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# CORRELATION OF NEUTROPHIL-LYMPHOCYTE AND PLATELET-LYMPHOCYTE RATIOS WITH IN-HOSPITAL MORTALITY IN PATIENT ACUTE DECOMPENSATED HEART FAILURE

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## ABSTRACT

**Background:** Acute Decompensated Heart Failure (ADHF) is a leading cause of hospitalization among heart failure (HF) patients, contributing to high rates of morbidity and mortality. In such acute decompensated conditions, the body's innate immune system is activated, leading to oxidative stress and the release of proinflammatory cytokines. The Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet-to-Lymphocyte Ratio (PLR) are emerging as cost-effective, easily measurable markers of inflammation that could act as potential prognostic indicators. **Objectives:** The goal of this study was to evaluate the association between NLR and PLR with in-hospital mortality in patients with ADHF. **Methods:** A cross-sectional study was carried out involving 69 ADHF patients who visited the Emergency Department of Muhammadiyah Lamongan Hospital between January 1 and June 30, 2021. NLR and PLR values were recorded at the time of admission. Pearson's correlation test was applied to assess the relationship between NLR, PLR, and in-hospital mortality. The cut-off values were established using the Receiver Operating Characteristic (ROC) curve. **Results:** This study shows that NLR (12.7±9.1 vs 6.8±3.4; r=0.418; p=0.001) and PLR (336.9±214.8 vs 205.4±78.5; r=0.402; p=0.001) values are higher in patients who died in the hospital compared to those who survived and have a statistically significant correlation with hospital mortality. The cut-off value for NLR was 8.33 (sensitivity 67.9%, specificity 68.3%, AUC 73.1%), while the cut-off for PLR was 216 (sensitivity 60.7%, specificity 58.5%, AUC 67%). **Conclusion:** Increased NLR and PLR values are strongly linked to in-hospital mortality and could act as straightforward, dependable prognostic indicators in ADHF patients.



## INTRODUCTION

Acute decompensated heart failure is a clinical syndrome characterized by the worsening of heart failure symptoms and signs, leading patients to present to the emergency department.[1] Along with the elevated rates of morbidity and mortality in the hospital, patients with ADHF often experience recurrent hospitalizations, which subsequently increase the risk of death due to cardiovascular complications.[2] The global number of individuals living with heart failure is estimated to be 64.3 million, while in Indonesia, the proportion of patients with symptomatic heart failure is approximately 0.31% of 968,997 respondents across 440 districts in 33 provinces, according to the 2007 Basic Health Research (Riskesdas).[3,4] Studies show that the mortality and readmission rates for acute heart failure patients one year after discharge from the hospital are around 45%, while 4 to 10 percent of patients experience in-hospital mortality.[5]

The exact mechanisms behind acute heart failure decompensation remain unclear, but numerous studies indicate that systemic inflammation is a critical factor in the development of acute heart failure.[6,7] In cases of acute heart failure, particularly those with severe symptoms, activation of

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inflammatory pathways is often observed.[8] Acute decompensated states trigger an innate immune response, leading to the release of oxidative stress and proinflammatory cytokines.[8–11]

NLR (Neutrophil-to-Lymphocyte Ratio) and PLR (Platelet-to-Lymphocyte Ratio) are systemic inflammation markers that are easily accessible in many hospitals at an affordable cost.[12] Several studies have shown that these markers can serve as prognostic indicators of mortality in cardiovascular diseases.[13] Research conducted on heart failure patients also suggests that the combination of NLR and PLR can be used for risk stratification.[14,15] The relationship between these combined markers and in-hospital mortality in patients with Acute Decompensated Heart Failure (ADHF) is not yet fully understood. On the other hand, evidence regarding the prognostic utility of combined NLR and PLR in predicting mortality among hospitalized ADHF patients remains scarce. Therefore, this study postulates that NLR and PLR, as readily accessible routine hematological markers, exhibit a strong association with in-hospital mortality in ADHF patients. Consequently, the findings of this study may contribute to refining risk stratification and serve as a predictive tool for mortality in ADHF patients, particularly in peripheral healthcare settings.

## METHODS

### Study design

This study is analytical observational research with a cross-sectional design, involving ADHF patients at Muhammadiyah Lamongan Hospital, conducted from January 1 to June 30, 2021.

### Ethical approval

This study has been approved by the Health Research Ethics Committee of Muhammadiyah Lamongan Hospital with the number 1560/KET/III.6.AU/F/ 2020. Prior to the study, all participants were provided with information about the research to ensure they understood the objectives, procedures, and potential risks involved. After understanding the information, participants signed the informed consent form.

### Participants & eligibility criteria

Patients were diagnosed with ADHF based on clinical symptoms, with an ejection fraction (EF) of less than 50% and aged over 40 years. Patients with heart failure who had immune disorders, hematological disorders, infections, malignancies, or who were using anti-inflammatory medications were excluded from the study.

### Data collection

This cross-sectional study was conducted from January 1, 2021, to June 30, 2021. During this period, a total of 69 patients who met the inclusion and exclusion criteria were consecutively enrolled. Patient characteristics data collected included gender, age, body mass index, NYHA class, medical history (hypertension, diabetes mellitus, arrhythmia, ischemic heart disease, stroke, previous heart failure), blood pressure, heart rate, LVEF, laboratory results (hemoglobin, leukocytes, platelets, neutrophils, lymphocytes, sodium, creatinine, random blood glucose), medications administered (ACE inhibitors, beta-blockers, furosemide, spironolactone), and in-hospital mortality or survival status, which were obtained from medical records.

### Measure

All study samples were then divided into two groups: the group that survived and was discharged from the hospital, and the group that died during hospitalization. Blood samples were collected from the antecubital vein when the patients first arrived at the emergency department (ED), and a Complete Blood Count (CBC) was performed using an automated CBC analyzer (Sysmex XN-1000, Sysmex Corporation, Kobe, Japan). From the CBC results, the neutrophil and platelet counts were divided by the total leukocyte count to obtain the Neutrophil-to-Lymphocyte Ratio (NLR) and Platelet-to-Lymphocyte Ratio (PLR). The NLR value is calculated by dividing the neutrophil count by the lymphocyte count, while the PLR value is determined by dividing the platelet count by the absolute lymphocyte count.[16,17]

### Statistical analysis

After the data were collected, the first step was to check for completeness. Incomplete data were supplemented by contacting the patients or the relevant laboratories. The complete data were then reviewed, coded, and entered into a computer for analysis. Data analysis was performed using the

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Statistical Package for the Social Sciences (SPSS) for Windows version 21.0. All study variables were described according to their measurement scales. Categorical data were described using absolute and relative frequencies (percentages), while numerical data were described using the mean and standard deviation. Normality testing was performed using the Kolmogorov-Smirnov test. To assess the relationship between dependent and independent variables, bivariate analysis was conducted using the independent t-test, Mann-Whitney test, and Chi-square test. Correlation testing was performed using the Pearson method. This study also determined the cut-off points for NLR and PLR in relation to in-hospital mortality in ADHF patients using Receiver Operating Characteristic (ROC) curves. A p-value of $< 0.05$ was considered statistically significant.

## RESULTS

### Patients' selection

This research was carried out on patients diagnosed with Acute Decompensated Heart Failure (ADHF) who sought care at the Emergency Department of Muhammadiyah Hospital Lamongan between January 1 and June 30, 2021. The study included 69 patients, 28 of whom died while hospitalized.

### Baseline characteristics

The table explores the relationship between clinical and demographic variables with in-hospital mortality. Patients who died were older (64.1±7.3 years) and had a higher BMI (25.2±1.6) compared to those who survived (58.9±8.2 years and 23.4±1.4, respectively), both showing statistically significant differences ($p<0.05$). A higher prevalence of hypertension (p=0.032) and previous heart failure (p=0.040) was observed in patients who died. However, other comorbidities such as diabetes, arrhythmia, ischemic heart disease, and cerebrovascular disease were not significantly different between the groups. Gender and the presence of NYHA III/IV classification also did not show statistically significant associations with mortality.

Clinical presentation revealed significant differences in systolic and diastolic blood pressure, as well as left ventricular ejection fraction (LVEF). Patients who died had lower systolic (131.4± 44.2 mmHg vs. 150.9±31.4 mmHg, p=0.035) and diastolic blood pressure (78.6±23.8 mmHg vs. 89.9±21.2 mmHg, p=0.041) and reduced LVEF (31.8±7.5% vs. 36.3±7.8%, p=0.013). Laboratory findings showed significant differences in neutrophil percentages (p=0.012) and lymphocyte percentages (p=0.001), with higher neutrophil and lower lymphocyte values in patients who died, indicating an association with inflammation. Other laboratory parameters, including hemoglobin, leukocyte count, platelet count, sodium, creatinine, and blood glucose, were not significantly different.

Regarding medications at admission, the use of ACEI/ARB (p=0.031), beta-blockers (p=0.011), and spironolactone (p=0.036) was significantly more common among survivors, suggesting a potential protective effect. Diuretic use did not differ significantly between groups (p=0.678). These findings highlight the importance of clinical and laboratory parameters, as well as medical therapy, in understanding and potentially predicting in-hospital mortality outcomes.

### Correlation Between NLR and PLR With In-Hospital Mortality

To determine the correlation between the neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) with in-hospital mortality, we compare these ratios with in-hospital mortality. The NLR was significantly higher in patients who died (12.7±9.1) compared to those who survived (6.8±3.4), with a correlation coefficient of 0.418 ($p<0.05$). Similarly, the PLR was significantly higher in patients who died (336.9±214.8) compared to those who survived (205.4±78.5), with a correlation coefficient of 0.402 ($p<0.05$). The Pearson correlation test was performed, and both correlations were statistically significant ($p<0.05$).

### Ethnicity sub-group analysis

The cut-off point for the neutrophil-to-lymphocyte ratio (NLR) is 8.33, with a sensitivity of 67.9% and a specificity of 68.3%. The area under the curve (AUC) is 73.1%, indicating moderate accuracy in distinguishing between positive and negative conditions.

The cut-off point for the platelet-to-lymphocyte ratio (PLR) is 216, with a sensitivity of 60.7% and a specificity of 58.5%. The area under the

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curve (AUC) is 67%, indicating a fair level of accuracy in distinguishing between positive and negative condition

**Table 1.** Basic Characteristics of Research on In-Hospital Mortality

| Variable | In-Hospital Mortality | | *p* |
|---|---|---|---|
| | Died (n=28) | Survived (n=41) | |
| Gender, male - n (%) | 10 (35.7) | 23 (56.1) | 0.096[c] |
| Age, mean±SD | 64.1±7.3 | 58.9±8.2 | 0.009[c*] |
| BMI, mean±SD | 25.2±1.6 | 23.4±1.4 | 0.000[a*] |
| NYHA III/IV, n (%) | 18 (64.3) | 23 (56.1) | 0.496[c] |
| **Past Medical History**, n (%) | | | |
| Hypertension | 15 (53.6) | 32 (78) | 0.032[c*] |
| Diabetes mellitus | 10 (35.7) | 11 (26.8) | 0.431[c] |
| Arrhythmia | 4 (14.3) | 3 (7.3) | 0.346[c] |
| Ischemic heart disease | 11 (39.3) | 22 (53.7) | 0.241[c] |
| Cerebrovascular disease | 4 (14.3) | 2 (4.9) | 0.173[c] |
| Previous heart failure | 21 (75) | 38 (92.7) | 0.040[c*] |
| **Clinical Presentation** | | | |
| Systolic blood pressure - mmHg | 131.4±44.2 | 150.9±31.4 | 0.035[a*] |
| Diastolic blood pressure - mmHg | 78.6±23.8 | 89.9±21.2 | 0.041[a*] |
| Heart rate - /minutes | 97.8±35.8 | 101.5±24.9 | 0.467[b] |
| LVEF - mean±SD | 31.8±7.5 | 36.3±7.8 | 0.013[b*] |
| **Laboratory Results**, mean±SD | | | |
| Haemoglobin - g/dL | 11.3±2.4 | 12.4±2.7 | 0.078[a] |
| Leukocyte - per $mm^3$ | 11.3±5.1 | 10.1±3.9 | 0.279[b] |
| Neutrophil - % | 82.5±8.2 | 77.3±7.1 | 0.012[b*] |
| Lymphocyte - % | 9.2±6.1 | 13.6±5.7 | 0.001[b*] |
| Platelet count - per $mm^3$ | 236±99.5 | 237.6±66 | 0.937[a*] |
| Sodium (Na) – mmol/L | 135.4±5.5 | 137.7±6.2 | 0.111[a] |
| Creatinine – mg/dL | 2.7±1.8 | 2.1±1.8 | 0.110[b] |
| Blood glucose level, mg/dL | 199.6±127.5 | 173.7±101.5 | 0.258[b] |
| **Medication at admission**, n (%) | | | |
| ACEI/ARB | 9 (32.1) | 24 (58.5) | 0.031[c*] |
| Betablockers | 7 (25) | 23 (56.1) | 0.011[c*] |
| Diuretics | 23 (82.1) | 32 (78) | 0.678[c] |
| Spironolactone | 12 (42.9) | 28 (68.3) | 0.036[c*] |

Note: ACEI/ARB, Angiotensin-Converting Enzyme Inhibitor/Angiotensin Receptor Blocker; BMI, Body Mass Index; LVEF, Left Ventricular Ejection Fraction; NYHA, New York Heart Association; SD, Standard Deviation. a: Independent test; b: Mann-Whitney test; c: Chi-square test; *$p < 0.05$ is considered significant*

**Table 2.** The Correlation Between NLR and PLR With In-Hospital Mortality

| Variable | In-Hospital Mortality | | *r* | *p*[a] |
|---|---|---|---|---|
| | Died (n=28) | Survived (n=41) | | |
| NLR | 12.7±9.1 | 6.8±3.4 | 0.418 | 0.000* |
| PLR | 336.9±214.8 | 205.4±78.5 | 0.402 | 0.001* |

Note: NLR, Neutrophil-to-Lymphocyte Ratio; PLR, Platelet-to-Lymphocyte Ratio; [a]Pearson correlation test; *$p$<0.05 significant

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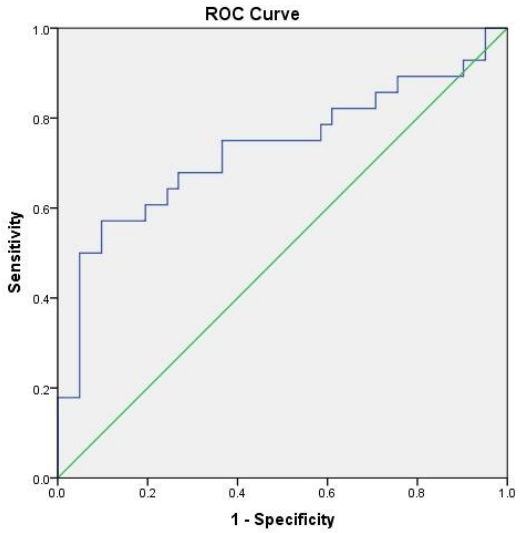


**Figure 1.** Receiver Operating Characteristic (ROC) curve used to observe the point where sensitivity and specificity intersect in the neutrophil-to-lymphocyte ratio (NLR).

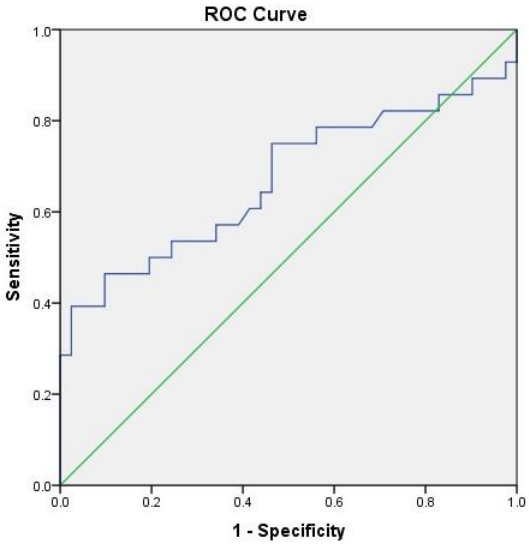


**Figure 2.** Receiver Operating Characteristic (ROC) curve used to examine the point of intersection between sensitivity and specificity in the platelet-to-lymphocyte ratio (PLR).

## DISCUSSION

This study was conducted on 69 patients diagnosed with ADHF, focusing on the relationship between NLR and PLR values and in-hospital mortality. The results showed that the majority of ADHF patients, namely 41 individuals (56.1% of whom were male) with an average age of 58.9 ± 8.2 years, survived and had lower NLR (6.8 ± 3.4 vs. 12.7 ± 9.1; r = 0.418; p = 0.001) and PLR (205.4 ± 78.5 vs. 336.9 ± 214.8; r = 0.402; p = 0.001) values compared to those who died during hospitalization.

Inflammation is a key factor influencing the development and progression of heart failure through multiple mechanisms.[9] This is especially apparent in acute heart failure, which is characterized by a marked rise in neurohormonal activity and systemic inflammation.[8] Several studies have shown that patients with ADHF hospitalized due to worsening symptoms experience an increase in inflammatory

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cytokine levels caused by sterile inflammation (without infection). The extent of systemic inflammation, assessed using different biomarkers, differs among studies.[8] One of the biomarkers that can be used as an indicator of systemic inflammation is the neutrophil-to-lymphocyte ratio (NLR) and the platelet-to-lymphocyte ratio (PLR).

PLR represents the balance between platelet aggregation and systemic inflammation, providing insight into inflammatory coagulation and platelet activation triggered by the inflammatory process. Meanwhile, NLR highlights subclinical inflammation by reflecting the innate immune response through neutrophils and the adaptive immune response via lymphocytes.[18] Recent studies have highlighted that the combination of these two biomarkers can be used to indicate poor outcomes in cardiovascular diseases. For example, this combination can be used to predict the severity of CAD in patients undergoing CAG, assess the risk of mortality during follow-up in heart failure patients, and evaluate the severity of peripheral artery conditions like arteriosclerosis obliterans.[19–21]

This study demonstrates that ADHF patients who died during hospitalization had a higher Neutrophil-to-Lymphocyte Ratio (NLR) compared to those who survived, with a significant positive correlation between these variables, indicating that elevated NLR is associated with an increased risk of in-hospital mortality in ADHF patients. The NLR cut-off value for predicting in-hospital mortality in this study was determined to be 8.33, with an area under the curve (AUC) of 73.1%. The sensitivity and specificity for this threshold were 67.9% and 68.3%, respectively, indicating a good level of predictive accuracy. These findings are consistent with a meta-analysis conducted by Ang et al. in 2023, which included 15 studies with a total of 15,995 ADHF patients. The study demonstrated that patients with significantly higher NLR values had a greater risk of in-hospital mortality [HR 1.54, 95% CI (1.18–2.00), $p$<0.001] and long-term all-cause mortality [HR 1.61, 95% CI (1.40–1.86), p<0.001] compared to those with lower NLR values.[22] Turfan M reported that an NLR (Neutrophil-to-Lymphocyte Ratio) value greater than 4.78 is independently a predictor of in-hospital mortality in patients with acute heart failure. This value demonstrated a sensitivity of 66.7% and a specificity of 60.5% (OR 1.156, 95% CI 1.001–1.334, $p$=0.048).[23]

Although the NLR cut-off value in this study is higher compared to previous research, it remains a useful prognostic marker for predicting mortality in patients with acute heart failure. This condition may be related to the presence of infection or ischemic processes, as mentioned in a study by Cho JH et al. The study stated that the cut-off value of NLR in heart failure patients with worsening conditions due to ischemia or infection tends to be higher than in those without such conditions.[24] The presence of inflammatory reactions and effects on sympathetic tone are two factors that explain how NLR contributes to increased mortality in patients with heart failure.[25] During inflammation, the release of pro-inflammatory cytokines such as myeloperoxidase, acid phosphatase, and elastase leads to reduced ventricular function, which negatively affects the myocardium.[26] On the other hand, decreased lymphocyte levels serve as an independent factor contributing to lower survival rates in heart failure patients.[27] This condition is linked to neurohormonal activation and disruptions in the regulation of lymphocyte differentiation, proliferation, and apoptosis.[28]

A similar trend was also observed in this study regarding PLR values, where higher PLR was associated with an increased incidence of in-hospital mortality in ADHF patients. Furthermore, this study identified a cut-off value with moderate predictive capability and a balance between sensitivity and specificity. A study conducted by Ye G. Lian on 443 patients with acute heart failure between 2010 and 2017 revealed that high PLR values have the potential to serve as a novel marker in the management of acute heart failure patients. This is based on findings indicating that high PLR values are associated with poor clinical outcomes in these patients.[29] A meta-analysis conducted in 2024, involving 5 studies with a total of 2,179 heart failure patients and an average age of 73.18±11.16 years, revealed that the in-hospital mortality rate in this patient group was associated with a PLR value of 192.83 (95% CI: 150.06–235.61).[30] Delcea et al demonstrated that a PLR cut off point of 154.78 could effectively predict in-hospital mortality, with an AUC of 0.658 (95% CI: 0.567–0.750, p = 0.001), a sensitivity of 54.05%, and a specificity of 67.40%.[31] The poor outcomes

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associated with increased PLR levels have been widely demonstrated in various studies. This is attributed to platelet aggregation and inflammatory processes, which serve as the main mechanisms underlying elevated PLR, making it a novel prognostic marker.[17] Similar to NLR, an increase in PLR is also associated with systemic inflammatory processes that can disrupt myocardial cells, further impairing the pumping function of the left ventricle. This condition is caused by the release of cytokines during the inflammatory process, such as tumors necrosis factor α (TNF-α), C-reactive protein (CRP), and interleukin 6 (IL-6).[32]

NLR and PLR, as easily accessible and affordable inflammatory markers, are used to predict mortality in heart failure patients, especially after treatment for acute decompensated heart failure, with their combination providing better predictive value, particularly in HFpEF patients, as they reflect different aspects of the inflammatory response related to non-cardiac comorbidities.[12] This study has some limitations, such as a limited sample size and being carried out at a single research center, which could affect the results and may not reflect a broader, global viewpoint. This limitation also necessitates cautious interpretation of the study results. A second limitation is that PLR and NLR are markers of systemic inflammation, which makes it difficult to entirely rule out the presence of subclinical chronic conditions in heart failure patients. Furthermore, other inflammatory markers were not measured in this study. The third limitation is the absence of repeated measurements of NLR and PLR during treatment or at discharge. This study relied solely on initial values obtained at the emergency department, making it impossible to assess the fluctuations in NLR and PLR levels.

## CONCLUSION

Patients with ADHF who died during hospitalization had elevated NLR and PLR values compared to those who survived. The NLR threshold of 8.3 and PLR threshold of 216 showed a promising ability to predict the prognosis in ADHF patients. Nevertheless, additional studies are required to confirm these results.

## ETHICAL APPROVAL

Ethical approval was granted by the Health Research Ethics Committee of Muhammadiyah Lamongan Hospital.

## CONFLICTS OF INTEREST

The authors report no conflicts of interest.

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## AUTHOR CONTRIBUTIONS

Conceptualization, AAB and ZR; methodology, AAB; software, AAB; validation, AAB, ZR, and LP; investigation, AAB; resources, ZR; data curation, LP; writing—review and editing, ZR; visualization, AAB; supervision, LP; project administration, AAB.

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