Inter Relationship Between Obesity And Periodontal Disease?

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Citation

Abstract

Obesity is a multifaceted subject. It has increased at an alarming rate in recent years. Being overweight increases 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 periodontium may be mediated through pro-inflammation cytokines and various other bioactive substances. This article focuses on the possible role of obesity as a potential contributor to periodontal disease and vice-versa. The outcome of these associations can be used for various diagnostic and treatment planning purposes.

INTRODUCTION

The global obesity epidemic has been described by the world health organization (2000) 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 significant contributor to the development of hypertension, diabetes mellitus, arteriosclerosis and cardiovascular and cerebrovascular diseases. 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 teeth resulting from the interaction between pathogenic bacteria and the host immune response (5).

DEFINITION OF OBESITY

Obesity occurs when the size and number of fat cells in a person's body increase. A normal person has 30-35 billion fat cells. When a person gains weight, these fat cells first increase in size and later in number. The WHO defines obesity as abnormal or excessive fat accumulation that may impair health and classifies obesity as a chronic disease (1). According to the NIHS, a person is considered obese if the BMI of the person is above 30.

PREVALENCE OF OBESITY

Being overweight and obesity are 2 major health hazards in today's world: according to the WHO, one billion people were overweight in 2005, and the number will be 1.5 billion by the year 2015. The prevalence of obese and overweight people has doubled in number between 1960-1990. In 2004, approximately 34.1% of the U.S. population was overweight, and about 32.2% obese. In 2008, 32% of women and 42% men above the age of 16 were obese in the U.K. The prevalence of periodontal disease is 76% higher among young obese individuals aged 18-34 years than in normal weight individuals, (7) and being overweight is associated with an increased risk of periodontitis among those aged 17-21 years (8).

EVALUATION OF OBESITY

Obesity is usually evaluated by calculating Body Mass Index (BMI), waist circumference, waist-hip ratio and fat percentage. Other methods of measuring obesity include the use of ultrasound, densitometry or imaging procedures (Computed Tomography, Nuclear Magnetic Resonance) and dual energy X-ray absorptiometry, but these methods vary in reliability and require expensive equipment and staff training (1).

Body Mass Index: It is also known as the Quetelet index, and is the ratio of body weight (in kg) to height (in meters) squared. It is easy to calculate and is considered more

appropriate than simple weighing (1).

Figure 1

$$BMI = Weight\,(kg)/height^2(m^2)$$

Body Mass Index (BMI) Classification is as under:

Figure 2

Classification	BMI
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+

The concept of BMI has certain limitations: it does not assess body fat distribution, or whether it is central, visceral or android. It also ignores many factors such as differences in muscle mass, bone mass and genetic makeup. Thus BMI alone may not be an entirely appropriate measurement of overweight or obesity (1).

WAIST CIRCUMFERENCE/WAIST-HIP RATIO

Recent advances in the equipment to measure waist circumference have made this technique a better disease risk indicator than BMI (3).

Waist circumference shows a close correlation with the amount visceral adipose tissue. Visceral adipose tissue has been shown to be more metabolically active than subcutaneous adipose tissue and it secretes far greater amount of cytokines and hormones (9).

Figure 3

Waist circumference	Values
<94 cm for men <80 cm for women	Low
94-102 cm for men 80-88 cm for women	High
>102 cm for men >88 cm for women	Very high

Waist-hip ratio (WHR) is the ratio of the circumference of the waist to that of the hips. It is calculated by measuring the waist circumference just above the upper hip bone and dividing it by the hip circumference at the widest hip part.

Figure 4

Values	WHR
<0.83 for men <0.9 for women	Non-obese
>0.83 for men >0.9 for women	Obese

Fat percentage is calculated by bioelectric impedance analysis (BIA). It is a method of estimating body fat percentage. The device measures the flow of electric signals as they pass through fat and lean areas and water in the human body. It works on the principle that fat-free mass (muscle) is a good conductor, as it contains a large amount of water and electrolytes, while fat is anhydrous and a poor conductor of electrical current.

Figure 5

Values	Fat percentage
25%	Non-obese
>25%	Obese

THE INTERRELATIONSHIP BETWEEN OBESITY AND 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 pathogenesis of periodontitis. The adverse effect of obesity on periodontium may be mediated through pro-inflammatory cytokines like interleukins (IL-1, IL-6 and TNF-a), 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 (5).

ADIPOSE TISSUE DERIVED CYTOKINES AND HARMONES: Interleukin-6 is secreted by human adipose tissue and is produced in greater amount by deep abdominal fat than by subcutaneous fat. Elevated levels of interleukin-6 have been found to be associated with an increased risk of cardiovascular events, lipolysis and weight gain (6).

Tumor Necrosis Factor a: Obesity-associated tumor necrosis factor-a (TNF-a) is primarily secreted from macrophages accumulated in abdominal adipose tissue. Increased circulating TNF-a from adipose tissue contributes to poor health outcomes by increasing insulin resistance, C-reactive peptide production and general systemic inflammation. TNF-a is a potent inhibitor of adiponectin, an important anti-inflammatory adipokine (6).

ADIPONECTIN: Adiponectin is a circulating hormone secreted by adipose tissue. It is involved in glucose and lipid metabolism and accounts for about 0.05% of total serum proteins. Contrary to other adipose-derived hormones, adiponectin levels are reduced in persons with obesity, insulin resistance or type 2 diabetes. Adiponectin 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 (11-12).

Leptin: Leptin is the best-known substance secreted from 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. Most patients suffering from obesity have leptin resistance. It has been reported that leptin is present within healthy and marginally inflamed gingiva and decreased in concentration as the adjacent probing depth increases. Thus, leptin may play an important role in the development of periodontitis (13).

RESISTIN: Resistin belongs to a family of resistin-like molecules (REIM) and is reported to be secreted by adipocytes 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 determined (2).

PLASMINOGEN ACTIVATOR INHIBITOR-1 (PAI-1): PAI-1 is an adipokine that generates agglutination of the 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 (14-15).

HOST IMUNITY: Obesity increases the host's susceptibility by modulating the host immune and inflammatory systems, leaving the patients with 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 rat models (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 overproduction 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 the progression of periodontitis (16-17).

BACTERIAL CONTRIBUTION TO OBESITY: Goodson J.M et al. suggested three mechanisms by which oral bacteria can contribute to development of obesity. First, the oral bacteria may contribute to increased metabolic efficiency, as suggested by infectobesity proponents. 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-a or reducing levels of adiponectin. By any of these mechanisms, even a small excess in caloric consumption with no change in diet or exercise could result in unacceptable weight gain (10).

CONCLUSION

Obesity is a complex and multifaceted disease. Its relationship with periodontal disease is well documented, but the underlying mechanism is under investigation. At this moment it is difficult to state whether obesity predisposes individuals to periodontal disease or if periodontal disease affects lipid metabolism. 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, prevention and management of obesity may be an adjunctive approach to improving periodontal health.

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