Al-Azhar International Medical Journal

Volume 5 | Issue 6 Article 1

5-31-2024
Section: Internal Medicine

Relationship of Neutrophil to Lymphocyte Ratio (NLR), Platelet to Lymphocyte Ratio (PLR) and C3, C4 levels with Acute Kidney Injury

Mona Abdel Raouf Abdel kader
Internal Medicine, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt

Noha Mahmoud Hamdy
Clinical Pathology, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt

Marwa Mohammed Abdulrehim Omar
Internal Medicine, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt

Shaymaa Mamdouh Eisa
Internal Medicine, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt,
shimaa.m.eisa@gmail.com

How to Cite This Article

Abdel kader, Mona Abdel Raouf; Hamdy, Noha Mahmoud; Omar, Marwa Mohammed Abdulrehim; and Eisa, Shaymaa Mamdouh (2024) "Relationship of Neutrophil to Lymphocyte Ratio (NLR), Platelet to Lymphocyte Ratio (PLR) and C3, C4 levels with Acute Kidney Injury," *Al-Azhar International Medical Journal*: Vol. 5: Iss. 6, Article 1.
DOI: https://doi.org/10.58675/2682-339X.2461

ORIGINAL ARTICLE

Relationship of Neutrophil to Lymphocyte Ratio (NLR), Platelet to Lymphocyte Ratio (PLR) and C3, C4 levels with Acute Kidney Injury

Mona A. Abdel kader [a], Noha M. Hamdy [b], Marwa M. A. omar [a], Shaymaa M. Eisa [a,*]

[a] Department of Internal Medicine, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt
[b] Department of Clinical Pathology, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt

Abstract

Background: Patients in need of intensive care due to acute kidney injury (AKI), a sudden decline in renal function commonly caused by severe clinical illness such as sepsis, it is common practice to employ acute-phase markers for systemic inflammation in clinical practice.

Aim and objectives: In critically ill patients, the relationship between AKI and neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and C3 and C4 levels should be examined.

Subjects and methods: The study, a case-control design, involved 80 patients of both sexes divided into two groups and was conducted by researchers from Al-Azhar University's Faculty of Medicine for Women.

Results: Regarding platelets, there were no statistically significant differences among the groups. More TLC and neutrophils were seen in Groups 2 and 3 compared to Group I and the control group. Even though groups 2 and 3 had significantly more lymphocytes than the control and group I did, the differences were not statistically significant. The control group had the lowest creatinine levels, followed by groups two and I, and then group 3. Group 3 had the highest urea levels, followed by groups 2 and I, and finally, the control group.

Conclusion: Both NLR and PLR show potential as low-cost, clinically relevant markers of activity and severity in the critically ill with acute kidney injury. It is crucial to note that NLR is not the only risk factor for renal prognosis in individuals with AKI and that it has only a modest impact on some key renal functions.

Keywords: Acute Kidney Injury; Platelet-To-Lymphocyte Ratio; Neutrophil-To-Lymphocyte Ratio; Prognosis

1. Introduction

AKI is a primary cause of death and disability, especially for those who are already in a severe condition. Inadequate detection of early stages of renal injury by urine output and serum creatinine continues to be a barrier to the timely diagnosis of AKI. No single biomarker has been universally adopted as an early sign of tubular injury, and the identification of biomarkers for the early detection of AKI is still an ongoing process. [1]

Following a comparison of the patient's baseline and maximal creatinine levels within the first seven days of their index intensive care unit stay, AKI was determined to be present. Results from this comparison were based on the KDIGO Clinical Practice Guidelines for Acute Kidney Injury. [2]

The numbers of neutrophils to lymphocytes in a blood sample is known as the neutrophil-to-lymphocyte ratio. The complete blood count (CBC) is a diagnostic tool for a wide range of medical issues, including anemia, infections, inflammation, and more. Although NLR is not novel, it does not see much use as an inflammation indicator because to its low cost and ease of availability. [3]

NLR is less expensive than CRP, IL6, or ferritin since it is used more frequently for cancer prognosis and less frequently for other purposes. Again, it has been in the spotlight due to the deaths and heart problems it has been associated with. [4]

One of the best nephrology groups found that the NLR was the only ratio with a lymphocyte in the denominator that helped predict death better than demographics, comorbidities, and albumin level. [5]

Accepted 21 May 2024.
Available online 31 May 2024

* Corresponding author at: Internal Medicine, Faculty of Medicine for Girls, Al-Azhar University, Cairo, Egypt.
E-mail address: shimaa.m.eisa@gmail.com (S. M. Eisa).

https://doi.org/10.58675/2682-339X.2461

When sepsis manifests, there is a strong indication that the complement system has been activated. Numerous complement anaphylatoxins (C3a, C4a, and C5) are produced via three primary pathways: the classical, the lectin, and the alternative. [6]

The study was conducted to learn more about the link between acute kidney injury and NLR and PLR ratios, as well as complement components C3 and C4.

2. Patients and methods

For this case-control study, researchers at Al-Azhar University's Faculty of Medicine for Girls divided 80 patients of both sexes into two groups. The patient group consisted of 60 people with acute renal impairment, who were further separated into three subgroups based on the severity of their AKI. The control group consisted of 20 people of the same age and gender who appeared healthy.

The Faculty of Medicine for Girls at Al-Azhar University reviewed and approved the study's protocol.

2.1.Subjects were classified into:

Sixty cases with acute renal impairment who were admitted to the intensive care unit at Ahmed Maher Teaching Hospital were included in the study. They were all over the age of± 44 (range 24-64).

According to the severity of their acute renal damage, the patients were separated into three categories: Twenty individuals (8 men and 12 women) with stage 1 AKI were enrolled in the study. Nine men and eleven women, ages 24 to 64 (mean ±44), all in Stage 2 of AKI, were included in the study. Twenty patients (10 men and ten females) with Stage 3 AKI with an age range of 34 to 64 (mean 49 years) were included in the study. Their ages varied from 27 to 52 (with a mean of 39.5).

The control group included twenty age- and sex-matched healthy individuals (9 men and 11 women). Their age ranged from 34 to 64 (mean ± 49 years).

Inclusion Criteria: Patients aged> 18 and <65 years old; patients in conservative treatment and control were healthy individuals with serum creatinine < 1.2 mg/dL.

Exclusion Criteria: renal transplant before admission; dialysis before admission; stage 5 chronic kidney disease (CKD) before admission; liver cirrhosis; malignancy; and autoimmune disease.

2.2.Sample size

Using the Fleiss technique and the correction factor, we can determine the sample size needed to investigate the outcomes of the current study with a significance level of P < 0.05 and a power of study of 80%.

$$n = \frac{\left[z_\alpha \sqrt{(1+1/m)\bar{p}(1-\bar{p})} + z_\beta \sqrt{p_0(1-p_0)/m + p_1(1-p_1)}\right]^2}{(p_0-p_1)^2}$$

$$\bar{p} = \frac{p_1 + m p_0}{m+1}$$

$$n_c = \frac{n}{4}\left(1 + \sqrt{1 + \frac{2(m+1)}{nm|p_0-p_1|}}\right)^2$$

In this context, α represents alpha, β represents 1 minus power, n stands for the sample size, nc for the continuity corrected sample size, and zp for the standard normal deviation for probability p. Rounding up to the nearest integer is done for n.

2.3.Methods

The following were applied to both the patients and the control group: Review of medical history, physical examination, radiographs (pelvic and abdominal ultrasonography), and blood work (complete blood count), Serum creatinine, urea, sodium, and potassium levels; liver enzymes (serum glutamic oxaloacetic transaminase; serum glutamic pyruvic transaminase); C-reactive protein; serum ferritin; complement: C3 and C4); estimated glomerular filtration rate (GFR) using the CKD EPI 2021 formula; calculated normalized liver ratio (NRR) and projected plasma lipid profile (PLR).

2.4.Sample collection and preparation:

After obtaining informed consent, blood was drawn from each subject's peripheral vein using a single-use plastic syringe kept in a completely sterile environment.

The first tube contained 2 ml of anticoagulated blood with ethylenediaminetetraacetic acid (EDTA) for CBC. It used a fully automated cell counter (Sysmex XS 500, serial 13235).

After 4 milliliters of blood had clotted at room temperature for 30 minutes, the second tube was spun at 3000 revolutions per minute for 20 minutes. Serum sodium, potassium, creatinine, serum blood urea, and liver function tests (SGOT, SGPT) were all measured from the separated serum. We used a Dimension Panda Plus automated chemical analyzer (Serial No. 20040827) to conduct these experiments.

2.5.Statistical methods

IBM SPSS statistics (Statistical Package for the Social Sciences), version 28.0, IBM Corp., Chicago, USA, 2021, was used to code, tabulate, and analyze the data that was gathered. The Shapiro-Wilk statistic was used to determine whether the numerical data was normal, and the findings were shown as the mean SD with the lowest and maximum values provided. For cells with a low predicted count, the Chi-square or Fisher's exact test are used for data analysis based on numerical and percentage values. The relationships between the variables were examined using Pearson's correlation coefficient. Post-hoc analysis was conducted using the Bonferroni technique. A p-value of less than 0.050 was considered

statistically significant in this investigation.

3. Results

Table 1. Demographic characteristics among the studied groups

MEASURES	CONTROL (TOTAL=20)	GROUP 1 (TOTAL=20)	GROUP 2 (TOTAL=20)	GROUP 3 (TOTAL=20)	P-VALUE
AGE (YEARS)					
MEAN±SD	50.2±11.1	50.6±12.2	52.0±9.5	52.9±10.8	^0.859
RANGE	32.0–64.0	24.0–64.0	34.0–64.0	27.0–64.0	
SEX (N, %)					
MALE	9 (45.0%)	8 (40.0%)	9 (45.0%)	10 (50.0%)	#0.939
FEMALE	11 (55.0%)	12 (60.0%)	11 (55.0%)	10 (50.0%)	

^ANOVA test. #Chi square test.

The results showed no statistically significant differences among the studied groups concerning age and gender.

Table 2. Correlations of NLR, PLR, C3, and C4 in the control group

VARIABLES	MEASURES	NLR	PLR	C3	C4
HEMOGLOBIN	r	-0.013	-0.215	-0.185	-0.484
	p-value	0.957	0.363	0.434	0.031*
PLATELETS	r	-0.011	0.814	-0.106	0.289
	p-value	0.962	<0.001*	0.655	0.217
TLC	r	0.655	0.143	0.157	-0.058
	p-value	0.002*	0.549	0.508	0.809
NEUTROPHILS	r	0.854	0.313	0.121	-0.018
	p-value	<0.001*	0.179	0.613	0.941
LYMPHOCYTES	r	-0.747	-0.415	0.256	0.021
	p-value	<0.001*	0.069	0.276	0.929
SGPT	r	0.209	0.283	-0.096	0.189
	p-value	0.375	0.227	0.687	0.425
SGOPT	r	0.117	-0.164	0.265	-0.127
	p-value	0.622	0.491	0.258	0.594
CREATININE	r	0.075	0.243	0.492	0.130
	p-value	0.754	0.302	0.028	0.104
UREA	r	0.050	0.345	0.287	0.260
	p-value	0.835	0.137	0.219	0300
NA	r	-0.214	-0.372	0.105	0.243
	p-value	0.364	0.106	0.659	0.302
K	r	0.272	0.129	-0.070	-0.151
	p-value	0.246	0.588	0.770	0.524
EGFR	r	-0.021	-0.391	-0.423	-0.527
	p-value	0.929	0.088	0.063	0.017*
CRP	r	0.586	0.206	-0.023	-0.147
	p-value	0.007*	0.383	0.923	0.535
FERRITIN	r	0.782	0.500	0.017	-0.250
	p-value	<0.001*	0.025*	0.943	0.287
PLR	r	0.434			
	p-value	0.056			
C3	r	-0.119	-0.196		
	p-value	0.616	0.408		
C4	r	-0.051	0.213	0.143	
	p-value	0.831	0.368	0.548	

NLR had significant positive correlations with TLC, neutrophils, CRP, and ferritin, as well as significant negative correlations with Lymphocytes. PLR had significant positive correlations with platelets and ferritin. C3 had no significant correlation with other lab findings. C4 had significant negative correlations with hemoglobin and eGFR.

Table 3. Correlations of NLR, PLR, C3, and C4 in Group I

VARIABLES	MEASURES	NLR	PLR	C3	C4
HEMOGLOBIN	r	0.195	0.002	0.274	0.281
	p-value	0.410	0.994	0.242	0.229
PLATELETS	r	-0.004	0.802	0.279	0.488
	p-value	0.987	<0.001*	0.234	0.029
TLC	r	0.455	-0.072	-0.127	-0.233
	p-value	0.044*	0.762	0.593	0.324
NEUTROPHILS	r	0.648	-0.036	-0.315	-0.277
	p-value	0.002*	0.880	0.177	0.238
LYMPHOCYTES	r	-0.707	-0.622	0.338	-0.178
	p-value	<0.001*	0.003*	0.145	0.453
SGPT	r	-0.114	-0.102	-0.044	-0.390
	p-value	0.631	0.669	0.854	0.089
SGOPT	r	0.241	0.091	-0.405	-0.294
	p-value	0.307	0.702	0.076	0.208
CREATININE	r	-0.223	-0.369	0.043	-0.347
	p-value	0.344	0.109	0.857	0.134
UREA	r	-0.261	-0.414	0.418	0.090
	p-value	0.267	0.069	0.067	0.705
NA	r	-0.220	0.125	0.223	0.215
	p-value	0.351	0.601	0.344	0.364
K	r	-0.358	-0.019	0.489	-0.221
	p-value	0.121	0.938	0.029*	0.350
EGFR	r	0.289	0.352	-0.002	0.312
	p-value	0.216	0.127	0.995	0.181
CRP	r	0.161	-0.261	-0.238	-0.274
	p-value	0.496	0.267	0.313	0.242
FERRITIN	r	0.016	-0.427	0.138	-0.420
	p-value	0.947	0.061	0.563	0.065
PLR	r	0.507			
	p-value	0.022*			
C3	r	-0.478	-0.059		
	p-value	0.033*	0.804		
C4	r	-0.030	0.388	0.321	
	p-value	0.899	0.091	0.168	

NLR had significant positive relationships with TLC, neutrophils, and PLR, as well as significant negative correlations with lymphocytes and C3. PLR had significant positive correlations with platelets as well as significant negative correlations with Lymphocytes. C3 had a significant positive correlation with K. C4 had no significant correlation with lab findings.

Table 4: Correlations of NLR, PLR, C3, and C4 in Group 2

VARIABLES	MEASURES	NLR	PLR	C3	C4
HEMOGLOBIN	r	0.150	-0.152	-0.051	-0.128
	p-value	0.528	0.521	0.831	0.591
PLATELETS	r	-0.167	0.756	-0.127	0.083
	p-value	0.481	<0.001*	0.594	0.729
TLC	r	0.892	-0.001	0.021	0.367
	p-value	<0.001*	0.996	0.931	0.111
NEUTROPHILS	r	0.913	-0.012	-0.001	0.414
	p-value	<0.001*	0.960	0.997	0.069
LYMPHOCYTES	r	-0.544	-0.540	0.259	-0.030
	p-value	0.013*	0.014*	0.271	0.899
SGPT	r	0.159	0.011	0.001	-0.138
	p-value	0.502	0.963	0.998	0.562
SGOPT	r	0.158	-0.269	-0.159	0.074
	p-value	0.507	0.252	0.503	0.755
CREATININE	r	-0.105	-0.447	0.041	0.013
	p-value	0.661	0.048*	0.862	0.957
UREA	r	0.143	-0.273	-0.102	0.033
	p-value	0.546	0.244	0.668	0.889
NA	r	-0.005	-0.351	0.426	-0.582
	p-value	0.982	0.129	0.061	0.007*
K	r	0.148	-0.488	-0.009	0.254
	p-value	0.532	0.029*	0.971	0.280
EGFR	r	-0.150	0.291	0.020	-0.060
	p-value	0.527	0.213	0.935	0.800
CRP	r	0.626	-0.042	-0.028	0.177
	p-value	0.003*	0.859	0.907	0.455
FERRITIN	r	0.337	-0.144	-0.186	-0.037
	p-value	0.146	0.545	0.432	0.876
PLR	r	0.292			
	p-value	0.212			
C3	r	-0.120	-0.317		
	p-value	0.615	0.173		
C4	r	0.394	0.163	-0.394	
	p-value	0.086	0.493	0.086	

NLR had significant positive correlations with TLC, neutrophils, and CRP, as well as significant negative correlations with Lymphocytes. PLR had significant positive correlations with platelets as well as significant negative correlations with lymphocytes, creatinine, and K. C3 had no significant correlation with lab findings. C4 had a significant negative correlation with Na.

Table 5. Correlations of NLR, PLR, C3, and C4 in Group 3

VARIABLES	MEASURES	NLR	PLR	C3	C4
HEMOGLOBIN	r	-0.085	-0.275	-0.185	-0.166
	p-value	0.720	0.240	0.436	0.485
PLATELETS	r	-0.101	0.790	0.591	0.282
	p-value	0.673	<0.001*	0.006*	0.228
TLC	r	0.213	-0.081	0.207	0.006
	p-value	0.366	0.734	0.382	0.979
NEUTROPHILS	r	0.604	-0.287	0.100	0.119
	p-value	0.005*	0.219	0.676	0.617
LYMPHOCYTES	r	-0.320	-0.421	0.211	0.246
	p-value	0.168	0.065	0.371	0.296
SGPT	r	0.285	-0.032	-0.271	-0.078
	p-value	0.224	0.893	0.248	0.743
SGOPT	r	0.150	0.226	-0.042	-0.080
	p-value	0.529	0.338	0.862	0.739
CREATININE	r	0.116	-0.100	-0.141	-0.374
	p-value	0.628	0.676	0.554	0.104
UREA	r	-0.095	-0.088	0.239	0.372
	p-value	0.691	0.711	0.310	0.107
NA	r	0.421	0.053	-0.018	-0.230
	p-value	0.064	0.824	0.939	0.330
K	r	0.445	0.125	0.138	-0.056
	p-value	0.049*	0.599	0.562	0.816
EGFR	r	-0.162	0.291	-0.151	0.344
	p-value	0.496	0.213	0.526	0.137
CRP	r	0.227	-0.238	-0.017	-0.204
	p-value	0.336	0.312	0.942	0.389
FERRITIN	r	0.061	-0.278	-0.319	-0.433
	p-value	0.800	0.235	0.170	0.056
PLR	r	0.022			
	p-value	0.928			
C3	r	-0.001	0.253		
	p-value	0.997	0.145		
C4	r	-0.068	0.051	0.130	
	p-value	0.775	0.831	0.585	

NLR had significant positive correlations with neutrophils, and K. PLR had significant positive correlations with platelets. C3 had significant positive correlations with platelets. C4 had no significant correlation with lab findings.

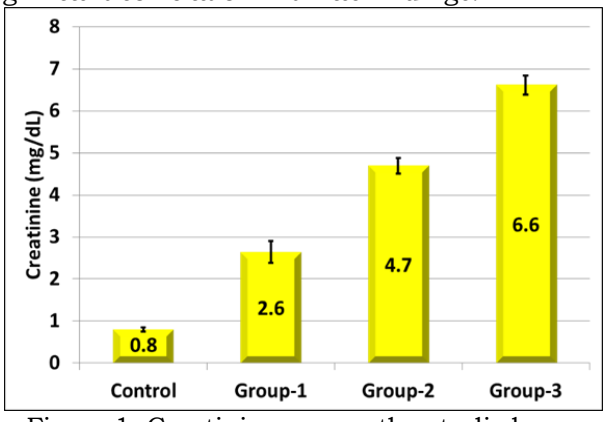

Figure 1. Creatinine among the studied groups

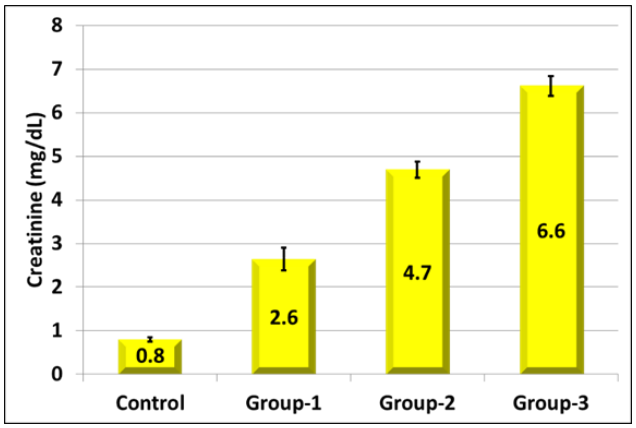

Figure 2. Urea among the studied groups

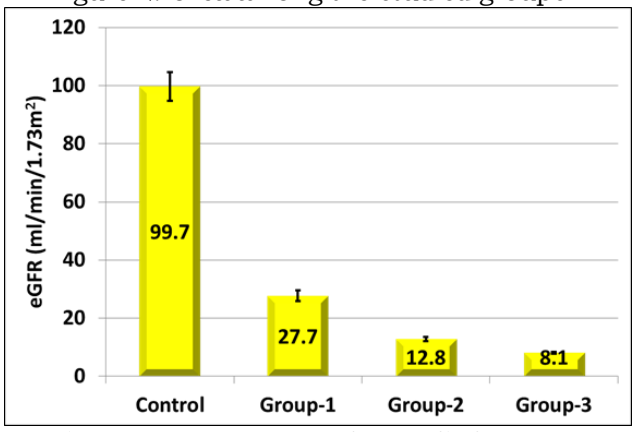

Figure 3. eGFR among the studied groups

4. Discussion

(AKI) occurs in between 16 and 67 percent of critically ill patients and has a mortality rate of more than 50 percent, depending on the criteria of AKI used. We know that sepsis and septic shock are major contributors to AKI. There is a strong correlation between septic AKI and negative outcomes such as chronic kidney disease and end-stage renal failure, and this condition is associated with high morbidity and mortality, prolonged hospital admissions, and increased healthcare expenses. [7]

Tang et al. split their subjects into four groups: Individuals with Systemic Lupus Erythematosus (SLE), Individuals with Lupus Nephritis (LN), Individuals with Healthy Controls (HC), and Individuals with Chronic Nephritis (CN). Additionally, they discovered no statistically significant changes in gender or age between groups 1, 2, and 3 of acute renal damage and the control group. These four groups are distinguished by age, gender, and other essential clinical characteristics. Among these four groups, there was no statistically significant difference in age. [8]

In our study, regarding correlations of NLR, PLR, C3, and C4 in the control group, we found that NLR had significant positive correlations with TLC, neutrophils, CRP, and ferritin, as well as significant negative correlations with Lymphocytes. PLR had significant positive correlations with platelets and ferritin.C3 had no significant correlations with other lab findings. C4 had significant negative correlations with hemoglobin and eGFR.

We found that Yu et al.'s findings were consistent with ours. Patients with acute kidney injury had elevated NLR and decreased hemoglobin compared to healthy controls. In contrast, cases with systemic lupus erythematosus had significantly elevated NLR ($P<0.001$) and a markedly decreased hemoglobin level ($P<0.001$). To learn more about how NLR and hemoglobin levels are connected in patients with acute renal damage, Patients' NLR levels were substantially higher than those of healthy female controls ($P<0.001$). This study had no statistically significant difference between male patients and healthy male controls. Both male and female cases with acute renal damage had significantly reduced hemoglobin levels. [9]

In the current study, our results showed that in group I, the NLR had significant positive correlations with TLC, Neutrophils, and PLR and significant negative correlations with Lymphocytes and C3. PLR had significant positive correlations with platelets and negative correlations with Lymphocytes.C3 had no significant correlations with lab findings other than NLR.C4 had no significant correlations with lab findings findingsndings corroborated those of Zou et al., who discovered a positive relationship between NLR and platelet count, neutrophil count, PLR, and serum C4, and a negative relationship between NLR and lymphocyte count. Serum C3 levels were not related to NLR in any significant manner. [10]

In the second group, we found that PLR, C3, and C4 correlations were While NLR was negatively correlated with lymphocytes, it was positively correlated with TLC, neutrophils, and CRP. Platelets and lymphocytes were positively correlated with PLR, while creatinine and lymphocytes were negatively correlated.Results from the lab did not show any significant relationships with C3.Na and C4 were significantly inversely related.

Our results corroborated those of Zou et al., which found a correlation between NLR and other markers in LN patients. There was a negative correlation between NLR and lymphocyte and eGFR counts, and a positive correlation with all other white blood cell, platelet, neutrophil, Scr, PLR, and serum C4 counts. [10]

Similarly, our results were in line with El-Said et al., who found that PLR had statically significant positive correl ations with lymphocytes and platelets but disagreed that PLR had significant positive correlations wi

th creatinine. [11]

Our results showed that in group 3, NLR had significant positive correlations with Neutrophils, PLR had significant positive correlations with platelets, C3 had significant positive correlations with platelets, and C4 had a significant negative correlation with Na.

Our results were in disparity with Umeres-Francia et al., who reported that NLR positively correlated with platelet and other laboratory findings, such as CRP, Neutrophil, and creatinine, with no significant correlations. PLR had a non-significant correlation with creatinine and a statistically significant positive correlation with nebetweenphil and hemoglobin. PLR had no significant correlation with another laboratory finding. [12]

4. Conclusion

NLR and PLR appear to be potentially effective and affordable indicators of activity and severity in AKI patients who are critically ill. NLR is not the primary risk factor for renal prognosis in individuals with AKI; nonetheless, it does have some influence on critical renal functions.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

Funding

No Funds : Yes

Conflicts of interest

There are no conflicts of interest.

References

1. Mansour SG, Zhang WR, Moledina DG, et al. The Association of Angiogenesis Markers With Acute Kidney Injury and Mortality After Cardiac Surgery. Am J Kidney Dis. 2019;74(1):36-46.
2. Margolis G, Gal-Oz A, Letourneau-Shesaf S, Khoury S, Keren G, Shacham Y. Acute kidney injury based on the KDIGO criteria among ST elevation myocardial infarction patients treated by primary percutaneous intervention. J Nephrol. 2018;31(3):423-428.
3. Li H, Lu X, Xiong R, Wang S. High Neutrophil-to-Lymphocyte Ratio Predicts Cardiovascular Mortality in Chronic Hemodialysis Patients. Mediators Inflamm. 2017;2017:9327136.
4. Zhao WM, Tao SM, Liu GL. Neutrophil-to-lymphocyte ratio in relation to the risk of all-cause mortality and cardiovascular events in patients with chronic kidney disease: a systematic review and meta-analysis. Ren Fail. 2020;42(1):1059-1066.
5. Catabay C, Obi Y, Streja E, et al. Lymphocyte Cell Ratios and Mortality among Incident Hemodialysis Patients. Am J Nephrol. 2017;46(5):408-416.
6. Fattahi F, Zetoune FS, Ward PA. Complement as a Major Inducer of Harmful Events in Infectious Sepsis. Shock. 2020;54(5):595-605.
7. Al-Husinat L, Alsabbah A, Hmaid AA, et al. Norepinephrine May Exacerbate Septic Acute Kidney Injury: A Narrative Review. J Clin Med. 2023;12(4):1373.
8. Tang D, Tang Q, Zhang L, Wang H. High Neutrophil-lymphocyte Ratio Predicts Serious Renal Insufficiency in Patients with Lupus Nephritis. Iran J Immunol. 2022;19(1):5.
9. Yu H, Jiang L, Yao L, et al. Predictive value of the neutrophil-to-lymphocyte ratio and hemoglobin insystemic lupus erythematosus. Exp Ther Med. 2018;16(2):1547-1553.
10. Zou G, Gao H. The relationship between neutrophil-lymphocyte ratio and early renal fibrosis and renal prognosis in patients with lupus nephritis. Am J Transl Res. 2021;13(3):1710-1716.
11. El-Said NY, El Adle S, Fathi HM. Clinical significance of platelet-lymphocyte ratio in systemic lupus erythematosus patients: Relation to disease activity and damage. The Egyptian Rheumatologist. 2022 Jun 1;44(3):225-9.
12. Umeres-Francia GE, Rojas-Fernández MV, Herrera-Añazco P, Benites-Zapata VA. Neutrophil-to-lymphocyte ratio and platelet-to-lymphocyte ratio as risk factors for mortality in Peruvian adults with chronic kidney disease. Renal Replacement Therapy. 2022 Jul 22;8(1):30.