ORIGINAL RESEARCH

Evaluation of Inflammatory Acute Phase Protein Level and Different Leukocyte Counts in Chronic Periodontitis Normolipidemic Patients after Nonsurgical Periodontal Therapy

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Abstract

Aim and objective: To evaluate the effect of nonsurgical periodontal therapy on periodontal parameters, serum C-reactive protein (CRP) level, total leukocyte count (TLC), and differential leukocyte count (DLC) in normolipidemic patients with generalized chronic periodontitis.

Materials and methods: A total of 60 subjects (38 males and 22 females) between 20 and 55 years of age were included in this study. Twenty subjects with generalized chronic gingivitis were assigned group I. Forty subjects with generalized chronic periodontitis were randomly divided into test groups, i.e., group II ($n = 20$) and control group, i.e., group III ($n = 20$). At baseline, clinical parameters (plaque and gingival indices, clinical attachment loss) were recorded and blood collected for lipid profile test, TLC, DLC, and CRP estimation. Patients with lipid values in the normal range continued the study. Groups I and II were provided nonsurgical periodontal therapy. Follow-up clinical examination and blood examination were done for CRP level, TLC, and DLC after 1 and 2 months.

Results: A significant improvement in the clinical parameters was evident following scaling and root planning in group II as compared to group III. A decrease in serum CRP and TLC count was also observed, but the difference was not significant. Moreover, a reduction was observed in neutrophils, monocytes, eosinophils post therapeutically in group II but the decrease was significant only for monocyte count.

Conclusion: Based on the findings of the study, it can be concluded that nonsurgical periodontal therapy can reduce the inflammatory component.

Clinical significance: Periodontal diseases comprise a wide range of inflammatory conditions affecting the supporting structures of teeth. Effect of nonsurgical periodontal therapy on chronic periodontitis can be evaluated by measuring the CRP and leukocyte concentration.

Keywords: C-reactive protein, Chronic periodontitis, Leukocyte count, Nonsurgical periodontal therapy.

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Introduction

Periodontitis, triggered by bacterial insult, mediates the destruction of periodontal tissues eventually resulting in tooth loss.[1,2] The pathogenic bacteria which form colonies at the gingivodental junction are the main causative factors for periodontitis. Dental plaque is the biofilm formed at the dentogingival junction by these pathogenic bacteria.[3,4] It consists of a large number of colonies of the pathogenic bacteria which are implanted in network of extracellular polymers present in the host. There is the formation of periodontal pockets by these pathogenic bacteria by causing transformation of sulcular epithelium and junctional epithelium. These dental plaque and periodontal pocket are responsible for the development of chronic periodontitis.[5,6]

Chronic periodontitis causes alveolar bone loss leading to mobility of teeth. This causes severe patient discomfort in the mastication of food. There can be associated pain and bleeding from gums along with complaints of food lodgement.[7,8] There can be eventual loss of tooth causing several problems to the patient like difficulty in mastication, speech, esthetics, occlusal disharmony, and many others. The management of chronic periodontitis can be both nonsurgical as well as surgical therapy.[9,10]

Nonsurgical periodontal therapy is usually the first step in the treatment procedure aimed at eliminating etiological factors of gingival disease and periodontal disease.[11,12] This results in halting of disease progression, which clinically manifests as decreased gingival bleeding, improved plaque score, reduction in probing pocket depth (PPD), and improved clinical attachment level (CAL).[13,14]

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The number of studies exploring the effect of nonsurgical periodontal therapy on hematological and serological parameters is scarce. Thus, the present study was undertaken to evaluate the effect of nonsurgical periodontal therapy on serum C-reactive protein (CRP) level, total leukocyte count (TLC), and differential leukocyte count (DLC) in patients with generalized chronic gingivitis and periodontitis. This would help in correlating the levels of these serological and hematological entities with the diseased and post-treatment status of the periodontium. In addition, it would also explore the relationship between serum CRP level, TLC, and DLC with low-grade chronic inflammatory disease of periodontium, which often passes through an acute phase.

Materials and Methods

It was a prospective study. A total of 60 subjects with BMI (body mass index) within healthy weight criteria, ranging between 20 and 55 years of age suffering from generalized chronic periodontitis or generalized chronic gingivitis were enrolled in this study spanning over 2 months time period. The patients were randomly selected from the out-patient department of a private dental college in Patna, Bihar.

The subjects were divided into three groups. Twenty subjects with generalized chronic gingivitis were assigned as group I. Forty subjects with generalized chronic periodontitis, were randomly divided into test groups, i.e., group II ($n = 20$) and control group, i.e., group III ($n = 20$). Groups I and II were provided nonsurgical periodontal therapy. Subjects of all the three groups were given instructions for oral hygiene maintenance like brushing twice daily with a fluoridated toothpaste, use of interdental aids, and eating a healthy diet limiting sugar intake.

Subjects with erythema, edema, and loss of stippling of the gingival tissue involving at least 20 teeth were included in the study. Nonsmoking patients with BMI within healthy weight criteria were included in the study. Study participants presenting with generalized chronic periodontitis (more than 30% of sites affected) with PPD or attachment loss ≥5 mm and radiographic evidence of alveolar bone loss were included.

Patients suffering from any other systemic illness were not included in the study. Patients with any history of periodontal treatment or antimicrobial treatment in the previous six months or treatment with any medication affecting the serum level of inflammatory markers such as anti-inflammatories, hormonal therapy steroids, statins, immunosuppressants, and anticoagulants were also excluded from the study. Pregnant and lactating women were also not included.

Procedure

A detailed medical and dental history of each participant was recorded. BMIs of all the patients were also recorded. Written consent was obtained from all the study participants and institutional ethical clearance was also obtained.

At baseline, under aseptic precautions, 5 mL of venous blood was collected from all the subjects after a fasting period of 12 - 14 hours. Two milliliters blood was kept in a plain vial for WBC count (using a complete blood cell analyzer) and the rest three milliliters blood sample was stored in an ethylenediaminetetraacetic acid coated vial and sent to the laboratory for lipid profile test (by photometry method in terms of mg/dl by semi-autoanalyzer) and quantitative determination of CRP (by means of particle-enhanced turbidimetric immunoassay using a semiautomated biochemical analyzer). The study was continued on participants with lipid profile values in the normal range.

Maxillary and mandibular alginate impressions were made, cast prepared and an acrylic stent made for standardized measurements of PPD and CAL. Clinical parameters were recorded at baseline. Periodontal status was assessed by measuring the PPD and/or CAL with the help of UNC-15 (Hu-Friedy Manufacturing Company, Chicago, USA) periodontal probe. Plaque index (PI) was scored according to the modified plaque scoring system given by Turesky et al. in the year 1970.[15] Bleeding on probing was assessed using gingival index (GI) as given by Loe and Silness in 1963.[16]

Full-mouth scaling and root planning (SRP) using ultrasonic scaler and Gracey curettes were done by a single operator in two consecutive days for groups I and II patients to avoid fatigue of both the patient and operator. No SRP was done in group III participants during the study period. CAL was recorded in group II but not in group III participants as the reference point in group III was covered with calculus since no SRP was done.

Patients were recalled after 1 and 2 months for follow-up clinical examination and blood sample collection. Only tests for CRP level and WBC (total and differential) count were repeated at follow-up visits.

Statistical Analysis

All statistical analyses were carried out using software Stata version 10 (Stat Corp. Texas, USA) and Excel of Microsoft Office 13. Two-way analysis of variance (F—statistics at appropriate degree of freedom) was used to compare the mean of continuous variables of two categorical factors, i.e., group (test, control, and healthy) and time (baseline, 1 month and 2 months). Scheffe test was used for comparing means of two groups in course of multiple comparisons. One-way analysis was used to compare the mean of three groups (test, control, and gingivitis) for comparing the baseline data. Finally *t*-test was used for comparing means of continuous variables between two groups (test and control) at the baseline and at 2 months. Chi-square was used for accessing association between two categorical variables.

Results

A total of 60 subjects were included in this interventional study who fulfilled the inclusive criteria. Fifty-six (93.3%) subjects completed the study among which nineteen were in group I, nineteen in group II, and eighteen in group III. Drop-out rate was 6.7%. A total of four subjects, one from group I, one from group II, and two from group III, discontinued the study. Data obtained at baseline, after 1 month and 2 months from 56 subjects were analyzed.

Group I consisted of 15 males and 4 females, group II consisted of 8 males and 11 females, and the control group, i.e., group III included 13 males and 5 females. Based on ANOVA, among the three groups, there was a significant difference of mean values of PI ($p = 0.001$), GI ($p = 0.002$), PPD ($p = 0.0001$), and monocyte (M) ($p = 0.042$) at baseline. Rest other variables, i.e., CRP ($p = 0.195$), TLC ($p = 0.723$), neutrophil (N) ($p = 0.56$), lymphocyte (L) ($p = 0.42$), and eosinophil (E) ($p = 0.55$) were similar among three groups (Tables 1 to 6).

A significant improvement in the clinical parameters was evident following SRP in group II as compared to group III. A decrease in serum CRP and TLC count was also observed, but the difference was not significant. Moreover, a reduction was observed in neutrophils, monocytes, eosinophils post-

Table 1: Comparison of baseline variables of periodontal clinical parameters (Mean ± SD) among the three groups

Variables	Group I (n = 19)	Group II (n = 19)	Group III (n = 18)	p-value	Group I vs Group II	Group II vs Group III	Group III vs Group I
PI	2.16 ± 0.46	2.82 ± 0.28	2.81 ± 0.34	0.001*	0.001	0.99	0.001
GI	1.24 ± 0.21	1.49 ± 0.27	1.55 ± 0.27	0.002*	0.07	0.523	0.004
PPD	1.83 ± 0.26	3.29 ± 0.57	3.16 ± 0.57	0.0001*	0.001	0.702	0.0001

*p-value <0.05—statistically significant

Table 2: Comparison of baseline variables of inflammatory markers (Mean ± SD) among the three groups

Variables	Group I (n = 19)	Group II (n = 19)	Group III (n = 18)	p-value	Group I vs Group II	Group II vs Group III	Group III vs Group I
CRP	3.19 ± 1.52	4.32 ± 2.59	4.24 ± 2.60	0.195	0.11	0.995	0.14
TLC	8068 ± 1734	8258 ± 3271	7800 ± 2141	0.723	0.82	0.875	0.677
N	59.74 ± 6.61	62.16 ± 7.41	62.27 ± 7.53	0.56	0.33	0.99	0.284
L	33.68 ± 5.89	31.68 ± 6.78	32.72 ± 6.81	0.42	0.34	0.898	0.648
M	2.89 ± 1.04	2.95 ± 0.85	2.11 ± 0.76	0.042*	0.87	0.02	0.013
E	3.63 ± 2.11	3.21 ± 1.84	2.88 ± 1.60	0.55	0.52	0.895	0.233

*p-value <0.05—statistically significant

Table 3: Comparison of periodontal parameters and inflammatory markers recorded at baseline, after 1 month and 2 months in group I

	Group I (n = 19)				
Variables	Baseline	After 1 month	p-value	After 2 months	p-value
PI	2.16 ± 0.46	2.11 ± 0.56	0.765	2.09 ± 0.64	0.71
GI	1.24 ± 0.21	1.22 ± 0.19	0.89	1.19 ± 0.28	0.537
PPD	1.83 ± 0.26	1.79 ± 0.26	0.724	1.74 ± 0.29	0.32
CRP	3.19 ± 1.52	3.14 ± 1.40	0.92	2.94 ± 1.41	0.61
TLC	8068 ± 1734	8037 ± 1835	0.96	7931 ± 1770	0.811
N	59.74 ± 6.61	60.26 ± 5.55	0.798	60.42 ± 7.15	0.766
L	33.68 ± 5.89	33.94 ± 5.37	0.88	33.73 ± 7.43	0.98
M	2.89 ± 1.04	2.84 ± 0.83	0.87	2.68 ± 0.94	0.518
E	3.63 ± 2.11	2.95 ± 1.87	0.31	3.16 ± 1.54	0.438

Table 4: Comparison of periodontal parameters and inflammatory markers recorded at baseline, after 1 month and 2 months in group II.

	Group II (n = 19)				
Variables	Baseline	After 1 month	p-value	After 2 months	p-value
PI	2.82 + 0.29	2.56 ± 0.34	0.016*	2.51 + 0.37	0.005*
GI	1.48 ± 0.27	1.18 ± 0.15	0.002*	1.14 ± 0.21	0.001*
PPD	3.29 ± 0.57	3.15 ± 0.36	0.377	3.05 ± 0.34	0.123
CRP	4.32 ± 2.59	4.06 ± 2.54	0.756	3.79 ± 2.13	0.495
TLC	8258 ± 3271	8174 ± 3202	0.932	8000 ± 3291	0.81
N	62.16 ± 7.41	61.89 ± 7.50	0.911	61.16 ± 6.54	0.662
L	31.68 ± 6.78	33.10 ± 6.82	0.524	33.84 ± 5.83	0.3
M	2.95 ± 0.85	2.11 ± 0.74	0.002*	2.32 ± 1.00	0.043*
E	3.21 ± 1.84	2.89 ± 1.56	0.57	2.68 ± 1.33	0.321

*p-value <0.05—statistically significant

therapeutically in group II but the decrease was significant only for monocyte count.

Table 5: Clinical attachment loss (CAL) (Mean ± SD) in group II over the period of time

Time	CAL (Mean ± SD)	F-stat (p-value)
Baseline	4.06 ± 0.10	3.35 (p = 0.042)*
1 month	3.8 ± 0.11	
2 months	3.61 ± 0.14	

*p-value <0.05—statistically significant

Table 6: Comparison of periodontal parameters and inflammatory markers recorded at baseline, after 1 month and 2 months in group III

	Group III (n = 18)				
Variables	Baseline	After 1 month	p-value	After 2 months	p-value
PI	2.80 ± 0.34	2.85 ± 0.34	0.66	2.92 ± 0.37	0.32
GI	1.55 ± 0.27	1.54 ± 0.24	0.864	1.62 ± 0.26	0.56
PPD	3.15 ± 0.56	3.32 ± 0.37	0.294	3.43 ± 0.37	0.09
CRP	4.24 ± 2.60	4.19 ± 2.50	0.953	4.31 ± 2.54	0.935
TLC	7800 ± 2141	7822 ± 2098	0.975	7850 ± 2071	0.943
N	62.27 ± 7.53	61.44 ± 6.61	0.727	62.72 ± 7.19	0.855
L	32.72 ± 6.81	33.61 ± 5.91	0.678	31.22 ± 6.66	0.51
M	2.11 ± 0.76	2.33 ± 1.03	0.471	2.89 ± 0.83	0.006*
E	2.88 ± 1.60	2.61 ± 1.33	0.585	3.16 ± 1.88	0.633

*p-value <0.05—statistically significant

Discussion

In the present study, a significant improvement in PI, GI, and CAL values was observed following SRP in the subjects, and similar findings were also documented in the previous studies.[20–24] The reduction in PI is attributed to good plaque control and maintenance of oral hygiene, which in turn ensured reduced

GIs' scores. The reduction in PPD and improvements in CALs are accredited to the reduced inflammatory component following intense periodontal therapy.[24]

The present study found a decrease in serum CRP following periodontal therapy, but the difference of declination was not significant, a finding similar to other studies.[21,25–27] This lack of statistical significance may be due to the relatively small number of participants enrolled in each group, and the fact that systemic inflammation is present in patients with periodontal disease.[1,21,28] On the contrary, several studies have shown significant reduction in serum CRP levels from baseline to the end of treatment after nonsurgical periodontal therapy in periodontitis patients.[23,24,29,30]

In the present study, patients with different grades of severity of periodontal inflammation were included. The mean CRP value was lower in group I patients, i.e., gingivitis patients in comparison to group II, periodontitis patients. These findings were in accordance with other studies, thus suggesting a direct relationship between periodontitis and CRP.[1,17,18,21] On the contrary, Mattila et al. deduced that periodontitis could increase CRP only in some individuals, and elevated baseline CRP levels were not associated with the severity of periodontitis.[31]

The present study also observed a 3.12% decrease in TLC from baseline to the end of treatment in periodontitis patients who underwent nonsurgical periodontal therapy, though the reduction was not statistically significant. A reduction was observed in DLC, i.e., for neutrophils, monocytes, eosinophils post-therapeutically but the decrease was significant only for monocyte counts. The present study failed to observe any difference in basophil count in pretherapy and post-therapy phases. Banthia et al.[32] and Siddeshappa et al.[33] reported a reduction in counts of individual WBCs, i.e., neutrophils, lymphocytes, eosinophils, and monocytes, but this decrease was statistically nonsignificant.

Several authors observed in their study that the leukocyte count between generalized chronic periodontitis patients, localized chronic periodontitis patients, and control group was significantly different, and this difference was primarily attributed to difference in number of neutrophils. This occurs due to a phenomenon known as "dose response," which results in an increase in the number of neutrophils, directly proportionate to the amount of bacteria present.[18,19] On the contrary, in the present study, gingivitis patients had a mean TLC of 8068 cells/mm^3 in comparison to 8035 cells/mm^3 of periodontitis patients at baseline, thereby, indicating no relationship between the severity of periodontitis and TLC.

Mohan et al. evaluated the impact of SRP on CRP levels of gingival crevicular fluid (GCF) and serum in chronic periodontitis patients with type 2 diabetes mellitus (T2DM-CP) and without type 2 diabetes mellitus (NDM-CP). They reported CRP levels in both GCF and serum were higher in T2DM-CP patients than in NDM-CP patients. Although there was a significant improvement in both the groups, greater improvement was observed in both GCF and serum samples of T2DM-CP patients.[34] The present study complies with this observation, i.e., periodontitis patients showed more improvement in clinical parameters after periodontal therapy.

Thus, to conclude, a significant improvement in the clinical parameters was evident following SRP in periodontitis patients. A decrease in serum CRP and TLC count following periodontal therapy was also observed, but the difference of declination was not significant. Moreover, a reduction was observed in DLC, i.e., for neutrophils, monocytes, eosinophils post therapeutically but the decrease was significant only for monocyte counts. Therefore, this study helped in correlating the levels of serological and hematological entities with the diseased and post-treatment status of the periodontium. In addition, it also explored the relationship between serum CRP level, TLC, and DLC with low-grade chronic inflammatory disease of periodontium, which often passes through an acute phase.

The finding obtained from this study that CRP is significantly correlated with periodontal indices which can be of important clinical significance. Measurement of CRP and leukocyte count before and after the periodontal therapy will help in better analysis of the periodontal status as well as prognosis of the periodontal therapy. Therefore hematological tests of the patients for CRP and leukocyte count should be conducted during the periodontal therapy for better analysis.

A short 2 months observation period failed to detect a significant improvement in CRP following the elimination of periodontal inflammation. Limitations of the present study were the small sample size, short term follow-up period, and lack of incorporation of more specific blood parameters. Moreover, it was also not clearly evident if the alteration in CRP was only associated with periodontal infection or there were some other confounding factors like age or mental stress influencing the level of inflammatory markers at the same time.

Conclusion

Based on the findings of the study, it can be concluded that nonsurgical periodontal therapy can reduce the inflammatory component. Prospective studies with a larger sample size, longer observation periods, and more added parameters are needed to further evaluate the benefits of reducing the episodes of periodontal inflammation on the serum level of CRP.

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