J Metab Bariatr Surg. 2026 Aug;15(2):59-76
https://doi.org/10.17476/jmbs.2026.15.2.59
pISSN 2287-2930·eISSN 2508-5956

Journal of Metabolic and Bariatric Surgery JMBS

Review Article

Check for updates

Early and Late Complications After Sleeve Gastrectomy and Roux-en-Y Gastric Bypass: A Narrative Review

Sa-Hong Kim, Seung Jong Oh

Department of Surgery, Inha University Hospital, Inha University College of Medicine, Incheon, Korea

OPEN ACCESS

Received: Jul 14, 2026
Revised: Aug 4, 2026
Accepted: Aug 5, 2026
Published online: Aug 18, 2026

Corresponding author:
Seung Jong Oh
Department of Surgery, Inha University Hospital, Inha University College of Medicine, 27 Inhang-ro, Jung-gu, Incheon 22332, Korea.
Tel: +82-32-890-1036
Fax: +82-32-890-3549
Email: md.sjoh@gmail.com

ORCID iDs
Sa-Hong Kim
https://orcid.org/0000-0003-0178-6570
Seung Jong Oh
https://orcid.org/0000-0002-4823-3917

Funding
No funding was received for this study.

Conflict of Interest
None of the authors have any conflict of interest.

ABSTRACT

This review aimed to provide an updated overview of early and late complications after metabolic and bariatric surgery (MBS), focusing on incidence, mechanisms, diagnosis, and management, particularly for sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB), the 2 most commonly performed procedures in Korea. We performed a narrative review, integrating evidence from recent meta-analyses, systematic reviews, large registry studies, and key guidelines or position statements, supplemented by representative Korean data and procedure-specific details. Early complications, including bleeding (SG 0.6–2.5%, RYGB 1–1.5%), leakage (SG ~1%, RYGB ~0.6%), and venous thromboembolism (0.3–2.4%), notably porto-mesenteric vein thrombosis (0.3–1.0%), remain uncommon yet potentially life-threatening. Advances in surgical technique, computed tomography-based evaluation, and thromboprophylaxis have reduced the incidence of early complications. Late complications are more prevalent and chronic, including gastroesophageal reflux disease (23–28%), sleeve stenosis or twist (0.5–3.5%), gastrojejunal anastomotic stricture (~6%), marginal ulcer (6–16%), internal hernia (0.2–9%), gallstone formation (up to 50%); nutritional deficiencies, including iron deficiency anemia (16–24%), vitamin B12, folate, vitamin D, and calcium deficiencies; and dumping syndrome (up to 40%) and post-bariatric hypoglycemia (~34%). MBS is a safe and effective treatment for severe obesity and its associated comorbidities. However, a procedure-specific understanding of complication patterns, risk factors, and stepwise management strategies is essential to minimize morbidity and to achieve long-term success through multidisciplinary care.

Keywords: Bariatric surgery; Metabolic surgery; Postoperative complications

INTRODUCTION

Morbid obesity is associated with more than 200 comorbidities, including obstructive sleep apnea, hypertension, type 2 diabetes mellitus, and metabolic syndrome, and several types of malignancies. A meta-analysis of 281 prospective studies reported that for every 5 kg/m^2 increase in body mass index (BMI), the risk of esophageal adenocarcinoma and thyroid cancer rose significantly in male individuals, whereas the risk of endometrial and renal cell carcinoma increased sharply in female individuals [1]. A review similarly reported that obesity is associated

Author Contributions
Conceptualization: Oh SJ; Investigation: Oh SJ, Kim SH; Supervision: Oh SJ; Visualization: Kim SH; Writing - original draft: Oh SJ, Kim SH; Writing - review & editing: Oh SJ.

with increased incidence across all 13 obesity-related cancers designated by the U.S. National Cancer Institute, including esophageal adenocarcinoma, gastric cardia cancer, colorectal cancer, hepatocellular carcinoma, and endometrial cancer, among others [2]. The prevalence of obesity in adults showed a continuously increasing trend, though stabilized over the last 3 years in Korea. In 2023, obesity affects 49.8% of men and 27.5% of women, with nearly 40% of adults and one-fifth of children and adolescents classified as obese or overweight [3].

The therapeutic landscape for obesity has expanded considerably in recent years. While older anti-obesity medications such as phentermine, topiramate, buprenorphine, and naltrexone yield modest weight reduction of 5–10%, newer pharmacological agents, including glucagon-like peptide-1 (GLP-1) receptor agonists and dual GLP-1 and glucose-dependent insulinotropic peptide (GIP) receptor agonists have shown promising efficacy with total body weight loss greater than 20% in patients with morbid obesity [4], though concerns regarding cost, adverse effects, treatment compliance, and long-term safety persist. Nevertheless, metabolic and bariatric surgery (MBS) remains the gold standard for the management of morbid obesity, achieving 30% to 40% total body weight loss with reliable resolution for obesity-related comorbidities such as type 2 diabetes, obstructive sleep apnea, metabolic syndrome, and pseudotumor cerebri.

In Korea, sleeve gastrectomy (SG) is the most commonly performed bariatric procedure, accounting for over 70% of all cases, followed by Roux-en-Y gastric bypass (RYGB) [5]. SG is a restrictive procedure that involves resecting approximately 80% of the stomach along the greater curvature [6] creating narrow, sleeve-shaped conduit that limits gastric volume and accelerates gastric emptying [7,8]. Resection of the greater curvature also reduces plasma ghrelin levels, a key hunger-mediating hormone, thereby suppressing appetite in most patients [9]. RYGB creates a small gastric pouch directly anastomosed to a jejunal Roux-en-Y limb, bypassing most of the stomach, duodenum, and a variable length of proximal small intestine, which alters the kinetics of digestive enzymes. Evidence suggested that a longer biliopancreatic limb leads to greater weight loss and superior glycemic control compared to a longer Roux limb [10].

Though generally safe when performed by experienced surgeons, complications after MBS can arise immediately or even years later after surgery. These complications range from subtle and gradually progressive conditions to acute and life-threatening manifestations requiring urgent intervention, encompassing nutritional deficiencies, gastroesophageal reflux disease (GERD), dumping syndrome, bleeding, anastomotic leakage or strictures, sleeve stenosis or twist, gastric and marginal ulcerations, gallstone formation, and venous thromboembolism (VTE). Timely recognition with systematic follow-up evaluation is essential to detect and manage these potential complications [11,12].

This review aimed to provide a comprehensive, procedure-specific overview of early and late complications following MBS, with particular focus on SG and RYGB in the context of Korean clinical practice.

EARLY COMPLICATIONS

1. Postoperative bleeding

Postoperative bleeding is the most common early complication of MBS, with reported incidences of 1% to 1.5% following RYGB and 0.6% to 2.5% following SG [13,14]. The reported incidence of post-SG bleeding ranges from 1% to 6% in Korean literature, with some studies

https://doi.org/10.17476/jmbs.2026.15.2.59

reporting rates as high as 15%. However, postoperative bleeding is often defined based on clinically significant events requiring blood transfusion or reoperation. In addition, the use of staple-line oversewing reinforcement may contribute to lower incidence of post-SG bleeding. Consequently, the clinically relevant incidence is often considered to be closer to 1% [15].

Bleeding after MBS may be either intraluminal or intra-abdominal. Intraluminal bleeding typically presents with hematemesis or melena, whereas intra-abdominal (extraluminal) bleeding often has a more nonspecific presentation and overt abdominal pain is not consistently or predominantly present; both may present with tachycardia, hypotension, and, in severe cases, hemodynamic instability. The need for endoscopic or surgical intervention depends largely on the bleeding site [16,17]. In hemodynamically unstable patients, the decision to perform surgical exploration should be considered. In contrast, conservative management, including fluid resuscitation, blood transfusion, antifibrinolytic agents, and correction of coagulopathy, may be appropriate in selected cases without clinical or radiologic evidence of active bleeding. Nevertheless, close monitoring is essential, as some studies have reported that delayed management for postoperative bleeding may be associated with subsequent late complications including leakage or intra-abdominal abscesses [18,19].

In selected patients with ongoing, secondary, or recurrent postoperative bleeding, radiologic intervention can provide a minimally invasive alternative or adjunct to reoperation. Pre-interventional contrast-enhanced computed tomography (CT) helps localize the bleeding focus and differentiate intraluminal from intra-abdominal hemorrhage. When active contrast extravasation suggesting arterial or pseudoaneurysm bleeding from splenic, gastroepiploic, or left gastric branches is identified, selective transcatheter angiographic embolization using coils, gelatin sponge, or other embolic materials achieves technical success rates above 90% even in hemodynamically unstable patients [20], when endoscopic or surgical control is expected to be difficult [21]. Therefore, management strategy for post-MBS bleeding should be multidisciplinary, integrating surgical, endoscopic, and interventional measures according to the location and timing of bleeding and the patient's hemodynamic status (**Table 1**).

Table 1. Overview of early and late complications following MBS

Category	Incidence	Symptoms	Diagnosis	Key management
Early complications				
Postoperative bleeding	• SG: 0.6–2.5% • RYGB: 1–1.5%	• Intraluminal: hematemesis or melena • Intra-abdominal: nonspecific presentation, abdominal pain not consistently present • Hemodynamic instability in severe cases; tachycardia, hypotension, etc.	• Endoscopy • Contrast-enhanced CT • Diagnostic angiography	• Conservative care for stable patients: fluid resuscitation, blood transfusion, antifibrinolytic agents, and correction of coagulopathy • Endoscopic hemostasis • Transcatheter angiographic embolization • Surgery
Leakage	• SG: ~1% • RYGB: ~0.6%	• Tachycardia, fever, abdominal pain, shoulder pain, nausea, vomiting • Hemodynamic instability in severe cases; tachycardia, hypotension, etc.	• CT with oral contrast • UGIS with oral contrast	• Conservative care for stable patients: broad-spectrum antibiotics, high dose PPIs, nutritional support • Fluoroscopy-guided percutaneous drainage • Endoscopic procedures: SEMS, OTSC, EID, EVT • Surgery
VTE	• Overall: 0.3–2.4% • DVT: 0.1–0.3% (large registries); ~2% (earlier studies with routine Doppler screening) • PE: 0.1–0.12% • PMVT: 0.3–1.0%	• DVT: limb swelling, pain, warmth, erythema • PE: dyspnea, pleuritic chest pain, tachycardia, hypoxia, shock • PMVT: abdominal pain, nausea, vomiting, diarrhea, bowel ischemia	• Duplex ultrasonography for DVT • Contrast-enhanced CT (abdomen, pelvis, pulmonary angiography, etc.)	• Prevention: the combination of mechanical and pharmacologic prophylaxis, with extended postoperative prophylaxis using rivaroxaban (10 mg once daily) for 7–10 days • Anticoagulation with LMWH or UFH followed by transition to direct oral anticoagulation for 3–6 months • Systemic thrombolysis for high-risk PE

(continued to the next page)

Table 1. (Continued) Overview of early and late complications following MBS

Category	Incidence	Symptoms	Diagnosis	Key management
Late complications				
GERD	• Worsening of pre-existing GERD: 19% • De novo GERD: 23–28% after SG	• Heartburn, regurgitation, epigastric burning • Atypical symptoms including chest discomfort, chronic cough, dysphagia	• Endoscopy • Ambulatory pH monitoring • Esophageal manometry	• PPI • Lifestyle modification • Dietary adjustment • Surgery: conversion of SG to RYGB for refractory cases
Sleeve stenosis and twist	0.5–3.5%	• Nausea, vomiting, food intolerance or dysphagia, particularly for solid food	• UGIS with oral contrast • Endoscopy	• Endoscopic balloon dilation or stenting • Surgery: conversion of SG to RYGB for refractory cases
Gastrojejunal anastomotic stricture	~6%	• Progressive dysphagia and vomiting, often without abdominal pain	• UGIS with oral contrast • Endoscopy	• Endoscopic balloon dilation • Surgery: revision for refractory cases
Marginal ulcer	6–16% after RYGB	• Usually presents months to years after RYGB • Epigastric or retrosternal pain aggravated by food, nausea, vomiting, upper gastrointestinal bleeding, perforation	• Endoscopy • Contrast-enhanced CT	• High-dose PPI • Sucralfate • Eradication of *H. pylori*, when indicated • Endoscopic procedures: oversewing, stenting • Surgery: revision of gastrojejunostomy of RYGB, reversal of RYGB
Internal hernia	0.2–9% after RYGB	• Recurrent crampy postprandial abdominal pain, nausea, vomiting	• Contrast-enhanced CT • Early diagnostic laparoscopy	• Surgery (low threshold for diagnostic laparoscopy)
Gallstone formation	10–50% after MBS	• Often asymptomatic; biliary colic, acute cholecystitis, choledocholithiasis, or gallstone pancreatitis in symptomatic cases	• Abdominal ultrasonography (first-line) • CT or MRCP as an adjunct for suspected choledocholithiasis or gallstone pancreatitis	• Prophylactic ursodeoxycholic acid • Cholecystectomy for symptomatic disease (routine prophylactic cholecystectomy not recommended)
Nutritional deficiencies	Iron deficiency anemia: 16–24% after RYGB	• Fatigue, reduced exercise tolerance, exertional dyspnea, palpitations, dizziness; pallor, brittle nails, hair loss, restless legs syndrome	• Laboratory evaluation (CBC, ferritin, transferrin saturation, serum iron, TIBC)	• Oral iron supplementation • IV iron supplementation for severe or refractory cases
	• Vitamin B12: ~24% • Folate: 1.9%	• Vitamin B12: glossitis, peripheral neuropathy, megaloblastic anemia • Folate: more often asymptomatic	• Serum vitamin B12, folate	• Oral multivitamin supplementation • IV vitamin B12 supplementation for symptomatic deficiency
	• Vitamin D: 35–54% • Calcium: ~2%	• Often asymptomatic • Bone pain, muscle weakness, or fragility fracture (secondary hyperparathyroidism)	• Serum calcium, 25-hydroxyvitamin D, PTH	• Oral elemental calcium (calcium citrate) • Oral vitamin D
Dumping syndrome and PBH	• Early dumping: ~40% • Late dumping: ~25% • PBH: ~34%	• Early: vasomotor (fatigue, flushing, palpitations, hypotension) and gastrointestinal symptoms (increased bowel sounds, diarrhea, bloating, nausea, abdominal cramping), within 1 hour after meals • Late: transient autonomic (adrenergic) symptoms (sweating, palpitations, tachycardia, tremors, hunger, anxiety), within 1–3 hours after meals • PBH: neuroglycopenic symptoms (confusion, impaired consciousness, seizure) due to postprandial hypoglycemia, typically presenting more than 1 year after MBS	• Dumping syndrome (early/late): clinical symptom profile (timing and pattern), Sigstad score, modified oral glucose tolerance test • PBH: Whipple's triad with fasting hypoglycemia, exclusion of hypoglycemia-inducing drugs use; capillary blood glucose confirmation; continuous glucose monitoring as an adjunct	• Dietary modification: small, frequent, protein-rich meals, restriction of simple carbohydrates • Pharmacologic therapy: acarbose, diazoxide, or GLP-1 receptor antagonists • Revisional surgery for refractory cases

MBS = metabolic and bariatric surgery, SG = sleeve gastrectomy, RYGB = Roux-en-Y gastric bypass, CT = computed tomography, UGIS = upper gastrointestinal series, PPI = proton pump inhibitor, SEMS = self-expandable metal stent, OTSC = over-the-scope clip, EID = endoscopic internal drainage, EVT = endoluminal vacuum therapy, VTE = venous thromboembolism, DVT = deep vein thrombosis, PE = pulmonary embolism, PMVT = porto-mesenteric vein thrombosis, LMWH = low-molecular-weight heparin, UFH = unfractionated heparin, GERD = gastroesophageal reflux disease, MRCP = magnetic resonance cholangiopancreatography, CBC = complete blood count, TIBC = total iron-binding capacity, IV = intravenous, PTH = parathyroid hormone, PBH = post-bariatric hypoglycemia, GLP-1 = glucagon-like peptide-1.

https://doi.org/10.17476/jmbs.2026.15.2.59

2. Leakage

The reported incidence of leakage is approximately 1% after SG and 0.6% after RYGB [22,23]. The higher leakage rate after SG compared to RYGB may be attributable to the long staple line and increased intraluminal pressure. In addition, from a technical aspect, inappropriate selection of staple height, excessive traction during stapling, and extensive perigastric dissection causing tissue ischemia have been implicated in postoperative leakage [24].

In RYGB, leakage most commonly occurs at the gastrojejunostomy, where technical aspects of anastomotic construction are considered the major determinants of leakage development. Anastomotic integrity is influenced by factors such as closure technique, tissue tension, and perfusion [25,26], variations in pouch size and anastomotic configuration have been discussed as additional contributing factors [27].

Additionally, a variety of patient-related risk factors have been implicated in postoperative leakage, including diabetes, chronic kidney disease, hypoalbuminemia, chronic steroid use, and smoking [28], most of which are well-established risk factors for general surgical procedures.

Clinical manifestations of leakage after bariatric surgery are often nonspecific and may include tachycardia, fever, abdominal pain, shoulder pain, nausea, vomiting, or respiratory symptoms such as tachypnea or hypoxia. In more advanced cases, signs of sepsis, including tachycardia, hypotension and altered mental status, may develop. Sustained tachycardia is the initial clinical warning sign observed in the majority of cases with early leakage [29,30]. The clinical presentation may be subtle and delayed, as obesity itself can mask early signs. Therefore, a high index of suspicion is essential when evaluating postoperative patients with unexplained tachycardia or systemic symptoms.

When leakage is suspected, prompt and systematic evaluation is required. Assessment should include the onset timing (early or late), anatomical location, which may differ according to the surgical procedure (e.g., along the staple line of gastric sleeve or anastomosis of RYGB), and clinical severity, including the presence of hemodynamic instability or organ failure [30,31].

Upper gastrointestinal series (UGIS) with oral contrast has a sensitivity of approximately 50% for detecting postoperative leakage, whereas abdominal CT with oral contrast has been reported to achieve a sensitivity exceeding 90% [32]. In this context, in patients with clinically suspected leakage after bariatric surgery, abdominal CT with oral contrast is generally favored over conventional UGIS as the initial diagnostic modality [33]. Beyond leakage detection, CT enables comprehensive assessment of associated intra-abdominal complications, including abscess formation and bowel obstruction, and offers the practical advantage of immediate and continuous availability in urgent clinical settings [33,34].

Management of postoperative leakage is determined by clinical severity and may range from conservative measures to radiologic, endoscopic, or surgical interventions. Conservative management may be appropriate in selected, clinically stable patients, and typically includes broad-spectrum antibiotics, high dose proton pump inhibitors (PPIs), nutritional support, and close monitoring [35,36]. Radiologic intervention may include fluoroscopy-guided percutaneous drainage, which plays a key role in the management of localized intra-abdominal fluid collections or abscesses from leakage site and often serves as an adjunct to conservative treatment [30]. Endoscopic interventions have become a cornerstone of

https://doi.org/10.17476/jmbs.2026.15.2.59

therapy for leakage, aiming to control sepsis, divert luminal contents, facilitate drainage, and promote closure of the fistulous tract. Fully covered self-expandable metal stents can divert luminal contents away from the leakage site and promote healing, with reported endoscopic success in approximately two-thirds of cases. They are particularly useful for early proximal staple-line leakage, although stent migration and patient discomfort remain important limitations [37]. Over-the-scope clips are suitable for small, well-circumscribed defects without a large associated cavity, especially in cases of early leakage, whereas larger or chronic defects with associated perigastric fluid collections are generally better managed with endoscopic internal drainage (EID) [38,39]. EID using double-pigtail plastic stents provides continuous internal drainage of fluid collection and promotes secondary cavity obliteration, achieving high clinical success, good tolerability, early resumption of oral intake, and lower complication rates than stent-only approaches [40]. More recently, endoluminal vacuum therapy has emerged as an effective option for complex or refractory leakage [41,42].

Surgical intervention is indicated in hemodynamically unstable, those with diffuse peritonitis, or after failure of nonoperative management. In those acute settings, the primary goal is effective source control through irrigation, debridement, and adequate drainage rather than definitive repair of leakage site. Primary repair may be attempted in selected patients with early leakage occurring within 3 days postoperatively [29], whereas persistent or recurrent leakage after SG may require conversion to RYGB or fistula-to-jejunostomy formation. As a preventive measure, staple-line reinforcement remains inconsistent in its reported efficacy. Oversewing has been shown to reduce leak rates [43,44] but the degree of benefit appears to vary by reinforcement method [44] and other studies have failed to demonstrate a significant reduction in leakage rates with reinforcement [45,46].

Because no single modality is universally effective, treatment should be individualized according to leakage timing, size, location, associated cavity, and the patient's clinical condition, often combining endoscopic techniques with radiologic or surgical approaches within a multidisciplinary strategy. Despite the complexity of management, patients who successfully recover from leakage generally achieve long-term weight loss and comorbidity resolution comparable to those without leakage-related complications (**Table 1**) [47].

3. VTE

VTE is an uncommon but potentially serious complication that can occur after bariatric surgery, with a reported incidence ranging from approximately 0.3% to 2.4% [48]. Contemporary series and registry data indicate that the majority of VTE events occur after discharge, with approximately 70–80% of cases diagnosed during the post-discharge period [49]. VTE may be presented as deep vein thrombosis (DVT), pulmonary embolism (PE), or porto-mesenteric vein thrombosis (PMVT). Despite its relatively low incidence, VTE is associated with substantial morbidity and mortality.

Obesity has been demonstrated to be associated with venous stasis, hypertension, and obstructive sleep apnea, which are recognized as risk factors for thrombotic complications. Established patient-related risk factors include high BMI, older age, current smoking, and exposure to exogenous hormones such as oral contraceptives or hormone replacement therapy, all of which have been consistently associated with increased risk of postoperative VTE [50]. Procedure-related factors include prolonged operative time, procedure type (e.g., RYGB vs. SG), and postoperative complications (e.g., infection, anastomotic leakage, or prolonged immobilization), which may further increase VTE risk [48-50].

https://doi.org/10.17476/jmbs.2026.15.2.59

A range of preventive strategies has been proposed to reduce the risk of VTE after MBS, including risk assessment, pulmonary toileting, early ambulation, minimization of opioid use, pharmacologic prophylaxis with unfractionated heparin (UFH) or low-molecular-weight heparin (LMWH), and mechanical measures such as compression devices or elastic stockings. Systematic reviews have evaluated several aspects of thromboprophylaxis in MBS, including comparisons of higher-dose versus standard-dose heparin (UFH or LMWH), heparin versus pentasaccharides (e.g., fondaparinux), and timing of initiation, specifically preoperative versus postoperative administration of pharmacologic prophylaxis, preoperative versus postoperative initiation of pharmacologic prophylaxis, and combined mechanical and pharmacologic prophylaxis versus mechanical prophylaxis alone. Among the evaluated strategies, the combination of mechanical and pharmacologic prophylaxis is the only approach that has been associated with a reduction in VTE compared with mechanical prophylaxis alone [51]. Meanwhile, an extended pharmacologic thromboprophylaxis has been investigated by a randomized clinical trial, demonstrating that postoperative rivaroxaban (10 mg once daily) for 7 days is safe and effective for VTE prevention after bariatric surgery [52,53].

The cornerstone of treatment for postoperative VTE is prompt initiation of therapeutic anticoagulation. Initial treatment generally consists of LMWH or intravenous UFH, followed by transition to direct oral anticoagulants (e.g., rivaroxaban or apixaban) or vitamin K antagonists when clinically appropriate [54,55]. Anticoagulation is typically continued for at least 3–6 months, with treatment duration individualized according to thrombus location [56,57], provoking factors, bleeding risk, and the risk of recurrence [48-50].

DVT is one of manifestations of postoperative VTE after bariatric surgery. The incidence of DVT after laparoscopic bariatric surgery is approximately 0.1% to 0.3% in large multicenter registries [58,59], though earlier single-center studies using routine postoperative Doppler ultrasound screening reported higher rates around 2% [60]. Patients typically present with unilateral lower extremity swelling, pain, warmth, or erythema, although asymptomatic cases may occur. Diagnosis is established using duplex ultrasonography as the first-line modality. In cases with inconclusive findings or suspected proximal or pelvic vein thrombosis, additional imaging such as CT or magnetic resonance venography may be considered. Management generally follows the aforementioned principles of anticoagulation.

PE is less common than DVT but represents the most severe manifestation of VTE, accounting for the majority of VTE-related mortality. The incidence of 30-day postoperative PE is approximately 0.1% for SG and 0.12% for RYGB. Clinical presentations include dyspnea, pleuritic chest pain, tachycardia, hypoxia, and, in severe cases, hemodynamic instability or shock. CT pulmonary angiography is the diagnostic modality of choice. In addition to the general anticoagulation principles described above, systemic thrombolysis should be considered in patients with high-risk PE presenting with sustained hypotension or shock.

PMVT is another rare but life-threatening complication after bariatric surgery. The incidence was reported as 0.3% to 1.0% [61-64]. It occurs more frequently after SG compared to other bariatric procedures, and is typically presented within the first month after surgery [15]. Clinical symptoms are often nonspecific, including vague abdominal pain, nausea, vomiting, and diarrhea, but may progress to peritonitis or shock due to bowel ischemia. Diagnosis is most reliably confirmed using contrast-enhanced abdominal CT, which can demonstrate portal and mesenteric venous thrombosis and associated bowel ischemia [65]. Although therapeutic anticoagulation remains the primary treatment, patients who develop bowel

https://doi.org/10.17476/jmbs.2026.15.2.59

infarction, perforation, or generalized peritonitis require urgent surgical exploration with resection of nonviable bowel when indicated (**Table 1**) [54].

LATE COMPLICATIONS

1. GERD

GERD is one of the most common long-term complications after SG. SG can induce GERD by disrupting the native anti-reflux barrier and creating an unfavorable pressure environment at the gastroesophageal (GE) junction. Resection of the fundus straightens the angle of His, divides sling fibers around GE junction, and may reduce lower esophageal sphincter (LES) pressure, while hiatal dissection can contribute to hiatal hernia formation. In addition, the narrow, low-compliance gastric sleeve increases intragastric pressure, which raises the pressure gradient across the LES and promotes reflux. Technical factors, including stenosis at the incisura angularis (a mid-gastric narrowing), gastric sleeve twisting, proximal sleeve dilation resulting in a neo-fundus, may create localized high-pressure zones and impair gastric emptying, thereby leading to prolonged esophageal acid exposure, particularly in patients with impaired esophageal motility. Although weight loss, reduced acid production, and accelerated gastric emptying are generally observed after SG and may improve symptoms in some patients, structural alterations often predispose to the development or worsening of GERD after SG [66,67].

A systematic review and meta-analysis reported worsening of pre-existing GERD in 19% of patients and de novo GERD in 23% after laparoscopic SG, while endoscopically diagnosed esophagitis was identified in 28% of patients, exceeding symptom-based estimates [68]. Another meta-analysis including patients with at least 4 years of follow-up demonstrated persistent reflux-related morbidity, with pooled incidences of 24.8% for de novo GERD, 27.9% for esophagitis, and 6.7% for Barrett's esophagus [69]. Collectively, these findings indicate that a substantial proportion of patients experience deterioration of pre-existing GERD or develop de novo reflux after SG.

Clinically, GERD after SG presents with typical reflux symptoms, including heartburn, regurgitation, and epigastric burning, as well as atypical symptoms such as chest discomfort, chronic cough, and dysphagia [66,67].

Evaluation is primarily based on gastroscopy to assess esophagitis, Barrett's esophagus, and anatomical abnormalities such as sleeve stenosis, proximal neo-fundus, or hiatal hernia. In selected patients, additional functional testing including ambulatory pH monitoring or esophageal manometry may be considered for further characterization of reflux and esophageal motility disorders [66,70].

First-line treatment consists of medical anti-reflux therapy, including PPIs, lifestyle modification, and dietary adjustment. In patients with persistent or refractory symptoms despite optimal medical therapy and without correctable structural abnormalities, conversion to RYGB is an effective surgical option (**Table 1**) [66,70].

2. Sleeve stenosis and twist

Sleeve stenosis is reported in approximately 0.5% to 3.5% of patients after SG, though population-based registry data report a lower incidence of 0.6% [15,71]. It most commonly

https://doi.org/10.17476/jmbs.2026.15.2.59

occurs at the incisura angularis and results from either mechanical narrowing, typically from stapling too close to the incisura over a narrow calibration bougie, or axial obstruction resulting from sleeve twist caused by uneven alignment of the anterior and posterior gastric walls during stapling; longer operative time and staple-line oversewing have been identified as additional risk factors. Patients present with nausea, vomiting, and food intolerance or dysphagia, particularly for solid food. Diagnosis relies on UGIS demonstrating abrupt luminal narrowing, supported by endoscopy to distinguish true stenosis from functional twisting. Endoscopic balloon dilation or stenting can be therapeutic options, while surgical conversion to RYGB is reserved for patients who are refractory to endoscopic treatment (**Table 1**) [72].

3. Gastrojejunal (GJ) anastomotic stricture

GJ anastomotic stricture after RYGB has a pooled incidence of approximately 6% [73], and its risk appears to be influenced by the size of the circular stapler used, with some comparisons favoring linear over circular stapling techniques [74].

Stricture formation has been attributed to a combination of localized ischemia, suboptimal anastomotic technique, and scar contracture unrelated to ischemia [75]. Patients typically present within weeks to months postoperatively with progressive worsening dysphagia and vomiting, often without abdominal pain. The diagnosis is usually confirmed by UGIS or endoscopy. Endoscopic balloon dilation is safe and highly effective, and surgical revision is generally reserved for refractory strictures (**Table 1**) [73].

4. Marginal ulcer (MU)

MU is one of the most common late complications after RYGB, with a reported incidence ranging from approximately 6–16% [76,77]. Risk factors include patient-related factors, such as smoking, nonsteroidal anti-inflammatory drugs (NSAIDs) use, diabetes, immunosuppression, alcohol consumption, and *Helicobacter pylori* infection, although the contribution of *H. pylori* remains controversial because of inconsistent findings across studies [78,79]. Technical factors include a large or ischemic gastric pouch, tension at the gastrojejunostomy, retained foreign bodies (e.g., nonabsorbable sutures, staples), and the presence of a gastro-gastric fistula (an abnormal communication between the gastric pouch and the excluded stomach) [80].

Clinically, MU usually presents months to years after RYGB with epigastric or retrosternal pain aggravated by food intake, nausea, vomiting, and reduced oral intake, but may also manifest as upper gastrointestinal bleeding, iron deficiency anemia, or, in severe cases, perforation with an acute abdomen. Diagnosis is primarily established by upper endoscopy, which permits direct visualization of the ulcer while simultaneously assessing associated complications, including stricture, fistula, or retained foreign material. CT is reserved for suspected perforation or other intra-abdominal complications. Initial treatment consists of high-dose PPIs, sucralfate, and eradication of *H. pylori* when indicated, with most MUs healing after prolonged medical therapy. However, refractory, recurrent, or complicated cases may require adjunctive medical therapy (e.g., misoprostol), endoscopic procedures (e.g., oversewing or stenting), or revisional surgery, including revision of gastrojejunostomy or reversal of RYGB [76,81].

Primary prevention focuses on routine PPI prophylaxis and aggressive modification of ulcerogenic risk factors. Current American Society for Metabolic and Bariatric Surgery

https://doi.org/10.17476/jmbs.2026.15.2.59

(ASMBS) literature reviews and observational studies support PPI therapy for at least 3 months after RYGB, with many centers extending to 6 months, which has been shown to reduce MU rates by approximately 50–70% (e.g., from 7.3% to 1.2%) [82]. Smoking cessation should be strongly encouraged, and patients should avoid NSAIDs and systemic corticosteroids whenever possible, since smoking and prolonged NSAID use are well-established modifiable risk factors for MU development. Low-dose aspirin can be used for patients with clear medical indications, preferably combined with concomitant PPI. *H. pylori* should be identified and eradicated when indicated (**Table 1**).

5. Internal hernia

Internal hernia is a well-recognized late complication after RYGB, with a reported incidence ranging from approximately 0.2% to 9%, although the cumulative incidence may exceed 10% in some cohorts, particularly when mesenteric defects or Petersen's space are not routinely closed. Reported incidences can vary according to defect closure technique, institutional practice, and duration of follow-up [83-88].

Internal hernia should be suspected in patients with new or recurrent crampy abdominal pain accompanied by nausea, vomiting, or obstructive symptoms, often postprandial, that may temporarily improve with positional changes, particularly forward flexion [89]. Diagnosis is primarily based on clinical suspicion, supported by contrast-enhanced CT, which may show the mesenteric swirl sign and abnormally clustered or displaced small bowel loops [90]. However, because imaging modality can be falsely negative, early diagnostic laparoscopy should be considered in patients with persistent or severe symptoms suggesting bowel ischemia.

Current ASMBS recommendations emphasize routine closure of all mesenteric defects, including Petersen's space, using nonabsorbable sutures or stapling techniques, and a low threshold for diagnostic laparoscopy because of the limitations of CT and the risk of bowel ischemia (**Table 1**) [84,91].

6. Gallstone formation

Gallstone formation is a common late complication after MBS, with reported incidences ranging from approximately 10% to 50%, driven primarily by rapid postoperative weight loss [92,93]. Rapid weight loss increases cholesterol saturation of bile and reduces gallbladder motility, favoring stone formation, typically within the first 6 to 12 months postoperatively [93,94]. Most gallstones remain asymptomatic, but a subset of patients develop biliary colic or complications such as acute cholecystitis, choledocholithiasis, or gallstone pancreatitis, which may require cholecystectomy [95]. Diagnosis is established by abdominal ultrasonography, the first-line imaging modality for gallstones and cholecystitis [95]. CT or magnetic resonance cholangiopancreatography is used as an adjunct when choledocholithiasis or gallstone pancreatitis is suspected [96].

Prophylactic ursodeoxycholic acid significantly reduces the overall incidence of gallstone formation after bariatric surgery, with a dose of 600 mg/day associated with improved compliance and superior outcomes regardless of procedure type, whereas higher doses (>600 mg/day) have not shown a significant benefit [97]. A subsequent, larger meta-analysis of 12 randomized controlled trials confirmed a significant reduction in overall gallstone incidence as well as symptomatic cholelithiasis and cholecystectomy rates [98]. Routine prophylactic cholecystectomy is not recommended, consistent with ASMBS guidance (**Table 1**) [99].

https://doi.org/10.17476/jmbs.2026.15.2.59

7. Nutritional deficiencies

Nutritional deficiencies are common long-term complications after bariatric surgery, particularly after procedures with a malabsorptive component such as RYGB. They result from reduced dietary intake, altered gastrointestinal anatomy, impaired nutrient absorption, and suboptimal adherence to lifelong supplementation. Current nutritional guidelines emphasize routine biochemical surveillance and procedure-specific supplementation because deficiencies of iron, vitamin B12, folate, calcium, vitamin D, and fat-soluble vitamins may occur after bariatric surgery [100-102].

Iron deficiency is one of the most frequent micronutrient deficiency and may progress to iron deficiency anemia if not detected and treated. After RYGB, the incidence of iron deficiency anemia ranges from 16–24%, depending on baseline nutritional status, procedure type, adherence to supplementation [103-105]. Major risk factors include female sex (particularly premenopausal women), younger age, low preoperative ferritin or preexisting anemia, heavy menstrual or gastrointestinal blood loss, the malabsorptive nature of RYGB, and poor adherence to postoperative iron supplementation [103,106]. The clinical spectrum of iron deficiency ranges from asymptomatic biochemical depletion to fatigue, reduced exercise tolerance, exertional dyspnea, palpitations, dizziness, and headache. Advanced deficiency may be associated with pallor, brittle nails, hair loss, or restless legs syndrome. Subtle cognitive impairment and reduced quality of life have also been reported in patients with iron deficiency. Diagnosis is based on laboratory evaluation showing microcytic or normocytic anemia with low serum ferritin (or ferritin in the low-normal range in the context of inflammation), reduced transferrin saturation, and low serum iron, often accompanied by elevated total iron-binding capacity. Because iron depletion often precedes overt anemia, routine biochemical surveillance is recommended for early detection. Treatment includes routine prophylactic multivitamin, mineral, and iron supplementation for all patients after bariatric surgery. Once iron deficiency is confirmed, oral iron supplementation should be escalated to therapeutic doses, preferably administered with vitamin C, while avoiding concomitant administration with calcium supplements or PPI to optimize absorption. Intravenous iron supplementation should be considered in patients with significant anemia, intolerance or malabsorption of oral iron, active bleeding, pregnancy, or persistently increased iron requirements. Contributory factors, including menstrual or gastrointestinal blood loss, MUs, and inadequate dietary intake, should be identified and corrected to prevent recurrent deficiency.

Vitamin B12 and folate deficiencies are also common (vitamin B12, ~24%; folate, ~9%, less common due to compensatory small bowel absorption) after RYGB, reflecting reduced intrinsic factor secretion, diminished gastric acid, and bypass of the duodenum and proximal jejunum [107]. Vitamin B12 deficiency manifests as glossitis, peripheral neuropathy, and megaloblastic anemia, whereas folate deficiency is more often asymptomatic. Diagnosis is based on serum Vitamin B12 and folate levels. Routine supplementation with a bariatric-specific multivitamin containing at least 350–1,000 μg of vitamin B12 and 400–800 μg of folic acid is recommended, with parenteral vitamin B12 reserved for symptomatic or malabsorptive patients [100-102,108].

Vitamin D and calcium deficiencies are common after RYGB. Vitamin D deficiency affects roughly 35–54% of patients depending on follow-up duration, while calcium deficiency (hypocalcemia) is comparatively less frequent, occurring in approximately 2% of RYGB patients [109,110]. This impaired vitamin D-calcium homeostasis predisposes to secondary

https://doi.org/10.17476/jmbs.2026.15.2.59

hyperparathyroidism and progressive bone loss, most pronounced at the hip, particularly in postmenopausal women [111]. Patients are often asymptomatic but may present with bone pain, muscle weakness, or fragility fracture. Diagnosis relies on serum calcium, 25-hydroxyvitamin D, and parathyroid hormone, with bone mineral density assessment in high-risk patients. Current guidelines recommend 1,200–1,500 mg/day of elemental calcium, preferably as calcium citrate given in divided doses, along with at least 3,000 IU/day of vitamin D. The vitamin D dose should be adjusted or titrated to maintain serum 25-hydroxyvitamin D level above 30 ng/mL (**Table 1**) [100-102,108].

8. Dumping syndrome and post-bariatric hypoglycemia (PBH)

Dumping syndrome is a common functional complication following MBS, with early dumping reported in up to 40% and late dumping in up to 25% of patients [112]. Early dumping results from rapid emptying of hyperosmolar gastric contents into the small intestine, causing osmotic fluid shifts into the bowel lumen and triggering early postprandial vasomotor (fatigue, flushing, palpitations, hypotension) and gastrointestinal symptoms (increased bowel sounds, diarrhea, bloating, nausea, abdominal cramping), within 1 hour after meal. Late dumping occurs 1–3 hours after meals, when a rapid rise in blood glucose provokes an exaggerated insulin response, resulting in postprandial hypoglycemia. Most patients experience mild, transient autonomic (adrenergic) symptoms such as sweating, palpitations, tachycardia, tremors, hunger, or anxiety.

PBH is considered a distinct clinical entity characterized by documented postprandial hypoglycemia accompanied by neuroglycopenic symptoms, such as confusion, impaired consciousness, or seizure due to inadequate glucose supply to the brain, and should be distinguished from the broader concept of late dumping syndrome [113].

Diagnosis of dumping syndrome is primarily clinical, based on the timing and pattern of postprandial symptoms and often supported by the Sigstad score. Modified oral glucose tolerance testing may be used in selected patients with suspected dumping syndrome. PBH is confirmed when 4 criteria are met: symptomatic postprandial hypoglycemia fulfilling Whipple's triad; a history of MBS performed more than 1 year before symptom onset; absence of fasting hypoglycemia; and no recent use of hypoglycemia-inducing drugs. Capillary blood glucose measurement is generally used to confirm hypoglycemic episodes in practice, while continuous glucose monitoring may serve as a useful adjunct, particularly for detecting nocturnal hypoglycemia, though it is not recommended as a standalone diagnostic tool [113].

Dietary modification is first-line, with small, frequent, protein-rich meals and restriction of simple carbohydrates, accompanied by pharmacologic therapy such as acarbose, diazoxide, or GLP-1 receptor antagonists, while revisional surgery is limited to refractory cases. Given its heterogeneous presentation and the potential risk of long-term morbidity, dumping syndrome and PBH require systematic screening and individualized management within the broader spectrum of post-bariatric surgical complications (**Table 1**) [113-115].

CONCLUSIONS

MBS is generally safe, effective, and durable treatment for severe obesity and its associated comorbidities, yet it carries a well-defined spectrum of complications that can differ by procedures. Early complications including leakage, bleeding, and VTE are uncommon in

https://doi.org/10.17476/jmbs.2026.15.2.59

experienced centers but can progress rapidly to life-threatening events if not recognized and managed promptly. Late complications, such as GERD, sleeve stenosis or twist after SG, GJ anastomotic stricture, marginal ulceration, internal hernia after RYGB, gallstone formation, nutritional deficiencies, and dumping syndrome and PBH, are more prevalent, often persist for years, and can meaningfully affect quality of life. A procedure-specific understanding of complication patterns, standardization of operative technique, modification of patient-related risk factors, and protocolized postoperative follow-up with appropriate medical, endoscopic, and surgical strategies are essential to minimize morbidity and preserve the long-term weight-loss and metabolic benefits of surgery.

REFERENCES

1. Renehan AG, Tyson M, Egger M, Heller RF, Zwahlen M. Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. Lancet 2008;371:569-78. PUBMED | CROSSREF
2. Lim JS, Kim S, Cho IY, Shin DW. Obesity and cancer: mechanisms, epidemiological evidence, and potential risk reduction. J Obes Metab Syndr 2026;35:14-37. PUBMED | CROSSREF
3. Kim E, Cho S, Kim J, Kim B, Seo HT, Nam GE, et al. 2025 Obesity fact sheet for Korea: prevalence of obesity, abdominal obesity from 2014 to 2023 and prevalence of chronic disease by obesity status. J Obes Metab Syndr 2026;35:176-87. PUBMED | CROSSREF
4. Moll H, Frey E, Gerber P, Geidl B, Kaufmann M, Braun J, et al. GLP-1 receptor agonists for weight reduction in people living with obesity but without diabetes: a living benefit-harm modelling study. EClinicalMedicine 2024;73:102661. PUBMED | CROSSREF
5. Lee H, Huh YJ, Seo WJ, Kim Y, Kim DJ; Informatics Committee of the Korean Society for Metabolic and Bariatric Surgery. A nationwide report on metabolic and bariatric surgery in 2019-2022: utilizing the Korean Society of Metabolic and Bariatric Surgery database registry. J Metab Bariatr Surg 2024;13:17-26. PUBMED | CROSSREF
6. Elbanna H, Emile S, El-Hawary GE, Abdelsalam N, Zaytoun HA, Elkaffas H, et al. Assessment of the correlation between preoperative and immediate postoperative gastric volume and weight loss after sleeve gastrectomy using computed tomography volumetry. World J Surg 2019;43:199-206. PUBMED | CROSSREF
7. Sioka E, Tzovaras G, Perivoliotis K, Bakalis V, Zachari E, Magouliotis D, et al. Impact of laparoscopic sleeve gastrectomy on gastrointestinal motility. Gastroenterol Res Pract 2018;2018:4135813. PUBMED | CROSSREF
8. Tomasicchio G, D'abramo FS, Dibra R, Trigiante G, Picciariello A, Dezi A, et al. Gastroesophageal reflux after sleeve gastrectomy. Fact or fiction? Surgery 2022;172:807-12. PUBMED | CROSSREF
9. Anderson B, Switzer NJ, Almamar A, Shi X, Birch DW, Karmali S. The impact of laparoscopic sleeve gastrectomy on plasma ghrelin levels: a systematic review. Obes Surg 2013;23:1476-80. PUBMED | CROSSREF
10. Kwon Y, Lee S, Kim D, ALRomi A, Park SH, Lee CM, et al. Biliopancreatic limb length as a potential key factor in superior glycemic outcomes after Roux-en-Y gastric bypass in patients with type 2 diabetes: a meta-analysis. Diabetes Care 2022;45:3091-100. PUBMED | CROSSREF
11. Lim R, Beekley A, Johnson DC, Davis KA. Early and late complications of bariatric operation. Trauma Surg Acute Care Open 2018;3:e000219. PUBMED | CROSSREF
12. Hsu JL, Ismail S, Hodges MM, Agala CB, Farrell TM. Bariatric surgery: trends in utilization, complications, conversions and revisions. Surg Endosc 2024;38:4613-23. PUBMED | CROSSREF
13. Mocanu V, Dang J, Ladak F, Switzer N, Birch DW, Karmali S. Predictors and outcomes of bleed after sleeve gastrectomy: an analysis of the MBSAQIP data registry. Surg Obes Relat Dis 2019;15:1675-81. PUBMED | CROSSREF
14. Seong BO, Ko CS, Oh SG, Jeong SA, Yook JH, Yoo MW, et al. Laparoscopic sleeve gastrectomy: ensuring safety and achieving an aesthetic gastric tube shape. J Surg Innov Educ 2024;1:22-5. CROSSREF
15. Park JY. Diagnosis and management of postoperative complications after sleeve gastrectomy. J Metab Bariatr Surg 2022;11:1-12. PUBMED | CROSSREF
16. Odovic M, Clerc D, Demartines N, Suter M. Early bleeding after laparoscopic Roux-en-Y gastric bypass: incidence, risk factors, and management - a 21-year experience. Obes Surg 2022;32:3232-8. PUBMED | CROSSREF

17. Kollmann L, Gruber M, Lock JF, Germer CT, Seyfried F. Clinical management of major postoperative bleeding after bariatric surgery. Obes Surg 2024;34:751-9. PUBMED | CROSSREF
18. Aboueisha MA, Freeman M, Allotey JK, Evans L, Caposole MZ, Tatum D, et al. Battle of the buttress: 5-year propensity-matched analysis of staple-line reinforcement techniques from the MBSAQIP database. Surg Endosc 2023;37:3090-102. PUBMED | CROSSREF
19. 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 2017;31:3547-51. PUBMED | CROSSREF
20. Chatani S, Inoue A, Ohta S, Takaki K, Sato S, Iwai T, et al. Transcatheter arterial embolization for postoperative bleeding following abdominal surgery. Cardiovasc Intervent Radiol 2018;41:1346-55. PUBMED | CROSSREF
21. Davrieux CF, Palermo M, Cúneo T, Zanutini D, Giménez ME. What is the role of image-guided endovascular surgery in postbariatric surgery bleeding complications? J Laparoendosc Adv Surg Tech A 2021;31:146-51. PUBMED | CROSSREF
22. Gagner M, Kemmeter P. Comparison of laparoscopic sleeve gastrectomy leak rates in five staple-line reinforcement options: a systematic review. Surg Endosc 2020;34:396-407. PUBMED | CROSSREF
23. Mocanu V, Dang J, Ladak F, Switzer N, Birch DW, Karmali S. Predictors and outcomes of leak after Roux-en-Y gastric bypass: an analysis of the MBSAQIP data registry. Surg Obes Relat Dis 2019;15:396-403. PUBMED | CROSSREF
24. Alanezi H, Alshehri A, Alrobiea A, Yoo MW. The causes, prevention, and management of gastric leakage after laparoscopic sleeve gastrectomy: a review article. J Metab Bariatr Surg 2019;8:28-33. CROSSREF
25. Vidarsson B, Sundbom M, Edholm D. Incidence and treatment of leak at the gastrojejunostomy in Roux-en-Y gastric bypass: a cohort study of 40,844 patients. Surg Obes Relat Dis 2019;15:1075-9. PUBMED | CROSSREF
26. Smith MD, Adeniji A, Wahed AS, Patterson E, Chapman W, Courcoulas AP, et al. Technical factors associated with anastomotic leak after Roux-en-Y gastric bypass. Surg Obes Relat Dis 2015;11:313-20. PUBMED | CROSSREF
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;30:2011-5. PUBMED | CROSSREF
28. Spiro C, Bennet S, Bhatia K. Meta-analysis of patient risk factors associated with post-bariatric surgery leak. Obes Sci Pract 2023;9:112-26. PUBMED | CROSSREF
29. 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;19:1672-7. PUBMED | CROSSREF
30. Rosenthal RJ, ; International Sleeve Gastrectomy Expert Panel, Diaz AA, Arvidsson D, Baker RS, Basso N, et al. International Sleeve Gastrectomy Expert Panel consensus statement: best practice guidelines based on experience of >12,000 cases. Surg Obes Relat Dis 2012;8:8-19. PUBMED | CROSSREF
31. Johari Y, Catchlove W, Tse M, Shaw K, Paul E, Chen R, et al. A 4-tier protocolized radiological classification system for leaks following sleeve gastrectomy. Ann Surg 2022;275:e401-9. PUBMED | CROSSREF
32. Musella M, Cantoni V, Green R, Acampa W, Velotti N, Maietta P, et al. Efficacy of postoperative upper gastrointestinal series (UGI) and computed tomography (CT) scan in bariatric surgery: a meta-analysis on 7516 patients. Obes Surg 2018;28:2396-405. PUBMED | CROSSREF
33. Xu T, Rosculet N, Steele K, Auster M. Comparison of upper gastrointestinal fluoroscopy versus computed tomography for evaluation of post-operative leak in a bariatric surgery patient. BJR Case Rep 2017;3:20160076. PUBMED | CROSSREF
34. Shin CI, Kim SH. Normal and abnormal postoperative imaging findings after gastric oncologic and bariatric surgery. Korean J Radiol 2020;21:793-811. PUBMED | CROSSREF
35. Spyropoulos C, Argentou MI, Petsas T, Thomopoulos K, Kehagias I, Kalfarentzos F. Management of gastrointestinal leaks after surgery for clinically severe obesity. Surg Obes Relat Dis 2012;8:609-15. PUBMED | CROSSREF
36. Kim J, Azagury D, Eisenberg D, DeMaria E, Campos GM; American Society for Metabolic and Bariatric Surgery Clinical Issues Committee. 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;11:739-48. PUBMED | CROSSREF
37. Martin Del Campo SE, Mikami DJ, Needleman BJ, Noria SF. Endoscopic stent placement for treatment of sleeve gastrectomy leak: a single institution experience with fully covered stents. Surg Obes Relat Dis 2018;14:453-61. PUBMED | CROSSREF

38. 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;25:1358-63. PUBMED | CROSSREF
39. Shoar S, Poliakin L, Khorgami Z, Rubenstein R, El-Matbouly M, Levin JL, et al. 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;27:2410-8. PUBMED | CROSSREF
40. Fuentes-Valenzuela E, García-Alonso FJ, Tejedor-Tejada J, Nájera-Muñoz R, de Benito Sanz M, Sánchez-Ocaña R, et al. Endoscopic internal drainage using transmural double-pigtail stents in leaks following upper gastrointestinal tract surgery. Rev Esp Enferm Dig 2021;113:698-703. PUBMED | CROSSREF
41. Rogalski P, Swidnicka-Siergiejko A, Wasielica-Berger J, Zienkiewicz D, Wieckowska B, Wroblewski E, et al. Endoscopic management of leaks and fistulas after bariatric surgery: a systematic review and meta-analysis. Surg Endosc 2021;35:1067-87. PUBMED | CROSSREF
42. Intriago JMV, de Moura DTH, do Monte Junior ES, Proença IM, Ribeiro IB, Sánchez-Luna SA, et al. Endoscopic vacuum therapy (EVT) for the treatment of post-bariatric surgery leaks and fistulas: a systematic review and meta-analysis. Obes Surg 2022;32:3435-51. PUBMED | CROSSREF
43. Coşkun M, Uprak TK, Günal Ö, Aliyeva A, Cingi A. Reinforcement in laparoscopic sleeve gastrectomy: is it effective? Surg Laparosc Endosc Percutan Tech 2024;34:290-4. PUBMED | CROSSREF
44. Aiolfi A, Gagner M, Zappa MA, Lastraioli C, Lombardo F, Panizzo V, et al. Staple line reinforcement during laparoscopic sleeve gastrectomy: systematic review and network meta-analysis of randomized controlled trials. Obes Surg 2022;32:1466-78. PUBMED | CROSSREF
45. Niaz O, Askari A, Currie A, McGlone ER, Zakeri R, Khan O, et al. Analysis of the effect of staple line reinforcement on leaking and bleeding after sleeve gastrectomy from the UK National Bariatric Surgery Registry. World J Surg 2024;48:1950-7. PUBMED | CROSSREF
46. Eckharter C, Heeren N, Mongelli F, Sykora M, Mühlhäusser J, Lottenbach N, et al. Partial staple line reinforcement with synthetic buttressing material in laparoscopic sleeve gastrectomy: a propensity score-matched analysis. Langenbecks Arch Surg 2023;408:47. PUBMED | CROSSREF
47. Abu-Abeid A, Litmanovich A, Abu-Abeid S, Eldar SM, Lahat G, Yuval JB. Long-term outcomes of patients with staple line leaks following sleeve gastrectomy. Obes Surg 2024;34:2523-9. PUBMED | CROSSREF
48. Carvalho L, Almeida RF, Nora M, Guimarães M. Thromboembolic complications after bariatric surgery: is the high risk real? Cureus 2023;15:e33444. PUBMED | CROSSREF
49. Winegar DA, Sherif B, Pate V, DeMaria EJ. Venous thromboembolism after bariatric surgery performed by Bariatric Surgery Center of Excellence participants: analysis of the Bariatric Outcomes Longitudinal Database. Surg Obes Relat Dis 2011;7:181-8. PUBMED | CROSSREF
50. Citla-Sridhar D, Rajpurkar M, Srivaths L, Bannow BS, Rosovsky RP, Sokkary N. Venous thromboembolism in obese hormonal contraceptive users: a large national database study. Res Pract Thromb Haemost 2025;9:103230. PUBMED | CROSSREF
51. Amaral FC, Baptista-Silva JC, Nakano LC, Flumignan RL. Pharmacological interventions for preventing venous thromboembolism in people undergoing bariatric surgery. Cochrane Database Syst Rev 2022;11:CD013683. PUBMED | CROSSREF
52. Kröll D, Nett PC, Rommers N, Borbély Y, Deichsel F, Nocito A, et al. Efficacy and safety of rivaroxaban for postoperative thromboprophylaxis in patients after bariatric surgery: a randomized clinical trial. JAMA Netw Open 2023;6:e2315241. PUBMED | CROSSREF
53. Rodríguez JI, Kobus V, Téllez I, Pérez G. Prophylaxis with rivaroxaban after laparoscopic sleeve gastrectomy could reduce the frequency of portomesenteric venous thrombosis. Ann R Coll Surg Engl 2020;102:712-6. PUBMED | CROSSREF
54. Acosta S, Salim S. Management of acute mesenteric venous thrombosis: a systematic review of contemporary studies. Scand J Surg 2021;110:123-9. PUBMED | CROSSREF
55. Gomes R, Costa-Pinho A, Ramalho-Vasconcelos F, Sousa-Pinto B, Santos-Sousa H, Resende F, et al. Portomesenteric venous thrombosis after bariatric surgery: a case series and systematic review comparing LSG and LRYGB. J Pers Med 2024;14:722. PUBMED | CROSSREF
56. Monaco G, Bucherini L, Stefanini B, Piscaglia F, Foschi FG, Ielasi L. Direct oral anticoagulants for the treatment of splanchnic vein thrombosis: a state of art. World J Gastroenterol 2023;29:4962-74. PUBMED | CROSSREF
57. Custo S, Tabone E, Aquilina A, Gatt A, Riva N. Splanchnic vein thrombosis: the state-of-the-art on anticoagulant treatment. Hamostaseologie 2024;44:242-54. PUBMED | CROSSREF
58. Helm MC, Simon K, Higgins R, Kindel TL, Gould JC. Perioperative complications increase the risk of venous thromboembolism following bariatric surgery. Am J Surg 2017;214:1135-40. PUBMED | CROSSREF

59. Thaher O, Wendt E, Hukauf M, Croner RS, Stroh C. Thromboembolic prophylaxis in bariatric and metabolic surgery: state-of-the-art according to the results of a nationwide registry study. Updates Surg 2025;77:1923-32. PUBMED | CROSSREF

60. Alsina E, Ruiz-Tovar J, Alpera MR, Ruiz-García JG, Lopez-Perez ME, Ramon-Sanchez JF, et al. Incidence of deep vein thrombosis and thrombosis of the portal-mesenteric axis after laparoscopic sleeve gastrectomy. J Laparoendosc Adv Surg Tech A 2014;24:601-5. PUBMED | CROSSREF

61. Tan SBM, Greenslade J, Martin D, Talbot M, Loi K, Hopkins G. Portomesenteric vein thrombosis in sleeve gastrectomy: a 10-year review. Surg Obes Relat Dis 2018;14:271-5. PUBMED | CROSSREF

62. Villagrán R, Smith G, Rodriguez W, Flores C, Cariaga M, Araya S, et al. Portomesenteric vein thrombosis after laparoscopic sleeve gastrectomy: incidence, analysis and follow-up in 1236 consecutive cases. Obes Surg 2016;26:2555-61. PUBMED | CROSSREF

63. Anewenah LS, Asif M, Francesco R, Ramachandra P. Portomesenteric vein thrombosis after laparoscopic sleeve gastrectomy for morbid obesity. BMJ Case Rep 2017;2017:bcr2016218264. PUBMED | CROSSREF

64. AlSabah SA, AlRuwaished M, Almazeedi S, Al Haddad E, Chouillard E. Portomesenteric vein thrombosis post-laparoscopic sleeve gastrectomy: case series and literature review. Obes Surg 2017;27:2360-9. PUBMED | CROSSREF

65. Alenazi NA, Ahmad KS, Essa MS, Alrushdan MS, Al-Shoaibi AM. Porto-mesenteric vein thrombosis following laparoscopic sleeve gastrectomy for morbid obesity: case series and literature review. Int J Surg Case Rep 2019;63:59-64. PUBMED | CROSSREF

66. Silecchia G, Iossa A. GERD and Barrett's esophagus as indications for revisional surgery after sleeve gastrectomy: experience of a bariatric center of excellence IFSO-EC and narrative review. Expert Rev Endocrinol Metab 2021;16:229-35. PUBMED | CROSSREF

67. Serra FE, Cohen RV. Gastroesophageal reflux disease after sleeve gastrectomy. Dig Med Res 2024;7:5. CROSSREF

68. Yeung KTD, Penney N, Ashrafian L, Darzi A, Ashrafian H. Does sleeve gastrectomy expose the distal esophagus to severe reflux?: a systematic review and meta-analysis. Ann Surg 2020;271:257-65. PUBMED | CROSSREF

69. Hajibandeh S, Hajibandeh S, Ghassemi N, Evans D, Cheruvu CVN. Meta-analysis of long-term de novo acid reflux-related outcomes following sleeve gastrectomy: evidence against the need for routine postoperative endoscopic surveillance. Curr Obes Rep 2023;12:395-405. PUBMED | CROSSREF

70. Chiappetta S, Lainas P, Kassir R, Valizadeh R, Bosco A, Kermansaravi M. Gastroesophageal reflux disease as an indication of revisional bariatric surgery-indication and results-a systematic review and metanalysis. Obes Surg 2022;32:3156-71. PUBMED | CROSSREF

71. Sillén L, Andersson E, Olbers T, Edholm D. Obstruction after sleeve gastrectomy, prevalence, and interventions: a cohort study of 9,726 patients with data from the Scandinavian Obesity Surgery Registry (SOReg). Obes Surg 2021;31:4701-7. PUBMED | CROSSREF

72. Eguchi M, Nogueira R, Balthazar da Silveira CA, Vidotto L, Kasakewitch JPG, Lech GE, et al. Endoscopic management of gastric stenosis after sleeve gastrectomy: a systematic review and single-arm meta-analysis. Surg Endosc 2025;39:5324-47. PUBMED | CROSSREF

73. Baumann AJ, Mramba LK, Hawkins RB, Carpenter AM, Fleisher MS, Ayzengart AL, et al. Endoscopic dilation of bariatric RNY anastomotic strictures: a systematic review and meta-analysis. Obes Surg 2018;28:4053-63. PUBMED | CROSSREF

74. Baccaro LM, Vunnamadala K, Sakharpe A, Wilhelm BJ, Aksade A. Stricture rate after laparoscopic Roux-en-Y gastric bypass with a 21-mm circular stapler versus a 25-mm linear stapler. Bariatr Surg Pract Patient Care 2015;10:33-7. PUBMED | CROSSREF

75. Dolce CJ, Dunnican WJ, Kushnir L, Bendana E, Ata A, Singh TP. Gastrojejunal strictures after Roux-en-Y gastric bypass with a 21-MM circular stapler. JSLS 2009;13:306-11. PUBMED

76. Vosburg RW, Nimeri A, Azagury D, Grover B, Noria S, Papasavas P, et al. ASMBS literature review on the treatment of marginal ulcers after metabolic and bariatric surgery. Surg Obes Relat Dis 2025;21:1-8. PUBMED | CROSSREF

77. Salame M, Jawhar N, Belluzzi A, Al-Kordi M, Storm AC, Abu Dayyeh BK, et al. Marginal ulcers after Roux-en-Y gastric bypass: etiology, diagnosis, and management. J Clin Med 2023;12:4336. PUBMED | CROSSREF

78. Beran A, Shaear M, Al-Mudares S, Sharma I, Matar R, Al-Haddad M, et al. Predictors of marginal ulcer after gastric bypass: a systematic review and meta-analysis. J Gastrointest Surg 2023;27:1066-77. PUBMED | CROSSREF

79. Rahman A, Mushtaq M, Nadeem F, Kabir SA. The impact of helicobacter pylori on marginal ulcer formation following gastric bypass: a systematic review and meta-analysis. Cureus 2025;17:e96787. PUBMED | CROSSREF

80. Wynn M, Tecson KM, Provost D. Marginal ulcers and associated risk factors after Roux-en-Y gastric bypass. Proc Bayl Univ Med Cent 2023;36:171-7. PUBMED | CROSSREF

81. Di Palma A, Liu B, Maeda A, Anvari M, Jackson T, Okrainec A. Marginal ulceration following Roux-en-Y gastric bypass: risk factors for ulcer development, recurrence and need for revisional surgery. Surg Endosc 2021;35:2347-53. PUBMED | CROSSREF

82. Vosburg RW, Nimeri A, Azagury D, Grover B, Noria S, Papasavas P, et al. American Society for Metabolic and Bariatric Surgery literature review on risk factors, screening recommendations, and prophylaxis for marginal ulcers after metabolic and bariatric surgery. Surg Obes Relat Dis 2025;21:101-8. PUBMED | CROSSREF

83. Ende V, Devas N, Zhang X, Yang J, Pryor AD. Internal hernia trends following gastric bypass surgery. Surg Endosc 2023;37:7183-91. PUBMED | CROSSREF

84. Altieri MS, Carter J, Aminian A, Docimo S Jr, Hinojosa MW, Cheguevara A, et al. American Society for Metabolic and Bariatric Surgery literature review on prevention, diagnosis, and management of internal hernias after Roux-en-Y gastric bypass. Surg Obes Relat Dis 2023;19:763-71. PUBMED | CROSSREF

85. 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;16:641-5. PUBMED | CROSSREF

86. Aghajani E, Nergaard BJ, Leifson BG, Hedenbro J, Gislason H. 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;31:3743-8. PUBMED | CROSSREF

87. 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;5:565-70. PUBMED | CROSSREF

88. Iannelli A, Facchiano E, Gugenheim J. Internal hernia after laparoscopic Roux-en-Y gastric bypass for morbid obesity. Obes Surg 2006;16:1265-71. PUBMED | CROSSREF

89. Tartamella F, Ziccarelli A, Cecchini S, Ferro M, Riccò M, Baldini E, et al. Abdominal pain and internal hernias after Roux-en-Y Gastric Bypass: are we dealing with the tip of an iceberg? Acta Biomed 2019;90:251-8. PUBMED | CROSSREF

90. 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. PUBMED | CROSSREF

91. Wesley Vosburg R, Carter J, Khorgami Z, Fam J, El Djouzi S, Groller KD, et al. A literature review on reoperative metabolic and bariatric surgery for chronic complications: American Society for Metabolic and Bariatric Surgery Clinical Issues Committee and Surgery Revision Task Force. Surg Obes Relat Dis 2026;22:823-31. PUBMED | CROSSREF

92. Verhoeff K, Mocanu V, Dang J, Switzer NJ, Birch DW, Karmali S. Characterization and risk factors for early biliary complications following elective bariatric surgery: an Mbsaqip analysis. Obes Surg 2022;32:1170-7. PUBMED | CROSSREF

93. Chen S, Zheng Y, Cai J, Wu Y, Chen X. Gallstones after bariatric surgery: mechanisms and prophylaxis. Front Surg 2025;12:1506780. PUBMED | CROSSREF

94. Dai Y, Luo B, Li W. Incidence and risk factors for cholelithiasis after bariatric surgery: a systematic review and meta-analysis. Lipids Health Dis 2023;22:5. PUBMED | CROSSREF

95. Patel H, Jepsen J. Gallstone disease: common questions and answers. Am Fam Physician 2024;109:518-24. PUBMED

96. Tran A, Hoff C, Polireddy K, Neymotin A, Maddu K. Beyond acute cholecystitis-gallstone-related complications and what the emergency radiologist should know. Emerg Radiol 2022;29:173-86. PUBMED | CROSSREF

97. Fearon NM, Kearns EC, Kennedy CA, Conneely JB, Heneghan HM. The impact of ursodeoxycholic acid on gallstone disease after bariatric surgery: a meta-analysis of randomized control trials. Surg Obes Relat Dis 2022;18:77-84. PUBMED | CROSSREF

98. Al-Huniti M, Alsardia Y, Odeh A, Bdour B, Hassanat R, Aloun A, et al. Ursodeoxycholic acid prophylaxis and the reduction of gallstone formation after bariatric surgery: an updated meta-analysis of randomized controlled trials. Cureus 2023;15:e50649. PUBMED | CROSSREF

99. Leyva-Alvizo A, Arredondo-Saldaña G, Leal-Isla-Flores V, Romanelli J, Sudan R, Gibbs KE, et al. Systematic review of management of gallbladder disease in patients undergoing minimally invasive bariatric surgery. Surg Obes Relat Dis 2020;16:158-64. PUBMED | CROSSREF

100. Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom KA, Greiman L. American Society for Metabolic and Bariatric Surgery integrated health nutritional guidelines for the surgical weight loss patient 2016 update: micronutrients. Surg Obes Relat Dis 2017;13:727-41. PUBMED | CROSSREF

101. Mechanick JI, Apovian C, Brethauer S, Garvey WT, Joffe AM, Kim J, et al. Clinical practice guidelines for the perioperative nutrition, metabolic, and nonsurgical support of patients undergoing bariatric procedures - 2019 update: cosponsored by American Association of Clinical Endocrinologists/American College of Endocrinology, the Obesity Society, American Society for Metabolic & Bariatric Surgery, Obesity Medicine Association, and American Society of Anesthesiologists - executive summary. Endocr Pract 2019;25:1346-59. PUBMED | CROSSREF

102. O'Kane M, Parretti HM, Pinkney J, Welbourn R, Hughes CA, Mok J, et al. British Obesity and Metabolic Surgery Society guidelines on perioperative and postoperative biochemical monitoring and micronutrient replacement for patients undergoing bariatric surgery-2020 update. Obes Rev 2020;21:e13087. PUBMED | CROSSREF

103. Enani G, Bilgic E, Lebedeva E, Delisle M, Vergis A, Hardy K. The incidence of iron deficiency anemia post-Roux-en-Y gastric bypass and sleeve gastrectomy: a systematic review. Surg Endosc 2020;34:3002-10. PUBMED | CROSSREF

104. Gowanlock Z, Lezhanska A, Conroy M, Crowther M, Tiboni M, Mbuagbaw L, et al. Iron deficiency following bariatric surgery: a retrospective cohort study. Blood Adv 2020;4:3639-47. PUBMED | CROSSREF

105. Sandvik J, Bjerkan KK, Græslie H, Hoff DAL, Johnsen G, Klöckner C, et al. Iron deficiency and anemia 10 years after Roux-en-Y Gastric bypass for severe obesity. Front Endocrinol (Lausanne) 2021;12:679066. PUBMED | CROSSREF

106. Gesquiere I, Lannoo M, Augustijns P, Matthys C, Van der Schueren B, Foulon V. Iron deficiency after Roux-en-Y gastric bypass: insufficient iron absorption from oral iron supplements. Obes Surg 2014;24:56-61. PUBMED | CROSSREF

107. Lewis CA, de Jersey S, Seymour M, Hopkins G, Hickman I, Osland E. Iron, vitamin B_{12}, folate and copper deficiency after bariatric surgery and the impact on anaemia: a systematic review. Obes Surg 2020;30:4542-91. PUBMED | CROSSREF

108. Heo J. Dietary management after bariatric surgery in patients with obesity and diabetes. J Korean Diabetes 2023;24:95-101. CROSSREF

109. Gao Z, Liang Y, Huang S, Wu Z, Li M, Yang J. Prevalence and associated factors of vitamin D deficiency after Roux-en-Y gastric bypass: a systematic review and meta-analysis. Int J Surg 2023;109:4273-85. PUBMED | CROSSREF

110. Shah M, Sharma A, Wermers RA, Kennel KA, Kellogg TA, Mundi MS. Hypocalcemia after bariatric surgery: prevalence and associated risk factors. Obes Surg 2017;27:2905-11. PUBMED | CROSSREF

111. Stein EM, Silverberg SJ. Bone loss after bariatric surgery: causes, consequences, and management. Lancet Diabetes Endocrinol 2014;2:165-74. PUBMED | CROSSREF

112. van Beek AP, Emous M, Laville M, Tack J. Dumping syndrome after esophageal, gastric or bariatric surgery: pathophysiology, diagnosis, and management. Obes Rev 2017;18:68-85. PUBMED | CROSSREF

113. Abdelgadir E, Rashid F, Al Awadi F, Al Busaidi NB, Alfaris N, Al Hadad M, et al. Post-bariatric hypoglycemia management: a gulf cooperation council consensus statement. J Endocr Soc 2026;10:bvaf225. PUBMED | CROSSREF

114. Iqbal A, Makin V. Hypoglycemia after bariatric surgery: management updates. Cleve Clin J Med 2025;92:103-8. PUBMED | CROSSREF

115. Ribeiro R, Rossoni C, Rocha C, Viveiros O, Taranu V, Eiró F, et al. Post-bariatric hypoglycemia: diagnosis, mechanisms and management-a case report-based review. J Clin Med 2026;15:3220. PUBMED | CROSSREF