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Neutrophil-Lymphocyte Ratio as an Early Predictor of Mortality in Patients with Acute Kidney Injury: A Hospital Based Study

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I. Introduction:

Acute kidney damage (AKI) is associated with a substantial mortality risk in critically ill patients. In developed nations, the in-hospital death rate for AKI is approximately 20%, and up to 50% for intensive care unit (ICU) patients. The only definitive treatment for the consequences of AKI is renal replacement therapy (RRT), which is expensive and not commonly available in resource-poor areas. [1]

Endothelial dysfunction, hemodynamic changes, tubular damage, and intrarenal inflammation characterise the pathogenesis of AKI. Inflammation is a crucial component of the pathogenesis of AKI. Recent investigations have attributed promising prognostic and predictive significance to different possible inflammatory biomarkers in AKI. [1]

The neutrophil-lymphocyte ratio (NLR) is an easily measurable potential inflammatory biomarker associated with an increased risk of acute kidney injury (AKI) and predictive of poor outcome in a variety of diseases, including cardiovascular disease, solid tumours, liver failure, and postoperative infection.[1] Consequently, we predicted that NLR might influence the prognosis of AKI.

To our knowledge, there have been very few studies regarding the relationship between mortality and NLR in AKI patients. We therefore undertook the study to check whether the NLR is predictive of AKI mortality on an early basis.

This study aimed to establish whether the neutrophil-to-lymphocyte ratio (NLR) evaluated at the time of intensive unit (ICU) admission is associated with in-hospital mortality.

II. Materials And Methods:

In a prospective observational study**,** 100 consecutive patients admitted in ICU at Dept of Medicine, Silchar Medical College & Hospital, with AKI were selected after fulfilling the inclusion and exclusion criteria for 1 year from June 2021 to May 2022. AKI was diagnosed according to the KDIGO-AKI criteria. The primary outcome of the study was in-hospital mortality of patients with AKI. The NLR was calculated for every patient and was correlated with in-hospital mortality.Statistical analysis and all the statistical graphs were prepared using Microsoft Excel and Statistical Package for Social Sciences (SPSS for Windows, version 20.0 Chicago, SPSS inc.). The Institutional ethics Committee approved the study after though review and written informed consent was taken from all the patients.

Patients with age ≥18 years of either sex; either increase in serum Creatinine by 0.3 mg/dL within 48 hours in ICU, increase in serum creatinine to 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days or urine volume <0.5 ml/kg/hr for atleast 6 hours were included in the study. Patients with known renal disease (CKD), established diabetic or hypertensive nephropathy, Bilateral shrunken kidneys, Polycystic Kidney disease, with a medical record of AKI before ICU admission (ICU-ad), renal replacement therapy (RRT) on the day of or before their hospital admission, serum creatinine (sCr) levels not achieving lesser than 4.0 mg/dL during their ICU stay were categorized as having ESRD, kidney transplant or undergoing and patients who did not give consent for the study were excluded.

III. Results:

Out of 100 AKI patients studied, 65% were males and 35% were females. The mean age of the study was 50.57±16.827 years. The most common age group affected was 41-60 years. The admitted patients had co-morbidities like Hypertension (10%), Diabetes (9%), Chronic Liver Disease (7%), COPD (4%), IHD(2%) and Hypothyroidism(1%). In our study, 57% died, of which 61.4% were males 35) and 38.6% were females (22). The most common etiology of mortality was Sepsis (52.6%) followed by Cardiogenic causes (15.8%) and AGE(8.8%).

The mean NLR of the survival group was significantly lower than that of the mortality group, and there was a statistically significant difference between the two groups ($P < 0.001$). (Tab.1)

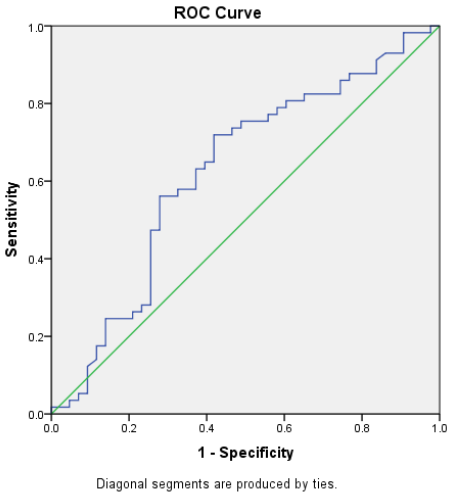
Tab. 1

*** Calculated using Unpaired t test**

	SURVIVOR GROUP (95%CI 8.58-16.4721)	NON-SURVIVOR GROUP (95%CI 12.536-18.8475)	P VALUE
MEAN NLR	12.53±12.82	15.69±11.89	<0.001*

The ROC curve showed that the AUC of the NLR for predicting in-hospital mortality in AKI patients was 0.625 (95% CI 0.511–0.739, $P < 0.05$) and the cutoff value was 8 (sensitivity, 73.7%; specificity, 51.2%).(Fig.1).

Fig. 1



IV. Discussion:

The hypothesis that NLR is associated with outcomes is based primarily on the physiological link between neutrophilia and lymphopenia with systemic inflammation and stress. First reported by Zahorec *et al*., the NLR may be indicative of the patient's response to inflammatory insult, with neutrophils rising in response to stress, which, when overwhelming, induces lymphocyte apoptosis.[2]

Previous reports have highlighted the association between increased NLR and worse outcomes in patients with cancer of the pancreas, breast, lung and colon. In addition to cohorts of oncology patients, in a prospective study, Suliman *et al.* found that a higher NLR was associated with higher rates of mortality in patients admitted with acute coronary syndrome. NLR has been investigated for its association with adverse outcomes in acute pancreatitis and has been identified as a significant predictor of ICU admission and a longer stay.[3]

In our study, we found the mean NLR of the survivor group was significantly lower than that of the non-survivor group among AKI patients, and there was a statistically significant difference between the two groups ($P < 0.001$).The ROC curve showed that the AUC of NLR for predicting in-hospital mortality in AKI patients was 0.625 (95% CI 0.511–0.739, $P < 0.05$) with a cut-off value of 8.(sensitivity, 73.7%; specificity, 51.2%).

Chen JJ et al revealed a linear relationship between composite outcome of AKI mortality and NLR in the medical ICU population.[4] Similar findings have also been reported in a pulmonary embolism population (NLR cut-off value for mortality ranged from 5.4 to 9.2)[5,] in a pancreatitis population (NLR cut-off value for severe pancreatitis: 9.6 with sensitivity: 81.7%, specificity: 55.3% and AUROC: 0.72 at day 0)[6,] and in a CKD population (HR for all-cause mortality: 1.45, HR for cardiovascular event: 1.52)[7.] In septic patients, the relationship between the NLR and mortality is more complex.

Zhang et al found that the NLR of the AKI group was significantly higher than that of the non-AKI group, and there was a statistically significant difference between the two groups ($P < 0.001$). The ROC curve showed that the AUC of the NLR for predicting septic AKI was 0.656 (95% CI 0.584–0.728, $P < 0.001$) and the cutoff value was 17.11 (sensitivity, 62.1%; specificity, 68.9%). But, in contrast to our study, they could not establish association between NLR and in-hospital mortality.[8] The limitations of our study were it was a single center study with small sample size, we only recorded the NLR at admission, and it may make more sense to monitor dynamical NLR changes, atypical lymphocytes and lymphocyte subsets were not considered in our analysis and it was required to carefully interpret NLR because NLR was influenced by medications and morbidities that could affect the neutrophil and lymphocyte count.

V. Conclusion:

NLR, a simple, widely available, and affordable laboratory variable, indicated that it may be a reliable biomarker for the early detection of AKI associated in-hospital mortality.

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