PROCEEDING

2nd Banten International Conference on Health Advancement & Research
binar@poltekkesbanten.ac.id
https://binar.poltekkesbanten.ac.id/
Vol. 1 No. 2 (2026)

ASSOCIATION OF NEUTROPHIL-TO-LYMPHOCYTE RATIO WITH REDUCED eGFR IN PATIENTS WITH HYPERTENSION AT TIGARAKSA REGIONAL GENERAL HOSPITAL

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ABSTRACT

Hypertension is an important risk factor for impaired kidney function, and systemic inflammation may contribute to renal injury. The neutrophil–to–lymphocyte ratio (NLR) is an accessible inflammatory marker derived from routine hematological testing. This study evaluated the association between NLR and reduced estimated glomerular filtration rate (eGFR) among patients with hypertension. An analytical cross–sectional study was conducted using secondary data from medical records at Tigaraksa Regional General Hospital. Patients with hypertension aged ≥30 years who had complete demographic and laboratory data were included using total sampling. NLR was calculated from neutrophil and lymphocyte values, while eGFR was estimated using the CKD–EPI equation. Reduced eGFR was defined as <60 mL/min/1.73 m². The primary exposure was categorized as NLR ≥3.0 versus <3.0. Associations were evaluated using chi–square and multivariable binary logistic regression. Receiver operating characteristic (ROC) analysis was performed to assess the discriminatory performance of continuous NLR. Ninety participants were analyzed. Reduced eGFR occurred in 53.5% of participants with NLR ≥3.0 and 36.2% of those with NLR <3.0. In the final adjusted model, NLR ≥3.0 was associated with higher odds of reduced eGFR (adjusted odds ratio [aOR] 2.48, 95% confidence interval [CI] 0.96–6.43; p=0.061). Continuous NLR showed limited discrimination (area under the curve 0.606, 95% CI 0.488–0.725; p=0.077). The NLR ≥3.0 category showed a positive but statistically non–significant association with reduced eGFR. NLR may have potential as an adjunctive marker of inflammatory burden, but its clinical utility for identifying reduced eGFR requires further evaluation.

Keywords: neutrophil-to-lymphocyte ratio; hypertension; estimated glomerular filtration rate; kidney function; inflammation

INTRODUCTION

Hypertension is a major risk factor for cardiovascular and kidney disease, and persistent elevation of blood pressure contributes to progressive renal injury (Lee et al., 2020). The pathophysiological relationship between hypertension and kidney dysfunction is complex and bidirectional. Sustained hypertension can promote vascular remodeling, endothelial dysfunction, oxidative stress, and renal tissue injury, whereas

impaired renal function can further contribute to blood pressure dysregulation through alterations in sodium handling, fluid balance, and neurohormonal mechanisms. Increasing evidence also indicates that immune and inflammatory mechanisms are involved in the development and progression of hypertension and its associated target-organ damage (Nguyen et al., 2024). Inflammatory and immune processes may contribute to renal injury through interactions between innate and adaptive immune responses, vascular dysfunction, oxidative stress, and inflammatory signaling. In hypertension, immune-cell activation can promote vascular and renal

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INTRODUCTION

Hypertension is a major risk factor for cardiovascular and kidney disease, and persistent elevation of blood pressure contributes to progressive renal injury (Lee et al., 2020). The pathophysiological relationship between hypertension and kidney dysfunction is complex and bidirectional. Sustained hypertension can promote vascular remodeling, endothelial dysfunction, oxidative stress, and renal tissue injury, whereas impaired renal function can further contribute to blood pressure dysregulation through alterations in sodium handling, fluid balance, and neurohormonal mechanisms. Increasing evidence also indicates that immune and inflammatory mechanisms are involved in the development and progression of hypertension and its associated target-organ damage (Nguyen et al., 2024). Inflammatory and immune processes may contribute to renal injury through interactions between innate and adaptive immune responses, vascular dysfunction, oxidative stress, and inflammatory signaling. In hypertension, immune-cell activation can promote vascular and renal

inflammation and may contribute to structural and functional changes in the kidney (An et al., 2022; Gao et al., 2023). These observations provide a biological rationale for investigating accessible hematological markers that may reflect systemic inflammatory and immune activity in patients with hypertension (Nguyen et al., 2024).

The neutrophil-to-lymphocyte ratio (NLR) is a simple hematological index calculated from routinely measured neutrophil and lymphocyte counts. By integrating information from two circulating leukocyte populations, NLR has been investigated as an indicator of systemic inflammatory and immune activity. Increasing primary research has linked higher NLR with impaired kidney function and adverse renal outcomes across different populations. In a large health-checkup population, Chen et al. reported that NLR was positively correlated with serum creatinine among hypertensive participants, with values >3 representing the highest NLR category (C. Chen et al., 2020). Similarly, a case-control study of hypertensive patients found that NLR was significantly higher in those with low eGFR than in those with normal eGFR and increased progressively across CKD stages. NLR was also significantly correlated with blood pressure, serum urea, creatinine, eGFR, albumin-to-creatinine ratio, and C-reactive protein, supporting its potential relevance to hypertensive renal dysfunction (Ibrahim et al., 2022). In a prospective cohort of non-dialysis patients with CKD, higher NLR was associated with lower eGFR and increased risks of mortality and composite renal outcomes, further supporting the potential relevance of NLR to renal dysfunction (Rashid et al., 2023).

In patients with H-type hypertension, higher NLR was also associated with renal damage and lower eGFR, with an NLR cut-off of 2.247 identified by ROC analysis (Z.-N. Chen et al., 2021). Among patients with type 2 diabetes, baseline NLR was independently associated with subsequent eGFR decline over two years, with participants in the highest NLR tertile showing substantially higher odds of rapid renal function decline (Akase et al., 2020). Similarly, higher NLR has been associated with diabetic nephropathy and albuminuria, with an AUC of 0.869 for NLR in discriminating diabetic nephropathy in one clinical study (Jaaban et al., 2021). In patients with established non-dialysis chronic kidney disease, the highest NLR tertile was associated with a greater incidence of composite kidney events, including substantial eGFR decline or initiation of renal replacement therapy (adjusted HR 3.33, 95% CI 1.43–7.76) (Kim et al., 2023). More recently, a prospective community-based cohort of 9,631 adults with preserved kidney function found a dose-dependent association between baseline NLR and incident CKD: each one-unit increase in NLR was associated with 19% higher odds of incident CKD, while NLR ≥3.0 was associated with 62% higher odds compared with NLR <1.5 (Yu et al., 2026). These findings collectively suggest that NLR may reflect renal risk across different stages of kidney disease, although differences in population characteristics, renal outcomes, and study designs preclude direct comparison of effect estimates and thresholds.

Despite these findings, the relationship between NLR and reduced estimated glomerular filtration rate (eGFR) specifically among patients with hypertension remains insufficiently characterized. A large health-checkup study of 4,997 individuals with hypertension reported associations between NLR and renal parameters, particularly serum creatinine and blood urea nitrogen, but did not specifically evaluate reduced eGFR as the primary outcome (C. Chen et al., 2020). In other populations, NLR has been associated with eGFR and kidney disease among patients with diabetes (Chunyu et al., 2025; Li et al., 2024), while prospective and cohort studies in broader or established CKD populations have primarily examined incident CKD, renal progression, or mortality rather than reduced eGFR at a single assessment (Lan et al., 2025; Liu et al., 2025; Yu et al., 2026). These differences in population characteristics, renal outcomes, disease stage, and study design make direct comparison of NLR–renal associations across populations challenging. Furthermore, NLR thresholds have varied across clinical populations and outcomes; for example, thresholds around 3.0 have been used in hypertensive or community-based populations, whereas a cut-off of 3.36 was identified for rapid eGFR decline in patients with established CKD (C. Chen et al., 2020; Lan et al., 2025; Yu et al., 2026). Therefore, further evaluation of NLR in defined hypertensive populations using clinically relevant measures of kidney function is warranted. This study aimed to evaluate the association between NLR and reduced eGFR among patients with hypertension at Tigaraksa Regional General Hospital and to assess the discriminatory performance of NLR for reduced eGFR.

MATERIALS AND METHODS

Study Design and Setting

This study used an analytical cross-sectional design based on secondary data obtained from patient medical records. The study evaluated the association between the neutrophil-to-lymphocyte ratio (NLR) and

kidney function assessed by estimated glomerular filtration rate (eGFR) among patients with hypertension. The study was conducted at Tigaraksa Regional General Hospital (RSUD Tigaraksa), Tangerang Regency, Banten, Indonesia. The study was conducted during April–June 2026.

Population and Sample

The target population consisted of patients diagnosed with hypertension, while the accessible population comprised hypertensive patients who underwent laboratory examinations at RSUD Tigaraksa and had complete medical-record data. Participants were selected from the accessible population according to the predefined inclusion and exclusion criteria.

The minimum sample size was estimated using a sample-size formula for correlation analysis between two numerical variables, assuming an expected correlation coefficient of 0.30, a significance level of 0.05, and 80% statistical power. The calculation yielded a minimum sample size of approximately 85 participants. All records meeting the eligibility criteria during the study period were subsequently included using total sampling, resulting in 90 participants for the final analysis. Participants were included if they had a documented diagnosis of hypertension, were aged ≥30 years, had laboratory data for neutrophils, lymphocytes, and serum creatinine, and had complete demographic information. Records were excluded when data were incomplete or when participants had acute infection, hematological disease, autoimmune disease, or malignancy.

Data Collection and Study Variables

Data were extracted from patient medical records using a standardized data-collection form. The variables included age, sex, systolic and diastolic blood pressure, body mass index (BMI), diabetes mellitus status, neutrophil and lymphocyte values, serum creatinine, NLR, and eGFR. NLR was calculated as the ratio of the neutrophil percentage to the lymphocyte percentage obtained from the differential leukocyte count. For the primary analysis, NLR was categorized using a literature-informed threshold of ≥3.0, with NLR <3.0 classified as non-elevated. The threshold was selected a priori based on previous evidence in hypertensive and community-based populations (C. Chen et al., 2020; Yu et al., 2026). Kidney function was assessed using estimated glomerular filtration rate (eGFR), calculated from serum creatinine, age, and sex using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) creatinine equation (Levey et al., 2009). Reduced eGFR was defined as <60 mL/min/1.73 m², whereas eGFR ≥60 mL/min/1.73 m² was classified as non-reduced eGFR (Stevens et al., 2024). For the primary outcome analysis, reduced eGFR was evaluated as a binary outcome. The eGFR categories G1, G2, G3a, G3b, G4, and G5 were also described to characterize the distribution of kidney function in the study population (Stevens et al., 2024).

Statistical Analysis

Statistical analyses were performed using Jamovi. Continuous variables were summarized as mean ± standard deviation or median with interquartile range, as appropriate, while categorical variables were presented as frequencies and percentages. The distribution of continuous variables was assessed using the Shapiro–Wilk test. The association between NLR category and reduced eGFR was evaluated using Pearson's chi-square test. Fisher's exact test was additionally reported. The crude odds ratio (OR) with its 95% confidence interval (CI) was calculated to quantify the unadjusted association between the NLR ≥3.0 category and reduced eGFR.

A binary logistic regression model was used to evaluate the association between the NLR ≥3.0 category and reduced eGFR (<60 mL/min/1.73 m²) after adjustment for potential confounding variables. The initial model included NLR category, age, sex, diabetes mellitus, systolic blood pressure, and BMI

category. Purposeful confounder assessment was performed by sequentially removing candidate covariates and evaluating the percentage change in the NLR regression coefficient. A 10% change in the NLR coefficient was used as a practical criterion for identifying potentially important confounding, while age was retained a priori based on its clinical and epidemiological relevance. The final model therefore included NLR category, age, systolic blood pressure, and BMI category.

Adjusted odds ratios (aORs), 95% CIs, and p-values were reported. A two-sided p-value <0.05 was considered statistically significant. Receiver operating characteristic (ROC) analysis was performed using continuous NLR to evaluate its ability to discriminate participants with reduced eGFR from those with eGFR ≥60 mL/min/1.73 m². The area under the ROC curve (AUC), sensitivity, specificity, positive and negative predictive values, likelihood ratios, and overall accuracy were calculated. The NLR value corresponding to the maximum Youden's Index was identified as the optimal sample-derived cut-off. This ROC-derived cut-off was considered exploratory and was not used to replace the literature-based NLR threshold of 3.0 in the primary association analysis.

Ethical Considerations

The study received ethical approval under Ethical Approval No. DP.03.04/F.XII.25/00221/2026, issued on 24 April 2026 and valid until 24 April 2027. The study used secondary data obtained from medical records and did not involve direct intervention or contact with participants. Personal identifiers were removed from the analytical dataset, and the results were reported in aggregate form.

RESULTS

Participant Characteristics

A total of 90 participants were included in the analysis, with no missing data for the variables evaluated. The mean age was 57.0 ± 10.7 years. Most participants were female (63, 70.0%), while 27 (30.0%) were male. The mean systolic and diastolic blood pressures were 151 ± 19.6 mmHg and 90.2 ± 14.3 mmHg, respectively. The mean BMI was 23.7 ± 4.38 kg/m². Based on the BMI categories used in the dataset, 11 participants (12.2%) were classified as underweight, 29 (32.2%) as normal weight, 21 (23.3%) as overweight, 22 (24.4%) as obese class I, and 7 (7.8%) as obese class II. Diabetes mellitus was present in 47 participants (52.2%), while 43 (47.8%) did not have diabetes mellitus.

The mean neutrophil and lymphocyte percentages were 67.6 ± 13.7% and 22.8 ± 11.5%, respectively. The median NLR was 2.88 (IQR, 1.82–6.73). Using the literature-based threshold of NLR ≥3.0, 43 participants (47.8%) were classified as having elevated NLR, while 47 (52.2%) had NLR <3.0. The serum creatinine concentration had a median of 1.00 mg/dL (IQR, 0.725–1.60), and the mean eGFR was 66.8 ± 32.7 mL/min/1.73 m². According to eGFR categories, 29 participants (32.2%) were classified as G1, 21 (23.3%) as G2, 11 (12.2%) as G3a, 14 (15.6%) as G3b, 9 (10.0%) as G4, and 6 (6.7%) as G5. Overall, 40 participants (44.4%) had reduced eGFR (<60 mL/min/1.73 m²), whereas 50 (55.6%) had eGFR ≥60 mL/min/1.73 m².

Table 1 Baseline Characteristics of the Study Population

Characteristic	Value
Age, years	57.0 ± 10.7
Sex, n (%)	
Male	27 (30.0)
Female	63 (70.0)
Systolic blood pressure, mmHg	151 ± 19.6
Diastolic blood pressure, mmHg	90.2 ± 14.3
BMI, kg/m²	23.7 ± 4.38

Characteristic	Value
BMI category, n (%)	
Underweight	11 (12.2)
Normal	29 (32.2)
Overweight	21 (23.3)
Obese class I	22 (24.4)
Obese class II	7 (7.8)
Diabetes mellitus, n (%)	
No	43 (47.8)
Yes	47 (52.2)
Neutrophil, %	67.6 ± 13.7
Lymphocyte, %	22.8 ± 11.5
NLR, median (IQR)	2.88 (1.82–6.73)
NLR category, n (%)	
NLR <3.0	47 (52.2)
NLR ≥3.0	43 (47.8)
Serum creatinine, mg/dL, median (IQR)	1.00 (0.725–1.60)
eGFR, mL/min/1.73 m²	66.8 ± 32.7
eGFR category, n (%)	
G1 (≥90)	29 (32.2)
G2 (60–89)	21 (23.3)
G3a (45–59)	11 (12.2)
G3b (30–44)	14 (15.6)
G4 (15–29)	9 (10.0)
G5 (<15)	6 (6.7)
eGFR <60	40 (44.4)
eGFR ≥60	50 (55.6)

Note: Values are presented as mean ± standard deviation, median (interquartile range), or n (%), as appropriate. NLR, neutrophil-to-lymphocyte ratio; eGFR, estimated glomerular filtration rate; BMI, body mass index.

Bivariate Association Between NLR and Reduced eGFR

Among participants with elevated NLR (≥3.0), 23 of 43 (53.5%) had reduced eGFR (<60 mL/min/1.73 m²), compared with 17 of 47 (36.2%) among participants with NLR <3.0. Pearson's chi-square test showed that the association between NLR category and reduced eGFR was not statistically significant ($\chi^2 = 2.73$, df = 1; p = 0.099). Fisher's exact test yielded a similar result (p = 0.137). The crude odds ratio was approximately 2.03 (95% CI, 0.87–4.72), indicating higher crude odds of reduced eGFR among participants with elevated NLR, although the confidence interval included the null value.

Table 2 Bivariate Association Between NLR Category and Reduced eGFR

NLR category	eGFR ≥60, n (%)	eGFR <60, n (%)	Total, n
NLR <3.0	30 (63.8)	17 (36.2)	47
NLR ≥3.0	20 (46.5)	23 (53.5)	43
Total	**50 (55.6)**	**40 (44.4)**	**90**

Pearson's $\chi^2 = 2.73$, df = 1, p = 0.099; Fisher's exact test, p = 0.137.

Multivariable Analysis

A binary logistic regression model was used to evaluate the association between elevated NLR (≥3.0) and reduced eGFR (<60 mL/min/1.73 m²). The initial model included NLR category, age, sex, diabetes mellitus, systolic blood pressure, and BMI category. Purposeful confounder assessment was performed by evaluating the change in the NLR regression coefficient following sequential removal of candidate covariates. Removal of sex and diabetes mellitus resulted

in minimal changes in the NLR coefficient, corresponding to changes of 0.61% and 0.74%, respectively. Age was retained as an a priori clinically relevant covariate, despite a change below the 10% confounding threshold. BMI category was retained because its removal resulted in a 12.51% change in the NLR coefficient.

The final model therefore included NLR category, age, systolic blood pressure, and BMI category, with sex and diabetes mellitus excluded from the final adjustment set. In the final adjusted model, elevated NLR was associated with higher odds of reduced eGFR, although the association did not reach conventional statistical significance (aOR = 2.48, 95% CI, 0.96–6.43; p = 0.061). Thus, participants with NLR ≥3.0 had approximately 2.5−fold higher adjusted odds of reduced eGFR than those with NLR <3.0; however, the confidence interval included 1.0.

Systolic blood pressure was independently associated with reduced eGFR (aOR = 1.032, 95% CI, 1.006–1.060; p = 0.016). Compared with participants with normal BMI, those classified as underweight had lower odds of reduced eGFR (aOR = 0.150, 95% CI, 0.026–0.863; p = 0.034). The other BMI categories were not statistically significant. The final model had a deviance of 107, an AIC of 123, and a McFadden's R^2 of 0.132.

Table 3 Final Multivariable Logistic Regression Model for Reduced eGFR

Predictor	Adjusted OR	95% CI	*p*
NLR ≥3.0 vs <3.0	**2.483**	**0.960–6.427**	**0.061**
Age, per year	1.032	0.983–1.084	0.207
Systolic blood pressure, per mmHg	1.032	1.006–1.060	0.016
BMI category			
Underweight vs normal	0.150	0.026–0.863	0.034
Overweight vs normal	0.331	0.094–1.167	0.088
Obese class I vs normal	0.504	0.148–1.718	0.273
Obese class II vs normal	2.037	0.297–13.973	0.469

Note: OR, odds ratio; CI, confidence interval; NLR, neutrophil−to−lymphocyte ratio; BMI, body mass index. The outcome was coded as 1 for eGFR <60 mL/min/1.73 m² and 0 for eGFR ≥60 mL/min/1.73 m². Reference categories were NLR <3.0 and normal BMI. Model deviance = 107; AIC = 123; McFadden's R^2 = 0.132; N = 90.

Discriminatory Performance of NLR for Reduced eGFR

Receiver operating characteristic (ROC) analysis was performed using continuous NLR to evaluate its ability to discriminate participants with reduced eGFR (<60 mL/min/1.73 m²) from those with eGFR ≥60 mL/min/1.73 m². The area under the ROC curve (AUC) was 0.606 (95% CI, 0.488–0.725; p = 0.077), indicating poor discriminatory performance with substantial uncertainty. The maximum Youden's Index was observed at an NLR cut−off of 3.445, with a sensitivity of 57.5% and specificity of 68.0% (Youden's Index = 0.255). At this cut−off, the positive predictive value was 59.0%, negative predictive value was 66.7%, and overall accuracy was 63.3%.

Table 4 ROC Analysis of NLR for Reduced eGFR

Measure	Estimate	95% CI
AUC	0.606	0.488–0.725
Optimal NLR cut-off	3.445	—
Sensitivity	57.5%	40.89–72.96%
Specificity	68.0%	53.30–80.48%
Positive likelihood ratio	1.797	1.107–2.915
Negative likelihood ratio	0.625	0.416–0.939
Positive predictive value	59.0%	46.98–69.99%
Negative predictive value	66.7%	57.09–75.04%
Accuracy	63.3%	52.51–73.25%

Note: AUC, area under the receiver operating characteristic curve; CI, confidence interval. The optimal cut-off was determined using the maximum Youden's Index. Reduced eGFR was defined as eGFR <60 mL/min/1.73 m².

DISCUSSION

Principal Findings

This study investigated the association between the neutrophil–to–lymphocyte ratio (NLR) and reduced estimated glomerular filtration rate (eGFR) among patients with hypertension. Participants with NLR ≥3.0 had a higher proportion of reduced eGFR than those with NLR <3.0 (53.5% vs. 36.2%). The corresponding crude odds ratio was approximately 2.03, indicating that participants with elevated NLR had approximately twice the odds of reduced eGFR compared with those with lower NLR. After adjustment for age, systolic blood pressure, and body mass index (BMI), the magnitude of the association increased, with an adjusted odds ratio (aOR) of 2.48 (95% CI 0.96–6.43). However, the confidence interval included the null value and the p–value was 0.061. Therefore, the present findings should be interpreted as a positive association that did not reach conventional statistical significance, rather than as evidence that elevated NLR is an independent predictor of reduced eGFR. The direction of the association nevertheless provides a clinically and biologically relevant signal. The approximately 2.5–fold adjusted odds observed for NLR ≥3.0 suggests that higher systemic inflammatory burden may coexist with reduced kidney filtration in hypertensive patients, although the precision of the estimate was limited by the sample size. The relatively wide 95% confidence interval also indicates substantial uncertainty around the estimated effect. Consequently, the present study supports the hypothesis that NLR may be related to renal impairment in hypertension, but it does not establish the strength or independence of this relationship with sufficient certainty. This distinction is particularly important because statistical non–significance should not be interpreted as proof of absence of an association, while the positive point estimate should likewise not be interpreted as definitive evidence of an independent effect.

The ROC analysis provided complementary evidence regarding the potential clinical utility of NLR. Continuous NLR demonstrated limited discriminatory ability for reduced eGFR, with an AUC of 0.606 (95% CI 0.488–0.725; p=0.077). The maximum Youden's Index was obtained at an NLR value of 3.445, corresponding to 57.5% sensitivity and 68.0% specificity. Thus, although the direction of the association observed in regression analysis was compatible with an inflammatory relationship, NLR alone provided insufficient discrimination to distinguish patients with reduced eGFR from those with eGFR ≥60 mL/min/1.73 m². The regression and ROC findings are therefore complementary: NLR may be associated with renal impairment at a population level without necessarily having adequate diagnostic performance as an individual–level screening marker.

Comparison with Previous Studies

The positive direction of the present association is consistent with a growing body of primary research linking NLR with impaired kidney function and adverse renal outcomes across different populations. A recent prospective community–based cohort of 9,631 adults provides particularly relevant evidence because NLR was assessed before the development of CKD. During follow–up, 400 participants developed incident CKD, and each one–unit

increase in NLR was associated with higher odds of incident CKD after multivariable adjustment (OR 1.19, 95% CI 1.06–1.32). A dose–response pattern was also observed, with participants with NLR ≥3.0 having greater odds of incident CKD than those with NLR <1.5 (OR 1.62). These findings support the possibility that elevated NLR may precede clinically apparent renal dysfunction, although the prospective design and larger sample provide stronger evidence for temporal association than the cross-sectional design of the present study (Yu et al., 2026). Evidence from populations with impaired kidney function further supports this association. In a longitudinal cohort of 1,224 multi-ethnic Asian adults with type 2 diabetes, elevated NLR was associated with subsequent renal function decline over a median follow-up of 1.8 years; the association remained significant after multivariable adjustment (HR 1.38, 95% CI 1.19–1.59). Addition of NLR to a model containing demographic, metabolic, renal, and treatment variables significantly improved risk discrimination (p=0.030) (Moh et al., 2022).

Similarly, a retrospective cohort study of 141 patients with non-dialysis CKD found that patients in the highest NLR tertile had lower eGFR and higher serum creatinine at baseline than those in the lowest tertile. During a mean follow-up of 5.45 years, the highest-NLR group had a significantly greater incidence of composite kidney events, defined as ≥50% eGFR decline or initiation of renal replacement therapy, and high NLR remained associated with these outcomes after adjustment (HR 3.33, 95% CI 1.43–7.76) (Kim et al., 2023). Longitudinal evidence from patients with type 2 diabetes similarly supports an association between NLR and subsequent renal deterioration. In a cohort of 1,224 adults with type 2 diabetes followed for a median of 2 years, higher baseline NLR independently predicted progressive kidney function decline (HR 1.39, 95% CI 1.21–1.60; P < 0.001) and albuminuria progression (HR 1.34, 95% CI 1.08–1.68; P = 0.009), while adding NLR to a multivariable base model significantly improved discrimination for kidney function decline (P = 0.022) (Moh et al., 2023). Studies using reduced eGFR as an outcome also provide supportive evidence. In a population-based analysis of 7,153 adults with type 2 diabetes from NHANES, higher NLR was positively associated with diabetic kidney disease, albuminuria, and low eGFR (<60 mL/min/1.73 m²), with associations remaining after multivariable adjustment. NLR also demonstrated discriminatory ability for identifying these renal outcomes in ROC analyses (Li et al., 2024). Evidence from related leukocyte-derived inflammatory indices also suggests that inflammatory biomarkers may be more strongly associated with specific manifestations of renal disease than with isolated eGFR decline. In a prospective cohort of hypertensive patients, higher circulating lymphocyte-to-monocyte ratio (CLR) was independently associated with incident proteinuric CKD and any incident CKD, whereas no significant association was observed with isolated eGFR decline. The addition of CLR to traditional risk models also improved risk reclassification, suggesting that inflammatory indices may provide complementary information beyond eGFR alone (Yang et al., 2026).

Evidence specifically from hypertensive populations is particularly relevant to the present study. In a health-checkup cohort of 15,219 individuals, including 4,997 participants with hypertension, NLR was positively correlated with serum creatinine and blood urea nitrogen among hypertensive participants. NLR was categorized into tertiles, with values >3 forming the highest category, providing population-specific evidence that NLR around the threshold examined in the present study is associated with renal parameters in hypertension (C. Chen et al., 2020). Among 3,067 adults with hypertension from the NHANES cohort, higher NLR was associated with increased all-cause and cardiovascular mortality, and eGFR partially mediated these associations (Zhang et al., 2024). Similarly, in a retrospective cohort of patients with coronary heart disease and hypertension, higher NLR was associated with increased all-cause and cardiovascular mortality after adjustment for potential confounders; higher NLR was also accompanied by lower eGFR (Song et al., 2024). More recent evidence also suggests that the relationship between NLR and renal function may extend to clinically meaningful renal progression. In a study of 377 patients with CKD stages 3–4, Lan et al. identified an NLR cut-off of 3.36 for rapid eGFR decline, with an AUC of 0.877 (95% CI 0.837–0.918), sensitivity of 88.6%, and specificity of 81.7% (Lan et al., 2025). In another clinical population, higher NLR was associated with lower eGFR and increased odds of CKD among uric acid stone formers, although the specific population and renal context differed substantially from the present hypertensive cohort (Tung et al., 2023). Although the population and outcome differed substantially from those of the present study, the proximity of this threshold to the sample-derived cut-off of 3.445 observed in our study is noteworthy. However, this similarity should be regarded as hypothesis-generating rather than as external validation.

Taken together, the available evidence suggests that the association between NLR and renal dysfunction is reproducible in direction but variable in magnitude. The stronger associations reported in prospective CKD cohorts and established CKD populations should not be directly extrapolated to our hospital-based hypertensive population. Differences in baseline renal function, age, comorbidity burden, inflammation, outcome definition, and follow-up duration may all influence the observed relationship. Our finding of an aOR of 2.48 with p=0.061 therefore represents a plausible but statistically imprecise estimate within this particular population.

Biological Interpretation

The observed positive association between elevated NLR and reduced eGFR is biologically plausible within the context of hypertension and renal injury. NLR reflects the relative balance between circulating neutrophils and lymphocytes and may capture systemic inflammatory and immune activity. Emerging evidence indicates that both innate and adaptive immune responses contribute to hypertension and its target-organ damage, with inflammatory cytokines, reactive oxygen species, and immune-cell activation implicated in vascular and renal injury (Nguyen et al., 2024). In the hypertensive kidney, innate immune cells, including neutrophils, can influence oxidative stress and inflammatory signaling, while adaptive immune responses can alter renal sodium and water handling (Lu & Crowley, 2020). In hypertension, persistent elevation of blood pressure can promote renal vascular injury, oxidative stress, and inflammatory activation. Immune cells, including neutrophils and lymphocytes, participate in the pathophysiology of hypertensive renal injury through interactions with renal vascular and parenchymal cells(Hao et al., 2024). A recent review of hypertensive nephropathy describes persistent hypertension as contributing to intrarenal arteriolar injury, ischemic renal parenchymal damage, glomerulosclerosis, tubular atrophy, and interstitial fibrosis, with inflammatory processes contributing to these changes (Hao et al., 2024). Experimental and mechanistic literature also indicates that innate and adaptive immune responses can influence blood pressure and renal sodium and water handling, providing a biological link between immune activation, hypertension, and renal injury (Lu & Crowley, 2020).

Neutrophils may contribute to this inflammatory environment through oxidative and proteolytic activity. Recent systematic evidence indicates that neutrophil dysfunction in CKD can include increased degranulation and basal reactive oxygen species production, particularly in patients receiving hemodialysis (Lauxen et al., 2025). More broadly, oxidative stress and pro-inflammatory signaling can impair endothelial function, reduce nitric oxide bioavailability, and promote leukocyte adhesion and vascular injury. These processes are relevant to renal microvascular dysfunction and may contribute to progressive impairment of kidney function (J.-H. Wang et al., 2025). The lymphocyte component of NLR may provide an additional dimension to this interpretation. An elevated NLR can arise not only from neutrophilia but also from relative lymphopenia (Buonacera et al., 2022). Chronic metabolic stress, inflammation, and the uremic environment can alter adaptive immune function, potentially producing an imbalance between innate and adaptive immune responses. Thus, NLR may provide an integrated indicator of the relative balance between innate and adaptive immune cell populations (R. Wang et al., 2024).

Importantly, these mechanisms should be interpreted as biological plausibility rather than evidence of causality in the present study. Because the present analysis was based on observational medical-record data and assessed NLR and eGFR within the same study period, it cannot establish whether elevated NLR contributes to declining kidney function, results from impaired kidney function, or reflects common upstream processes such as hypertension-related vascular injury, metabolic stress, or other comorbid conditions. This distinction is particularly important because recent literature has identified a strong association between NLR and adverse renal outcomes but has also emphasized the need for prospective studies to clarify temporal relationships and establish clinically useful thresholds (Burlacu et al., 2025). The biological interpretation also helps explain why NLR may show an association with renal dysfunction without demonstrating strong diagnostic discrimination. NLR integrates several nonspecific inflammatory and immune processes and is therefore influenced by multiple conditions beyond renal injury. The AUC of 0.606 observed in the present study supports this interpretation: although higher NLR was associated with a tendency toward reduced eGFR, NLR alone provided limited separation between participants with and without reduced eGFR. Accordingly, NLR is more appropriately viewed as a potential adjunctive marker of inflammatory burden or risk stratification, rather than a standalone indicator of reduced kidney function.

Role of Covariates and Confounding

The purposeful confounder assessment indicated that the estimated association between NLR ≥3.0 and reduced eGFR remained directionally stable after sequential adjustment. Sex and diabetes mellitus produced minimal

changes in the NLR coefficient when removed from the model, whereas removal of BMI resulted in a 12.51% change in the NLR coefficient. BMI was therefore retained in the final model. Age was also retained based on its prespecified clinical and epidemiological relevance. The use of change in the exposure coefficient to assess potential confounding is consistent with the change-in-estimate approach commonly used in epidemiological analyses. However, the 10% criterion should be regarded as a practical rule rather than an absolute statistical threshold. In logistic regression, changes in regression estimates after adjustment can also arise from noncollapsibility of the odds ratio, even in the absence of conventional confounding (Schuster et al., 2021). Accordingly, the present variable-selection process should be interpreted as a structured modeling strategy rather than definitive proof that individual covariates were or were not biological confounders.

Systolic blood pressure remained independently associated with reduced eGFR in the final model (aOR = 1.032, 95% CI 1.006–1.060; p = 0.016). This finding is consistent with longitudinal evidence from hypertensive populations showing that blood-pressure characteristics are associated with subsequent renal function decline (Cheng et al., 2019). The biological plausibility of this relationship is further supported by evidence that hypertension-related hemodynamic and vascular disturbances contribute to renal injury. In parallel, BMI has been associated with subsequent CKD development and renal function decline in hypertensive populations, supporting its consideration as a clinically relevant covariate (Xie et al., 2018). The potential relevance of BMI to the NLR–renal association is also supported by population-based evidence showing that higher NLR was associated with CKD among overweight/obese individuals, whereas this association was not observed in normal-weight participants (Lin et al., 2022). The retention of BMI in the present model was based primarily on its influence on the estimated NLR effect rather than on the statistical significance of its individual categories. This distinction is important because a covariate can influence the exposure–outcome estimate without itself demonstrating a statistically significant independent association with the outcome. In the present analysis, removal of BMI produced a 12.51% change in the NLR coefficient and therefore altered the estimated association sufficiently to justify its retention within the prespecified modeling strategy.

The inverse association observed for the underweight category should nevertheless be interpreted cautiously. Participants classified as underweight had lower adjusted odds of reduced eGFR than those with normal BMI (aOR = 0.150, 95% CI 0.026–0.863; p = 0.034). This isolated finding should not be interpreted as evidence of a protective effect of low BMI. Recent population-based evidence has demonstrated a U-shaped association between BMI and kidney function decline, with both underweight and obesity associated with increased risk of subsequent eGFR decline (Nakamura et al., 2026). Other longitudinal studies have similarly demonstrated complex, non-linear relationships between BMI and renal outcomes. Therefore, the apparently lower odds observed in the present underweight subgroup may reflect the small number of participants in this category, residual confounding, or imprecision rather than a true protective effect. Overall, adjustment for age, systolic blood pressure, and BMI did not eliminate the positive association between NLR ≥3.0 and reduced eGFR, but the association remained statistically uncertain. The final adjusted estimate (aOR = 2.48, 95% CI 0.96–6.43; p = 0.061) therefore supports a possible association rather than establishing NLR as an independent determinant of reduced eGFR. The direction of this association is also consistent with previous primary research in hypertensive populations linking higher NLR with adverse renal parameters (C. Chen et al., 2020; Yu et al., 2026).

Interpretation of the NLR Threshold

The literature-informed threshold of NLR ≥3.0 used in the primary analysis was supported by previous evidence in hypertensive and community-based populations. In a large hypertensive health-checkup population, NLR values >3 were used to define the highest tertile, while a recent prospective community-based cohort explicitly categorized NLR ≥3.0 as a high-NLR group and demonstrated a dose-dependent association with incident CKD (C. Chen et al.,

2020; Yu et al., 2026). Thus, the threshold of 3.0 in the present study was used as a literature-informed analytical threshold rather than as a cut-off derived from our own data. The ROC analysis produced a sample-derived threshold of 3.445, which was numerically close to the literature-informed threshold of 3.0. Interestingly, a recent study by Lan et al. involving 377 patients with CKD stages 3–4 identified an NLR cut-off of 3.36 for predicting rapid eGFR decline, with an AUC of 0.877 (95% CI 0.837–0.918), sensitivity of 88.6%, and specificity of 81.7%. However, that study evaluated rapid renal function decline in patients with established CKD rather than reduced eGFR in a hypertensive population at a single assessment. Moreover, none of the evaluated blood-cell indices independently predicted transition to ESRD (Lan et al., 2025).

The numerical similarity between 3.445 in our study and 3.36 reported by Lan et al. is therefore noteworthy but should not be interpreted as external validation of the threshold (Lan et al., 2025). The markedly different AUC values—0.606 in the present study compared with 0.877 in Lan et al.—illustrate that the discriminatory performance of NLR depends strongly on the study population and outcome definition. A threshold that performs well for predicting rapid eGFR decline in patients with established CKD may not provide equivalent discrimination for identifying reduced eGFR among hypertensive patients. This distinction is important for the clinical interpretation of the present findings. The ROC-derived value of 3.445 was generated from the same 90 participants used for the primary analysis and therefore represents an internally derived exploratory threshold. Without external validation, the sensitivity and specificity observed in this sample may be unstable and may not be reproducible in another population. Accordingly, the present study should not be used to propose 3.445 as a clinical decision threshold.

Diagnostic Performance and Clinical Implications

The limited AUC of 0.606 indicates that NLR alone has poor-to-limited discriminatory ability for reduced eGFR in this population. The 95% CI included values close to no discrimination, and the AUC did not differ significantly from 0.50 ($p=0.077$). This finding is important because an association between a biomarker and an outcome does not necessarily imply adequate diagnostic discrimination. A biomarker may demonstrate a statistically or clinically meaningful association at the population level while still performing poorly when used to classify individual patients.

Recent evidence reinforces this distinction. The 2026 prospective cohort study found that NLR was independently and dose-dependently associated with incident CKD, but the study addressed risk association over time, not necessarily diagnostic discrimination at a single clinical encounter. Similarly, recent studies support the prognostic relevance of NLR in CKD but also emphasize heterogeneity and limitations in the certainty of evidence (Burlacu et al., 2025; Yu et al., 2026). Therefore, NLR should currently be regarded as a potential complementary inflammatory marker rather than a substitute for established measures of kidney function. In routine clinical practice, renal assessment remains dependent on serum creatinine-derived eGFR and, where indicated, markers of kidney damage such as albuminuria (Stevens et al., 2024). NLR may provide additional contextual information about inflammatory burden, particularly when interpreted together with blood pressure, metabolic factors, and established renal markers, but the present findings do not support using NLR alone to identify patients with reduced eGFR.

Strengths and Limitations

This study has several strengths. First, it addresses a clinically relevant question in a defined population of patients with hypertension, in whom kidney dysfunction is an important target-organ complication. Second, NLR was derived from routinely available hematological parameters, making the marker potentially feasible for use in settings where more specialized inflammatory biomarkers are not readily accessible. Third, the study combined categorical association analysis, multivariable adjustment, and ROC analysis, allowing evaluation of both the relationship between NLR and reduced eGFR and its discriminatory performance. Fourth, the final regression model was developed with explicit consideration of confounding rather than relying solely on unadjusted comparisons. Several limitations should be considered when interpreting the findings. The cross-sectional design prevents determination of temporal sequence and precludes causal inference. The study was conducted at a single regional hospital and included only 90 participants, which limits statistical power and generalizability. The relatively small number of outcome events relative to the number of model parameters may also have limited the precision and stability of the multivariable estimates.

Another important limitation is the use of a single eGFR assessment. Although an eGFR below 60 mL/min/1.73 m^2 identifies reduced filtration at the time of measurement, a single measurement does not establish persistence and therefore does not by itself establish chronic kidney disease. In addition, the retrospective nature of the study limited the availability of potentially important variables, including albuminuria and duration of hypertension, which may have provided more comprehensive characterization of kidney damage and disease severity. Finally, the ROC-derived NLR threshold of 3.445 was generated and

evaluated in the same sample. This creates a risk of optimism in the estimated diagnostic performance and means that the threshold requires independent validation before any clinical application. The present study should therefore be regarded as an exploratory evaluation of the NLR–renal function relationship rather than a validation study for an NLR diagnostic cut-off.

Implications and Future Research

Future research should prioritize larger prospective and multicenter studies to clarify the temporal relationship between NLR and renal function decline in patients with hypertension. Serial measurements of NLR would be particularly valuable because they could determine whether persistent or increasing inflammatory activity is associated with subsequent deterioration in eGFR rather than simply reflecting a single cross-sectional inflammatory state. Prospective designs would also help distinguish whether NLR is an antecedent marker, a consequence of declining renal function, or a marker reflecting shared underlying processes. Future studies should additionally incorporate established markers of kidney damage, particularly albuminuria, together with longitudinal eGFR measurements (Stevens et al., 2024). Combining NLR with age, blood pressure, diabetes mellitus, BMI, albuminuria, and other routinely available clinical variables may provide greater predictive value than NLR alone. Recent evidence suggests that NLR may contribute to renal risk stratification, but the incremental value of NLR beyond established risk factors remains insufficiently defined (Burlacu et al., 2025; Liu et al., 2025).

External validation of candidate NLR thresholds is also essential. The proximity of the thresholds observed in the present study (3.445) and Lan et al. (3.36) provides a useful hypothesis for future investigation, but the substantial difference in discriminatory performance demonstrates that threshold transferability cannot be assumed (Lan et al., 2025). A future multicenter study should therefore prespecify candidate thresholds, evaluate calibration and discrimination in independent samples, and determine whether adding NLR to established renal risk models meaningfully improves clinical prediction.

CONCLUSION

This study found a positive but statistically non-significant association between elevated neutrophil-to-lymphocyte ratio (NLR) and reduced eGFR among patients with hypertension. After adjustment for age, systolic blood pressure, and BMI category, participants with NLR ≥3.0 had approximately 2.5-fold higher odds of reduced eGFR compared with those with NLR <3.0 (aOR = 2.48, 95% CI 0.96–6.43; p = 0.061). The association therefore suggests a potential relationship between the NLR ≥3.0 category and reduced kidney function, but the present data do not provide sufficient statistical evidence to establish an independent association between NLR and reduced eGFR. Continuous NLR also demonstrated limited discriminatory performance for reduced eGFR (AUC = 0.606, 95% CI 0.488–0.725; p = 0.077). The sample-derived optimal threshold of 3.445 yielded 57.5% sensitivity and 68.0% specificity but should be regarded as exploratory rather than as an established clinical cut-off. Overall, NLR may have potential as an adjunctive marker of systemic inflammatory burden in hypertensive patients, but its clinical utility for identifying reduced eGFR requires confirmation in larger prospective studies with external validation.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest related to this study or its publication.

REFERENCES

Akase, T., Kawamoto, R., Ninomiya, D., Kikuchi, A., & Kumagi, T. (2020). Neutrophil-to-lymphocyte ratio is a predictor of renal dysfunction in Japanese patients with type 2 diabetes. *Diabetes & Metabolic Syndrome*, *14*(4), 481–487. https://doi.org/10.1016/j.dsx.2020.04.029

An, C., Jiao, B., Du, H., Tran, M., Zhou, D., & Wang, Y. (2022). Myeloid PTEN deficiency aggravates renal inflammation and fibrosis in angiotensin II-induced hypertension. *Journal of Cellular Physiology*, *237*(1), 983–991. https://doi.org/10.1002/jcp.30574

Buonacera, A., Stancanelli, B., Colaci, M., & Malatino, L. (2022). Neutrophil to Lymphocyte Ratio: An Emerging Marker of the Relationships between the Immune System and Diseases. *International Journal of Molecular Sciences*, *23*(7), 3636. https://doi.org/10.3390/ijms23073636

Burlacu, A., Namolovan, C. A., Brinza, C., Covic, A., Floria, M., Voroneanu, L., & Covic, A. (2025). Neutrophil-to-Lymphocyte Ratio (NLR)-Independent Prognostic Marker of Renal Function Decline in Chronic Kidney Disease: A Systematic Review and Meta-Analysis. *Journal of Clinical Medicine*, *14*(19). https://doi.org/10.3390/jcm14196822

Chen, C., Zhao, H. Y., & Zhang, Y. H. (2020). Correlation between neutrophil-to-lymphocyte ratio and kidney dysfunction in undiagnosed hypertensive population from general health checkup. *Journal of Clinical Hypertension (Greenwich, Conn.)*, *22*(1), 47–56. https://doi.org/10.1111/jch.13749

Chen, Z.-N., Huang, Y.-R., Chen, X., Liu, K., Li, S.-J., Yang, H., Chen, W., Ren, B.-Q., & Luo, Z.-H. (2021). Value of Neutrophil-To-Lymphocyte Ratio As A Marker of Renal Damage in Patients with H-Type Hypertension. *Biomarkers in Medicine*, *15*(9), 637–646. https://doi.org/10.2217/bmm-2020-0638

Cheng, D., Tang, Y., Li, H., Li, Y., & Sang, H. (2019). Nighttime blood pressure decline as a predictor of renal injury in patients with hypertension: a population-based cohort study. *Aging*, *11*(13), 4310–4322. https://doi.org/10.18632/aging.101873

Chunyu, J. I. A., WANG, G., WANG, J., CHEN, G., ZHENG, K., & Xuemei, L. I. (2025). Correlation Between Neutrophil to Lymphocyte Ratio and eGFR in Diabetic Patients: A Cross-sectional Analysis Based on NHANES Data. *Medical Journal of Peking Union Medical College Hospital*, *16*(2), 379–385. https://doi.org/https://doi.org/10.12290/xhyxzz.2024-0908

Gao, Y., Yu, W., Song, J., Nie, J., Cui, Z., Wen, S., Liu, B., & Liang, H. (2023). JMJD3 ablation in myeloid cells confers renoprotection in mice with DOCA/salt-induced hypertension. *Hypertension Research*, *46*(8), 1934–1948. https://doi.org/10.1038/s41440-023-01312-z

Hao, X.-M., Liu, Y., Hailaiti, D., Gong, Y., Zhang, X.-D., Yue, B.-N., Liu, J.-P., Wu, X.-L., Yang, K.-Z., Wang, J., & Liu, Q.-G. (2024). Mechanisms of inflammation modulation by different immune cells in hypertensive nephropathy. *Frontiers in Immunology*, *15*, 1333170. https://doi.org/10.3389/fimmu.2024.1333170

Ibrahim, M. E., El Shahawy, E. M. L., Mohammed, S. M., Mahmoud, A. T., Hefnawy, A. A., & Mansour, A. E. (2022). Neutrophil-to-Lymphocyte Ratio as a Novel Marker for Early Detection of Hypertensive Nephropathy and as a Predictor of Worsening Renal Functions in Hypertensive Patients. *The Egyptian Journal of Hospital Medicine*, *89*(1), 5521–5527. https://doi.org/10.21608/ejhm.2022.264853

Jaaban, M., Zetoune, A. B., Hesenow, S., & Hessenow, R. (2021). Neutrophil-lymphocyte ratio and platelet-lymphocyte ratio as novel risk markers for diabetic nephropathy in patients with type 2 diabetes. *Heliyon*, *7*(7), e07564. https://doi.org/10.1016/j.heliyon.2021.e07564

Kim, J., Song, S. H., Oh, T. R., Suh, S. H., Choi, H. S., Kim, C. S., Ma, S. K., Kim, S. W., & Bae, E. H. (2023). Prognostic role of the neutrophil-to-lymphocyte ratio in patients with chronic kidney disease. *The Korean Journal of Internal Medicine*, *38*(5), 725–733. https://doi.org/10.3904/kjim.2023.171

Lan, D. T. N., Coradduzza, D., Van An, L., Paliogiannis, P., Chessa, C., Zinellu, A., Mangoni, A. A., & Carru, C. (2025). Role of Blood Cell Indexes in Progresses to ESRD. *Indian Journal of Clinical Biochemistry : IJCB*, *40*(2), 307–315. https://doi.org/10.1007/s12291-024-01184-1

Lauxen, J. S., Vondenhoff, S., Junho, C. V. C., Martin, P., Fleig, S., Schütt, K., Schulze-Späte, U., Soehnlein, O., Prates-Roma, L., Döring, Y., Baaten, C. C. F. M. J., & Noels, H. (2025). Neutrophil Function in Patients With Chronic Kidney Disease: A Systematic Review and Meta-Analysis. *Acta Physiologica (Oxford, England)*, *241*(6), e70057. https://doi.org/10.1111/apha.70057

Lee, K. P., Kim, Y. S., Yoon, S. A., Han, K., & Kim, Y. O. (2020). Association between Blood Pressure and Renal Progression in Korean Adults with Normal Renal Function. *Journal of Korean Medical Science*, *35*(34). https://doi.org/10.3346/jkms.2020.35.e312

Levey, A. S., Stevens, L. A., Schmid, C. H., Zhang, Y. (Lucy), Castro, A. F., Feldman, H. I., Kusek, J. W., Eggers, P., Van Lente, F., Greene, T., & Coresh, J. (2009). A New Equation to Estimate Glomerular Filtration Rate. *Annals of Internal Medicine*, *150*(9), 604–612. https://doi.org/10.7326/0003-4819-150-9-200905050-00006

Li, X., Wang, L., Liu, M., Zhou, H., & Xu, H. (2024). Association between neutrophil-to-lymphocyte ratio and diabetic kidney disease in type 2 diabetes mellitus patients: a cross-sectional study. *Frontiers in Endocrinology*, *14*. https://doi.org/10.3389/fendo.2023.1285509

Lin, C.-H., Li, Y.-H., Wang, Y.-Y., & Chang, W.-D. (2022). Higher Neutrophil-To-Lymphocyte Ratio Was Associated with Increased Risk of Chronic Kidney Disease in Overweight/Obese but Not Normal-

Weight Individuals. *International Journal of Environmental Research and Public Health*, *19*(13), 8077. https://doi.org/10.3390/ijerph19138077

Liu, Y., Wang, R., Yuan, J., & Zhao, J. (2025). The role of neutrophil-to-lymphocyte ratio in the prognosis of chronic kidney disease: insights from the NHANES cohort study. *Frontiers in Systems Biology*, *5*, 1656683. https://doi.org/10.3389/fsysb.2025.1656683

Lu, X., & Crowley, S. D. (2020). Actions of immune cells in the hypertensive kidney. *Current Opinion in Nephrology and Hypertension*, *29*(5), 515–522. https://doi.org/10.1097/MNH.0000000000000635

Moh, M. C., Low, S., Shao, Y.-M., Subramaniam, T., Sum, C. F., & Lim, S. C. (2022). HIGH NEUTROPHIL/LYMPHOCYTE RATIO PREDICTS RENAL FUNCTION DECLINE IN MULTI-ETHNIC ASIANS WITH TYPE 2 DIABETES – A LONGITUDINAL COHORT STUDY. *Journal of the ASEAN Federation of Endocrine Societies*, *37*(2), 7. https://doi.org/10.15605/jafes.037.AFES.07

Moh, M. C., Low, S., Shao, Y.-M., Subramaniam, T., Sum, C. F., & Lim, S. C. (2023). Association between neutrophil/lymphocyte ratio and kidney impairment in type 2 diabetes mellitus: A role of extracellular water/total body water ratio. *Diabetes Research and Clinical Practice*, *199*, 110634. https://doi.org/10.1016/j.diabres.2023.110634

Nakamura, M., Yamazaki, C., Hiromura, K., Goto, A., & Hamazaki, K. (2026). Analysis of the relationship between body mass index and kidney function decline in a middle-aged Japanese population: A population-based retrospective cohort study. *PloS One*, *21*(5), e0349621. https://doi.org/10.1371/journal.pone.0349621

Nguyen, B. A., Alexander, M. R., & Harrison, D. G. (2024). Immune mechanisms in the pathophysiology of hypertension. *Nature Reviews Nephrology*, *20*(8), 530–540. https://doi.org/10.1038/s41581-024-00838-w

Rashid, I., Tiwari, P., D'Cruz, S., & Jaswal, S. (2023). Prognostic importance of neutrophil-lymphocyte ratio in non-dialysis chronic kidney disease patients—a hospital-based prospective cohort. *Exploration of Medicine*, 299–313. https://doi.org/10.37349/emed.2023.00141

Schuster, N. A., Twisk, J. W. R., Ter Riet, G., Heymans, M. W., & Rijnhart, J. J. M. (2021). Noncollapsibility and its role in quantifying confounding bias in logistic regression. *BMC Medical Research Methodology*, *21*(1), 136. https://doi.org/10.1186/s12874-021-01316-8

Song, S., Chen, L., Yu, R., & Zhu, J. (2024). Neutrophil-to-lymphocyte ratio as a predictor of all-cause and cardiovascular mortality in coronary heart disease and hypertensive patients: a retrospective cohort study. *Frontiers in Endocrinology*, *15*. https://doi.org/10.3389/fendo.2024.1442165

Stevens, P. E., Ahmed, S. B., Carrero, J. J., Foster, B., Francis, A., Hall, R. K., Herrington, W. G., Hill, G., Inker, L. A., Kazancıoğlu, R., Lamb, E., Lin, P., Madero, M., McIntyre, N., Morrow, K., Roberts, G., Sabanayagam, D., Schaeffner, E., Shlipak, M., … Levin, A. (2024). KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. *Kidney International*, *105*(4), S117–S314. https://doi.org/10.1016/j.kint.2023.10.018

Tung, H.-T., Liu, C.-M., Huang, H.-S., Lu, Z., & Liu, C.-J. (2023). Increased risk of chronic kidney disease in uric acid stone formers with high neutrophil-to-lymphocyte ratio. *Scientific Reports*, *13*(1), 17686. https://doi.org/10.1038/s41598-023-45034-1

Wang, J.-H., Lin, Y.-L., & Hsu, B.-G. (2025). Endothelial dysfunction in chronic kidney disease: Mechanisms, biomarkers, diagnostics, and therapeutic strategies. *Tzu Chi Medical Journal*, *37*(2), 125–134. https://doi.org/10.4103/tcmj.tcmj_284_24

Wang, R., Lan, C., Benlagha, K., Camara, N. O. S., Miller, H., Kubo, M., Heegaard, S., Lee, P., Yang, L., Forsman, H., Li, X., Zhai, Z., & Liu, C. (2024). The interaction of innate immune and adaptive immune system. *MedComm*, *5*(10). https://doi.org/10.1002/mco2.714

Xie, L., Wang, B., Jiang, C., Zhang, X., Song, Y., Li, Y., Wang, J., Yu, Y., Liang, M., Wang, G., Zhang, Y., Li, J., Huo, Y., Xu, X., Nie, J., & Qin, X. (2018). BMI is associated with the development of chronic kidney diseases in hypertensive patients with normal renal function. *Journal of Hypertension*, *36*(10), 2085–2091. https://doi.org/10.1097/HJH.0000000000001817

Yang, W., Jiang, W., Zhu, Q., Wang, M., Mulalibieke, H., Qin, L., Wu, T., Guo, B., Hong, J., & Li, N. (2026). Phenotype-specific association of C-reactive protein-to-lymphocyte ratio with incident proteinuric CKD versus isolated eGFR decline: a real-world retrospective cohort study. *Frontiers in Endocrinology*, *17*. https://doi.org/10.3389/fendo.2026.1769059

Yu, Y., Lin, Z., Zhang, W., Jiang, L., Zhang, C., Ni, X., Yu, J., & Jiang, Y. (2026). The neutrophil-to-lymphocyte ratio and incident chronic kidney disease in a community-based cohort: a prospective study. *Scientific Reports*. https://doi.org/10.1038/s41598-026-58556-1

Zhang, X., Wei, R., Wang, X., Zhang, W., Li, M., Ni, T., Weng, W., & Li, Q. (2024). The neutrophil-to-lymphocyte ratio is associated with all-cause and cardiovascular mortality among individuals with hypertension. *Cardiovascular Diabetology*, *23*(1), 117. https://doi.org/10.1186/s12933-024-02191-5