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Dental status in patients after bariatric surgery (literature review)

ANNOTATION

The study developed modern methods for reducing the weight of patients for the treatment of morbid obesity, including methods of bariatric surgery. The most effective and safe treatment for all ages is bariatric surgery. Manifestations following bariatric surgery include gastroesophageal, respiratory, cardiovascular, endocrine, and psychological changes. The most common gastrointestinal complications are duodenal stenosis, gastric ulcers, diarrhea, chronic vomiting, reflux and gastroesophageal cancer. Related to demineralization is dental erosion, which is defined as the chemical dissolution of tooth tissue through a chemical process without the participation of bacteria.

Key words: dental manifestations, bariatric surgery, methods

Modern methods have been developed to reduce the weight of patients for the treatment of morbid obesity, including bariatric surgery methods [22,30]. The most effective and safe treatment for all ages is bariatric surgery, with the prevalence increasing over the years [22,30]. Since its introduction, surgical techniques have undergone great changes over time, both to improve instrumentation and postoperative outcomes[1]. Currently, there are 3 main surgical methods known. One method is gastric banding, in which a silicone ring is placed around the stomach, thus creating two compartments: a small one (15-20 ml) on top, which will store a small amount of food, thus creating a feeling of fullness, and another part larger and placed below, which will take up part of normal digestion. Another option is Roux -en-Y gastric bypass, which creates a small pouch (15 to 30 ml) that sutures the stomach itself, limiting the amount of food that can be consumed. And part of the small intestine is diverted, delaying the mixing of food with gastric juice. Method, Misuse Biliopancreatic is performed in such a way that ³/₄ of the stomach is removed and the intestines are shortened,







reducing the time food is in contact with the intestines, significantly reducing nutrient absorption.

Numerous factors may affect the results. Of note, older patients are more likely to develop postoperative complications due to the lower functional reserve of this age group, as well as the presence of other metabolic diseases such as diabetes mellitus, which has led to significant consequences in these patients. Manifestations following bariatric surgery include gastroesophageal, respiratory, cardiovascular, endocrine, and psychological changes. In operated patients, the most common gastrointestinal complications are duodenal stenosis, gastric ulcers, diarrhea, chronic vomiting, reflux and cancer of the gastroesophageal tract. There may also be an increased risk of iron, vitamin B12, vitamin D and calcium deficiencies, mainly related to poor absorption of nutrients by the stomach and intestines.

Some researchers report nearly a 90% reduction in asthma and sleep apnea associated with weight loss. In patients at high risk of cardiovascular disease, surgical patients experience a decrease in systolic diastolic blood pressure with a subsequent decrease in the risk of hypertension and coronary heart disease.

There may also be a decrease in total cholesterol, triglycerides and uric acid, as well as an increase in the HDL cholesterol fraction [26]. Patients undergoing gastroplasty surgery, with weight loss, diabetes rates and the risk for non-diabetics are reduced. Researchers have reported changes in plasma hormone levels associated with ovulation that are lower than normal due to changes in absorption in the gastrointestinal tract.

Considering physiological factors, psychological and emotional factors should be taken into account. Postoperative consequences may affect the effectiveness of treatment. At weight loss results in increased self-esteem, as well as improved social relationships, decreased anxiety and depression. Some patients may develop self-denial, psychotic behavior, eating disorders, and return to their original weight. In a review of the literature, researchers suggest that patients with psychological complications after surgery, especially fear of returning to previous weight, induce vomiting [18], attributing the psychological problem to oral manifestations at this point. Based on the above data, the purpose of this article was to provide a summary of dental manifestations in bariatric patients.

Heling et al. [14] (2006) conducted a study involving 113 patients (about 30–50 years old) who had undergone bariatric surgery 4–5 years previously. Scientists examined bariatric patients' self-perceptions regarding their dental health. 79% of patients noted vomiting as the most common postoperative phenomenon; 37% reported that after surgery they began to eat more sweets; 20% noted improved oral hygiene; 73% did not change their oral hygiene habits; 34%







reported increasing the number of dental visits; 60% did not change the frequency of visits to the dentist; 37% reported severe hypersensitivity after surgery; 44% reported vomiting associated with high sensitivity; 32% suffer from indigestion after surgery, of which 59% report increased sensitivity; and 80% of patients visited the dentist due to increased sensitivity. Results from case reports have shown an association between bariatric surgery and increased caries [11–13].

A decrease in pH after bariatric surgery can compromise dental health, which is manifested by a high frequency of sugar consumption, as well as gastroesophageal reflux disease (GER). GER is a chronic disease that occurs as a result of retrograde reflux of gastroduodenal contents (mainly stomach acids such as hydrochloric acid) into the esophagus or adjacent organs, such as the oral cavity. The pH of gastric juice is around 1.2, which poses a potential risk of tooth demineralization since the critical pH for dental apatite dissolution is around 5.5 [17]. Also, patients experienced decreased saliva production [23], due to low absorption of nutrients in the intestine, which, in turn, may contribute to the dissolution of minerals. A decrease in pH can lead to tooth demineralization (caries and erosion) and increased sensitivity [14]. The undoubted consequences of these injuries are loss of enamel and hypersensitivity due to the impact on the dentinal tubules.

Dental erosion. Among the lesions associated with demineralization is dental erosion, which is defined as the chemical dissolution of tooth tissue as a result of a chemical process (acid or chelating agents) without the participation of bacteria [21]. The etiology of erosion is multifactorial and not fully understood. The most important sources of acids are those found in the diet, such as acidic foods and drinks [20], and those produced in the stomach, such as gastric acid from regurgitation and reflex disorders.

Current data indicate that increased consumption of acidic foods and soft drinks are important factors in the development of erosive wear [19]. Acid attack leads to irreversible loss of hard tooth tissue, which is accompanied by progressive softening of the surface [19]. This softened zone is more sensitive to mechanical influences, such as abrasion [27], which, in turn, have virtually no effect on healthy hard dental tissues [2].

The clinical picture of early enamel erosion looks like a smooth, silky-shiny glazed surface. Typical of facial erosions is a ridge of enamel separating the defect from the marginal gingiva. Collision erosion is characterized by rounded tubercles and concavities. As occlusal erosion progresses, it results in distinct cuspal striations and restorations that rise above the surface of the adjacent tooth. In cases of severe erosion, all occlusal or facial morphology disappears. When dentin is reached, increased sensitivity to cold, heat, and osmotic pressure is commonly reported. Other consequences of dental erosion are diastema, thin and broken







incisal edges, loss of vertical dimension, open pseudobite and convexity of esthetic restorations [10].

Caries. Dental caries is considered a multifactorial disease, the etiology of which is related to the presence of plaque composed of cariogenic bacteria that can metabolize sugars such as sucrose. As a result of this metabolism, organic acids are formed, such as lactic acid, which, in turn, can cause demineralization of dental tissues [16,29]. Gradually, the biofilm becomes saturated with minerals that are released from the dental structure, promoting precipitation and the formation of the initial subsurface carious lesion [6,12]. An early sign of enamel damage is a white spot (also known as a non-cavitating lesion) due to subsurface demineralization. As a result of increased bacterial metabolism, the intact surface layer can be destroyed, leading to cavity formation, bacterial spread and progression of the lesion to the dentin. When the dentin is exposed to masses of bacteria in the cavity, the most superficial part of the dentin soon decomposes under the action of acids and proteolytic enzymes. This zone is called the destruction zone. Tubular invasion of bacteria is often observed in this area. Bacterial invasion leads to pulpal inflammation, which can have serious consequences such as pain, pulp necrosis and periapical lesions [7,16].

Hypersensitivity. Dentin hypersensitivity has been defined as an acute, brief pain arising from exposed dentin in response to stimuli, typically thermal, evaporative, tactile, osmotic, chemical, and which cannot be attributed to any other form of dental defect or pathology [31]. The authors believe that short-term and acute pain symptoms are associated with hydrodynamic load. The most affected patients are between 20 and 40 years of age; premolars and incisors are usually the most sensitive teeth, as the pain is localized on the facial surface. Sensitive teeth have a much larger number of open tubules per unit area, and the average diameter of the tubules is almost 2 times larger than the tubules in non-sensitive teeth [32]. Dentin hypersensitivity is a condition of presumed multifactorial pathology. For its development, two processes are necessary: (1) dentin must be exposed due to genetic disorders, enamel defects (lamellae and spindles), enamel loss (erosion, abrasion, abrasion, abfraction), gingival recession with rapid loss of cementum, and (2). The dentinal tubules must be open to both the oral cavity and the pulp. The diagnostic protocol for this condition consisted of medical, dental, dietary, oral hygiene history, and intraoral air index examinations. In-office procedures may be made from substances that can create a smear layer on the dentin surface, clogging the dentinal tubules with insoluble sediments and stimulating the production of reparative dentin swelling and sclerosis. This can be achieved chemically using substances such as potassium, calcium and fluoride, or physically [32-33].

Conclusion. These findings suggest that postoperative diet and gastric reflux may increase the risk of dental disease, especially in the presence of other risk



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factors such as consumption of sweet-tasting foods and acidic drinks. Due to lack of data, these and other questions require further study based on the analysis of clinical materials related to dental manifestations of those who underwent bariatric surgery.

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