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Role of Obesity in Chronic Periodontal Disease : A Literature Review

Dental Science

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ABSTRACT

Obesity is a chronic disease with a multifarious origin where in there in increase 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.

Introduction :

Well documented increased prevalence of obesity is been observed in recent years and is the fastest growing health-related problem in the world. Obesity is caused by eating more calories than the amount of calories burn in daily routine activity. As the prevalence of obesity is increasing, the other health related problems like cardiovascular disease, diabetes mellitus, 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 focus 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 (kg), divided by the square of his/her height (meter). The normal range is given below in table 1.

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

Not only the total body fat matter but also the pattern of distribution of fat matter. Excess visceral fat, also referred to as central obesity, also 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 adipokines or adipocytokines and can cause disease through dysregulated immune responses. Adipokines represents a number of different role such as

(a) hormone-like proteins [e.g. leptin and adiponectin],
(b) classical cytokines [e.g. TNF-α and IL-6],
(c) proteins involved in vascular hemostasis [e.g. PAI-1 and tissue factor],
(d) regulators of blood pressure [angiotensinogen],
(e) promoters of angiogenesis [e.g. VEGF]
(f) acute phase respondents [e.g. CRP]

These adipocytokines plays an important role in initiation of periodontal disease by activating monocytes which in-

creases 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 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 part of immune response and host defense mechanisms. A study done by Johnson RB and Serio FG suggests that leptin is present within healthy gingiva 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 from 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 also related to the activation of inflammatory cells to secrete TNF-α and IL-6. It also impairs the anti-inflammatory effects of adiponectin. With increased obesity (the major contributing risk factor for developing type II diabetes mellitus and periodontits) there is 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

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.

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 various 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:

Chaffee BW10 et al did a systematic review of 70 cross sectional studies, of which 41 studies suggested a positive association between chronic periodontal disease and obesity. A 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 A11 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.

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. Interventional studies on periodontal clinical response between obese and normal-weight patients undergoing non-surgical periodontal treatment are few, thus more evidences are required to evaluate for its effect.

Reference:

1. Altay U, Gurgan CA, Agbaht K. Changes in Inflammatory and Metabolic Parameters After Periodontal Treatment in Patients With and Without Obesity. J Periodontol. 2013;84:13-23.
2. Cancello R, Clement K. Is obesity an inflammatory illness? Role of low-grade inflammation and macrophage infiltration in human white adipose tissue. BJOG. 2006;113:1141-47.
3. Karlsson EA, Beck MA. The burden of obesity on infectious disease. Exp Biol Med 2010;235:1412-24.
4. Segula D. Complications of obesity in adults: A short review of the literature. Malawi Medical Journal. 2014; 26:20-24.
5. Suresh S, Mahendra J. Multifactorial Relationship of Obesity and Periodontal Disease. J Clin Diagn Res. 2014;8:01-03.
6. Johnson RB, Serio FG. Leptin within healthy and diseased human gingiva. J Periodontol. 2001;72:1254–7.
7. Iwayama T, Yanagita M, Mori K, Sawada K, Ozasa M. Adiponectin regulates functions of gingival fibroblasts and periodontal ligament cells. J Periodontal Res. 2012;47:563-71.
8. Furugen R, Hayashida H, Yamaguchi N, Yoshihara A, Ogawa H, Miyazaki H, et al. The relationship between periodontal condition and serum levels of resistin and adiponectin in elderly Japanese. J Periodontal Res. 2008;43:556-62.
9. Tabari ZA, Azadmehr A, Nohekhan A, Naddafpour N, Ghaedi FB. Salivary Visfatin Concentrations in Patients With Chronic Periodontitis. J periodontal. 2014;85:1081-5.
10. Chaffee BW, Weston SJ. Association Between Chronic Periodontal Disease and Obesity: A Systematic Review and Meta-Analysis. J periodontal. 2010;81:1708-24.
11. Keller A, Rohde JF, Raymond K, Heitmann BL. Association Between Periodontal Disease and Overweight and Obesity: A Systematic Review. J periodontal. 2015;86:766-76.