Research Article

Assessment of serum levels of monocyte chemoattractant protein 1 (MCP 1) in patients with periodontitis and atherosclerotic cardiovascular disease

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Abstract: Background: Monocyte chemotactic protein-1 (MCP-1) is a chemokine expressed by inflammatory and endothelial cells. It has a crucial role in initiating, regulating, and mobilizing monocytes to active sites of periodontal inflammation. Its expression is also elevated in response to pro-inflammatory stimuli and tissue injury, both of which are linked to atherosclerotic lesions. Aim of the study: To determine the serum level of MCP-1 in patients with periodontitis and atherosclerotic cardiovascular disease in comparison to healthy control and evaluate the biomarker's correlations with periodontal parameters. methods: This study enrolled 88 subjects, both males and females, ranging in age from 36-66 years old, and divided into four groups: 1ST group with atherosclerotic cardiovascular disease (ASCVD) without periodontal disease (25 patients), 2nd group with periodontitis and systemically healthy, (25 patients),3rdgroup having both ASCVD and periodontitis (25 patients), and the 4th is the control group without any systemic disease and with good oral hygiene (13 subjects). The clinical periodontal parameters plaque index (PL I), Bleeding on probing (BOP), Probing Pocket depth (PPD) and clinical attachment level (CAL) were used to evaluate periodontal health status. Atherosclerotic cardiovascular disease patients were chosen after clinical examination by specialists and diagnoses confirmed with catheterization. Following clinical assessment, 5ml of venous blood was drawn from each participant MCP-1 levels in the blood were then measured using enzyme-linked-immunosorbent assay (ELISA). Results: According to the findings of this study, the mean values of PLI and BOP were higher in periodontitis group and athero+periodontitis group than in athero group and control group, PPD and CAL mean values were greater in athero+periodontitis group than in periodontitis group. The serum level of MCP-1 was higher in athero+periodontitis group than in athero, periodontitis and control groups. Regarding the correlations between MCP-1 and clinical periodontal parameters. In periodontitis group there was a positive correlation with PPD and CAL and there was a positive correlation with CAL in athero+periodontitis. Conclusion: This study revealed that periodontitis with higher MCP-1 level may be linked to an increased risk of atherosclerosis.

Keywords: Periodontitis, Cardiovascular, Plasminogen activators, Monocyte chemoattractant.

Introduction

Periodontal diseases (PDs) are inflammatory diseases that affect the tissues that support the teeth. Periodontitis is defined as a loss of supportive periodontal tissue, manifested clinically as clinical loss of attachment with gingival bleeding, and pocketing, while loss of alveolar bone is determined by radiography [(1)]. Atherosclerosis is a chronic inflammatory disease of the arteries caused by cholesterol accumulation and other physiological factors, and it is the leading cause of heart disease.

Cardiovascular disease (CVD) is the leading cause of death worldwide. CVD is anticipated to kill 17.3 million people per year, accounting for 30% of all fatalities, and this figure is expected to rise to 23.3 million

by 2030 owing to enhanced aging and obesity prevalence [2]. For over a century, there has been speculation that there is a link between dental health and CVD and according to epidemiological data, persons with periodontitis are more prone to develop the atherosclerotic disease than people without [3]. The investigation into potential links between periodontitis and atherosclerosis has intensified, and various correlations and causalities are being studied [4,5] Two publications looked at the scientific evidence for these associations: one looked at the function of invading microbes and infections [6], whereas the other is based on inflammatory processes [7]. This academic evidence now suggests that periodontitis leads to the entry of bacteria or their products into the circulatory system, which increases the host's inflammatory response through numerous pathways, favoring the formation and worsening of atheromatous lesions [8].

Monocyte chemoattractant protein-1 (MCP-1) is a strong factor for macrophage and monocyte chemotaxis, reflecting the very first identified. human CC chemokine with monocyte chemotactic functions [9]. A number of various cells, such as epithelial, endothelial, smooth muscle, astrocytic, mesangial, microglial, monocytic, and fibroblastic cells producing MCP-1 Following oxidative stress, particular cytokine or growth factor action which is either biologically derived or stimulated [9]. The key role of MCP-1 is the regulation of monocytes, macrophages, natural killer (NK) cells, and memory T lymphocyte recruitment and aggregation [10]. In atherosclerotic CVD, research findings have shown that MCP-1 promotes endothelial cell activity with chemokine receptors and enhances the vulnerability of plaques [11]. Furthermore; MCP-1 was shown to be selectively present at affected periodontal sites and displayed a distinct distribution pattern in the periodontium, as it is expressed in the inflammatory infiltrate along the basal layer of the oral epithelium and by endothelial cells, fibroblasts, and mononuclear phagocytes [12].

Material and methods

Study design

Eighty-eight subjects were selected according to inclusion and exclusion criteria in a period from March to July 2021. The study protocol was approved by the ethical committee of the College of the Dentistry/University of Baghdad follow the guidelines of Helsinki and Tokyo for humans (the reference no.249 in 18/2/2021). All the individuals were informed about the purpose of this investigation. Informed consent was obtained from each participant, and a questionnaire was used to record the background information. Subjects were divided into four main groups:

•Athero group, 25 Patients with Atherosclerotic Cardiovascular Disease (ASCVD) who have had catheterization for less than one year and are taking anticoagulant medicines (Plavix) and have stenosis of about fifty percent or more of at least one coronary artery and with healthy periodontium.

• Periodontitis group, 25 patients diagnosed to have periodontitis and didn't have any systemic disease. A patient is a periodontitis case in the context of clinical care with Interdental CAL is detectable at ≥2 non-adjacent teeth, or buccal or oral CAL ≥3 mm with pocketing >3 mm is detectable at ≥2 teeth [13].

•Athero+periodontitis group, 25 patients with both periodontitis and atherosclerosis,

•control group, 13 subjects with clinically healthy periodontium and no systemic disease, in which bleeding on probing less than 10% and Probing pocket depth of ≤ 3 mm [14]. This group represents baseline data for the serum level of MCP-1.

The exclusion criteria were: Patients with a history of another chronic, systemic disease with a known link with periodontitis, such as diabetes mellitus, rheumatoid arthritis, and so on, medication (anti-inflammatory or antibacterial medication) during the past three months and smokers, pregnancy, and contraceptive pills.

Examiner alignment

For inter-examiner calibration, the periodontal parameters (BOP, PPD, and CAL) for 5 subjects were measured by the researcher and the supervisor at the same time. The measurements were assessed using interclass correlation coefficient (ICC), and there was a good level of agreement for all parameters 0.785,0.760, and 0.753 respectively. For intra examiner calibration, the periodontal parameters (BOP, PPD, and CAL) for 5 subjects were measured twice by the researcher with a two-hour interval between the two measurements. There was a good level of agreement for BOP and PPD, and excellent for CAL [16].

Clinical assessment

Assessment of the periodontal status was carried out for all participants in which full mouth plaque score by O'Leary (1972) is used to detect presence or absence of plaque at four surfaces of each tooth (buccal, palatal/lingual, mesial & distal) by using a disclosing agent [15], full mouth bleeding on probing score recorded as present (1) or absent (0), probing pocket depth and clinical attachment level were recorded using a periodontal probe of William's (marking at 1,2,3,5,7,8,9 and 10 mm). PPD was measured from the gingival margin to the base of the pocket while CAL is the distance measured from cemento-enamel junction to the base of the pocket/sulcus at six sites per tooth.

MCP-1 measurement

5ml of venous blood was collected from each participant from the cubital fossa using 5ml plastic disposable syringe, then transferred into jell separating tubes, centrifuged for 5 minutes at (3000 RPM) and then sera were separated, then the tubes were labeled and stored at (-20°C) for later analysis by Enzyme-Linked Immuno-Sorbent Assay (ELISA) for the quantitative determination of serum MCP-1.

Statistical analysis

Data description, analysis, and presentation were performed using Statistical Package for Social Science (SPSS version 21) (Chicago, USA, Illinois). Mean, and standard deviation (SD) for nominal variables, also Inferential Statistics as Levene test, Pearson correlation (r), interclass correlation coefficient (ICC), two independent sample T-test, Shapiro Wilk and D'agostino Pearson test for the normality distribution of the quantitative variables, and One Way Analysis Of Variance (ANOVA) with Games-Howell posthoc test and Tukey Kramer HSD (according to levene's p-value) were used to analyze data.

Results

All studied variables were found to be normally distributed (parametric data) using Shapiro-Wilk and D'Agostino Pearson at ($p>0.05$). For PLI analysis, the mean value of PLI was found to be higher in athero+periodontitis group followed by periodontitis group then athero group, and lowest in the control group with a significant difference at P.value <0.05. While in BOP it was found to be higher in periodontitis group, followed by athero+periodontitis group, then athero group, and also lowest in control group with a significant difference at P.value <0.05 **table.1** Levene statistics=1.904, p value=0.135 NS, * = Significant at $p<0.05$. for PLI Levene statistics=15.375, p value=0.000 sig., *= Significant at $p<0.05$.for BOP

Following multiple pairwise comparisons (MPC), the only non-significant finding was found when comparing control group with athero group and periodontitis group with athero+periodontitis group while other results were significant, BOP showed the same results table 2. For PPD and CAL,

athero+periodontitis group showed a higher mean value and SD than periodontitis group with a significant difference between them at P.value <0.05 as shown in table 3.

Table 1: Statistical test of PLI and BOP among groups using One Way ANOVA.

Groups	**PLI (Mean±SD) of Score 1**	**P-value**	**BOP (Mean±SD) of Score 1**	**P-value**
Athero	25.080±8.411		5.080±1.656	
periodontitis	70.400±13.342		39.360±18.154	
Athero+periodontitis	70.640±11.139	0.000	35.440±15.594	0.000
control	24.538±13.956	Sig	5.154±2.853	Sig

Table 2: Inter groups multiple pairwise comparisons of the mean values of PLI and BOP.

(A) Groups	**(B) Groups**	**Mean Difference (A-B) of PLI**	**P-value**	**Mean Difference (A-B) of BOP**	**P-value**
Athero	**Periodontitis**	-45.3200	0.000 *	-34.2800	0.0000 *
	Athero+periodontitis	-45.5600	0.000 *	-30.3600	0.0000 *
	Control	0.5415	0.9991 ^	-0.0738	0.9998 ^
periodontitis	**Athero+periodontitis**	-0.2400	0.9999 ^	3.9200	0.8452 ^
	Control	45.8615	0.000 *	34.2062	0.0000*
Athero+periodontitis	**Control**	46.1015	0.000 *	30.2862	0.0000*

*=significant at p<0.05, ^=not significant at p>0.05

Table 3: A statistical test of PPD and CAL among groups using Independent Sample T- test.

Variables	**Periodontitis**	**Athero+periodontitis**	T-test	P-value
PPD (mean±SD)	4.759±0.727	5.169±0.583	2.202	0.032* Sig
CAL (mean±SD)	3.459±1.033	4.649±0.999	4.143	0.000* Sig

*=significant at p<0.05

Primary outcomes

The current study showed that MCP-1 was higher in athero+periodontitis group followed by athero group, and then periodontitis group, while lower in control group with a significant difference at P.value <0.05 table 4.

After doing MPC the only non-significant result was found when comparing athero group with periodontitis group while other findings were significant as shown in table 5. Concerning the correlation of MCP-1 and clinical periodontal parameters in the current study, there was a significant strong positive correlation with PPD (r=0.503; p=0.0104), and CAL (r=0.501; p=0.0107) in periodontitis group and there was a significant strong positive correlation with CAL (r=0.510; p=0.009) in athero+periodontitis group table 6.

Table 4: A statistical test of MCP-1 among groups using One Way ANOVA.

Groups	Mean	±SD	P-value
athero	83.857	33.742	
Periodontitis	82.433	26.499	0.000* Sig
Athero+peridontitis	148.177	51.693	
control	48.594	14.586	

Table 5: Inter groups multiple pairwise comparisons of the mean values of MCP-1 Multiple Comparisons (Games-Howell) Dependent Variable: MCP-1 (ng/L)

(A) Groups	(B) Groups	lean Difference (A-B)	Sig.
Athero	**Periodontitis**	1.4244	0.9983 ^
	Athero+periodontitis	-64.3200	0.0000*
	control	35.2632	0.0004*
Periodontitis	**Athero+periodontitis**	-65.7444	0.0000*
	control	33.8388	0.0001*
Athero+periodontitis	**control**	99.5832	0.0000*

Levene statistics=3.730, p value=0.014 Sig. *= significant at p<0.05
*=significant at p<0.05, ^=not significant at p>0.05

Table 6: Correlation between MCP-1 and periodontal parameters by each group..

MCP-1 (ng/ L)		r	p-value
Athero.	PLI	0.477	0.016 *
	BOP	0.215	0.302
	PLI	0.071	0.736
	BOP	0.079	0.709
	PPD (mm)	0.503	0.0104*
Periodontitis	CAL (mm)	0.501	0.0107*
	PLI	0.291	0.158
	BOP	0.318	0.121
	PPD (mm)	0.011	0.958
Athero+periodontitis	CAL (mm)	0.510	0.009*
	PLI	0.175	0.569
Control	BOP	-0.028	0.927

Discussion

The current study showed that the mean values of PPD and CAL in athero+periodontitis group were greater than in periodontitis group with significant difference between them. These results agreed with other previous studies [17-20]. This could be due to an increase in plaque and bacterial invasion with its toxin, which induces further damage of the alveolar bone tissue, sulcular and junctional epithelium, and other supporting tissue, ultimately increasing the supply of nutrients required for the multiplication of bacteria [21]. In terms of increased periodontal damage when atherosclerosis is present, there are two possible explanations: either a direct effect of atherosclerosis on the immune system, resulting in the release of enzymes and pro-inflammatory cytokines such as IL-1α, IL-6, MMPs, and TNF-α, or the effect of plaque accumulation. Endothelial dysfunction has also been linked to more periodontal destruction [22]. Also, the increased loss of periodontal attachment may be due to the host immune responses to the presence of pathogens leading to collagen and bone destruction by the production of cytokines from cells such as PMNs and monocytes that played an active role in inflammatory processes and were involved in numerous activities that would enhance the amount of damage. Furthermore, the existence of atherosclerosis would result in a rise in cytokine release, greater damage, and hence an increase in CAL levels [23]. Results as well showed that the mean value of serum MCP-1 was higher in athero+periodontitis group than in athero group, periodontitis group and the control group with highly significant difference between them this agreed with previous findings [21]. Regarding the increased level of MCP-1 with

periodontitis, the possible explanation is that Pathogens such as (P. gingivalis, Fusobacterium nucleatum, Aggregatibacter actinomycetemcomitans) may cause local production of MCP-1 by various cells at diseased periodontal sites, attracting circulating monocytes to the periodontal tissues, where they mature into macrophages upon exposure to various stimuli, including cytokines.

Macrophages are found in large numbers in inflamed gingival tissues in periodontal disease and are considered to have a key role in pathogen killing and the production of pro-inflammatory mediators and cytokines. In addition to that, IL-1 and TNF-α, which are produced by macrophages, are known to cause bone resorption in addition to their pro-inflammatory characteristics [24]. Regarding the increased level of MCP-1 with atherosclerosis, the observed increase of MCP-1 protein levels in the circulation of individuals with atherosclerosis can be attributed to at least two processes.1st , it might be a direct result of the progression of atherosclerosis. Endothelia and macrophage-like cells are the major sources of MCP-1, which are known to play a key role in the development of atherosclerosis and plaque formation. Endothelial cells can generate MCP-1 in response to low-density lipoprotein (LDL), a key atherosclerotic trigger factor, according to in vitro studies [25, 26].

2nd, elevated MCP-1 levels may reflect the progression of the inflammatory response in the heart and brain. MCP-1 and Macrophage inflammatory protein-1beta (MlP-lbeta) are two major chemokines that coordinate the infiltration of blood-derived monocytes and lymphocytes into the ischemic region, according to several research on animal models of stroke.

In addition, a significant positive correlation was found with PPD and CAL. Our results were in agreement with [27-29], The possible explanation for these findings is that in periodontal disease, MCP-1/CCL2 is thought to be the main chemoattractant for macrophages. Macrophages are abundant in inflamed gingival tissues and are hypothesized to play a key role in pathogen killing as well as the release of pro-inflammatory mediators such TNF-, IL-1, and nitric oxide [24, 30, 31]. These mediators also promote the cellular immune response, which could help with periodontopathogen control.

In contrast, macrophages produce inflammatory products that have been shown to promote osteoclast formation and maturation, which leads to bone resorption [31]. As a result, the attraction of macrophages by MCP-1 may result in increased periodontal disease severity, a theory confirmed by data revealing that more macrophages were detected in active periodontitis sites. MCP1/CCL2 is also linked to osteoclast chemotaxis and differentiation, most likely due to its interaction with the CCR2 receptor [32, 33]

Conclusion

According to the findings of this study the significant positive correlations between serum level of MCP-1 and clinical periodontal parameters (PPD and CAL) in both periodontitis and athero+periodontitis group and assuming that serum MCP-1 level rise proportionally to the severity of periodontal disease, and suggesting that MCP-1 could be used as an inflammatory biomarker in periodontal disease.

Conflict of interest: None.

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العنوان: تقييم مستويات مصل الدم للبروتين الجاذب لأحادي الخلية 1 في مرضى التهاب دواعم الاسنان و مرض التصلب العصيدي

الباحثون: صفا علي حمد , مها شكري محمود

المستخلص:

الخلفية: البروتين الجاذب لأحادي الخلية –1 هو مادة كيميائية يتم التعبير عنها بواسطة الخلايا الالتهابية والبطانية، ويلعب دورًا مهما في بدء وتنظيم وتعبئة الخلايا الاحادية في المواقع النشطة لالتهاب اللثة. يرتفع تعبيره أيضًا استجابةً للمنبهات المؤيدة للالتهابات وإصابة الأنسجة، وكلاهما مرتبط بآفات تصلب الشرايين.الأهداف: قياس المعلمات السريرية اللثوية مثل مؤشر الصفيحة الجرثومية ومؤشر النزف عند التسبير وقيم مؤشر عمق الجيوب وفقدان الانسجة الرابطة، قياس مستويات المصل للبروتين الجاذب لأحادي الخلية –1 باستخدام فحص المقايسة الامتصاصية المناعية للأنزيم المرتبط، في كل من مجموعات الدراسة ومجموعة الضبط، ومقارنة مستويات المصل للبروتين الجاذب لأحادي الخلية –1 في مجموعات الدراسة والضبط وربطها بالمعلمات السريرية اللثوية. الأشخاص، المواد وطرق العمل: ضمت هذه الدراسة 88 شخصا، ذكورًا وإناثًا، تراوحت أعماره من 36 إلى 66 عامًا، تم تقسيمهم إلى أربع مجموعات: المجموعة الأولى تضم 25 شخصا لديهم مرض التصلب العصيدي مع لثة سليمة، المجموعة الثانية تضم 25 شخصا لديهم مرض التهاب دواعم الاسنان وبدون أي امراض جهازية، المجموعة الثالثة تضم 25 شخصا لديهم كلا المرضين والمجموعة الرابعة تضم 13 شخصا لديهم لثة سليمة وبدون أي مرض جهازي مزمن. تم استخدام المعلمات السريرية اللثوية لتقييم الحالة الصحية للثة. وبعد التقييم السريري، تم سحب 5 مل من الدم الوريدي من كل مشارك وفصل مصل الدم لقياس مستوى البروتين الجاذب لأحادي الخلية MCP-1 باستخدام فحص المقايسة الامتصاصية المناعية للأنزيم المرتبط ELISA.النتائج: وفقًا لنتائج هذه الدراسة، فإن قيم مؤشر الصفيحة الجرثومية ومؤشر النزف عند التسبير كان أعلى في مجموعة 3 و2 منه في المجموعة 1 و4 .في المقابل. كانت قيم مؤشر عمق الجيوب وفقدان الانسجة الرابطة أكبر في المجموعة 3 منها في المجموعة 2، كما وجد ان مستوى البروتين الجاذب لأحادي الخلية في مصل الدم أعلى في المجموعة 3 منه في المجموعة 1و 2و 4، وفيما يتعلق بالارتباطات بين MCP -1 والمعلمات السريرية اللثوية. في المجموعة 2 كان هناك ارتباط إيجابي معنوي مع قيم مؤشر عمق الجيوب وفقدان الانسجة الرابطة وفي المجموعة 3 كان هناك ارتباط إيجابي معنوي مع مؤشر فقدان الانسجة الرابطة. الاستنتاج: كشفت هذه الدراسة أن مستوى البروتين الجاذب لأحادي الخلية في مصل الدم في المجموعة الثالثة كان أعلى بشكل ملحوظ منه في المجموعة الأولى والثانية. نتيجة لذلك، قد يرتبط ارتفاع مستوى البروتين الجاذب لأحادي الخلية المرتبط بالتهاب دواعم الاسنان بزيادة خطر الإصابة بتصلب الشرايين.