Gastrointestinal Complications of Bariatric Surgery

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The stomach is a slave that must accept everything that is given to it, but which avenges wrongs as slyly as does the slave.

—Émile Souvestre

7.1 Introduction

Prevalence of bariatric surgery is on increasing trend all over the globe due to sustainable weight loss, improvement in quality of life, and comorbidity resolution. Therapies for obesity can be divided broadly into four groups: behavioural (primarily diet and exercise), drugs, devices (intragastric balloon), and surgery. The complications of bariatric surgery include gastrointestinal complications, venous thromboembolism, and nutritional complications. The overall complication rate is low ranging from 0.29% to 0.78% [1], which is similar to most elective abdominal operations like cholecystectomy. The reported common early complications include leak, bleeding, obstruction, venous thromboembolism while the late complications include internal hernias, dumping, nutritional deficiencies, and gastro-oesophageal reflux. In this chapter, we will discuss the common metabolic GI complications specific to each procedure. Hormonal and physiological changes following bariatric surgery are covered in another chapter and therefore, will not be discussed here. The delayed and long-term surgical complications like Internal hernia, obstruction, stenosis etc. will also not be discussed as the focus of the book is on metabolic complications. Additionally, liver complications, nesidioblastosis, and nutritional complications have been discussed in other chapters and will not be discussed.

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A. G. Bhasker et al. (eds.), *Management of Nutritional and Metabolic Complications of Bariatric Surgery*,
https://doi.org/10.1007/978-981-33-4702-1_7

Table 7.1 Gastrointestinal and Metabolic complications of Bariatric surgical procedures

	AGB	SG	RYGB	OAGB
Nutritional complications	+	+	+	+
Reflux	+	+	–	+
Nausea	+	+	+	+
Vomiting	+	+	+	+
Marginal ulcers	–	–	+	+
Oesophagitis	+	+	+	–
Barrett's oesophagus	+	+	+	–
Diarrhoea	–	+	+	+
Steatorrhoea	–	–	+	+
Constipation	+	+	+	+
Early dumping syndrome	–	+	+	+
Late dumping	–	–	+	+
SIBO	–	–	+	+
Liver dysfunction	–	–	+	+
Gallstones	+	+	+	+
Alteration in taste	–	+	+	+
De novo food intolerance	–	+	+	+
Nesidioblastosis	–	–	+	+

AGB Adjustable Gastric Banding, *SG* Sleeve Gastrectomy, *RYGB* Roux-en-Y Gastric Bypass, *OAGB* One Anastamoses Gastric Bypass, *SIBO* small intestinal bacterial over-growth

The metabolic GI complications after bariatric surgery are summarised in Table 7.1.

Table 7.1 shows the commonly seen metabolic complications after various bariatric surgeries.

7.2 Reflux

The prevalence of gastroesophageal reflux disease (GERD) is 10–20% in normal population as compared to 37–72% in patients with obesity [2]. In general, weight loss brings about a decrease in intra-abdominal pressure leading to a decrease in GERD. However, after bariatric surgery, depending on the procedure patients can be at an increased or decreased risk of GERD.

7.2.1 Adjustable Gastric Band

Functional or anatomical obstruction due to band can lead to oesophageal dilatation and ineffective motility. This can happen either because of overtight bands or large bites of food being stuck up in the pouch above the band. Patients with oesophageal dilatation present with reflux-like symptoms [3]. These symptoms require band deflation/band removal and conversion to a sleeve gastrectomy or a Roux en Y gastric bypass (RYGB) to maintain weight loss.

7.2.2 Laparoscopic Sleeve Gastrectomy (SG)

GERD is known to decrease following RYGB, but its incidence after SG is controversial. In a systematic review, four studies showed an increase in incidence of GERD and seven studies found a decrease in GERD postoperatively [4]. At our institute, we found that GERD improved following SG as assessed by symptom questionnaire as well as endoscopy. De novo GERD was seen in some patients on scintigraphy, however, that might not be pathologically significant because of a reduction in total acid amount after sleeve [5]. Some patients have an improvement in their reflux symptoms. Possible mechanisms include accelerated gastric emptying, decreased total acid production, and reduced intrabdominal pressure due to weight loss. In a recent metanalysis, there was a slight trend towards an increase in GERD but a definitive conclusion could not be made. The results of oesophageal function tests and 24 h pH monitoring also yielded conflicting results in different studies [2]. There are various mechanisms that might be responsible for de novo GERD after SG. In a single centre study a triphasic response to GERD after SG was observed [6]. In the first year after SG, reflux increased probably due to disruption of angle of His, incompetence of sling fibres at cardia (these fibres are transacted during LSG), decreased tone of lower oesophageal sphincter, decreased distensibility of stomach, and increased intragastric pressure. Second year onwards, the reflux is decreased due to weight loss and a decrease in intra-abdominal pressure. In another paper, the same authors have [7] demonstrated a 22% incidence of post-SG GERD after 1 year, which came down to 3% after 3 years. However, with time, a neofundus is formed. Neo fundus occurs when a part of fundus is left behind at the level of left crus, which results in a conical sleeve with narrowing at mid-body region leading to weight regain and a mid-stomach functional stenosis leading to a second peak in GERD [6]. This can predispose the patients undergoing SG to develop GERD [8]. Other anatomical factors like twisting of sleeve causing a functional obstruction as well as incisural stenosis can cause reflux. We strongly believe that a missed hiatal hernia might be an important factor for persistence/development of symptoms of GERD (Fig. 7.1).

Despite this, not all patients develop GERD following sleeve. A common notion that sleeve leads to worsening of reflux should be considered with caution. Precautions such as avoiding narrowing at incisura, not leaving any fundus behind the left crus, and selecting proper bougie size should be undertaken during surgery. A low threshold should be maintained for dissection of hiatus on suspicion during laparoscopy and for repairing any concomitant hiatal hernia. There is also a need to standardize the technique of sleeve gastrectomy to decrease the incidence of GERD following it.

Most patients who develop GERD can be managed by dietary modification, proton pump inhibitors (PPIs), and prokinetics. Endoscopy and a contrast study should be done to rule out mechanical causes or a missed hiatal hernia. Patients with symptoms unresponsive to medical management as well as severe nocturnal symptoms require conversion to RYGB. In our experience post-SG, we have encountered

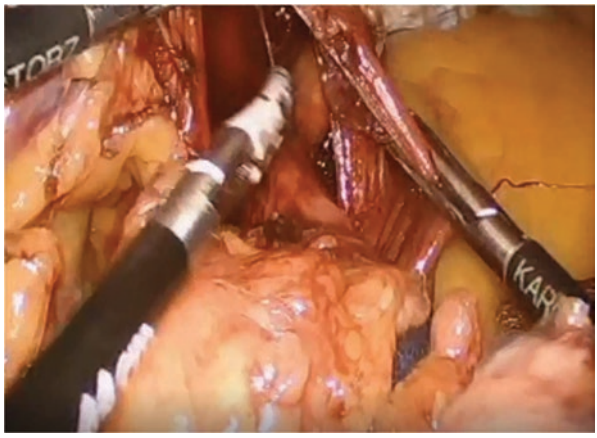

Fig. 7.1 Hiatal hernia after dissection which was missed initially

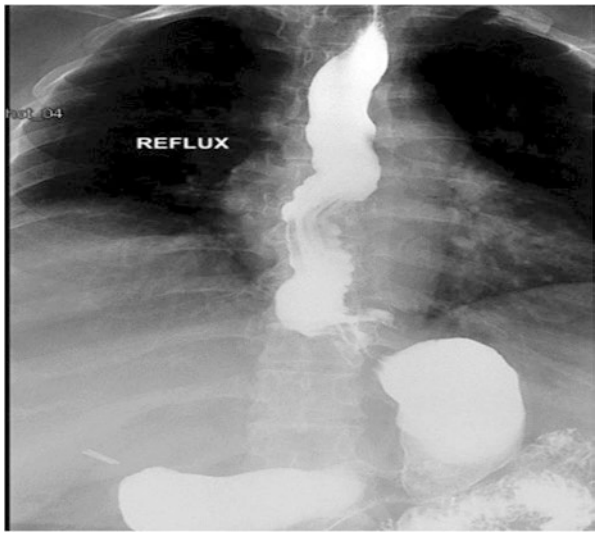

Fig. 7.2 Reflux after LSG

GERD (Fig. 7.2) due to remnant fundus which got twisted, twisting of sleeve in mid-part due to dense adhesions with the left lobe, and hiatus hernia with excessive posterior fundus. All these patients were converted to RYGB.

7.2.3 One Anastomosis Gastric Bypass (OAGB)

One anastomosis gastric bypass is usually associated with biliary reflux. It is defined as the presence of bile in the gastric pouch and into the oesophagus with/without symptoms of gastroesophageal reflux disease (GERD). The occurrence of GERD correlates with a gastric pouch shorter than 9 cm in length and with the presence of

preoperative GERD [9]. Usually, 10–12 cm length of the gastric pouch is recommended [9]. It can be identified by clinical findings through validated questionnaires [10]. In a study by Tolone et al., 15 patients underwent oesophagogastric junction function evaluation by endoscopy, 24 h pH-impedance monitoring, and high-resolution impedance manometry performed both preoperatively and 1 year after surgery. No patient reported heartburn or de novo regurgitation postoperatively. On endoscopy at 1 year no patient had oesophagitis or biliary gastritis. There was no difference in manometric features and patterns after surgery, whereas gastroesophageal pressure gradient (GEPG) and intragastric pressures (IGP) were statistically diminished [11]. On the contrary, laparoscopic sleeve gastrectomy (SG) showed a high-pressure gastric pouch with gastroesophageal reflux [12].

The correct anatomical configuration including creating a long and narrow pouch without any twist at gastrojejunostomy site is necessary. In addition, we take an anti-reflux stitch at top of gastrojejunostomy site to align anastomosis vertically and hence preventing bile reflux. Gastric and/or oesophageal symptomatic bile reflux is quite rare (0–0.7%) in OAGB [13, 14]. Chevallier et al. had reported that seven patients following OAGB presented with intractable biliary reflux. They were re-operated after a mean of 23 months when the mean BMI was 25.7 kg/m^2. All patients got cured of intractable bile reflux after conversion to a Roux en Y gastric bypass [13].

Dietary and lifestyle modifications, PPIs (40 mg twice a day for 6 months), and sucralfate (1 g before every meal and before bedtime for 3 months, followed by 1 g before bedtime for another 3 months) [15]. If conservative treatment fails, a revisional surgery is advised with conversion to either RYGB or Braun side-to-side anastomosis between the afferent and the efferent limb about 15–20 cm beyond the gastro-jejunal anastomosis [16].

7.3 Barret's Oesophagus and Adenocarcinoma

Reflux can further complicate the long-term course of sleeve gastrectomy patients as it can predispose to Barrett's oesophagus (BE). Limited literature is available in regard to the incidence of BE in post-sleeve gastrectomy patients. The incidence of BE has been reported with a wide range, varying from 1.2% to 17.2% in recent studies [17–19]. However, the authors did not follow the strict criteria to take a biopsy at least 1 cm above the gastro-oesophageal junction, which might have resulted in a falsely positive Barrett's. A biopsy within 1 cm of he gastroesophageal junction can have normal gastric mucosa, which is misinterpreted as BE [20].

Since BE is a known premalignant condition, it is imperative that adequate studies should be performed to formulate guidelines for its screening and treatment. There have been three case reports of oesophageal adenocarcinoma following SG. However, preoperative endoscopy was performed only in one of them. [21–23]. In another case report oesophageal adenocarcinoma developed on a pre-existing BE (diagnosed on preoperative endoscopy), 3 years after SG [24]. These are rare case reports in literature considering the number of sleeve gastrectomies performed all

over the globe. Moreover, preoperative endoscopy was done only in one patient and other two cases might already have an underlying dysplasia. De novo/worsening of GERD may be avoided by proper preoperative evaluation of reflux symptoms and intraoperative precautions, keeping in mind that the benefits of sleeve gastrectomy far outweigh its potential complications.

After OAGB, there are serious concerns not only regarding the symptomatic biliary reflux (BR) into the stomach and the oesophagus [25, 26], but also the increased risk of malignancy after OAGB [27, 28]. BR is known to cause histological changes in oesophagus and gastric pouch, secondary to acute and chronic inflammatory changes in oesophageal and gastric mucosa. These changes might progress to premalignant condition, Barrett's oesophagus [29]. Under the influence of constant BR, Barrett's oesophagus can progress to an incomplete intestinal metaplasia (type III) instead of complete intestinal metaplasia (type I), which has a higher risk of gastro-oesophageal cancer development [30]. We recently reported probably the first case of adenocarcinoma of oesophagus involving gastro-oesophageal junction following OAGB within 2 years of surgery [31] (Fig. 7.3).

7.4 Nausea and Vomiting

Nausea and vomiting after SG can be due to anatomical or functional causes. The most common site for anatomical stenosis after SG is at the incisura angularis. The common causes being inappropriate placement of bougie and oversewing of staple line, especially when it is done without a bougie in place. Functional stenosis can also occur due to a twist in the sleeve. Twisting usually occurs due to excessive traction on anterior/posterior wall of the stomach when firing the stapler. The twist can be localized at an area between body and antrum or along the full length of sleeve resulting in a spiral sleeve. Diagnosis of anatomical stenosis can be made by endoscopy; however, the diagnosis of functional stenosis is difficult as the endoscope can easily negotiate the twist. CT scan with three-dimensional reconstruction is best to diagnose spiral sleeve [32]. Endoscopic balloon dilatation is the first line of

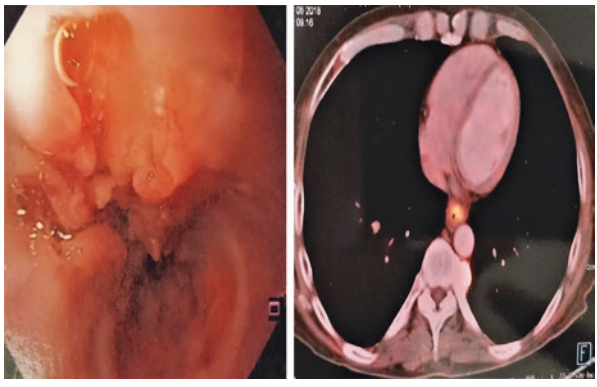

Fig. 7.3 Endoscopic image of growth in lower oesophagus (left) and Positron Emission Tomography (PET) showing increased uptake in lower oesophageal growth (right)

Table 7.2 Grades of anastomotic stricture on the basis of endoscopy

Grade: I	Mild stenosis, which allows a 10.5-mm endoscope to pass
Grade: II	Moderate stenosis, which allows an 8.5-mm paediatric endoscope to pass
Grade: III	Severe stenosis, which allows only guide-wire to pass
Grade: IV	Complete obstruction, which is non-traversable

treatment requiring multiple sessions over several weeks. Patients are kept on a liquid diet, ensuring adequate calorie intake and nutritional supplementation. Failure of endoscopic treatment and long segment stricture requires conversion to RYGB, which is considered the gold standard for this complication.

Anastomotic Stricture An anastomotic stricture post-RYGB can happen due to anastomotic tension, ischemia, and subclinical leaks. Patients with stricture usually present with dysphagia, nausea, vomiting, and painless post-prandial regurgitation. Upper gastro-intestinal endoscopy (UGIE) is required for confirmation of diagnosis. Table 7.2 gives a classification for the degree of anastomotic stricture.

Treatment includes endoscopic balloon dilatation to at least 15 mm in the first sitting to decrease the recurrence rate by the use of through-the-scope (TTS) balloon catheters [33]. They lead to circumferential dilatation and gradual expansion of stenosis and thus prevent excess pain and minimizes the risk of perforation. In 95% of cases, obstruction is relieved by two separate sessions of dilatation [34], however, re-stenosis may occur in 3% of these patients [35]. Patients not responding to endoscopic therapy even after four sessions of balloon dilatation are candidates for surgical revision of Gastro-jejunal (G-J) anastomosis, which is required in less than 0.4% cases [36].

To prevent stenosis after OAGB, an anastomotic size of ≥2.5 cm is highly recommended. We perform an anastomotic size of 5–6 cm. This complication can be managed by pneumatic endoscopic dilations successfully or conversion to laparoscopic Roux en Y gastric bypass if endoscopic dilatations fail.

7.5 Marginal Ulcers

Marginal ulcer is characterized by the development of mucosal erosion at the gastrojejunal (G-J) anastomosis, usually on the jejunal side as it does not have the acid buffering capacity of the duodenum and hence becomes vulnerable to ulcer formation [37]. Reported incidence of marginal ulcers is variable, ranging from 0.6 to 16% [38, 39] after RYGB. Marginal ulcer rate is lower following OAGB (0.5–4%) [40]. In a systematic review of 11 studies (1 RCT, 5 prospective, 5 retrospectives) were analyzed; of which 1174 patients underwent RYGB and 767 patients underwent MGB-OAGB. Marginal ulcer was reported in 7/362 (1.9%) of RYGB and 15/523 (2.86%) of MGB-OAGB patients [41]. In our own experience, marginal ulcer was detected in 4/46 (13.04%) of OAGB patients all of them being asymptomatic. So routine surveillance by endoscopy may be recommended in all OAGB

patients. Marginal ulcer rates and need for surgical revisions after OAGB in some large series and long-term follow-up papers are shown in Table 7.3.

The actual incidence is much higher than reported, as documented ulcers represent only those that are diagnosed on endoscopy, but many are treated medically based on symptoms without undergoing any endoscopic evaluation. Risk factors for development of marginal ulcer include smoking (causes mucosal ischaemia), use of NSAIDS (causes mucosal breakdown) and risk decreases with the use of proton pump inhibitors. The exact aetiology of marginal ulcers is not clear, but the possible mechanisms proposed are increased acid production in an oversized pouch, ischaemia of the pouch due to tension on roux limb, presence of *Helicobacter pylori* infection, staple line disruption, and presence of suture material within the pouch [42]. The use of absorbable suture instead of permanent suture was found to significantly reduce the incidence of postoperative marginal ulcers [43] (Fig. 7.4).

7.5.1 Diagnosis

Patients with marginal ulcers usually present with abdominal pain (especially post prandial), nausea and vomiting, and in extreme cases with haematemesis or malena

Table 7.3 Incidence of marginal ulcers after OAGB

OAGB series	Percentage of marginal ulcer reported (%)	Surgical revisions
Musella et al. n-974	1.43%	4
Carbajo et al. n-1200	0.5%	0
Chevallier et al. n-1000	2%	2
Lee et al. n-1163	NA	7
Kular et al. n-1054	0.47%	0

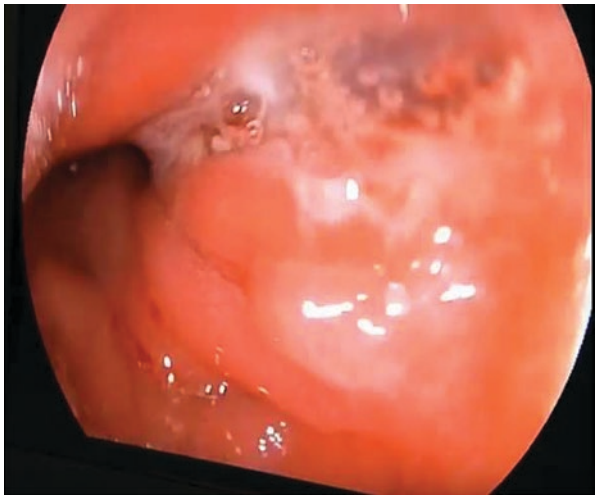

Fig. 7.4 Endoscopic image of marginal ulcer after 1 year of RYGB. Ulcer is at 2 o'clock position

or even perforation [44]. Patel et al. reported abdominal pain (66.6%) as the most common presentation of marginal ulcers [45]. Upper GI endoscopy is the diagnostic study of choice and biopsies should be taken to evaluate for *H. pylori* during same sitting [46].

7.5.2 Management

Treatment depends on the aetiology of marginal ulcer (Fig. 7.5). For smokers, smoking cessation is imperative and in patients on NSAIDs, they should be stopped. The use of proton pump inhibitors in the immediate post-operative period, for the first 3–6 months, is critical from a prophylactic perspective. However, there is no consensus about the exact duration of usage. In the literature duration of post-operative PPI, administration ranges from 30 days to 2 years [47]. In our practice, we prescribe PPI for 3 months and then subsequently on the basis of symptoms. For a patient with documented marginal ulcer either by symptoms or on endoscopy, initial treatment includes the use of proton pump inhibitor and sucralfate suspension (1 g oral liquid/6 h) for a period of 3–6 months. For comprehensive therapy, urea breath test, serology, or endoscopic biopsies should be performed for *H. pylori* and if found to be positive medical eradication should be considered using two antibiotics and a proton pump inhibitor popularly known as triple regimen [48]. If ulcer is left untreated or persists despite medical treatment, it may lead to stricture formation and ultimately gastric outlet obstruction, which may require multiple endoscopic dilatations [49]. Thus, it is of utmost importance to assess whether the ulcer is responding to medical treatment and has evidence of healing on repeat endoscopy.

Surgical intervention is required in case of failure of medical management or if ulcer leads to perforation. The operative approach includes excision of the ulcer and revision of the gastrojejunal (G-J) anastomosis, reduction in the size of gastric pouch if it is oversized. If the ulcer is associated with suture material, it should be

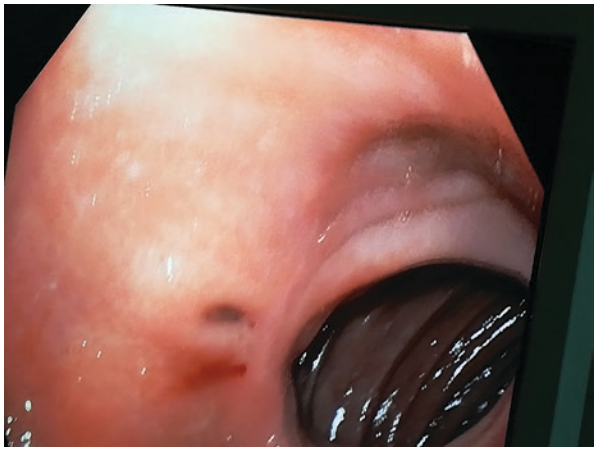

Fig. 7.5 Marginal ulcer in a female, non-smoker asymptomatic post MGB-OAGB patient

removed and the roux limb should be mobilized if there is excess tension leading to mucosal ischaemia [50].

7.6 Diarrhoea

Up to 75% of patients report a change in bowel habits after bariatric surgery. Diarrhoea is more common after bypass procedures and is more predominant when there is a short common absorptive channel. This accounts for the more predominance of diarrhoea in distal RYGB as compared to proximal RYGB and Bilio-Pancreatic Diversion (BPD) with a short common absorptive channel.

Diarrhoea can be secondary to many causes—decreased intestinal transit time, dumping syndrome, and small intestinal bacterial over-growth (SIBO.) Rare cases of gastro-colic fistulas have also been implicated in diarrhoea.

Most bypass patients report episodes of 3–4 bowel motions 2–3 times a year. Most of these episodes subside with diet alteration, fibre supplements, and anti-motility medications. If associated with steatorrhoea, pancreatic enzyme supplementation should be given. Associated symptoms like palpitation, dizziness, fainting should prompt for evaluation of dumping syndrome and should be treated with small non-sugary meals with a high-fibre content and avoidance of water with food. Non-responding foul-smelling diarrhoea should arise the suspicion of SIBO and should be treated accordingly.

7.7 Steatorrhoea and Pancreatic Exocrine Insufficiency

Steatorrhoea can occur both after SG and bypass surgeries. The prevalence after proximal and distal RYGB is around 19% and 48%, respectively. The causes include changes in the food and caloric content, an altered pancreatic response to the food, shorter time of contact of chyme with food due to bypass, degradation of the enzymes in Bilio-Pancreatic (BP) limb due to absence of food. The actual secretion from the pancreas may or may not be altered. Factors primarily decreasing the pancreatic output include decreased level of gastrin, cholecystokinin (CCK), and pancreatic polypeptide (PP) due to gastric resection, exclusion of the duodenum, and vagal nerve damage during dissection at the time of pouch formation. The primary stimulus for pancreatic secretion is a decrease in duodenal pH post meals. Decrease in the amount of acid reaching the duodenum de to decreased stomach volume (SG) and bypass of duodenum (RYGB) occurs after bariatric surgery. Additionally, ileal infusion of nutrients as in distal RYGB can decrease the pancreatic enzyme secretion. Rarely, some patients who develop non-insulinoma pancreatic hypersecretion (nesidioblastosis) and are forced to undergo pancreatectomy can develop primary pancreatic insufficiency. Symptoms of pancreatic insufficiency include steatorrhoea, borborygmi post meals, and weight loss. However, these symptoms are non-specific in a post-bariatric patient. Pancreatic insufficiency is defined by symptoms and faecal pancreatic elastase-1 (PE-1) <200 μg/g stool or a faecal PE-1 between 200 and 500 μg/g stool and the patient has benefited from pancreatic enzyme

replacement [51]. Direct stimulation testing can be done by intubating the duodenum and measuring the pancreatic response to CCK or secretin. This is technically not feasible in RYGB patients. Measurement of faecal elastase is easier, however, patients with a normal value can still suffer from pancreatic insufficiency due to indirect factors discussed above. Regardless, pancreatic enzyme replacement therapy is the treatment of choice. It is important to remove the acid protective coating over the supplements for better absorption as the acid levels in stomach post-bariatric surgery are usually lower [52].

7.8 Constipation

Bowel habits after bariatric surgery is an important issue for the patient and affect their quality of life. Many obese individuals have a pre-existing bowel disturbance. In a systematic review, it was observed that the rates of faecal incontinence and diarrhoea were higher in obese patients compared with non-obese patients. Constipation rates were similar [53].

Bariatric and metabolic surgeries have been claimed to have a diverse effect on bowel habits. Some authors have reported higher prevalence of diarrhoea after RYGB and Bilio-Pancreatic Diversion-Duodenal Switch (BPD-DS) and constipation after adjustable gastric band (AGB) and sleeve gastrectomy [54, 55].

Other authors on the other hand have reported resolution of loose stools after RYGB [56].

These contrasting findings may be the result of heterogeneity of patient cohort, food habits, assessment tools, and procedure variations. Afshar et al. [57] studied bariatric patients prior and after their surgery with a validated food frequency questionnaire and 7 day Bristol Stool Form Scale that has been validated and found to correlate with whole gut transit time. RYGB, sleeve gastrectomy, and intra-gastric balloon were assessed. They found that the frequency of stool decreased and stools were more formed at a median follow-up of 6.4 months after surgery.

This increased rate of constipation after bariatric surgery may be explained by the decrease in dietary fibre [57] and increase in GI hormones such as Glucagon-Like Peptide-1 (GLP-1) and Polypeptide YY (PYY) seen as an effect after surgery. Based on the literature and our own experience, we may opine that constipation is a frequent complaint of the bariatric patient. Clinicians involved in the care of bariatric patients should be aware of these findings and treat constipation as and when required with laxatives. They should also encourage patients to increase the fibre content in the diet. Paying attention to these details may go a long way in improving the quality of life of these patients.

7.9 Dumping Syndrome

Dumping syndrome was first described by Hertz in 1913, correlating the symptoms with accelerated gastric emptying and the term “dumping” was coined by Mix in 1922 [58]. It is the most common syndrome that may occur after any form of gastrectomy

or damage to vagus nerve during upper gastrointestinal (GI) surgeries. Dumping may occur in sleeve gastrectomy but is more common after gastric bypass surgery and may cause both gastrointestinal and autonomic symptoms. It occurs when hyperosmolar undigested food gets directly "dumped" from stomach pouch into the small intestine without being properly digested. Post RYGB, the prevalence of dumping syndrome is around 13% with a median follow-up of 4.5 years and is more commonly found in young females [59]. There are two types of dumping syndrome: Early and Late.

Early dumping occurs 10–30 min after a meal and is characterized by gastrointestinal symptoms such as nausea, vomiting, bloating, cramping abdominal pain, diarrhoea, dizziness, and fatigue. It is caused by shift of osmotically driven fluid from the blood vessels to the gastrointestinal lumen. Late dumping occurs 1–3 h after a meal and is characterized by autonomic symptoms like weakness, sweating, and dizziness due to rebound hypoglycaemia when insulin surge overcompensates for the glucose load delivered to the portal circulation, and the blood glucose level falls precipitously. Dumping syndrome leads to desired behaviour modification post RYGB that prevents individuals from consuming calorie-dense foods and thereby contributes to weight loss [60].

7.9.1 Diagnosis

a) History and clinical examination: Importance of proper history and evaluation of signs and symptoms cannot be overlooked and it gives the first clue to the diagnosis.
b) Oral glucose tolerance test: 50 g of glucose is given in water. Blood sugar, haematocrit, and the pulse are then recorded at 30 min intervals for 3 h. The diagnosis is confirmed if there is initial hyperglycaemia followed by hypoglycaemia (<60 g/dl or 3.33 mmol/l) [61].
c) Gastric emptying test: A radiotracer material is added to food to assess how quickly food moves through the stomach.
d) Diagnostic Questionnaires: Sigstad Score Scale (Table 7.4) and the Arts Dumping Questionnaire can be used to identify clinically significant symptoms. Sigstad scores ≥7, after glucose intake, is considered diagnostic of dumping syndrome [62].

Treatment of dumping syndrome is based on delaying the gastric emptying time. The symptoms of early dumping are likely to resolve on its own in three months but if they are troublesome and affecting the quality of life then treatment includes:

1. Lifestyle and dietary modification such as:
   - Eating smaller meals—Try to eat five or six small meals a day instead of three large meals.
   - Avoiding fluids with meals—Drink liquids either 30 min before meals or 30 min after meals.
   - Dietary changes—Eat more protein and complex carbohydrates rather than simple sugars. Limit high-sugar foods, such as candy, table sugar, syrup, sodas, and juices.

Table 7.4 Sigstad score

Pre-shock or shock	+5
Loss of consciousness, fainting	+4
Will lie down or sit	+4
Dyspnoea	+3
Physical fatigue, exhaustion	+3
Sleep, listlessness, blurred vision	+3
Palpitation	+3
Restlessness, agitation	+2
Dizziness, vertigo	+2
Headache	+1
Feeling hot, sweating, pallor, clammy skin	+1
Nausea	+1
Abdominal distension	+1
Borborygmi	+1
Eructation	−1
Vomiting	−4

A score of >7 is suggestive of dumping syndrome. A score less than <4 is suggestive of an alternate diagnosis

- Increasing fibre intake—Psyllium husk, guar gum, and pectin in food delay the absorption of carbohydrates in the small intestine and also prolongs gastric emptying time.

2. Pharmacological management:
   - Acarbose (alpha-glucosidase inhibitor)—Can be used to delay the digestion of carbohydrates, but its use is limited due to lack of efficacy and occurrence of side effects such as flatulence and diarrhoea.
   - Octreotide (somatostatin analogues)—Acts by slowing the emptying of food into the intestine. It can be administered subcutaneously (three times daily) for early symptoms or intramuscularly every 2 or 4 weeks for late symptoms. Possible side effects include nausea, vomiting, and stomach upset [61].
3. Surgical management:
   - Surgical treatment is reserved only for severely affected patients, with intense and disabling symptoms, not resolved by the above measures. Possible options include reconstruction of a gastric reservoir, adding restrictive intervention like band, insertion of a short anti-peristaltic loop. The last resort is the reversal of the operation [63].

7.10 Small Intestinal Bacterial Overgrowth

Small intestinal bacterial overgrowth (SIBO) is defined as an excessive number of bacteria (>10^5 CFU/ml) in the small bowel. The incidence is 2.5% in a healthy population; however, it increases up to 41% in patients with obesity due to impaired small intestinal motility. SIBO can also occur in patients post-gastric bypass sugery [51]. SIBO is associated with bypass procedures and is not related to weight loss as the rate of bacterial overgrowth is similar both before and after the restrictive

procedures. The symptoms may be diarrhoea, malabsorption, pain abdomen, obstruction, or extra -digestive complaints (arthritis, dermatologic abnormalities). Studies have shown bacterial overgrowth in both the pouch and the remnant stomach after RYGB. Hypotheses for development of SIBO after gastric bypass are alteration of the anatomy, influence of proton pump inhibitors, modifications in the caloric intake, and dietary composition. The commonly used tests for the diagnosis of SIBO include hydrogen breath test with the gold standard being endoscopic sampling of the bowel yielding bacterial count $>10^5$ CFU/ml. In the breath test, the patient is given lactulose that is fermented and the hydrogen in breath is used as a marker. In post-gastric bypass patients, breath test is unreliable due to exclusion of the part of a bowel from normal pathway. Routine endoscopy is technically not feasible in the bypassed bowel making aspiration and culture difficult. Moreover, the consequences of SIBO in post-bariatric patients are of questionable value. RYGB patients with and without SIBO report a similar percentage of digestive symptoms making the clinical significance of SIBO uncertain. The nutrients which escape digestion in the bowel due to SIBO can get fermented to a short- and medium-chain fatty acids in the colon and can theoretically increase the caloric uptake. However, studies have failed to prove such hypothesis.

An infection of clinical significance is *Clostridium difficile*-associated colitis. It might present in the form of protein-losing enteropathy without any signs of inflammation. The diagnosis requires measurement of *C. difficile* toxin in the stool samples. These tests need to be evaluated carefully taking into consideration the patients' symptoms to avoid treating asymptomatic carriers. The first line of treatment is probiotics with oral metronidazole. Oral treatment can be suspect in post RYGB patients due to altered anatomy and a high percentage of absorption of metronidazole in the small intestine. Intravenous metronidazole or vancomycin might be used in cases of fulminant colitis. Faecal microbiota transplant is a new treatment option but is not usually practiced.

7.11 Gallstones

The incidence of hepatobiliary complications after bariatric surgery is around 5.5 cases/1000 patient years, with biliary colic amounting to three cases/100 patient years. Rapid weight loss (>25% of original weight) following bariatric surgery is one of the main factors responsible for the formation of gall stones as it results in higher biliary cholesterol levels. Other factors include gallbladder stasis, increased production of calcium, and arachidonic acid derivative with the disturbed enterohepatic circulation. Patients can thus present with gall stones and their complications including common bile duct stones, which can be difficult to manage via ERCP due to altered anatomy (RYGB).

Prophylactic concomitant cholecystectomy could reduce the incidence of such complications; however, it has been seen that concomitant surgery increases the perioperative morbidity as compared to bariatric surgery alone. A concomitant procedure also poses a diagnostic dilemma in case a complication such as a leak is

suspected in the bariatric surgery. Moreover, it is technically difficult due to visceral obesity, large liver size, torque, and different port placement. Overall a concomitant procedure has a higher rate of post-operative minor complications with a similar rate of severe post-operative complications (Clavien Dindo ≥IIIa) and increases the operative time. Prophylactic cholecystectomy should be avoided given the lower rate of biliary symptoms after bariatric surgery. Moreover, many of the patients who develop gall stones remain asymptomatic and do not require treatment. Prophylaxis with ursodiol at a dose of 600 mg/day for 6 months can further reduce the incidence with the effect lasting until 1 year [64, 65].

7.12 Taste Alteration

The taste preferences have also been found to be altered post-operatively after bariatric surgery. It has been seen in a systematic review that there is decreased mesolimbic activation to high energy foods and decreased preference for sweet and fatty stimuli. The taste sensitivity to sweet stimuli increases resulting in a lesser intake of sweets, which might also help in persistent weight loss after bariatric surgery [66].

7.13 De Novo Food Intolerance

There are reports of development of intolerance to certain food products after bariatric surgery. In a study by Boerlage et al. [67] patients reported de novo intolerance to a median of four food items after surgery. The most commonly reported items were fried products, carbonated drinks, and cakes. Intolerance to meat was usually associated with dysphagia. Nicoletti et al. [68] have also reported red meat intolerance after gastric bypass. The patients usually present with other commonly reported GI symptoms like nausea, vomiting, and diarrhoea. No significant impacts of these alterations have been noted on the nutritional profile.

7.14 Nesidioblastosis

Nesidioblastosis, also known as non-insulinoma pancreatic hypersecretion is due to increased beta cell–trophic polypeptides or incretins, such as glucagon-like peptide 1, PYY, GIP, and Oxyntomodulin. The increased levels of these hormones lead to the hypertrophy of pancreatic beta cells. The details of this are discussed in the chapter on endocrine complications.

7.14.1 Portal and Mesenteric Vein Thrombosis

Although portal and mesenteric vein thrombosis are early complications after bariatric surgery, there have been reports of delayed portal vein thrombosis (PVT) and

mesenteric vein thrombosis (MVT) [69]. A hypercoagulable state with protein C and protein S deficiencies, oral contraceptive pill (OCP) intake, smoking, as well as chronic dehydration have been implicated. Postoperative abdominal pain associated with nausea, vomiting, fever, and leucocytosis should arouse suspicion for venous thrombosis. Treatment have been attempted with unfractionated heparin, vitamin K antagonists, low molecular weight heparin, and thrombolytics. Bowel resection and/or splenectomy may be required.

7.14.1.1 Bile Acid Malabsorption

Bile acids play an important role in the metabolic improvement post surgery. Farsenoid X receptor (FXR) and the G protein-coupled bile acid receptor (Gpbar1/TGR5) are the targets through which bile acids suppress hyperphagia and improve glucose metabolism. It has been shown that the amount of circulating bile acids increases after SG and RYGB due to diversion of bile to mid-jejunum resulting in increased secretion of GLP-1. The action of bile acids through FXR allows many diabetic patients to stop oral hypoglycaemic drugs just after surgery even when there is hardly any weight loss. Improved circulation of bile acids also results in a change in the gut microbiome.

However, bile acid malabsorption also can occur due to altered enterohepatic cycle and bile acid production. Majority of the bile acids are absorbed in the ileum. Malabsorption can stem from a variety of causes including ileal dysfunction, such as after resection, ileal dysmotility, SIBO, post cholecystectomy, change in microbiota, or changes in food consumption. It can be idiopathic also and can result in irritable bowel syndrome—diarrhoeal type as seen sometimes in post-cholecystectomy patients. Many patients with obesity also undergo cholecystectomy concomitantly or afterwards due to gall stones. Bile acid malabsorption might be more common in patients with short common channel (distal RYGB or BPD), however, studies to support this are lacking. The diagnostic tests include faecal bile acid determination, radiolabelled Selenium homotaurocholic acid or the serum-C4-concentration measurement. These are expensive tests and difficult to apply in clinical practice. Treatment is relatively simpler with cholestyramine—a raisin that binds bile acids with up to 95% response rate [51].

7.14.1.2 Short Bowel Syndrome

Short bowel syndrome (SBS) is defined by lack of absorptive surface resulting in failure of intestine to absorb macro- and micronutrients to fulfil the demands of the body. In the most commonly performed RYGB with alimentary limb (AL) of 100–150 cm and a Bilio-Pancreatic (BP) limb of 40–80 cm, the common channel is not measured. In OAGB also the common channel is not measured generally. Moreover, there is a constant debate regarding optimal limb lengths. The major causes of SBS post-bariatric surgery include intestinal herniation through the mesenteric defect and mesenteric thrombosis. There is no consensus on the routine closure of the mesenteric defect. There should be a high index of suspicion (pain out of proportion to the expected) for intestinal herniation and a low threshold for diagnostic laparoscopy in such cases. Mesenteric venous thrombosis is a rare cause with suspected etiologies being splanchnic blood flow alteration during bariatric surgery

and reverse Trendelenberg position. Routine anticoagulation is usually advised post-bariatric surgery. Overall, SBS occurs in up to 4% of the patients post-bariatric surgery [70]. This might be averted by routine measurement of whole of the bowel length, but this also predisposes to the risk of iatrogenic bowel injury. Nonetheless, the initial treatment consists of conservative measures including parentral nutrition; surgical options are lengthening of the common channel, enteral nutrition via gastrostomy tube in the remnant stomach, and the last resort is restoration of normal anatomy or intestinal transplantation. Such patients also might have associated liver disease and require a liver inclusive intestinal transplant. The waiting list mortality for isolated intestinal transplant is lower as compared to combined transplant due to absence of liver disease in the former group. Isolated liver is another option in patients whom intestinal failure is reversible, and the transplant is required for liver disease. To summarize, SBS is a devastating complication requiring multidisciplinary approach. The initial treatment includes medical and surgical techniques to lengthen the bowel. Transplantation is a last resort in such patients.

Key Points

- The late GI complications after bariatric surgery are related to altered motility, bile flow alteration, reduction of gastric size, anatomical gut rearrangement and altered flow of nutrients, vagal manipulation, and enteric and adipose hormones modulation.
- Commonly seen complications are Reflux, Esophagitis, Barrett's oesophagus, Nausea, Vomiting, Marginal ulcers, Diarrhoea, Steatorrhoea, Constipation, Early Dumping syndrome, Late dumping, SIBO, Liver dysfunction, Gallstones, Alteration in taste, de novo food intolerance, and Nesidioblastosis.
- Most complications are mild and respond to changes in diet and pharmacotherapy. Some like reflux, esophagitis, gallstones, and nesidioblastosis may require surgical intervention.

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