DOI: https://doi.org/10.31351/vol35iss2pp125-135

Assessing the Clinical Significance of Monocyte Chemoattractant Protein 1 and Protein Oxidation Products in Patients with Chronic Periodontitis

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Received 12/11/2024, Accepted 2/11/2025, Published 24/6/2026

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Abstract

An essential chemokine that triggers, regulates, and draws monocytes to regions of severe periodontal inflammation is monocyte chemoattractant protein-1 (MCP-1). The aim of this study is to evaluate the serum levels of MCP-1 and protein oxidation products in patients with chronic periodontitis, specifically Advanced Oxidation Protein Products (AOPP), 3-Nitrotyrosine (3-NT), and Protein Carbonyl (PC). Additionally, it seeks to determine whether there are any connections between MCP-1 levels and the protein oxidation byproducts. Forty healthy people and forty-five patients who were involved in the current investigation had severe chronic periodontitis. The serum sample of each participant were examined for the levels of MCP-1, AOPP, 3-NT, and PC using the enzyme-linked immunosorbent assay (ELISA). The statistical software R was used to perform correlation and linear regression tests. The periodontitis group's serum levels of MCP-1, AOPP, 3-NT, and PC were significantly greater than the control groups. Protein oxidation measurements and MCP-1 were found to be positively correlated. Increased levels of MCP-1, AOPP, 3-NT, and PC have been thought to be possible indicators of periodontal disease and as potentially important during inflammation.
Keywords: Monocyte chemoattractant protein-1, Nitrotyrosine, Oxidative stress, Periodontitis, Protein carbonyl

Introduction

A complicated infectious disease is periodontitis that affects the tissues that support teeth due to an imbalanced bacterial population. By producing proinflammatory cytokines and mediators, the body's inflammatory response, triggered by bacterial infection, degrades these periodontal tissues [(1)]. Different cell types are preferentially activated by a class of polypeptides called chemokines. MCP-1, or monocyte chemoattractant protein, is one possible way to activate and attract monocytes. It is a strong chemoattractant for particular macrophage, monocyte, and lymphocyte subsets. Monocytes, fibroblasts, endothelial cells, and T cells can all release MCP-1. Numerous diseases, including osteoarthritis, diabetes mellitus, rheumatoid arthritis, cancer, idiopathic pulmonary fibrosis, and atherosclerosis are influenced by it. Monocyte chemotactic ability has also been linked to MCP-1 in relation to oral infections [(2)]. These conditions are mostly brought on by oxidative stress, and it is thought that oxidative stress from periodontal diseases may have a significant impact on systemic inflammation. Peripheral blood levels of oxidative stress markers were lower in individuals with periodontitis than in those with periodontal health, per certain epidemiological investigations [(3)].

However, markers of oxidative stress in the peripheral blood of people with chronic periodontitis vary from study to study and are not always consistent. Reactive oxygen species (ROS) have short half-lives and are highly reactive in biological materials and are therefore challenging to detect directly. Toxic byproducts of ROS generation are therefore commonly employed to assess the effects of oxidative stress-related illnesses. Two commonly utilized indicators for assessing oxidative protein damage are 3-nitrotyrosine (3-NT) and advanced oxidation protein products (AOPP). However, nothing is known about the changes in local 3-NT levels in periodontitis; the only study that discusses this topic is [(4)]. Therefore, from a clinical and scientific perspective, a comprehensive examination of oxidative stress markers in periodontal tissues is crucial. Additionally, systemic oxidative stress could be a major cause of the combined exacerbation of periodontitis. Despite the fact that the severity of periodontitis and bone degradation has been linked to oxidative stress [(5)]. The precise significance of protein oxidation in periodontitis and its participation in the different pathogenic pathways of periodontal disorders stay ambiguous. When oxidants and plasma proteins interact, advanced oxidation protein products AOPP) are created. For determining the degree of

oxidant-mediated protein damage, these are regarded as trustworthy indicators [6]. Nitrotyrosine, or NT-Tyr, is a byproduct of tyrosine's modification when peroxynitrite is present. Autophagy, ferroptosis, pyroptosis, and parthanatos are among the mechanisms that might result in cell death, and One biomarker for oxidative stress (OS) and nitrosative stress (NS) is nitrotyrosine (NT-Tyr) [7]. Proteins can undergo structural or unfolding changes due to reactive oxygen species (ROS), however some of these alterations are comparatively innocuous. However, irreversible protein alterations can cause some proteins to become inactive, which could have detrimental effects on cells for a long time. Reactive oxygen species (ROS) cause a form of irreversible, non-enzymatic protein modification called carbonylation. A number of oxidative mechanisms, such as direct interactions with ROS and interactions with lipids and carbohydrates, can cause proteins to absorb carbonyl groups. As a result, reactive carbonyl species are created, which then react with proteins to create protein carbonyl derivatives including ketones and aldehydes [8]. The carbonyls produced by direct protein oxidation and those made by the addition of pre-oxidized molecules cannot be distinguished in studies on the production of protein carbonyls. Protein carbonyls must therefore be regarded as a universal oxidation indicator [9].

The MCP-1 serum levels in periodontal health and illness have not yet been documented, despite earlier research showing elevated GCF levels of MCP-1 in periodontitis. Thus, this study was conducted to quantify MCP-1 serum level in participants with chronic periodontitis, gingivitis, and a clinically healthy periodontium in light of the previously reported findings. It has been discovered that oxidative stress is linked to the degree of bone loss and periodontitis. But how protein oxidation impacts periodontitis and contributes to the variations in its pathogenic processes is still unclear [5]. The current work attempts to fill this gap by comparing the effects of protein oxidation (3-nitrotyrosine, advanced oxidation protein products, and protein carbonyl) on chronic periodontitis and identifying the part that protein oxidation plays in the disease's pathophysiology. Thus, this study looks at the amounts of 3-nitrotyrosine, protein carbonyl, and AOPP in the blood of those who have chronic periodontitis. The association between oxidative protein indicators and clinical periodontal state was examined, and the data collected were contrasted with those from healthy controls.

Materials and Methods

The study included 45 individuals (28 men and 17 women) with a history of chronic periodontitis, ages 27 to 63. Additionally, 40 healthy controls—25 men and 15 women—who were chosen from dental clinics in Erbil City and matched in age and gender were involved in the research. Those with hepatitis, immunologic abnormalities, pregnant or lactating women, those who during the previous six months, underwent periodontal therapy, and those on antibiotics, anti-inflammatory medications, or antioxidants were excluded. Patients who were systemically healthy and had a clinically healthy periodontium met the inclusion criteria for the matched controls. However, the following were the exclusion criteria for the matched controls: pregnant or nursing women, people with severe oral pathology or problems of the salivary glands, and active smokers or tobacco users.

Collection of blood samples

Each participant had about 5 mL of venous blood extracted and gathered in serum separator tubes (SST) with gold caps. Following a 15-minute standing period, the Samples were centrifuged at 3000 rpm for 15 minutes in order to separate the blood cells from the serum. The extracted serum was promptly moved to Eppendorf tubes that were already coded and labeled, and it was kept at -20°C for further examination

Biochemical assays

The BioVision Company's kits were utilized to evaluate the amounts of MCP-1, AOPP, 3-NT, and PC in serum using the sandwich enzyme-linked immunosorbent assay (ELISA) method.

Sample size estimation

The German software G*Power 3.0.1 was used to do a power analysis. It was shown that, with an effect size of 0.93 and a significance threshold of 0.05, a total calculated sample size of 40 subjects (20 in Group 1 and 20 in Group 2) would provide 80% power to detect significant changes. The sample size was raised to 85 participants in consideration of possible dropouts (45 participants in Group 1 and 40 participants in Group 2).

Statistical analysis

Statistical data analysis was carried out by version 9 of GraphPad Prism. Mean ± SE were used to present bar graphs and results of test statistics. The test of normality (Gaussian distribution) of the data was determined by the Shapiro-Wilk (S-W) and Kolmogorov-Smirnov (K-S) tests. The t-test or Mann-Whitney U test which was unpaired was to compare the patient group's mean study parameters with those of the healthy controls. To assess statistical correlations between MCP-1, the main research parameter, and other study variables within the patient group, Spearman's correlation test (r) was used. According to a 95% confidence interval (CI), a p-value < 0.05 was deemed statistically significant.

Results and Discussion

Monocyte chemoattractant protein 1 (MCP- 1) serum levels

Chemoattractant protein 1 levels in the serum were significantly higher (P<0.0001) in patients (99.428±6.578 pg/µL) than controls (57.591±4.602 pg/µL), as shown in Table 1 and Figure 1. These results are consistent with earlier research [10,11]. It can be argued that the noted increase can be attributed to the fact that plaque and bacterial invasion increase accompanied by the relevant toxins could further damage the sulcular and junctional epithelium, alveolar bone structure, and other supportive tissues. As a result, there may be more nutrients available, which is important for bacterial development. Pathogens such Porphyromonas gingivalis, *Fusobacterium nucleatum*, and *Aggregatibacter actinomycetem-comitans* may promote local synthesis of MCP-1 by different cells at diseased periodontal sites, which could account for the elevated level of MCP-1 associated with periodontitis. Circulating monocytes are drawn to periodontal tissues by MCP-1, and when they are exposed to other stimuli, such as cytokines, they develop into macrophages. In periodontal disease, macrophages are abundant in inflamed gingival tissues and are essential for the destruction of pathogens as well as the synthesis of cytokines and pro-inflammatory mediators. In addition to their pro-inflammatory actions, macrophage-produced IL-1 and TNF-α are known to induce bone resorption [11]. Connective tissue and alveolar bone are among the tissues that break down in periodontitis as a result of the interaction of bacteria or bacterial chemicals with host cells and the host's immunological response. Wang et al. [12] claim that bacterial plaque causes host inflammation, and this is followed by periodontal pathogens such as *Prevotella intermedia*, *Porphyromonas gingivalis*, and *Aggregatibacter actinomycetemcomitans*. In progressing periodontal lesions, monocytes and macrophages are increasingly common, indicating their contribution to the disease formation. Research on the function of MCP-1 in periodontal health and disease has revealed that MCP-1 is released by osteoblasts, mononuclear phagocytes, and endothelial cells. It has been discovered that patients with periodontal disease had higher MCP-1 levels in their gingival biopsies and gingival crevicular fluid (GCF) [13].

Table 1. Monocyte chemoattractant protein-1 levels in patient and control sera

Parameter	Healthy controls	Periodontitis patients	P-Value
MCP-1 (pg/µl)	57.591±4.602	99.428± 6.578	P<0.0001

P < 0.05 indicates statistical significance; values are Mean ± SE.

Inflammatory chemokines begin their actions by attaching to target cell receptors. Specific stimuli cause them to be activated. In order to regulate integrin-dependent adhesion, cytoskeletal rearrangement, and the binding and detachment of cells from their substrates, chemokine receptors and their ligands interact to affect leukocyte migration [14]. Inflammatory cells are drawn to the location of host-bacterial interactions, which promotes tissue degradation into deeper tissue planes. MCP-1 is one chemokine that contributes to cell motility during inflammation. Endothelial cells and vascular smooth muscle cells that have been activated by cytokines create it to aid monocyte migration to the inflammatory area [2]. In one of the studies, the expression of the MCP-1 gene on periodontal tissues and the chemotaxis of the monocytes in the gingival crest fluid (GCF) of the patient with periodontal diseases were examined. They concluded that the patients with Chronic periodontitis expressed much more MCP-1 gene in their gingiva tissues than their periodontally healthy counterparts. This finding demonstrates that MCP-1 expression is important in infiltration of monocytes in the periodontal disease-impacted gingival tissues. Additionally, a study that looked at MCP-1 expression in gingival tissues that were constantly irritated discovered that tissues that were substantially inflamed had significantly higher MCP-1 expression than tissues that were just moderately or minimally impacted. The level of MCP-1 increased as the condition deteriorated and decreased after treatment for periodontal disease, based on studies examining GCF MCP-1 levels in periodontal health and sickness [15]. According to earlier studies, aggressive and chronic periodontitis were associated with higher GCF MCP-1; our findings also indicated that chronic periodontitis had higher MCP-1 than healthy participants.

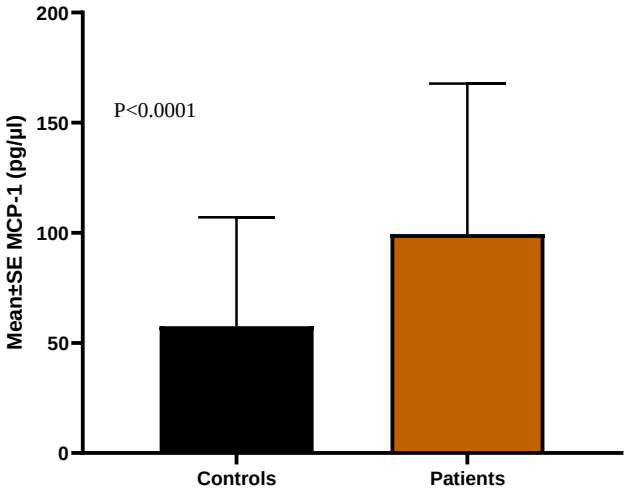

Figure 1. Comparing the serum MCP- 1 Levels in Patients and Controls

Advanced Oxidation Protein Products (AOPP) serum level

Circulating levels of AOPP in patients were higher significantly (P<0.0001) than in healthy controls (5.079± 0.954 ng/mL), according to the data in Figure 2 and Table 2. The present study finding is in a good agreement with many previous study reports [16].

Table 2. Protein oxidation product levels in control and patient sera

Parameters	Controls	Patients	P-Value
Advanced oxidation protein products (ng/mL)	5.079± 0.954	9.504± 1.378	P<0.0001
3-Nitrotyrosine (ng/L)	34.80± 9.864	327.13± 53.46	P<0.0001
Protein Carbonyl (ng/L)	70.337± 5.040	210.43± 12.7	P<0.0001

P < 0.05 indicates statistical significance; values are Mean ± SE.

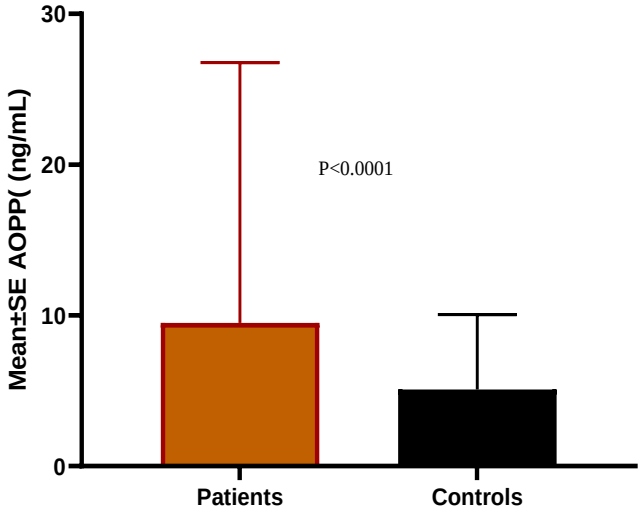

Figure 2. Comparing the serum AOPP Levels in Patients and Controls

In inflammatory conditions, when polymorphonuclear leukocytes are activated, as is frequently the case in periodontitis, reactive oxygen species (ROS) are generated. Oxidative stress is a complicated biological phenomenon that is distinguished by an excess of reactive oxygen species (ROS), which disrupts the redox equilibrium of the body and results in oxidative damage. Every physiological function, including the balance of nucleic acids, is impacted by oxidative stress. ROS

can cause tissue oxidative damage through a variety of mechanisms, including as protein oxidation, DNA damage, and lipid peroxidation (LPO) damage [17]. When periodontal pathogenic bacteria in biofilm trigger host defensive responses, neutrophils are the leading inflammatory cells that accumulate in the periodontal tissue and the gingival sulcus [18]. It is thought that neutrophils are the primary source of ROS in periodontitis. When neutrophils phagocytose periodontal pathobionts, they create an excess of reactive oxygen species (ROS) via the NADPH oxidase pathway [19]. However, it can be challenging to directly identify ROS due to their short half-life. Byproducts associated with ROS and non-enzymatic and enzymatic antioxidant activity are therefore significant pointers in determining the effect of oxidative stress on the pathogenesis of periodontitis [17]. The presence of local differences in the concentrations of biomarkers of oxidative stress are closely associated with the development of the disease, which indicates that the biomarkers can be useful in the diagnosis and monitoring treatment response [20]. Oxidative stress is believed to be a pathophysiological factor in the deterioration of periodontal tissue [21]. Studies have shown that compared to gingivitis and healthy groups, the periodontium of patients with periodontal disease shows noticeably higher levels of oxidative stress. Furthermore, there is a linear relationship between bleeding on probing (BOP) and the degree of periodontal disease [22]. Antioxidant enzymes and ROS-induced tissue damage markers are elevated in inflammatory periodontal tissue and gingival fluid from patients with periodontitis [23]. ROS can alter proteins in a variety of ways, such as by oxidizing certain amino acids, altering the electrical charge of proteins, breaking peptide chains, and creating protein cross-links. These modifications increase the susceptibility of proteins to proteolysis by specific proteases [24]. Advanced oxidation protein products, which are employed to identify oxidative alterations in proteins, are a precise indicator of protein oxidation generated during oxidative stress. It has been shown that in vivo-generated AOPP, an inflammatory mediator, causes neutrophil and monocyte oxidative bursts [15]. Salivary AOPP levels are greater in those with periodontitis, according to publicly accessible data [25]. The periodontitis group (CP) of the study recorded more AOPP serum levels as compared to the controls, which highlights the role played by inflammation of the gingivae in oxidative stress. AOPP levels in serum were statistically significant in both the CP and periodontitis chronic oxidative stress (PCOS) groups, with the CP group having the highest values. Both PCOS and CP cases and an enhanced risk of periodontitis and a local/periodontal prooxidative state have been linked to higher levels of AOPP, an oxidative stress marker, in women with PCOS. Moreover, the study also found that the AOPP of PCOS was lower than the periodontitis and this could be due to the fact that people with periodontitis were experiencing an intensification in their inflammatory stage.

3-Nitrotyrosine (3-NT) serum level

3-nitrotyrosine serum level in patients was highly significant ($P<0.0001$) than controls (34.80± 9.864 ng/L) at 327.13± 53.46 ng/L, as shown in Figure 3 and Table 2. One of the main pathogenic factors in periodontitis has been shown to be oxidative stress [26]. As the primary target of oxidative damage, 3-nitrotyrosine (3-NT) is widely acknowledged as a crucial assay for evaluating oxidative protein damage. Nitrotyrosine, a frequently utilized biomarker for protein oxidation, is expressed in several tissues from patients suffering from periodontitis [27]. The current study found that serum 3-NT levels were considerably higher in patients with periodontal disease. The reactive nitrogen species (RNS), including peroxynitrite and nitrogen dioxide, occur when the inducible form of NO synthase (iNOS) synthesizes excess nitric oxide (NO) in immunological and inflammatory mechanisms in lung diseases. NO and superoxide anions produce these RNS or the pathway of NO production through the action of H2O2/peroxidase-dependent oxidation of nitrite. Tyrosine residue nitration, lipid peroxidation, and tissue damage can result from excessive RNS, according to [28]. One metabolite that symbolizes the in vivo production of RNS is 3-nitrotyrosine, a derivative of amino acids. Numerous inflammatory lung diseases, including chronic obstructive pulmonary disease, have been shown to generate 3-nitrotyrosine [29]. When 3-nitrotyrosine was initially identified, it was believed to be a trustworthy marker of RNS synthesis. Nevertheless, more recent studies have demonstrated that 3-nitrotyrosine itself can act as a "footprint" for RNS and also cause morphological changes, growth inhibition, and cytotoxicity in cultured cells [28].

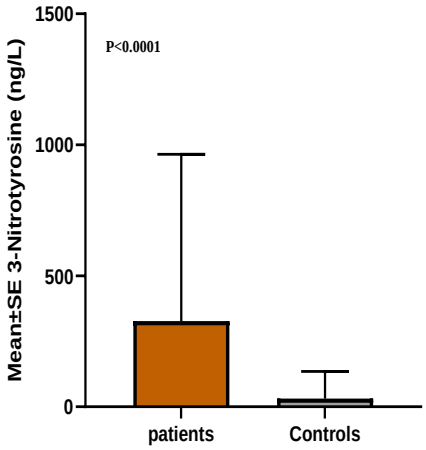

Figure 3. Comparing the serum 3-Nitrotyrosine Levels in Patients and Controls

Protein Carbonyl (PC) serum level

The serum level of protein carbonyl in patients was substantially (P<0.0001) greater in Table 2 and Fig. 4 than in controls (70.337± 5.040 ng/L). The results of the current study on PC are consistent with those of a prior investigation [5]. Oxidative damage to proteins can be classified as either irreversible or reversible [30]. Protein carbonylation, an irreversible protein alteration caused by oxidative stress, typically results in the loss of protein function [31]. Carbonylation is frequently used as a biomarker for evaluating protein oxidative degradation because it can occur selectively in various amino acids [32]. Protein kinases and phosphatases, on the other hand, may oxidize proteins containing cysteine residues close by in a reversible manner. This mechanism can regulate protein activity and redox signaling pathways in a variety of stress reactions [33]. Protein carbonyl levels have not been extensively studied, but several oxidative stress markers and their effects on periodontal diseases have been examined [34, 35]. The connection between oxidative stress and the molecular and clinical differences between aggressive and chronic periodontitis is a field of increasing research interest [34]. Therefore, a better knowledge of the role oxidative stress plays in periodontitis may provide new information on the causes of the chronic or severe course that periodontal infections are known to take. Protein carbonylation can cause oxidative damage that can impair protein function [5]. Because of their early synthesis and stability, carbonyls have shown promise as oxidative stress markers. Additionally, earlier researches highlight the link between oxidative stress, protein carbonylation, and pathogenic situations [36].

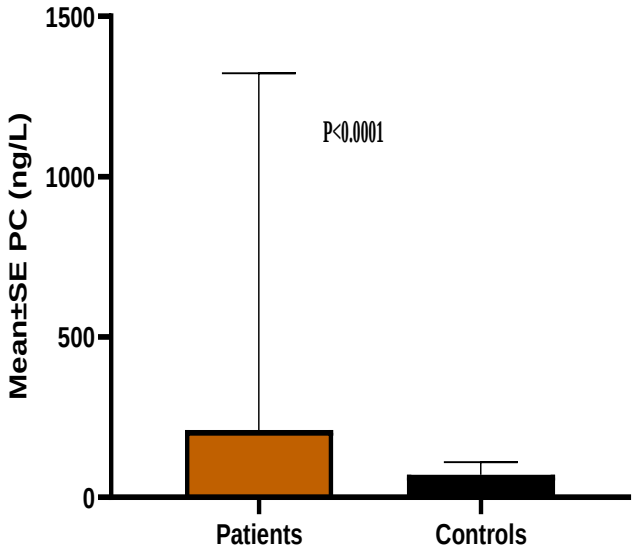

Figure 4. Comparing the Serum Protein Carbonyl (PC) Levels in Patients and Controls

Correlation between MCP -1 and protein oxidation markers

The relationship between MCP-1 levels and the biochemical markers that were measured is displayed in Figures 5, 6, and 7. The results, as shown in Figure 5, indicated that there was no statistically significant positive connection between 3-Nitrotyrosine (3-NT) and serum MCP-1 (r=0.087; P=0.307). Nevertheless, as shown in Figures 6 and 7, serum MCP-1 and PC and AOPP showed highly significant positive connections, with correlation coefficients of r=0.442; P=0.046 and r=0.627; P<0.0001, respectively.

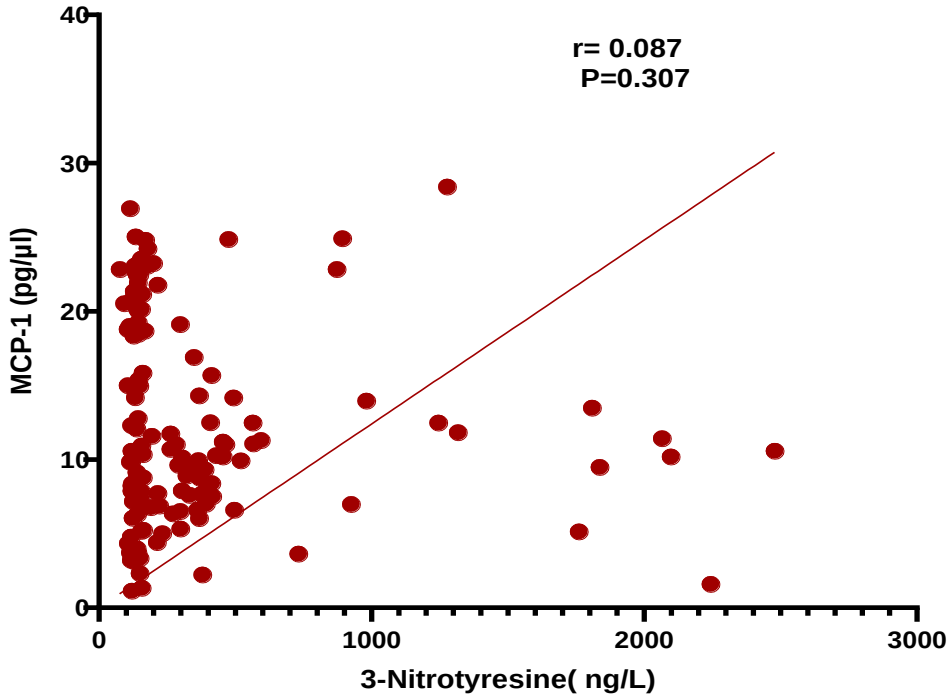

Figure 5. Serum MCP-1 vs. 3-NT levels in patients with periodontitis: a scatter plot

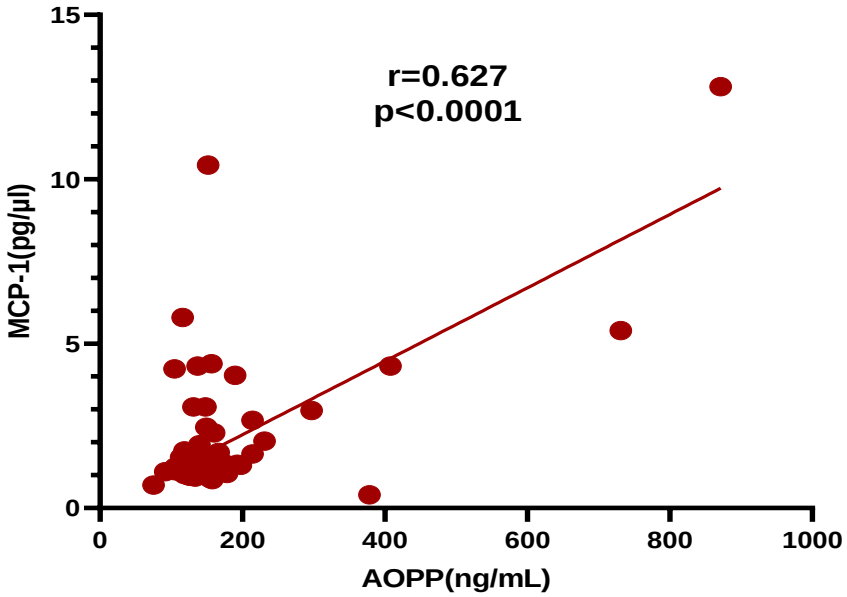

Figure 6. Serum MCP-1 vs. AOPP levels in patients with Periodontitis: a scatter plot

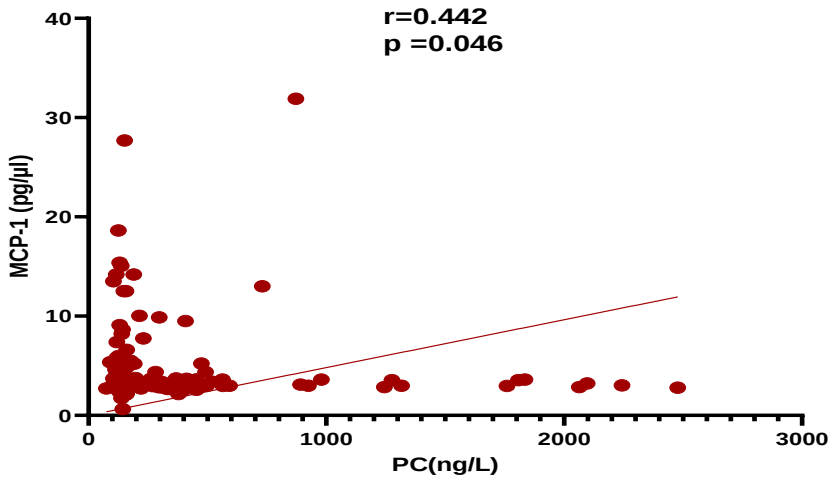

Figure 7. Serum MCP-1 vs. PC levels in patients with Periodontitis: a scatter plot

Serum MCP-1 levels and clinical markers are positively correlated in chronic periodontitis patients. Oxidative stress is a major contributor to the development of periodontitis. Anaerobic Gram-negative bacteria become the dominant population in the oral microbiome, which leads to the start of periodontitis [37]. Oxidative stress, which results from this microbial shift, directly harms cells and tissues, producing more damages to tissue and triggering inflammatory responses that exacerbate microbial dysbiosis [38]. Together, these elements—oxidative stress, lipopolysaccharides (LPS), and the transition to an anaerobic Gram-negative bacterial community—cause persistent inflammation and worsen periodontal disorders [39]. These studies show a reciprocal association between oxidative stress and periodontitis, whereby oxidative stress exacerbates damage to periodontal tissues and influences the development of periodontitis. The predominant inflammatory cells that build up in gingival sulcus and periodontal tissue are neutrophils, which are produced when periodontal pathogenic bacteria in biofilm trigger host defensive responses [40]. In periodontitis, it is believed that neutrophils are the primary generator of ROS [41]. Through the NADPH oxidase pathway, neutrophils can create excess ROS when they phagocytose periodontal pathobionts [42]. However, ROS are difficult to detect and have a relatively short half-life. Thus, the best ways to ascertain how oxidative stress-related events impact the pathological process of periodontitis are through ROS-related degradants and enzymatic and non-enzymatic antioxidant activity [43]. The onset of periodontitis is tightly linked to changes in the local concentrations of oxidative stress indicators. Periodontitis can be diagnosed and treatment efficacy evaluated using the oxidative stress biomarker level [41].

Limitations

Although our study provides insight into the serum levels of MCP-1 and protein oxidation products, as well as the possible correlation between these biomarkers and levels in periodontitis patients, some cross-sectional study limitations limit the ability to make predictions. Our sample size is relatively small, which prevents us from conclusively establishing the association of these pertinent biomarkers (protein oxidation products) with monocyte chemoattractant protein 1 or correlating them with resident periodontal infection. Additionally, confounding factors (food habits). Our data may have been impacted by dietary practices that are controlled by lifestyle factors, which are important for health because they regulate inflammation and oxidative stress. However, our results may help guide future studies and medical procedures.

Conclusion

It is important to look at MCP-1 as a possible risk factor for severe chronic periodontitis, as the current study highlights. According to our research, periodontal disease may be one of the primary effects of protein degradation pathways. The levels of serum advanced oxidation protein products (AOPP) can be utilized as a biomarker to identify protein damage brought on by oxidative stress. It has been demonstrated that there is a considerable positive correlation between the measures of monocyte chemoattractant protein-1, AOPP, 3-Nitrotyrosine (3-NT) and protein carbonyl (PC). The mechanisms behind inflammatory states in periodontal illnesses may be revealed by this connection. However, more research in the form of longitudinal prospective studies is required to corroborate the findings of our analysis. The goal of future treatment approaches should be to reduce

periodontal damage by focusing on oxidative stress pathways. Potential tactics that could break the inflammatory-oxidative cycle in periodontitis include antioxidant treatments (such as vitamin E or N-acetylcysteine) or measures to lessen MCP-1-mediated monocyte recruitment. To determine if these strategies are effective in slowing the advancement of the disease, clinical trials assessing them are necessary.

Acknowledgments

Our sincere gratitude is extended to the Erbil City dental clinic personnel for providing the samples.

Conflict of Interests

No conflicts of interest are disclosed by the authors.

Funding

There was no institutional funding for this study; BioVision merely supplied the ELISA kits.

Author's Contributions

Hazhar M. Balaky wrote and edited the first draft, and Parween Abdulsamad Ismail helped with the research, data gathering, and critical evaluation. The study's concept was influenced by both authors, with Parween offering final approval and project management.

Ethical Approval

The research was permitted by Ethics Committee of Nursing Department - Mergasor Technical Institute Council/ Erbil Polytechnic University. Issue Number (11198), Date: 12/11/2023. Ethical consent statements were obtained for all participating persons.

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تقييم الأهمية السريرية لبروتين الجاذب الكيميائي الوحيد ١ ومنتجات أكسدة البروتين في المرضى الذين يعانون من التهاب اللثة المزمن

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الخلاصة

إن الكيموكين الأساسي المسمى بروتين الجاذب الكيميائي الوحيد المسؤول عن بدء ومراقبة وتعبئة الوحيدات إلى مواقع التهاب اللثة الحاد النشط. الغرض من الدراسة الحالية هو تقييم مستويات مصل بروتين الجاذب الكيميائي الوحيد ومنتجات أكسدة البروتين لدى مرضى التهاب اللثة المزمن، و منتجات بروتين الأكسدة المتقدمة، نيتروتيروسين، وبروتين كاربونيل. تقييم العلاقات المحتملة بين مستويات بروتين الجاذب الكيميائي الوحيد والمنتجات الثانوية لأكسدة البروتين. شارك في البحث الحالي ٤٥ مريضاً يعانون من التهاب اللثة المزمن الشديد و40 فرداً سليماً. تم استخدام مقايسة الامتصاص المناعي المرتبط بالإنزيم لقياس كميات بروتين الجاذب الكيميائي الوحيد ومنتجات أكسدة البروتين. باستخدام البرنامج الإحصائي، تم إجراء اختبارات الارتباط والانحدار الخطي. كانت مستويات مصل بروتين الجاذب الكيميائي الوحيد منتجات بروتين الأكسدة المتقدمة، نيتروتيروسين ، وبروتين كاربونيل اعلى بكثير في مجموعة التهاب اللثة مقارنة بالمجموعة الضابطة. أظهرت مقاييس أكسدة البروتين و بروتين الجاذب الكيميائي الوحيد وجود علاقة إيجابية. يشار إلى أن المستويات المرتفعة من أكسدة البروتين و بروتين الجاذب الكيميائي الوحيد يمكن أن تكون علامة حيوية محتملة لاضطرابات اللثة وأن هذه المستويات قد تلعب دورًا مهمًا أثناء التهاب اللثة .

الكلمات المفتاحية: بروتين جاذب كيميائي وحيد الخلية -١، نيتروتيروسين ، الإجهاد التأكسدي، التهاب اللثة، بروتين كربونيل