Research Article

The Accuracy of Systemic Inflammatory Indices as Preeclampsia Markers: A Retrospective Study

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Abstract
Objective: This study aimed to assess the association between systemic inflammatory indices, including the systemic immune inflammation index (SII), neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), systemic inflammation response index (SIRI), and pan immune inflammation value (PIV), and the development of preeclampsia.
Methods: A retrospective study was performed at a tertiary healthcare facility from January to June 2025. This study employed a cross sectional design involving 100 samples, divided into two groups: the preeclampsia group (n=50) and the control group (n=50). Hematological parameters and inflammatory indices were compared, with a p-value < 0.05 indicating statistical significance.
Results: Normality tests, receiver operating characteristic (ROC) curve analyses, and correlation tests were conducted to evaluate the systemic immune index (SII), neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), systemic inflammation response index (SIRI), and pan immune inflammation value (PIV). Both the correlation test and ROC curve analyses showed no significant results for any variable ($p > 0.05$), with sensitivity and specificity ranging from 50 – 56% and 36 – 62%, respectively.
Conclusion: The preeclampsia group tended to have higher inflammatory parameters, but the differences were not statistically significant. Future research could include additional inflammatory markers, such as CRP and fibrinogen, and consider confounding factors for more accurate results.

Keywords: Hematological parameters; inflammatory index; preeclampsia

Akurasi Indeks Inflamasi Sistemik Sebagai Penanda Preeklamsia: Sebuah Studi Retrospektif

Abstrak
Tujuan: Penelitian ini bertujuan untuk menilai hubungan antara indeks inflamasi sistemik yaitu *systemic immune-inflammation index* (SII), *neutrophil to lymphocyte ratio* (NLR), *monocyte to lymphocyte ratio* (MLR), *systemic inflammation response index* (SIRI), dan *pan immune inflammation value* (PIV) dengan kejadian preeklampsia.
Metode: Studi restropektif dilakukan di fasilitas kesehatan tersier periode Januari – Juni 2025. Metode penelitian yang digunakan dalam penelitian ini adalah *cross-sectional*. Studi ini meliputi 100 sampel, dibagi menjadi 2 kelompok: grup preeklamsia (n=50) dan kontrol (n=50). Parameter hematologi dan indeks inflamasi dibandingkan untuk dianalisis dengan nilai $p< 0,05$ dianggap signifikan secara statistik.
Hasil: Uji normalitas, *receiver operating characteristics* (ROC) *curve*, dan uji korelasi dilakukan untuk menganalisis *systemic immune index* (SII), *neutrophil to lymphocyte ratio* (NLR), *monocyte to lymphocyte ratio* (MLR), *systemic inflammation response index* (SIRI), dan *pan-immune inflammation value* (PIV). Hasil uji korelasi dan ROC *curve* tidak menunjukkan hasil bermakna pada seluruh variabel ($p> 0,05$) dengan rentang sensitivitas dan spesifisitas masing-masing sebesar 50 – 56% dan 36 – 62%.
Kesimpulan: Kelompok preeklamsia cenderung mempunyai parameter inflamasi lebih tinggi, tetapi tidak berbeda signifikan. Studi berikutnya dapat mengggunakan parameter inflamasi lebih banyak seperti CRP dan fibrinogen, dan menganalisis faktor perancu supaya hasil studi lebih akurat.

Kata kunci: Indeks inflamasi; parameter hematologi; preeklamsia

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Introduction

Preeclampsia is a leading cause of maternal mortality as well as perinatal morbidity and mortality.[1,2] The estimated incidence of preeclampsia ranges from 2% to 10%. The International Society for the Study of Hypertension in Pregnancy (ISSHP) defines preeclampsia as the presence of arterial hypertension accompanied by proteinuria ≥ 300 mg/24 hours after 20 weeks of gestation, or arterial hypertension associated with one or more of the following signs or symptoms: renal failure, hepatic dysfunction, hematological or neurological issues, intrauterine growth restriction (IUGR), or uteroplacental insufficiency. Pregnancy normally involves mild systemic inflammation; however, in preeclampsia, a more significant inflammatory response occurs due to inadequate placentation, which plays a key role in the pathophysiology of preeclampsia.[2-4]

Eclampsia and life-threatening complications such as Hemolysis Elevated Liver Enzymes and Low Platelet Count (HELLP) syndrome and hemorrhage can develop rapidly, often with minimal or no warning signs of preeclampsia. Preeclampsia involves excessive and progressive immune activation, along with increased proinflammatory and antiangiogenic factors in both the intrauterine environment and maternal endothelium, leading to placental dysfunction and maternal complications. Systemic inflammation in preeclampsia also negatively impacts fetal and perinatal outcomes through impaired uteroplacental perfusion and chronic hypoxia. Persistent inflammatory activation hampers placental vascular development and nutrient transport, raising the risk of fetal growth restriction, preterm birth, and low birth weight. The clinical usefulness of systemic immune inflammatory response indices in assessing, diagnosing, and predicting preeclampsia remains uncertain.[1-3,5] Therefore, this study aimed to evaluate the association between systemic inflammatory indices, including the systemic immune inflammation index (SII), neutrophil to lymphocyte ratio (NLR), monocyte to lymphocyte ratio (MLR), systemic inflammation response index (SIRI), and pan immune inflammation value (PIV), and the development of preeclampsia.

Methods

This study was a cross-sectional research conducted among women in their third trimester of pregnancy. Data collection began after obtaining ethical approval from the Ethical Committee of R.T. Notopuro General Hospital, Sidoarjo, with letter number 000.9.2/069/438.5.2.11/2024. Initially, screening was performed using electronic medical records from January to June 2025. A total of 100 participants meeting the inclusion criteria were enrolled and divided into two groups: the preeclampsia group (n = 50) and the control group (n = 50). The inclusion criteria included singleton pregnancies in the third trimester, with or without preeclampsia, and no presence of comorbid conditions such as infections or autoimmune diseases. The exclusion criteria involved incomplete medical records, multiple pregnancies, autoimmune diseases, liver disease, heart disease, renal disease, and infections. The sample size met the minimum calculated requirement using an appropriate formula.

The minimum sample size for this cross-sectional study was determined using the standard formula . A 95% confidence level was used, with a prevalence of 0.13 based on previous data for the cross-sectional study. The margin of error was set at 0.1. Based on this calculation, the minimum required sample size was 43 subjects per group.

The inflammatory markers were calculated as follows: SII: neutrophil count*platelet count/lymphocyte count,

NLR: neutrophil count/lymphocyte count, MLR: monocyte count/lymphocyte count, SIRI: neutrophil count*monocyte count/lymphocyte count, PIV: neutrophil count*platelet count*monocyte count/ lymphocyte count.[3,6] Demographic data and laboratory examination results were recorded by the investigators. Data normality was assessed using the Kolmogorov-Smirnov test. Descriptive analysis was performed on demographic characteristics and presented as mean values for normally distributed continuous variables and median values for abnormally distributed variables, while categorical variables were reported as frequencies and percentages. There were no significant differences in sociodemographic data, including confounding factors such as age, BMI, and gestational age, between groups, indicating the data analysis was valid. Receiver operating characteristic (ROC) curve analysis was conducted to determine the sensitivity, specificity, and statistical significance of each inflammatory index in relation to preeclampsia. Data analysis was performed using the Statistical Package for Social Sciences (SPSS) version 24.0, and a p-value < 0.05 was considered statistically significant.

Result

The average age of participants in the preeclampsia (PE) and control groups was 31.46 and 28.86 years, respectively. Most participants in both groups were multigravida, making up 76% and 65%, respectively. Regarding parity, the PE group was mainly nulliparous (32%), while the control group was mainly parity one (46%).

According to the Asia Pacific BMI classification, most pregnant women in both the PE and control groups were classified as obese, with proportions of 78% and 74%, respectively. In the PE group, the proportion of women with normal BMI was higher than that of women who were overweight (16% vs. 6%), whereas in the control group, the proportion of overweight women was higher than that of women with normal BMI (18% vs. 8%). Most women in the PE group delivered by cesarean section (72%), while most women in the control group delivered vaginally (66%).

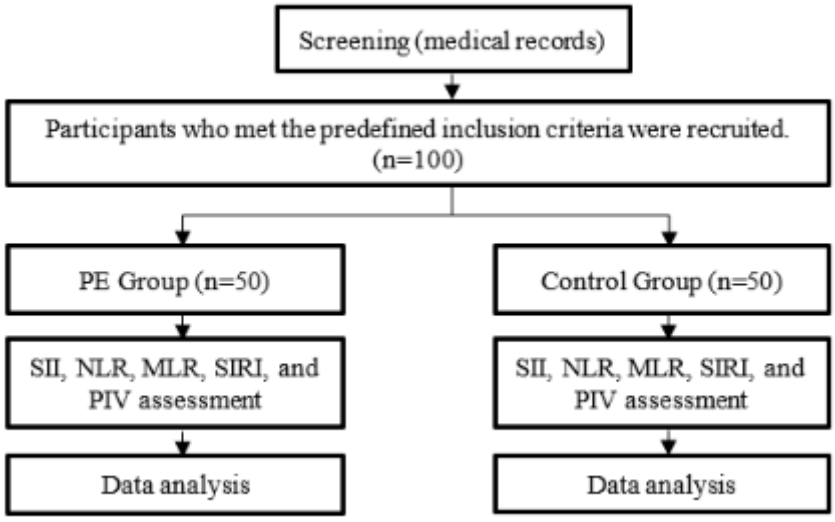


Figure 1 Sample Recruitment

The mean APGAR scores at 0 and 5 minutes were higher in the control group compared to the PE group (7.32 and 9.02 vs. 6.44 and 8.06). The preeclampsia group had lower mean birth weight and gestational age compared to the control group (2,551.6 g vs. 2,829 g; 36.52 weeks vs. 38.36 weeks) (Table 1).

Table 2 shows the average hematological parameters for both groups. In this study, the preeclampsia group had higher mean values for hematological parameters compared to the control group, except for platelet count (263,160/mm³ vs. 263,360/mm³). The comparison of leukocyte, neutrophil, lymphocyte, and monocyte counts between the two groups (Table 2) suggests a tendency toward increased inflammatory status in the preeclampsia group.

The Mann-Whitney U test was used to compare the systemic inflammatory indices between groups. Although the pre-eclampsia group had higher indices than the control group, the differences were not statistically

Table 1 Demographic Characteristics of Respondents

Characteristic	Preeclampsia (n = 50)	Control (n = 50)
Age	31.46 years	28.88 years
Gravida	Primigravida (24%) Multigravida (76%)	Primigravida (34%) Multigravida 66%)
Parity	0 (32%) 1 (22%) 2 (28%) 3 (12%) 4 (6%)	0 (44%) 1 (46%) 2 (8%) 3 (2%) 4 (0)
Body Mass Index (BMI)	Normal (16%) Overweight (6%) Obese (78%)	Normal (8%) Overweight (18%) Obese (74%)
Mode of Delivery	Vaginal delivery (28%) Cesarean section (72%)	Vaginal delivery (66%) Cesarean section (34%)
Gestational Age	36.52 weeks	38.36 weeks
Birth Weight	2,551.6 grams	2,829 grams
APGAR Score	0 min: 6.44 5 mins: 8.06	0 min: 7.32 5 mins: .02

Table 2 The Comparison of Hematological Parameters

Parameter	Preeclampsia (n = 50)	Control (n = 50)
Hemoglobin	11.91 g/dL	11.396 g/dL
Neutrophils	9.4×10^9/L	8.242×10^9/L
Leukocytes	11,525/mm³	10,665/mm³
Lymphocytes	1.55×10^9/L	2.278×10^9/L
Monocytes	0.99×10^9/L	0.65×10^9/L
Platelets	263,160/mm³	263,360/mm³

Table 3 The Comparison of Systemic Inflammatory Indices

Parameter	Preeclampsia (n = 50)	Control (n = 50)	*p-value*
SII	1,534.692 ± 1,315	1,261.071 ± 829	0.363
NLR	7.715 ± 4.519	4.661 ± 2.263	0.770
MLR	0.503 ± 0.591	0.354 ± 0.129	0.893
SIRI	5.041 ± 6.041	3.036 ± 1.825	0.299
PIV	1,399.516 ± 1,708.801	817.62 ± 559.882	0.352

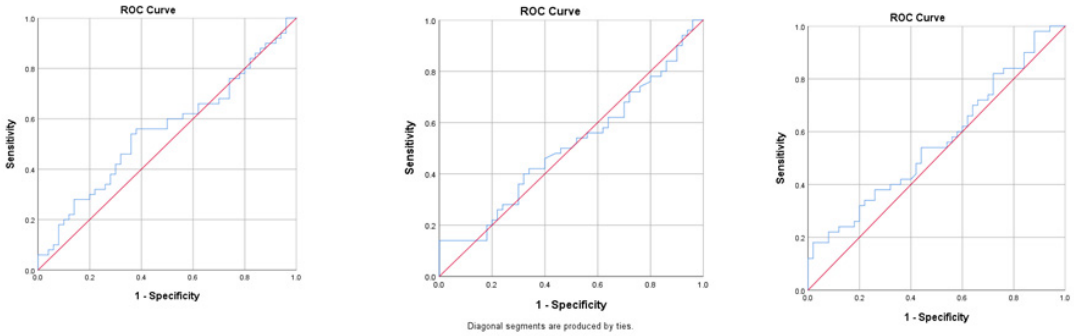


Figure 2 ROC of SII, NLR and MLR

significant ($p > 0.05$) (Table 3).

ROC curve analysis showed that SII, NLR, MLR, SIRI, and PIV were not significantly linked to the occurrence of preeclampsia ($p > 0.05$). According to ROC analysis, the best cut-off value for SII was 1,200, with a sensitivity of 54% and a specificity of 62% (Figure 2).

The optimal cut-off values for NLR, MLR, SIRI, and PIV were 4.08, 0.32, 2.52, and 751.1, respectively (Figures 2-3). The sensitivity and specificity for NLR, MLR, SIRI, and PIV were 50% and 36%, 50% and 50%, 56% and 44%, and 52% and 48%, respectively (Figures 1–2). Correlation analysis showed no significant association between any inflammatory variables and the occurrence of preeclampsia. All analytical results for each inflammatory index are summarized in Table 4.

Logistic regression analysis revealed that age and body mass index were statistically significant factors, with P values of 0.018 and 0.005, respectively. This suggests that differences in age and body mass index between the two groups may affect the values of other variables; therefore, these variables were kept in the logistic regression model. In this analysis, the other inflammatory indicator variables did not have statistically significant P values. This finding aligns with the results of the ROC curve analysis, which also did not show statistically significant P values. Nevertheless, it can be observed that increasing age, BMI, and NLR values are risk factors for preeclampsia. This is demonstrated in the table below (Table 5).

Based on these results, it can be concluded that after controlling for age and body mass index, the inflammatory indicator variables examined in this study were not statistically significantly associated with preeclampsia. However, NLR was the only inflammatory indicator with an Exp (B) value greater than 1, specifically 2.342 (95% CI: 0.961–5.709). This indicates a 2.3-fold

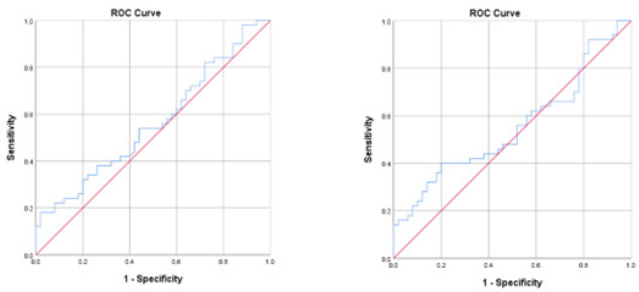


Figure 3 ROC SIRI dan PIV Curve

Table 4 Cut-off Values, Sensitivity, Specificity, AUC and p-values of Each Inflammatory Index

Inflammatory Index	Cut-off	Sensitivity	Specificity	AUC	p-value
SII	1,200	54%	62%	0.447	0.108
NLR	4.08	50%	36%	0.483	0.157
MLR	0.32	50%	50%	0.492	1.000
SIRI	2.52	56%	44%	0.438	1.000
PIV	751.1	52%	48%	0.446	1.000

Table 5 Results of Logistic Regression Analysis for Preeclampsia

Variables	β	Exp (B)	95% CI	P-value
Age	0.098	1.103	1.017 - 1.197	0.018
BMI	0.175	1.191	1.054 - 1.345	0.005
SII	-0.003	0.997	0.993 - 1.000	0.065
NLR	0.851	2.342	0.961 - 5.709	0.061
MLR	-0.437	0.646	0.013 - 33.138	0.828
SIRI	-0.928	0.395	0.107 - 1.460	0.164
PIV	0.004	1.004	0.999 - 1.009	0.082

increased risk of preeclampsia with higher NLR after adjusting for age, BMI, and other inflammatory indicators.

The SII variable had an Exp (B) value of 0.997, which is very close to 1, indicating minimal influence on predicting the risk of preeclampsia. MLR had an Exp (B) value of 0.646, indicating a protective effect against preeclampsia with each increase in MLR. Higher MLR values were associated with a 35.4% reduction in the risk of preeclampsia; however, this finding was not statistically significant (P = 0.828). The inflammatory variable SIRI was also found to be a strong protective factor, with an Exp (B) value of 0.395; however, similar to MLR, this variable was not supported by a statistically significant P value (P = 0.164). The PIV variable showed no meaningful effect on preeclampsia after adjustment for other variables, as evidenced by a very small β and an Exp(B) close to 1.

Thus, among all the inflammatory variables examined, NLR seems to be a relatively reliable risk indicator for preeclampsia, while MLR and SIRI serve as protective factors. However, SII and PIV do not appear to affect the risk of preeclampsia.

Discussion

During early normal pregnancy, fetal trophoblasts invade the maternal spiral

arteries of the uterine decidua, leading to the remodeling of these vessels from narrow, high-resistance arteries into wider, more compliant vessels to ensure proper blood flow, as seen in healthy placentation. In preeclampsia, trophoblast invasion is confined to the superficial parts of the maternal spiral arteries, causing placental hypoperfusion and hypoxia. The second stage of preeclampsia occurs after 20 weeks of gestation, during which placental hypoxia results in placental ischemia, oxidative stress, and the release of placental microparticles into the maternal circulation. This cascade then triggers endothelial activation, the release of coagulation proteins, immune cell activation, and increased production of proinflammatory cytokines, eventually manifesting as the clinical signs and symptoms of preeclampsia. Identifying and managing preeclampsia are essential to safeguarding maternal and fetal health well-being.[3,4]

Preeclampsia has detrimental effects on both the mother and fetus. Although the exact mechanisms behind PE need further study, several inflammatory processes are known to be involved. In normal pregnancy, trophoblast invasion and spiral artery remodeling depend heavily on the maternal immune system to regulate these vital processes. During trophoblast invasion, the decidua lining the uterus, which forms the maternal part of the placenta, is rich in immune cells essential for proper trophoblast migration. These immune cells help ensure successful implantation and regulate invasion to prevent excessive or insufficient invasion. In preeclampsia, immune responses and immune cell and cytokine profiles become dysregulated. Placental ischemia in preeclampsia causes an immune imbalance, leading to chronic inflammation that resembles autoimmune disease. This imbalance is marked by increased proinflammatory immune cells and cytokines and decreased regulatory immune cells and cytokines, fueling uncontrolled chronic inflammation. These immune changes contribute to the overall pathology of preeclampsia, including increased production of reactive oxygen species (ROS), higher endothelin 1 levels, and the formation of B cells that produce autoantibodies against the angiotensin II type 1 receptor (AT1-AA), which are involved in developing hypertension during pregnancy.[3,5] This systemic inflammation shows up as alterations in circulating immune cell profiles, such as neutrophilia, relative lymphopenia, and monocyte activation, which form the basis of composite inflammatory indices like NLR, MLR, SII, SIRI, and PIV.[6-8]

Pregnancy itself is a low-grade inflammatory condition. The inflammatory state in preeclampsia plays a crucial role in causing endothelial injury and microvascular dysfunction. Activated neutrophils and monocytes release reactive oxygen species, proteolytic enzymes, and proinflammatory cytokines, which further increase oxidative stress and damage the endothelium. These processes lead to increased vascular permeability, vasoconstriction, and activation of the coagulation cascade, eventually resulting in hypertension, proteinuria, and involvement of other organs. Notably, renal injury in preeclampsia has been linked to heightened systemic inflammation, emphasizing the pathogenic connection between immune activation and organ-specific damage. Throughout pregnancy, the maternal immune system works to defend both the mother and fetus from pathogens while avoiding rejection of the fetal placental allograft. Paternal major histocompatibility complex (MHC) class I antigens (HLA-C) present on trophoblast surfaces are recognized by antigen-presenting cells (APCs), which then stimulate maternal T helper cells. Once activated, lymphocytes release various pro- and anti-inflammatory cytokines. Th1 cells mainly produce interferon-γ (IFN-γ), along with interleukin-1β (IL-1β), IL-22, IL-12,

IL-15, and tumor necrosis factor-α (TNF-α). Conversely, Th2 cells predominantly secrete IL-10, as well as IL-4, IL-5, IL-6, IL-9, IL-13, and transforming growth factor-β1 (TGF-β1). Th1 cytokines are involved in delayed-type hypersensitivity reactions and cellular immune responses, whereas Th2 cytokines activate B cells and promote humoral immune responses.[4,6]

IL-10 is an immunosuppressive cytokine that primarily inhibits Th1 responses by acting on APCs, promoting T-cell energy, and encouraging regulatory T cells to secrete IL-10 and TGF-β. During a normal first-trimester pregnancy, peripheral blood mononuclear cells mainly produce Th1 cytokines rather than Th2 cytokines. Understanding immunological changes during pregnancy and their effects is crucial, as it may help clinicians and pregnant women reduce the physiological burden of pregnancy and improve fetal outcomes.[9,10]

In this study, no significant link was found between inflammatory markers and the occurrence of preeclampsia; however, the preeclampsia group showed higher levels of inflammatory parameters. This aligns with a systematic review by Black et al., which reported that no inflammatory marker can be reliably used to predict or diagnose preeclampsia. Normal pregnancy has three immunological phases aligned with the trimesters. The first trimester is a proinflammatory phase that supports implantation, placentation, blastocyst adhesion, trophoblast invasion, and uterine tissue remodeling. The second phase is a period of fetal growth marked by an anti-inflammatory state. The final phase involves labor and delivery, which require activation of systemic inflammation. These inflammatory responses are controlled by signaling molecules like cytokines and C-reactive protein, along with cytokines released from blood cells and other tissues. Since this study was conducted in the third trimester, this might explain the absence of a significant link between inflammatory indices and preeclampsia, whereas a study by Seyhanli et al. showed significant associations when conducted during the first trimester, a proinflammatory phase of pregnancy.[4,10,11]

Kapci et al. did not find a significant correlation between systemic inflammatory indices and pre-eclampsia, although pre-eclampsia was associated with higher inflammatory indices. The relationship between NLR and pre-eclampsia remains inconsistent and warrants further research.[12,13] Mensah et al also did not find a connection between systemic inflammatory indices and pre-eclampsia. The conflicting results among studies may be related to the severity of PE, which the present study did not categorize participants by. One study found that NLR and PLR were higher in severe PE cases inthan to mild cases. Sample sizes may also contribute, as other studies have larger samples than this one.[14,15] We controlled for demographic factors such as age and BMI, so they were not confounding factors in our findings.

A study by Imaralu et al. reported similar findings, indicating that the use of inflammatory markers to predict preeclampsia remains limited, although inflammatory markers are elevated and associated with disease severity. However, they used different inflammatory markers, namely high-sensitivity C-reactive protein (hsCRP) and fibrinogen. The dominance of proinflammatory M1 macrophages shifts toward anti-inflammatory M2 macrophages, which persists through the second and third trimesters of pregnancy. In addition to supporting fetal growth, the anti-inflammatory state of the second trimester also helps protect pregnancy from premature uterine contractions. Beyond the M1–M2 balance, T lymphocytes play a key role in maintaining pregnancy. Hormonal changes and placental trophoblast-derived immunomodulatory

molecules promote a shift toward Th2 and regulatory T cells (Treg), which suppress allograft rejection responses through the production of IL-4 and IL-10.

Gao et al. reported that systemic inflammatory biomarkers, including NLR, MLR, PLR, SII, and SIRI, are significantly associated with preeclampsia-related kidney injury, suggesting that these indices may better reflect end-organ involvement and disease progression rather than serve as standalone diagnostic markers for preeclampsia.[6] Preeclampsia (PE) develops in two stages. The first occurs at the maternal-fetal junction, where uteroplacental arterial flow and placental perfusion become inadequate due to insufficient invasion of cytotrophoblasts into the uterine wall and spiral arteries, leading to hypoxia and the release of reactive oxygen species. This contributes to placental dysfunction and oxidative stress. Hypoxia induces inflammation through several mechanisms, including the activation of monocytes and neutrophils. Neutrophils play a crucial role in PE pathogenesis, with their activation triggered by oxidized lipids secreted by the placenta and passing through the intervillous space. Conversely, lymphocyte levels decrease in PE. An increase in neutrophils and a decrease in lymphocytes raise the NLR, but the link between elevated NLR and PE remains inconsistent. Monocytes also play a key role in placental inflammation; their increase alongside decreased lymphocytes results in a higher MLR in PE. The second stage involves activated neutrophils infiltrating maternal vascular tissues, which is associated with maternal systemic inflammation, leading to platelet activation, vasoconstriction, hypertension, endothelial dysfunction, and end-organ ischemia.[6,16,17]

Since PE affects neutrophil, monocyte, thrombocyte, and lymphocyte counts, it also influences other inflammatory markers, including PIV, SII, and SIRI, because SIRI combines neutrophil, monocyte, and lymphocyte counts, reflecting innate immune activation and regulation dysfunction. These markers have been considered indicators for forecasting the presence and severity of preeclampsia.[17,18] This aligns with the findings of the current study, where inflammatory indices tended to be higher in the preeclampsia group but were not significantly linked to the occurrence of preeclampsia itself. This suggests a potential role in risk assessment and prognosis rather than early detection. It may also be influenced by BMI and gestational age, as most participants in both groups were obese and in their last trimester. Janscura et al. found no significant difference in inflammatory markers during the third trimester between the PE and control groups among obese women.[19] It is hypothesized that obese women might have higher levels of anti-inflammatory markers as a compensatory response. This could be an example of an immune-mediated adaptation, where chronic inflammation triggers increased production of anti-inflammatory cytokines production.[20-24]

This study has both strengths and limitations. The strengths include the use of multiple inflammatory indices, such as SII, NLR, MLR, SIRI, and PIV. Although no significant association was found between inflammatory indices and preeclampsia, the preeclampsia group tended to display higher inflammatory parameters than the control group. This finding aligns with a study by Sahit et al. (2025), which reported higher inflammatory indices in preeclampsia during the first trimester and suggested their potential usefulness for predicting more severe preeclampsia in later trimesters, although only the lymphocyte to monocyte ratio (LMR) reached statistical significance. The limitations of this study include a single-center design and the absence of confounding factor analysis. Future studies should include additional inflammatory parameters, such as CRP and fibrinogen, and control for

confounding factors to achieve more accurate results.

Conclusion

Preeclampsia is one of the most common pregnancy-related disorders and a leading cause of maternal death as well as perinatal morbidity and mortality. Pregnancy involves changes in immunological status, and the development of preeclampsia is closely linked to inflammatory processes. Although inflammatory markers such as SII, NLR, MLR, SIRI, and PIV were not significantly associated with the occurrence of preeclampsia, the preeclampsia group tended to show higher inflammatory parameter values. More research is needed to identify reliable markers for diagnosing and predicting preeclampsia, which could help with early management and better outcomes for patients.

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