DOI: 10.14744/cm.2025.63935
Comprehensive Medicine 2025;17(2):124-129

RESEARCH ARTICLE

Prognostic Value of Systemic Immune-inflammation Index in Early- and Late-Onset Preeclampsia

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ABSTRACT

Objective: The study aimed to determine the role of the systemic immune-inflammation index (SII) on prognosis and its cut-off value in early- and late-onset preeclampsia.

Materials and Methods: The retrospectively designed study was conducted with 195 women diagnosed with preeclampsia. The study group was divided into 92 patients with early-onset preeclampsia and 103 patients with late-onset preeclampsia. Demographic and clinical data; leukocyte, platelet, neutrophil, monocyte, and lymphocyte values; neutrophil-lymphocyte ratio (NLR); platelet-lymphocyte ratio (PLR); monocyte-lymphocyte ratio (MLR); and systemic immune-inflammation index (SII) parameters were recorded. Perinatal mortality was considered an unfavorable prognostic criterion, and its relationship with inflammatory markers was compared in two groups according to the groups' prognosis.

Results: The parameters of leukocyte, platelet, monocyte, neutrophil, lymphocyte, MLR, NLR, PLR, and SII measured at the time of diagnosis were not significant in early- and late-onset preeclampsia. However, PLR and SII values were significantly higher in the non-survivor group of early-onset preeclampsia (p=0.04, p=0.045, respectively). The ROC curve analyzed that the cut-off point for SII ≥1050.8 was 0.645 (95% CI: 0.493–0.796), and for PLR ≥146.7, it was 0.648 (0.499–0.797). Multiple logistic regression analysis revealed that SII and PLR were not independent predictors of adverse prognosis (p=0.829, p=0.534).

Conclusion: High PLR and SII values were not statistically significant in predicting perinatal mortality in early-onset preeclampsia. However, with the support of more comprehensive studies and examination of other practical factors as adverse prognostic criteria, the contribution of inflammatory markers in managing early- and late-onset preeclampsia may provide clinicians with a different approach to determining prognosis.

Keywords: Preeclampsia, pregnancy, systemic immune-inflammation index

How to cite this article: Çetin Arslan H, Yılmaz N, Arslan K. Prognostic Value of Systemic Immune-inflammation Index in Early- and Late-Onset Preeclampsia. CM 2025;17(2):124-129

INTRODUCTION

Hypertensive diseases of pregnancy, such as preeclampsia, can lead to severe feto-maternal morbidity and mortality.[1] Preeclampsia is a multisystem disease characterized by hypertension and end-organ dysfunction, with or without proteinuria, after the 20th week of pregnancy.[2] Although its frequency varies, it complicates 3–5% of pregnancies worldwide.[3] Preeclampsia can progress with maternal complications such as renal, hepatic, pulmonary, neurological, and hematological diseases.[4] This process, which can also affect the fetus, can lead to severe conditions such as oligohydramnios, intrauterine growth restriction, preterm birth, and fetal loss.[5] Inadequate trophoblastic invasion, abnormal placentation, and widespread endothelial damage are some known causes of preeclampsia, of which more than one factor is responsible for its pathogenesis. It can also be defined as abnormal placental cytokine release, oxidative stress, free radical release, adaptation of leukocytes and macrophages, complement activation, and endothelial damage due to abnormal inflammatory processes.[6] However, it is assumed that different factors are effective in the pathophysiology of early- and late-onset preeclampsia. Placental factors such as abnormal spiral artery remodeling play an essential role in the pathogenesis of early-onset preeclampsia. In contrast,

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Received date: 13.12.2024
Revised date: 20.03.2025
Accepted date: 26.03.2025
Online date: 28.03.2025

maternal factors are more effective in the pathophysiology of late-onset preeclampsia. In addition, when looking at prognosis, it has been shown that fetal well-being is better in late-onset preeclampsia than in early-onset preeclampsia.[7] It has also been reported that the incidence of late-onset preeclampsia is approximately seven times higher than in early-onset preeclampsia.[8] Early diagnosis of a disease associated with severe morbidity, such as preeclampsia, is extremely valuable for clinicians in clinical follow-up to prolong pregnancy, improve pregnancy outcomes, and control the disease. Based on studies showing that inflammatory markers obtained from peripheral blood samples can contribute to the diagnosis of systemic and local inflammatory diseases, this study aimed to determine the prognostic power of inflammatory markers in early and late preeclampsia.

MATERIALS and METHODS

This retrospective observational study was conducted on patients diagnosed with preeclampsia between January 2022 and September 2023 at the Obstetrics and Gynecology Clinic of the University of Health Sciences, Kanuni Sultan Suleyman Training and Research Hospital. The study was started using the principles of the Helsinki Declaration after the Kanuni Sultan Suleyman Training and Research Hospital Ethics Committee's approval (date: 25.10.2023, number: 152). Informed consent was obtained from all patients.

Preeclampsia was diagnosed based on the ACOG Practice Bulletin, Number 222 guideline (American College of Obstetricians and Gynecologists): (1) in a normotensive pregnant woman, systolic blood pressure is above 140 mmHg and diastolic blood pressure is above 90 mmHg at least twice after the twentieth week of pregnancy, or a single value of ≥160 mmHg or diastolic blood pressures of ≥110 mmHg; and (2) proteinuria (>300 mg/24 hours or dipstick ≥ +1 or spot urine protein/creatinine >0.3); or at least one of the following without proteinuria: (3) end-organ symptoms: (i) thrombocytopenia (platelet count of <100,000/mL); (ii) liver dysfunction (increased transaminases to twice normal); (iii) renal dysfunction (serum creatinine > 1.1 mg/dL); (iiii) pulmonary edema, cerebral or visual symptoms. Preeclampsia is classified into early (before 34 weeks) and late (after 34 weeks) onset preeclampsia, depending on the time of diagnosis.[9]

Criteria for inclusion in the study: (1) over the age of 18; (2) singleton; (3) patients diagnosed with preeclampsia based on the ACOG Practice Bulletin, Number 222 criteria.

The exclusion criteria: (1) multiple pregnancies; (2) fetal genetic diseases; (3) previous pregnancy complicated by preeclampsia; (4) pregnancies obtained by artificial reproductive technology; (5) the presence of any known systemic diseases and infections; (6) smoking, alcohol consumption, and drug usage; (7) missing data.

The study population count was determined with the G*Power 3.1 program. For the t-tests, when the effect size is 0.5, type 1 error α=0.05, study power (1-β) is 0.8, and degrees of freedom (sd) is 1, 158 patients should be included in the study.[10]

A total of 195 preeclampsia patients were included in the study: 92 with early-onset preeclampsia and 103 with late-onset preeclampsia. Age, BMI, gravida, parity, cesarean delivery, emergency delivery, 5th-minute APGAR score, birth weight, need for NICU, and perinatal mortality were recorded as demographic and clinical outcomes. Leukocyte, platelet, monocyte, lymphocyte, and neutrophil values; platelet-lymphocyte ratio (PLR); neutrophil-lymphocyte ratio (NLR); monocyte-lymphocyte ratio (MLR); and systemic immune-inflammation index: neutrophil × platelet/lymphocyte ratio (SII) were obtained from peripheral blood samples during hospitalization. Perinatal mortality was considered an unfavorable prognostic criterion. Patients were divided into two groups based on prognosis: the survivor and the non-survivor. The predictive role of inflammatory parameters was analyzed.

Statistical Analysis

Statistical analyses of the study were performed using SPSS 26.0 (SPSS Inc., Chicago, USA). Descriptive analyses were shown as numbers, percentages, and median values. Normal distribution of variables was assessed using the Shapiro-Wilk test and histogram. The Mann-Whitney U test was used for quantitative variables that did not show normal distribution. Chi-square and Fisher's exact tests were used for categorical variables. Multivariate regression analysis determined that SII and PLR were not independent predictors of adverse prognosis (p=0.829, p=0.534). Logistic regression was analyzed using the odds ratio (OR) and 95% confidence interval (CI). A p-value of less than 0.05 was accepted to indicate a significant result.

RESULTS

Demographic and clinical outcomes of patients diagnosed with early- and late-onset preeclampsia were compared (Table 1). No significant difference was found between the groups regarding age, body mass index, gravida, parity, cesarean section, or emergency delivery. Fetal outcomes such as birth weight and 5th APGAR score were significantly lower in the early-onset group, and the need for NICU and perinatal mortality was higher (p=0.001). The patients with late-on-

Table 1. Distribution of demographic and clinical outcomes in early and late-onset preeclampsia

	All population (n=195)		Early-onset preeclampsia (n=92)		Late-onset preeclampsia (n=103)		p
	n	%	n	%	n	%	
Age (years)	31.5 (18–49)		31 (21–43)		31 (18–49)		0.788
BMI (kg/m²)	27.9±2.9		27.8±3.0		28.1±2.8		0.724
Gravida	2.7±1.9		2.9±2.1		2.6±1.7		0.477
Parity	1.2±1.4		1.0±1.3		1.3±1.5		0.344
5-minute APGAR	8.1±1.5		8.0±1.9		8.5±0.7		<0.001
Birth weight (g)	2431±866		2065±919		2758±667		<0.001
Birth with cesarean	157	80.5	78	84.8	79	76.7	0.155
Emergency delivery	53	27.1	30	32.6	23	22.3	0.107
Need for NICU	73	37.4	48	52.2	25	24.3	<0.001
Perinatal mortality	27	13.8	21	77.8	6	22.2	<0.001

Values are expressed as the mean±SD; the number of patients (n) and percentage. BMI: Body mass index; APGAR: Appearance, pulse, grimace, activity, and respiration; NICU: Neonatal intensive care unit; SD: Standard deviation.

Table 2. Comparative analysis of hematological parameters and related indices in late-onset preeclampsia

	Group survivor (n=97)	Group non-survivor (n=6)	p
Leukocyte, $mm^3 \times 10^3$	10.5±2.4	11.9±3.2	0.297
Platelet, $mm^3 \times 10^3$	265.9±143.7	260.6±78.6	0.805
Monocyte, $\times 10^3/\mu L$	0.5±0.2	0.4±0.3	0.084
Neutrophil, $\times 10^3/\mu L$	7.8±2.2	9.3±2.9	0.098
Lymphocyte, $\times 10^3/\mu L$	2.1±0.6	2.9±1.9	0.386
MLR	0.3±0.8	0.1±0.1	0.07
NLR	5.2±12.2	4.1±2.3	0.642
PLR	182.9±501	102.5±35	0.272
SII	1297.4±2493.6	1021.5±572	0.989

Values are expressed as the mean±SD. MLR: Monocyte-to-lymphocyte ratio; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio SII: Systemic Immune-inflammation index.

set preeclampsia (n=103) were divided into two groups: the survivor group without perinatal mortality (n=97) and the non-survivor group with perinatal mortality (n=6) (Table 2). The groups had no significant difference in leukocytes, platelets, monocytes, neutrophils, lymphocytes, MLR, NLR, PLR, and SII. In addition, cases of early-onset preeclampsia (n=92) were also divided into two groups according to unfavorable prognosis: the survivor group (n=71) and the non-survivor group (n=21) (Table 3). In cases of early-onset preeclampsia, there was no significant difference in the leukocytes, platelets, monocytes, neutrophils, lymphocytes, MLR, and NLR; however, PLR and SII values were significantly higher in the non-survivor group (p=0.04; p=0.045).

In the ROC curve analysis of the predictive values of SII and PLR ratios among biomarkers, the cut-off value for SII was ≥1050.8, AUC (Area Under Curve) was 0.645 (95% CI: 0.493–0.796); the cut-off value for PLR was ≥ 146.7, AUC was 0.648 (95% CI: 0.499–0.797) (Table 4). Multiple logistic regression analysis concluded that SII and PLR were not independent predictors of perinatal mortality (p=0.829 and p=0.534, respectively) (Table 5).

DISCUSSION

This study with patients with early- and late-onset preeclampsia investigated the relationship between inflammatory markers and perinatal mortality. In late-onset pre-

Table 3. Comparative analysis of hematological parameters and related indices in early-onset preeclampsia

	Group survivor (n=71)	Group non-survivor (n=21)	p
Leukocyte,$mm^3 \times 10^3$	11.0± 2.6	10.9±2.1	0.672
Platelet, $mm^3 \times 10^3$	240.3 ± 67.5	249.8±92.9	0.874
Monocyte,$\times 10^3/\mu L$	0.7±0.3	0.5±0.3	0.256
Neutrophil,$\times 10^3/\mu L$	8.2±2.1	8.5±2.1	0.387
Lymphocyte,$\times 10^3/\mu L$	2.0±0.6	1.7±0.7	0.162
MLR	0.3±0.2	0.3±0.1	0.952
NLR	4.4±2.4	6.4±4.8	0.174
PLR	126.3±53.5	179.2±134.8	0.04
SII	1051.3±602.5	1580.4±1340.9	0.045

Values are expressed as the mean±SD. MLR: Monocyte-to-lymphocyte ratio; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; SII: Systemic Immune-inflammation index; SD: Standard deviation.

Table 4. Prognostic performance of PLR and SII for predicting in perinatal mortality

	Cut-off	Sensitivity	Specificity	AUC (95% CI)
PLR	146.7	0.571	0.817	0.648 (0.499–0.797)
SII	1050.8	0.667	0.676	0.645 (0.493–0.796)

AUC: Area under curve; CI: Confidence interval (minimum-maximum).

Table 5. The multivariate logistic regression analysis results.

Variables	OR	95% CI (min-max)	p
PLR	1.006	0.988–1.023	0.534
SII	1.000	0.999–1.002	0.829
Constant	0.107		

OR: Odds ratio.

eclampsia, the association of leukocyte, platelet, monocyte, neutrophil, lymphocyte, MLR, NLR, PLR, and SII values obtained at diagnosis with an adverse prognosis was not found. The need for NICU and perinatal mortality were significantly higher in early-onset preeclampsia. In the early-onset preeclampsia group, PLR and SII values were considerably higher in the non-survivor group, in contrast to other parameters. In the ROC curve analysis, the AUC for PLR was 0.648 (0.499–0.797), and the AUC for SII was 0.645 (0.493–0.796). However, the multivariate regression analysis concluded that SII and PLR are not independent predictors of perinatal mortality.

It is known that preeclampsia is a progressive disease caused by insufficient trophoblastic invasion of the spiral arteries in the placental bed.[11] In pathophysiology, it is assumed that neutrophil activation is responsible for arteriopathy and endothelial damage associated with preeclampsia; however, it is not fully understood whether neutrophil activation causes or results in endothelial damage.[12] A study examining increased neutrophil-endothelial binding and inflammatory responses in preeclampsia also showed that in normotensive pregnant women and preeclampsia, neutrophils and endothelial cells produce IL-6, sIL-6R, and sgp130 in different capacities, thus inducing neutrophil activation of the placenta in preeclampsia.[13] However, this abnormal placentation and the resulting placental insufficiency are associated with adverse outcomes in the mother and fetus and even fetal loss. In the modeling study, it has been shown that pregnancy losses can be prevented by blocking the complement activation regions.[14]

Studies are showing the predictive role of SII and SIRI inflammation markers on mortality and functional limitation in acute ischemic stroke in whom neuroinflammation plays a significant role.[15] It has also been stated that SII, NLR, and PLR can predict poor prognosis in critical traumatic brain injury cases, enable early intervention of patients and planning of aggressive treatments, and thus contribute to reducing mortality.[16] The role of inflammatory markers in determining prognosis has also been investigated in preeclampsia accompanied by an inflammatory process. In a study comparing NLR levels with severe preeclampsia, mild preeclampsia, and healthy pregnant women, it was observed that NLR was

significantly higher in severe preeclampsia, and it was stated that it could be used in clinical practice.[17] In another study investigating the severity of preeclampsia and inflammatory markers such as NLR, PLR, MLR, and SII, MLR was the only significant marker in mild preeclampsia. The cut-off point for MLR was ≥ 3.24, sensitivity was 58%, specificity was 58%, AUC was 0.57 (p=0.04), and other markers were not associated with the severity of preeclampsia.[18] Seyhanli et al.[19] stated that the cut-off value for the predictive value in detecting preeclampsia was 1.5, with 56.2% sensitivity and 55.6% specificity for the first-trimester systemic inflammation response index (SIRI) (p=0.012), and 394.4 for the pan-immune inflammation value (PIV), with 55.2% sensitivity and 55% specificity (p=0.013). However, NLR, PLR, MLR, SII, and β-hCG to PAPP-A were insignificant in detecting preeclampsia. Gezer et al.,[20] otherwise, stated that high NLR and PLR in the 1st trimester predict the following preeclampsia. The cut-off values for NLR and PLR were 3.08 and 126.8, respectively, with 74.6% and 71.8% sensitivity and 70.1% and 72.4% specificity. Unlike the literature, our study analyzed the role of inflammatory markers in predicting adverse prognosis in early- and late-onset preeclampsia. No correlation was found between inflammatory markers and adverse prognosis in late-onset preeclampsia, and only PLR and SII were significant in early-onset cases. The cut-off value for PLR ≥146.7, AUC 0.648 (0.499–0.797), p=0.534; and the cut-off value for SII ≥1050.8, AUC 0.645 (0.493–0.796), p=0.829. PLR and SII were not independent predictors of adverse prognosis in early-onset preeclampsia and had poor prognostic values in logistic regression analysis. Orgul et al.[21] similarly compared the first-trimester CBC values of early (n=21) and late-onset preeclampsia (n=42) and healthy pregnant women (n=123). Increased leukocyte and neutrophil counts in the first trimester were observed to be significant in early-onset preeclampsia. The differences between the groups did not significantly differ in hemoglobin, hematocrit, eosinophils, basophils, monocytes, lymphocytes, and platelets.

The study's retrospective design and the inability to analyze the data independently of risk factors such as systemic diseases, autoimmune diseases, antiphospholipid syndrome, body mass index, and short interbirth interval were considered limitations.

CONCLUSION

In clinical practice, estimating the prognosis of preeclampsia with a peripheral blood sample, which is a cheap and accessible test, is extremely valuable in terms of early diagnosis and long-term prevention of complications. Our study evaluated the ability of inflammatory markers to predict perinatal mortality in early- and late-onset preeclampsia. Assessing the relationship between the systemic immune-inflammation index and other prognostic factors such as the newborn's NICU requirement, APGAR score value, and cord pH value will help clinicians in a complex and difficult-to-manage process such as preeclampsia.

Disclosures

Ethics Committee Approval: The study was approved by the Kanuni Sultan Suleyman Training and Research Hospital Ethics Committee (No: 152, Date: 25/10/2023).

Authorship Contributions: Concept: H.Ç.A., N.Y.; Design: H.Ç.A., K.A.; Supervision: K.A.; Materials: H.Ç.A., N.Y.; Data Collection or Processing: H.Ç.A., N.Y.; Analysis or Interpretation: K.A.; Literature Search: H.Ç.A., K.A.; Writing: H.Ç.A., K.A.; Critical review: N.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

Informed Consent: Written informed consent was obtained from all patients.

Use of AI for Writing Assistance: Artificial intelligence (AI) supported technologies (such as Large Language Models [LLM], chatbots or image generators, ChatGPT) were not used in this study.

Financial Disclosure: The authors declared that this study received no financial support.

Peer-review: Externally peer reviewed.

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