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Original Research Article

Evaluation of serum alkaline phosphatase and serum creatinine level in chronic periodontitis patients before and after non-surgical periodontal therapy

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ABSTRACT

Background: This study aimed to quantitatively evaluate and compare serum alkaline phosphatase (ALP) and serum creatinine (SCr) levels in chronic periodontitis patients before and after non-surgical periodontal therapy (NSPT), and compare these levels with periodontally healthy individuals.
Methods: Eighty participants, aged 30-50 years, were recruited: 40 with chronic generalized periodontitis (study group) and 40 periodontally healthy individuals (control group). Clinical parameters including simplified oral hygiene index (OHI-S), gingival index (GI), probing depth (PD), and clinical attachment level (CAL) were measured at baseline for both groups. Blood samples were collected from both groups at baseline for ALP and SCr estimation. The both groups received phase I therapy. On day 30 post-NSPT, clinical parameters and blood samples were re-evaluated for the study group. The results were statistically analysed using paired t test and one way ANOVA.
Results: At baseline, the chronic periodontitis group exhibited significantly higher serum ALP levels compared to the healthy control group. Following NSPT, a significant decrease in serum ALP levels (<0.001) was observed in the chronic periodontitis group, accompanied by significant improvements clinical parameters. SCr levels were found to be significantly higher in the periodontitis group at baseline compared to healthy controls. However, no statistically significant (≈0.53) change was observed in SCr levels in the periodontitis group following NSPT.
Conclusions: These results indicate that serum ALP may serve as a responsive biomarker of disease activity and therapeutic response in chronic periodontitis, while SCr though elevated at baseline does not respond to short term NSPT.

Keywords: Biomarkers, Chronic periodontitis, Non-surgical periodontal therapy, Serum alkaline phosphatase, Serum creatinine

INTRODUCTION

Chronic periodontitis is a common inflammatory disease characterized by the progressive destruction of the supporting structures of the teeth, including the periodontal ligament and alveolar bone. This condition arises primarily due to the accumulation of bacterial plaque and the host's immune-inflammatory response to the microbial challenge, ultimately leading to tooth mobility and loss if left untreated.[1] While conventional clinical parameters such as probing depth, clinical attachment level, and bleeding on probing remain the cornerstone of periodontal diagnosis and monitoring, the search for reliable biochemical markers that reflect disease activity and systemic involvement has gained momentum in recent years.[2]

Among such markers, serum alkaline phosphatase (ALP), an enzyme associated with bone turnover and inflammation, has shown elevated levels in individuals

with active periodontal disease, indicating ongoing bone metabolism and tissue destruction.[3] Similarly, serum creatinine- commonly used as a renal function indicator- has also been implicated in systemic inflammatory states, and its levels may be altered in periodontal disease due to the systemic inflammatory burden.[4]

Nonsurgical periodontal therapy (NSPT), primarily consisting of scaling and root planing (SRP), remains the gold standard for initial periodontal treatment. NSPT aims to reduce the microbial load and inflammation in periodontal pockets, promoting tissue healing and halting disease progression. Emerging evidence suggests that in addition to improving local periodontal health, NSPT may also exert systemic effects by modulating the levels of inflammatory biomarkers in circulation.[5]

In light of these considerations, the present study investigated changes in serum ALP and creatinine levels in patients with chronic periodontitis before and after NSPT, compared with healthy controls. The goal was to evaluate the potential of these serum biomarkers as non-invasive indicators of periodontal disease activity and the systemic impact of periodontal therapy.

METHODS

A clinical study was conducted at the department of periodontology from March 2025 to May 2025 as a total number of 80 participants with the age range of 30-50 years were selected. The sample size was calculated using F test; repeated ANOVA between the factors employing G–power software, version 3.1.9.7 (Heinrich-Heine-university, Dusseldorf, Germany).[6,7] Considering the power of the study of 80% at 98% confidence interval; of which, group A (study group) consisted of 40 patients (chronic generalized periodontitis) group B (control group) consisted of 40 individuals (periodontally healthy individuals) and were selected from the outpatient division of periodontics.

Ethical clearance

The study protocol received approval (No: IEC GDCH/PER.4/2025) from the institutional Ethics committee of Government Dental College and Hospital, Ahmedabad and was conducted in full compliance with the ethical principles outlined in the declaration of Helsinki (World Medical Association 2013). All the participants participated in the study were informed about the nature of the study and all the participants signed an informed consent form.

Inclusion criteria

The inclusion criteria for group A (the study group) comprised: (a) interdental clinical attachment loss (CAL) of at least 4 mm at two or more sites on different teeth; (b) radiographic bone loss affecting 15-33% of root length, extending from the coronal third down to the middle or apical third; (c) periodontal probing depths (PPD) ≥5 mm at two or more interdental sites on separate teeth; (d) loss of no more than four teeth attributed to periodontitis- or none at all; (e) a ratio of bone loss to age falling between 0.25 and 1.0; (f) a mild to moderate disease progression rate; and (g) destruction of periodontal structures that correlated with the level of plaque accumulation.

Exclusion criteria

Exclusion criteria were: (a) renal impairment (serum creatinine > 1.2 mg/dl); (b) systemic illnesses such as diabetes, cardiovascular disease, or liver conditions; (c) current smokers; (d) use of medications affecting periodontal tissues; (e) any surgical periodontal treatment within the last six months; (f) contraindications to periodontal therapy; (g) pregnancy or lactation; (h) stage I or IV periodontitis- either too mild (stage I) or too severe with fewer than 20 teeth remaining (stage IV); and (i) grade C periodontitis, indicative of a highly aggressive disease course. Group A served as the control cohort, consisting of individuals with periodontal health as defined by Lang and Bartold.[8] Similarly, group B was characterized by minimal or no clinical inflammation and stable periodontal support; it included patients diagnosed with stage II or III periodontitis of grades A or B, who also received non surgical periodontal therapy (NSPT).

Informed consent was obtained from all the individuals who participated in the study. All the participants were subjected to measurement of clinical indices including simplified oral hygiene index (OHI-S) given by Greene and Vermillion, gingival index given by Loe and Silness, pocket probing depth and CAL at baseline for the both group followed by blood sample collection (Figure 1) Baseline samples (5 ml of venous blood) were collected from both groups using aseptic techniques.

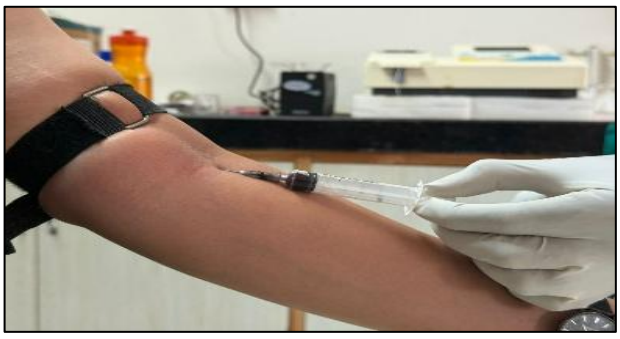

Figure 1: Blood collection.

Blood samples were centrifuged, and serum was separated for analysis (Figure 2). Parameters analyzed: serum alkaline phosphatase (ALP) (IU/l) and serum creatinine (mg/dl). Biochemical analysis was performed using an automated clinical chemistry analyzer in a standardized laboratory setting (Figure 7). Following sample collection, complete ultrasonic scaling was

performed at day 1 for patients with chronic periodontitis. Complete root planing was done within 15 days from the baseline in two subsequent visits. On the 30th day, after the completion of phase I periodontal therapy, patients were reviewed and blood samples were collected and analysed again for ALP and serum creatinine activity.

Figure 2: Serum separation.

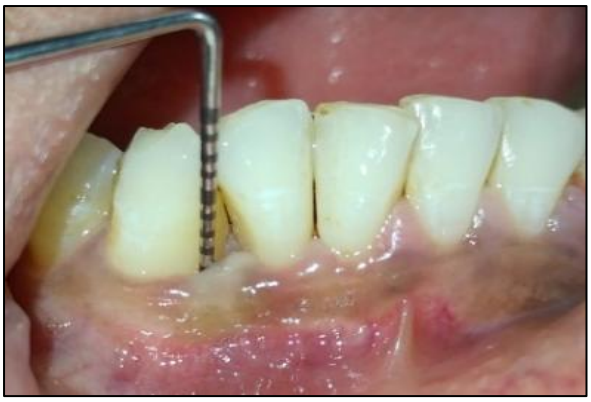

Figure 3: Pocket probing depth at baseline.

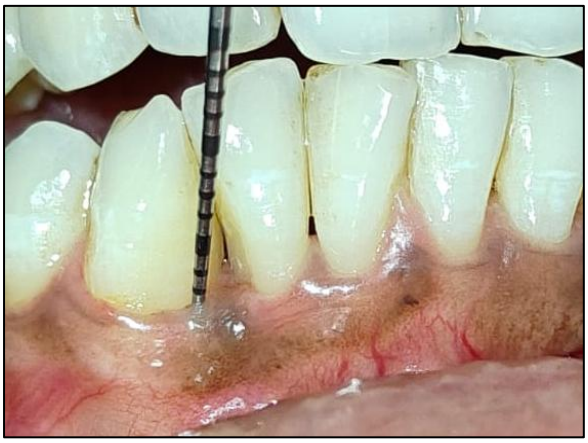

Figure 4: Pocket probing depth after 1 month of NSPT.

Figure 5: Alkaline phosphatase reagent kit.

Figure 6: Creatinine reagent kit.

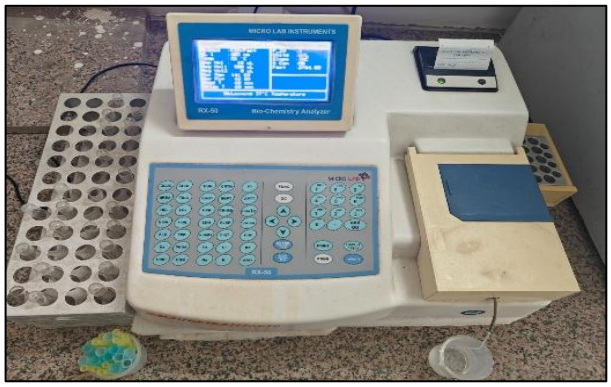

Figure 7: Automated chemical analyser.

Statistical analysis

The obtained data were tabulated, and the data collected were participant to statistical analysis through SPSS (SPSS V:24.0, Softonic International SA© 1997-2018). The paired-t test was used to assess the baseline and 1-month values of clinical parameters and one-way ANOVA was used to compare the enzyme levels in serum between the study group at baseline and postoperatively along with control group.

RESULTS

This clinical study was conducted to evaluate the levels of serum alkaline phosphatase and serum creatinine in patients with generalized chronic periodontitis before and after nonsurgical periodontal therapy and to compare the outcomes with healthy participants. All clinical parameters were measured at baseline with blood sample collected on the same day and then 30 days after the phase I periodontology therapy. The collected sample biochemical analysis was performed using an automated clinical chemistry analyser in a standardized laboratory setting.

ALP levels significantly decreased from baseline to 1month post-NSPT ($p<0.001$), suggesting a reduction in periodontal inflammation and bone turnover after treatment. This supports its potential as a systemic biomarker for periodontal disease activity. No statistically significant change was observed in serum creatinine levels ($p\approx0.53$), indicating that short-term NSPT may not have a notable impact on systemic renal-related markers or that creatinine is not a sensitive biomarker for periodontal inflammation in otherwise systemically healthy individuals. All clinical periodontal parameters showed statistically significant improvement after NSPT ($p<0.001$), reflecting the effectiveness of non-surgical therapy in reducing inflammation, improving oral hygiene, and halting disease progression.

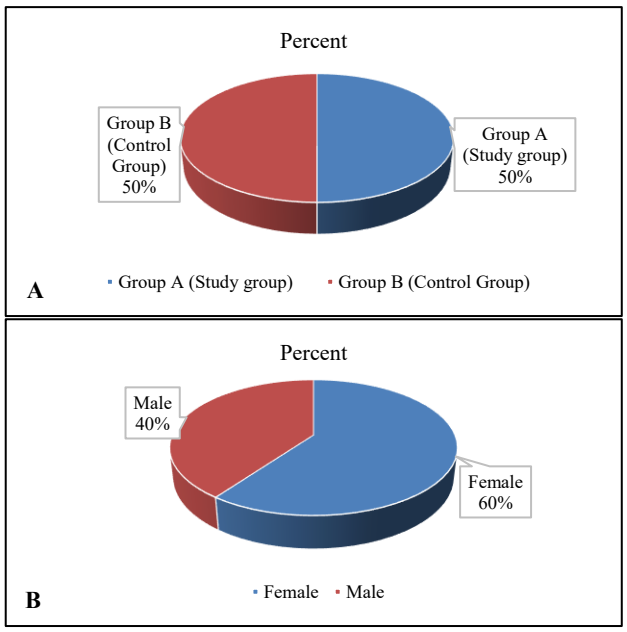

Figure 8 (A and B): Demographic data.

Table 1: Comparison of clinical parameters, serum ALP and serum creatinine level in group A (study group) at baseline and 1 month.

Parameters	Baseline mean±SD	1 Month mean±SD	Mean difference	P value
Serum alkaline phosphate (IU/l)	160.690±25.643	82.534±22.566	78.156	<0.001 (S)
Serum creatine mg/dl	0.979±0.09932	0.9605±0.15778	0.019	≈0.53 (NS)
Gingival index	2.380±0.270	0.685±0.242	1.694	<0.001 (S)
OHI-S	3.013±0.411	1.473±0.296	1.540	<0.001 (S)
Probing pocket depth	6.013±0.594	5.150±0.635	0.863	<0.001 (S)
CAL	6.369±0.555	5.181±0.494	1.188	<0.001 (S)

S- significant, NS-non significant, GI- gingival index, OHI-S- oral hygiene index- simplified, PPD- pocket probing depth, CAL- clinical attachment level.

Table 2: Comparison of clinical parameters, serum ALP and serum creatinine level in group A and group B at baseline and 1 month.

Parameters		Group A (mean±SD)	Group B (mean±SD)	P value
Serum Alkaline Phosphate U/l	Baseline	160.690±25.643	80.0390±17.8787	<0.001 (S)
	1 Month	82.534±22.566	77.1118±14.8240	≈0.208 (NS)
Serum creatine mg/dl	Baseline	0.979±0.09932	0.9690±0.0993	≈0.654 (NS)
	1 Month	0.9065±0.1578	0.9075±0.1578	≈0.977 (NS)
Gingival index	Baseline	2.380±0.270	1.8175±0.4056	<0.001 (S)
	1 Month	0.685±0.242	0.6853±0.2419	≈0.996 (NS)
Oral hygiene index - simplified	Baseline	3.013±0.411	2.4988±0.5757	≈0.0016 (S)
	1 Month	1.473±0.296	1.4725±0.2961	≈0.994 (NS)
Probing pocket depth	Baseline	6.013±0.594	3.8675±0.4760	<0.001 (S)
	1 Month	5.150±0.635	3.7750±0.7224	<0.001 (S)
Clinical attachment level	Baseline	6.369±0.555	0.3168±0.3335	<0.001 (S)
	1 Month	5.181±0.494	0.1333±0.2182	<0.001 (S)

Comparison between chronic periodontitis patients (group A) and healthy controls (group B) revealed significantly elevated serum ALP levels in diseased individuals at baseline ($p<0.001$), supporting its role as a potential systemic biomarker for periodontal inflammation. However, post-treatment ALP levels in group A reduced markedly, showing no statistically significant difference from healthy controls ($p\approx0.208$), indicating a favorable systemic response to NSPT. Serum creatinine levels did not differ significantly between groups at any time point, suggesting limited utility of creatinine as a periodontal disease biomarker in systemically healthy individuals.

Clinically, all baseline periodontal indices (GI, OHI-S, PPD, CAL) were significantly worse in group A compared to controls. Post-therapy, gingival and hygiene indices normalized, but probing depths and attachment levels, although improved, remained significantly different, reflecting the chronic nature of tissue destruction in periodontitis and the time required for full recovery.

DISCUSSION

The term "biomarker" refers to biological substances that can be quantified and assessed as indicators of biological health, disease progression, environmental exposures, or pharmacological reactions to treatment.[9] Among the various biomarkers associated with periodontal disease activity, alkaline phosphatase (ALP) serves as a phenotype marker of bone turnover. ALP levels are elevated in numerous bone disorders, with the highest increases noted in Paget's disease (osteitis deformans). Other conditions such as osteomalacia, rickets, hyperparathyroidism, and osteogenic sarcoma also show elevated ALP. Additionally, increased levels occur during bone healing post-fracture and periods of physiological bone growth.[10]

In present study significantly improve in all clinical parameters after nonsurgical periodontal therapy in patients with periodontitis due to reduce the microbial load in supporting periodontal tissue. On comparing the clinical parameters for group A (study group) (Table 1) Forty patients with stage II-III periodontitis completed the one-month intervention. Significant clinical improvements were observed: the gingival index decreased from 2.38±0.27 to 0.69±0.24 ($p<0.001$), and the simplified oral hygiene index improved from 3.01±0.41 to 1.47±0.30 ($p < 0.001$). Periodontal parameters also showed marked gains, with probing pocket depth reducing from 6.01±0.59 mm to 5.15±0.64 mm ($p<0.001$) and clinical attachment level improving from 6.37±0.56 mm to 5.18±0.49 mm ($p<0.001$). The similar result found by study conducted by Dabra and Singh in 2012, ALP is enriched in the membranes of mineralizing tissue cells (e.g. osteoblasts) and is also present in polymorphonuclear leukocytes (PMN) granules.[11]

The results of present study showed that ALP levels were increased in serum in patients with chronic generalized periodontitis which was in accordance with the study conducted by Malhotra et al, in 2010.[12] The study also showed that the following phase I periodontal treatment, there was a significant decrease (Table 1) in the serum ALP levels in patients with chronic generalized periodontitis in accordance with the results obtained from the study conducted by Jeyasree et al along with an added improvement in the clinical parameters following phase I periodontal therapy.[2] ALP is produced by some oral bacteria, including gram-negative microorganisms found in subgingival plaque. In contrast, Gi-bert et al found that patients with attachment loss had significantly lower bone-specific ALP activity than controls, with no difference in total ALP.[13] It suggests that the lowest values for bone-type ALP in the patients with attachment loss resulted either from diminished passage into the general circulation or from diminished local synthesis

Serum creatinine is a breakdown product of creatine, which is predominantly found in skeletal muscle.[14] Recent research has identified low serum creatinine as a risk factor for type 2 diabetes- observed in Japanese men and in Caucasian individuals with morbid obesity.[14,15] The mechanism behind this association remains unclear, but one hypothesis suggests that low creatinine levels may reflect reduced skeletal muscle mass, which is a primary site for insulin action.[14] The present study evaluates the effects of NSPT on serum creatinine levels in systemically healthy individuals with stage II and III and grade A and B periodontitis (group A). In the current study, we observed higher serum creatinine levels in group A than group B at the base line which was statistically significant. While after completion of phase I therapy in patients with periodontitis there is no significant difference in serum creatinine level.

Elevated serum creatinine in periodontitis has been reported previously by Hattatoglu-Sönmez et al and Kshirsagar et al, which supports our results.[16,17] The former focused on premenopausal women, while the latter included healthy adult men and women. However, Caúla et al reported an inverse relationship, finding between lower serum creatinine in severe periodontitis among healthy individuals.[18] Their methodology differed: they used Page and Eke's classification and included patients with at least 15 teeth, while our study used Lang and Bartold's staging/grading and only included subjects with ≤4 teeth missing due to periodontitis. This could explain the discrepancy.[19]

Similarly, Shimazaki et al found lower probing depth and attachment loss with increased serum creatinine among Japanese middle-aged male self-defence force members, which contrasts with our findings- likely due to the homogenous male military sample compared to our diverse general population.[20] More recently, Vrinda et al studied chronic periodontitis patients with and without chronic kidney disease in Tamil Nadu, India, and found

no significant differences in serum creatinine across groups- again supporting our observations.[21] Elevated serum alkaline phosphatase (ALP) has been identified as a predictor of cardiovascular events and mortality among individuals with chronic kidney disease (CKD). There are well-established associations linking periodontal disease with both cardiovascular disease and CKD, although direct causation remains under investigation.[22-24] Our findings strengthen the evidence for these interconnections and suggest a novel mechanistic pathway connecting periodontal health to systemic outcomes.

Despite these findings, caution is essential when interpreting the results. Given the cross-sectional design- where exposure and outcome are assessed simultaneously- causality cannot be established nor the underlying mechanisms confirmed. Such studies are vulnerable to reverse causality, confounding, and biases inherent to prevalence assessments, and therefore are best viewed as hypothesis-generating rather than definitive evidence. Future research should include well-powered prospective longitudinal studies to explore the temporal sequence and causal pathways linking serum biomarkers to chronic periodontitis, and to clarify their role in its association with systemic diseases

Within the limitations of this study, elevated serum ALP appears useful for identifying active periodontal disease and monitoring response to phase I therapy. Although serum creatinine was higher in periodontitis cases than in healthy controls, non-surgical periodontal treatment had little effect on its levels. These findings should be interpreted cautiously, and further, larger-scale studies are needed to validate the clinical utility of these biomarkers.

CONCLUSION

This study underscored the potential utility of serum alkaline phosphatase (ALP) as a systemic biomarker for monitoring periodontal disease activity and response to nonsurgical periodontal therapy (NSPT). Significant improvements in clinical periodontal parameters and a marked reduction in serum ALP levels following phase I therapy suggest that ALP may serve as a reliable indicator of periodontal healing and disease resolution. While elevated serum creatinine was observed in patients with periodontitis at baseline, its levels remained largely unchanged post-treatment, indicating limited diagnostic value for short-term periodontal monitoring in systemically healthy individuals. By integrating biochemical markers with clinical assessments, this research advances the understanding of the systemic manifestations of periodontal disease and highlights the complex interplay between oral and systemic health. These findings contribute to the growing body of evidence supporting the role of periodontal inflammation in systemic disease pathways and call for further longitudinal studies to establish causal relationships and validate the clinical relevance of serum biomarkers in periodontal diagnostics and management.

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Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee (No: IEC GDCH/PER.4/2025)

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