loops (79.4%) followed by small bowel obstruction (73.5%) and swirled mesentery (61.8%) [25].

In order to find a more systematic approach toward diagnosis and management of internal hernia after RYGB, Altieri et al. created an algorithm, based on clinical situation of the patient, CT scan, and neutrophilia; they found that sensitivity of CT scan increased from 76% alone to 96% when adding neutrophilia to the equation (Fig. 13.8) [28]. Since in 20% of cases, radiological imaging can be negative [2, 18], Ahmed et al. suggested that in a stable patient where CT scan is negative, a second imaging modality can be helpful, for example, in their series, CT scan was negative in three patients, they went on upper GI, and there were findings suggestive of internal hernia in two out of three patients [24].

It is important to stress again that undiagnosed internal hernia can lead to devastating results; hence, we should have a high index of suspicion, even in absence of diagnostic evidence, and patients should get an early intervention for diagnosis and treatment of internal hernia and other causes of small bowel obstruction.

Management

No matter what was the method of diagnosis, the management of internal hernia after RYGB remains surgical. The urgency of surgical intervention relies on the clinical condition of the patient, for example, a patient with toxic signs and full-blown picture of obstruction should be taken urgently to exploration, even if only based on high index of clinical suspicion. While in patients who have intermittent symptoms, surgical intervention should strongly be recommended [18, 29]. Gandhi et al. suggested that usually patients who present with small bowel obstruction after RYGB have usually preceding intermittent symptoms of obstruction. They named these symptoms "herald symptoms": abdominal pain with bloating and transient small bowel obstruction [29]. In their series, among 11 patients presenting with

"herald symptoms," 9/11 agreed to undergo surgical intervention, and they were all found to have internal hernia, while the 2 patients who refused the operation presented later on with small bowel obstruction and underwent emergency operations and were also found to have internal hernia. Moreover three out of nine patients who underwent the operation under elective conditions were found to have small bowel volvulus; hence, early surgical intervention is important [29].

We should always keep in mind that, as in any bowel obstruction case, it's important to stabilize and improve the general condition of the patient. Proper intravenous fluid therapy should be initialed with normal saline or lactated Ringer's. Potassium should be corrected, and patients should be admitted to the surgical floor [30].

Although gastric decompression has a crucial role in the management of usual small bowel obstruction, however, in case of gastric bypass, extreme caution should be taken during the placement of nasogastric tubes (NGT), so one does not disrupt the anastomosis and cause leak [31].

In the literature, very few cases of hollow viscus perforation were reported after RYGBP; however, the PVC NGT available in the market are of 122 cm in length, and these tubes are designed for patients with normal gastric anatomy, especially that the last 8 cm has multiple holes from which fluid can be aspirated or infused [32]. In the normal stomach, excess length of NGT can be coiled inside the stomach with almost no potential harm, but in RYGBP the gastric pouch is only 5–6 cm in length, either the proximal end of the 8 cm fenestrated part of NGT might fall in the esophagus or the distal end may travers the gastrojejunostomy to the Roux limb [32]. According to Hanson et al., there is a formula we can use in patients with normal gastric anatomy to estimate the length of NGT needed, so the tip can lie in the body of the stomach, [(NEX-50)/2] + 50, where NEX is the distance from the nostrils to the earlobe to xiphoid in centimeters. In most cases it is between 51 and 73 cm [33]. According to Dinter et al., subtracting 10–15 cm from Hanson's way of measuring the length of the NGT may help avoid the risk of perforation in RYGBP patients. He also suggested that either the surgeon or the nurse who are going to place this NGT should coordinate with the radiologist, so it can be done under fluoroscopic guidance to minimize the risk of perforation [32].

In cases of severe gastric remnant distension, where placement of NGT will not be beneficial, placement of percutaneous decompression was successful, while in other cases, patients had to be taken for re-exploration and surgical gastrostomy tube insertion [34].

Surgical intervention should be attempted laparoscopically first if possible; some surgeons used the same trocars of the index procedure. Higa et al. described in their series that majority of their patients were successfully managed laparoscopically. Only 5/65 patients have to be converted to open because of peritonitis, extremely dilated bowel, and confusing anatomy [18]. In other series, Filip et al. had to convert three out of five patients to open because of grossly dilated small bowel loops [21]. On the other hand, Elms et al. managed all of their 26 cases laparoscopically [11].

Whether laparoscopic or open approach was done, there are several important points that should be taken into consideration during the repair of any internal hernia. Bowel should be fully run, preferably starting from a fixed point like ligament

of Treitz or ileocecal junction [35]. Although many surgeons recommend starting from the cecum rather than ligament of Treitz, as small bowel would be collapsed distally, hence easier to handle during running of small bowel, another added benefit is identifying the anatomy. If we start from the ligament of Treitz, Roux limb, and biliopancreatic limbs, identification can be confusing. All potential hernia sites should be carefully examined, any hernia should be reduced, and herniated bowel should be assessed for viability; in cases of strangulation, resection and anastomosis should be performed. All of the defects (even the ones discovered during a surgical procedure for different reasons) should be closed either by continuous running or purse-string manner with nonabsorbable sutures [18, 36].

In cases of intussusception, although sometimes it can spontaneously resolve but in most of the cases once diagnosis is made, surgical intervention is warranted to avoid bowel necrosis. In some cases, simple reduction might be enough; in other cases resection of nonviable segment should be performed with termino-terminal anastomosis. If the jejuno-jejunostomy is involved, reconstruction of this anastomosis should be done [6].

In rare scenarios, when the cause of obstruction is an intraluminal clot, some surgeons advice resection of the whole bowel segment containing the clot, with end-to-end anastomosis, while others found that opening the small bowel, then evacuating the clot, and closing the bowel segment can safely be performed when no evidence of active bleeding is seen [5]. In our own experience, in cases of jejuno-jejunostomy blood clot, opening the blind end of the biliopancreatic limb can give quicker and easier access to the clot, without the need of revision of anastomosis.

Important Points in Prevention of Internal Hernia

Different variations in the technique of performing RYGB were described in the literature. However, two important points are still a matter of debate in surgical societies across the world: closure vs. non-closure of mesenteric and peritoneal defects and antecolic vs. retrocolic positioning of the Roux limb. With regard to closure vs. non-closure of potential hernia spaces, there is a trend in most of studies in the literature toward closure of these defects by continuous nonabsorbable sutures. Bauman et al. found in a large series of RYGB patients a reduction in the rate of internal hernia at Petersen's space from 6.2% to 0% after changing their practice to closure technique [14]. On the other hand, Brolin and Kella also reported a decline in the incidence of internal hernia from 2.6% to 0.5% after adopting the closure technique of the mesenteric defects [13].

With regard to positioning of the Roux limb, in current practice antecolic position is gaining more popularity and acceptance among bariatric surgeons, because it eliminates one of the potential sites of internal hernia, namely, the transvers mesocolic defect. Koppman et al. published a very large series of 9527 patients and reported significantly higher rate of internal hernia after retrocolic vs. antecolic positioning of the Roux limb (2.4% vs. 0.3%), respectively, ($p < 0.0001$) [3]. This point was also reinforced by Escalona et al. who reported also an increased rate of

internal hernia with retrocolic vs. antecolic position of the Roux limb (9.3% vs 1.8%), respectively ($p > 0.001$) [37]. Other important considerations in the prevention of internal hernia are counterclockwise rotation of the Roux limb as mentioned by Nandipati et al. who reported that counterclockwise rotation of the Roux limb allows the jejuno-jejunostomy to lie in a more natural position on the left side of the abdomen to the left of the mesenteric axis. The study also reported a decline in the rate of internal hernia with counterclockwise vs. clockwise rotation (0.7% vs 6.9%), respectively ($p = 0.0018$), as it is believed that it renders the mesenteric defect easier to close completely [38]. Some other points include non-minimal or minimal division of small bowel mesentery, division of the omentum with placing the edges on either sides of the Roux limb, fashioning a long jejuno-jejunostomy and placing it above the colon in the left upper quadrant, and finally opting for 40 cm biliopancreatic limb [14, 39–42].

References

1. Brethauer SA, Kim J, El Chaar M, Papasavas P, Eisenberg D, Rogers A, Ballem N, Kligman M, Kothari S, Committee ACI. Standardized outcomes reporting in metabolic and bariatric surgery. Obes Surg. 2015;25(4):587–606.
2. Obeid A, McNeal S, Breland M, Stahl R, Clements RH, Grams J. Internal hernia after laparoscopic Roux-en-Y gastric bypass. J Gastrointest Surg. 2014;18(2):250–5. discussion 255–6
3. Koppman JS, Li C, Gandsas A. Small bowel obstruction after laparoscopic Roux-en-Y gastric bypass: a review of 9,527 patients. J Am Coll Surg. 2008;206(3):571–84.
4. Husain S, Ahmed AR, Johnson J, Boss T, O'Malley W. Small-bowel obstruction after laparoscopic Roux-en-Y gastric bypass: etiology, diagnosis, and management. Arch Surg. 2007;142(10):988–93.
5. Green J, Ikuine T, Hacker S, Urrego H, Tuggle K. Acute small bowel obstruction due to a large intraluminal blood clot after laparoscopic Roux-en-Y gastric bypass. J Surg Case Rep. 2016;2016(8):rjw143.
6. Daellenbach L, Suter M. Jejunojejunal intussusception after Roux-en-Y gastric bypass: a review. Obes Surg. 2011;21(2):253–63.
7. Hwang RF, Swartz DE, Felix EL. Causes of small bowel obstruction after laparoscopic gastric bypass. Surg Endosc. 2004;18(11):1631–5.
8. Sarhan M, Shyamali B, Fakulujo A, Ahmed L. Jejunal Bezoar causing obstruction after laparoscopic Roux-en-Y gastric bypass. JSLS. 2010;14(4):592–5.
9. Garza E Jr, Kuhn J, Arnold D, Nicholson W, Reddy S, McCarty T. Internal hernias after laparoscopic Roux-en-Y gastric bypass. Am J Surg. 2004;188(6):796–800.
10. Champion JK, Williams M. Small bowel obstruction and internal hernias after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2003;13(4):596–600.
11. Elms L, Moon RC, Varnadore S, Teixeira AF, Jawad MA. Causes of small bowel obstruction after Roux-en-Y gastric bypass: a review of 2,395 cases at a single institution. Surg Endosc. 2014;28(5):1624–8.
12. Schauer PR, Ikramuddin S, Hamad G, Eid GM, Mattar S, Cottam D, Ramanathan R, Gourash W. Laparoscopic gastric bypass surgery: current technique. J Laparoendosc Adv Surg Tech A. 2003;13(4):229–39.
13. Brolin RE, Kella VN. Impact of complete mesenteric closure on small bowel obstruction and internal mesenteric hernia after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2013;9(6):850–4.

14. Bauman RW, Pirrello JR. Internal hernia at Petersen's space after laparoscopic Roux-en-Y gastric bypass: 6.2% incidence without closure – a single surgeon series of 1047 cases. Surg Obes Relat Dis. 2009;5(5):565–70.
15. Schneider C, Cobb W, Scott J, Carbonell A, Myers K, Bour E. Rapid excess weight loss following laparoscopic gastric bypass leads to increased risk of internal hernia. Surg Endosc. 2011;25(5):1594–8.
16. Blachar A, Federle MP. Internal hernia: an increasingly common cause of small bowel obstruction. Semin Ultrasound CT MR. 2002;23(2):174–83.
17. Faria G, Preto J, Oliveira M, Pimenta T, Baptista M, Costa-Maia J. Petersen's space hernia: a rare but expanding diagnosis. Int J Surg Case Rep. 2011;2(6):141–3.
18. Higa KD, Ho T, Boone KB. Internal hernias after laparoscopic Roux-en-Y gastric bypass: incidence, treatment and prevention. Obes Surg. 2003;13(3):350–4.
19. Paroz A, Calmes JM, Romy S, Giusti V, Suter M. A new type of internal hernia after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2009;19(4):527–30.
20. Marinis A, Yiallourou A, Samanides L, Dafnios N, Anastasopoulos G, Vassiliou I, Theodosopoulos T. Intussusception of the bowel in adults: a review. World J Gastroenterol. 2009;15(4):407–11.
21. Filip JE, Mattar SG, Bowers SP, Smith CD. Internal hernia formation after laparoscopic Roux-en-Y gastric bypass for morbid obesity. Am Surg. 2002;68(7):640–3.
22. Spector D, Perry Z, Shah S, Kim JJ, Tarnoff ME, Shikora SA. Roux-en-Y gastric bypass: hyperamylasemia is associated with small bowel obstruction. Surg Obes Relat Dis. 2015;11(1):38–43.
23. Lockhart ME, Tessler FN, Canon CL, Smith JK, Larrison MC, Fineberg NS, Roy BP, Clements RH. Internal hernia after gastric bypass: sensitivity and specificity of seven CT signs with surgical correlation and controls. AJR Am J Roentgenol. 2007;188(3):745–50.
24. Ahmed AR, Rickards G, Johnson J, Boss T, O'Malley W. Radiological findings in symptomatic internal hernias after laparoscopic gastric bypass. Obes Surg. 2009;19(11):1530–5.
25. Kawkabani Marchini A, Denys A, Paroz A, Romy S, Suter M, Desmartines N, Meuli R, Schmidt S. The four different types of internal hernia occurring after laparascopic Roux-en-Y gastric bypass performed for morbid obesity: are there any multidetector computed tomography (MDCT) features permitting their distinction? Obes Surg. 2011;21(4):506–16.
26. Onopchenko A. Radiological diagnosis of internal hernia after Roux-en-Y gastric bypass. Obes Surg. 2005;15(5):606–11.
27. Iannuccilli JD, Grand D, Murphy BL, Evangelista P, Roye GD, Mayo-Smith W. Sensitivity and specificity of eight CT signs in the preoperative diagnosis of internal mesenteric hernia following Roux-en-Y gastric bypass surgery. Clin Radiol. 2009;64(4):373–80.
28. Altieri MS, Pryor AD, Telem DA, Hall K, Brathwaite C, Zawin M. Algorithmic approach to utilization of CT scans for detection of internal hernia in the gastric bypass patient. Surg Obes Relat Dis. 2015;11(6):1207–11.
29. Gandhi AD, Patel RA, Brolin RE. Elective laparoscopy for herald symptoms of mesenteric/internal hernia after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5(2):144–9. discussion 149
30. Dorsey ST, Harrington ET, Iv WF, Emerman CL. Ileus and small bowel obstruction in an emergency department observation unit: are there outcome predictors? West J Emerg Med. 2011;12(4):404–7.
31. Higa KD, Boone KB, Ho T. Complications of the laparoscopic Roux-en-Y gastric bypass: 1,040 patients – what have we learned? Obes Surg. 2000;10(6):509–13.
32. Van Dinter TG, Jr, John L, Guileyardo JM, John SF. Intestinal perforation caused by insertion of a nasogastric tube late after gastric bypass. Proc (Bayl Univ Med Cent). 2013;26(1):11–5.
33. Hanson RL. Predictive criteria for length of nasogastric tube insertion for tube feeding. JPEN J Parenter Enteral Nutr. 1979;3(3):160–3.
34. Byrne TK. Complications of surgery for obesity. Surg Clin North Am. 2001;81(5):1181–93. vii–viii

35. Aghajani E, Jacobsen HJ, Nergaard BJ, Hedenbro JL, Leifson BG, Gislason H. Internal hernia after gastric bypass: a new and simplified technique for laparoscopic primary closure of the mesenteric defects. J Gastrointest Surg. 2012;16(3):641–5.
36. O'Rourke RW. Management strategies for internal hernia after gastric bypass. J Gastrointest Surg. 2011;15(6):1049–54.
37. Escalona A, Devaud N, Perez G, Crovari F, Boza C, Viviani P, Ibanez L, Guzman S. Antecolic versus retrocolic alimentary limb in laparoscopic Roux-en-Y gastric bypass: a comparative study. Surg Obes Relat Dis. 2007;3(4):423–7.
38. Nandipati KC, Lin E, Husain F, Srinivasan J, Sweeney JF, Davis SS. Counterclockwise rotation of Roux-en-Y limb significantly reduces internal herniation in laparoscopic Roux-en-Y gastric bypass (LRYGB). J Gastrointest Surg. 2012;16(4):675–81.
39. Ortega J, Cassinello N, Sanchez-Antunez D, Sebastian C, Martinez-Soriano F. Anatomical basis for the low incidence of internal hernia after a laparoscopic Roux-en-Y gastric bypass without mesenteric closure. Obes Surg. 2013;23(8):1273–80.
40. Cho M, Pinto D, Carrodeguas L, Lascano C, Soto F, Whipple O, Simpfendorfer C, Gonzalvo JP, Zundel N, Szomstein S, Rosenthal RJ. Frequency and management of internal hernias after laparoscopic antecolic antegastric Roux-en-Y gastric bypass without division of the small bowel mesentery or closure of mesenteric defects: review of 1400 consecutive cases. Surg Obes Relat Dis. 2006;2(2):87–91.
41. Iannelli A, Buratti MS, Novellas S, Dahman M, Amor IB, Sejor E, Facchiano E, Addeo P, Gugenheim J. Internal hernia as a complication of laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2007;17(10):1283–6.
42. Madan AK, Lo Menzo E, Dhawan N, Tichansky DS. Internal hernias and nonclosure of mesenteric defects during laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2009;19(5):549–52.

Endoscopic Interventions for Complications in Bariatric Surgery

14

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Introduction

The treatment of bariatric surgery complications implies in early recognition of their signs and symptoms. Traditional surgical management options may be associated with increased morbidity and mortality. In this context, bariatric endoscopy aims to treat those complications with a less invasive nature and decreased morbidity.

Bariatric endoscopy is defined as an interface between advanced therapeutic endoscopy and bariatric surgery, involving the treatment of complications in a minimally invasive manner. Bariatric endoscopy is considered a new and unknown field by most endoscopists and surgeons. Literature about devices like dilation balloons, clips, scissors, and stents in bariatric surgery complications is growing [1, 2].

On this chapter, we present the role of endoscopy in the diagnosis and treatment of the main complications of bariatric surgery.

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Roux-en-Y Gastric Bypass (RYGB)

Anastomotic Stricture

Gastrojejunal anastomotic stricture is one of the most common complications of RYGB, defined when the diameter is <10 mm and common endoscope (9.8 mm) passage is not possible. Most common symptoms are food intolerance, vomiting, and epigastric pain, usually becoming symptomatic when solid diet is started. The treatment can be done through endoscopic hydrostatic balloon dilation, using TTS (through the scope) balloons, with diameters usually up to 15 mm. This approach reaches success in 98% of cases, in a mean of 1.7 sessions per patient, with a complication rate of 2.5%, mostly perforations and bleeding [3, 4]. Some reports also use the Savary-Gilliard bougie for dilation [5]. In cases of failure, endoscopic stenotomy can be performed, using an endoscopic cautery to make incisions on the stricture, followed by balloon dilation.

Marginal Ulcers

Marginal ulcers after RYGB have a multifactorial etiology, including tobacco and alcohol consumption, nonsteroidal anti-inflammatory drug (NSAID) usage, gastrogastric fistulas, and foreign bodies. Ulcers are more common at the jejunal side of the anastomosis, with varying size and depth [6]. Main symptoms are epigastric pain, dysphagia, nausea, and vomiting, with some cases being asymptomatic [7]. Any foreign bodies, such as visible sutures and staples, should be endoscopically removed to improve ulcer healing, together with proton pump inhibitor and sucralfate prescription [8]. Ulcers can cause strictures due to fibrotic scar formation, and these can be treated through stenotomy and balloon dilation [4] (Fig. 14.1).

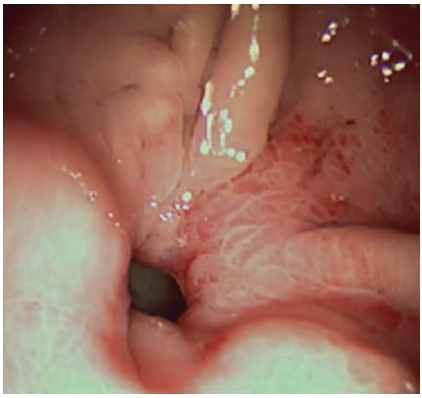

Fig. 14.1 Anastomotic stricture secondary to ulcer fibrotic scar tissue formation

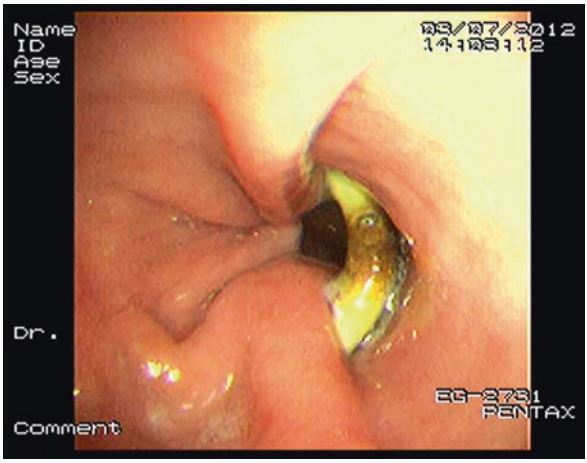

Fig. 14.2 Endoscopic view of eroded ring after RYGB, showing approximately 50% erosion; endoscopic removal with scissors

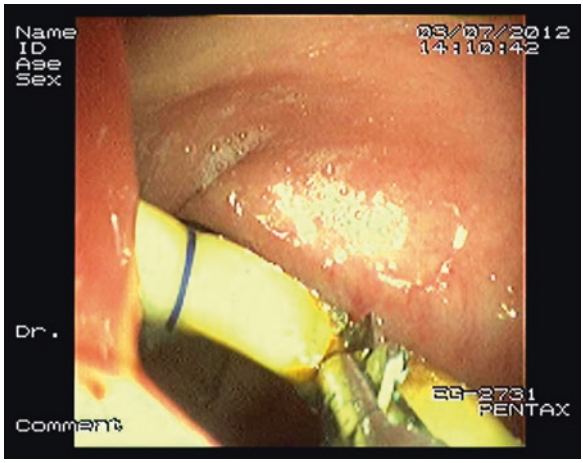

Fig. 14.3 Endoscopic view of eroded ring after RYGB, showing approximately 50% erosion; endoscopic removal with scissors

Ring Complications: Intragastric Erosion

This complication has an incidence of 0,9 a 7%; most common symptoms of intragastric ring erosion are weight regain, nausea, vomiting, and bleeding [9]. Endoscopy may show the ring inside the gastric pouch, and in early stages, an ulcer at the erosion site can be the only visible sign. In this case, PPIs should be prescribed until complete ring erosion, with surveillance endoscopy performed. Once >30% of the ring circumference is visible inside the gastric pouch, endoscopic removal is the gold standard. Removal is done using endoscopic scissors to section the ring. In cases of failure in cutting the ring, a gastric band cutter or lithotripter may be used. Treatment should be scheduled as soon as possible, due to the risk of gastric wall bleeding or food impaction [10] (Figs. 14.2 and 14.3).

Ring Slippage

Distal ring slippage promotes an angulation of the longitudinal axis of the gastric pouch, and proximal gastric dilatation, leading to obstructive symptoms with an incidence lower than 1% [11]. Diagnosis can be made with contrast x-ray, showing an area of contrast retention, and endoscopy, which may show food stasis and convergence of the mucosal folds, caused by the jejunal obstruction just beneath the anastomosis [12].

Management can be done through endoscopic 30 mm achalasia balloon dilation that stretches or ruptures the thread running inside the ring, thus relieving symptoms with a low complication rate [11]. Stent placement can also be used for removal, causing an inflammatory/ischemic reaction around the ring, promoting intragastric erosion, with stent and ring removal possible after 10–15 days. A fibrotic scar tissue forms in the ring erosion area, restricting the pouch diameter, with better weight control when compared to dilation [13–15] (Figs. 14.4 and 14.5).

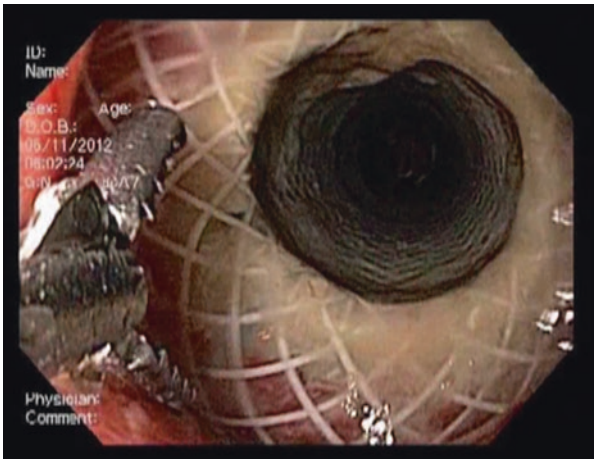

Fig. 14.4 Endoscopic view of plastic stent placed to induce ring erosion – possible to visualize ring compression on stent – and ring completely eroded after stent removal

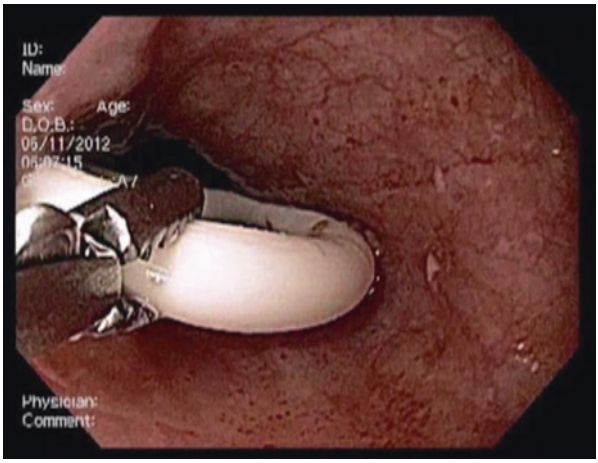

Fig. 14.5 Endoscopic view of plastic stent placed to induce ring erosion – possible to visualize ring compression on stent – and ring completely eroded after stent removal

Food Intolerance

In some cases, vomiting episodes may occur after RYGB even when there is no ring slippage or gastric pouch/anastomotic stricture, which can be defined as ring-related food intolerance. This affects quality of life, with difficulties in food ingestion. Symptoms are similar to when there is a stricture: dysphagia, solid food intolerance, postprandial vomiting, and, in advanced stages, excessive weight loss, dehydration, and malnutrition.

These patients can also be treated by ring dilation or stenting, leading to improvement of symptoms in more than 96% of cases. The procedure has low cost and morbidity, with nonsignificant weight regain [16].

Sleeve Gastrectomy

Gastric Stricture

In post-sleeve gastric strictures, endoscopy can show a reduction in the gastric lumen, usually near the *incisura*, associated to a difficult endoscope progression or axis deviation. Typical symptoms include dysphagia, vomiting, and excessive weight loss. Treatment can be done with pneumatic 30 mm achalasia balloon dilation, associated to stenotomy when necessary [17, 18]. When endoscopic treatment fails, surgical management can be done through RYGB conversion, or, in some cases, total gastrectomy if surgical manipulation is too difficult [19] (Fig. 14.6).

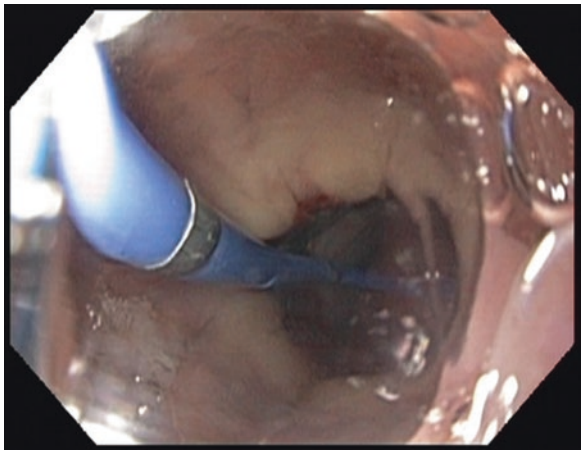

Fig. 14.6 Dilation of sleeve gastrectomy stricture with 30 mm achalasia balloon

RYGB and SG Leaks

Gastric leaks represent one of the most feared complications after RYGB and SG. Treatment modality is made according to patient status and local fistula conditions. Initial measures include drainage (surgical or percutaneous), antibiotics, and nutritional support [20, 21]. When there is contamination of the abdominal cavity – peritonitis/perigastric abscess – surgical drainage can be an option. In a stable patient with a functioning and well-located peritoneal drain, conservative management may be suitable. Also, the perigastric abscess can be approached through percutaneous drainage or, in selected cases, internal drainage through endoscopy [22].

Early diagnostic endoscopy allows evaluation of the leak internal orifice and identification of associated strictures and helps in correct positioning of abdominal drains and performance of internal abscess drainage. After initial leak control, specific surgical or endoscopic measures are taken. The endoscopic management is linked to decreased morbidity, involving internal drainage, septotomy, dilation, endoscopic suturing, clips, and, in most cases, endoscopic stenting [23–29].

Endoscopic therapy has the aim of solving the three main issues perpetuating the leak: distal gastric stricture, increased intragastric pressure, and fistulous tract persistence. In SG, additionally, there can be an axis deviation with associated increased intragastric pressure [18].

Treatment choice is made according to time of onset, divided in four phases:

- Acute (<7 days): stent
- Early (1–6 weeks): stent + balloon dilation + septotomy (rare)
- Late (6–12 weeks): septotomy + balloon dilation + stent (rare)
- Chronic (>12 weeks): septotomy + balloon dilation [19]

In *acute and early leaks*, self-expandable metallic stents (SEMS) act promoting occlusion of the leak orifice, correction of axis deviation, and distal strictures, also decreasing intragastric pressure, which leads to leak closure [19, 29].

Bariatric customized stents have been developed with a design customized for SG, with promising initial results and decreased complications [26, 30–33] (Fig. 14.7) Stents should not be left in place for 1–2 months, what is usually enough to correct strictures and deviations, with lower migration and easier removal [23]. After initial leak control, stent is removed even if complete orifice closure is not achieved. When needed, endoscopic treatment continues through septotomy, stenotomy, and balloon dilations, which will lead to complete fistula closure. In some early cases, internal drainage with pigtail drains has been described with success, especially in smaller leaks (<10 mm) with associated perigastric abscess [29, 34]. Other endoscopic approaches include usage of endoscopic clips, biologic glue, and tissue sealants, with controversial results [35, 36]. Endoscopic vacuum therapy has also been described [37, 38].

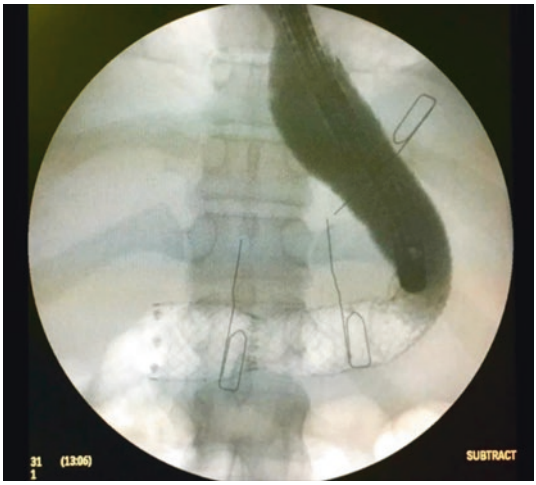

Fig. 14.7 Radioscopic view of long bariatric stent placed for sleeve gastrectomy leak treatment

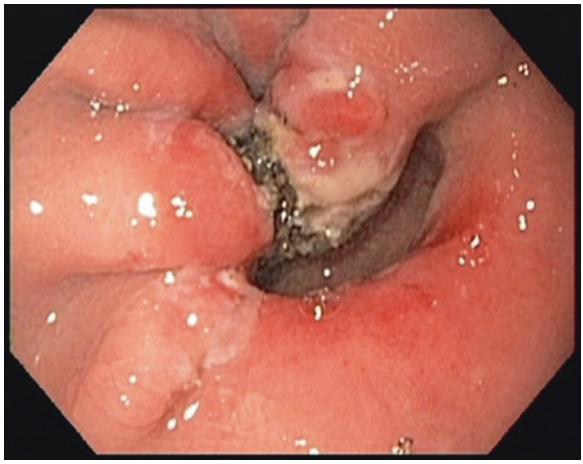

Fig. 14.8 Endoscopic view of septotomy: leak orifice on the left side, septum, and gastric lumen on the right side

For *late and chronic leaks*, endoscopic multimodal treatment is usually done through multiple sessions using different techniques. When there is a septum adjacent to the fistulous orifice, septotomy is performed, decreasing flow of gastric contents through the fistula [39]. Septotomy is done with needle knife or argon plasma coagulation (associated to less bleeding), followed by balloon dilation (Fig. 14.8). When there is stenosis and fibrotic tissue associated, stenotomy associated to balloon dilation may be used. This endoscopic therapy can be performed on an outpatient basis, with low morbidity and mortality and better quality of life. The correction in digestive contents flow will eventually lead to leak closure [18]. Stents can be used in selected cases, especially when there are anatomical defects.

Adjustable Gastric Band

Intragastric Band Erosion

This complication is one of the most common after this procedure, occurring in about 1.6% of patients, with nonspecific symptomatology [40]. The patient may present with weight regain, epigastric pain, portal infection, dysphagia, fever, hemorrhage, or obstruction. Endoscopy shows the presence of a segment of the gastric band in the gastric lumen, near the cardia, with better visualization under retroflection [4, 5].

In early stages of band erosion and asymptomatic patients, conservative approach is recommended, until intragastric erosion is greater than 50% of the circumference of the band. During this period, surveillance is essential, with use of proton pump inhibitor, due to the risk of gastrointestinal bleeding or perigastric abscess [41, 42]. When there is enough intragastric migration (>50% of its circumference), endoscopic cutting and removal can be performed with high success and low complication rates, using endoscopic scissors or a gastric band cutter [41] (Fig. 14.9).

Band Slippage

Distal band slippage can cause proximal gastric reservoir dilation, with associated obstructive symptoms, like nausea, vomiting, dehydration, halitosis, excessive weight loss, heartburn, and abdominal pain [9]. Diagnosis can be confirmed with a

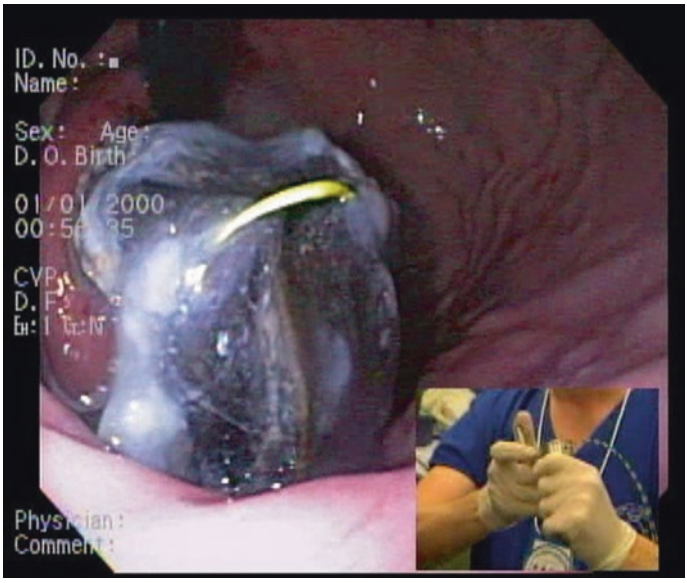

Fig. 14.9 Eroded adjustable gastric band, with removal using a gastric band cutter (similar to a lithotripter)

contrast x-ray or endoscopy, which will show a dilation in the gastric pouch with food stasis above the compression area of the band. Under retroflection, a retraction of the mucosa is seen in the slippage area, with exuberant and edematous folds, and difficult passage to the antrum [9].

For temporary symptoms relief, an endoscopic maneuver can be done: hyperinflation of the gastric body, below the compression level, which can lead to band repositioning. The success of this maneuver does not modify the need for band removal [10].

Classical laparoscopic removal is the appropriate therapy, with attention to the risk of bronchial aspiration. To avoid this, and for temporary symptoms relief, endoscopic approach under light sedation can be done, with aspiration of gastric contents. After passing of the endoscope past the band compression area, the stomach is hyperinflated, which will promote proximal slippage of the device, leading to a repositioning of the band to its usual site. This will relieve symptoms and decrease risks until definitive surgical removal is performed [43].

Endoscopic Treatment of Weight Regain

One of the potential causes of weight regain after RYGB is dilation of the gastrojejunal anastomosis and gastric pouch enlargement. In a study of 165 patients, it was found that the diameter of the anastomosis is a risk factor for weight regain after RYGB and that this variable should be included as a predictor of weight regain [44]. Recently, Ramos et al. published a study evaluating the size of the gastrojejunal anastomosis and its influence on weight loss. In a 2-year follow-up, a stoma diameter of 15 mm presented statistically better results than a 45-mm anastomosis [45]. The ideal anastomosis should have an approximate diameter of 10 mm, not exceeding 14 mm [44].

The most relevant aspects to indicate endoscopic treatment of weight regain after RYGB are a large gastric pouch and gastrojejunal anastomosis. However, there is a need to evaluate other alterations that may be leading the patient to gain weight, like gastrogastric fistula, ring slippage or stricture, and any type stenosis [46]. A chronic stenosis causes food intolerance for which patients will, over the years, select food that most appeals to them, which are usually carbohydrates, sweets, and caloric liquids, which pass the stricture easily.

Development of endoluminal therapies for pouch and stoma revision can be a less invasive approach for failure or weight regain after bariatric surgery. Argon plasma coagulation leads to fusion of tissues and induces an inflammatory and fibrotic response. This fibrotic response is a side effect that is positive if used in order to reduce the anastomosis after RYGB [47]. Argon plasma coagulation can only be employed to narrow the anastomosis as it is not indicated in cases of enlarged pouches. The cost is low; it is more accessible and does not need a service of high complexity, with sedation being the means of anesthesia.

To produce the desired effect, the coagulation should be done in a circumferential way, involving the entire gastric side of the anastomosis, extending for 1–2 cm

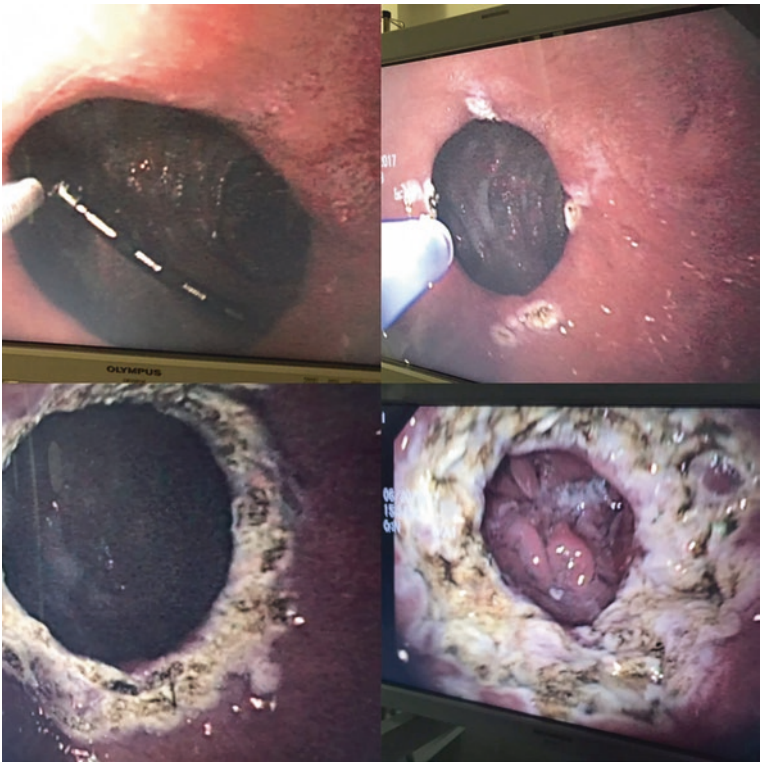

Fig. 14.10 Dilated RYGB gastrojejunal anastomosis, performance of argon plasma coagulation for stoma reduction

(Fig. 14.10). There is an initial edema and inflammatory response, causing immediate restriction. This effect decreases over time, and the edema is substituted by fibrosis. More than one session is usually necessary in order to achieve long lasting effects [48]. Argon coagulation reduces the diameter of the anastomosis and consequently delays gastric emptying and early satiety and improves weight reduction [49]. One of the possible complications or argon plasma use is stricture of the coagulated anastomosis. This can be treated through endoscopic dilation with TTS balloons. In some cases, the obstructive symptoms will resolve by itself, hindering the need of intervention.

The use of sutures allows the concomitant treatment of a dilated anastomosis and a large pouch or the treatment of one or the other in isolation. A retrospective series of eight patients evaluated safety and short-term efficacy of the Overstitch™ in patients with weight regain and dilated gastrojejunostomy. Pre-procedure pouch size varied from 2 to 6 cm and stoma size from 20 to 40 mm. Procedure time ranged from 20 to 60 min (mean 38 min), all but one patient had three stitches applied, reducing stoma size to a 10 mm diameter. Post-procedure weight loss in a 90 days follow-up varied from 6 to 8 kg, with a mean % of regained weight loss of 28%. No postoperative complications were recorded [50].

References

1. Sullivan S, Kumar N, Edmundowicz SA, Abu Dayyeh BK, Jonnalagadda SS, Larsen M, et al. ASGE position statement on endoscopic bariatric therapies in clinical practice. Gastrointest Endosc. 2015;82(5):767–72.
2. Cai JX, Schweitzer MA, Kumbhari V. Endoscopic management of bariatric surgery complications. Surg Laparosc Endosc Percutan Tech. 2016;26(2):93–101.
3. Campos JM, Mello FS, Ferraz AA, Brito JN, Nassif PA, Galvao-Neto Mdos P. Endoscopic dilation of gastrojejunal anastomosis after gastric bypass. Arq Bras Cir Dig. 2012;25(4):283–9.
4. de Moura EG, Orso IR, Aurelio EF, de Moura ET, de Moura DT, Santo MA. Factors associated with complications or failure of endoscopic balloon dilation of anastomotic stricture secondary to roux-en-Y gastric bypass surgery. Surg Obes Relat Dis. 2016;12(3):582–6.
5. Escalona A, Devaud N, Boza C, Perez G, Fernandez J, Ibanez L, et al. Gastrojejunal anastomotic stricture after Roux-en-Y gastric bypass: ambulatory management with the Savary-Gilliard dilator. Surg Endosc. 2007;21(5):765–8.
6. Sapala JA, Wood MH, Sapala MA, Flake TM Jr. Marginal ulcer after gastric bypass: a prospective 3-year study of 173 patients. Obes Surg. 1998;8(5):505–16.
7. Huang CS, Forse RA, Jacobson BC, Farraye FA. Endoscopic findings and their clinical correlations in patients with symptoms after gastric bypass surgery. Gastrointest Endosc. 2003;58(6):859–66.
8. Garrido AB Jr, Rossi M, Lima SE Jr, Brenner AS, Gomes CAR Jr. Early marginal ulcer following Roux-en-Y gastric bypass under proton pump inhibitor treatment: prospective multicentric study. Arq Gastroenterol. 2010;47:130–4.
9. Huang CS, Farraye FA. Endoscopy in the bariatric surgical patient. Gastroenterol Clin N Am. 2005;34(1):151–66.
10. Fobi M, Lee H, Igwe D, Felahy B, James E, Stanczyk M, et al. Band erosion: incidence, etiology, management and outcome after banded vertical gastric bypass. Obes Surg. 2001;11(6):699–707.
11. Campos JM, Evangelista LF, Ferraz AA, Galvao Neto MP, De Moura EG, Sakai P, et al. Treatment of ring slippage after gastric bypass: long-term results after endoscopic dilation with an achalasia balloon (with videos). Gastrointest Endosc. 2010;72(1):44–9.
12. Espinel J, Pinedo E. Stenosis in gastric bypass: endoscopic management. World J Gastrointest Endosc. 2012;4(7):290–5.
13. Marins Campos J, Moon RC, Magalhaes Neto GE, Teixeira AF, Jawad MA, Bezerra Silva L, et al. Endoscopic treatment of food intolerance after a banded gastric bypass: inducing band erosion for removal using a plastic stent. Endoscopy. 2016;48(6):516–20.
14. Blero D, Eisendrath P, Vandermeeren A, Closset J, Mehdi A, Le Moine O, et al. Endoscopic removal of dysfunctioning bands or rings after restrictive bariatric procedures. Gastrointest Endosc. 2010;71(3):468–74.
15. Wilson TD, Miller N, Brown N, Snyder BE, Wilson EB. Stent induced gastric wall erosion and endoscopic retrieval of nonadjustable gastric band: a new technique. Surg Endosc. 2013;27(5):1617–21.
16. Ferraz A, Campos J, Dib V, Silva LB, de Paula PS, Gordejuela A, et al. Food intolerance after banded gastric bypass without stenosis: aggressive endoscopic dilation avoids reoperation. Obes Surg. 2013;23(7):959–64.
17. Shnell M, Fishman S, Eldar S, Goitein D, Santo E. Balloon dilatation for symptomatic gastric sleeve stricture. Gastrointest Endosc. 2014;79(3):521–4.
18. Zundel N, Hernandez JD, Galvao Neto M, Campos J. Strictures after laparoscopic sleeve gastrectomy. Surg Laparosc Endosc Percutan Tech. 2010;20(3):154–8.
19. Rosenthal RJ, Diaz AA, Arvidsson D, Baker RS, Basso N, Bellanger D, et al. International sleeve gastrectomy expert panel consensus statement: best practice guidelines based on experience of >12,000 cases. Surg Obes Relat Dis. 2012;8(1):8–19.

20. Vix M, Diana M, Marx L, Callari C, Wu HS, Perretta S, et al. Management of staple line leaks after sleeve gastrectomy in a consecutive series of 378 patients. Surg Laparosc Endosc Percutan Tech. 2015;25(1):89–93.
21. Bhayani NH, Swanstrom LL. Endoscopic therapies for leaks and fistulas after bariatric surgery. Surg Innov. 2014;21(1):90–7.
22. Baretta G, Campos J, Correia S, Alhinho H, Marchesini JB, Lima JH, et al. Bariatric postoperative fistula: a life-saving endoscopic procedure. Surg Endosc. 2015;29(7):1714–20.
23. Campos JM, Pereira EF, Evangelista LF, Siqueira L, Neto MG, Dib V, et al. Gastrobronchial fistula after sleeve gastrectomy and gastric bypass: endoscopic management and prevention. Obes Surg. 2011;21(10):1520–9.
24. Silva LB, Moon RC, Teixeira AF, Jawad MA, Ferraz AA, Neto MG, et al. Gastrobronchial fistula in sleeve gastrectomy and Roux-en-Y gastric bypass-a systematic review. Obes Surg. 2015;25(10):1959–65.
25. Puli SR, Spofford IS, Thompson CC. Use of self-expandable stents in the treatment of bariatric surgery leaks: a systematic review and meta-analysis. Gastrointest Endosc. 2012;75(2):287–93.
26. Basha J, Appasani S, Sinha SK, Siddappa P, Dhaliwal HS, Verma GR, et al. Mega stents: a new option for management of leaks following laparoscopic sleeve gastrectomy. Endoscopy. 2014;46(Suppl 1 UCTN):E49–50.
27. Shehab HM, Hakky SM, Gawdat KA. An endoscopic strategy combining mega stents and over-the-scope clips for the management of post-bariatric surgery leaks and fistulas (with video). Obes Surg. 2015;26:941.
28. Fischer A, Bausch D, Richter-Schrag HJ. Use of a specially designed partially covered self-expandable metal stent (PSEMS) with a 40-mm diameter for the treatment of upper gastrointestinal suture or staple line leaks in 11 cases. Surg Endosc. 2013;27(2):642–7.
29. Nedelcu M, Manos T, Cotirlet A, Noel P, Gagner M. Outcome of leaks after sleeve gastrectomy based on a new algorithm adressing leak size and gastric stenosis. Obes Surg. 2015;25(3):559–63.
30. van Wezenbeek MR, de Milliano MM, Nienhuijs SW, Friederich P, Gilissen LP. A specifically designed stent for anastomotic leaks after bariatric surgery: experiences in a tertiary referral hospital. Obes Surg. 2015;
31. Fishman MB, Sedov VM, Lantsberg L. Laparoscopic adjustable gastric banding in treatment of patients with obesity. Vestn Khir Im I I Grek. 2008;167(1):29–32.
32. Galloro G, Magno L, Musella M, Manta R, Zullo A, Forestieri P. A novel dedicated endoscopic stent for staple-line leaks after laparoscopic sleeve gastrectomy: a case series. Surg Obes Relat Dis. 2014;10(4):607–11.
33. Bezerra Silva L, Galvao Neto M, Marchesini JC, SNG E, Campos J. Sleeve gastrectomy leak: endoscopic management through a customized long bariatric stent. Gastrointest Endosc. 2017;85(4):865–6.
34. Pequignot A, Fuks D, Verhaeghe P, Dhahri A, Brehant O, Bartoli E, et al. Is there a place for pigtail drains in the management of gastric leaks after laparoscopic sleeve gastrectomy? Obes Surg. 2012;22(5):712–20.
35. Caballero Y, Lopez-Tomassetti E, Castellot A, Hernandez JR. Endoscopic management of a gastric leak after laparoscopic sleeve gastrectomy using the over-the-scope-clip (Ovesco(R)) system. Rev Esp Enferm Dig. 2016;108
36. Keren D, Eyal O, Sroka G, Rainis T, Raziel A, Sakran N, et al. Over-the-scope clip (OTSC) system for sleeve gastrectomy leaks. Obes Surg. 2015;25(8):1358–63.
37. Seyfried F, Reimer S, Miras AD, Kenn W, Germer CT, Scheurlen M, et al. Successful treatment of a gastric leak after bariatric surgery using endoluminal vacuum therapy. Endoscopy. 2013;45(Suppl 2 UCTN):E267–8.
38. Hwang JJ, Jeong YS, Park YS, Yoon H, Shin CM, Kim N, et al. Comparison of endoscopic vacuum therapy and endoscopic stent implantation with self-expandable metal stent in treating postsurgical gastroesophageal leakage. Medicine (Baltimore). 2016;95(16):e3416.

39. Campos JM, Ferreira FC, Teixeira AF, Lima JS, Moon RC, D'Assuncao MA, et al. Septotomy and balloon dilation to treat chronic leak after sleeve gastrectomy: technical principles. Obes Surg. 2016;
40. Nocca D, Frering V, Gallix B, De Seguin des Hons C, Noel P, Foulonge MA, et al. Migration of adjustable gastric banding from a cohort study of 4236 patients. Surg Endosc. 2005;19(7):947–50.
41. Neto MP, Ramos AC, Campos JM, Murakami AH, Falcao M, Moura EH, et al. Endoscopic removal of eroded adjustable gastric band: lessons learned after 5 years and 78 cases. Surg Obes Relat Dis. 2010;6(4):423–7.
42. Campos JM, Evangelista LF, Galvao Neto MP, Ramos AC, Martins JP, dos Santos MA Jr, et al. Small erosion of adjustable gastric band: endoscopic removal through incision in gastric wall. Surg Laparosc Endosc Percutan Tech. 2010;20(6):e215–7.
43. Kang SH, Kim KC, Kim KH. Endoscopic treatment of gastric band prolapse. Obes Surg. 2014;24(6):954–7.
44. Abu Dayyeh BK, Lautz DB, Thompson CC. Gastrojejunal stoma diameter predicts weight regain after Roux-en-Y gastric bypass. Clin Gastroenterol Hepatol. 2011;9(3):228–33.
45. Ramos AC, Marchesini JC, de Souza Bastos EL, Ramos MG, de Souza MDG, Campos JM, et al. The role of gastrojejunostomy size on gastric bypass weight loss. Obes Surg. 2017;27:2317.
46. Yimcharoen P, Heneghan HM, Singh M, Brethauer S, Schauer P, Rogula T, et al. Endoscopic findings and outcomes of revisional procedures for patients with weight recidivism after gastric bypass. Surg Endosc. 2011;25(10):3345–52.
47. Fernandez-Esparrach G, Lautz DB, Thompson CC. Peroral endoscopic anastomotic reduction improves intractable dumping syndrome in Roux-en-Y gastric bypass patients. Surg Obes Relat Dis. 2010;6(1):36–40.
48. Aly A. Argon plasma coagulation and gastric bypass – a novel solution to stomal dilation. Obes Surg. 2009;19(6):788–90.
49. Baretta GA, Alhinho HC, Matias JE, Marchesini JB, de Lima JH, Empinotti C, et al. Argon plasma coagulation of gastrojejunal anastomosis for weight regain after gastric bypass. Obes Surg. 2015;25(1):72–9.
50. Galvão Neto M, Rodriguez L, Zundel N, Ayala JC, Campos J, Ramos A. Endoscopic revision of Roux-en-Y gastric bypass stomal dilation with a suturing device: preliminary results of a first out-of-United States series. Bariatric Times. 2011;8(6):32–4.

Pregnancy in the Bariatric Patient

15

Maria S. Altieri and Aurora D. Pryor

Introduction

The prevalence of adult obesity is increasing in the United States and around the world. Bariatric surgery is proving to be the only efficacious means for treatment of obesity and obesity-related comorbidities. With the parallel rise of bariatric surgery, we are faced with more people who have undergone bariatric procedures. As nearly one in two women of childbearing age is considered either overweight or obese [1] and over 80,000 women of childbearing age are undergoing bariatric surgery each year [2–4], bariatric surgeons should be well versed on management considerations for pregnant women following bariatric surgery. It is important to provide care as a multidisciplinary team consisting of the surgeon, family physician, and the obstetricians and gynecologists, for these patients to assure a safe and healthy pregnancy.

This chapter will review the effects of bariatric surgery on pregnancy, taking care of a bariatric patient during pregnancy and delivery and dealing with complications due to bariatric surgery that may present during pregnancy.

The Effects of Bariatric Surgery on Pregnancy

Obesity during pregnancy increases the risk of various short- and long-term maternal and fetal complications such as pregnancy-induced hypertension (PIH), gestational diabetes, thrombosis, difficulty in delivery leading to higher cesarean section (CS) rates, hemorrhage, miscarriage, fetal abnormality, prematurity, macrosomia, birth injury, still birth, and maternal and neonatal death [5, 6]. The relationship between obesity and infertility is well established, as obesity can cause a state of hyperandrogenism, leading to amenorrhea and endocrine infertility [7]. The

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association between polycystic ovarian syndrome (PCOS), a common endocrine system disorder among women, which causes infertility, menstrual dysfunction, and miscarriages, and obesity is also well known [7, 8].

Bariatric surgery leads to rapid weight loss, which can reverse the mechanism of infertility. The menstrual cycle disorders may completely resolve after bariatric surgery [9]. Deitel et al. reported improvement of menstrual irregularities post-bariatric surgery (40.4% versus 4.6%, $p < 0.001$). Infertility problems were also present in 29.3% of preoperative obese women. All women who tried to conceive postoperatively were successful [10]. Milone et al. performed a systematic review and meta-analysis of the literature and reported a high incidence (58%) of infertile women who became spontaneously pregnant after bariatric surgery [11]. In addition, PCOS symptoms resolve postoperatively [12, 13]. Eid et al. reported an observational study of 24 women with diagnosis of PCOS, who underwent Roux-en-Y gastric bypass. Post-procedure menstrual cyclicity improved in all women. Twenty-one percent of women conceived naturally [13]. Studies report successful conception post-bariatric surgery to be between 15% and 44% [5, 14–16].

Although the majority of studies show that the rates of conception are improved, some studies still show that it is more difficult to conceive even after bariatric surgery when compared to the normal weight population. When compared to the general population, post-bariatric patients had a higher need for fertility treatment (6.7% versus 2.3%) [17]. Aricha-Tamir showed no difference in rates of infertility treatment prior to surgery [18]. However, the extent of weight loss may play a role in the potential for conception [19].

Timing of Pregnancy Following Bariatric Surgery

The first year following bariatric surgery is associated with an active catabolic state due to weight loss. In addition, due to decreased intake, there is a concern for nutritional instabilities. These are particularly common with malabsorptive types of bariatric surgery, such as RYGB and BPD/DS, or with non-compliance with supplements. Common deficiencies occur commonly with iron, vitamin B12, folate, vitamins K and A, and calcium, which can lead to maternal and fetal complications. Thus, the American Congress of Obstetricians and Gynecologists (ACOG) suggests a waiting period of at least 12–24 months following bariatric surgery [5] prior to pregnancy. However, data regarding the risks of conception shortly following bariatric procedures is limited and conflicting.

In a study comparing 21 patients who became pregnant within the first year after surgery compared to 13 that became pregnant over 1 year postoperatively, there was no difference in terms of fetal weight, term pregnancy, or complications [20]. A more extensive study, assessing a cohort of 286 women following RYGB, showed no difference between women who conceived during or after the first year of surgery in terms of birth weight, generational age, preeclampsia, gestational diabetes mellitus, labor induction, and need for cesarean section, among other variables [21].

Other studies have shown a higher spontaneous abortion rate and more frequent preterm deliveries [22–24] following bariatric surgery. Printen and Scott showed high rate of premature births in the first 2 years following RYGB [24]. A rate of 29% of spontaneous abortions was reported in a study of patients who conceived in the first 2 years post-procedure [23]. Given the mixed studies, a waiting period of 12–24 months should be recommended to all women who wish to conceive following bariatric surgery. If the patient becomes pregnant during that period, both the patient and the fetus should be closely monitored [5]. If nutritional supplementation is required, overall surgical weight loss may also be jeopardized.

Use of Contraception

The use of contraception is an important topic for patients who are of childbearing age and are undergoing bariatric surgery. As fertility rates can improve following bariatric surgery and the recommended period of waiting for conception is 12–24 months, the use of contraception should be discussed. Malabsorption of oral contraceptives has been suggested, as there is the potential for decreased absorption and lower effectiveness [15, 25]. Thus, ACOG recommends the use of non-oral contraception, with barrier methods as one preferred method following bariatric surgery [5].

Patients should be thoroughly counseled regarding effectiveness and adverse effects of methods of contraception. Alarmingly, Mody et al. reported that only 21% of post-bariatric patients were referred to a gynecologist for contraceptive counseling [16].

Nutritional Status

Nutritional deficiencies may vary depending on the type of procedure, as they are less common during gastric-specific procedures and more common following malabsorptive procedures. Following malabsorptive procedures, nutritional deficiencies, such as iron, folate, thiamine, vitamin B 12, fat-soluble vitamins (vitamins A, D, E, K), calcium, and protein, are not infrequent. Most pregnant women are advised to take prenatal vitamins. As the nutritional requirements are higher during pregnancy, in addition to a higher incidence of nausea and vomiting, following bariatric surgery the potential for clinically relevant deficiencies is increased. These deficiencies can lead to fetal intracranial hemorrhage, neurologic and developmental impairment, neural tube defects, or vision problems [26, 27].

Supplementation of multivitamins and micronutrients is important for patients following surgery. In case of the pregnant bariatric patient, it is vital that supplementation is used and counseling is provided. Ideally, patients should be screened prior to conception for any deficiencies. In addition, in case the patient desires pregnancy or is pregnant, counseling regarding specific supplementation is necessary, as some of the supplementation may have teratogenic effects on the fetus, as in the

case of retinol-based vitamin A. Compliance is important [5]. Screening should be used with some experts suggesting monitoring for deficiencies each trimester [5]. When deficiency has been established, oral supplementation should be initiated. In case patients are not tolerating a tablet or capsule, a chewable or liquid form taken with food can be prescribed. In addition to vitamin and mineral supplementation, protein supplementation should be considered as well for patients who have lost weight or are not gaining weight or for fetal growth below the 50th percentile [28].

Complications Encountered in the Pregnant Bariatric Patient

In general, management of the bariatric pregnant patient should comprise of a multidisciplinary approach, involving surgeons, obstetricians, primary care physicians, and dietitians. Thus, both the health of the mother and the fetus can be addressed. As complications of bariatric surgery have been reported to lead to morbidity and mortality [4, 29], the bariatric surgeon should be involved early to minimize these risks.

Post-bariatric pregnant patients may develop procedure-specific complications during pregnancy. Pregnancy predisposes to increased intra-abdominal pressure, reduced gastric volume, displacement of intra-abdominal contents, and predisposition to nausea and vomiting. It is vital to distinguish between complications due to bariatric surgery and physiological manifestations of pregnancy. Nausea, vomiting, and occasional cramping/abdominal pain can be normal during pregnancy. However, the provider should have a high suspicion for complications due to history of bariatric surgery in the pregnant bariatric patient as these symptoms can represent a more serious problem that may necessitate surgical intervention. Thus, an urgent surgical consultation should be sought. The provider's suspicion should be based on the type of bariatric procedure.

Radiology Considerations in the Pregnant Bariatric Patient

Diagnosis of complications in post-bariatric patients will often involve the use of radiographic studies, including an abdominal radiograph, an upper gastrointestinal series (upper GI), and/or a computer tomography (CT scan). All of these studies have some degree of radiation exposure. Due to that many physicians will be reluctant to obtain these studies or may substitute MR imaging as appropriate.

In pregnant patients, although the use of a single diagnostic procedure may be less than 5 rads, depending on the trimester, there are concerns of radiation exposure to the fetus. Recently, ACOG published their statement on diagnostic imaging during pregnancy and lactation [30]. Growth restriction, microcephaly, and intellectual disabilities are the most common effects of radiation exposure, with minimal threshold effects between 60 and 310 mGy. A single abdominal X-ray leads to radiation exposure to the fetus of 0.1–3 mGy, and even multiple X-rays rarely amount to a 50 mGy dose which is set as the cutoff for safety. Abdominal CT exposes the fetus

to 1.3–35 mGy of radiation [30]. If concerned about an intra-abdominal complication in the pregnant post-bariatric patient, the concern for radiation exposure should not preclude further work-up, as the prompt diagnosis and treatment far outweigh any fetal risks of teratogenicity.

Complications Related to Laparoscopic Adjustable Gastric Banding

Although the laparoscopic adjustable gastric banding has fallen out of favor and the number of newly placed devices has significantly decreased, there are still patients who may present during pregnancy who have had this procedure. Studies have shown that LAGB is tolerable in pregnancy and babies born to women with LAGB are as healthy as children born to the general population [31]. Similar to nonpregnant patients, common complications during pregnancy following LAGB include uncontrollable nausea and vomiting, band erosion, port leaks, pouch dilation, and prolapse/slip of the band. A review of 728 pregnancies in 638 patients reported a low rate complication of 2.3% involving the band during pregnancy [32]. Most reported interventions during pregnancy involved adjustments of the band due to vomiting or risk of nutrient deficiencies [31, 33].

Pouch Dilation and Prolapse/Slip of the Band

Pouch dilation and band prolapse/slippage have been well described in pregnant women with about 1.2% incidence during pregnancy and 1.1% postpartum [32]. Symptoms tend to be nonspecific and can include abdominal pain, nausea and vomiting, and reflux. Initial evaluations usually include an abdominal X-ray but can also be performed with a fluoroscopic water-soluble contrast swallow in the nonpregnant patients. Figure 15.1 shows a normally located band.

If concerns for band prolapse, initial treatment involves desufflating the band. If symptoms persist, the surgeons can obtain an esophagogram or limited fluoroscopic water-soluble contrast swallow study. If the diagnosis is confirmed and symptoms do not resolve with desufflation, laparoscopic removal should be performed. Depending on the stage of pregnancy, the abdomen can be entered either via a Veress needle or open trocar placement. Following removal of the band, an air-leak test can be performed, which will avoid fluoroscopic testing.

Band Erosion

Although incidence in the pregnant population is not well known, band erosion in the general population is around 12% [34–36]. In the minimally symptomatic patient, removal of the band can wait. If symptomatic, the band should be removed. Although most bands can be removed laparoscopically, endoscopic removal in pregnant patients has been described [36, 37].

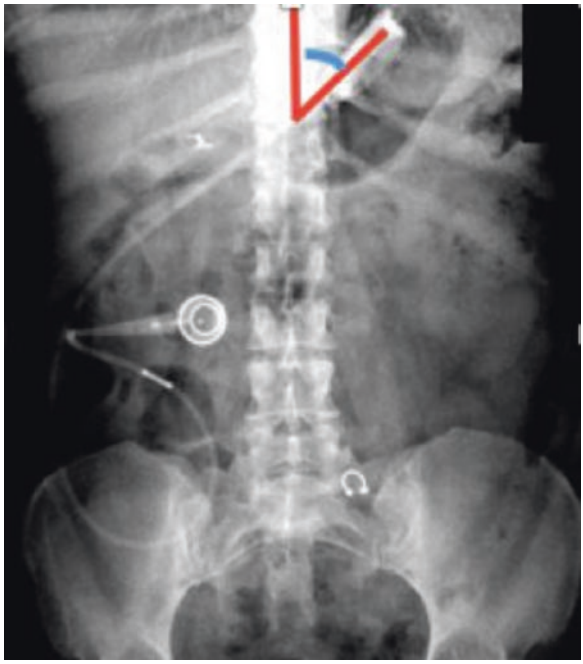

Fig. 15.1 Normal LAGB position

Complications Related to Roux-en-Y Gastric Bypass

Laparoscopic RYGB is the gold standard of bariatric procedures. Although the numbers of RYGB have steadily declined in the past several years, as the numbers of SG increase, there are still a substantial number of postoperative RYGB patients that may become pregnant. One of the reasons why SG is gaining popularity is the perceived lower long-term complication profile compared to RYGB. Common late complications are similar to those in nonpregnant patients and include small bowel obstruction, internal hernia, anastomotic strictures, marginal ulcer formation, fistula formation, and nutritional deficiencies.

Internal Hernia/Small Bowel Obstruction

Internal hernia (IH) is a well-known, serious complication and is the most common cause of small bowel obstruction (SBO) following RYGB. IH are likely due to the presence of potential mesenteric defects, although an internal hernia is possible due to an adhesive band. Either two or three potential mesenteric defects can be created, depending if the Roux limb is antecolic or retrocolic: transverse mesocolon, Petersen's space (between the Roux limb and the transverse mesocolon), and at the site of the jejunojejunostomy (JJ).

Table 15.1 Common findings concerning for internal hernia prebsent on CT scans

Swirled appearance of mesenteric fat or vessels
Mushroom shape of hernia
Tubular distal mesenteric fat surrounded by bowel loops
Small bowel obstruction
Clustered loops of small bowel
Small bowel other than duodenum posterior to the superior mesenteric artery
Right-sided location of the distal jejunal anastomosis

IH has an incidence of up to 16% in some series [38]. Since delay of diagnosis is associated with bowel necrosis and high mortality, patients presenting with abdominal pain and/or emesis must be urgently evaluated. However, pregnancy presents a challenge as these symptoms can be common. A literature review showed that internal hernia following RYGB presents in pregnant patients at a young age with most patients waiting at least 2 days prior to seeking help [39]. It is important that this patient population is aware of the morbidity and mortality of IH and the importance of consulting for abdominal pain. In addition, initial vital signs and laboratory studies can be normal in some cases; thus emergency department physicians and obstetricians should be aware of the potential of internal hernia.

We have previously created an algorithmic approach to expedite the diagnosis of internal hernia. Initial work-up includes laboratory studies, such as CBC with differential, chemistries, and lactic acid. Persistently elevated WBC, neutrophilia, and lactic acid despite fluid resuscitation are worrisome for intra-abdominal pathology and may require further investigation. If laboratory studies are normal or improve following fluid resuscitation, further work-up is dependent on the physical exam. If the patient presents with benign abdomen, further studies can be used. If patient presents with peritonitis, the emergent operative treatment should be planned.

In case of a benign abdomen, initial work-up can include a plain abdominal radiograph, which can provide some important findings, such as dilated bowel loops, paucity of intestinal air, or intraluminal air-fluid levels. If any of these are present, an emergent surgical exploration is needed. If no specific findings are seen on the plain abdominal radiograph, further studies are warranted. In the early pregnancy (first trimester), the patient can be either observed with serial abdominal exams or an MRI can be performed. In second or third trimester, computed tomography (CT) of the abdomen and pelvis can be performed. Several findings on CT scan can be worrisome for the presence of an internal hernia. These include small bowel loops in the upper quadrants, small bowel mesentery crossing the transverse mesocolon, twisting, swirling, crowding, and engorgement of the main mesenteric trunk [40–42]. Lockhart et al. examined the findings of 18 patients with surgically proven internal hernia and compared to 18 controls. The scans were reviewed by three radiologists for the findings of the findings based on Table 15.1. The authors concluded that the presence of a mesenteric swirl is the best indicator of an internal hernia [43].

A previous study performed by our group examined the sensitivity and specificity of CT scans in detection of internal hernia. Laboratory studies and CT scan findings were examined in 50 patients. The sensitivity and specificity of CT scans to detect an internal hernia were 76% and 60%, respectively. When we combined CT scan findings with the presence of neutrophilia, the sensitivity increased to 96% [44].

Marginal Ulceration

Marginal ulcer is a common complication following RYGB with a reported incidence of up to 16% [45–48]. While the presence of a marginal ulcer can be the cause of abdominal pain, dysphagia, nausea, and vomiting, it may lead to perforation, which is a surgical emergency as it can be the cause of morbidity and mortality to both mother and fetus. The incidence of marginal ulcer or perforation is not well documented in the pregnant population, but the incidence is about 1% per year in the general population [49].

Evaluation of the pregnant patient who presents with symptoms concerning for a marginal ulcer is performed with an endoscopy with *H. pylori* testing or biopsies if indicated. If diagnosis is confirmed, in most cases patients are treated conservatively. However, although the usual therapy in the general population is proton pump inhibitors (PPI) and cytoprotective agents, such as misoprostol, these are not recommended in pregnancy or for women who are breastfeeding. While some state that PPIs are safe in pregnancy [50], a meta-analysis, which examined 1530 pregnant women taking PPIs, reported an odds ratio of 1.12 (95% CI 0.84–1.45) for congenital malformations, without any significant difference in the odds ratios for spontaneous abortion or preterm delivery [51]. Safety of omeprazole, a common PPI used to treat marginal ulcers, has not been studied in this population. Alternatively, cimetidine or ranitidine (histamine-receptor antagonists) can be used.

In the case of perforation, surgery is mandatory in order to decrease both morbidity and mortality for mother and fetus. Fluid resuscitation and correction of electrolyte imbalance should be done prior to surgery. In the case of a duodenal perforation, Graham patch closure is the preferred treatment. In case of premature labor in the preterm patient, intramuscular steroid administration for fetal lung maturation should be considered [52].

Anastomotic Strictures and Leaks

While anastomotic strictures can present in pregnancy, anastomotic leaks are not common, as most occur early following surgery. Although not described in the literature, as it can lead to high morbidity and mortality, it should be considered in the pregnant bariatric patient. Leaks can be managed with surgery, stenting, or percutaneous drainage.

Anastomotic strictures can present during pregnancy, although true incidence is not known. In case of a stricture at the gastrojejunostomy (GJ) anastomosis, it can be managed by endoscopic dilation with a CRE balloon inflated to 18 mm. Multiple dilation procedures can be necessary and are not contraindicated during pregnancy. Persistent strictures may require conservative treatment during pregnancy and surgical intervention following delivery.

Complications Related to Sleeve Gastrectomy

Sleeve gastrectomy (SG) has gained popularity, as it is currently the most commonly performed procedure in the United States. Although it has a relatively low complication rate, complications can occur, such as reflux and strictures.

Gastroesophageal Reflux (GERD)

The presence of reflux following SG is a highly debated topic. Reflux is a common symptom in pregnancy, and prevalence increases with gestational age [53]. Reflux occurs in approximately 30–80% of pregnant women [54]. It is usually a de novo problem that arises during pregnancy and resolves with delivery.

The predominant mechanism of pregnancy-induced reflux is due to a decrease in the lower esophageal sphincter pressure caused by hormones during pregnancy, especially progesterone. Other contributing factors include an altered mucosal barrier, an increased intra-abdominal pressure, and impaired clearance of refluxate. Sleeve gastrectomy has also postulated to be refluxogenic [55, 56]. Several mechanisms have been proposed that lead to increased reflux after sleeve: dissection of the phrenoesophageal ligament and angle of His, intact or incompetent pylorus with narrowing of the gastric tube, intrathoracic sleeve migration, narrowing of the incisura, or fundus regrowth [57–60]. These factors confound to increase the incidence of reflux in pregnancy.

Initial treatment of reflux in pregnancy includes lifestyle and dietary modifications. Most women with mild symptoms do well following lifestyle modifications. If symptoms persist, first-line medications include antacids, either magnesium- or aluminum-containing products, or sucralfate. Sodium bicarbonate containing antacids can lead to metabolic alkalosis; thus it should be avoided. For persistent symptoms, histamine-receptor antagonists, preferably ranitidine, may be used, while PPIs are only reserved for women with intractable symptoms (discussed above). Nizatidine should be avoided during lactation [54]. The preferred PPI is lansoprazole. If symptoms are persistent, the endoscopy should be performed, but it should be delayed until the second trimester to avoid any effects from anesthetic agents [54]. Figure 15.2 shows a proposed algorithm for treatment of reflux of patients.

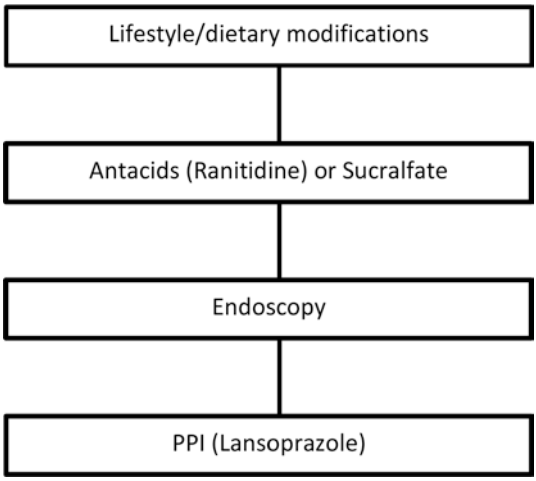

Fig. 15.2 Algorithm for management of reflux in the post-bariatric pregnant patient

Stricture

Strictures following SG can be caused by kinking or twisting, ischemia, or a leak. Strictures generally present with an inability to tolerate oral nutrition. Treatment of strictures in SG patients who are pregnant should involve a temporizing approach, such as endoscopic stenting or placement of supplemental feeding tubes. Definitive repair is delayed until postpartum or at a minimum the second trimester.

Conclusions

There are many considerations for bariatric surgical patients who become pregnant. A good understanding of nutrition and possible complications is important to safely manage these patients. There are certain complications that need to be considered when encountering these patients, as these can affect both mother and fetus. An integrated multidisciplinary approach is needed, including the obstetrician, primary care physician, nutritionist, and surgeon.

References

1. Vahratian A. Prevalence of overweight and obesity among women of childbearing age: results from the 2002 National Survey of Family Growth. Matern Child Health J. 2008;13(2):268–73.
2. Kakarla N, Dailey C, Marino T, Shikora S, Chelmow D. Pregnancy after gastric bypass surgery and internal hernia formation. Obstet Gynecol. 2005;105(5 Pt 2):1195–8.
3. Efthimiou E, Stein L, Court O, Christou N. Internal hernia after gastric bypass surgery during middle trimester pregnancy resulting in fetal loss: risk of internal hernia never ends. Surg Obes Relat Dis. 2009;5:378–80.

4. Moore KA, Ouyang DW, Whang EE. Maternal and fetal deaths after gastric bypass surgery for morbid obesity. New Engl J Med. 2004;351:721–2.
5. American College of Obstetricians and Gynecologists. ACOG practice bulletin no. 105: bariatric surgery and pregnancy. Obstet Gynecol. 2009;113(6):1405–13.
6. Wax JR. Risks and management of obesity in pregnancy: current controversies. Curr Opin Obstet Gynecol. 2009;21(2):117–23.
7. Ehrmann DA. Polycystic ovary syndrome. N Engl J Med. 2005;352:1223–36.
8. McCartney CR, Marshall JC. Polycystic ovary syndrome. N Engl J Med. 2016;375(14):1398–9.
9. Teitelman M, Grotegut CA, Williams NN, Lewis JD. The impact of bariatric surgery on menstrual patterns. Obes Surg. 2006;16:1457–63.
10. Deitel M, Stone E, Kassam HA, Wilk EJ, Sutherland DJ. Gynecologic-obstetric changes after loss of massive excess weight following bariatric surgery. J Am Coll Nutr. 1988;7(2):147–53.
11. Milone M, De Placido G, Musella M, et al. Incidence of successful pregnancy after weight loss interventions in infertile women: a systematic review and meta-analysis of the literature. Obes Surg. 2016;26(2):443–51.
12. Escobar-Morreale HF, Botella-Carretero JI, Alvarez-Blasco F, Sancho J, San Millán JL. The polycystic ovary syndrome associated with morbid obesity may resolve after weight loss induced by bariatric surgery. J Clin Endocrinol Metab. 2005;90(12):6364–9.
13. Eid GM, Cottam DR, Velcu LM, et al. Effective treatment of polycystic ovarian syndrome with Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2005;1(2):77–80.
14. Roehrig HR, Xanthakos SA, Sweeney J, et al. Pregnancy after gastric bypass surgery in adolescents. Obes Surg. 2007;17:873–7.
15. Merhi ZO. Weight loss by bariatric surgery and subsequent fertility. Gynecol Obstet Investig. 2007;64:100–2.
16. Mody SK, et al. Contraceptive counseling for women who undergo bariatric surgery. J Women's Health. 2011;20(12):1785–8.
17. Sheiner E, et al. Pregnancy after bariatric surgery is not associated with adverse perinatal outcomes. Am J Obstet Gynecol. 2004;190(5):1335–40.
18. Aricha-Tamir et al. Downsizing pregnancy complications: a study of paired pregnancy outcomes before and after bariatric surgery. Surg Obes Relat Dis. 2012;8(4):434–9.
19. Musella M, Milone M, Bellini M, et al. Effect of bariatric surgery on obesity-related infertility. Surg Obes Relat Dis. 2012;8(4):445–9.
20. Dao T, et al. Pregnancy outcomes after gastric-bypass surgery. Am J Surg. 2006;192(6):762–6.
21. Kjaer MM, Nilas L. Timing of pregnancy after gastric bypass- a national register-based cohort study. Obes Surg. 2013;23:1281–5.
22. Patel JA, Patel NA, Thomas RL, Nelms JK, Colella JJ. Pregnancy outcomes after laparoscopic Roux-en Y gastric bypass. Surg Obes Relat Dis. 2008;4(1):39–45.
23. Weiss HG, Nehoda H, Labeck B, et al. Pregnancies after adjustable gastric banding. Obes Surg. 2001;11(3):303–6.
24. Printen KJ, Scott D. Pregnancy following gastric bypass for the treatment of morbid obesity. Am Surg. 1982;48(8):363–5.
25. Centers of Disease Control and Prevention (CDC). U.S medical eligibility criteria for contraceptive use. MMWR Recomm Rep. 2010;59:1–86.
26. Maggard MA, Yermilov I, Li Z, et al. Pregnancy and fertility following bariatric surgery: a systematic review. JAMA. 2008;300:2286–96.
27. Jans G, Matthys C, Bogaerts A, et al. Maternal micronutrient deficiencies and related adverse neonatal outcomes after bariatric surgery: a systematic review. Adv Nutr. 2015;6:762–6.
28. Lesko J, Peacman A. Pregnancy outcomes in women after bariatric surgery compared with obese and morbidly obese controls. Obstet Gynecol. 2012;119:547–54.
29. Loar PV, Sachez-Ramos L, Kaunitz A, et al. Maternal death caused by midgut volvulus after bariatric surgery. Am J Obstet Gynecol. 2005;193:1748–9.
30. American College of Obstetricians and Gynecologists. ACOG Committee Opinion N0. 656: guidelines for diagnostic imaging during pregnancy and lactation. Obstet Gynecol. 2016;127(2):e75–80.

31. Carelli AM, Ren CJ, Youn HA, et al. Impact of laparoscopic adjustable gastric banding on pregnancy, maternal weight, and neonatal health. Obes Surg. 2011;21(10):1552–8.
32. Vrebosch L, Bel S, Vansant G, Guelinckx I, Devlieger R. Maternal and neonatal outcome after laparoscopic adjustable gastric banding: a systematic review. Obes Surg. 2012;22:1568–79.
33. Pilone V, Hasani A, Di Micco R, et al. Pregnancy after laparoscopic gastric banding: maternal and neonatal outcomes. Int J Surg. 2014;12:S136–9.
34. Miller K, Hell E. Laparoscopic adjustable gastric banding: a prospective 4-year follow-up study. Obes Surg. 1999;9:183–7.
35. Westling A, Bjurling K, Ohrvall M, Gustavsson S. Silicone adjustable gastric banding: disappointing results. Obes Surg. 1998;8:467–74.
36. Dogan UB, Akin MS, Yalaki S, Akova A, Yilmaz S. Endoscopic management of gastric band erosions: a 7-year series of 14 patients. Can J Surg. 2014;57(2):106–11.
37. Gochnour DC, Snyder BE, Wilson EB, Walker PA, Shah SK. Endoscopic removal of eroded adjustable gastric band in a pregnant patient. J Dig Endosc. 2015;6:126–9.
38. Higa K, Ho T, Tercero F, Yunus T, Boone KB. Laparoscopic Roux-en-Y gastric bypass: 10-year follow-up. Surg Obes Relat Dis. 2011;7:516–25.
39. Leal-Gonzalez R, De la Garza-Ramos R, Guajardo-Perez H, Ayala-Aguilera F, Rumbaul R. Internal hernias in pregnant women with history of gastric bypass surgery: case series and review of literature. Int J Surg Case Rep. 2012;4:44–7.
40. Scheirey CD, Scholz FJ, Shah PC, Brams DM, Wong BB, Pedrosa M. Radiology of the laparoscopic Roux-en-y gastric bypass procedure: conceptualization and precise interpretation of results. Radiographics. 2006;26:1355–71.
41. Hamdan K, Somers S, Chand M. Management of late postoperative complications of bariatric surgery. Br J Surg. 2011;98:1345–55.
42. Iannuccilli JD, Grand D, Murphy BL, Evangelista P, Roye GD, Mayo-Smith W. Sensitivity and specificity of eight CT signs in the preoperative diagnosis of internal mesenteric hernia following Roux-en-y gastric bypass surgery. Clin Radiol. 2009;64:373–80.
43. Lockhart ME, Tessler FN, Canon CL, Smith JK, Larrison MC, Fineberg NS, Roy BP, Clements RH. Internal hernia after gastric bypass: sensitivity and specificity of seven CT signs with surgical correlation and controls. AJR Am J Roentgenol. 2007;188(3):745–50.
44. Altieri MS, Pryor AD, Telem DA, Hall K, Brathwaite C, Zawin M. Algorithmic approach to utilization of CT scans for detection of internal hernia in the gastric bypass patient. Surg Obes Relat Dis. 2015;11(6):1207–11.
45. Jordan JH, Hocking MP, Rout WR, et al. Marginal ulcer following gastric bypass for morbid obesity. Am Surg. 1991;57:286–8.
46. Sapala JA, Wood MH, Sapala MA, Flake TM Jr. Marginal ulcer after gastric bypass: a prospective 3-year study of 173 patients. Obes Surg. 1998;8(5):505–16.
47. Dallal RM, Bailey LA. Ulcer disease after gastric bypass surgery. Surg Obes Relat Dis. 2006;2:455–9.
48. Gumbs AA, Duffy AJ, Bell R. Incidence and management of marginal ulceration after laparoscopic Roux-Y gastric bypass. Surg Obes Relat Dis. 2006;2(4):460–3.
49. Lublin M, McCoy M, Waldrep DJ. Perforating marginal ulcers after laparoscopic gastric bypass. Surg Endosc. 2006;20(1):51–4.
50. Nava-Ocampo AA, Velázquez-Armenta EY, Han JY, Koren G. Use of proton pump inhibitors during pregnancy and breastfeeding. Can Fam Physician. 2006;52:853–4.
51. Gill SK, O'Brien L, Einarson TR, Koren G. The safety of proton pump inhibitors (PPIs) in pregnancy: a meta-analysis. Am J Gastroenterol. 2009;104(6):1541–5.
52. Essilfie P, Hussain M, Bolaji I. Perforated duodenal ulcer in pregnancy-a rare cause of acute abdominal pain in pregnancy: a case report and literature review. Case Rep Obstet Gynecol. 2011;2011:263016.
53. Marrero JM, Goggin PM, de Caestecker JS, et al. Determinants of pregnancy heartburn. Brit J Obstetr Gynaecol. 1992;99:731–4.
54. Richter JE. Gastroesophageal reflux disease during pregnancy. Gastroenterol Clin N Am. 2003;32(1):235–61.

55. Santoro S. Technical aspects in sleeve gastrectomy. Obes Surg. 2007;17:1534–5.
56. Rawlins L, Rawlins MP, Brown CC, Schumacher DL. Sleeve gastrectomy: 5-year outcomes of a single institution. Surg Obes Relat Dis. 2013;9(1):21–5.
57. Dupree CE, Blair K, Steele SR, Martin MJ. Laparoscopic sleeve gastrectomy in patients with preexisting gastroesophageal reflux disease: a national analysis. JAMA. 2014;149(4):328–34.
58. Himpens J, Dapri G, Cadiere GB. A prospective randomized study between laparoscopic gastric banding and laparoscopic isolated sleeve gastrectomy: results after 1 and 3 years. Obes Surg. 2006;16:1450–6.
59. Braghetto I, Csendes A, Korn O, Valladares H, Gonzalez P, Henriquez A. Gastroesophageal relux disease after sleeve gastrectomy. Surg Laparosc Endosc Percutan Tech. 2010;20:148–53.
60. Burgerhart JS, Schotborgh C, Schoon EJ, et al. Effect of sleeve gastrectomy on gastroesophageal reflux. Obes Surg. 2014;24(9):1436–41.

Acute and Chronic Complications Following Biliopancreatic Diversion with Duodenal Switch

16

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Background

Although presently duodenal switch (DS) and recent modifications such as single anastomosis versions (SIPS or SADI) represent a minority of bariatric procedures, there is considerable increasing interest. This inquisitiveness is secondary to an increasing number of patients presenting with inadequate weight loss or weight regain following more widely used bariatric procedures [1, 2]. In addition, long-term outcomes for bariatric procedures such as Roux-en-Y gastric bypass (RYGB) or vertical sleeve gastrectomy (VSG) are suboptimal in the subclass of super morbidly obese or insulin-dependent diabetics. In comparison with RYGB, several randomized perspective and comparative case series have shown greater weight loss and resolution of comorbid metabolic derangements in DS patients [3, 4]. Recent matched cohort analysis studies have demonstrated that DS increases weight loss by 30% as compared to VSG [5]. Weight loss continues approximately 14–18 months as opposed to approximately 9 months following VSG [5]. Furthermore, conversion of VSG to DS or modified versions is an appealing option for many bariatric surgeons [6, 7]. As a result, it is our expectation that the prevalence of these procedures will grow rapidly and knowledge of potential complications will be imperative.

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Development of the DS and the Modified DS

Bariatric surgical procedures evolved following the awareness that patients that required gastrectomy or extensive small bowel resection lost weight. As a result, initial bariatric procedures focused on either making the stomach smaller or reducing intestinal length.

The duodenal switch is an operation that combines a longitudinal gastrectomy with an aggressive intestinal bypass. Most consider it a malabsorptive procedure; however the gastric resection certainly is responsible for a considerable amount of weight loss. Therefore, it is best to consider it as hybrid or gastric and intestinal procedure. The advantage of using both the stomach and intestine is higher average weight loss, a lower percentage of patients with weight loss failure, and a smaller rate or weight regain or recidivism. The disadvantage is that the greater proportion of bowel that is bypassed or not in contact with food, the higher the likelihood of consequences of having a shortened bowel. Besides frequent bowel movements, issues can include protein and vitamin deficiency, peri-rectal pathology such as abscess or fistula, and long-term issues such as bone loss and anemia.

The historical development of the DS traces back to the first malabsorptive procedure – the jejunoileal bypass (JIB). This was a procedure introduced in the 1950s which divided the jejunum about 10–15 in. from the ligament of Treitz and then anastomosed this segment to the ileum approximately 4 in. proximal to the ileocecal valve, in essence bypassing almost the entire small bowel [8].

In the classic Payne version of the JIB, the cut end of the proximal jejunum was anastomosed to the side of the terminal ileum [9]. However, with this short bowel length, there was severely poor absorption, with inability to absorb bile salts as well as irritation of the colon leading to water and protein loss. In addition, as in short bowel syndrome, bacterial overgrowth entered the portal system. There were ten deaths related to hepatic failure reported in Payne's series of 230 JIB as well as a high rate of protein deficiency and metabolic derangements [10]. In summary, this operation caused short bowel syndrome. For weight loss it was effective, but not conducive for proper nutrition and electrolyte balance. It was clear that longer intestine was required and that there had to be flow of at least digestive juices through the entire bowel. No segment could be a blind loop, meaning not have at least flow of digestive juices from the pancreas or liver.

In an attempt to minimize bowel length yet provide for adequate nutrition, fluid, and electrolyte status, Scopinaro introduced the biliopancreatic diversion (Scopinaro Procedure) in 1979. The operation combined a horizontal distal gastrectomy with a division of the small bowel 250 cm from the terminal ileum. At this point the bowel was attached to the stomach. The proximal staple line was reattached to the ileum 50 cm from the colon [11]. This created a 200 cm alimentary limb and 50 cm common channel. Bile salts could be absorbed through the entire biliopancreatic limb and common channel, so less damage would be inflicted on the colon. While this operation is very effective for weight loss, considerable issues remained. Malnutrition occurred and there was 2% reoperation rate for protein deficiency. Another major concern was the marginal ulcer rate of 8% [12].

Development of the Duodenal Switch

In 1998, Dr. Douglas Hess became interested in the research of Dr. Thomas DeMeester on duodenal gastric reflux and his concept of attaching a small segment of the duodenum to a Roux limb of small bowel. Hess wanted to utilize a similar approach to Scopinaro on revisions but encountered dense adhesions and following these challenging cases a high rate of marginal ulceration. As a result, Hess developed the concept of combining a vertical gastrectomy of the greater curvature with a duodenal division preserving a small cuff of the duodenum [13].

Hess calibrated his gastrectomy using a 40 Fr bougie or dilator and then dividing the stomach two finger breaths lateral to this leaving a larger stomach than is commonly done in VSG or modern DS. The length of small bowel was determined by measuring the total intestinal length from the stomach to the cecum. Then, 40% of the total length was used to create the alimentary limb and 10% of the total length to create the common channel [13].

Advantages of the duodenal switch include preservation of the pylorus and the reduction of marginal ulcer rates. In a 1998 report, Hess reported an 85% excess weight loss with 10 of 440 patients requiring revision for either protein malnutrition or diarrhea [13]. The duodenal switch has become the most common version of biliopancreatic diversion. Within North America, Dr. Gary Anthone [14] and Drs. Marceau and Biron [15] have published extensive series with lengthy follow-up. In contrast to Hess, they have used fixed bowel length rather than calculating based on total intestinal length.

No procedure is without complication, and no bariatric procedure is without side effects. Duodenal switch, although developed to minimize the complications and adverse effects of prior surgeries, is no exception. While the current methodology has reduced the incidence of short bowel syndrome, there still remain cases where many of the above issues can occur. In this chapter, our goal is to explain the differences between this procedure and other more prevalent bariatric procedures. The major alterations are the duodenal dissection and bypass of greater length of intestine. The latter results in overall greater weight loss than VSG or RYGB, at the cost of nutritional deficiencies and consequences of frequent bowel movements.

DS: Perception Versus Reality

A major reservation regarding DS is the impression that it is technically demanding and the complication profile is higher. However, it is important to note that the majority of the literature describing duodenal switch is not randomized. Often, this operation is chosen for patients with a higher degree of obesity and more preexisting conditions than other bariatric surgeries. In a cohort study comparing DS and gastric bypass, diabetes, hypertension, hyperlipidemia, GERD, congestive heart failure, sleep apnea, and poor functional status were all more prevalent in the DS ($p < 0.05$) [16]. As this is the case, some of the perioperative morbidity and post-op complications can be related to this more “high-risk” patient population. To account

for this, Dorman et al. matched RYGB and DS patients and found no difference in complication level [17]. Further potentiating this bias is that many insurance companies exclude DS coverage for patients with a BMI less than 50 [18]. This is in direct contradiction to published material that DS patients with BMI of 40–50 have excellent results [19]. Additionally, this policy suggests that larger patients have a greater reserve or are healthier to withstand the rigors of the more aggressive procedure. However, the converse is true: our largest patients are higher risk and do not have increased nutritional or physiologic reserve. What is true is that DS offers the best opportunity for super obese patients to not be morbidly obese 5 years after initial surgery.

In a meta-analysis that was performed to determine the mortality rates of bariatric procedures, they found that total mortality (for all bariatric procedures) at 30 days was 0.28% and total mortality at >30 days to 2 years was 0.24% [20]. Restrictive procedures showed a 30-day mortality of 0.30% when performed open and 0.07% when performed laparoscopically, whereas restrictive/malabsorptive procedures had a mortality of 0.41% when done open and 0.16% when performed laparoscopic [20]. Purely malabsorptive procedures had the highest rate of mortality at 0.76% for open and 1.11% for laparoscopic [20]. They included BPD/DS in this final category [20]. It is important to note, however, that in this study patients undergoing DS had the highest BMI average of all the surgeries [20]. In addition, this study did not include a comparison of comorbidities across groups [20]. In a series of 1000 DS patients, Biertho et al. published a mortality rate of 0.1% 1/1000, challenging some of the above misconceptions [21].

Technical Challenges

Duodenal switch is one of the longer and more challenging bariatric procedures. Therefore, one important consideration is that longer cases require longer anesthesia time which theoretically increases cardiopulmonary risk. However, as DS is a newer procedure, and as surgeons become more familiar with it, the risk factors associated with operative time could improve. In a large cohort, morbidly obese patients undergoing DS were compared with those undergoing gastric bypass [16]. DS was associated with longer operating times, mean of 113.8 min for GB and 191.2 min for DS ($p < 0.001$). However, only 50% of DS patients underwent laparoscopic approach in this study (leaving 50% open surgery) versus 92% laparoscopic for GB ($P < 0.001$) [16]. In addition, in this study, DS patients more frequently underwent concurrent cholecystectomy and liver biopsy. Solid organ injury occurred more frequently in DS than GB (0.5% vs. 0.2%, $p = 0.01$) [16]. Whether concurrent cholecystectomy is indicated remains controversial. The majority of laparoscopic surgeons performing the procedure are not routinely removing the gallbladder. Arguments for cholecystectomy include difficulty for ERCP access in the future (it requires an enteral approach and concurrent surgical procedure) and the estimated 10–30% risk of future stones. In contrast, most report a minimal incidence of future cholecystectomy and rarely are these cases complicated. Cardiac events occurred

0.1% of the time in DS, bleeding events occurred 0.2% of the time, and hollow viscous injury occurred 0.2% of the time [16].

The DS combines a VSG with an aggressive intestinal bypass. There are several aspects of the procedures that differ from primary VSG and RYGB. The longitudinal gastrectomy in DS should be larger than primary VSG. Marceau et al. advocated for a 60 French bougie [15]. Hess used a 40 Fr bougie but then moved two finger lengths laterally [13]. An issue with several frequent studies is that the bougie size used was as small as 32 Fr. It is essential to realize that the difference between a 36 or 42 bougie is 2 mm diameter. However, it is important to understand that the sleeve should be larger enough to allow adequate protein intake. It is estimated depending on the intestinal reconstructions that 1.5× the normal amount of protein is required. In SADI, Torres and Sanchez have suggested a 54 bougie [20]. In SIPS, Roslin and Cottam have utilized a 42 bougie and state the importance of not being overly aggressive [21]. There are several potential advantages of the larger bougie size. Data from primary VSG suggests a lower leak rate with larger bougie [22]. Additionally, with proper technique the risk of stricture should be nullified with this larger size. Theoretically, by having the intestinal conduit, a larger sleeve can be performed, and it is plausible that this can negate certain of the burdensome primary complications that can occur following VSG.

The second difference that is a source of trepidation is the transection of the duodenum. This creates a duodenal stump, and the dissection is near key blood vessels and the common bile duct. There are several techniques that have been utilized for the duodenal dissection. Many now continue the greater curvature dissection, taking the posterior adhesions, and then elevate the duodenum. Anterior to the gastroduodenal artery, the duodenum is transected. At no point should there be any dissection near or involving the pancreas. Accidental injury to the pancreas can lead to pancreatitis and or precipitate a duodenal stump leak. Should this occur, this becomes a most difficult management situation and requires drainage, avoiding feeding and potentially reoperative surgery. It is best to be prevented by dissecting in the proper plane.

Another intraoperative situation that potentially may be encountered is injury to the duodenum during dissection. Should this occur attempt to place a staple line distal to the injury and then try to utilize the opening as part of the duodenotomy for the eventual anastomosis.

Encircling the duodenum should be relatively simple, and fibrosis or adhesions should not be encountered posteriorly. If this is found, it can be an indication of peptic ulcer disease, and consideration should be given to aborting, leaving the patient with a VSG and endoscopy postoperatively.

Early Postoperative Complications

As with any bariatric procedure, there are risks of bleeding, leakage, and venous thromboembolism. The risk of bleeding is similar to any other major bariatric procedure. Leaks can occur from the VSG, the duodenal-enteral anastomosis, or in

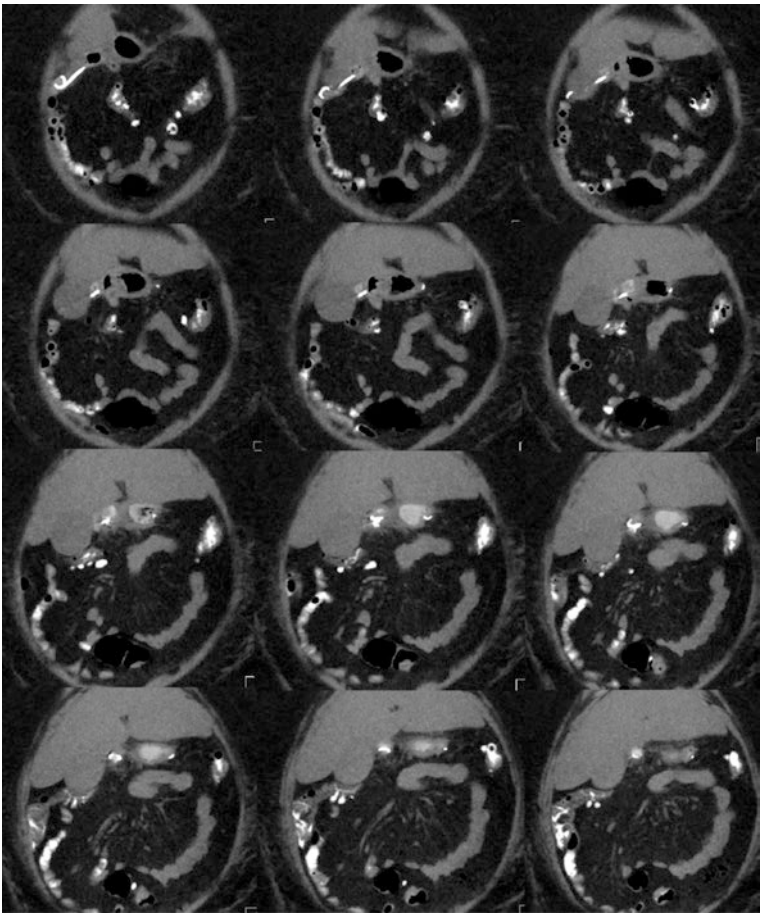

Fig. 16.1 CT scan showing leak and J stent

a Roux or classic DS the distal anastomosis. An advantage of the DS is the ability and suggestion to make a larger sleeve. As a result, many believe that the risk of leakage or stricture can be reduced (Fig. 16.1).

The risk of anastomotic leaks of the duodenal-enteral anastomosis is a real concern. As with any surgical procedure, proper technique can reduce the incidence. Two aspects appear critical – preserving an adequate blood supply to the duodenal cuff and sufficient mobilization so that there is no tension on the anastomosis. To accomplish, in our technique we lyse all posterior adhesions but do not ligate or use power source on any of the lesser curvature blood supply.

Early anastomotic leaks that occur within several days of surgery are probably best handled with reoperation. Additionally, there are important differences as to whether the leak occurred in a patient with a single anastomosis or Roux configuration. If an early leak occurs in a single anastomosis patient, the injured

area will be subjected to the biliopancreatic secretions. Additionally, there is not a logical place to leave a jejunostomy tube. As a result, in addition to repair of the leak, our suggestion would be to convert to a Roux or perform a Braun enteroenterostomy. An advantage to the post-pyloric reconstruction is, in the worst-case scenario, that duodenal cuff can be resected and gastric bypass performed.

Leaks that occur 7–10 days later are generally treated with percutaneous drainage, making sure that there is no distal obstruction. Patients with a Roux configuration are very amenable to the placement of a stent through the leaking anastomosis. Again, patients with a loop configuration represent a new challenge. We have been successful treating a POD 14 leak with an internally placed double pigtail, placing the pigtail through the leak. In general, if there is no distal obstruction, no duodenal stump leak, sepsis contained, and adequate nutrition provided, these leaks will heal without additional surgery. Presently, there are no dual limb covered stents; thus single anastomosis leaks may not be amenable to stenting.

Diarrhea and Malnutrition

A major concern for DS is the consequences of shorter bowel length causing diarrhea, perirectal complications, and protein and vitamin deficiency. Since revisions were common after the initial BPD, Topart and Becouarn did a literature review of revisions comparing the rate for DS and BPD. They found the rate of revision was 0.5–4.9% for DS and 3–18.5% for BPD [23]. The chief reason for reoperation was protein malnutrition, accounting for 43–60% of the reasons for reversal and revision in two of the main studies Topart and Becouarn reviewed [23]. Another reason, however less common at 30%, is for control of frequent diarrhea or flatus. Hamoui et al. reported a series of DS requiring and undergoing revision, and the most common indications for revision were malnutrition (20/33), diarrhea (9/33), metabolic abnormalities (5/33), abdominal pain (3/33), liver disease (2/33), and emesis (2/33) [24]. In this series of patients undergoing revision, complications occurred 15% of the time, with two wound infections, one respiratory failure, one GI bleed, and one SBO perioperatively [24]. The revision surgery was successful for those having the operation for diarrhea, with the median number of daily bowel movements being reduced from 5 to 1 [24]. Almost all patients had improvement in albumin levels to >3.5 g/dL except for one patient whose albumin increased from 2.5 to 2.8 g/dL [24]. Interestingly, although DS had resolved 10/11 patients with diabetes, 3/10 redeveloped diabetes after revision [24]. With revision surgery, the common channel is usually elongated, allowing for a longer segment of bowel in contact with food. Most DS revisions report a 100 cm common channel elongation, while Scopinaro recommended a 150 cm increased for BPD [12].

The advantage of the DS is that it offers the greatest weight loss. As a result, it is not surprising that in certain patients it overshoots the mark. There are several reasons that this can occur. Most people do not get evidence of short bowel syndrome if more than 2 m of bowel are in contact with food and the ileocolic valve is functional. Certain patients may be more sensitive. Sanchez et al. were able to

decrease the amount of revisions required in SADI by increasing the bowel length from 200 to 250 cm [25]. There can be individual differences, and certain individuals may require greater bowel length. Furthermore, the measurement of bowel is a very inexact science, and if there are frequent bowel movements with nutritional derangement, revision should be considered. Additionally, diet habits are essential. A purpose of the procedure is to interfere with fat absorption. Thus a diet high in fat is going to cause voluminous smelly and frequent bowel movements. Additionally, a diet high in simple carbohydrates and starch can cause bloating, diarrhea, and bacterial overgrowth.

Protein intake after surgery should be the primary nutrition goal for a number of reasons. During periods of rapid weight loss, the body will need to conserve lean body mass for an increased metabolism and ability to burn calories. High-quality protein sources increase satiety as well as aid in tissue healing. It is generally recommended that patients consume 80–100 g protein/day (1.0–1.5 g/kg/IBW). While this high quantity may be difficult in the first stage of the liquid phase, protein shakes and liquid supplements will likely be necessary to achieve this goal. Ideally, feedings should be distributed throughout the day.

In the analysis of cohorts of patients undergoing DS, Strain et al. found a rate of 7.3% for nutritional deficiency, 5.1% of which required TPN [26]. At 9 years, 30% of patients were protein deficient, with 20% of patients having low albumin levels [26].

What is interesting is that Lebel et al. in a study that compared DS with 200 cm common channel vs. 100 cm common channel discovered that the longer channel group had lower severe protein deficiency (11% vs. 19%). Additionally, fewer patients with the longer channel required vitamins A and D supplementation ($p < 0.05$). Patients also had fewer bowel movements (2.0 vs. 2.9, $p = 0.03$) [27]. This was with no significant difference in weight loss. This paper suggests that some of the main complications of the DS can be possibly reduced with lengthening the common channel while still keeping the weight loss robust.

It is our goal to keep bowel movements to two to four per day. Blood work is obtained at 3, 6, and 12 months. If there is diarrhea or nutritional issues, a complete dietary assessment is performed. The goal is to combine a protein source with a leafy fibrous vegetable and encourage the consumption of 90–100 g of protein/day while avoiding high saturated fats and simple carbohydrates. Hair loss may be common within first few months due to weight loss, but if carried in the long term, it is important to review zinc, vitamin A, and iron status. For frequent bowel movements, reducing liquid intake and making sure drinking and eating are separated can be helpful. If bowel movements are malodorous, we usually will prescribe bismuth subgallate. If bad breath is present and there is considerable abdominal bloating, small intestinal bacterial overgrowth (SIBO) should be considered. This can be confirmed by breath hydrogen and methane test and treated with antibiotic and probiotic supplements [28]. If not treated appropriately, long-term micronutrient deficiencies can arise due to dysbiosis in gut microflora which can interfere with iron and fat absorption.

Micronutrient Considerations

Optimizing postoperative patient outcomes and nutrition status begins within the preoperative process. Intensive preoperative nutritional counseling is crucial to gauge patients' motivation, predicted compliance, and ability to change habits. Patients should be educated before and after surgery on the expected nutrient type, dietary behaviors, and weight loss goals to support long-term outcomes. Invasive alterations to physiology, digestion, absorption, metabolism, and excretion are associated with higher nutrient deficiencies and should be reviewed with patients [29].

Laboratory markers are considered imperative for completing the initial nutrition assessment and continued in follow-up care. Baseline values help distinguish between postoperative complications, deficiencies related to surgery, and noncompliance with recommended supplementation. Any nutrient deficiencies identified presurgery should be repleted following the RDA in addition to any individualized recommendations.

Common deficiencies include the following.

Vit A

Vitamin A deficiency has been reported to be at 52% at 1 year and 69% at 4 years after DS [30]. Early symptoms of vit A deficiency are night blindness and changes in conjunctiva of the eyes. Treatment includes 10,000 IU po. Iron, zinc, and protein levels need to be corrected to normalize vit A levels. Additionally vit A deficiency has been found to be associated with low serum prealbumin.

Calcium and Vitamin D

Calcium and vitamin D are important for bone formation, blood coagulation, muscle contraction, and myocardial conduction. An acidic environment and adequate levels of D are needed for proper absorption of calcium and other minerals. Limited intake and/or decreased absorption of one or both can lead to osteopenia, osteoporosis, and/or osteomalacia. While calcium and vit D deficiencies have higher incidences after malabsorptive procedures, bone mineral depletion directly correlates with the amount of weight lost in an individual, regardless of the cause of weight loss [31].

Calcium citrate supplementation is preferred as it requires minimal acid for absorption, and a supplement including magnesium and vit D enhances absorption. DS patient requires higher calcium doses than other bariatric surgery patients, typically 1800–2400 mg divided into doses of 500 mg per dose. Vit D deficiency is prevalent even before weight loss surgery with reports of 16–57% [32]. Vit D supplementation may consist of up to 50,000 IU weekly for up to 9 weeks and 5000 IU daily thereafter [33]. Parathyroid (PTH) is the best indicator of calcium

status; when PTH increases, bone resorption of calcium increases in order to maintain normal blood levels of calcium. It is generally recommended that the PTH level be kept below 100 pg/ml to reduce the risk of metabolic bone disease [34]. Bone density should be tracked serially. Blood work should include a minimum of annual albumin, calcium, PTH, and 25-OHD levels to assess bone health. It is important to note that elevated PTH values are commonly found in patients who have had DS or RYGB even with normal vitamin D levels and no change on bone density scans. The significance is not yet known.

Folic Acid

Typically 100 mcg of folate is excreted in bile daily; most is resorbed in the upper third portion of the unaltered small intestine but may be absorbed throughout the entire small bowel [35]. Since much of the small bowel is bypassed, daily excretion of folate is greater, and deficiency may occur rapidly without adequate supplementation of minimum 400 mcg daily which can be found in multivitamins. Folate and vitamin B12 are codependent, and deficiency of either can contribute to macrocytic anemia.

Zinc

Zinc deficiency can be suspected with hair loss, poor wound healing, diarrhea, glossitis, dermatitis, and hypogeusia. Zinc deficiency may arise to due lack of absorption in the proximal jejunum, intolerance to zinc-rich foods such as meat, and fat malabsorption. Supplementing with elemental zinc of 30–50 mg daily or every other day may be suggested [36].

Iron

Iron deficiency anemia is the most common micronutrient deficiency after DS [12]. Iron absorption is compromised due to reduced stomach size and less exposure to hydrochloric acid. Furthermore, the principal sites of iron absorption (duodenum and proximal jejunum) are bypassed in the DS [37]. It is important to rule out other causes of anemia, such as deficiency of protein, vit b12, folate, selenium, zinc, and copper [38]. The 2016 ASMBS Nutritional Guidelines recommend 150–200 mg of elemental iron in the form of ferrous fumarate, sulfate, or gluconate for treatment in iron deficiency through repletion [33]. If oral supplementation is not effective, intravenous iron infusions containing ferric gluconate may be necessary (Table 16.1, Fig. 16.2).

The majority of patients that follow the advised diet and take supplements can have a high quality of life with minimal nutritional disturbances. However, even with the increased common channel in SIPS and SADI, there will be complaints of

Table 16.1 Recommended prophylactic treatment

Iron (ferrous gluconate, fumarate, or sulfate)	150–200 mg elemental iron compounded with vit C for better absorption
Folic acid	400 mcg
Vit A (retinol)	10,000 IU/day
Calcium (citrate)	1800–2400 mg with vit D separated in doses of 500 mg each
Vit D	Minimum 3000 IU/day
	Up to 50,000 IU weekly if severe deficiency
Zinc	15 mg/day

*Supplementation recommended by ASMBS Integrated Health Nutritional Guidelines for the Surgical Weight Loss Patient 2016 Update [33]

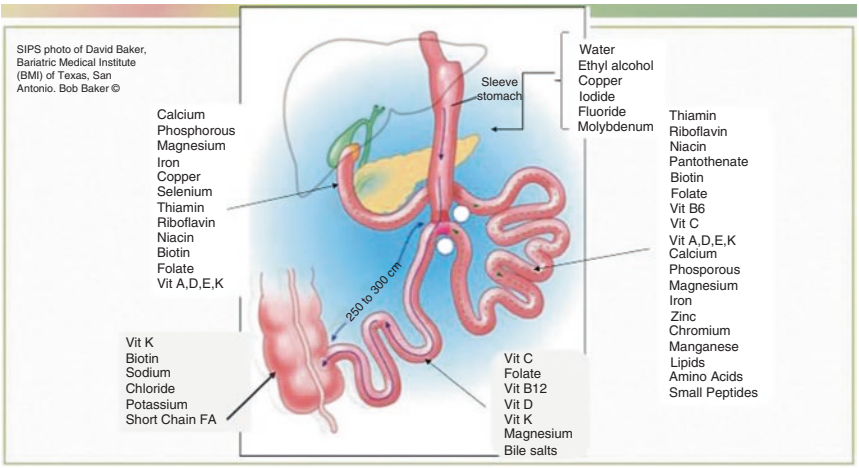

Fig. 16.2 Macronutrient and micronutrient absorptions in the GI tract

increased bowel movements. When encountered the initial steps would be objective nutritional assessment with lab work and detailed review of dietary intake. Empirical treatment with flagyl is reasonable for potential SIBO (small intestine bacterial overgrowth). For patients with persistent symptoms, lengthening of the bowel should be considered. Whereas most advocate for a course of TPN followed by increased counseling, we suggest earlier consideration for laparoscopic revision. In our experience this is an unusual event, and the issues causing are not likely to improve without aggressive treatment. Although after surgery the bowel will eventually hypertrophy, the risk of reconstruction is far lower than the risk of getting irreversible changes from chronic malnutrition. To place in perspective, in our practice the incidence of patients presenting with frequent bowel movements or malnutrition is a much lower incidence than the amount of patients that have inadequate weight loss or weight regain following sleeve gastrectomy. To handle bowel issues, the common channel is lengthened, and the biliopancreatic limb is reduced. Our recommendation for standard DS is to identify the distal anastomosis and measure 1.5 m

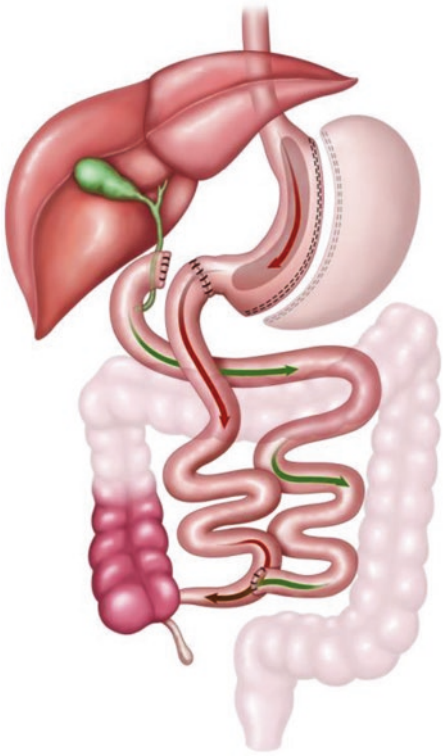

Fig. 16.3 Duodenal switch (Courtesy of Ethicon Endo-surgery Ltd.)

of the biliopancreatic limb (Fig. 16.3). This point is attached to the proximal Roux. There is no need to divide the alimentary limb. For those with a single anastomosis, there are two options. The anastomosis can be taken apart and the small bowel reclosed being careful to not narrow, and another single anastomosis is performed more proximally. Alternatively the small bowel can be divided proximal to the loop, and this point attached approximately 50 cm downstream. Then 1.5 m is measured from this new staple line, and that point is attached to the proximal Roux limb.

Emerging Trends

It is our expectation that modifications of the duodenal switch will become the fastest-growing procedures in bariatric surgery. Advantages of a post-pyloric construction seem to avoid marginal ulcer. Additionally, loop or single anastomosis procedures can potentially reduce the risk of small bowel obstruction. A major concern has been the risk of protein malnutrition and vitamin/micronutrient deficiency. Expanding common channel length can potentially reduce complications, and early results do not indicate a significant reduction in efficacy. As VSG becomes even more prevalent, there will be an increasing number of patients with recidivism and inadequate weight loss. Conversion to DS or similar procedure that expands the

biliopancreatic limb will be the only effective option. RYGB will be reserved for those with GERD or dysphagia following VSG. Additionally, we believe that the single anastomosis DS procedures have significant advantages over one anastomosis gastric bypass. The post-pyloric position and remnant resection reduce marginal ulcer rates. Resection of the remnant offers a greater likelihood of hunger suppression secondary to altered ghrelin level. As a result, knowledge of these procedures is imperative, and as surgeon become more familiar with the duodenal dissection and anastomoses, we expect they will be offered to an increasing number of patients.

References

1. Magro DO, Geloneze B, Delfini R, et al. Obes Surg. 2008;18:648. https://doi.org/10.1007/s11695-007-9265-1.
2. Daniel MF, Felix BL, et al. Surg Obes Relat Dis. 2016;12(Issue 9):1651–4.
3. Zaveri, et al. Springer Plus. 2016;5:1740. https://doi.org/10.1186/s40064-016-3392.
4. Rajan S, Mathew LM, et al. Comparative, effectiveness of primary bariatric operations in the United States. Surg Obes Relat Dis. https://doi.org/10.1016/j.soard.2017.01.021
5. Cottam A, Cottam D, Roslin M, et al. Obes Surg. 2016;26:2363. https://doi.org/10.1007/s11695-016-2133-0.
6. Keshishian A, Zahriya K, Hartoonian T, et al. Obes Surg. 2004;14:1187. https://doi.org/10.1381/0960892042387066.
7. Manish P, Alfons P, Michel G. Surg Obes Relat Dis. 2007;3:611–8. https://doi.org/10.1016/j.soard.2007.07.010.
8. Payne JH, DeWind LT. Surgical treatment of obesity. Am J Surg. 1969;118(2):141–7. https://doi.org/10.1016/0002-9610(69)90113-5.
9. Payn JH, et al. Metabolic observations in patients with jejunocolic shunts. Am J Surg. 1963;106:273–89.
10. DeWind LT, Payne JH. Intestinal bypass surgery for morbid obesity. Long-term results. JAMA. 1976;236(20):2298–301.
11. Scopinaro N, Gianetta E, Civalleri D, Bonalumi U, Bachi V. Bilio-pancreatic bypass for obesity: 1. An experimental study in dogs. Br J Surg. 1979;66(9):613–7.
12. Scopinaro N, Adami GF, Marinari GM, et al. Biliopancreatic diversion. World J Surg. 1998;22(9):936–46.
13. Hess DS, Hess DW. Biliopancreatic diversion with a duodenal switch. Obes Surg. 1998;8(3):267–82.
14. Anthone GJ, Lord RV, DeMeester TR, Crookes PF. The duodenal switch operation for the treatment of morbid obesity. Ann Surg. 2003;238(4):618–27.
15. Marceau P, Biron S, Hould FS, et al. Obes Surg. 2007;17:1421. https://doi.org/10.1007/s11695-008-9435-9.
16. Nelson DW, Blair KS, Martin MJ. Analysis of obesity-related outcomes and bariatric failure rates with the duodenal switch vs gastric bypass for morbid obesity. Arch Surg. 2012;147(9):847–54.
17. Dorman RB, Rasmus NF, al-Haddad BJ, et al. Benefits and complications of the duodenal switch/biliopancreatic diversion compared to the Roux-en-Y gastric bypass. Surgery. 2012;152(4):758–65.
18. Drummond MF, Sculpher MJ, Torrance GW, O'Brien BJ, Stoddart GL. Methods for the economic evaluation of health care programmes. 3rd ed. Oxford: Oxford University Press; 2005.
19. Rezvani M, Sucandy I, Klar A, Bonanni F, Antanavicius G. Is laparoscopic single-stage biliopancreatic diversion with duodenal switch safe in super morbidly obese patients? Surg Obes Relat Dis. 2014;10:427–30.

20. Andrés SP, Torres A, et al. Single anastomosis Duodeno–Ileal bypass with sleeve gastrectomy (SADI-S). One to three-year follow-up. Obes Surg. 2010;20(12):1720–6.
21. Mitzman B, Cottam D, Roslin M, et al. Obes Surg. 2016;26:2098. https://doi.org/10.1007/s11695-016-2077-4.
22. Yuval JB, Mintz Y, Cohen MJ, et al. Obes Surg. 2013;23:1685. https://doi.org/10.1007/s11695-013-1047-3.
23. Topart PA, Becouarn G. Revision and reversal after biliopancreatic diversion for excessive side effects or ineffective weight loss: a review of the current literature on indications and procedures. Surg Obes Relat Dis. 2015;11(4):965–72.
24. Hamoui N, Chock B, Anthone GJ, Crookes PF. Revision of the duodenal switch: indications, technique, and outcomes. J Am Coll Surg. 2007;204(4):603–8.
25. Sánchez-Pernaute A, et al. Proximal duodenal-ileal end-to-side bypass with sleeve gastrectomy: proposed technique. Obes Surg. 2007;17:1614–8.
26. Strain GW, Torghabeh MH, Gagner M, et al. Obes Surg. 2017;27:787. https://doi.org/10.1007/s11695-016-2371-1.
27. Lebel S, Dion G, Marceau S, Biron S, Robert M, Biertho L. Clinical outcomes of duodenal switch with a 200-cm common channel: a matched, controlled trial. Surg Obes Relat Dis. 2016;12(5):1014–20.
28. Bures J, Cyrany J, Kohoutova D, et al. Small intestinal bacterial overgrowth syndrome. World J Gastroenterol. 2010;16:2978–90. https://doi.org/10.3748/wjg.v16.i24.2978.
29. Cummings S, et al. Metabolic and bariatric surgery. JADA. 2015;146(10):767–72. https://doi.org/10.1016/j.adaj.2015.06.004
30. Slater GH, Ren CJ, Seigel N. Serum fat-soluble vitamin deficiency and abnormal calcium metabolism after malabsorptive bariatric surgery. J Gastrointest Surg. 2004;8(1):48–55.
31. Guney E, et al. Effect of weight loss on bone metabolism. Comparison of vertical banded gastroplasty and medical intervention. Obes Surg. 2003;13:383–8.
32. Toh SY, et al. Prevalence of nutrient deficiencied in bariatric patients. Nutrition. 2009;25(11–12):1150–6. https://doi.org/10.1016/j.nut.2009.03.012
33. Parrott J, et al. ASMBS integrated health nutritional guidelines for the surgical weight loss patient—2016 update: micronutrients. Surg Obes Relat Dis. https://doi.org/10.1016/j.soard.2016.12.01816.12.018
34. Hamoui N, Anthone G, Crookes P. Calcium metabolism in the morbidly obese. Obes Surg. 2004;14:9–12.
35. Zeigler O, et al. Medical follow up after bariatric surgery: nutritional and drug issues. General recommendations for the prevention and treatment of nutritional deficiencies. Diabetes Metab. 2009;35(6 Pt 2):544–57. https://doi.org/10.1016/S1262-3636(09)73464-0.
36. Bal BS, Finelli FC, Shope TR, Koch TR. Nutritional deficiencies after bariatric surgery. Nat Rev Endocrinol. 2012;8(9):544–56.
37. Herron DH, Roohipour R, et al. Gastrointest Endosc Clin N Am. 2011;21:213–38.
38. Mechanick JI, Youdim A, Jones DB, et al. Clinical practice guidelines for the perioperative nutritional, metabolic, and nonsurgical support of the bariatric surgery patient–2013 update: cosponsored by American Association of Clinical Endocrinologists, The Obesity Society, and American Society for Metabolic & Bariatric Surgery. Obesity (Silver Spring). 2013;21(suppl 1):S1–S27.

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