



A RESEARCH ARTICLE

Association of Complete Blood Count -Derived Inflammatory Markers Indices with Renal Function in Patients with Chronic Kidney Disease: A Case–Control Study from Basrah, Iraq

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Abstract

Chronic kidney disease (CKD) is defined as progressive deterioration of renal function, with persistent activation of inflammatory pathways contributing to disease progression and complications. Traditional inflammatory markers are useful as they routine application is easy and can be widely used. Recently, complete blood count (CBC)-derived inflammatory indices, including neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), and systemic inflammation response index (SIRI), have emerged as simple, inexpensive, and readily available biomarkers of systemic inflammatory status. However, the relationship between these indices and renal function across different populations remains poorly understood. This case–control study aimed to evaluate the association between CBC-derived inflammatory indices and renal function in patients with CKD. A total of 161 subjects were enrolled, comprising 79 patients with CKD and 82 healthy controls. Routine hematological parameters, serum urea, creatinine, and estimated glomerular filtration rate (eGFR) were assessed, and inflammatory indices were calculated from CBC-derived data. Statistical tests were used to compare groups and spearman correlation were employed to explore associations between indices and kidney function. Serum levels of urea, creatinine, NLR, and SIRI were significantly higher in CKD patients compared to healthy controls, whereas eGFR values were markedly decreased (all $P < 0.001$). eGFR showed strong inverse correlations with creatinine and urea, as well as significant negative associations with NLR and SIRI. Conversely, SII did not differ significantly between groups ($P = 0.105$); however, in multivariable analysis, SII remained independently associated with eGFR, suggesting a potential relationship with renal function beyond group-level differences. Among the evaluated inflammatory indices, SIRI demonstrated the most consistent and robust association with impaired renal function. These findings suggest that CBC-derived inflammatory indices, particularly SIRI, may serve as affordable and accessible biomarkers for assessing systemic inflammation and renal dysfunction in CKD. When integrated with conventional renal parameters, they could enhance clinical risk assessment and disease monitoring. Nevertheless, further validation through large-scale, multicenter prospective studies is warranted to confirm these findings and establish their clinical utility.

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1. Introduction

Chronic kidney disease (CKD) is a progressive disorder characterized by a gradual decline in renal function and is associated with substantial morbidity and mortality worldwide. Although CKD is traditionally considered a disease of impaired filtration and structural kidney damage, increasing evidence indicates that systemic inflammatory responses play a central role in its development and progression. Persistent inflammation contributes to renal injury through mechanisms involving oxidative stress, endothelial dysfunction, immune dysregulation, and activation of profibrotic pathways, ultimately accelerating functional deterioration of the kidney [1, 2].

Inflammation in CKD involves complex interactions between innate and adaptive immune responses. Activation of neutrophils and monocytes, together with impaired lymphocyte-mediated immune regulation, contributes to the chronic inflammatory environment observed in patients with renal dysfunction. These alterations in circulating immune cells have encouraged researchers to explore hematological parameters as accessible indicators of systemic inflammation [1, 3].

Composite inflammatory indices based on routine complete blood count (CBC) parameters have received attention recently because of their simplicity, low cost and potential clinical applicability. The NLR is a measure of the balance between inflammatory activation and immune regulation. The SII, however, integrates the counts of neutrophils, lymphocytes and platelets to provide a broader picture of the inflammatory state. Also, the systemic inflammation response index (SIRI) based on neutrophil, monocyte and lymphocyte counts has emerged as a potential marker of systemic immune activation [3-7].

Inflammatory indices obtained from the CBC have previously been shown to be associated with poor outcomes in a variety of chronic diseases, including cardiovascular disease, cancer and inflammatory diseases [1-3].

In CKD, increased inflammatory indices have been linked with disease severity, cardiovascular complications, and mortality. However, the relative contribution and clinical relevance of individual inflammatory indices remain unclear, particularly when evaluated simultaneously within the same patient population [8-10].

Given that CBC analysis is routinely performed in clinical practice, clarification of the relationship between these inflammatory indices and renal function may provide a practical approach for identifying patients with increased inflammatory burden. However, further investigation is required to determine which CBC-derived index most consistently reflects renal impairment [9-11].

Therefore, the present study aimed to evaluate the association between NLR, SII, and SIRI and renal function parameters in patients with CKD compared with healthy controls. Further investigated whether these readily available inflammatory indices could provide additional information regarding renal dysfunction beyond conventional laboratory markers.

2. Materials and Methods

2.1. Study design and participants

In this case-control study, we aimed to determine the relationship between inflammatory indices derived from the CBC and renal function parameters in dialysis patients who attending at Basrah Nephrology and transplantation center. The study was approved by scientific committee of dentistry College University of basrah (BDC -8-1-26-3) as well as it was approved by Ministry of Health & Environment/ Iraq (No. 1124). A total of 161 subjects were enrolled in the study (79 male and 82 female), their age ranging from (20-80) years ; including 76 patients with chronic kidney disease on hemodialysis for at least 6 months ago and 85 apparently healthy participants with no signs and symptoms of any systemic disease as a control group .All information related to their medical history and health state were collected according specific questioner was prepared for the study purpose and after explaining the aims and objectives of the study, written informed consent was obtained from all participants .They were free for participation or refuse. The samples were collected from December 2025 to Aprile 2026

Clinical evaluation, laboratory findings and evidence of impaired renal function were used to identify patients with CKD. All people with acute inflammatory conditions or active infections, malignancies, or other systemic conditions likely to independently affect inflammatory markers were excluded . Control group included people without a history of renal disease or chronic inflammatory disorders.

2.2. Laboratory measurements

Three ml of blood was taken from each participated in the study by using sterile a disposable syringe. The blood sample was divided into two parts: The first part is one ml of blood was put in a tube with EDTA as anticoagulant for direct making complete blood count (CBC) in Full automated 5 part hematology analyzer from Mindray for evaluation of total white blood cell, neutrophil, lymphocyte, monocyte and platelet counts . The remained 2 ml of blood was put in a gel tube for measurement of serum creatinine and urea concentrations were used to assess renal function by using BS 240 biochemistry analyzer from Mindray .

The eGFR (estimated glomerular filtration rate) standard clinical equations, was used as the primary indicator of renal function. This was used as it didn't depend on race factor [12].

2.3. Calculation of inflammatory indices

Inflammatory indices from CBC were calculated using previously established formulas:

Neutrophil-to-lymphocyte ratio (NLR) [13]:

$$NLR = \frac{\text{Neutrophil count}}{\text{Lymphocyte count}}$$

Systemic immune-inflammation index (SII) [14]:

$$SII = \frac{\text{Platelet count} \times \text{Neutrophil count}}{\text{Lymphocyte count}}$$

Systemic inflammation response index (SIRI) [14]:

$$SIRI = \frac{\text{Neutrophil count} \times \text{Monocyte count}}{\text{Lymphocyte count}}$$

These indices were selected as they combine several circulating immune cell populations and may better represent systemic inflammatory activity than individual hematological parameters [15, 16].

2.4. Statistical analysis

SPSS version 26 (IBM Corp., Armonk, NY, USA). was used to analyze data. Continuous variables are presented as mean $\pm$ standard deviation (SD) and categorical variables as frequency (%) and were used to compare laboratory characteristics and inflammatory indices between patients with CKD and healthy controls. Group differences were compared using independent T- tests and Pearson's chi-square test

categorical variables were analyzed using respective methods. Correlation between inflammatory indices and renal function parameters and eGFR was assessed using Pearson correlation coefficients (r).

Box and-whisker plots were used to illustrate the distribution of renal biomarkers and inflammatory indices between CKD patients and healthy controls.Independent associations between inflammatory indices and renal function with adjustment for relevant clinical variables. Results were presented as correlation coefficients, regression coefficients and respective P values. Statistical significance was set at a two-sided P value < 0.05.

3. Results

3.1. Participant characteristics

A total of 161 participants were enrolled, comprising 76 patients with CKD and 85 healthy controls. **Table 1** presents the demographic characteristics. Both age and sex distribution did not differ significantly between the two groups .As in age (48.49 ± 11.59 vs. 46.44 ± 12.73 years with $p = 0.286$), indicating that the two groups were comparable with respect to age and sex $p=0.392$

The median duration of dialysis among CKD patients was 18 months, reflecting established renal impairment.

Table 1. Demographic characteristics of CKD patients and healthy controls

Characteristic	CKD patients (n = 76)	Healthy controls (n = 85)	P-value
Age (years), mean $\pm$ SD	48.49 ± 11.59	46.44 ± 12.73	0.286
Sex, n (%)			
Male	40 (52.6%)	39 (45.9%)	0.392
Female	36 (47.4%)	46 (54.1%)	

Data are presented as mean $\pm$ standard deviation (SD) or number (percentage). P-value for age was calculated using Welch's independent-samples t-test; P-value for sex was calculated using Pearson's chi-square test. Statistical significance was set at $P < 0.05$.

3.2. Hematological characteristics

Table 2. Compares CBC parameters in CKD patients and healthy controls. Total WBC , neutrophil, lymphocyte ,monocytes ,eosinophil as well as platelet counts were comparable between two groups .Both of total WBC and neutrophil show non-significant increase in CKD patients .However significant decrease in lymphocyte and platelet counts and significant increase in both monocyte and eosinophil levels was observed in CKD patients. These indicate that CKD is associated with specific changes in circulating immune cell distributions, but not generalized leukocytosis.

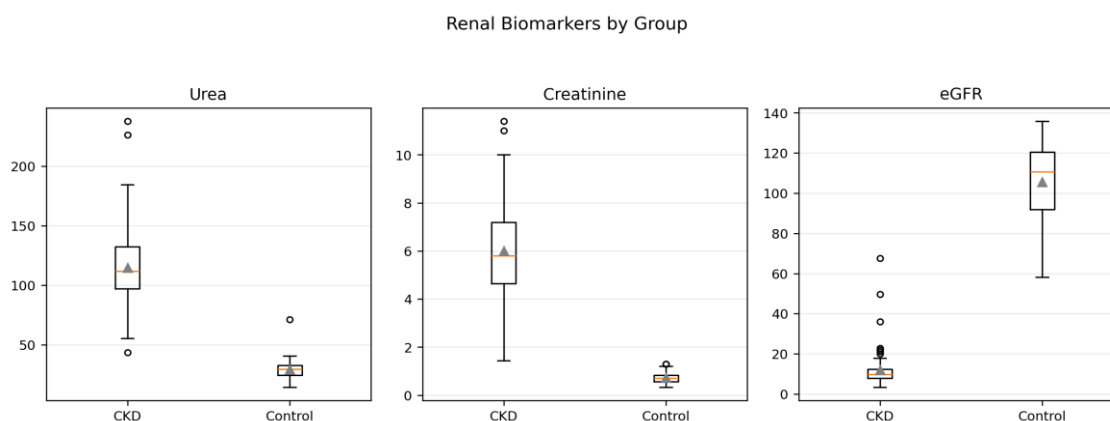
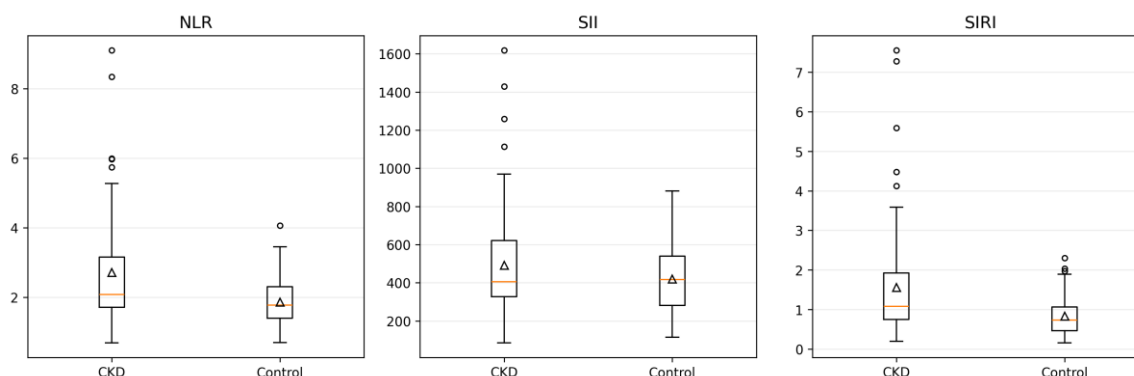
Table 2. Comparison of CBC and inflammatory indices between CKD patients and healthy controls

Variable	CKD Patients (n=76)	Healthy Controls (n=85)	P-value	Statistical Significance
	Mean $\pm$ SD	Mean $\pm$ SD		
WBC	6.90 $\pm$ 2.89	6.66 $\pm$ 1.36	0.512	NS
NEU	4.34 $\pm$ 2.52	3.94 $\pm$ 1.23	0.218	NS
LYM	1.75 $\pm$ 0.70	2.22 $\pm$ 0.58	<0.001	S
MON	0.55 $\pm$ 0.31	0.44 $\pm$ 0.16	0.006	S
EOS	0.25 $\pm$ 0.34	0.14 $\pm$ 0.14	0.007	S
PLT	191.28 $\pm$ 67.43	225.80 $\pm$ 48.84	<0.001	S
NLR	2.72 $\pm$ 1.59	1.87 $\pm$ 0.67	<0.001	S
SII	492.49 $\pm$ 284.26	420.01 $\pm$ 166.82	0.054	NS
SIRI	1.56 $\pm$ 1.39	0.84 $\pm$ 0.45	<0.001	S
Urea	114.89 $\pm$ 35.07	29.01 $\pm$ 7.66	<0.001	S
Creatinine	6.00 $\pm$ 2.09	0.72 $\pm$ 0.22	<0.001	S
eGFR	11.96 $\pm$ 9.34	105.58 $\pm$ 20.52	<0.001	S

S = Significant, NS = Not significant

3.3. Renal function biomarkers and inflammatory indices

Table 2 also presents a summary of renal function markers analysis. Patients with CKD showed significantly higher serum urea and creatinine levels than healthy controls, along with a marked reduction in eGFR (all $P < 0.001$) (**Figure 1**). Regarding inflammatory indices, CKD patients had significantly higher NLR ($P < 0.001$), and systemic inflammatory indices SIRI ($P < 0.001$) values compared to controls. In contrast, SII show non-significant increase in CKD patients ($P = 0.054$) (**Figure 2**). Among the evaluated indices, NLR and SIRI showed greater discriminatory power between CKD patients and controls, indicating a stronger association with inflammatory changes in renal dysfunction.

**Figure 1.** Distribution of biomarkers of renal function in CKD patients and healthy controls**Figure 2.** Distribution of CBC-derived inflammatory indices in CKD patients and healthy subjects

3.4. Correlation between inflammatory indices and renal function

The associations between eGFR, renal biomarkers, and inflammatory indices are presented in **Table 3**

As expected, eGFR showed strong inverse correlations with serum urea ($r = -0.858$, $P < 0.001$) and creatinine concentrations ($r = -0.887$, $P < 0.001$). This indicates progressive deterioration of renal function with increasing biochemical markers of kidney injury.

Among CBC-derived inflammatory indices, eGFR showed significant negative correlations with NLR ($r = -0.302$, $P < 0.001$) and SIRI ($r = -0.312$, $P < 0.001$). No significant association was observed between eGFR and SII ($r = -0.128$, $P = 0.105$). The overall correlation structure among renal biomarkers, hematological parameters, and inflammatory indices is summarized in (**Figure 3**).

Table 3. Correlation Analysis of eGFR with Renal Biomarkers and Inflammatory Indices

Variable vs. eGFR	Pearson r	P value	Significance
Urea	-0.858	<0.001	S
Creatinine	-0.887	<0.001	S
NLR	-0.302	<0.001	S
SII	-0.128	0.105	NS
SIRI	-0.312	<0.001	S

All samples: CKD patients ($n=76$) + healthy controls ($n=85$); total $n=161$ • Pearson correlation

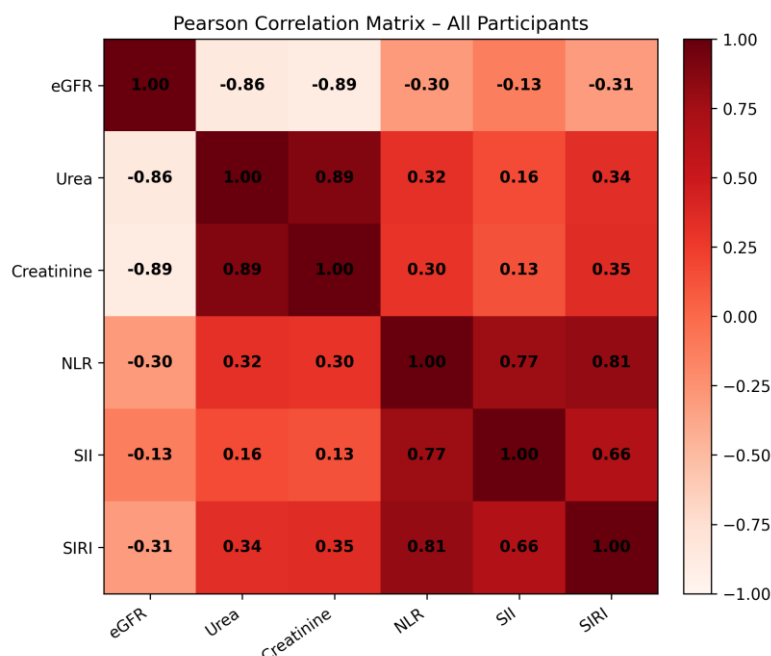


Figure 3. Pearson Correlation Matrix. eGFR and Inflammatory Indices – All Participants ($n = 161$). The values inside the squares represent Pearson correlation coefficients (r). Negative values indicate inverse correlations, whereas positive values indicate direct correlations.

4. Discussion

There is growing recognition that CKD is a systemic disorder, with persistent activation of inflammation contributing to disease progression beyond a simple decline in renal filtration capacity. This study focus on three inflammation-based indices derived from the CBC, namely NLR, SII, and SIRI, and their association with renal function parameters in patients with CKD [17]. The results showed that CKD patients had distinct inflammatory alterations characterized by increased NLR and SIRI values, together with significant association between these two indices and reduced eGFR.

The relationship between inflammation and CKD progression has been widely recognized. Chronic inflammatory activation in CKD is driven by multiple interconnected mechanisms, including oxidative stress, endothelial dysfunction, accumulation of uremic toxins, immune cell imbalance, and activation of pro-inflammatory signaling pathways. These processes contribute to ongoing renal injury,

fibrosis development, and increased cardiovascular risk among affected patients [18, 19]. In agreement with previous investigations, results of this study demonstrated higher NLR and SII values in CKD patients compared with healthy controls. NLR reflects the balance between neutrophil-associated inflammatory activity and lymphocyte-mediated immune regulation, and previous studies have reported its association with CKD severity and adverse outcomes [3, 20, 21]. The increased NLR may therefore represent a consequence of the persistent low-grade inflammatory state commonly observed in CKD.

Among the evaluated inflammatory indices, SIRI showed the strongest and most consistent relationship with impaired renal function. Unlike NLR, which incorporates only neutrophil and lymphocyte counts, SIRI additionally includes monocyte levels and may therefore capture a broader spectrum of immune activation. Previous population-based studies have also reported associations between elevated SIRI values and CKD prevalence, disease severity, and unfavorable clinical outcomes [2, 22, 23]. The biological relevance of SIRI may be related to the important role of monocytes in chronic renal inflammation. Activated monocytes contribute to cytokine production, macrophage recruitment, tissue remodeling, and fibrotic responses, all of which are involved in CKD progression.

In contrast, SII did not demonstrate a significant difference between CKD patients and controls in the unadjusted analysis, although it remained independently associated with eGFR after multivariable adjustment. This finding highlights the complexity of interpreting platelet-containing inflammatory indices in CKD populations. Platelet abnormalities are frequently observed in patients with renal dysfunction due to uremic toxicity, altered platelet production, dialysis-related effects, and changes in platelet activation pathways [24-26]. Therefore, although SII may provide additional information regarding inflammatory status, its interpretation should consider the hematological alterations accompanying CKD.

The observed changes in individual blood cell populations further support the presence of immune dysregulation in CKD. Although total leukocyte and neutrophil counts were not significantly different between groups, CKD patients showed lower lymphocyte counts, increased monocyte counts, and reduced platelet levels. Similar alterations have been described in chronic inflammatory conditions and may explain why composite inflammatory indices provide additional information compared with isolated hematological parameters [27-29].

The inverse associations observed between eGFR and inflammatory indices are consistent with previous studies demonstrating that increased systemic inflammation is linked to worsening renal function and higher cardiovascular risk in CKD populations [2, 3, 22, 30]. However, unlike some previous reports, SII did not differ significantly between groups in our cohort. This discrepancy may be explained by differences in the severity of CKD, dialysis status, demographic characteristics, underlying comorbidities, and population-specific characteristics [31-33].

There are a few practical advantages to CBC-based inflammatory indices from a clinical viewpoint. These are inexpensive, easy to compute from routinely available laboratory parameters and can be obtained without requiring additional testing. These traits may be especially useful in health care settings where advanced inflammatory biomarkers are not readily available. In this study, SIRI was most strongly associated with renal dysfunction, indicating that it may provide complementary information to conventional renal markers such as serum creatinine and eGFR [15, 34, 35].

The strengths point of this study, it is the first one in Basrah that use simultaneous evaluation of multiple CBC-derived inflammatory indices NLR, SII, and SIRI and assessment of their independent relationships with renal function urea, creatinine, and their equation estimated glomerular filtration rate (eGFR). Most previous studies were focused either on other inflammatory factors such as C-reactive protein (CRP) and ferritin, and percentage of CBC cells [36] or estimate biochemical parameters to eGFR and overlooked other inflammatory factors [37] or focused on just immunological markers such as high-sensitivity C-reactive protein (hsCRP), complement C3, and complement C4 from Erbil [37]. However, some limitations should be taken into account. The single-center case-control design limits causal inference and may limit generalizability to other CKD populations. In

addition, confounding factors such as diabetes status, hypertension, use of medications, and dialysis-related factors could not be fully included in the statistical models.

Future multicenter longitudinal studies in larger populations are required to confirm these findings and determine whether serial assessment of CBC-derived inflammatory indices can improve prediction of CKD progression and clinical outcomes. Integration of these accessible markers with conventional renal evaluation may provide a practical approach for improving inflammatory risk assessment in patients with CKD.

5. Conclusion

In this case-control study, we demonstrated that CBC-derived inflammatory indices are associated with impaired renal function in patients with chronic kidney disease. CKD patients showed significant alterations in inflammatory profiles, including increased NLR and SII values, together with marked reductions in eGFR and elevations in conventional renal biomarkers.

Among the evaluated indices, SII exhibited the most consistent association with renal dysfunction and remained independently related to eGFR after multivariable adjustment. This finding suggests that incorporating monocyte-related inflammatory information may provide additional insight into the systemic inflammatory burden associated with CKD. Although SII did not differ significantly between patients and controls, its independent association with eGFR indicates that different inflammatory indices may capture distinct aspects of CKD-related immune alterations.

Given that NLR, SII, and SII can be calculated from routinely available complete blood count parameters without additional laboratory costs, these markers may represent practical complementary tools for inflammatory assessment and risk stratification in CKD, particularly in settings with limited access to advanced biomarker testing.

Future investigations should evaluate whether serial monitoring of CBC-derived inflammatory markers can improve prediction of CKD progression, cardiovascular complications, and long-term clinical outcomes.

Abbreviations

CBC: Complete Blood Count

CKD: Chronic Kidney Disease

CKD-EPI: Chronic Kidney Disease Epidemiology Collaboration

eGFR: Estimated Glomerular Filtration Rate

NLR: Neutrophil-to-Lymphocyte Ratio

SII: Systemic Immune-Inflammation Index

SII: Systemic Inflammation Response Index

WBC: White Blood Cell

NEU: Neutrophil

LYM: Lymphocyte

MON: Monocyte

PLT: Platelet

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Ethics Approval Statement and Patient Consent

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Ethical approval was obtained from the appropriate institutional ethics committee prior to study initiation (BDC -8-1-26-3). Written informed consent was obtained from all participants before enrollment and blood sample collection.

Data Availability Statement

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

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The author received no specific funding for this work.

Conflict of Interest Disclosure

The author declares that there are no conflicts of interest regarding the publication of this article.

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دراسة حالة-مراقبة في البصرة، العراق: العلاقة بين مؤشرات الالتهاب المشتقة من تعداد الدم الكامل ووظائف الكلى لدى مرضى القصور الكلوي المزمن

الملخص

يُعرف القصور الكلوي المزمن بأنه تدهور تدريجي في وظائف الكلى، مع استمرار تنشيط مسارات الالتهاب التي تُسهم في تطور المرض ومضاعفاته. تُعد مؤشرات الالتهاب التقليدية مفيدة لسهولة استخدامها الروتيني وانتشارها الواسع. في الآونة الأخيرة، برزت مؤشرات الالتهاب المشتقة من تعداد الدم الكامل، بما في ذلك نسبة العدلات إلى اللمفاويات، ومؤشر الالتهاب المناعي الجهازى، ومؤشر الاستجابة الالتهابية الجهازية، كمؤشرات حيوية بسيطة وغير مكلفة ومتوفرة بسهولة لحالة الالتهاب الجهازى. ومع ذلك، لا تزال العلاقة بين هذه المؤشرات ووظائف الكلى غير مفهومة بشكل كافٍ بين مختلف الفئات السكانية. هدفت هذه الدراسة، التي اعتمدت على تصميم الحالة-المراقبة، إلى تقييم العلاقة بين مؤشرات الالتهاب المشتقة من تعداد الدم الكامل ووظائف الكلى لدى مرضى القصور الكلوي المزمن. تم تسجيل 161 شخصاً في الدراسة، منهم 79 مريضاً مصاباً بمرض الكلى المزمن و82 شخصاً سليماً كمجموعة ضابطة. تم تقييم المؤشرات الدموية الروتينية، ومستويات اليوريا والكرياتينين في الدم، ومعدل الترشيح الكبيبي المقدر (eGFR)، وحُسبت مؤشرات الالتهاب من بيانات تعداد الدم الكامل. استُخدمت الاختبارات الإحصائية لمقارنة المجموعات، كما استُخدم معامل ارتباط سيرمان لاستكشاف العلاقات بين المؤشرات ووظائف الكلى. كانت مستويات اليوريا والكرياتينين ونسبة العدلات إلى اللمفاويات (NLR) ومؤشر الالتهاب الجهازى (SIRI) في الدم أعلى بشكل ملحوظ لدى مرضى الكلى المزمن مقارنةً بالمجموعة الضابطة، بينما انخفضت قيم eGFR بشكل ملحوظ (جميع قيم $P < 0.001$). أظهر eGFR ارتباطات عكسية قوية مع الكرياتينين واليوريا، بالإضافة إلى ارتباطات سلبية مع NLR و SIRI. في المقابل، لم يختلف مؤشر الالتهاب الجهازى (SII) بشكل معنوي بين المجموعات. ($P = 0.105$) مع ذلك، في التحليل متعدد المتغيرات، ظل مؤشر الالتهاب الجهازى (SII) مرتبطاً بشكل مستقل بمعدل الترشيح الكبيبي المقدر (eGFR)، مما يشير إلى وجود علاقة محتملة بوظائف الكلى تتجاوز الاختلافات على مستوى المجموعة. ومن بين مؤشرات الالتهاب التي تم تقييمها، أظهر مؤشر استجابة الالتهاب الجهازى (SIRI) ارتباطاً أكثر اتساقاً وقوة باختلال وظائف الكلى. تشير هذه النتائج إلى أن مؤشرات الالتهاب المشتقة من تعداد الدم الكامل، وخاصة مؤشر استجابة الالتهاب الجهازى (SIRI)، قد تُستخدم كعلامات حيوية ميسورة التكلفة وسهلة الوصول لتقييم الالتهاب الجهازى واختلال وظائف الكلى في مرض الكلى المزمن. وعند دمجها مع معايير الكلى التقليدية، يمكنها تحسين تقييم المخاطر السريرية ومراقبة المرض. ومع ذلك، يلزم إجراء المزيد من الدراسات المستقبلية واسعة النطاق ومتعددة المراكز للتحقق من صحة هذه النتائج وتحديد جدواها السريرية.

الكلمات المفتاحية: مرض الكلى المزمن؛ الالتهاب؛ نسبة العدلات إلى اللمفاويات؛ مؤشر استجابة الالتهاب الجهازى؛ معدل الترشيح الكبيبي المقدر