VER
Vascular & Endovascular Review

Evaluation of ALP and CRP in Chronic Periodontitis and Healthy Controls Following Scaling and Root Planing

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ABSTRACT

Chronic periodontitis is a common inflammatory condition that leads to the destruction of supporting tooth structures. Biomarkers such as alkaline phosphatase (ALP) and C-reactive protein (CRP) have been linked with inflammation and tissue damage, making them potential indicators of periodontal disease activity. Aim: To evaluate and compare serum levels of ALP and CRP in patients with chronic periodontitis and healthy individuals, and to assess changes following scaling and root planning (SRP). Methods: This study included 86 participants—43 with chronic periodontitis and 43 healthy controls—recruited from the Department of Periodontology, NIMS Dental College, Jaipur. Periodontitis patients underwent SRP, and serum samples were collected at three intervals: before treatment, 1 week after, and 1 month after SRP. Serum ALP and CRP levels were measured using ERBA diagnostic kits with a semi-auto analyzer (ERBA CHEM-7). Data were analyzed for intergroup and intragroup differences using appropriate statistical tests. Results: Baseline serum ALP and CRP levels were significantly higher in chronic periodontitis patients than in healthy controls (ALP: 180.62 ± 33.86 vs 80.81 ± 17.61 U/L; CRP: 15.97 ± 3.72 vs 2.08 ± 0.98 U/L; $p < 0.01$). After SRP, both markers showed a marked and progressive reduction—at 1 week (ALP: 153.28 ± 24.89 U/L; CRP: 10.42 ± 2.83 U/L) and at 1 month (ALP: 110.24 ± 12.25 U/L; CRP: 5.99 ± 1.56 U/L)—with statistically significant improvements ($p < 0.01$). Conclusion: Serum ALP and CRP levels are elevated in chronic periodontitis, reflecting active inflammation. Their significant reduction after scaling and root planning indicates periodontal healing and highlights their potential utility as biomarkers for disease monitoring and therapeutic response.

KEYWORDS: Chronic periodontitis, Alkaline phosphatase, C-reactive protein, Biomarkers, Scaling and root planning, Inflammation.

How to Cite: Raghvendra Saini, Sushma BJ, Neetha Bhargava, (2025) Evaluation of ALP and CRP in Chronic Periodontitis and Healthy Controls Following Scaling and Root Planing, Vascular and Endovascular Review, Vol.8, No.6s, 468-472..

INTRODUCTION

The field of dentistry known as periodontology, or periodontics, focuses on diseases affecting teeth and their supporting structures. The periodontium, comprising cementum, alveolar bone, gingiva, and periodontal ligament, provides the essential support for teeth. One of the most prevalent inflammatory disorders affecting these tissues is chronic periodontitis. [1] It is a degenerative condition characterized by progressive tissue destruction, leading to pocket formation, loss of attachment, and ultimately tooth loss if untreated. The prevalence of chronic periodontitis increases with age and remains a significant public health concern due to its impact on both oral and general health. [2] the disease is primarily caused by specific bacteria or clusters of microorganisms that lead to gingival recession, increased probing depth, and, if persistent, tooth loss. This condition affects mastication, phonation, and aesthetics, thereby diminishing the quality of life. [3] in periodontitis, bacteria act as the initiating agents, while the host response and the complexity of the associated microbiota influence disease progression. These multifactorial interactions make it difficult to identify specific indicators of periodontal disease. [4] Bacterial virulence factors contribute to tissue damage both directly and indirectly by stimulating the host to release biologic mediators. The host response generates proteinases, cytokines, and prostaglandins, which further promote tissue destruction and serve as potential biomarkers for periodontal diagnosis. In addition to these host-derived factors, bacterial enzymes such as collagenases, elastase-like proteases, trypsin-like proteases, aminopeptidases, and dipeptidyl peptidases play significant roles in tissue degradation. [5] Alkaline phosphatase (ALP) is one such biomarker associated with bone metabolism and tissue remodelling, making it a valuable indicator of periodontal disease activity. [6] Chronic periodontitis often progresses silently, with symptoms appearing only after considerable damage. Regular dental checkups are crucial for early detection. Common signs include red, swollen, or tender gums, bleeding gums, gum recession, tooth sensitivity or mobility, painful chewing, altered bite alignment, and changes in denture fitting. [7]

The primary cause of chronic periodontitis is dental plaque biofilm—a bacterial population adhering to non-shedding surfaces such as teeth, restorations, and prostheses. [7] The microbial Etiology involves several key factors. Pathogenic bacteria within dysbiotic biofilms, including members of the “red complex” such as *Tannerella forsythia*, *Treponema denticola*, and *Porphyromonas gingivalis*, play a central role.[8] These microorganisms possess virulence factors enabling them to evade host defense and induce inflammation.[9] Biofilm formation provides bacterial protection from host immune responses, allowing persistence and progression of disease.[10] Microbial dysbiosis—an imbalance favouring pathogenic species—further drives the

onset and severity of periodontitis.[11] Elevated ALP levels are also noted in several bone disorders such as Paget's disease (osteitis deformans), osteocalcin abnormalities, rickets, hyperparathyroidism, and osteogenic sarcoma. Increased ALP activity is additionally observed during bone growth and fracture healing. [12] C-reactive protein (CRP) serves as another marker, reflecting systemic inflammation in response to infectious, traumatic, or viral stimuli.[13] CRP elevation is influenced by various factors including diabetes, lipid disorders, smoking, obesity, and periodontal disease.[14] In periodontitis, inflammatory changes in periodontal tissues can alter the cellular and molecular profile of peripheral blood.[15] Although CRP is primarily synthesized by hepatocytes in the liver, vascular tissues may also produce it locally, marking it as a key indicator of systemic inflammation.[16]

MATERIALS AND METHODS

Study Setting and Ethical Approval

The present study was conducted in the Department of Biochemistry in collaboration with the Department of Periodontics, NIMS Dental College, Jaipur. Ethical clearance for the study was obtained from the Institutional Ethics Committee (Approval No. IEC/P596-/2024). All procedures were performed in accordance with the ethical standards of the institutional and national research committees. Written informed consent was obtained from all participants prior to enrollment.

Study Design, Population, and Selection Criteria

This study was a comparative prospective follow-up design conducted to evaluate and compare the serum levels of alkaline phosphatase (ALP) and C-reactive protein (CRP) in patients with chronic periodontitis and healthy controls. It also aimed to assess the changes in these biochemical parameters following non-surgical periodontal therapy, specifically scaling and root planing (SRP). A total of eighty-six participants of both sexes, aged between twenty and sixty years, were included. Among them, forty-three patients were clinically diagnosed with chronic periodontitis, while forty-three periodontally healthy individuals were taken as controls. The diagnosis of chronic periodontitis was established based on clinical criteria, including gingival inflammation, probing depth, and loss of attachment. Participants within the age range of twenty to sixty years, of either sex, and clinically diagnosed with chronic periodontitis were included after providing informed consent. Exclusion criteria were strictly applied to eliminate confounding factors. Pregnant or lactating women and individuals suffering from systemic conditions such as diabetes mellitus, rheumatoid arthritis, cardiovascular, gastrointestinal, respiratory, or hepatic disorders were excluded. Participants with deleterious habits such as smoking or alcohol consumption were not considered for the study. Those who had taken antibiotics in the last three months, non-steroidal anti-inflammatory drugs (NSAIDs) in the previous six months, or had undergone any form of periodontal therapy in the preceding year were excluded. Individuals who were unable to maintain satisfactory oral hygiene were also not included in the study.

Sample Collection and Processing

For biochemical analysis, five millilitres of venous blood was collected aseptically from the antecubital vein of each participant. In patients with chronic periodontitis, blood samples were drawn at three time points—before scaling and root planing (SRP), one week after SRP, and one month after SRP. In healthy control subjects, a single 5 mL blood sample was collected for comparison. All collected blood samples were centrifuged at 3500 revolutions per minute for fifteen minutes to obtain clear serum, which was used for biochemical estimations.

Biochemical Analysis

The serum levels of alkaline phosphatase (ALP) and C-reactive protein (CRP) were estimated using a semi-auto analyzer (ERBA CHEM-7). ALP was determined using the p-nitrophenyl phosphate (PNPP) method with commercially available ERBA Diagnostic Kits (Lot No. S012457). CRP was measured using the latex-enhanced turbidimetric immunoassay (LTIA) method, also with ERBA Diagnostic Kits (Lot No. S022480). All analyses were conducted according to the manufacturer's instructions to ensure accuracy and reproducibility.

Statistical Analysis

The collected data were compiled and statistically analyzed using the Statistical Package for the Social Sciences (SPSS) software, version 23 (USA Inc.). The mean and standard deviation (SD) of all parameters were calculated. The chi-square test was used to analyze non-parametric variables, while Pearson's correlation coefficient was applied to assess relationships between continuous variables. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: Comparison of the parameters in Chronic Periodontitis and Healthy Controls

Parameters	Chronic Periodontitis	Healthy Controls Mean ±SD	P Value
Alkaline Phosphatase	180.62±33.86	80.81±17.61	<0.01*
C-Reactive Protein	15.97±3.72	2.08±0.98	<0.01*
* Significant at 1% Level of significance			

Table 2: Comparison of Alkaline Phosphatase before SRP, after 1 week and after 1 month of scaling in Chronic periodontitis

Alkaline Phosphatase (ALP)	Mean	SD	P Value
ALP before SRP	180.6200	33.86	<0.01*
ALP after 1 week	153.28	24.89	
ALP before SRP	180.6200	33.86	<0.01*
ALP after 1 month	110.24	12.25	
ALP after 1 week	153.28	24.89	<0.01*
ALP after 1 month	110.24	12.25	
* Significant at 1% Level of significance			

Table 3: Comparison of C-Reactive Protein before SRP, after 1 week and after 1 month of scaling in Chronic periodontitis

C-Reactive Protein (CRP)	Mean	SD	P Value
CRP before SRP	15.97	3.72	<0.01*
CRP after 1 week	10.42	2.83	
CRP before SRP	15.97	3.72	<0.01*
CRP after 1 month	5.99	1.56	
CRP after 1 week	10.42	2.83	<0.01*
CRP after 1 month	5.99	1.56	
* Significant at 1% Level of significance			

Table 4: Correlation between Alkaline Phosphatase and C-Reactive Protein in Chronic periodontitis Patients

Parameters	Mean ±SD	Karl Pearson coefficient of Correlation (r)	P Value
Alkaline Phosphatase	180.62±33.86	0.09	0.556 (NS)
C-Reactive Protein	15.97±3.72		

DISCUSSION:

The present study was carried out at Department of Biochemistry in association with department of Periodontics in NIMS Dental college when it was carried by Institutional ethical approval. The nature of study was comparative prospective follow-up study. The Comparison of Alkaline Phosphatase and C-Reactive Protein in Chronic periodontitis and Healthy controls shows an increase in levels of ALP and CRP in subjects of chronic periodontitis than the healthy control. The ALP levels Mean ±SD in chronic periodontitis is 180.62±33.86 and healthy control is 80.81±17.61. the difference was statically significant (p <0.01). This clearly indicates that serum ALP levels are elevated in chronic periodontitis patients compared to healthy individuals. This shows that ALP activity increase in chronic periodontitis due to bone turnover, tissue destruction, and inflammatory changes. ALP is an enzyme for bone metabolism and tissue remodeling. When Comparison of Alkaline Phosphatase before SRP, after 1 week and after 1 month of scaling in Chronic periodontitis is done the values of Mean ± SD is in the way that before SRP. It is 180.62±33.86. The Mean ± SD after one week of SRP is 153.28±24.89 and after one month of SRP is 110.24±12.25. The ALP levels decrease after one week and gradually more decrease after one month of SRP. This gradual decrease indicates that non – surgical periodontal therapy (SRP) helps in reducing inflammation and tissue destruction. Several previous studies support the findings of the present study that ALP levels decrease after periodontal therapy. Parihar et al.[17], Koppolu et al.[18], Jeyasree et al.[19], and Caúla et al.[20] all reported significant reductions in ALP after SRP, reflecting reduced inflammation and tissue healing. In chronic periodontitis, neutrophils, macrophages, and fibroblasts release ALP due to tissue and bone damage. After SRP, bacterial load and inflammation decrease, leading to lower ALP levels. The further decline after one month indicates ongoing healing and bone stabilization, as supported by Koppolu et al.[18] and Hutomo et al.[21]. When Comparison of CRP before SRP, after 1 week and after 1 month of scaling in Chronic periodontitis is done the values of Mean ± SD is in the way that before SRP it is 15.97±3.72. The Mean ± SD after one week of SRP it is 10.42±2.83 and after one month of SRP is 5.99±1.56. This the CRP levels decrease after one week and gradually more decreases after one month of SRP. This shows a significant decrease (P <0.01). CRP is produced by the liver during inflammation and its high level indicates active infection. After SRP, bacterial load and inflammation decrease, so CRP levels drops as healing begins. Several previous studies support the findings of the present study by Sharma

et al.[22], Kopczyńska et al.[23], Kumar et al.[24], Manocha A et al.[25], and — all showing a fall in CRP after periodontal therapy. Thus, our study confirms that SRP effectively reduces systemic inflammation, and CRP can be used as a marker to monitor treatment success. Previous studies have also reported that elevated CRP levels are linked to periodontal inflammation, supporting the study that CRP reflects the severity of tissue damage. These findings along with the present study, confirm that periodontal inflammation contribute to increase CRP and that SRP is effective in lowering this response both locally and systemically. After SRP, bacterial plaque is removed, inflammation decrease and the liver reduces CRP synthesis. The Correlation between Alkaline Phosphatase and C-Reactive Protein in Chronic periodontitis Patients was shown by Karl Pearson, which was found to be non-significant as shown by p-value (0.556) and r- value (0.09).

CONCLUSION

In this study we conclude that the levels of ALP and CRP gradually decrease after one week and one month of SRP in chronic periodontitis as compared to before SRP. There was no correlation between ALP and CRP in patients of Chronic periodontitis.

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