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KETOGENIC DIET AND BARIATRIC SURGERY: RISK REDUCTION PRE, INTRA AND PERI-OPERATORS

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Abstract

Many diseases are caused or influenced by environmental factors. The term environment includes the various environments in which the life of an individual takes place - external environments, confined environments and work environments. The air we breathe, the food and water we consume and the toxic substances to which we are exposed in each of these environments have important effects on our health. These factors are added to those relating to the personal environment ("subjective environment"): consumption of tobacco, alcohol, drugs and substances "for recreational purposes", diet and the like. In general, personal environmental factors have greater effects on health than "public" environmental factors.

Diseases caused by environmental agents are caused not only by exposure to chemical or physical agents present in the environment or in the work environment and in personal environments, but also by nutrition disorders.

Obesity has reached epidemic proportions. Between 1960 and 2008 the incidence of obesity increased from 13 to 34% and in 2009 68% of Americans between 20-75 years were overweight. Equally alarming is childhood obesity, a predictor of obesity in adults, which is also increased 2-3 times in the same period.

Obesity increases the risk of various important diseases, such as diabetes and hypertension and is therefore a serious public health problem, with an increase in health care costs, which in 2009 in the United States rose to around 147 billion dollars / year for obesity.

An ample scientific literature documents that, before dealing with a bariatric surgery, it is useful to lose weight in general using, strictly monitoring it, a ketogenic diet.

Keywords: Ketogenic diet, Obesity, bariatric surgery, environmental factors.

Introduction

Obesity is defined as the increase in body weight, caused by the accumulation of adipose tissue, which is likely to cause side effects on health. There are variously sophisticated techniques for calculating the accumulated fatty tissue, but the measurements used in clinical practice are as follows:

- Body mass index (Body Mass Index, BMI)
- Skin folds

• Circumference of various body regions, in particular the waist / hip ratio (Waist-to-Hip Ratio). BMI is closely related to body fat. The normal BMI ranges from 18.5 to 25 kg / m2, the overweight is defined by a BMI of 25-30 kg / m2 and obesity by a BMI greater than 30 kg / m^2 . It is a shared opinion that a BMI greater than 30 kg / m^2 is a risk factor for health.

The side effects of obesity depend not only on the overall body weight but also on the distribution of body fat. Central or visceral obesity with fat accumulation in the trunk and abdominal cavity is associated with a high risk of developing various diseases compared to obesity with widespread accumulation in the subcutaneous tissue.

The etiology of obesity is complex and not completely clarified. Genetic, environmental and psychological factors are involved. Obesity is a disturbance of the energy balance; the two elements of the energy equation, intake and consumption, are finely regulated by hormonal and neuronal mechanisms, so as to maintain body weight in a narrow range for several years. This fine balance seems to depend on an internal "set point" called iipostat which detects stored energy (in adipose tissue) and regulates food intake and energy consumption.

A key element of energy homeostasis is the LEP gene, which encodes leptin. Leptin, secreted by adipocytes, belongs to the cytokine family and regulates both elements of the energy equation - energy intake and consumption. The net effect of leptin is to reduce food intake and increase energy consumption.

The neuro-humoral mechanisms that regulate the energy balance and body weight are composed of three elements (Fib.1).

• The peripheral or afferent system generates signals from various locations.

The main components are leptin and adiponectin produced by adipocytes, pancreatic insulin, gastric ghrelin and intestinal YY (produced peptide ileus by and colon). Leptin reduces food intake, ghrelin stimulates the appetite and acts as a signal to start a meal. The YY peptide, released in the postprandial phase of endocrine cells of the ileum and colon, is a sign of satiety.

• The hypothalamic arcuate nucleus receives and integrates the peripheral signals and generates new signals transmitted by: 1) POMC / pro-oppiomelanocortin neurons) and CART (cocaine-and amphetamine-regulated transcript peptide; 2) NPY neurons (neuropeptide Y) and AgRP (Agouti-related protein (AgRP), also called agouti-related peptide).

• The efferent system is formed by hypothalamic neurons regulated by the arcuate nucleus. POMC / CART neurons activate efferent neurons that stimulate energy consumption, inducing weight loss, while NPY/ AgRP neurons activate afferent neurons that induce food intake and weight gain. The signals transmitted by efferent neurons also communicate with the centers of the forebrain and mesencephalon that control the autonomic nervous system. (1)

Leptin

Leptin secretion is regulated by stored fat with complex and little-known mechanisms. The presence of abundant adipose tissue stimulates the secretion of leptin, which reaches the hypothalamus, where it inhibits food intake by stimulating POMC / CART neurons and inhibiting NPY/AgRP neurons.

If the adipose tissue is scarce the opposite effect occurs.

Leptin also increases energy consumption by stimulating physical activity, energy consumption and thermogenesis, probably the main metabolic effects of leptin are mediated by the hypothalamus. Adipocytes express beta-adrenergic receptors which, when stimulated by norepinephrine, cause hydrolysis of fatty acids and decoupling of energy production from stored fat. In rodents and humans, mutations with loss of function of leptin pathway components cause massive obesity. Mutations in the melanocortin 4 receptor (MCAR) gene are found in 4-5% of patients with massive obesity.

Serum leptin levels are often elevated in the obese, suggesting that leptin resistance is widespread in the human race.

Adipose tissue

In addition to leptin, adipose tissue produces other mediators, such as adiponectin, cytokines, chemokines and steroid hormones, which allow adipose tissue to act as a link between lipid metabolism, nutrition and inflammatory responses. The total number of adipocytes is determined during adolescence and is higher in subjects who

during adolescence and is higher in subjects who have been obese as children, which is another reason to prevent childhood obesity.

In adults about 10% of adipocytes undergo an annual turnover, but their number remains constant, regardless of body mass. Diets fail in part because fat loss from adipocytes reduces leptin levels, stimulating appetite and reducing energy consumption.

Intestinal hormones

Intestinal hormones are initiators and terminators of rapid voluntary feeding.

Ghrelin is produced by the stomach and is the only intestinal peptide that stimulates food intake, probably stimulates hypothalamic NPY/AgRP neurons. Ghrelin increases before meals and is reduced 1-2 hours after meals, but the reduction is attenuated in the obese. PYY is secreted by endocrine cells of the ileum and colon in response to meals. It works by stimulating the POMC / CART receptors in the hypothalamus and thus reducing food intake.

Clinical consequences of obesity

Obesity is associated with insulin resistance and hyperinsulinism, key aspects of type 2 diabetes. The excess of insulin generates, in turn, sodium retention, hypervolemia, excess adrenaline production and smooth muscle proliferation, all typical signs of hypertension. Regardless of the mechanism, the risk of hypertension in previously normotensive subjects increases proportionally to weight.

Obese people generally also have hypertriglyceridemia and low levels of HDL cholesterol, risk factors for coronary heart disease. However, the association between obesity and heart disease is not direct and depends mainly on the association of obesity with diabetes and hypertension, rather than on the weight itself.

Overweight is associated with an increased risk of cancer, esophagus, thyroid, colon, kidney, endometrium and gallbladder neoplasms. The pathogenesis is multifactorial, and it has been hypothesized that hyperinsulinism is one of the responsible factors. Insulin increases the levels of insulin-like growth factor 1 (IGF-1), which promotes the growth and survival of various types of neoplastic cells, through the IGF-1R receptor. Hyperestrogenism is associated with an increased risk of endometrial cancer and obesity increases estrogen levels.

Non-alcoholic fatty liver disease is frequently associated with obesity and type 2 diabetes. The disease is also called non-alcoholic fatty liver disease and can cause fibrosis and cirrhosis.

Cholelithiasis is 6 times more common in the obese than in lean subjects. The pathogenesis is mainly linked to the increase in total cholesterol, cholesterol metabolism and cholesterol excretion in bile salts, which predisposes to the predisposition to the formation of cholesterol stones.

The hypoventilation syndrome, a constellation of respiratory disorders of the large obese (Pickwick syndrome), is characterized by nocturnal and diurnal hypersomnia, often caused by sleep apnea, polycythemia and right heart failure. Severe adiposity risk factor is а for arthropathy (osteoarthritis). These forms of arthritis, typical of the elderly, are mainly caused by the cumulative effects of wear on the joints. Over time, joint trauma is proportional to fat mass.

Inflammatory markers, such as C-reactive protein (PCR), and inflammatory cytokines, such as TNF, are often elevated in the obese. The origin of the inflammation is not clear; it has been hypothesized to be due to the direct inflammatory effect of hyperlipidemia and the release of cytokines from fat-laden adipocytes. Regardless of the cause, chronic inflammation is thought to cause various complications of obesity, such as insulin resistance,

metabolic changes, thrombosis, heart disease and cancer.

Ketogenic diet

The ketogenic diet is a nutritional strategy based on the reduction of dietary carbohydrates, which forces the body to independently produce the glucose necessary for survival and to increase the energy consumption of the fat contained in adipose tissue. (2)

Regularly produced in minimal quantities and easily disposable with urine and pulmonary ventilation, ketone bodies reach a higher level than the normal condition.

The presence of ketone bodies in the blood has different effects on the body: some are considered useful in the process of weight loss, others are of a collateral type.

There is not just one type of ketogenic diet and all dietary styles that supply less calories, carbohydrates and sometimes less protein than necessary are ketogenic; the Atkins diet and LCHF (low card, high fat-low carbohydrate, high fat) are certainly low carbs and potentially ketogenic.

The ketogenic diet (keto diet) is a nutritional scheme:

• low in calories (low-calorie diet)

• low percentage and absolute carbohydrate content (low carb diet)

• high percentage of protein

• with a high percentage of lipids.

A rather strict guideline for a correct ketogenic diet involves an energy distribution of:

• 10% from carbohydrates

•15-25% protein (proteins also contain glucogenic amino acids that help sustain blood glucose levels)

• 70% or more fat.

This food strategy is used above all in three very different contexts:

• Weight Loss

• food therapy of metabolic pathologies such as chronic hyperglycemia, hypertriglyceridemia, arterial hypertension and metabolic syndrome (never in the presence of pathologies or suffering of heart and / or kidneys).

• reduction of symptoms associated with childhood epilepsy, only when the subject is not responding to drug therapy.

In the ketogenic diet it is necessary to eat foods that do not contain carbohydrates, limit those that bring few and avoid the foods that are rich in them. The recommended foods are:

• meat, fishery products and eggs - the fundamental food group

• cheeses - The fundamental group of foods

• fats and seasoning oils - V fundamental group of foods

• vegetables - VI and VII fundamental food group The foods advised against are:

• cereals, potatoes and derivatives - III basic food group

• legumes - IV fundamental food group

• fruits - VI and VII fundamental group of foods

• sweet drinks, various sweets, beer, etc.

Generally, it is recommended to maintain a carbohydrate intake of less than or equal to 50 g / day, ideally organized in 3 portions with 20 g each. In the last period more and more numerous studies have demonstrated the validity of a treatment with the ketogenic diet undertaken before the bariatric surgery to be practiced.

Following the keto diet even for a short period of three weeks is very useful, as it results in a weight reduction of about 10%. This weight reduction, consisting mainly of a decrease in body fat, allows easier operative access and consequently also easier manual execution of the operation. Reducing the subcutaneous fat in the abdominal area, particularly in the vicinity of the liver, is very important for the success of the bariatric surgery.

Not only before, but also after surgery, the ketogenic dietary approach, to be repeated in cycles, can serve to maintain the benefits of surgery thanks to a further reduction of insulin resistance, a metabolic condition almost always present in large obese.

The ketogenic diet thus becomes an invaluable ally of surgery, making complex operations succeed in the best way, without too many risks for the patient's health. To identify a possible state of ketosis, it is possible to carry out urine tests (with special urine strips), blood (blood ketone gauges) or breath (ketone analyzer in the breath). However, you can also rely on certain "revealing" symptoms, which require no tests:

• Dry mouth and feeling thirsty

• Increase in diuresis (for the filtration of acetoacetate)

• Breath or acetonic sweat (due to the presence of acetone) that escapes through our breath

• Reduced appetite

• Tiredness.

There is no real distinction between ketosis and non-ketosis. The level of these compounds is influenced by diet and lifestyle. However, it is possible to state that there is an optimal range for the correct functioning of the ketogenic diet:

• Below 0.5 mmol of ketones per liter of blood is not considered ketosis.

• Between 0.5-1.5 mmol / l it is called light ketosis

• With 1.5-3 mmol / I the ketosis is defined as optimal

• Values of over 3 mmol / I, in addition to not being more effective, compromise the state of health (especially in the case of type 1 diabetes mellitus)

• Values over 8-10 mmol / l are difficult to achieve with the diet. Sometimes they are obtained in diseases or through inadequate physical activity; relate to even very serious symptoms.

The functioning mechanism of the ketogenic diet is based on the reduction of calories and food carbohydrates which, in association with a correct level of proteins and a high percentage of fat, should improve lipolysis and cellular lipid oxidation, therefore total consumption fat optimizing slimming. The production of ketone bodies, which must be absolutely controlled, has the function of moderating appetite stimulation - due to their anorectic effect.

Ketogenic metabolism

Cellular energy production takes place thanks to the metabolization of some substrates, above all glucose and fatty acids. Mostly, this process begins in the cytoplasm (anaerobic glycolysis - without oxygen) and ends in the mitochondria (Krebs cycle with oxygen - and ATP recharge). Note: the muscle cells are also able to oxidize good amounts of branched amino acids. However, two fundamental aspects must be emphasized:

Some tissues, like the nervous one, work "almost" exclusively with glucose

The correct cellular use of fatty acids is subordinated to the presence of glucose which, if deficient, is produced by the liver by means of neoglucogenesis (starting from substrates such as glucogenic amino acids and glycerol).

Neoglucogenesis is not able to definitively satisfy, in the long term, the metabolic demands of the whole organism. This is why carbohydrates, although they cannot be defined as "essential", must be considered essential nutrients and a minimum intake of 180 g / day is recommended (the minimum quantity to guarantee the full functionality of the central nervous system).

Residual ketone bodies

We now explain how the release of ketone bodies occurs.

During energy production, the fatty acids are first reduced to CoA (coenzyme A) and, immediately afterwards, allowed to enter the Krebs cycle. Here they bind to the oxalacetate to reach a further oxidation, until it ends with the release of carbon dioxide and water. When the production of acetyl CoA by lipolysis exceeds the absorption capacity of oxalacetate, the formation of the so-called ketone bodies occurs.

Types of ketone bodies

The ketone bodies are of three types:

- Acetone
- Acetoacetate
- 3-hydroxybutyrate.

Disposal of ketone bodies

The ketone bodies can be further oxidized, in particular from muscle cells, from the heart and to a lesser extent from the brain (which uses them especially in the absence of glucose), or eliminated with urine and pulmonary ventilation. Needless to specify that increasing the ketone bodies in the blood also increases the workload of the kidneys. If the production of ketone bodies exceeds the disposal capacity of the organism, they accumulate in the blood, giving rise to the so-called ketosis. Ketosis, ketoacidosis and metabolic acidosis Also called ketoacidosis, this condition lowers the blood pH defining the typical picture of metabolic acidosis (typical of untreated diabetics). In extreme cases, acidosis can lead to coma and even death.

Motor activity and ketoacidosis

The role of motor activity on ketoacidosis is, in a sense, contradictory. Starting from the assumption that the use of the ketogenic diet is however a metabolic forcing - which in the long run can lead to unpleasant consequences, even in a young and well-trained organism - it is necessary to specify that:

• On the one hand, intense physical exercise increases the energy demands of glucose favoring the production and accumulation of ketone bodies.

• On the other hand, moderate exercise increases the oxidation of ketone bodies by opposing their accumulation and the negative effects they can have on the body.

Neoglucogenesis

The body still needs glucose and, if it is not taken with the diet, it must be produced with neoglucogenesis. Indispensable for the correct functioning of nerve tissue in particular, glucose is also necessary to complete lipid oxidation.

Gluconeogesis is a process that leads to the formation of glucose starting from the carbon skeleton of some amino acids (called glucogenic, or that give rise to oxalacetate); to a lesser extent, also from glycerol and lactic acid. This process ensures a constant supply of energy even in conditions of glucose deficiency, but forces the liver and kidneys to work harder to eliminate nitrogen.

There are different types of ketogenic diet and not all of them offer the same results.

This is because, apart from personal attitudes, that of the ketogenic diet is a system that requires rather accurate control and monitoring. Furthermore, it cannot (or rather should not) be continued for too long. The ketogenic diet can have advantages:

- Facilitates weight loss thanks to:
- Reduction of total calories
- Constant blood glucose and insulin maintenance
- Increased consumption of fat for energy purposes

• Increased global calorie expenditure due to increased specific dynamic action and "metabolic work"

• It has an anorectic effect

• It can be useful in countering the symptoms of epilepsy that does not respond to drugs, especially in children.

Disadvantages

The ketogenic diet can also show several disadvantages, most of which depend on the levels of ketone bodies present in the blood:

• Increased renal filtration and diuresis (excretion of ketone bodies and nitrogenous waste)

• Tendency to dehydration

• Increased workload of the kidneys

• Possible toxic effect on the kidneys by ketone bodies

- Possible hypoglycemia
- Possible hypotension

• Keto-flu or "keto-flu" in English; it is a syndrome linked to the poor adaptation of the organism after 2-3 days from the start of the ketogenic diet. It Includes:

- Headache
- Fatigue
- Dizziness
- Light nausea
- Irritability.

• In the most sensitive subjects, increased chance of fainting (due to the previous two)

- Greater tendency to:
- Muscle cramps
- Constipation
- Sensation of heart palpitations

• Increased liver workload due to increased neoglucogenesis, transamination and deamination processes

- In the presence of intense and / or prolonged motor activity, muscle catabolism
- It is unbalanced and tends to limit the intake of some very important nutrients
- It can be particularly harmful for

Advantages

- Malnourished subjects such as, for example, those affected by eating disorders (DCA)
- type I diabetics
- pregnant and nursing mothers
 - those who already suffer from liver and / or kidney diseases.

Bariatric surgery

Obesity is a chronic condition that is often difficult to treat with a simple diet combined with regular exercise. In these cases, bariatric surgery is a valid therapeutic option, especially for severely obese people who suffer from serious health problems exacerbated by excess weight.

Bariatric surgery includes a variety of procedures that promote weight loss by reducing food intake and / or absorption. Weight loss can be achieved by reducing the size of the stomach with a gastric bandage, by surgical resection (partial vertical gastrectomy or biliopancreatic diversion with duodenal switch) or by creating a small gastric pouch connected directly to a section of the small intestine (gastric bypass and variants). The best outcome is obtained when the patient who undergoes surgery is strongly determined to adhere to strict dietary guidelines and to perform regular physical activity after the operation. In addition, the subject must agree to commit to long-term followup and post-operative medical treatment. These behaviors are essential to maintain the results obtained with bariatric surgery.

Indications

Currently, bariatric surgery is a suitable option for patients who:

• They present with severe obesity;

• Failed to achieve effective results with a controlled food program (with or without pharmacological support);

• They have associated pathologies, such as hypertension, reduced glucose tolerance, diabetes mellitus, hyperlipidemia and obstructive sleep apnea.

The body mass index (BMI) is used to define the levels of obesity, an indicator of the weight state of an individual who compares height and weight. A subject with $BMI \ge 30$ is considered obese.

Bariatric surgery is recommended only for people with at least one of the following characteristics:

• BMI> 40 (obesity of Illa class / very serious);

• BMI> 35 (2nd / 2nd class obesity), associated with at least one pathological condition linked to obesity that can improve with weight loss.

However, recent research suggests that bariatric surgery may also be appropriate for people with a BMI of 35-40 with no associated disease or a BMI of 30-35 and significant comorbidities.

Anyone considering the possibility of undergoing bariatric surgery to achieve significant weight loss should be aware of the risks and benefits of the treatment.

The patient can be considered suitable for a bariatric surgery if:

• It fails to reach or maintain a beneficial level of weight loss (for at least six months) by adopting appropriate non-surgical solutions, such as diet, medication and exercise.

• He agrees to commit himself long term, after the operation, to adopt a healthy diet and to follow a regular physical activity; it is therefore aware of the limits it will have to face to its future food choices and the need to undergo regular follow-ups.

• It does not present any medical or psychological obstacles to surgery or to the use of anesthesia, it does not abuse alcohol and / or drugs.

• Is motivated to improve their health conditions and is aware of how life can change after surgery (for example, patients must adapt to side effects, such as the need to chew food well or the inability to eat large amount of food).

There is no absolutely safe method, including surgery, to produce significant weight loss and maintain it over time. Some subjects who undergo a bariatric surgery procedure may experience a lower weight loss than expected; others may regain part of the weight lost over time. This recovery can vary depending on the degree of obesity and the type of surgery. Even some bad habits, such as lack of exercise or frequent consumption of high-calorie snacks can affect the long-term outcome of the treatment.

Classification

Bariatric procedures can be grouped into three main categories:

Malabsorption interventions. Malabsorption surgical procedures reduce the absorption of food. They involve an irreversible reduction in the size of the stomach and their effectiveness derives mainly from the creation of a physiological condition: the gastric cavity is connected to the terminal part of the small intestine, with consequent limitation of the absorption of calories and nutrients.

They belong to this type:

• Biliopancreatic diversion (wider form of gastric bypass, with the gastric pouch combined with the ileum. It produces the most extreme malabsorption);

• jejuno-ileal by-pass;

• Restrictive procedures. Gastro-destructive interventions limit the introduction of food through a prevailing mechanical action. They are based on the formation of a small gastric pouch in the upper part of the stomach, which limits the gastric volume and leaves the alimentary canal in continuity through a narrow and non-dilatable orifice. Restrictive procedures act to reduce the amount of food taken orally.

They belong to this type:

- Adjustable gastric banding;
- Vertical gastroplasty;
- Sleeve gastrectomy (partial vertical gastrectomy);

• Intragastric flask (transient non-surgical treatment).

• Mixed interventions. Mixed bariatric procedures apply both techniques simultaneously, as in the case of gastric bypass or sleeve gastrectomy with duodenal switch.

The type of surgery that more than any other can help an obese person depends on a number of factors. Patients should discuss with the reference surgeon which option is best suited to their needs. Bariatric surgery can be performed through approaches, which "open" standard include laparotomy with incision of the abdominal wall, or by laparoscopy. With the second technique, doctors insert surgical instruments through small cuts made on the abdomen, guided by a small camera that transmits the images to a monitor. Currently, in most cases, laparoscopic bariatric procedures are performed, because they are minimally invasive, require smaller incisions, create less tissue damage and are associated with fewer post-operative problems. However, not all patients are suitable for laparoscopy. Extremely obese patients (eg> 350kg) who have undergone previous stomach surgery or who have complex health problems (severe heart and lung disease) may require an open approach. (3)

Surgical options

There are four types of operations most commonly practiced: adjustable gastric banding (AGB), Roux-en-Y gastric bypass (RYGB), biliopancreatic diversion with duodenal switch (BPD-DS) and vertical sleeve gastrectomy (or sleeve gastrectomy, VSG).

Adjustable gastric (AGB): banding a gastroreductor action that reduces food intake by placing an elastic silicone band around the upper portion of the stomach. This allows you to create a small gastric pouch that communicates with the rest of the stomach through а narrow unbleachable emptying orifice. The containment capacity of the gastric pouch can be adjusted according to the patient's needs without resorting to further surgery; in fact the bandage houses a saline solution that can be increased or decreased, varying the constrictive effect, by means of a thin catheter connecting it to a tank placed just below the skin.

Weight loss is mainly due to the limited amount of food that can be ingested in a single meal (early satiety) and the increase in the time needed to digest introduced foods. It is often performed by laparoscopy (LAGB) and represents a reversible intervention: the gastric cavity is not sectioned and the bandage can be removed. Weight loss: about 50% of excess weight.

• Roux-en-Y gastric bypass (RYGB): it is a mixed operation, which limits both the intake and absorption of food. The amount of food eaten is limited by reducing (by surgical resection) the stomach to a small bag, similar in size to the pocket created with the gastric band. Furthermore, this small sac is connected, through a jejunalloop, directly to the small intestine (at the jejunum), excluding the digestive tract responsible for absorbing nutrients (part of the stomach, duodenum and biliary tract). The RYGB is considered an irreversible intervention, but, in some cases, the procedure can be partially reversed. Weight loss: around 60-70% of the excess weight

• Biliopancreatic diversion with duodenal switch (BPD-DS): usually referred to as "duodenal switch" (duodenal inversion), it is a complex bariatric surgery with three

Effectiveness

The purpose of bariatric surgery is to reduce the risk of disease or death associated with obesity. In general, malabsorptive procedures induce greater weight loss than restrictive procedures, but they have a higher risk profile.

Recovery after bariatric surgery

Immediately after bariatric surgery, the patient is limited to a liquid diet, which includes foods such as broth or diluted fruit juices. This line is adopted until the complete recovery of the gastrointestinal tract from the operation. In the later stages, the patient is "forced" to take only modest quantities of food, because if he exceeds the containment capacity of the stomach he may experience nausea, headache, vomiting, diarrhea, dysphagia and so on. Dietary restrictions depend in part on the type of surgery. Many patients, for example, will need to take a multivitamin daily for life, to compensate for the reduced absorption of essential nutrients (4).

Side effects

A variety of complications can be associated with bariatric surgery procedures. The risks depend on the type of intervention and any other intestinal, nausea and vomiting (due to excess food or stenosis in the surgical site). Other problems that can occur are related to nutrient deficiencies, typical of subjects subjected to health problems before the operation. In the postoperative period, some shortterm complications (within 1-6 weeks after surgery) include bleeding, infection of surgical may wounds, venous thromboembolism (deep vein thrombosis in the legs and pulmonary embolism), heart attack, pneumonia, urinary tract infections, gastrointestinal ulcers, gastric and / or intestinal fistula, stenoses and hemias internal hemia).

Scientific evidence

Elective surgery in obese adults carries a higher risk of post-operative infection and prolonged hospital stays, and surgeons may postpone surgery for patients with obesity until they lose weight. A dietitian-led Very Low Caloric Ketogenoc Diet protocol achieved sufficient weight loss to facilitate elective surgery for most patients. The approach was feasible, highly valued by patients and surgeons, and resulted in perceived surgical benefits (5-7). A large scientific literature documents that it is useful to lose weight before facing a bariatric surgery, generally using a ketogenic diet while closely monitoring it. The motivations according to the care team are basically two: the first is surgical, as reducing the weight allows an easier intervention, primarily for the patient and secondarily for the whole surgical team involved. Even the anesthesiologist will benefit from it, the intervention will be shorter and with less operator risk. The second motivation is to be able to assess how much the patient is really interested in facing this path, how much is his real commitment.

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