Suboptimal Weight Loss After Bariatric Surgery: Mechanisms and Treatment Algorithms

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Contents

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Abstract

Bariatric surgery is the most effective treatment for severe obesity; however, on an individual level, weight loss is highly variable. Improvements in weight-related comorbidities following bariatric surgery are related to the amount of weight loss achieved. Therefore, it is important to maximize weight loss. Cases of insufficient weight loss after bariatric surgery include those who have never achieved a good weight loss response from surgery (primary suboptimal responder) and those who had a good initial weight loss and then experienced weight regain (secondary suboptimal responder). The etiology and biology and, therefore, treatment for these patients are different and remain one of the most challenging problems facing bariatric surgery as they are associated with failure of comorbidities resolution or recurrence of their weight-related comorbidities. Attempts have been made to determine preoperative characteristics of patients that predict postoperative weight trajectories and also the underlying driving biology. This chapter aims to summarize the current literature surrounding suboptimal weight loss and weight regain and current treatment options.

Keywords

Weight regain · Suboptimal weight loss · Bariatric surgery

Introduction

Bariatric surgery is currently the most effective treatment for people with severe obesity, resulting in marked, sustained weight loss, resolution of obesity-related comorbidities, such as type 2 diabetes mellitus (T2DM), hypertension, non-alcoholic fatty liver disease (NAFLD), and overall increased life expectancy [1, 2]. Although, on a population level, bariatric surgery is highly effective for treating obesity, with a mean total weight loss of 20–30% reported in most studies, there is a large variation on an individual level.

Maximizing postsurgical weight loss is important as there is evidence showing that long-term T2DM remission depends on the extent of weight loss sustained. Those who achieved more than 25% weight loss were more likely to achieve T2DM remission compared to those who did not [3]. Resolutions of other conditions such as hypertension, dyslipidemia, obstructive sleep apnea, and NAFLD are positively correlated with percentage weight loss following surgery [4]. More recently, meta-analysis looking at reduction of major adverse cardiovascular events (MACE) after bariatric surgery found significantly lower odds of MACE following bariatric surgery compared to no surgery [5]. Therefore, it is not surprising that greater weight loss following bariatric surgery is correlated with greater improvement in health-related quality of life [6].

The SM-BOSS randomized controlled trial (RCT) compared the total weight loss between sleeve gastrectomy (SG) and Roux-en-Y gastric bypass (RYGB), as well as presented the distribution of weight loss in all the participants. They demonstrated a subset of patients who had never achieved more than 20% weight loss (primary suboptimal responder) and others who had good initial weight loss and then regained weight subsequently (secondary suboptimal responder) [7]. Manning et al. [8] also reported a highly variable but normally distributed maximal weight loss response following RYGB and SG. The multicenter Swedish Obese Subjects (SOS) study [9], a large, prospective observational study comparing bariatric surgery and conservative medical treatment, showed that at 10 years, subjects regained 34% and 38% of the maximal weight loss following RYGB and SG, respectively. The Longitudinal Assessment of Bariatric Surgery study [10] reported suboptimal weight loss (20% total body weight) at 7 years in 25% of patients. However, these studies did not differentiate between those who had primary suboptimal weight loss or subsequently weight regained. This may be important due to the different underlying mechanisms responsible for primary suboptimal responders and those with weight regain (secondary suboptimal responders).

Definition of Weight Regain

Primary suboptimal weight loss and weight regain following bariatric surgery are among the main challenges in bariatric surgery. Currently, there is no reliable way of predicting the weight loss outcome for the individual patient prior to surgery, and the etiology remains poorly understood. There is a wide variation in the definition of "suboptimal weight loss" in the literature, which creates difficulty when comparing data. Many studies have used the cutoff less than 50% excess body weight loss as a definition of suboptimal weight loss [11]. However, this increases the likelihood of suboptimal weight loss in those with higher starting weight as excess body weight is calculated using the "ideal" body mass index (BMI) of 25 kg/m^2. Percentage total body weight loss (%WL) has been shown to be a better measure for weight loss outcomes as it is less influenced by preoperative BMI [12]. The threshold for "suboptimal weight loss" has not been universally defined. However, some authors have described a less than 20% WL as suboptimal weight loss from bariatric surgery as it is correlated with re-emergence of weight-related comorbidities [8, 13].

Weight regain following bariatric surgery is defined as progressive weight regain that occurs after the achievement of an initially successful weight loss. Definitions used include calculations based on percentage weight increase from nadir (>25% of total lost weight [14]), 25% percentage excess weight loss from nadir weight [15], and >50% excess weight loss from nadir [16].

A recent systematic review reporting definitions of primary and secondary suboptimal responders showed that out of 112 papers that reported weight loss failure only 42 described a definition, with 23 different definitions in total, whilst weight regain was mentioned in 77 papers. However, only 21 provided a definition. The lack of standardization in the definition of suboptimal weight loss from bariatric surgery means that data on the prevalence of suboptimal weight loss and weight regain is unknown. There is a need for standardizing the reporting of both primary and secondary suboptimal responders.

Mechanisms of Weight Regain

Several mechanisms and risk factors have been studied and found to contribute to weight regain following bariatric surgery. These are summarized in Fig. 1 and Table 1.

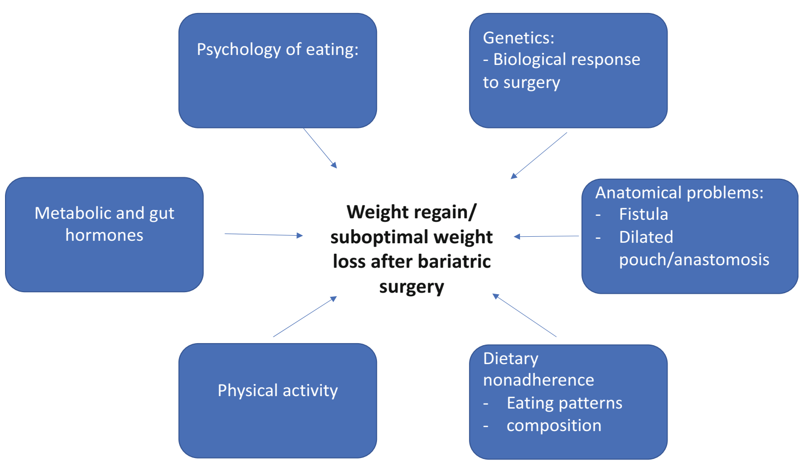

Fig. 1 Causes of poor weight loss outcome following bariatric surgery

Table 1 Etiology of weight regain and suboptimal weight loss following bariatric surgery

Mechanisms	Evidence
Genetics	Presence of obesity-associated polymorphism FTO rs16945088 minor allele presence
Gut hormones	Lower levels of glucagon-like peptide 1 and peptide YY in patients with poor weight loss following roux-en-Y gastric bypass (RYGB) Higher ghrelin pre- and postop in patients who had poor weight loss following RYGB
Physical activity	Sedentary behavior, presence of barriers to exercise
Dietary noncompliance	Increase caloric intake with time, change in composition and macronutrient, grazing, lack of dietary follow-up
Mental health	Depression, presence of maladaptive eating patterns: Binge eating disorder, loss of control of eating
Anatomical factors	Dilatation of gastric sleeve in sleeve gastrectomy Dilatation of gastric pouch, dilatation of gastrojejunostomy stoma outlet and presence of gastro-gastric fistula in RYGB
Risk factors	Increase age, high preoperative body mass index, presence of type 2 diabetes mellitus, lower socioeconomic status, lack of social support
Medications	Antipsychotics and mood stabilizers (clozapine, haloperidol, lithium, olanzapine, quetiapine, risperidone)[a] Antidepressants (mirtazapine, citalopram, amitriptyline)[a] Antihyperglycemics (insulin, gliclazide, glimepiride, pioglitazone)[a] Antihypertensives (atenolol)[a] Others: prednisolone[a]

[a]All medications listed associated with >3 kg weight change [75]

Genetics

The etiology of obesity is complex and influenced by multiple factors, including genetics, medications, environment, and social deprivation. Genome-wide association and linkage studies have identified multiple susceptibility foci for obesity. The high degree of variability in weight loss following bariatric surgery may be due to a genetic predisposition to resist weight loss. Twins experimental studies demonstrated high variability in weight loss following a very low-calorie diet between pairs of identical twins but not within pairs of identical twins [17]. The SOS study found that the minor allele at the FTO rs16945088 locus was associated with less weight loss after bariatric surgery. They found that the G-allele carriers achieved 4.1 kg less weight loss compared to common allele homozygotes (A/A). A study of 77 patients who underwent laparoscopic gastric band (LAGB) and 277 who had one anastomosis gastric bypass (OAGB) found that the UCP2 rs660339 was associated with higher weight loss in the T allele carriers compared to the CC genotype (difference of 4 BMI units at 12 months) [18]. However, there was no difference between genotypes following OAGB group. Hatoum et al. [19] found that first-degree relative pairs had a similar weight loss response to RYGB, which was not seen with cohabitating individuals and unrelated individuals. Gerhard and colleagues [20] reported high allelic burden from multiple obesity-associated polymorphisms may be associated with less weight loss after RYGB. These data demonstrate that genetic factors potentially explain a significant portion of the variation in weight loss after bariatric surgery, which is also dependent on the surgery type.

Gut Hormones

Obesity occurs when energy intake chronically exceeds energy expenditure. By consistently overriding homeostatic signals of energy availability, eating becomes disjointed from energy requirements and results in dysregulation of the metabolic mechanisms controlling energy homeostasis, including impaired gut hormone secretion (Table 2). People living with obesity have been found to have a blunted ghrelin reduction post-meal together with reduced levels of anorectic gut

Table 2 Gut hormone and hunger response changes following bariatric surgery and in patients with suboptimal weight loss or weight regain

Gut hormones	Person with obesity	Changes after bariatric surgery	Changes seen in patients with suboptimal weight loss after bariatric surgery
Glucagon-like peptide 1 (GLP1)	Blunted postprandial response	Significant increase in postprandial GLP1	Blunted postprandial GLP1 response compared to patients with good weight loss
Ghrelin	High ghrelin	Significant reduction in both fasting and postprandial ghrelin	Higher ghrelin levels compared to those weight good weight loss
Peptide YY (PYY)	Blunted postprandial response	Significant increase in postprandial PYY	Blunted postprandial PYY compared to good weight loss
Perceived hunger	Increase hunger compared to person without obesity	Lower perceived hunger following surgery	Higher perceived hunger compared to those with good weight loss
Perceived satiety	Reduction in perceived satiety compared to person without obesity	Increased perceived satiety	Not reported

hormones, peptide YY (PYY), glucagon-like peptide 1 (GLP-1), and neurotensin (NT), compared to individuals with normal weight people. Following bariatric procedures such as RYGB and SG, patients report a marked change in eating behavior, with reduced appetite and changes in taste and food. This is associated with a dramatic increase in nutrient-stimulated circulating levels of several anorectic hormones, including PYY and GLP-1, following both RYGB and SG, as well as a reduction in ghrelin with more marked changes following SG. These gut hormone changes are thought to play a key role in mediating changes in appetite, eating behavior, and sustained weight loss after SG and RYGB.

Cross-sectional studies investigating the role of gut hormones in primary suboptimal weight loss and weight regain after bariatric surgery have shown differential gut hormone response following bariatric surgery, with some patients experiencing marked changes whilst others remained to have blunted satiety hormones even after surgery [21]. Santo et al. [21] studied gut hormone profiles in patients who had RYGB with good weight loss compared to those who had weight regain of >50% of the weight lost. They found higher postprandial glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 in patients with good weight loss compared to those with weight regain but no difference in ghrelin levels. Dirksen et al. [21, 22] investigated patients with good weight loss compared to those with primary suboptimal response at 1 year after RYGB. They found lower levels of postprandial GLP-1 in patients who had suboptimal weight as well as blunted postprandial ghrelin suppression. In terms of ghrelin, one study found higher levels of ghrelin pre- and postoperatively were associated with lower weight regain after RYGB [23]. Similar findings were observed with SG, where slightly higher levels of ghrelin were found in patients with weight regain, although not statistically significant [24].

This blunted gut hormone response seen in some patients who have primary suboptimal weight loss may point to underlying genetic determinants to the success of bariatric surgery.

There is a lack of longitudinal data on gut hormone profiles to fully understand how these changes evolve with time and whether there are differences in patients with primary suboptimal weight loss versus those with weight regain. Research is needed to establish whether hormonal response can be predicted pre-surgery to optimize selection of patients for surgery.

Physical Activity

During rapid weight loss following bariatric surgery, there is a decrease in muscle mass. However, a recent study has suggested that muscle strength is improved postoperatively despite the reduction in muscle mass [25]. A number of studies have reported a positive relationship between physical activity levels and weight loss. Higher preoperative levels of physical activities have also been found to have higher postoperative physical activity. In terms of outcomes, it is not surprising that patients who performed some physical activity had a lower incidence of weight regain when compared to sedentary patients [26]. Therefore, it may be prudent to encourage an increase in physical activity preoperatively to remove the barriers to physical activity prior to surgery.

Dietary Nonadherence

Several studies have reported poor postoperative adherence to dietary recommendations being associated with a gradual increase in caloric intake during the postoperative period and poorer weight loss outcomes. Brolin et al. found that in the postoperative period following RYGB, patients consumed from 890 +/− 407 kcal/day at 6 months postop to 1386 +/− 578 kcal/day after 2.5 years. The SOS study also found that following bariatric surgery mean energy intake gradually increased over a period of 10 years, contributing to weight regain. Poorer food quality, an increased in excess snacks and sweets, as well as oils and fatty food, was associated with higher weight regain after bariatric surgery reported in a study by Freire et al. [26]. The importance of nutritional counseling on long-term weight maintenance has been demonstrated with studies showing lack of appropriate nutritional follow-up was significantly associated with weight regain after surgery [27].

Maladaptive Eating Behaviors

Binge eating disorder (BED) is common in the bariatric surgery populations, with a prevalence of 5–49%, depending on the definition used for BED. Maladaptive eating patterns (including disinhibited, grazing, nocturnal eating, emotional eating, and loss of control of eating) have been linked with increased caloric intake and poorer weight loss outcomes following bariatric surgery, although the results are inconsistent [28]. However, the presence of loss of control (LOC) of eating following bariatric surgery has been consistently shown to correlate with poorer weight loss outcomes [29–31]. LOC of eating is defined as a subjective sense of loss of control while eating or difficulty stopping eating regardless of the amount consumed. Conceicao et al. [29] reported 39.5% of patients developed new maladaptive eating patterns following bariatric surgery, which predicted poorer postoperative weight loss outcomes, whilst the presence of preoperative maladaptive eating patterns did not correlate with postoperative weight loss. These eating patterns include picking and nibbling, frequency of which increased overtime postoperatively. It is not clear why some patients develop these maladaptive eating patterns following surgery. However, insight into the cause of this may provide future treatment targets.

Anatomical or Surgical Factors

Surgical techniques have been explored as potential causes. However, results are conflicting, and they have not shown any conclusive evidence influencing differential weight loss. Factors such as different limb lengths in RYGB and the size of the calibration tube used for SG have all shown no difference in weight loss, as evidenced by well-designed RCTs [32–34]. Heneghan et al. [35] evaluated the size of gastric pouch using endoscopy following RYGB in good weight loss ($n = 175$) versus suboptimal weight loss patients ($n = 205$) and found no difference. A similar study using barium swallow as an evaluation tool also found no difference [36]. The presence of a gastro-gastric fistula, a communication between the gastric pouch and remnant stomach, is uncommon, reported at 1.2% in a series of 1292 consecutive cases from one center, and is treated with transection of the fistula, which restores weight loss [37]. These typically present with more rapid weight regain.

Moreover, large series reported from single centers, using the same surgical techniques, have consistently shown a suboptimal weight loss rate of around 20–25% [38]. Therefore, there is little evidence to suggest surgical techniques influence weight loss variability. Postoperatively, anatomical changes are found after both SG and RYGB, and some have attributed these to increase calorie intake and weight regain. In patients who had SG, dilatation of the gastric sleeve has been correlated with postoperative BMI, where dilatation leads to an increase in food intake and, therefore, reduction in satiety and subsequent weight regain [39]. Studies have shown a gradual increase in gastric volume 5 years after SG, which may contribute to weight regain [40]. Following RYGB, dilatation of the gastric pouch or gastrojejunostomy (GJ) stoma outlet is well recognized and has been shown to correlate with weight regain [35, 41]. Stoma size of more than 2 cm was associated independently with weight regain [35], which was found to be present in 58.9% of patients who had weight regain [41].

Preoperative Risk Factors

Although the mechanisms of suboptimal weight loss remain poorly understood, risk factors for suboptimal weight loss following bariatric surgery have been identified. Demographic and patient factors, including increasing age, greater initial BMI, presence of T2DM and liver fibrosis, lower socioeconomic status and lower educational level, unemployment, and lack of social support, have been identified to be associated with poorer weight loss response [42–45].

There has been a lot of focus on preoperative psychological predictors in the literature, with studies postulating the effects of preoperative eating disorders on weight loss after bariatric surgery. However, a recent meta-analysis, which included 19 studies, reported no correlation between the preoperative presence of BED and weight loss after bariatric surgery [46].

Management Options

Management of weight loss following bariatric surgery requires a multidisciplinary approach with a comprehensive assessment of dietary patterns, physical activity levels, psychological factors, medications, and personal factors. This is summarized in Table 3.

Prevention and Early Identification

Manning et al. [8]showed that slow early weight loss from SG or RYGB (at 6 weeks) was associated with lower overall total body weight loss. Therefore, early weight loss can be used to identify those predicted to have suboptimal weight loss trajectories. Early identification of "poor responders" from surgery is important so that dietary and physical activity recommendations can be further reinforced with a more intense postoperative lifestyle and behavioral support to enhance weight loss response. Centile charts have been published to be used in order to support people with poor weight loss from 12 weeks and then later to identify those with weight regain [47]. Early interventions with multidisciplinary

Table 3 Treatment options

Type	Intervention
Behavioral therapy	Ten weeks cognitive-based therapy, remote acceptance-based therapy, mindfulness eating techniques
Dietary	Structured dietary counseling, protein-based structured dietary intake
Pharmacotherapy	FDA-approved: orlistat, phentermine, liraglutide 3.0 mg, semaglutide 2.4 mg, bupropion/naltrexone, topiramate
Surgical	Revisional surgery: Sleeve gastrectomy (SG): Re-sleeve, conversion to Roux-en-Y gastric bypass (RYGB), OAGB, BPD/DS, SADI RYGB: distalization, revision to banded gastric bypass
Endoscopic approach	RYGB: transoral outlet reduction using endoscopic suture +/− argon plasma coagulation, sclerotherapy SG: endoscopy sleeve gastroplasty

team support are important in improving postoperative weight loss [48]. Interventions such as enrolling in physical activity programs have shown to be helpful for weight maintenance. Those who have not previously been physically active could get support from exercise therapists. Dietary interventions such as food diaries to record daily intake could be helpful in regular self-monitoring [49]. Several studies have looked at postoperative dietary interventions. An RCT reviewed post-RYGB patients that had a dietary intervention versus standard care and found that they had significantly greater weight loss and lower portion sizes [49].

Behavioral Therapy

Psychological interventions are well established in the treatment of eating pathologies such as BED and LOC. The aim of behavioral therapy is to enable patients to identify maladaptive eating patterns so that they have the skills and mechanisms to change these maladaptive eating patterns. Cognitive behavioral therapy is the most widely studied and consists of self-monitoring, dietary change, and cognitive restructuring to promote healthy eating patterns and change maladaptive eating attitudes [50]. In bariatric surgery, behavioral interventions have been shown to have an impact on maladaptive eating patterns, demonstrated in a systematic review that included six RCTs of postoperative behavioral intervention and their impact on weight loss [51]. One study used a 10-week behavioral intervention for patients experiencing weight regain following bariatric surgery. They found that the intervention was able to stop the patients from regaining weight and helped in increasing weight loss as well as reducing maladaptive eating patterns [52]. A study using a 6-week behavioral intervention, utilizing techniques of cognitive-behavioral therapy and dialectical behavioral therapy, resulted in a reduction in weight following bariatric surgery [53]. Similarly, online and remote behavioral interventions have also shown to be feasible, acceptable, and effective at delivering similar interventions [54].

Dietary

Dietary interventions after bariatric surgery have yielded mixed results in promoting additional weight loss or preventing weight regain in the long term. One RCT demonstrated improvement in weight loss using a comprehensive nutritional and lifestyle education program 6 months following bariatric surgery compared to the usual group. They received group education sessions every other week for 6 weeks [49]. Another study used a structured dietary intervention that used portion-controlled foods compared to the usual care. This RCT found a significant reduction in calorie intake at 4 months and increased weight loss compared to the usual group [55]. Gomes et al. [56] evaluated the effect of whey protein supplementation combined with a low-calorie diet on the weight and body composition of women with weight regain after RYGB. They observed a greater weight and fat mass loss compared to the control group. Dietary interventions that combine the use of a high protein diet and exercise may further improve weight loss and loss of fat mass whilst preserving fat-free mass [57]. However, more studies need to be done to confirm this.

Pharmacotherapy

Insights from the biological changes that followed bariatric surgery have led to the discovery of new targets for pharmacotherapy for obesity. Currently available antiobesity medications are summarized in Table 3. There are multiple publications showing that adding pharmacotherapy can lead to similar weight loss in patients after surgery compared to patients who have not undergone surgery.

GLP-1 analogs have an established role in the management of T2DM, with weight loss being an added benefit [58]. The SCALE trial showed that higher dose formula, such as liraglutide 3.0 mg once a day subcutaneous injections, was effective at inducing more than 5% weight loss in 63% of participants and more than 10% weight loss achieved in 33% of participants [59]. The use of liraglutide as a treatment for patients with poor weight loss and weight regain following bariatric surgery has been reported in cohort studies that were found to induce significant %WL following both RYGB and SG. However, the proportion of patients who attained more than 5% weight loss was lower than those in the clinical trials of patients who have not had bariatric surgery (38% compared to 63%) [60]. Suliman et al. [61] compared the use of liraglutide 3.0 mg in people with obesity and patients who had undergone bariatric surgery. Weight loss achieved was similar in those who had bariatric surgery compared to those who had not. There is one RCT that used liraglutide 1.8 mg in post-bariatric surgery patients who had failed to achieve remission of T2DM from bariatric surgery. In addition, to an improvement in glycemic control, they also found a reduction in body weight of >5% in 50% of participants who were randomized to liraglutide 1.8 mg when compared to placebo [62]. Drawing from the evidence that some patients with poor weight loss exhibit lower GLP-1 response, a targeted pharmacotherapy approach to those with suboptimal GLP-1 response may increase the proportion of patients achieving >5% WL from GLP-1 receptor agonists. Until recently, pharmacotherapy for the treatment of obesity has had limited efficacy. However, there are newer medications that have achieved a higher %WL in people who have not had bariatric surgery. Semaglutide is a once-weekly GLP-1 receptor agonist that has been shown to induce an average weight loss of 14.9% in people with obesity without T2DM (STEP1 trial). This has not been studied in patients who have undergone bariatric surgery with weight reduction. There are multiple treatments in development based on gut hormones targeting amylin, PYY, gastric inhibitory peptide (GIP), growth differentiator factor 15, ghrelin, and fibroblast growth factor. Combination therapy has shown promising results and is likely to be more efficacious in inducing a larger degree of weight loss and playing an important role in the management of weight regain after bariatric surgery. Tirzepatide, a novel dual GLP1 and GIP analog, is being investigated at weekly subcutaneous doses for patients with T2DM in the SURPASS-2 phase 3 clinical trial compared to semaglutide 1.0 mg. This showed dose-dependent, greater reductions in body weight in all three doses of tirzepatide (5 mg dose led to 8.2% body weight loss, 10 mg dose led to 9.3%, and 15 mg led to 11.9% body weight loss) compared to semaglutide 1.0 mg (6.1% body weight loss) [63]. Understanding the gut hormone changes that may be driving suboptimal weight loss and weight regain after bariatric surgery may be key to providing targeted treatment in order to achieve best outcome for the patients.

The optimal timing of starting adjuvant pharmacotherapy is still being evaluated. The United States has reported a number of studies where they have used adjuvant pharmacotherapy much more widely. They have found that patients achieve a higher total weight loss compared to their preoperative level if they were prescribed medications at their weight plateau following bariatric surgery rather than after weight regain has started [64]. Therefore, early identification of those at risk of weight regain and suboptimal weight loss is important for timely administration of adjuvant pharmacotherapy.

Surgical Intervention

Revisional surgery after primary bariatric surgery for weight regain has been widely examined, and it is one of the most common indications worldwide for revisional surgery. The evidence for revisional surgery is discussed in detail in other parts of the book. Majority of the studies are from case series without any long-term outcomes. The %WL achieved following surgery is variable, but most reported additional weight loss following revisional surgery. There is also a lack of direct comparisons with techniques and randomized trials to understand the superiority of one technique over another. Short-term complications such as nutritional deficiencies were higher amongst those who underwent revisional surgery. A systematic review for revisional surgery after SG found that revisional rates were 22.6% after SG, with the most common reason being a failure in weight loss.

Endoscopic Treatment

Esophagogastroduodenoscopy (EGD) is an important part of the assessment of a patient with weight regain following bariatric surgery, allowing the evaluation of the altered anatomy and surgical complications such as fistula, estimation of gastric pouch size and anastomosis size, sleeve dilatation, and other complications such as ulcers.

Several studies have demonstrated a correlation between patients with larger pouch size and large diameter of GJ anastomosis being associated with weight regain following bariatric surgery [35, 65, 66]. Many centers defined enlarged GJ as >2 cm in diameter. Pouch dilation is defined as enlarged in some studies if the diameter exceeds 5 cm. Endoscopic interventions have generally been suggested to be safe and may be completed with minimal morbidity. Techniques such as sclerotherapy, argon plasma coagulation (APC), and endoscopic suturing have been used for weight regain following bariatric surgery.

Endoscopic suturing at the GJ anastomosis to reduce the gastric outlet size has been trialed since early 2000s. Reports suggest that amongst those who had suboptimal weight loss and weight regain following RYGB, enlargement at the anastomosis found on OGD following RYGB occur as often as 58.9% [41]. Thompson et al. [67] showed that endoscopic suturing was feasible to reduce the GJ anastomosis to an average of 10 mm, leading to weight loss in 75% of RYGB patients and an excess body weight loss of 23.4%. Five-year outcome data has been published by some centers showing durable total body weight loss of 8.8% following transoral outlet reduction using endoscopic suturing [68]. Both full-thickness endoscopic surgery and superficial thickness endoscopic surgery have been described. Evidence is stronger for full-thickness endoscopic suturing, with a meta-analysis showing 20% EWL at 1 year for full-thickness endoscopic suturing compared to 11% for superficial endoscopic suturing [69]. Endoscopic suturing, together with APC, has been shown to be effective and offered additional weight loss when compared to endoscopic suturing without APC [69]. APC is a non-contact electrocoagulation method in which radiofrequency energy is applied to the tissue through ionized argon gas. It produces GJ anastomosis size reduction and tissue compliance through healing following the electrocoagulation [70].

Sclerotherapy involves an injection of morrhuate sodium circumferentially around the GJ anastomosis using an endoscopic needle to reduce the size of GJ diameter and tissue compliance. This solution is injected around the rim of the GJ anastomosis in 1–2 mL aliquots with a total of 10–30 mL injected. Complications include bleeding and late perforation. Subsequently, repeat sessions are scheduled every 3 months until GJ measures less than 12 mm [71]. Loewen et al. [72] performed this on patients with weight regain and found that 66% of patients who underwent sclerotherapy were able to either maintain their weight or lose further weight, whilst 34% continued to have weight gain.

As SG is performed more widely worldwide, an endoscopic approach to improve weight loss following SG is becoming more popular. Although studies reporting its efficacy are still limited, Eid [73] published a series of five patients with dilated sleeves who underwent endoluminal plication using an endoscopic suturing device. They showed a %WL between 6.7% and 17.2% at 1 year, following the procedure. de Moura et al. [74] published a series of 34 patients who underwent endoscopic sleeve gastroplasty for weight regain after SG. The reported %WL was 13.2% at 6 months and 18.3% at 1 year. Longer-term data is lacking for these procedures, and more studies are needed to further evaluate the optimal technique, safety, and efficacy profiles of these endoscopic treatments for weight regain following SG.

An Algorithm for Management of Weight Regain Following Bariatric Surgery

The etiology for weight regain following bariatric surgery is complex and multifactorial; therefore, a comprehensive multidisciplinary approach is necessary (Fig. 2). This also includes reviewing

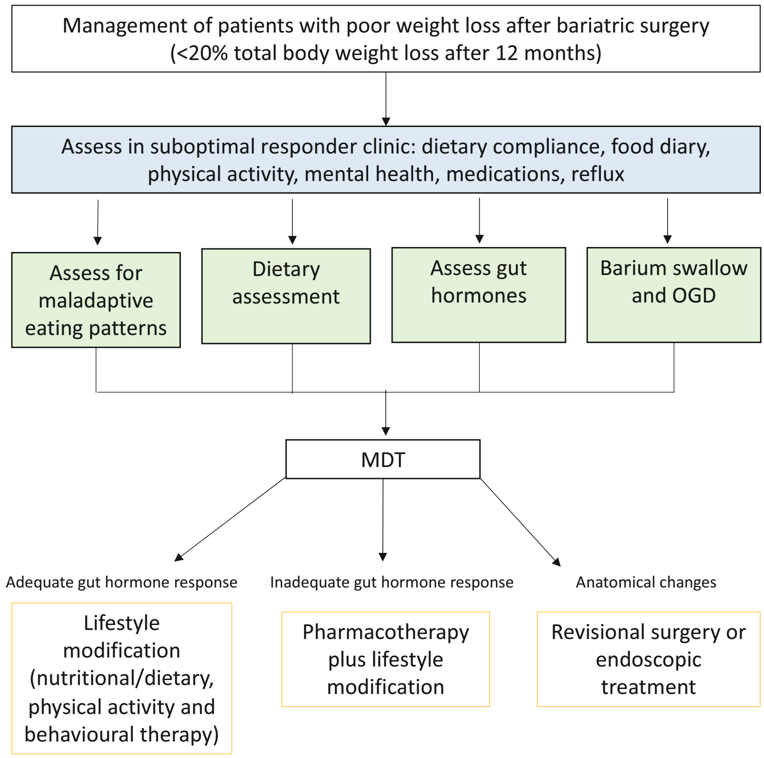

Fig. 2 An algorithm for management of suboptimal weight loss following bariatric surgery

medical comorbidities as well as medications to ensure patients are on weight-neutral medications. Identification of those who require intervention for additional support is the first step to management and having a standardized definition is prudent. Thereafter investigations will be targeted toward discussing and identifying all the potential risk factors that may be playing a role in the suboptimal weight loss and weight regain. A dedicated suboptimal weight loss clinic that offers patients a methodical, organized, and multidisciplinary plan may serve as a useful template for practitioners struggling with patients experiencing suboptimal weight loss and weight regain following bariatric surgery.

Summary

Obesity is a progressive chronic disease. Weight loss after any intervention is highly variable, and weight regain occurs after any weight-loss interventions. Following bariatric surgery, weight regain is common and often occurs 12 months after surgery. Lower weight loss early in postoperative period suggests a likelihood of a suboptimal weight loss trajectory and weight regain later in the postoperative period. Early identification and intervention and engaging patients early is likely to be more successful in treating suboptimal weight loss and weight regain to mitigate full/complete weight regain. A comprehensive approach is required with bariatric surgeons, obesity physicians, dietitians, psychologists, and exercise physiologists. Although there are a number of factors identified here that may contribute to suboptimal weight loss and weight regain, the most influential factor is likely to be genetic. Until we understand the genetic determinants of good and poor weight loss, more efforts in research should focus on first getting a universal definition for weight regain and suboptimal weight loss. Then, large series should focus on identifying factors that may influence suboptimal weight loss and weight regain so that treatment can be guided toward the cause.

Key Learning Points

- Weight regain and suboptimal weight loss following bariatric surgery are common, affecting up to one in five patients undergoing surgery.
- Standardized definition for weight regain can lead to more accurate estimates of the true prevalence.
- Early weight loss is an important indicator for long-term weight loss and weight regain, allowing for early identification of those at risk of weight regain.
- Multidisciplinary approach with a dedicated suboptimal responder clinic to fully assess the contributors for weight regain may improve outcomes so that treatment can be targeted.
- Circulating levels of the gut hormones are different in those with good weight loss versus poor weight loss after surgery. This may be useful in targeted pharmacotherapy and behavioral therapy based on gut hormone changes.

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