

BARIATRIC SURGERY AND PERIODONTAL DISEASE

CIRURGIA BARIÁTRICA E DOENÇA PERIODONTAL

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Kaliane Rocha Soledade

Doutora em Processos Interativos dos Órgãos e Sistemas pela Universidade Federal da Bahia, Docente dos Programas de Mestrado Profissional em Biotecnologia e Mestrado Profissional em Desenvolvimento Regional e Meio Ambiente da Faculdade Maria Milza

Instituição: Colegiado de Odontologia, Faculdade Maria Milza

Endereço: Rodovia BR-101 - Km 215 - Governador Mangabeira / Bahia, Brasil

E-mail: krsoledade@gmail.com

Larissa Rolim Borges-Paluch

Doutorado em Ciências Biológicas pela Universidade Federal do Paraná
Docente do Programas de Mestrado Profissional Desenvolvimento Regional e Meio Ambiente da Faculdade Maria Milza

Instituição: Faculdade Maria Milza

Endereço: Rodovia BR-101 - Km 215 - Governador Mangabeira / Bahia, Brasil

E-mail: larissapaluch@gmail.com

Airton Francisco de Souza

Discente do Programa de Mestrado Profissional em Desenvolvimento Regional e Meio Ambiente da Faculdade Maria Milza

Instituição: Faculdade Maria Milza

Endereço: Rua Leonel Ribas, 104. Centro. Cruz das Almas – Bahia, Brasil

E-mail: asouza101@gmail.com

Magno Andrade dos Santos

Bolsista Integral do Programa de Mestrado Profissional em Desenvolvimento Regional e Meio Ambiente da Faculdade Maria Milza

Instituição: Faculdade Maria Milza

Endereço: Rua João Elizeu de Melo, SN, casa, Centro, São Felipe – Bahia, Brasil

E-mail: mgno.andrade@gmail.com

José Carlos Barbosa Andrade Júnior

Bolsista do Programa de Mestrado Profissional em Biotecnologia da Faculdade Maria Milza

Instituição: Faculdade Maria Milza

Endereço: Largo 2 de Julho- Maragogipe – Bahia, Brasil

E-mail: junior.andrade.11@hotmail.com

Clara Renata de Almeida Sampaio

Acadêmica do Curso de Bacharelado em Odontologia da Faculdade Maria Milza

Instituição: Faculdade Maria Milza

Endereço: Rodovia BR-101 - Km 215 - Governador Mangabeira / Bahia, Brasil

E-mail: renatasampaio1@outlook.com

Mônica Dourado Silva Barbosa

Doutora em Periodontia pela Universidade de São Paulo
Professora Adjunto da Escola Bahiana de Medicina e Saúde Pública
Instituição: Escola Bahiana de Medicina e Saúde Pública

Endereço: Escola Bahiana de Medicina e Saúde Pública. Av. Silveira Martins, 3386, Cabula
41150100 - Salvador, Bahia, Brasil
E-mail: monicadourados@uol.com.br

Isaac Suzart Gomes Filho

Doutor em Periodontia pela Universidade de São Paulo – Bauru
Professor Titular Pleno do Curso de Odontologia e do Programa de Pós-Graduação em Saúde
Coletiva da Universidad Estadual de Feira de Santana

Instituição: Universidade Estadual de Feira de Santana, Departamento de Saúde, Odontologia
Endereço: Av. Universitária, Km 3 - Br 116, Departamento de Saúde. Campus Universitário.
44031460 - Feira de Santana, Bahia, Brasil
E-mail: isuzart@gmail.com

ABSTRACT

Current epidemiological studies have showed a growing number of obese patients worldwide. New surgical treatments for obesity that can provide consistent weight loss and effective management of associated comorbidities have been increasingly investigated. The effects of bariatric surgery include long-term benefits such as lower blood glucose levels with good management of type 2 diabetes, hyperlipidemia, metabolic syndrome, reduced risk of coronary heart disease, better quality of life and improved life expectancy. However, bariatric surgery is a procedure that implies malabsorption of nutrients and can result in nutritional deficiencies, anemia, hypocalcemia, low absorption of vitamin D and hyperparathyroidism, causing decreased bone mineral density and increased risk of osteoporosis and may play a major role in the progression and severity of periodontal disease.

Keywords: Obesity, Bariatric Surgery, Periodontal Disease, Osteoporosis.

RESUMO

Estudos epidemiológicos actuais têm mostrado um número crescente de doentes obesos em todo o mundo. Novos tratamentos cirúrgicos para a obesidade que podem proporcionar uma perda de peso consistente e uma gestão eficaz das comorbilidades associadas têm sido cada vez mais investigados. Os efeitos da cirurgia bariátrica incluem benefícios a longo prazo, tais como níveis mais baixos de glucose no sangue com uma boa gestão da diabetes tipo 2, hiperlipidemia, síndrome metabólica, redução do risco de doença coronária, melhor qualidade de vida e melhoria da esperança de vida. No entanto, a cirurgia bariátrica é um procedimento que implica má absorção de nutrientes e pode resultar em deficiências nutricionais, anemia, hipocalcemia, baixa absorção de vitamina D e hiperparatiroidismo, causando diminuição da densidade mineral óssea e aumento do risco de osteoporose e pode desempenhar um papel importante na progressão e gravidade da doença periodontal.

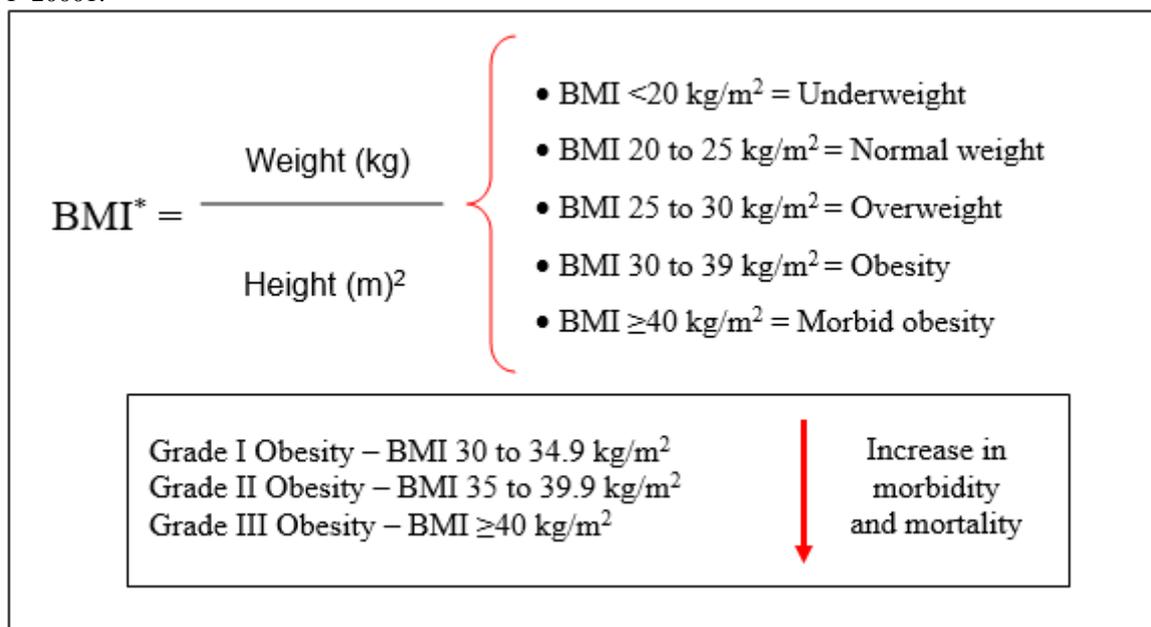
Palavras - Chave: Obesidade, Cirurgia Bariátrica, Doença Periodontal, Osteoporose.

1 INTRODUCTION

Obesity prevalence has been substantially increasing worldwide. Modern lifestyle, changes in eating habits, increased workload and stress have all been identified as factors for an increase in body weight seen in industrialized and mainly in developing countries.¹ Obesity is a multifactorial disease associated with genetic predisposition, high-calorie diet and physical inactivity.² It is determined using the body mass index (BMI) ($\geq 30.0 \text{ kg/m}^2$) and is regarded a major public health concern nowadays.³

The BMI is a ratio between height and weight and is calculated by dividing weight (kilograms) by the square of height (meters) (Figure 1). The National Institutes of Health Consensus Conference proposed in 1991 a classification system for risk based on BMI: patients with BMI between 25.0 and 29.9 are classified as overweight and those with BMI $\geq 30 \text{ kg/m}^2$ are classified as obese. Obesity was later subclassified according to health risk in: grade I (BMI between 30.0 and 34.9 kg/m^2); grade II (BMI between 35.0 and 39.9 kg/m^2); and grade III (BMI $\geq 40 \text{ kg/m}^2$).¹

Figure 1 – Classification of obesity according to World Health Organization (WHO) based on the calculation of body mass index* (BMI) and obesity risk classification based on the National Institutes of Health Consensus Conference, 1991–20001.



Current epidemiological data suggest that about 1.7 billion people worldwide are obese.⁴ Obesity prevalence doubled between 1978 and 1999 in the United States.¹ In 2004, approximately 34.1% of US population were overweight and about 32.2% were obese.³ Currently, it is estimated that obesity grade III affects one in every 20 Americans.⁵ In Brazil, in 2006, about 40% of the

population were overweight compared to 16% 30 years ago and about 10% of public health expenditures were allocated to obesity.⁶ At that time it was also estimated that approximately one million Brazilians had morbid obesity.⁷

High prevalence of obesity is associated with increased prevalence of comorbidities, namely type II diabetes, hyperlipidemia, hypertension, sleep apnea, heart disease, stroke, asthma, degenerative conditions, depression, chronic inflammatory diseases such as periodontitis, various forms of cancer, among other.^{1, 3, 4, 8, 9, 10} Several studies have evidenced obese individuals have an increased risk of developing different types of cancer, which might suggest immune deficiency.^{9,11} These comorbidities account for 2.5 million deaths per year worldwide. Compared to normal weight, 25-year-old obese individuals have a reduction of about 22% in their life expectancy, a reduction of around 12 years.⁴

Conservative treatments for obesity (low-calorie diets, drug therapy and physical exercise) have not been effective for most patients in achieving sustained weight loss. Hence, there has been a significantly increasing demand for surgical procedures that can provide sustained weight loss and effective management of comorbidities. In this scenario, bariatric surgery has become the focus of a discussion regarding its short- and long-term benefits and adverse health effects. The objective of the present study was to conduct a literature review on the main consequences of bariatric surgery while seeking to find a biological plausibility of its potential association with progression and severity of periodontal disease.

2 MAJOR CONSEQUENCES OF BARIATRIC SURGERY

Bariatric surgery was first described in 1954 when Kremer et al. performed a jejunoileal bypass. The term bariatric comes from the Greek words *baros* (weight) and *iatrike* (treatment).¹² This surgical procedure involves three main operation approaches: restrictive and malabsorptive techniques and a combination of both (Fobi and Capella technique). The expected results of bariatric surgery include weight loss, better management of associated comorbidities and improved quality of life.^{13,14,15,16} The literature describes improved quality of life and improved respiratory and cardiovascular (chest pain, dyspnea, sleep apnea and hypertension) and metabolic parameters (diabetes and lipid disorders) associated with substantial weight loss.^{2,15,17,18,19} Perioperative mortality is around 0.3%–1.6%.^{20,24,26}

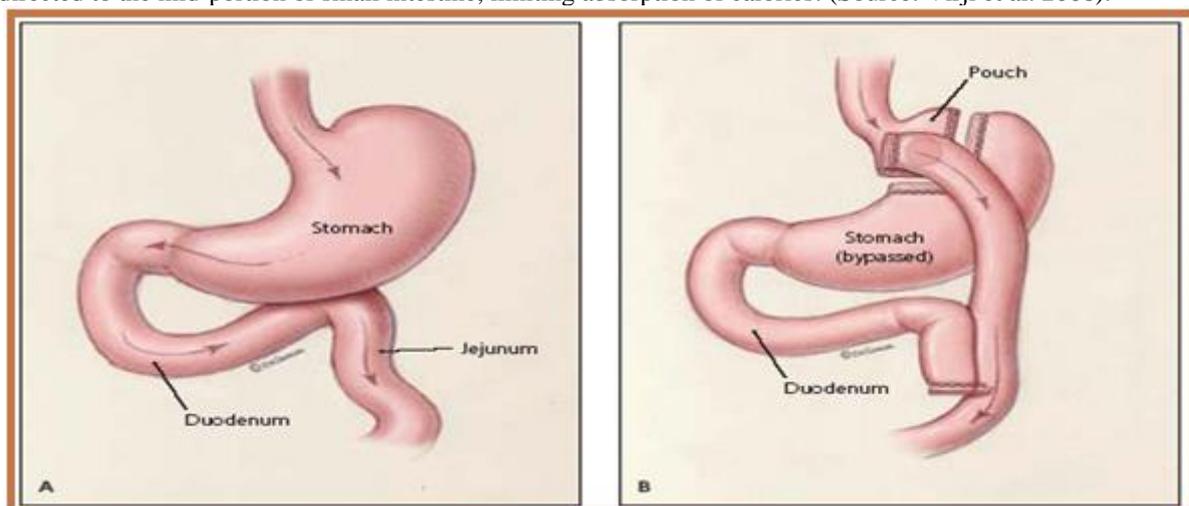
Postoperative complications of bariatric surgery are classified into early and late complications. Early common complications include infections, stomal stenosis, marginal ulceration and constipation. Infections are more common in open than laparoscopic procedures

and are seen in approximately 20% of patients. Vomiting and intolerance to fluid meals are often seen after gastric bypass procedures. Stomal stenosis can occur in 9%–20% of patients while marginal or anastomotic ulcers are seen in about 1%–16%.²⁷

A longitudinal study involving 10 obese patients 36 months postoperative of weight reduction surgery found that weight loss resulted in significant reduction of leptin, IL-6, α -defensins and C-reactive protein and significant increase in adiponectin. There were no changes in TNF- α and *bactericidal permeability-increasing protein (BPI)*. The authors concluded that weight loss after bariatric surgery can partially reverse low-grade inflammation.²⁸

Literature data on the results of *Roux-en-Y gastric bypass (RYGB) only* (Figure 2), *which is* currently the most widely used surgical procedure, include fast weight loss of up to 70% in two to three years, of which 30%–40% of body weight or 40%–60% of excess weight in the first year, with excellent control of comorbidities through a qualitative diet and moderate need for dietary restriction.

Figure 2 – (A) Stomach before bariatric surgery. (B) Stomach after surgery by Fobi & Chapel technique, the bolus is redirected to the mid-portion of small intestine, limiting absorption of calories. (Source: Virji et al. 2006).



RYGB surgery showed the highest rates of complications in the immediate postoperative period such as prolonged impaired absorption of calcium, iron and vitamins but at a lesser extent compared with purely malabsorptive procedures. RYGB is difficult to reverse and the stomach and duodenum become inaccessible for clinical investigation with moderate regurgitation and vomiting during the adaptation phase, difficult laparoscopic approach and prolonged hospital stay.^{2,8,29} A study evaluating postoperative alterations of Fobi-Capella procedure, using upper gastrointestinal series in 41 patients undergoing surgery from May 2000 to June 2005, found changes in 51.2% of radiological evaluations. Gastroesophageal reflux was the most common

alteration, followed by hiatal hernia (seven patients), and both (three patients).⁷ The expected re-operation rate due to postoperative complications is 6%–9% and pulmonary complications is 4–7%.³⁰

Long-term complications reported in the literature include chronic renal failure, symptomatic cholelithiasis, dumping syndrome, persistent vomiting, nutritional deficiencies and disorders of bone metabolism (Table 1). The prevalence of chronic renal failure (CRF) is 50% in the postoperative period. Hypovolemia, tubular occlusion, metabolic acidosis and increased free radicals are the main causes of CRF and its predictive factors are advanced age, hypoalbuminemia, and sepsis.⁸ CRF is characterized by progressive, irreversible loss of functional nephrons resulting in reduced glomerular filtration rate. The kidneys play an important role in the metabolism of calcium and phosphate and production of vitamin D as well. Renal failure patients have decreased vitamin D synthesis, which leads to reduced serum calcium and secondary hyperparathyroidism.³¹

Cholelithiasis is a common consequence of fast weight loss after surgery seen in about 50% of patients.^{1,20,27,32} Dumping syndrome is a group of procholinergetic symptoms resulting from an influx of undigested carbohydrates into the jejunum. It is a physiological response to simple sugars that is characterized by tremors, sweating, malaise, tachycardia and often severe diarrhea,^{1,27,32,33} and is due to the osmotic power of simple sugars than insulin release, which is often associated with malabsorptive procedures.^{8,27}

Patients undergoing malabsorptive procedures are at high risk for nutritional deficiencies compared with purely restrictive procedures.²⁷ Common complications are malnutrition (21% of patients), anemia (30%) and metabolic bone disease (73%).³⁴ Iron, B₁₂ and folate deficiencies are very commonly seen, especially among childbearing age women, as a result of lower absorption of iron due to decreased exposure of foods to stomach acids and no exposure of nutrients in the duodenum and proximal jejunum that are primary sites for the absorption of these nutrients.⁸ Deficiencies of iron, vitamin B₁₂ and folate have been reported in approximately 50%, 70% and 40% of patients, respectively, after Fobi-Capella surgery.^{34,35,36}

In addition, when the main calcium absorption sites in the duodenum and proximal jejunum are removed, patients have increased risk of hypocalcemia. The remaining small intestine can absorb calcium; however, the paracellular mechanism is not as effective and absorbs less than 20% of the calcium ingested orally.² Vitamin D is essential for calcium absorption. Thus, changes in bone mineral density may arise as a result of calcium and vitamin D malabsorption and secondary hyperparathyroidism, leading to osteopenia and/or osteoporosis.^{2,34,35,37,38}

A case report on caries development in a patient undergoing RYGB surgery showed that an increased intake of fermentable carbohydrates, from 3 times/day to 6 times/day, apparently may explain the higher rate of carious lesions seen in these patients. In addition, these patients usually develop lactose intolerance postoperatively due to reduced lactase production, thus hindering intake of high-calcium food sources. Calcium phosphate and casein are known to act as major anticariogenic agents due to their ability to protective tooth enamel and prevent or reduce enamel demineralization. It was also reported in this case another factor that may have contributed to caries development after surgery: xerostomia.⁵

TABLES:

Table 1 – Adverse consequences of surgical treatment techniques for obesity

Source	Type of surgery	Study design (N)	Findings
Berarducci, et al. 2009 ³⁷	Roux-en-Y Gastric Bypass (Fobi-Capella)	Longitudinal (N=167)	- Increased risk of bone fractures - Development of osteopenia and osteoporosis
Carrasco, et al., 2009 ³⁹	Roux-en-Y Gastric Bypass (Fobi-Capella)	Longitudinal (N=42)	- Significant reduction in bone mineral density
Coates, et al., 2008 ⁴⁰	Roux-en-Y Gastric Bypass (Fobi-Capella)	Longitudinal (N=15)	- Increased bone resorption and bone loss
Gasteyger, et al., 2008 ⁴¹	Roux-en-Y Gastric Bypass (Fobi-Capella)	Retrospective (N=137)	- Nutritional deficiencies regardless of postoperative nutrient supplementation.
Mahdy, et al. 2008 ⁴²	Roux-en-Y Gastric Bypass (Fobi-Capella)	Cross-sectional (N=70)	- Significant reduction in bone mass and density.
Hague, et al., 2008 ⁰⁵	Roux-en-Y Gastric Bypass (Fobi-Capella)	Case report	- Increased caries activity - Xerostomia
Pereira, et al. 2007 ⁴³	Roux-en-Y Gastric Bypass (Fobi-Capella)	Longitudinal (N=16)	- Normal PTH levels - Growth factor-1 bound to significantly less insulin - Significant bone loss (femur), not affected by overload reduction but associated with hormonal factors.
McCullough, et al, 2006 ³⁰	Gastric Banding and Roux-en-Y Gastric Bypass (Fobi-Capella)	Cross-sectional (N=106)	- Postoperative reduction in cardiorespiratory capacity in the short-term
Sánchez-Hernández, et al., 2005 ⁴⁴	Roux-en-Y Gastric Bypass (Fobi-Capella)	Prospective cohort (N=64)	- Vitamin D deficiency - Increased PTH levels - Hyperparathyroidism - Deficiency of calcium absorption
Vazquéz, et al., 2005 ⁴⁵	Gastric Banding and Roux-en-Y Gastric Bypass (Fobi-Capella)	Cross-sectional (N=52)	- No change in the levels of IL-6, TNF- α and soluble receptors.
Diniz, et al. 2004 ⁴⁶	Roux-en-Y Gastric Bypass (Fobi-Capella)	Longitudinal (N=110)	- High levels of PTH Hypocalcemia
Pugnale, et al, 2003 ³⁴	Gastric Banding	Longitudinal (n=31)	- Increased bone resorption

3 CONSEQUENCES OF BARIATRIC SURGERY FOR PERIODONTAL DISEASE

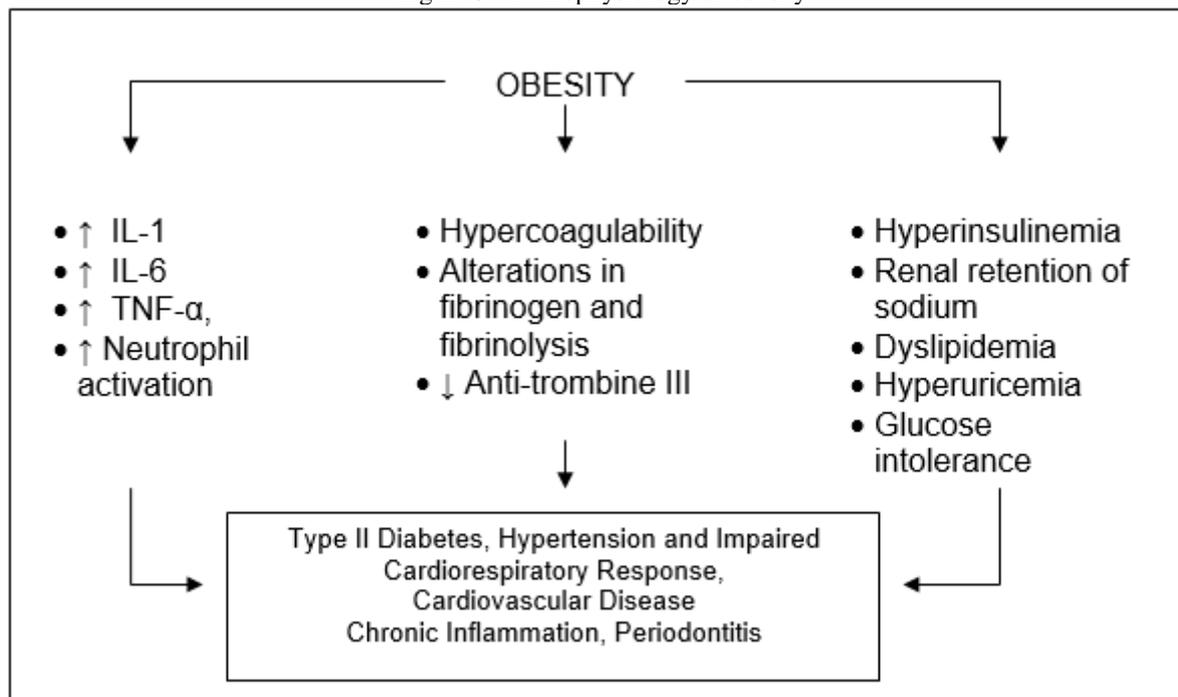
Periodontal disease (PD) is an inflammation of teeth-supporting tissues with specific histopathologic characteristics that can lead to loss of tissue attachment and bone resorption,

predisposing the individual to tooth loss and edentulism.^{47,48} It is the second most prevalent oral disease worldwide characterized by an abnormal inflammatory response to periodontal flora with production of cytokines, increased lymphocyte infiltrate, bone resorption and extracellular matrix dissolution.⁴⁹ IL-1, IL-6 and TNF- α are powerful agents for signaling osteoclast action stimulating bone resorption. IL-1 also stimulates the release of metalloproteinases that promote extracellular matrix degradation and prostaglandin E2 that causes vasodilation, edema and bone resorption as well.^{47,50,51}

Systemic disorders have been considered secondary risk factors for PD by modifying both disease onset and progression. Factors associated with PD include advanced age, bacterial plaques, immune dysfunction, nutritional deficiencies, non-steroid and steroid drug therapy, gender, stress, smoking, genetic and systemic conditions including neutrophil disorders, diabetes, pregnancy and other hormonal disturbances, and osteoporosis.^{47,52,53,54} These disorders and systemic conditions can modify the composition and physiology of some tissues, including periodontal tissues, compromising barrier integrity and host defenses against periodontal infection resulting in more destructive disease.⁵⁵ Furthermore, recent studies have showed periodontal infections potentially have adverse effects on systemic health and they have been associated in the literature with coronary heart disease,^{56,57,58} diabetes mellitus,⁵⁹ stroke, premature birth and low birth weight babies,^{60,61} and respiratory diseases.⁶²

Obesity, like periodontal disease, is also characterized by a chronic inflammatory state that compromises both immune function and metabolism including hypercoagulability and insulin resistance^{8,63} (Figure 3). Adipose tissue has a powerful ability to release major inflammatory agents such as TNF- α and IL-6. There are also changes in neutrophil mobility and activation, high levels of fibrinogen and plasminogen activator inhibitor,¹ decreased antithrombin III and fibrinolysis, and high levels of C-reactive protein.^{8,9,64,65,66,67} Inflammatory mediators are also high in patients with comorbidities of obesity regardless of their body mass.^{2,9,64,66} Dyslipidemia, hyperuricemia and glucose intolerance associated with insulin resistance create a state known as metabolic syndrome, a risk condition for the development of systemic atherosclerotic disease, especially coronary disease, and clearly associated with type 2 diabetes,^{8,68,69} and increased risk of periodontal disease.⁷⁰

Figure 3 – Pathophysiology of obesity



Several recent cross-sectional and prospective studies have evidenced a correlation between obesity and chronic inflammatory diseases, including periodontal disease, with the finding of proinflammatory cytokines that can make the link between obesity and severity of periodontal disease.^{10,52} In addition to poor glucose tolerance and dyslipidemia, excess fat tissue appears to be a large reservoir of adipokines that act promoting periodontal destruction.^{3,71,72,73} IL-1, IL-6 and TNF- α are found at high levels in obesity, mostly produced by activated macrophages in adipose tissue.^{70,74} Obesity has a powerful action on the immune system in the presence of high serum fatty acids and cholesterol levels along with lipid solubility of some vitamins, including vitamin D, resulting in reduced vitamin availability and affecting bone turnover.^{75,76}

Since bariatric surgery has gained importance as a major treatment modality for obesity, there is a growing concern about its long-term health consequences. Studies have suggested that bariatric surgery may cause malabsorption of fat-soluble vitamins due to poor mixing with bile salts with greater decrease in total vitamin D available and thus lower calcium absorption.^{2,38,51}

One of the body's mechanisms to maintain normocalcemia would be an increase in the production of 1,25-dihydroxy vitamin D (cholecalciferol) and, more importantly, an increase in bone calcium reabsorption.² Cholecalciferol deficiency caused by inadequate absorption of dietary calcium and phosphate results in increased secretion of parathyroid hormone (PTH),⁴⁶ which in turn mobilizes bone calcium, leading to secondary hyperparathyroidism and consequent

osteopenia, osteoporosis, and eventually, osteomalacia.^{2,34,35,36,77} The magnitude of bone loss varies from 3% to 9% depending on the body site examined in the first year after surgery.^{35,36} An evaluation of 26 patients 10 years after surgery, compared with controls, found lower serum calcium, increased alkaline phosphatase, lower cholecalciferol and decreased bone mass.⁴³

More specifically in obese individuals, the availability of vitamin D, and consequently cholecalciferol, is limited by their solubility in adipose tissue.⁷⁸ In addition to a skeletal homeostasis action, cholecalciferol has antimicrobial and anti-inflammatory effects by modulating cytokines production by immune cells and stimulating the secretion of peptides with antibacterial actions by monocyte-macrophage cells.^{79,80} With accumulating evidence on the role of genetic alterations in vitamin D receptors (VDR) in bone metabolism and homeostasis, osteopenia and systemic bone loss resulting from reduced dietary calcium and vitamin D absorption has been directly associated with alveolar bone, clinical attachment loss and tooth loss.^{80,81,82} The results of some studies point to a significant association between VDR gene polymorphism and development of chronic periodontitis⁸³ and generalized aggressive periodontitis.⁸⁴

A study compared the effects on bone metabolism of purely restrictive with malabsorptive procedures and found that patients undergoing restrictive surgery had early increase in bone turnover, particularly increased bone resorption markers. As for the consequences of malabsorptive procedures, the literature has demonstrated strong evidence of vitamin D deficiency, secondary hyperparathyroidism with increased bone turnover, osteopenia, osteoporosis and osteomalacia.^{34,43,85}

Osteopenia is a condition characterized by low bone mass and osteoporosis is the most severe form of osteopenia with episodes of pain, deformities and fractures.^{39,47} Osteoporosis is a condition associated to age, gender and physiology with bone mass loss and changes in bone structure affecting both cortical and trabecular bone.⁵⁴ Bone mineral loss is twice as high in women than men.^{39,47} Contributing factors for the development of osteoporosis include inadequate intake of calcium, physical inactivity, weight loss and heredity.^{39,87}

Recent studies have revealed that reduced bone density can indicate a risk for periodontal disease.^{88,89,90} Treatment strategies that are used to prevent and treat osteoporosis can also inhibit periodontal bone loss. Patients with systemic bone density loss are likely to have decreased bone density in the maxilla and mandible, including bone loss in the alveolar process associated with periodontal infection.⁴⁷ which may require different treatment strategies for these patients.⁹¹ Similar to periodontal disease, osteoporosis has a silent onset with no early symptoms, and both conditions share common risk factors including increased prevalence with age, smoking, presence

of systemic disorders and drug use.⁵⁰ Thus, alveolar bone loss is more likely to occur in individuals with osteoporosis, especially those with preexisting periodontitis. It would indicate that osteoporosis or low bone mineral density should be considered a risk factor for progression of periodontal disease.⁸⁸ One hypothesis that may explain this association is that osteoporosis causes alveolar bone crest loss as it can be stimulated by the presence of periodontitis.⁴⁷

The interactions between many systemic health changes occurring in pre- and postoperative bariatric patients seem to contribute to the progression and severity of periodontal disease.

4 FINAL CONSIDERATIONS

As patients are increasingly turning to bariatric surgery as an effective resort to fight obesity and its comorbidities, the consequences of this surgical treatment are gaining importance and attracting the interest of the scientific community.

Candidates for bariatric surgery are required to be patients with high-grade obesity and/or comorbidities, and possibly a great deal of them also suffer from periodontal disease. And since postoperative patients can have bone metabolism changes with reduced bone mass concentration and density along with nutritional deficiencies, their periodontal disease can become more serious leading to tooth loss. On the other hand, periodontally healthy patients undergoing bariatric surgery may have periodontal disease as long-term adverse effects of surgery, including larger daily amounts of food intake without adequate oral hygiene, and osteoporosis, among other factors, may contribute to its development.

This is an evidence-based hypothesis formulated based on current literature. However, further investigations are needed to test this hypothesis, including data from prevalence studies. It is thus recommended the evaluation of this issue while monitoring bariatric patients.

STATEMENT

The authors state no conflict of interest in this scientific study.

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