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# Role of Platelets to Lymphocytes Ratio and Neutrophils to Lymphocytes Ratio in Detection of Acute Kidney Injury in Intensive Care Unit Patients

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**Abstract**

Intensive Care Unit (ICU)-acquired acute kidney injury (AKI) is a frequent consequence with many risk factors. When AKI develops, inflammatory mediators are a key factor. The ratios of neutrophils to lymphocytes (NLR) and platelets to lymphocytes (PLR) have been linked in many studies to the incidence of AKI. Our goal was to determine whether the PLR and the NLR might be used as a simple method for detecting acute kidney injury in ICU patients. 80 critically ill patients were included in our cross-sectional study, recruited from AL-Zahraa University Hospital. Their age ranged from 18 to 60 years for both sexes with in less than 6 hours from ICU admission with normal serum creatinine on admission. The following were done for the patients: a detailed medical history, complete examination, routine laboratory investigation including serum creatinine on admission then daily during ICU stay and estimation of GFR, using MDRD formula, complete blood count (CBC) daily follow up (to calculate PLR and NLR), fasting lipid profile, FBS, liver function tests, ESR, CRP, measurement of 24hr UOP, Na, K and ABG. On comparison of the AKI group to the non-AKI group, there was a highly significant rise in PLR and NLR at admission. However, NLR and PLR did not significantly rise 48 hours after admission. NLR was excellent (AUC= 0.940) in discriminating patients with AKI from patients without AKI at cut off value of >2.5 with sensitivity (83.6%), specificity (96%), PPV (97.9%), NPV (72.7%) and P-value (<0.001). PLR was good (AUC= 0.744) in discriminating patients with AKI from patients without AKI at cut off value of >134.8 with sensitivity (63.6%), specificity (96%), PPV (97.2%), NPV (54.5%) and P-value (<0.001). AKI in the ICU has been correlated with increased morbidity and mortality, so early detection is essential. NLR and PLR are both useful early indicators for the diagnosis of AKI.

**Keywords:** Platelets, lymphocytes, neutrophils, Acute Kidney Injury, Intensive care unit patients

## 1. Introduction

Acute kidney injury (AKI) is a common consequence among hospitalized patients following an intercurrent disease such as sepsis, hepatic, or major adverse cardiovascular diseases [1]. Acute kidney injury (AKI) is described by Kidney Disease Improving Global Outcome (KDIGO) as one of the following: a decrease in urine output of less than 0.5 mL/kg/h for six hours; 1.5 times baseline or more rise of serum creatinine within seven days; or 0.3 mg/dL or more rise within 48 hours [2].

The rate of AKI is increasing, now affecting 20–23% of all patients admitted to hospitals. It is well-established that AKI is linked to elevated rates of mortality and morbidity. Among patients with sepsis, AKI complicates about 5–7% of hospital admissions and can occur as many as 30% of those admitted to intensive care units (ICU) [3].

Numerous pathophysiologic events, such as shock, tubular damage or blockage, microvascular thrombi, endothelial dysfunction, and inflammation, are present in most of the multifactorial AKI cases [4]. Several investigations have demonstrated the critical role that immune-mediated inflammatory processes play in the pathogenesis of AKI. Early detection of inflammatory cells, including neutrophils and lymphocytes, in kidneys with AKI suggests that these cells have a crucial participation in renal pathology. So, some settings have explored the use of readily accessible data from a complete blood count (CBC) to provide an early diagnosis of AKI [5].

Lymphocytes affect the kidneys and other organ systems by producing inflammatory mediators such as cytokines and chemokines. While platelet adherence to the endothelium leads to migration and adhesion of leukocytes, platelets' anti-thrombotic actions can progress into atherogenesis through the inflammatory cytokines. The platelet-to-lymphocyte ratio (PLR) has emerged as a key indicator of inflammation in both cardiovascular disease (CVD) and tumors, which are recognized as diseases associated with inflammation. Researches have shown a positive relationship between elevated PLR and unfavorable outcomes in these conditions. [6].

The neutrophil-to-lymphocyte ratio (NLR) is a convenient biomarker indicative of systemic inflammation that can be easily computed using a complete blood count. The NLR is thought to represent the equilibrium between innate (neutrophils) and adaptive (lymphocytes) immunity. Past studies have demonstrated that increased levels correspond with higher amounts of several pro-inflammatory cytokines, which could lead to cellular DNA damage. Given that inflammation plays an essential function in the physiopathology of AKI, impacting the kidney and the body as a whole might serve as an inflammation label that can forecast the onset of AKI [7].

Measures associated with inflammation that can predict the development of AKI include the PLR and the NLR, both derived from CBC. PLR has been recognized as an effective indicator for identifying variations in platelet and lymphocyte levels due to acute inflammation. Changes in PLR are valuable in evaluating the intensity of inflammation and different associated health issues, as shown by numerous comprehensive observational studies [8]. This research seeks to estimate the significance of platelets to lymphocytes and neutrophils to lymphocytes ratios as a simple method for the detection of acute kidney injury in ICU patients.

## 2. Patients and Methods

This cross-sectional study was conducted on 80 patients, 41 (51.2%) were males and 39 (48.8%) were females, and their mean age was 52.8 ± 9.9 years. Patients were indiscriminately chosen from the ICU of Alzahraa Hospital (March 2024 - March 2025). Written and oral consent were taken from the subjects included, and approval of

the ethical panel of Al-Azhar University was acquired.

**2.1 Inclusion criteria:**

ICU Patients (18-60) years old of both sexes, were admitted (less than 6 hours) with normal serum creatinine level. After 48 hours patient were categorized into AKI and non-AKI groups based on KDIGO criteria 2012.

**2.2 Exclusion criteria:**

- Chronic kidney disease, e.g., diabetic nephropathy, glomerulonephritis, or any history of renal disease.
- End-stage renal disease (ESRD) or patients receiving haemodialysis.
- Pregnancy.
- Patients who had taken any antiplatelet agents during the last one week.
- Patients who have received a platelet transfusion within 24 hours.
- Patients with hematological malignancies and other terminal malignancies.
- Patients on drugs known to cause bone marrow suppression.

**2.3 Methods:**

**2.3.1 Our patients were submitted to :**

1. Complete medical history.
2. A physical examination which includes weight, height, BMI, and vital signs. Severity scoring systems, APACHE is an example of a mortality scoring system, such as acute physiology and chronic health evaluation II (APACHE II) and SOFA scores. APACHE II is counted within 24 hours of admission to the ICU. It ranged from 0-71, and Greater scores indicate more serious illness and a higher chance of death (9).
3. 24-hour measurement of urine output.
4. Routine laboratory investigation: Daily Serum creatinine and calculation of GFR by the MDRD formula. GFR (mL/min/1.73 $m^2$) $=175 \times (Scr)^{-1.154} \times (Age)^{0.203} \times$ (0.742 if female) × (1.212 if African American) [8]. Complete blood count (CBC) daily follow-up, including Total leukocytic count (TLC) and its differential count using an automated hematology analyzer. Lipid profile (normal fasting cholesterol up to 150 mg/dl and triglyceride up to 200 mg/dl). Fasting blood sugar (FBS) is normal to less than 100 mg/dl. Liver function tests: AST, ALT (normal less than 38 IU/L), serum albumin (3.5_5.5 g/dl), bilirubin (1.2 mg/dl), international normalized ratio (INR) normal 1 IU. Erythrocyte sedimentation rate (ESR) by western method, normal range in the 1$^{st}$ hour, 0-22 for males and 0-29 for females. C-reactive protein (CRP) normal level below 3 mg/dl, complete Urine analysis, arterial blood gases (ABG), serum Sodium and potassium.
5. Markers of our study: NLR and PLR.
6. Pelviabdominal ultrasound was done to prove normal kidney parameters.
7. ECG and Echocardiography.

8. Follow up patients as regards UOP/h, mortality, and requiring dialysis or mechanical ventilation.

### 2.3.2 Samples collection:

For measurement of complete blood count (CBC): (2ml) of blood was collected into EDITA containing tube for CBC.

### 2.4 Statistical Analysis

Version 25 of the Statistics Package for Social Sciences (SPSS) was used to analyse the data. Frequencies and percentages were used to express the qualitative data. The mean ± standard deviation (Mean ±SD) was used to express continuous quantitative data. P-value < 0.05 was deemed significant, P-value < 0.001 was deemed highly significant, and P-value > 0.05 was deemed insignificant.

## 3. Results

As show in table 1 there is no difference between the two groups as regard demographic data. As show in table 2 there is a significant decrease in mean arterial pressure (MAP), a significant increase in pulse, and a highly significant increase in APACHE II and SOFA scores in the AKI group, in comparison to those without AKI.As show in table 3 there is a highly significant decrease in lymphocytes, a significant decrease in eGFR, PH & HCO3, while a highly significant increase in PLR & NLR in patients with AKI, when compared with those of patients without AKI.As show in table 4 regarding CBC after 48 hours of admission, there was a significant decrease in lymphocytes and Hematocrit (HCT) in the AKI group in comparison to non-AKI. As regards renal function tests after 48 hours of admission, there was a highly significant increase in serum creatinine & urea, while there was a highly significant decrease in eGFR and UOP (after 24 hours and 48 hours) in the AKI group. As show in table 5 this is a highly significant positive correlation between NLR and both APACHE II score and SOFA score in the studied patients with AKI.As show in table 6 using roc curve, it shows that NLR was excellent (AUC= 0.940) in distinctive patients with AKI from patients without AKI at cut off value of >2.5 with sensitivity (83.6%), specificity (96%), PPV (97.9%), NPV (72.7%) and (P <0.001) and it shows that PLR was good (AUC= 0.744) in distinctive patients with AKI from patients without AKI at cut off value of >134.8 with sensitivity (63.6%), specificity (96%), PPV (97.2%), NPV (54.5%) and (P <0.001)

**Table 1:** Comparison between patients with and without AKI as regards demographic data

| | | AKI (n= 55) | | Non-AKI (n= 25) | | T | P-value |
|---|---|---|---|---|---|---|---|
| Sex | Males | 25 | 45.5% | 16 | 64.0% | $X^2$= 2.37 | 0.124 NS |
| | Females | 30 | 54.5% | 9 | 36.0% | | |
| Age (years) | Mean ± SD | 53.7 ± 8.8 | | 50.8 ± 11.8 | | 1.210 | 0.230 NS |
| | Min – Max | 26 – 60 | | 20 – 60 | | | |
| Height (cm) | Mean ± SD | 166.9 ± 4.6 | | 168.8 ± 3.5 | | -1.845 | 0.069 NS |
| | Min – Max | 158 – 180 | | 160 – 176 | | | |
| Weight (Kg) | Mean ± SD | 75.1 ± 8.3 | | 78.6 ± 9.9 | | -1.671 | 0.099 NS |
| | Min – Max | 60 – 90 | | 65 – 90 | | | |
| BMI ($kg/m^2$) | Mean ± SD | 27 ± 2.9 | | 27.6 ± 3.3 | | -0.836 | 0.405 NS |
| | Min – Max | 20.8 – 33.2 | | 22.5 – 33.1 | | | |

T: independent sample T test. X2: chi-square test.
There is no difference between the two groups as regard demographic data.

**Table 1**: Comparison between groups as regards vital data, APACHE II, and SOFA score

| | | AKI (n= 55) | Non-AKI (n= 25) | T | P-value |
|---|---|---|---|---|---|
| MAP | Mean ± SD | 74.6 ± 14.2 | 84.6 ± 17.5 | -2.702 | 0.008 S |
| | Min – Max | 60 – 116.6 | 60 – 110 | | |
| Pulse | Mean ± SD | 102.6 ± 15.1 | 94 ± 15.8 | 2.323 | 0.023 S |
| | Min – Max | 68 – 140 | 62 – 120 | | |
| RR | Mean ± SD | 27.3 ± 5.6 | 28.2 ± 4.3 | -0.74 | 0.464 NS |
| | Min – Max | 12 – 39 | 20 – 36 | | |
| Temp. | Mean ± SD | 37.6 ± 0.6 | 37.5 ± 0.6 | 0.265 | 0.792 NS |
| | Min – Max | 36.5 – 39.5 | 36 – 39 | | |
| GCS | Mean ± SD | 11.9 ± 3.2 | 12.7 ± 3.7 | -0.974 | 0.333 NS |
| | Min – Max | 3 – 15 | 3 – 15 | | |
| FiO2 | Mean ± SD | 34.2 ± 22.5 | 32.5 ± 22.3 | 0.311 | 0.757 NS |
| | Min – Max | 21 – 100 | 21 – 100 | | |
| APACHE II score | Mean ± SD | 18.1 ± 6.9 | 11.2 ± 6.2 | 4.313 | <0.001 HS |
| | Min – Max | 6 – 42 | 5 – 28 | | |
| SOFA Score | Mean ± SD | 7.5 ± 3.3 | 3.2 ± 2.2 | 6.083 | <0.001 HS |
| | Min – Max | 3 – 16 | 0 – 8 | | |
| MV | Yes<br>No | 28 %50.9<br>27 %49.1 | 8 %32.0<br>17 %68.0 | 2.48 | 0.115 NS |

**Table 3:** CBC and renal function tests in both groups on admission

| CBC | | AKI(n= 55) | Non-AKI(n= 25) | T | P-value |
|---|---|---|---|---|---|
| TLC | Mean ± SD | 7.9 ± 1.9 | 8.4 ± 2.4 | -0.967 | 0.336 NS |
| | Min – Max | 4.5 – 14 | 5.9 – 15.9 | | |
| Neutrophils | Mean ± SD | 5.7 ± 1.4 | 5.4 ± 1.8 | 0.962 | 0.339 NS |
| | Min – Max | 3 – 9.4 | 2.8 – 11.2 | | |
| Lymphocytes | Mean ± SD | 1.9 ± 0.5 | 2.7 ± 0.6 | -6.875 | <0.001 HS |
| | Min – Max | 0.9 – 3.2 | 2 – 4 | | |
| NLR | Mean ± SD | 3.3 ± 1.1 | 2 ± 0.3 | 5.742 | <0.001 HS |
| | Min – Max | 1.3 – 7.6 | 1.1 – 2.8 | | |
| Platelets | Mean ± SD | 268.8 ± 71.1 | 276.8 ± 64.3 | -0.479 | 0.634 NS |
| | Min – Max | 100 – 468 | 190 – 461 | | |
| PLR | Mean ± SD | 152.2 ± 52.8 | 105.7 ± 21.1 | 4.244 | <0.001 HS |
| | Min – Max | 62.5 – 272 | 73.1 – 179.6 | | |
| RBCS | Mean ± SD | 4.1 ± 0.7 | 4.2 ± 0.8 | -0.911 | 0.365 NS |
| | Min – Max | 2.7 – 5.8 | 2.5 – 6.1 | | |
| HB | Mean ± SD | 11.1 ± 1.8 | 11.5 ± 2.6 | -0.812 | 0.419 NS |
| | Min – Max | 7.7 – 16 | 7.5 – 18.9 | | |
| HCT | Mean ± SD | 32.8 ± 5 | 35.1 ± 7.9 | -1.564 | 0.122 NS |
| | Min – Max | 20 – 45 | 19.6 – 58.9 | | |
| MCV | Mean ± SD | 80.4 ± 5.3 | 81.6 ± 4.7 | -0.933 | 0.354 NS |
| | Min – Max | 57.2 – 90.8 | 74.3 – 96.8 | | |
| MCH | Mean ± SD | 26.6 ± 2 | 27.1 ± 2 | -0.885 | 0.379 NS |
| | Min – Max | 19 – 30 | 24 – 30.5 | | |
| MCHC | Mean ± SD | 31.9 ± 1.6 | 31.7 ± 2.1 | 0.541 | 0.590 NS |
| | Min – Max | 28.2 – 35.5 | 26 – 37.2 | | |
| S. Creatinine (mg/dl) | Mean ± SD | 0.8 ± 0.2 | 0.7 ± 0.2 | 1.686 | 0.096 NS |
| | Min – Max | 0.5 – 1.1 | 0.5 – 0.9 | | |
| S. Urea (mg/dl) | Mean ± SD | 32.9 ± 8.5 | 29 ± 6.1 | 2.093 | 0.04 S |
| | Min – Max | 18 – 60 | 18 – 39 | | |
| eGFR | Mean ± SD | 93.6 ± 29.3 | 110.6 ± 34.8 | -2.27 | 0.026 S |
| | Min – Max | 60.4 – 172 | 63.9 – 195.2 | | |
| PH | Mean ± SD | 7.32 ± 0.11 | 7.38 ± 0.1 | -2.289 | 0.025 S |
| | Min – Max | 7.09 – 7.54 | 7.02 – 7.6 | | |
| HCO3 | Mean ± SD | 16.9 ± 6.6 | 21.2 ± 6.9 | -2.673 | 0.009 S |
| | Min – Max | 6.6 – 40.5 | 8.3 – 42.2 | | |
| CO2 | Mean ± SD | 32.6 ± 11.5 | 34 ± 11.6 | -0.493 | 0.623 NS |
| | Min – Max | 14 – 82 | 17.4 – 80 | | |
| PaO2 | Mean ± SD | 71.5 ± 10.5 | 72.2 ± 10.7 | 1.686 | 0.096 NS |
| | Min – Max | 54 – 99 | 56 – 94 | | |

**Table 4:** CBC, renal function, and UOP in both groups after 48 hours from admission

| CBC (2) | | AKI(n= 55) | Non-AKI(n= 25) | T | P-value |
|---|---|---|---|---|---|
| TLC | Mean ± SD | 16.5 ± 5.7 | 17.7 ± 4.9 | -0.912 | 0.364 NS |
| | Min – Max | 4.7 – 34.1 | 7.5 – 29.3 | | |
| Neutrophils | Mean ± SD | 14.1 ± 5.4 | 14.4 ± 4.6 | -0.249 | 0.804 NS |
| | Min – Max | 3.7 – 31.6 | 5.5 – 26 | | |
| Lymphocytes | Mean ± SD | 1.3 ± 0.6 | 1.7 ± 0.8 | -2.685 | 0.009 S |
| | Min – Max | 0.2 – 3.1 | 0.6 – 4.7 | | |
| NLR | Mean ± SD | 13.7 ± 11.2 | 9.6 ± 4.3 | 1.741 | 0.086 NS |
| | Min – Max | 3.6 – 82 | 3.2 – 20.5 | | |
| Platelets | Mean ± SD | 250.9 ± 97.1 | 238.5 ± 94.4 | 0.534 | 0.595 NS |
| | Min – Max | 50 – 518 | 110 – 467 | | |
| PLR | Mean ± SD | 255.2 ± 291.5 | 152.4 ± 65.6 | 1.738 | 0.086 NS |
| | Min – Max | 36.4 – 2260 | 92.3 – 382.8 | | |
| RBCS | Mean ± SD | 4 ± 1.4 | 4.3 ± 0.9 | -0.818 | 0.416 NS |
| | Min – Max | 2.4 – 12.5 | 2.3 – 6.3 | | |
| HB | Mean ± SD | 10.4 ± 2.4 | 11.6 ± 2.7 | -1.951 | 0.055 NS |
| | Min – Max | 4.4 – 17.7 | 7.1 – 18.3 | | |
| HCT | Mean ± SD | 31.9 ± 6.8 | 36 ± 8.5 | -2.329 | 0.022 S |
| | Min – Max | 16 – 51.9 | 19.2 – 61.4 | | |
| MCV | Mean ± SD | 81.1 ± 6 | 81.9 ± 5 | -0.569 | 0.571 NS |
| | Min – Max | 60.5 – 90.9 | 74 – 98.2 | | |
| MCH | Mean ± SD | 26.7 ± 2.6 | 27 ± 1.6 | -0.573 | 0.568 NS |
| | Min – Max | 16.3 – 30 | 23.6 – 29.9 | | |
| MCHC | Mean ± SD | 31.9 ± 2 | 31.6 ± 2.1 | 0.781 | 0.437 NS |
| | Min – Max | 28.2 – 40.1 | 26.3 – 36.3 | | |
| S. Creatinine (mg/dl) | Mean ± SD | 4.2 ± 3.5 | 0.8 ± 0.2 | 4.829 | <0.001 HS |
| | Min – Max | 1.1 – 19 | 0.5 – 1 | | |
| S. Urea (mg/dl) | Mean ± SD | 169.6 ± 92.7 | 46 ± 10.1 | 6.630 | <0.001 HS |
| | Min – Max | 54 – 485 | 33 – 75 | | |
| eGFR | Mean ± SD | 21.9 ± 14 | 106.4 ± 34 | -15.769 | <0.001 HS |
| | Min – Max | 2.1 – 50.7 | 63.9 – 195.2 | | |
| UOP1 | Mean ± SD | 1271 ± 178 | 1696 ± 195 | -9.61 | <0.001 HS |
| | Min – Max | 1000 – 1800 | 1300 – 2000 | | |
| UOP2 | Mean ± SD | 548 ± 226 | 1624 ± 244 | -19.3 | <0.001 HS |
| | Min – Max | 200 – 1000 | 1000 – 2000 | | |

**Table 5:** Correlation between NLR and PLR and other parameters on admission in patients with AKI.

| Patients with AKI (n=55) | | | | | |
|---|---|---|---|---|---|
| | NLR | | | PLR | |
| | r | P-value | | r | P-value |
| APACHE II score | 0.620 | <0.001 | APACHE II score | 0.164 | 0.231 |
| SOFA score | 0.494 | <0.001 | SOFA score | 0.087 | 0.528 |
| Creatinine | -0.193 | 0.159 | Creatinine | -0.086 | 0.534 |
| eGFR | 0.159 | 0.245 | eGFR | 0.169 | 0.217 |
| UOP | -0.115 | 0.403 | UOP | -0.159 | 0.245 |

(r): Pearson correlation coefficient.

**Table 6:** Diagnostic performance of NLR and PLR on admission for distinctive patients with AKI from those without AKI.

| | Cut off | AUC | Sensitivity | Specificity | PPV | NPV | p-value |
|---|---|---|---|---|---|---|---|
| NLR | >2.5 | 0.940 | 83.6% | 96% | 97.9% | 72.7% | <0.001 |
| PLR | >134.8 | 0.744 | 63.6% | 96% | 97.2% | 54.5% | <0.001 |

PPV: positive predictive value. AUC: Area under curve

NPV: negative predictive value.

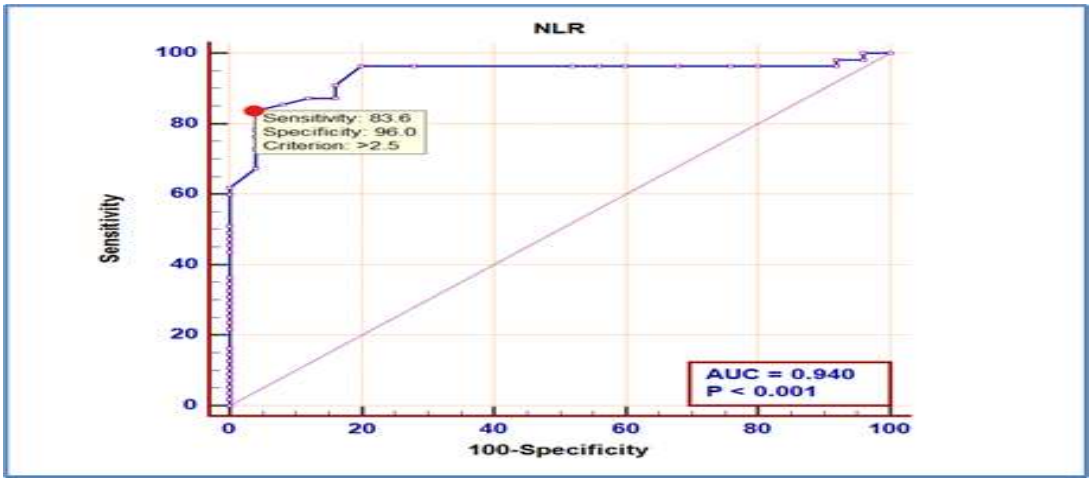


**Fig. 1:** Roc curve of NLR for distinctive AKI patients from those without AKI.

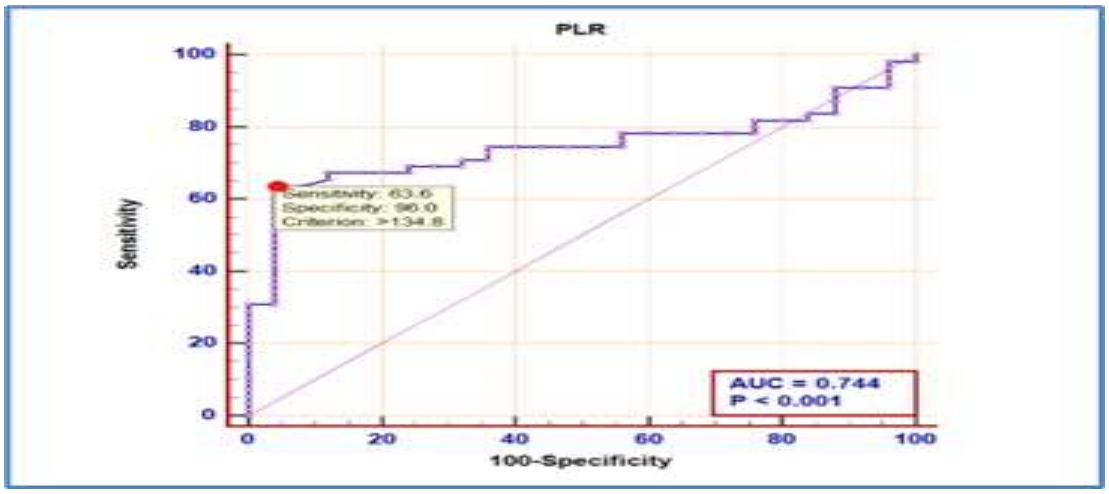


**Fig. 2:** Roc curve of PLR for distinctive AKI patients from those without AKI.

## 4. Discussion

ICU-acquired AKI is a usual condition with numerous risk factors. Inflammatory mediators have a considerable role in the development of AKI. Several studies suggested a link between the PLR and the NLR and the occurrence of AKI [4]. This research aimed to assess the importance of the platelets to lymphocytes ratio and the neutrophils to lymphocytes ratio as a simple method for the detection of AKI in ICU patients. Our prospective research included 80 patients who were indiscriminately chosen from different ICUs of AL-Zahraa Hospital from March 2024 to March 2025.

Our results showed that 41 (51.2%) of the studied patients were males and 39 (48.8%) were females. The mean age was (52.8 ± 9.9) years and ranged from 20 years to 60 years, the mean weight was (76.2 ± 8.9) kg, the mean height was (167.5 ± 4.3) cm, and the mean BMI was (27.2 ± 3). As regards comorbidities, 16 patients (20%) were diagnosed with DM, 22 patients (27.5%) with HTN, 15 patients (18.8%) with IHD, 5 patients (6.3%) with HF, and 5 patients (6.3%) with COPD.

Those patients were admitted to the ICU (within 6 hours) with normal creatinine level. 48 hours later, patients were classified based on KIDGO guidelines [11] into the AKI group, which included 55

patients (68.75%), and the non-AKI group, which included 25 patients (31.25%). As regards causes of AKI, sepsis was the major factor in 22 patients (40%), followed by hypoperfusion in 17 patients (30.9%), then cardiorenal syndrome in 9 patients (16.4%), and lastly, hepatorenal syndrome in 7 patients (12.7%).

Our findings revealed that MAP was significantly decreased, and Pulse was significantly increased in patients with AKI compared to patients without AKI. Decreased MAP may be related to the cause, either hypovolemic, septic, or cardiogenic shock, or as a result of AKI. AKI can cause fluid overload, electrolyte imbalances, or uremic cardiomyopathy, which worsens MAP. Inflammatory responses (e.g., in sepsis) may induce vasodilatation and hypotension, leading to renal hypoperfusion. Also effect of medications as nephrotoxic drugs, can impair renal autoregulation. Other drugs like diuretics, antihypertensives, or vasodilators cause further lowering of MAP [12].

Also, AKI results from volume depletion (e.g., diarrhea, hemorrhage, sepsis), the body compensates with tachycardia to maintain cardiac output when stroke volume is reduced. In sepsis-associated AKI, systemic inflammation directly stimulates tachycardia. Electrolyte imbalances such as hyperkalemia (common in AKI), can cause arrhythmias or reflex tachycardia, and acidosis (from AKI) stimulates chemoreceptors, increasing heart rate and respiratory rate. Pain or distress due to obstructive nephrolithiasis induces AKI, increasing catecholamine release and HR [13].

In consensus with our findings, Okba et al. (14) revealed that most of AKI patients in their study were shocked (72.5%). Also, Khanna et al. [15] reported that decreasing MAP was strongly associated with myocardial and renal injury in postoperative ICU patients. Vasopressors, significant fluid changes, multiple simultaneous insults, and preexisting and surgical organ system impairment are all possible outcomes for post-operative intensive care unit patients. Some were also feverish, which increases the need for perfusion and metabolic rate even more. Additionally, they imply that the kidneys continue to be extremely vulnerable to the effects of hypotension, most likely at thresholds higher than those of the heart.

We found that patients with AKI had significantly higher APACHE II and SOFA scores compared to those without AKI. Additionally, predicted mortality rates based on both scoring systems were significantly elevated in the AKI group. Stratification by SOFA score mortality risk further showed that a notable proportion of AKI patients fell into higher risk categories (≥50%), while all non-AKI patients remained in the low-risk category (<33%). These findings suggested that the AKI was strongly related to increased severity and higher mortality risk in ICU patients.

As regards initial CBC, we found that lymphocytes were significantly decreased, PLR&NLR were significantly increased in AKI patients and a highly significant association between NLR and both APACHE II score and SOFA score in patients with AKI. However, there was no significant difference between the two studied groups in the same parameter after 48 hours.

In agreement with our findings, Bu et al. [16] found that the NLR was predictive of AKI development in sepsis. Also, they revealed that the first NLR done at ICU admission was linked to the development of AKI in patients with sepsis and septic shock also, APACHE II and SOFA scores were significantly higher in the AKI patients in comparison to those without AKI ($P < 0.05$).

Wagner et al. [17] were in concurrency to our research as they discovered that during hospitalisation, lymphopenic patients are more prone to acquire an AKI (68% with ALC < 1000 cells/mL vs. 33% with ALC >1000 cells/mL); P = .01. Zheng et al. [18] reported higher PLR among AKI patients

in ICU (28.5 ± 2.56). also, Okba et al. (14) showed that the PLR ratio among AKI patients in the ICU was 15.6 ± 6.4 and increased significantly at different time points, reaching a peak at discharge.

Also, Xie et al. [19] who included 1238 septic patients in their research, concluded that a higher NLR and lower prognostic nutritional index (PNI) on admission were significantly linked with an increased risk of AKI.

Our study demonstrated decreased hematocrit (HCT) in AKI patients. It may be due to: Hemodilution (fluid overload), which is common in sepsis, heart failure, or post-resuscitation AKI. Also, AKI leads to impaired fluid excretion, and hemodilution occurs, leading to artificially low Hct. As the kidneys are responsible for Erythropoietin (EPO) production so AKI disrupts this, causing acute anemia. studies show EPO drops within 24-48 hours of AKI onset. Blood loss (uremia-induced platelet dysfunction), inflammation, and cytokine storm (e.g., in septic-AKI) suppress RBC production [20, 21].

Sukmark et al. [22] have found that HCT<30% was independently associated with an increased risk of worsening renal function in ICU patients, with a QR of 1.81 (95% CI: 1.50-2.19, P<0.001).

Su et al. [23] showed that in patients with acute myocardial infarction (AMI), a lower haematocrit was an independent risk factor for AKI.

Regarding renal function tests after 48 hours, our results showed a highly significant deterioration in patients with acute kidney injury compared to those without AKI. Patients had markedly elevated serum creatinine and urea levels and a significantly reduced eGFR, confirming substantial renal impairment. Also, our results showed that there was a highly significant decrease in UOP in patients with AKI compared to patients without AKI after 24 and 48 hours from admission to the ICU.

Our findings demonstrated that NLR was excellent (AUC= 0.940) in discriminating patients with AKI from patients without AKI at cut off value of >2.5 with sensitivity (83.6%), specificity (96%), PPV (97.9%), NPV (72.7%). PLR was good (AUC= 0.744) in discriminating patients with AKI from patients without AKI at cut off value of >134.8 with sensitivity (63.6%), specificity (96%), PPV (97.2%), and NPV (54.5%). Both NLR and PLR were statistically significant and clinically relevant inflammatory markers for distinguishing AKI among ICU patients.

In harmony with our findings, Abu Alfeilat et al. [7] stated that AKI patients can be identified with a 78% sensitivity and 65% specificity with a threshold value of 5.5. NLR is also a useful biomarker to rule out the AKI progression within the first hour of presentation as it has extremely strong negative predictive value at this cutoff.

Also, Kurtul et al. [24] who demonstrated that NLR was a significant predictor for AKI where NLR was above 3.46 and had a 73% specificity and 70% sensitivity for developing AKI among cardiac patients. Also, Yilmaz et al. [25] using a cut-off point of 10.15 for NLR, reported a sensitivity of 90.2% and a specificity 92.9% for predicting AKI in sepsis patients.

A meta-analysis by Chen et al. [26] who assessed NLR's diagnostic utility in adult patients with AKI. A mean sensitivity of 0.736 (95% CI 0.675–0.790) and specificity of 0.686 (95% CI 0.601–0.759) were obtained by bivariate analysis. According to their findings, the NLR is a trustworthy investigation for AKI prediction

PLR significantly contributed to the early prognostic prediction for short-term ICU patients with AKI, as Purkayastha et al. [8] showed. With the ideal cutoff, the PLR's ROC curve produced a value of 0.803 [95% CI,0.720–0.886; p<0.001]. value of 107.905 with a sensitivity of 82.5% and a specificity of 51.2% for the PLR used to evaluate prognosis.

Variations in inclusion and exclusion criteria account for the little variation in

NLR cutoffs amongst studies. For instance, if only septic patients were involved, it led to lower lymphocyte and greater neutrophil counts. Given that NLR values vary by race, demographic differences may also be a contributing cause to the heterogeneity in NLR across studies [27].

## 5. Conclusion

Both NLR and PLR were elevated in patients with AKI and serve as valuable inflammatory markers for its early detection. NLR showed excellent diagnostic performance with high sensitivity, specificity, and predictive values, making it a reliable tool for identifying AKI among critically ill patients.

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