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Obesity and its association with chronic periodontitis: A cross-sectional study

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Abstract:

BACKGROUND: Relationship among periodontitis, obesity, and chronic diseases may be multidirectional. Inflammatory diseases such as 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 lipopolysaccharides from periodontal Gram-negative bacteria promotes hepatic dyslipidemia and decreases insulin sensitivity.

AIM: To study the association of obesity with the severity of periodontitis and to compare the blood glucose levels and plasma lipid profile in obese and non-obese subjects with chronic periodontitis.

METHODOLOGY: A total of 84 systemically healthy subjects, aged 30–60 years, were included in the study. Periodontal status of the subjects was assessed by recording Community Periodontal Index (CPI) and Gingival Index (Loe and Silness, 1963). Body mass index (BMI) and waist circumference (WC) were used as measures to assess obesity. The fasting plasma lipids level and fasting blood glucose level were measured. Data were analyzed by unpaired 't'test, Fisher's Exact test, and Chi-square test.

RESULTS: There was a statistically significant difference in Gingival Index and Community Periodontal Index between the obese and non-obese group. Total cholesterol, triglycerides, and low-density lipoprotein (LDL) levels showed significant difference between the two groups. There was no statistically significant difference in mean age, high-density lipoprotein (HDL) levels, and fasting blood glucose levels between the two groups.

CONCLUSION: The present study showed a positive association between obesity and chronic periodontitis. Further long-term studies involving larger sample size are required to confirm this association.

Keywords:

Body mass index, lipid profile, obesity, periodontitis, waist circumference

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Introduction

Obesity is one of the growing health problems around the world and also the most common underlying factor responsible for several systemic diseases. According to the Global Health Observatory of the WHO, worldwide, at least 2.8 million people die each year as a result of being overweight or obese. Adverse metabolic effects of overweight and obesity affect blood pressure, cholesterol, triglycerides (TGs), and insulin

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resistance. There is a steady increase in the risks of coronary heart disease, ischemic stroke, and type 2 diabetes mellitus with increasing body mass index (BMI).^[1]

It has been suggested that obesity is second to smoking as the strongest risk factor for inflammatory periodontal tissue destruction.^[2] The first report on the relationship between obesity and periodontal disease appeared in 1977 when Perlstein and Bissada observed histopathologic changes in the periodontium in hereditary obese Zucker rats. Using

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ligature-induced periodontitis, they found alveolar bone resorption to be greater in obese animals compared with non-obese rats.^[3] The association between obesity and periodontal disease was first time shown in Japanese individuals by Sato *et al.*^[4] The National Health and Nutrition Examination Survey (NHANES III) data had shown a positive correlation with BMI and severity of periodontal attachment loss and also the relationship is modulated by insulin resistance.^[5]

Recently, increased interest in the link between obesity and oral health has suggested that obesity is also associated with periodontitis.^[6-10]

Adipose tissue secretes hormones and cytokines that are intimately involved in inflammatory processes, suggesting that similar pathways may be involved in the pathophysiology of obesity, diabetes, and periodontitis.^[11]

These adipose tissue-derived cytokines and hormones can be considered to play a key role in the underlying biological mechanisms for obesity with periodontitis association. Fat tissue not only acts as a reservoir for TGs but is also responsible for the production of large amounts of adipokines or adipocytokines, which may modulate periodontitis.^[12]

This study was conducted to ascertain the association of obesity with the severity of periodontitis and to compare the blood glucose levels and plasma lipid profile in obese and non-obese subjects with chronic periodontitis.

Methodology

The data were collected from the subjects visiting the outpatient section of the Department of Periodontology, Yenepoya Dental College, Mangalore, India. The Institutional Ethical Committee approved the study protocol (YUEC 153/20/11/2013, date: 19.11.2013). All the participants were provided with verbal explanation of the nature of the study, and informed consent was obtained. The sample size was calculated at 80% of power and 0.1% significance level. In this cross-sectional study, the study population included both the genders and consisted of 84 systemically healthy subjects in the age group of 30-60 years that were further categorized into two groups comprising 42 in each group. According to the BMI classification for adult Asians,^[13] the subjects with BMI >25 kg/m² have been grouped into obese category (Group I) and those with BMI <24.9 kg/m² have been grouped into non-obese category (Group II).

• Group I (obese): Subjects with BMI ≥25 kg/m² with chronic periodontitis (probing depth >4 mm and/or attachment loss >3 mm in at least 30% teeth in the oral cavity).

• Group II (non-obese): Subjects with BMI ≤ 24.9 kg/m² with chronic periodontitis (probing depth >4 mm and/or attachment loss >3 mm in at least 30% teeth in the oral cavity).

Subjects having any systemic disease including diabetes mellitus or any other endocrine disorders, subjects taking drugs for hypercholesterolemia, smokers, subjects who have undergone surgery within 6 months, pregnant women or lactating women, or women taking oral contraceptive pills were excluded from the study. BMI was computed from body weight in kilograms divided by the square of height in meters.^[14] Waist circumference (WC) of all the subjects were recorded using a measuring tape. According to guidelines for weight classification in adult Asians, cutoffs for waist circumstance will now be 90 cm for Indian men and 80 cm for Indian women.^[13] Values above these were considered obese. Community Periodontal Index (CPI) and Gingival Index (GI) were used to assess the periodontal and gingival status, respectively.[15] The fasting plasma lipid level and fasting blood glucose level in all the 84 subjects were measured.

Fasting lipid profile was measured using the enzymatic method. The components of the lipid profile were total cholesterol (T.Ch), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL). Fasting blood glucose level was estimated using glucose oxidase-peroxidase method. Data were analyzed by unpaired *t*-test, Fisher's exact test, and Chi-square test using SPSS software version 17 (SPSS Inc., Version 17.0. Chicago, USA). A p -value of <0.001 was considered to be statistically significant.

Results

The mean age of the subjects was 46.95 ± 98 and 47.1 ± 8.042 years for non-obese and obese groups, respectively. In the present study, the mean Gingival Index score among the non-obese group (1.0811 ± 0.1973) was significantly less than that of the obese group (1.619 ± 0.4915) (P < 0.001) [Table 1]. The mean BMI was $30.571 \pm 4.429 \text{ kg/m}^2$, and mean WC was 94.857 ± 6.9232 cm in the obese group, while the mean BMI in the non-obese group was $23.206 \pm 1.306 \text{ kg/m}^2$ and WC was 82.2 ± 6.613 cm. A statistically significant difference between the two groups was observed using unpaired *t*-test, with higher BMI and WC in the obese group (P < 0.001) [Table 1]. The mean total cholesterol among the non-obese was 119.62 ± 31.385 and among the obese subjects was 200.36 ± 28.508; mean TG level among the non-obese was 93.64 ± 23.147 and among the obese was 216.74 ± 87.314 ; mean LDL level among the nonobese was 73.79 ± 27.224 and among the obese was 119.21 ± 27.285 ; and mean VLDL level among the nonobese was 23.38 \pm 6.424 and among the obese was 33.45 \pm 13.923, which was statistically significant (*P* < 0.001) [Table 2].

There was no statistically significant difference in the mean age among the two groups (P = 0.942) [Table 1]. The mean fasting blood glucose among the non-obese was 89.81 ± 13.199 and among the obese was 83.4 ± 8.673 , which was not statistically significant (P = 0.011) [Table 2]. The mean HDL among the non-obese was 33.76 ± 7.628 and among the obese was 32.29 ± 11.066 , which was not statistically significant (P = 0.479) [Table 2]. CPI score showed statistically significant differences between the non-obese and obese group. Chi-square value for probing depth (PD) [Table 3] and loss of attachment (LOA) components between the two groups was 16.513 and 16.429, respectively, which was statistically significant (P < 0.001) for both the parameters [Table 4].

Discussion

This cross-sectional study was conducted on subjects aged 30–60 years of both genders to evaluate the relationship between obesity and chronic periodontitis. The present study showed a higher Gingival Index in the obese group compared to the non-obese group [Table 1]. There was higher probing depth and LOA observed in the obese group compared to the non-obese group, which was statistically significant [Tables 3 and 4].

Certain studies have analyzed the association between overweight/obesity (categorized using BMI) and periodontitis using clinical attachment loss and bleeding on probing (BOP) components in a cross-sectional design which showed that BMI was positively related to BOP. The results obtained in previous studies are similar to the results of our study.^[7,8,16]

The mean age between the obese and non-obese groups showed no statistically significant difference [Table 1]. However, Al-Zahrani *et al.*^[8] observed a higher prevalence of obesity among the younger age group people, which is in contrast to the present study.

Our study showed no statistically significant difference in the fasting blood glucose levels between the two groups [Table 2]. In fasting lipid profile, the total cholesterol, TGs, LDL level, and VLDL level were significantly higher in the obese group, which is comparable with previously reported studies^[4,17,18] [Table 2].

According to Fentoğlu *et al.*,^[19] the serum levels of total cholesterol, LDL-cholesterol, and TGs have been observed to be higher in periodontal patients;

Table 1: Mean values of age, gingival index, waist circumference, and body mass index in the nonobese and obese groups

obcsc groups							
	GROUP	n	Mean	Std.Deviation	t	df	р
AGE	NON OBESE	42	46.95	9.8	-0.073	82	0.942
	OBESE	42	47.1	8.042			
GINGIVAL INDEX	NON OBESE	42	1.08119	0.197314	-6.581	53.88	<0.001
	OBESE	42	1.619048	0.491507			
WAIST	NON OBESE	42	82.2	6.163	-8.85	82	<0.001
CIRCUMFERENCE	OBESE	42	94.857	6.9232			
BMI (kg/m ²)	NON OBESE	42	23.2064	1.36002	-10.3	48.664	<0.001
	OBESE	42	30.5714	4.42908			

BMI=Body mass index

Table 2: Mean values of fasting blood sugar, total cholesterol, triglycerides, high-density lipoprotein, low-density lipoprotein in non-obese and obese groups

	GROUP	n	Mean	Std.Deviation	t	df	р
FASTING BLOOD	NON OBESE	42	89.81	13.199	2.628	70.843	0.011
SUGAR	OBESE	42	83.4	8.673			
T. Ch.	NON OBESE	42	119.62	31.385	-12.34	82	<0.001
	OBESE	42	200.36	28.508			
TGs	NON OBESE	42	93.64	23.147	-8.825	46.726	<0.001
	OBESE	42	216.74	87.384			
HDL	NON OBESE	42	33.76	7.628	0.712	72.787	0.479
	OBESE	42	32.29	11.066			
LDL	NON OBESE	42	73.79	27.224	-7.638	82	<0.001
	OBESE	42	119.21	27.285			
VLDL	NON OBESE	42	23.38	6.424	-4.257	82	<0.001
	OBESE	42	33.45	13.923			

T.Ch=Total cholesterol, TGs=Triglycerides, HDL=High-density lipoprotein, LDL=Low-density lipoprotein, VLDL=Very low-density lipoprotein

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Table 3: Comparison of Probing Depth (PD)
component of Community Periodontal Index between
non-obese and obese groups

		GROUP		TOTAL	
		NON OBESE	OBESE		
PD	1 count	4	0	4	
	% within group	9.5%	0.0%	4.8%	
	2 count	20	11	31	
	% within group	47.6%	26.2%	36.9%	
	3 count	18	22	40	
	% within group	42.9%	52.4%	47.6%	
	4 count	0	9	9	
	% within group	0.0%	21.4%	10.7%	
Total	count	42	42	84	
	% within group	100.0%	100.0%	100.0%	
Chi-squ	are Tests				
		Value	Exact Sig	. (2- sided)	
Fisher's Exact Test		16.513	0.001		
N of Valid Cases		84			

Table 4: Comparison of loss of attachment (LOA) component of Community Periodontal Index between non-obese and obese groups

		GROUP		TOTAL
		NON OBESE	OBESE	
LOA	0 count	8	0	8
	% within group	19.0%	0.0%	9.5%
	1 count	28	23	51
	% within group	66.7%	54.8%	60.7%
	2 count	6	14	20
	% within group	14.3%	33.3%	23.8%
	3 count	0	3	3
	% within group	0.0%	7.1%	3.6%
	4 count	0	2	2
	% within group	0.0%	4.8%	2.4%
Total	count	42	42	84
	% within group	100.0%	100.0%	100.0%
Chi-squ	are Tests			
		Value	Exact Sig.	(2- sided)
Fisher's Exact Test		16.429	0.0	01
N of Valid Cases		84		

periodontitis may also be a risk factor for hyperlipidemia. These findings indicate that obesity and periodontitis can, independently or jointly, alter the local and systemic levels of adipocytokines, leading to the vicious cycle of events seen in these individuals.

However, studies have indicated that fat distribution pattern plays a crucial role in the association with periodontitis.^[20] BMI is highly correlated with fat mass, morbidity, and mortality and, therefore, sufficiently reflects obesity-related disease risk in a wide range of populations; however, there are some limitations. BMI describes relative weight for height and is significantly correlated with total body fat content. More importantly, it does not assess body fat distribution. WC is positively correlated with abdominal fat content. It provides a clinically acceptable measurement for assessing a patient's abdominal fat content before and during weight loss treatment.

In the present study, both the anthropometric parameters (BMI and WC) were measured to remove any ambiguity in comparing the effects of central adiposity to overall adiposity on the risk of periodontal disease. The results of the anthropometric parameters in this study are similar to those reported in previous studies^[8,17,20] [Table 1].

The mechanism by which obesity may affect the periodontium has not yet been determined. Studies in animals and humans suggest that overnutrition is associated with immune competence alterations.^[10] The adipose tissue is a complex and metabolically active endocrine organ that secretes numerous immunomodulatory factors such as adipocytokines, tumor necrosis factor-alpha (TNF- α), leptin, adiponectin, and resistin; it can also affect the periodontal response or can also be affected during periodontal infections.^[21,22]

Due to the predominant role of Gram-negative bacteria, the ulcerated pocket epithelium becomes a chronic source of bacterial products as well as locally produced inflammatory mediators. As a consequence of the high vascularity, the inflamed periodontium may act as an endocrine-like source of inflammatory mediators (such as TNF- α , interleukin [IL] IL-6, and IL-1) which are significant in periodontal inflammation and may also influence glucose and lipid metabolism.^[18]

Elevated levels of TNF- α are a well-known risk factor for destruction due to periodontal disease and contribute to the onset of destructive periodontal disease. Previous studies on human subjects reported on the elevated levels of TNF- α in gingival crevicular fluid of obese individuals.^[23]

Furthermore, the plasminogen-activating system has been shown to play an important role in gingival inflammation. Plasminogen activator inhibitor-1 has an increased gene expression in visceral fat and induces agglutination of blood. This, in turn, may decrease periodontal blood flow in obese individuals, promoting initiation of periodontitis. All these could be the probable mechanisms underlying the periodontitis–dyslipidemia relationship in obese individuals.^[9,24]

The potential implications of the present study would help both oral health providers and patients in screening for periodontitis and obesity, which could have a significant impact on obesity prevention and

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management, as many individuals who seek dental care do not routinely seek medical care.

Conclusion

The results of this study showed a significant positive association between the gingival and periodontal findings and anthropological parameters among obese and non-obese groups. Further long-term, well-designed prospective studies involving larger sample size are required to validate the relationships between periodontal health and systemic health.

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Conflicts of interest

There are no conflicts of interest.

References

- World Health Organization. Global Health Observatory (GHO) Data. Geneva: World Health Organization; 2008. Available from: http://www.who.int/gho/ncd/risk_factors/obesity_text/ en/. [Last accessed on 2018 Feb 03].
- Nishida N, Tanaka M, Hayashi N, Nagata H, Takeshita T, Nakayama K, *et al.* Determination of smoking and obesity as periodontitis risks using the classification and regression tree method. J Periodontol 2005;76:923-8.
- Perlstein MI, Bissada NF. Influence of obesity and hypertension on the severity of periodontitis in rats. Oral Surg Oral Med Oral Pathol 1977;43:707-19.
- 4. Saito T, Shimazaki Y, Sakamoto M. Obesity and periodontitis. N Engl J Med 1998;339:482-3.
- Genco RJ, Grossi SG, Ho A, Nishimura F, Murayama Y. A proposed model linking inflammation to obesity, diabetes, and periodontal infections. J Periodontol 2005;76 Suppl 11S: 2075-84.
- Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. J Dent Res 2001;80:1631-6.
- Saito T, Shimazaki Y, Kiyohara Y, Kato I, Kubo M, Iida M, et al. Relationship between obesity, glucose tolerance, and periodontal disease in Japanese women: The Hisayama study. J Periodontal Res 2005;40:346-53.
- 8. Al-Zahrani MS, Bissada NF, Borawskit EA. Obesity and

periodontal disease in young, middle-aged, and older adults. J Periodontol 2003;74:610-5.

- Wood N, Johnson RB, Streckfus CF. Comparison of body composition and periodontal disease using nutritional assessment techniques: Third national health and nutrition examination survey (NHANES III). J Clin Periodontol 2003;30:321-7.
- Dalla Vecchia CF, Susin C, Rösing CK, Oppermann RV, Albandar JM. Overweight and obesity as risk indicators for periodontitis in adults. J Periodontol 2005;76:1721-8.
- Pischon N, Heng N, Bernimoulin JP, Kleber BM, Willich SN, Pischon T. Obesity, inflammation, and periodontal disease. J Dent Res 2007;86:400-9.
- 12. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. J Clin Endocrinol Metab 2004;89:2548-56.
- Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, et al. Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. J Assoc Physicians India 2009;57:163-70.
- Beck JD, Offenbacher S. Systemic effects of periodontitis: Epidemiology of periodontal disease and cardiovascular disease. J Periodontol 2005;76:2089-100.
- 15. Löe H. The gingival index, the plaque index and the retention index systems. J Periodontol 1967;38 (6) Suppl:610-6.
- Kongstad J, Hvidtfeldt UA, Grønbaek M, Stoltze K, Holmstrup P. The relationship between body mass index and periodontitis in the Copenhagen city heart study. J Periodontol 2009;80:1246-53.
- Manjunath NS. Comparison of plasma lipid and blood glucose level in obese and non obese patients with moderate periodontitis. Int J Contemp Dent 2012;3:63-7.
- Grossi SG, Genco RJ. Periodontal disease and diabetes mellitus: A two-way relationship. Ann Periodontol 1998;3:51-61.
- Fentoglu O, Oz G, Tasdelen P, Uskun E, Aykac Y, Bozkurt Y. Periodontal status in subjects with hyperlipidemia. J Periodontol. 2009;80:267–73.
- Suvan J, Petrie A, Moles DR, Nibali L, Patel K, Darbar U, *et al.* Body mass index as a predictive factor of periodontal therapy outcomes. J Dent Res 2014;93:49-54.
- 21. Ritchie CS. Obesity and periodontal disease. Periodontol 2000 2007;44:154-63.
- Sun WL, Chen LL, Zhang SZ, Wu YM, Ren YZ, Qin GM. Inflammatory cytokines, adiponectin, insulin resistance and metabolic control after periodontal intervention in patients with type 2 diabetes and chronic periodontitis. Intern Med 2011;50:1569-74.
- Lundin M, Yucel-Lindberg T, Dahllöf G, Marcus C, Modéer T. Correlation between TNFalpha in gingival crevicular fluid and body mass index in obese subjects. Acta Odontol Scand 2004;62:273-7.
- 24. Shimomura I, Funahashi T, Takahashi M, Maeda K, Kotani K, Nakamura T, *et al.* Enhanced expression of PAI-1 in visceral fat: Possible contributor to vascular disease in obesity. Nat Med 1996;2:800-3.