Depicting the levels of resistin and adiponectin in GCF (gingival crevicular fluid) in chronic periodontitis subjects before and following treatment

Nayana Borah, Nidhi Srivastava, Shalabh Mehrotra

Abstract Adipokines including resistin and adiponectin are vital in periodontal inflammation along with host response and infection. Adiponectin also had anti-inflammatory activity. Assessment of resistin and adiponectin in gingival crevicular fluid (GCF) of subjects with chronic periodontitis serves as a biomarker to predict the active phase of periodontitis. The purpose of this research was to determine the relationship between resistin and adiponectin levels in GCF of subjects in subjects with chronic periodontitis before and following treatment. 70 subjects were split into two groups where Group I had 35 healthy subjects and Group II had 35 subjects having periodontitis. Group III comprised subjects with periodontitis after the treatment. Samples of GCF were taken earlier to therapy 21 days after therapy. Stages of resistin and adiponectin were assessed with ELISA. The levels and clinical indicators were linked, and conclusions were drawn. The study results presented a reduction in mean stages of resistin and an increase was seen in the mean levels of adiponectin after the periodontal therapy in subjects with chronic periodontitis. The study concludes that adiponectin is an anti-inflammatory compound that increases in subjects following periodontal treatment, whereas resistin is an inflammatory component that shows a reduction after periodontal treatment.

Keywords: adiponectin, chronic periodontitis, periodontal therapy, periodontitis, resistin

1. Introduction

Chronic periodontitis is a chronic and contagious illness affecting some of the tissues that support teeth, and untreated dental problems are likely to lead to tooth loss. Periodontitis mainly has a multifactorial etiology caused by the interaction of causative factors, including inflammatory mediators, immune cells, bacteria, and their products (Guilherme et al 2019). Immune cells are activated by the immune-inflammatory process of the host, initiating the release of inflammatory mediators, including arachidonic acid metabolites, adipokines, and/or chemokines. These cytokines play a crucial role in the development of periodontal illness and tissue destruction (Hajishengallis et al 2021).

Adipokines constitute a set of molecules with biological activity constructed by adipose tissues. Among these adipokines, resistin plays a vital role in subjects with periodontal inflammation. These cytokines and adipokines also play a crucial role in the reaction of the host toward periodontal inflammation and infection (Mårginean et al 2020). Adiponectin imparts anti-inflammatory action by stimulating heme oxygenase-1 and IL-10 (interleukin-10), which are anti-inflammatory components that inhibit LPS (lipopolysaccharide) and stimulate NF-kB (nuclear factor kappa beta) translocation. Adiponectin also hinders the generation of proteolytic and anti-inflammatory compounds in periodontal cells (Koka et al 2020). Figure 1 shows adiponectin’s function in periodontitis.

Resistin causes an increase in monocyte chemoattractant protein-1, IL-12, TNF-α tumor necrosis factor, and proinflammatory cytokines (IL-1 and IL-6), which are all examples of proinflammatory cytokines. In the hepatic stellate cells, macrophages, and peripheral blood mononuclear cells of the humans by NF-kB pathway. A protein hormone called resistin was first identified in mice and subsequently proven to exist in humans. Its levels are often higher in people who are obese and have insulin resistance since it is largely released by macrophages and adipocytes (fat cells). Resistin is considered to have a role in the etiology of several inflammatory illnesses, including periodontal disease, and has been linked to the emergence of persistent low-grade inflammation. This finding indicates that resistin has the main role in inflammation (Carrion et al 2019). Nonsurgical periodontal treatment particularly aims to restore gingival health after the complete removal of the causative and etiologic factors leading to gingival inflammation in the oral cavity, including endotoxins,
calculus, and plaque-causing periodontal inflammation and destruction (Nongrum et al. 2022). Ultrasound and hand-used periodontal instruments can drastically reduce the number of microorganisms seen subgingivally in periodontal diseases. Despite the different treatment modalities available for treating periodontal diseases, nonsurgical periodontal therapy, including scaling and root planing, is still the gold standard technique for treating periodontal diseases (Tsuchiya and Fujio, 2022).

Researchers have examined resistin and adiponectin as possible periodontal disease biomarkers in the GCF. According to research, resistin levels may be higher in the GCF of people who have periodontitis, suggesting a potential link between resistin and the inflammatory processes in periodontal tissues. The GCF of people with periodontitis has been discovered to have lower levels of adiponectin, indicating that this adipokine may have a protective function in maintaining periodontal health. The previous literature has limited data concerning the association of resistin and adiponectin with the clinical parameters in the GCF of subjects with periodontal disease. A biomarker for predicting periodontal damage and the active stage of periodontal disease is the measurement of adiponectin and resistin in the GCF of persons with chronic periodontitis. In previous studies, the association of resistin and adiponectin GCF levels and clinical indicators in periodontal disease has not been well established (Wielento et al. 2023). Hence, the present biochemical-clinical study was performed to assess the stages of resistin and adiponectin in the GCF of individuals with persistent periodontitis and periodontal health before and following scaling and root planing (SRP).

Brement et al. (2019) assessed the levels of three adipokines in typical canine skin. Punch biopsy specimens were taken from the thinly hairy caudal ventral skin belly of a clinically sound dog without a history of skin problems. The samples were formalin-fixed and paraffin wax-embedded. Utilizing rabbit polyclonal primary antibodies that are specific for leptin, adiponectin, and resistin, immunohistochemistry was used. Insulin resistance, visceral adiposity, dyslipidemia, hyperglycemia, and hypertension are all associated with obesity. are just a few of the metabolic and cardiovascular complications that make up metabolic syndrome (MetS). These conditions all raise the risk of cardiovascular diseases (CVDs) and type 2 diabetes (DM2). An increased risk of CVD occurs in those who have rheumatic conditions such as rheumatoid arthritis and osteoarthritis. Since MetS discovered a potential relationship between arthritic conditions and cardiovascular disease, there have been recent improvements in treating arthritic conditions. The prevalence of CVD also remains high (Rezaee et al. 2020). Cai et al. (2020) investigated the connection between Kawasaki disease (KD) and the levels of circulating adipokines in the blood. Resistin may play a significant role in the pathogenesis of KD, and as a result, it may serve as a biomarker for the identification of KD. In contrast, adiponectin may only play a significant pathogenic role in coronary artery lesions (CALs), and as a result, it might serve as a biomarker to identify CALs from noncoronary artery lesions (NCALs).

Musovic et al. (2021) examined the cellular and molecular level control of the secretion of resistin in primary and cultured adipocytes from mice. Adipose tissue hormone resistin is thought to have a role in metabolic illness. Resistin is produced by various cell types inside white adipose tissue in humans as opposed to rats, where it is released by white adipocytes. Asthma is a long-term inflammatory condition of the airways, with a wide range of overlapping pathologies and phenotypes that significantly alter how it manifests clinically. Obesity may alter the prognosis, phenotype, and risk of asthma. Systemic inflammation is one theory for the relationship between fat and asthma. It was proposed that a connection between overweight and asthma may be made via adipokines released by adipose tissue (Osman et al. 2023). Adipocytokines have been intimately related in recent studies to the development of fibrosis and liver inflammation in people with nonalcoholic liver disease. According to the length of antiviral medication, the goal of this research was to ascertain how blood levels of adiponectin and resistin related to how severe the fibrous liver was in patients with chronic hepatitis B (CHB) (Custovic and Rasic, 2022). Habib et al. (2021) determined the relationship between body adiposity indices in healthy adult

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men and the blood levels of resistin, adiponectin, and the adiponectin/resistin ratio (AR ratio). With respect, adiponectin serum levels were favorably associated, and resistin was adversely linked to physical condition assessments that used having an appropriate body composition, a smaller percentage of a greater percentage of fat-free mass, and increased body adiposity.

2. Materials and Methods

The present biochemical-clinical research was performed to assess the concentrations of adiponectin and resistin in the GCF of individuals with persistent periodontitis and prior periodontal health following SRP. The study population contributed subjects from the Institute’s Department of Periodontology. After the detailed study design was explained to all the subjects, informed consent was obtained in both written and verbal formats.

The research involved 70 participants who were split into two groups: 35 periodontally healthy subjects and 35 subjects with periodontal disease. The subjects with periodontal diseases were included according to the following standards: subjects with at least 20 teeth present, periodontally and systemically using healthy volunteers and a probing depth of <3 mm for healthy subjects and ≥5 mm for the periodontal disease group (recorded with 15 mm periodontal probe), individuals who did not undergo any dental care, the period within the past 6 months, and subjects with 6 minimum sites with bleeding on probing in the periodontal disease group.

The 70 2 groups of subjects were formed of 35 subjects each, where Group I had healthy subjects with a probing depth of <3 mm, Group II had subjects with chronic periodontitis with a probing depth of >5 mm, and Group III comprised 35 subjects of Group II after completion of SRP.

The clinical indicators assessed were CAL (clinical attachment level), probing pocket depth, sulcus bleeding index (Klewin-Steinböck and Wyganowska 2023), GI (gingival index) (Seleem et al 2021) and plaque index (Masood et al 2019). All these parameters were assessed at baseline and after 21 days following SRP. The GCF was gathered from the test site after isolation with the cotton roll and air-drying the site, and without touching the marginal gingiva, a supragingival plaque was removed. The GCF sample was collected with a calibrated capillary pipette. Two microliters of GCF were collected from each test site by placing the pipette in the gingival sulcus for 30 seconds. The samples of GCF collected if there was blood or saliva contamination were discarded. All samples were collected at the 1st visit in both groups, and samples gathered on the 21st day contributed to Group III. After collection, the samples were labeled, stored, and processed after being sent to the laboratory.

SRP was performed in all subjects at the same appointment after the collection of GCF as a part of nonsurgical periodontal therapy. The whole mouth underwent ultrasonic scaling at the baseline initial visit. This was followed by root planing under local anesthesia with adrenaline at 1:80,000 with Hu-FriedyGracey curettes. Twenty-one days later, GCF was gathered for Group III clinical parameters and was collected at the same spot and reassessed after 21 days. Resistin and adiponectin were evaluated biochemically with ELISA (enzyme-linked immunosorbent assay). Adiponectin levels were assessed first, followed by resistin levels.

The data gathered were statistically analyzed using a paired t test and Pearson correlation analysis with Version 19 of SPSS, which was released by IBM Company in Armonk, New York, USA. The significance levels were kept at p<0.05.

3. Results

The 70 study subjects were split into two groups of 35 subjects each. Group I had wholesome themes with a probing depth of <3 mm, Group II had subjects with chronic periodontitis with a probing depth of >5 mm, and Group III comprised 35 subjects from Group II after the completion of SRP. For the assessment of clinical parameters in Groups II and III, it was seen that CAL was 4.81±0.34 mm and 2.65±0.43 mm for Groups II and III, respectively, which was expressively greater for Group II with p<0.001. The probing depth was also significantly higher in Group II (5.35±0.45 mm) than in Group III (3.22±0.56 mm) (p<0.001). The sulcus bleeding index, gingival index, and plaque index were significantly higher in Group II (3.45±0.47, 2.64±0.61, and 2.84±0.59, respectively) than in Group III (1.94±0.54, 1.75±0.34, and 1.74±0.26, respectively), and the p value was <0.001 for all three parameters, as shown in Table 1. Figure 2 depicts the study parameter comparison between Group II and Group III participants.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Parameters</th>
<th>Group II</th>
<th>Group III</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>CAL (mm)</td>
<td>4.81±0.34</td>
<td>2.65±0.43</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>2.</td>
<td>Probing depth (mm)</td>
<td>5.35±0.45</td>
<td>3.22±0.56</td>
<td>&lt;0.001</td>
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<tr>
<td>3.</td>
<td>Sulcus bleeding index</td>
<td>3.45±0.47</td>
<td>1.94±0.54</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>4.</td>
<td>Gingival index</td>
<td>2.64±0.61</td>
<td>1.75±0.34</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>5.</td>
<td>Plaque Index</td>
<td>2.84±0.59</td>
<td>1.74±0.26</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>
On comparing the resistance levels in the three research groups, it was observed that in the comparison of Group I and Group II, the resistin level was 7.17±0.36, which was quantitatively significant with p< 0.05. On comparing Groups II and III, the resistin levels were 18.14±0.45 pg/ml, which exhibited statistical significance with p< 0.05, and for Groups I and III, it was 11.26±0.37 pg/ml, which was statistically nonsignificant with p>0.05, as depicted in Table 2. Figure 3 shows the subjects in Groups I, II, and III resistin.

Concerning the assessment of the adiponectin levels in the three study groups, it was observed that in Group I in comparison to Group II, the mean adiponectin levels were 20.16±0.28 ng/ml, which was a difference that was quantitatively significant with p< 0.05. For Comparing Group II to Group III, the mean adiponectin levels were found to be 9.94±0.29 ng/ml with a substantial difference in terms of statistics and p<0.05. For the comparison of Group III to Group I, the mean value of adiponectin was 15.44±0.34 ng/ml, which was statistically nonsignificant with p>0.05, as shown in Table 3. Figure 4 denotes the adiponectin levels of the subjects in Groups I, II, and III.

Table 2 Comparison of levels of resistin in Group I, II, and III subjects.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Resistin (pg/ml)</th>
<th>Comparative groups</th>
<th>Mean± S. D</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Group I</td>
<td>Groups I and II</td>
<td>7.17±0.36</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>2.</td>
<td>Group II</td>
<td>Groups II and III</td>
<td>18.14±0.45</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>3.</td>
<td>Group III</td>
<td>Groups I and III</td>
<td>11.26±0.37</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Table 3 Comparison of levels of adiponectin in Group I, II, and III subjects.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Adiponectin (ng/ml)</th>
<th>Comparative groups</th>
<th>Mean± S. D</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Group I</td>
<td>Groups I and II</td>
<td>20.16±0.28</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>2.</td>
<td>Group II</td>
<td>Groups II and III</td>
<td>9.94±0.29</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>3.</td>
<td>Group III</td>
<td>Groups I and III</td>
<td>15.44±0.34</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>
4. Discussion

In the current biochemical-clinical study, patients with chronic periodontitis had their levels of resistin and adiponectin measured in GCF and prior periodontal health following SRP. In the 70 studies, 2 groups of subjects were formed of 35 subjects each, where Group I had healthy subjects with a probing depth of <3 mm, Group II had subjects with chronic periodontitis with a probing depth of >5 mm, and Group III comprised 35 subjects from Group II after the completion of SRP. For assessment of clinical standards in Groups II and III, it was seen that CAL was 4.81±0.34 mm and 2.65±0.43 mm for Groups II and III, respectively, which was much greater for Group II with p<0.001. The probing depth was also substantially greater in Group II (5.35±0.45 mm) than in Group III (3.22±0.56 mm) (p<0.001). The sulcus bleeding index, gingival index, and plaque index were significantly higher in Group II (3.45±0.47, 2.64±0.61, and 2.84±0.59, respectively) than in Group III (1.94±0.54, 1.75±0.34, and 1.74±0.26, respectively), and the p value was < 0.001 for all three parameters. These findings are consistent with the earlier research of (Rode et al 2019) and (Suresh et al 2018), where authors reported similar parameters in their study subjects as in the present study.

Concerning the comparison of the resistin levels in the three study groups, it was seen that in the comparison of Group I and Group II, the resistin level was 7.17±0.36, which was quantitatively significant with p< 0.05. Comparing Groups II and III, the resistin levels were 18.14±0.45 pg/ml, which exhibited statistical significance with p< 0.05, and for Groups I and III, the resistin levels were 11.26±0.37 pg/ml, which was not statistically significant with p>0.05. These outcomes were in agreement with the previous studies of Alkan et al (2020) and Borilova Linhartova et al (2019), where similar levels to the present study were reported for leptin by authors in their respective studies.

For assessing the adiponectin levels in the three study groups, it was observed that in Group I in comparison to Group II, the mean adiponectin levels were 20.16±0.28 ng/ml, which was a distinction with statistical significance at p< 0.05. For Comparing Group II to Group III, the mean adiponectin levels were found to be 9.94±0.29 ng/ml with a substantial difference in terms of statistics and p<0.05. For group comparison III to Group I, the mean value of adiponectin was 15.44±0.34 ng/ml, which was statistically nonsignificant with p>0.05. These results were in line with the previous outcomes of Nepomuceno et al (2019) and Pamuk and Kantarci (2022), where adiponectin levels reported by the authors were comparable to the results of the present study.

4. Conclusions

The current research derives conclusions despite its limitations that adiponectin is an anti-inflammatory compound that increases in subjects following periodontal treatment, whereas resistin is an inflammatory component that shows a reduction after periodontal treatment. Adiponectin can be considered a biomarker in periodontal diseases. Resistin is an adipokine that is largely released by adipose tissue and has been linked to insulin resistance and inflammation. Resistin levels have been discovered to be higher in people with periodontitis than in those with healthy periodontium in the GCF, which is related to periodontal disease. This shows that resistin could aggravate the development of periodontal disease by promoting inflammation in periodontal tissues. However, further investigation is required to completely comprehend the precise pathways via which resistin affects periodontal health. However, further long-term studies with more samples should be performed to reach a definitive conclusion.

Ethical considerations

Not applicable.
Declaration of interest

The authors declare no conflicts of interest.

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