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THE ASSOCIATION OF OBESITY AND PERIODONTAL DISEASE: A NARRATIVE REVIEW

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ABSTRACT

Introduction: Obesity can be defined as an abnormal or excessive accumulation of fat in adipose tissue related to the lean mass. The body mass index (BMI) is the classification widely used and characterize an individual with obesity when it results in a value equal or greater than 30 Kg/m^2. It has been recognized the role of this disease in exacerbating periodontal inflammation and increasing the risk of developing periodontitis. Aim: The aim of this study was to explore and summarize the pathways involved in the relationship between obesity and periodontal disease. Material and methods: An advanced search was made in the PubMed database using combinations of the terms “obesity,” “overweight,” “periodontitis,” and “periodontal disease.” Additionally, a thorough examination of the reference lists of the included articles was performed without any constraints. Data was synthesized in a narrative fashion. Results: Obesity and periodontal disease are chronic conditions with overlapping risk factors. Their association can be elucidated through multiple mechanisms, primarily involving sustained inflammation in individuals with obesity, mediated by cytokine secretion, leading to compromised immune function. Furthermore, this relationship appears to be bidirectional, as tooth loss resulting from periodontal disease can disrupt impaired eating patterns, potentially contributing to obesity. Conclusion: It can be concluded that there appears to be a bidirectional association between obesity and periodontitis, which can be attributed to several underlying mechanisms, especially related to the inflammatory state of these conditions.

Key words: Obesity. Periodontal disease. Periodontitis. Inflammation.

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RESUMO

Associação entre obesidade e doença periodontal: revisão narrativa

Introdução: Obesidade pode ser definida como o acúmulo anormal ou excessivo de tecido adiposo em relação à massa magra. O Índice Massa Corporal (IMC) é a classificação largamente utilizada e caracteriza um indivíduo com obesidade quando resulta em um valor igual ou maior que 30 Kg/m^2. Tem sido reconhecido o papel dessa doença em exacerbar a inflamação periodontal e aumentar o risco para o desenvolvimento da periodontite. Objetivo: O objetivo desse estudo foi explorar e sumarizar os mecanismos envolvidos na relação entre obesidade e doença periodontal. Materiais e métodos: Uma busca avançada foi realizada na base de dados PubMed usando uma combinação dos termos “obesidade”, “sobrepeso”, “periodontite” e “doença periodontal”. Além disso, as listas de referências dos artigos incluídos foram revisadas sem impor qualquer restrição. Os dados foram sintetizados de forma narrativa. Resultados: A obesidade e a doença periodontal são condições crônicas com fatores de risco sobrepostos. Suas associações podem ser elucidadas por meio de múltiplos mecanismos, primariamente envolvendo inflamação crônica em indivíduos com obesidade, mediada pela secreção de citocinas, levando a um comprometimento da função imune. Além disso, essa relação parece ser bidirecional, pois a perda dentária que resulta da doença periodontal pode desencadear padrões alimentares deficientes, potencializando fatores que contribuem para a obesidade. Conclusão: Foi concluído que parece haver uma associação bidirecional entre obesidade e periodontite, a qual pode ser atribuída por vários mecanismos, especialmente aqueles relacionados com o estado inflamatório dessas condições.

Palavras-chave: Obesidade. Doenças periodontais. Periodontite. Inflamação.

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INTRODUCTION

According to the World Health Organization (WHO), obesity is a chronic and multifactorial disease whose prevalence has increased considerably in recent decades, more than doubling between 1990 and 2022 (WHO, 2024).

Affecting approximately 16% of the world's adult population, this disease, in which there is an increase in body fat deposition related to the lean mass, can cause low-grade chronic inflammation (Ellulu et al., 2017; WHO, 2024).

A classification widely used by the WHO to determine an obese individual is the body mass index (BMI), calculated by dividing the weight in kilograms by the square of the person's height in meters (Kg/m^2). Based on this calculation, a BMI ≥25 Kg/m^2 is considered overweight and a BMI ≥30 Kg/m^2 is classified as obesity (WHO, 2020; WHO, 2024).

Because it is a multifactorial condition, there are several etiological factors involved in the development of obesity, including genetic, biological, social, and behavioral factors, such as excessive intake of a high-calorie diet, associated with a sedentary lifestyle (Khan et al., 2020).

Obesity has several negative implications for an individual's systemic condition, including hypertension, type II diabetes, coronary heart disease, stroke, osteoarthritis, and its presence significantly reduces life expectancy (Kim e Popkin, 2006).

In the context of oral health, the role of obesity in exacerbating periodontal inflammation and increasing the risk of developing periodontitis through deficient immune responses has been recognized (Jepsen et al., 2018).

This chronic, multifactorial, and inflammatory disease is characterized by the progressive destruction of the tooth-supporting apparatus and associated with dysbiotic biofilm (Papapanou et al., 2018).

Among periodontal diseases, periodontitis is the most frequently correlated with obesity, and it also presents a greater number of biological mechanisms that explain this association (Çetiner et al., 2019).

Adipose tissue is responsible for the metabolism and conversion of energy and is associated with the secretion of chemical substances and inflammatory responses (Bray, 2004; Lyon et al., 2003).

The large quantities of tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), interleukin-8 (IL-8) and plasminogen activator inhibitor-1 (PAI-1) secreted by adipose tissue are proportional to the BMI of obese individuals (Nishimura e Murayama, 2001).

These substances increase the production of degradation enzymes and cause low-grade inflammation, playing a significant role in the development of periodontitis (Martinez-Herrera et al., 2017; Ritchie, 2007; Thomas et al., 2020; Usui et al., 2021).

An additional mechanism linking obesity and periodontal disease is elucidated through the presence of type II diabetes (Pamuk e Kantarci, 2022).

This metabolic disorder, characterized by excess weight as a primary risk factor, serves as both a predisposing and modifying factor for periodontitis (Genco e Borgnakke, 2020; Papapanou et al., 2018). Consequently, individuals with obesity are at an increased susceptibility to developing diabetes, consequently heightening their risk for periodontal disease.

Given these facts, this narrative review aims to explore and summarize the main pathways involved in the relationship between obesity and periodontal disease.

MATERIALS AND METHODS

The studies included in this review were identified through comprehensive searches conducted in the PubMed database.

Various combinations of the following terms were employed as search criteria, requiring their presence in the title or abstract of the article: "obesity," "overweight," "periodontitis," and "periodontal disease." Furthermore, supplementary searches were conducted within the reference lists of the chosen studies.

All articles regardless of study design were included and there were no language restrictions. Articles were selected based on their titles indicating a primary focus on investigating the relationship between obesity and periodontal disease.

Articles addressing diseases or comorbidities in their titles beyond this scope were excluded. Data synthesis was performed in a narrative fashion.

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Obesity

Obesity is considered the most common metabolic and nutritional disorder among the world's population and can be defined as an abnormal or excessive accumulation of fat in adipose tissue (Bray, 2003; Izaola et al., 2015).

Excess weight is a major public health problem worldwide, representing health risks and being responsible for 3.4 million deaths annually (Hruby e Hu, 2015; Ng et al., 2014).

he development of obesity is influenced by multiple factors, including genetics, behavior, social environment, metabolism, and hormones (Franco et al., 2020).

These factors likely interact with each other, resulting in a persistent imbalance between the consumption and expenditure of energy, which characterizes obesity as a chronic condition (Khan, et al., 2020).

The BMI classification for obese individuals is subdivided into: Grade I, which BMI ranges from 30-34.5 Kg/m^2; Grade II from 34.5-40 Kg/m^2; Grade III >40 Kg/m^2 (Tjepkema, 2006).

Another measure of obesity is waist circumference, which is strongly associated with cardiovascular disease and consequently greater morbidity (Abu-Shawish et al., 2022).

Studies have indicated that this measurement may be a better predictor of disease risk than BMI (Wang et al., 2005; Yusuf et al., 2005). The cut-off point for abdominal obesity is 88cm for women and 102cm for men (Panel, 1998).

Waist circumference has a close correlation with the amount of visceral adipose tissue, located between various organs, which is metabolically more active, secreting greater amounts of cytokines when compared to subcutaneous tissue (Berg Scherer, 2005; Wanjchenberg, 2000).

As an active endocrine organ, adipose tissue releases various immunoregulatory factors, modulating metabolic and vascular biology, as well as producing and secreting a wide variety of pro-inflammatory molecules (Coelho et al., 2013; Izaola et al., 2015).

Adipocytes, the most abundant cells in this tissue, secrete adipokines, bioactive peptides responsible for changes in systemic inflammation (Cruz-Ávila et al., 2022).

The large quantity of these peptides promotes a shift in macrophages towards a pro-inflammatory state (Franco et al., 2020).

These cells, present in adipose tissue in a proportion related to the level of adiposity, release various molecules, also related to the pro-inflammatory state, such as leptin, TNF-α, interleukin 1β (IL-1β), and IL-6 (Franco et al., 2020).

Because of these diverse factors, obesity commonly correlates with elevated blood lipid and glucose levels, concomitant with a state of low-grade inflammation (Denke et al., 1994; Dixon e O'Brien, 2001; Hsieh et al., 2000; Tanaka et al., 2001).

The imbalance in the immune system can be considered a risk factor or aggravating factor for a series of chronic non-communicable diseases, such as cardiovascular diseases, hypertension, type II diabetes, osteoarthritis, cancer, and potentially also for periodontitis (Ard, 2015; Field et al., 2001; Piche et al., 2020; Smith e Smith, 2016; Suvan et al., 2011).

Periodontal disease

Periodontitis represent a group of disorders located in the periodontium, affecting the supporting structures of the teeth (the cementum, periodontal ligament, and alveolar bone), constituting one of the main causes of tooth loss in adults (Girano-Castaños e Robello-Malatto, 2020; Silva-Boghossian e Dezonn, 2021; Toker et al., 2018).

Gingivitis and periodontitis are the two most predominant diseases among periodontal conditions, with periodontitis being the most severe. Its global prevalence is estimated at 62%, representing a global public health problem (Dumitrescu, 2015).

The most common clinical manifestations are gingival inflammation and gingival recession, alveolar bone loss, and tooth mobility (Lang et al., 1990; Page, 1998).

Patients with periodontal diseases have an increased risk for edentulism and masticatory dysfunction, negatively impacting their nutrition, quality of life, and self-esteem (Reynolds e Duane, 2018).

Gingivitis is an inflammatory reaction of the gingival margin, induced by pathogens residing in the bacterial plaque which is accumulated on the dental surface.

In this disease, when the local etiologic factor is removed, there is healing without the destruction of structures or sequels. On the other hand, periodontitis is considered a destructive and irreversible inflammatory disease, resulting from the progression of

untreated gingivitis (Larsen e Fiehn, 2017; Van Dyke e Sheilesh, 2005).

Periodontitis is characterized by inflammation that extends deep into the periodontal tissues, causing degeneration and destruction of tooth support (Martínez-Aguilar et al., 2017).

The etiology implies that a bacterial infection is the main cause, and certain genetic and acquired risk factors can modulate susceptibility or resistance to the disease (Genco e Borgnakke, 2020; Salvi et al., 1997; Van Dyke e Sheilesh, 2005).

When bacterial plaque sustains gingival inflammation, it can progress to the destruction of the underlying connective tissue and alveolar bone due to toxic byproducts associated with microbial changes found in the gingival sulcus (Ebersole et al., 1993; Lang et al., 2009; Schatzle et al., 2004).

Lipopolysaccharides prompt a local inflammatory response, characterized by the infiltration of neutrophils, lymphocytes, and macrophages, followed by the secretion of pro-inflammatory cytokines.

Subsequently, there is activation of host metalloproteinases, leading to the breakdown of periodontal ligament fibers, and migration of the junctional epithelium, resulting in the apical dissemination of subgingival bacterial biofilm along the root surface (Ebersole et al., 1993; Page, 1991; Socransky e Haffajee, 2005; Tonetti et al., 2018).

In this biofilm, some proteolytic bacteria closely associated with periodontitis predominate, including Porphyromona gingivalis, Prevotella intermedia, Tannarella forsythia, and Treponema denticola, contributing to dysbiosis and the consequent initiation of an inflammatory response and disease state (Hajishengallis e Lamont, 2012; Socransky e Haffajee, 2005).

Clinically, the diagnosis is established by detecting interproximal clinical attachment loss in two or more non-adjacent sites, or clinical attachment loss of 3mm or more on the buccal or lingual face of at least two teeth, added to bleeding on probing indicative of inflammation (Tonetti et al., 2018).

Classification of periodontitis is done according to stages and grades. The stage categorizes the severity, extent, and complexity of the disease and it is classified from I, an early phase of the disease, to IV, the most advanced one.

The grading system relies on a retrospective evaluation of the disease progression rate, considering the risk of future advancement, given the variability in progression rates among individuals and variations in treatment response. It is classified in grade A, B and C and this classification system incorporates smoking and diabetes as risk factors and grade modifiers (Papapanou et al., 2018; Tonetti et al., 2018).

Periodontitis therapies include scaling and root planing, often associated with the systemic administration of antibiotics and oral antiseptics (Lin et al., 2022). The aim is to reduce the number of microorganisms in the biofilm and disrupt its ecology, eliminating both live bacteria and those in the calcified biofilm (Adriaens e Adriaens, 2004; Nascimento et al., 2016).

Once periodontal therapy is complete, personalized maintenance protocols should be established, with time intervals between appointments determined according to each patient's risk factors (Kumar, 2019).

Supportive therapy is essential for improving periodontal health, and helping to reduce tooth loss (Trombelli et al., 2015).

Pathways for the relationship between obesity and periodontal disease

The growing interest in the relationship between obesity and oral health has suggested an association between obesity and periodontitis (Dalla Vecchia et al., 2005; Saito et al., 2001; Wood et al., 2003).

Both conditions are non-communicable chronic inflammatory diseases sharing several risk determinants, including age, socioeconomic factors, diet, and lifestyle (Jepsen et al., 2020).

The first association between obesity and periodontal disease in humans was identified in a study by Saito et al., (1998), with periodontal parameters affected by this relationship, such as increased probing depth and clinical attachment loss.

Several pathways explain this relationship, with systemic inflammation being a primary mechanism (Figure 1) (Marruganti et al., 2023).

Obesity can induce or exacerbate a chronic pro-inflammatory state. This could alter the microenvironment of periodontal sites, promote the growth and complexity of oral microflora, and affecting immune responses

due to an imbalance in the levels of pro-inflammatory cytokines in the bloodstream (Aronson et al., 2004; Ellulu et al., 2017; Genco et al., 2005; Pischon et al., 2007; Zelkha et al., 2010).

Individuals with obesity may exhibit an altered composition and diversity of the periodontal microbiota, with higher proportions of periodontopathogens, such as Tannarella forsythia, when compared to normal weight individuals (Haffajee e Socransky, 2009).

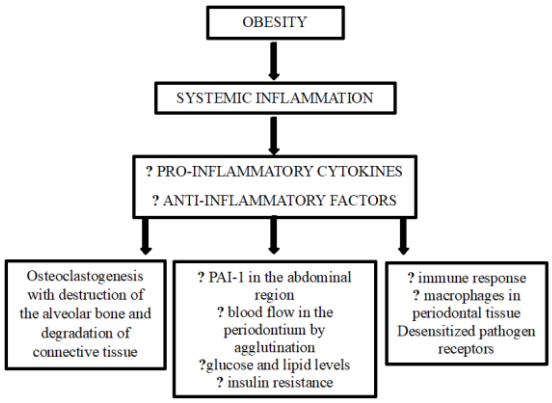

Figure 1 - Several pathways explaining systemic inflammation.
Legend: PAI-1: Plasminogen Activator Inhibitor - 1

Relative to its size and the individual's BMI, adipose tissue secretes substantial amounts of pro-inflammatory adipokines, including IL-1β, IL-6, IL-8, TNF-α, PAI-1, and C-reactive protein (Coelho et al., 2013; Nishimura e Murayama, 2001).

IL-6 and TNF-α, involved in the pathophysiology of obesity and periodontitis, besides leptin, resistin, and others, are secreted in greater quantities in obesity. At the same time, the anti-inflammatory factors have their quantities (Genco et al., 2005; Nakamura et al., 2014).

They cause changes in glucose metabolism, resulting in increased secretion, elevated BMI, and enlarged adipocyte size (Crujeiras et al., 2015; Castilhos et al., 2012; Lee e Jeong, 2020; Seymour e Gemmell, 2001). The pro-inflammatory markers, in response to external and internal stimuli, are also released by the immune cells of the periodontium, such as macrophages and lymphocytes (Slots, 2017).

TNF-α is released by these cells in the junctional epithelium around the gingival sulcus and the host response to pathogens, they induce osteoclastogenesis, with consequent destruction of the alveolar bone, as well as degradation of the connective tissue (Khosravi et al., 2013; Offenbacher, 1996).

It is crucial to consider that hypertrophy of adipose tissue can lead to hypoxia in certain adipocytes, thereby enhancing the chemoattraction of monocytes to the tissue. Consequently, there is an escalation in the secretion of pro-inflammatory cytokines, contributing to the development of low-grade systemic inflammation (Cancello e Clement, 2006).

The immune response of the obese host is then weakened and the migration of macrophages to the periodontal tissues is reduced (Huang et al., 2016).

Furthermore, cytokines desensitize important receptors involved in pathogen recognition within periodontal tissue cells, impairing their ability to effectively identify periodontopathogens (Suvan et al., 2011).

Alterations in pro-inflammatory markers observed in saliva and gingival crevicular fluid correlate with adipose tissue excess and contribute to the initiation and exacerbation of inflammatory disorders such as gingivitis (De Castilhos et al., 2012).

With the predominant role of gram-negative bacteria in destructive periodontal disease, the epithelium of the ulcerated pocket becomes a chronic source of bacterial products, as well as locally produced inflammatory mediators (Grossi e Genco, 1998).

With an inflamed periodontium and consequent increased vascularization, the lipopolysaccharides of these bacteria trigger the secretion of IL-6 and TNF-α, which predispose to periodontal inflammation and metabolic disorders such as hepatic dyslipidemia, insulin resistance and increase the risk of obesity and type II diabetes mellitus (Nishimura et al., 2003; Saito et al., 2001).

It highlights that a typical condition observed in individuals with obesity is compensatory hyperinsulinemia, in which higher levels of insulin are produced by the pancreas, inducing dyslipidemia and consequently altering plasma concentrations of total cholesterol, triglycerides, and fatty acids (Genco et al., 2005).

Insulin resistance is induced by apoptosis of the beta cells of the pancreas, which can predispose to diabetes and also periodontal disease progression (Genco et al., 2005; Nishimura et al., 2003).

Furthermore, the distribution of body fat plays an important role in the host's immune

response. The main pro-inflammatory adipokines are produced mostly by abdominal adipose tissue and the accumulation of fat in this region can increase in glucose and lipid levels and also in insulin resistance (Brochu et al, 2000).

In addition, PAI-1 has an increased genetic expression in this area, inducing blood agglutination. In turn, blood flow in the periodontium region is reduced, increasing the risk of developing periodontitis (Shimomura et al., 1996; Wood et al., 2003).

Among the adipokines secreted by adipose tissue, leptin has been shown to negatively interfere with the regenerative capacity of periodontal ligament cells, causing impaired healing (Nokhbehsaim et al., 2014).

C-reactive protein is an acute-phase protein expressed in response to systemic inflammation and there are reports of an increase in its serum levels in obese individuals (López et al., 2012; Thanakun et al., 2017).

In contrast, adiponectin is a circulating hormone involved in glucose and lipid metabolism. It improves insulin sensitivity, has anti-inflammatory properties, and is found at lower levels in obese individuals (Nakamura et al., 2014).

A possible bidirectional association between obesity and periodontitis underscores the intricate relationship between these conditions (Figure 2).

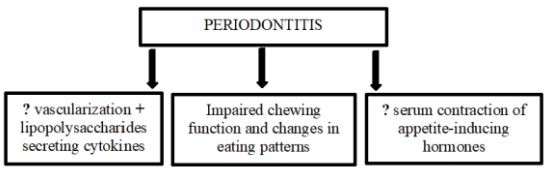

Figure 2 - Possible bidirectional association between obesity and periodontitis.

The impairment of masticatory function by periodontitis, caused by tooth mobility and tooth loss, lead to change in their eating patterns and consume more palatable, soft foods that are high in fat and calories, leading to weight gain and obesity (Borges et al., 2013; Kosaka et al., 2016).

In addition, individuals with periodontitis have been found to have higher serum concentrations of appetite-inducing hormones such as ghrelin (Yılmaz et al., 2014).

There is also the possibility that endotoxemia induced by Porphyroma gingivalis affects obesity by altering the endocrine functions of adipose tissue (Hatasa et al., 2021).

This process involves the action of virulence factors from periodontal pathogens on macrophages, monocytes, and fibroblasts, eliciting an inflammatory response in periodontal tissues mediated by inflammatory mediators and facilitating the progression of tissue destruction.

Host-derived and microbial factors escape from the site via the bloodstream, contributing to the pathophysiology of various systemic diseases, including obesity (Konkel et al., 2019; Iwashita et al., 2021).

CONCLUSION

This review highlights the association between obesity and periodontal disease, which can manifest through several pathways.
While obesity often contributes to the onset or progression of periodontal disease, the reverse relationship may also occur.

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