

Research Article

Mean Platelet Volume and Neutrophil-to-Lymphocyte Ratio in Preeclampsia and Normal Pregnancy

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Abstract

Objectives: To compare mean platelet volume (MPV) and neutrophil-to-lymphocyte ratio (NLR) values between women with preeclampsia and those with normal pregnancies.

Methods: This cross-sectional study used secondary data obtained from the medical records of A. Yani Islamic Hospital, Surabaya. A total of 63 third-trimester pregnant women were selected using purposive sampling, including 30 women with preeclampsia and 33 women with normal pregnancies. MPV and NLR values were obtained from hematological examination records at the time of diagnosis. Data normality was assessed using the Shapiro-Wilk test. As the data were normally distributed, an independent t-test was performed to compare the mean MPV and NLR values between the two groups. Statistical significance was set at $p < 0.05$.

Results: The mean MPV did not differ significantly between the normal pregnancy and preeclampsia groups (9.05 ± 0.79 vs. 9.12 ± 1.10 fL, respectively; $p = 0.756$). The mean NLR was significantly higher in the preeclampsia group than in the normal pregnancy group (5.34 ± 2.94 vs. 4.11 ± 1.07 , respectively; $p = 0.028$).

Conclusion: Women with preeclampsia had significantly higher NLR values than women with normal pregnancies, whereas MPV values did not differ significantly between the groups. These findings suggest that NLR may be associated with preeclampsia, whereas MPV was not significantly different between women with preeclampsia and those with normal pregnancies.

Keywords: hematological parameter, mean platelet volume (MPV), neutrophil lymphocyte ratio (NLR), preeclampsia, pregnancy.

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INTRODUCTION

Preeclampsia is a pregnancy - specific hypertensive disorder and a major cause of maternal and perinatal morbidity and mortality worldwide. Hypertensive disorders of pregnancy, including preeclampsia and eclampsia, account for approximately 16% of maternal deaths globally, corresponding to approximately 42,000 deaths in 2023, with a disproportionately high burden in low-resource settings.¹ In addition to increasing maternal morbidity and mortality, preeclampsia is associated with adverse maternal, fetal, and

neonatal outcomes, including stroke, pulmonary edema, thromboembolic events, disseminated intravascular coagulation, multiorgan failure, fetal growth restriction, preterm birth, low birth weight, stillbirth, and the need for prolonged neonatal care.² Women with a history of preeclampsia are also at increased long-term risk of hypertension, heart failure, ischemic heart disease, stroke, and cardiovascular death. Established risk factors include a previous hypertensive disorder of pregnancy, chronic kidney disease, chronic hypertension, type 1 or type 2 diabetes mellitus, nulliparity, advanced maternal age, obesity, and

multiple pregnancy.²

The management of preeclampsia includes blood pressure control to reduce the risk of severe maternal complications. An Indonesian multicenter hospital-based study reported that antihypertensive agents, including methyldopa, nifedipine, and amlodipine, were prescribed rationally and effectively reduced blood pressure among women with preeclampsia.³ However, pharmacological management primarily controls maternal hypertension and does not directly address the underlying pathophysiological processes of preeclampsia. Therefore, early identification of women at risk remains important for preventing disease progression and adverse maternal and perinatal outcomes.

The pathophysiology of preeclampsia is multifactorial; however, particularly in early-onset preeclampsia, abnormal placentation resulting from inadequate extravillous trophoblast invasion and defective spiral artery remodeling is considered a central initiating mechanism.⁴ These abnormalities reduce uteroplacental perfusion, leading to placental ischemia, oxidative stress, and progressive hypoxia.⁵ In response, the ischemic placenta releases reactive oxygen species, pro-inflammatory cytokines, chemokines, and antiangiogenic factors into the maternal circulation, resulting in systemic endothelial dysfunction and an exaggerated inflammatory response.⁵ Furthermore, an imbalance between proangiogenic signaling mediated by Vascular Endothelial Growth Factor (VEGF), Placental Growth Factor (PlGF), and Transforming Growth Factor- β (TGF- β) and antiangiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng) contributes to maternal endothelial dysfunction in preeclampsia.⁶

Endothelial dysfunction promotes platelet activation and increased platelet turnover, resulting in hematological alterations that may be detected using routine laboratory parameters. Mean platelet volume (MPV) reflects platelet activation and increased platelet production in response to platelet consumption and has been reported to increase with the severity of preeclampsia.^{7,8} In addition, the neutrophil-to-lymphocyte ratio (NLR) reflects the systemic inflammatory response associated with endothelial dysfunction and the release of inflammatory mediators.⁷ Because MPV and NLR are inexpensive, readily available, and routinely measured hematological parameters, they have attracted considerable attention as potential

indicators associated with preeclampsia.

Although several studies have evaluated MPV and NLR in women with preeclampsia, inconsistent findings have been reported across different populations and clinical settings. Therefore, further evaluation of these hematological parameters remains necessary. This study aimed to compare the mean MPV and NLR values between women with preeclampsia and those with normal pregnancies at A. Yani Islamic Hospital, Surabaya.

METHODS

This analytical cross-sectional study was conducted using secondary data obtained from the medical records of pregnant women at A. Yani Islamic Hospital, Surabaya, Indonesia. Medical records from January to June 2024 were reviewed. The study population consisted of third-trimester pregnant women who attended the hospital during the study period. Eligible participants were classified into two groups according to pregnancy status: women with normal pregnancies and women diagnosed with preeclampsia.

A total of 63 eligible pregnant women, comprising 33 women with normal pregnancies and 30 women with preeclampsia, were selected using purposive sampling from medical records that met the predefined inclusion and exclusion criteria. Women aged 20–40 years with either a normal pregnancy or preeclampsia were eligible for inclusion. Preeclampsia was diagnosed after 20 weeks of gestation based on a systolic blood pressure of ≥ 140 mmHg and/or a diastolic blood pressure of ≥ 90 mmHg measured on two occasions at least four hours apart using a calibrated sphygmomanometer. The diagnosis was confirmed by proteinuria, defined as a dipstick result of $\geq 2+$, 24-hours urinary protein of ≥ 300 mg, or a protein-to-creatinine ratio of ≥ 0.3 mg/dL. In the absence of proteinuria, preeclampsia was diagnosed in the presence of at least one severe feature, including thrombocytopenia ($< 100,000/\mu\text{L}$), renal insufficiency (serum creatinine > 1.1 mg/dL), impaired liver function with elevated liver transaminases, pulmonary edema, or persistent neurological or visual symptoms. Only participants with complete medical records, including complete blood count results, were included in the study.

Pregnant women with ruptured membranes, eclampsia, HELLP syndrome, multiple pregnancies,

congenital fetal anomalies, fetal chromosomal abnormalities, intrauterine fetal death, anemia, infectious diseases during pregnancy, or maternal comorbidities, including chronic kidney disease, vascular disease, hypothyroidism, malignancy, diabetes mellitus, and ischemic heart disease, were excluded. Medical records with incomplete information were also excluded from the analysis.

Maternal age, gestational age, pregnancy status, mean platelet volume (MPV), and neutrophil-to-lymphocyte ratio (NLR) were extracted from the medical records. MPV and NLR values were obtained from complete blood count examinations performed during the third trimester at the time of preeclampsia diagnosis. Pregnancy status (normal pregnancy or preeclampsia) served as the grouping variable, while maternal age, gestational age, MPV, and NLR were compared between the two groups.

Continuous variables were presented as mean $\pm$ standard deviation (SD). Data normality was assessed using the Shapiro–Wilk test because

each study group consisted of fewer than 50 participants. Homogeneity of variances was assessed using Levene's test. As both assumptions were satisfied, independent t-tests were performed to compare maternal age, gestational age, MPV, and NLR between women with preeclampsia and those with normal pregnancies. A two-tailed *p*-value of <0.05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics version 26.

Ethical approval for this study was obtained from the Research Ethics Committee, Faculty of Medicine, Universitas Nahdlatul Ulama Surabaya (Approval No. 280/KEPK-UNUSA/X/2024).

RESULTS

A total of 63 third-trimester pregnant women were included in this study, comprising 33 women with normal pregnancies and 30 women with preeclampsia. The baseline characteristics of the study participants are presented in Table 1.

Table 1. Baseline Characteristics of Study Participants

Characteristic	Groups		P-value
	Normal (n=33) mean $\pm$ SD	Preeclampsia (n=30) mean $\pm$ SD	
Maternal age (years)	29.45 $\pm$ 5.23	31.00 $\pm$ 4.71	0.224
Gestational age (weeks)	38.29 $\pm$ 1.58	37.75 $\pm$ 1.77	0.206

There were no significant differences in maternal age (*p* = 0.224) or gestational age (*p* = 0.206) between women with normal pregnancies and those with preeclampsia, indicating that the two groups were comparable with respect to these baseline characteristics.

Table 2. Comparison of MPV and NLR Between Study Groups

Variable	Normal mean $\pm$ SD	Preeclampsia mean $\pm$ SD	P-value
Mean Platelet Volume / MPv (fL)	9.05 $\pm$ 0.79	9.12 $\pm$ 1.10	0.756
Neutrophil Lymphocyte Ratio / NLR	4.11 $\pm$ 1.07	5.34 $\pm$ 2.94	0.028

As shown in Table 2, the mean MPV did not differ significantly between women with normal pregnancies and those with preeclampsia (9.05 $\pm$ 0.79 vs. 9.12 $\pm$ 1.10 fL, respectively; *p* = 0.756). In contrast, the mean NLR was significantly higher in women with preeclampsia than in those with normal pregnancies (5.34 $\pm$ 2.94 vs. 4.11 $\pm$ 1.07, respectively; *p* = 0.028). Thus, NLR showed a significant between-group difference, whereas MPV did not.

DISCUSSION

Preeclampsia is a pregnancy-specific multisystem disorder characterized by hypertension after 20 weeks of gestation accompanied by proteinuria or maternal organ dysfunction resulting from widespread endothelial dysfunction.^{9,10} It affects approximately 2–8% of pregnancies worldwide and remains one of the leading causes of maternal and perinatal

morbidity and mortality.^{11,12} Severe preeclampsia may progress to multiorgan complications, including thrombocytopenia, acute kidney injury, hepatic dysfunction, pulmonary edema, and neurological complications, while fetal consequences include fetal growth restriction, preterm birth, placental abruption, and perinatal death.^{13,14} The pathophysiology of preeclampsia involves abnormal placentation, placental ischemia, oxidative stress, systemic inflammation, and endothelial injury, all of which may contribute to hematological alterations during pregnancy.¹⁵

The baseline characteristics of the present study showed no significant differences in maternal age or gestational age between the two groups, indicating that they were generally comparable with respect to these characteristics. Nevertheless, because adjustment for other potential confounding variables was not performed, the possibility of residual confounding cannot be completely excluded.

The present study demonstrated that the mean MPV was slightly higher in women with preeclampsia than in those with normal pregnancies; however, the difference was not statistically significant. This finding is consistent with previous studies reporting no significant difference in MPV between women with normal pregnancies and those with preeclampsia.¹³ In contrast, other studies have reported significantly higher MPV values, particularly among women with severe preeclampsia.¹⁴ These discrepancies may be explained by differences in disease severity, gestational age at blood sampling, laboratory measurement techniques, study populations, and sample sizes.

MPV reflects platelet size and may serve as an indicator of platelet activation and turnover. Endothelial injury associated with preeclampsia promotes platelet activation and consumption, followed by compensatory thrombopoiesis and the release of larger, younger platelets into the circulation.¹⁵ Platelet activation may be further amplified by inflammatory mediators released in response to placental ischemia, contributing to endothelial dysfunction.^{16,17} In addition, endothelial injury caused by placental hypoperfusion may activate coagulation pathways and increase platelet turnover, providing a biological rationale for evaluating MPV in preeclampsia.¹⁸ However, the clinical performance of MPV has remained inconsistent across studies. MPV may be affected by several factors unrelated to preeclampsia, including preanalytical conditions,

anticoagulant exposure time, hematology analyzer differences, and physiological variations during pregnancy.^{19,20} Furthermore, platelet activation may vary according to the severity and stage of preeclampsia. Therefore, MPV should be interpreted cautiously and in conjunction with other clinical and laboratory findings rather than as an isolated hematological parameter.

In contrast, the present study demonstrated that NLR was significantly higher in women with preeclampsia than in those with normal pregnancies. This finding is consistent with previous studies reporting elevated NLR values among women with preeclampsia.²¹ Similar observations have been reported across different populations, supporting the potential role of NLR as a marker of the systemic inflammatory response associated with preeclampsia.⁷ The significantly higher NLR observed in the present study is also consistent with the established role of inflammatory activation in the pathophysiology of preeclampsia.

The elevated NLR observed in the present study is biologically plausible. Failure of spiral artery remodeling results in persistent placental hypoperfusion, promoting the release of antiangiogenic factors, including soluble fms-like tyrosine kinase-1 (sFlt-1), and pro-inflammatory mediators that contribute to endothelial dysfunction and systemic inflammation. This inflammatory environment may stimulate neutrophil recruitment and activation in the maternal circulation. Activated neutrophils produce reactive oxygen species, matrix metalloproteinases, and pro-inflammatory cytokines, including interleukin-8 and tumor necrosis factor- α , which may further aggravate endothelial injury and vascular dysfunction.^{22,23} Persistent systemic inflammation may also be accompanied by relative lymphocyte suppression, resulting in an increased NLR.²⁴⁻²⁶ Thus, the higher NLR observed in women with preeclampsia is consistent with the inflammatory mechanisms underlying the disorder.

NLR integrates relative changes in circulating neutrophils and lymphocytes into a single index and may therefore provide an indirect measure of systemic inflammatory and immune activity.²⁷ Because it is derived from routinely available complete blood count parameters, NLR is inexpensive and readily accessible. Increased NLR in women with preeclampsia has been associated with systemic inflammation, neutrophil activation, and endothelial dysfunction, generating

interest in its potential role as an adjunctive hematological marker.²⁸ However, NLR should not be considered a standalone diagnostic or predictive biomarker. Rather, it may complement established diagnostic criteria, clinical findings, and other validated biomarkers when available.

The present findings may have potential clinical implications for routine antenatal care. Because complete blood count examinations are commonly performed during pregnancy, NLR may provide additional information regarding systemic inflammatory status without requiring additional laboratory testing or substantial healthcare costs. Nevertheless, its clinical utility should be interpreted cautiously, particularly because NLR may be influenced by factors other than preeclampsia. Further studies are therefore needed to establish appropriate reference values and determine whether NLR can provide clinically meaningful additional information beyond established risk factors and diagnostic parameters.

Several limitations should be considered when interpreting the findings of this study. First, the cross-sectional design precludes determination of temporal or causal relationships between hematological parameters and preeclampsia. Therefore, the observed differences should be interpreted as between-group differences rather than evidence of predictive ability. Second, the study was conducted at a single hospital with a relatively small sample size, which may limit the external validity and generalizability of the findings. Third, because secondary medical record data were used, adjustment for potential confounding variables, such as body mass index, parity, chronic hypertension, and other maternal characteristics, could not be performed, leaving the possibility of residual confounding. In addition, the use of non-probability sampling and retrospective medical records may have introduced selection bias and further limited the generalizability of the findings to the broader obstetric population.

Overall, the present study demonstrated that women with preeclampsia had significantly higher NLR values than women with normal pregnancies, whereas MPV values did not differ significantly between the two groups. These findings suggest that NLR may better reflect the systemic inflammatory response associated with preeclampsia than MPV and may serve as a practical adjunctive hematological marker in routine clinical assessment. However,

further multicenter prospective studies with larger sample sizes, adjustment for potential confounding variables, and multivariable analyses are warranted to validate these findings and determine the independent clinical value of NLR and MPV in preeclampsia.

CONCLUSION

Women with preeclampsia had significantly higher neutrophil-to-lymphocyte ratio (NLR) values than women with normal pregnancies, whereas mean platelet volume (MPV) values did not differ significantly between the two groups. These findings suggest that NLR may better reflect the systemic inflammatory response associated with preeclampsia than MPV. Further prospective multicenter studies with larger sample sizes and adjustment for potential confounding factors are warranted to validate these findings and clarify the independent clinical value of NLR and MPV in preeclampsia.

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