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

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
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. Hyperinsulinemia: 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 seric levels of cholesterol and triglyceride due to the lipid metabolism alterations on the neutrophil functions, and the inhibition of growth factors by macrophages, reducing the healing capacity of the tissues. The other hand, chronic exposure to bacteria LPS promotes the assimilation of the circulating glucose, which contribute to the alteration of the lipid metabolism.

Insulin Resistance

Insulin resistance is found in obesity and in infections, as the result of an increased secretion of cytokines by the adipose tissue and the macrophages, which are hyperactive because of the hyperlipidemia in obesity5,6. TNF-a interferes in insulin signaling and blocks the translocation of the glucose transporter (GLUT-4) to the cell membrane, as well the assimilation of the circulating glucose by the cell; as a result, a state of hyperglycemia can rise. Insulin resistance also interferes with lipid metabolism because the adipocytes resistant to insulin cannot assimilate the circulating fatty acids. Reaching the liver, these fatty acids will be broken down in triglycerides and cholesterol8, which will worsen the insulin resistance state.

Insulin resistance can be the link between periodontal disease, obesity and diabetes because in these three situations there is the production of chronic regulated cytokines. This chronic regulation contributes to the insulin resistance state, leading to metabolic alterations9.

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 hyperlipidemia9.

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 healing9.

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 hyperlipidemic 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) ≥3.5mm. 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-a14, 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 alterations19, such as insulin
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