Obesity and Periodontitis: An Inflammatory Relationship

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Obesity and co-morbidities: A Global Concern

Obesity has been a serious social problem since 1980.[1] According to recent WHO reports, the number of obese people in the world has doubled. In 2008, about 35% of adults over the age of 20 years were overweight and 11% were obese. As much as 65% of the world's population live in countries where overweight and obesity cause more fatalities than underweight. In addition, the problem increasingly affects children. In 2011, over 40 million children under 5 years of age were overweight. Over 30 million overweight children live in developing countries and 10 million live in developed countries. Moreover, overweight and obesity are the fifth-leading cause of death worldwide. At least 2.8 million people die because of excessive weight and obesity every year.[2] According to WHO, obesity and overweight are defined as excessive accumulation of adipose tissue, resulting in unfavourable health effects.[2] Obesity is a risk factor for hypertension, hypercholesterolemia, type 2 diabetes, periodontal diseases, heart problems, myocardial infarction and some types of cancer.[3] This disease is so common among the world population that it is becoming the most important contributors to ill health. This is a complex and multi-factorial disease arising from excessive storage of fat, resulting from the interaction of social, behavioral, cultural, psychological, metabolic and genetic factors.[4]

Overweight and obesity have long been recognized as important determinants of elevated blood pressure levels. Mechanisms that have been implicated in the development of obesity-related hypertension include increased sympathetic nerve activity, sodium and volume retention, renal abnormalities, insulin resistance, hyperleptinemia and increased secretion of angiotensinogen from adipocytes.[5,6]

Obese persons have a more than 10-fold increased risk of developing type 2 diabetes compared with normal-weight persons. Type 2 diabetes develops due to an interaction between insulin resistance and beta cell failure. Several factors, including lipotoxicity and glucose toxicity, as well as obesity-derived cytokines, have been implicated in these processes.[7,8]

Obesity is also associated with an about 2-fold higher risk of heart failure and a 50% increased risk of atrial fibrillation.[9,10] A clinical presentation of obesity associated periodontitis reported to department of Periodontology, Rural Dental College of Pravara Institute of Medical Sciences, Loni, Maharashtra, India is illustrated in Figure1.

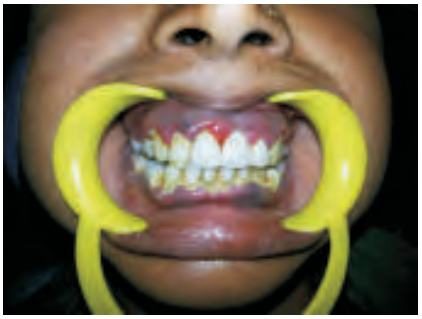

Figure 1: Obesity associated Periodontitis

Obesity is associated with bone knee and hip arthritis, and with arthritis involving the carpometacarpal joints of the hand. Recent studies have proved that being overweight antedates the development of knee osteoarthritis and increases the risk of radiographic progression.[11]

Obesity is the major reversible risk factor for obstructive sleep apnea syndrome. The prevalence rises from 2% to 4% in the general population to a prevalence of at least

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40% in morbidly obese patients. Orofacial findings of this syndrome includes a retrognathic mandible, narrow palate, large neck circumference, long soft palate, tonsillar hypertrophy, nasal septal deviation and relative macroglossia.

The metabolic syndrome is a concept that encompasses metabolic abnormalities that co-occur to a greater degree than would be expected by chance alone, and which predisposes individuals at a high risk to develop cardiovascular disease. This approach is supported by a growing number of studies showing that the adipose tissue itself is capable of producing several hormones and proteins, which are involved in the development of obesity-related diseases. [11,12]

Periodontitis is a destructive inflammatory disease of the supporting tissues of the teeth and is caused either by specific microorganisms or by a group of specific microorganisms, resulting in progressive destruction of periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession, or both.[13] Bacteria are the prime etiological agents in periodontal disease, and it is estimated that more than 500 different bacterial species are capable of colonizing the adult mouth [14] and the lesions of the oral cavity have an immense impact on the quality of life of patient with complex advance diseases.[15] Periodontitis has been proposed as having an etiological or modulating role in cardiovascular and cerebrovascular disease, diabetes, respiratory disease and adverse pregnancy outcome and several mechanisms have been proposed to explain or support such theories.[16] The oral cavity appears as an open ecosystem, with a dynamic balance between the entrance of microorganisms, colonization modalities and host defenses aimed to their removal: To avoid elimination, bacteria need to adhere to either hard dental surfaces or epithelial surfaces. The oral biofilm formation and development, and the inside selection of specific microorganisms have been correlated with the most common oral pathologies, such as dental caries, periodontal disease and peri-implantitis.[17] The cooperative communal nature of a microbial community provides advantages to the participating microorganisms. These advantages include a broader habitat range for growth, an enhanced resistance to antimicrobial agents and host defence, and an enhanced ability to cause disease.[18] Microorganisms in dental plaque interact with neutrophils/monocytes resulting in acute-phase inflammatory response by the systemic dissemination of locally produced mediators, such as C-reactive protein (CRP), interleukins-1 beta (IL-1â), interleukins-6 (IL-6) and tumor necrosis factor alpha (TNF-á). Thus, the acute-phase response might be useful as biomarkers of periodontitis contribution to systemic disease, as well as providing a prospective mechanistic link between the local and systemic manifestations of periodontitis.

Relationship between obesity and periodontitis

The relationship between obesity and periodontitis was first reported by Perlstein& Bissada in 1977 when histopathologic changes were observed in the periodontium of obese rats subjected to ligature induced periodontitis.[19] In response to plaque accumulation, periodontal inflammation and alveolar bone resorption were found to be greater in the obese rats as compared to nonobese controls. In humans, an association between obesity and periodontitis was first reported in an epidemiological study conducted by Saito et al in Japan using 241 healthy individuals to which the community periodontal index of treatment needs (CPITN) was applied.[20] After adjusting for confounding variables, they found that the relative risk for periodontitis was 3.4 in persons with a BMI of 25-29.9 kg/m2 and 8.6 in those with a BMI e" 30 kg/m2.

An additional study by Saito et al utilizing 643 Japanese individuals who had at least one tooth per sextant with a probe depth of e"4 mm showed that high upper-body obesity and high total body fat were associated with a higher risk of periodontitis as compared to normal weight individuals.[21] A longitudinal study recently published by Morita et al involving 2,787 Japanese men and 803 Japanese women, whose BMI and the incidence of periodontal disease as defined by a probing depth >4 mm, were evaluated over a 5-year period. The study reported that the men and women in the BMI groups of 25-29.9 kg/m2 and e"30 kg/m2 were statistically more likely to develop periodontitis than those whose BMI was <22 kg/m2.[22] Moreover, these findings demonstrated a dose-response relationship between BMI and the development of periodontal disease in the population studied.

Boesing *et al*., 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.[23] A study that analyzed the National health and nutrition examination survey-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.[24]

Giri DK et al, conducted a study on the relationship between periodontitis and obesity on Indian population and concluded that good oral hygiene and normal body weight can reduce the overall inflammatory burden, thereby reducing the risk for development of periodontal disease. [25]

Conclusion

Periodontists must be aware of the increasing numbers of obese persons and of the significance of obesity as a multiple-risk-factor syndrome for overall and oral health. Proinflammatory cytokines may be a multidirectional link among periodontitis, obesity and other chronic diseases. The adipose tissue is a large reservoir of biologically active mediators, such as TNF-á and other adipokines. Studies have demonstrated a close involvement of the adipokines – such as leptin, resistin and adiponectin – in inflammatory processes.[26] 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.[27] In current scenario, it may not be incorrect to construe that maintaining standard body weight may be significant in reducing the risk of developing periodontal disease. Further, maintenance of adequate oral hygiene and normal body weight can reduce the overall inflammatory burden.

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