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

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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 lipoprotein (LDL), along with decreased levels of high density lipoprotein (HDL). 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. Cardiovascular disease of atherosclerotic origin is one of the main causes of sudden death in developed and developing countries. 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. However, it has been suggested that infections may be responsible for accelerated development of atherosclerosis.
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-\(\alpha\)), interleukin (IL)-1\(\beta\), and IL-6.\(^7,8\) 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 antilipemic 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) \(\geq 4\) 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...
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 (≥200 mg/dl), LDL (≥130 mg/dl), HDL (≤35 mg/dl), and TG (≥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 ± 12.3 and 51.5 ± 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.[20,23,27,34-36] 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.

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

| Table 1: Socio-demographic characteristics of the study and control group participant |
| Variables | Study group | Control group | Total |
| 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) | | | | | |
| 35-37 | 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 and control groups at baseline |
| Variables | Baseline periodontal status | P |
| Control group | Study group | |
| | Mean±SD | 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
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. A similar reduction in GI has been reported among hyperlipidemic patients on statin therapy. 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.

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. 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 A (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. 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.

### Table 3: Lipid profile of the study and control groups at baseline

| Lipid profile | Baseline | Study group | P value |
|---------------|----------|-------------|---------|
| HDL (mg/dl)   | Mean±SD  | Mean±SD     |         |
| Control group | 33.60±9.97 | 33.13±6.60 | 0.881   |
| Study group   | 33.13±6.60 | 33.13±6.60 |         |
| LDL (mg/dl)   | Mean±SD  | Mean±SD     |         |
| Control group | 169.47±23.53 | 181.53±18.11 | 0.127   |
| Study group   | 181.53±18.11 | 181.53±18.11 |         |
| TG (mg/dl)    | Mean±SD  | Mean±SD     |         |
| Control group | 224.40±36.91 | 214.40±39.52 | 0.052   |
| Study group   | 214.40±39.52 | 214.40±39.52 |         |
| TC (mg/dl)    | Mean±SD  | Mean±SD     |         |
| Control group | 203.40±22.02 | 194.40±22.02 | 0.124   |
| Study group   | 194.40±22.02 | 194.40±22.02 |         |
| CRP (mg/l)    | Mean±SD  | Mean±SD     |         |
| Control group | 1.17±0.27 | 1.17±0.27 | 0.001   |
| Study group   | 1.17±0.27 | 1.17±0.27 |         |

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.

### Table 4: Periodontal status at baseline and after periodontal therapy in the control and study groups

| Variables | Baseline | APT | P value |
|-----------|----------|-----|---------|
| PI        | Mean±SD  | Mean±SD     |         |
| Control group | 1.62±0.50 | 1.17±0.27 | 0.005   |
| Study group   | 1.17±0.27 | 1.17±0.27 |         |
| FTB        | Mean±SD  | Mean±SD     |         |
| Control group | 1.20±0.94 | 1.60±0.63 | 0.183   |
| Study group   | 1.60±0.63 | 1.60±0.63 |         |
| PD (mm)    | Mean±SD  | Mean±SD     |         |
| Control group | 3.13±0.62 | 3.13±0.62 | 1       |
| Study group   | 3.13±0.62 | 3.13±0.62 |         |
| GI         | Mean±SD  | Mean±SD     |         |
| Control group | 1.74±0.40 | 1.32±0.30 | 0.003   |
| Study group   | 1.32±0.30 | 1.32±0.30 |         |
| ATL (mm)   | Mean±SD  | Mean±SD     |         |
| Control group | 4.11±0.76 | 4.10±0.74 | 0.976   |
| Study group   | 4.10±0.74 | 4.10±0.74 |         |

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 independent "t" test.

### Table 5: Lipid profile and CRP at baseline and after periodontal therapy in the control and study groups

| Lipid profile | Baseline | APT | P value |
|---------------|----------|-----|---------|
| HDL (mg/dl)   | Mean±SD  | Mean±SD     |         |
| Control group | 33.60±9.97 | 31.87±9.88 | 0.636   |
| Study group   | 31.87±9.88 | 31.87±9.88 |         |
| LDL (mg/dl)   | Mean±SD  | Mean±SD     |         |
| Control group | 169.47±23.53 | 176.47±22.84 | 0.415   |
| Study group   | 176.47±22.84 | 176.47±22.84 |         |
| TG (mg/dl)    | Mean±SD  | Mean±SD     |         |
| Control group | 224.40±36.91 | 214.20±39.52 | 0.471   |
| Study group   | 214.20±39.52 | 214.20±39.52 |         |
| TC (mg/dl)    | Mean±SD  | Mean±SD     |         |
| Control group | 203.40±22.02 | 194.40±22.02 | 0.479   |
| Study group   | 194.40±22.02 | 194.40±22.02 |         |
| CRP (mg/l)    | Mean±SD  | Mean±SD     |         |
| Control group | 1.17±0.27 | 1.17±0.27 | 0.001   |
| Study group   | 1.17±0.27 | 1.17±0.27 |         |

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 independent "t" test.
CRP 3 months after the completion of the non-surgical periodontal therapy. 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. 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.

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 increase in the serum levels of CRP (from 2.3 ± 0.7 to 1.8 ± 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). 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 turbidometric immunoassay with CRP turbilatex kit, 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. 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 Boehringer nephelometer II analyser, among chronic periodontitis patients. 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.

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