Relation between Triglyceride Levels and Body Mass Index with respect to Periodontitis in Gulbarga population in India

V Patil, P Deshpande, S Desai

Citation

V Patil, P Deshpande, S Desai. *Relation between Triglyceride Levels and Body Mass Index with respect to Periodontitis in Gulbarga population in India*. The Internet Journal of Dental Science. 2009 Volume 8 Number 2.

Abstract

Aim: The purpose of this study was to correlate Triglyceride level and periodontal disease status in normal weight and overweight/ obese individuals. Materials and Methods: A total of sixty patients diagnosed for chronic generalized periodontitis were included in the study, out of which thirty patients were in normal weight group and thirty were in over weight group. Weight (in kilograms) and height (in meters) were measured. Body Mass Index (BMI) was calculated by dividing the individual's body weight by the square of his/her height and was accordingly divided into two groups: Group I- BMI < 25 (normal weight) and Group II- BMI ≥ 25 (overweight). Periodontal status was recorded using Community Periodontal Index of Treatment Needs (CPITN) Index. Twelve hours fasting venous blood was drawn and sent to biochemistry department for assessment of Triglyceride levels. Mann Whitney U test was used to correlate BMI and CPITN score, Students't' test was used to correlate Triglyceride and BMI, and Karl Pearsons correlation coefficient was used to correlate CPITN and Triglyceride levels. Data were analysed.Results: A significant correlation between BMI and mean CPITN scores at the confidence interval of 95%, and also a positive correlation between mean Triglyceride levels and CPITN scores were found.Conclusion: Patients with increased BMI and increased Triglyceride levels have significantly higher periodontal destruction incidence. There was a positive correlation between BMI, Triglycerides and Periodontal destruction.

INTRODUCTION

Periodontitis is an inflammatory disease resulting from the infection and interaction of specific subgingival bacterial species with components of the host immune response, influenced by many systemic factors in disease. Systemic factors play a major role in the pathogenesis and progression of periodontal disease. Many systemic factors have been implicated for periodontal disease like diabetes mellitus, obesity and coronary heart diseases (1). Overweight is recently considered as one of the risk factor for periodontal disease. There are many studies showing significant correlation between overweight and prevalence of periodontitis (2-4). Clarke et al. (1995) proposed a model in which they focused on personal risk factors like smoking, alcohol consumption and overweight/ obesity and concluded that obesity can act as risk factor for periodontal disease (5). Robert et al. (2005) also stated that increased BMI is related to severe periodontal attachment loss (6).

It has also been shown that lipid levels are directly associated with extent of periodontal destruction (7). There are many studies showing correlation between hyper-triglyceridemia and periodontal health (8, 9). Kuramitsu et al. (2003) found that Porphyromonas gingivalis (Pg) in presence of lipid induces foaming of macrophages (10). Lipids may interact directly with the macrophage cell membrane interfering with membrane bound receptors and enzyme systems. Hence altering macrophage gene expression for essential polypeptide growth factors and pro-inflammatory cytokines: like Tumour Necrosis Factor-α and Interleukin 1-β (11, 12). These are believed to be associated with periodontal destruction (13, 14). The aim of the present study was to correlate Triglyceride levels and periodontal disease status in normal weight and overweight/ obese individuals.

MATERIALS AND METHODS

Sixty patients visiting the outpatient section, Department of Periodontics, H.K.E society's S. Nijalingappa Institute of Dental Sciences and Research, Gulbarga, Karnataka, India were screened and included in the study. Patients of both genders of age 18 years and above, having at least one site

with both a probing depth of > 4 mm and a clinical attachment loss of > 3 mm (to diagnose as Periodontitis) were included. Patient with nephrosis, diabetes mellitus, endocrine disturbances, patients who had previously undergone periodontal treatment, pregnant women, smokers, and patient taking any medication for hyperlipidemia were excluded from the study.

Body weight in kilogram and height in meter were recorded and BMI was calculated for each individual using the formula: body weight divided by the square of height (15). Patients were divided into two group, Group – I with thirty patients of normal weight (BMI < 25 for Normal weight) and Group-II with thirty patients of overweight/ obese (BMI > 25 for Overweight/ obese) depending on BMI decided by world health organization criteria (15).

Periodontal status was assessed by using Community Periodontal Index of Treatment Needs (CPITN), using a CPITN probe. All records were taken by single examiner. The highest CPITN score found among all examined sites were concluded as CPITN score for that individual.

Triglyceride levels were assessed using "Wako and the modification by Mc Gowan et al. (1983) (17) et al. method." In these methods Triglycerides is converted into quinoneimine dye. Quinoneimine dye is formed at a rate proportional to Triglyceride concentration in the serum. Since it is a coloured compound it can be detected and quantified by a colorimeter (16,17). Patients abstained from eating or drinking, except water for 12 hrs and three millilitres of venous blood was collected using a sterile disposable syringe and the blood sample was allowed to clot, serum was extracted and stored at 4°c till it was subjected to above mentioned Triglyceride estimation test.

STATISTICAL ANALYSIS

Mean and standard deviation were calculated for CPITN scores and tested at P-value of less than 0.05 for significance in both groups using Mann Whitney U test. Also mean and standard deviation of Triglyceride levels in each group was calculated and both groups were compared using student's t-test. The correlation between Triglyceride levels and CPITN scores were calculated using Karl Pearson's correlation co-efficient at a significance level of $P \leq 0.05$.

RESULTS

Mean CPITN score in the Group-I was 3.33 with a standard deviation of 0.48 whereas the mean CPITN score in Group-II was 3.66 with a standard deviation of 0.48 as shown in Table (1).

Figure 1

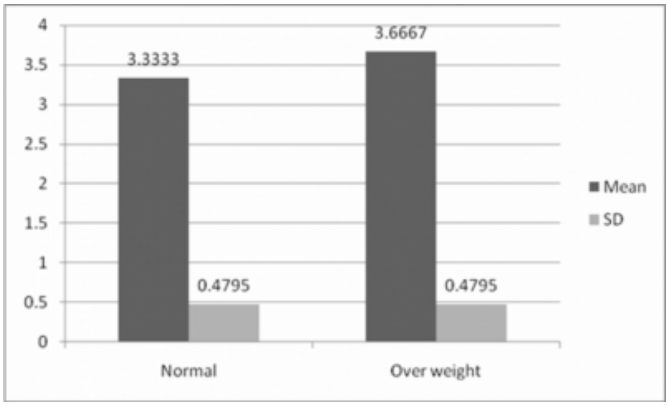
Table (1): Comparison of Group-I and Group-II with respect to CPITN scores by Mann Whitney U test

Group	n	Mean CPITN Score	SD	p-value	Significance
Group-I	30	3.3333	0.4795	0.0266	S
Group-II	30	3.6667	0.4795		

When mean CPITN score were compared between groups, it showed that mean CPITN score was significantly greater in Group-II than that in Group-I as shown in Figure- 1.

Figure 2

Figure- 1: Comparison of CPITN scores between Group-I and Group-II



Relationship between Triglyceride levels and CPITN scores was calculated using Karl Pearson correlation coefficient as shown in Table (2).

Figure 3

Table (2): Correlation between CPITN and Triglyceride scores (Karl Pearson's correlation coefficient)

Variables	CPITN Score	Triglyceride levels
Total CPITN	1.000	
Triglyceride levels	0.4541*	1.000

*Significant at 0.05% level of significance (p<0.05)

Mean CPITN score was significantly correlated to mean Triglyceride levels. The mean Triglyceride level in Group-I was 109.80 with a standard deviation of 71.93, and in Group- II, the mean Triglyceride level was 150.80 with standard deviation of 72.84 (Table 3).

Figure 4

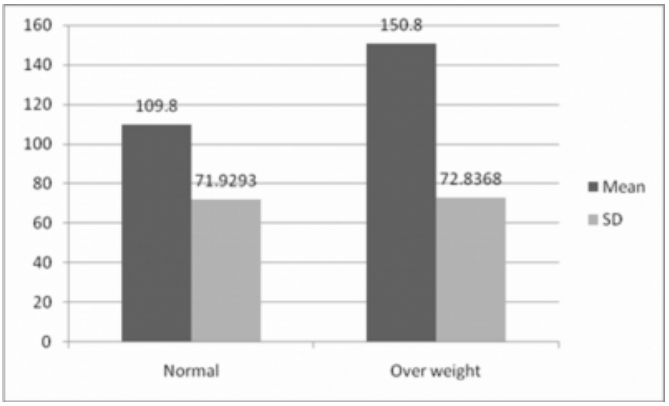
Table-3: Comparison of Group-I and Group-II BMI with respect to Triglyceride scores by students't'-test

Group	n	Mean Triglyceride Score	SD	p-value	Significance
Normal	30	109.8000	71.9293	0.0323	S
Over weight	30	150.8000	72.8368		

Triglycerides levels were compared between Group-I and Group-II and found that Group- II had significantly higher amount of Triglycerides levels than Group- I (Figure-2).

Figure 5

Figure-2: Comparison of Triglyceride levels between Group I and Group II.



DISCUSSION

Obesity is one of the recently observed risk factor for many chronic diseases like diabetes, coronary heart disease, osteoarthritis and hypertension (1). There are many studies showing that increase in the Body Mass is also one of the significant aggravating factors for periodontal disease (2-4,6). The present study also supplements the above finding that overweight individuals have increased periodontal destruction. Similarly, Al-Zahrani et al. (2003) in their study concluded that BMI and periodontitis were significantly correlated (4). There was one more study correlating smoking and obesity with periodontitis, concluding that obesity may act as an independent risk factor for periodontitis (18).

In our study we have taken patients in the age group of 18yrs and above, similar to the study done by Genco et al. (2005) (6) where they correlated obesity, diabetes and periodontal infection. In our study CPITN index was used because of its simplicity, speed and objectivity.

Lipid levels in blood have been seen as one of the important risk factors for periodontal destruction (2,4,8,9). In the present study there was a correlation between Triglyceride levels and periodontal destruction. As it has been stated earlier hyperlipidemia may trigger foaming of macrophages (10), leading to release of pre-inflammatory cytokines like Tumour Necrosis Factor-, Interleukin 1 (11,12). These cytokines are significantly related with periodontal destruction (13,14). It has been shown that P. gingivalis in presence of lipid induces foaming of macrophages (10). Hence Triglycerides may have priming effect on inflammatory cells leading to increased periodontal destruction. In our study we have seen a significant correlation between BMI and Triglyceride levels. Hence overweight may act as a risk factor for periodontal destruction through the pathway of increased Triglyceride levels.

CONCLUSION

To conclude the present study demonstrates a positive correlation between BMI and periodontal destruction as well as a positive correlation between Triglyceride levels in blood and periodontal destruction. A positive correlation between Triglyceride levels and BMI was also shown; hence it may be one of the pathways of periodontal destruction.

References

1. Kopelman PG. Obesity as a medical problem. Nature 2000 Apr 6; 404(6778):635-43.
2. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relation between upper body obesity and periodontitis. J Dent Res 2001; 80(7): 1631- 1636.
3. Wood N, Johnson RB, Steckfus CF. Comparison of body composition and periodontal disease using nutritional assessment techniques. Third National Health and Nutritional Survey (NHANES-III). J Clin Periodontol 2003 Apr; 30(4): 321- 327.
4. Al-Zaharani MS, Bissada NF, Borawskit EA. Obesity and periodontal disease in young, middle-aged, and older adults. J Periodontol 2003 May; 74(5): 610- 615.
5. Clarke NG, Hirsch RS. Personal risk factors for generalized periodontitis. J Clin Periodontol 1995 Feb; 22(2): 136- 145.
6. Genco RJ, Grossi SG, Alex H, F Nishimura and Murayama Y. A proposed model linking inflammation to obesity, diabetes, and periodontal infections. J Periodontol 2005; 76(11s): 2075- 2084.
7. Pussinen PJ, Vilkuna- RantianenT, Alfthean G. Severe periodontitis enhances macrophages activation in increased serum lipopolysaccharides. Arterioscler Thromb Vase Biol 2004; 24: 2174- 2180.
8. Thomas CI, Jackson RL, Ohlweiler DF, Ku G. Multiple lipid oxidation products in low density lipoprotein induced interleukin-1 release from human blood mononuclear cells. J Lipid Res 1994 Mar; 35(3): 417- 427.
9. Van der Poll T, Braxton CC, Coyle SM, Calvano SE, Hask CE, Lowry SF. Effect of hypertriglyceridemia on endotoxin response in humans. Infect Immun 1995 Sep; 63(9): 3396- 3400.
10. Kuramitsu HK, Kang IC, Qi M. Interaction of

Porphyromonas gingivalis with host cells: Implication for cardiovascular disease. J Periodontol 2003; 74: 85-89.
11. Doxey DL, Ng MC, Dill RE, Lacopino AM. Platelet derived growth factor levels in wounds of diabetic rats. Life Sci 1995 Aug; 57 (11): 1111- 1123.
12. Chu X, Newman J, Park B, Nara S, Ordonez G, Lacopino AM. In vitro alteration of macrophages phenotype and function by serum lipids. Cell Tissue Res 1999; 296(1): 331-337.
13. Stashenko P, Fujiyoshi P, Obernesser MS, Prostak L, Hafajee AD, Sacransky SS. Levels of interleukin 1 in tissue sites of active periodontal disease. J Clin periodontol 1991; 18: 548-554.
14. Heasman PA, Collins JG, Offenbacher S. Changes in crevicular fluid level of interleukin 1, Leukotriene B4, Prostanglandin E2, Thromboxane B2, and tumour necrosis factor in experimental gingivitis in humans. J Periodontal Res 1993; 28 (4): 241- 247.
15. National Institute of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in aults- the evidence report. National Institute of Health. Obes Res 1998; 6 (suppl): 51s- 209s (erratum: 1998; 6: 464).
16. Product Data Sheet, Triglyceride- G code no: 997-69801, WAKO Pure Chemical Industries Ltd., Dallas, TX.
17. McGowan MW, Artiss JD, Strandbergh DR, Zak B. A Peroxidase-Coupled Method for the Colorimetric Determination of Serum Triglycerides. Clin Chem 1983; 29 (3): 538.
18. Nishida N, Tanaka M, Hayashi N, Nagata H, Takeshita T, Nakayama K et al. Determination of Smoking and Obesity as Periodontitis Risk Using the Classification and Regression Tree Method. J Periodontol 2005; 76 (6): 923-928.

Author Information

Veena A. Patil, MDS
Head of Department, Department of Periodontics, HKE's SN Dental College

Pavan S. Deshpande, BDS
Post graduate Student, HKE's SN Dental College

Shrikar R. Desai, MDS
Associate professor, HKE's SN Dental College