

Research article

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Obesity and its Role in Periodontal Disease - A Review

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ABSTRACT:

Obesity is a multifactorial problem and its rate has increased at an alarming rate now a days. Patients with overweight increases the chances of having associated so many health and social problems which will affect dental services and management. An overall review among the literatures on obesity and its role in oral health suggested that they both are depend on each other. Obesity itself has been considered as a major risk factor for most of the periodontal diseases. Adverse effects of obesity/over weight on the periodontal structures and other oral structures may be mediated mainly through pro-inflammatory cytokines and some other bioactive substances which release. In this article our views tries to focus on the some reasonable role of obesity associated diseases, which may be a potential contributor to periodontal and other oral diseases and vice versa. These associations between oral health and obesity can be useful for various diagnostic, investigatory and treatment planning purposes which may helpful in proper dental and general services towards patients.

KEYWORDS: Obesity, Periodontal disease, Salivary proteome modifications, Adipokines.

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INTRODUCTION:

The world level epidemic of obesity has been described by, WHO (2002) as one of the most blatantly visible but yet most neglected public health problems that threatens to overwhelm both more and less developed countries^{1,2,3}. Main cause of obesity represents by the complex interaction between so many factors such as, genetics, diet, metabolism and physical activity levels, etc. In person's body, when increase size and number of fat cells occurs always results in obesity. Obesity defined usually by, Body Mass Index (BMI) and evaluated in terms of waist-hip ratio and closely related to percentage of body fat and total body fat^{1,4,5}.

EVALUATION OF OBESITY: Obesity is usually evaluated by three standard techniques

1. Body Mass Index (BMI)^{1,4}:

It is also known as, Quetelet index and is calculated by the ratio of body weight in Kgs to height in metres squared. Body mass index is considered more appropriate than simple weighing of total body weight.

BMI= Weight (Kg) /Height² (m²)

BMI classification is as under¹:

Table- no 1. "BMI Classification"

CLASSIFICATION	BMI
Underweight	< 18.5
Normal	18.5 24.9
Overweight	25.0 – 29.9
Obesity Class 1	30.0 – 34.9
Obesity Class 2	35.0 – 39.9
Obesity Class 3	>40

2. Waist-hip ratio (WHR)⁵: WHR is calculated by the ratio of the waist to that of the hips. It is usually calculated by measuring the waist circumference just above the upper hip bone and dividing it by the hip circumference at the widest hip part.

Table no-2 "WHR Classification"

VALUES	WHR	
< 0.83 for Men	Non Obese	
< 0.9 for Women		
> 0.83 for Men	Obese	
> 0.9 for Women		

3.Fat percentage: It is usually calculated by the method of Bioelectric Impedance Analysis^{1,4}.

Table no.3 "Fat Percentage"

VALUES	FAT PERCENTAGE
25%	Non obese
>25%	Obese

Other techniques which evaluate the obesity includes, skin fold by means of a calliper, Ultrasound, Densitometry or imaging procedures like Computed tomography, Nuclear magnetic resonance.etc^{1,5,6}.

OBESITY AND SYSTEMIC HEALTH:

Obesity usually affecting multisystem in the human body and a major contributor to development so many systemic conditions such as hypertension, Type2 diabetes, arteriosclerosis, cardio vascular and Cerebro vascular diseases, Sleep apnoea, Gastric reflux, Gout, Infertility, Osteoarthritis, Respiratory disorders, Metabolic syndrome, Hyperlipidemia and some types of cancer etc^{3,7}. Three metabolic alterations are responsible for the obesity characteristics:

- 1. Hyperinsulinemia
- 2. Hyperglycemia
- 3. Hyperlipidemia

These alterations seen in obesity are also associated with localised chronic or generalised acute infections⁷. Some associated factors linking obesity with the following systemic diseases as follows.

Hypertension – Not all obese candidates are affected by hypertension, weight gain is almost invariably associated with an increased BP. Raise in BP is very closely related to the raise in weight gain level and even moderate weight gain is associated with high risk of hypertensive state among people⁴.

Type2 diabetes – It occurs due to an interaction between beta cell failure and insulin resistance. Several factors such as obesity derived cytokines and some other pro inflammatory agents as well as lipotoxicity and glucose toxicity have been implicated in developing type2 diabetes ^{8,9,10,11}.

Cardiovascular disease – The majority of risk factors associated with development of cardiovascular disease are also considered to be risk factors for developing Periodontitis. The

presumed association between the poor oral hygiene maintenance and atherosclerosis can be explained by the chronic inflammatory disease effect on blood components and Th1 reaction^{4,12,13}.

Osteoarthritis – Obesity is considered as a major risk factor for development of osteoarthritis, but it is a modifiable risk factor only not an initiating factor. As the epidemic of obesity continues to grow, the incidence level of osteoarthritis will definitely follow¹⁴.

Respiratory disorders – Fat accumulation in visceral areas may leads to restrictive respiratory function with reduced level of expiratory reserve volume and forced vital capacity. Obesity is also considered as the major risk factor for obstructive sleep apnoea syndrome, etc¹⁵.

Metabolic syndrome – Although the exact etiology of metabolic syndrome is not known, the more recent descriptions mainly the focus on abdominal obesity than the overall obesity as its core component. These results supported by most of the studies, that the adipose tissue itself able to produce several proteins and hormones, which may involved in the development of obesity related diseases ^{10,16,17,18}.

PERIODONTITIS -AS A DISEASE ENTITY:

Periodontal disease refers to the process of destruction of the peri tooth structures that supports and helps in functions of the teeth. Chronic progressive destruction of these supporting tissue structures may leads to the eventual loss of teeth and associated functions. Recent notice on the importance of periodontitis and its perfect impact on the perpetuation and management of systemic diseases directs the globe to focus on control of periodontal disease^{12, 16, 19}.

MECHANISM LINKING OBESITY AND PERIODONTITIS:

The mechanism by which, obesity shows their effects on the periodontium is currently very poorly understood, but the obesity has several harmful biololgical effects that might be related to pathogenesis, progression and severity of Periodontitis. The adverse effects of obesity on periodontium may be usually mediated through some pro inflammatory cytokines such as interleukins (IL-1, IL-6 and TNF-α), adipokines (leptin, adiponectin, resistin, plasminogen activator inhibitors-1, chemerin, vaspin, visfatin, retinol binding protein-4) and several other bioactive substances such as reactive oxygen species which may affect the periodontal tissue structures directly or indirectly ^{18,20,21}.

Pro inflammatory cytokines:

- 1. Interleukins which secreted by the human adipose tissue especially from the abdominal fat than subcutaneous fat. Elevated levels of adipose tissue related interleukins usually associated with risk of lipolysis, cardiovascular events and weight gain, etc²².
- 2. TNF- α Obesity associated tumour necrosis factor- α is primarily secreted from macrophage cells which accumulated in the abdominal adipose tissue. Increased TNF- α might leads to increasing levels of insulin resistance, C-reactive peptide production in systemic inflammation. TNF- α is a most potent inhibitor of adiponectin, which is an important anti inflammatory adipokine²².

Adipokines:

- 1. Adiponectin: It is a circulating adipokine or hormone which secreted from the adipose tissue and involved in lipid and glucose metabolism. But Contrary to other types of adipokines, adiponectin levels are usually reduced in individuals with obesity, type2 DM or insulin resistance^{4,22}.
- 2. Leptin: It plays a most important role in regulation between energy intake, energy expenditure which includes appetite and metabolism. It's action similar to insulin in most of the ways. Leptin is usually present both within healthy and also in marginally inflammed gingiva but reduces in concentration levels when the adjacent probing depth increases^{22,23}.
- 3. Resistin: It causes insulin resistance in animal models. Recent evidence suggests that in human beings, resistin is more closely associated with inflammatory process than the insulin resistance³.
- 4. Plasminogen activator inhibitors-1: It generates agglutination of blood components and increases the risk of gingival inflammation and even ischemic vascular diseases. It eventually reduces the bloodflow to the periodontium of obese individuals, accelerates the development of Periodontal diseases^{24,25}.
- 5. Chemerin: It is usually associated with insulin resistance, adiposity, and degree of non alcoholic fatty liver. Additionally, it modulates the innate immune system through its binding to the orphan G-protein coupled receptor such as, chemokine like receptor-1. It also modulates chemotaxis of immature dentritic cells and macrophages²⁶.
- 6. Recent additions to this list of adipokines include visfatin, vaspin and Retinol binding protein-4 induces insulin resistance by interfering with insulin receptor substrate-1 adds to the list of plasma abnormalities that link obesity with development of T2D²⁶.

Reactive oxygen species: Even though reactive oxygen species are products of normal cellular metabolism, but overproduction of any reactive oxygen species induces damage by oxidizing DNA, lipids and proteins, which in turn increases the risk of gingival oxidative damage and progression of Periodontal diseases^{4,27}.

ASSOCIATION BETWEEN PERIODONTITIS AND OBESITY RELATED DIABETES:

The risk of developing Periodontitis is increased by threefold in candidates with diabetic when compared with non-diabetic individuals. The level of glycemic control such as HbA_{1c} , is of key importance in determining raise in risk status. For example, in the US National Health and Nutrition Examination Survey (NHANES) III, adults with HbA_{1c} level of >9% had a significantly higher prevalence of severe Periodontitis than those without diabetes after controlling for age, ethnicity, education, sex and smoking. Type 2 diabetes and decreased insulin sensitivity are related with the over production of advanced glycation end products (AGE), which induces the inflammatory cytokine production, and thats way it may progressing the inflammatory conditions such as Periodontitis^{8,9,11}.

ASSOCIATION BETWEEN PERIODONTITIS AND OBESITY RELATED CHRONIC DISEASES:

Obesity is usually accompanied by systemic inflammatory state, and raised levels of C-reactive protein, which is a marker of low grade inflammation, have been reported in obese individuals (Visser *et al.*1999, Rexrode *et al.*2003, Nazmi *et al.*2008). Pischon *et al.*(2007) suggested that the secretion of inflammatory cytokines by adipose tissue could be triggered by lipopolysacchride(LPS) of gram-negative periodontal bacteria, might leads to reduction in insulin sensitivity and hepatic dyslipidemia. These features would be enhanced in individuals with increased level of adipose tissue and would aggrevate the systemic inflammatory condition which might predisposing to the aggrevation or establishment of inflammatory conditions such as Periodontal diseases ^{13,28}.

SALIVARY PROTEOME MODIFICATIONS IN OBESE:

Range H *et al.* Suggested that, in whole saliva samples, SELDI-TOF-MS analysis detected eight putative markers. Among the total, Six markers were increased and identified in obese individuals versus controls (Albumin, α and β haemoglobin chains, α - defensins 1, 2 and 3). Alpha – defensins were less abundant in saliva of obese periodontal disease patients (36.47 \pm 19.84 μ A)

versus non-periodontitis obese patients ($43.44 \pm 30.34\mu A$), whereas α -defensins were more abundant in obese patients ($40.99 \pm 26.66\mu A$) versus controls ($27.1 \pm 23.98\mu A$). Alpha defensins may play a big role in gingival inflammatory conditions, and be involved in the higher speciality of obese patients to periodontal diseases²⁹.

BACTERIAL CONTRIBUTION TO OBESITY:

Goodson J.M *et al.*, Suggested three mechanisms by which oral bacteria can contribute to development of obesity³⁰.

- 1. Oral bacteria may contribute to increased metabolic efficiency, as suggested by infectobesity proponents.
- 2. Oral bacteria could increase weight gain by increasing appetite.
- 3. Oral bacteria redirect energy metabolism by facilitating insulin resistance through increasing levels of TNF- α or reducing levels of adiponectin.

ROLE OF A DENTIST:

A dentist can evaluate patients for their symptoms and signs of obesity-related diseases. Mathus-vliegen *et al.*, documented that obesity is related to several aspects of oral health. Dentists should have sufficient knowledge about the signs, symptoms and diagnostic tests for obesity, metabolic syndrome and other related conditions. Dentist should refer their obese periodontally affected patients for their weight reduction intervention procedures which includes diet therapy, pharmacotherapy, behavioural therapy and even surgical procedures ^{4,30}. So that they can have better control over progression of the periodontal inflammation. Recommendation of proper balanced diet which may includes anti-obesity food substances such as pomegranate etc. may not only reduce the chances of obesity and also the periodontal disease progression and leads to proper restore of oral and periodontal health³¹.

CONCLUSION:

Obesity usually has a negative feedback on a person's not only the oral and also the overall health. In patients, Promotion of healthy nutrition and adequate physical activity may help in general body resistance and also prevent or slow down the progression of periodontal disease conditions. Oral health care providers including dentists who can have an important role on treatment outcomes by analyzing the patients at high risk and addressing these concerns. The devastating general and oral health hazards of obesity suggest further research is necessary regarding the inter relationship among periodontal diseases, obesity and other chronic and systemic diseases. Studies should includes

the role of oral health providers who plays important role in management of dental problems and associated medical problems and even suggestions for life style modification etc.

REFERENCES:

- 1. Reilly RD, Boyle CA, Craig DC. Obesity and dentistry: A growing problem. Br Dent J 2009; 207:171-8.
- 2. Lalit kumar Mathur, Balaji Manohar, Rajesh Shankarapillai, *et al.* Obesity and Periodontitis: A clinical study. J Ind Soc Periodontol 2011;15(3):240-4.
- 3. N.Pischon, N.Heng. Obesity, inflammation, and periodontal disease. J Dent Res 2007; 86:400-9.
- 4. Preethe Paddmanabhan. Is there any link between cardiovascular disease, obesity and periodontal disease? J Dent Med Research 2013; 3(6):04-06.
- 5. Khader.Y.S, Bavv-adi H.A, Haroun T.F *et al.* The association between periodontal disease and Obesity among adults in Jordan. J Clin Periodontol 2009;36:18-24.
- 6. Ylostalo p, Suominen Taipale L, Reunanen A *et al*. Association between body weight and periodontal infection. J Clin Periodontol 2008;35:297-304.
- 7. Beatriz Bezerra, Enilson Sallum, Antonia Sallum. Obesity and periodontal disease: why suggest such relationship? An overview. Braz J Oral Sci. 2007;6(23):1420-2.
- 8. P. M. Preshaw, A. L. Alba, D. Herrera *et al.* Periodontitis and diabetes: a two-way relationship. Diabetologia. 2012; 55:21–31.
- 9. Stumvoll M, Goldstein BJ, Van Haeften TW,. Type 2 diabetes: principles of pathogenesis and therapy. Lancet.2005; 365:1333-46.
- 10. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet. 2005;365:1415-28.
- 11. Lindsay RS, Funahashi T, Hanson RL *et al.* Adiponectin and development of type 2 diabetes in the Pima Indian population. Lancet.2002; 360:57-8.
- 12. Zelkha SA, Frielich RW, Amar S. Periodonal innate immunal mechanisms relevant to atherosclerosis&obesity. Periodontol 2000 2010; 54(1):207-21.
- 13. Beck JD, Offenbacher S. Systemic effects of Periodontitis: epidemiology of periodontal disease and cardiovascular disease. J Periodontol. 2005; 76(11):2089-100.
- 14. Peter W. Lementowski, MD, and Stephen B. Zelicof, MD, PhD. Obesity and osteoarthritis. Am J Orthop. 2008; 37:148-151.

- 15. Magliocca KR, Helman JI. Obstructive sleep apnoea: diagnosis, medical management and dental implications. J Am Dent Assoc. 2005; 136:1121-9.
- 16. Socransky SS, Haffajee AD. The bacterial etiology of destructive periodontal disease: current concepts. J periodontal 1992; 63(4):322-31.
- 17. Toshiyuki Satio, Yoshihiro Shimazaki. Metabolic disorders related to obesity and periodontal disease. Periodontol 2000 2007; 43:254-66.
- 18. Ritchie CS. Obesity and periodontal disease. Periodontol 2000. 2007;44:154-63.
- 19. Roberto Franchini, Antonella Petri, Mario Migliari *et al.*. Poor oral hygiene and gingivitis are associated with obesity and overweight status in pediatric subjects. J Clin Periodontol 2011; 38(11):1021-8.
- 20. Joerg Meyle, Jose R Gonzales. Influence of systemic diseases as Periodontitis in children and adolescents. Periodontol 2000 2001;26:92-112.
- 21. Enricco Marchetti, Annalisa Monaco, Laura Procaccinin *et al.* Obesity and periodontal damage. Nutr Metab. 2012;9:88.Ritchie CS. Obesity and periodontal disease. Periodontol 2000. 2007; 44:154-63.
- 22. Emma O' Keefe. Periodontitis associated with obesity although the magnitude of association unclear. Evidence based dentistry 2012; 13(1):12-3.
- 23. Raul I Garcia, Michelle M Henshaw, Elizabeth A Krall. Relationship between periodontal disease and systemic health. Periodontol 2000 2001; 25:21-36.
- 24. Sunitha Jagannathachary, Dinesh Kamaraj. Obesity and periodontal disease. J Ind Soc Periodontol 2010; 14(2):96-100.
- 25. Saito T, Shimazaki Y, Logs T *et al.* A relationship between upper body obesity and periodontal disease. I Dent Res. 2001; 80(7):1631-6.
- 26. Robert J.Genco, Sara G. Grossi, Alex Ho *et al.* A proposed model linking inflammation to obesity, Diabetes, and periodontal infections. J periodontal 2005; 76:2075-84.
- 27. Christine, Seel, Ritchie. Nutrition, inflammation and periodontal disease. Nutrition 2003; 19:475-6.
- 28. Suvan J, D'Aiuto F, Moles DR *et al.* Association between overweight/obesity and Periodontitis in adults: A systemic review. Obes Rev 2011; 12:e 381-404.
- 29. Range H, Leger T, Huchon C *et al.* Salivary proteome modifications associated with Periodontitis in obese patients. J Clin Periodontol 2012; 39:799-806.

- 30. J.M.Goodson, D.Groppo, S.Halem, *et al.* Is obesity an oral bacterial disease? J Dent Res 2009; 88(6):519-23.
- 31. Elizabeth K Kaye. Nutrition, dietary guidelines and optimal periodontal health. Periodontol 2000, 2012; 58:93-110.