EFFECT OF SMOKING ON C-REACTIVE PROTEIN LEVELS IN CHRONIC PERIODONTITIS

FARZEEN SHAFIQ WASEEM
FARAH NAZ
ASHAR AFAQ
MASOOD A QURESHI

ABSTRACT

The objective of the study was to compare the effect of smoking on the level of C-reactive protein (CRP) in chronic periodontal disease. The response of periodontal treatment and its effect on the level of the CRP was also observed in both groups.

Hundred patients with chronic periodontitis were taken. Smoking history of the patients was assessed and accordingly, they were categorized as: Group A (smokers) and Group B (non-smokers). Periodontal evaluation was done on every patient and blood samples were collected for noting the levels of CRP. All patients were then given treatment of chronic periodontitis by mechanical removal of calculus as well as prescription of antimicrobials and mouth rinses. Periodontal evaluation and blood samples were repeated three months after the given treatment to check the change in CRP levels.

At Baseline, CRP levels of smoker group was more than non-smoker group. Three months after the given periodontal treatment, the periodontal health was significantly improved. Mean CRP level in both smoker group and non-smoker group was significantly decreased.

Chronic periodontitis has significant effect on CRP levels. Chronic periodontitis may add to the inflammatory burden of an individual and this can be reverted to some extent if appropriate treatment is given. This response will not be affected by adverse effects of smoking.

Key Words: C-reactive protein, Chronic periodontitis, Smoking.

INTRODUCTION

Chronic Periodontal infections has been identified as a potential risk factor for systemic pathologies such as cardiovascular disease, atherosclerosis, stroke, diabetes mellitus, preterm labor, stroke, respiratory conditions.1-3 Fortunately, it is a modifiable risk factor since periodontal disease can be easily prevented and treated.4

There is an increasing interest among the researchers to observe role of periodontitis on level of C-reactive protein in diseased as well otherwise healthy individuals.5,6,7

C-reactive protein is an acute phase protein synthesized in liver and is normally present as a trace constituent of plasma or serum in diseased as well otherwise healthy individuals. Elevated CRP goes hand in hand with traditional risk factors for heart disease, such as smoking, obesity, high blood pressure or elevated cholesterol, and rarely occurs in their absence. Recently, it is being preferred on ESR (Erythrocyte Sedimentation Rate), as a routine prognostic test and has become valuable as cholesterol level in lipid profile to predict cardiovascular diseases.8,9,10

Smoking has been strongly associated as a major etiological factor of the cardiovascular disease, high blood pressure, brain stem infarction11, and periodontal disease. It is involved in with the periodontal attachment loss resulting in the active progression of disease. The deleterious effects of active smoking on the musculoskeletal system, soft tissue and bone wound healing have also been recorded.12

In spite of its hazardous health risks, smoking is very common in Pakistan. One out of every two to three middle-aged men in Pakistan, smoke cigarettes.13 Several epidemiological studies have proved smoking as primary behavioral risk factor for the increased accumulation of plaque and Periodontal attachment loss.14,15

METHODOLOGY

Patient Selection

One hundred patients were included for the conduction of this interventional study visiting the
Periodontology Department of the Fatima Jinnah Dental College, Karachi. The study protocols were approved by the Board of Ethical Committee of the institute. All participants signed written informed consent and the whole procedure was verbally explained to patient before inclusion in the study. Patients having chronic periodontitis with a periodontal index of 6 and/or 8 were selected for the study. Exclusion criteria were pregnancy, lactation, and pathologies such as diabetes mellitus, hypertension, asthma, epilepsy, and coronary artery disease, skin, or chronic diseases which can also increase the C-reactive protein levels.

Smoking history was assessed by the number of cigarettes smoked per day, the packs per month and the period since the person started smoking. The subjects were then categorized as: Group A (smokers) and Group B (nonsmokers) based on their smoking status.

**Periodontal Evaluation**

This was assured by four measures of periodontal status i.e. Periodontal pocket depth (PD) using a CPITN probe, Gingival Index (GI), Gingival bleeding on probing (GB) and periodontal attachment level (AL). All four measurements were carried out in Ramford teeth and their mean values were determined for each patient, respectively. The readings were taken before treatment and three months after the treatment.

**Determination of the C-reactive protein**

In this study we used whole blood as a sample, as it needed no storage, and the results were obtained quickly at the chairside of patient. Blood samples were collected from all patients, before and after treatment for chronic periodontitis. NycoCard CRP Single Test kit was used for rapid determination of CRP C-reactive protein in human whole blood. The normal reference range for CRP in serum is usually reported as less than 0-5mg/L.

**Periodontal Treatment**

Chair side treatment included mechanical debridement of the plaque and calculi by manual and ultrasonic scaling as well as deep curettage of gingival and periodontal tissues. An antibiotic course of Metronidazole 400mg 8 hourly was prescribed as it is very effective against most anaerobes involved in periodontal diseases. The Antibiotic cover was given for 5 to 7 days, depending on the destruction of periodontal tissues that varied among patients. Proper brushing and flossing techniques were also demonstrated to the patient along with a prescription of a mouthwash containing Chlorhexidine. After 3 months of periodontal treatment, the patients were recalled for the follow up. On the follow up visit the periodontal evaluation was repeated and the blood sample was taken to check the change in CRP levels.

**Statistical Analysis**

The data was recorded in a proforma and analysis was done using SPSS version 16. Data analysis included descriptive statistics such as frequency distribution, percentage and cross tabulation. Wilcoxon signed matched paired test was applied to measure the difference of periodontal index and CRP, before and after treatment. The level of significance was set at <0.01 p-value. Mann-Whitney U test was applied to compare the periodontal index and the CRP between the smokers and nonsmokers.

**RESULTS**

Total number of patients was 100. There were 38 smokers and 62 non-smokers.

**At Baseline**

The CRP levels were relatively higher in the Group A (smokers) than in the Group B (nonsmokers). There were no significant differences in the reading of Periodontal Index between both groups. (Table 1 and 2)

**Three months after the given treatment**

Three months after the given periodontal treatment the periodontal health was significantly improved as present in Table 1. The mean CRP level in smoker group was 6.79±2.8 and in nonsmokers 6.06±1.9 which was significantly decreased to 6.11±2.5 and 5.23±1.6 respectively.

**DISCUSSION**

The chronic periodontitis is a local inflammatory disease resulting in the inflammation of the gums and repeated exposure of the bloodstream to bacterial invasion leading to bacteremia. Tooth brushing, Tooth

<table>
<thead>
<tr>
<th>CRP</th>
<th>Before treatment</th>
<th>After treatment</th>
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<tbody>
<tr>
<td>Smokers</td>
<td>6.79</td>
<td>*6.11</td>
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<tr>
<td></td>
<td>SD 2.8</td>
<td>SD 2.5</td>
</tr>
<tr>
<td></td>
<td>Range 2-12</td>
<td>Range 2-12</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>6.06</td>
<td>*5.23</td>
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<td></td>
<td>SD 1.9</td>
<td>SD 1.6</td>
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<td></td>
<td>Range 2-12</td>
<td>Range 2-1</td>
</tr>
</tbody>
</table>

Values are expressed as Mean, standard deviation (SD) and *P<0.0001 as compared To CRP levels before and after periodontal treatment

<table>
<thead>
<tr>
<th>CRP</th>
<th>Before treatment</th>
<th>After treatment</th>
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<tbody>
<tr>
<td>Smokers</td>
<td>7.21</td>
<td>*4.42</td>
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<tr>
<td></td>
<td>SD 0.99</td>
<td>SD 1.98</td>
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<tr>
<td></td>
<td>Range 6-8</td>
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<tr>
<td>Non-smokers</td>
<td>7.35</td>
<td>*4.6</td>
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<tr>
<td></td>
<td>SD 0.94</td>
<td>SD 1.91</td>
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<td>Range 6-8</td>
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Values are expressed as Mean, standard deviation (SD) and *P<0.0001 as compared to Periodontal Index (PI) before and after periodontal treatment.
extractions, and periodontal therapy may be the reason of the entrance of bacteria in the blood circulation.16

The chronic infections leads to the activation of our immune system; thus releasing the C-reactive protein in the bloodstream.17, 18 Chronic periodontitis triggers the level of CRP, IL-6, thus increasing the cardiovascular diseases.1 The combination of smoking, periodontal disease can increase the systemic CRP level to a level that is very risky for health. In our study we tried to disclose some aspects of association among these three parameters.

In present study the periodontal index was not poor in smokers as compared to non-smokers as we expected. This controversial result may be due to the fact that in smokers localized vascular dysfunction and reduced gingival blood flow decreases the localized inflammation of gums.

The gingival blood flow is again dependent on the number of the cigarettes, age and mode of smoking.19 The oral hygiene practices of the individual may also balance out the destructive effect caused by the smoking on the periodontal health.

Tobacco is known for reducing the blood supply to gums depriving them of oxygen and nutrients, leaving them vulnerable to the bacterial infections.20, 21 Smoking increases the CRP by its effect on the accumulation of plaque and effects on the host response. Jennifer O’Loghlin et al have shown positive relationship between smoking status and elevated CRP in adolescents, and in particular among the heavier smokers.22 Rima Azar, also proved that the active and passive smoking, both can result in the elevation of CRP levels.23 Kristina Bertl et al have shown that smoking increases the salivary Histamine and CRP levels in periodontal disease. In the present study CRP levels were quite high in the smokers, which is in line with above studies.

CONCLUSION

Chronic periodontitis has significant effect on CRP levels. Periodontal Treatment decreased the CRP level in smokers as well as non-smokers. Our study concluded that the chronic periodontitis may add to the inflammatory burden of an individual and this can be reverted to some extent if appropriate treatment is given.

Our study proved that if the proper periodontal management is provided to the patient the response to this treatment in terms of periodontal health will not be effected by the adverse effect of smoking.

REFERENCES


