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Overview of Bariatric Surgeries: Management and Complications

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ABSTRACT

Around 1.9 billion individuals and children who were 18 years old and older were overweight. Approximately 650 million people were obese. 39% of adults over the age of 18 were overweight or obese in 2016, with 13% of them being obese. Overweight and obesity cause more fatalities than underweight in the majority of the world's population. Many factors could contribute to the rising obesity epidemic. The initial treatment goal is usually a modest weight loss 5% to 10% of your total weight. The burden of obesity is even more complex as the impact of obesity is a result of its comorbidities rather than a direct effect, which makes it more difficult to estimate the burden of obesity. The treatment methods that are right for you depend on your obesity severity, your overall health and your willingness to participate in your weight-loss plan. Bariatric surgeries are classified as having restrictive or malabsorptive properties. Restrictive surgeries reduce the volume of food that can be consumed at one time, leading to reduced total caloric intake. The aim of the present study was to review the bariatric surgeries management and its associated complications.

Keywords: Obesity ; Bariatric Surgeries: Management ; Complications

Introduction

There were 38 million children under five who were overweight or obese in 2019. In 2016, there were more than 340 million overweight or obese kids and teenagers between the ages of 5 and 19 **(1).** According to the World Health Organization (WHO), Egypt ranks 18th with the highest prevalence of obesity worldwide. Deaths attributable to non-communicable diseases represent about 71% of the total mortality burden. Very few studies have been published about the burden of diseases in Egypt in general. There is not any published study which has specifically attempted to evaluate the economic burden of obesity in Egypt **(2).**

Estimating the societal burden of obesity is an essential step in setting priorities for research and public health interventions. It also helps to raise public awareness about the negative impacts of obesity and provides health policy decision-makers with information about the magnitude of health problems. Some studies do not consider obesity as a disease, as it may not have substantial direct costs or related pain, although it is included in the International Classification of Diseases and Related Health Problems. The clear fact is that obesity is a risk factor for several costly and disabling diseases **(3).**

Obesity ensues when an individual's body accumulates abnormal amounts of fat. This takes place when energy intake exceeds energy expenditure over time **(4).** Increased fast food consumption has been linked with obesity in the recent years. Many families, especially those with two parents working outside the home, went for these places as they are often favored by

their children and are both convenient and inexpensive. Foods served at fast food restaurants tend to contain a high number of calories with low nutritional values **(5)**.

Globally, the obesity pademic is largely the consequence of increased energy consumption. However, in individual patients, there may be several reasons why a person has an increased caloric intake or decreased energy expenditure, which may even be modifiable. Often, there is a complex interplay of multiple social, psychological, and biological factors altogether resulting in excess energy intake **(6)**. Some individuals may overeat as a coping strategy for other, psychological factors such as emotions. Next, a decreased quantity or quality of sleep can induce weight gain. This may lead to a desire for high caloric food, imbalance of appetite hormones, as well as increased hypothalamic–pituitary–adrenal-axis reactivity yielding higher cortisol levels which may also enhance obesity. Circadian misalignment such as in shift work is associated with decreased daily energy expenditure and increased caloric intake. As for sleep quality, obstructive sleep apnea (OSA) is especially notable as it seems to have a bidirectional relation with obesity (**7).**

Medication can affect the energy homeostasis mainly by promoting hunger or by decreasing resting metabolism. Drugs that are frequently used in psychiatry, such as specific selective serotonin reuptake inhibitors or antipsychotic agents, are well-known to promote weight gain. The strongest weight change is seen in amitriptyline, mirtazapine, and paroxetine. As for antipsychotic drugs, olanzapine and clozapine induce the highest weight gain. Also, several anti-epileptic drugs should be noted. Although most prescribers are aware of these side effects, in patients with severe obesity, a medication switch to less obesogenic drugs should be considered if possible (**5).**

It is well-known that systemic corticosteroids can cause weight gain. Local corticosteroids may also have systemic effects resulting in weight gain, in a similar matter as local corticosteroids having other systemic effects, such as adrenal insufficiency. A weight gaining systemic effect of local corticosteroids is likely in patients using large quantities on a frequent basis, particularly if they show additional Cushioned features, such as abdominal obesity, peripheral atrophy, plethora, and purple striae **(8).**

Monogenic (non-syndromic) causes of obesity are characterized by a young age of onset and hyperphagia, with usually no intellectual deficit. Additionally, other clinical signs of monogenetic obesity may differ depending on the affected gene. These include red or ruddy hair, pale skin, and adrenocorticotropic hormone (ACTH) deficiency (pro-opiomelanocortin [*POMC*] gene defects), central hypothyroidism, hypogonadotropic hypogonadism and frequent infections (leptin deficiency, caused by autosomal recessive *LEP* gene mutations), increased linear growth and increased lean mass, severe hyperinsulinemia, and mild central hypothyroidism (caused by autosomal dominant or recessive *MC4R* mutations) and neonatal diarrhea, recurrent hypoglycemia, and global endocrine dysfunction (caused by prohormone convertase-1 [*PCSK1*] mutations) **(9).**

Prader-Willi syndrome characterized by hypotonia and feeding problems in infancy, and later in life hyperphagia and obesity, short stature, intellectual deficit and hypogonadotropic hypogonadism, Bardet-Biedl syndrome (intellectual deficit, retinal dystrophy or pigmentary retinopathy, polydactyly, hypogonadism, and nephropathy), There is a large variability of these symptoms among affected patients. Mild to moderate intellectual disability is also seen in patients with pseudohypoparathyroidism type 1 (PHP1a), caused by maternally inherited heterozygous mutations in *GNAS*. PHP1a is associated with the clinical phenotype of Albright's hereditary osteodystrophy (AHO), which encompasses short stature, round facies, and skeletal abnormalities **(9).**

A relatively sudden increase in weight may suggest a neuroendocrine cause, screening for hypothyroidismisd recommended, as this is associated with a modest weight gain. This is especially recommended if patients present with other symptoms such as dry skin, feeling cold, etc. However, the weight gain in hypothyroidism seems mostly due to additional edema. Also, obesity is often associated with a slightly increased TSH that is most often the result of excess adipose tissue rather than the cause of obesity. This can be explained by the presence of peripheral thyroid resistance and also by increased levels of leptin, stimulating TRH and subsequently TSH. Weight loss usually reverses this form of hyperthyrotropinemia (**10).**

Cushing syndrome (CS), specific signs of CS including easy bruising, facial plethora, proximal myopathy, and recent purple striae. should be considered, as most patients with obesity will have the more non-specific CS signs such as central obesity, fatigue, hypertension, and decreased libido. Due to the large number of corticosteroid users, iatrogenic CS should also be considered **(7)**. Also, acommon endocrinopathy affecting between 6%-10% of reproductive-age women **(11).**

PCOS presents with the cardinal features of hyperandrogenism, reproductive and metabolic dysfunction. PCOS associates with insulin resistance, independently of (but amplified by) obesity. Insulin resistance in PCOS is characterized by abnormal post-receptor signalling within the phosphatidylinositol-kinase (PI3-K) pathway. Multiple factors (including most notably, weight gain) contribute towards the severity of insulin resistance in PCOS. Compensatory hyperinsulinaemia ensues, resulting in over-stimulation of the (intact) post-receptor mitogen-activated protein kinase (MAP-K) insulin pathway, with consequent implications for steroidogenesis and ovarian function **(12)**.

Binge-eating disorder is characterized by recurrent binge-eating episodes where more food is consumed than is normal for most people and where feelings of lack of control and distress play a role. Importantly, binge-eating can also be a sign of hyperphagia and may thus be a diagnostic clue for either genetic or hypothalamic obesity **(13).**

Evidence is mounting that stress leads to more appetite (in comfort food), induces abdominal obesity, and may counteract the effects of a healthy diet. Additionally, the weight stigma that individuals with obesity often suffer from may also lead to extra weight gain. It is therefore conceivable that a non-stigmatizing attitude, as well as stress reduction, is beneficial when treating obesity **(11)**.

Hypothalamic obesity, typically accompanied by hyperphagia, can occur after various insults leading to damage of the hypothalamic region. It is seen in patients with abnormalities in the hypothalamic region, eg, craniopharyngeoma (especially following surgery), inflammatory processes such as sarcoidosis and tuberculosis, vascular damage, head trauma, or after cranial radiotherapy, but also some of the genetic mutations that were previously mentioned can be considered hypothalamic obesity **(14)**.

Management of obesity

I Physical activity

The role of physical activity in weight management is 2-fold: to support an energy deficit and to preserve lean muscle mass. Total energy expenditure (TEE) is partitioned into basal metabolic rate (BMR), diet-induced thermogenesis (DIT), nonexercise activity thermogenesis (NEAT), and exercise, both cardiovascular and resistance training (RT), is the only component of TEE that is significantly modifiable. The combination of diet and exercise always results in greater weight loss than either modality alone **(15).**

Fasting is known to be one of the most ancient traditions in the world, and has been practiced among various communities for either cultural or religious reasons. Interestingly, it has also been used as a healing method for diseases in the past **(16).**

Intermittent fasting refers to eating patterns that target a pattern of dedicated periods of time (ranging from 12 h to several days) with consumption of little or no calories. It is voluntary, has a fixed duration, and caloric drinks are consumed regularly. It has been found to more likely mimic human eating patterns. Intermittent fasting has had increasing popularity as an alternative to continuous CR, with emerging data supporting promise in delivering similar benefits with respect to weight loss and cardio-metabolic health. As opposed to traditional CR paradigms, IF is a only requires fasting for discrete periods of time **(17).**

- **low-calorie diets**

Low-calorie diets - also referred to as low-energy diets, hypocaloric diets, or calorie restriction (CR) - constitute weightloss strategies for individuals with overweight or obesity that aim at improving metabolic health and diminishing the risk of obesity-associated disorders. Although the goal of weight loss in most interventions is set at a realistic 10%, in patients with BMI > 35 or in those with BMI > 30 and T2D a weight loss of 15 – 20% is recommended to achieve substantial health benefits. The diet is typically composed of a weight reduction phase (e.g. 4 – 12 weeks), followed by a weight stabilization phase. The LCD should be planned according to individual energy demands, taking into account the patient's sex, age, and physical activity level, as well as the degree of obesity, accompanying diseases, and previous treatment **(18).**

Macronutrient composition

The three primary dietary macronutrients are fat, carbohydrate and protein, which provide 9, 3.75 and 4 kilocalories per gram, respectively. Fat is the least satiating, most readily absorbed and calorie-dense macronutrient, making it the most appealing target for weight loss intervention **(19).**

II -Pharmacotherapy

NICE currently recommends pharmacological treatment for weight loss maintenance in addition to a reduced-calorie diet and optimal physical exercise **(20).**

- **Orlistat**

Orlistat is a reversible inhibitor of pancreatic and gastric lipases, which reduces the absorption of fat from the intestine. Administered at the standard dose of 120 mg three times daily before meals, orlistat prevents the absorption of approximately 30% of dietary fat, thereby reducing caloric intake. The most common side effects of orlistat are gastrointestinal (due to the non-absorbed fats in the intestine) and may include steatorrhea, frequent bowel movements, flatus with discharge, and fecal incontinence. Because orlistat inhibits the absorption of lipid-soluble vitamins, vitamin A, D, E and K supplements should be taken when using orlistat **(21).**

- **Phentermine/topiramate**

Phentermine is a potent inhibitor of the norepinephrine transporter and acts as an appetite suppressant via activation of the hypothalamic proopiomelanocortin (POMC) arcuate nucleus neurons. Topiramate is an antiepileptic drug, which suppresses appetite through modulation of voltage-gated ion channels, increased activity of the γ-aminobutyric acid (GABA)-A receptor-mediated inhibitory currents and/or inhibition of α-amino3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA)/ kainite glutamate receptors **(22).** A combination

of phentermine (a centrally acting appetite suppressant) and topiramate (an antiepileptic) that appears to induce weight loss, possibly by increased energy utilization **(23).**

- **Naltrexone/bupropion**

Naltrexone/bupropion combines the dopamine/norepinephrine reuptake inhibitor bupropion with the opioid antagonist naltrexone. Bupropion monotherapy is FDA approved as an antidepressant and to assist with smoking cessation. Inhibiting reuptake of dopamine and norepinephrine modulates the central reward pathways triggered by food. Naltrexone monotherapy is FDA approved for the treatment of alcohol and opioid dependence. Naltrexone antagonizes an inhibitory feedback loop that would otherwise limit bupropion's anorectic properties, and the combination of naltrexone/bupropion has been shown to activate POMC neurons in the arcuate nucleus of the hypothalamus **(24).**

Ideal candidates for naltrexone/bupropion include patients who describe strong cravings for food and/or addictive behaviors related to food. Patients who have concomitant depression, are trying to quit smoking or decrease alcohol consumption are also good candidates **(24).**

- **Liraglutide**

Liraglutide (3 mg daily) is a glucagon-like peptide-1 (GLP-1) receptor agonist (GLP1-RA) currently approved in both the United States and Europe for the treatment of obesity even in the absence of T2DM, which was its original indication. An initial dose-finding study showed that the optimal dose for weight loss is 3 mg daily, as opposed to 1.8 mg daily currently approved for T2DM. The subsequent SCALE clinical program displayed substantial weight loss for up to 3 years **(25).** Liraglutide was initially approved for the management of T2DM and later on, based on the results of clinical trials demonstrating the ability of GLP-1 analogs to induce weight loss, it was also approved as a weight loss agent. The weight loss with liraglutide appears to be mediated by appetite suppression and delayed gastric emptying **(26).**

- **Lorcaserin**

A serotonin (5-HT) agonist acting centrally to suppress appetite. It facilitates sustained weight loss without an increase in cardiovascular risk factors **(27).**

Minimal invasive therapy

- **Intragastric balloon:**

IT is a temporary and minimally invasive therapy for weight loss, currently being the main choice for mild obesity. As a space-occupying device, it reduces stomach capacity, resulting in decreased hunger and food intake. There are different balloon models, filled with liquid or air. The most used is the non-adjustable liquid-filled balloon, due to its lower rate of complications (Fig. 1). The mechanism of action is multifactorial, involving physiological and neurohormonal changes. The device functions as an artificial bezoar, filling the stomach and leading to early satiety (**28).**

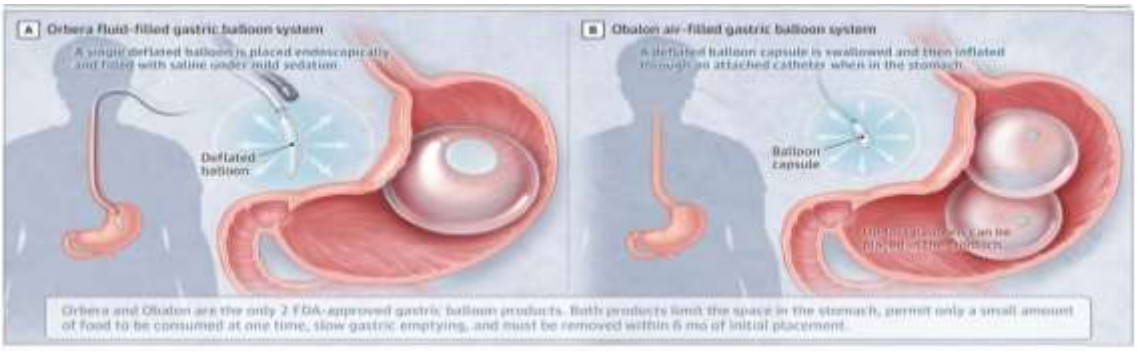


Figure (1): US Food and Drug Administration (FDA)–Approved Gastric Balloon Products for Weight Loss. **(28)**

Surgical Treatment of Morbid Obesity

Joint guidelines from the American Association of Clinical Endocrinologists, the Obesity Society, and the American Society for Metabolic and Bariatric Surgery advise that WLS should be considered for patients whose BMI is over 40 regardless of comorbidities, for patients with BMI of 35–40 in the presence of a severe obesity-related comorbidity, and for patients with BMI 30–35 in the presence of a severe obesity-related comorbidity such as diabetes. Recent guidelines recommend WLS for any diabetic patient with BMI over 40 or with BMI 35–40 and poor glycemic control despite aggressive medical therapy. They advise consideration of WLS for those with poorly controlled diabetes in patients with a BMI between 30 and 35 **(29).**

WLS reliably induces rapid, marked, and durable weight loss among obese patients, and reduces the burden of multiple obesity-associated comorbidities including diabetes, cardiovascular disease including hypertension, stroke, coronary artery disease, and heart failure. WLS is also effective in improving OSA. Excess weight and diabetes are associated with risk of multiple primary cancers, and WLS may protect against malignancy **(30).**

Types of bariatric surgery

The history of bariatric surgery has been described as a science that has progressed not as a single idea by one person, but rather in small collaborative steps that take decades to accept. The first recorded case of a bariatric procedure was in 1952 by a Swedish surgeon, **Dr Victor Henrikson**. He noticed that small bowel resections performed for other disease processes usually produced no change in the patient's general status however, in some cases, resulted in significant weight loss. on his observations, he resected 105 cm of small intestine from a 32-year-old obese female who could not complete a weight loss program. Interestingly, the patient lost only a small amount of weight but was noted to have an improved quality of life **(30).**

The quest to develop a surgical option with improved weight loss and acceptable complications resulted in six dominant bariatric procedures to date. Chronologically, these procedures are: jejunoileal bypass (JIB), Roux-en-Y gastric bypass (RYGB), vertical banded gastroplasty (VBG), biliopancreatic diversion with or without duodenal switch (BPD-DS), adjustable gastric banding (AGB), and SG **(Fig. 2)**. The JIB was first reported by Kremen and colleagues in 1954, but first performed by Dr. Varco in 1953. Although it achieved excellent weight loss, the procedure was abandoned because of severe complications. Similarly, the

VBG fell out of favor due to complications and disappointing weight loss, and complications such as gastric outlet obstruction and mesh erosion **(31).**

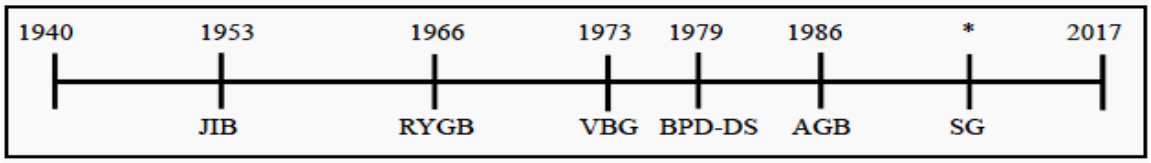
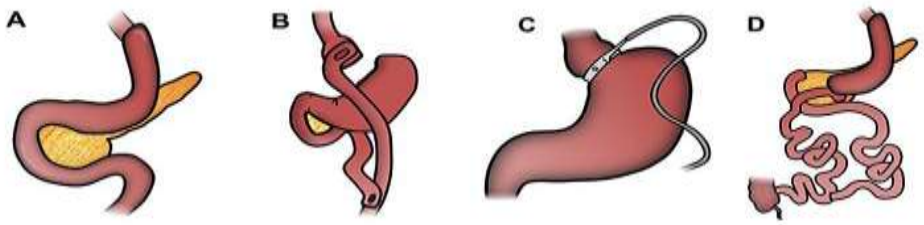


Fig. (2): Chronological bariatric procedures. JIB: jejunoileal bypass; RYGB: Roux-en-Y gastric bypass; VBG: vertical banded gastroplasty; BPD-DS: biliopancreatic diversion with duodenal switch; AGB: adjustable gastric banding; SG: sleeve gastrectomy**. (31)**

Bariatric surgery is currently most often performed laparoscopically. Laparoscopy became the standard approach due to its low perioperative risk, reduced complications, improved patient recovery, and reduced mortality **(Fig. 3).** From the original six bariatric procedures initially developed, four major procedures are currently performed: 1) RYGB; 2) BPD-DS; 3) AG B; and 4) SG **(32)**.



Type of Procedure	SG	RYGB	ABG	BPD-DS
In-hospital Mortality	.04%	.08%	.05%	.33%
In-hospital Morbidity	5.3%	9.4%	5.8%	10.6%
BMI Weight Loss at 1 year (kg/m2)	13.6	15.8	7.2	19.9

Figure (3): Bariatric procedure type and outcomes SG: sleeve gastrectomy; RYGB: Roux-en-Y gastric bypass; AGB: adjustable gastric banding; BPD-DS: biliopancreatic diversion with duodenal switch; BMI: Body Mass Index **(32)**.

I. Laparoscopic sleeve gastrectomy

As a primary bariatric procedure for morbidly obese patients (BMI = 40 kg/m2 – 2.5 BMI points lower for Asians) with multiple failed attempts at weight loss using conservative, non-surgical methods. As a primary bariatric procedure for patients with BMI = 35 kg/m2 (BMI = 35 kg/m2 – 2.5 BMI points lower for Asians) with 2 or more comorbidities. As an adjunct to metabolic procedures like iliac transposition, duodeno-jejunal bypass or jejuno-ileostomy. Preferred bariatric procedure in Asian countries like Korea and Japan which are endemic for cancer of the stomach. Patients with a large hiatal hernia, grade 3 or 4 flap valve on endoscopy (as per Hill‘s classification) or grade C or D esophagitis, as per Los Angeles (LA) classification. Pyloric outlet obstruction **(33).**

Contraindications

Specific contraindications for LSG are few and include the Barrett’s esophagus. It is generally agreed that LRYGB is superior to LSG in patients with long-standing diabetes, although recurrent diabetes is easier to treat after sleeve than bypass. Other general

contraindications apply, as for any bariatric surgical procedure, and include the American Society of Anesthesiologists (ASA) grade 4 patients not likely to withstand the surgery, patients with end-stage organ dysfunction of the heart, lungs, or both that are unlikely to improve, patients with malignancy, and cirrhotic liver with severe portal hypertension. **(34).**

II. Adjustable gastric banding (AGB)

The laparoscopic AGB involves the placement of an adjustable band around the upper portion of the stomach, creating a gastric pouch above the band. The diameter of the opening between the gastric pouch and the rest of the stomach can be adjusted by adding or removing saline from the balloon inside the band **(35).**

III. The laparoscopic Roux-en-Y gastric bypass (RYGB)

The laparoscopic RYGB is considered the gold standard for bariatric surgery. First, a small gastric pouch, approximately 30ml in size, is created by stapling and separating the cardia from the rest of the stomach **(Fig. 4)**. Then, the jejunum is divided into two limbs 30-40 cm distal to the ligament of Treitz. The alimentary limb (distal jejunum segment, or Roux limb) is brought up and connected to the newly created gastric pouch. Then the proximal limb of the resected jejunum is connected to the Roux limb at approximately 75-150 cm from its connection to the gastric pouch **(36).**

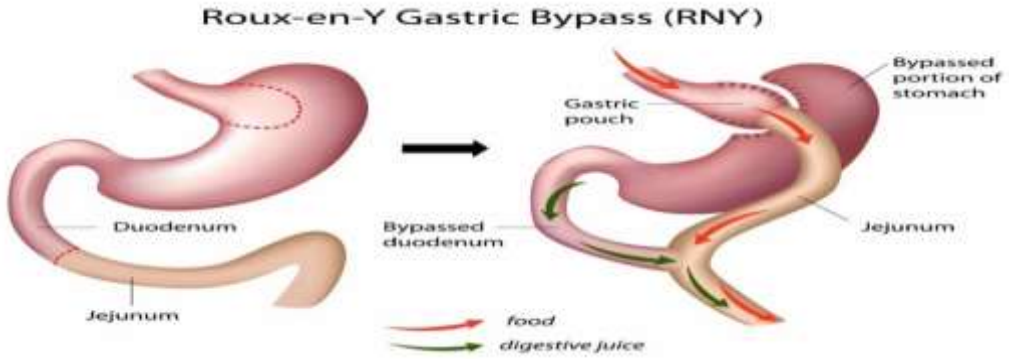


Figure (4) Roux-en-Y gastric bypass **(36)**

IV. The laparoscopic bilio-pancreatic diversion with duodenal switch (BPD/DS)

The Duodenal Switch (DS) is a mixed operation that consists of two techniques, a gastric surgery, the Sleeve-forming Vertical Gastrectomy (SFG) to reduce intake and also an intestinal surgery, the bilio-pancreatic diversion (BPD) that produces intestinal malabsorption. It is the most complex operation in Bariatric Surgery (BS) **(37).** This operation is now the least performed operation worldwide and has fallen into disfavor due to unacceptable rates of severe nutritional deficiencies and high rates of reversals or revisions, problems that have been known since the mid-1980s. Numbers have shown a progressive decline in the United States, as well as worldwide **(38).**

Complications bariatrics surgeries and management:

I. Early complications:

- **Haemorrhage**

The incidence of hemorrhage after LSG is reported to range from 1 to 6 %. The hemorrhage can be extraluminal or intraluminal. The causes of extraluminal hemorrhage are

bleeding from the staple line, omental vessel, spleen injury, liver laceration or trochar sites. Intraluminal bleeding is uncommon, and is a result of staple line bleed. Patients with extraluminal hemorrhage ususally experience tachycardia, sudden hypotension, and sanguineous drain output, with adrop in hematocrit. Patients are resuscitated and serial monitoring of pulse rate, blood pressure, and haematocrit is done. An urgent relaparoscopy or laparotomy should be done if bleeding results in hypotension, especially within 12 h after LSG. In most experiences, most patients can be managed conservatively if they are hemodynamically stable. Anticoagulants should always be discontinued in such cases **(39)**.

- **Leakage**

The rate of staple line and anastomotic leaks in adult bariatric literature has historically been reported to be as high as 4–5% for both LSG and RYGB. On the other hand, staple line leak after pediatric LSG appear to occur rather rarely (0.9–1.9%). The factors increasing the leak rates are thought to be related to inadvertent inclusion of weaker esophagogastric junction tissue in the cephalic portion of the staple line and creation of a relative distal obstruction due to significant narrowing or twisting of the gastric sleeve near the incisura. Avoidance of tension and twisting of the stomach. (**40)**

Leakage is the most dreaded complication after bariatric surgery is a leak from the staple line. Leak can be classified as early or late, depending upon the time interval of presentation after surgery. Early leak is defined as a leak that is diagnosed within 3 days after surgery. Late leaks are those diagnosed a week after surgery. The presentation of leak is often varied ranging from absence of symptoms to diffuse peritonitis. The earliest signs of leak are tachycardia, agitation, tachypnea, and fever. Pulse rate is the single most reliable parameter to diagnose leak in obese patients. Any tachycardia or fever warrants further evaluation by contrast enhanced computer tomography of the abdomen and/or gastrografin study to diagnose the leak. If the leak is diagnosed or suspected within 48–72 h, relaparoscopy is done. At the time of relaparoscopy, the leak is repaired, peritoneal lavage is done, a drain is placed, and a feeding jejunostomy is done. After 72 h, repair of leak is not recommended because of the extensive inflammatory changes. **(41)**.

II. Late complications:

- **Stricture**

The incidence of stricture following LSG is reported to be 1–2 % **(42)**. Stenosis can result from surgical technique or ischemia with subsequent stricture development. Clinically significant stenosis occurs in 0.5%–3.5% of cases, most often a short segment located at mid-body, near the incisura. Initial management consists of endoscopic balloon dilatation. Many patients require two to four dilation sessions, with long-term success rates reported at 95%–100%. The risk of perforation associated with dilation is 2%–5%. For those patients in whom endoscopic dilation fails, treatment options include endoscopic stenting, longitudinal seromyotomy, median gastrectomy with gastrogastric anastomosis, and conversion to Roux-en-Y gastric bypass **(42)**.

Chronic strictures usually require further intervention. These include either endoscopic or surgical treatments. Treatment options depend on the length of stenosis. Endoscopic dilatation is an invaluable tool used in this setting of a short segment stenosis **(43).**

Successive treatments in 4- to 6-week intervals are adequate to treat stricture and ameliorate patient symptoms. In contrast, long segment stenosis and failure of endoscopic

management demands a surgical intervention. Options include laparoscopic or open seromyotomy or conversion to Roux-en-Y gastric bypass **(44).**

- **Gastroesophageal reflux disease**

The most frequent complication of LSG is gastroesophageal reflux disease (GERD). The Second International Consensus Summit for Sleeve Gastrectomy stated that the average incidence of de novo reflux symptoms after this form of bariatric surgery is 6.5%. However, the incidence varies widely between centers with more than 1 in 5 patients complaining of new reflux symptoms after LSG in some reports, and underreporting by patients on acid suppressants may be an issue **(45).**

For those patients who do develop reflux after LSG, treatment usually consists of proton-pump inhibitor medication or conversion to Roux-en-Y bypass. A recent pilot study including 6 selected patients demonstrated feasibility of a laparoscopic anterior fundoplication of the proximal sleeve with posterior crura approximation for treatment of post-LSG reflux, given that preoperative evaluation demonstrated enough tissue volume of the proximal gastric pouch to allow fundoplication **(46).**

- **Nutritional deficiency**

There is a high prevalence of micronutrient deficiency among obese patients prior to bariatric surgery. These nutrient deficiencies may increase or occur de novo after the bariatric procedure. The mechanism of their development differs according to surgical procedures and individuals. Vitamin B_{12}, folic acid, iron, calcium, and thiamine are some of the most common nutritional deficiencies among patients who undergo bariatric surgery for weight loss. However, there are not many specific reports of nutritional deficiencies among SG patients. Patients undergoing SG are at risk of these deficiencies because of decreased hydrochloric acid and intrinsic factor secretion, reduced intake, poor food choices, postoperative vomiting and nausea, as well as food avoidance due to intolerance **(47).**

- **Gallstone disease**

Patients who have had bariatric operation develop gallstones at a higher incidence than the average population. Alterations in enterohepatic circulation, hormonal changes associated with weight loss, and perhaps increased biliary stasis contribute to the development of cholelithiasis. Bariatric operation results in rerouting of food through the alimentary limb and may change or delay the release of the usual gut hormones that stimulate gallbladder contraction, resulting in atypical symptoms or non-postprandial pain. Symptomatic cholelithiasis and cholecystitis can be treated with laparoscopic cholecystectomy **(48).**

However, the management of choledocholithiasis is complicated because the usual route to the ampulla of Vater for endoscopic retrograde cholangiopancreatography (ERCP) is bypassed. Pediatric colonoscopes or double-balloon endoscopy can allow highly skilled endoscopists to pass a scope all the way down the alimentary limb through the JJA and back up the biliopancreatic limb to the ampulla of Vater, but this is time-consuming and not always in the armamentarium of the endoscopist. Hence, the three options available to the surgeon for treatment of choledocholithiasis after gastric bypass are percutaneous transhepatic cholangiography, surgical common bile duct exploration, or the so-called "rendezvous" procedure where the surgeon laparoscopically provides access to the bypassed stomach

remnant to allow the gastroenterologist to approach the ampulla of Vater with a standard side-viewing ERCP scope. Biliopancreatic diversion/DS patients have only the first two options, as these patients typically do not have retained stomach for this access. Some institutions have created algorithms to treat these patients that require complex multidisciplinary procedures (**49,50).**

CONCLUSION:

Obesity surgery is justified as it produces sustained weight loss, increased life expectancy and reduces the complications of obesity. For this reason, increasing numbers of patients are undergoing this surgery. Bariatric surgery has been shown to affect several of the key obesity-related hormones.

General practitioners and emergency departments must be aware of the management of these patients. Their complications are rare and the most insidious are deficiencies which may result in irreversible neurological complications. If the least doubt is present, a medical or surgical consultation should be requested with a specialist practitioner in the management of obese patients as death rates increase with delayed diagnosis.

No conflict of interest.

References:

1- Nittari, G., Scuri, S., Sagaro, G. G., Petrelli, F., & Grappasonni, I. (2020). Epidemiology of obesity in children and adolescents. Teamwork in Healthcare, 20.
2- World Health Organization. (2020). The double burden of malnutrition: Priority actions on ending childhood obesity. World Health Organization. Regional Office for South-East Asia.
3- Gebremichael, M. A., Mengesha, M. M., Hailegebrea, S., Abdulkadir, H., & Wolde, B. B. (2021). Prevalence of Overweight/Obesity and Associated Factors Among Under-Five Children in Ethiopia: Evidence From the 2019 Ethiopia Mini Demographic and Health Survey (Emdhs); A Multilevel Analysis.
4- Deniz, S., Şirin, H., Kıvrak, M., Kaplan, Z., Ketrez, G., & Üner, S. (2020). Factors associated with overweight and obesity in students of 5-14 age group in Mersin. Gülhane Tip Dergisi, 62(4), 245.
5- Soliman, N., Huseth-Zosel, A., & Soliman, A. T. (2021). Obesity and overweight in children below 5 years of age: environmental, behavioural and social factors and their active use in prevention and management. World Nutrition, 12(4), 190-212.
6- Le, G. B. (2022). Prevalence and associated factors of overweight and obesity among primary school children: a cross-sectional study in Thanhhoa City, Vietnam. BMJ open, 12(4), e058504.
7- Tanveer, M., Hohmann, A., Roy, N., Zeba, A., Tanveer, U., & Siener, M. (2022). The current prevalence of underweight, overweight, and obesity associated with demographic factors among Pakistan school-aged children and adolescents—An empirical cross-sectional study. International Journal of Environmental Research and Public Health, 19(18), 11619.
8- Oray, M., Abu Samra, K., Ebrahimiadib, N., Meese, H., & Foster, C. S. (2016). Long-term side effects of glucocorticoids. Expert opinion on drug safety, 15(4), 457-465.
9- Gunay-Aygun, M., Cassidy, S. B., & Nicholls, R. D. (1997). Prader–Willi and other syndromes associated with obesity and mental retardation. Behavior genetics, 27, 307-324.
10- Crinò, A., Fintini, D., Bocchini, S., & Grugni, G. (2018). Obesity management in Prader–Willi syndrome: Current perspectives. Diabetes, metabolic syndrome and obesity: targets and therapy, 579-593.

11- Diamanti-Kandarakis, E., Papalou, O., Kandaraki, E. A., & Kassi, G. (2017). Mechanisms in endocrinology: nutrition as a mediator of oxidative stress in metabolic and reproductive disorders in women. European journal of endocrinology, 176(2), R79-R99.
12- Barber, T. M., & Franks, S. (2021). Obesity and polycystic ovary syndrome. Clinical endocrinology, 95(4), 531-541.
13- Gordon-Elliott, J. (2016). Fundamentals of diagnosing and treating eating disorders: A clinical casebook. Springer.
14- Thaler, J. P., Guyenet, S. J., Dorfman, M. D., Wisse, B. E., & Schwartz, M. W. (2013). Hypothalamic inflammation: marker or mechanism of obesity pathogenesis?. Diabetes, 62(8), 2629-2634.
15- Rippe, J. M., & Hess, S. (1998). The role of physical activity in the prevention and management of obesity. Journal of the American Dietetic Association, 98(10), S31-S38.
16- Hills, A. P., & Byrne, N. M. (2004). Physical activity in the management of obesity. Clinics in Dermatology, 22(4), 315-318.
17- Leermakers, E. A., Dunn, A. L., & Blair, S. N. (2000). Exercise management of obesity. Medical Clinics, 84(2), 419-440.
18- Hession, M., Rolland, C., Kulkarni, U., Wise, A., & Broom, J. (2009). Systematic review of randomized controlled trials of low-carbohydrate vs. low-fat/low-calorie diets in the management of obesity and its comorbidities. Obesity reviews, 10(1), 36-50.
19- Botchlett, R., & Wu, C. (2018). Diet composition for the management of obesity and obesity-related disorders. Journal of diabetes mellitus and metabolic syndrome, 3, 10.
20- Patel, D. (2015). Pharmacotherapy for the management of obesity. Metabolism, 64(11), 1376-1385.
21- Henness, S., & Perry, C. M. (2006). Orlistat: a review of its use in the management of obesity. Drugs, 66, 1625-1656.
22- Smith, S. M., Meyer, M., & Trinkley, K. E. (2013). Phentermine/topiramate for the treatment of obesity. Annals of Pharmacotherapy, 47(3), 340-349.
23- Sweeting, A. N., Tabet, E., Caterson, I. D., & Markovic, T. P. (2014). Management of obesity and cardiometabolic risk–role of phentermine/extended release topiramate. Diabetes, metabolic syndrome and obesity: targets and therapy, 35-44.
24- Apovian, C. M. (2016). Naltrexone/bupropion for the treatment of obesity and obesity with Type 2 diabetes. Future cardiology, 12(2), 129-138.
25- Scott, L. J. (2015). Liraglutide: a review of its use in the management of obesity. Drugs, 75, 899-910.
26- Lin, C. H., Shao, L., Zhang, Y. M., Tu, Y. J., Zhang, Y., Tomlinson, B., Liu, Z. (2020). An evaluation of liraglutide including its efficacy and safety for the treatment of obesity. Expert opinion on pharmacotherapy, 21(3), 275-285.
27- Hess, R., & Cross, L. B. (2013). The safety and efficacy of lorcaserin in the management of obesity. Postgraduate medicine, 125(6), 62-72.
28- Shah, R., Davitkov, P., Dayyeh, B. K. A., Saumoy, M., & Murad, M. H. (2021). AGA technical review on intragastric balloons in the management of obesity. Gastroenterology, 160(5), 1811-1830.
29- Farina, M. G., Baratta, R., Nigro, A., Vinciguerra, F., Puglisi, C., Schembri, R., ... & Frittitta, L. (2012). Intragastric balloon in association with lifestyle and/or pharmacotherapy in the long-term management of obesity. Obesity surgery, 22, 565-571.
30- Shah, R., Davitkov, P., Dayyeh, B. K. A., Saumoy, M., & Murad, M. H. (2021). AGA technical review on intragastric balloons in the management of obesity. Gastroenterology, 160(5), 1811-1830.
31- Hachem, A., & Brennan, L. (2016). Quality of life outcomes of bariatric surgery: a systematic review. Obesity surgery, 26, 395-409.
32- Piche, M. E., Auclair, A., Harvey, J., Marceau, S., & Poirier, P. (2015). How to choose and use bariatric surgery in 2015. Canadian Journal of Cardiology, 31(2), 153-166.
33- Diamantis, T., Apostolou, K. G., Alexandrou, A., Griniatsos, J., Felekouras, E., & Tsigris, C. (2014). Review of long-term weight loss results after laparoscopic sleeve gastrectomy. Surgery for Obesity and Related Diseases, 10(1), 177-183.

34- Pequignot, A., Prevot, F., Dhahri, A., Rebibo, L., Badaoui, R., & Regimbeau, J. M. (2015). Is sleeve gastrectomy still contraindicated for patients aged≥ 60 years? A case-matched study with 24 months of follow-up. Surgery for Obesity and Related Diseases, 11(5), 1008-1013.
35- Lazzati, A., De Antonio, M., Paolino, L., Martini, F., Azoulay, D., Iannelli, A., & Katsahian, S. (2017). Natural history of adjustable gastric banding: lifespan and revisional rate. Annals of surgery, 265(3), 439-445.
36- Lee, W. J., Ser, K. H., Lee, Y. C., Tsou, J. J., Chen, S. C., Chen, J. C. (2012). Laparoscopic Roux-en-Y vs. mini-gastric bypass for the treatment of morbid obesity: a 10-year experience. Obesity surgery, 22, 1827-1834.
37- Biertho, L., Simon-Hould, F., Marceau, S., Lebel, S., Lescelleur, O., & Biron, S. (2016). Current outcomes of laparoscopic duodenal switch. Annals of surgical innovation and research, 10, 1-5.
38- Marceau, P., Biron, S., Marceau, S., Hould, F. S., Lebel, S., Lescelleur, O., Kral, J. G. (2014). Biliopancreatic diversion-duodenal switch: independent contributions of sleeve resection and duodenal exclusion. Obesity surgery, 24, 1843-1849.
39- Lim, R., Beekley, A., Johnson, D. C., & Davis, K. A. (2018). Early and late complications of bariatric operation. Trauma surgery & acute care open, 3(1).
40- Montravers, P., Augustin, P., Zappella, N., Dufour, G., Arapis, K., Chosidow, D., Desmard, M. (2015). Diagnosis and management of the postoperative surgical and medical complications of bariatric surgery. Anaesthesia Critical Care & Pain Medicine, 34(1), 45-52.
41- Walsh, C., & Karmali, S. (2015). Endoscopic management of bariatric complications: a review and update. World Journal of Gastrointestinal Endoscopy, 7(5), 518.
42- Vedantam, S., & Roberts, J. (2020). Endoscopic stents in the management of bariatric complications: our algorithm and outcomes. Obesity Surgery, 30, 1150-1158.
43- Hamdan, K., Somers, S., & Chand, M. (2011). Management of late postoperative complications of bariatric surgery. Journal of British Surgery, 98(10), 1345-1355.
44- Abell, T. L., & Minocha, A. (2006). Gastrointestinal complications of bariatric surgery: diagnosis and therapy. The American journal of the medical sciences, 331(4), 214-218.
45- Richter, J. E. (2013). Gastroesophageal reflux disease treatment: side effects and complications of fundoplication. Clinical Gastroenterology and Hepatology, 11(5), 465-471.
46- Ashrafi, D., Osland, E., & Memon, M. A. (2020). Bariatric surgery and gastroesophageal reflux disease. Annals of Translational Medicine, 8(Suppl 1).
47- Becker, D. A., Balcer, L. J., & Galetta, S. L. (2012). The neurological complications of nutritional deficiency following bariatric surgery. Journal of obesity, 2012.
48- Portincasa, P., Di Ciaula, A., de Bari, O., Garruti, G., Palmieri, V. O., & Wang, D. H. (2016). Management of gallstones and its related complications. Expert review of gastroenterology & hepatology, 10(1), 93-112.
49- Morais, M., Faria, G., Preto, J., & Costa-Maia, J. (2016). Gallstones and bariatric surgery: to treat or not to treat?. World journal of surgery, 40, 2904-2910.
50- Chang, J., Corcelles, R., Boules, M., Jamal, M. H., Schauer, P. R., & Kroh, M. D. (2016). Predictive factors of biliary complications after bariatric surgery. Surgery for Obesity and Related Diseases, 12(9), 1706-1710.