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RESEARCH

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Platelet to lymphocyte ratio: can it be an early economical mortality predictor of AKI patients?

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Abstract

Background Acute kidney injury (AKI) affects over 13 million individuals annually worldwide, resulting in 1.7 million deaths. The potential long-term progression to chronic kidney disease (CKD) and renal failure, as well as the acute use of health care resources associated with acute kidney injury (AKI), impose enormous costs on society. The platelet-to-lymphocyte ratio (PLR) has emerged as a useful economical marker for detecting changes in platelet and lymphocyte counts owing to acute inflammatory and prothrombotic states. This study aimed to determine the PLR in patients with AKI and evaluate the in-hospital mortality.

Results The median PLR was compared between the non-survivor and survivor groups, and it was determined that the non-survivor group had a significantly higher PLR. ($p < 0.001$) For further subgroup analysis, the PLR was stratified into three groups: ≤ 100, 101–200, and > 200. Significantly more patients were demised in the PLR group 101–200 than in the PLR group ≤ 100, while all of the patients died in the PLR group greater than 200. The group with a PLR > 200 had a higher SOFA score > 10 ($p = 0.006$), a lower eGFR ($p = 0.001$), and higher platelet counts ($p = 0.001$), higher serum creatinine ($p = 0.001$), BUN ($p < 0.001$), and procalcitonin levels ($p = 0.007$). In multivariate Logistic regression analysis to predict the mortality outcome, PLR (OR 1.051; 95% CI, 1.016–1.087; $p = 0.004$) was identified as one of the significant indicators predicting AKI mortality. Other statistically significant indicators included SOFA scores (OR 2.789; 95% CI, 1.478–5.260; $p = 0.002$), procalcitonin levels (OR 0.898; 95% CI, 0.818–0.987; $p = 0.025$), and duration of hospital stay (OR 0.494; 95% CI, 0.276–0.886; $p = 0.017$). The ROC curve for the PLR yielded a value of 0.803 [95% CI, 0.720–0.886; $p < 0.001$] with the optimal cutoff value for the PLR to determine prognosis being 107.905, with a sensitivity of 82.5% and a specificity of 51.2%.

Conclusion PLR plays a significant role in the early prediction of prognosis (survival or death) for patients with AKI in ICU on a short-term basis.

Keywords PLR, AKI, ICU, SOFA score

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Background

Acute kidney injury (AKI) affects over 13 million individuals annually worldwide, resulting in 1.7 million deaths. Up to 20% of hospitalised patients and 30–60% of critically ill patients may be diagnosed. It causes organ dysfunction in intensive care units (ICUs) involving the liver, brain, and lungs more frequently. Even moderate AKI is associated with a 50% increased mortality risk. The potential long-term progression to chronic kidney disease (CKD) and renal failure, as well as the acute use of health care resources associated with acute kidney injury (AKI), impose enormous costs on society [1].

In patients with critical illness, systemic inflammation plays a significant role in disease progression and is frequently associated with sepsis, resulting in an increased mortality risk. Along with morphological and functional alterations in vascular endothelial cells and tubular epithelium, inflammation is a crucial factor in the initiation and progression of AKI in patients. Leukocytes, including lymphocytes, infiltrate the injured kidneys and the entire body via the circulatory system, inducing the production of inflammatory mediators including cytokines and chemokines, which damage multiple organs, including the kidneys. Platelet antithrombotic actions can lead to atherogenesis via the release of proinflammatory cytokines, whereas platelet attachment to endothelial cells can cause leukocyte transmigration and adhesion, especially under shear stress [2].

Inflammation-related measures that are predictive of the onset of AKI include the platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR), which are based on total blood counts [3]. The platelet-to-lymphocyte ratio (PLR) has emerged as a useful marker for detecting changes in platelet and lymphocyte counts owing to acute inflammatory and prothrombotic states. PLR shifts are useful for assessing the severity of systemic inflammation and predicting infections and other comorbidities, as demonstrated by a number of extensive observational studies [4].

A positive monotonic association between a high PLR and an unfavourable prognosis for diseases such as hypertension, hepatocellular carcinoma, and myocardial infarction has been reported. It is plausible to hypothesise that the PLR may influence the prognosis of AKI, contingent upon their findings. To date, however, very few epidemiological studies have investigated the prognostic impact of the PLR in AKI patients [2].

The main objective of this study was to determine the ratio of platelets to lymphocytes in patients with acute kidney injury and evaluate the patients' hospital outcomes and its correlation with other parameters, with the assumption that PLR may be significant in inflammatory states like in patients with AKI.

Methods

It was a prospective hospital-based observational study conducted at the Intensive Care Unit of the Department of Medicine at Silchar Medical College & Hospital from 1 June 2021 to 31 May 2022. The diagnosis of AKI was based on the KDIGO-AKI criteria. The primary outcome of the investigation was the in-hospital mortality rate of AKI patients. The PLR was computed for each individual patient and correlated with in-hospital mortality. A consecutive sampling method was used for the selection of study participants. The study was approved by the institution's ethical committee board after its thorough review. Informed consent was obtained from all participants, and the confidentiality of the data of all the patients has been maintained.

Out of 956 patients admitted to the ICU of the Department of Medicine, 100 consecutive patients met the inclusion and exclusion criteria and were included in the study during the specified time period. All patients aged 18 years or older and those who stayed for 48 h or longer were included in the study. Patients with a medical record of AKI before admission, those who underwent renal replacement therapy (RRT) on the day of or before their hospital admission, and those who did not achieve serum creatinine levels below 4.0 mg/dL during their stay at the end of day 7 were classified as having ESRD, thus ruling out false positive cases of chronic kidney disease rather than including as non-recovered AKI. The patients' eGFR was computed using the 2021 CKD-EPI creatinine equation.

Microsoft Excel 2013 and IBM SPSS 20.0 were utilized for statistical analysis. Continuous variables were expressed as mean (SD) or median (IQR), as appropriate for parametric and non-parametric data, respectively. Student's t-test and analysis of variance were used for parametric data, and Mann–Whitney U or Kruskal–Wallis test was used for non-parametric data, as appropriate. Categorical data were expressed as proportions and compared using the chi-square or Fisher exact test as appropriate. A p-value of < 0.05 was deemed statistically significant, and the appropriate tests for significance were applied based on the normal distribution of the patients. If the study population did not exhibit a normal distribution, non-parametric tests were performed. Multivariate logistic regression analysis was done for PLR, MV, eGFR, serum procalcitonin levels, SOFA score, Haemodialysis, and duration of hospital stay. The receiver operating characteristics curve was also plotted for PLR against other indicators such as NLR and BUN-creatinine ratio.

Results

In our investigation, the mean age was 50.57 (16.87) years. Sixty-five percent of the 100 patients were male, while the rest 35% were females. (male-to-female ratio = 1.85:1). Table 1 displays the other baseline characteristics of AKI patients. The proportions of patients in stages 1, 2, and 3 of AKI were 65%, 13%, and 22%, respectively. Pre-renal and intrinsic-renal insults were the leading causes of AKI, accounting for 45% and 55% of cases, respectively. Non-survivors tended to be younger and had a higher prevalence of diabetes, while the prevalence of hypertension and cardiac disease was comparable. The non-survivors required RRT and vasopressors at significantly higher rates than the survivors. In addition, they had high SOFA scores, reduced eGFR levels, higher serum bilirubin levels, and shorter hospital stays. Notably, neither the prevalence of hypertension, cardiovascular disease, or diabetes nor the WBC and platelet counts

Table 1 Baseline information of patients with AKI at day 1 of ICU admission

Characteristics	Total patients (*n* = 100)	Survivors (*n* = 43)	Non-survivors (*n* = 57)	*p*-value	Test
Age (years)	50.57 [16.87]	54.3 [17.149]	47.75 [16.248]	0.056	Unpaired *t* test
Gender (M/F)	65/35	30/13	35/22	0.384	Chi square test
PR (bpm)	98 [90.5–108.00]	94 [88–103]	102 [94–112]	**0.002**	Mann–Whitney *U* test
MAP (mmHg)	80 [74.15–89.175]	83.3 [80–88.35]	76.7 [70–86.7]	**0.023**	Mann–Whitney *U* test
SOFA score	8.29 [4.326]	4.81 [3.018]	10.91 [3.158]	**< 0.001**	Unpaired *t* test
MV (n/y)	87/13	40/3	47/10	0.144	Chi square test
VASOPRESSOR (N/Y)	84/16	40/3	44/13	**0.033**	Chi square test
CRRT (N/Y)	83/17	41/2	42/15	**0.004**	Chi square test
Hospital stay (days)	4 [2–7.3]	7 [4–8.5]	3 [1–5]	**< 0.001**	Mann–Whitney *U* test
eGFR (ml/min)	19 [11–29]	28 [18–35]	14 [10–22]	**< 0.001**	Mann–Whitney *U* test
Hb (g/dL)	9.49 [2.98]	9.48 [3.23]	9.50 [2.81]	0.971	Unpaired *t* test
Bil (mg/dl)	1.435 [0.6925–3.6950]	0.99 [0.65–1.53]	2.54 [0.91–5.12]	**0.001**	Mann–Whitney *U* test
UO (avg; ml/h)	35.42 [29.17–39.58]	37.5 [33.33–43.75]	29.17 [25–37.5]	**< 0.001**	Mann–Whitney *U* test
WBC (/cu.mm)	16590 [11487.5–23553.25]	15610 [10900–19715]	18700 [13020–25040]	0.168	Mann–Whitney *U* test
PLT (/cu.mm)	1.79 [1.20–2.60]	1.9 [1.15–2.35]	1.7 [1.2–2.9]	0.376	Mann–Whitney *U* test
ALC (/cu.mm)	1.355 [0.870–1.880]	1.68 [1.36–2.38]	1.02 [0.72–1.5]	**< 0.001**	Mann–Whitney *U* test
NLR	9.495 [5.035–16.675]	6.42 [4.31–9.83]	13.19 [8.7–18.76]	**< 0.001**	Mann–Whitney *U* test
PLR	138.62 [93.61–198.51]	107.38 [68.17–141.9]	176.47 [121.88–284.77]	**< 0.001**	Mann–Whitney *U* test
BUN (mg/dl)	45.72 [28.62–78.98]	34.74 [20.71–49.99]	65.09 [37.22–86.14]	**0.001**	Mann–Whitney *U* test
S.Cr (mg/dl)	2.59 [1.675–4.2625]	1.74 [1.57–2.64]	3.01 [2.05–5.38]	**< 0.001**	Mann–Whitney *U* test
PCT (ng/mL)	1.15 [0.30–10.375]	0.3 [0.2–2.85]	11.32 [18.00]	**< 0.001**	Mann–Whitney *U* test
ntProBNP (pg/mL)	2140 [655.50–8005.00]	1120 [585.5–5680.5]	3200 [1090–8670]	0.611	Mann–Whitney *U* test
Types of AKI (*n*%)				**0.003**	Fisher exact test
Pre-renal	45 (45.0%)	25 (58.14%)	20 (35.09%)		
Renal	50 (50.0%)	14 (32.56%)	36 (63.16%)		
Post-renal	5 (5.0%)	4 (9.3%)	1 (1.75%)		
Comorbidities (*n*%)					
HTN	10 (25.64%)	5 (29.41%)	5 (22.72%)	0.741	Fisher exact test
COPD	4 (10.26%)	4 (23.52%)	0 (0%)	**0.031**	Fisher exact test
DM	10 (25.64%)	2 (11.76%)	8 (36.36%)	0.181	Fisher exact test
IHD	8 (20.51%)	4 (23.52%)	4 (18.18%)	0.722	Fisher exact test
CLD	7 (17.95%)	2 (11.76%)	5 (22.72%)	0.695	Fisher exact test
AKI stage (KDIGO) (*n*%)				**0.006**	Chi square test
1	65 (65.0%)	34 (79.07%)	31 (54.39%)		
2	13 (13.0%)	6 (13.95%)	7 (12.28%)		
3	22 (22.0%)	3 (6.97%)	19 (33.33%)		

Numerals in bold denote statistical significance

PR pulse rate, *MV* mechanical ventilation, *MAP* mean arterial pressure, *SOFA* Sequential organ failure assessment, *RRT* renal replacement therapy, *NLR* neutrophil-to-lyphocyte ratio, *eGFR* estimated glomerular filtration rate, *WBC* white blood cell count, *PLT* platelet count, *ALC* absolute lymphocyte count, *BUN* blood urea nitrogen, *nt pro BNP* n-terminal pro brain natriuretic peptide

differed significantly between the two groups of patients. As in our study, the population was not normally distributed for PLR; the median PLR was compared between the non-survivor and survivor groups, and it was determined that the non-survivor group had a significantly higher PLR ($p < 0.001$).

For further subgroup analysis, the PLR was stratified into three groups: ≤ 100, 101–200, and > 200. The lower limit cut-off was chosen arbitrarily depending upon the studies which varied between 90 and 150 [2, 5–7]. Significantly more patients perished in the PLR group 101–200 than in the PLR group ≤ 100, with 23 patients dying in the PLR group > 200. The group with a PLR > 200 had higher SOFA scores > 10 ($p = 0.006$), a lower eGFR ($p < 0.001$), and greater platelet counts ($p = 0.001$), serum creatinine ($p = 0.001$), BUN ($p < 0.001$), and procalcitonin levels ($p = 0.007$). In the PLR group 101–200, pre-renal and intrinsic renal AKI predominated. The remaining characteristics are detailed in Table 2.

Multivariate logistic regression was performed to predict the mortality in AKI patients and PLR (OR 1.051; 95% CI, 1.016–1.087; $p = 0.004$) was identified as one of the indicators predicting AKI mortality. Other statistically significant indicators included SOFA scores (OR 2.789; 95% CI, 1.478–5.260; $p = 0.002$), procalcitonin levels (OR 0.898; 95% CI, 0.818–0.987; $p = 0.025$), and duration of hospital stay (OR 0.494; 95% CI, 0.276–0.886; $p = 0.017$). Details of which can be found in Table 3.

After performing logistic regression, we plotted the ROC curve for the PLR and obtained an area under the receiver operating characteristics curve (AUROC) value of 0.803 [95% CI, 0.720–0.886; $p < 0.001$], with the optimal cutoff value for the PLR to determine prognosis being 107.905, with a sensitivity of 82.5% and a specificity of 51.2%. The ROC curve is illustrated in Fig. 1.

Discussion

As an early predictor of mortality in AKI patients admitted to the ICU, our study revealed a correlation between High PLR and mortality. In a massive cohort of cancer patients, Proctor et al. discovered a correlation between the PLR and overall survival. Using a similar PLR criterion as our study, they demonstrated a positive correlation between PLR and mortality (PLR < 150, HR 1; PLR 150–300, HR 1.19; *P* 0.001; PLR > 300, HR 1.71; *P* 0.001) [5].

In contrast to our findings, Zheng, CF, et al. demonstrated a U-shaped correlation between the PLR and 30-day and 90-day mortality. Both low and high PLRs were associated with elevated mortality rates [2].

Shen Y et al. demonstrated that the OR for PLRs > 200 was statistically significant (OR 1.0002; 95% CI, 1.00001 to 1.0004) following adjustment for covariates such as the SOFA score with higher mortality [6]. In our study also, the association between PLR > 200 and mortality was statistically significant (OR = 1.051; 95% CI = 1.016–1.087; $p = 0.004$). But in contrast to their study, our study found a statistically significant association between PLR > 200 and higher SOFA score > 10($p = 0.006$).

Chen Y et al. showed the prognostic value of PLR for patients with septic AKI, along with the optimal cutoff value being 120, with a sensitivity of 70.7%, and a specificity being 65.4% [7]. Meanwhile in our study, we found a comparatively lower cut-off value of 107.905, along with a better sensitivity of 82.5% but lower specificity of 51.2%.

Yaprak et al. evaluated the correlation between the PLR and mortality in a small cohort of patients with end-stage kidney disease and demonstrated that the PLR could predict mortality from all causes in this population independently. This disparity is primarily due to the insufficient quantity of patients with low PLRs [8]. AKI and CKD contribute to local and systemic inflammation. In addition, numerous observational studies have reported elevated levels of inflammatory mediators including blood cells, endothelial cell components, platelets, lymphocytes, macrophages, mast cells, and fibroblasts, as well as negative outcomes for these conditions [9]. According to Yanfei Shen et al., PLR was associated with a higher risk of mortality in sepsis patients as noted in our study with higher SOFA scores with higher PLR [6].

Balta et al. demonstrated that in ESRD, the PLR predicts inflammation more accurately than the neutrophil-to-lymphocyte ratio. On the basis of the relationship between PLR-related inflammation and disease severity, we hypothesised that extremely elevated PLRs may predict the same adverse outcomes as other inflammatory biomarkers in AKI populations as well [10].

In addition, Kweon et al. examined median PLRs in a healthy Korean population and proposed that PLR cutoff values for illness assessment be individually determined based on age [11]. However, in our study, age was not statistically significant with different categories of PLR.

PLR is a strong predictive factor in pancreatic cancer patients, according to a previous Smith et al. In the current study, it was similarly proposed that PLR could be helpful for predicting the early progression to septic AKI [12].

However, AKI in the ICU is associated with a high mortality rate; it appears that other factors also contribute to poor outcomes. For instance, blood pressure, renal function, urine output, and additional clinical indicators may all influence the outcome of AKI.

Nevertheless, the strengths and limitations of the study were as follows: PLR can be useful for predicting the progression of AKI. The study found a significant association between higher PLR (> 200) and higher SOFA

Table 2 Baseline information of variables in patients across different groups of PLR with AKI on day 1 of ICU admission

	PLR ≤ 100	PLR 101–200	PLR > 200	*p*-value	Test
Outcome				**< 0.001**	
Survivors	21 (70%)	22 (46.8%)	0 (0%)		Chi square test
Non-survivors	9 (30%)	25 (53.12%)	23 (100%)		
Age (years)	54.20 (19.21)	47.98 (15.41)	51.13 (16.29)	0.286	ANOVA
Gender (M\F)	20\10	28\19	17\6	0.485	Chi square test
PR (bpm)	98 [92–105.5]	98 [91–111]	98 [90–107]	0.786	Kruskal–Wallis test
MAP (mmHg)	81.65 [76.7–89.18]	80 [70–85]	83.3 [75–90]	0.41	Kruskal–Wallis test
SOFA score	6.4 (3.94)	8.62 (4.75)	10.09 (2.84)	**0.006**	ANOVA
Hospital stay (days)	3.5 [2–5.75]	4 [2–6]	8 [7–10.5]	0.798	Kruskal–Wallis test
MV (n\y)	5\25	5\42	3\20	0.802	Fisher exact test
Vasopressor (N\Y)	26\4	38\9	20\3	0.776	Fisher exact test
RRT (N\Y)	29\1	38\9	16\7	**0.022**	Fisher exact test
Hemoglobin (g/dL)	9.97 (3.69)	9.38 (2.65)	9.10 (2.63)	0.543	ANOVA
S. bilirubin (mg/dl)	1.22 [0.72–2.23]	1.43 [0.7–3.23]	3.12 [0.72–5.72]	0.335	Kruskal–Wallis test
Urine output (avg; ml/kg/h)	37.5 [31.25–39.58]	35.42 [29.17–39.58]	29.17 [26.04–38.54]	0.129	Kruskal–Wallis test
eGFR (ml/min)	28 [20.25–35.5]	16 [10–27.5]	12 [8.5–21.5]	**< 0.001**	Kruskal–Wallis test
WBC (/cu.mm)	15745 [11690–24397.5]	17438 [11960–21530]	18700 [11575–26170]	0.765	Kruskal–Wallis test
PLT (/cu.mm)	1.55 [1–2.05]	1.78 [1.2–2.35]	2.9 [1.7–3.7]	**0.001**	Kruskal–Wallis test
ALC (/cu.mm)	2.12 [1.55–3.41]	1.19 [0.91–1.6]	0.8 [0.61–1.13]	**< 0.001**	Kruskal–Wallis test
NLR	5.06 [3.71–7.79]	10.73 [7.29–14.76]	17.27 [12.35–32.69]	**< 0.001**	Kruskal–Wallis test
BUN (mg/dl)	35.25 [20.96–45.1]	50.66 [28.82–80.28]	72.01 [46.28–93.31]	**< 0.001**	Kruskal–Wallis test
S.creatinine(mg/dl)	1.81 [1.62–2.42]	2.73 [1.7–4.2]	3.15 [2.63–6.46]	**0.001**	Kruskal–Wallis test
Procalcitonin (ng/ml)	0.35 [0.24–1.35]	3.5 [0.4–12.6]	3.1 [0.4–11.45]	**0.007**	Kruskal–Wallis test
ntProBNP (pg/mL)	1533 [670.25–6050]	2300 [657–9515]	3540 [887–7720]	0.552	Kruskal–Wallis test
Types of AKI				**0.004**	Fisher exact test
Pre-renal	21	18	6		
Renal	7	27	16		
Post-renal	2	2	1		
Comorbidities					
Hypertension	5	3	2	0.392	Fisher exact test
Chronic obstructive pulmonary disease	3	1	0	0.19	Fisher exact test
Diabetes mellitus	3	4	3	0.909	Fisher exact test
Ischaemic heart disease	4	3	1	0.503	Fisher exact test
Chronic liver disease	2	2	3	0.409	Fisher exact test
AKI stage (KDIGO)				**0.016**	Fisher exact test
1	26	29	10		
2	2	7	4		
3	2	11	9		

For normal distribution, mean (standard deviation) was used, while in non- normal distribution, median (interquartile range) was used to depict the individual variables of the patients

Numerals in bold denote statistical significance

PR pulse rate, *MV* mechanical ventilation, *MAP* mean arterial pressure, *SOFA* Sequential organ failure assessment, *RRT* renal replacement therapy, *NLR* neutrophil-to-lyphocyte ratio, *eGFR* estimated glomerular filtration rate, *WBC* white blood cell count, *PLT* platelet count, *ALC* absolute lymphocyte count, *BUN* blood urea nitrogen, *nt pro BNP* n-terminal pro brain natriuretic peptide

score (> 10), thus identifying morbid individuals at an early stage. It can be a useful maker to know the probable outcome of the patients as there was a significant correlation between PLR and mortality. The study yielded a lower cut-off value of PLR, increasing its sensitivity to determine beforehand the patients at risk. Since this was a single-center study, differing conclusions could be drawn if patient data from other institutions were included. Therefore, subject selection bias cannot be ignored, necessitating prospective multicenter research.

Table 3 Multivariate logistic regression analysis for PLR to predict AKI mortality in ICU patients

Variables	OR	95% C.I. for OR		*p*-value
		Lower	Upper	
PLR	1.051	1.016	1.087	**0.004**
SOFA score	2.789	1.478	5.260	**0.002**
PCT	0.898	0.818	0.987	**0.025**
HD	0.144	0.000	86.271	0.553
MV	11.942	0.389	366.503	0.156
Hospital stay	0.494	0.276	0.881	**0.017**
eGFR	1.062	0.944	1.194	0.319

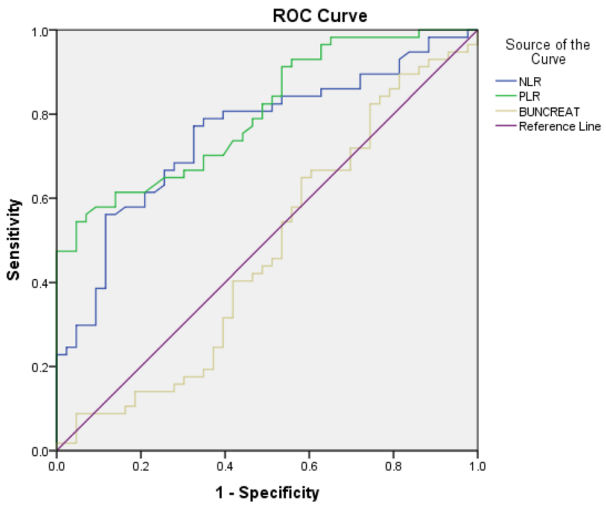
Numerals in bold denote statistical significance

PLR platelet to lymphocyte ratio, *SOFA* Sequential organ failure assessment, *PCT* procalcitonin, *MV* mechanical ventilation, *eGFR* estimated glomerular filtration rate

marker, researchers must validate its clinical utility. In statistical studies, the cutoff value must be determined in one patient cohort and evaluated in another, and the number of patients in each cohort must be taken into account. Due to a lack of pertinent data, we did not analyse the effect of sepsis and shock, both of which may worsen patient morbidity and predict more substantial mortality among patients with AKI, on the relationship between PLR and outcomes.

Conclusion

All of the aforementioned evidence demonstrates that the PLR plays a significant role in the early prediction of prognosis (survival or death) for patients with AKI in ICU on a short-term basis. Despite of the drawbacks, evidence suggests that PLR can provide valuable information to



Diagonal segments are produced by ties.

MARKER	BEST CUT OFF	SENSITIVITY(%)	SPECIFICITY(%)	AUC(95% CI)	p-value
PLR	107.905	82.5	51.2	0.803(0.720-0.886)	**<0.001**
NLR	7.73	80.7	60.5	0.753(0.658-0.848)	**<0.001**
BUNCREAT	18.2066	43.9	51.2	0.473(0.355-0.591)	0.648

Fig. 1 Illustration of ROC curve

Due to a lack of data on kidney function prior to 3 months before patient arrival, we were unable to investigate the prevalence of CKD among patients with AKI or determine the significance of CKD in relation to the PLR and mortality. Patients cannot be evaluated for PLR until they are admitted to the ICU. In addition, a single PLR measurement does not completely reflect inflammation, which is best evaluated by assessing additional inflammatory mediators simultaneously or subsequent repeat measurements. Preliminary findings suggest that the PLR could be a risk adjustment instrument with implications for AKI prognosis. To establish PLR as a predictive clinicians who encounter multisystem manifestations of Acute Kidney Injury, which are reflected by changes in platelet, lymphocyte, neutrophil, or monocyte counts. Interpretation of PLR in conjunction with complementary hematologic indices is recommended for more accurate prediction of related comorbidities and can be used as an early, potentially valuable, and cost-effective clinical marker.

Abbreviations

PLR Platelet to lymphocyte ratio
NLR Neutrophil to lymphocyte ratio

AKI	Acute kidney injury
CKD	Chronic kidney disease
EPI	Epidemiology collaboration
RRT	Renal replacement therapy
ESRD	End stage renal disease
ICU	Intensive care unit/s
S.Cr	Serum creatinine
BUN	Blood urea nitrogen
OR	Odds ratio
HR	Hazard ratio
SOFA	Sequential organ failure assessment
CI	Confidence interval
ROC curve	Receiver operating characteristics curve
eGFR	Estimated glomerular filtration rate
KDIGO	Kidney disease improving global outcomes

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s43162-023-00267-4.

Additional file 1.

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Authors' contributions
AP: Manuscript writing, Data Collection and Statistical Analysis; AK: Design, Manuscript writing and Final approval of the manuscript; ZF: Data collection and Manuscript proof-reading; BKN: Modification of Manuscript and manuscript writing; PB: Modification of Manuscript.

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Availability of data and materials
The data is included as a Supplemental file with the manuscript.

Declarations

Ethics approval and consent to participate
After a comprehensive review, the study was approved by the institution's ethical committee.
Consent to participate in the investigation was obtained from all participants.
The confidentiality of the data of all the patients has been maintained.

Competing interests
The authors declare that they have no competing interests.

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