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Review Article

Role of Obesity in Chronic Periodontal Disease - A Literature Review

Amrutiya MR* and Deshpande N
Department of Periodontics, K.M. Shah Dental College & Hospital, India

***Corresponding author:** Amrutiya MR, Department of Periodontics, K.M. Shah Dental College & Hospital, At. & Po. Piparia, Gujarat, India

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Abstract

Obesity is a chronic disease with a multifarious origin where there is increase in deposition of fat in the adipose tissue. Obesity has become an important health problem as its prevalence is increasing worldwide. Periodontitis is a chronic inflammatory disease that affects the supporting structures of the tooth. Chronic periodontitis has a multifaceted origin with numerous risk factors that contribute to disease progression. Obesity represents a systemic condition capable of causing the onset and progression of periodontal disease. The probable mechanism for this association has been suggested to involve the proinflammatory state that exists in patients with obesity, which results in insulin resistance and oxidative stress. So the aim of this article is to get an overview of the association between obesity and periodontitis and to review the association between adipose tissue derived cytokines and periodontal disease.

Keywords: Obesity; Chronic periodontal disease; Adipose tissue; Body mass index

Introduction

An increased prevalence of obesity has been observed in recent years and is one of the fastest growing health-related problems in the world. Obesity is caused by eating more calories than the calories burn in daily routine activity. As the prevalence of obesity is increasing, the other health related problems like cardiovascular disease, diabetes mellitus, and hypertension have also increased in conjunction with obesity. Obesity contributes to an overall inflammatory condition through its metabolic and immune parameters, thereby increasing susceptibility to periodontal disease [1]. Periodontitis is a chronic inflammatory disease affecting the tooth supporting structures. Periodontal disease is chronic in nature and can prevail in the absence of treatment. The risk factors linked to a higher risk of periodontitis include smoking, hormonal changes in females, diabetes, genetics, AIDS, cancer. The mechanism(s) whereby obesity may affect periodontal health is so far unclear. Focusing on the biological aspects, alterations in oral conditions or a low grade chronic inflammation might be because of the excessive adipose tissue. It has also been suggested that there is a high production of pro-inflammatory cytokines, such as interleukin (IL)-1β, tumor necrosis factor (TNF)-α, IL-6, by the adipocytes and macrophages of the white adipose tissue [2]. These cytokines play a significant role in the development and progression of periodontal disease because the release of inflammatory cytokines is closely linked to a higher susceptibility to bacterial infection, caused by an alteration in the host immune response [3]. This review focuses on the effects of obesity on infectious disease and surmise on possible mechanisms for increased susceptibility of the obese host.

Obesity: definition, assessment and complication

Obesity is the excess amount of body fat in proportion to lean body mass, to such an extent that the health is impaired. The explanation of obesity is based on Body Mass Index (BMI), that is person's weight (in kilogram), divided by the square of his/her height (in meter). The normal range is given below in Table 1.

Not only the total body fat matters but also the pattern of distribution of fat matter. Excess visceral fat, also referred to as central obesity, shows a strong association with cardiovascular disease compared to subcutaneous fat (mainly deposited around the hips and buttocks). Central obesity produces a characteristic body shape which resembles an apple and thus is also referred to as "apple shaped" obesity as opposed to "pear shaped" obesity in which fat is deposited on the hips and buttocks.4 Waist Circumference (WC) is used to measure the body fat distribution, the cut off point for abdominal obesity in men 102 cm and in women 88 cm.

Various health consequences are associated with obesity that includes impaired glucose tolerance and diabetes mellitus, hypertension, heart disease like coronary artery disease, heart failure, Dyslipidaemia, Cerebrovascular Disease like haemorrhagic and ischaemic stroke, Metabolic syndrome like insulin resistance, Pulmonary abnormalities like obstructive sleep apnea, asthma, Gastrointestinal abnormalities like Gastroesophageal reflux disease, cholelithiasis, Osteoarthritis, Reproductive disease like Polycystic ovary syndrome in females and impotence and infertility in males, Cancer like cancer of gallbladder, esophagus (adenocarcinoma), thyroid, kidney, uterus, colon and breast, and Pyschosocial problems.

Obesity – role of adipose tissue derived cytokines and oral health

The adipose tissue, shown to function as endocrine organ, is a loose connective tissue composed of adipocytes which not only acts as a passive triglyceride reservoir but also produces high levels of cytokines and hormones collectively called as adipokines or adipocytokines and can cause disease through dysregulated immune responses. Adipokines represents a number of different roles such as

(a) hormone-like proteins [e.g. leptin and adiponectin],

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(b) classical cytokines [e.g. tumor necrosis factor alpha (TNF-α) and interleukin 6 (IL-6)],

(c) proteins involved in vascular hemostasis [e.g. plasminogen activator inhibitor-1 (PAI-1) and tissue factor],

(d) regulators of blood pressure [angiotensinogen],

(e) promoters of angiogenesis [e.g. vascular endothelial growth factor]

(f) acute phase respondents [e.g. C-reactive peptide]

These adipocytokines plays an important role in initiation of periodontal disease by activating monocytes which increases the production of inflammatory cytokines. The release of inflammatory cytokines causes an alteration in the host immune response that links to a higher susceptibility to bacterial infection.

Leptin was the first adipocyte hormone discovered and is mainly produced by adipocytes. Leptin enhances the host immune mechanism by activation of monocytes and macrophage function and manage activities like phagocytosis and cytokine production, chemotaxis and oxidative species production by stimulated neutrophils [5]. It also plays an important role in development of natural killer cells and shifting T-cell responses towards Th1 cytokine type and inhibit Th2 cell. Thus the overall increase in leptin during infection and inflammation indicates that leptin is a part of immune response and host defense mechanisms. A study done by Johnson RB and Serio FG suggests that leptin is present within healthy gingival and its concentration declines coincident to the severity of gingival inflammation and periodontal pocket formation [6].

Adiponectin is produced primarily by adipocytes and plasma levels of adiponectin decrease in obese subjects compared to normal weight subjects. Adiponectin has certain constructive outcomes like anti inflammatory, vasoprotective and antidiabetic effects. These defensive effects occur due to suppression of tumor necrosis factor-α, interleukin-6 and along with induction of interleukin-1 receptor antagonist. Iwayama T et al. in his study demonstrated that adiponectin exerts anti-inflammatory effects on Human Gingival Fibroblasts (HGFs) and Mouse Gingival Fibroblasts (MGFs), and promotes the activities of osteoblastogenesis of Human Periodontal Ligament (HPDL) cells and concluded that adiponectin has potent beneficial functions to maintain the homeostasis of periodontal health, improve periodontal lesions, and contribute to wound healing and tissue regeneration [7].

Resistin does not directly originate from adipocytes but may originate from inflammatory cells infiltrating the fat tissue. Resistin acts on adipocytes resulting in insulin resistance. It is related to the activation of inflammatory cells to secrete TNF-α and IL-6. TNF-α and IL-6 is primarily secreted by human adipose tissue. Elevated levels of TNF- α and IL-6 has shown an important link between obesity and periodontitis. Resistin impairs the anti-inflammatory effects of adiponectin. With increased obesity (the major contributing risk factor for developing type II diabetes mellitus and periodontits) there are increased levels of resistin. Furugen R et al. concluded that increased serum resistin levels were significantly associated with periodontal condition, especially when considering bleeding on probing, in elderly Japanese people [8].

Table 1: BMI range and values.

Categorization	BMI values
Underweight	<18.5
Normal	18.5 – 24.9
Overweight	25.0 – 29.9
Obesity class I	30.0 – 34.9
Obesity class II	35.0 – 39.9
Obesity class III	>40.0

The most recently identified adipocytokine is visfatin, produced by visceral adipose tissue and has insulin-mimetic action. Visfatin, exerts hypoglycaemic effect by binding to insulin receptor at a site distinct from insulin. Levels of visfatin increase in inflammatory conditions like periodontitis. A study done by Tabari et al. concluded that there is significant increase in the concentration of salivary vasfatin level in patients with chronic periodontitis [9]. Visfatin can be considered as an inflammatory marker and can be used in future as a potential therapeutic target in the treatment of periodontal disease.

Evidences showing association between obesity and periodontal disease

Saxlin T [10] et al. performed a longitudinal study to investigate the association between body weight and periodontal infection. He concluded that this longitudinal study does not provide evidence that overweight and obesity can be considered as significant risk factors in the pathogenesis of periodontal infection.

Singh MP et al. [11] did a study to evaluate the relationship between obesity and periodontitis and showed that the prevalence of periodontitis was significantly more in obese than in non obese. It was concluded that strong correlation exists between obesity& periodontitis. Obese with high serum triglycerides & LDL could be at higher risk of periodontitis.

Palle AR et al. [12] demonstrated significant association between measures of overall and abdominal obesity (BMI and WC) and periodontal disease showed significant association in his cross sectional study.

Giri DK et al. [13] did a study to determine the association of obesity with periodontal disease in a semi urban Indian population. No association was obtained between BMI and periodontitis. Hence it was concluded, that good oral hygiene and normal body weight can reduce the overall inflammatory burden, thereby reducing the risk for development of periodontal disease.

Chaffee BW [14] et al. in a systematic review of 70 cross sectional studies, suggested that 41 studies showed positive association between chronic periodontal disease and obesity. Also positive association between periodontal disease and obesity was suggested across diverse populations. The prevalence of periodontal disease is likely to be higher among obese patients, although there is no current evidence to recommend differences in treatment planning.

Keller A [15] et al. reviewed 8 longitudinal and 5 interventional studies of which 2 of the longitudinal studies showed a direct association between degree of overweight and risk of developing periodontitis. 3 studies found a direct association between obesity and development of periodontitis among adults. Two intervention

studies on the influence of obesity on periodontal treatment effects found that the response to non-surgical periodontal treatment was better among lean than obese patients; the remaining three studies did not report treatment differences between obese and lean participants. The review suggests that overweight, obesity, weight gain, and increased waist circumference may be risk factors for development of periodontitis or worsening of periodontal measures.

Effect of Non Surgical Periodontal Therapy (NSPT) on obesity and periodontal disease

Goncalves TE et al. [16] did a study to evaluate the effects of Scaling and Root Planing (SRP) on clinical parameters and circulating levels of leptin and adiponectin in obese patients with Chronic Periodontitis (CP). It was seen that there was no changes in the serum levels of leptin and adiponectin in groups with and without obesity after scaling and root planning therapy. It was concluded that patients with obesity and CP presented lower reductions in PD than patients without obesity with CP at 6 months after SRP. Furthermore, the treatment did not affect the circulating levels of leptin and adiponectin in any group.

Zuza EP et al. [17] performed a study to assess the role of obesity as a modifying factor on periodontal clinical parameters and on circulating proinflammatory cytokine levels in subjects undergoing non-surgical periodontal treatment. The results of the study showed that Obesity does not seem to play a negative role by interfering in the improvement of the periodontal clinical response or decreasing circulating proinflammatory cytokine levels after periodontal treatment.

Altay U et al. [18] aimed to evaluate the short-term changes in systemic inflammatory, lipid, and glucose parameters in the presence of obesity after periodontal treatment. The treatment was associated with a decrease in serum TNF-α and IL-6 levels and Homeostasis Model Assessment of the Insulin Resistance (HOMA-IR) scores in individuals with obesity and with a decrease in IL-6 levels in patients without obesity. Conversely, there were insignificant decreases in lipid profiles and serum fasting glucose of patients with obesity. It was concluded that the non-surgical periodontal treatment causes a decrease in the levels of some circulating proinflammatory cytokines and may be associated with a decrease in insulin resistance in the obese population.

Conclusion

Obesity is a complex and has a multifarious origin. Various evidences have well-documented a positive association between obesity and periodontal disease but the exact mechanism behind it is still not clear and requires further investigation. It is quite difficult to say if obesity predisposes to periodontal disease or periodontal disease affects lipid metabolism or both. Further longitudinal studies are needed to focus the causality and to determine if obesity is a true risk factor for periodontal disease.

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