Review Article

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Obesity and Periodontal Disease: An Overview

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ABSTRACT

Background: Obesity is one of the most common, yet among the most neglected, public health problems in both developed and developing countries. It remains a leading public health problem because of its complications and resistance to change, despite record rates of dieting. There are various factors responsible for obesity such as genetics, behavioral, social and cultural factors. Obesity is associated with risk factors such as diabetes, hyperlipidemia, hypertension, atherosclerosis, cardiovascular disease. Obesity is characterized by a chronic inflammatory state, cytokines such as TNF-a which is produced by adipose tissue which is responsible for these alterations. Studies have shown alveolar bone loss in ligature induced periodontal disease in obese animals depicting relationship between obesity and periodontal disease. Also BMI was positively correlated with the severity of periodontal attachment loss. Adipose tissue secretes pro-inflammatory cytokines such as tumor necrosis factor-alpha(TNF-a) and interleukin-6(IL-6) which also induces release of acute-phase protein production such as C-reactive protein(CRP). Periodontitis is also related with release of these pro-inflammatory cytokines which is triggered by LPS of gram negative periodontal pathogenic bacteria promoting dyslipidemia and decrease insulin. Type 2 diabetes and decrease insulin are associated with the production of glycated end products (AGE), which in turn trigger the inflammatory cytokine production, thus predisposing to inflammatory condition such as periodontitis. Thus these observations suggest a potential association of obesity with periodontitis.

Keywords: Obesity, Periodontitis, Adipokines, Pro-inflammatory cytokines, Inflammatory disorders.

INTRODUCTION

According to the World Health Organization (WHO), obesity is one of the most common, yet among the most neglected, public health problems in both developed and developing countries (1). This epidemic has received both national and International attention because of obesity's detrimental impact on health, the enormous economic burden it imposes and its increasing prevalence. It remains a leading public health problem because of its complications and resistance to change, despite record rates of dieting. Risk factors exist at both population and individual levels, thus obesity has diverse etiologies and consequences. In addition to the increased morbidity and functional limitations associated with obesity, approximately 325,000 deaths in the United States each year among non-smokers are attributable to obesity (2).

PREVALENCE

The WHO-World Health statistics report 2012, states globally one in six adults is obese and nearly 2.8 million individuals die each year due to overweight or obesity (1). According to NHANES In 2011–2014, the prevalence of obesity was just over 36% in adults and 17% in youth. The prevalence of obesity was higher in women (38.3%) than in men (34.3%). Among all youth, no difference was seen by

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sex. From 1999 through 2014, obesity prevalence increased among adults and youth. Overall, the prevalence of obesity among preschool-aged children (2–5 years) (8.9%) was lower than among school-aged children (6–11 years) (17.5%) and adolescents (12–19 years) (20.5%). The same pattern was seen in both males and females (3).

The prevalence of obesity has increased substantially over the past decades in most industrialized countries. India, with 1.2 billion people is the second most populous country in the world and is currently experiencing rapid epidemiological transition. Undernutrition due to poverty which dominated in the past, is being rapidly replaced by obesity. According to national health and family survey 3 NHFS 3 (2005-06) which interviewed all women age 15-49 and all men age 15-54 from across the 29 states of India, Obesity, is a substantial problem among several groups of women in India, particularly urban women, well-educated women, women from households with a high standard of living, and among Sikhs. Fifteen percent of ever-married women are overweight or obese, up from 11 percent in NFHS-2. Obesity is particularly prevalent for both men and women in Delhi, Kerala, and Punjab (4).

DEFINITION AND ASSESSMENT OF OBESITY

The definition of obesity is based on the body mass index (BMI, also called Quetelet Index), which is the ratio of body weight (in kg) to body height (in m) squared (1). However, there are some limitations. For example, for the same BMI, older persons tend to have a higher body fat composition, and, therefore, risk assessment by BMI is less accurate in older people (over 65 yrs of age). Furthermore, current BMI cut-off points for overweight and obesity are probably too high for Asian populations). More importantly, the BMI does not assess body fat distribution. It is well-known that abdominal (central, visceral, android) obesity, which is usually observed in men, is associated with a higher morbidity than the gluteofemoral (peripheral) obesity typically observed in women (Expert Panel, 1998).Recent large studies have indicated that measurement of waist circumference or waist-hip ratio may be a better disease risk predictor than BMI (5)and there is still intensive research ongoing as to whether BMI, waist circumference, or both should be used to assess disease risk. Several other diagnostic tools are available to assess body fat composition, such as measurement of (subcutaneous) skin fold by means of a caliper or ultrasound, bioelectrical impedance analysis (BIA), densitometry, or imaging procedures (CT, NMR); however, most of these procedures are not readily available in clinical practice, and do not add substantial information for risk assessment in an individual beyond BMI and waist circumference (6)

CLASSIFICATION OF OBESITY

In adults, overweight is defined by a BMI of ≥25.0 kg/m2 , and obesity is defined by a BMI of ≥30.0 kg/m2 , regardless of sex. The World Health Organization distinguishes several BMI categories based on increasing health risks (Table 1).

Table 1: World Health Organization's body mass index (BMI) categories based on increasing health risks established for non-Asian population (1)

Classification	BMI(Kg/m2)	Disease risk* relative to normal weight and waist circumference Men ≤102 cm Women ≤88 cm	Disease risk* relative to normal weight and waist circumference Men >102 cm Women >88 cm
Underweight	<18.5	-	-
Normal	18.5-24.9	-	-
Overweight	25.0-29.9	INCREASED	HIGH
Obese –class I	30.0-34.9	HIGH	VERY HIGH
Obese- class II	35.0-39.9	VERY HIGH	VERY HIGH
Obese class III	≥40	EXTREMELY HIGH	EXTREMELY HIGH

*Disease risk for diabetes type 2,hypertension and cardiovascular disease.

The recently proposed classification for Asian population is BMI <18.5-UNDERWEIGHT: 18.5-22.9,NORMAL WEIGHT: 23.0-24.9,OVERWEIGHT: 25.0-29.9, OBESE CLASS I ≥30.0, OBESE CLASS II(7)

ETIOLOGY

Three major factors are believed to contribute to the etiology of obesity: metabolic factors, diet, and physical inactivity. In turn, each is influenced by genetic traits. It is the result of genetic, behavioral, environmental, physiological, social, and cultural factors that result in energy imbalance and promote excessive fat deposition(8).

1. ROLE OF GENETICS

The genetic contribution to obesity has been elucidated by studies of Stunkard et al(9) involving twins, in which concordance rates for varying degrees of overweight were twice as high among monozygotic than dizygotic male twin pairs at age 20 years.

Bouchard et al(10) demonstrated that the amount of body weight and fat gained, as well as the distribution of fat gained in response to overfeeding, had greater similarity within than between twin pairs, further supporting the heritability of the tendency to become overweight or obese. One of the mechanisms by which genotype affects body weight is in the regulation of energy expenditure. It is estimated that approximately 40% of the variance in daily energy expenditure (excluding vigorous physical activity) is attributable to genotype. Thus there is substantial evidence for the role of genetics in body weight regulation. However, population-wide genetic variations alone cannot be solely responsible for the spread of obesity in epidemic proportions. Rather, behavioral and environmental factors are largely accountable. Therefore, the current obesity epidemic appears to be the result of environmental and behavioral factors along with genetic susceptibility.

2. ROLE OF METABOLIC FACTORS ENERGY EXPENDITURE

The development of obesity is dependent on an imbalance between energy intake and energy expenditure during an extended period of time. The cause may be viewed as excess energy intake relative to daily energy expenditure, or as low energy expenditure relative to daily energy intake. The three components of total energy expenditure include resting energy expenditure, thermic effect of food, and activity-related energy expenditure. Resting energy expenditure, which generally represents about 60% of daily energy expenditure, depends largely on body mass, especially fat-free mass, which is more metabolically active than fat tissue (11).

On an absolute basis, resting energy expenditure is usually higher in obese persons because of their greater body size, including fat-free mass. Thus, relatively low resting energy expenditure values could be associated with weight gain. Several longitudinal studies have addressed this possibility. A relatively low total daily energy expenditure (measured in a metabolic chamber) has been found to be strongly correlated with the rate of weight gain (r520.39, P ,0.001) (12). the available evidence suggests that reduced activity-related energy expenditure is a potentially important contributor to the predisposition to obesity.

DIETARY PATTERNS

Many studies have reported a relationship between dietary fat consumption as a percent of total caloric intake and adiposity, although the association appears to be highly variable (13). Despite an increased focus on nutrition, a heightened awareness of the energy and fat content of foods, and the availability of various reduced-fat, fat-free, and sugar-free foods and beverages, obesity continues to increase. Modern society facilitates excessive consumption with its abundance of inexpensive, energy-dense foods, numerous conveniently located eating establishments that promote dining away from home, a high variety of foods at mealtime, and unreasonably large portion sizes. Food manufacturers strive to enhance the appearance and flavor of packaged foods and, through effective advertising, promote overeating.

3. ROLE OF PHYSICAL INACTIVITY

There is an inverse relationship between physical activity and adiposity, with correlation coefficients in the order of -0.4(14). However, the amount of energy expended differs according to the

type of physical activity chosen, and the energy economy of the activity (energy expenditure/work performed) is modified by its relative intensity, the muscle groups used, and the range of motion involved (15). Prentice and Jebb (16) concluded that a modern inactive lifestyle must play an important, and perhaps dominant, role in the increasing prevalence of obesity. A 5-year prospective study of over 12,000 Finnish adults found that sedentary individuals were almost twice as likely to experience substantial weight gain as physically active men and women (17). By contrast, greater energy intake was associated with weight gain only among women. Thus, population wide, the relationship of physical inactivity to weight gain appeared to be more consistent than the relationship of excess energy intake to weight gain.

OBESITY AND PERIODONTAL HEALTH

Obesity is a chronic metabolic condition that has public health implications because it is a risk factor for many diseases, such as diabetes, hyperlipidemia, hypertension, atherosclerosis, cardiovascular disease, among others (18).

Three metabolic alterations are responsible for the obesity characteristics:

(1) Hyperinsulinemia: there is increased production of insulin by beta cells to compensate the resistance in the tissues, especially the adipose, muscular and hepatic ones.

(2) Hyperglycemia: since there is a resistance to insulin, the circulating glucose is not taken by the cells and there is an increase of the blood glucose levels.

(3) Hyperlipidemia: there is an elevation of the seric levels of cholesterol and triglyceride due to the lipid metabolism alteration – there is an increase in the liver lipogenesis and lipolysis in the adipocytes.

These alterations seen in obesity are also found in localized chronic or generalized acute infections. Cytokines, especially TNF-a, produced by the adipose tissue, are the ones responsible for those alterations. Obesity is then characterized as a chronic inflammatory state. It has been suggested that obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction.[19]. The first report on the relationship between obesity and periodontal disease appeared in 1977, when Perlstein et al found alveolar bone resorption due to ligature induced periodontitis to be greater in obese animals compared with non-obese Zucker rats. Also, it seemed that under healthy oral conditions, obesity per se does not promote pathologic periodontal alterations; however, in response to bacterial plaque accumulation, periodontal inflammation and destruction were more severe in obese animals.[20] Genco et al. analyzed National Health and Nutrition Examination Survey (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.[21]

ROLE OF ADIPOSE TISSUE DERIVED CYTOKINES IN PERIODONTAL TISSUE DESTRUCTION

Adipose tissue secretes pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-**α**) and interleukin-6 (IL-6). TNF-**α** and IL-6 are the main inducers of acute-phase hepatic protein production, including C-reactive protein (CRP).

Adiponectin is a hormone secreted by adipose tissue that is involved in glucose and lipid metabolism. It accounts for about 0.05% of total serum proteins. 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, and 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. [22,23]

Leptin regulates adipose tissue mass. It acts as a lipostat and works through a negative feedback mechanism, elevated leptin concentrations result in increased energy expenditure, decreased food intake and a negative energy balance. In contrast, most overweight and obese persons show resistance to leptin at the receptor level and therefore have higher leptin levels than non-overweight individuals[24]. Leptin has been shown to be involved in bone metabolism. Conflicting evidence exists that leptin may decrease bone formation via central nervous

pathways and may stimulate bone formation via direct peripheral effects on bone cells. The net result on bone formation may depend on various general and bone-specific factors, such as species, age, gender, serum leptin levels, blood-brain barrier permeability, bone tissue, skeletal maturity and signaling pathways [25]. In periodontal disease, leptin regulation has still to be examined, especially with respect to the epidemiological association between obesity and periodontitis. One study implied decreasing leptin levels in gingival biopsies with increasing pocket-probing depths, which would be contrary to the cited data on other inflammatory diseases [26].

Resistin belongs to a family of resistin-like molecules (RELM) and has been reported to be secreted by adipocytes and 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 defined.[27]

INFLAMMATION- A COMMON PATHWAY FOR CHRONIC DISORDERS

Inflammatory diseases like periodontitis induce the production of pro-inflammatory cytokines such as TNF-**α**, IL-1 and IL-6. It has been suggested that the secretion of TNF-**α** by adipose tissue triggered by LPS from periodontal gram-negative bacteria promotes hepatic dyslipidemia and decreases insulin. Type 2 diabetes and decreased insulin sensitivity are associated with the production of advanced glycation end products (AGE), which trigger inflammatory cytokine production, thus predisposing to inflammatory diseases such as periodontitis (28).These observations suggest a potential interaction among obesity, periodontitis and chronic disease incidence, although present studies are insufficient to conclude whether such associations are causal. Thus in addition to being a risk factor for type 2 diabetes and coronary heart disease, obesity-related inflammation may also promote periodontitis. In this context, it is interesting to note that periodontal treatment has been shown to reduce circulating TNF-**α** and serum levels of glycosylated hemoglobin (21). TNF-a helps exacerbate the periodontal disease and perpetuate the insulin resistance seen in obesity. Periodontal disease may also unbalance lipid metabolism worsening the hyperlipidemic state in the obese patients. In a case-control study by Noack et al (29) the patients with hyperlipidemia had higher periodontal inflammation than the control patients, with a higher percentage of sites and sextants with probing depth (PD) Ñ 3.5mm.

OBESITY AND DENTAL PRACTICE

Knowledge about obesity by dental professionals

Very little information exists regarding the experience of dental professionals with obesity management. Given the potentially negative effect of obesity on oral health-related outcomes, dental professionals would benefit from the inclusion of obesity education in the curricula and continuing medical education.

Screening

Health screening by oral health providers could have a significant impact on obesity prevention and management, as many individuals seek dental care who do not routinely seek medical care. In Glick's evaluation of National Health and Nutrition Examination Survey data, he identified 332,262 men who had a moderate risk of experiencing a cardiovascular event within 10 years, had not seen a physician in the previous 12 months, but had seen a dentist (30). These individuals would greatly benefit from obesity screening.

Assessment

Oral health care providers could consider obtaining waist circumferences in patients with a body mass index greater than 25, but <35, as these individuals may carry more risk than their body mass index alone may suggest. In addition, the medical history should take note of comorbid conditions, including hypertension, dyslipidemia, coronary heart disease, type 2 diabetes, and sleep apnea.

Counseling

The NHLBI Guidelines recommend that all patients with a body mass index of 30 or higher

should attempt to lose weight. Those with a body mass index of 25–29.9 kg/m2, and who have one or no risk factors, should work on maintaining their current weight rather than embark on a weight reduction program. The decision to lose weight must be made in the context of other risk factors (e.g. quitting smoking is more important than losing weight) and patient preferences. Patient motivation and confidence in making a change is crucial to success. Although the guidelines recommend the loss of 10% of baseline weight at a rate of 0.5–1 kg per week.

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. Many comorbidities are associated with obesity and have consequences for oral health professionals. Because many patients see their dentist more often than their primary care provider, the oral health provider can serve to screen and identify patients with obesity. Obesity is characterized by a chronic inflammatory state, which can worsen the preexisting periodontal disease. The adipose tissue is a large reservoir of biologically active mediators, such as TNF-α and other adipokines which play a role in inflammatory processes. Further longitudinal studies need to be done to establish the exact etiopathogenesis of obesity related periodontitis, until then risk assessment and counselling can be done to prevent obesity related periodontal problems.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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