e - ISSN - 2348 - 2168
Print ISSN - 2348 - 215X

Acta Biomedica Scientia

Journal homepage: www.mcmed.us/journal/abs

OBESITY AND PERIODONTAL DISEASE

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Article Info

Received 29/09/2015
Revised 16/10/2015
Accepted 19/10/2015

Keywords :-
Obesity, Periodontitis, Inflammatory disease.

ABSTRACT

Obesity is a chronic disease of mutifactorial origin, where there is increase in body fat. The global obesity epidemic has been described by the World Health Organization as one of the most blatantly visible but yet most neglected, public health problems that threatens to overwhelm both more and less developed countries. Periodontitis is an inflammatory disease of tooth supporting tissues resulting in destruction of periodontal ligament and alveolar bone. Periodontitis and obesity are both chronic health problems and the literature supports this association. A hyperinflammatory state observed in obesity is proposed as a mechanism to explain this association. This low grade inflammation in obese subjects triggers the worsening of non transmissible chronic diseases like periodontitis.

INTRODUCTION

The prevalence of obesity is increasing worldwide. According to WHO the global obesity epidemic continues to increase [1].

This disease is so common in the world's population that is becoming more important contributors to health. This is a complex and multifactorial disease resulting from excessive fat storage, resulting from the interaction of social, behavioral, cultural, psychological, metabolic and genetic factors. Obesity causes or exacerbates many health problems, both independently and in association with other disorders (comorbidity). These diseases can be the type 2 diabetes, hypertension, hypoventilation, sleep apnea, venous stasis, cancer, regenerative joint disease, and others [2-3].

Periodontitis is a chronic inflammatory joint hearing of the adult population is characterized by an inflammatory response gum against a pathogenic bacterial microflora, resulting in alveolar bone loss and eventually tooth loss. Studies of risk assessment identified the inclusion of age, male gender, smoking and diabetes mellitus as some features object-level associated with periodontal disease severity and progression [4].

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DISCUSSION

Overweight and obesity have been suggested to be associated with periodontitis, because obesity can have some effects on systemic health by changing the host susceptibility to periodontitis due to inflammatory mediators. The link between periodontal disease and obesity may have important implications for public health because both diseases are important risk factor for cardiovascular disease.

However, this association is not entirely clear in the literature, because there are controversies about finding studies [5].

A recent meta-analysis concluded that an increase in body mass index (BMI), waist circumference (WC), and other indicators of obesity were significantly associated with a higher prevalence of periodontal disease.

Research continues to support the role of overweight and obesity as an independent risk factor for the development of an aggravation of many chronic diseases. Biological mechanisms linking obesity with other inflammatory diseases, such as coronary heart disease, metabolic syndrome, diabetes mellitus, and osteoarthritis, can lead to oxidative stress, adipokines, and other related hormones. Proinflammatory cytokines produced in response to periodontal disease such as IL-1β, and interferon-γ, as well as Gram-negative lipopolysaccharide,

can interfere with lipid metabolism and further contribute to obesity and obesity-related co-morbidity [6].

Oxidative stress is involved in many pathologic conditions like obesity, diabetes and cardio vascular disease. In obesity, the organism cannot adapt and maintain homeostasis under continuous energy and nutrient exposure, and the consequent emergence of metabolic and oxidative stress leads to inflammation responses and cell organelle dysfunction. Adipocytokines are also responsible for production of reactive oxygen and nitrogen by macrophages and monocytes leading to increased oxidative stress. Angiotensin II secreted by adipocytes stimulate nicotinamide adenine dinucleotide phosphate (NADPH) oxidase activity. NADPH oxidase is responsible for ROS production in adipocyte [7].

Obesity increases the mechanical load and myocardial metabolism, therefore oxygen consumption is increased. Increased oxygen consumption leads to increased ROS production derived from increase in mitochondrial respiration. The effect of high triglyceride (TG) affects functioning of the mitochondrial respiratory chain, inhibits translocation of adenine nucleotides and promotes the generation of superoxide.

Certainly it has been well established that inflammation is a key component in the development of atherosclerosis and observational studies have shown that periodontitis is associated with a moderate but significantly increased risk of coronary heart disease. Interventional studies that have examined the effects of antibiotic treatment on cardiovascular risk have generally failed to show any beneficial effect; However, these studies were mostly of short duration (less than one year of treatment) and have studied the effects of a single secondary prevention. Inflammatory diseases such as periodontitis induce the production of proinflammatory cytokines such as tumor necrosis factor-α, interleukin-1 and interleukin-6. It has been suggested that the secretion of TNF-α from adipose tissue triggered by lipopolysaccharides of Gram-negative periodontal promotes hepatic dyslipidemia and decreases insulin [8-9].

Obesity seems to affect bone metabolism through multiple mechanisms. First, osteoblasts and adipocytes derive from a common mesenchymal cell in the bone marrow. Obesity can induce adipogenesis while decreasing osteoblastogenesis. Another mechanism by which obesity can affect bone metabolism is through receptor activator of NF-κB (RANK), RANK ligand (RANK-L), and osteoprotegerin (OPG), by which osteoblasts are capable of regulating the differentiation and activity of osteoclasts. The overproduction of proinflammatory cytokines such as IL-1, IL-6, and TNF-α by adipocytes are capable of stimulating the activity of osteoclasts by regulating RANK/RANKL/OPG. Adipocytes may also directly regulate hematopoietic progenitor cells of osteoclasts [10].

The direct or indirect production of leptin and adiponectin related to obesity may influence bone metabolism. Circulating leptin levels are high in cases of obesity. The action of leptin on bone seems to be variable; however, it seems to negatively affect the bone metabolism of animals subjected to high-fat-diet-induced obesity [11]. With regard to adiponectin, it has been observed that cytokine secreted by adipocytes has an anti-inflammatory action which is associated with the reduction of osteoclastogenesis, bone resorption, and stimulation of osteoblastogenesis. However, the plasma concentration of this cytokine is decreased in obese compared with non-obese individuals. Finally, the calcium absorption seems to be diminished in the presence of high-fat diets [12-13].

The type 2 diabetes and decreased insulin sensitivity are associated with the production of glycation end products (AGE), which activate the production of inflammatory cytokines, thus preparing in inflammatory diseases like periodontitis. These observations suggest a potential interaction between obesity, periodontitis and the incidence of chronic diseases, although current studies are insufficient to determine whether these associations are causal. So in addition to being a risk factor for type 2 diabetes and coronary heart disease, obesity-related inflammation can also promote periodontitis. In contrast, periodontitis, once there, can promote systemic inflammation and thus increase the risk of coronary heart disease [14-15].

CONCLUSION

Obesity is a complex disease and multifactorial. His relationship with periodontal disease and other chronic diseases is well documented, but the underlying mechanism is under investigation. It 'very difficult to say if obesity predisposes an individual to periodontal disease or periodontal disease affects lipid metabolism, or both.

There is a link between obesity and periodontal disease, but the risk factors that aggravate these diseases should be clarified to clarify the meaning of this association. However, oral health measures should be implemented in obese patients. Working with paired samples and avoid confounding factors may contribute to the homogeneity of the studies. These tips can improve the scientific evidence that can respond to these concerns.

In conclusion, normal weight and overweight, non-diabetic, white males with rapid rates of weight gain toilet, and AFA had more periodontal disease and progression of periodontitis than those who had smaller gains. Further longitudinal studies are needed to confirm these results and to extend them to the most diverse and current populations. Further evaluation of longitudinal changes in weight and adiposity with developing periodontitis also clarify the potential biological mechanisms.

ACKNOWLEDGEMENT: None

CONFLICT OF INTEREST:
The authors declare that they have no conflict of interest.

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