Obesity and periodontal disease: A review

Mohammad Shoyab Khan¹, Mohammed Alasqah¹, Lamia M. Alammar², Yousef Alkhaibari¹

¹Department of Preventive Dental Sciences, College of Dentistry, Prince Sattam Bin Abdulaziz University, Al-Kharj, ²Resident, King Fahad Medical City, Riyadh, Saudi Arabia

ABSTRACT

Periodontal diseases usually refer to inflammatory disorders that are caused by pathogenic bacteria in the subgingival biofilm in association with impaired host immune response and connective tissue breakdown. The bacterial challenge exacerbates the cytokine production by the gingival epithelium, resulting in an uncontrolled inflammation that leads to tooth loss in adults from different populations. The prevalence of these diseases increases with aging, longer retention of teeth, and increased incidence of obesity and diabetes among the population. The prevalence demonstrates an increasing trend and a correlation with numerous comorbidities. Hence, as a family physician one should have the in-depth knowledge regarding the relationship between obesity and periodontitis to create awareness among people to provide primary care. Thus, it is relevant to develop new methods capable of detecting these diseases in the early stages and following up on their progression.

Keywords: Obesity, overweight, body mass index, periodontal disease, association, systemic conditions

Introduction

Periodontal diseases are inflammatory diseases affecting the surrounding and supporting tissues of teeth, the periodontium. Gingivitis and destructive periodontal disease (periodontitis) are the two most common forms of periodontal diseases.¹ Gingivitis is an inflammatory reaction that often induces the pathogens residing in dental plaque (biofilm), which forms on the adjacent tooth surfaces. Destructive periodontal disease results in an apical loss of epithelial attachment along with the periodontal soft and hard tissues.² Unlike gingivitis, which is cured following the removal of local etiological factors, destructive periodontal disease is irreversible. Destructive periodontal disease is mediated by various intrinsic and acquired factors; two individuals with similar microbiological profile could show different susceptibility to periodontal diseases.³ Several case-control and cohort studies have reported the contribution of systemic conditions and diseases in the onset and exacerbation of destruction periodontal disease. Preterm birth, cardiovascular diseases, and diabetes are examples of this conditions.⁴⁻⁶

In addition, a growing body of evidence during the last decade suggests obesity as a risk factor for destructive periodontal disease.⁷ Although the majority of studies on destructive periodontal disease in individuals with obesity or metabolic syndrome concentrated on adults, some studies reported on evidence proposing that this potential link in children and adolescence exists.⁸ In this paper, we review the evidence suggesting that destructive periodontal disease is linked to obesity and metabolic syndrome, as an example of inter-organ crosstalk under inflammatory conditions.

Obesity is a multifactorial condition with a wide range of etiological factors including genetic, biological, social, and behavioral factors, all of which likely interact to ultimately lead to a chronic imbalance between energy intake and energy expenditure. This imbalance could cause excessive fat accumulation and...
result in adverse health consequences. Obesity has reached epidemic proportions worldwide, largely because of increased consumption of a high caloric diet and a sedentary lifestyle. According to the World Health Organization, approximately 2.3 billion adults will be overweight and more than 700 million will be obese by 2015. This phenomenon affects particularly in developed countries. Over the past two decades, the overall obesity rates have reached 24.1% and 34.4% in Canada and the United States, respectively. Based on current Health Canada guidelines, a body mass index (BMI, kg/m²) of 25–30 and over 30 are considered overweight and obese, respectively. Obesity is categorized into three classes according to the increased health risks associated with increasing BMI levels: class I (BMI 30–34.9), class II (BMI 35–39.9), and class III (BMI ≥40).

Pediatric obesity has also become a public health concern since it is more common for children to experience the negative health consequences of obesity, which used to be only seen in adulthood. In 2010, more than 40 million children under the age of 5 were estimated to be overweight worldwide. In 2004, 26% of Canadian children and adolescents aged 2–17 years were overweight or obese. Obesity has been associated with a wide spectrum of comorbidities, such as coronary artery disease, strokes, diabetes, arthritis, reproductive dysfunctions, and various cancers.

**Role of Leptin, Adiponectin, Tumor Necrosis Factor-alpha**

For many years, adipose tissue was considered as an inert organ that stored triglycerides. It is now clear that adipose tissue is a complex and metabolically active endocrine organ that secretes numerous immune-modulatory factors and plays a major role in regulating metabolic and vascular biology. Adipose cells, which include adipocytes, pre-adipocytes, and macrophages, secrete more than 50 bioactive molecules, known collectively as adipokines. Some of these adipokines act locally, whereas others are released into the systemic circulation where they act as signaling molecules to the liver, muscle, and endothelium. Adipokines play a number of different roles, such as hormone-like proteins (e.g., leptin and adiponectin), classical cytokines (e.g., tumor necrosis factor-alpha, interleukin-6), proteins involved in vascular hemostasis (e.g., plasminogen activator inhibitor-1, tissue factor), regulators of blood pressure (angiotensinogen), promoters of angiogenesis (e.g., vascular endothelial growth factor), and acute-phase respondents (e.g., C-reactive peptide).

**Leptin** is secreted almost exclusively by adipocytes. Leptin signals through the central nervous system and peripheral pathways to suppress appetite and increase energy expenditure. Leptin mimics some of the actions of insulin by increasing glucose uptake in muscle and adipose tissue and by lowering hepatic glucose production. Most obese individuals have elevated leptin levels that do not suppress appetite. Many consider this leptin resistance to be one of the features contributing to obesity’s pathology. In obese patients with leptin resistance, leptin may elevate blood pressure and contribute to atherosclerosis and cardiovascular diseases.

**Adiponectin** is produced primarily by adipocytes but surprisingly decreased in obese patients, especially those with abdominal obesity. Clinical studies demonstrate inverse associations between adiponectin and serum markers of inflammation.

**Tumor necrosis factor-alpha**

Obesity-associated tumor necrosis alpha is primarily secreted from macrophages accumulated in abdominal adipose tissue. Although studies have not shown completely consistent results, it is thought that increased circulating tumor necrosis factor-alpha from adipose tissue contributes to poor health outcomes by increasing insulin resistance and by inducing C reactive peptide production and general systemic inflammation.

**Adipokines and Obesity**

Obesity is associated with an increased risk for developing characteristic features of metabolic syndrome, including hypertension, type 2 diabetes, and dyslipidemia. Interestingly, chronic exposure to periodontal pathogens endotoxin and increased cytokine production have been proposed to enhance the risk of causing type 2 diabetes and cardiovascular complications. Obesity has also recently been reported to be associated with periodontitis. Obesity induces macrophage accumulation in adipose tissue, promotes chronic low-grade inflammation, and increases adipokines derived from adipocytes. In this review, we summarize recent advances in understanding the roles of adipokines in chronic inflammatory states such as periodontitis and focus primarily on adiponectin, leptin, and resistin. Understanding the role of adipokines may help elucidate relationships among periodontitis, obesity, type 2 diabetes and cardiovascular diseases.

**Relationship of Obesity and Telomeres**

Obesity is a chronic disease characterized by a high inflammatory burden which has been associated with shorter telomeres. A case-control study with 793 French children and young people aged 2-17 years old demonstrated that obese individuals presented telomeres 24% shorter than non-obese controls. A great concern about this result is that, interestingly, another study found that former obese individuals present shorter telomeres in adipose tissue cells when compared to control individuals, which means that alteration in telomere length may be an irreversible phenomenon. However, the relationship between adipocytes and leukocyte telomere length still needs further investigations.

**Polyphenols and periodontal disease**

Inflammatory stimulation by periodontal pathogens increases the production of crevicular fluid and induces the chemotaxis of polymorphonuclear leukocytes, which, in order to inactivate periodontal pathogens, release singlet oxygen and hypochlorous acid into the crevicular fluid. The consequent oxidative stress is
countered by the antioxidant activity of ascorbate, albumin and urate present in the crevicular fluid and derived from plasma. However, this local oxidative stress may be increased by external factors or systemic conditions, such as smoking, diabetes, obesity and metabolic syndrome. When there is a disequilibrium between oxidative stress and antioxidant activity, periodontal tissue destruction may appear. These observations suggest that antioxidant-rich diets might inhibit periodontal disease development and progression, particularly in subjects exposed to environmental and dietary sources of oxidative stress.[6,9,10] Several studies also report that decreased antioxidant activities of crevicular fluid and saliva are associated with the development of periodontitis.[11,12] Polyphenols may contribute to increasing the antioxidant activity of oral fluids. Indeed, delivery of tea Polyphenols by holding green or black tea in the mouth for 2–5 min increases the antioxidant capacity of saliva, and daily consumption of two fresh grapefruits for 2 weeks increases the phagocytic capacity of the polymorphonuclear leucocytes in the gingival crevicular fluid.[13] The finding of an association between obesity and periodontitis adds to the small number of relatively recent studies that found a similar relationship.[14,15,16] Several studies found that the positive association between body mass and periodontitis was most pronounced among women.[17,18,19] Despite all the evidence about obesity and periodontitis, recommendations on treatment planning, primary health care professionals need to be aware of the complexities of obesity to educate their patients about the importance of maintaining healthy body weight and good oral hygiene.

**Conclusion**

Periodontal diseases, chronic inflammation by its nature, have been linked to many systemic conditions. Despite all these pieces of evidence indicating the higher risk of destructive periodontal disease in obesity and metabolic syndrome, the underlying biological mechanism(s) is yet to be fully understood. Studying the common etiological factors in obesity, metabolic syndrome, and destructive periodontal disease would be a potential approach to delineate biological mechanisms explaining the higher risk of destructive periodontal disease under these conditions. Inflammation is indeed one of the common factors in the pathogenesis of the destructive periodontal disease, obesity, and metabolic syndrome. Knowing this fact helps family physicians to provide primary care for the people.

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**Conflicts of interest**

There are no conflicts of interest.

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