Effects of non-surgical periodontal therapy on serum lipids and C-reactive protein among hyperlipidemic patients with chronic periodontitis

Ahmed Tawfig

Department of Preventive Dentistry, Division of Periodontics, Riyadh Colleges of Dentistry and Pharmacy, Riyadh, Saudi Arabia

Corresponding author (email: <drataufig@gmail.com>)

Dr. Ahmed Tawfig, Department of Preventive Dentistry, Division of Periodontics, Annamuthajiya Campus, Riyadh Colleges of Dentistry and Pharmacy, Post Box: 84891, Riyadh - 11681, Saudi Arabia.

Abstract

Aim: To evaluate the effect of non-surgical periodontal therapy on plasma lipid levels in hyperlipidemic patients with chronic periodontitis. Materials and Methods: After considering the inclusion and exclusion criteria, 30 hyperlipidemic patients with chronic periodontitis in the age group of 30-70 years, undergoing treatment in Ahmed Gasim Cardiac and Renal transplant Centre in north Sudan were recruited for the study. Patients were randomly assigned to the study and control groups. The study group received non-surgical periodontal therapy – oral hygiene instructions, scaling and root planing. The control group participants received only oral hygiene instructions. Lipid profile [total cholesterol (TC), low density lipoprotein (LDL), high density lipoprotein (HDL), triglycerides (TG)], C-reactive protein (CRP), and periodontal parameters [Plaque index (PI), Gingival index (GI), probing pocket depth (PD), and attachment loss (ATL)] were measured and compared at baseline and after 3 months of the respective intervention. Between-groups analysis was done using independent "t" test and within-group analysis was done using dependent "t" test. **Results:** At baseline, groups were comparable based on lipid profile and periodontal parameters. After 3 months, the control group showed significant decrease in the PI and GI scores while there was no significant change in the other parameters. However, the study group showed significant decrease in the LDL and CRP levels along with a significant decrease in PD, ATL, PI, and GI scores, compared to the baseline values. **Conclusion:** Local non-surgical periodontal therapy resulted in improved periodontal health, with significant decrease in the LDL and CRP levels in hyperlipidemic patients with chronic periodontitis. Hence, local non-surgical periodontal therapy may be considered as an adjunct in the control of hyperlipidemia, along with standard care.

Key words: Chronic periodontitis, C-reactive protein, non-surgical periodontal therapy, serum lipids

INTRODUCTION

Hyperlipidemia refers to an abnormal lipid profile characterized by elevated blood concentrations of total cholesterol (TC), triglycerides (TG), and low density

Access this article online				
Quick Response Code:	Website:			
	www.jispcd.org			
2012 2012 05 A 2012 2013 2014 05 A 2014	DOI:			
	10.4103/2231-0762.156524			

lipoprotein (LDL), along with decreased levels of high density lipoprotein (HDL).^[1,2] The underlying problem could be due to hereditary factors, or more commonly, it is an acquired condition. Moreover, hyperlipidemia is a strong risk factor for cardiovascular diseases.^[11] Cardiovascular disease of atherosclerotic origin is one of the main causes of sudden death in developed and developing countries.^[3] Higher serum lipid levels are involved in the pathogenesis of atherosclerosis – an inflammatory process initiated following the focal accumulation of lipids in the arterial intima.^[4] However, it has been suggested that infections may be responsible for accelerated development of atherosclerosis.^[5] Recently, the European Federation of Periodontology and the American Academy of Periodontology have categorized periodontitis as a chronic multifactorial inflammatory disease caused by microorganisms, characterized by progressive destruction of the tooth supporting apparatus leading to tooth loss. Periodontitis is a major public health issue as it reduces the quality of life; it causes tooth loss, masticatory dysfunction, impaired aesthetics, impaired general health, and also contributes to escalating dental costs. Periodontitis disproportionately affects certain groups: It is more prevalent and severe in (i) socially disadvantaged and specific ethnic groups and (ii) in smokers, diabetics, and the obese.^[6]

Periodontal diseases are mainly caused by gram-negative, anaerobic bacteria that induce local and systemic elevations of pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-a), IL-6.^[7,8] interleukin (IL)-1b, and Increased inflammatory cytokines mobilize lipids from the liver and adipose tissue.^[9]

Two-way relationships exist between periodontal disease and hyperlipidemia. Several studies have pointed out that the subjects with periodontal disease have increased serum levels of TC, LDL, and TG, when compared to the subjects with healthy periodontium.^[10-14] Likewise, patients diagnosed with hyperlipidemia have shown significantly higher levels of periodontal disease indicators than the control subjects with a normal metabolic status.^[15-19] Therefore, this interrelationship between periodontitis and hyperlipidemia provides an example of a systemic disease predisposing to oral infection, and once the oral infection is established, it exacerbates the systemic disease.^[20]

C-reactive protein (CRP) is an acute phase reactant whose blood levels rise as a non-specific response to infections and non-infectious inflammatory processes.^[21] Measurement of CRP in the blood helps in the evaluation of the amount of injury to the body tissues. The level of CRP in blood is positively correlated with an increased probability of cardiovascular disease.^[22] Available evidence indicates that periodontal disease severity correlates with increased levels of circulating CRP.^[23,24]

Studies from different parts of the world have suggested that the control of periodontal infection can lead to decrease in blood lipid and CRP levels among hyperlipidemic patients with periodontitis.^[25-27] However, until now, there is no reported data on the effect of non-surgical periodontal therapy on the blood lipid profile and CRP level of hyperlipidemic patients on antilipidemic therapy. Hence, we conceived the present study to evaluate the effect of local non-surgical periodontal therapy on the plasma lipid profile and CRP levels among hyperlipidemic patients who have chronic periodontitis.

MATERIALS AND METHODS

Ethical clearance

The study protocol was approved by the Sudan Medical Specialization Board (SMSB), the Federal Ministry of Health (FMoH), and Ahmed Gasim Cardiac and Renal transplant Centre (AGCRC).

Subjects

The study was conducted at the AGCRC, Khartoum, North Sudan. We screened the AGCRC records for hyperlipidemic patients on anti-hyperlipidemia therapy (statin) and with chronic periodontitis [pocket depth (PD) ≥ 4 mm], who were within 30–70 years of age and were of either gender, and identified 38 subjects. Our exclusion criteria were as follows: Any known systemic disease other than heart disease, smoking history, pregnancy, any periodontal therapy during the last 6 months, and antibiotic therapy during the last 3 months. Eight patients were (diabetic- 4, antibiotic therapy- 2, and smokers- 2) excluded from the study based on our exclusion criteria. Thirty patients (females- 11, males- 19) who fulfilled the inclusion/exclusion criteria were invited to participate in the study. Study participants were informed about the objectives and methodology of the study and a written informed consent was obtained from each study subject [Figure 1].

Periodontal parameters

A single trained examiner (ATG) conducted all the oral examinations. The periodontal parameters of each participant were evaluated at six sites (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, and disto-lingual). We selected tooth numbers 16, 21, 24, 36, 41, and 44 to achieve the same. If an index tooth was absent, a tooth nearest in the sextant was examined. If all the teeth of the sextant were missing, that sextant was excluded. Teeth exhibiting extensive decayed crowns or un erupted teeth were also excluded from the examination.

We recorded plaque index (PI) by Silness and Loe method,^[28] gingival index (GI) by Loe and Silness

Tawfig: Effects of non-surgical periodontal therapy in hyperlipidemic patients with periodontitis

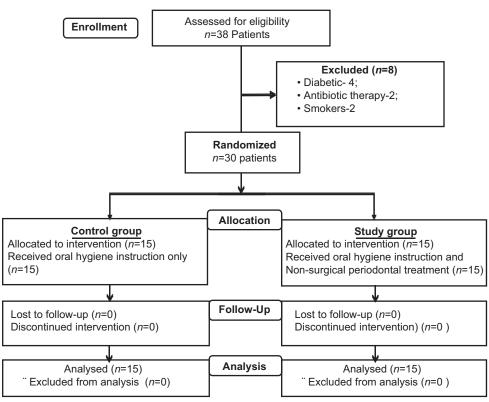


Figure 1: Flow chart of trial phases

method,^[29] PD, attachment loss (ATL) by Glavind and Loe method,^[30] and the frequency of tooth brushing (FTB). All assessments were performed using Williams's periodontal probe. PD was measured in millimetres as the distance between the gingival margin and the base of the periodontal pocket. ATL was also measured in millimetres as the distance between the cemento-enamel junction and the base of the gingival sulcus.

Lipid parameters

Fasting (overnight 12 h) blood sample (2.5 ml) was collected from the antecubital vein and stored in sodium heparin solution containers. Then, the whole blood samples were centrifuged at a moderate speed of 3000 RPM for 3-5 min using digital centrifuge (high-performance centrifuge 5417 R; Eppendorf AG, Hamburg, Germany). Approximately 1 ml of separated plasma was collected and placed in appropriately labelled Eppendorf tubes. The separated plasma was then treated with specific reagents (Biosystems lipid profile reagents A15/A25°; Biosystems S. A., Barcelona, Spain) and analyzed by enzymatic method to determine the serum lipid profile (TC, TG, LDL, HDL) and CRP using a digital spectrophotometer (SM11250-33 Thermolyne/Barnstead, Dubuque, Iowa, USA). Manufacturer recommended cut-off values of TC (\geq 200 mg/dl), LDL (\geq 130 mg/dl), HDL (\leq 35 mg/dl), and TG (\geq 150 mg/dl) were used to identify abnormal lipid profile among the study participants.

Based on the periodontal examination and serum lipid levels, participants were randomly divided (15 in each group) into study group and control group. Study group participants were given oral hygiene instructions and were provided a non-surgical periodontal therapy consisting of scaling and root planing, whereas the control group participants received only oral hygiene instructions. Oral hygiene instructions were reinforced after 4 weeks to both the groups. The blood samples were obtained at baseline and after 3 months.

Statistical analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (PASW SPSS 18). Subject characteristics were summarized using descriptive statistics (frequencies, percentages, means, and standard deviations). Independent samples "t" test was applied to compare the means of biochemical and periodontal parameters between the study and control groups both before and after intervention. The level of statistical significance was set at $P \leq 0.05$.

RESULTS

Majority of the participants in the study group were males (73.3%), as compared to control group (53.3%). Mean age of the study and control group participants were 56 \pm 12.3 and 51.5 \pm 10.9 years, respectively. Socio-demographic data of the participants are shown in Table 1.

At baseline, the groups were comparable based on periodontal status (PI, GI, FTB, PD, and ATL) and lipid parameters (LDL, HDL, TG, TC and CRP) as shown in Tables 2 and 3, respectively.

In the control group, the mean PI score and GI score reduced significantly following oral hygiene instructions as compared to the baseline scores. However, no such significant differences were observed in the mean scores of FTB, PD, and ATL [Table 4] and lipid profile [Table 5]. The study group showed a significant decrease in mean PI, GI, PD, and ATL compared to the baseline scores after non-surgical periodontal therapy [Table 4]. Similarly, a significant decrease in the mean values of LDL and CRP levels compared to baseline and after 3 months was observed. There were no statistically significant changes in the mean values of TC, TG, and HDL [Table 5].

DISCUSSION

It is well recognized now that periodontal disease produces numerous changes in systemic health by altering the blood biochemistry with an increase in inflammatory mediators such as CRP and lipids. Moreover, transient and recurrent bacteraemia caused by periodontal infection induces local and systemic inflammatory response, leading to changes in the whole body. These factors may partly explain the probable association between periodontitis and the susceptibility to certain systemic diseases, such as hyperlipidemia, and increased risk of cardiovascular disease, which affects many people globally.^[31-33]

Studies conducted in many places have assessed the effect of periodontal therapy on serum lipids and lipoprotein-related inflammatory mediators and suggested that the treatment of periodontal disease has positive effects on lipid metabolism.^[20,25,27,34-36] But none of the studies reported were on non-surgical periodontal therapy. Hence, the present study was undertaken to evaluate the effect of non-surgical periodontal therapy for periodontitis on plasma lipid parameters in hyperlipidemic patients on antilipemic medication.

Table 1: Socio-demographic characteristics of the
study and control group participant

Variables	Study group		Control		Total	
			gro	group		
	No.	%	No.	%	No.	%
Gender						
Male	11	73.3	8	53.3	19	63.3
Female	4	26.7	7	46.7	11	36.7
Total	15	100	15	100	30	100
Age group (years)						
33-37	1	6.7	2	13.3	3	13.3
38-42	2	13.3	4	26.7	6	16.7
43-47	1	6.7	2	13.3	3	16.7
48-52	3	20	2	13.3	5	6.7
53-57	0	0	1	6.7	1	6.7
58-62	1	6.7	4	26.7	5	36.7
63-70	7	46.7	0	0	7	46.7
Total	15	100	15	100	30	100
Marital status						
Married	13	86.7	14	93.3	27	90
Single	2	13.3	0	0	2	6.7
Widowed	0	0	1	6.7	1	3.3
Divorced	0	0	0	0	0	0
Total	15	100	15	100	30	100
Occupation						
Farmer	3	20	2	13.3	5	16.7
Business	2	13.3	3	20	5	16.7
Merchant	1	6.7	0	0	1	3.3
Free worker	2	13.3	3	20	5	16.7
Housewife	1	6.7	5	33.3	6	20
Retired	1	6.7	2	13.3	3	10
Worker	5	33.3	0	0	5	16.7
Total	15	100	15	100	30	100

Table 2: Periodontal status of the study andcontrol groups at baseline					
Variables Baseline periodontal status P					
	Control group Mean±SD	Study group Mean±SD			
PI	1.62 ± 0.50	1.62 ± 0.45	1.000		
FTB	1.20 ± 0.94	1.40 ± 1.06	0.588		
PD (mm)	3.13 ± 0.62	3.34 ± 0.40	0.263		
GI	1.74 ± 0.40	1.77 ± 0.44	0.872		
ATL (mm)	4.11 ± 0.76	4.27 ± 0.52	0.514		

PI=Plaque index, FTB=Frequency of tooth brushing, PD=Pocket depth, GI=Gingival index, ATL=Attachment loss, SD=Standard deviation. Comparison between the groups was done using independent "t" test

In our study, all the patients were on antilipemic drug regimen statin (10–20 mg) with a good compliance as observed at the baseline. Therefore, no attempt was made to change the prescribed statin therapy and dosage. In this study, groups were comparable at baseline based on lipid profile and periodontal status. However, after 3 months, the control group showed a significant decrease in the PI and GI scores. This could be due to the effect of oral hygiene instructions provided at/during the study period. Hawthorne effect and pleiotropic anti-inflammatory effect of statin medication could have partly played a role in reducing gingival inflammation.^[37] A similar reduction in GI has been reported among hyperlipidemic patients on statin therapy.^[38] Compared to the baseline levels, after 3 months, the mean value of FTB increased, ATL decreased, and PD remained the same without any statistical significance. Similarly, in the control group, we observed a decrease in the mean values of LDL, HDL, TG, and CRP, whereas TC values increased without any statistical significance. These changes could be attributed to the drug therapy and improvement in the gingival status observed after 3 months.

Table 3: Lipid profile of the study and controlgroups at baseline				
Lipid profile	Baseline lip	oid profile	P	
	Control group	Study group		
	Mean±SD	Mean±SD		
HDL (mg/dl)	33.60 ± 9.97	33.13±6.66	0.881	
LDL (mg/dl)	169.47 ± 23.53	181.53 ± 18.11	0.127	
TG (mg/dl)	224.40 ± 36.91	194.40 ± 43.83	0.052	
TC (mg/dl)	203.40 ± 22.02	214.67 ± 17.76	0.134	
CRP (mg/l)	1.00 ± 0.000	1.00 ± 0.000	-	

HDL=High density lipoprotein, LDL=Low density lipoprotein, TG=Triglycerides, TC=Total cholesterol, CRP=C-reactive protein, SD=Standard deviation. Comparison between the groups was done using independent "t" test The study group showed highly statistically significant reductions in PI, GI, PD, and ATL after 3 months compared to the baseline values. This finding is in agreement with other studies reported earlier.^[20,25,26,34,35] As stated earlier, this reduction could be due to the pleiotropic anti-inflammatory effects of lipid-lowering drug along with the combined effects of scaling/root planing. Statins are 3-hydroxy-3-methylglutarylcoenzyme А (HMG CoA) reductase inhibitors and primarily approved as lipid-lowering agents to prevent cardiovascular events. Statins lower low density lipoprotein-C (LDL-C), but recent studies provide convincing evidence that statins also possess anti-inflammatory activity, independent of their lipid-lowering effects.^[39,40] In addition, potential pleiotropic effects of statins comprised immunomodulation, antioxidant effect, antithrombotic and endothelium stabilization actions, angiogenesis promotion effect, increased osteoblast differentiation effect, and bone formation inducing effects.^[41,42]

The results of the present study reveal that periodontal therapy led to a decrease in atheroma bolstering lipid profile among hyperlipidemic patients. This finding was similar to that reported by Oz *et al.*,^[27] Duan *et al.*,^[25] and Fentaglo *et al.*,^[26] who have reported the beneficial effects of periodontal therapy on the metabolic control of hypercholesterolemia. Past studies conducted among hyperlipidemic patients on statin therapy revealed a significant decrease in serum TC and LDL levels and

Table 4: Periodontal status at baseline and after periodontal therapy in the control and study groups							
Control group							
Variables	Baseline	APT	P value	Baseline	APT	Р	
	Mean±SD	Mean±SD		Mean±SD	Mean±SD		
PI	1.62 ± 0.50	1.17 ± 0.27	0.005	1.62 ± 0.45	1.06 ± 0.24	< 0.001	
FTB	1.20 ± 0.94	1.60 ± 0.63	0.183	1.40 ± 1.06	2.00 ± 0.65	0.072	
PD (mm)	3.13 ± 0.62	3.13 ± 0.60	1	3.34 ± 0.40	2.53 ± 0.40	< 0.001	
GI	1.74 ± 0.40	1.32 ± 0.30	0.003	1.77 ± 0.44	1.03 ± 0.22	< 0.001	
ATL (mm)	4.11 ± 0.76	4.10 ± 0.74	0.976	4.27 ± 0.52	$3.38 {\pm} 0.57$	< 0.001	

APT=After periodontal therapy, PI=Plaque index, FTB=Frequency of tooth brushing, PD=Pocket depth, GI=Gingival index, ATL=Attachment loss, SD=Standard deviation. Within-group analysis was done using dependent "t" test

Table 5: Lipid protile and ()	'DD at bacaling and attar pariodontal	I therapy in the control and study groups
	NF at baseline and alter benoutlina	

Control group				Study group		
Lipid profile	Baseline	APT	P	Baseline	APT	Р
	Mean±SD	Mean±SD		Mean±SD	Mean±SD	
HDL (mg/dl)	33.60 ± 9.97	31.87±9.88	0.636	33.13 ± 6.66	36.40±9.61	0.289
LDL (mg/dl)	169.47 ± 23.53	176.47 ± 22.84	0.415	181.53 ± 18.11	166.60 ± 20.90	0.046
TG (mg/dl)	224.40 ± 36.91	214.20 ± 39.52	0.471	194.40 ± 43.83	176.53 ± 36.03	0.233
TC (mg/dl)	203.40 ± 22.02	209.00 ± 20.66	0.479	214.67 ± 17.76	203.00 ± 22.00	0.121
CRP (mg/l)	1.00 ± 0.00	0.93 ± 0.26	0.326	1.00 ± 0.00	0.07 ± 0.26	< 0.001

HDL=High density lipoprotein, LDL=Low density lipoprotein, TG=Triglycerides, TC=Total cholesterol, CRP=C-reactive protein, SD=Standard deviation. Within-group analysis was done using dependent "t" test CRP 3 months after the completion of the non-surgical periodontal therapy.^[20,26,35] Similar significant reductions in LDL and CRP levels were observed in the present study.

Three types of assays have been developed for measuring CRP in serum or body fluids. These include: Conventional CRP (CRP), high-sensitivity CRP (hsCRP), and cardiac CRP (cCRP). Conventional CRP is indicated for use in the evaluation of infection, tissue injury, and inflammatory disorders. For conventional CRP assays, test values are considered clinically significant at levels above 10 mg/l. In apparently healthy individuals, blood CRP levels are below 5 mg/l, while in various conditions, this threshold is often exceeded within 4-8 h after an acute inflammatory event, with CRP values reaching approximately 20–500 mg/l.^[43] However, the recent development of high-sensitivity assays for CRP (hsCRP) has permitted detection of mild elevation of CRP, even within the normal range. Reliable and fully automated high-sensitivity assays for CRP are now widely utilized. It was suggested that hsCRP level of <1, 1–3 and >3 mg/l be used to represent lower, moderate, and higher vascular risk for global risk prediction.[44]

In the present study, conventional CRP assay was utilized to detect the CRP level in the plasma due to the non-availability of the equipment that could detect hsCRP levels in the blood. A significant decrease in the level of CRP (from 1.00 ± 0.00 to 0.07 ± 0.26 mg/l, P < 0.001) was evident in the study group after non-surgical periodontal therapy, as compared to the control group. A study by Kamil et al. Showed a significant decrease in the serum levels of CRP (from 2.3 \pm 0.7 to 1.8 \pm 0.6 mg/dl) measured by an immunoturbidimetric high-sensitivity assay with a lower limit of detection of 0.03 mg/dl (Tina-quant CRP immunoturbidimetric assay performed on a Cobas Integra analyzer; Roche Diagnostics, GmbH, Mannheim, Germany).^[35] Similarly, a study by Mohan et al. showed a mean change of -0.29 ± 0.89 mg/dl in the serum CRP level among type 2 diabetes mellitus patients on utilizing quantitative turbidimetric immunoassay with CRP turbilatex kit,[45] and Mattila et al. reported a mean CRP decrease of 0.34 mg/l using sensitive sandwich enzyme immunoassay (UC CRP ELISA, Eucardio Laboratory) technique.^[46] By contrast, Ide et al. have reported no effect of periodontal therapy on the plasma CRP level, as measured by high-sensitivity latex anti-CRP monoclonal antibody kit with immunonephelometry (Dade Behring, Milton

Keynes, UK) on a Boehring nephelometer II analyser, among chronic periodontitis patients.^[47] Hence, it can be inferred from the present study that the non-surgical periodontal therapy results in significant reduction of plasma CRP level among hyperlipidemic patients.

The present study has many subject variables such as physical activity, food habits, socioeconomic conditions, obesity, age, stress, and lifestyle. These variables are difficult to control and may have influenced the results. Because of the small number of patients examined, data cannot be generalized for all patients affected by periodontitis. More investigations are needed for further exploration of the relationship between periodontitis, periodontal therapy, and lipid metabolism. Larger studies and clinical intervention trials are necessary to better define the periodontitis study participants in whom local infection causes significant systemic inflammation and to find whether these findings are true or confounded by other important factors like smoking, nutrition, socioeconomic status, or age.

CONCLUSION

Local non-surgical periodontal therapy resulted in improved periodontal health with a significant decrease in the LDL and CRP levels among hyperlipidemic patients having chronic periodontitis. Hence, local non-surgical periodontal therapy may be considered as an adjunct in the control of hyperlipidemia, along with standard care.

ACKNOWLEDGMENTS

I would like to express my profound and sincere gratitude to my Professor, Dr. Abdel Rahman M. Ramadan, who was highly instrumental in supporting me in this study. I also wish to extend my warmest regards to my colleague, Dr. Hani Eltayeb, and other colleagues working in Ahmed Gassim Cardiac Centre for their help and support during the study. Moreover, I am very thankful to all the study participants who had volunteered in this study.

REFERENCES

- 1. Nelson RH. Hyperlipidemia as a risk factor for cardiovascular disease. Prim Care 2013;40:195-211.
- Saxlin T, Suominen-Taipale L, Kattainen A, Marniemi J, Knuuttila M, Ylöstalo P. Association between serum lipid levels and periodontal infection. J Clin Periodontol 2008;35:1040-7.
- Mehra R. Global public health problem of sudden cardiac death. J Electrocardiol 2007;40(Suppl):S118-22.
- 4. Ross R. Atherosclerosis–an inflammatory disease. N Engl J Med 1999;340:115-26.
- 5. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med 2005;352:1685-95.

- Tonetti MS, Van Dyke TE; Working group 1 of the joint EFP/AAP workshop. Periodontitis and atherosclerotic cardiovascular disease: Consensus report of the Joint EFP/ AAP Workshop on Periodontitis and Systemic Diseases. J Clin Periodontol 2013;40(Suppl 14):S24-9.
- 7. Page RC. The role of inflammatory mediators in the pathogenesis of periodontal disease. J Periodontal Res 1991;26:230-42.
- 8. Page RC, Kornman KS. The pathogenesis of human periodontitis: An introduction. Periodontol 2000 1997;14:9-11.
- 9. Iacopino AM, Cutler CW. Pathophysiological relationships between periodontitis and systemic disease: Recent concepts involving serum lipids. J periodontol 2000;71:1375-84.
- Cutler CW, Shinedling EA, Nunn M, Jotwani R, Kim BO, Nares S, *et al.* Association between periodontitis and hyperlipidemia: Cause or effect? J Periodontol 1999;70:1429-34.
- Losche W, Karapetow F, Pohl A, Pohl C, Kocher T. Plasma lipid and blood glucose levels in patients with destructive periodontal disease. J Clin Periodontol 2000;27:537-41.
- Moeintaghavi A, Haerian-Ardakani A, Talebi-Ardakani M, Tabatabaie I. Hyperlipidemia in patients with periodontitis. J Contemp Dent Pract 2005;6:78-85.
- Penumarthy S, Penmetsa GS, Mannem S. Assessment of serum levels of triglycerides, total cholesterol, high-density lipoprotein cholesterol, and low-density lipoprotein cholesterol in periodontitis patients. J Indian Soc Periodontol 2013;17:30-5.
- 14. Sandi RM, Pol KG, Basavaraj P, Khuller N, Singh S. Association of serum cholesterol, triglyceride, high and low density lipoprotein (HDL and LDL) levels in chronic periodontitis subjects with risk for cardiovascular disease (CVD): A cross sectional study. J Clin Diagn Res 2014;8:214-6.
- 15. Awartani F, Atassi F. Evaluation of periodontal status in subjects with hyperlipidemia. J Contemp Dent Pract 2010;11:033-40.
- Fentoğlu O, Oz G, Taşdelen P, Uskun E, Aykaç Y, Bozkurt FY. Periodontal status in subjects with hyperlipidemia. J Periodontol 2009;80:267-73.
- 17. Lee JB, Yi HY, Bae KH. The association between periodontitis and dyslipidemia based on the Fourth Korea National Health and Nutrition Examination Survey. J Clin Periodontol 2013;40:437-42.
- Noack B, Jachmann I, Roscher S, Sieber L, Kopprasch S, Lück C, *et al.* Metabolic diseases and their possible link to risk indicators of periodontitis. J Periodontol 2000;71:898-903.
- Shivakumar T, Patil VA, Desai MH. Periodontal status in subjects with hyperlipidemia and determination of association between hyperlipidemia and periodontal health: A clinicobiochemical study. J Contemp Dent Pract 2013;14:785-9.
- 20. Fentoğlu O, Sözen T, Oz SG, Kale B, Sönmez Y, Tonguç MO, *et al.* Short-term effects of periodontal therapy as an adjunct to anti-lipemic treatment. Oral Dis 2010;16:648-54.
- Kushner I. The phenomenon of the acute phase response. Ann NY Acad Sci 1982;389:39-48.
- 22. Ridker PM. Clinical application of C-reactive protein for cardiovascular disease detection and prevention. Circulation 2003;107:363-9.
- Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillen PM, Van der Velden U. Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients. J Periodontol 2000;71:1528-34.
- Salzberg TN, Overstreet BT, Rogers JD, Califano JV, Best AM, Schenkein HA. C-reactive protein levels in patients with

aggressive periodontitis. J Periodontol 2006;77:933-9.

- Duan JY, Ou-Yang XY, Zhou YX. Effect of periodontal initial therapy on the serum level of lipid in the patients with both periodontitis and hyperlipidemia. Beijing Da Xue Xue Bao 2009;41:36-9.
- Fentoğlu O, Kirzioğlu FY, Ozdem M, Koçak H, Sütçü R, Sert T. Proinflammatory cytokine levels in hyperlipidaemic patients with periodontitis after periodontal therapy. Oral Dis 2012;18:299-306.
- Oz SG, Fentoglu O, Kilicarslan A, Guven GS, Tanrtover MD, Aykac Y, *et al.* Beneficial effects of periodontal therapy on metabolic control of hypercholesterolemia. South Med J 2007;100:686-91.
- Silness J, Loe H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. Acta Odontol Scand 1964;22:121-35.
- 29. Loe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. Acta Odontol Scand 1963;21:533-51.
- Glavind L, Löe H. Errors in the clinical assessment of periodontal destruction. J Periodontal Res 1967;2:180-4.
- Cutler CW, Shinedling EA, Nunn M, Jotwani R, Kim BO, Nares S, *et al.* Association between periodontitis and hyperlipidemia: Cause or effect? J Periodontol 1999;70:1429-34.
- Machado AC, Quirino MR, Nascimento LF. Relation between chronic periodontal disease and plasmatic levels of triglycerides, total cholesterol and fractions. Braz Oral Res 2005;19:284-9.
- Schenkein HA, Loos BG. Inflammatory mechanisms linking periodontal diseases to cardiovascular diseases. J Clin Periodontol 2013;40(Suppl 14) S51-69.
- D'Aiuto F, Nibali L, Parkar M, Suvan J, Tonetti MS. Short-term effect of intensive periodontal therapy on inflammatory markers and cholestrol. J Dent Res 2005;84:269-73.
- 35. Kamil W, Al Habashneh R, Khader Y, Al Bayati L, Taani D. Effects of nonsurgical periodontal therapy on C-reactive protein and serum lipids in Jordanian adults with advanced periodontitis. J Periodontal Res 2011;46:616-21.
- 36. Rai B, Kharb S, Anand SC. Effect of treatment of periodontitis on creactive protein, tissue plasminogen activator and high-serum/low density lipoprotein in cholesterol: A pilot study. J Clin Diagn Res 2008;2:786-8.
- 37. Suresh S, Narayana S, Jayakumar P, Sudhakar U, Pramod V. Evaluation of anti-inflammatory effect of statins in chronic periodontitis. Indian J Pharmacol 2013;45:391-4.
- Sangwan A, Tewari S, Singh H, Sharma RK, Narula SC. Periodontal status and hyperlipidemia: Statin users versus non-users. J Periodontol 2013;84:3-12.
- Jain MK, Ridker PM. Anti-inflammatory effects of statins: Clinical evidence and basic mechnasims. Nat Rev Drug Discov 2005;4:977-87.
- Schönbeck U, Libby P. Inflammation, Immunity, and HMG-CoA reductase inhibitors: Statins as antiinflammatory agents? Circulation 2004;109(Suppl 1):II18-26.
- Landmesser U, Bahlmann F, Mueller M, Spiekermann S, Kirchhoff N, Schulz S, *et al.* Simvastatin versus ezetimibe: Pleiotropic and lipid-lowering effects on endothelial function in humans. Circulation 2005;111:2356-63.
- Maeda T, Matsunuma A, Kurahashi I, Yanagawa T, Yoshida H, Horiuchi N. Induction of osteoblast differentiation indices by statins in MC3T3-E1 cells. J Cell Biochem 2004;92:458-71.
- 43. FDA.GOV. Review Criteria for Assessment of C-Reactive Protein (CRP), High Sensitivity C-Reactive Protein (hsCRP) and Cardiac C-Reactive Protein (cCRP) Assays. Rockville, MD: 2005.

Available from: http://www.fda.gov/RegulatoryInformation/ Guidances/ucm077167.htm. [Last accessed on 2005 Feb 19].

- 44. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO 3rd, Criqui M, *et al.* Centers for Disease Control and Prevention; American Heart Association. Markers of inflammation and cardiovascular disease: Application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation 2003;107:499-511.
- 45. Mohan M, Jhingran R, Bains VK, Gupta V, Madan R, Rizvi I, et al. Impact of scaling and root planing on C-reactive protein levels in gingival crevicular fluid and serum in chronic periodontitis patients with or without diabetes mellitus.

J Periodontal Implant Sci 2014;44:158-68.

- Mattila K, Vesanen M, Valtonen V, Nieminen M, Palosuo T, Rasi V, *et al.* Effect of treating periodontitis on C-reactive protein levels: A pilot study. BMC Infect Dis 2002;2:30.
- 47. Ide M, McPartlin D, Coward PY, Crook M, Lumb P, Wilson RF. Effect of treatment of chronic periodontitis on levels of serum markers of acute-phase inflammatory and vascular responses. J Clin Periodontol 2003;30:334-40.

How to cite this article: Tawfig A. Effects of non-surgical periodontal therapy on serum lipids and C-reactive protein among hyperlipidemic patients with chronic periodontitis. J Int Soc Prevent Communit Dent 2015;5:49-56.

Source of Support: Nil, Conflict of Interest: None declared.