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Research Article

The Role of Serum MCP-1 in the Diagnosis and Assessment of Rheumatoid Arthritis

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Abstract

Background: RA is a chronic autoimmune disease characterized by synovial inflammation and joint destruction. Monocyte chemoattractant protein-1 (MCP-1) plays a key role in recruiting immune cells, contributing to both physiological immune responses and pathological conditions such as RA. The aim of this study is to evaluate the diagnostic and prognostic role of serum MCP-1 levels in patients with rheumatoid arthritis, particularly in relation to comorbid conditions such as diabetes mellitus.

Methods: A case-control study was conducted in Kerbala province between November 2024 and March 2025. It included 90 participants, comprising 20 rheumatoid arthritis (RA) patients with diabetes mellitus, 25 RA patients without diabetes, and 45 apparently healthy individuals as controls. Body mass index (BMI) was calculated, and family medical history was recorded. Laboratory measurements included serum levels of MCP-1 using Enzyme-Linked Immunosorbent Assay (ELISA), rheumatoid factor (RF), anti-cyclic citrullinated peptide (ACCP), and erythrocyte sedimentation rate (ESR).

Results: ACCP and RF levels were significantly elevated in RA patients, especially those with diabetes, compared to both non-diabetic RA patients and healthy controls. Obesity was also more prevalent among diabetic RA patients. ROC curve analysis showed excellent diagnostic performance of MCP-1 in distinguishing RA patients from controls, with high sensitivity and specificity.

Conclusions: The findings indicate that MCP-1, ACCP, and RF were valuable biomarkers for RA diagnosis and disease assessment, particularly in patients with metabolic comorbidities such as diabetes and obesity.

Keywords: Rheumatoid arthritis, Monocyte chemoattractant protein-1, inflammation, RF, ESR, DM

Introduction

Rheumatoid arthritis (RA) is a predominant systemic autoimmune inflammatory joint disorder. It is characterized by persistent joint inflammation, which may lead to cartilage and bone destruction, joint deformities, and functional disability if not properly managed [1]. The clinical manifestations of rheumatoid arthritis (RA) markedly vary between the early stages and the inadequately managed latter stages of this disease. Generalized signs of the disease, including fatigue, flu-like symptoms, inflamed and tender joints, and morning stiffness, indicate early-stage RA. These symptoms are accompanied by a higher level of C-reactive protein, also known as CRP, and a raised erythrocyte sedimentation rate (ESR) [2].

Over the past few decades, research has shown that innate immunity played a major role in the development and progression of rheumatoid arthritis [3]. The inflammatory response found in RA patients involves a variety of innate immune cells, such as macrophages, monocytes, and dendritic cells, and also triggers the activation of the adaptive immune system, which is essential in the later stages of the disease [4]. Autoantibodies are a characteristic of positive RA and a biomarker of an erosive condition of the joint surface, which means that the pathophysiology of rheumatoid arthritis may be diverse and present with various presentations [5]. Additionally, several changes may occur before the disease's clinical manifestation; these changes may last for years and be typified by the presence of autoantibodies without any indications of arthritis [6]. The generation of autoantibodies is a hallmark of RA, and these are produced by autoreactive B cells [7].

Autoantibody-positive and autoantibody-negative RA patients could be distinguished based on the presence of autoantibodies [8]. These autoantibodies are predominantly detected in the serum and synovial fluid (SF) of RA patients [9].

Cytokines are produced by specific cells and affect the function of many other cells [10-11]. They encompass chemokines that mediate chemotaxis, interleukins that coordinate leukocyte communication, interferons involved in innate immunity, lymphokines, and tumor necrosis factor (TNF), which exhibits potent proinflammatory activity [12]. The development of RA involves cytokine-mediated pathways, which are defined by the deregulation of anti-inflammatory cytokines and an elevated production of cytokines primarily with pro-inflammatory functions. This promotes the initiation of autoimmunity by triggering signaling pathways linked to chronic inflammation, excessive generation of autoantibodies, and effects on end organs [13-14]. The class of small chemotactic cytokines known as chemokines usually has a molecular weight of 8–15 kDa. Leukocyte migration during inflammation is regulated by chemokine ligand-receptor interactions, which encourage migration from the circulation into the extravascular space in inflammatory tissues [15].

Active monocytes and fibroblasts create Monocyte chemoattractant protein 1 (MCP-1) at inflammatory areas, which is crucial chemokine that controls monocyte and macrophage movement and infiltration. Synovial fibroblasts mostly release it in reaction to inflammatory cytokines like interleukin (IL)-1, interferon-γ, and tumor necrosis factor-α. MCP-1 not only draws in monocytes but also stimulates macrophages and monocytes, causing them to release IL-1 and IL-6 [16]. The MCP-1 is a beta chemokine that can be constitutively generated by endothelial cells, monocytes, synoviocytes, and fibroblasts, or it can be activated by growth factors, cytokines, or oxidative stress. By attracting monocytes to the site of inflammation that are stimulated to develop into macrophages in the synovial tissue, it plays a significant role in the immune system [17-18]. One of the primary features of RA is the migration of inflammatory cells mediated by chemokines into the joints [19]. Prior research has demonstrated that RA serum has a higher amount of MCP-1 expression than normal serum [19-20]. It has also been demonstrated that the disease activity score is linked to CCL2 levels and may be a useful biomarker for assessing the activity of RA disease [21].

The aim of the current study was to assess the role of serum MCP-1 in patients with rheumatoid arthritis as a diagnostic and prognostic marker.

Materials and Methods

Patients

A case-control study was conducted at Al-Imam Al-Hassan Al-Mujtaba Teaching Hospital between November 2024 to March 2025, A convenience sampling method was applied. A total of 90 participants were enrolled and categorized into three groups: group I was 20 patients diagnosed with rheumatoid arthritis (RA) and type 2 diabetes mellitus (T2DM), group II was 25 patients with RA without diabetes, and group III (control group) was 45 apparently healthy individuals with no history of autoimmune or metabolic diseases. The participant's age ranged between 30 and 70 years.

Clinical data and medical history were collected using a structured questionnaire and verified through patient interviews and medical records. Detailed information regarding disease duration, comorbidities, medications, and laboratory results was obtained for each subject. The hospital's ethics committee approved the study protocol.

Inclusion and exclusion criteria

Patients with RA have completely met the RA classification criteria of American College of Rheumatology (ACR)/European League against Rheumatism (EULAR) in 2010. The control group were healthy people without any rheumatologically disorder. Exclusion criteria included pregnant patient, patient with malignancy, and thyroid disorder.

Assay

The concentration of MCP-1 was determined using an Enzyme-Linked Immunosorbent Assay (ELISA) kit (BT LAB, China, CAT. NO E0124Hu). A microplate pre-coated with MCP-1 antibodies was used. Standards and samples were added to the wells to bind with the antibodies, followed by the addition of a biotinylated anti-MCP-1 antibody and Streptavidin-HRP. After washing to remove unbound substances, substrate solutions were added, and a colorimetric reaction was developed. The reaction was stopped with an acidic stop solution, and the optical density was measured at 450 nm using a microplate reader. The rheumatoid factor (RF) concentration was assessed utilizing a fluorescent sandwich immunoassay (FIA) method on the Ichroma™ analyzer (Boditech/Korea).

Hematological assays

Erythrocyte Sedimentation Rate (ESR) was measured by adding 1.5 ml of blood to a tube containing 3.8% sodium citrate, mixing thoroughly, and placing the tube vertically in a designated rack for 30 minutes. Afterward, the distance the red blood cells had sedimented was measured, and the result was recorded in millimeters per hour (mm/hr). The normal value of ESR is 0-15 mm/hr for males and 0-20 mm/hr for females.

Ethical approval

The ethics committee team provides the ethical approvals from the College of Medicine at the University of Kerbela and the Kerbela Health Directorates/Kerbela–Iraq, by reference number 20 on 14 April 2025.

Statistical analysis

With SPSS 22, the statistical analysis was conducted. A chi-square test was used to look at categorical variables. Independent ANOVA samples were used to analyze continuous normal distribution variables with a mean and a standard deviation for between-group comparisons. The determination of the ideal sensitivity and specificity threshold for critical cases was reached by executing receiver operating characteristic (ROC) analysis.

Results

The majority of participants in all study groups were female. Compared to 84.0% (21 out of 25) in the RA without DM group and 80.0% (36 out of 45), women accounted for 95.0% (19 out of 20) in the RA with DM group. Men made just 5.0%, 16.0%, and 20.0% in the corresponding groups.

According to Table 1, participants in the study were categorized into four age groups. The predominant age group among all participants was 51–60 years, which constituted 38.9% of the entire sample. In the RA with DM and RA without DM groups, 30.0% and 36.0% of participants, respectively, were classified in this category, whereas 42.2% of the control group fell within this range. Individuals under 39 years of age were the least represented, especially in the RA without DM group, which included no participants from this demographic.

A significant prevalence of obesity was noted among all categories. In the RA with DM group 70.0% of participants were categorized as obese, in contrast to 52.0% in the RA without DM cohort and 48.9% in the control group. The percentage of participants with a healthy BMI was minimal in the RA with DM group (5.0%) and maximal in the control group (11.1%). A positive family history was exclusively reported within the illness groups. In the RA with DM cohort, 55.0% indicated a familial history of RA, whereas 48.0% stated the same in the RA without DM cohort.

The current study examined various significant indicators across three groups: patients with rheumatoid arthritis (RA) and diabetes mellitus (DM), patients with RA without DM, and a control group. The findings indicate considerable disparity in the levels of these markers among the categories. The immunological and hematological test results for both patient groups and controls are demonstrated in Table 2.

Table 1: Demographic and clinical characterization

Variable	RA with DM (n=20)	RA without DM (n=25)	Control (n=45)	Total No (n=90)
Sex				
Male	1 (5.0%)	4 (16.0%)	9 (20.0%)	14 (15.6%)
Female	19 (95.0%)	21 (84.0%)	36 (80.0%)	76 (84.4%)
Statistical	X^2= 2.38; P> 0.05			
Age groups				
Less than 39 years	3 (15.0%)	0 (0.0%)	5 (11.1%)	8 (8.9%)
40–50 years	7 (35.0%)	9 (36.0%)	14 (31.1%)	30 (33.3%)
51–60 years	6 (30.0%)	9 (36.0%)	19 (42.2%)	35 (38.9%)
61 years and above	4 (20.0%)	7 (28.0%)	7 (15.6%)	17 (18.9%)
BMI groups				
Healthy weight	1 (5.0%)	2 (8.0%)	5 (11.1%)	8 (8.9%)
Overweight	5 (25.0%)	10 (40.0%)	18 (40.0%)	33 (36.7%)
Obesity	14 (70.0%)	13 (52.0%)	22 (48.9%)	49 (54.4%)
Family history				
Yes	11 (55.0%)	12 (48.0%)	0 (0.0%)	23 (25.6%)
No	9 (45.0%)	13 (52.0%)	45 (100%)	67 (74.4%)

RA: rheumatoid arthritis, DM: diabetes mellitus, BMI: body mass index

Table 2: Distribution of parameters among study groups

Variable	RA with DM (n=20)	RA without DM (n=25)	Control (n=45)	p-value
MCP-1 (ng/mL)	250.53 ± 39.42[a]	209.04 ± 49.82[b]	64.40 ± 57.60[c]	<0.01[S]
ESR (mm/hr)	33.50 ± 13.45	38.16 ± 11.80	13.80 ± 4.17	<0.01[S]
RF (IU/mL)	32.40 ± 14.22[b]	27.22 ± 11.15[b]	7.11 ± 3.89[a]	<0.01[S]
ACCP (U/mL)	135.38 ± 60.70[c]	97.19 ± 41.05[b]	2.15 ± 1.24[a]	<0.01[S]

RA: rheumatoid arthritis, DM: diabetes mellitus, BMI: body mass index, MCP-1: monocyte chemoattractant protein-1, RF: rheumatoid factor ACCP: anti-cyclic citrullinated peptide, ESR: erythrocyte sedimentation rate

Primarily, concerning MCP-1 levels, it was observed that individuals with RA and DM had an average level of 250.53 ng/mL, whereas it was 209.04 ng/mL in patients without DM. Conversely, MCP-1 concentrations in the control group were markedly low, measuring 64.40 ng/mL. The data demonstrated that both groups of RA patients exhibit increased MCP-1 levels, indicating heightened inflammatory activity. The ESR data showed that patients with DM had a rate of 33.50 mm/hr, whereas the average for the non-DM group was 38.16 mm/hr. The control group presented a rate of 13.80 mm/hr. The increased rates in both RA patient groups indicate active inflammation, while the differences between the groups are not statistically significant.

In RA patients with DM, rheumatoid factor levels were 32.40 IU/mL, compared to 27.22 IU/mL in patients without DM, while the control group demonstrated a low measurement of 7.11 IU/mL. The data demonstrated evident disease activity in both RA patient cohorts, with a tendency for elevated RF levels in those with DM.

Finally, anti-citrullinated protein antibodies (ACCP) averaged 135.38 U/mL in RA patients with DM, 97.19 in RA patients without DM, and 2.15 in the control group. These discrepancies indicate that RA patients, especially those with DM, had an active immune response.

Biomarker correlation

The results of the correlations among the studied biomarkers of RA patients were presented in Table 3. The correlation study of MCP-1 levels with various clinical and demographic characteristics identified many statistically significant associations. A considerable association was identified between MCP-1 and rheumatoid factor ($r = 0.674$, $P < 0.01$), indicating a common inflammatory mechanism influencing disease activity in RA patients. The ESR demonstrated a moderate to significant positive connection with MCP-1 ($r = 0.592$, $P < 0.01$), suggesting that MCP-1 may signify systemic inflammatory burden.

Furthermore, significant positive correlations were observed between ACCP antibodies and MCP-1 ($r = 0.553$, $P < 0.01$), suggesting a possible association between this chemokine and autoantibody-driven pathogenesis. Age exhibited a small yet significant correlation with MCP-1 levels ($r = 0.303$, $P = 0.043$), indicating a potential involvement of age-associated inflammatory mechanisms. Additionally, duration of disease showed a positive correlation with MCP-1 ($r = 0.755$, $P < 0.01$), potentially indicating a sustained elevation of inflammatory mediators throughout the progression of the disease. These data highlight the significance of MCP-1 as a prospective biomarker for disease activity and chronic inflammation, especially in RA patients with extended disease duration and complications.

Receiver-operating characteristic (ROC) curve analysis was applied to assess the diagnostic performance of serum MCP-1 in RA patients versus the healthy control group (Figure 1). At a cut-off value of 148.5 ng/mL, it showed high diagnostic accuracy, achieving a sensitivity of 93.3% and a specificity of 91%. The AUC was 0.97, demonstrating exceptional capability. The findings were statistically significant ($P = 0.000$), with a 95% confidence interval of 0.94 to 1.00 (Table 4).

Table 3: Correlation between Serum MCP-1 Levels and Clinical Parameters in rheumatoid arthritis patients

Parameter	MCP1 (r)	p-value
MCP-1 (ng/mL)	1	-
RF(IU/mL)	0.674	<0.01 [S]
ESR	0.592	<0.01 [S]
ACCP U/mL	0.553	<0.01 [S]
Age	0.303	0.043 [S]
Duration of disease	0.755	<0.01 [S]

MCP-1: monocyte chemoattractant protein-1, RF: rheumatoid factor ACCP: anti-cyclic citrullinated peptide, ESR: erythrocyte sedimentation rate

Table 4: AUC, Optimal Threshold, Sensitivity, and Specificity For MCP-1

Parameter	Cut-off points ng/mL	Sensitivity	Specificity	AUC	p-value	95% CL	
MCP-1	148.5	93.3%	91%	0.97	0.000	0.94	1.000

MCP-1: monocyte chemoattractant protein-1, AUC: receiver–operating characteristic

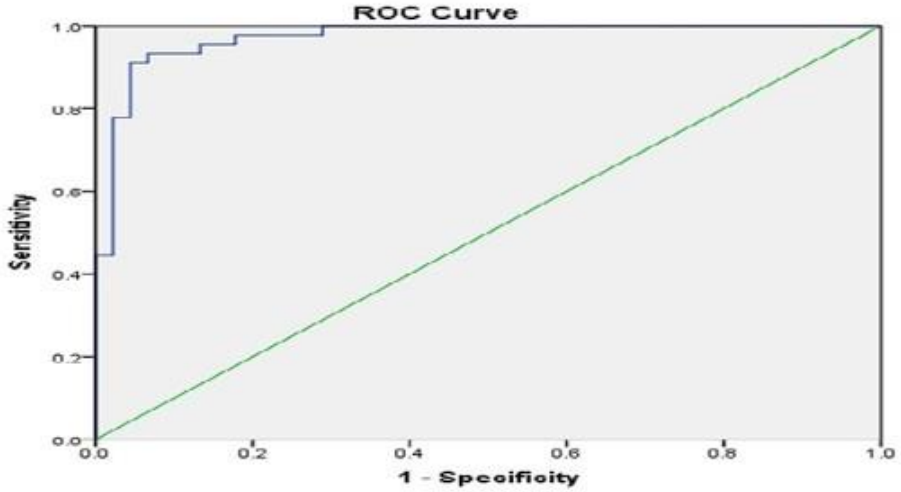

Figure 1: Receiver operating characteristics (ROC) curve for MCP-1 level in rheumatoid arthritis patients

Discussion

The present study's findings indicated that females were the predominant percentage in all groups, with almost 95% in the RA with DM group, 84% in the rheumatoid arthritis without DM group, and 80% in the control group. This finding aligns with the 2014 study conducted in Taiwan by Lu *et al.* (2014), which demonstrated that females with diabetes had a higher tendency to develop rheumatoid arthritis than males, highlighting a distinct sex disparity in this disease intersection [22]. And this also agreed with Tentolouris *et al.* (2018), which showed that all cases of diabetes-related rheumatoid arthritis were in women [23]. This supports the idea that immunological and hormonal factors could play a role in the higher incidence of this overlap in women.

The age distribution of the three groups in this study showed that the majority of cases occurred in middle age, particularly in the fifth and sixth decades of life, while the proportions in both younger and older age groups were comparatively lower. These results are consistent with earlier investigations by Al-Saleh *et al.* (2024), which found that rheumatoid arthritis cases were most frequent among individuals in middle age, with fewer cases reported among those under forty and over sixty [24]. According to research findings, 70% of rheumatoid arthritis patients with diabetes were obese, which was greater than the percentages of non-diabetic RA patients (52%) and the control group (48%). These findings demonstrated a strong correlation between rheumatoid arthritis and obesity, particularly in the presence of diabetes. Similar to previous research by Singh (2019), a study conducted in India demonstrated that RA patients with diabetes had significantly higher body mass index (BMI) levels compared to those without diabetes [25]. This finding supported the association between obesity and the increased prevalence of diabetes among RA patients, which also agreed with Hammoda *et al.* (2021), who concluded that overweight and obesity were frequent among Egyptian RA patients [26]. It was associated with high disease activity and extra-articular manifestations.

In this study, a significant elevation in the mean concentration of MCP-1 was observed among RA patients with diabetes, compared to RA patients without diabetes and the control group. This increase was statistically significant. This elevation in MCP-1 was interpreted as a result of its activation as part of the chronic inflammatory response associated with RA. MCP-1 is mostly secreted by immune and synovial cells and plays a crucial role in recruiting monocytes to the site of joint inflammation, thereby contributing to increased inflammatory activity and progressive tissue damage. Also, the presence of diabetes, which is characterized by a low-grade chronic

inflammatory state, was believed to stimulate MCP-1 production via common inflammatory pathways. Further, leading to higher levels of this chemokine compared to non-diabetic RA patients. These findings suggested that MCP-1 served as a possible common mediator between RA and diabetes in promoting chronic inflammation. These results were consistent with previous studies, such as Ellingsen *et al.* (2001), which reported the involvement of MCP-1 in joint inflammation severity [27], and Liou *et al.* (2013), which demonstrated a correlation between MCP-1 levels and the Disease Activity Score 28 (DAS28), highlighting the potential of MCP-1 as a therapeutic target in RA [28].

In the present study, both anti-cyclic citrullinated peptide (ACCP) and rheumatoid factor (RF) levels were significantly elevated in patients with RA compared to the control group. Specifically, ACCP levels were highest in RA patients with diabetes mellitus, followed by RA patients without diabetes, and lowest in the control group. Similarly, RF levels were elevated in RA patients with diabetes. These findings align with previous research [29], underscoring the diagnostic and prognostic significance of ACCP and RF in RA.

According to table 3, MCP-1 levels showed a clear positive association with several clinical markers of RA activity, such as RF, ESR, ACCP, patient age, and disease duration. These findings were consistent with previous research [30]. An Egyptian study demonstrated that serum MCP-1 levels were notably higher in RA patients than in healthy individuals and were positively correlated with disease activity markers like RF and DAS28-CRP. This supports the notion that MCP-1 could be a helpful biomarker for assessing inflammation and the course of RA. The correlation study of the serum MCP-1 in rheumatoid arthritis patients revealed a significant positive correlation between serum MCP-1 and ESR. These results are in agreement with Liou (2003), who recorded a significant increase in serum MCP-1 and ESR levels in RA patients compared to the control group and attributed these findings to the fact that RA patients frequently have inflamed synovial joints [31]. In this study, serum MCP-1 showed excellent diagnostic performance in distinguishing RA patients from healthy controls, with high sensitivity and specificity based on ROC curve analysis. These results align with previous findings [32], that also reported strong diagnostic accuracy for MCP-1, despite using a lower cut-off value.

Study limitations

This study has several limitations that should be acknowledged. First, the sample size was relatively small, particularly within the subgroup of RA patients with diabetes, which may affect the generalizability of the findings. Second, the study was conducted in a single geographical location (Karbala province), potentially limiting the applicability of results to broader populations with different genetic, environmental, or lifestyle factors. Third, a lack of genetic studies to be included in such a study.

Conclusions

The findings indicate that MCP-1, ACCP, and RF were valuable biomarkers for RA diagnosis and disease assessment, particularly in patients with metabolic comorbidities such as diabetes and obesity. These results highlight the importance of integrating metabolic evaluations in the clinical management of RA.

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