



Neutrophil-to-Lymphocyte Ratio as a Predictor of Renal Function Decline Based on Serum Urea, Creatinine, and Estimated Glomerular Filtration Rate in Non-Hemodialysis Chronic Kidney Disease Patients

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Abstract

Chronic kidney disease (CKD) is a progressive condition affecting 9–13% of the global population, with diabetes and hypertension being the predominant risk factors. Chronic low-grade inflammation contributes to CKD progression, and the neutrophil-to-lymphocyte ratio (NLR) provides a simple and routinely available inflammatory index. This study examined whether NLR was associated with serum urea, serum creatinine, and estimated glomerular filtration rate (eGFR) in non-hemodialysis CKD patients. A cross-sectional analytical study was conducted at Dr. H. Abdul Moeloek Regional Hospital, Bandar Lampung, from March to May 2026. Using consecutive sampling, 264 patients with CKD stages G2–G4 were enrolled. Spearman's correlation analysis was applied with a significance threshold of $p < 0.05$. The mean NLR was 2.52 ± 1.32 . NLR demonstrated a positive correlation with serum creatinine ($r_s = +0.151$; $p = 0.014$) and a negative correlation with eGFR ($r_s = -0.155$; $p = 0.012$), whereas no significant correlation was observed with serum urea ($r_s = +0.066$; $p = 0.284$). The consistent direction of NLR associations with creatinine and eGFR supports its potential as a cost-effective supplementary marker for monitoring renal function decline in resource-limited settings.

INTRODUCTION

Chronic kidney disease (CKD) is defined as a structural or functional kidney abnormality persisting for three months or longer, with implications for health. According to the KDIGO 2012 Clinical Practice Guideline, CKD is classified into five stages (G1–G5) based on estimated glomerular filtration rate (eGFR) thresholds, with stage G3b and beyond carrying substantially elevated risks of disease progression and cardiovascular events (Levin et al., 2024). The global prevalence of CKD is estimated at 9–13% of the adult population, a figure that continues to increase in parallel with the worldwide burden of diabetes mellitus and hypertension (Hill et al., 2016). In Indonesia, both comorbidities have independent prognostic significance: data from Dr. H. Abdul Moeloek Regional Hospital demonstrated that diabetes ($p = 0.003$) and hypertension ($p = 0.019$) were each significantly associated with all-cause mortality in patients with CKD (Yonata & Islamy, 2025). These findings reflect a substantial clinical burden, while monitoring tools in many district hospitals remain limited to a small panel of basic laboratory tests.

Renal function is conventionally assessed through serum creatinine, serum urea, and eGFR. Creatinine is freely filtered by the glomerulus with minimal tubular secretion, making its serum concentration a reliable inverse surrogate marker of GFR. Its reference range and rate of increase in CKD are well established in standard nephrology literature (Bazilinski, 2000;

Matsushita et al., 2012). Creatinine, a metabolic byproduct of muscle turnover, increases predictably as glomerular filtration declines and remains a fundamental marker in clinical nephrology (Syamsi et al., 2021). Serum urea serves as a supplementary marker; however, its concentration is influenced by protein intake, hydration status, gastrointestinal bleeding, and catabolic states, limiting its specificity as a direct indicator of glomerular function (Syamsi et al., 2021). The CKD-EPI 2009 equation represented a major advancement in GFR estimation by improving accuracy across a broader range of kidney function compared with the MDRD formula (Levey et al., 2009). The CKD-EPI 2021 creatinine equation, developed without race as a variable, provides eGFR estimates that are more equitable and accurate across diverse populations (Inker et al., 2021). Together, these three parameters allow clinicians to stage CKD and monitor disease trajectories; however, none directly captures the inflammatory milieu that independently contributes to renal damage progression (Amdur et al., 2016; Okyay et al., 2013; Tonyali et al., 2018; Turkmen et al., 2012).

Systemic low-grade inflammation is increasingly recognized as a core mechanism in CKD progression rather than merely a consequence of reduced renal clearance. Neutrophil activation, cytokine release, and depletion of regulatory T cells collectively contribute to a cycle of tubulointerstitial fibrosis and glomerular hypertension (Akchurin & Kaskel, 2015). Stenvinkel et al. demonstrated that the transcription factor Nrf2, a key regulator of antioxidant defense, is progressively suppressed as CKD advances, creating an environment conducive to sustained oxidative stress and immune-mediated tubular injury that further accelerates GFR decline (Stenvinkel et al., 2021). The neutrophil-to-lymphocyte ratio (NLR) quantifies this imbalance using data derived from a standard complete blood count: an increased NLR reflects enhanced innate immune activity accompanied by relative adaptive immune suppression. Zahorec first described NLR as a rapid bedside index of systemic inflammatory stress in critically ill patients, and subsequent studies have expanded its application to cardiovascular disease, malignancy, and, more recently, CKD. Chen et al. (2020), in a cross-sectional study involving 15,219 participants undergoing routine health screening, found that NLR was positively correlated with serum creatinine ($r = 0.142$; $p < 0.001$) and blood urea nitrogen ($r = 0.090$; $p < 0.001$) among hypertensive participants, suggesting that inflammatory dysregulation may be detected even before CKD is formally diagnosed (Chen et al., 2020).

Despite this body of evidence, most published studies on NLR and kidney function have examined either eGFR or creatinine alone, involved populations outside Southeast Asia, or combined hemodialysis and non-hemodialysis patients in ways that obscure relationships during earlier CKD stages. No published study from Indonesia has simultaneously evaluated NLR in relation to urea, creatinine, and eGFR calculated using the CKD-EPI 2021 equation in a non-hemodialysis CKD cohort. The combination of an accessible biomarker (NLR) and three routinely collected renal indices provides a clinically practical framework that has not yet been characterized in this population (Burlacu et al., 2025; Mihai et al., 2018; Navarro-González & Mora-Fernández, 2008; Yoshitomi et al., 2019).

This study evaluated the correlation between NLR and three renal function parameters in patients with CKD stages G2–G4 at a regional referral hospital in Lampung Province. The primary research question was whether NLR, derived from a routine blood count, was associated with the degree of renal impairment as measured by serum creatinine, serum urea, and eGFR. Demonstrating such an association would support the potential use of NLR as a low-

cost adjunctive marker in settings where repeated nephrology referral or advanced biomarker testing is not feasible (Ananti, 2021; Angkananard et al., 2018; Kocyigit et al., 2013; Templeton et al., 2014; Zhao et al., 2020).

METHOD

Study Design

This study was an analytical observational study with a cross-sectional design conducted at the Department of Internal Medicine, Dr. H. Abdul Moeloek Regional Hospital, Bandar Lampung, from March to May 2026. The design was selected to evaluate the correlation between inflammatory and renal indices in patients with CKD.

Sample

The target population comprised adults with a confirmed diagnosis of CKD who were not receiving hemodialysis or any other form of renal replacement therapy. Consecutive sampling was applied: every patient who attended the inpatient or outpatient nephrology service and met eligibility criteria was enrolled until the target sample size was reached.

Inclusion criteria were: (1) CKD stage G2–G4 defined by eGFR 15–89 mL/min/1.73 m² using CKD-EPI 2021; (2) age above 17 years; and (3) a complete set of recorded laboratory results including absolute neutrophil and lymphocyte counts, serum urea, serum creatinine, age, and sex. Patients were excluded if they had an active acute infection, haematological or solid malignancy, systemic autoimmune disease, or were taking long-term systemic corticosteroids, as each of these independently perturbs NLR. A total of 264 patients were ultimately enrolled.

Data Collection Technique

Data were extracted from medical records after institutional approval. Variables collected were: age (years), sex, absolute neutrophil count (10⁹/L), absolute lymphocyte count (10⁹/L), serum urea (mg/dL), and serum creatinine (mg/dL). NLR was computed as the ratio of absolute neutrophil to absolute lymphocyte count. eGFR was calculated individually for each subject using the CKD-EPI 2021 creatinine equation, incorporating age and sex, without a race coefficient.⁸

Data Analysis Technique

All analyses were performed in SPSS version 26.0. Continuous variables are presented as mean ± SD or as median with interquartile range (IQR), depending on distribution. Categorical variables are presented as frequency and percentage. Normality was assessed using the Kolmogorov-Smirnov test. Because none of the key continuous variables satisfied normality assumptions, Spearman's rank correlation (ρ) was used to evaluate the relationship between NLR and each renal parameter. A two-tailed p-value below 0.05 was considered statistically significant. Correlation strength was interpreted according to Evans' criteria: very weak ($r_s < 0.20$), weak (0.20–0.39), moderate (0.40–0.59), strong (0.60–0.79), and very strong (≥ 0.80).

Ethical Consideration

Ethical clearance was obtained from the Abdul Moeloek Hospital Ethic Committee. As data were extracted retrospectively from existing medical records without direct patient contact, a formal individual informed consent waiver was granted in accordance with the Helsinki Declaration. Patient identifiers were removed before analysis, and data were used solely for this study.

RESULTS AND DISCUSSION

The 264 enrolled patients had a mean age of 50.79 ± 12.45 years, with the largest single group falling in the 46–55-year bracket (early elderly, 32.2%). Sex distribution was nearly balanced (136 male, 51.5%). By CKD stage, the cohort spread across G2 (26.5%), G3a (24.6%), G3b (29.2%), and G4 (19.7%), with no patient having an eGFR below 15 mL/min/1.73 m². Characteristics are summarised in Table 1.

Table 1. Participant Characteristics

Variable	N	%
Sex		
Male	136	51.5
Female	128	48.5
Age Group (yr)		
17–25 yr	7	2.7
26–35 yr	19	7.2
36–45 yr	66	25.0
46–55 yr	85	32.2
56–65 yr	51	19.3
>65 yr	36	13.6
eGFR (mL/min/1.73 m²)		
60–<90	70	26.5
45–<60	65	24.6
30–<45	77	29.2
15–<30	52	19.7

Source: Author's analysis based on medical record data (2026).

Descriptive statistics for the key continuous variables are shown in Table 2. Mean NLR was 2.52 ± 1.32 (median 2.24, IQR 1.62–3.21). Mean serum urea was 86.93 ± 37.73 mg/dL and mean serum creatinine was 1.78 ± 0.70 mg/dL. Mean eGFR was 46.81 ± 17.70 mL/min/1.73 m², consistent with a predominantly G3 population. All continuous variables failed the Kolmogorov-Smirnov normality test ($p < 0.05$), justifying the use of Spearman's correlation throughout.

Spearman correlation results are presented in Table 3. NLR showed a significant positive correlation with serum creatinine ($r = +0.151$; $p = 0.014$) and a significant negative correlation with eGFR ($r = -0.155$; $p = 0.012$). No statistically significant correlation was found between NLR and serum urea ($r = +0.066$; $p = 0.284$).

Table 2. Descriptive Statistics for Continuous Variables

Variable	Mean $\pm$ SD	Median	IQR	Min	Max
NLR	2.52 ± 1.32	2.24	1.62–3.21	0.50	10.00
Urea (mg/dL)	86.93 ± 37.73	85.55	57.93–116.15	15.0	149.0
Creatinine (mg/dL)	1.78 ± 0.70	1.58	1.24–2.23	0.78	4.57
eGFR (mL/min/1.73 m ²)	46.81 ± 17.70	45.50	33.73–60.53	16.00	85.10
Age (years)	50.79 ± 12.45	50.00	41.00–60.00	18	79

Source: Author's analysis based on clinical laboratory data (2026).

The relationship between NLR and serum urea, serum creatinine, and eGFR were Pearson/Spearman correlation as shown in table 3.

Table 3. Spearman Correlation Between NLR and Renal Function Parameters

Variable Pair	r	p-value	Correlation Strength
NLR vs Creatinine	+0.151	0.014*	Very weak
NLR vs Urea	+0.066	0.284	Not significant
NLR vs eGFR	−0.155	0.012*	Very weak

Source: Author's statistical analysis using Spearman correlation test (2026).

The findings of this study answer all four research questions posed. First, NLR demonstrated a statistically significant positive correlation with serum creatinine in non-hemodialysis CKD patients stages G2–G4 ($r_s = +0.151$; $p = 0.014$), indicating that higher NLR values are associated with higher serum creatinine levels. Second, no significant correlation was found between NLR and serum urea ($r_s = +0.066$; $p = 0.284$), suggesting that NLR does not track meaningfully with urea in this population. Third, NLR showed a statistically significant negative correlation with eGFR ($r_s = -0.155$; $p = 0.012$), confirming that higher NLR values correspond to lower estimated glomerular filtration rate. Taken together, NLR is significantly associated with two of the three renal function parameters evaluated—serum creatinine and eGFR, but not with serum urea, with all correlations classified as very weak in magnitude according to Evans' criteria. These results indicate that NLR, derived from a standard complete blood count, carries information about the degree of renal function decline in non-hemodialysis CKD patients and may therefore serve as a low-cost supplementary index alongside conventional renal markers in clinical practice.

The significant and directionally consistent associations between NLR and both creatinine and eGFR are biologically plausible. Akchurin and Kaskel documented that persistent low-grade inflammation in CKD, characterised by elevated pro-inflammatory cytokines (IL-6, TNF- α , IL-1 β) and oxidative stress, directly correlates with declining GFR and contributes to tubulointerstitial fibrosis and glomerulosclerosis. Navarro-González and Mora-Fernández showed that these same cytokines, particularly IL-6 and TNF- α , drive mesangial cell proliferation, podocyte injury, and proteinuria in the context of diabetic nephropathy, a pathway shared with non-diabetic CKD progression. Activated neutrophils release reactive oxygen species and matrix metalloproteinases that damage the glomerular basement membrane and accelerate tubular atrophy. Mihai et al. provided a systematic framework linking these inflammatory mechanisms to CKD prediction and outcome, demonstrating that circulating inflammatory markers, including CRP, IL-6, and TNF- α , independently predict both disease progression and cardiovascular mortality in CKD patients. Simultaneously, regulatory T-lymphocyte depletion removes a key brake on this inflammatory cascade. The CRIC study, a large prospective cohort of 3,939 CKD patients, confirmed this relationship longitudinally: baseline levels of CRP, fibrinogen, and IL-6 were each independently associated with faster eGFR decline and higher risk of ESRD over a median follow-up of 3.5 years.¹⁷ NLR captures precisely this dynamic: a numerator driven upward by innate immune activation and a denominator pulled down by adaptive immune suppression. Zahorec's original conceptualisation of NLR as a stress index in critical illness translates directly to the chronic inflammatory milieu of advanced CKD, where the immune imbalance is less acute but more sustained.

The absence of a significant NLR-urea correlation is not unexpected. Unlike creatinine, which is released at a relatively constant rate from skeletal muscle and eliminated almost exclusively by glomerular filtration, urea production varies substantially with dietary protein

intake, catabolic state, gastrointestinal bleeding, and hepatic urea cycle activity.⁶ These non-renal determinants introduce biological noise that attenuates its correlation with an inflammatory marker. Chen et al. (2020) observed a similar pattern: the NLR-BUN association was significant only in the highest NLR tertile (>3) and in the hypertensive subgroup, suggesting that detectable correlation with urea may require a threshold level of systemic inflammation not reached in most G2–G4 patients.

Several prior studies support these findings. Okayay et al. (2013) found that NLR was significantly higher across each CKD subgroup of predialysis, haemodialysis, and peritoneal dialysis, compared with healthy controls, and that it correlated positively with IL-6 and high-sensitivity CRP.¹⁸ Turkmen et al. (2012) reported that ESRD patients with $\text{NLR} \geq 3.5$ had significantly higher TNF- α levels compared with $\text{NLR} < 3.5$ patients. This finding established NLR as a valid inflammatory index even at the most advanced stage of kidney disease.¹⁹ Tonyali et al. (2018), examining NLR in a mixed cohort that included CKD outpatients and patients after partial or radical nephrectomy, found that NLR was significantly lower in healthy controls than in CKD patients (2.14 vs 3.53; $p = 0.005$) and inversely correlated with GFR.²⁰ Yoshitomi et al. (2019), in a prospective Japanese cohort of 350 CKD patients followed over a median of 39 months, showed that those with high NLR faced a hazard ratio of 1.67 (95% CI 1.02–2.77) for reaching a composite endpoint of end-stage renal disease or death.¹⁴ Kocyigit et al. (2013) identified NLR as an independent predictor of progression to dialysis in stage 4 CKD, and Burlacu et al. (2025), in a systematic review and meta-analysis, concluded that elevated NLR independently predicts renal function decline in CKD patients.^{16,21} At the population level, Zhao et al. (2020), pooling data from 10 cohort studies ($n = 1,442$), found NLR to be associated with increased risks of all-cause mortality (HR 1.45; 95% CI 1.20–1.75) and cardiovascular events (HR 1.52; 95% CI 1.33–1.72) in CKD patients.²²

The utility of NLR extends well beyond nephrology. In a systematic review of 38 studies totalling 76,002 participants, Angkananard et al. (2018) found that high NLR carried significantly elevated odds of coronary artery disease (OR 1.62; 95% CI 1.38–1.91), acute coronary syndrome, and stroke.²³ The oncology literature is similarly consistent: Templeton et al. (2014), pooling 100 studies across multiple solid tumour types and 40,559 patients, established NLR as an independent prognostic marker with high NLR predicting poor overall survival (HR 1.81; 95% CI 1.67–1.97).²⁴ In patients with type 2 diabetes mellitus undergoing percutaneous coronary intervention, elevated NLR has been linked to worse major adverse cardiovascular events.²⁴ Zahorec's original work demonstrated that NLR rises dramatically in sepsis and other critical states,¹¹ confirming that its sensitivity to systemic inflammatory burden cuts across disease categories rather than being organ-specific.

At a district hospital level in Indonesia, where access to advanced biomarkers such as high-sensitivity CRP, IL-6, or fibroblast growth factor-23 is limited, NLR derived from a routine complete blood count carries no additional cost. Its associations with creatinine and eGFR in this cohort indicate that clinicians reviewing a CKD patient's blood count could extract prognostic information that is currently overlooked. The progressive nature of CKD from early structural damage through to end-stage disease, underscores the clinical importance of identifying accessible markers that can signal deterioration before it becomes irreversible, particularly in patients who may not have ready access to specialist nephrology services. Yonata and Islamy (2025) have already shown that diabetes and hypertension independently predict

mortality in this hospital's CKD population;3 NLR may serve as an additional, inflammation-derived signal within the same monitoring framework. Practically, an NLR trending upward in a patient with stable creatinine may warrant closer monitoring and earlier specialist input.

Several limitations temper these findings. The cross-sectional design precludes causal inference; whether elevated NLR precedes and drives renal function loss, or merely reflects it, cannot be determined from these data. The single-centre setting limits generalisability to other hospital types and ethnic compositions within Indonesia. Potential confounders including nutritional status, subclinical infection, and concurrent medication use were incompletely captured in retrospective records. Comparative inflammatory markers such as CRP, albumin, or ferritin were not available for all patients, preventing adjustment for the broader inflammatory profile. The weak correlation coefficients observed (r_s approximately 0.15), while statistically significant at $n = 264$, represent modest explanatory power and should not be over-interpreted as evidence of a strong mechanistic link at the individual patient level.

CONCLUSION

NLR, derived from a routine complete blood count, correlates significantly with both serum creatinine ($r_s = +0.151$; $p = 0.014$) and estimated glomerular filtration rate (eGFR) ($r_s = -0.155$; $p = 0.012$) in non-hemodialysis CKD patients at stages G2–G4. No significant relationship was found with serum urea, which is consistent with urea's well-established sensitivity to non-renal factors. The directional consistency of the NLR–creatinine and NLR–eGFR associations supports its biological plausibility as an inflammatory index associated with renal function decline. In settings where advanced biomarkers are not routinely available, incorporating NLR interpretation into standard blood count assessments may provide a practical and cost-neutral approach to CKD monitoring. Prospective multicentre cohort studies with longer follow-up periods, standardised control of confounding variables, and receiver operating characteristic (ROC) curve analyses to establish clinically actionable NLR thresholds are required before this marker can be formally recommended for inclusion in CKD management protocols.

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