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Comparison of Systemic Inflammatory Response Markers Derived from Complete Blood Count between Women with Preeclampsia and Healthy Pregnant Women

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Abstract

Background: Preeclampsia is a significant pregnancy complication with an underlying inflammatory basis, typically occurring after the 20th week of gestation and associated with increased maternal and fetal risks. Identifying simple and effective markers for its prediction and monitoring can play an important role in early diagnosis and effective management of the condition.

Objectives: This study aimed to compare systemic inflammatory response markers derived from complete blood count between women with preeclampsia and normotensive pregnant women.

Methods: In this case-control study, 324 pregnant women, including 162 with preeclampsia and 162 healthy pregnant women, were evaluated. The CBC indices were calculated from the first blood sample obtained at hospital admission and compared between the two groups and between the subgroups of non-severe and severe preeclampsia. Statistical analyses were applied to examine correlations by using SPSS version 20.

Results: This study demonstrated statistically significant differences in NLR, PLR, and SII indices between women with preeclampsia and healthy pregnant women, whereas the MLR index did not differ significantly. In the comparison between non-severe and severe preeclampsia subgroups, only NLR and MLR showed statistically significant differences.

Conclusion: The findings of this study suggest that specific inflammatory indices derived from complete blood count may play an important role in the diagnosis and assessment of preeclampsia severity. These indices are simple, accessible, and cost-effective, and can serve as complementary tools alongside standard clinical and laboratory criteria to support early diagnosis and optimal patient management.

Keywords: Preeclampsia, Neutrophil-to-Lymphocyte Ratio (NLR), Platelet-to-Lymphocyte Ratio (PLR), Monocyte-to-Lymphocyte Ratio (MLR), Systemic Immune-Inflammation Index (SII).

1. Background

Preeclampsia is a multisystem hypertensive disorder of pregnancy affecting 2–8% of pregnancies worldwide and significantly contributing to maternal and neonatal morbidity and mortality (1). It is characterized by new-onset hypertension (≥140/90 mmHg) accompanied by proteinuria or other signs of end-organ dysfunction after the twentieth week of gestation (2). Although the precise pathophysiology remains incompletely understood, abnormal placentation and an exaggerated maternal inflammatory response are considered central drivers of the disorder (3). Systemic inflammation is a key feature of preeclampsia, promoting endothelial injury and vascular dysfunction by elevating proinflammatory cytokines and

altering immune cell activation (4). While normal pregnancy induces physiological changes in hematological parameters, including mild leukocytosis and variations in platelet indices (5), these alterations are typically more pronounced in preeclampsia, reflecting underlying inflammatory and thrombotic processes (6). Hematological indices derived from complete blood count, such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII), have gained attention as accessible, noninvasive, and cost-effective markers of systemic inflammation (7, 8). Comparing these indices between preeclamptic and normal pregnancies can provide valuable insight into disease severity (9).

2. Objective

This study aimed to compare systemic inflammatory response markers derived from complete blood count between women with preeclampsia and healthy pregnant women, and to determine significant differences in these indices. Accordingly, the study evaluates these markers in both groups, aiming to elucidate the inflammatory pathways involved in disease pathogenesis and to explore their applications for diagnostic, predictive, and therapeutic monitoring.

3. Methods

Study design and setting

This case-control study was conducted at the Department of Obstetrics and Gynecology, Qaem Hospital, Mashhad, Iran, among pregnant women referred to the hospital between January and December 2024. A total of 324 pregnant women were enrolled and divided into 162 women with preeclampsia, who were diagnosed according to ACOG criteria as the case group (2), and 162 healthy pregnant women with normal blood pressure as the control group, matched for age, gestational age, and body mass index, who were admitted for routine delivery during the same period. The study was approved on 20 December 2023 by the Research Ethics Committee of Mashhad Medical Science, Islamic Azad University (IR.IAU.MSHD.REC.1402.161), and written informed consent was obtained from all participants.

Participants

Patients were selected using convenience sampling. Inclusion criteria were single pregnancies, gestational age ≥20 weeks and exclusion criteria included chronic hypertension, gestational hypertension, pregestational or gestational diabetes, multiple gestation, thyroid disorders, tuberculosis, previous malignancy or vascular disease, active infection, alcohol or tobacco use, premature rupture of membranes, asthma, hematological disorders, anemia (hemoglobin <10 g/dL), use of medications affecting inflammatory markers (corticosteroids, immunosuppressants) and autoimmune diseases.

Clinical definitions

Diagnosis of preeclampsia was based on the American College of Obstetricians and Gynecologists criteria, defined as systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg on at least two occasions with a minimum interval of four hours, along with proteinuria >0.3 g/day after 20 weeks of gestation. Severe preeclampsia was defined as systolic blood pressure ≥160 mm Hg or diastolic blood pressure ≥110 mm Hg, or the presence of at least one of the following: pulmonary edema, microvascular complications, thrombocytopenia, hepatic dysfunction, or central nervous system involvement such as visual disturbances or headache (2). The inflammatory indices

were calculated as follows:

- NLR = Absolute neutrophil count (cells/µL) / Absolute lymphocyte count (cells/µL)
- PLR = Absolute platelet count (cells/µL) / Absolute lymphocyte count (cells/µL)
- MLR = Absolute monocyte count (cells/µL) / Absolute lymphocyte count (cells/µL)
- SII = [Absolute neutrophil count (cells/µL) × Absolute platelet count (cells/µL)] / Absolute lymphocyte count (cells/µL)

Sampling

The sample size was calculated based on the study by Thombare et al. (10), which reported the mean neutrophil-to-lymphocyte ratio in the preeclampsia and control groups, 3.52 ± 1.05 and 3.22 ± 0.88, respectively. Considering a 95% confidence level (α=0.05) and 80% power (β=0.20), the required sample size for each group was calculated using the formula $n = ((S1^2 + S2^2) \times (Z1\text{-}\alpha/2 + Z1\text{-}\beta)^2) / (\bar{X}1 - \bar{X}2)^2$, which resulted in 162 participants per group.

Data Gathering

Demographic and clinical data, including maternal age, body mass index, gestational age, and preeclampsia status, were extracted from hospital records. Laboratory data from the first complete blood count at admission, before delivery, and before any treatment were used to calculate the neutrophil-to-lymphocyte ratio, monocyte-to-lymphocyte ratio, platelet-to-lymphocyte ratio, and systemic immune-inflammation index. The blood pressure of study participants was measured using a calibrated mercury sphygmomanometer with a suitable cuff size, with patients in the sitting posture after 10 minutes of rest. Two readings were collected 4 hours apart, and the average was calculated. Peripheral blood samples were taken in tubes containing EDTA at the time of hospital admission, before receiving any drugs (such as magnesium sulfate or antihypertensives), and before delivery. Complete blood count was performed using an automated hematology analyzer. Data were recorded in a structured checklist and verified by the supervising investigator.

Statistical Analysis

All data were entered into IBM SPSS version 20 for statistical analysis. The normality of continuous variables was examined using the Shapiro–Wilk test, which is recommended for small to moderate sample sizes due to its greater sensitivity than the Kolmogorov–Smirnov test. Because the distributions of the variables significantly deviated from normality ($p < 0.05$), nonparametric methods were used. Specifically, comparisons between groups were performed using the Mann–Whitney U test. Statistical significance was defined as $p < 0.05$. Patient confidentiality was strictly maintained, and no additional interventions or costs were imposed beyond standard care.

4.Result

T A total of 324 pregnant women were studied, including 162 with preeclampsia and 162 healthy pregnant women. As shown in Table 1, information on age, body mass index, and gestational age of the patients was recorded, and there was no significant statistical correlation between age, gestational age, and body mass index within either group of the sample ($p>0.05$). According to Table 2, women with preeclampsia exhibited significantly higher NLR ($p < 0.001$) and SII ($p = 0.009$) than the control group, and PLR was slightly lower in the PE group than in controls ($p = 0.0081$). Also, the MLR index did not differ significantly between groups ($p = 0.58$). Comparison of inflammatory markers by PE severity is shown in Table 3. Within the preeclampsia group, severe cases, as defined in the previous section, exhibited significantly higher NLR ($p = 0.01$) and MLR

(p = 0.01) than non-severe cases. No significant differences were observed for PLR (p = 0.78) or SII (p = 0.61) between severe and non-severe patients. Consequently, the sensitivity and specificity were calculated for diagnosing preeclampsia. The study computed and compared NLR, PLR, MLR, and SII values between case and control groups. The Area Under the Curve (AUC), optimal cut-off points, sensitivity, and specificity values for each marker are reported in Table 5. For diagnosing preeclampsia, the following thresholds were established: NLR >0.495 (55% sensitivity, 84% specificity); PLR >66.02 (93% sensitivity, 30% specificity); MLR >0.16 (80% sensitivity, 30% specificity); and SII >339.07 (94% sensitivity, 13% specificity). The results are summarized in Table 4 and Figures 1–2.

Table 1. Baseline characteristics of participants

Characteristic	Preeclampsia (n=162)Median (IQR)	Controls (n=162)Median (IQR)	p-value
Maternal age (years)	33.5(11.0)	33.55(7.3)	0.17
Gestational age (weeks)	37 (2)	38 (2)	0.061
BMI (kg/m²)	27.0 (5.27)	26 (5.26)	0.051

Table 2. CBC-derived inflammatory indices

Marker	Preeclampsia (n=162)Median (IQR)	Controls (n=162)Median (IQR)	Effect sizes (CI 95%)	p-value
NLR	4.53 (4.0)	3.26 (1.0)	0.47 (0.31, 0.62)	<0.001
MLR	0.25 (0.0)	0.22 (0.0)	0.04 (-0.12, 0.20	0.58
PLR	89.28 (71.0)	104.10 (42.0)	0.21 (0.06, 0.36)	0.0081
SII	735.5 (689.0)	644.4 (316.0)	0.21 (0.06, 0.35)	0.009

Table 3. CBC-derived inflammatory indices in severe and non-severe preeclampsia

Marker	Severe PE (n=79)Median (IQR)	Mild PE (n=83)Median (IQR)	Effect sizes (CI 95%)	p-value
NLR	5.5 (5.0)	4.15 (3.0)	0.2 (0.04, 0.35)	0.01
MLR	0.26 (0.0)	0.21 (0.0)	0.2 (0.04, 0.36)	0.01
PLR	84.47 (78.0)	89.28 (63.0)	0.02 (0.13, 0.18)	0.78
SII	734.7 (984.0)	725.16 (440.0)	0.04 (-0.12,0.19)	0.61

Table 4. Multiple logistic regression analysis

Predictor	Estimate	SE	p-value	OR	95% CI for OR
Age (years)	−0.032	0.022	0.146	0.97	0.93 – 1.01
Gestational age (weeks)	−0.553	0.123	<0.001	0.58	0.45 – 0.73
BMI (kg/m²)	0.121	0.047	0.009	1.13	1.03 – 1.24
NLR	0.251	0.137	0.066	1.29	0.98 – 1.68
PLR	−0.026	0.006	<0.001	0.98	0.96 – 0.99
MLR	−0.926	1.727	0.592	0.40	0.01 – 11.69
SII	0.002	0.001	0.035	1.00	1.00 – 1.00

Table 5. Comparison of inflammatory markers derived from CBC for identifying patients with preeclampsia

Marker	AUC	Cut-off	Sensitivity	Specificity
NLR	0.69	0.495	55%	84%
PLR	0.59	66.02	93%	30%
MLR	0.58	0.16	80%	30%
SII	0.58	339.07	94%	13%

AUC: Area under the receiver operating characteristic curve; NLR: Neutrophil-to-Lymphocyte ratio; MLR: Monocyte-to-lymphocyte ratio; PLR: Platelet-to-Lymphocyte ratio; SII: Systemic Immune Inflammatory Index.

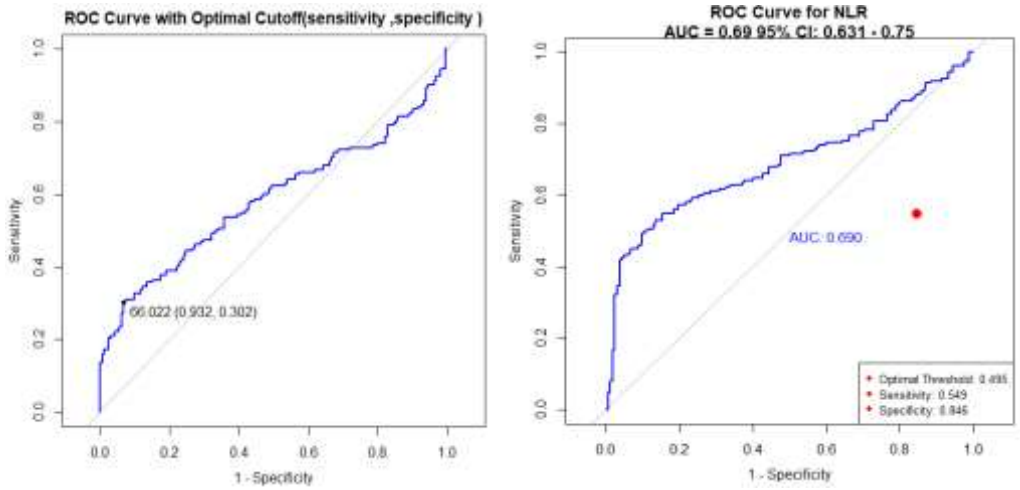

Figure 1. ROC curves for NLR (Left) and PLR (Right) in predicting the diagnosis of preeclampsia

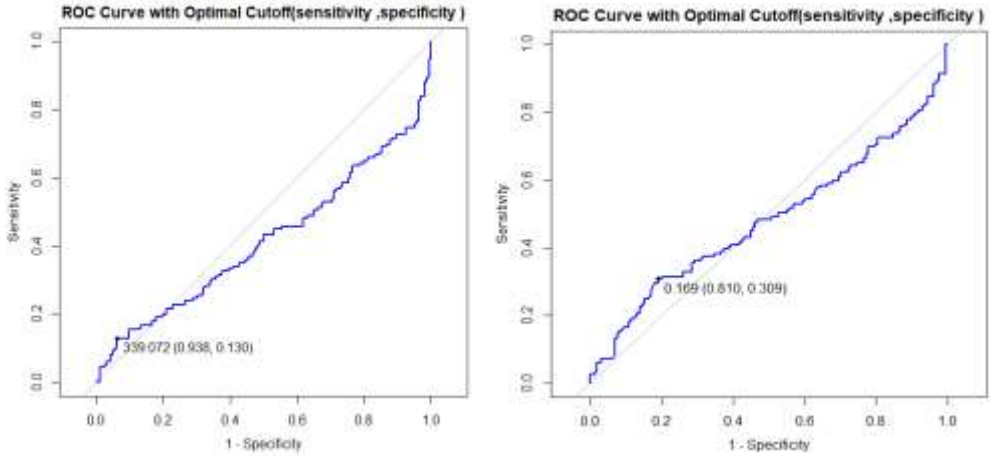

Figure 2. ROC curves for MLR (Left) and SII (Right) in predicting the diagnosis of preeclampsia

5. Discussion

This study aimed to compare systemic inflammatory response markers between women with preeclampsia and healthy pregnant women. The findings show that NLR and SII are significantly higher in patients with preeclampsia, and PLR is lower in the affected group, whereas MLR did not show a significant difference between the two groups. Also, disease severity was associated with increased NLR and MLR, but PLR and SII were not statistically significantly different between severe and non-severe patients. Our findings of increased NLR and SII are consistent with the known pathophysiology of preeclampsia. In this disease, defective placental vasculature leads to relative ischemia and oxidative stress. This condition causes the release of proinflammatory mediators (such as TNF-α, IL-6, and IL-8) from the placenta into the maternal circulation. These inflammatory cytokines activate neutrophils and increase their number, suppress lymphocytes and decrease their number, and damage endothelial cells, leading to vascular dysfunction. Therefore, the increase in the neutrophil-lymphocyte ratio (NLR) well reflects this systemic inflammatory state and the activation of the innate immune system in preeclampsia.

The results of the study by Thombare et al. (2023) on 140 pregnant women are consistent

with the present study in terms of NLR but differ in PLR for patients with preeclampsia compared with healthy controls (3.52±1.05 versus 3.22±0.88, P-value<0.001 for NLR and 98.08±18.27 versus 85.25±12.36, P-value<0.001 for PLR) (10). This agreement regarding NLR likely stems from both studies controlling for demographic variables such as age, gestational age, and body mass index, minimizing confounding effects. In contrast, the difference regarding PLR might be explained by the higher prevalence of thrombocytopenia in our severe preeclampsia subgroup or by differences in sampling timing relative to disease progression. However, a key difference emerged when analyzing subgroups: Thombare et al. found significant differences in NLR and PLR between severe and non-severe preeclampsia, whereas the present study identified significance only in NLR and MLR. This variation could be due to sample size: Thombare's study included 140 participants, while the present study included 324. Therefore, increasing sample size and designing more homogeneous studies may improve the accuracy of inflammatory indices, like NLR and PLR, for assessing preeclampsia severity.

Zhuang et al. (2025) evaluated 749 pregnant women (11). They reported that inflammatory markers NLR and SII were higher in patients with preeclampsia than in the control group (5.16±3.23 vs. 3.99±1.47, P-value<0.001 for NLR; 1165.64±802.78 vs. 877.98±356.45, P-value<0.001 for SII). It agrees with our study. This finding may be due to the increased systemic inflammatory response in preeclampsia. It also indicates the potential of these markers as predictors of the disease and its adverse outcomes.

The study by Khan et al. (2022), with a sample size of 194 patients, and the study by Serin et al. (2016), with a sample size of 107 patients, are both consistent with the results of the present study (9, 12). In both studies, the NLR index in patients with preeclampsia, both severe and non-severe, was reported to be significantly higher than that of healthy pregnant women and to increase with disease severity (4.07±1.25 versus 3.52±1.27 and 4.08±3.7 versus 3.9±2.3, respectively). The present study, with a larger sample size, confirms the findings of both studies and underscores the importance of NLR as an inflammatory index, accessible and predictive of preeclampsia severity. This consistency could be due to the similarity of demographic characteristics such as age and gestational age, which reduces the effect of confounding factors.

In the study by Toptas et al. (2016) with a sample size of 187 participants and the study of Yavuzcan et al. (2014) with a sample size of 101 patients, which contrast with the findings of the present study, no significant difference was reported in the values of NLR and PLR between patients with preeclampsia and healthy pregnant women (Toptas findings were 7.4±5.2 versus 7.2±3.7, P-value =0.7 for NLR and 134.4±64.5 compared to 130.5±86.52, P-value =0.898 for PLR and Yavuzcan findings were 4.04±2.03 versus 3.76±1.28, P-value =0.000 for NLR and 109.81±54.99 versus 127.41±41.17, P-value =0.098 for PLR) (13, 14). Toptas et al. (2016) found that the NLR index did not differ between severe and non-severe preeclampsia (6.8 ± 3.9 versus 5.9 ± 3.5, P-value = 0.314), but the PLR was higher in the severe group (149.8 ± 67.3 versus 122.9 ± 92.0, P-value = 0.024). Yavuzcan, however, showed that NLR and PLR did not have significant diagnostic value in differentiating severe preeclampsia from normal pregnancy, although NLR was higher than in non-pregnant women (4.04±2.03 versus 2.10±0.85, P-value =0.000). In contrast, the present study determined a significant increase in both indices in patients with preeclampsia and a relationship between NLR and disease severity. The difference in results could be due to the smaller sample size of these studies, different sampling times during pregnancy, the limitation of the Yavuzcan study to severe preeclamptic patients, and racial differences of

the participants that may affect inflammatory markers, all of which confirm the methodology, larger sample size, and more reliable results in our study.

A meta-analysis by Kang et al. in 2020, which included 15 studies with 3982 patients (15), reported results consistent with our study on the increase in the mean NLR in patients with preeclampsia compared to healthy pregnant women (3270 women, MD=1.44, 95%CI [1.04,1.83]), as well as the higher ratio in severe preeclampsia compared to the non-severe form (1287 women, MD=1.12, 95%CI [0.69,1.56]). Also, Wang et al. in 2019, examining 367 patients with preeclampsia and 172 healthy pregnant women, showed that the NLR and MLR indices in patients with preeclampsia were significantly higher than those in the control group (4.60±1.83 compared to 3.51±0.82, P-value <0.01 for NLR and 0.39±0.23 versus 0.26±0.76, P-value <0.01 for MLR), and that the NLR also played a role in predicting the severity of the disease (16). This consistency across the two studies, despite the smaller sample size in our study, indicates the validity of the findings and confirms and strengthens previous results.

The present study had several limitations, including a single-center design with convenience sampling, and the case-control design prevents causal inference about the relationship between inflammatory markers and preeclampsia development. Also, blood sampling at admission might not reflect dynamic changes throughout pregnancy; also, we lacked data on specific inflammatory cytokines (e.g., CRP, IL-6). CBC markers were also assessed without assessing maternal or neonatal outcomes. It is recommended that future prospective, multicenter studies with longitudinal sampling throughout pregnancy, measurement of specific inflammatory biomarkers, and assessment of clinical outcomes might be conducted to establish the definitive diagnostic and prognostic utility of these indices. In this study, we did not conduct more advanced statistical analyses, such as factor interaction analyses and complex multivariate modeling. These analyses are necessary to better understand the relationships between inflammatory factors and preeclampsia, but were not included in this study due to sampling and study design limitations. We suggest that these analyses be performed in future studies because they help identify high-risk groups, improve the accuracy of disease prediction, and clarify the complex relationships among factors.

6. Conclusion

The study found that the neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR), and systemic immune-inflammatory index (SII) showed statistically significant differences between the two groups, indicating the roles of inflammatory and coagulation pathways in the pathophysiology of preeclampsia. In contrast, the monocyte-lymphocyte ratio (MLR) did not show a significant difference in the overall comparison. However, when comparing the non-severe and severe preeclampsia subgroups, both NLR and MLR showed significant differences. In particular, the increase in NLR in the severe type could indicate greater severity of the inflammatory response, and MLR, although not significant in the overall comparison, showed potential to predict disease severity or prognosis in subgroups. In conclusion, our findings suggest that CBC-derived inflammatory markers, particularly the NLR, may serve as accessible adjunct tools for identifying and assessing preeclampsia severity. However, Future prospective multicenter studies with longitudinal sampling throughout pregnancy are needed to establish the definitive diagnostic and prognostic utility of these indices.

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Author contributions: Conceptualization: ZM; Methodology and Formal analysis: ZM; Investigation and Data curation: RA and NJ; Writing – Original draft: RA, SM, and NJ; Writing – Review & Editing: ZM, RA, SM.

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