Baghdad Journal of Biochemistry and Applied Biological Sciences

2026, VOL. 7, NO. 1, 42–47, e-ISSN: 2706-9915, p-ISSN: 2706-9907

RESEARCH ARTICLE

Relationship of Monocyte Chemoattractant Protein-1 (MCP-1) with Some Biochemical Parameters in CKD-5 Stage Patients

Saif Bashar Mohammed[1*], Hameed Hussein Ali[1], Amer Jihad Hussein[2]

[1]*Department of Chemistry, College of Science, University of Anbar, Ramadi, Iraq.*
[2]*Iraqi Ministry of Health, Al-Anbar Health Department, Al-Ramadi Teaching Hospital, Ramadi, Anbar, Iraq.*

Article Info.

Keywords:
Biochemical markers, Chemokines, End-stage renal disease (ESRD), Inflammation, Monocyte chemoattractant protein-1.

Received: 06.26.2025
Accepted: 08.19.2025
Publishedonline: 01.09.2026
Published: 01.09.2026

Abstract

Background: Monocyte chemoattractant protein-1 (MCP-1) is a pro-inflammatory chemokine increasingly recognized as a biomarker in chronic kidney disease (CKD). Its novelty lies in linking inflammation with renal dysfunction and mineral metabolism disturbances. However, its clinical utility in advanced CKD is not fully established.
Objective: This study evaluated the association of serum MCP-1 with renal, metabolic, and nutritional parameters in end-stage renal disease (ESRD, CKD stage 5) patients undergoing hemodialysis, and assessed its diagnostic performance.
Methods: A case–control study included 58 ESRD patients undergoing maintenance hemodialysis and 30 matched healthy controls. Patients with acute infection, autoimmune disease, malignancy, and liver diseases were excluded. Blood samples were collected before dialysis to measure serum MCP-1, renal function markers, bone-mineral parameters, and nutritional indices. Statistical analyses included independent *t*-test, Pearson's correlation, and receiver operator characterization (ROC) curve analysis.
Results: MCP-1 levels were significantly elevated in ESRD, compared to controls (1090.69 ± 465.04 pg/mL vs. 195.33 ± 95.19 pg/mL, $P < 0.0001$). MCP-1 correlated positively with creatinine, urea, uric acid, phosphorus, and parathyroid hormone, and negatively with estimated glomerular filtration rate, calcium, vitamin D3, albumin, total protein, hemoglobin, and hematocrit ($P < 0.01$). ROC analysis revealed excellent diagnostic accuracy (area under curve: 0.99), with sensitivity = 94.8%, specificity = 96.7%, and an optimal cut-off of 382.5 pg/mL.
Conclusion: MCP-1 is markedly elevated in ESRD and strongly associated with renal dysfunction, mineral imbalance, and inflammation. Its high diagnostic accuracy supports its role as a candidate biomarker in advanced CKD, although larger prospective studies are needed to confirm prognostic value and therapeutic implications.

1. Introduction

End-stage renal disease (ESRD) is the end stage of chronic kidney disease (CKD), as less than 15% of the kidney conducts the process of filtration. A decrease in filtration is associated with multiple comorbidities such as hypertension, anemia, malnutrition, bone disease, neuropathy, and impaired quality of life, when function of the kidney decreases to a level that patients cannot survive without renal replacement therapy, dialysis, or kidney transplantation [1], severe CKD is associated with major disturbances in mineral and bone metabolism collected under the term of CKD–mineral and bone disorder (CKD–MBD) that strongly contribute to skeletal complications and cardiovascular risks [2].

As a member of chemokine family, monocyte chemoattractant protein-1 (MCP-1), also called chemokine ligand 2 (CCL2), is involved in regulating innate immunity and tissue inflammation [3]. MCP-1 is one of the first chemokines to be cloned, and its importance in renal inflammatory disease is

*Corresponding author: Saif Bashar Mohammed: sai22s3011@uoanbar.edu.iq

How to cite this article: Mohammed, SB., Hussein, HA., Hussein, AJ. Relationship of Monocyte Chemoattractant Protein-1 (MCP-1) with Some Biochemical Parameters in CKD-5 Stage Patients. Baghdad Journal of Biochemistry and Applied Biological Sciences, 2026, VOL. 7, NO. 1, 42–47. https://doi.org/10.47419/bjbabs. v7i1.420

significant. MCP-1 is also a major chemokine to send call for monocytes to come forth from the bone marrow [4]. MCP-1 is secreted by activated cells, such as macrophages, lymphocytes, renal tubular epithelial cells, endothelial cells, and fibroblasts. MCP-1 is a vital chemokine in the control of biological functions in monocytes/macrophages. It affects a lot of inflammatory processes involving the kidneys. MCP-1 has caused considerable strides as a biomarker for kidney disease among different diseases [4].

It is well known that serum creatinine, urea, phosphate, calcium, parathyroid hormone (PTH), vitamin D, and total protein concentration as well as albumin levels, which are all biochemical markers, are essential to assess functioning of the kidney, bone (metabolism), and the (overall) health of ESRD patients. These biochemical parameters are often disturbed in ESRD and may be responsible for modifications in MCP-1 levels. The association of MCP-1 with these biochemical indices may provide clues to the potential role of MCP-1 as a predictor for ESRD-related complications [5].

Diabetes, the leading cause of ESRD, is associated with chronic hyperglycemia-induced activation of nuclear factor kappa B (NF-κB) and oxidative stress pathways, which markedly upregulate MCP-1 expression in renal and vascular tissues [6]. Accordingly, diabetic ESRD patients may exhibit higher MCP-1 levels than non-diabetic counterparts, independent of severity of renal dysfunction. Furthermore, medications widely used in ESRD, including statins, renin–angiotensin system blockers, and vitamin D analogs, have demonstrated anti-inflammatory properties that may attenuate MCP-1 production [7].

The present study is designed to examine the relationship between serum MCP-1 levels and multiple biochemical parameters in patients with ESRD on hemodialysis. To our knowledge, studies have not elucidated how these associations relate to one another or how they may influence one another in ESRD. We aim to examine these associations to inform a better understanding of MCP-1 function in ESRD and its possible clinical implications.

2. Materials and Methods

This case–control study included 58 patients (28 males and 30 females), aged 19–75 years; patients who provided blood in pre-dialysis condition were included in the study. It was carried out in the hemodialysis unit of Al-Ramadi Teaching Hospital, Al-Anbar, Iraq, from December 27, 2024 to February 8, 2025. In all, 30 healthy subjects (15 males and 15 females), aged 35–71 years, were included in the control group. The selection of control group was based on the medical history of each participant. The study was approved by the ethical committee, University of Anbar, Iraq, on December 26, 2024, with ID 270.

2.1. Collection of blood samples

A total of 5-mL venous blood was obtained from fasting patients and control subjects, and blood of all the patients was collected prior to dialysis. Blood was separated into two tubes: blood in the first tube (1 mL) was added to an EDTA tube for Hb and hematocrit (Hct) determination, and blood in the second tube (4 mL) was put into a gel tube for serum extraction. The blood was centrifuged at 2,500 ×*g* for 10 min after clotting at room temperature (18–25°C). The supernatant was separated into two volumes for biochemical parameter measurement (renal function by Abbott device; vitamin D and PTH were measurement by Cobas e411 device that followed the ECLIA technology). The rest of sera was stored at -20°C in order to measure serum MCP-1 using the enzyme-linked-immunosorbent serologic assay (ELISA) kit (ID ELK5252; ELK Biotechnology, China).

2.2. Inclusion and exclusion criteria

The inclusion criteria in the study for CKD stage 5 patients were proteinuria, hematuria of renal origin, hydronephrosis, clinical features, such as fatigue, anorexia, nausea, and vomiting, anemia, and bone-mineral disorders. The exclusion criteria were the patients under or above study design age, having normal renal function, liver diseases, acute infection, autoimmune disease, and malignancy.

2.3. Statistical analysis

Analyses were performed with SPSS-28. Results were reported as mean and SD. Differences between two independent mean values were examined using Student's *t*-test, and $P \leq 0.05$ was considered statistically significant. Bivariate relationships were also assessed using Pearson's correlation coefficient. Area under curve (AUC) and receiver operator characterization (ROC) were performed by SPSS with 95% confidence interval (CI) to determine the specificity and sensitivity of MCP-1 between study groups.

3. Results

The study results found that hypertension was the main cause of ESRD in 32/58 patients (55.17%), but the second cause of ESRD was diabetes mellitus in 20/58 patients (34.48%), and other reasons in 6/58 patients (10.35%) (see Table 1).

The investigation was performed in order to demonstrate the correlation between MCP-1 and other parameters in patients with end-stage renal failure. Table 2

Table (1): Causes of ESRD in study patients.

Causes	Number of patients	Percentage (%)
Hypertension	32	55.17
Diabetes mellitus	20	34.48
Other reasons	6	10.35
Total	58	100

Table (2): Measured variables in the patient and control groups (mean ± SD).

Variable	Unit	Patient group (mean ± SD)	Control group (mean ± SD)	*P* value
MCP-1	pg/mL	1090.69 ± 465.04	195.33 ± 95.19	<0.0001*
Creatinine	mg/dL	7.15±1.69	0.79±0.13	<0.0001*
Urea	mg/dL	155.38±37.26	31.80±2.95	<0.0001*
Uric acid	mg/dL	7.036 ± 1.29	4.307 ± 0.97	<0.0001*
eGFR	mL/min/1.73 m^2	8.28 ± 2.16	111.53 ± 13.50	<0.0001*
Albumin	g/dL	3.13 ± 0.26	3.89 ± 0.33	<0.0001*
Total protein	g/dL	5.81 ± 0.71	6.90 ± 0.34	<0.0001*
Ca^{+2}	mg/dL	7.73 ± 0.54	9.08 ± 0.48	<0.0001*
D3	ng/mL	11.19 ± 3.78	33.56 ± 12.47	<0.0001*
PTH	pg/mL	311.3 ± 138.1	43.64 ± 18.02	<0.0001*
PO_4^{-3}	mg/dL	5.33±0.89	3.23±0.49	<0.0001*
Hb	g/dL	8.59 ± 1.22	13.31 ± 1.61	<0.0001*
Hct	%	25.17 ± 3.38	42.14 ± 5.17	<0.0001*
Age	Years	54.8 ± 15.9	53.1 ± 10.4	0.597

P values from paired *t*-test.
PTH: parathyroid hormone; Hct: hematocrit; Hb: hemoglobin.

presents the basic experimental information of all subjects. The estimated glomerular filtration rate (eGFR; mL/min) in the patient group with ESRD was severely low (8.28±2.16), which was significant, compared to the control group (111.53±13.50; $P < 0.001$), indicating significantly high differences between patient and control groups. The levels of Hb and Hct were markedly lower in ESRD patients than those in the control group (Hb: 8.59 vs. 13.31 g/dL, and Hct: 25.17 vs. 42.14%). These results indicate that hemoglobin (Hb) and hematocrit (Hct) levels were significantly lower in ESRD patients compared with the control groups. Serum levels of calcium, vitamin D3, total protein, and albumin were significantly reduced in ESRD patients, compared with the control subjects ($P < 0.05$). The P values indicate statistically significant differences between the two study groups. The mean values of calcium, vitamin D3, total protein, and albumin were 7.7 vs. 9.08 mg/dL, 11.19 vs. 33.56 ng/mL, 5.81 vs. 6.90 g/dL, and 3.13 vs. 3.89 g/dL, respectively. Compared to the control, significant increase was observed ($P = 0.0001$) in the levels of serum of MCP-1 (1090.69 vs. 195.33 pg/mL), creatinine (7.15 vs. 0.79 mg/dL), urea (155.38 vs. 31.80 mg/dL), uric acid (7.036 vs. 4.307 mg/dL), parathyroid (311.31 vs. 43.64 pg/mL), and phosphorus (5.33 vs. 3.23 mg/dL) in ESRD patients.

The findings of the present study indicated a positive association of MCP-1 with urea, creatinine, uric acid, phosphorus, and PTH, and showed that MCP-1 had no correlation with eGFR, D3, Ca, Hb, Hct, total protein, and albumin, as indicated in Table 3.

The AUC for MCP-1 was 0.99, showing good diagnostic power. There was a specificity of 96.7%, sensitivity of 94.8%, and a cut-off value of 382.5 (Table 4 and Figure 1). This significant finding indicates the validity of MCP-1 as a biomarker for the diagnosis of end-stage renal failure.

4. Discussion

The present study was designed to investigate the serum levels of MCP-1 and its interaction with diverse biochemical parameters in patients with ESRD (i.e., CKD stage 5). The findings demonstrated that the levels of MCP-1 were significantly increased in ESRD patients who were on hemodialysis, compared to the control subjects. These results were consistent with the increasing evidence that MCP-1 is an essential factor in renal inflammation and disease. The results of the current study agreed with the results of other studies [8–10].

MCP-1 (CCL2) plays a central role in the progression of ESRD by forcing chronic renal inflammation and fibrosis. In uremic conditions, glomerular and tubular epithelial cells, endothelial cells, and infiltrating leukocytes upregulate MCP-1 expression in response to oxidative stress, advanced glycation end products, and pro-inflammatory cytokines, such as tumor necrosis factor-α (TNF-α) and interlukin 1β (IL-1β). Elevated MCP-1 binds to its receptor C-C chemokine receptor type 2 (CCR2) on circulating monocytes, promoting their recruitment into the renal interstitium. Once infiltrated, these monocytes differentiate into macrophages that secrete additional cytokines, growth factors, and reactive oxygen species (ROS), thereby amplifying local inflammation. Persistent MCP-1/CCR2 signaling sustains macrophage activation and enhances production of transforming growth factor-beta (TGF-β) and other pro-fibrotic mediators, which stimulate fibroblast proliferation and extracellular matrix deposition [6].

MCP-1 activates and differentiates osteoclasts, which are necessary for bone resorption. High MCP-1 levels in ESRD are likely to exacerbate bone loss via promoting osteoclastic activity, and this elevated MCP-1 reflects increased oxidative stress and inflammation, which

Table (3): Correlation between MCP-1 and studied variables in the patient and control groups.

Variables	Coefficient correlation	MCP-1 (pg/mL)
Age	*R*	0.024
	Sig. (2-tailed)	0.821
Urea (mg/dL)	*R*	0.780**
	Sig. (2-tailed)	0.0001
Creatinine (mg/dL)	*R*	0.830**
	Sig. (2-tailed)	0.0001
Uric acid (mg/dL)	*R*	0.593**
	Sig. (2-tailed)	0.0001
GFR (mL/min/1.73 m^2)	*R*	−0.751**
	Sig. (2-tailed)	0.0001
Albumin (g/dL)	*R*	−0.509**
	Sig. (2-tailed)	0.0001
Total protein (g/dL)	*R*	−0.475**
	Sig. (2-tailed)	0.0001
Calcium (mg/dL)	*R*	−0.668**
	Sig. (2-tailed)	0.0001
Phosphorus (mg/dL)	*R*	0.661**
	Sig. (2-tailed)	0.0001
Vitamin D3 (ng/mL)	*R*	−0.624**
	Sig. (2-tailed)	0.0001
PTH (pg/mL)	*R*	0.631**
	Sig. (2-tailed)	0.0001
Hb (g/dL)	*R*	−0.695**
	Sig. (2-tailed)	0.0001
Hct (%)	R	−0.714**
	Sig. (2-tailed)	0.0001

Notes. **Correlation is significant at the 0.01 level (2-tailed);
R: Pearson's correlation.
PTH: parathyroid hormone; Hct: hematocrit; Hb: hemoglobin.

Table (4): Specificity and sensitivity with cut-off and area under curve (AUC) values for MCP-1 in the study groups.

Variables	AUC	Spec.	Sen.	Cut-off	*P* value
MCP-1	0.99	96.7%	94.8%	382.5	0.0001

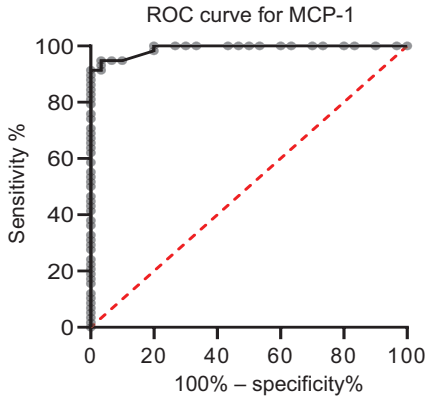

Figure (1): Receiver operator characterization (ROC) curve for MCP-1.

contribute to cardiovascular diseases and atherogenesis; MCP-1 recruits monocytes to atheroma lesions, accelerating the development of vascular complications in this population [11]. This elevation reflects increased inflammation associated with systemic inflammatory markers and malnutrition, predicting increased all-cause and cardiovascular mortality, all of which contribute to poor clinical outcomes in this cohort [12]. MCP-1/CCL2 plays a pivotal role in the progression of kidney disease by mediating inflammatory cell recruitment. Renal injury, proteinuria, oxidative stress, hyperglycemia, and uremic toxins stimulate MCP-1 expression in tubular and glomerular cells via NF-κB activation. Angiotensin II and advanced glycation end products further enhance its production. Recruited monocytes/macrophages amplify inflammation and fibrosis through a feedback loop. Thus, elevated MCP-1 reflects both local and systemic renal injury mechanisms [13].

A significantly increased level of serum MCP-1 in ESRD patients (1090.69 ± 465.04 pg/mL), compared to controls (195.33 ± 95.19 pg/mL), indicated their active participation in inflammatory reactions typical of advanced renal failure. This finding agreed with earlier studies of MCP-1 upregulation in renal pathologies as a result of chronic inflammation, oxidative stress, and immune dysregulation [14].

Biochemically, ESRD patients had higher values for urea, creatinine, uric acid, phosphorus, and PTH, and lower levels of calcium, vitamin D3, albumin, total protein, Hb, and Hct. These changes reflected systemic effects of decreasing renal function (e.g., disorders of mineral metabolism [CKD–MBD], anemia, and malnutrition) [15].

MCP-1 was associated with serum creatinine, urea, uric acid, phosphorus, and intact parathyroid hormone (iPTH), markers of renal dysfunction and metabolic derangements. These associations of MCP-1 with these markers support an additional role for MCP-1 as the indicator of decline in renal function, including its association with complications of CKD, such as hyperphosphatemia and secondary hyperparathyroidism. These associations support that MCP-1 may not only be indicative of the inflammatory state in ESRD patients but also of their biochemical imbalances [3].

MCP-1 was inversely related to GFR, calcium, vitamin D3, albumin, total protein, Hb, and Hct. These inverse correlations also suggested that MCP-1 could serve as an indicator of disease severity. The lower levels of eGFR were representative of severe damage to the kidneys, which correlated with the increased levels of MCP-1. Moreover, hypocalcemia and low vitamin D levels represented the features of CKD–MBD that were frequently correlated with adverse outcomes in ESRD patients. Hypoalbuminaemia and low protein levels were indicative of malnutrition–inflammation complex syndrome, which often occurs in dialysis patients and is associated with increased mortality [16].

The result of the study done by Gregg *et al.* showed that the level of MCP-1 in CKD patients was significantly elevated, compared to non-CKD individuals, suggesting its role in renal inflammation [8]. Diagnostic variables

supported the idea that MCP-1 could be a potential marker to monitor inflammation, early diagnosis, or risk stratification in subjects at risk of developing ESRD [17]. MCP-1 levels tend to rise with decline in kidney functions. A negative correlation is often observed between MCP-1 and eGFR, while a positive correlation is discovered with serum creatinine [18]. This supports the role of MCP-1 in kidney inflammation and damage. Proteinuria, especially albuminuria, is both marker and promoter of kidney damage. Elevated MCP-1 levels in urine are positively correlated with the degree of proteinuria, suggesting tubular injury and inflammatory cell infiltration [19]. Since MCP-1 is an inflammatory marker, its levels often correlate with systemic inflammatory markers such as C-reactive protein (CRP). Increased levels of CRP and MCP-1 reflect a heightened pro-inflammatory state in CKD stage 5 patients [20].

Levels of MCP-1 may show a positive correlation with serum urea and uric acid, as uremic toxins promote inflammatory cytokine release, further worsening renal inflammation and damage [21]. Chronic inflammation mediated by MCP-1 can impair erythropoiesis and contribute to anemia in CKD. Inflammatory cytokines suppress erythropoietin production and iron utilization, thereby linking MCP-1 levels with Hb and ferritin concentrations [22].

The relatively small sample size and single-center, case–control design limit study's generalizability and prevent causal inference. Important confounders, including diabetes, hypertension, dialysis-related inflammation, and medications such as statins, RAAS blockers, vitamin D analogs, and erythropoiesis-stimulating agents, could have influenced MCP-1 levels but were not fully controlled. The cross-sectional design with single pre-dialysis measurements did not allow evaluation of longitudinal changes or prediction of clinical outcomes. Moreover, MCP-1 is a non-specific chemokine elevated in many inflammatory conditions, which could reduce diagnostic specificity. Finally, MCP-1 was studied in isolation without validation against other biomarkers. Larger, multi-center, prospective studies are needed to confirm the diagnostic and prognostic value of MCP-1 in advanced CKD.

Conclusions

The results of the study demonstrate the precise significance of MCP-1 in ESRD, showing close correlation with major parameters of renal function, mineral metabolism, and inflammation. These results enhance the role of MCP-1 as a candidate of diagnostic and prognostic marker in advanced level renal disease. Larger studies and longitudinal designs are warranted to confirm its role in CKD as well as to investigate the possibility of a therapeutic target on MCP-1 leading to potential clinical benefits in the treatment of CKD.

Conflict of Interest

The authors declared no conflict of interest.

Author Contributions

Contributor role	Degree of contribution		
	Lead	Equal	Supporting
Conceptualization	SBM	HHA	
Data curation	SBM		
Formal analysis	SBM	HHA	AJH
Funding acquisition	SBM		
Investigation		HHA	
Methodology	SBM	HHA	AJH
Project administration	SBM	HHA	AJH
Resources	SBM		
Software	SBM	HHA	
Supervision		HHA	AJH
Validation		HHA	AJH
Visualization	SBM		
Writing—original draft	SBM		
Writing—review & editing		HHA	

References

[1] Wouk, N. End-stage renal disease: Medical management. Am Family Phy. 104(5):493–499, 2021.

[2] Kazancioğlu, R. Risk factors for chronic kidney disease: An update. Kidney Int Supp. 3(4):368–371, 2013. https://doi.org/10.1038/kisup.2013.79.

[3] Liu, Y.; Xu, K.; Xiang, Y.; Ma, B.; Li, H.; Li, Y.; et al. Role of MCP-1 as an inflammatory biomarker in nephropathy. Front Immunol. 14:1303076, 2023. https://doi.org/10.3389/fimmu.2023.1303076.

[4] Haller, H.; Bertram, A.; Nadrowitz, F.; Menne, J. Monocyte chemoattractant protein-1 and the kidney. Curr Opinion Nephrol Hypertension. 25(1):42–49, 2016. https://doi.org/10.1097/mnh.0000000000000186.

[5] Treacy, O.; Brown, N.N.; Dimeski, G. Biochemical evaluation of kidney disease. Transl Androl Urol. 8(Suppl 2):S214–S223, 2019. https://doi.org/10.21037/tau.2018.10.02.

[6] Tesch, G.H. MCP-1/CCL2: A new diagnostic marker and therapeutic target for progressive renal injury in diabetic nephropathy. Am J Physiol Renal Physiol. 294(4):F697–F701, 2008. https://doi.org/10.1152/ajprenal.00016. 2008.

[7] Navarro-González, J.F.; Mora-Fernández, C.; de Fuentes, M.M.; García-Pérez, J. Inflammatory molecules and pathways in the pathogenesis of diabetic nephropathy. Nature Rev Nephrol. 7(6):327–340, 2011. https://doi.org/10.1038/nrneph.2011.51.

[8] Gregg, L.P.; Tio, M.C.; Li, X.; Adams-Huet, B.; de Lemos, J.A.; Hedayati, S.S. Association of monocyte chemoattractant protein-1 with death and atherosclerotic events in chronic kidney disease. Am J Nephrol. 47(6):395–405, 2018. https://doi.org/10.1159/000488806.

[9] Wu, C-L.; Wu, H-M.; Chiu, P-F.; Liou, H-H.; Chang, C-B.; Tarng, D-C.; *et al*. Associations between the duration of dialysis, endotoxemia, monocyte chemoattractant protein-1, and the effects of a short-dwell exchange in patients requiring continuous ambulatory peritoneal dialysis. PLoS One. 9(10):e109558, 2014. https://doi.org/10.1371/journal.pone.0109558

[10] Tawfik, G.A.; Khalil, F.A.; Omar, S.A.; Keshawy, M.M.; Sliem, H. Monocyte chemoattractant protien-1 (MCP-1) and atherosclerosis in end stage renal disease patients. Br J Med

Medical Res. 12(9):1-8, 2016. https://doi.org/10.9734/BJMMR/2016/22852

[11] Pawlak, K.; Pawlak, D.; Mysliwiec, M. Possible new role of monocyte chemoattractant protein-1 in hemodialysis patients with cardiovascular disease. Am J Nephrol. 24(6):635–640, 2004. https://doi.org/10.1159/000082936.

[12] Ko, K.I.; Park, K.S.; Lee, M.J.; Doh, F.M.; Kim, C.H.; Koo, H.M.; *et al.* Increased dialysate MCP-1 is associated with cardiovascular mortality in peritoneal dialysis patients: A prospective observational study. Am J Nephrol. 40(4):291–299, 2014. https://doi.org/10.1159/000368201.

[13] Perkins, B.A.; Ficociello, L.H.; Ostrander, B.E.; Silva, K.H.; Weinberg, J.; Warram, J.H.; *et al.* Microalbuminuria and the risk for early progressive renal function decline in type 1 diabetes. J Am Soc Nephrol. 18(4):1353–1361, 2007. https://doi.org/10.1681/asn.2006080872.

[14] Pradeep, A.R.; Daisy, H.; Hadge, P. Serum levels of monocyte chemoattractant protein-1 in periodontal health and disease. Cytokine. 47(2):77–81, 2009. https://doi.org/10.1016/j.cyto.2009.05.012.

[15] Al Ani, H.T.; Al-Lami, M.Q. Evaluation of some biochemical and hematological parameters in patients with chronic kidney disease. J Faculty Med Baghdad. 66(2):154–161, 2024. https://doi.org/10.32007/jfacmedbagdad.2269.

[16] Eardley, K.S.; Zehnder, D.; Quinkler, M.; Lepenies, J.; Bates, R.L.; Savage, C.O.; et al. The relationship between albuminuria, MCP-1/CCL2, and interstitial macrophages in chronic kidney disease. Kidney Int. 69(7):1189–1197, 2006. https://doi.org/10.1038/sj.ki.5000212.

[17] Chang, H.; Lv, J.; Zheng, Y.; Li, D.; Li, Y. The diagnostic value of serum MCP-1 combined with OPN detection for early renal injury in gout patients. Int J Gen Med. 18:1423–1429, 2025. https://doi.org/10.2147/ijgm.s508220.

[18] Youssouf, S.; Harris, T.; O'Donoghue, D. More than a kidney disease: A patient-centred approach to improving care in autosomal dominant polycystic kidney disease. Nephrol Dialy Transplant. 30(5):693–695, 2015. https://doi.org/10.1093/ndt/gfv058.

[19] Kiattisunthorn, K.; Wutyam, K.; Indranoi, A.; Vasuvattakul, S. Randomized trial comparing pulse calcitriol and alfacalcidol for the treatment of secondary hyperparathyroidism in haemodialysis patients. Nephrology (Carlton, Vic). 16(3):277–284, 2011. https://doi.org/10.1111/j.1440-1797.2010.01398.x.

[20] Zager, R.A.; Johnson, A.C.; Lund, S. "Endotoxin tolerance": TNF-alpha hyper-reactivity and tubular cytoresistance in a renal cholesterol loading state. Kidney Int. 71(6):496–503, 2007. https://doi.org/10.1038/sj.ki.5002092.

[21] Kanellis, J.; Watanabe, S.; Li, J.H.; Kang, D.H.; Li, P.; Nakagawa, T.; et al. Uric acid stimulates monocyte chemoattractant protein-1 production in vascular smooth muscle cells via mitogen-activated protein kinase and cyclooxygenase-2. Hypertension (Dallas, TX). 41(6):1287–1293, 2003. https://doi.org/doi:10.1161/01.HYP.0000072820.07472.3B.

[22] Stenvinkel, P.; Heimbürger, O.; Paultre, F.; Diczfalusy, U.; Wang, T.; Berglund, L.; *et al.* Strong association between malnutrition, inflammation, and atherosclerosis in chronic renal failure. Kidney Int. 55(5):1899–1911, 1999. https://doi.org/10.1046/j.1523-1755.1999.00422.x.