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Research Article

Systemic Inflammatory Indices in Preterm Preeclampsia Versus Term Preeclampsia and Healthy Pregnancies: A Retrospective Case-Control Study

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Abstract

Aim: This study aimed to investigate whether systemic inflammatory indices derived from first-trimester complete blood counts—namely the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), and pan-immune-inflammation value (PIV)—differ among women with preterm preeclampsia, term preeclampsia, and healthy pregnancies.

Material and Method: In this retrospective case–control study, 197 pregnant women were evaluated and categorized into three groups: preterm preeclampsia (n=39), term preeclampsia (n=59), and healthy controls (n=99). Demographic, perinatal, and hematological data were retrieved from medical records. Inflammatory indices were calculated from complete blood count parameters obtained during the first trimester. Statistical comparisons across groups were performed using one-way ANOVA or Kruskal–Wallis test, with a significance threshold of $p<0.05$.

Results: While neutrophil counts and hemoglobin levels were significantly higher in preeclampsia groups compared to controls ($p=0.001$ and $p<0.001$, respectively), there were no statistically significant differences among groups in terms of NLR ($p=0.063$), PLR ($p=0.750$), SII ($p=0.100$), SIRI ($p=0.110$), or PIV ($p=0.091$). Birth weight, birth length, and Apgar scores were significantly lower in the preterm preeclampsia group ($p<0.001$ for all), reflecting more severe neonatal outcomes.

Conclusion: Despite differences in neutrophil count and hemoglobin concentration, systemic inflammatory indices derived from first-trimester blood counts did not significantly differentiate preterm preeclampsia from term preeclampsia or healthy pregnancies. These findings suggest limited utility of these indices as standalone diagnostic markers in early pregnancy. Future prospective studies incorporating serial measurements and multimodal predictive models are warranted.

Keywords: First-trimester screening, maternal biomarkers, preeclampsia, preterm preeclampsia , systemic inflammation

INTRODUCTION

Preeclampsia is a complex multisystem hypertensive condition distinct to pregnancy, typically presenting beyond 20 weeks of gestation, and defined by the onset of hypertension accompanied by proteinuria or signs of maternal organ disturbance and uteroplacental insufficiency. Affecting roughly 2–8% of pregnancies globally, it continues to be a major contributor to perinatal and maternal mortality and morbidity (1,2). Among its clinical subtypes, preterm preeclampsia—which necessitates delivery before full-term gestation is achieved—constitutes a more severe phenotype, often associated with adverse outcomes such as impaired fetal development, premature placental detachment, and subsequent neonatal intensive care unit hospitalization (3,4).

Despite substantial scientific investigations, the development of preeclampsia is still not fully elucidated. Central to its development is deficient trophoblast invasionand defective spiral artery reorganization, leading

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to placental hypoperfusion and the subsequent maternal systemic release of antiangiogenic and proinflammatory mediators (5,6). These events collectively contribute to oxidative stress, endothelial dysfunction, and systemic maternal inflammation—key processes implicated in disease progression and severity (7). Elevated circulating concentrations of proinflammatory cytokines (interleukin-6 and tumor necrosis factor-alpha) along with neutrophil and monocyte activation, underscore the involvement of immune dysregulation in preeclampsia's immunopathophysiology (8,9).

In light of this inflammatory milieu, Inflammatory blood markers measured through conventional complete blood count assessments have garnered attention as potential indicators of systemic immune activation in pregnancy. Among these, platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR) are the most widely studied due to their availability and cost-effectiveness. These ratios indicate the equilibrium between innate immune components (neutrophils, platelets) and adaptive immune cells (lymphocytes), and may be perturbed in inflammatory states such as preeclampsia (10,11).

Expanding on these parameters, composite indices including the Systemic Inflammation Response Index (SIRI), Systemic Immune-Inflammation Index (SII) and Pan-Immune-Inflammation Value (PIV) have been developed to present a more integrative assessment of the immune-inflammatory axis. SII is calculated as (platelet count × neutrophil count) / lymphocyte count, SIRI as (neutrophil × monocyte) / lymphocyte count, and PIV as (neutrophil × monocyte × platelet) / lymphocyte count (12). These indices have demonstrated clinical utility in predicting disease severity and prognosis across a range of inflammatory and neoplastic conditions, including sepsis, cardiovascular disease, and malignancy (13,14).

In obstetric research, the application of these systemic indices for the early prediction or diagnosis of preeclampsia remains limited and yields inconsistent findings. While some studies have reported elevated SII, SIRI, or PIV levels in women who later developed preeclampsia—particularly during the first trimester—others have found no significant associations (15,16). Importantly, few investigations have specifically addressed their diagnostic potential in preterm preeclampsia, a clinically distinct entity with possibly divergent immunologic underpinnings compared to term disease (17,18). This represents a critical gap in the literature, particularly given the higher burden of morbidity associated with early-onset forms.

Accordingly, the present study aimed to evaluate whether systemic inflammatory indices—namely SII, PIV, NLR, PLR and SIRI—differ significantly among pregnant women with preterm preeclampsia, term preeclampsia, and normotensive healthy pregnancies. We hypothesized that these indices, derived from first-trimester complete blood counts, would be significantly elevated in women with preeclampsia—particularly in those with the preterm phenotype—reflecting a heightened early inflammatory state. This investigation therefore aims to contribute to the development of accessible and cost-effective early biomarkers that may support individualized risk stratification and timely intervention in high-risk pregnancies.

MATERIAL AND METHOD

Research Design and Setting

This retrospective case–control study was conducted at Tekirdağ Namık Kemal University Medical Faculty Hospital. Medical records were reviewed for pregnant women who received antenatal care and delivered between January 1, 2021, and August 1, 2023. Ethical approval for the study protocol was obtained from the institutional ethics committee Namık Kemal University Faculty of Medicine and all procedures adhered to the principles of the Declaration of Helsinki.

Participant Group

Research cohort consisted of three groups of pregnant women:

- **Group 1:** Patients diagnosed with preterm preeclampsia, defined as preeclampsia requiring delivery before 34 weeks of gestation.
- **Group 2:** Patients with term preeclampsia, defined as preeclampsia diagnosed and delivered at or after 37 weeks.
- **Group 3 (Control):** Healthy pregnant women with no history of hypertensive or systemic disease, who delivered at term without maternal or fetal complications.

Preeclampsia was defined as new-onset hypertension—systolic blood pressure of 140 mmHg or higher, or diastolic pressure of at least 90 mmHg—emerging after 20 weeks of pregnancy, accompanied by either proteinuria (300 mg or more in a 24-hour urine sample) or clinical signs of maternal organ dysfunction.

Maternal age was among the exclusion criteria below 18 years, multiple gestation, pre-existing hypertension or diabetes mellitus, chronic inflammatory or autoimmune disease, and presence of any acute infection at the time of blood sampling.

Data Collection

Demographic and clinical variables incorporating variables such as maternal age, gravidity, and parity, abortion history, birth weight, birth length, and Apgar scores were retrieved from hospital records. Complete blood count (CBC) results from the first trimester of pregnancy were recorded for all participants. Blood samples were processed with an automated hematology analyzer in the hospital laboratory under standard protocols.

The following hematological parameters were collected:

- Neutrophil count (10^3/μL)
- Lymphocyte count (10^3/μL)
- Monocyte count (10^3/μL)
- Platelet count (10^3/μL)
- Hemoglobin concentration (g/dL)

Based on these values, calculation of inflammatory indices was performed as described below:

- **Neutrophil-to-Lymphocyte Ratio (NLR):** Neutrophil count / Lymphocyte count
- **Platelet-to-Lymphocyte Ratio (PLR):** Platelet count / Lymphocyte count
- **Systemic Immune-Inflammation Index (SII):** (Platelet × Neutrophil) / Lymphocyte
- **Systemic Inflammation Response Index (SIRI):** (Neutrophil × Monocyte) / Lymphocyte
- **Pan-Immune-Inflammation Value (PIV):** (Neutrophil × Monocyte × Platelet) / Lymphocyte

All calculations were performed using values from the same laboratory reports to ensure consistency and reliability.

Sample Size

All suitable patients according to the inclusion criteria within the study period were included. A total of 197 participants were analyzed: 39 in the preterm preeclampsia group, 59 in the term preeclampsia group, and 99 in the control group.

Statistical Analysis

The data were evaluated using SPSS software, version 22.0. To assess the normality of continuous data, the Kolmogorov–Smirnov test was applied. Non-normally distributed variables were described using medians, along with their respective minimum and maximum values. Group comparisons among the three study cohorts were conducted using one-way ANOVA for variables with normal distribution, while the Kruskal–Wallis H test was applied for those without normal distribution. Categorical data were analyzed using the Chi-square test when applicable. A p-value below 0.05 was considered indicative of statistical significance. When significant differences were identified among groups, post-hoc pairwise comparisons were conducted.

RESULTS

A total of 197 pregnant women were participated in the study, comprising 39 cases of preterm preeclampsia, 59 cases of term preeclampsia, and 99 healthy pregnant women as controls.

Demographic features and neonatal outcomes of the three groups are presented in Table 1. Median maternal age was similar across groups: 26 years (range: 17–46) in the preterm preeclampsia group, 26 years (19–45) in the term preeclampsia group, and 27 years (18–44) in the control group (p=0.385). No significant differences were observed among groups in terms of gravidity (p=0.221), parity (p=0.177), or number of previous abortions (p=0.883).

Table 1. Demographic and neonatal characteristics of the study groups.

	Preterm preeclampsia (n=39) Median (min-max)	Preeclampsia (n=59) Median (min-max)	Control (n=99) Median (min-max)	p
Age (years)	26 (17-46)	26 (19-45)	27 (18-44)	0.385
Gravida	2 (1-8)	2 (1-6)	2 (1-8)	0.221
Parita	1 (1-7)	1(1-7)	2 (1-7)	0.177
Abortus	0 (0-2)	0 (0-2)	0 (0-3)	0.883
Weight (gram)	1470 (310-2405)	2257 (550-3980)	3143 (2330-4210)	<0.001
Length (cm)	38 (23-48)	46 (28-53)	49 (42-53)	<0.001
Apgar scores at 1st min	6 (1-8)	8 (0-9)	9 (7-10)	<0.001
Apgar scores at 5st min	8 (1-9)	9 (0-10)	9 (7-10)	<0.001

Neonatal birth weight showed statistically significant differences, with median values of 1470 g (310–2405) in the preterm preeclampsia group, 2257 g (550–3980) in the term preeclampsia group, and 3143 g (2330–4210) in the control group (p<0.001). Similarly, birth length was significantly lower in the preterm preeclampsia group (38 cm, range: 23–48) compared to the term preeclampsia (46 cm, 28–53) and control groups (49 cm, 42–53) (p<0.001). Apgar scores at both the 1st and 5th minutes differed significantly between groups. Median Apgar scores at 1 minute were 6 (range: 1–8), 8 (0–9), and 9 (7–10) in the preterm, term, and control groups, respectively (p<0.001). At 5 minutes, the respective medians were 8 (1–9), 9 (0–10), and 9 (7–10) (p<0.001).

A comparison of blood parameters and derived inflammatory indices is presented in Table 2. Neutrophil counts differed significantly across groups, with medians of 7.2 ×10^3/μL (1.53–15.0), 7.5 ×10^3/μL (3.3–26.2), and 6.3 ×10^3/μL (1.1–13.5) for preterm preeclampsia, term preeclampsia, and controls, respectively (p=0.001).

Table 2. Comparison of hematological parameters and systemic inflammatory indices among groups

	Preterm preeclampsia (n=39) Median (min-max)	Preeclampsia (n=59) Median (min-max)	Control (n=99) Median (min-max)	p
Neutrophil (10^3µL)	7.2 (1.53-15.0)	7.5 (3.3-26.2)	6.3 (1.1 -13.5)	0.001
Lymphocyte (10^3µL)	1.6 (0.67-3.4)	1.81 (0.7-3.4)	1.9 (0.3-3.4)	0.458
Monocyte (10^3µL)	0.53 (0.18-3.3)	0.58 (0.2-9.3)	0.54 (0.06-6.3)	0.474
Hemoglobin (g/dL)	12.2 (9.4 -14.3)	12.3 (8.4-15.7)	11.2 (7.1 -14.5)	<0.001
Platelet (10^3µL)	220 (280-391)	251 (49-578)	236 (35- 431)	0.071
NLR	3.7 (0.94-14.2)	4.1 (1.6-17.6)	3.3 (0.8-17.7)	0.063
PLR	451.3 (42.6-7755.8)	139.1 (24.4-446.5)	135.8 (16.7-703.2)	0.750
SII	804.1 (157.7-4147.5)	1005.1 (204.1-5848.7)	805.8 (96.2-3757.9)	0.100
SIRI	2.22 (0.25-20.8)	2.3 (0.49-49.4)	1.7 (0.16-17.3)	0.110
PIV	451.3 (42.6-7755.9)	599.1 (80.9-17880.4)	445.1 (44.5-5110.7)	0.091

NLR: neutrophil lymphocyte ratio, PLR: platelet lymphocyte ratio, LMR: lymphocyte monocyte ratio, SII: systemic inflammatory index (neutrophilxplatelet/lymphocyte count), SIRI: systemic inflammatory response index (neutrophil×monocyte/lymphocyte count), PIV: pan-immune inflammation value (neutrophil×plateletxmonocyte/lymphocyte count)

No statistically significant differences were observed among groups for lymphocyte counts (p=0.458), monocyte counts (p=0.474), or platelet counts (p=0.071). Hemoglobin levels were significantly different across groups: 12.2 g/dL (9.4–14.3) in preterm preeclampsia, 12.3 g/dL (8.4–15.7) in term preeclampsia, and 11.2 g/dL (7.1–14.5) in the control group (p<0.001).

The median NLR was 3.7 (0.94–14.2) in preterm preeclampsia, 4.1 (1.6–17.6) in term preeclampsia, and 3.3 (0.8–17.7) in controls (p=0.063). PLR showed wide variability but no significant difference among groups: 451.3 (42.6–7755.8), 139.1 (24.4–446.5), and 135.8 (16.7–703.2) (p=0.750).

SII was 804.1 (157.7–4147.5) in preterm preeclampsia, 1005.1 (204.1–5848.7) in term preeclampsia, and 805.8 (96.2–3757.9) in controls (p=0.100). SIRI values were 2.22 (0.25–20.8), 2.30 (0.49–49.4), and 1.70 (0.16–17.3) in the respective groups (p=0.110). PIV was 451.3 (42.6–7755.9), 599.1 (80.9–17880.4), and 445.1 (44.5–5110.7) (p=0.091).

DISCUSSION

The aim of this study was to evaluate whether systemic inflammatory indices derived from routine complete blood counts differ significantly among pregnant women with preterm preeclampsia, term preeclampsia, and normotensive healthy pregnancies. The primary objective was to determine whether early hematologic markers—particularly NLR, PLR, SII, SIRI, and PIV—might serve as accessible diagnostic or risk stratification tools, especially for more severe, early-onset forms of preeclampsia. Our findings showed that although neutrophil counts and hemoglobin levels differed significantly between groups, the composite inflammatory indices did not demonstrate statistically significant intergroup differences. These results suggest limited utility of these markers for distinguishing preterm preeclampsia from either term preeclampsia or healthy pregnancies in a clinical setting.

Our findings regarding elevated neutrophil counts in preeclampsia are consistent with earlier studies, which have demonstrated leukocyte activation and systemic inflammatory responses as central features in preeclamptic pathophysiology (1,5,7). Neutrophils have been shown to contribute to endothelial dysfunction through increased generation of reactive oxygen species, proteases, and inflammatory cytokines (6,8). This proinflammatory profile may explain why neutrophil counts are elevated in both preterm and term preeclampsia, yet it is noteworthy that the derived NLR did not differ significantly. This finding aligns with prior reports that question the sensitivity of NLR as a solitary marker in predicting or diagnosing preeclampsia (10,11).

Similarly, while platelet counts trended lower in preeclampsia—particularly in preterm cases, possibly reflecting platelet activation and consumption—the PLR exhibited high variability and showed no significant differences between groups. Several studies have shown inconsistent associations between PLR and preeclampsia, with some suggesting a potential diagnostic role and others reporting negligible or non-significant differences (10,11,15). These discrepancies may be due to differences in study populations, timing of sample collection, and varying definitions of preeclampsia severity.

More importantly, our study evaluated newer composite indices such as the SII, PIV and SIRI which were hypothesized to provide a broader view of systemic inflammation. Contrary to expectations, none of these indices demonstrated statistically significant differences

between groups. Although the median values of SII and PIV were numerically higher in the term preeclampsia group, the wide ranges and overlapping distributions precluded meaningful discrimination. These findings suggest that the degree of systemic inflammation reflected by these indices may not differ substantially between early- and late-onset preeclampsia, or between preeclamptic and healthy pregnancies, when measured in the first trimester.

Our results partly contrast with a study published recently by Seyhanli et al., which reported significantly elevated first-trimester SIRI and PIV values in women who later developed preeclampsia, suggesting a potential predictive role (15). However, their study combined both preterm and term preeclampsia and focused on predictive rather than diagnostic value. In our study, group comparisons were performed after the diagnosis of preeclampsia, which may explain the lack of observed differences. Furthermore, our sample included only cases with clearly defined preterm and term classifications, allowing for finer subgroup analysis but also potentially reducing statistical power.

Another study by Genç and Erdal found that PIV and SII were elevated in women with early pregnancy losses, suggesting that these indices may reflect a heightened proinflammatory state (16). Their relevance to preeclampsia remains uncertain, as inflammatory patterns in spontaneous abortion and hypertensive disorders of pregnancy may differ significantly. Nevertheless, these findings underscore the need for further exploration of how hematologic indices relate to diverse obstetric outcomes.

While our study did not identify significant diagnostic value for these markers, it is noteworthy that some research in non-obstetric populations—such as oncology and critical illness—has demonstrated strong correlations between systemic inflammatory indices and disease severity or prognosis (13,14). It is possible that in preeclampsia, systemic inflammation occurs on a spectrum and that these indices reflect only part of the complex pathophysiological process. Additionally, the heterogeneity of immune responses among pregnant women, influenced by factors such as parity, age, genetic predisposition, and environmental exposures, may obscure any consistent pattern of hematologic change.

This study has several strengths. It is among the few that directly compare systemic inflammatory indices in preterm versus term preeclampsia, using a well-defined control group and laboratory data obtained in the first trimester. The inclusion of novel indices such as SIRI and PIV adds value to the existing literature, as few studies have examined their role in obstetrics. Furthermore, the use of routine and widely available hematological parameters enhances the potential applicability of the findings in clinical settings.

Nonetheless, several limitations must be acknowledged. The retrospective design limits control over confounding variables, such as undetected subclinical infections or other inflammatory conditions. The study also lacked adjustment for body mass index (BMI), smoking status, and other lifestyle-related inflammatory modifiers. In addition, inflammatory indices were derived from single time-point measurements, which may not capture dynamic changes throughout pregnancy. Serial measurements could provide deeper insights into the temporal evolution of inflammatory profiles in preeclampsia. Moreover, although first-trimester blood samples were prioritized, it is possible that not all cases had uniform timing of laboratory assessment. Finally, the sample size, while adequate for group-level comparisons, may have been underpowered for detecting small effect sizes, particularly for highly variable indices such as PLR and PIV.

In light of these findings, systemic inflammatory indices derived from complete blood counts should be interpreted with caution in the context of preeclampsia diagnosis or stratification. While they offer a cost-effective and accessible approach to evaluating systemic immune activity, their standalone diagnostic accuracy appears limited. Future research should aim to integrate these indices into multifactorial prediction models incorporating biochemical, angiogenic, and Doppler ultrasound parameters to improve early detection and risk classification of preeclampsia. Prospective studies with larger, diverse cohorts and serial measurements are warranted to validate the predictive potential of these indices, particularly in high-risk pregnancies.

CONCLUSION

This study demonstrates that although neutrophil counts and hemoglobin levels were significantly elevated in women with preeclampsia, systemic inflammatory indices derived from first-trimester complete blood counts—namely SII, NLR, SIRI, PLR and PIV—did not differ significantly among patients with preterm preeclampsia, term preeclampsia, and healthy pregnancies. These findings suggest that such indices, when used in isolation, may have limited diagnostic utility in distinguishing early-onset and late-onset preeclampsia during early gestation. Given their accessibility and cost-effectiveness, these hematologic markers may still hold value when integrated into multifactorial prediction models. Future prospective studies incorporating serial measurements, larger sample sizes, and additional biochemical or imaging parameters are warranted to better elucidate their role in early risk stratification of preeclampsia.

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Ethical approval: *The study protocol was approved by the Ethics Committee of Tekirdağ Namık Kemal University Faculty of Medicine (Approval No: 2023.161.09.11, Date: 26.09.2023). All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional or national research committee and with the 1964 Helsinki Declaration and its later amendments.*

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