Obesity and periodontal disease: why suggest such relationship? An overview

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Abstract
Obesity is a chronic condition that has social and economic implications for public health. It can be associated with periodontal disease since the metabolic alterations observed in that condition could have some influence in immunity. The elevated levels of lipid and glucose can be associated with periodontal disease, contributing to an exacerbated host inflammatory response, alterations in neutrophil function, and with the inhibition of macrophage growth factors, reducing tissue-healing capacity. In this way, obese individuals could have higher chances of undergoing tissue destruction in the presence of periodontal infection. On the other hand, periodontitis may be involved in alterations of lipid metabolism, since gram-negative bacteria could promote a rise in cholesterol and triglyceride levels due to chronic exposure to low levels of LPS in circulating blood, leading to the production of cytokines, which could initiate the production of lipoproteins by the liver. The objective was to review the literature about obesity and periodontal disease and provide a better understanding of their relationship.

Key words:
obesity, periodontitis, insulin resistance, hyperlipidemia

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Introduction

Obesity is a chronic condition, which can be associated with periodontal disease, since metabolic alterations that are present in this condition could have some influence on the host immunity. Elevations in the levels of lipids and glucose can be associated with periodontal disease and may contribute to an exacerbated inflammatory host response, alterations on the neutrophil functions, and the inhibition of growth factors by macrophages, reducing the healing capacity of the tissues. On the other hand, chronic exposure to bacteria LPS promotes the production of cytokines, which contribute to the alteration of the lipid metabolism.

Literature Review

Obesity

Obesity is a chronic metabolic condition that has public health implications because it is a risk factor for many diseases, such as diabetes, hyperlipidemia, hypertension, atherosclerosis, cardiovascular disease, among others1-2. Three metabolic alterations are responsible for the obesity characteristics:

1. Hiperinsulinemia: there is increased production of insulin by beta cells to compensate the resistance in the tissues, especially the adipose, muscular and hepatic ones.
2. Hyperglycemia: since there is a resistance to insulin, the circulating glucose is not taken by the cells and there is an increase of the blood glucose levels.
3. Hyperlipidemia: there is an elevation of the serum levels of cholesterol and triglyceride due to the lipid metabolism alteration – there is an increase in the liver lipogenesis and lipolysis in the adipocytes.

These alterations seen in obesity are also found in localized chronic or generalized acute infections. Cytokines, especially TNF-a, produced by the adipose tissue, are the ones responsible for those alterations. It is known that the adipose tissue stores energy as triglycerides and has a secretory capacity of the tissues. On the other hand, chronic exposure to bacteria LPS promotes the production of cytokines, which contribute to the alteration of the lipid metabolism.

Insulin Resistance

Insulin resistance is a metabolic condition in which the beta cells of the pancreas are unable to produce insulin in a sufficient amount to maintain normal blood glucose levels. This condition is often associated with obesity and can lead to hyperglycemia, hyperinsulinemia, and other metabolic abnormalities. Insulin resistance is a risk factor for the development of type 2 diabetes, cardiovascular disease, and other chronic conditions.

Relationship between hyperlipidemia and periodontal disease

The relationship between periodontal disease and hyperlipidemia has already been studied by other authors5,6,10-12, who verified that patients with periodontal disease could present elevated levels of triglycerides and cholesterol. This is because the systemic involvement, since the periodontal infection is a chronic one. Moreover, chronic exposure to bacteria LPS promotes the recruitment of defense cells, specifically macrophages that secrete TNF-a and IL-1b, increasing lipogenesis and lipolysis and leading to a state of hyperlipidemia5.

The increased lipid levels promote alterations, for example, phagocytosis and chemotaxis alterations of the defense cells (polimorphonuclear cells and macrophages). These cells release a greater amount of growth factors by the macrophages, which impair tissue healing5.

Why suggest such a relationship?

New evidences5,9,13,14 have been showing that insulin resistance is a link between obesity and periodontal disease due to the TNF-a produced in both conditions. TNF-a produced by the adipose tissue helps exacerbate the periodontal disease and the one produced by periodontal disease helps perpetuate the insulin resistance seen in obesity.

Periodontal disease may also unbalance lipid metabolism worsening the hiperlipidemic state in the obese patients5,6,10-12. In a case-control study by Noack et al.15 the patients with hyperlipidemia had higher periodontal inflammation than the control patients, with a higher percentage of sites and sextants with probing depth (PD) ³ 3.5mm. A positive relationship between the obesity indicators, body mass index (BMI) and waist-hip ratio (WHR), and periodontal disease was found in some studies14,16-18. Furthermore, a significant correlation between BMI and the plasmatic concentrations of TNF-a5, which may suggest that obesity may lead to an exacerbation of the periodontal disease because of the higher load of circulating cytokines. Nevertheless, the BMI is not a good obesity indicator because it is based on the total fat of the patient. Waist-hip ratio is a more reliable indicator since it measures the waist circumference, which shows a close correlation with the amount of visceral adipose tissue9. Visceral adipose tissue was shown to be metabolically active and to secrete a great amount of cytokines and hormones, this way being responsible for some metabolic alterations9, such as insulin resistance.
resistance and lipid profile alterations. In addition, in the hip area, there is the greatest amount of muscle in the body, and its mass and function are closely related to the systemic sensitivity to insulin\textsuperscript{20}. This way, many studies are using the WHR as an obesity indicator and have found a stronger positive relationship with periodontal disease than the BMI\textsuperscript{11,14,16-17}.

Final considerations
Obesity is characterized by a chronic inflammatory state, which can worsen the preexisting periodontal disease. Periodontal disease has shown to induce metabolic alterations in the lipid metabolism contributing to the hyperlipidemic state of obesity. Further studies are necessary to elucidate the real relationship between obesity and periodontal disease. Molecular biology studies are necessary to better understand the mechanism and biological foundation of the association between obesity, periodontal disease and insulin resistance. Longitudinal studies are also necessary to show a causal relationship. However, before any progress in the understanding of this relationship, periodontists should counsel obese patients in relation to the possible oral complications of obesity, to diminish morbidity for these individuals. This counseling should include the measurement of BMI and WHR for periodontal risk evaluation on a regular basis\textsuperscript{21}.

References