Review Article

Obesity, periodontal and general health: Relationship and management

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ABSTRACT

Obesity is a multifaceted subject. It has increased at an alarming rate in recent years. Being overweight increases the likelihood of a patient having associated health and social problems which may affect dental services and dental management. A review of the literature on obesity and periodontal disease suggested that they both confound each other and obesity itself has been recognized as a major risk factor for periodontal disease. It has been found that adverse effects of obesity on the periodontium may be mediated through pro-inflammatory cytokines and various other bioactive substances. This article tries to focus on the possible role of obesity and obesity-related diseases like diabetes and coronary heart diseases (CHD), as a potential contributor to periodontal disease and vice versa. The meanings of these associations can be useful for various diagnostic and treatment planning purposes.

Key words: Adipokine, body mass index, obesity, periodontitis

Introduction

The global obesity epidemic has been described by the World Health Organization as the most neglected public health problem that threatens to overwhelm both more and less developed countries.[1] The etiology of obesity represents a complex interaction of genetics, diet metabolism and physical activity levels. In addition to consumption of high-energy food, physical activity is a key factor in the energy balance equation.

Obesity is a multisystem condition and a major contributor to the development of hypertension, diabetes mellitus, arteriosclerosis, cardiovascular and cerebrovascular diseases.[2-5] Besides these risk factors, obesity has also been suggested to be a risk factor for periodontitis which is a disease of the supporting structures of the teeth resulting from the interaction between pathogenic bacteria and the host immune response.[6]

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Obesity—Definition and Assessment

The definition of obesity is based on body mass index (BMI, also called Quetelet Index), which is the ratio of body weight (in kg) to body height (in m) squared.[7] BMI is highly correlated with fat mass and morbidity and mortality and therefore sufficiently reflects obesity-related disease risk in a wide range of populations; however, there are some limitations. For example, for the same BMI, older persons tend to have a higher body fat composition; and therefore, risk assessment by BMI is less accurate in older people (over 65 years of age). Body fat distribution is assessed by the measurement of waist circumference, with 102 cm in men and 88 cm in women, respectively, being the cutoff point for abdominal obesity associated with an increased risk of morbidity.[7] Waist circumference shows a close correlation with the amount of visceral adipose tissue, and visceral adipose tissue has been shown to be metabolically more active and to secrete far greater amounts of cytokines and hormones compared with subcutaneous adipose tissue.[8] Recently, numerous studies have indicated

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that measurement of the waist circumference or waist-hip ratio may be a better disease risk predictor than BMI, and there is still intensive research ongoing as to whether BMI, waist circumference or both should be used to assess disease risk.[9,10]

Periodontitis as a Disease Entity

Periodontal disease refers to the processes of destruction of the peri-tooth structures that support the teeth. These are composed of the gingiva, the periodontal ligament, the cementum, and the alveolar bone. The chronic destruction of these supporting tissues leads to the eventual loss of teeth and hence partial or complete loss of teeth. Epidemiological studies reveal that more than two-thirds of the world's population suffers from one of the chronic forms of periodontal disease. Recent recognition of the importance of periodontal disease and its impact on the perpetuation and management of systemic diseases calls for a global effort to control periodontal disease. Two forms of periodontitis have been proposed: One is chronic periodontitis (previously termed "adult periodontitis"), which affects primarily the adult population who are > 35 years of age. This type of periodontitis is frequently associated with an elevated number and frequency of *Porphyromonas (P.) gingivalis*, *Treponema (T.) denticola*, and *Tanerella (T.) forsythia* detected in the subgingival microbial community.[11] Contributing local factors consisting of conspicuous dental plaque, calculus, root surface accretions, and overhanging restorations are closely associated quantitatively or qualitatively with disease expression. The other form, aggressive periodontitis (previously referred to as "early-onset periodontitis"), is associated with young adults (< 35 years of age) and is characterized by rapid destruction with minimal signs of gingival inflammation. *Actinobacillus (A.) actinomycetemcomitans* is the key pathogen for aggressive periodontitis.[12]

Prevalence of Obesity and Periodontitis

Between 1960–80, the prevalence of overweight and obesity among adults and of overweight among children was relatively constant. About 13% of adults were obese and 5% of children were overweight.[13] However, according to the third National Health and National Examination Survey (NHANES), between 1988–91, the prevalence of obesity among adults has doubled, and the prevalence of overweight among children and adolescents has tripled. In the year 2004, approximately 34.1% of the US population was overweight and about 32.2% obese.[7,14] The prevalence of overweight in an urban population of India, as found in the National Family Health Survey during 2005–6 was 11.38% and prevalence of obesity was 2.24%.[15]

The prevalence of periodontal disease is 76% higher among young obese individuals aged 18–34 years than in normal weight individuals[16] and overweight is associated with an increased risk of periodontitis among those aged 17–21 years.[17]

Interrelationship of Obesity, Periodontitis and Chronic Inflammation

It has been now well established that inflammation is an essential component in the development of atherosclerosis, and observational studies showed that periodontitis is associated with a moderately, but significantly, higher risk of coronary heart disease.[18-20] Interventional studies that examined the effects of antibiotic treatment on cardiovascular risk have generally failed to show any beneficial effect; however, these studies have mostly been of short duration (less than one year of treatment) and have investigated the effects on secondary prevention only. Inflammatory diseases like periodontitis induce the production of proinflammatory cytokines such as Tumor Necrosis Factor-α, Interleukin-1 and Interleukin-6.[18] It has been suggested that the secretion of TNF-α by adipose tissue triggered by lipopolysaccharides from periodontal Gram-negative bacteria promotes hepatic dyslipidemia and decreases insulin.

Type 2 diabetes and decreased insulin sensitivity are associated with the production of advanced glycation end products (AGE), which trigger inflammatory cytokine production, thus predisposing to inflammatory diseases such as periodontitis. These observations suggest a potential interaction among obesity, periodontitis and chronic disease incidence, although present studies are insufficient to conclude whether such associations are causal. Thus in addition to being a risk factor for Type 2 diabetes and coronary heart disease, obesity-related inflammation may also promote periodontitis. Conversely, periodontitis, once it exists, may promote systemic inflammation and thereby increase the risk of coronary heart disease. This multi-directional association has been shown in Figure 1.[2]

Mechanism Linking Obesity with Periodontal Disease

The mechanism of how obesity affects the periodontium is currently poorly understood but what is known is that obesity has several harmful biological effects that might be related to the pathogenesis of periodontitis.

The adverse effect of obesity on the periodontium may

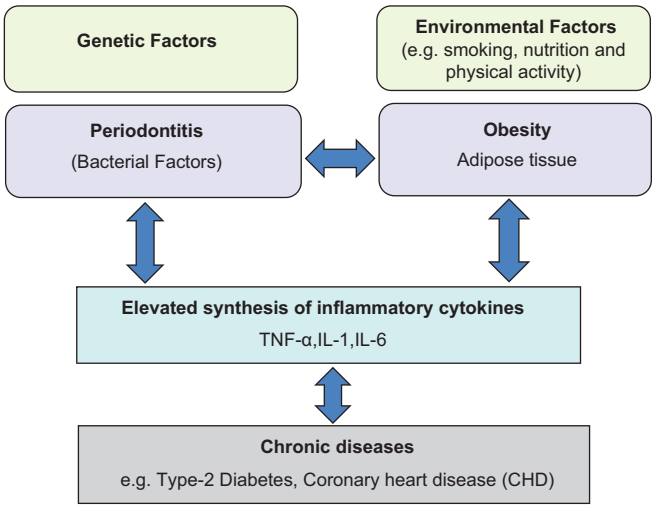

Figure 1: Model linking Periodontitis, Obesity and Obesity-related chronic diseases

be mediated through pro-inflammatory cytokines like interleukins (IL-1, IL-6 and TNF-α), adipokines (leptin, adiponectin, resistin and plasminogen activator inhibitors-1) and several other bioactive substances like reactive oxygen species (ROS), which may affect the periodontal tissues directly.[6]

Adipose Tissue-Derived Cytokines and Hormones

Interleukin-6: Interleukin-6 is secreted by the human adipose tissue and is produced in greater amounts by deep abdominal fat than by subcutaneous fat. Elevated levels of interleukin-6 have been found to be associated with increased risk of cardiovascular events, lipolysis and weight gain.[13]

TNF-a: Obesity-associated tumor necrosis factor-α (TNF-α) is primarily secreted from macrophages accumulated in the abdominal adipose tissue. Increased circulating TNF-α from the adipose tissue contributes to poor health outcomes by increasing insulin resistance, C-reactive peptide production and general systemic inflammation. TNF-α is a potent inhibitor of adiponectin, an important anti-inflammatory adipokine.[13]

Adiponectin: Adiponectin is a circulating hormone secreted by the adipose tissue. It is involved in glucose and lipid metabolism and accounts for about 0.05% of the total serum proteins. Contrary to other adipose-derived hormones, adiponectin levels are reduced in persons with obesity, insulin resistance or Type 2 diabetes.

Leptin: Leptin is the best known substance secreted from the adipose tissue. It plays an important role in regulating energy intake and energy expenditure, including appetite and metabolism.

It is similar in some action to insulin. It has been reported that leptin is present within healthy and marginally inflamed gingiva and decreases in concentration as the adjacent probing depth increases. Thus, leptin may play an important role in the development of periodontitis.[21]

Resistin: Resistin belongs to a family of resistin-like molecules (RELM) and has been reported to be secreted by adipocytes and to cause insulin resistance in animal models. Current evidence suggests that in humans, resistin is more closely related to inflammatory processes than to insulin resistance. Whether or not resistin plays a role in inflammatory periodontal disease remains to be defined.[2]

Plasminogen activator inhibitors-1 (PAI-1): PAI-1 is an adipokine which generates agglutination of blood and raises the risk of ischemic vascular disease and gingival inflammation. PAI-1 may decrease blood flow in the periodontium of obese patients and promotes development of periodontitis.[22,23]

Host Immunity

Obesity increases hosts susceptibility by modulating the host immune and inflammatory system, leaving the patient with a greater risk of periodontitis. Obesity affects host immunity. It impairs the cell-mediated immune response and decreases lymphocyte immune function and natural killer T-cell activity as seen in the rat model.[4]

Reactive Oxygen Species

It is also believed that there is a close association of obesity and periodontitis with chronic inflammation. Reactive oxygen species are products of normal cellular metabolism but over-production of reactive oxygen species induces damage by oxidizing DNA, lipids and proteins. Obesity increases the circulation of reactive oxygen species which in turn causes gingival oxidative damage and progression of periodontitis.[24,25]

Periodontal Pathogens' Contribution to Obesity

Goodson *et al*., suggested three mechanisms by which oral bacteria may contribute to development of obesity. First, the oral bacteria may contribute to increased metabolic efficiency, as suggested by the infectobesity proponent. The second hypothesis is that oral bacteria could increase weight gain by increasing appetite. The third hypothesis is that oral bacteria redirect energy metabolism by facilitating insulin

resistance through increasing levels of TNF-α or reducing levels of adiponectin. By any of these mechanisms, even a small excess in calorie consumption with no change in diet or exercise could result in unacceptable weight gain.[26]

Evidences for Association Between Obesity and Periodontal Disease

The first report on the relationship between obesity and periodontal disease appeared in 1977 when Perlstein *et al.,* found alveolar bone resorption to be greater in obese animals compared to non-obese zucker rats.[27] Since then a large number of studies were carried out to get an overview of the association between obesity and periodontal disease. In 2005, CFD Vecchia *et al*., evaluated the relationship between overweight, obesity and periodontitis and found that obesity was significantly associated with periodontitis in adult and non-smoker women. However, overweight was not significantly associated with periodontitis.[28]

In their research work, Genco *et al*., concluded that obesity is associated with high plasma levels of TNF-α and its soluble receptors, which in turn may lead to a hyper-inflammatory state increasing the risk for periodontal disease.[29]A study on metabolic disorders related to obesity and periodontal disease concluded that the association among periodontal disease, diabetes and obesity confounds each other.[4] An epidemiologic study on obesity and periodontitis in 60-70-year-old men found a positive correlation between obesity and periodontal disease.[30]

Sarlati *et al*., conducted a study on the relationship between obesity and periodontal status in a sample of young Iranian adults and found that the overall and abdominal obesity were associated with the extent of periodontal disease and hence prevention and management of obesity may be an additional factor for improving periodontal health.[31]A study on a young Japanese adult population concluded that BMI could be a potential risk factor for periodontitis among healthy young individuals i.e. those with BMI of less than 30 kg/m^2.[32]

Khader *et al*., did a study on the association between periodontal disease and obesity among adults in Jordan and came to the conclusion that BMI, high waist circumference and high fat percentage were significantly associated with increased odds of having periodontitis.[3] In Copenhagen city, an inverse relation between obesity and clinical attachment level and a slight association between BMI and bleeding on probing was found.[33]

Boesing *et al*., (2009) conducted a study on the interface between obesity and periodontitis with emphasis on oxidative stress and inflammatory response and found obesity to participate in the multifactorial phenomenon of occurrence of periodontitis through the increased production of reactive oxygen species.[34]

Effect of Periodontal Treatment on Obesity and Other Related Diseases and Vice Versa

Tandon *et al*., found that patients of chronic generalized periodontitis who were offered periodontal therapy showed improvement in the various lipid parameters except high density lipoproteins-cholesterol, which was not significantly altered. Chronic periodontitis in otherwise healthy individuals may therefore, be contributing to the systemic inflammatory burden in these patients and adversely altering the lipid profile.[35]

A randomized controlled clinical trial by Singh *et al*., involving 45 Type 2 diabetic patients showed that periodontal therapy had a role to play in improved glycemic control, more so in those patients who were subjected to periodontal treatment and adjunctive doxycycline (100 mg daily for 14 days).[36]

Periodontal treatment resulted in significant decrease in bleeding on probing, pocket depth, and lowered serum inflammatory markers in patients with coronary heart disease (CHD) and no coronary heart disease (NCHD). This may result in decreased risk of CHD in treated patients.[37]

Some studies have indicated that maintaining a normal weight by regular physical activity is associated with lower periodontitis prevalence.[38-40] Individuals who pursued regular exercise have lower plasma levels of inflammatory markers, such as IL-6 and C-reactive protein (CRP), and show an increased insulin sensitivity that may beneficially affect periodontal health.[38,39] A study that analyzed the NHANES-III study population demonstrated that individuals who maintained a normal weight, pursued regular exercise, and consumed a diet in conformity with the Dietary Guidelines for Americans and the Food Guide Pyramid recommendations were 40% less likely to have periodontitis.[39]

Role of the Physician and Dentist in Managing Obesity and Periodontal Disease

Role of the dentist

Dentists may wonder what their role should be in the management of obesity and obesity-related diseases like

diabetes and atherosclerosis. The diagnosis of such patients is in the realm of physicians. But a dentist can evaluate patients for signs and symptoms of obesity-related diseases. Mathus-Vliegen *et al*., documented that obesity is related to several aspects of oral health, such as dental caries, periodontitis and xerostomia.[41] Dentists should have ample knowledge of the signs, symptoms and diagnostic tests for metabolic syndrome and related diseases. The most common oral manifestations of diabetes are burning mouth syndrome, candidiasis, dental caries, and glossodynia, and periodontal manifestations includes multiple gingival and periodontal abscesses, inflammatory gingival enlargements, deep periodontal pockets, gingival polyps and mobile teeth.[42]

Dentists usually do not communicate with patients with diabetes mellitus (DM) and their physicians consistently. The results of a study of general dentists and periodontists showed that only 35% of periodontists and 14% of general-practice dentists consistently communicated with physicians concerning their patients with DM.[43] A good communication among dentists, physicians and patients is an area ripe for improvement.

Dentist should refer their overweight and obese periodontal patient for weight reduction interventions like diet therapy, behavioral therapy, pharmacotherapy and surgical procedures, so that they can have better control over periodontal inflammation. In the future, if obesity is to be acknowledged as a multiple-risk-factor syndrome for overall and oral health, general and oral risk assessment in the dental office should include the evaluation of BMI on a regular basis.

Role of physician

By simply asking patients when they last visited a dentist, physicians may start an important conversation and enhance interactions among patients, dentists and physicians. Physicians should be aware of the signs and symptoms of periodontal disease that includes gingival bleeding, reddish or bluish discoloration and puffiness of gingiva, halitosis, feeling of itching in gums, sensitivity to hot and cold, toothache in the absence of caries, and mobility, extrusion or migration of teeth. In such conditions the patient should be referred to a dentist or a periodontist.[44]

Physicians, nurses and diabetes educators may play an important role in educating patients with obesity, diabetes and coronary heart disease, about the importance of good oral hygiene and how poor oral hygiene adversely affects the general health. One of the most important things physicians can do to help their dental colleagues is to provide the results of laboratory tests such as HbA1c to dentists on request.

Conclusion

Obesity is a complex and multifactorial disease. Its relationship with periodontal disease and other chronic diseases is well documented but the underlying mechanism is under investigation. It is quite difficult to say whether obesity predisposes an individual to periodontal disease or periodontal disease affects lipid metabolism, or both. Further prospective studies are needed to address the question of causality and to determine if obesity is a true risk factor for periodontal disease, especially among the younger population. If this proves to be the case, periodontal disease prevention could be included in planned intervention campaigns designed to prevent obesity-related diseases. Conversely, the prevention and management of obesity may be an adjunctive approach to improving periodontal health.

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