Gastroesophageal Reflux, Obesity, and Bariatric Surgery

Balaji Nagammapudur and Hrishikesh Salgaonkar

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Abstract

Bariatric surgery has been widely adopted as the most effective treatment modality for Obesity achieving long-term weight loss. It prevents the development and/or progression of obesity-associated chronic conditions and metabolic disorders. Obesity and its impact on a wide range of comorbidities are well known, including gastroesophageal reflux disease (GERD). GERD if left untreated, can lead to a wide array of complications ranging from esophagitis, stricture, Barrett's metaplasia, and cancer. There is accumulating evidence that obesity itself is a risk factor for longstanding GERD. It is increasingly accepted that symptoms are an unreliable marker of the presence or absence of GERD in the obese population. It should be expected that bariatric surgery

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should result in improvement in symptoms of GERD and reduce the impact of its associated complications by the achieving weight loss alone. In reality, however, different bariatric surgical procedures have varying impact on pre-existing GERD and can themselves be risk factors for the development of *de novo* GERD.

While Roux-en-Y gastric bypass (RYGB) appears to produce a beneficial effect on GERD, other procedures like sleeve gastrectomy (SG), one anastomosis gastric bypass (OAGB), and adjustable gastric banding (AGB) have been linked with increased prevalence of GERD. Adequate evaluation and patient counseling are paramount before planning surgical intervention to achieve durable weight loss and GERD resolution. It is also extremely important to explain the risk of GERD to patients undergoing refluxogenic bariatric surgery procedures and the potential long-term complications.

Keywords

GERD · Gastroesophageal reflux disease · Bariatric surgery · Obesity · Roux-en-Y gastric bypass · Sleeve gastrectomy

Introduction

Gastro-esophageal reflux disease (GERD)) is defined as symptoms and/or complications occurring as a result of reflux of gastric contents up into the esophagus or beyond [39]. Classical symptoms of GERD include retrosternal burning or heartburn and regurgitation. While atypical symptoms include bloating, belching, dysphagia, nausea, vomiting, epigastric pain, and rarely, extra-esophageal symptoms such as cough, hoarseness, and throat irritation [35, 39]. Long-term GERD may result in erosive esophagitis (EE) and Barrett's metaplasia which can progress to cancer. There is no clear correlation between the frequency and severity of symptoms with the development and progression of complications [1, 2]. GERD is relatively common and is known to affect roughly a third of the population. This incidence rises to up to 70% in patients with severe obesity, highlighting the impact on economic and healthcare resources [1, 5, 7].

Obesity itself has reached pandemic proportions worldwide, developing into a global health problem. Over the last few decades, the prevalence of obesity has almost tripled, and worldwide, roughly 650 million adults are obese. In the United States (US), roughly one-third of the population is obese [4, 9]. In the United Kingdom, the obesity prevalence has increased steeply since 1993 from 14% to 28% in 2018 as per the Health Survey for England. The same study found that in 2018, the majority of adults in England (63%) were either overweight or obese. The impact of the rise in obesity on GERD and its associated sequelae is on the rise [26]. In the US, the incidence of GERD in the general population is around 15%, while the same ranges between 22% to 70% in the obese population [1, 6]. This highlights the enormous health and socioeconomic impact on the healthcare system. Obesity remains an independent risk factor for GERD, and weight loss plays a positive role with a significant improvement seen in symptoms of GERD [20].

Pathophysiology of GERD Associated with Obesity

The precise mechanism of the lower esophageal sphincter (LES) at the gastroesophageal junction (GEJ) has been a matter of extensive investigation [38]. The anatomy and its functioning are fairly complex, involving various anti-reflux barrier mechanisms. This mainly includes, but are not solely limited to the LES, diaphragmatic crura, intra-abdominal length of the esophagus, the intra-abdominal pressure, angle of His, gastric sling fibers, and the phrenoesophageal ligament, which anchors the distal esophagus and GEJ to the diaphragm. Impairment of these mechanisms or the neuronal and hormonal control will promote GERD.

GERD can be caused by the following multifactorial mechanisms in obesity (Table 1):

Table 1 Obesity and its contributory factors to gastroesophageal reflux disease

Transient lower esophageal sphincter (LES) relaxation [relaxation of LES in absence of swallow]
Reduced LES pressure
Disruption of the normal anatomy of the gastroesophageal junction and its resultant changes at the hiatus, e.g., hiatal hernia
Gastric distention
Increased intra-abdominal pressure
Transdiaphragmatic pressure gradient [23]
Associated esophageal dysfunction [24, 37]
Contribution of hyposalivation impacting esophageal clearance [21]

- Transient LES relaxation (relaxation of LES in the absence of swallows)
- Reduced LES pressure
- Disruption of the normal anatomy of the GEJ and its resultant changes to the physiology of LES, e.g., hiatal hernia
- Gastric distention
- Increased intra-abdominal pressure
- Transdiaphragmatic pressure gradient [16]
- Associated esophageal dysfunction [12, 15]
- Contribution of hyposalivation impacting esophageal clearance [36]

Increased susceptibility of obese patients to GERD is directly related to the effect of multiple variables of obesity like increased gastric capacity (higher gastric distensibility with GEJ muscle fibers disruptions), increased intragastric pressure, increased intra-abdominal pressure, and its interplay with the negative intra-thoracic pressure [8, 14, 18]. Ayazi et al. [4], in their study, demonstrated that a mechanically defective LES and concomitant hiatal hernia became more prevalent with a rise in body mass index (BMI). Similarly, Herbella et al. [27], in their study, reported that in obese patients, for every 5-point increment in BMI, we should expect an increase of 3 units in the DeMeester score. Obesity-related hormonal changes cause irregularities in leptin and adiponectin secretion from the adipose tissue cells. This has been proposed as a possible factor between obesity and esophageal metaplasia [3, 12]. Abdominal obesity, by increasing the intra-abdominal pressure, promotes the development of hiatal hernia and/or reflux [5]. El-Serag et al. [22] in their study used computed tomography (CT) to measure and compare the abdominal fat composition in patients with EE, Barrett's esophagus (BE), and esophageal adenocarcinoma (EAC). They demonstrated that patients with BE, EE, and EAC had a greater intra-abdominal visceral adiposity as compared to control subjects. Duggan et al. [18] reported that the metabolically active visceral abdominal fat itself could predispose an individual to BE and EAC by mechanisms that are independent of GERD. They documented alterations in the levels of cytokines, chemokines, and adipokines along with interactions of insulin/insulin growth factor pathways, ultimately resulting in the progression from inflammation to metaplasia and neoplasia.

A meta-analysis by Hampel et al. [27] exploring the link between GERD and obesity demonstrated a statistically significant association between increasing BMI with progressive risk of GERD, EE, and EAC. They postulated that the mechanisms are primarily driven by raised intra-abdominal pressure and the gastroesophageal pressure gradient. Pandilfino et al. [31], in their study, subjected 285 patients to high-resolution manometry. They simultaneously recorded intra-esophageal and intra-gastric pressure using transnasal sensors. They could demonstrate a significant correlation between waist circumference and BMI with intra-gastric pressure and gastroesophageal pressure gradient. They also found that obesity was associated with the separation of GEJ pressure components. They concluded that obesity increases the flow of gastric contents into the esophagus by causing GEJ incompetence and has an adverse effect on GEJ physiology.

Bariatric Surgery and Gastroesophageal Reflux

Beneficial (Anti-Refluxogenic) Effects of Bariatric Surgery on GERD

The pure and simple assumption of the potential of improvement in GERD after bariatric surgery purely based on weight loss alone has a very strong argument to be made of, based on logistical

reasoning and evidence. The mitigation of the majority of the contributing factors to promoting GERD mentioned in Table 1 on its own accord would stand ground.

In addition to the above, certain other anatomical and physiological factors that are individual components/consequences of different types of surgeries would explain the differential benefit between surgeries to improve reflux further.

Reduction and/or Repair of the Hiatal Hernia

It is known that there is a relatively higher incidence of hiatal hernia among the obese population [16]. Careful preoperative evaluation, intraoperative assessment, and appropriate surgical techniques such as repair of a hiatal hernia (if felt necessary) will have a beneficial effect. On the same note, routine dissection of the hiatus in all patients can be a double-edged sword with a potential to disrupt preexisting protective mechanisms and predispose to the formation of a hiatal hernia or GERD after surgery.

Reduction in the Acid Exposure (Production) Due to Resection (All Surgeries with a Sleeve Gastrectomy Component)

The reduction in the total capacity of the stomach (and hence, capacity to produce acid) in a sleeve gastrectomy due to the "resection" component, in theory, would have a beneficial effect. However, the resultant increase in intragastric pressure may work in an opposing way against the beneficial reduction in acid production.

Reduction in Acid Exposure Due to Exclusion (All Types of Bypasses)

The exclusion of the remnant stomach in the bypass surgeries, although of different magnitude between an RYGB and OAGB, reduces the exposure to the acid of the esophagogastric junction (OG) junction and the potential of reflux.

Creation of a Low-Pressure System (All Types of Bypasses)

RYGB and OAGB, in principle, create a low-pressure system that aids a preferential caudal (distal) flow of food and acid, reducing the potential to GERD. However, the presence of added bile as a component of the new gastric pouch/reservoir in the OAGB can add to the volume passing across the pouch and a theoretical potentiation to combined reflux of acid and bile.

Mechanical Caudal Pull Effect (All Types of Bypasses)

The downward pull exerted by the anastomosis in the bypass techniques (RYGB and OAGB) serves as a protective factor against the potential to herniation of the gastric pouch, which can be possible due to dissection around the hiatus compromising the integrity of the OG junction.

Potential Refluxogenic Effects of Bariatric Surgery on GEJ

The most performed bariatric surgery procedures are SG, RYGB, OAGB, and gastric banding. The contribution of weight loss after bariatric surgery is, in fact, an independent mechanism in providing relief from GERD. However, different procedures have a varying effect on GERD depending on the inherent technical nature/configuration of the surgery [1, 2].

The possible mechanisms with which certain bariatric procedures may alter the protective mechanisms against GERD are:

- Division of the phreno-esophageal ligaments
- Blunting of the angle of His
- Division of the sling fibers impairing the effectiveness of the lower esophageal sphincter mechanism.
- Restrictive gastric pouch/sleeve leads to increased gastric pressure by reducing gastric compliance, e.g., SG
- Delayed gastric emptying
- Impaired esophageal peristalsis and esophageal dilatation
- Bile reflux, e.g., OAGB
- Potential to hiatal hernia
- Outflow obstruction, e.g., kink, stricture, or anastomotic narrowing.

Based on the potential effects the commonest types of surgeries may have on the anti-reflux barrier mechanism, we have postulated a table with the refluxogenic potential of different surgeries (Table 2).

It is interesting to note that all types of bariatric surgery, in theory, have a potential to be refluxogenic. Although not the case in real life due to the balance between the refluxogenic and anti-refluxogenic effects slanting toward one side or the other, it gives us an understanding of the mechanisms of action that can be at interplay when patients present with symptoms of gastroesophageal reflux after bariatric surgery.

Hence, it is essential to understand and accept that GERD can be a problem after bariatric surgery irrespective of the type of surgery and deserves careful attention to symptoms, assessment, and further management.

Take home message – ***"The problem of GERD can exist after any type of bariatric surgery based on pure reason and logic."***

Evidence

Reflux After Laparoscopic Sleeve Gastrectomy (LSG/SG)

SG is currently the most performed bariatric surgery worldwide. With increasing experience, literature, and follow-up data, it has been subject to discussion with special reference to the incidence of reflux disease after surgery.

The beneficial effects of weight loss are variably comparable to the other procedures but there has been a growing interest in the incidence of gastroesophageal reflux after SG in comparison to the other procedures [5, 9, 21]. There has been increasing evidence in the literature of the new development of symptoms, endoscopic changes [15], and BE [17, 18, 27] with ad hoc reports of adenocarcinoma [18, 22] of GEJ. It is imperative that this needs to be considered with the increasing adoption of the SG worldwide.

There are two categories of patients to be considered within the context of GERD and SG. Those with pre-existing GERD prior to surgery and the patients with new-onset reflux (*de novo* GERD). It is also to be borne in mind that the absence of symptoms prior to surgery is a very unreliable indicator of the true absence of pathological reflux in the obese population.

However, it is imperative that technical and functional issues are excluded and investigated as a part of the work-up of the patients presenting with symptoms of GERD after an SG (Table 3).

Coupaye et al. [14] in 2018 had performed manometric and pH analysis in all patients before and after SG with a one-year follow-up and had found that 66% of patients had no GERD and 34% had GERD prior to surgery. It was found that 56% of patients developed *de novo* GERD based on pH studies (increased acid exposure times), and 37% of patients with GERD prior to surgery had worsening symptoms. However, 63% of patients with prior GERD had no or improved symptoms after surgery.

Table 2 Inherent reflexogenic score after bariatric surgery

	Sleeve Gastrectomy	Roux Y Gastric Bypass	One Anastomosis Gastric Bypass	SADI-S Duodenal Switch	SASI
Esophageal function	+/-	+/-	+/-	+/-	+/-
Angle of His disturbed	++	++	++	++	++
Phreno-oesophageal ligament	++	++	++	++	++
Sling/Clasp fibres LES	++	++	++	++	++
Potential to hiatal herniation	+++	++	++	++	++
Intragastric pressure	+++	-	-	++	-
Gastric contents (Acid/Bile)	+	-	+	+	+
TOTAL REFLUXOGENIC SCORE	6 (or 7)	4 (or 5)	5 (or 6)	6 (or 7)	5 (or 6)

Table 3 Anatomical factors (non-Inherent) contributing to reflux after a laparoscopic sleeve gastrectomy (LSG)

Anatomical factors (non-Inherent) contributing to reflux after LSG
Esophageal
Undiagnosed esophageal motility disorder
Esophageal hypersensitivity
Gastric
Large fundic pouch
Intrathoracic herniation of the sleeve
Tight sleeve
Stricture in the sleeve
Twisted/Kinked sleeve
Delayed gastric emptying
Functional disorders

Perretta et al. [32] in 2020 had performed a prospective study in 35 patients with endoscopic, manometric, pH, and magnetic resonance imaging studies before and after SG. At one-year follow-up there was a significant mean increase in the angle of His, reflux episodes, acid exposure times, and a decrease in the length plus LES pressure after surgery. Esophageal motility was, however not affected.

Csendes et al. in 2019 [15] reported their 10 year follow-up in 93% of patients after an SG with a detailed clinical, radiological, endoscopic, and histological data in 97 patients and reported *de novo* GERD in 58% of patients and a hiatal hernia in 38%, with a 4% incidence of BE. They mention a complete resolution of GERD in 13% of patients after surgery.

The Austrian [25] series update to a previous study with over 100 patients and a 10-year follow-up found that 33% of SG needed conversion, with 34% needing it due to GERD. The patients had full endoscopic, manometric, and pH studies before and after surgery. They had also noted the development of Barrett's esophagus in 14% of their patients and the presence of a hiatal hernia in over 55% of the patients with GERD.

There have been at least four studies with a total patient load of over 600 patients, and an average follow-up of 5 years demonstrating a mean incidence of 10.7% for the new development of BE [6, 19, 30].

Interestingly, Ikramuddin et al. [28], in a multicenter study with 8362 patients, had compared a 1:1 match between RYGB and SG and had noted GERD in both subsets. They had noted a postoperative GERD occurrence of 60.2% in SG vs. 55.6% in RYGB but a greater incidence of *de novo* GERD in the SG group. They noted a greater incidence of BE in the RYGB population (0.7% vs. 1.1%). They argue for no added advantage of the RYGB over an LSG in terms of reflux.

A recent systematic survey and meta-analysis by Gu et al. in 2019 comparing laparoscopic Roux-en-Y gastric bypass (LRYGB) and LSG had concluded that although both procedures had resulted in improved symptoms. LSG had a higher incidence of *de novo* GERD, and RYGB may be a better choice for patients undergoing bariatric surgery with preexisting GERD.

The American Society for Metabolic and Bariatric Surgery (ASMBS) and International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO) recommendations [10] state that all patients undergoing LSG and OAGB should undergo an upper gastrointestinal (GI) endoscopy at one and five years after surgery to rule out BE and the complications of GERD.

Take home message – ***"There is literature evidence to suggest the increased incidence and potential of reflux (+/− BE) after SG."***

Reflux After OAGB

The increasing acceptance of OAGB over the past decade has once again presented the opportunity to evaluate larger data and evaluate outcomes with long-term follow-up. The effectiveness of the procedure in terms of weight loss and comorbidity resolution remains undisputed and has established its place as a mainstream procedure in the realm of bariatric and metabolic surgery.

On a parallel note, concerns on the potential to enhance bile reflux and gastroesophageal reflux remain an area of debate. Based on pure logic and reason, the surgery does involve dissection around the hiatus and carries identical procedural steps and effects on the anti-reflux barrier mechanism at the hiatus, such as the SG or the RYGB.

Hence, it carries the same refluxogenic potential by interfering with the angle of His, phrenesophageal membrane, sling fibers, LES pressure, etc. The longer pouch created to counteract the potential of bile reflux carries with it an added acid-secreting load and aids the mixing of bile and acid within the pouch.

However, the anti-refluxogenic effect of the procedure stems from creating a low-pressure system due to the gastrojejunal anastomosis (GJA) and the added downward traction from the anastomotic mechanism preventing the formation of a hiatal hernia after surgery. Furthermore, certain modifications from the original technique to create a valve have been proposed and variably followed.

The evidence regarding the association between OAGB and GERD bile reflux is variable. The proponents of the procedure quote a very low incidence of postoperative GERD, and the critics understandably otherwise. Overall, it seems to be comfortably placed between the LRYGB and the SG in terms of GERD control, worsening of preexisting reflux, and development of *de novo* reflux.

The literature evidence is not as robust as with the data from the SG group but is increasingly being available and comparative studies between procedures are being performed to answer the question.

However, it is imperative that technical and functional issues are excluded and investigated as a part of the workup of the patients presenting with symptoms of GERD after an OAGB (Table 4).

Musella et al. [30] in a prospective randomized study of 58 patients (LSG-30 and OAGB-28), observed that the acid exposure time had increased after SG and there was no change after an OAGB [29]. There was no worsening in esophagitis in OAGB compared to SG and OAGB was proposed as a preferred approach in patients with preoperative subclinical reflux or grade A esophagitis. However, there was a nonsignificant increase in the number of non-acid reflux episodes in the distal (approximately 50% increase) and proximal esophagus at 6 and 12 months after the OAGB. Hence, although not significant, there exists a potentiation of non-acid reflux episodes after the OAGB, which is not surprising.

Table 4 Anatomical factors (non-Inherent) contributing to reflux after a one anastomosis gastric bypass (OAGB)

Anatomical factors (non-Inherent) contributing to reflux after OAGB
Esophageal
Undiagnosed esophageal motility disorder
Esophageal hypersensitivity
Gastric pouch related
Large fundic portion within the gastric pouch
Intrathoracic herniation of the gastric pouch
Tight pouch
Stricture in the pouch
Twisted/kinked pouch
Gastro-jejunal ulcer/stricture
Gastrogastric fistula
Small bowel related causes
Twisted bowel related to the anastomosis
Internal herniation
Distal obstruction (Adhesions etc.)
Functional disorders

Another 2018 study analyzed the multichannel intraluminal impedance-pH-metry (MII-pH) parameters in 11 patients after an OAGB and found some interesting variables [17]. The number of acid reflux episodes had decreased after OAGB but there was a significant increase in the number of non-acid refluxes both in the distal and proximal esophagus as noted in the Musella study (although not significant in the Musella study). The researchers had found that the DeMeester score had increased despite the decreased number of acid reflux episodes because of the increased bolus transit and acid clearance times. They had noted *de novo* reflux in 28% of patients and worsening of reflux in all patients with preexisting GERD.

A more recent retrospective study of 89 patients with a hiatal hernia, symptomatic GERD, or esophagitis found that in the symptomatic GERD subset of 34 patients, 61% had postoperative GERD, with 14% needing conversion to RYGB and 55% needing daily proton pump inhibitors (PPIs) [40]. Of the smaller group of nine patients with preoperative endoscopic changes, 44% continued the need for once-daily PPIs. The authors, however, accept the limitation with the

absence of routine postoperative endoscopic or GI physiology studies. It is also widely accepted that symptoms alone may be an inadequate marker for GERD and there is a potential to underestimate the incidence in the obese population.

The YOMEGA trial reported a 10% incidence of esophagitis in the OAGB group in comparison to 3% in the RYGB group [34]., Saarinen et al., in their study to detect bile reflux using scintigraphic and endoscopic methods, had found postoperative scintigraphic bile reflux in the stomach pouch in 28% and scintigraphic bile reflux in the esophagus in 2.8% of patients. However, they also quote that 22% of patients had GERD (persistent plus *de novo* reflux), and 22.5% had esophagitis on endoscopy, with 14.5% being new-onset esophagitis.

Nehmeh et al. in 2021 [30] had investigated 43 patients with symptomatic GERD after OAGB with endoscopy and MII-pH studies. Abnormal acid reflux was found in 30% of patients, non-acid reflux in 27%, and mixed refluxate in 11.6% of patients. More than 50% of the subjects showed esophagitis after surgery in comparison to 11% prior to it.

Although it is not the intention of the author to pick out the articles suggesting the incidence of GERD after an OAGB, it should be accepted that OAGB is not necessarily a reflux curing procedure, and patients should be selected, warned, and investigated when symptoms present or persist after OAGB.

The ASMBS and IFSO recommendations [10] state that all patients undergoing LSG and OAGB should undergo an upper GI endoscopy at one and five years after surgery to rule out BE and the complications of GERD.

Take home message - "***Although relatively less compared to SG, there is literature evidence to suggest the incidence and potential to reflux after OAGB.***"

Reflux After LRYGB or RYGB

The RYGB configuration in the LRYGB understandably has the most promising effect on control of GERD in preexisting GERD and the least chance of *de novo* GERD. It has always been treated and assailed as the go-to option/holy grail in anyone with GERD prior to surgery or with postoperative GERD after other forms of bariatric surgery.

However, based on some logical reasoning that has been placed earlier in the chapter, the technique of surgery that involves dissection around the hiatus, loss of angle of His, potential to postoperative hiatal herniation, division of the phrenoesophageal membrane, etc. which is common to all the three types of surgery (LSG/OAGB and LRYGB) does not make the LRYGB theoretically "free" of any chance of postoperative reflux.

Despite the above, the compensatory mechanisms below offer a significant comparative advantage over the LSG and OAGB.

1. The low-pressure system due to the GJA (common to OAGB).
2. The relatively smallest amount of residual acid-secreting pouch of all three procedures.
3. The downward caudal pull exerts a protective mechanism against hiatal herniation (common to OAGB).
4. The Roux-en-Y configuration being protective against bile reflux (unique to the LRYGB).

It is to be, however, noted that there can be other mechanisms contributing to reflux after an LRYGB, which need consideration and assessment when evaluating patients with symptoms of GERD after an LRYGB (Table 5).

The increasing availability of data and long-term outcomes in the medical literature is witnessing some accumulating evidence (although less in comparison to LSG and OAGB) of the incidence of symptomatic, anatomical, endoscopic, and physiological evidence on the occurrence of GERD to a smaller extent in the postoperative follow-up period that may need to be considered.

Lenglinger et al. [6] investigated 47 patients presenting with symptomatic reflux or symptoms suggestive of reflux after LRYGB over a 3-year period with a median follow-up of 3.8 years. The limiting factor of this study was that the common denominator of the base population on the total

Table 5 Anatomical factors (non-Inherent) contributing to reflux after a Roux Y Gastric Bypass

Anatomical factors (non-Inherent) contributing to reflux after a Roux Y gastric Bypass
Esophageal
Undiagnosed esophageal motility disorder
Esophageal hypersensitivity
Gastric pouch related
Large fundic portion within the gastric pouch
Intrathoracic herniation of the gastric pouch
Inappropriately large/long pouch (increased acid secreting capacity)
Candy cane syndrome
Gastrogastric fistula
Pouch stasis syndrome
Marginal ulcer and stricture
Alimentary limb related
Short limb
Roux -O- configurations
Roux-Y stasis syndrome/Altered limb motility
Distal obstruction (JJ stricture, intussusception, adhesions)
Internal hernias (Peterson's and or mesenteric defect herniation)
Functional disorders

number of LRYGB performed in that period or otherwise, was not available. The preoperative symptoms or investigative data is also not available. However, the paper gives insight into the potential causes for reflux after LRYGB, and the authors should be appreciated on the strict workup of these patients. Patients were assessed using a symptom questionnaire, endoscopy, GI physiology (manometry and MII-pH), and a barium swallow. It was noted that 53.2% of patients had hiatal hernias, 23.4% had esophagitis of grade B or above, 37.8% had some form of esophageal dysmotility, a hypotensive lower esophageal sphincter was found in 57.8%, and a pathological amount of acid refluxes in 68.2%. Only 12.8% were deemed functional after undergoing all investigations. Although it has no preoperative data or the total number of RYGB of which these groups form a percentage, it cannot be disputed that there was a group of patients in the symptomatic reflux group who were investigated and had postoperative reflux after an RYGB.

Arnoldner et al. [3] had performed an interesting study in the evaluation of intrathoracic migration (ITM) of the gastric pouch in a select group of patients undergoing revisional surgery after an RYGB due to weight regain. They had compared the sensitivity of a 3D CT with a view to measure ITM and pouch volume with gastroscopy in 30 patients. It was found that the 3D CT had found a hiatal herniation of the pouch (ITM) in 66% (20 of 30) of the patients. Although the relationship between the ITM and GERD has not been studied in this study, it can be generalized that a hiatal hernia in this setting is due to disruption of the anti-reflux barrier mechanisms at the hiatus contributed by a previous RYGB.

Clapp et al. [13], in 2020, had reported on a series of eight patients from a single institution with hiatal hernias and symptomatic reflux after LRYGB needing surgery. This study, too, suffers from the limitation as in the previously quoted study by Lenglinger et al. [6], with no information of the total number of LRYGB procedures among which the study population formed a subset. However, in a qualitative sense, the occurrence of a hiatal hernia and symptomatic reflux after the LRYGB cannot be denied.

Rebecchi et al. [33] assessed the medium- and long-term effects of LRYGB on acid exposure at the GEJ. They had studied two groups of patients after LRYGB – (Group A with 54 patients with no GERD preoperatively and Group B with 32 patients who had GERD preoperatively based on preoperative MII-pH monitoring). Endoscopy and MII-pH studies were performed at 12 months and 60 months in all patients. The incidence of acid reflux, weak acid reflux (WAR), and combined reflux was studied. Esophagitis was found in 14 group A patients (30%) and in 18 group B patients (69%). There was a significant improvement in the acid reflux parameters in both groups with return to normal levels in group B. However, it was noted that there was a concomitant increase in WAR in both groups. All patients with esophagitis had WAR at 24-h MII-pH monitoring. They go on to propose the potential need for endoscopic surveillance even after an LRYGB in the context of non-acid/WAR refluxes. However, the exact

definition, acceptance, and relevance of WAR refluxes remain unknown.

Take home message - "***Although significantly less compared to the SG and the OAGB, there is literature evidence to suggest the incidence and potential to reflux after LRYGB"*** (Fig. 1).

Approach to the Patient with Symptomatic Reflux After Bariatric Surgery

A standardized approach should be adopted when assessing patients presenting with symptoms of GERD after bariatric surgery (Fig. 2).

History Taking

A complete and detailed history of the exact symptoms, precipitants, relieving factors, surgical history, smoking history, etc. forms the initial stepping stone to assess the patient. Relief with PPI may give an idea as to if the symptoms may be related to acid reflux or otherwise. The timing of reflux after meal ingestion may suggest an esophageal cause/distal cause. Associated symptoms such as dysphagia, vomiting, and abdominal pain should be explored to look for alternate/added pathologies.

Obtaining the operative notes may be of added value as it is not infrequent that some patients are unaware of the exact type of procedures they may

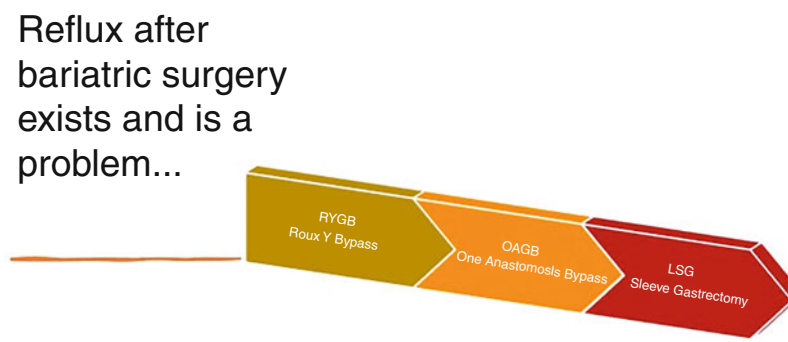


Fig. 1 Reflux after bariatric Surgery

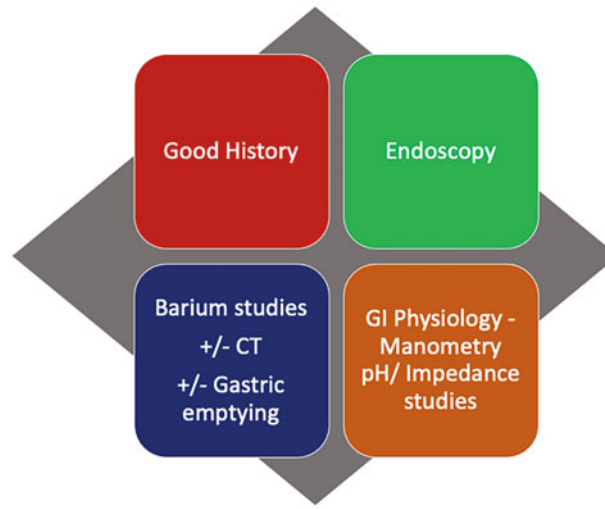


Fig. 2 Assessment of the patient with Gastro esophageal reflux after bariatric surgery

have had in the era of medical tourism, countries, and continents away from the place of residence. On the same note, innovative procedures have been seen to be performed without the full awareness/consent of patients.

Investigations

Upper GI endoscopy – Ideally performed by the surgical team to assess for anatomical variations, esophagitis, hiatal hernia, marginal ulcers, pouch volume, anastomotic stricture, etc. Correlation of the symptoms with the findings at endoscopy could offer valuable clues to a way forward.

Barium studies – Video fluoroscopic barium studies offer valuable information to the anatomy, the presence or absence of a hiatal hernia, pouch/sleeve configuration, kinking, strictures, reflux, candy cane, gastro-gastric fistula, etc. It can be more sensitive than endoscopy in most instances except for detecting intraluminal pathology.

GI Physiology and pH/impedance studies – Esophageal manometry and pH/impedance studies are vital to exclude contributory esophageal motility issues and for confirmation of pathological reflux. The use of impedance studies is more useful after OAGB and RYGB to look for and record pathological concomitant non-acid reflux.

CT (+/− 3D reconstruction) – Will serve as a valuable adjunctive tool for the need to rule out internal hernias, herniation of the pouch, pouch volume, alternate pathology, etc.

Gastric emptying studies – Selective usage to assess gastric emptying after an SG.

Exploratory laparoscopy – May be considered if all the investigations are negative and there is a convincing history with a less likely chance of a functional problem.

Management

Following a complete assessment of the patient and confirmation of GERD after any form of bariatric surgery, the options can be broadly medical or surgical based on the symptoms, intensity, the effect on the quality of life, and the associated pathologies found on investigation.

The presence of anatomical variants contributing to postoperative GERD that are not a part of the intended original surgery is better served by surgical correction. These may include the following

- Large hiatal hernia/thoracic migration of the sleeve, gastric pouch
- Long candy cane with GERD
- Gastro-gastric fistula
- Twisted, strictures sleeve, gastric pouch
- Resistant/recalcitrant strictures, marginal ulcers
- Adhesions
- Internal hernias, intussusception, etc.
- Suboptimal bowel length during bypass configuration contributing to reflux

In the absence of any of the above factors, a choice of alternatives could be considered after discussion with the patient and ideally involving a multidisciplinary team to aid decision-making.

Medical Management

In the absence of a contributing anatomical factor that can only be served by surgical correction, medical management can be an accepted treatment with PPI, Gavison, etc. It should, however, be counseled that it is likely to be a lifelong treatment modality.

Endoscopic Management

It is not within the context of this chapter to elaborate on them, but endoscopic therapies such as the radiofrequency ablation (Stretta) could be considered based on the availability within different health systems, appropriateness, and expertise.

Surgical Management

A broad overview based on algorithms is presented in the figures for GERD after

- SG- (Fig. 3)
- OAGB- (Fig. 4)
- RYGB- (Fig. 5)

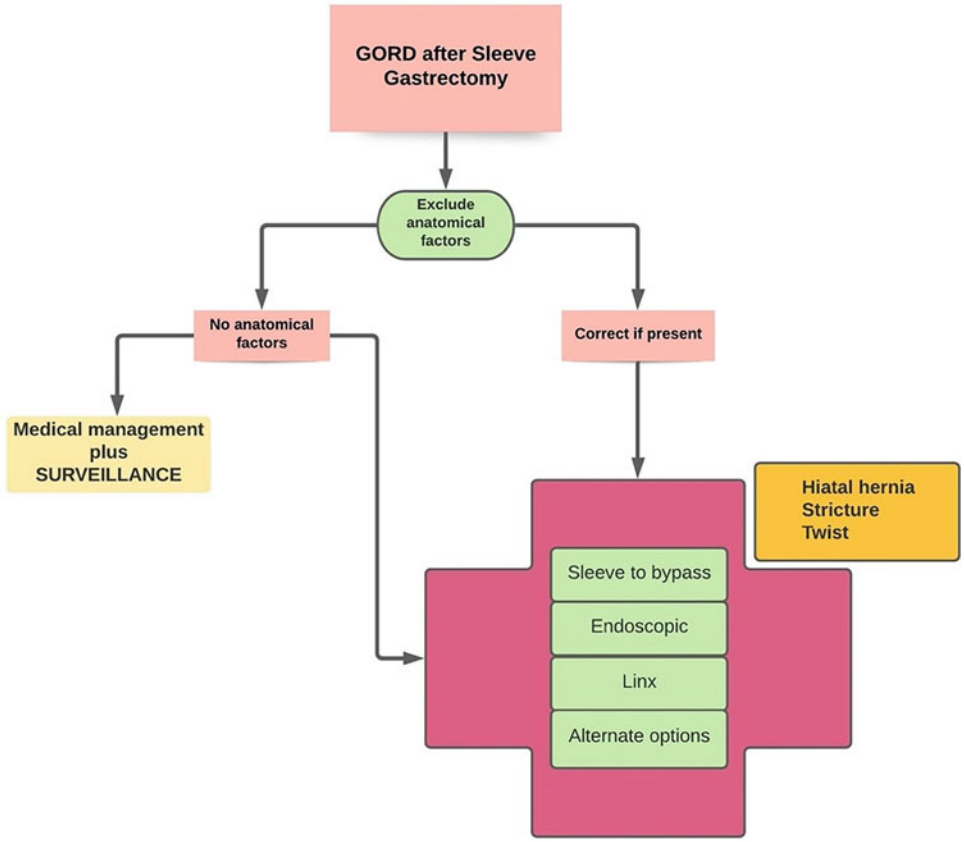


Fig. 3 Management of GERD after laparoscopic sleeve gastrectomy (LSG)

Newer modalities such as the magnetic sphincter augmentation device (LINX), remnant stomach fundoplication, Belsey type fundoplication, ligament teres reinforcement, etc., have been reported with varying success rates and could be offered if felt appropriate, taking into context the individual circumstance and expertise available.

Surveillance

It is increasingly being accepted that surveillance should be part and parcel of the care after refluxogenic types of bariatric surgery. The IFSO [10] and ASMBS [11] statements have made specific recommendations on the same.

- Upper GI endoscopy should be undertaken routinely for all patients after bariatric surgery at one year and then every two-three years for patients who have undergone LSG or OAGB to enable early detection of BE or upper GI malignancy until more data is available to confirm the incidence of these cancers in practice.
- Upper GI endoscopy should be performed following gastric banding and RYGB on the basis of upper GI symptoms.

Summary

Based on the above discussion, we can accept with a combination of reason, logic, science, and the available literature that obesity contributes to the development and potentiation of GERD.

Bariatric surgery remains the most effective treatment available to maximize weight loss and improve medical comorbidities associated with

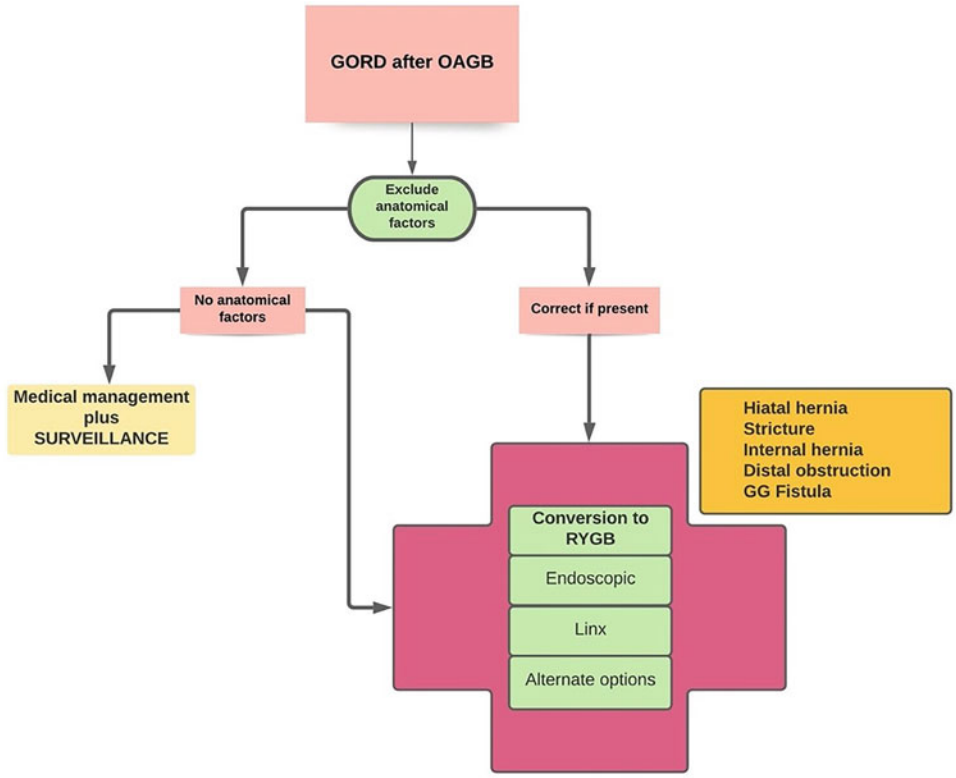


Fig. 4 Management of GERD after one anastomosis gastric bypass (OAGB)

obesity. Reflux remains an important comorbidity in patients with reflux. Symptoms alone are not a reliable indicator of reflux as many patients suffer silent GERD in the absence of symptoms. Thorough preoperative evaluation is needed in patients with symptoms of reflux and can be generalized for a routine recommendation for endoscopy prior to any form of bariatric surgery. The IFSO recommendations [10] support the idea of routine endoscopy prior to bariatric surgery.

The presence or absence of reflux is likely to influence the choice of surgery when taken into consideration with other associated factors that may influence the decision, such as Type 2 diabetes mellitus, previous surgery, inflammatory bowel disease, smoking, very high BMI, patient on immunosuppression, etc.

All types of bariatric surgery have the potential to improve reflux merely by contribution to significant weight loss. However, it should be accepted that different surgeries offer differing levels of reflux protection, and all do have a refluxogenic potential for the development of new-onset reflux and/or worsening of existing reflux. We strongly believe that it is important that the patient is counseled prior to surgery on the potential to develop reflux after all types of surgery (Figs. 2 and 6).

The most protective and lesser chance of reflux after bariatric surgery resides with the LRYGB. However, the patients are not immune from developing reflux after a LRYGB. In comparison to LRYGB, the OAGB and the SG have a relatively less protective action on reflux and a higher chance of new-onset GERD. The LSG carries a higher chance of persistent and new-onset reflux of all the current available forms of bariatric surgery. This should be borne in mind when a patient is counseled for surgery. Complex, revisional surgeries that incorporate SG as a part of the procedure such as the single anastomosis duodeno-ileal bypass with sleeve gastrectomy and the duodenal

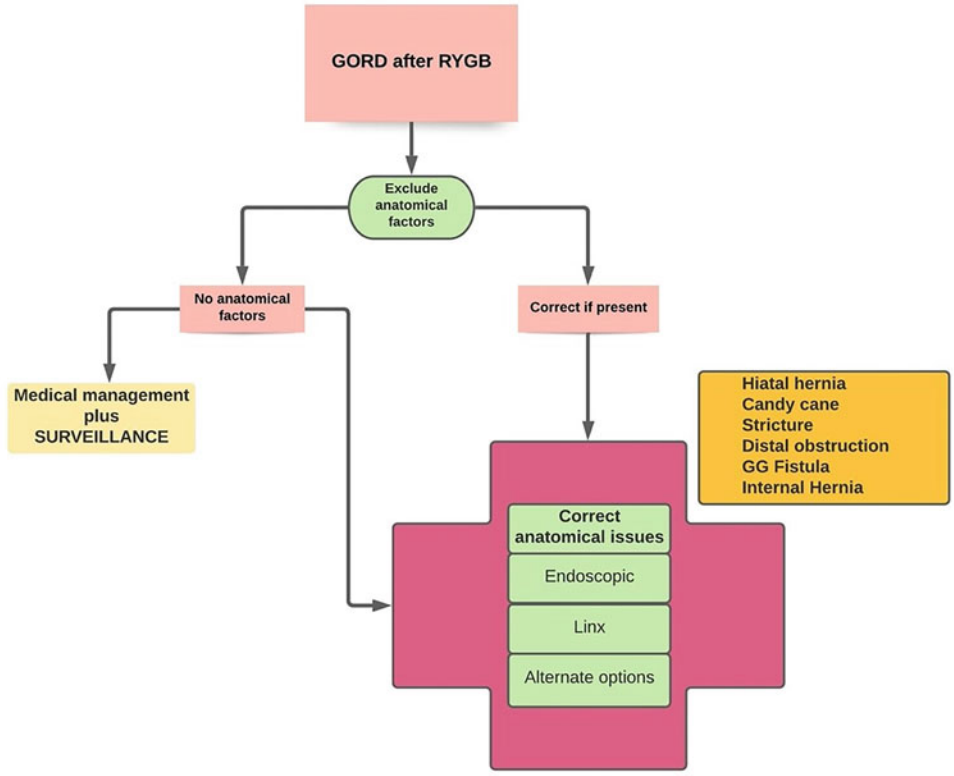
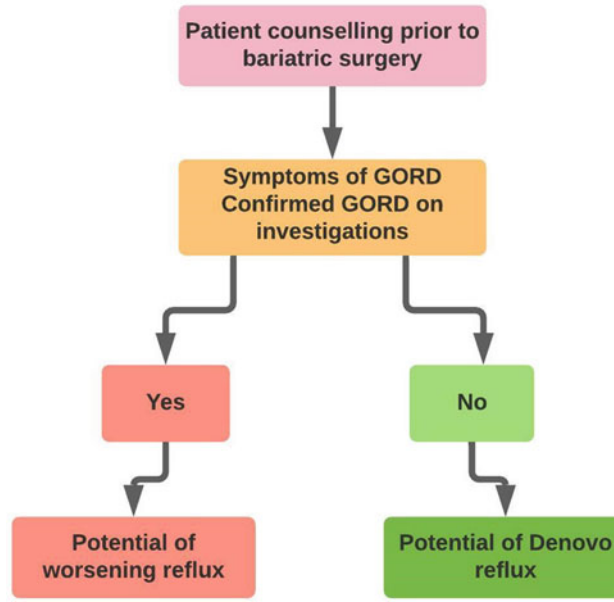


Fig. 5 Management of GERD after Roux Y Gastric bypass (RYGB)

Fig. 6 Patient counselling prior to bariatric surgery



switch surgery retain the chance to be refluxogenic due to the sleeve component despite a wider pouch.

Key Learning Points

- GERD is common in obesity and has a multifactorial cause. Symptoms alone may not be reliable in the diagnosis of GERD when planning bariatric surgery and all bariatric procedures have the potential to induce GERD after primary surgery.
- GERD is more common after SG and least after RYGB.
- Postoperative surveillance is recommended after LSG and OAGB as per recent recommendations from international bodies.
- Evaluation of GERD after bariatric surgery should include a complete evaluation with a thorough history taking, endoscopy, GI physiology studies, and barium examinations in all patients. The use of CT scanning, gastric emptying studies, and diagnostic laparoscopy is to be considered judiciously when felt appropriate.
- Decisions of management of GERD after bariatric surgery include a combination of medical, endoscopic, and surgical interventions. The involvement of a multi-disciplinary team decision is recommended.

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