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The Role of Neutrophil Lymphocyte Ratio (NLR) and Platelet Lymphocyte Ratio (PLR) as Predictors of Reduced Left Ventricular Ejection Fraction (LVEF) in Heart Failure Patients

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ABSTRACT

Background: Heart failure can be considered an emerging epidemic due to increasing cases along with the increasing population. The pathogenesis of heart failure is closely related to inflammation. Some of the biomarkers that can be used to assess the severity of inflammation in patients are neutrophil lymphocyte ratio (NLR) and platelet lymphocyte ratio (PLR).

Objectives: The purpose of this study was to determine the relationship between NLR and PLR with left ventricular ejection fraction (LVEF) and to determine the role of NLR and PLR as predictors of reduced LVEF.

Methods: This observational cross-sectional study was conducted among heart failure patients at Dr. H. Abdul Moeloek Hospital, Lampung Province, in 2023. A total of 88 patients with heart failure were included in this study. This study used the Spearman rank correlation test and receiver operating characteristics (ROC) analysis test to determine the cut-off point of NLR and PLR as predictors of reduced left ventricular ejection fraction. In this study, left ventricular ejection fraction was divided into 2 groups (≤40% and >40%).

Result: NLR has a significant relationship with LVEF (r: -0.290, p-value: 0.006), and PLR also has a significant relationship with LVEF (r: -0.297, p-value: 0.005). NLR >2.67 can be used as a predictor of reduced left ventricular ejection fraction with a sensitivity of 70.7% and a specificity of 70% (AUC: 0.747, p-value: <0.001). PLR >119.69 can be used as a predictor of reduced left ventricular ejection fraction with a sensitivity of 62.1% and a specificity of 63.3% (AUC: 0.707, p-value: 0.001).

Conclusion: NLR and PLR are associated with LVEF in patients with heart failure. NLR >2.67 and PLR >119.69 can be used as a cheap, simple, and fast predictor of reduced LVEF in heart failure patients.

INTISARI

Latar Belakang: Gagal jantung dapat disebut sebagai *emerging epidemic* karena jumlah kasus yang terus meningkat seiring bertambahnya populasi. Patogenesis gagal jantung memiliki kaitan yang erat dengan peradangan. Salah satu biomarker yang dapat digunakan untuk menilai tingkat keparahan peradangan dari pasien adalah rasio neutrofil limfosit (RNL) dan rasio platelet limfosit (RPL).

Tujuan: Tujuan dari penelitian ini adalah mengetahui hubungan RNL dan RPL dengan fraksi ejeksi ventrikel kiri (FEVK) serta mengetahui peran RNL dan RPL sebagai prediktor FEVK yang menurun.

Metode: Penelitian ini merupakan penelitian observasional analitik dengan desain potong lintang pada penderita gagal jantung di RSUD Dr. H. Abdul Moeloek Provinsi Lampung sepanjang tahun 2023.

Terdapat 88 pasien dengan gagal jantung yang diikutsertakan dalam penelitian ini. Penelitian ini menggunakan uji korelasi Spearman rank dan uji analisis *receiver operating characteristics* (ROC) untuk mencari titik potong RNL dan RPL sebagai prediktor fraksi ejeksi ventrikel kiri yang menurun. Dalam penelitian ini, fraksi ejeksi ventrikel kiri dibagi menjadi 2 kelompok, yaitu ≤40% dan >40%.

Hasil: RNL memiliki hubungan yang signifikan dengan FEVK (*r*: -0.290, *p-value*: 0.006), dan RPL juga memiliki hubungan yang signifikan dengan FEVK (*r*: -0.297, *p-value*: 0.005). RNL >2.67 dapat dijadikan sebagai prediktor fraksi ejeksi ventrikel kiri yang menurun dengan sensitivitas 70.7% dan spesifisitas 70% (AUC: 0.747, *p-value*: <0.001). RPL >119.69 dapat dijadikan sebagai prediktor fraksi ejeksi ventrikel kiri yang menurun dengan sensitivitas 62.1% dan spesifisitas 63.3% (AUC: 0.707, *p-value*: 0.001).

Kesimpulan: NLR dan PLR berhubungan dengan FEVK pada penderita gagal jantung. RNL >2.67 dan RPL >119.69 dapat dijadikan sebagai prediktor yang murah, sederhana, dan cepatdalam memprediksi FEVK yang menurun pada penderita gagal jantung.

Kata Kunci: rasio neutrofil limfosit; rasio platelet limfosit; fraksi ejeksi ventrikel kiri; gagal jantung

INTRODUCTION

Heart failure can be considered an emerging epidemic epidemic, with the number of heart failure cases continuing to increase along with the increasing population, especially the elderly population[1]. The incidence of heart failure globally ranges from 1 to 9 cases in every 1000 people[2]. The one-year mortality rate for patients with acute heart failure is quite high, reaching 23.6%, and 6.4% in chronic heart failure. From 2005 to 2009 in the United States, the 5-year mortality rate for heart failure reached 75.4%[3].

The pathogenesis of heart failure is closely related to inflammation and is interrelated[4]. The pathogenesis of heart failure is related to maladaptive responses in the innate and adaptive immune systems that contribute to adverse cardiac remodeling, fibrosis, and disorders outside the heart[5]. There are many biomarkers that can be used to assess the inflammatory process that occurs, such as the neutrophil lymphocyte ratio (NLR) and platelet lymphocyte ratio (PLR)[4,6].

One of the indicators that can be used to assess prognosis and determine the therapy process is the left ventricular ejection fraction (LVEF)[7]. Therefore, this study will examine the role of NLR and PLR in predicting reduced LVEF in heart failure patients at Dr. H. Abdul Moeloek Hospital.

METHODS

This study was an observational analytical study with a cross-sectional design conducted among patients diagnosed with heart failure at Dr. H. Abdul Moeloek Hospital, Lampung Province, throughout 2023. A total of 88 patients with heart failure were included in this study. The inclusion criteria in this study were patients (1) diagnosed with heart failure at Dr. H. Abdul Moeloek Hospital throughout 2023 and[2] have medical record data that includes complete blood count and echocardiography. The exclusion criteria in this study were[1] patients with immunocompromised conditions[2], patients with chronic inflammatory diseases, such as rheumatoid arthritis[3], patients with autoimmune diseases, and[4] patients with leukemia.

Data were analyzed using the Spearman rank correlation and receiver operating characteristic (ROC) curve analysis test to determine the cut-off point for NLR and PLR as predictors of reduced left ventricular ejection fraction. In this study, the left ventricular ejection fraction was divided into 2 groups, namely ≤40% and >40%.

RESULT

Patient characteristics are presented in Table 1.

Table 1. Frequency Distribution of Sample Characteristics

Characteristics	Frequency (%)	Total (%)
Gender		
Male	54 (61.4)	88 (100)
Female	34 (38.6)	
Age		
<65	62 (70.5)	88 (100)
≥65	26 (29.5)	

In this study, the sample was dominated by the male gender, which was 54 samples (61.4%), with the female gender as many as 34 samples (38.6%). The age of the sample was dominated by the age group <65 years, with as many as 62 samples (70.5%), and followed by the age group ≥65 years, with as many as 26 samples (29.5%). Samples with reduced LVEF (≤40%) were 58 samples (65.9%), and samples with LVEF above 40% were 30 samples (34.1%).

Table 2. Variable Characteristics

Heart Failure Patient		
NLR	Mean (SD)	5.55 (6.89)
	Median	3.58
	Range	46.50
PLR	Mean (SD)	168.33 (152.69)
	Median	124.61
	Range	972.41
LVEF	Mean (SD)	36.39 (10.59)
	Median	35
	Range	69

Table 2 presents the mean values of the research variables, including NLR, PLR, and LVEF. The average NLR is 5.5 with a standard deviation of 6.89. The average PLR is 168.33 with a standard deviation of 152.69. While the average LVEF in this study is 36.39 with a standard deviation of 10.59.

Table 3. Spearman rank analysis result

Parameter	*r*	*p-value*
NLR	-0.290	0.006
PLR	-0.297	0.005

Table 4. ROC analysis

Area Under the Curve					
Test Resul Variable	Area	Std. Error	Asymptotic Sig.	Asymptotic Confidence Interval 95%	
				Lower Bound	Upper Bound
NLR	.747	.057	.000	.634	.859
PLR	.707	.061	.001	.588	.827

Table 4 shows that there is a significant relationship between NLR and PLR with LVEF. The accuracy of predicting reduced left ventricular ejection fraction (≤40%) can be observed in figure 1. NLR has the highest area under the curve (AUC: 0.747, *p-value* <0.001) followed by PLR (AUC: 0.707, *p-value* 0.001).

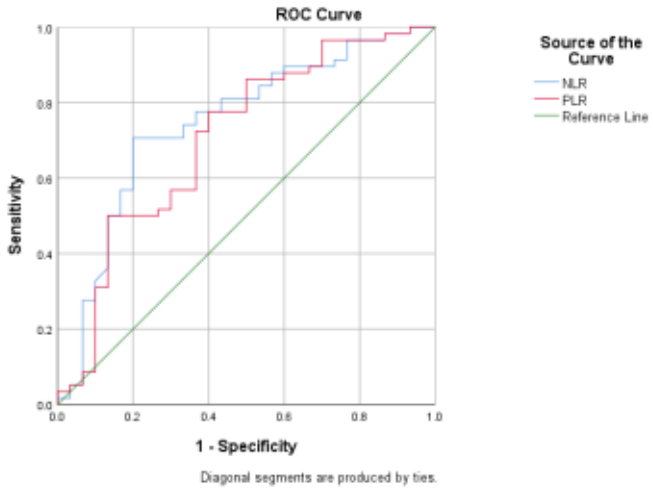

Figure 1. NLR and PLR ROC Curve

The best cut-off point of NLR to predict reduced LVEF (≤40%) is at 2.67 with a sensitivity of 70.7% and a specificity of 70%. The best cut-off point of PLR to predict low LVEF (≤40%) is at 119.69 with a sensitivity of 62.1% and a specificity of 63.3%.

Table 5. Hypothesis Analysis of Neutrophil Lymphocyte Ratio (NLR) with Left Ventricular Ejection Fraction (LVEF)

		Neutrophil Lymphocyte Ratio				PR (CI 95%)	p-value
		>2.67		<2.67			
		n	%	n	%		
LVEF	≤40%	21	55.3	17	44.7		<0.001
	>40%	9	18	41	82	3.07 (1.591-5.923)	

Samples with a high NLR (>2.67) experienced reduced LVEF (≤40%) in 21 samples (55.3%) more than those with LVEF >40%. The prevalence ratio was 3.07 (p < 0.001, 95% CI 1.591-5.923), which means that samples with a neutrophil lymphocyte ratio >2.67 had a prevalence of reduced LVEF (≤40%) that was 3.07 times higher.

Table 6. Hypothesis Analysis of Platelet Lymphocyte Ratio (PLR) with Left Ventricular Ejection Fraction (LVEF)

		Platelet Lymphocyte Ratio				PR (CI 95%)	p-value
		>119.69		<119.69			
		n	%	n	%		
LVEF	≤40%	19	46.3	22	53.7		<0.001
	>40%	11	23.4	36	76.6	1.98 (1.072-3.656)	

Samples with a high PLR (>119.69) experienced reduced LVEF (≤40%) in 19 samples (46.3%) more than those with LVEF >40%. The prevalence ratio was 1.98 (p < 0.001, 95% CI 1.072-3.656), which means that samples with neutrophil lymphocyte ratio >119.69 had a prevalence of reduced LVEF (≤40%) that was 3.07 times higher.

DISCUSSION

Most participants were male, with a percentage of 61.4%, followed by women at 38.6%. This is in line with research from Curran in 2021, which explained that the incidence of heart failure was higher in the male group than in the female group[8]. Hari Hu's research in 2022 also stated that the majority of heart failure patients studied were male, which was around 53.06%[9]. Men have a higher lifetime risk of developing heart failure with reduced ejection fraction than women, with a risk of 10.6% in men and 5.8% in women[1]. Men have a higher proportion of cigarette and alcohol consumption, a higher distribution of body mass index, a higher prevalence of diabetes mellitus, and a higher prevalence of ischemic heart disease[10].

Based on age, the study sample was dominated by the age group <65 years with 70.5%, with the age group ≥65 years

at 29.5%. This is in line with research by Deek in Lebanon with an average age of heart failure samples of 57.98 years[11]. Research from THESUS-HF also stated that the average age of heart failure patients was 52.3 years. Research in the Persian Gulf by Gulf CARE also stated that the average age of heart failure patients was 59 years[12]. The older the age of heart failure patients, the more at risk they are of experiencing more comorbidities[13]. Older patients with heart failure have a higher mortality rate than older patients. However, sudden death is the most common cause for younger patients with heart failure (≤ 55 years), while older patients (≥85 years) are more often affected by noncardiovascular causes[14].

Inflammation is a major contributor to the pathophysiology of heart failure, especially in heart failure with reduced ejection fraction[15]. Although the exact mechanism of inflammation's contribution to determining heart failure prognosis is not fully understood, the role of inflammation in the pathophysiology of heart failure is related to inflammatory biomarkers[8]. Elevated levels of inflammation are also linked to poor prognosis in coronary artery disease (CAD). White blood cells and their subtypes serve as significant inflammatory indicators in cardiovascular diseases (CVDs). In response to inflammatory stimuli, leukocytes secrete various inflammatory cytokines, such as TNF-α, IL-6, and CRP, along with proteolytic enzymes. These pro-inflammatory mediators exert detrimental effects on the myocardium, leading to impaired left ventricular function and the development of heart failure[16].

Neutrophils are one of the leukocytes that act as important modulators in the inflammatory process[17]. Neutrophils secrete the enzyme myeloperoxidase, which enhances their phagocytic activity; however, elevated levels of this enzyme lead to excessive production of free radicals, resulting in tissue damage[18]. Neutrophils are sensitive to inflammation that occurs in heart failure, where there will be an increase in cell age due to delayed apoptosis and will lead to neutrophilia. Neutrophilia itself is independently related to the severity and prognosis of heart failure patients[4]. Neutrophils exhibit a high turnover rate and can be affected by acute clinical conditions. Therefore, conducting serial baseline measurements could have provided more informative results[8].

Platelets are considered to be a link between inflammatory status, thrombosis, and hemostasis[19]. Platelet involvement and activation in heart failure have a reciprocal relationship, creating a vicious cycle of persistent inflammation. Heart failure patients will have levels of C-C chemokines secreted by platelets and they will be proportional to the level of damage that occurs. Platelet activation in heart failure is further evidenced by increased aggregability, elevated platelet-derived adhesion molecules, greater mean platelet volume, and higher soluble P-selectin levels, regardless of cardiac etiology[6]. Increased platelet count in circulation has the potential to cause complications in congestive heart failure[20].

Lymphocytes consist of various types of cells that function as adaptive immunity by providing specific responses to inflammation[21]. Increased systemic cytokines in heart failure promote lymphocyte apoptosis[6]. Lymphocytes exert immunomodulatory effects by inducing tissue inhibitor of metalloproteinase-1 expression. In heart failure, activation of the hypothalamic–pituitary–adrenal axis increases cortisol secretion, which promotes lymphocyte apoptosis and leads to lymphocytopenia[22]. In addition, lymphocytes, which consist of various types (such as B cells, CD4-positive T cells, CD4/CD8-negative or CD8-positive T cells, and natural killer T cells), play a crucial role in adaptive immunity by mounting specific responses to antigens that are regulated by major histocompatibility complex (MHC) class I[21].

The division of neutrophils and lymphocytes will produce a new index, namely the neutrophil lymphocyte ratio (NLR), which can be used as a predictor of the occurrence and prognosis of cardiovascular disease[22]. The division between platelets and lymphocytes will produce an index called the platelet lymphocyte ratio (PLR) which is reported to function as a clinical predictor for various diseases, such as kidney disease, cardiovascular disease, and malignancy[23].

In this study, NLR had a negative relationship with LVEF (*r*: -0.290, *p-value* 0.006). This is in line with research from Durmus, which stated a negative relationship between NLR and LVEF in patients with heart failure (*r*: -0.409, *p-value* <0.001)[16]. Based on the ROC curve analysis, this study indicated that subjects with high NLR (>2.67) had a higher prevalence of reduced LVEF 3.07 times, with AUC value of 0.747, a sensitivity of 70.7%, and a specificity of 70%. In a different study, an NLR threshold of 3.0 was identified as the optimal cut-off for predicting heart failure, yielding a sensitivity of 86.3% and a specificity of 77.5% (AUC = 0.868, p < 0.001) (16). Beside that, it was stated that NLR >3.70 can function as a predictor of 1-year mortality for heart failure patients (AUC: 0.705)[24].

PLR also showed a negative correlation with LVEF (r = -0.297, *p-value* = 0.005), indicating that patients with high PLR values tend to have lower LVEF. This is reinforced by research from Delcea in 2023, which stated that an increase in PLR is proportionally related to reduced LVEF, increased NT-proBNP and troponin levels[6]. PLR >119.69 can predict reduced LVEF (≤40%) in heart failure patients. This study indicated that subjects with high PLR (>119.69) had a higher prevalence of reduced LVEF 1.98 times, with AUC value of 0.707, a sensitivity of 62.1%, and a specificity of 63.3%. In another study, it was stated that PLR >137.3 can be a predictor of the incidence of heart failure with a sensitivity of 70% and a specificity of 60% (AUC: 0.689)[16].

The NLR and PLR cut-off points identified in this study (>2.67 and >119.69, respectively) are slightly lower than those reported by Durmus et al. (NLR >3.70; PLR >137.3), which may be related to ethnic and population differences, disease severity, or treatment variations. Despite these differences, the overall direction of correlation aligns with previous findings, reinforcing that inflammatory markers are consistent predictors of cardiac dysfunction across populations.

Clinically, the simplicity and cost-effectiveness of NLR and PLR measurements make them attractive for early

identification of heart failure patients at risk of reduced LVEF, particularly in settings with limited access to echocardiography or advanced biomarkers such as NT-proBNP. Therefore, NLR and PLR are cheap, simple, and rapid inflammatory parameters in assessing prognosis in patients, especially heart failure[6, 28].

This study presents several limitations. First, it was a single-center, retrospective analysis with a relatively small sample size, which may restrict the generalizability of the findings. Second, due to its cross-sectional design, a causal relationship between inflammation and LVEF reduction cannot be determined. Future multicenter, prospective studies are needed to validate the cut-off points and explore the dynamic changes of NLR and PLR in response to treatment or during acute decompensation.

CONCLUSION

NLR and PLR are associated with LVEF in patients with heart failure. NLR >2.67 and PLR >119.69 can be used as cheap, simple, and fast predictors of reduced LVEF in patients with heart failure.

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DISCLOSURES AND ETHICS

The authors declare that there is no conflict of interest. This study has been approved by The Research Ethics Committee of Dr. H. Abdul Moeloek Hospital, number 377/KEPK-RSUDAM/X/2024. The need for patient consent was waived due to the retrospective nature of the study and anonymized data usage.

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