Original Article

Effect of scaling & root planing on the activity of ALP in GCF & serum of patients with gingivitis, chronic and aggressive periodontitis: A comparative study

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ARTICLE INFO

Article history:

Received 10 January 2017

Accepted 16 March 2017

Available online 27 March 2017

Keywords:

GCF

Alkaline phosphatase

Gingivitis

Chronic periodontitis

Aggressive periodontitis

ABSTRACT

Introduction: Host response and environmental factors are known to modify periodontal status adversely. Presently serum, saliva, and GCF are being investigated for its biochemical constituents. GCF contains array of biochemical factors, offering potential use as a diagnostic or prognostic biomarker of the biologic state of the periodontium in health and disease. Alkaline phosphatase is produced locally in the periodontium and shows positive correlation with disease activity and PD. Present study was designed to analyze the levels of ALP in GCF and serum of patients with gingivitis, chronic & aggressive periodontitis before and after SRP & to compare the difference within the study groups.

Methods: OPD patients grouped into: Gingivitis, Aggressive periodontitis & chronic periodontitis patients. Clinical parameters recorded for each patient prior to therapy. Pooled GCF samples collected using micro capillary tubes from the deepest pocket sites for each patient and stored at -70°C . Serum samples also collected and stored at -20°C . Each patient was subjected to scaling and root planing with two weeks maintenance recall. After 6 to 8 weeks GCF and serum samples collected again and all clinical parameters rerecorded. GCF and serum samples analyzed for levels of ALP by using para nitro phenol assay for the three groups.

Results: ALP levels in GCF increased significantly during active phase of disease followed by statistically significant reduction after phase I therapy. Baseline levels of ALP in GCF was $\text{CP} > \text{AP} > \text{G}$ with maximum reduction in GCF ALP after SRP in $\text{G} > \text{CP} > \text{AP}$ group.

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1. Introduction

The traditional clinical diagnosis of periodontal diseases cannot reliably identify sites with ongoing periodontal destruction and does not provide any information on the patient's susceptibility to disease, whether the disease is progressing, or is in remission, or whether the response to therapy will be positive or negative. Though specific microorganisms are cited as a cause for periodontitis, various other aspects of tissue alterations are known to modify the periodontal status adversely. Based on this concept, presently serum, saliva, tissue biopsy specimens and gingival crevicular fluid have been investigated for their biochemical constituents.^{1,2}

GCF contains a vast array of biochemical factors, offering potential use as a diagnostic or prognostic biomarker of the biologic state of the periodontium in health and disease.³ GCF can be collected in stimulated or non stimulated form, non invasively from different area of mouth and has proved as a reliable indicator of disease activity.^{4–6}

Due to increase in disease activity many proteins and enzymes are increased in the GCF. Alkaline phosphatase is one such enzyme produced by many cells within the area of periodontium and gingival crevice. Neutrophils are probably the main source of enzymes in the gingival sulcus, although it has been shown that the oral bacteria including some gram negative microorganisms which are typical for sub gingival plaque also produce this enzyme.⁷

Level of alkaline phosphatase in GCF varies from $60\ \mu\text{m IU/site}$ in gingivitis and $120\ \mu\text{m IU/site}$ in periodontitis. The mean alkaline phosphatase activity in gingival crevicular fluid is 20 times more

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Table 1

Comparative evaluation of treatment based on ALP (GCF).

ALP (GCF)	Group I	Group II	Group III	Significance
Pre	1972.98 ± 727.75	2616.19 ± 475.85	2208.94 ± 801.98	F = 3.405; p = 0.043 [*]
Post	869.32 ± 203.22	1275.31 ± 390.67	1820.73 ± 919.13	F = 9.874; p < 0.001 [*]
Mean change	1103.65 ± 611.09	1340.89 ± 527.84	388.21 ± 267.33	–
% Change	55.9%	51.3%	17.6%	–
P value	<0.001 [*]	<0.001 [*]	<0.001 [*]	–

^{*} Comparative evaluation of treatment based on ALP (GCF).

than that of serum. These observations suggest that alkaline phosphatase is produced locally in the periodontal tissues. Ishikawa and Cimasoni, in 1970 found a significant correlation between pocket depth and levels of alkaline phosphatase enzyme in GCF.⁸

At present there is no definite method to determine the active or inactive state of periodontal disease. The precise determination of disease activity will have a direct influence on the diagnosis, prognosis and therapy. In the light of the above facts, the present study is designed to estimate alkaline phosphatase activity in serum and gingival crevicular fluid in active sites in patients with gingivitis, chronic periodontitis and aggressive periodontitis before and after SRP.

2. Materials and method

2.1. Source of data

Patients recruited from the outpatient department of OXFORD DENTAL COLLEGE AND RESEARCH CENTRE.

2.2. Inclusion criteria

1. Age groups between 20 to 60 years.
2. No antibiotic use in the previous 3 months before the study and no history of chronic antibiotic use.
3. No history of any systemic conditions which might influence the course of periodontal disease or influence periodontal therapy.
4. Patients coming to dental clinic for the first time.

2.3. Exclusion criteria

1. Smoker
2. Medically compromised patients
3. Patients with systemic disease like diabetes mellitus
4. Patients on antibiotics
5. Pregnant females

2.4. Study design

1. Patients recruited from OPD divided into 3 groups

- a Gingivitis
- b Aggressive periodontitis patients
- c Chronic periodontitis patients.

2. Bleeding on probing, probing pocket depths, loss of attachment levels along with Plaque index, gingival index and bleeding index was noted for each patient prior to any therapy.

3. GCF samples collected using micro capillary tubes from the deepest pocket sites for each patient along with 2 ml of blood drawn from antecubital vein for serum analysis was collected prior to any treatment.

4. GCF samples and serum samples were stored at –70 °C and –20 °C respectively till the time of analysis.

5. Patients subjected to thorough scaling and root planing and oral hygiene instructions given.

6. Patients recalled every 2 weeks for maintenance and evaluation of oral hygiene status.

7. After 6–8 weeks GCF samples were again collected and recording of bleeding on probing, pocket depth and loss of attachment levels was repeated.

8. GCF samples along with serum sample before and after treatment for each patient was analyzed for levels of alkaline phosphatase by using Para nitro phenol phosphate assay.

9. Results were analyzed and compared for the 3 chosen groups using ANOVA, student t-test and t-test of a correlation coefficient.

3. Results

The results from this study showed that Serum ALP levels did not have any significant relationship with disease activity where as ALP levels in GCF increased significantly during active phase of disease followed by statistically significant reduction of ALP level after phase I therapy. Baseline levels of ALP in GCF was maximum in chronic periodontitis group (2616.19 ± 475.85) followed by aggressive group and then gingivitis group. Maximum reduction in the level of GCF ALP after scaling and root planing was seen in gingivitis group (55.9%) followed by chronic periodontitis (51.3%) and aggressive periodontitis group (17.6%)(Table 1). Levels of ALP in GCF were also positively correlated to probing pocket depth, plaque index and gingival index in all the three study groups (Table 2). Baseline difference in levels of ALP in GCF was also statistically significant within the study groups but the range was not much. But due to small sample size we could not reach a

Table 2

Pearson's correlation of changes in clinical parameters with changes in ALP levels in GCF and serum.

Pair	Group I	Group II	Group III
Change in PI vs Change in ALP(Serum)	r = –0.482; p = 0.069	r = –0.260; P = 0.350	r = –0.214; P = 0.443
Change in GI vs Change in ALP (Serum)	r = –0.417; p = 0.122	r = –0.265; P = 0.339	r = –0.079; P = 0.779
Change in BI vs Change in ALP (Serum)	r = 0.129; p = 0.647	r = –0.337; P = 0.166	r = –0.047; P = 0.869
Change in PD (A) vs Change in ALP(Serum)	r = 0.025; p = 0.928	r = 0.328; P = 0.232	r = –0.372; P = 0.173
Change in PI vs Change in ALP (GCF)	r = 0.694; p = 0.004**	r = 0.642; p = 0.010*	r = 0.107; p = 0.704
Change in GI vs Change in ALP (GCF)	r = 0.504; p = 0.055+	r = 0.639; p = 0.010*	r = –0.024; p = 0.932
Change in BI vs Change in ALP (GCF)	r = 0.512; p = 0.050*	r = 0.240; p = 0.388	r = –0.321; p = 0.243
Change in PD (A) vs Change in ALP(GCF)	r = 0.196; p = 0.483	r = 0.704; p = 0.003**	r = 0.368; p = 0.160

Comparative evaluation of treatment based on ALP (GCF)

Fig. 1. Comparative evaluation of treatment based on ALP (GCF).

standard measured value of ALP to diagnose a particular type of periodontal disease and differentiate between progressive and stable sites.

4. Discussion

Since increase in alkaline phosphatase in serum have been associated with bone disease, local elevations in GCF could also reflect local tissue destruction in active periodontal disease.⁹ The potential value of alkaline phosphatase as a marker of periodontal disease activity identified by Ishikawa and Cimasoni. Based on their findings, the present study was designed to analyze the levels of alkaline phosphatase enzyme in gingivitis, chronic periodontitis and aggressive periodontitis cases before and after SRP and to compare the difference in the levels of this enzyme in the selected study groups. The efficacy of SRP in controlling the disease activity was assessed by comparing the levels of alkaline phosphatase enzyme with clinical parameters like PI, GI, BOP and probing pocket depth.

Unlike other markers in serum like C-reactive proteins, fibrinogen, glycated Hb, glucose or lipid profile, serum ALP was not found to be positively correlated to disease activity. The changes in the level of alkaline phosphatase in serum after SRP was not statistically significant in the study groups and it was also

negatively correlated with clinical parameters like PI, GI, BI and PD (Table 2) (Fig. 2). This could have been probably due to the fact that the local changes in periodontium may not have a direct effect on the levels of this enzyme in serum. Its changing concentration in serum depends on function of other organ systems like bone, kidney, liver etc.^{9,10}

Baseline ALP levels in GCF was maximum for chronic periodontitis i.e. group II (2616.19 ± 475.85), followed by aggressive periodontitis (Gr III) and gingivitis (Gr I) (Fig 1). This result is in accordance with the study done by Nakashima et al. in 1994 where he found that levels of ALP in GCF was more in periodontitis as compared to gingivitis and healthy sites.¹¹ In our study, the chronic periodontitis group had both increased levels of plaque and gingival index as compared to gingivitis and aggressive periodontitis groups. Hence chronic periodontitis sites at baseline were associated with increased inflammation as compared to aggressive and gingivitis group. This could be attributed to increased levels of neutrophils recruited in GCF as a result of increased permeability of JE along with increased microbial load that could have contributed to raised levels of alkaline phosphatase in chronic periodontitis cases. Aggressive periodontitis is associated with increased colonization of *A.comitans* and *P. gingivalis* and also hyper responsive neutrophils.^{12–14} All these factors could have resulted in increased levels of ALP in this group at baseline even though the

Comparative evaluation of treatment based on ALP (Serum)

Fig. 2. Comparative evaluation of treatment based on ALP (Serum).

PI was minimum i.e. 1.49 ± 0.20 . In gingivitis, inflammation is restricted to gingiva without affecting deeper periodontal structures, therefore contribution of ALP by periodontal fibroblast and osteoblast is minimum and also there is less periodontopathic microbial load associated with gingivitis. This could be the reason why baseline levels of ALP in gingivitis was minimum in comparison to chronic and aggressive groups.

Decreased levels of GCF ALP in aggressive periodontitis cases as compared to chronic periodontitis could be attributed to defective functions of neutrophils and this can further give us a clue that more than the recent citations of hyper responsive nature of neutrophils, it is the defect in neutrophil functions that contributes in the pathogenesis of aggressive disease.¹⁵

After SRP, changes in levels of ALP in all the three groups was statistically significant, but maximum change was seen in gingivitis group i.e. 55.9%, followed by chronic periodontitis (51.3%) and then aggressive periodontitis (17.6%). This means that disease activity was reduced to a maximum after SRP in gingivitis group than in the other two groups, as gingivitis does not involve deeper periodontal tissues unlike chronic and aggressive periodontitis groups. So local factors and microbial load contributing to inflammation could be more efficiently removed in gingivitis group, compared with chronic periodontitis and aggressive group.¹⁶ However, in aggressive periodontitis cases, host immune defects and presence of tissue invasive microorganisms could not be altered by SRP alone.^{17,15} Therefore disease control in aggressive group was minimally achieved by scaling and root planing, and this reflects least change in gingival crevicular fluid levels of ALP found in our present study.

In our study, the changes in levels of ALP in GCF is positively correlated to changes in clinical parameters like PI, GI, PD in gingivitis and chronic periodontitis groups. This is because SRP is successful in decreasing disease activity in these groups owing to its association with plaque induced inflammation. Also, there seems to be a negative correlation between changes in GI and BI with changes in levels of ALP in GCF in aggressive periodontitis group. This is because the changes in total alkaline phosphatase activity in GCF is detectable even before the occurrence of changes in gingival index.¹²

In accordance to the study done by Ishikawa and Cimasoni, our study too showed a positive and statistically significant correlation with levels of ALP in GCF and pocket depth i.e sites with deeper pocket showed increased levels of alkaline phosphatase thus indicating increased disease activity. Minimum changes in levels of GCF ALP in aggressive periodontitis cases as compared to other groups could thus be attributed to lack of antimicrobial therapy in aggressive cases. This reinforces the rationale of antimicrobial therapy in aggressive cases, since only SRP in such cases are not sufficient to arrest the disease and additional antibiotics and surgical intervention forms a part of complete treatment plan.^{17,15}

Similarly, decrease in ALP levels in chronic periodontitis after SRP was less as compared to gingivitis group. This point to the fact that in contrast to gingivitis, chronic periodontitis cases with deep pockets, require additional surgical therapy for complete arrest of disease activity. But the question that remains unanswered in our

study is the exact cut off value/range for ALP levels in GCF that could help us decide if a particular site needs surgical therapy or only non surgical therapy which includes SRP followed by maintenance that would be sufficient.

Conclusion: We thus conclude that ALP levels in GCF increased in gingivitis, chronic periodontitis and aggressive periodontitis and its value decreased significantly after SRP in group I and II. Thus it suggests that it could be used as a fairly accurate risk marker of disease for an extent. Estimation of changes in the levels of this enzyme has the potential to aid in monitoring treatment outcomes. Nevertheless, we could not arrive at a suitable cut off value of ALP to diagnose specifically gingivitis, chronic periodontitis and aggressive periodontitis due to small sample size. This warrants the need for longitudinal studies to concur, whether the changes in the levels of ALP in GCF at baseline and after SRP can be used accurately to evaluate the treatment outcome.

Conflict of interest

The authors have none to declare.

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