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Obesity and Periodontal Disease – An Emerging Link

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ABSTRACT *Obesity has increased in prevalence and is a major contributor to worldwide morbidity. It has been a recognized as a major public health problem and evidence exists of its role as a major risk factor for a number of conditions, such as cardiovascular diseases, diabetes mellitus, cancer, osteoarthritis and gall bladder disease. One consequence of obesity is an increased risk for periodontal disease, although periodontal inflammation might, in turn, exacerbate the metabolic syndrome of which obesity is one component.*

This review, thus aims to show the positive association between obesity and periodontal disease.

Introduction

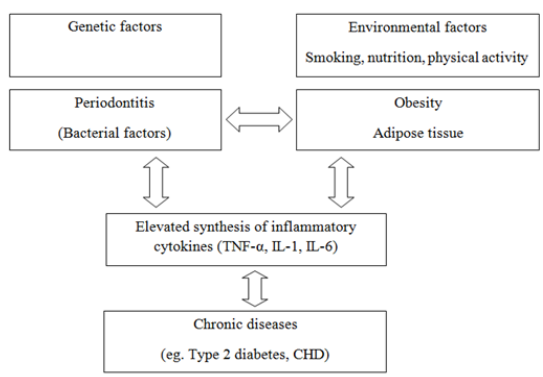
Obesity can be seen as the first wave of a defined cluster of non-communicable diseases called "New World Syndrome," creating an enormous socioeconomic and public health burden in poorer countries. The World Health Organization has described obesity as one of today's most neglected public health problems, affecting every region of the globe (Reddy MN, Kumar K and Jamil K, 2012).

In India, obesity is emerging as an important health problem particularly in urban areas. Almost 30-65% of adult urban Indians are either overweight or obese (Giri DK, Kundapur PP, Bhat GS, Bhat KM and Guddattu V, 2013). The rising prevalence of obesity in India has a direct correlation with the increasing prevalence of related co-morbidities; hypertension, the metabolic syndrome, dyslipidemia, type 2 diabetes mellitus (T2DM), and cardiovascular disease (CVD).

It has been suggested that obesity contributes to an overall systemic inflammatory state through its effect on metabolic and immune parameters; thereby increasing susceptibility to periodontal disease (Lacopino AM, 2009). Adipose tissue secretes several cytokines and hormones that are involved in inflammatory processes, suggesting that similar pathways are involved in the pathophysiology of obesity and periodontitis (Pischon N, Heng N, Bernimoulin JP, Kleber BM, Willich SN and Pischon T, 2007).

The present article provides an overview of association between obesity and periodontal disease.

Figure-1 : Model linking periodontitis and obesity with inflammatory related chronic diseases



TNF-α : Tumour Necrosis Factor- α
IL-1 : Interleukin-1
IL-6 : Interleukin-6
CHD : Coronary Heart Disease

Definition And Assessment Of Obesity

Obesity is an excess amount of body fat in proportion to lean body mass, to the extent that health is impaired. Obesity is defined based on body mass index (BMI), also called Quetelet Index, which is the ratio of body weight (in kg) to body height squared (in m^2). 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, older people (over 65 years) tend to have a higher body fat composition and therefore risk assessment by BMI is less accurate in them (Pischon N et al, 2007).

The World Health Organization and the National Heart, Lung and Blood Institute (NHLBI) define overweight as a body mass index of 25–29.9 and obesity as a body mass index of ≥30. It is important to note that Body Mass Index does not assess body fat distribution.

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Recent large studies have indicated that measurements of waist circumference or waist–hip ratio may be a better disease risk predictor than BMI.

Other diagnostic tools available to assess body fat composition are-

- Skin fold thickness
- Bio-impedance
- Underwater Weighing (Densitometry)
- Air-Displacement Plethysmography
- Dilution Method (Hydrometry)
- Dual Energy X-ray Absorptiometry (DEXA)
- Computerized Tomography (CT) and Magnetic Resonance Imaging (MRI)

Prevalence of Obesity in Indian Population

Very few studies on the prevalence of obesity have been reported from developing countries like India. Under-nutrition is more prevalent in rural areas, whereas overweight and obesity are more than three times higher in urban areas. This may be due to lesser physical activity in the urban areas. The additional burden of obesity due to increasing sedentary lifestyle, junk food habits in some urban and economically sound areas is really alarming (Garg C and Khan SA, 2010).

Obesity Related Important Diseases

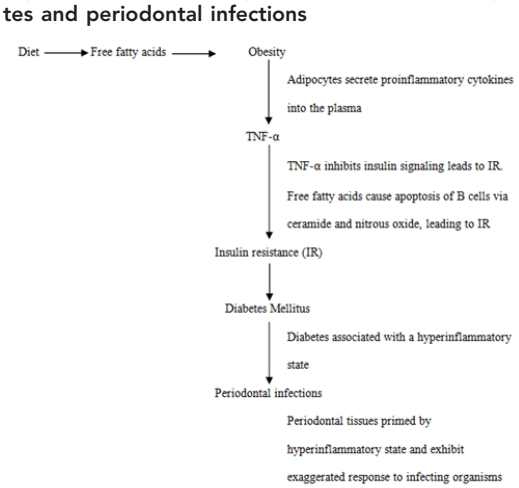
Hypertension

Overweight and obesity have long been recognized as important determinants of elevated blood pressure levels. It is well established that weight gain is consistently associated with increase blood pressure and that weight loss decreases blood pressure independent of changes in sodium intake. The mechanisms that have been implicated in the development of obesity related hypertension include increase sympathetic nerve activity, sodium and volume retention, renal abnormalities, insulin resistance, hyperleptinemia and increase secretion of angiotensinogen from adipocytes (Ritchie CS, 2007).

Type 2 Diabetes

There is close relationship between obesity and type 2 diabetes. Obese persons have a more ten-fold increase risk of developing type 2 diabetes compared with normal weight persons. This develops due to an interaction between insulin resistance and beta cell failure.

Figure- 2 : Model linking inflammation to obesity, diabetes and periodontal infections



TNF-α : Tumour Necrosis Factor- α

IR : Insulin Resistance

Cardiovascular Disease

Obese people have a 1.5 fold increased risk of cardiovascular disease (including coronary heart disease and cerebrovascular disease). It is also associated with a two-fold higher risk of heart failure and 50% increase risk of atrial fibrillation. This is probably a result of combination of factors including associated hypertension, type-2 diabetes, dislipedemia, diabetes and accelerated artherosclerosis, all of which increase with obesity (Pischon N et al, 2007).

Metabolic syndrome

It is the clustering of interrelated risk factors that identify individuals at risk for type-2 diabetes and cardiovascular disease. Insulin resistance is considered to be a major underlying abnormality (Ritchie CS, 2007).When three or more of the five listed criteria are present, the diagnosis of metabolic syndrome can be made.

Table-1 : Criteria for the diagnosis of metabolic syndrome

Elevated waist circumference Men Women	 ≥40 inches (102cm) ≥ 35 inches (88 cm)
Elevated triglycerides	≥150mg/dl
Reduced HDL(Good cholesterol) Men Women	 < 40mg/dl < 50mg/dl
Elevated blood pressure	≥130/85mmHg
Elevated fasting glucose	≥100mg/dl

HDL : High Density Lipoprotein

Relationship Between Obesity And Periodontal Disease

An association between obesity and periodontitis was first reported in 1977 when Perlstein et al. observed histopathologic changes in the periodontium in hereditary obese Zucker rats. Using ligature induced periodontitis; they found alveolar bone resorption to be greater in obese animals compared with non-obese rats. In obese and hypertensive rats, plaque accumulation caused even more pronounced periodontal destruction, suggesting that a combination of risk factors, such as those defined by the metabolic syndrome; elicit the most severe periodontal effects (Ritchie CS, 2007).

In 1998, Saito et al. analyzed 241 healthy Japanese individuals and showed, for the first time, an association between obesity and periodontal disease in humans. In addition, studies have indicated that the fat distribution pattern plays a crucial role in the association with periodontitis. It was found that high upper body obesity and high total body fat were correlated with a higher risk of periodontal disease, compared with normal weight persons. An exami-

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nation of the NHANES III data demonstrated that waist-to-hip ratio, BMI, fat-free mass, and log sum of subcutaneous fat significantly correlated with periodontal disease. Genco et al, in 2005 analyzed NHANES III data and demonstrated that BMI was positively correlated with the severity of periodontal attachment loss; they found that this relationship is modulated by insulin resistance.

In general, data indicate that increased body mass index, waist circumference ratio (abdominal obesity), serum lipid levels and percentage of subcutaneous body fat are associated with increased risk for periodontitis. After adjusting for confounding factors such as smoking, age and systemic conditions, the risk association appears to be linear. For instance, more bleeding on probing, deeper periodontal pockets and more bone loss were noticed in individuals with higher indicators of obesity (Wood N, Johnson RB and Streckfus CT, 2003).

Amelie Keller (2015) conducted a systematic review in which intervention and longitudinal studies with overweight or obesity as exposure and periodontitis as outcome were considered. Two of the longitudinal studies found a direct association between degree of overweight at baseline and subsequent risk of developing periodontitis, and a further three 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. Among the eight longitudinal studies, one study adjusted for C-reactive protein (CRP) and biologic markers of inflammation such as, interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and inflammation markers were analyzed separately in three of the five intervention studies. This systematic review suggested that overweight, obesity, weight gain and increased waist circumference may be risk factors for development of periodontitis or worsening of periodontal measures (Keller A, Rohde JF, Raymond K and Heitmann BL, 2015).

Obesity, Periodontal Disease and Chronic Inflammation
Adipose Tissue-

It is a loose connective tissue composed of adipocyte. 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 (adipocytes, preadipocytes and macrophages), secrete more than 50 bioactive molecules, known collectively as adipokines. Some of these act locally whereas others are released into the systemic circulation, where they act as signaling molecules to the liver, muscle and endothelium (Ritchie CS, 2007).

Adipokines play a number of different roles such as -

1. Hormone-like proteins, e.g. Leptin and adiponectin
2. Classical cytokines, e.g. TNF-α and IL-6
3. Protein involved in vascular haemostasis, e.g. Plasminogen activator inhibitor-1, tissue factor
4. Promoters of angiogenesis, e.g. Vascular endothelial growth factor
5. Acute phase respondents, e.g. CRP
   Obese individual have reported to have elevated levels of circulating TNF-α, IL-6 as compared to normal–weight individuals.

I) Leptin
Leptin, a non-glycosylated polypeptide that acts as cytokine and a hormone, is involved in a multitude of biological processes including energy metabolism, endocrine functions, reproduction and bone metabolism. It regulates adipose tissue mass. As a negative feedback mechanism elevated leptin concentrations result in increased energy expenditure, decreased food intake, and a negative energy balance. Most of the patients suffering from obesity have leptin resistance. Leptin is also involved in bone metabolism. In inflammatory periodontal disease leptin regulation has still to be examined.

II) Adiponectin
Adiponectin, a circulating hormone improves insulin sensitivity and may have anti-atherogenic and anti-inflammatory properties. Low plasma adiponectin levels have been shown to predict type-2 diabetes and coronary heart disease in humans. Experimental models suggest that adiponectin could play a role as a mediator of inflammation; however, the exact role of adiponectin in inflammatory disease remains to be elucidated (Pischon N, 2007).

III) Resistins
Resistin belongs to a family of resistin–like molecules (RELM) and has been reported to be secreted by adipocytes and cause insulin resistance in animal models. Current evidence suggests that human resistin is more closely related to inflammatory processes. The role of resistins in inflammatory periodontal disease remains to be defined.

IV) Cytokines (TNF- α and IL-6)
Obesity-associated TNF-α is primarily secreted from macrophages accumulated in abdominal adipose tissue. Increased circulating TNF-α from adipose tissue contributes to poor health outcomes by increasing insulin resistance and by inducing CRP production and general systemic inflammation. It also facilitates monocyte recruitment into developing atherosclerotic lesions and is a potent inhibitor of adiponectin, an important anti-inflammatory adipokine.

IL-6 is secreted by human adipose tissue and is produced in greater amounts by deep abdominal fat rather than by subcutaneous fat. It is a procoagulant cytokine and increases plasma concentrations of fibrinogen, plasminogen activator inhibitor-1 and CRP. Elevated levels of IL-6 are associated with increased risk of cardiovascular events in healthy men (Ritchie CS, 2007).

V) C-Reactive Peptide
Elevated CRP levels are associated with obesity, the development of cardiovascular disease and the risk of progression to type-2 diabetes mellitus.

VI) Plasminogen Activator Inhibitor-1
Plasminogen activator inhibitor-1 is a regulatory protein of the coagulation cascade. It prevents the dissolution of clots by inhibiting extracellular matrix degradation and fibrinolysis. It is produced both by adipocytes and stromal cells surrounding the adipocytes.

Association of Periodontitis and Obesity Related Chronic Disease
Proinflammatory cytokines may play a link between periodontitis and obesity related chronic diseases. This association may be multidirectional. Inflammatory diseases like periodontitis induce the production of pro-inflammatory cytokines such as TNF-α, IL-6 and IL-1. Lipopolysaccharide from periodontal gram negative bacteria stimulates

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the adipose tissue to secrete TNF-α which promotes hepatic dyslipidemia and decreases insulin sensitivity. This leads to type-2 diabetes associated with the production of advanced glycation end-products (AGE's), triggering inflammatory cytokine production and thus predisposing for inflammatory disease such as periodontitis (Genco et al, 2005).

These observations suggest a potential interaction among obesity, periodontitis and chronic disease incidence. Conversely periodontitis, once it exists, may promote systemic inflammation and thereby increase the risk of coronary heart disease (Beck and Offenbacher, 2005 and Loss, 2005). Periodontal treatment has been shown to reduce circulating TNF- α and serum levels of glycosylated hemoglobin, and has beneficial effects on the control of type-2 diabetes.

Risk and Risk Assessment in Periodontal Office

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 body mass index on a regular basis. Oral health care providers should consider obtaining waist circumferences in patients with a body mass index of ≥25, but <35, as these individuals may carry more risk than their body mass index alone may suggest. Besides the suggested association between periodontal disease and obesity, periodontists need to be aware of the health problems related to obesity, and should take them into account during treatment. The medical history should take note of comorbid conditions, including hypertension, dyslipidemia, coronary heart disease, type-2 diabetes and sleep apnoea. Higher incidences of infections and post-surgical hematoma formation have been reported among obese persons. The vulnerability to wound complications increases not only morbidity and mortality of obese persons, but also the length of individual treatment sessions, the overall length of the treatment, and, consequently, the economic costs of treatment. Pharmacological aspects, such as altered pharmacokinetics due to the person's increased blood volume or fat mass and technical incompatibilities, such as small dental chairs or tight blood pressure cuffs, should be considered. Also, a close collaboration with the general physician and the dietician may be beneficial to ensure effective periodontal treatment.

Conclusion

Obesity has taken on epidemic proportions internationally. Much comorbidity is associated with obesity and has consequences for oral health professionals. What has emerged from literature, however, is that an association between obesity and periodontitis exists and this association most likely lies in the commonality of their inflammatory pathways. 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. Obesity is a complex disease, and its relationship to oral status has been realized by the scientific community in recent years. Although this relationship needs further investigation, periodontists should counsel obese persons regarding the possible oral complications of obesity, to diminish morbidity for these individuals. This includes the measurement of body mass index and waist circumference for periodontal risk assessment on a regular basis.

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