Gastrointestinal

Gastrointestinal complications of Roux-en-Y gastric bypass surgery

SS Siddique[1,2], JD Feuerstein[1*]

Abstract

Introduction

Obesity is a major health problem worldwide. Gastric bypass surgery is increasingly being used to manage obesity. Roux-en-Y gastric bypass surgery is associated with a number of early and late gastrointestinal complications. This manuscript aims to review the early and late gastrointestinal complications of Roux-en-Y gastric bypass surgery.

Discussion

Predictors of complications include patient comorbidities: Age > 65, BMI > 50, obstructive sleep apnoea, gastroesophageal reflux disease, history of a thrombotic event, diabetes, hyperlipidaemia, peripheral vascular disease hypertension, renal impairment, arthritis and asthma. Additionally, surgical technique (e.g open vs laparoscopic) and intraoperative complications also predict post-operative complications. Early complications include gastrointestinal bleeding (GIB), anastamotic leaks and small bowel obstruction. Late complications include hernias, intestinal obstruction, marginal ulcers, fistulas, anastamotic strictures, cholelithiasis, intussusception, nutritional deficiencies, and dumping syndrome.

Conclusion

Overall, the complications resulting from Roux-en-Y gastric bypass are usually amenable to endoscopic or surgical treatment. However, early recognition and treatment of these complications are critical to reduce overall morbidity and mortality.

*Corresponding author
Email: jfeuerst@bidmc.harvard.edu

[1] Department of Medicine and Division of Gastroenterology, Beth Israel Deaconess Medical Center, Harvard Medical School
[2] Department of Medicine, Mount Auburn Hospital, Harvard Medical School

Introduction

Obesity is a major health problem worldwide and of epidemic proportion in the Western world. In the United States, over 1/3 of individuals are obese[1]. In 2010[2] up to 27.2% of US adults were reported to be obese, with a BMI > 30 kg/m[2] and 6.55% were morbidly obese, with a BMI > 40 kg/m[2]. It is one of the leading causes of preventable death[1]. Obesity is associated with multiple complications including stroke, cancer, sleep apnoea, hypertension, hyperlipidaemia, heart disease, and type II diabetes[1]. Obese patients, who do not achieve a substantial and sustained weight loss with improvement of comorbidities with diet, exercise, and medications should be referred for bariatric surgery.

The currently FDA approved gastric bypass surgeries include Roux-en-Y, sleeve gastrectomy and laparoscopic banding. While these surgeries are all efficacious in achieving significant weight loss and resolution of complications secondary to obesity, each surgery carries both acute and late gastrointestinal related complications[3,4,5,6,7]. This article will focus on the gastrointestinal complications of Roux-en-Y gastric bypass (RYGB).

Complications can be divided into early or late. Early complications are defined as those occurring within 2 weeks post-operatively or within 30 days of the surgery. For this paper, we will refer to early complications as those within 2 weeks of the surgery.

Early complications include gastro-intestinal bleeding (GIB), anastamotic leaks and small bowel obstruction. Late complications for this manuscript refer to those arising after the second postoperative week and include hernias, intestinal obstruction, marginal ulcers, fistulas, anastamotic strictures, cholelithiasis, intussus-ception, nutritional deficiencies, and dumping syndrome[6,7].

Discussion

Predictors of complications

There are multiple risk factors for post-surgical complications including both patient specific and surgery related. Patients with severe morbid obesity with a BMI > 50 kg/m[2] and age >65 are at higher risk of complications. Importantly though, the subgroup with a BMI > 50 are at high risk of death from their obesity if they do not undergo bariatric surgery. Other predictors include preoperative obstructive sleep apnoea (OSA), gastroesophageal reflux disease (GERD), history of a thrombotic event, diabetes, hyperlipidaemia, peripheral vascular disease hypertension, renal impairment, arthritis and asthma[6].

Additionally, open procedures compared to laparoscopic surgery and occurrence of intra-operative complications also predict post-operative complications[8].

Early complications

Anastomotic or staple line leaks

Anastomotic or staple line leaks are a dreaded and potentially devastating complication of RYGB, with a mortality rate of nearly 50% if not treated quickly[9,10].

A study of 3000 patients undergoing RYGB found that an anastomotic leak (ASL) is one of the strongest independent risk factors for post-operative death[9]. Early recognition and treatment is critical. The incidence of ASL is relatively low at 0.4%–5.2%[9,10,11,12] and does not differ significantly between open and laparoscopic RYGB[9].

ASL usually develops within 5-7 days post-operatively[12].

The exact pathophysiology is unknown but the most likely aetiology is secondary to ischemia and/or technical error. There are five potential sites where a leak may form: gastrojejunostomy (most common site), gastric pouch staple line, gastric

OA Minimally Invasive Surgery
Open Access

remnant staple line, Roux limb staple line and jejunojejunal anastomosis[7,9,10].

Risk factors: Risk factors for ASL include older males with elevated BMI, multiple comorbidities, previous abdominal surgery, or revision of bariatric procedures[12,13].

One technical factor that may increase the risk of ALS is the Roux limb orientation after laparoscopic RYGB[14,15,16]. It has been postulated that the retrocolic Roux limb has a more direct path to the gastric pouch, compared with the antecolic route that may be associated with lower gastrojejunal anastomotic tension[13].

However, studies have reported conflicting results. Edwards et al. reported that ASLs may occur more commonly after antecolic (3%) versus retrocolic (0.5%) laparoscopic RYGB[14], but Bertucci et al. reported no ASLs after 141 retrocolic and 200 antecolic procedures[15], and Carrasquilla et al. reported an ASL rate of 0.1% after 1000 antecolic procedures versus 1.85% after 108 retrocolic procedures[16].

Diagnosis and presenting symptoms: The diagnosis of ASL is based on a high clinical suspicion. Symptoms are nonspecific and variable including tachycardia, fever, abdominal pain, nausea, and vomiting and purulent drain output. In one study, sustained tachycardia > 120 beats per minute in the post-operative setting was highly specific for an ASL[17]. Radiographic studies are occasionally helpful and may show fluid collection adjacent to the pouch, diffuse abdominal fluid or rarely, intraperitoneal air.

Importantly, though, a normal radiograph does not rule out this diagnosis. If suspicion remains then intraoperative exploration and diagnosis is necessary[18].

Treatment: Early operative management is the standard of care in any haemodynamically unstable patient[18]. In patients who are haemodynamically stable, conservative management with intravenous fluids, broad-spectrum antibiotics, bowel rest, enteral nutrition and nasogastric decompression can be considered[18].

ASLs can be reduced by careful operative technique, choosing the appropriate size and placement of the staples (see discussion below on late complication of fistula for further details), reinforcement of the anastomosis with biologic buttress material[19] and fibrin sealant[20] and performing an intraoperative leak test[13].

Gastrointestinal Bleeding
Gastrointestinal bleeding (GIB) after RYGB can be life threatening if not recognized and treated early. In a systematic review comparing open versus laparoscopic RYGB, the frequency of GIB was significantly higher in the laparoscopic RYGB series (1.9% vs. 0.6%, respectively)[21]. The overall incidence is between 1.9% and 4.4%[22,23].

GIB after laparoscopic RYGB can be intraperitoneal or intraluminal and most commonly originates at one of the five potential staple lines: the gastric pouch, excluded stomach, Roux limb staple line, gastrojejunostomy, and jejunojejunostomy[24,25]. Staple-line bleeding occurs at the transected tissue edges or at the sites of staple penetration of the tissue. Additional sites of bleeding include injuries to the liver, spleen, or trocar sites[24].

Diagnosis and presenting symptoms: Patients may display significant amounts of bloody output from the abdominal drains, tachycardia, hematemesis, melena, haematochezia, positive occult blood and a drop in the haemoglobin/hematrocrit[17].

The diagnosis is confirmed endoscopically and/or surgically[24].

Treatment: Depending on the location of the GIB, lesions can be treated endoscopically by gastroenterologists, surgically assisted deep enteroscopy, or via repeated surgical exploration.

Treatment is dependent on the location of the lesion and size of the lesion. If a visible vessel is present or stigmata of a recent bleed is noted as per the Forrest classification[26] on endoscopy, endoscopic clips and epinephrine can be utilized. If the bleed is around a recent staple line, cautery should be used with caution

as it may cause greater mucosal damage[27].

Similarly, if endoscopic intervention fails, angiographic intervention should be used with caution given the risk of mucosal ischemia to a recent anastomosis[28].

Occasionally, in severe cases of GIB, surgical oversewing or complete revision may be necessary[22,23].

Small bowel obstruction
In the very immediate post-operative period, acute stenosis at the gastrojejunal anastomosis may develop secondary to surrounding tissue edema. Otherwise, the most common cause of small bowel obstruction (SBO) is secondary to an internal hernia[28,29].

This can happen as an early or late complication of RYGB. Obstruction at the jejunojejunal anastomosis is the second most common cause of SBO occurring in approximately 1.8% of antecolic laparoscopic RYGB procedures[15]. Early obstructions at the jejunojejunal anastomosis can be the result of bowel kinking, narrowing, or acute angulation of the anastomosis[30].

Other causes include postsurgical anastomotic edema, stenosis, ischemia, and staple-line bleeding with intraluminal thrombus formation[29,30].

Early SBOs at other locations usually develop secondary to oedema and extrinsic compression of the retrocolic Roux limb as it crosses the transverse mesocolic defect from a thickened cicatrix formation in this area[31]. Less common causes of SBO after laparoscopic RYGB include trocar site incisional hernias, adhesive bands, bezoars, anastomotic strictures and jejunojejunal anastomosis intussus-ception[31].

Diagnosis and presenting symptoms: Symptoms of a gastrojejunal stenosis are usually acute onset nausea, vomiting and abdominal pain[28,29]. In stenosis developing more distally, the symptoms may be more gradual in onset[28,29]. Diagnosis can be made via radiologic findings of contrast or barium not flowing past the stenotic area[32]. Other findings may include incarcerated bowel, abdominal wall defects, dilated stomach, or dilated

bowel at or above the obstruction with collapse of the distal bowel loops [32,33].

Treatment: A small bowel obstruction after gastric bypass can result in a closed loop obstruction, which can be lethal. Therefore, if a patient appears ill or septic, an immediate exploration is necessary[28,29]. In cases of tissue oedema, nasogastric decompression and bowel rest may assist in resolution of the oedema and stenosis[34]. When the stenosis is at the gastrojejunal anastomosis, balloon dilation with an EGD can be used[35].

In cases of a stenosis at the jejunojejunal anastomosis a single balloon enteroscope can be used to dilate the stenotic area[35].

While these lesions are often treatable, there is a risk of morbidity with the management of postoperative obstruction with perforation and death[28,36].

Late complications
Gastrointestinal bleeding (GIB)
GIB occurs in up to 1.9% of RYGB[27]. It can present as an early or late complication. Early GIB usually develops along the anastomotic staple lines and has been discussed previously. The most common form of late GIB is a marginal ulcer (MU). MU develops at the gastrojejunal anastomosis, and may occur in up to 20% of cases[11,37]. They typically form 4 to 10 weeks after surgery, but can develop at any time[38].

The pathophysiology of these ulcers are unclear and are most likely multifactorial with a significant component of ischaemia, acid exposure, and foreign body reaction from sutures or staples[39].

Risk factors: Patient specific risk factors that may compromise the gastric mucosal barrier resulting in MU include: tobacco use, nonsteroidal anti-inflammatory drug (NSAID), helicobacter pylori infection and diabetes[39].

Diagnosis and presenting symptoms: Affected individuals typically present with epigastric pain, nausea, vomiting, hematemesis, melena or occult bleeding[39]. Bleeding from these ulcers

can be massive and life threatening. Marginal ulcers usually occur as solitary ulcers of varying size. In untreated cases, the ulcers can lead to stricture or fistula formation[39].

Treatment: Given the multifactorial aetiology of marginal ulcers, treatment is often challenging. If a visible vessel is present or stigmata of a recent bleed is noted on endoscopy, endoscopic clips and epinephrine can be utilized. Cautery should be used with caution as it may cause greater mucosal damage[27] or perforation given the possible ischemic aetiology for these ulcers.

Similarly, if endoscopic intervention fails, angiographic intervention should be used with caution given the risk of mucosal ischaemia[27].

Additionally, some advocate that if any suture material is around the marginal ulcer, this should be removed endoscopically as foreign bodies may play a role in the formation of these ulcers[27]. In cases of recurrent bleeding despite endoscopic intervention, surgical revision may be necessary. In all cases, maximal acid suppression with a proton pump inhibitor should be administered given the potential role acid may play in the development of these ulcers. Helicobacter pylori should be checked with gastric biopsies, breath test, or stool antigen and treated if positive. Blood IgG testing is not ideal, as this does not indicate active infection but rather prior exposure to helicobacter pylori[39].

Hernias
Internal and ventral hernias can present as a late postoperative complications at any time after RYGB. Hernias can lead to clinically significant complications, such as a closed loop bowel obstruction with or without strangulation and have been reported to be the most common cause of small bowel obstruction (SBO) after laparoscopic RYGB[28,29].

The type of internal hernia is determined most often by the configuration of the Roux limb with three potential locations: jejunojejunal mesenteric defect, Petersen's space and the transverse mesocolic defect,

which is unique to the retrocolic approach. Transverse mesocolic hernias are the most common with the retrocolic approach, followed by jejunojejunostomy and Petersen's space hernias[40].

In an antecolic approach, however, both Petersen's and jejunojejunostomy mesenteric defect hernias are reported with similar frequencies[41].

Risk factors: Compared with the open approach, the incidence of internal hernia is greater after laparoscopic RYGB ranging from 3% to 4.5%[40].

Reduced bowel manipulation and peritoneal irritation with the laparoscopic approach causes fewer postoperative adhesions, compared with the open approach. This results in reduced fixation of the Roux limb and less scarring to help close mesenteric defects. This in effect allows the free bowel loops to herniate through the mesenteric defects[42].

Diagnosis and presenting symptoms: Abdominal pain is the most common presentation of an internal hernia. Although patients can present in the immediate postoperative period, usually the presentation is delayed, occurring months to years after the operation. Some patients report previous episodes of gastrointestinal upset and frequent mild symptoms of self-limited abdominal pain, nausea and/or vomiting before their presentation with persistent symptoms.

The small bowel may intermittently become incarcerated and then reduced at the site of the hernia, causing this subtle presentation and atypical bowel obstruction features[40].

The diagnosis of a hernia can be difficult and radiologic evaluation may be non-diagnostic. The small bowel may intermittently become trapped and then reduced at the site of the hernia, causing a subtle presentation. Computed tomography (CT) signs of hernias have variable diagnostic predictive values, and signs of bowel obstruction may not be present until strangulation is imminent. The "mesenteric swirl sign" has a high specificity for this diagnosis[43]. The high frequency of negative imaging may be due to the fact that scans may not be obtained during an episode of

OA Minimally Invasive Surgery
Open Access

herniation or that incarceration of a short segment of the biliary limb may not cause recognizable small bowel dilation. Therefore, the occurrence of abdominal colic after a gastric bypass should raise suspicion of an obstruction, and recurrent symptoms should be surgically explored unless a clear alternative diagnosis is established[44].

Treatment: Cases of internal hernias and ventral hernias that are not incarcerated can be electively repaired[45]. Any case of incarcerated bowel requires emergent surgery due to risk of strangulation, ischemia, and perforation[45]. Reduction of hernias and repair of defects with non-absorbable suture can usually be accomplished through a laparoscopic technique. Incarceration, which in many cases is transient, may not be found at exploration, but closure of defects nonetheless achieves good results with relief of pain in the majority of patients[45].

Anastomotic stricture
The incidence of a stricture of the gastrojejunal anastomosis ranges from 2.9% to 23.0% of patients and has been noted to be substantially more frequent with the laparoscopic than the open approach[46,47]. They typically develop between 4-10 weeks postoperatively[47]. Strictures can form at any of the anastomosis as discussed in the early complications section. Postulated mechanisms include post-procedure ischemia causing scarring, chronic ischemia resulting from tension at the anastomosis, decreased healing capacity of individual patients, non-ischemic excessive scar formation, recurrent marginal ulcers, tension or malposition of the anastomosis, and surgical technique.

Technical factors that might contribute to increased stricture formation are type of stapler used (circular vs. linear), stapler size, hand-sewing, and surgeon experience. Circular staplers offer surgeons a reproducible anastomosis, eliminating any technique-dependent variability. Multiple studies have shown that the incidence of gastrojejunal anastomotic stricture is higher when a 21 mm

versus a 25 mm circular stapler is used in construction of the gastrojejunostomy[47,48,49,50].

In contrast, Frutos et. al. reported a 3.4% incidence of stenosis with the use of 21 mm circular staplers. They argued that restrictive procedures fail when the stoma is too wide. Therefore, they advocated the use of a 21 mm stapler instead of the 25 mm stapler, because the stapler is less difficult to insert through the abdominal wall and small intestine, but, more importantly, because the small diameter of the anastomosis delays gastric pouch emptying and consequently increases weight loss in the long-term[51].

Diagnosis and presenting symptoms: Patients with anastomotic stricture may present weeks to months postoperatively with progressive dysphagia, postprandial vomiting, bloating, and upper abdominal pain. Obstructive symptoms from strictures at the gastrojejunal anastomosis tend to develop shortly after meals, whereas vomiting associated with strictures at the jejunojejunal anastomosis occur 1 hour or more after meals. Diagnostic upper endoscopy should be performed in all suspected cases. More distal strictures can also be evaluated radiologically with oral contrast or with a single balloon enteroscopy.

Treatment: Endoscopic balloon dilation is the first line of treatment for anastomotic strictures[52]. Strictures at the gastrojejunal anastomosis can be dilated via esophago-gastroduodenoscopy (EGD) with balloon dilation. More distal strictures can sometimes be reached with a single balloon enteroscope. Findings have shown that 17% to 67% of cases respond to the first dilation, whereas 3% to 8% of cases require three or more dilations[52]. In all cases of balloon dilation, the main risk is perforation. To minimize this risk, dilations should be done to the smallest effective size[47]. If endoscopy fails, surgical revision may be required[53,54].

Fistulas
A gastrogastric fistula (GGF) is an abnormal communication between

the gastric pouch and the excluded stomach. The most common cause is secondary to incomplete gastric transection at the original surgery. However, gastrogastric fistula may form secondary to ulceration, ASL, or from a gastrojejunal stricture[7,11,55]. The incidence ranges between 1.5% and 6.0%[55].

Diagnosis and presenting symptoms: The most common symptom is inadequate weight loss or weight gain. Patients may also present with epigastric pain and/or worsening acid reflux secondary to the new connection with the remnant stomach. Diagnosis is made via endoscopic evaluation of the fistulous tract or via imaging with oral contrast flowing through the fistulous tract[47,55].

Treatment: Asymptomatic fistulous tracts are usually not a concern. However, symptomatic fistulous tracts can be closed endoscopically using specialized large endoclips to approximate the tissue surrounding the fistula[47]. Alternatively, if the anatomy and location of the fistula is amenable, a stent can be placed to exclude the fistulous tract and allow the mucosa to heal naturally while it is relatively diverted [47]. Others have used a fibrin sealant to seal the fistula with variable success[47]. If this modality fails, then surgical revision may be necessary. Any ongoing acid related symptoms can be treated with proton pump inhibitors as needed[47,55].

Cholelithiasis
Cholelithiasis occurs in about one-third of patients who undergo bariatric surgery. The rapid post-surgical weight loss is a significant contributor to the development of gallstones as is the pre-surgical obesity[56]. Studies indicate that 22% to 45% of patients who undergo bariatric surgery will develop gallstones within the first few months after surgery but in many cases the gallstones resolve on their own[57].

Diagnosis and presenting symptoms: Patient with cholelithiasis usually do not have any symptoms unless there is acute inflammation of the gallbladder or impaction of a stone within the biliary system. Patients present with nausea, vomiting, and biliary colic

Review

localized to the epigastrium and right upper quadrant. Diagnosis is confirmed with ultrasound or CT imaging[56,57].

Treatment: Six months of post-operative ursodeoxycholic acid therapy is a commonly used regimen supported by a multi-society consensus statement to reduce the risk of developing gallstones[58]. Ursodeoxycholic acid prophylaxis post-gastric bypass has reduced the prevalence rate of cholelithiasis to less than 3%[59,60]. The role of concomitant prophylactic cholecystectomy during gastric bypass is controversial given the difficulties in treating acute cholecystectomy complications in patients with a RYGB anatomy[61]. Cholecystectomy is indicated in bariatric surgery patients only in the context of symptomatic cholelithiasis, similar to non-bariatric patients[61].

Intussusception
Small bowel intussusceptions may be transient or fixed and may result in an SBO after gastric bypass surgery. They typically occur at or near the jejunojejunal anastomosis, with the staple line at the anastomosis presumably acting as the lead point for the intussusception. Altered small bowel motility near the anastomosis may also be a contributing factor[62,63].

Diagnosis and presenting symptoms: Intussusception may occur months or years after gastric bypass and is associated with nausea, vomiting, abdominal pain, and bowel obstruction. Like internal hernia, intussusception may be transient or chronic.

Intussusception is often retrograde with the jejunojejunostomy acting as a lead point and progressing proximally along either the alimentary or biliopancreatic limb.

Antegrade intussusception has also been reported. Plain abdominal films, gastrointestinal contrast studies, and abdominal ultrasound are unreliable diagnostic tools. CT scan may reveal a "target sign" or dilated excluded stomach, but these findings are relatively insensitive[62,63].

Treatment: A high level of clinical suspicion is usually the impetus for surgical exploration. Strangulation mandates resection of the affected area. In the absence of strangulation, resection or reduction and plication are accepted approaches with most surgeons favouring the former[63].

Nutritional deficiencies
Alterations of gastrointestinal anatomy and physiology after gastric bypass elicit imbalances owing to reduced oral intake or excessive losses secondary to reconfiguration of GI motility, pH and enzymatic profile[64]. Anaemia is the most common complication and is estimated to occur in 20%–49% of patients' post-operatively[65]. This is due to iron, folate and vitamin B12 deficiencies[66]. Major deficits are also noted in magnesium, calcium, zinc, 25-hydroxyvitamin D, thiamine and β-carotene[64,66].

Diagnosis and presenting symptoms: Patients may be asymptomatic or present with symptoms manifesting a particular nutritional deficiency. Patients with anaemia may present with dyspnoea, fatigue and inability to concentrate. Zinc deficiency can result in hair loss and low calcium levels can result in osteoporosis and fractures. Blood work is recommended to detect and monitor vitamin and mineral deficiencies[64,65,66,67].

Treatment: Patients who undergo gastric bypass require a referral to a registered dietician for nutrition counselling, and lifelong vitamin and mineral supplementation is recommended[67].

Dumping syndrome
Dumping syndrome is a common complication following RYGB reported in up to 70% of patients[68]. It occurs as a consequence of ingesting large amounts of concentrated sugars, which results in the stomach rapidly, and without regulation, emptying its contents into the small intestine[69]. Patients who undergo laparoscopic RYGB and patients who have preexisting hyperlipidaemia or gastroesophageal reflux disease appear to be at especially high risk for dumping syndrome[6].

Diagnosis and presenting symptoms: Dumping syndrome can be subdivided into early and late types. Early dumping syndrome develops rapidly within 15 minutes of eating food. As the food enters the alimentary limb, the hyperosmolality of the food triggers a rapid fluid shift from the plasma into the bowel. Symptoms include abdominal pain, diarrhoea, nausea, and tachycardia[69]. Late dumping syndrome develops 2-3 hours after eating. This results from the insulin released after a large bolus of food and eventually hypoglycaemia develops. Symptoms include classic hypoglycaemic symptoms including weakness, fatigue, diaphoresis, and in severe cases could include syncope[70,71,72].

Treatment: Treatment is dietary avoidance. Foods that are high in concentrated sugar content should be avoided. Small frequent meals and separated solids and liquids from each other can mitigate symptoms. Usually symptoms will resolve with time[71,72].

Conclusion
Roux-en-Y gastric bypass offers excellent long-term results in obese patients. However, it is associated with clinically important early and late gastrointestinal complications, which have varying degrees of morbidity and mortality risk. It is essential for physicians to be familiar with these complications and facilitate early recognition and prompt intervention to minimize the morbidity and mortality associated with gastric bypass.

References
1. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of Childhood and Adult Obesity in the United States, 2011-2012. JAMA. 2014;311(8):806-814.
2. Sturm, Roland, and Aiko Hattori. "Morbid obesity rates continue to rise rapidly in the United States." International Journal of Obesity 37.6. 2013: 889-891.
3. Flum DR, Belle SH, King WC, et al. Perioperative safety in the longitudinal assessment of bariatric surgery. N Engl J Med. 2009; 361(5):445-54.
4. Chapman AE, Kiroff G, Game P, et al. Laparoscopic adjustable gastric

banding in the treatment of obesity: a systematic literature review. Surgery. 2004; 135(3):32.

5. Shen R, Dugay G, Rajaram K, et al. Impact of patient follow-up on weight loss after bariatric surgery. Obes Surg. 2004; 14(4):514-9.

6. Cawley J, Sweeney MJ, Kurian M, & Beane S. Predicting complications after bariatric surgery using obesity-related co-morbidities. Obesity surgery. 2007, 17(11), 1451-1456.

7. Al Harakeh AB. Complications of laparoscopic Roux-en-Y gastric bypass. Surgical Clinics of North America. 2011, 91(6), 1225-1237.

8. Stenberg, Erik, et al. "Early Complications After Laparoscopic Gastric Bypass Surgery: Results From the Scandinavian Obesity Surgery Registry." Annals of surgery. 2013.

9. Fernandez AZ Jr, DeMaria EJ, Tichansky DS, et al. Experience with over 3,000 open and laparoscopic bariatric procedures: multivariate analysis of factors related to leak and resultant mortality. Surg Endosc. 2004;18(2):193–7.

10. Ballesta C, Berindoague R, Cabrera M, et al. Management of anastomotic leaks after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2008;18(6):623–30.

11. Griffith PS, Birch DW, Sharma AM, & Karmali S. Managing complications associated with laparoscopic Roux-en-Y gastric bypass for morbid obesity. Canadian Journal of Surgery. 2012, 55(5), 329.

12. Baker RS, Foote J, Kemmeter P, et al. The science of stapling and leaks. Obes Surg. 2004;14(10):1290–8.

13. Fullum TM, Aluka KJ, Turner PL. Decreasing anastomotic and staple line leaks after laparoscopic Roux-en-Y gastric bypass. Surg Endosc. 2009; 23(6):1403–8.

14. Edwards MA, Jones DB, Ellsmere J, et al. Anastomotic leak following antecolic versus retrocolic laparoscopic Roux-en-Y gastric bypass for morbid obesity. Obes Surg. 2007; 17(3):292–7.

15. Bertucci W, Yadegar J, Takahashi A, et al. Antecolic laparoscopic Roux-en-Y gastric bypass is not associated with higher complication rates. Am Surg. 2005; 71(9):735–7.

16. Carrasquilla C, English WJ, Esposito P, et al. Total stapled, total intra-abdominal (TSTI) laparoscopic Roux-en-Y gastric bypass: one leak in 1000 cases. Obes Surg. 2004; 14(5):613–7.

17. Bellorin O, Abdemur A, Sucandy I, et al. Understanding the significance, reasons and patterns of abnormal vital signs after gastric bypass for morbid obesity. Obes Surg. 2011; 21:707-13.

18. Gonzalez R, Sarr MG, Smith CD, et al. Diagnosis and contemporary management of anastomotic leaks after gastric bypass for obesity. J Am Coll Surg. 2007;204:47-55.

19. Miller KA, Pump A. Use of bioabsorbable staple reinforcement material in gastric bypass: a prospective randomized clinical trial. Surg Obes Relat Dis. 2007;3(4): 417–21 [discussion: 422].

20. Lee MG, Provost DA, Jones DB. Use of fibrin sealant in laparoscopic gastric bypass for the morbidly obese. Obes Surg. 2004;14(10):1321–6.

21. Podnos YD, Jimenez JC, Wilson SE, et al. Complications after laparoscopic gastric bypass: a review of 3464 cases. Arch Surg. 2003; 138:957-61.

22. Nguyen NT, Rivers R, Wolfe BM. Early gastrointestinal hemorrhage after laparoscopic gastric bypass. Obes Surg. 2003;13:62-5.

23. Rosenthal RJ, Szomstein S, Kennedy CI, et al. Laparoscopic surgery for morbid obesity: 1,001 consecutive bariatric operations performed at the Bariatric Institute, Cleveland Clinic Florida. Obes Surg. 2006;16: 119-24.

24. Nguyen NT, Longoria M, Chalifoux S, et al. Gastrointestinal hemorrhage after laparoscopic gastric bypass. Obes Surg. 2004;14(10):1308–12.

25. Nguyen NT, Rivers R, Wolfe BM. Early gastrointestinal hemorrhage after laparoscopic gastric bypass. Obes Surg. 2003;13:62-5.

26. Forrest JH, Finlayson NDC, & Shearman DJC. Endoscopy in gastrointestinal bleeding. The Lancet. 1974, 304(7877), 394-397.

27. Thompson CC, Roslin MS, Bipan C, et al. RESTORe: Randomized Evaluation of Endoscopic Suturing Transorally for Anastomotic Outlet Reduction: a double-blind, sham-controlled multicenter study for treatment of inadequate weight loss or weight regain following Roux-en-Y gastric bypass. Gastroenterology. 2010; 138(Suppl 1):S-388.

28. Champion JK, Williams M. Small bowel obstruction and internal hernias after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2003;13(4):596–600.

29. Capella RF, Iannace VA, Capella JF. Bowel obstruction after open and laparoscopic gastric bypass surgery for morbid obesity. J Am Coll Surg. 2006;203(3): 328–35.

30. Lewis CE, Jensen C, Tejirian T, et al. Early jejunojejunostomy obstruction after laparoscopic gastric bypass: case series and treatment algorithm. Surg Obes Relat Dis. 2009;5(2):203–7.

31. Ahmed AR, Rickards G, Messing S, et al. Roux limb obstruction secondary to constriction at transverse mesocolon rent after laparoscopic Roux-en-Y gastric bypass. Surg Obes Relat Dis. 2009;5(2):194–8.

32. Carucci LR. Role of imaging in bariatric procedures: Roux-en-Y gastric bypass and laparoscopic adjustable gastric banding. Imaging Med. 2011;3(1):81–92.

33. Tucker ON, Escalante-Tattersfield T, Szomstein S, Rosenthal RJ. The ABC system: a simplified classification system for small bowel obstruction after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2007;17(12): 1549–1554.

34. Hayanga, Awori J., Kirsten Bass-Wilkins, and Gregory B. Bulkley. "Current management of small-bowel obstruction." Advances in surgery. 2005, 39: 1-33.

35. Ahmad J, et al. "Endoscopic balloon dilation of gastroenteric anastomotic stricture after laparoscopic gastric bypass." Endoscopy. 2003, 35.09: 725-728.

36. Higa KD, HoT, BooneKB.Internal hernias after laparoscopic Roux-en-Y gastric bypass: incidence, treatment and prevention. Obes Surg. 2003;13: 350-4.

37. 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):1090–1094.

38. El-Hayek K, et al. "Marginal ulcer after Roux-en-Y gastric bypass: what have we really learned?." Surgical endoscopy 2012, 26.10: 2789-2796.

39. 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):505–516.

40. Garza Jr E, Kuhn J, Arnold D, Nicholson W, Reddy S, & McCarty T. Internal hernias after laparoscopic Roux-en-Y gastric bypass. The American journal of surgery. 2004, 188(6), 796-800.

41. Ahmed AR, Rickards G, Husain S, Johnson J, Boss T, & O'Malley W. Trends in internal hernia incidence after laparoscopic Roux-en-Y gastric bypass. Obesity surgery. 2007, 17(12), 1563-1566.

42. Hope, William W, et al. "Failure of mesenteric defect closure after Roux-en-Y gastric bypass." JSLS: Journal of the Society of Laparoendoscopic Surgeons. 2010, 14.2: 213.

43. Carucci LR, Turner MA, Shaylor SD. Internal hernia following Roux-en-Y gastric bypass surgery for morbid obesity: evaluation of radiographic findings at small-bowel exami- nation. Radiology. 2009;251(3):762–770.

44. Greenstein, Alexander J., and Robert W. O'Rourke. "Abdominal pain after gastric bypass: suspects and solutions." The American Journal of Surgery. 2011, 201.6: 819-827.

45. Gandhi, Alok D, Patel RA, and Brolin RE. "Elective laparoscopy for herald symptoms of mesenteric/internal hernia after laparoscopic Roux-en-Y gastric bypass." Surgery for Obesity and Related Diseases. 2009, 5.2: 144-149.

46. Mathew A, Veliuona MA, DePalma FJ, et al. Gastrojejunal stricture after gastric bypass and efficacy of endoscopic intervention. Dig Dis Sci. 2009;54:1971-8.

47. Nguyen NT, Stevens CM, Wolfe BM. Incidence and outcome of anastomotic stricture after laparoscopic gastric bypass. J Gastrointest Surg. 2003;7:997-1003.

48. Fisher, Barry L, James D. Atkinson, and Daniel Cottam. "Incidence of gastroenterostomy stenosis in laparoscopic Roux-en-Y gastric bypass using 21-or 25-mm circular stapler: a randomized prospective blinded study." Surgery for Obesity and Related Diseases. 2007, 3.2: 176-179.

49. Suggs, Jay W, et al. "Complications at gastrojejunostomy after laparoscopic Roux-en-Y gastric bypass: comparison between 21-and 25-mm circular staplers." Surgery for Obesity and Related Diseases. 2007, 3.5: 508-514.

50. Dolce, Charles J, et al. "Gastrojejunal strictures after Roux-en-Y gastric bypass with a 21-mm circular stapler." JSLS, Journal of the Society of Laparoendoscopic Surgeons. 2009, 13.3: 306-311.

51. Frutos MD, Luja´n J, Garc´ıa A, et al. Gastrojejunal anastomotic stenosis in laparo- scopic gastric bypass with a circular stapler (21 mm): incidence, treatment and long-term follow-up. Obes Surg. 2009;19(12):1631–5.

52. Ryskina KL, Miller KM, Aisenberg J, et al. Routine management of stricture after gastric bypass and predictors of subsequent weight loss. Surg Endosc. 2010; 24(3):554–60.

53. Jha S, Levine MS, Rubesin SE, et al. Detection of strictures on upper gastrointestinal tract radiographic examinations after laparoscopic Roux-en-Y gastric bypass surgery: importance of projection. AJR Am J Roent- genol. 2006;186(4): 1090–1093.

54. Buckwalter JA, Herbst CA Jr. Leaks occurring after gastric bariatric operations. Surgery. 1988;103(2):156–160.

55. Cucchi SG, Pories WJ, MacDonald KG, et al. Gastrogastric fistulas. A complication of divided gastric bypass surgery. Ann Surg. 1995;221: 387-91.

56. Shiffman, MITCHELL L., et al. "Changes in gallbladder bile composition following gallstone formation and weight reduction." Gastroenterology. 1992, 103.1: 214-21.

57. Shiffman ML, Sugerman HJ, Kellum JM, Brewer WH, & Moore EW. Gallstone formation after rapid weight loss: a prospective study in patients undergoing gastric bypass surgery for treatment of morbid obesity. American Journal of Gastroenterology. 1991, 86(8).

58. Mechanick JI, Kushner RF, Sugerman HJ, 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. Obesity. 2009;17(Suppl 1):S1–70. V.

59. Wudel LJ, Jr, Wright JK, Debelak JP, Allos TM, Shyr Y, Chapman WC. Prevention of gallstone formation in morbidly obese patients undergoing rapid weight loss: results of a randomized controlled pilot study. J Surg Res. 2002;102(1):50–6.

60. Miller K, Hell E, Lang B, Lengauer E. Gallstone formation prophylaxis after gastric restrictive procedures for weight loss: a randomized double-blind placebo-controlled trial. Ann Surg. 2003;238:697–702.

61. Mason EE, Renquist KE. Gallbladder management in obesity surgery. Obes Surg. 2002;12(2):222–9.

62. Coster DD, Sundberg SM, Kermode DS, et al. Small bowel obstruction due to antegrade and retrograde intussusception after gastric bypass: three case reports in two patients, literature review, and recommendations for diagnosis and treatment. Surg Obes Relat Dis. 2008;4:69–72.

63. Zainabadi K, Ramanathan R. Intussusception after laparoscopic Roux-en-Y gastric bypass. Obes Surg. 2007;17:1619–1623.

64. Dalcanale L, Oliveira CP, Faintuch J, et al. Long-term nutritional outcome after gastric Bypass. Obes Surg. 2010; 20:181-7.

65. Aills L, Blankenship J, Buffington C, et al.; Allied Health Sciences Section Ad Hoc Nutrition Committee. ASMBS Allied Health Nutri- tional Guidelines for the surgical weight loss patient. Surg Obes Relat Dis. 2008;4(Suppl):S73-108.

66. Lopez PP, Patel NA, Koche LS. Outpatient complications encountered

following Roux-en-Y gastric bypass. Med Clin North Am. 2007;91:471-83.

67. Karmali S, Johnson Stoklossa C, Sharma A, et al. Bariatric surgery: a primer. Can Fam Physician. 2010;56: 873-9.

68. DeMaria, Eric J. "Bariatric surgery for morbid obesity." New England Journal of Medicine. 2007, 356.21: 2176-2183.

69. Shuster MH, Vazquez JA. Nutritional concerns related to Roux-en-Y gastric bypass: what every clinician needs to know. Crit Care Nurs Q. 2005;28(3):227-60.

70. Blackwood HS. Obesity: a rapidly expanding challenge. Nurs Manage. 2004;35(5):27-35.

71. Deitel M. The Change in the Dumping Syndrome Concept. Obesity Surgery. 2008;18(12):1622-1624.

72. Foster-Schubert KE. Hypoglycemia Complicating Bariatric Surgery: Incidence and Mechanisms. Current opinion in endocrinology, diabetes, and obesity. 2011;18(2):129.