An Estimation of Serum C Reactive Protein in Patients with Chronic Generalized Periodontitis

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Abstract
Recently there has been growing interest in studies that concern with C reactive protein (CRP) in various diseases. Several studies have shown the role of CRP in inflammatory disorder including oral diseases. However considering its high prevalence in India, chronic generalized periodontitis has not been widely investigated with respect to CRP. This definitely has developed a responsibility over investigators to find out exact role of CRP in chronic generalized periodontitis. With this view in mind, the current study was undertaken to estimate and correlate serum CRP levels in chronic generalized periodontitis patients with healthy controls. This may provide a useful, simple and reproducible clinical tool and could be used as a biomarker.

Key words: chronic generalized periodontitis, C reactive protein

Introduction:
C-reactive protein, a member of the pentaxin protein family was first identified by Tilet & Francis (1930) in the plasma of patient with pneumonia and was named because of its ability to bind & precipitate the C-polysaccharide of pneumococcus. It is an alpha globulin with a molecular weight of 110,000 to 140,000 Daltons and is composed of five identical subunits which are non covalently assembled as a cyclic pentamer. Liver is the site of its synthesis & normally present as trace constituent of serum of plasma at levels less than 0.3 mg/dl.¹ Its physiological roles are numerous and varied, but mainly functions in host defense. CRP binds to specific macrophage receptors for IgG (FcγRI and FcγRIIa) a function important for opsonisation of bacteria. Moreover, it activates the early parts of the classical pathway of the complement system through binding to C1q and subsequent generation of fragments of C3 that are
recognized by macrophage receptors. CRP however does not fully activate the classical pathway as it binds to the complement regulatory protein factor H. In this CRP enhances bacterial clearance without generating pro-inflammatory products. It was subsequently found that, in mammals, CRP reacts with a group of protein bound to nucleic acids. It is suggested that CRP reaction with the nuclei and membranes of damaged cells may serve to prevent autoantibody response. It was also shown that CRP reaction with nuclei of apoptotic cells contributes to the clearance of these to further decrease inflammation.2

The synthesis of CRP in the hepatocytes may be regulated by pro-inflammatory cytokines like interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor-necrosis factor (TNF), which have been linked with inflammatory disorders. Therefore, these pro-inflammatory cytokines are currently the subject of intense studies as influencing factors in various types of inflammatory disorders.5 CRP is an easily identifiable biomarker which may differentiate periodontitis patients from healthy individuals.

Materials and method
The present study was carried out during the period of from Nov 2011 to Oct.2012

Selection of cases:
Of routine OPD patients reporting to periodontology department, subjects clinically suspicious of chronic generalized periodontitis were selected. Equal number of age and sex matched healthy subjects without any tissue abuse habits or without any clinically obvious periodontitis or oral lesions or systematic diseases were selected as control group. The subjects were grouped as follows:
1. Test group- 120 patients of chronic generalized periodontitis
2. Control group- 120 healthy controls

Sample Collection: Under all aseptic precaution fasting venous blood was collected from antecubital vein in the morning from control and test group. The sample of blood was processed to separate the serum. This serum was used for estimation C - reactive protein by using Kit method (CRP latex Biosystems SA Spain). The results obtained were tabulated analyzed and interpreted.

Results:
In the present study the level of C - reactive protein were compared between test group and control group. (Table 1) From the statistical analysis it was observed that the difference in levels of C-reactive protein between test group and control group was statistically significant.

Table 1: Comparison of serum C - reactive protein in test and control group.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Test group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum C-reactive</td>
<td>4.28±0.38*</td>
<td>2.70±0.49</td>
</tr>
</tbody>
</table>

* P < 0.001 highly significant

The levels of Serum C-reactive protein were compared between male and female of test and control group. (Table 2) The statistical evaluation was done and it was observed that the difference in levels of C-reactive protein was highly significant (P<0.001).

The levels of Serum C-reactive protein were compared between periodontitis patients in four age groups as compared with their control. (Table 3) From the statistical analysis it was observed that the difference in levels of C-reactive protein between periodontitis patients in four age groups as compared with their control was statistically significant (P<0.001).
In the present study, mean C-RP levels were compared between test group and control group. These levels in periodontitis patients of four age groups were compared with their respective controls (p<0.001) in accordance with previous studies by Slade G et al, Salzberg T N. and Chitsazi et al.3-5

Table 2: Comparison of serum C - reactive protein between males and females of test and control group.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Male Test group</th>
<th>Male Control group</th>
<th>Female Test group</th>
<th>Female Control group</th>
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<tbody>
<tr>
<td>C-reactive protein</td>
<td>4.33±0.17*</td>
<td>2.63±0.54</td>
<td>4.24±0.52*</td>
<td>2.76±0.46</td>
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Table 3: Serum C - reactive protein in periodontitis patients.

<table>
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<tr>
<th>Serum C-Reactive Protein</th>
<th>Group I (2nd decade)</th>
<th>Group II (3rd decade)</th>
<th>Group III (4th decade)</th>
<th>Group IV (5th decade)</th>
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</thead>
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<tr>
<td>Test Group</td>
<td>4.39</td>
<td>4.17</td>
<td>4.31</td>
<td>4.27</td>
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<td>Control Group</td>
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Discussion

In present study, mean C-RP levels were compared between test group and control group. These levels in periodontitis patients of four age groups were compared with their respective controls (p<0.001) in accordance with previous studies by Slade G et al, Salzberg T N. and Chitsazi et al.3-5

The elevated levels of C-RP in periodontitis patients occur when bacteria and bacterial products, such as lipopolysaccharide (LPS), as well as locally produced pro-inflammatory cytokines enter the circulation. Increased levels of C-RP indicate a more pronounced systemic inflammation in the periodontitis patient's group. Multiple virulence factors generated by surface proteins, capsules and fimbriae of the periodontal pathogens Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans and others trigger an inflammatory response in the host. The inflammatory infiltrate from gingival tissue initiates destruction of connective tissue and alveolar bone via activation of pro-inflammatory cytokines including IL-1, IL-6 and TNF, which then enters the systemic circulation.6

Endotoxins, derived from Gram negative microorganisms, induces high levels acute phase proteins like C-RP, after its interaction with receptors expressed on the surface of neutrophils and monocytes, which are present in large numbers in periodontal inflammation. C-RP may activate complement in damaged vessel walls whereas IL-6 has pro-inflammatory properties and a procoagulant effect. These properties may contribute to the pathogenesis of coronary syndromes.2

The inflammatory response is an essential component in the initiation and evolution of atherosclerosis. Thus, this inflammatory marker may serve as an intermediate variable, linking periodontal disease to CVD risk.2 In the present study, patients with periodontitis showed higher levels of C-RP than the control group (p<0.001). This is because it has an adverse effect on fibroblast function, chemotaxis and phagocytosis by neutrophils, immunoglobulin production and induction of peripheral vasoconstriction. Elevation of various proinflammatory

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cytokines increase leukocyte-endothelial cell interaction leading to leukocyte recruitment. Thus, periodontitis along with smoking fuels the fire of inflammation in the blood and at the vessel wall.

C-RP is a very early and sensitive marker of chronic inflammation. In a systemic environment that does not cloud its levels, its value can yield very useful information for the diagnosis of the disease. It was observed that there was a significant increase (P < 0.001) in the levels of C-reactive protein in periodontitis patients when compared to their respective controls. C-reactive protein is the most important markers of inflammation and "inflammation" is a common risk factor for both periodontitis. It was observed that there was significant increase (P < 0.001) in the levels of serum C-reactive proteins in periodontitis patients in all four age groups as compared with their controls. Serum C-reactive proteins are the two most important markers of inflammation and "inflammation" is a common risk factor for both periodontitis and CVD. Elevated levels of C-reactive proteins in patients with periodontitis, which is an indicator for early CVD, which may explain the link between periodontitis and increased risk of CVD. There is also an increase in the levels of inflammatory markers like C-reactive proteins. It was observed that periodontitis is associated with an imbalance of all these risk factors for CVD.2

**Conclusion**

CRP values can never be diagnostic on their own and can only be interpreted at the bedside, in full knowledge of all other clinical and pathological results. However, they can contribute powerfully to management, just as universal recording of the patient’s temperature, an equally nonspecific parameter, is of great clinical utility. However, CRP is a nonspecific marker of inflammation, and additional studies of specific cytokines that regulate acute-phase response are necessary to elucidate the mechanisms by which inflammation influences the risk of CVD & also future studies should recruit larger sample with various other types of periodontitis.

**References:**

4. Salzberg TN, Rogers JD, Abort DM. C-reactive protein levels in generalised aggressive periodontitis patients. Salzberg TN Thesis 2006; School of Dentistry, Virginia Commonwealth University, 521 North 11th Street, Richmond, Virginia.

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**Conflict of interest:** None declared