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Original Article

Estimation of Monocyte Chemoattractant Protein-1 (MCP-1) Levels as an Inflammatory Cytokine in Chronic Generalized Periodontitis Patients

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ABSTRACT

Background: Monocyte chemoattractant protein-1 (MCP-1) performs assignment of inflammatory cells by triggering the chemotaxis of monocytes and also various cellular events related with chemotaxis. Its increased GCF, saliva and serum levels in periodontal disease have been reported in previous studies. The present study is aimed to evaluate the part of MCP-1 in periodontal disease continuation and also to find out the effect of periodontal treatment on MCP-1 concentration in serum and saliva. **Materials and Methods:** A total of 60 subjects were divided into three groups (n = 30): chronic periodontitis pre scaling and root planning (group I), chronic periodontitis consisted of 30 subjects from group I, 4-6 weeks after treatment (group II) and healthy control (group III). Clinical parameters evaluated were gingival index (GI), oral hygiene index-simplified (OHI-S), probing pocket depth (PPD), clinical attachment level (CAL), and serum and saliva samples collected from each patient were quantified for MCP-1 using ELISA. **Results:** The mean MCP-1 concentration in serum and saliva was found to be the highest in group I, i.e. 160.54 pg/ml and 444.62 pg/ml respectively. The mean MCP-1 concentration in group II was 81.79 pg/ml and 262.98 pg/ml and in group III, it was 43.43 pg/ml and 151.91 pg/ml respectively. **Conclusion:** MCP-1 level in serum and saliva rise with advancement of disease and decline after phase I therapy. Due to its certain correlation with clinical parameters, it can be examined as an inflammatory biomarker in periodontal disease. Hence, MCP-1 needs to be further reviewed as a therapeutic target.

Keywords: Chemokine, Chronic generalized periodontitis, Inflammatory biomarker, Monocyte Chemoattractant Protein -1 (MCP-1), Non-surgical periodontal therapy.

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INTRODUCTION

In India, periodontal infections are among the most prevalent chronic inflammatory illnesses. According to Chandra *et al.*[1] periodontal disease affects 95% of the population in India. Gingivitis and periodontitis are two frequent periodontal disorders.

Gingivitis is a moderate form of periodontal disease that causes irritation, redness, and swelling of the gingiva, the area of the oral mucosa that covers the alveolar processes of jaw and surrounds the necks of the teeth.[2] Gingivitis can progress to periodontitis, a more serious gum disease that can result in tooth loss.

The three biological phases of biochemical signaling, or the host immune response are inflammation, connective tissue deterioration, and alveolar bone turnover i.e involved in the periodontal inflammatory disease process.[3] The generation of cytokines, which increases inflammatory processes to play a key role in leukocyte recruitment and may directly or indirectly affect osteoclast formation, is an intermediary step between bacterial stimulation and tissue death. It has been found that there is increased level of circulating molecules in these biological phases, in the patient's whole saliva, with periodontal disease.[4]

Saliva has a lot of potential as a diagnostic fluid because of its non-invasive collection method, lower sample aliquots, cost efficiency, and ease of storage and transit compared to other biological fluids.[5] However, new research suggests that individuals with periodontitis have higher levels of systemic inflammation than controls, as seen by higher blood levels of several inflammatory markers.[6] In light of these findings, it is sense to investigate the levels of these markers in the peripheral circulation and entire saliva of patients with periodontal disease.

The immune system responds to the occurence of subgingival microorganisms and their released compounds, implicated in periodontitis. This activation is not only harmful to these microorganisms, but it also harms the periodontium. A related group of cytokines and chemokines secreted continually by diverse immune cells drives the inflammatory process.[7] Chemokines have been studied as one of the mediators likely implicated in leukocyte migration to the periodontal environment. Chemokines are chemotactic cytokines that, unlike traditional leukocyte chemoattractants, cause the recruitment of well-defined leukocyte subsets, resulting in the creation of a particular inflammatory infiltrate.[8]

MCP-1 (monocyte chemoattractant protein-1), also known as small inducible cytokine A2 (SCYA2) or CCL2 (CC chemokine 2 ligand), is a well-characterized member of the CC chemokine family that was first recognised as a powerful monocyte chemoattractant. MCP-1 works primarily through its contact with the CCR2 receptor (CC chemokine 2 receptor).[9]

MCP-1 was found to be preferentially expressed in diseased periodontal sites and have a differential spatial distribution in periodontal tissues as it is revealed by fibroblasts, endothelial cells and mononuclear phagocytes in the inflammatory infiltrate. It is produced in response to different signals, typically tumor necrosis factor-alpha [TNF-α], interleukin- 1β [IL-1β], interferon-γ [IFN-γ], bacterial lipopolysaccharide present on

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cell wall of bacteria, platelet-derived growth factor [PDGF], and oxidized low-density lipoproteins.[10]

Apart from its expression at diseased site, MCP-1 has been included in the pathogenesis of various systemic diseases like atherosclerosis; diabetes mellitus; idiopathic pulmonary fibrosis; tumors; rheumatoid arthritis; osteoarthritis; and delayed type hypersensitivity reactions.[11]

Periodontal diseases may serve as risk factors or indicators for important medical outcomes related to various systemic conditions.[11] Hence, it becomes essential to find out the response of periodontal disease on systemic levels of inflammatory mediators as it may reduce the possibility of morbidity and mortality from systemic diseases. Thus, the present study aimed to estimate the levels of serum and salivary MCP-1 in healthy individuals and patients with chronic periodontitis, pre and post phase 1 therapy and to correlate their levels with the clinical parameters of periodontal disease.

MATERIALS AND METHODS

Study Population

The study population was selected from patients attending the outpatient section of the Department of Periodontics, People's College of Dental Sciences and Research Center, Bhopal, India. A total of 60 patients were selected. The study protocol was approved by the institutional ethical committee with the institutional ethical clearance number EC201913. It was compulsory for all participants to read and sign the consent form before inclusion in the present study. Serum and saliva samples were collected from chosen patients with chronic generalized periodontitis and healthy controls.

Sample size and groups

Patients were divided into three groups:

Group I (Pre scaling and root planing [SRP]): 30 patients which were systemically healthy and clinically diagnosed with chronic generalized periodontitis (CGP), at baseline.

Group II (Post SRP): 30 patients which were systemically healthy and clinically diagnosed with chronic generalized periodontitis (CGP), 6 weeks after phase I therapy.

Group III (Control): 30 patients with clinically healthy periodontium and systemically healthy as controls (C).

Inclusion criteria

- Patients having moderate to severe periodontitis affecting more than 30% of sites of test groups.
- Subjects with the age, 25-65 years and should be systemically healthy.
- Minimum 20 teeth should be present, excluding third molars.
- Subjects with GI >3, OHI-S >3, mean PPD ≥ 4mm, mean CAL ≥ 3mm and bleeding on probing.

Exclusion criteria

- Active smokers and tobacco users.
- Pregnant women and lactating females.
- Patients with aggressive periodontitis, gross oral pathology, xerostomia or salivary gland disorders and who had undergone phase I therapy in past 3 months, prior sample collection.
- Subjects on medication like contraceptives or any type of antibiotics.
- Subjects with any systemic conditions such as diabetes, hypertension, etc.

Parameters to be Measured

Clinical parameters which had to be measured pre and post non-surgical periodontal therapy, with the help of mouth mirror and UNC-15 probe, were:

a. Gingival Index (GI)
b. Oral hygiene index- Simplified (OHI-S)
c. Probing Pocket Depth (PPD)
d. Clinical Attachment Level (CAL)

Parameters to be evaluated in the laboratory by ELISA test were serum and saliva levels of MCP-1, at the baseline and post non-surgical periodontal therapy.

Sample collection method

Before performing any clinical examination, unstimulated whole saliva sample was collected into sterile collection vials according to Navazesh method.[12] 5ml blood sample collected by veni-puncture method was centrifuged and serum was separated. The samples were shifted to laboratory in CSRD (Centre for Scientific Research and Development) where it was be stored at -20°C and biochemical analysis was performed.

Estimation of MCP-1 Levels

All reagents, samples, and standards (EliKine™ Human CCL2 ELISA Kit) were brought to room temperature before use. All samples were diluted with Sample Dilution Buffer. 100 µl of standard or sample was added into appropriate wells and covered to incubate for 90 min at 37°C temperature. Plate was washed and aspirated twice. Then 100 µl of prepared antibody working solution was added to each well and incubation done for 1 h at room temperature. Plate was washed and aspirated thrice. Then 100 µl prepared Streptavidin solution was added and incubated for 30 min at room temperature. Plate was washed and aspirated 5 times. Then 90 µl of TMB (Tetra Methyl Benzidine) one-step substrate reagent was added to each well and incubation done for 10-20 min at room temperature. The reaction was ended by adding 50 µl of stop solution to each well and read at 450 nm in ELISA reader, immediately.

RESULTS

This study was conducted to evaluate the level of MCP-1 in serum and saliva of CGP patients; pre and post phase 1 therapy. A total of 60 patients were selected and were divided into three groups aged between 25- 65 years. (Table 1).

The data so collected was entered into MS excel and analyzed using SPSS version 20.0 (IBM; Chicago). Descriptive data was presented as mean + S.D. Kruskal Wallis test was applied to compare any statistical difference

Table 1: Comparison of participant's age among the diseased and control groups.

Group Mean	Age				**p value
	Minimum	Maximum	Standard Deviation		
Group I (Pre *SRP) and Group II (Post *SRP)	42.90	31.00	58.00	±6.42	0.108
Group III (Control)	39.87	28.00	52.00	±5.24	

*SRP: Scaling and Root Planing, **p >0.001 [Not significant]

Table 2: Comparison of clinical parameters and serum and salivary MCP-1 levels (pg/µl) among the diseased and control groups.

	Group						Kruskal Wallis statistics	**p value
	Group I (Pre *SRP)		Group II (Post *SRP)		Group III (Control)			
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation		
†GI	1.72	±0.34	0.77	±0.48	0.55	±0.16	51.240	<0.001
‡OHI-S	4.34	±0.64	2.34	±0.49	1.03	±0.22	78.789	<0.001
§PPD	3.23	±0.34	2.63	±0.33	1.81	±0.26	71.44	<0.001
¡CAL	4.23	±0.49	3.17	±0.41	2.98	±0.64	45.357	<0.001
Saliva	444.62	±34.64	262.98	±15.74	151.91	±12.52	79.125	<0.001
Serum	160.54	±15.70	81.79	±8.29	43.43	±6.93	79.123	<0.001

*SRP- Scaling and Root Planing, †GI: Gingival index, ‡OHI-S: Oral hygiene index- Simplified, §PPD: Probing Pocket Depth, ¡CAL: Clinical Attachment Level, **p <0.001 [Highly Significant]

Table 3: Pearson correlation test among Saliva and Serum MCP-1 levels.

		Saliva	Serum	**p value
Saliva	Pearson Correlation	1	0.962**	
	Sig. (2-tailed)		0.000	<0.001
	N	90	90	
Serum	Pearson Correlation	0.962**	1	
	Sig. (2-tailed)	0.000		<0.001
	N	90	90	

**p<0.001 [Highly Significant]

between groups for clinical parameters, MCP-1 level in serum and saliva. Pearson correlation test was used to find any relation between serum and salivary MCP-1 level among all the groups. A significance level of 5% was set ($p < 0.05$).

The mean GI of the participants among Group I was 1.72, Group II was 0.77 and in Group III, the estimated value was 0.55. The mean OHI-S of the participants among Group I was 4.34, Group II was 2.34 and in Group III, the estimated value was 1.03. The mean PPD of the participants among Group I was 3.23mm, Group II was 2.63mm and in Group III it was 1.81mm. The mean CAL of the participants among Group I was 4.23mm, Group II was 3.17mm and in Group III it was 2.98mm. There was a statistically significant difference found among the diseased and control groups ($p < 0.001$) regarding all the clinical parameters. (Table 2).

The mean salivary MCP-1 level among Group I was 444.62 pg/µl, Group II was 262.98 pg/µl, and in Group III it was 151.91 pg/µl. The mean serum MCP-1 level among Group I was 160.54 pg/µl, Group II was 81.79 pg/µl, and in Group III it was 43.43 pg/µl. There was a statistically significant difference found among the diseased and control groups ($p < 0.001$). (Table 2).

Pearson correlation test showed a significant correlation of salivary MCP-1 levels with serum MCP-1 levels and vice versa. There was a statistically significant difference found ($p < 0.001$). (Table 3).

DISCUSSION

Various studies have illustrated the assertion of MCP-1 by the periodontal pathogens. Hanazawa *et al.*[13] stated that the cell components of *Porphyromonas gingivalis and Fusobacterium nucleatum* can induce marked gene expression of MCP-1 in the gingival tissues of patients with periodontal disease. Jiang and Graves[14] reported that *Aggregatibacter actinomycetemcomitans* induces synthesis of MCP-1 by mononuclear and bone derived cells. These MCP-1 produced, recruit circulating monocytes to the periodontal tissues, where upon presentation to various stimuli, including cytokines, they grow into macrophages. Products derived from the macrophages, such as IL-1 and TNF-α, besides presenting their pro-inflammatory properties, are known to induce bone resorption.[15] In this way, chemoattractants for monocytes/macrophages may contribute towards the maintenance of the chronic inflammatory reaction and progression of periodontal disease.

Saliva is commonly preferred to predict a person's periodontitis susceptibility. Saliva analysis, according to Costa *et al.*,[16] is a reliable and safe method for diagnosing and monitoring periodontal disease development. However, in order to recognize the influence of periodontal disorders on systemic illnesses, serum levels of inflammatory biomarkers must be assessed. It can be useful in determining the state of an illness and its development, as well as in offering therapeutic assistance. There is confirmation that periodontal infection is a problem that can affect the systemic physiology of alveolar bone.[17]

Sheridan[18] discovered that the incidence and severity of periodontal disease rises with the patient's age. (Table 1) The current study also showed that, as a patient's age grows, so does the prevalence and gravity of periodontal disease. According to Oliver and Tervonen,[19] altered collagen metabolism could predict the contribution to the advancement of periodontal disease.

The results of the present study showed that mean value of GI, OHI-S, PPD and CAL increased from periodontal health to chronic periodontitis and marked reduction in mean value observed after the non surgical periodontal therapy. These results are in accordance with studies done by Pradeep *et al.*,[20] Yilmaz *et al.*[21] and Nisha *et al.*[22] which showed that plaque and calculus could aggravate periodontal disease in comparison to those who maintain good oral hygiene. The reason behind this is the attachment loss between bone and tooth due to inflammation in the periodontal connective tissue, leading to apical migration of epithelial cells along the root surface, thus resulting in the establishment of deep pockets. The range of inflammatory infiltrate is in direct proportion with the depth of periodontal pocket.[23] After SRP, local irritating factors are eliminated due to which inflammation gets subsided and so the depths of periodontal pockets get decreased.

The mean salivary MCP-1 level shows a significant difference between the diseased and control groups. The results of this study are in accordance with the study done by Gupta *et al.*[24] and Ridha *et al.*[25] whose result showed increased level of MCP-1 in saliva of patients with chronic

periodontitis and reduction in its level after non-surgical periodontal therapy was performed. In our study, it was observed that the salivary levels of MCP-1 reduced from group I i.e chronic periodontitis patients toward group II i.e after SRP. When CGP patients were treated with SRP along with the maintenance of good oral hygiene protocols, the mean value of MCP-1 in saliva reduced after treatment. Also the positive correlation of decreased values of clinical parameters with reduced value of MCP-1, indicated its relation with the intensity of disease. This firmly recommends that any mediatory therapy planned to upgrade the periodontal status influence the degree of inflammatory markers in the oral micro-habitat.

The mean serum MCP-1 level shows a significant difference between the groups, but on comparing between the groups, Group I show higher value along with Group II as compared to Group III. The result is in accordance with the study done by Kurtis *et al*.[26] and Babu *et al*.[27] who reported higher levels of MCP-1 in serum in periodontitis patients when weigh up to healthy individuals. The developing disease in the periodontal pocket results in systemic inflammatory response which could be the possible cause for the elevated MCP-1 level in the serum. When CGP patients were treated with SRP along with the maintenance of good oral hygiene protocols, the mean value of MCP-1 in serum reduced after treatment but were still more than healthy control subjects.

The study showed a certain statistical correlation among the serum and saliva levels of MCP-1, with a highly significant p value of <0.001, among all the groups. Other studies which assessed this biomarker in saliva or serum have shown similar positive correlation (Pradeep *et al*.[20] and Emingil *et al*.).[28] The elevated level of MCP-1 in serum and saliva and its association with clinical parameters indicate their part in the pathogenesis of inflammatory periodontal disease.

Numerous studies have been conducted across the world to identify biomarkers for distinguishing the individuals with periodontal disease from periodontally healthy individuals. But the level of an inflammatory biomarker determined through a clinical study, can be helpful when its sensitivity and accuracy can differentiate and identify the particular disease. In terms of identifying periodontitis, MCP-1 had 100% sensitivity and specificity.[29] The results of present study indicate that serum and salivary concentrations of MCP-1 can discriminate periodontitis from health and patients with periodontitis who came back to clinical health continue to process inflammatory mediators for weeks after dental prophylaxis.

CONCLUSION

It is well documented that one of the many potential risk factors for systemic conditions is periodontal disease. Based on findings of this study, role of MCP-1 in the progression of periodontal disease can be recommended. Serum and salivary MCP-1 levels increased successively with the development of disease and reduced after nonsurgical periodontal therapy. Levels of MCP-1 associated positively with clinical parameters like GI, OHI-S, PPD and CAL and thus, it can be contemplated as an inflammatory biomarker in periodontal disease.

Further studies are needed to affirm the findings of our study. Future investigations are entitled to survey, if there is any related risk of other diseases due to increased serum and salivary MCP-1 levels in periodontal disease and also to inspect MCP-1 as a innovative therapeutic subject.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

C: Controls; **CAL:** Clinical attachment level; **CCL2:** CC chemokine 2 ligand; **CCR2 receptor:** CC chemokine 2 receptor; **GI:** Gingival Index

GI: Gingival index (GI); **INF:** Interferon; **MCP-1:** Monocyte chemoattractant protein-1; **OHI-S:** Oral hygiene index- Simplified; **OHI-S:** Oral hygiene index-simplified; **PDGF:** Platelet-derived growth factor; **PPD:** Probing pocket depth; **SCYA2:** Small inducible cytokine A2; **SPSS:** Statistical package for the social sciences; **SRP:** scaling and root planing; **TNF- α:** tumor necrosis factor-alpha.

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