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Obesity and Microbes: The Role of Bariatric Surgery

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Abstract

Obesity is a worldwide ongoing pandemic and its consequences have a dramatic effect on society. Bariatric surgery is a key method to solve obesity, with 800,000 ca cases worldwide yearly and high outcome variability, which includes gut microbiota change. The relationship between weight and gut microbiota should be studied as it can affect the efficacy of weight-losing techniques.

Introduction

Obesity is defined as the accumulation of excess fat in the body that can compromise health: this corresponds to a BMI equal to or greater than 30 kg/m^2, with three different likely levels that are class I (BMI = 30.0 – 39.9 kg/m^2), class II (BMI = 40.0 – 49.9 kg/m^2) and class III (BMI > 50 kg/m^2) [1].

Obesity is considered a risk factor for various chronic illnesses such as cardiovascular diseases and hypertension, type 2 diabetes and thyreopathies, non-alcoholic fatty liver disease, and cancer. Over the past thirty years, the prevalence of obesity is rapidly getting higher not only in developed countries but also in developing countries. Even though physical inactivity and excessive food intake are usually thought of as the cause of obesity, its etiology is quite complex. There are many factors to be taken into account, such as environment, genetics, and lifestyle [2]. Besides, the microbiota has been reported as one of the key factors in obesity etiology [3-6].

Obesity is one of the easiest diseases to diagnose but one of the hardest to treat and it should be treated effectively to avoid its consequences on health. Obesity management must be planned in a personalized way [7]. At the moment, treatment methods for obesity are behavior modification therapy, diet therapy, medical treatment, and surgical treatment [8]. Surgery is not always the first choice and it should be applied only if the appropriate indications are present; after its application, body weight loss occurs with changes in the metabolism of bile acids, gastric pH, the metabolism of hormones, and in microbiota [9].

Bariatric Surgery

Bariatric surgery is one of the most effective therapeutic treatments for obesity and complications [10]. Thanks to it, long-term permanent body weight loss is achieved, metabolic effects of obesity are reduced, many diseases are prevented and quality of life is markedly increased [11]. Body weight loss with bariatric surgery is fulfilled through the change of food preferences, reduction of nutrient digestion, acceleration of gastric void, regulation of hormonal fluctuation (e.g. glucagon-like peptide 1, GLP-1, and peptide tyrosine tyrosine, PYY), and alterations in the metabolism of bile acids. In spite of the fact that bariatric surgery is suitable for obesity treatment, some complications can rarely occur and they should be taken into account in evaluating surgical risk. These are gastroesophageal reflux, nutritional deficiencies, gastric outlet obstruction, mesh erosion and marginal ulcerations, slippage, and internal herniation [12]. Indications for bariatric surgery were established by the United States National Institute of Health in 1991 (Table 1) [13].

There are various bariatric surgical methods according to their effect mechanisms (Figure 1) [14].

Surgical techniques

The most common surgical techniques in bariatric surgery are described as follows.

RYBG: Roux-en-Y gastric bypass is the gold standard in the surgical treatment of obesity so it is the most commonly practiced bariatric surgery in the world [15]. This method consists of two steps. First of all, the stomach capacity is left to be about 30 cm3; Roux sputum can then be pulled up from the stomach, the front of the colon and back of the stomach, or behind the colon and stomach for gastro-jejunostomy [16]. The input of food and energy goes down due to the reduction in stomach volume. In certain cases, fat malabsorption can happen [17].

LSG: Laparoscopic sleeve gastrectomy consists of the removal of 80% ca of the lateral aspect of the stomach in a vertical fashion, leaving a long gastric tube [18]. LSG is preferred for patients who have super obesity and a BMI < 50 kg/m^2 [19]. Due to the reduction of gastric volume, nutrient, and energy intake are restricted; however, there is a reduction in plasma levels of ghrelin [20].

LAGB: Laparoscopic adjustable gastric banding involves the placement of an adjustable silicone band around the upper part of the stomach, thus forming a small gastric space over the gastric band. The size of the gap between the upper stomach space and the posterior

Table 1: Indication for surgical operation in obesity according to the United States National Institute of Health 1991.

Indication for surgical operation [13]
BMI=40 kg/m^2 (or) BMI > 35 kg/m^2 + type 2 diabetes, hypertension, sleep apnea, or hyperlipidemia
Acceptance of surgical risk
Failure of nonsurgical treatments
Psychiatric stability, no alcohol and drug dependence
Well-established motivation, knowledge of the operation and its sequelae
No medical problems that will harm the surgeon
No uncontrolled psychotic and depressive disorder
Complete family and social support

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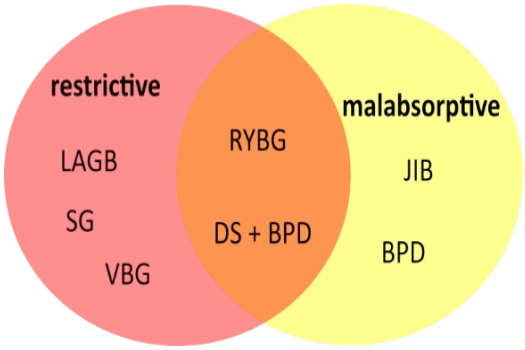

Figure 1: Scheme of the main techniques in bariatric surgery. **LABG**: laparoscopic adjustable gastric banding; **SG**: sleeve gastrectomy; **VBG**: vertical banding gastroplasty; **JIB**: jejunoileal bypass; **RYBG**: Roux-en-y gastric bypass; **DS**: Duodenal switch; **BPD**: biliopancreatic diversion [14].

Table 2: Behavioural and biochemical changes in RYBG, LSG and LAGB. RYBG: Roux-en-y gastric bypass, LSG: laparoscopic sleeve gastrectomy, LAGB: laparoscopic adjustable gastric banding. GLP-1: glucagon-like peptide 1, PYY: peptide tyrosine tyrosine.

Changes	RYBG	LSG	LAGB
Food intake	↓	↓	↓
Food transit time	↑	=	↓
Food choices	↓preference for food rich in fats and sugars	↓	↑preference for food poor in fibers
Chewing time	↑	↑	↑
Acid production	disrupted	=	=
Ghrelin	=	↓	=
GLP-1 and PYY	↑	=	=
Insulin	↓	↓	↓
Leptin	↓	↓	↓
Adiponectin	↑	↑	↑

part of the stomach can be tailored by filling it with sterile 0.9% saline solution injected through the abdominal wall. Adjustment of the band can be done piecemeal during postoperative follow-up [21]. This method provides body weight loss by reducing nutrient uptake with a completely restrictive effect [14].

BPD: Biliopancreatic diversion (BPD) is composed of three main components: a gastric tube with preserved pylorus, distal ileoanal anastomosis, and anastomosis of the proximal duodenal bile duct. Body weight loss is provided by the reduction of gastric volume and decrease of ghrelin hormone, increasing PYY, as it happens in LSG. In this technique, hormonal changes with anatomical changes are thought to be the route to body weight loss [22].

Surgery induces important changes in both intrinsic and extrinsic factors, as well as in the anatomy of the gastrointestinal system (Table 2) [23, 24].

The gut microbiome

The human body is colonized by a huge variety of microbes, commonly referred to as the human microbiota. It comprises commensal, symbiotic, and pathogenic microbes. Microbiome, instead, is the genetic pool of microbiota living in a specific place and their relation with the environment [25]. It is estimated that there are about 1,014 microorganisms in the human body, more than 70% of which are in the colon, and more than 35,000 bacterial strains in the gastrointestinal tract [26]. Microbiota is determined by various factors such as delivery type, breastfeeding time, transition time to complementary feeding, diet, and use of antibiotics from birth to death but also host weight and sugar consumption [27].

The gut microbiota is a complex ecosystem, which provides major functions to the host such as metabolism regulation, modulation of the immune system, and thus protection against pathogens [28-31]. Gut microbiota can be classified into six bacterial clusters in healthy individuals and these include Firmicutes (including gram+ve strains of Clostridium, Eubacterium, Ruminococcus, Butyrivibrio, Anaerostipes, Roseburia, Faecalibacterium, etc.), Bacteroidetes (including gram-negative strains of Bacteroides, Porphyromonas, Prevotella, etc.), Proteobacteria (including gram- strains such as Enterobacteriaceae), Actinobacteria (including the gram+ve Bifidobacterium genus), Fusobacteria and Verrucomicrobia (including Akkermansia, etc.) [32]. Bacteroidetes and Firmicutes form more than 90% of the total intestinal microbiota. The most important components of the human intestinal microbiota are obligate anaerobes of the genus Bacteroides, Eubacterium, Clostridium, Ruminococcus, Peptococcus, Peptostreptococcus, Bifidobacterium, and Fusobacterium and facultative anaerobes such as Escherichia, Enterobacter, Enterococcus, Klebsiella, Lactobacillus, and Proteus. Methanogenic archaea have also been pointed out and the most important in the human gut is Methanobrevibacter smithii [33]. Changes in microbiota content shape human health at a significant level. It is outlined that many non-communicable diseases such as obesity, type 2 diabetes, asthma and allergies, inflammatory bowel disease, metabolic syndrome, and atherosclerosis are intimately linked with gut microbiota [34].

Gut disbiosis in obesity

It is reported that genetic and environmental factors affect obesity etiology. Researchers have also noted that intestinal microbiota contributes to the regulation of energy and fat metabolism and that it affects obesity and its complications [35]. It has been outlined that obese patients have less variability in the intestinal microbiota than normal-weight individuals [36]. The fundamental function that splits up microbial strains from obese and thin individuals is the inability to obtain fermentation; another difference is that short-chain fatty acids cannot be produced from indigestible food [37].

Intestinal microbiota studies in both human and animal models have helped in understanding the role of microbial activity in the etiology of obesity. It has been outlined that patients with obesity have fewer Bacteroidetes and more Firmicutes in their microbiota than normal-weight people. It is well known that diets rich in saturated fatty acids lead to obesity and hepatic steatosis, increasing the Firmicutes/Bacteroidetes ratio in the gut microbiota [38]. On the other hand, fat and carbohydrate-restricted diets and body weight loss cause the amount of Bacteroidetes to increase and thus the Firmicutes/Bacteriodetes ratio to decrease [39]. These shreds of evidence are controversial so that some studies show that there is no relationship between BMI and Firmicutes/Bacteroidetes [40,41] although other studies display an increase in Firmicutes/Bacteroidetes ratio in obesity and insulin resistance. Reduction of carbohydrate intake in obese patients decreases the butyrate levels in feces which corresponds to a decrease in the level of Roseburia spp. and Eubacterium rectale [42]. The microbiota is affected by body weight loss caused by diet and exercise. It has been reported that the quantities of Bacteroides and Lactobacillus increase as a result of energy restriction and exercise in patients with obesity. Nonetheless, no changes were seen in overweight adolescents who lost less than 2 kg in body weight [43].

Obesity can affect the human immune system in a significant way and gut microbiota is likely to express a notable function. This is a controversial statement because there is much disagreeing evidence in scientific literature. For example, it has been outlined that a higher BMI compromises immunization following COVID-19 vaccination [44] but other studies, instead, have reported that is no relationship between BMI and COVID-19 severity, even in the most critical cases [45]. Moreover, it has been stated that obesity could have a protective role against infectious diseases: that is the case of pouchitis by Clostridioides difficile. [46]

What is true is far from easy to be defined. This is mostly due to the fact that obesity is complex to the extent that it cannot be evaluated as a single variable. In any case, intestinal microbiota modification may be a therapeutic treatment for the prevention or even reversal of obesity.

Bariatric surgery and microbiota

It is reported that significant changes in gut microbiota occur after bariatric surgery; the most likely mechanisms include changes in food choices and preferences, reduction of food intake, and nutrient malabsorption [47].

First of all, short-term dietary changes may cause fast changes in the composition of intestinal microbiota. As an example, it has been outlined that Prevotella enterotypes are associated with complex carbohydrate-rich and simple carbohydrate-rich diets, whereas Bacteroidetes enterotype is correlated with a typical "Western diet", full of animal protein and saturated fatty acids [48]. In detail, some diets can affect the quantity of specific strains of gut microbiota, such as diets low in fats and high in carbohydrates but also diets high in carbohydrates with a low glycemic index [49].

A second factor regulating the change in gut microbiota after bariatric surgery has been stated to be bile acids [8]. Bile acids can rule their synthesis and their intestinal reabsorption through modulation of the nuclear-located farnesoid X receptor (FXR). Another pathway of auto regulation is the G-linked protein TGR5, but this pathway is yet to be 100% understood [50]. Recently, the physiological role of bile acids has been associated with pancreatic beta cell function and thus glucose homeostasis but also energy consumption. Even these roles of bile acids are correlated with FXR and TGR5 pathways [8]. Bile and pancreatic secretions are separated from nutrients in RYGB and they come together only in the more distal part of the intestine; as a result, the distal jejunum and proximal ileum are excessively exposed to the nutrients. Dietary lipids are surrounded by the bile acids, while bile acids cycling in the upper intestine become blunted: this leads to an increase of serum bile acids level and of serum FGF15/19 levels that normalize the postprandial bile acids answer after surgery [51]. The pathway underlying the beneficial effects of bariatric surgery has been outlined to be changes in bile acids metabolism [43]. The change in bile acids flow has a definite effect on the alterations in gut microbiota after bariatric surgery, too. In the proximal jejunum, the absence of nutrient transit and the decrease in mobility alter the number of bacteria [24]. The changes in bile acids flow also change the 7α-dehydroxylation capacity of the intestinal microbiota, which is implied in the synthesis of the secondary (intermediate) bile acids. In these terms, administration of a diet supplemented with the primary bile acid colic acid to rats increases the presence of Firmicutes, which contains the enzyme 7α-hydroxylase such as *Clostridium* spp [52].

Even hormones, such as leptin and ghrelin, may change after bariatric surgery. Hormonal changes are linked to both energy metabolism and microbiota [53]. Despite the relationship between gut microbiota and ghrelin is not clearly comprehended, prebiotics are reported to modulate gut microbiota and decrease serum ghrelin levels [54]. On the other hand, leptin has a controversial role. Serum leptin levels have been outlined to have a positive correlation with Mucispirillum, Lactococcus. Another study stated that leptin has a positive correlation with Bifidobacterium and Lactobacillus whilst a negative correlation with Bacteroides, Clostridium, and Prevotella [55]. Researchers have emphasized that further studies are necessary, even though hormones have been reported to influence the intestinal microbiota [38, 56-58].

Another important factor affecting microbiota is changes in pH. After surgery, pH increases as the volume of the stomach decreases. The changing pH influences every part of the gastrointestinal system after the stomach. Increased pH can affect microbiota at an important level. It has been reported that Bacteroidetes decrease due to pH fluctuations after surgery, while Firmicutes and Actinobacteria increase [59].

After bariatric surgery, microbiota diversity changes due to the reasons mentioned above. Table 3 briefs how microorganisms are affected by bariatric surgery [8].

Table 3: Bacterial diversity of the intestinal microbiota after surgery.

↑ bacteria	↓ bacteria
Proteobacteria	Gelicobacter spp
Gammaproteobacteria	Treponema pallidum
Escherichia coli	Bachyspira hyodysenteriae
Klebsiella pneumoniae	Archae spp
Shigella boydii	
Salmonella enterica	
Enterobacter cancerogenus	
Enterobacter hormaechei	
Citrobacter spp	
Pseudomonas spp	
Enterococcus faecalis	
Fusobacterium nucleatum	

Conclusion

Bariatric surgery is one of the main treatments of obesity. It is considerably effective in achieving and protecting weight loss. The effectiveness of obesity treatments after bariatric surgery is not only related to food consumption but also to microbiota alteration. Malabsorption status after bariatric surgery, changes in the metabolism of bile acids, gastric pH, and the metabolism of hormones give rise to gut microbiota alteration. Changes in microbiota also influence energy homeostasis. Because of these reasons, microbiota should be highlighted as a key factor in body weight loss after bariatric surgery.

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