Effect of non surgical periodontal therapy on systemic inflammatory markers in chronic periodontitis

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Abstract

Aim: To elicit the effect of non surgical periodontal therapy on the levels of systemic inflammatory markers (Fibrinogen and total white blood cell count) in the chronic generalized periodontitis patients.

Materials and Methods: 40 subjects with chronic generalized periodontitis were included in the study. Oral hygiene instructions were given and scaling and root planing was performed in all the recruited subjects. Data was analysed by Paired t-test. A P-value of less than 0.05 was considered statistically significant.

Results: Non surgical periodontal therapy (scaling and root planing) resulted in statistically significant decrease in the levels of inflammatory markers.

Conclusion: Non surgical periodontal therapy resulted in reduction of systemic inflammatory markers Fibrinogen and WBCS in chronic generalized severe periodontitis patients and thus can help to reduce the risk of systemic diseases.

Keywords: Non Surgical periodontal therapy, systemic inflammatory markers, fibrinogen, WBCS

1. Introduction

Due to the chronic bacterial colonization of supra and subgingival aspects of teeth, the juxtaposed gingival tissue often demonstrates some level of localized inflammation. As periodontitis ensues there are alterations in local host inflammation mediators, the initiation of a localized specific host response and finally a serum antibody response is observed to the bacteria. These findings would support some ability of the localized inflammation and/or infection to manifest systemically within the affected host. Bacterial infections frequently provide a strong stimulus for systemic acute phase response initiated and coordinated by large number of diverse inflammatory mediators including cytokines and glucocorticoids resulting in production of strong, moderate and weak acute phase proteins [1].

Fibrinogen is an acute phase protein that functions as a blood coagulation factor in primary homeostasis in support of platelet aggregation and in secondary homeostasis in fibrin clot formation at the site of vessel injury. Besides its role in blood coagulation, fibrinogen can increase inflammation in three different ways; i) by providing a framework for the accumulation of inflammatory cells, ii) promoting the immune response, and iii) aiding in bacterial colonization, adhesion and invasion. Fibrinogen is expressed constitutively at basal levels and can be elevated 2 to 10 fold during an inflammatory process. Normal levels of fibrinogen are about 1.5–3 g/L. Higher levels are associated with cardiovascular disease (>3.43 g/L) [2,3].

Neutrophils are the first cell type that arrive at an injury site and play a critical role in the host defense against periodontal disease. Increases in the levels of neutrophils affect blood rheology. They adhere to the endothelial membrane and release harmful oxygen radicals and proteolytic enzymes thus contributing to increased inflammatory activity in the atherosclerotic lesions [4,5].

Since periodontal diseases are treatable, we may expect a reduction in the level of these inflammatory markers after effective periodontal therapy. This can help reduce the associated risk of atherosclerotic complications [6,7]. The present study was carried out to evaluate any alterations in the levels of Fibrinogen and WBC count in chronic periodontitis patients before
and after non-surgical periodontal therapy.

2. Materials and Methods

The study population comprised 40 subjects aged 25-50 years. All subjects gave informed consent for the study. Subjects presenting with severe (probing pocket depths greater than 6 mm and marginal alveolar bone loss greater than 30%); generalized (at least 50% of teeth affected) periodontitis were invited to participate in the study. Subjects with a history of cardiovascular disease, diabetes, hepatitis or any other illness likely to increase fibrinogen or white cell count were excluded. All the subjects received oral hygiene instructions and full mouth scaling using ultra-sonics scalers (magnetostrictivescaler) followed by root planing (using Gracey curettes) at baseline. Clinical parameters and inflammatory parameters were recorded at baseline and at the end of 3 months.

2.1 Clinical parameters

Dental disease was scored by: plaque index, gingival index and pocket depth.

2.2 Inflammatory makers

Venous blood was sampled for measurement of inflammatory markers fibrinogen and white cell count. Sysmex CA 50 blood coagulation analyzer was used for fibrinogen and measured by gm/l.

2.3 Principle of the Method

Fibrinogen is a plasma protein which is converted from a soluble protein to an insoluble polymer by the action of thrombin resulting in the formation of a fibrin clot. The thrombin clotting time of dilute plasma is inversely proportional to the fibrinogen concentration of the plasma [8, 9]. Using this principle, Clauss [8] developed a simple quantitative assay for fibrinogen by measuring the clotting time of dilute plasma when excess thrombin is added. The clotting time obtained is then compared with that of a standardized fibrinogen preparation.

2.4 Reagents

2.4.1 Materials Provided

Dade Fibrinogen Determination Reagents Test Kit, Code No. B4233-15SY with

6 x for 1 mL Dade Thrombin Reagent
1 x for 1 mL Dade Fibrinogen Standard
3 x 15 mL Dade Owren’s Veronal Buffer

2.5 Composition

2.5.1 Thrombin Reagent: A lyophilized preparation of bovine thrombin (approximately 100 NIH units/mL) with stabilizers and buffers.

2.5.2 Fibrinogen Standard: Citrated plasma (human), with HEPES buffer solution (12 g/L), stabilized and lyophilized. The fibrinogen content is tested with a method for determining coagulable proteins. The concentration of the Standard is given on the vial label.

2.5.3 Owren’s Veronal Buffer: 2.84 x 10-2 M sodium barbital in 1.25 x 10-1 M sodium chloride solution, pH. WBCs were measured by Sysmex XT-2000i hematological autoanlyser, which uses Fluorescence flow cytometry to measure WBCs [10]. The system employs a 633 nm semi-conductor laser for flow cytometry analysis. For the measurement by flow cytometry of the proportional count, expressed as percent of the total WBC, neutrophils (NEUT), lymphocytes (LYMPH), monocytes (MONO), and eosinophils (EOS), white cells are stained with fluorescent dyes that bind to both DNA and RNA. Side Scatter (SSC) is employed to determine the internal complexity of the cell—the size, shape, and density of the nucleus and granules of the cell. Fluorescence and scatter measurements are combined to characterize white cell populations. Basophils (BASO) are measured separately using cell size and SSC properties.

2.6 Statistical analysis

The data obtained was tabulated and analyzed statistically. Student’s t test was used for intra group comparison. A p-value of less than 0.05 was considered statistically significant.

3. Results

During the study period, subjects did not report changes in lifestyle issues, including exercise, diet, smoking, and medications. Table 1 shows that age and sex distributions of subjects.

Table 1: Demographic characteristics

<table>
<thead>
<tr>
<th>Number</th>
<th>40</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males/females</td>
<td>22/18</td>
</tr>
<tr>
<td>Mean Age</td>
<td>36</td>
</tr>
</tbody>
</table>

Clinical parameters

Table 2 shows the change in periodontal parameters at baseline and 3 months. There is statistically significant improvement in periodontal parameters at the end of 3 months.

Table 2: Intragroup comparison of clinical parameters at baseline and at 3 months.

<table>
<thead>
<tr>
<th>Clinical parameters</th>
<th>Mean ± SD</th>
<th>P - value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plaque index</td>
<td>Baseline</td>
<td>2.43 ± .23</td>
</tr>
<tr>
<td></td>
<td>3 months</td>
<td>1.62 ± .18</td>
</tr>
<tr>
<td>Gingival index</td>
<td>Baseline</td>
<td>2.02 ± .20</td>
</tr>
<tr>
<td></td>
<td>3 months</td>
<td>1.56 ± .22</td>
</tr>
<tr>
<td>Pocket depth</td>
<td>Baseline</td>
<td>5.06 ± .49</td>
</tr>
<tr>
<td></td>
<td>3 months</td>
<td>3.34 ± .52</td>
</tr>
</tbody>
</table>

Inflammatory markers

Table 3 shows the change in inflammatory markers from baseline to 3 months, statistically significant reduction in inflammatory markers was seen at the end of 3 months.

Table 3: Intra group comparison of inflammatory markers at baseline and 3 months

<table>
<thead>
<tr>
<th>Inflammatory markers</th>
<th>Mean ±SD</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibrinogen</td>
<td>Baseline</td>
<td>3 ± .59</td>
</tr>
<tr>
<td></td>
<td>3 months</td>
<td>2.2 ± .61</td>
</tr>
<tr>
<td>WBCS</td>
<td>Baseline</td>
<td>8690 ± 2270.1</td>
</tr>
<tr>
<td></td>
<td>3 months</td>
<td>7395 ± 1232.2</td>
</tr>
</tbody>
</table>

4. Discussion

This study was designed to examine the impact of scaling and root planing (SRP) on Fibrinogen, and White Blood Cell (W.B.C) count in adults with generalized chronic periodontitis. The results of this study showed increase in levels of fibrinogen and wpcs counts in chronic periodontitis patients. These findings presumably reflect the infection aspect of periodontitis as well as manifestations of acute and chronic...
inflammation that exists in periodontium. Such elevations carry a high relative risk of coronary heart disease possibly because there are several plausible mechanisms by which fibrinogen and white cells may promote atherosclerosis, thrombosis and myocardial ischemia\(^{[11]}\).

The traditional treatment modality of SRP remains the gold standard for the non-surgical management of chronic periodontitis\(^{[12]}\). In the present study all the three clinical periodontal parameters (GI, PI and PPD) showed a statistically significant decrease (\(P<0.001\)), following conventional mechanical treatment (SRP). This signifies that there was a significant improvement in the periodontal health of the patients following SRP. Fibrinogen levels showed a statistically significant decrease (\(P<0.05\)) from baseline to 3 months after SRP. These findings are consistent with the study carried out by Vidal F. \textit{et al.} who evaluated the effects of non-surgical periodontal therapy on plasma levels of fibrinogen in patients with severe periodontitis and refractory arterial hypertension and found that the treatment significantly reduced the blood levels of fibrinogen\(^{[13]}\).

WBC count showed a statistically significant (\(P<0.05\)) decrease from baseline to 3 month. Similar results were shown by Fredriksson M. \textit{et al.}\(^{[7]}\), Christgau M. \textit{et al.}\(^{[14]}\), Wakai K. \textit{et al.}\(^{[15]}\), Loos B.G. \textit{et al.}\(^{[6]}\) and Christian C. \textit{et al.}\(^{[16]}\).

This speculates that elevation in systemic inflammatory markers is due to periodontal infection and thorough periodontal treatment is required to eliminate the systemic markers and to reduce the risk of cardiovascular disease. However additional longitudinal studies of larger, better stratified populations will be required to validate usefulness of examining acute phase reactants in monitoring periodontal disease.

5. Conclusion

It can be concluded that acute phase reactants are increased in periodontitis patients and scaling and root planing resulted in reduction of systemic inflammatory markers fibrinogen and WBCS in chronic generalized periodontitis patients and thus can help to reduce the risk of systemic diseases.

6. Conflicts of Interest: None

7. Source of Support: Nil

8. References


13. Vidal F, Figueredo CM, Cordovil I, Fischer RG. Periodontal therapy reduces plasma levels of interleukin-

