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Obesity as a Risk Factor for Periodontal Disease

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Abstract

Obesity is a chronic, multifactorial pathological entity characterized by increased body fat associated with increased health risk and is considered a global epidemic that constitutes a major public health problem in developed countries and even in developing countries. process of development. It is a risk factor for different systemic diseases, including periodontal disease. This supports that both are chronic inflammatory entities and their strength of association linked to a common pathophysiology, chronic inflammation, has been justified by different studies, as well as both presenting a high incidence rate worldwide. Therefore, it is proposed in this work to delve into the influence of obesity on chronic immunoinflammatory periodontal disease to contribute to the improvement of oral health.

Keywords: periodontal disease; obesity; periodontitis

Introduction

Obesity is a chronic, multifactorial pathological entity characterized by increased body fat associated with greater health risk. It is described as the hypertrophy of adipose tissue in the body and develops from an interaction between genotypic and environmental, it is commonly defined as a complex, chronic and multifactorial metabolic disorder [1,2] It is the most common nutritional disorder in America and It is a significant risk factor due to its various morbidities such as: diabetes mellitus, cerebrovascular and cardiovascular diseases, systemic arterial hypertension, dyslipidemia, arthropathies in the lower limbs, some varieties of neoplasms, among other diseases; which have contributed to it being considered a relevant problem from an epidemiological point of view [1,3].

The World Health Organization (WHO) defines obesity as a body mass index greater than 30 kg/m2. It is found by calculating the individual's body weight in kg and dividing it by the square of their height in meters [4,5]. Similarly, the body mass index (BMI) is used to determine its existence, defining overweight when the BMI is 25-29 and obesity as a BMI equal to or greater than 30. BMI (kg/ m2) Interpretation: < 18.50 Underweight; 18.5-24.99 Normal; 25.0-29.9 Overweight and 30.0-34.9 Obesity [4,6]. It is considered a global epidemic since it constitutes a major public health problem in developed countries and even in developing countries. Epidemiological studies suggest that the main causes are related to environmental and lifestyle changes that have occurred in recent decades. Although obesity is a multifactorial disorder, excessive intake of energy-dense foods and a sedentary lifestyle are considered the main triggers and, consequently, the prevention of overweight and obesity should be based on changes in these factors. Although it may seem like an antithesis, the greatest increase in cases of obesity and overweight occurs in countries associated with famine such as Africa and India [7,8]. In Mexico, the results of the National Health and Nutrition Survey (Ensanut) 2020, published on June 11, show that the adult population is 74% obese, establishing differences between the female sex with 76%. and the male only with 72% [9]. In the United States, it is estimated that 40% of adults and 18.5% of children age suffer from obesity; Through the examination of the 2012 National Health and Nutrition Examination Survey, NHANES, for its acronym in English, reported that in less than two decades, the obesity rate among adults increased 30%, while that among minors, it increased by 33% [10]. In Europe, the numbers of the problem are somewhat lower than those of the United States but also alarming, noting that the prevalence of obesity is higher in women [11]. In the Americas, according to the FAO, 58% of the inhabitants live with overweight and obesity for a total of 360 people, with Chile at 63% and the Bahamas at

69% representing the highest rates. It should be noted that this increase in Latin America and the Caribbean disproportionately impacts women in more than 20 countries, being 10% higher than in the opposite sex [8]. In Cuba, data provided by national risk factor surveys conducted indicate that overall overweight has increased in the population by 42% and by 13% in children [12].

Regarding periodontal diseases (PD), such as gingivitis induced by dental biofilm and periodontitis, they are stealthy infections that do not cause discomfort or pain in their initial stages, which is why most patients and professionals overlook them and go undetected. opportunely. They are among the most common conditions of the human race. It encompasses a group of clinical conditions of multifactorial origin that affect the structures of the periodontium in the form of single inflammatory processes (gingivitis) or associated with destructive processes (periodontitis). These diseases are caused by the accumulation of bacteria (biofilm), which would act on a susceptible host. Gingivitis, periodontitis or both affect approximately more than 70% of the adult population [13,14].

Epidemiological studies carried out around the world have confirmed that gingival inflammation is present in the majority of populations, but that the most severe phases of periodontal disease, although not as prevalent as previously believed, are still of considerable magnitude. and affect 15-20% of most population groups over 35 years of age [15,5]. In Cuba it has been shown that approximately 80% of school-age children and 70% of the adult population have suffered from gingivitis or periodontitis, or both [16].

The first report of the relationship between obesity and periodontal disease appeared in 1977, when Perlstein and collaborators found that bone resorption was greater in obese Zucker rats compared to non-obese ones; Based on this, a series of studies have been carried out to determine whether or not there is a relationship between obesity and periodontal disease [1,17]; However, it was not until 1998 when Saito et al; reported for the first time in humans the links between these two pathologies.

A cross-sectional study of adolescents in NHANES III found that obese young adults between 18 and 34 years of age had 76% (95% CI: 19-161%) increased prevalence of periodontitis compared with normal weight subjects. Which suggests that periodontitis may be related to lifestyles associated with adiposity. Various clinical articles on obesity and periodontal disease have reported a large number of systematic comments. The medical literature more specifically suggests that obesity may negatively affect periodontitis, but evidence from clinical data or biological plausibility that periodontitis may affect obesity is limited [18,19].

The association between obesity and periodontal disease is one of the most recent fields of research in periodontics. It is said that adipose tissue releases proinflammatory cytokines and hormones known as adipocytokines, which induce inflammatory processes, generating a similar pathophysiology between both diseases (2,7). Therefore, the objective of this work is to delve deeper into the influence of obesity. in chronic immunoinflammatory periodontal disease.

Development

Obesity can be considered a low-grade chronic inflammatory pathology, characterized by an elevation in plasma levels of proinflammatory cytokines. An important fact in the inflammatory state associated with obesity is that it appears to be predominantly provoked and reside in white adipose tissue although other key metabolic organs may also be involved in the course of inflammation. Therefore, this inflammatory state has been proposed as a link between periodontal disease. Its strength of association linked to a common pathophysiology, chronic inflammation, has been justified by different studies, as well as both have a high incidence rate worldwide [20].

Inflammation is generally defined as a localized response subsequent to damage to a biological tissue. The harmful agent can be external, of a chemical, physical, or biological nature and can also be derived from within the body, as part of an existing pathology, highlighting here autoimmune diseases and cancer. The inflammatory response that occurs is a form of protection for the affected organism, since inflammatory processes induce the isolation, containment and destruction of the causal agent [21]. Chronic low-grade inflammation is characterized by elevation in circulating levels of inflammatory cytokines, infiltration of macrophages in peripheral tissues. This process does not induce injury or loss of functionality of the infiltrated tissue. It is mainly associated with the development of cardiometabolic diseases in patients with obesity, which is why this immune evolution is considered meta inflammation. In contrast, chronic inflammation can last months or

years, even after the acute inflammation that triggered it disappears. Some of the possible causes that may develop this response are: failures in the resolution of acute inflammation due to pathogens, persistence of foreign bodies and/or autoimmunity problems. Consequently, chronic inflammation generates serious damage, triggering neovascularization, fibrosis and tissue necrosis, causing damage to organs and increasing the risk of diseases, therefore, there is no favorable recovery and, in addition to this, positive feedback is generated. of the inflammatory process [22].

The connection between obesity and periodontal disease is due to the 50 bioactive substances that are secreted by adipose tissue. It is a loose tissue formed by adipocytes, whose main role includes insulation, cushioning and nutritional reserve. It can be formally considered an endocrine tissue, since it produces and secretes peptides with different effects that exert their action in distant tissues (endocrine effect), in contrast to local effects (paracrine or autocrine). Endocrine cells are classically controlled by external stimuli that generate a feedback mechanism. This tissue also participates in the processes of inflammation, metabolic energy regulation, atherosclerotic vascular disease, metabolic syndrome and cancer [1,2,4].

Generally, the adipocytes of an obese person suffer a thickening, causing the adipose tissue to lose its original shape, altering its correct functioning and increasing the proliferation of adipokines, which generates changes in the metabolism, producing inflammation. The release of adipokines with anti-inflammatory action compared to those with pro-inflammatory action and their imbalance promote adipocyte dysfunction and influence the development of metabolic disorders that accompany obesity [23].

Adiponectin is a protein hormone that modulates the number of metabolic processes, including glucose regulation, blood pressure, fatty acid catabolism, and has an inverse relationship with markers of inflammation. This means that it is an anti-inflammatory that is reduced in obese patients. These anti-inflammatory properties act as endogenous modulators of obesity-related diseases. Because down-regulation of adiponectin gene expression in adipose tissue directly or indirectly affects C-reactive protein levels, elevation of adiponectin gene expression might be necessary to explain the precise relationship between obesity and PD. These pathologies can, independently or together, alter local and systemic levels of adipocytokines, especially in favor of pro-inflammation. High levels of adipokines from visceral fat induce blood agglutination in the microvasculature, decreasing blood flow to the gingiva in obese individuals and thus facilitating the progression of periodontal disease [4].

The biological plausibility that correlates to both pathologies is based on the involvement of pro-inflammatory cytokines such as TNF α, IL-1, IL-6 and PAI-1, derived from adipose tissue, which are molecules that affect the whole-body metabolism and contribute to the development of low-grade systemic inflammation. Therefore, if they increase, it translates into a greater activation of the host response to inflammation, therefore the obese subject is more susceptible to PD [23].

The TNFα found in gingival crevicular fluid is derived from the adipose tissue of obese patients. Now if this cytokine is elevated, it activates the plasogen activator inhibitor -1 (PAI-1), which will inhibit the plasmogenes, which results in increased blood agglutination that produces a decrease in blood flow in the microvasculature of the gingiva, that is, it increases the risk of developing PD.

IL-6 initiates the production of pro-inflammatory proteins, which leads to the destruction of the collagen contained in the connective tissue of the gum and the destruction of this tissue occurs and loss of attachment appears as clinical changes in the patient. On the other hand, IL-1, when activated, induces fibroblasts to increase the production of collagenase, causing destruction of periodontal connective tissue and stimulates osteoblasts to generate chemical signals to osteoclasts to resorb periodontal bone structures [23].

Therefore, these mechanisms explain how these conditions can negatively affect each other, which points to a bidirectional adverse relationship.

Conclusions

The strength of the association between the two is framed in immune and metabolic parameters, because obesity can increase the host's susceptibility to periodontal disease, although it is believed that this association occurs in severe cases of this disease. The biological plausibility lies in the fact that adipose tissue produces adipokines, which could modulate the response in periodontal diseases.

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