

Association of Serum Chemerin With Albuminuria in Patients With Diabetic Nephropathy: A Case-Control Study

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Abstract

Background: Chemerin, an adipokine that links metabolic regulation with inflammation, has been put forward as a candidate biomarker for diabetic kidney disease.

Objective: Measuring serum chemerin levels among diabetic patients, comparing those with and without nephropathy and examining how these levels correspond with albuminuria and other measures of kidney function.

Subjects and Methods: A total of 180 adults from Duhok, Iraq, were recruited for this case-control design and divided evenly into three groups of 60: healthy controls, diabetics without nephropathy, and diabetics with nephropathy (DN). Chemerin concentrations measurement was done by using a sandwich ELISA kit, and renal function evaluation were done through three parameters: serum creatinine, estimated glomerular filtration rate (eGFR), and urinary albumin-to-creatinine ratio (ACR). The assessment of associations among these variables was done through multiple statistical analysis using SPSS software.

Results: Chemerin levels climbed progressively from one group to the next, with medians of 343.5 ng/mL among controls, 449.5 ng/mL in the diabetic-only group, and 518.0 ng/mL in the DN group ($p < 0.001$). Compared with the other two groups, patients with DN had the highest creatinine and ACR values along with the lowest eGFR (all $p < 0.001$). Within the DN group specifically, chemerin correlated positively with ACR ($p = 0.306$, $p = 0.017$), though no meaningful correlation emerged with creatinine, urea, eGFR, or HbA1c. ACR was the only variable to stand out in the multiple linear regression, independently predicting serum chemerin ($\beta = 0.523$, $p < 0.001$).

Conclusions: Serum chemerin is raised in DN and independently linked to albuminuria, which supports its possible role as a marker of renal involvement in diabetic kidney disease.

Keywords: Diabetes mellitus; diabetic nephropathy; chemerin; albuminuria.

Introduction

One of the most prevalent chronic metabolic diseases, diabetes mellitus (DM) has a significant impact on global health systems. Its global burden keeps mounting, bringing with it a heavy load of illness, premature death, and healthcare spending.^{1,2} Persistent hyperglycemia underlies the long-term microvascular and macrovascular complications that drive much of this burden and erode quality of life. Diabetic nephropathy (DN) remains one of the most important causes of chronic kidney disease (CKD) and eventually leading to end-stage renal disease (ESRD) globally.^{3,4} In spite of huge technological and medical advances in managing diabetes, the percentage of DN continues to increase, highlighting the need for improved strategies for early detection and risk assessment.^{3,4,5}

The pathophysiology of DN is much more complicated than it looks, and it involves multiple interacting steps, including persistent hyperglycemia, hemodynamic alterations, release of free radicals and oxidative stress, inflammation, and injury to endothelium and dysfunction.^{5,6,7,8} Chronic low-grade inflammation is a major factor in the initiation and developing DN, promoting glomerular injury, extracellular matrix accumulation, and progressive renal fibrosis.^{6,8} Although urinary albumin excretion and eGFR are widely used in the diagnosis and monitoring of DN, these markers primarily reflect established renal injury rather than the underlying biological processes involved in disease progression.^{3,4} Consequently, considerable attention has been directed toward identifying novel biomarkers that may better reflect ongoing metabolic and inflammatory alterations associated with diabetic kidney disease.

Adipose tissue is now viewed less as inert storage and more as an important organ of the endocrine system, releasing bioactive molecules termed adipokines that help regulate glucose metabolism, insulin sensitivity, inflammation, and vascular homeostasis.⁹ When this secretion becomes disordered, it has been tied to obesity, resistance to insulin, developing type 2 diabetes mellitus, and most of the vascular complications that follow.^{9,10} Chemerin, a more recently described adipokine which is encoded by the retinoic acid receptor responder 2 (RARRES2) gene, has drawn

particular interest in its joint metabolic and inflammatory roles.^{10,11,12,13} Adipogenesis, inflammatory signaling, and immune cell recruitment are all facilitated by signaling via receptors like chemokine-like receptor 1 (CMKLR1).^{11,12,14} Activating the chemerin/ChemR23 pathway in experimental mice has caused oxidative stress, inflammation, and endothelial dysfunction in renal tissue, suggesting it may play a role in the pathophysiology of DN.^{15,16,17}

At the molecular level, chemerin is produced as a largely inactive precursor, prochemerin, and is converted to its biologically active forms through sequential proteolytic cleavage of the carboxy-terminal end of the peptide. Because this processing is governed by the local balance of activating and inactivating proteases, the net biological activity of chemerin can vary considerably between tissues and disease states, even when total circulating concentrations are comparable. Chemerin acts through three receptors — CMKLR1, GPR1, and CCRL2 — but these are not equivalent: CMKLR1 carries most of the downstream signaling, whereas CCRL2 does not itself transduce a signal and instead captures chemerin and presents it to CMKLR1 on neighboring cells.^{13,14} What makes this relevant to kidney disease is where these components are found. Chemerin and its receptors are expressed not only in adipose tissue but also in the liver, the vasculature, and the kidney — a distribution that positions the molecule at the intersection of metabolic regulation, vascular function, and inflammation, the same processes that converge in DN.^{13,15}

Several clinical studies have documented elevated circulating chemerin in persons having diabetes, and in patients having CKD, and DN.^{18,19,20,21} Pfau et al. found that serum chemerin rises as renal function falls, proposing that reduced renal clearance drives circulating levels higher in chronic kidney disease.²⁰ Yamamoto et al. reported much the same, linking higher circulating chemerin to poorer clinical outcomes among dialysis patients.²² Blaszkak et al. went further, attributing the rise in serum chemerin in chronic kidney disease chiefly to declining kidney function rather than to greater adipose tissue production.²³

Taken together, these observations raise an important interpretive question. Because chemerin is partly eliminated by renal clearance, part of the rise seen in chronic kidney disease is likely to reflect reduced renal elimination rather than disease-specific overproduction.^{20,23} A clearance-driven increase, however, does not preclude a biologically meaningful link between chemerin and the glomerular injury that characterizes early DN. Albuminuria, unlike serum creatinine or eGFR, typically signals glomerular damage at a stage when overall filtration may still be preserved, and it is therefore regarded as one of the earliest clinical markers of diabetic kidney involvement.^{3,4} It remains unclear whether circulating chemerin tracks more closely with this early marker of injury or with the later fall in filtration.

Few studies have looked at serum chemerin against both albuminuria and filtration-based measures in the same well-characterized diabetic cohort, and fewer still have asked which of those associations survives adjustment for metabolic confounders such as body mass index and glycemic control. That gap matters clinically: a marker tied specifically to albuminuria could add to conventional filtration measures and flag kidney involvement earlier, at a stage when it may still be more modifiable.

Against this background, the present study serum chemerin was measured in diabetic patients, and in patients not having DN, and compared them with healthy controls. Our primary aim was to determine how serum chemerin relates to albuminuria, expressed as the urinary ACR. Secondary aims were to examine its associations with other renal and metabolic measures and to identify the factors that independently predict its concentration.

Materials and Methods

Study Design and Participants

Conducted in Duhok Governorate, Iraq, the present case-control study ran from June to October 2025. Overall, 180 adults aged 18 years or older were recruited and divided into three equal arms: a healthy control group (n = 60), a diabetes mellitus group without nephropathy (n = 60), and a diabetic nephropathy group (n = 60).

Control participants had no documented history of diabetes, kidney disease, or any other chronic systemic condition. Diabetic patients were enrolled through the Diabetic Center of Azadi Teaching Hospital, whereas those with diabetic nephropathy were drawn from the outpatient clinic of the Duhok Kidney Center. Diabetes mellitus was confirmed from each patient's medical records together with clinical assessment. A diagnosis of diabetic nephropathy rested on clinical and laboratory evidence of diabetes-related kidney involvement, such as albuminuria and/or reduced renal function attributable to diabetes. Exclusion criteria comprised acute kidney injury, recognized non-diabetic renal disease, chronic inflammatory or autoimmune conditions, cancer, active infection, pregnancy, and missing clinical or laboratory records.

Sample Collection and Laboratory Measurements

Under aseptic conditions, a venous blood sample was collected from every participant. Each sample was distributed into ethylenediaminetetraacetic acid (EDTA) tubes for glycated hemoglobin (HbA1c) testing and for serum separation we used plain gel tubes. The gel tubes were centrifuged at 3000 rounds per minute for at least 10 minutes and the sample was analyzed. Midstream urine sample was also collected from each participant to determine urinary albumin and creatinine, from which the urinary ACR was derived and valued in mg/g.

Routine biochemistry investigation was performed at Duhok Central Laboratory on a Cobas (Roche) automated analyzer. While for the estimated glomerular filtration rate (mL/min/1.73 m²) the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation was used to calculate it.²⁴

Serum Chemerin Measurement

The quantification of serum chemerin was done by using the ELISA sandwich assay which is directed against human retinoic acid receptor responder 2 (RARRES2) produced by (BT LAB, Shanghai, China; Cat. No. E1435Hu). Every step was taken following the manufacturer's rules. Chemerin measurements were calculated by interpolating absorbance at 450 nm from the standard calibration curve. The coefficient of variation of intra- and inter-assay stayed below 8% and 10%, and the assay's lower limit of detection was 4.99 ng/mL.

Analysis of Statistics

Data analyses were analyzed in SPSS 26.0 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk test assessed normality of distribution. Group differences were examined using one-way ANOVA with Tukey's post hoc test for normally distributed data, and the Kruskal–Wallis test with Dunn's post hoc test for non-normally distributed data. Correlations between variables were evaluated using Spearman's rank correlation.

Ethical Acceptance

The Directorate of Health in Duhok, Iraq, authorized the study protocol (Reference No. 13042025-3-6). Each participant gave their consent through signing a form before enrollment, and the study followed the guidelines of the Declaration of Helsinki.

Results

Participant Characteristics

The analysis covered all 180 enrolled participants: 60 healthy controls, 60 patients with diabetes mellitus but no nephropathy, and 60 with diabetic nephropathy. Age and sex matched groups were participated in the study with $p > 0.05$ in both categories. However, both diabetic groups had a considerably larger body mass index (BMI) in comparison to controls ($p < 0.001$). HbA1c, likewise, showed significantly worse glycemic control in the diabetes and diabetic nephropathy groups than in controls ($p < 0.001$) (Table 1).

Table 1 Study groups' baseline clinical and demographic variables

Parameter	Healthy	Diabetic	Diabetic Nephropathy	p-value
Age (years)	53.17 ± 10.06	54.57 ± 9.54	56.53 ± 11.51	0.28
Sex, n (%)				
– Female	34 (56.7%)	38 (63.3%)	33 (55.0%)	0.62
Body Mass Index (kg/m ²)	25.16 ± 2.43	28.29 ± 2.55	28.51 ± 1.54	< 0.001†
HbA1c (%)	5.25 ± 0.33	7.84 ± 1.47	7.91 ± 1.55	< 0.001†

Mean ± SD is used to express values. $p < 0.05$ is considered statistically significant.

Renal Function Parameters and Serum Chemerin Levels

Renal function indices varied significantly across the groups (Table 2). Compared with both controls and diabetic patients without nephropathy, those with diabetic nephropathy had significantly raised serum urea, creatinine, urinary ACR, and uric acid (all $p < 0.001$). Their eGFR, conversely, was significantly lower ($p < 0.001$).

Serum chemerin climbed stepwise from one group to the next. Median values were 343.5 ng/mL in controls, 449.5 ng/mL in the diabetes group, and 518.0 ng/mL in those with diabetic nephropathy. This across-group difference reached statistical significance ($p < 0.001$) (Table 2, Figure 1).

Table 2 Renal parameters compared to chemerin levels among participants

Parameter	Control group (n = 60)	Diabetic (n = 60)	Diabetic Nephropathy (n = 60)	p-value
Urea (mg/dL)	30.20 ± 8.25	35.62 ± 12.57	80.06 ± 29.54	< 0.001†
Creatinine (mg/dL)	0.77 ± 0.19	1.00 ± 0.21	2.19 ± 1.23	< 0.001†
eGFR (mL/min/1.73 m ²)	102.27 ± 19.63	71.69 ± 16.20	37.78 ± 14.70	< 0.001†
ACR (mg/g)	6.65 (6.13)	22.5 (3.85)	180.0 (123.75)	< 0.001†
Uric acid (mg/dL)	4.19 ± 1.42	4.84 ± 1.19	6.13 ± 1.82	< 0.001†
Chemerin (ng/mL)	343.5 (138)	449.5 (189)	518.0 (328)	< 0.001 †

Values are expressed as mean ± SD or median (IQR). † Statistically significant ($p < 0.05$).

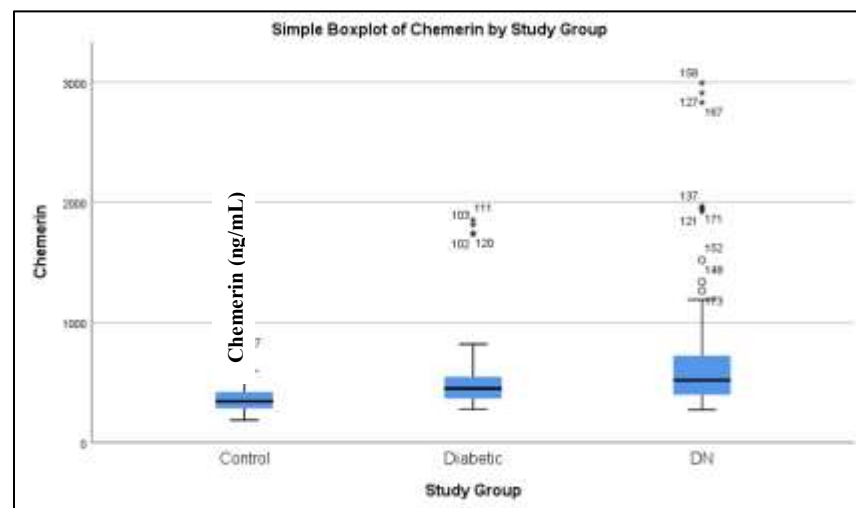


Figure 1 Box-and-whisker plot showing serum chemerin concentrations across the study groups.

Correlation Between Serum Chemerin and Clinical Parameters

Spearman correlations were computed within each group separately (Table 3). Among controls, chemerin was weakly and inversely related to serum urea ($\rho = -0.262$, $p = 0.043$). None of the other parameters examined correlated significantly with chemerin in this group.

Among patients with diabetes mellitus without nephropathy, serum chemerin was not significantly associated with any of the evaluated renal or metabolic parameters.

Within the diabetic nephropathy group, chemerin was positively and significantly correlated with the urinary ACR ($\rho = 0.306$, $p = 0.017$). No significant link was found with serum creatinine, urea, eGFR, or HbA1c.

Table 3 Correlation between serum chemerin and renal and metabolic parameters within each study group (Spearman's rho)

Parameter	Control		Diabetic		Diabetic nephropathy	
	(ρ)	p-value	(ρ)	p-value	(ρ)	p-value
eGFR	-0.029	0.826	-0.124	0.346	-0.063	0.632
ACR	-0.056	0.670	0.105	0.423	0.306	0.017†
Creatinine	-0.088	0.504	0.147	0.264	0.034	0.794
Urea	-0.262	0.043†	0.018	0.894	-0.135	0.303
HbA1c	0.158	0.227	0.100	0.446	0.161	0.220

Values are Spearman's rank correlation coefficient (ρ). † Statistically significant ($p < 0.05$).

Multiple Linear Regression Analysis

Serum chemerin was examined as the outcome variable in a multiple linear regression that included all 180 participants in the cohort (Table 4). Six predictors — age, BMI, HbA1c, serum creatinine, ACR, and uric acid — were entered into the model together. ACR was the only parameter with statistical significance, having a positive relationship with chemerin levels ($\beta = 0.523$, standardized; $p < 0.001$), while none of the other variables contributed independently. Taken as a whole, the model reached statistical significance ($F(6, 173) = 6.083$, $p < 0.001$) and accounted for roughly 17.4% of the variability in serum chemerin levels ($R^2 = 0.174$; adjusted $R^2 = 0.146$).

Table 4: Multiple linear regression identifying independent predictors of serum chemerin Dependent variable: Serum chemerin (ng/mL)

Predictor	B (Unstandardized)	SE	Standardized β	p-value
Age (years)	1.324	2.801	0.040	0.637
BMI (kg/m ²)	-8.701	14.858	-0.053	0.559
HbA1c (%)	-4.061	20.056	-0.016	0.840
Creatinine (mg/dL)	-56.455	42.477	-0.122	0.186
ACR (mg/g)	1.846	0.352	0.523	< 0.001†
Uric acid (mg/dL)	-19.928	20.656	-0.077	0.336

β denotes the standardized regression coefficient, B the unstandardized regression coefficient, and SE the standard error. † Statistically significant ($p < 0.05$).

Discussion

The study showed the relationship between serum chemerin concentrations and renal function indicators in patients with diabetes mellitus, both with and without diabetic nephropathy. Three findings stood out: (1) serum chemerin differed markedly across the three groups, rising step by step from healthy controls through diabetes mellitus to reach its highest level in diabetic nephropathy; (2) within the diabetic nephropathy group, serum chemerin correlated positively and significantly with the urinary ACR; and (3) in multivariable regression, ACR emerged as the only independent predictor of serum chemerin concentrations.

The stepwise rise in serum chemerin across the control, diabetes mellitus, and diabetic nephropathy groups adds to the growing evidence linking chemerin to the metabolic and renal complications of diabetes. Our findings align with Hu and Feng, who tied chemerin to renal impairment and reported markedly higher blood concentrations in type 2 diabetes mellitus. A role in diabetes-related vascular injury is plausible, given that Gu et al. found elevated circulating chemerin in diabetic patients with microvascular complications.¹⁹ El-Gohary et al. likewise reported higher serum chemerin in diabetic nephropathy and proposed that it may track disease progression.²⁵ Collectively, this evidence supports the view that chemerin concentrations increase as the metabolic and renal burden of diabetes progresses.

A key result of the present study was the strong positive correlation observed between serum chemerin and albuminuria, measured as the urinary albumin-to-creatinine ratio. Albuminuria is a well-established marker of glomerular injury and among the earliest clinical signs of diabetic nephropathy.^{3,4} Its association with chemerin implies that elevated concentrations may mirror the degree of renal involvement in diabetic kidney disease. These observations agree with El-Tahir et al., who described a distinct relationship between circulating chemerin and the degree of albuminuria in patients with diabetic kidney disease.²⁶ Similarly, Lin et al. linked chemerin to diabetic nephropathy and proposed that it may parallel disease severity.²⁷

Importantly, the link between chemerin and albuminuria remained even after adjustment for several possible confounders. Entered together in the multivariable model, ACR alone independently predicted serum chemerin, while age, BMI, HbA1c, serum creatinine, and uric acid did not. This finding suggests that albuminuria may have a closer relationship with chemerin than conventional markers of renal filtration function. Although serum creatinine and eGFR are widely used indicators of renal function, albuminuria often reflects earlier glomerular injury and may therefore be more sensitive to the inflammatory and vascular processes associated with chemerin dysregulation.^{28,29} This pattern contrasts with the earlier reports of Pfau et al. and Blaszak et al., in which circulating chemerin was linked primarily to declining glomerular filtration;^{20,23} in our diabetic nephropathy group, chemerin showed no significant correlation with eGFR, serum creatinine, or urea and was instead associated specifically with albuminuria. However, because chemerin is renally cleared and the diabetic nephropathy group had a markedly reduced eGFR (mean 37.78 mL/min/1.73 m²), the absence of a significant chemerin–eGFR correlation within this group may partly reflect the limited subgroup size (n = 60) rather than a true lack of association, and this should be considered when interpreting our findings.

Several biological mechanisms may explain the observed relationship between chemerin and diabetic nephropathy. Chemerin is a multifunctional adipokine involved in metabolic regulation, inflammation, and vascular homeostasis.^{11,12,13} Experimental studies have demonstrated that activation of the chemerin/ChemR23 signaling pathway promotes inflammatory cell recruitment, endothelial dysfunction, oxidative stress, and tissue fibrosis.^{15,16,17} Shang et al. demonstrated that activation of the chemerin/ChemR23 axis promoted inflammatory responses in glomerular endothelial cells exposed to diabetic conditions, supporting a direct role for chemerin in renal injury.¹⁵ Furthermore, Wang et al. reported that chemerin signaling may contribute to endothelial-to-mesenchymal transition, a process implicated in renal fibrosis and progression of diabetic nephropathy.¹⁶ These mechanisms provide biological plausibility for the association observed between chemerin and albuminuria in the present study. In addition to serving as a biomarker, chemerin may represent a potential therapeutic target. Signaling via CMKLR1 has been linked to inflammatory cell recruitment, endothelial dysfunction, and renal fibrosis, and inhibiting this pathway could, in theory, restrain the glomerular injury underlying diabetic nephropathy.^{15,16} Experimental findings support this notion: in animal studies, agents that suppress chemerin activity lessen inflammation and delay the progression of diabetes-related kidney damage.¹⁷ It remains uncertain whether such approaches will yield clinical benefit; nonetheless, the specific association between chemerin and albuminuria observed here strengthens the case for exploring the chemerin/ChemR23 axis as a therapeutic option in diabetic kidney disease.

This study has several limitations. First, its case–control design cannot establish the timing or causal direction of the relationship between serum chemerin and diabetic nephropathy. Second, it was conducted at a single center with a relatively modest sample, which may limit how far the findings generalize. Third, inflammatory markers other than chemerin were not measured, leaving the underlying mechanisms incompletely explored. Fourth, within the diabetic nephropathy group the chemerin–albuminuria association, though significant, was modest in magnitude (Spearman $\rho = 0.306$); the larger standardized coefficient in the pooled multivariable model ($\beta = 0.523$) reflects the full cohort, in which between-group differences may have amplified the apparent effect, so the within-group correlation should be read as the more conservative estimate. Despite these limitations, the study drew on well-characterized participant groups and adds further evidence for an association between serum chemerin and albuminuria in diabetic nephropathy.

Clinically, our findings indicate that serum chemerin levels might be helpful as an additional biomarker for detection and follow-up of diabetic renal disease regarding albuminuria. Since chemerin was independently related to ACR instead of serum creatinine, it might be useful in conjunction with traditional indices for early detection of diabetic kidney damage. Larger, multicenter prospective and longitudinal studies are now needed to confirm these findings, establish temporality and causality, and clarify the prognostic and therapeutic value of chemerin in diabetic nephropathy.

Several limitations restrict how readily these findings can be applied in clinical settings. No standardized reference range for circulating chemerin has yet been established, and measured concentrations vary considerably across assay platforms, complicating direct comparison between studies and the definition of clinically useful thresholds. Chemerin concentrations are further influenced by comorbidities that frequently coexist with diabetic kidney disease, including obesity, systemic inflammation, and cardiovascular disease, so a single reading may reflect several overlapping processes rather than renal involvement by itself. Overall, these considerations indicate that although chemerin is a promising marker of albuminuric kidney injury, its clinical adoption will require assay standardization and careful control for confounders before it can be used in routine practice.

Conclusion

Serum chemerin differed markedly between the study groups, climbing steadily and peaking in DN patients. Within DN patients, it rose in step with albuminuria, and the urinary ACR stood out as the only independent predictor of chemerin levels. These results point to chemerin as a candidate marker of renal involvement in diabetic nephropathy, though prospective studies will be needed to establish its clinical usefulness and prognostic value in diabetic kidney disease.

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Data Availability Statement

The data for this study are unavailable publicly; however, they can be given whenever asked by the corresponding author.

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