ORIGINAL RESEARCH

The effect of periodontal treatment on C-reactive protein level

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ABSTRACT

Background: Chronic periodontitis in a multifactorial inflammatory disease which is caused by various microorganisms. Many studies have found close association between chronic periodontitis and C-reactive protein (CRP). CRP is an inflammatory marker which increases in all inflammatory condition. **Aims and Objective**: The present clinical study was designed to show the effect of periodontal treatment on the CRP levels of gingival crevicular fluid and to determine the effect of nonsurgical therapy in minimizing the CRP levels in chronic generalized periodontitis. **Material and Method**: Gingival crevicular fluid was collected using a micro capillary pipette that was hand calibrated at every 1 mm till 10 mm, from selected sites in the subjects on the 1st, 14th and 45th days. **Results**: Bleeding index showed an improvement of 61% and 72% on the 14th and 45th days, respectively. Probing depth decreased by 29% on the 14th day and 43% on the 45th day. Similarly, the clinical attachment level showed improvement of 35% and 48% on the 14th and 45th days, respectively. **Conclusion**: The findings support the underlying inflammatory component of the disease activity in chronic periodontitis by demonstrating that the presence of CRP is more substantial in gingival crevicular fluid.

Keywords: C-reactive protein, GCF, periodontitis.

INTRODUCTION

Periodontal disease, especially its mild and moderate forms, is highly prevalent in adult-aged populations all over the world, with prevalence rates around 50%[1], while its severe form increases especially between the third and fourth decades of life, with the global prevalence being around 10%.[2] Certain demographic characteristics, such as age, gender, ethnicity, and socioeconomic status, influence the prevalence of periodontitis. Other strongly contributing factors include smoking, diabetes mellitus, metabolic syndrome, and obesity.[3,4] It is noteworthy that smoking and diabetes can expose individuals to the advanced form of periodontal disease already in adolescence and early adulthood.[5-7] There is also a strong relation of smoking to tooth loss in young individuals [8]. Severe periodontitis, the major cause of tooth loss in adults is typically complicated by the drifting and hypermobility of teeth, eventually resulting in the collapsed bite function of an affected individual.[9,10] Moreover, periodontal disease as well as tooth loss are considered to have an association with a variety of chronic diseases and conditions affecting general health.

Even in periodontal health, immune cells are constantly present in the gingiva, thus supporting the balance between oral biofilms and the host.[11] This constant communication keeps the immune response active, being a reciprocal, synergistic, and dynamic interaction. In the periodontium, the immune response carries characteristics of that of any other part of the body; the first action against microbes is due to non-specific innate response, while extended pathogenic challenge activates specific adaptive responses.

Elevated levels of CRP and decreased plasma adiponectin are associated with increased risk of atherosclerosis. As periodontal disease has been suggested to act as a risk factor for atherosclerosis, Iwamoto and Nishimura (2003) examined the effect of antimicrobial periodontal treatment on CRP, adiponectin and TNF-α levels. Periodontal treatment is effective in reducing CRP and TNF-α, while adiponectin does not appear to be influenced by periodontal treatment. Elevated levels of CRP and TNF-α may be associated with increased risk for further development of a thrombosclerosis in periodontitis patients.[12]

Hence, the present study was conducted to evaluate the effect of periodontal treatment on C-reactive protein.

MATERIAL AND METHODS

For the study, 100 individuals with radiographic evidence of bone loss and a pocket depth of less than 5 mm due to chronic generalised periodontitis were chosen. In the study, participants between the ages of 35 and 55 who had been diagnosed with chronic generalised periodontitis, had radiographic evidence of bone loss, and had pockets deeper than 5 mm were included.

Patients who had received oral prophylaxis or taken antibiotics six months before the study's start were not included. Patients who were breastfeeding babies, pregnant, or unable to attend the follow-up visit were also excluded.

On the 0 day (before phase I therapy), the 14th day, and the 45th day, gingival crevicular fluid was collected from specific sites in the subjects using a micro capillary pipette that was manually calibrated at every 1 mm until 10 mm. The gingival index (Loe H and Sillness J), sulcus bleeding index (Mulheman), clinical probing pocket depth, and clinical attachment loss were then recorded in a prepared chart. Oral hygiene tips were reiterated and gingival crevicular fluid was taken for the measurement of CRP on every recall visit, or the 14th and 45th day. Clinical indicators such the gingival index, bleeding index, depth of the clinical probing pocket, and clinical attachment loss were noted.

RESULTS

The aim of this research study is to stabilize the CRP as a gingival crevicular fluid biomarker. CRP levels in patients with periodontitis could prove valuable in identifying patients with enhanced disease susceptibility.

When gingival index was compared, it showed an improvement of 47% on the 14th day and 94% on the 45th day [Table 1].

Table 1: Mean gingival Index before and after treatment

Gingival index	Mean + SD	% change from baseline	P value
Baseline	2.301±0.45		0.00
14th day	1.213±0.36	47%	0.00
45th day	0.127±0.39	94%	0.00

Bleeding index showed an improvement of 61% and 72% on the 14th and 45th days, respectively. Probing depth decreased by 29% on the 14th day and 43% on the 45th day. Similarly, the clinical attachment level showed improvement of 35% and 48% on the 14th and 45th days, respectively.

Table 2: Mean C - reactive protein level before and after treatment

CRP level	Mean + SD	% change from baseline	P value
Baseline	7.431±4.387		0.00
15th day	3.413±1.971	54%	0.00
45th day	1.221±0.099	83%	0.00

The mean score for CRP level at baseline was 7.431 ± 4.387, which reduced to 3.413 ± 1.971 at the end of the 14th day, and further reduced to 1.221 ± 0.099 at the 45th day.

DISCUSSION

While most studies of periodontitis have emphasized the local nature of periodontitis,[13,14] it appears that systemic manifestations of this disease are also detected through the production of CRP and other acute-phase proteins and pro-coagulant mediators.[15] As a response to the presence of bacteria and bacterial products, such as lipopolysaccharides, cell-mediated inflammation is triggered and a number of proinflammatory cytokines (tumor necrosis factor [TNF], interleukin [IL]-1 and IL-8) are synthesized. Systemic inflammation primed by periodontal infection and the release of lipopolysaccharides into the periphery activates both inflammatory cells and endothelial cells and cytokines are carried to the liver where they induce the production of acute-phase proteins such as CRP. The reason for the interest in plasma/serum levels of CRP in periodontitis is due to the epidemiological research indicating association of periodontitis with CVD and that it is an exceptionally stable analyte in plasma and immunoassays for it are robust, well standardized, reproducible and readily available.[16]

Hence, this study was conducted to evaluate the effect of periodontal treatment on CRP level.

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Gains in clinical attachment levels and reduction in pocket probing depth are the most common

parameters used to measure clinical improvement. There was a significant decrease in the clinical attachment loss between baseline and 45th day, which was found to be 48%. Statistically significant reduction in the probing pocket depth was also observed at the end of the 45th day, which was 43%. These findings are in agreement with other studies by Persson et al.[17] and Adonogianaki et al.[18]

CONCLUSION

In gingival crevicular fluid, the level of CRP is more significant and shows the presence of an underlying inflammatory component to the disease activity in chronic periodontitis.

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